

**“A Retrospective Study of Visual Outcome and Factors
Affecting it in Suprasellar Tumours in a Tertiary Care Centre”**



*Dissertation submitted for the partial fulfilment for the
requirement of the degree of
M.Ch Neurosurgery*

Dr Shah Shreykumar Pravinchandra

M.Ch Neurosurgery Resident

**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND
TECHNOLOGY, THIRUVANANTHAPURAM,
KERALA 695011, INDIA**

2020

DECLARATION

This thesis titled **“A retrospective study of visual outcomes and factors affecting it in suprasellar tumours in a tertiary care center”**

is a consolidated report based on a bonafide study of the period from January 2015 to December 2019, done by me under the Department of Neurosurgery, Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram.

This thesis is submitted to SCTIMST in partial fulfilment of rules and regulations of MCh Neurosurgery examination.




A handwritten signature in blue ink, reading "Shreykumar", with a horizontal line underneath it.

Dr. Shah Shreykumar P,
Department of Neurosurgery,
SCTIMST, Thiruvananthapuram.


29 Aug 2020

CERTIFICATE

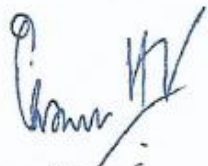
This is to certify that the thesis entitled - "A retrospective study of visual outcome and factors affecting it in suprasellar tumours in a tertiary care center" is a bonafide work of Dr Shah Shreykumar Pravinchandra and was conducted in the Department of Neurosurgery, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram (SCTIMST) under my guidance and supervision.


Dr Krishnakumar (Guide)
Professor
Department of Neurosurgery
SCTIMST
Thiruvananthapuram

Dr. KRISHNAKUMAR .K MS, MCh
Regn No. : 24080 (TCMC)
Professor in Neurosurgery
SCTIMST, Trivandrum-695 011
Kerala, India


Dr Prakash Nair (Co-guide)
Associate Professor
Department of Neurosurgery
SCTIMST
Thiruvananthapuram

CONSULTANT IN NEURO SURGERY
SREE CHITRA TIRUNAL INSTITUTE FOR
MEDICAL SCIENCES AND TECHNOLOGY
THIRUVANANTHAPURAM - 695 011


Dr Easwer H V
Professor and Head
Department of Neurosurgery
SCTIMST
Thiruvananthapuram

Dr. Easwer H.V,
Professor
Department of Neurosurgery
SCTIMST, Thiruvananthapuram

ACKNOWLEDGEMENT

I begin with expressing my undying gratitude to my father Sri. Pravinchandra H Shah and my mother Smt Kusum P Shah., for their everlasting blessing and encouragement.

I would like to thank Prof Dr Easwer H V, Head of the Department of Neurosurgery for his invaluable support encouragement and inspiration which was crucial to complete this study.

I would like to thank my guide Prof. Krishnakumar, Professor of the Department of Neurosurgery, whose support had been invaluable. I am eternally grateful and indebted for his contributions and suggestions, which were of crucial help during the entire work. He will always be a constant source of inspiration to me.

I would also like to thank Prof. Mathew Abraham, Professor of the Department of Neurosurgery for his constant guidance, support, critical appraisal and suggestions without which this study would have not reached this high standard

I owe a deep sense of gratitude to Dr. Prakash Nair, Associate Professor, Department of Neurosurgery, SCTIMST for his invaluable advice, encouragement, and guidance, without which this work would not have been possible. His critical remarks, suggestions, helped me in achieving a high standard of work.

I am deeply indebted to Dr. George Vilanilam, Dr. Jayanand Sudhir, Dr Tobin George and Dr. Ganesh Divakar. I thank them for their constant encouragement and support.

I owe a deep sense of gratitude to all my patients without whom this work would not have been possible.

I am very grateful to my family, especially my wife Dr Meghana Narendran for her constant support and valuable advice

I am also thankful to my seniors and colleagues, Dr Jaypalsinh, Dr Amjad Dr Ninad, Dr Biren, Dr Arun Gowda, Dr Harshvardhan, Dr Arvind Singh, Dr Sanjay HM, Dr Goutham for their constant encouragement and moral support.

ABBREVIATIONS

CSF- Cerebrospinal fluid

PCA- Posterior cerebral artery

CT- Computed Tomography

MRI- Magnetic Resonance imaging

ICA- Internal Cerebral Artery

ACA- Anterior Cerebral artery

MRA- Magnetic resonance angiography

PcoA- Posterior Communicating artery

FSH- Follicular Stimulating Hormone

LH- Luteinizing Hormone

ACTH- Adenocorticotropic Hormone

GH- Growth Hormone

DI- Diabetes Insipidus

VF- Visual field

VA- Visual Acuity

CONTENTS

Title	Page No.
Synopsis	1
Introduction	5
Review of Literature	6
Aim and Objectives	44
Materials and Methods	45
Results	48
Discussion	67
Conclusion	74
Limitations and Recommendations	75
References	76
Annexures	
Annexure A- WHO Classification of visual Impairment	88
Annexure B- Proforma	89
Annexure C- IEC approval letter	91
Annexure D- Plagiarism Report	93

List of Tables

Table No.	Title	Pg. No.
1.	Various pathology and their radiological findings	25
2.	Age distribution among the participants	48
3.	Gender distribution among the participants	48
4.	Visual deficit present in one or both eyes	49
5.	Severity of decline in visual acuity in both eyes	50
6.	Types of visual field defect in both eyes	51
7.	Presence of associated symptoms in the study population	52
8.	Findings of fundus examination	53
9.	Duration of visual decline before surgery	53
10.	Types of surgical approaches	54
11.	Extent of resection of the tumour	55
12.	Various pathology of suprasellar tumours in our study	56
13.	Improvement in visual deficit on discharge and follow up	57
14.	Improvement of visual field at 6 months	59
15.	Improvement in visual acuity and visual field on follow up	59
16.	Visual improvement in study population with blindness	60
17.	Complications after surgery for suprasellar tumours	61
18.	Visual outcome in recurrent disease	62
19.	Variables affecting the visual outcome and their association	63

List of Figures

Fig. No.	Title	Pg. No.
1.	Relation of suprasellar cistern to other cisterns	6
2.	Relation and location of optic chiasma in suprasellar region	9
3.	Types of chiasm	9
4.	Anatomy of circle of Willis	11
5.	Relations of floor of third ventricle to suprasellar cistern	12
6.	Relation of stalk in suprasellar region	13
7.	Case 1 - preoperative and postoperative MRI and visual field testing	40
8.	Case 2 - preoperative and postoperative MRI images with visual field testing	41
9.	Case 3 - preoperative and postoperative MRI and visual field testing	42
10.	Age distribution among the participants	48
11.	Gender distribution among the participants	49
12.	Visual deficit present in one or both eyes	49
13.	Severity of decline in visual acuity in both eyes	50
14.	Types of visual field defect in both eyes	51
15.	Presence of associated symptoms in the study population	52
16.	Findings of fundus examination	53
17.	Duration of visual decline before surgery	54
18.	Types of surgical approaches	55
19.	Extent of resection of the tumour	56
20.	Various pathology of suprasellar tumours in our study	57
21.	Improvement in visual deficit on discharge and follow up	58
22.	Improvement of visual field at 6 months	59
23.	Improvement in visual acuity and visual field on follow up	60
24.	Visual improvement in study population with blindness	61

25.	Complications after surgery for suprasellar tumours	62
26.	Visual outcome in recurrent disease	63



SYNOPSIS

Introduction:

TUMORS of the suprasellar region assume importance because of their anatomical proximity to the anterior visual apparatus and neuroendocrine structures and their delayed clinical presentation. Major modes of presentation are in the form of visual symptoms (visual acuity decline, visual field defects, and involvement of ocular movements), hormonal disturbances, or raised intracranial pressure. The majority of these patients present with visual symptoms only, and are initially treated by ophthalmologists and then referred to neurosurgeons, which further accounts for the delay in initiation of proper treatment. Blurring of vision is the most common ophthalmic presentation in suprasellar tumor. This visual deterioration, due to optic nerve compression is a common indication for decompressive surgery. Tumors in this region consist of pituitary adenomas, meningiomas, craniopharyngiomas, epidermoid cysts, germinomas, chordomas, and so on. Pituitary adenomas are the most common type of suprasellar tumors followed by meningiomas and craniopharyngiomas. Depending on the tumor type, presentation may be acute (for example, pituitary apoplexy) or delayed (for example, epidermoid meningiomas). A few of these patients present with blindness, which may be uniocular or binocular, and if left untreated may progress to binocular blindness. Blindness is usually caused by optic nerve atrophy due to tumor compression, which is generally regarded as irreversible. Suprasellar tumors have a close relationship with anterior visual apparatus, arteries of anterior circulation and neuroendocrine structures. This anatomical proximity makes the safe surgical resection difficult. There is a growing body of literature describing visual outcomes following optic nerve decompression. Both the transphenoidal and transcranial approaches show improvement in visual acuity when compared to preoperative assessment. Although abundant data exist in western literature regarding deterioration in visual function with suprasellar lesions and its improvement following surgery, little information is available regarding the same in Indian population. The rationale of this study is two folds: Firstly to provide local data regarding postoperative visual outcome in patients with suprasellar tumors with preexisting preoperative visual deficit and secondly, to provide data in order to help counsel patients and their caretakers preoperatively to aid decision-making and set expectations.

Aim and Objectives:

To study: Post-operative visual status in patients with suprasellar tumors with pre-existing

preoperative corrected visual deficit after surgical resection who underwent Transcranial or Transsphenoidal surgery.

Objectives:

1. To study the clinical profile among adults with preoperative visual deficits and underwent surgical excision of suprasellar tumour.
2. To evaluate the visual profile on admission, at the time of discharge and on follow up till six months of surgery and operative procedures.
3. To study the predictive factors on visual outcome after surgery.

Study Period:

This study was carried out from January 2015 to December 2019

Study Design:

All patients with suprasellar tumors with either unocular or binocular blindness (no perception of light) at admission, who underwent surgical removal of the tumor in the Department of Neurosurgery between January 2015 and December 2019 with 6 months follow up data were included in this study. The records of these patients, including their case files and outpatient follow-up files were retrospectively analyzed. Data regarding age; sex; duration of symptoms; mode of presentation; and visual acuity and visual field status at admission, discharge, and follow-up were collected from the files. Visual acuity was recorded using the Snellen chart. Visual field charting was performed using Goldman perimetry. Imaging investigations done at that time included CT scanning, MR imaging, or both. The patients underwent either transcranial or transsphenoidal surgery depending on the tumor type, tumor location, and imaging findings. Visual improvement after surgery was defined as when the patient improved to perception of light or better vision or better visual field at discharge from the hospital and on follow up. The multifactorial effect of variables such as age, sex, duration of visual decline, duration of blindness, operative findings regarding consistency of tumor, extent of resection, recurrent disease and histopathology was studied on the visual outcome.

Results:

In our study, patient age ranged from 15-60 years (mean age 38.4 ± 7.46 years). The male to female ratio was (1.01) showing slight male predominance. Pituitary adenoma was the commonest tumour seen in 284 patients (65.3%) followed by craniopharyngioma in 74(17%), and

meningioma in 61 patients (14%). The most common associated symptom with visual decline was headache seen in 60.91 %. In the present study, the patients with suprasellar tumors, presented with impaired vision including decline in visual acuity and visual field in both eyes seen in 373 patients (85.75%) than unioocular seen in 62 patients (14.25%). 63% patients in our study had normal to mild impairment of visual acuity in accordance with WHO classification for visual impairment and 88.51 % had moderate to severe field defects showing that visual fields are more affected than acuity in suprasellar lesion. Normal or minimal visual field deficit was present in just 11.49% patients. The most common field defect in our study was hemianopia and included unilateral or bilateral hemifield defect on nasal or temporal or both sides in both eyes. Out of these bitemporal hemianopia was the most common seen in 195 patients. In our study a total of 119 eyes (14%) were blind (defined as visual acuity worse than 3/60 according to WHO classification) preoperatively, out of which 45 eyes (37.8 %) improved. Out of 435 patients on follow up at 6 months visual improvement was seen in 397 (91.3%). Among 397 improved, patient's visual acuity improved in 75.31 % and visual field in 97.48% of the total improvement seen. Out of the 36 patients who had worsened initially 30 improved and 6 (1.4%) remained the same. Out of 136 with unchanged vision 104 improved and 32 (7.4%) remained the same. Visual improvement was present in 93.8% of patients who underwent transsphenoidal operation, while improvement was 86.01% in patients who were operated with transcranial approach (p-value <0.001). In our group of patients, who had symptoms for less than 12 months, a postoperative visual acuity improvement was noted in 93.8% of the cases. 28.4% of those patients who had symptoms for more than 12 months showed no improvement showing the importance of early diagnosis and surgery. In our study 95.4% patients who had gross total excision had significant improvement in visual outcomes whereas 16 (4.6%) patients did not show vision improvement. Bivariate analysis revealed the following to be statistically significant favourable outcome factors: duration of blindness < 12 months, surgical approach, extent of resection, preoperative visual deficit, recurrent disease and tumor histopathology (pituitary adenoma compared with craniopharyngioma or meningioma). Post-operative visual status was not statistically related to age and sex of the patients

Limitations:

It is a retrospective study. Evaluation of visual acuity and visual field separately may not be uniform and there is a need for uniform evaluation as these are the main factors affecting the visual outcome

Conclusion:

We concluded that patients with suprasellar tumors with pre-existing preoperative visual deficit experienced significant improvement of vision after surgery. Significant factors affecting the visual outcome were preoperative visual deficit, duration of blindness < 12 months, surgical approach, extent of resection, recurrent disease and tumor histopathology. Severe visual deficit or blindness should not be considered as a negative or deterrent factor while considering surgical decompression

INTRODUCTION

TUMOURS of suprasellar region are important because they lie in close proximity to visual apparatus and neuroendocrine structures. Coupled with their anatomical location is their delayed clinical presentation. These tumours have major modes of presentation in the form of visual symptoms (visual acuity decline, defect in the visual field and involvement of ocular movements), hormonal disturbances, or raised intracranial pressure. When their size increases, they can cause hydrocephalus by obstructing the Foramen of Monro. The majority of these patients present with visual symptoms only, and are initially treated by ophthalmologists and then referred to neurosurgeons, which further accounts for the delay in initiation of proper treatment.¹ Blurring of vision is one of the most common complaint of visual pathway in suprasellar tumours.² This visual deterioration, due to optic nerve compression is common indication for surgery. Tumours in this region consist of pituitary adenomas, meningiomas, craniopharyngiomas, epidermoid cysts, germinomas, chordomas, and so on. Commonest tumour is pituitary adenomas in suprasellar region followed by meningiomas and craniopharyngiomas.^{3,4} Depending on the tumour type, presentation may be acute (for example, pituitary apoplexy) or delayed (for example, epidermoid meningiomas). A few of these patients present with blindness, which may be uniocular or binocular, and if left untreated may progress to binocular blindness. Blindness is because of optic nerve atrophy due to tumour compression and is regarded as irreversible. Suprasellar lesions have close relation with anterior visual pathway, anterior circulation and endocrine structures^{3,5} making safe surgical resection difficult. There is growing body of literature assessing visual outcomes following surgery.^{3,5,6} Bulters et al reported improved visual acuity following optic nerve decompression.⁷ Trans-sphenoidal approach is ideal for midline suprasellar tumours. It gives direct access and less time consuming than transcranial operation. However transcranial approach is indicated if tumours are having significant extension into middle or anterior cranial fossa allowing better access to these lesions. Both surgical approaches show improvement in vision when compared to preoperative assessment.^{8,9} Abundant data exist in western region about deterioration in visual function with suprasellar tumours and the improvement following surgery. There is little information available in Indian population about the same. Therefore, rationale of doing this study is two folds:

- 1) To provide data regarding postoperative visual outcome in patients with suprasellar lesions with preoperative visual deficit
- 2) To provide data to help counsel patients and their relatives preoperatively to help decision-making and set expectations.

REVIEW OF LITERATURE

Anatomy

Suprasellar cistern is related to Sylvian, interhemispheric cistern, crural cistern, and interpeduncular cisterns.¹⁰

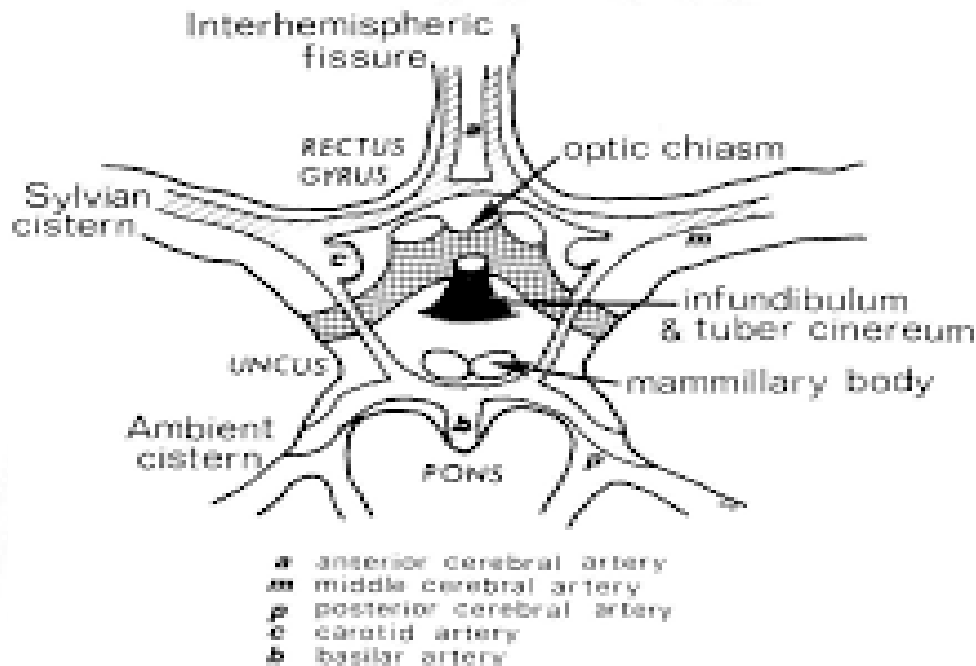


Figure 1 - Relation of suprasellar cistern to other cisterns

Contents of suprasellar cistern are:

- 1- circle of Willis
- 2- optic chiasm
- 3- hypothalamus
- 4- pituitary stalk.^{11,12}

Lilliquist membrane separates the suprasellar cistern from interpeduncular cistern,¹³ and due to multiple small perforations, it allows flow of CSF between them.¹⁴ Boundaries of the cisternal space are:

- 1- Anterior- gyrus rectus with interhemispheric fissure
- 2- Lateral- uncus and amygdala
- 3- Posterior- pons and cerebral peduncles
- 4- Inferior- diaphragm sella, sella turcica with pituitary gland

5- Superior-anterior perforated substance and floor of third ventricle.^{12,15}

Anterior- The gyrus rectus lies medial to olfactory sulci near the olfactory tracts in the medial basifrontal region. The olfactory tracts divide into lateral stria, medial olfactory stria forming the olfactory trigones. The base of this trigone is made by anterior perforated substance through which striate arteries pass.^{13, 16}

Lateral- The uncus and amygdaloid complex lie lateral to suprasellar cistern. A lateral concavity is produced in margin of suprasellar cistern by uncus.^{12,17, 18} Vessels in close to uncus:

- 1) posterior communicating artery,
- 2) anterior choroidal artery
- 3) basal vein of Rosenthal.

Uncal herniation in presence of increased intracranial pressure compresses these structures and causes important clinical features.¹⁹ Superior and posterior to uncus lies inferior choroidal point through which anterior choroidal artery enters temporal horn.²⁰ The choroidal fissure incorporates choroid plexus with choroidal vessels.

Medially- Sylvian fissure forms the anterolateral recess of the suprasellar region.¹⁶ It contains:

- a) first segment of middle cerebral artery courses towards the Sylvian fissure through this cistern towards limen insula.
- b) Striatal branches (medial, lateral, and intermediate)- These arborize to 50 branches and enter anterior perforated substance.¹⁶
- c) Deep middle cerebral vein draining into basal vein of Rosenthal.

Posterolateral- The ambient cistern which opens anteriorly into crural cistern.²⁰ It contains:

- a) posterior cerebral artery
- b) Anterior Choroidal artery
- c) Basal Vein of Rosenthal.²⁰
- d) Perforators from Posterior Communicating Artery, P1, P2 segments of the PCA.^{21,22}
- e) The distal basilar artery and bifurcation in the interpeduncular. Perforating branches from basilar tip supply midbrain.^{21,23}
- f) The oculomotor nerve traversing between posterior cerebral and superior cerebellar arteries and enters the suprasellar cistern en-route to the cavernous sinus. ^{23,24}

The cerebral peduncles which lie along the posterolateral borders contain the pyramidal tracts which are efferent fibre to spinal cord, substantia nigra and the red nuclei. ²⁵

Contents:

THE OPTIC CHIASM

a) lies centrally within the cistern.

b) The axis is at an angle 45 degrees from antero-inferior to postero-superior.^{26,27}

c) Types of chiasma:

1- Prefixed – Chiasma lies anterior to tuberculum sella. Present in 9%.

2- Post fixed – Chiasma lies over the dorsum sella.²⁸

d) In 70% lies it inferior to the A1 segment-Anterior Communicating Artery junction and 30% anteriorly over optic nerves²⁹

Blood supply to chiasma- Arterial supply:

a) superior group - from anterior cerebral artery, Anterior Communicating Artery. The superior group supplies the lateral portions of the chiasm.

b) inferior group from basilar artery, posterior communicating artery, posterior cerebral artery, and supraclinoid internal carotid arteries and they supply the entire chiasm along with decussating fibres

Optic nerves and tracts receive tributaries from both the anterior and posterior circulation groups.^{22,30,31}

Venous drainage- a) superior surface is drained anterior cerebral veins. b) inferior chiasm is drained by basal vein of Rosenthal.¹⁰

Radiology-Both CT and MRI demonstrate chiasma. On axial sections optic nerves with chiasm appear as U-shaped structure and on higher sections appear as boomerang-shaped structure. Optic recess causes a low density in the central region in optic chiasm.³² The structures present in floor of third ventricle appear posterior to chiasm. Normal range of dimensions of the chiasm 9 to 18 mm transversely (average 15 mm) x 4 to 9 mm anterior- posterior (average 8 mm) x 3 to 6 mm in thickness (average 4 mm).^{10, 32}

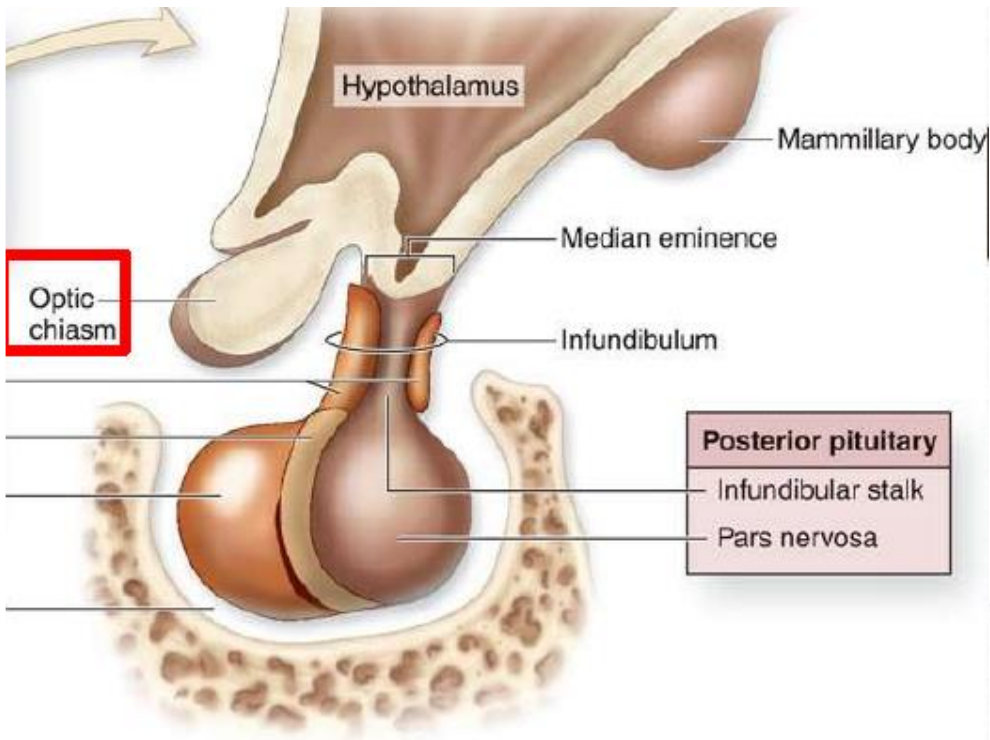


Figure 2- Relation and location of optic chiasma in suprasellar region

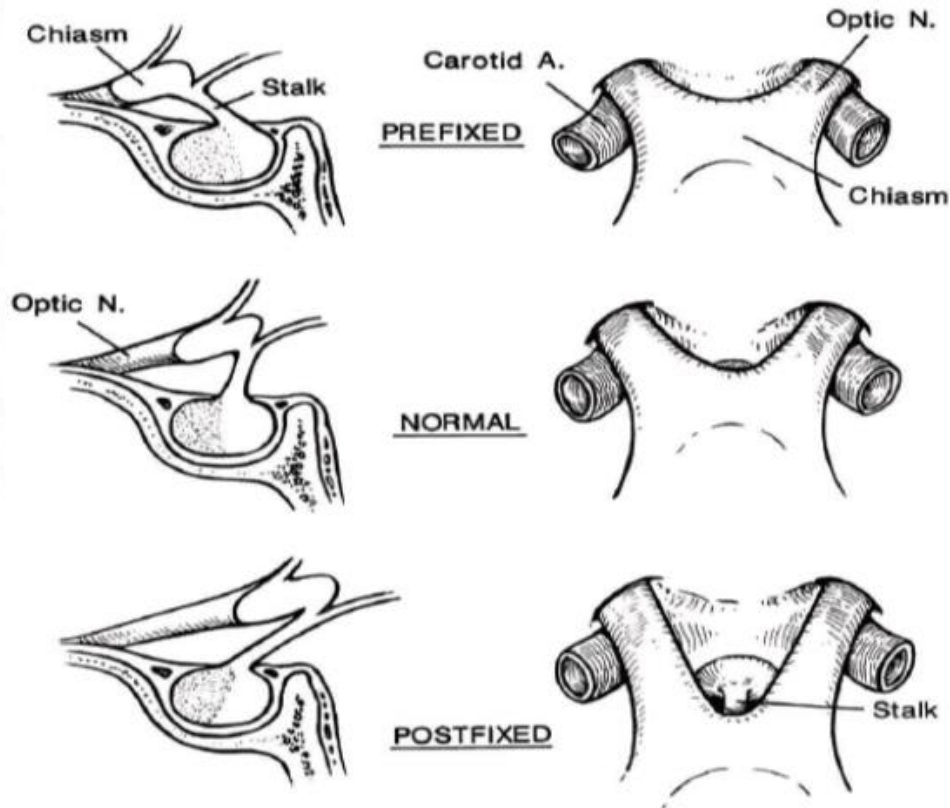


Figure 3 - Types of chiasm

CIRCLE OF WILLIS

The vascular ring in suprasellar cistern bridging both anterior circulations and anterior with posterior circulations (ICA and vertebral artery circulations) is known as Circle of Willis. Circle of Willis is composed of:

- a) Anterior Communicating Artery,
- b) A1 segment of the ACA,
- c) Posterior Communicating Artery,
- d) P1 segment of PCA.

20% of individuals have normal complete circle of Willis.¹⁹ Others may have variations like:

- 1) hypoplasia or aplasia
- 2) Duplications
- 3) Fenestrations
- 4) infundibular widening
- 5) "fetal" vessels.^{19,31}

Anterior communicating artery-

- a) It is the anterior most component.²⁹
- b) It connects first segment of right and left ACAs.
- c) A normal Anterior communicating artery complex is defined when the communicating artery connects right and left A1 segments of equal size and they should be sufficient to allow cross circulation.²⁹
- d) It is defined as hypoplastic if the diameter < 1.0 mm - 1.5 mm.^{29,31} Hypoplasia of A1 segment is related with high frequency of Anterior Communicating Artery aneurysms.
- e) Duplicated anterior communicating artery can be present in 14 to 30% of individuals,^{29,31}
- f) Anterior Communicating Artery may be rarely absent: as studied by found by Wollschlaeger.³¹

Posterior communicating artery-

- a) Connects the supraclinoid Internal Cerebral artery to the P1-P2 junction of Posterior Cerebral artery.
- b) They are nearly equal but smaller than the main vessels.³¹
- c) They may be aplastic or hypoplastic in 2% individuals.³¹
- d) Fetal circulation is defined as large Posterior Communicating Artery with same diameter as P2 segment and hypoplastic P1 segment wherein blood supply to the PCA territory is from the ICA.
- e) Other abnormalities are infundibular widening at its origin seen in around 13% of cases.³¹

Circle of Willis is investigated by conventional angiography, CT angiography and MRI with MRA. On CT angiography it is seen enhancing vascular ring in suprasellar cistern.³³ 3D CT of circle of Willis has been described.³⁴ Excellent anatomic delineation of circle of Willis with phase contrast MRA techniques,²⁵⁻³⁹ Conventional angiography remains "gold standard" for assessing it.

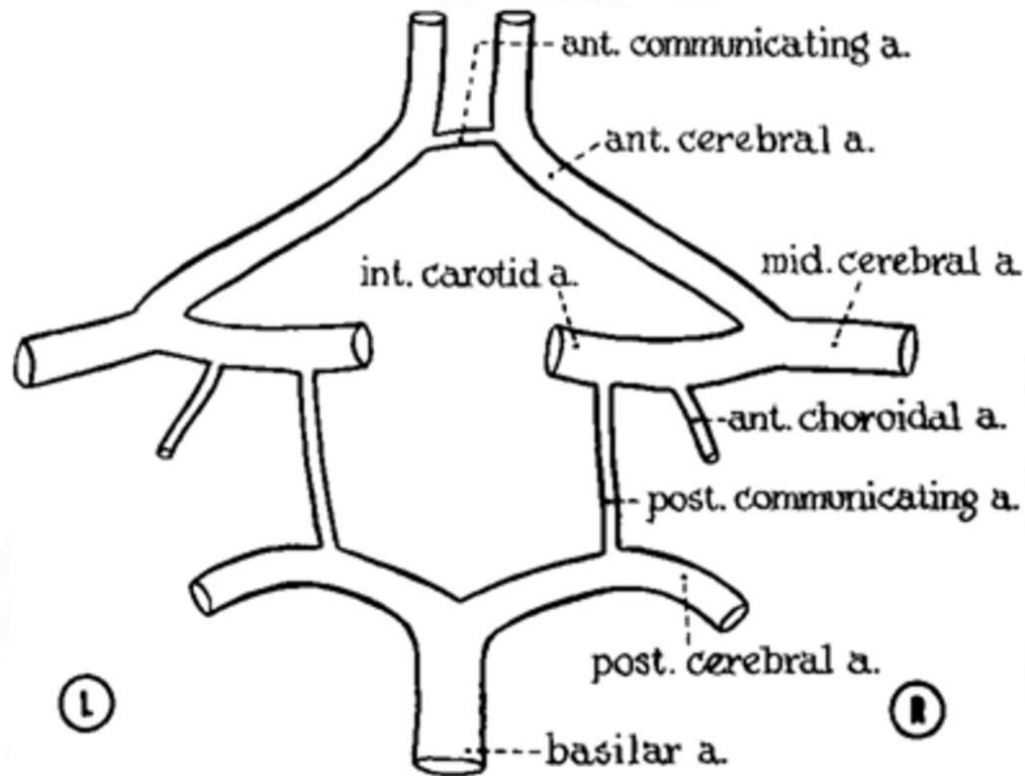


Figure 4- Anatomy of circle of Willis

FLOOR OF THIRD VENTRICLE

Roof of suprasellar cistern is made by floor of third ventricle. From anterior to posterior, structures present in the roof are:

- a) the optic recess- invagination of anterior part of the floor behind the lamina terminalis
- b) Infundibulum- Funnel shaped structure that lies posterior to chiasm anterior to tuber cinereum and axons innervate posterior pituitary gland.²¹
- c) tuber cinereum- Prominence of grey matter which merges anteriorly with infundibulum to form median eminence.⁴⁰
- d) mammillary bodies- Paired rounded structures are located lateral to the midline posterior to tuber cinereum.²¹ Forniceal tracts terminate into these structures and are thus related to limbic system and therefore may be implicated in memory, but this conclusion is not proven yet.⁴¹
- e) Posterior perforated substance- Gray matter substance located behind the mammillary bodies

and anterior to cerebral peduncles. The anterior thalamoperforators from posterior segment of Posterior communicating artery with posterior thalamoperforators from the P1 segment enter this grey matter and supply structures in the floor and lateral walls of third ventricle including the thalamus.²⁰⁻²²

The premammillary artery arises from the central third of the PCoA.⁴² The hypothalamus located in wall and floor of third ventricle functions in visceral activities of the body, including water balance, glucose and fat metabolism, and temperature regulation.⁴³

The lamina terminalis is thin sheet of grey matter which forms anterior wall of third ventricle and links the rostrum and the anterior commissure with optic chiasm and is separated by the optic recess.⁴⁴ The organum vasculosum is one of a circumventricular organs and it serves as a outlet for hypothalamic peptides.^{45,46} On sagittal MR scans, the tuber cinereum can be seen as convexity in the floor of third ventricle and enhance just like the pituitary stalk.⁴⁷ Posterior to it is the mammillary bodies, which are seen as intermediate intensity structures on coronal or sagittal MR scans.

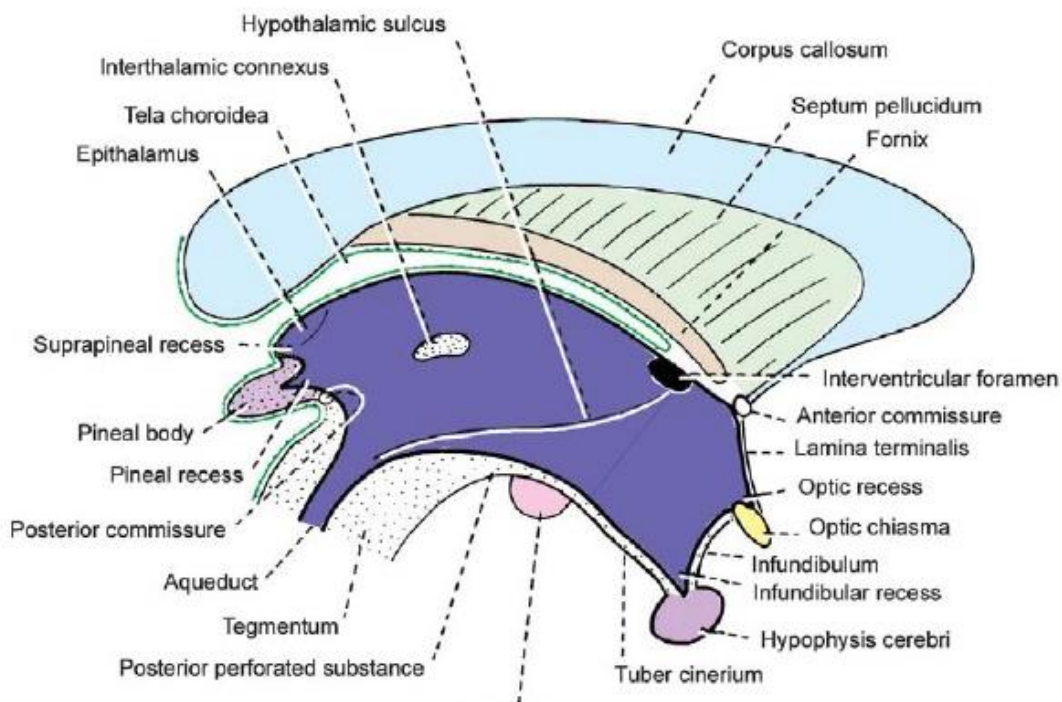


Figure 5 - Relations of floor of third ventricle to suprasellar cistern

THE PITUITARY STALK

The pituitary stalk connects the hypothalamus to the neurohypophysis. Functions of the stalk are:

- 1) carries oxytocin and vasopressin from the respective nuclei to the posterior pituitary.
- 2) neurotransmitters like dopamine to anterior pituitary.⁴⁰

The infundibulum lies obliquely in mid sagittal plane and is directed posterosuperior to anteroinferior. Blood supply to stalk is provided by:

- a) superior hypophyseal artery (branch of the supraclinoid ICA), - ring around the upper infundibulum
- b) inferior hypophyseal artery (branch of meningohypophyseal trunk)⁴⁰ - ring around posterior lobe of pituitary and lower infundibulum.

After entering the stalk, they break up into sinusoids and form the hypophyseal portal vessels and supply the anterior pituitary. They convey humoral substances from the tuberohypophyseal tract to the anterior pituitary.⁴⁰ On imaging the stalk is a midline structure posterior to optic chiasm measures approximately 4.0 mm (dorsum sella) and 4.5 mm (above the dorsum sella).⁴⁸ Usually it is noted that the stalk should be less than the nearby basilar artery.⁴⁸ Recent MR measurements indicate a normal diameter of $3.25 \text{ mm} \pm 0.56 \text{ mm}$ (near optic chiasm) and $1.91 \pm 0.40 \text{ mm}$ (near insertion in the pituitary).⁴⁷ An earlier MR report indicated that the upper limits of normal as 3.5 mm at median eminence and 2.8 mm at its midpoint.⁴⁹ It should taper smoothly superior to inferior in both the sagittal and coronal planes.⁴⁷ Because of absence of blood/brain barrier, the stalk enhances .

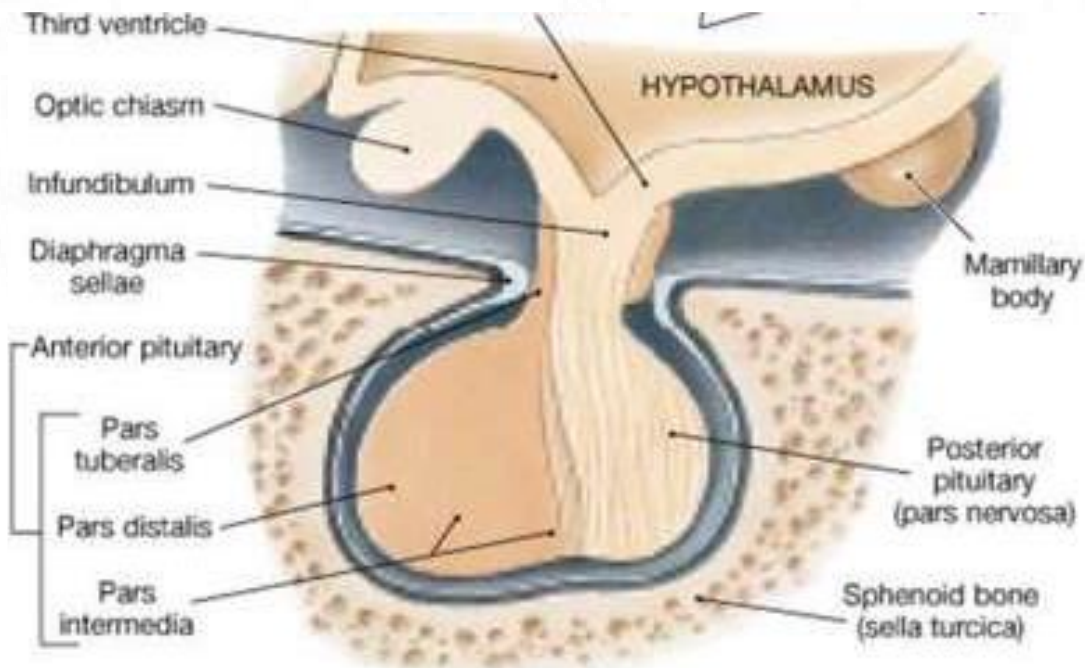


Figure 6 - Relation of stalk in suprasellar region

Clinical features

Tumours in this region are known to cause visual and endocrinological symptoms. This association was established in the 19th century. In 16th century a autopsy report written by Dutch professor Pieter Pauw (1564– 1617) had described a 18-year-old girl who had polyuria along with total blindness due to cystic tumour compressing optic chiasm and though he attributed the diabetes insipidus to the patient's kidneys, his report is the earliest known accounts of blindness and diabetes insipidus caused by an arachnoid cyst, Rathke cleft cyst, or craniopharyngioma in the region of Rathke pouch.⁵⁰

Depending on the location, mass effect can cause loss of vision (e.g. visual field defect, decreased visual acuity), cranial nerve dysfunction (e.g. diplopia, ptosis), or headache. A lesion that causes acute visual loss requires immediate evaluation and treatment.

Hormone Deficiency

Endocrine consequences of a pituitary lesion affect the reproductive system most commonly.⁵¹

a) Decreases FSH/ LH/ Testosterone- Men often have symptoms of hypogonadism—decreased libido, erectile dysfunction causing infertility. Women develop irregular menses, amenorrhea, or infertility.

b) Decreased growth hormone- in fatigue in adults and arrested or decreased growth rate in children and adolescents.

c) cortisol deficiency include fatigue, headache, weight loss, diminished appetite, and in some patients' hypotension and syncope

d) thyroid hormone deficiency include fatigue, cold intolerance, weight gain, bowel disturbance like constipation, difficulty concentrating, and memory problems

e) Deficiency of the vasopressin causes polyuria (particularly nocturia), polydipsia, and potentially volume depletion

The two most important hormones are cortisol and thyroid hormone, both of which are necessary for life.

Pituitary Hypersecretion

Excessive hormone secretion causes symptoms and signs associated with overproduction of prolactin, growth hormone (acromegaly), or adrenocorticotropic hormone (ACTH; Cushing disease).

a) Prolactinoma- most common type in secretory pituitary adenoma. Clinical features in women include irregular menses, amenorrhea, or infertility, galactorrhoea. Men develop reduced libido, erectile dysfunction causing infertility, gynecomastia, and galactorrhoea. They seek medical attention late in course of disease, visual loss may be the presenting feature.

b) Acromegaly- Excessive GH secretion after puberty causes enlargement of the bones of the face, hands, and feet. Other features include sleep apnoea, arthralgia, spinal stenosis, carpal tunnel syndrome, diabetes mellitus, hypertension, colon polyps, excessive sweating, oily skin, and cardiomyopathy with heart failure. Increase Growth Hormone levels before puberty results in gigantism with excessive linear growth.

c) Cushing disease - include weight gain, diabetes mellitus, hypertension, osteoporosis, bone fractures, depression, and memory loss

d) thyroid-stimulating hormone (TSH)–secreting tumour -clinical or subclinical hyperthyroidism (weight loss, tachycardia, frequent bowel movements, and anxiety).

Hormone Deficiency

The most important issue is to determine whether a patient has secondary adrenal insufficiency and/or secondary hypothyroidism. Low morning serum cortisol and ACTH levels may be adequate to diagnose secondary adrenal insufficiency. If there is doubt, a stimulation test such as an ACTH stimulation test or insulin-induced hypoglycaemia is indicated. This test is the most rigorous and reliable study; this test is also the most accurate for diagnosing GH deficiency. This test must be monitored by a physician and is not indicated in patients with coronary artery disease, a seizure disorder, or general debility.

For thyroid hormone deficiency, both the serum free thyroxine (free T4) and TSH levels should be measured. A low serum free T4 is the most reliable test. Patients with pituitary disease may have a “normal” serum TSH with low free T4 level. DI is primarily a clinical diagnosis (polyuria, polydipsia, excessive thirst, and in particular frequent nocturia—i.e. urination every 30–60 minutes during the night). Serum sodium and serum osmolality are normal in patient with intact thirst sensation and no restriction of fluid intake; serum osmolality may be normal, but urine specific gravity should be low. Gonadotropin deficiency in men is diagnosed by clinical symptoms and by measuring the serum testosterone and luteinizing hormone (LH). In gonadotropin deficiency, the serum testosterone level will be low in case of a “normal” serum LH (not normal for low testosterone). In women of reproductive age, irregular menses, amenorrhea, and infertility are the best indicators of gonadal dysfunction. Serum oestradiol may be low or “normal” depending on the day of the menstrual cycle, and serum LH is normally within the “normal”

range, but lack of regular menses or ovulation indicates gonadal dysfunction. GH deficiency is diagnosed by a decreased insulin-like growth factor 1 (IGF-1) level in case of several pituitary hormone deficiencies or by a subnormal GH response to a stimulation test (e.g. insulin-induced hypoglycaemia or arginine infusion).

Visual dysfunction

From an ophthalmic point of view visual system consist of three parts. The afferent system can be thought of as a mapping function, taking the outside world into our consciousness. For performance of this function the light is refracted by the anterior segment onto the photosensitive retina and it then converts the electromagnetic radiation into multiple impulses which are conducted by the optic nerves via visual pathway to the cortex, wherein interpretation is done. Disruption along these pathways causes various symptoms in visual function.

Afferent information is carried through the optic nerves, which has approximately 1.2 million axons from the ganglion cells. As the fovea is located nasal to the disc, fibres from temporal part of retina arc around the macula and separate fibres that are above the temporal horizontal midline from those below. This occurs in the retina at its disc and is maintained throughout in optic nerve. Therefore, any pathology affecting optic nerve follow nasal horizontal midline. This is the basis of anatomic origin of arcuate field defects. The most sensitive portion of retina is fovea and that is why it has the highest spatial resolution with relatively high (1:1 or 1:2) ganglion-to-receptor cell ratio. It does not have rods but has highest concentration of cones for colour vision causing colour abnormalities.⁵² The optic nerve is around 1.5 mm in diameter when it exits and extends <1mm through the sclera, becomes myelinated to increase in size to around 3 mm. Increased intracranial pressure causes disc oedema due to its surrounding optic nerve sheath and trabecular arachnoid which contains cerebrospinal fluid. Optic nerve sheath meningioma compromises the venous outflow causing formation of optociliary shunt vessels.⁵³ The length of optic canal is 6 to 8 mm directed superiorly and medially. The optic canal exits under falciform ligament and compression of superior portion of nerve against this ligament causes inferior field defect. Length of intracranial portion of optic nerve is around 10 mm. A post fixed chiasma can produce central scotomas and arcuate visual field defects. In preplaced chiasm there can be homonymous visual field defects.⁵⁴ Because most of the pupillary fibres cross, pathology affecting the optic tract may produce contralateral afferent pupillary defect.⁵⁵ Damage to tract has a distinguishing characteristic (not seen in cortical or optic radiation pathology) because retrograde degeneration will cause bowtie atrophy in the opposite optic nerve head. It occurs in weeks to months and will not be seen in acute tract lesion where opposite afferent pupillary defect develops acutely.

Pathology arising from the sella affects the anteroinferior part of the chiasm producing superior bitemporal field defect seen.^{56,57} The macular fibres cross posteriorly in the chiasm. Recent studies have demonstrated that looping of crossing fibres into contralateral optic nerve is an artifact.⁵⁸ The so-called Wilbrand knee⁵⁹ pathology that affecting the optic nerve anterior to chiasma produces central scotoma with relative temporal defect in the opposite eye. This still remains a useful localizing sign. Rarely, a lesion may cause junctional syndrome of Traquair.⁶⁰

Compression or ischemic involvement of optic nerve, chiasm, or optic tracts produces decreased vision or field defects.^{54,61} Most important question is whether this is uniocular or binocular. Loss of vision in one eye may be due to pathology in eyeball, retina, or optic nerve. If either eye is involved, there may be ocular pathology or chiasmal or retro chiasmal pathology. The next important question is of the onset which should include if visual loss is bilateral central, or one side, constant / intermittent with aggravating and relieving factors, associated neurologic or systemic complaints Patients should be enquired about previous history of comorbidities; inflammatory disease (demyelinating disease, infections, autoimmune conditions malignancy, or migraine. The patient's occupation is also important to look for exposure to toxins and agents. Prior records like ophthalmic evaluation, including records of previous ophthalmologists or results of visual screening, like for the Department of Motor Vehicles or military. Bitemporal field defect is often unnoticed.⁶² Formed visual hallucinations can occur with occipital involvement. Additional uncommon manifestations of pathology affecting the afferent system are visual obscurations⁶³ typical in patients with disc oedema due to raised intracranial pressure. Transient visual loss with exercise (Uhthoff phenomena) may be due to long-standing inflammation involving the optic nerves⁶⁴ Monocular loss of vision lasting minutes can be caused due to thromboembolic phenomena. Second most common complaint is double vision.⁶⁵ This could be monocular or binocular. Failure to resolve if the eye is covered shows that it is a monocular problem.

Various reasons have been postulated for visual loss and field defects in suprasellar tumours. Harvey Cushing⁶⁶ described classic chiasmal syndrome consisting of primary optic atrophy with symmetrical bitemporal hemianopia. However, symmetry of bitemporal field defects has been rare in later published series.^{67,68} In another report, Bergland and Ray⁶⁹ who studied arterial supply of chiasma in 480 autopsies, observed that the blood supply of central part is from below while that of the lateral part is from the lateral and superior aspects. Hence tumours growing from below would cause temporal field defects. However, why tumours arising from above the chiasm cause temporal field cuts could not be explained. McIlwaine et al.⁷⁰ postulated the preferential susceptibility of nasal crossing fibres.

Double vision which resolves by covering one eye is caused by ocular malalignment. Usually in incomitant deviations the cause is a cranial nerve palsy.⁷¹ It is necessary to recognize other causes which causes a skew deviation or internuclear ophthalmoplegia. Supranuclear palsies due to cortical or corticobulbar pathway pathology may limit motility but usually do not produce malalignment.

They can present with signs of raised intracranial pressure, when they lead to hydrocephalus by extension into third ventricle.⁷² Tumour associated with hydrocephalus are craniopharyngioma, optic pathway glioma. Hydrocephalus is found in 30% of craniopharyngioma patients upon presentation. Up to 55% of patients with glioma develop hydrocephalus that requires surgical intervention. Only 5% of pituitary adenomas become large enough cause hydrocephalus. Therefore, although large pituitary adenomas causing hydrocephalus to have been described in the literature.⁷³ Germ cell tumours can infiltrate third ventricle and rarely present with hydrocephalus.⁷⁴ Suprasellar arachnoid cysts can cause hydrocephalus.

Obstructive type of hydrocephalus is seen in suprasellar tumours. Hydrocephalus may result from obstruction at the following levels: (1) the basal cisterns, (2) invasion and obstruction of the inlet and outlet of third ventricle, (3) occlusion of the foramen of Monro, and, rarely, (4) posterior displacement of the brainstem with occlusion of sylvian aqueduct.

Since suprasellar tumours are usually slow-growing tumours, they may reach a significant size before causing any symptoms. Once the CSF flow becomes obstructed, the patients have symptoms and signs of increased intracranial pressure. Most commonly they present with headaches (80%), nausea and vomiting (60%), visual loss due to papilledema or direct pressure on the optic pathways (40%), short stature due to endocrinological deficiencies (30%), and mentation disturbances (5%). Patients with hydrocephalus due to suprasellar arachnoid cysts manifest with raised intracranial pressure (60%), ataxia (60%), macrocephaly (70%), reduced visual acuity (20%-30%), seizures (30%), and developmental delay (30%). Head bobbing is considered a pathognomonic symptom of suprasellar arachnoid cysts, occurs in only 10% of patients.

Investigations

Pituitary Hypersecretion

Serum prolactin should be measured to determine if a pituitary lesion is a prolactinoma, because medical therapy with dopamine agonist is the main treatment for a prolactinoma. The prolactin level should be correlated with the diameter of the lesion. Any lesion in this region may cause a

mild elevation of serum prolactin (interference with the prolactin inhibitor hormone, dopamine, through the pituitary stalk). In case of macroadenoma (>10 mm), serum prolactin level should be greater than 200 ng/mL for a true prolactinoma. This assessment is necessary to determine the course of treatment—medical therapy or surgery. If there is elevated serum prolactin level (>200ng/mL), the serum specimen must be diluted to obtain an accurate value (prolactin >200 ng/mL is not sufficient), and the physician must request that the laboratory perform the necessary dilutions to obtain the actual value. A value of >200 ng/mL may be 2000 or 20,000 or greater. To assess the response to medical therapy, an accurate baseline prolactin value is also required.

Acromegaly is diagnosed by clinical features, an elevated serum IGF-1 level, and a serum GH level that does not decline to <1 ng/mL after oral glucose (75 or 100 g). There are important considerations about these tests. Although the IGF-1 assay is reliable and reproducible, over last two years there have been false elevations of IGF-1 related to the database for the range of normal according to age. This problem is being addressed but still not yet fully resolved. The definitive test for acromegaly is the GH response to an oral glucose challenge (oral glucose tolerance test, or OGTT). The test must be performed correctly to interpret the results. Baseline serum glucose and GH are measured, the patient drinks a glucose solution (75 or 100 g), and the serum glucose and GH levels are measured every half hour for 2 hours. The current guideline for a normal response is a serum GH level of <1 ng/ml. Some patients with acromegaly may have a paradoxical increase in GH.⁷⁵

Cushing disease is diagnosed by demonstrating a consistent overproduction of cortisol even if there is detectable or elevated serum ACTH. Cushing disease is the most problematic of all pituitary adenoma diagnoses for several reasons, including overlap of the clinical features with those of other disorders (polycystic ovarian syndrome, obesity, depression) and the variable sensitivity and specificity of tests. Consistent overproduction of cortisol is demonstrated by three types of screening tests: elevated 24-hour urine free cortisol (preferably measured by tandem mass spectrometry), loss of circadian rhythm with elevated night time salivary cortisol levels, and failure of the serum cortisol to decline to <1.8 µg/dL at 8 AM after ingestion of dexamethasone at 11 PM the previous night.⁷⁶ These three types of screening tests are ~92% accurate; repeated tests are necessary to establish the diagnosis. Serum ACTH may be in the normal range or elevated. High-dose dexamethasone suppression test (given as 8 milligrams overnight or 2 milligrams every six hours for 48 hours) was developed to distinguish between pituitary dependent or ectopic ACTH syndrome. Unfortunately, these tests are not sufficiently sensitive or specific to exclude ectopic ACTH production by a tumour in the lung, pancreas, or thyroid gland. Because ~50% of patients with a pituitary adenoma causing Cushing disease have no visible lesion on dynamic MRI

with a pituitary protocol and because 10% of adults with normal pituitary function have a visible lesion in pituitary gland (“incidentaloma”), an MRI study is not sufficient to recommend pituitary surgery. The inferior petrosal sinus sampling (IPSS) study is the one of most precise method to determine if the source of ACTH is pituitary gland and to exclude ectopic ACTH syndrome. This test involves comparing the central (petrosal sinus, left and right) and peripheral (inferior vena cava) ACTH levels before and after administration of CRH. A ratio of the basal central to the peripheral ACTH level of >2 or a ratio in CRH stimulated patients of >3 indicates a pituitary aetiology. This invasive study should be performed only by an experienced interventional radiologist or neuroradiologist. This study is not without risk, including thrombosis and stroke, emphasizing the requirement that it be performed by an experienced radiologist.

An uncommon type of secretory adenoma produces excessive α subunit, which causes no specific clinical features but often causes hypogonadism. Measuring serum α subunit serves as a tumour marker before and after surgical removal of the adenoma. Although many pituitary adenomas are gonadotroph tumours by immunohistochemical criteria, they rarely secrete excessive amounts of LH or follicle-stimulating hormone (FSH). Measurement of the serum LH and FSH serves as a tumour marker in the event the tumour produces an excessive amount of one or both hormones

Ophthalmic evaluation

Ophthalmic evaluations can be a) qualitative - localizing in nature, to confirm the presence of lesion, direct additional work-up b) quantitative- assessment of natural history of disease and whether therapy is useful. Central vision is measured by Snellen chart. It represents only macular function. It is the lowest Snellen optotype seen at the distance of 20 feet. The upper case of the fraction (e.g. 20/40) indicates testing distance and lower case denotes smallest optotype is seen. The optotype is size of the letter that subtends 5 minutes of arc at a particular distance. Corrected visual acuity should be recorded. Near vision should be checked routinely. An important part of evaluation of vision is visual fields.^{60,77} This can be done using:

- 1) confrontation testing,
- 2) tangent screen testing,
- 3) Goldmann or automated static perimetry.⁷⁸

On assessing the visual field defects if they follow the vertical midline it can be chiasmal or retro chiasmal compression and if horizontal midline is respected then it can be optic nerve pathology. Right visual field is on the right and the left to the left and the temporal fields are laterally and nasal fields are medially. Visual field defects are helpful in localizing pathology:

- A) Bitemporal field defect- area of the chiasm.
- B) Homonymous field defects - post-chiasmal.

C) central scotoma or arcuate field defects -Involvement of optic nerve

Evaluation of the afferent system involves:

1) assessment of pupillary response-

a) regulates the amount of light coming into the visual system.⁷⁹

b) regularity and symmetry should be examined. Anisocoria may be physiological but the difference should be the same in light and dark.⁸⁰

c) asymmetric pathology of optic nerve causes afferent pupillary defect.⁸¹ It may be quantified by keeping neutral density filters in front of better-functioning eye⁸²

2) the retina

3) optic disc.

Funduscopy evaluation:

a) helps us to look at the initial part of optic nerve.

b) Raised intracranial pressure may produce papilledema due to blockage of axonal transport and if persistent will produce atrophy.

c) Suprasellar tumours may present with bilateral disc oedema (due to increased intracranial pressure) or may have unilateral disc oedema and contralateral optic atrophy (Foster Kennedy syndrome- due to compression of one optic nerve).⁸³

d) Grading of papilledema:

1) Grade I papilledema - C-shaped halo with a temporal gap

2) Grade II papilledema- halo becomes circumferential

3) Grade III papilledema -loss of major vessels when they are leaving the disc

4) Grade IV papilledema - loss of major vessels on the disc.

5) Grade V papilledema - grade IV + plus partial or total obscuration of all vessels

Optical coherence topography (OCT):

It allows more quantitative assessment of optic nerve, optic disc, and nerve fibre layer. There is a delay in the thinning of the nerve fibre layer for weeks to months, between acute onset of optic nerve dysfunction and thinning seen on OCT. The OCT will not show zero because of the support structures in retina and they will keep nerve layer thickness measurements in the range of upper 30s - lower 40s range. Thinning of nerve fibre on OCT shows that lesion is long standing and useful for prognostication of recovery of already damaged nerve. The lack of OCT thinning may be taken as a relatively good prognostic sign for potential recovery. Converse is also true and that even with significant thinning, there may be substantial improvement in optic nerve function, particularly central acuity and even by visual fields.

Radiology

On CT, suprasellar cistern is hypodense because of its CSF content. On axial scans it most frequently appears as a hexagon.⁸⁴ The cistern usually is symmetric in shape, but mild asymmetry is seen in 5% of cases.⁸⁵ Anterior limbs of this star is formed by posterior part of interhemispheric fissure. Both anterolateral limbs of the star formed by medial part of Sylvian fissures. Posterolateral points on both sides of the star are formed by the crural cisterns. interpeduncular cistern forms the posterior point.

CT Scan

Advantages

1. Images obtained in less time
2. Superior in detecting bone, calcification, and acute intracranial haemorrhage
3. Wider availability
4. Costs less

Disadvantages

1. Relatively poor soft tissue contrast
2. Artefacts from bone and metallic objects
3. Lack of direct sagittal scanning capability
4. Difficulty in positioning patients to perform a coronal Scan

Magnetic Resonance Imaging

Neuro imaging study of choice for demonstration of soft tissue anatomy and pathology.

Views can be obtained directly in any plane allowing excellent localization and three-dimensional reconstruction of images.

Advantages

1. Multiplanar capability
2. Increased soft tissue contrast

Disadvantages

1. Requires a scanning time of several minutes per sequence
2. Difficult in claustrophobic patients
3. Contra indicated in patients with cardiac pacemakers, metallic implants. Etc
4. More expensive

Intensity of CSF varies with sequence. Short TR/TE images it has low signal and increased signal

intensity on long TR/TE images. It is the direct sagittal plane capability of MRI that demonstrates the anterior and posterior recesses of suprasellar cistern and their relationship.

Imaging

CT is excellent to provide information on sellar floor or calcification⁸⁶ and MRI is better than CT in providing information about lesion morphology and its extent.⁸⁷ Most microadenomas are best shown on thin postcontrast images sagittal T1-weighted sections. The fat saturation pulses may also be beneficial.⁸⁸ Dynamic T1 weighted images after administration of contrast delineates the pathology better. Typical scanning time for dynamic images are of 10 to 12 seconds. On these sequences it shows characteristic pattern of centrifugal enhancement. Kucharczyk et al,⁸⁹ using a dynamic MR technique and found an increased rates of adenoma detection. Tabarin et al,⁹⁰ in contrary demonstrated that if pattern of enhancement is not understood it can lead to false-positive interpretations. Pierallini et al⁹¹ suggested that adenomas may be classified as soft and hard groups based on their apparent diffusion coefficient values obtained by DWI.

Imaging Anatomy

Elster's rule that pituitary gland measures 6 mm in infant and children, 8 mm in men and postmenopausal women, 10 mm in women of childbearing age, and 12 mm in pregnant and postpartum women.⁹² Adenohypophysis is isointense whereas the neurohypophysis has a characteristic hyperintensity on T1-weighted imaging due to possibly presence of neurophysin (vasopressin-associated carrier protein). Any stalk pathology can lead to bright spot occupying an "ectopic" position.⁹³ The normal stalk has a diameter of 4 mm, smoothly tapers into the gland, and enhances fairly homogeneously. Optic chiasm is best evaluated on the coronal images.

Differential diagnosis on radiology

Pathology in suprasellar area- Cystic Sellar/Suprasellar Lesions

Craniopharyngiomas

Craniopharyngiomas as a benign, slow-growing tumors originate from squamous epithelial cells of Rathke pouch. It accounts for 2-4 % of intracranial tumors in adults, and 6-10 % of all tumours in children.⁹⁴ There is no gender predominance.⁹⁵⁻⁹⁷ There are two peaks: one in childhood around the age of 10 years, and a second one in the 5th and 6th decade of life.^{94,98-100} The papillary variety is likely to present in older individuals as a solid enhancing tumors, although solid-cystic pattern may be seen. Most of the cystic craniopharyngiomas remain localized to the suprasellar region.¹⁰² Cystic giant craniopharyngiomas with extensions into adjacent areas are described.¹⁰³ Multi-

lobulation of the cysts is a common finding.¹⁰⁴

Suprasellar Cysts

Rathke cleft cysts present as discrete non enhancing lesions on MRI. The signal intensities depend on function of protein concentration. Average age of 57 years in Rathke cyst is higher than for arachnoid cysts with 9 years.¹⁰⁵

Pars intermedia cysts are found between adenohypophysis and neurohypophysis and incidentally detected. Usually seen as T1 hypointensity, T2 hyperintensity on MRI with no enhancement

Arachnoid cysts arise from incomplete perforation of Lilliequist membrane.

Arachnoid cysts are relatively rare and account for about 10 % of all arachnoid cysts.¹⁰⁶ The etiology remains unclear- may exist congenital forms or acquired symptomatic forms.¹⁰⁷

Hemorrhage into an arachnoid cyst is possible and results in signal changes over the time.

Epidermoid and Dermoid Cysts

These developmental lesions can be recognized by their characteristic MRI signal intensities. They are well delineated from the surrounding tissue¹⁰⁸ often despite a large volume without mass effect. The CT examination often detects calcifications in dermoid cyst as compared to epidermoid cyst.¹⁰⁹ Epidermoid tumours show characteristic restriction on DWI differentiating from an arachnoid cyst. Rupture of dermoid cyst can result in chemical meningitis. It is seen on MRI as high T1 signal intensity due to fat droplets in the subarachnoid spaces.¹¹⁰ The long T1, T2 relaxation time of the epidermoid are known¹¹¹ but in dermoid there is reduction of T1 values resulting in a hyperintense signal on T1-weighted images.

Meningiomas and Schwannomas

10% of meningiomas are found in this region. They arise from dura on planum sphenoidale, diaphragm sellae, cavernous sinus, and optic nerve sheath. Meningiomas involving cavernous sinus and surround the cavernous internal carotid artery, they constrict it, as compared to macroadenomas which does not constrict it. Schwannomas have heterogenous enhancement and show foci of cystic change or hemorrhage. They demonstrate a tubular shape, conforming to the nerve of origin.^{112, 113}

Germ Cell Tumors

Germinoma is a rare lesion that can occur in pineal region and suprasellar region.¹¹¹ Clinical findings include visual disturbances, diabetes insipidus, occlusive hydrocephalus, and headache. Midline enhancing solid suprasellar lesion with a concomitant pineal lesion, is usually germinoma

occult neuro-hypophyseal germinoma is germ cell tumors in a young patient with diabetes insipidus that may not be seen on initial imaging and follow-up MRI may reveal its presence and is therefore warranted.¹¹⁴

Chordomas and Chondrosarcomas

They arise from clivus. they may present as suprasellar masses. Chondrosarcomas are usually centered at Petro clival fissure. Chordomas are more midline in location. The bone destruction is best seen on CT. On MRI, heterogeneous signal intensity due to hemorrhage, calcification, proteinaceous material is visible.¹¹⁵

Gliomas/astrocytoma's

Some authors summarize and classify the optic chiasmatic glioma as pilocytic astrocytoma.¹¹¹ They comprise approximately 3 % of all brain malignancies.¹¹¹ and may associated with NF type 1. These gliomas are from the histologic point of view benign lesions and slow growth. They occur usually between 1 and 10 years of age. A second peak is described for the higher age.¹¹⁶ MRI is helpful for the diagnostic and differential diagnostic evaluation.¹¹⁷

Inflammatory diseases

Sarcoidosis and tuberculosis

A major differential diagnosis are inflammatory diseases. Neurosarcoidosis usually affects the pituitary stalk causing diabetes insipidus.¹¹⁸ On MR images pathologic Dural enhancement as well as an infiltration of pituitary stalk is seen. An important clinical and neuroradiologic differential diagnosis is tuberculosis. Both can cause granulomas. Use of MRI is more specific in the diagnostic evaluation of tuberculosis.¹¹⁹ Some authors describe a "target sign" (central bright spot with a hypointense rim) on T2- weighted images, which is classical for tuberculosis.¹¹⁹

Table 1 – Pathology and radiological findings of suprasellar tumours

Sl.No.	Pathology	CT	MRI		
			T1	T2	Contrast
1.	Rathke's cleft cyst	Follows CSF density (may be hyperintense)	Hyperintense Variable (depending on protein content)	Hyperintense Variable (depending on protein content)	Peripheral enhancement
2.	Meningioma	Isodense or hyperdense to	Hypointense to slightly	Hypointense to slightly	Intense homogeneous

		cerebral cortex Calcifications Hyperostosis Bone erosion	hypointense	hyperintense	enhancement with enhancing Dural tail
3.	Chordoma	Hypodense with calcification	Hypointense	Hyperintense with interlobular septae	Variable
4.	Chiasmatic and hypothalamic glioma		Hypointense	Hyperintense	Variable
5.	Arachnoid cyst	Follows CSF imaging characteristics			Nil
6.	Epidermoid	Hypodense	Hypointense	Hyperintense	Mild or no enhancement
7.	Adamantinomatous type	Cystic- Follows CSF density Solid- Soft- tissue density Stippled calcification (90%)	Cystic- Isointense to hyperintense (depending on protein content) solid- Hypointense	Variable	Heterogenous enhancement
8.	Papillary type (prominent solid content with very few cysts)	Cyst- Follows CSF density (if present are very small) solid- Soft- tissue density calcification Rare	Cystic- Variable Solid- Isointense to hypointense	Variable	Intense enhancement
9.	Pituitary Microadenoma	Not clearly visualized	Not clearly visualized	Not clearly visualized	Early phase no enhancement compared to normal pituitary

					gland enhancement Delayed contrast enhancement
10.	Pituitary Macroadenoma	Hypodense Contrast enhancement	Hypointense	Hyperintense Hypointense (in functioning GH-secreting tumour)	Early intense contrast enhancement
11	Germ cell tumour	Hyperdense calcification	hypointense	hyperintense	Heterogenous enhancement

Surgical management

Surgical resection is the main therapeutic option.

Advantages-

- 1) adequate decompression of compressed neurovascular structures
- 2) establish histopathological diagnosis
- 3) relieves raised intracranial pressure
- 4) allows for complete cure.

Disadvantages-

- 1) is technically difficult
- 2) demands an optimal approach
- 3) brain retraction
- 4) traction/injury to neurovascular structures.

Anatomical nuances for surgery

In 1975, Rhoton, together with Renn dissected and analysed the anatomy of 50 adult sellar regions removed en bloc¹²⁰. Their aim was to investigate implications and variations in anatomy held for different surgical approaches and the incidence of each variation.¹²⁰ Important Anatomical factors:

(a) a prefixed chiasm (10%) and a normal chiasm $\leq 2\text{mm}$ between chiasm and tuberculum sella (14%)

(b) an acute angle between optic nerves when they are forming chiasm (25%);

(c) a prominent tuberculum sellae protruding above line connecting optic nerves when they enter optic canals (44%),

(d) carotid arteries within ≤ 4 mm of the midline around sella (12%) – ‘kissing carotids’

The importance of relationship of optic chiasm:

- 1- first determined by the criteria of Bergland et al.¹²¹
- 2- choice of approach
- 3- difficult the surgery will be.

Post fixed chiasm:

- 1- easier approach and resection
- 2- tumour is accessible between the tuberculum sellae and the front of the chiasm,
- 3- No need to work between optic apparatus and the ICA

Types of surgical approaches:

1. Transcranial
2. Endoscopic

Transcranial approach

The first transcranial surgery was done in 1893 that had been postulated by Sir Victor Horsley.¹²² The surgery was not successful as the patient died. While Horsley’s mortality rate for transcranial route was 20% and his surgical colleagues had mortality rates around 50–80%.^{123,124} Therefore, attempts were made to access via other means and hence transsphenoidal approaches were developed and carried lower rate of mortality. However, the initial transsphenoidal techniques were limited by the technology and medical knowledge.¹²² Patients had high rate of cerebrospinal fluid leaks and meningitis. It was not until the discovery of the operating microscope that the transsphenoidal approach regained its popularity.¹²² Recently with technology development the endoscopic are far superior routes for most tumours within suprasellar region regions. Approximately 1–4% require surgical resection via a transcranial route. Those are:

- 1) Pituitary tumours with extensive superior parasellar extension
- 2) Parasellar lesions extending into cavernous sinus
- 3) dumbbell-shaped sellar lesions with a narrow neck across the diaphragma sellae
- 4) patients may have contraindications to transsphenoidal surgery.
- 5) ectatic “kissing” carotid arteries
- 6) the morphology of the tumour like fibrous tumour, high vascularity
- 7) previously failed transsphenoidal exploration.

Transcranial approaches provide a large field of view. They have very flexible working angles

and expose cranial nerves and neurovascular anatomy early in the operation.

The principles in surgical management are: -

- 1-to relieve mass effect on the visual apparatus
- 2-normalize pituitary hypersecretion,
- 3-preserve or restore normal pituitary function,
- 4-prevent tumour recurrence
- 5-provide tissue for pathological and scientific study.

Ideal surgical approach is the one with –

- 1- the shortest route to the lesion,
- 2- confers minimal trauma to surrounding structures,
- 3- provides adequate exposure
- 4- permit the manipulation necessary to resect the lesion

Another option is to either perform both procedures simultaneously as advocated by Barrow et al¹²⁵ and Alleyne et al.¹²⁶ or to stage the procedure performing the transsphenoidal procedure first.

Advantage of performing the transsphenoidal procedure:

- 1- adequate decompression of optic apparatus, the principal reason for the surgery,
- 2- with tumour descent, permit resection of sufficient tumour, making a subsequent craniotomy superfluous.

Disadvantages of performing the transcranial approach first:

- 1- Increases postoperative CSF fistula after the subsequent transsphenoidal operation.
- 2- residual tumour beyond the narrow exposure of the transsphenoidal approach will then not descend into the operative field due to the development of fibrosis and adhesions.

Suprasellar meningiomas have been usually approached transcranially because it helps to completely resect Dural tails to prevent recurrence. Now there are series of extended endoscopic procedures for meningiomas.¹²⁷ Such procedures should be undertaken by experienced pituitary surgeons' patients having midline lesions with minimal lateral, anterior or posterior Dural extensions.

Transcranial approach

Pterional Trans-Sylvian Approach

First shown by Silbermark¹²⁸, and then later by Heuer¹²⁹ and Adson¹³⁰ provides shortest distance to suprasellar region and is used most frequently. In craniopharyngioma series by Fahlbusch et al.¹³¹ It was used in 39.2% procedures. Ideal when optic chiasm is post fixed

Side of approach –

- 1) side of the tumour- aid in preventing untoward retraction.
- 2) If side not a determinant then side with greater visual deficit should be selected
- 3) If neither of the above, then non dominant hemisphere is the side for approach.

Frontobasal Interhemispheric Approach

Indications- Large midline lesions with bilateral extension along optic pathway with post fixed chiasm

Advantage-

- 1) wide view of optic and lamina terminalis.
- 2) earlier identification of important midline structures

Risk-

- 1) frontal lobe retraction causing seizures
- 2) loss of smell

In this approach it is necessary to extend the exposure up to ACF floor to minimize brain retractions. It is necessary to open the subarachnoid space early to drain CSF and maximize allow brain relaxation. One needs to expose the optic nerves, optic chiasm, A2 segments and anterior communicating artery. It is considered the best transcranial approach for large retrochiasmatic and suprasellar craniopharyngiomas which can be exposed through lamina terminalis giving you a midline view into the interpeduncular cistern.¹³¹

Unilateral Sub frontal

Indications- lesions that extend anteriorly or laterally on one side.

Advantage- requires less surgical time as compared to bifrontal exposure and provide same exposure of midline structures without the need for ligation of superior sagittal sinus or dissection of the olfactory bulb and tract.¹³²

Supraorbital Keyhole Approach

Similar to the unilateral sub frontal approach.

Indications- tumours superior to pituitary gland and around the optic nerve.

Advantages –

- 1) minimal bone removal
- 2) much smaller skin incision.

Disadvantages- the intracranial exposure is more restrictive.

Orbitozygomatic Approach

Indications-

- 1) preferred approach when additional bony removal is necessary for resection of large lesions with superior and posterior extensions
- 2) tumours that extend superiorly into third ventricle or anteriorly into the posterior third of the orbit

Advantages-

- 1) provide the versatility of both lateral and anterior access
- 2) absolutely minimal brain retraction
- 3) Expanded corridor

Technically, this is a more difficult exposure. One should preserve supraorbital and supratrochlear nerves. If they are sacrificed, they can cause lot of postoperative discomfort. Tumour lies at depth of 2cm more from Pterional approach.¹³³

Interhemispheric Transcallosal Approach

Indications-

- 1) large septated craniopharyngiomas¹³⁴
- 2) third ventricular tumours.

Dilatation of lateral ventricles is necessary in the exposure. One must avoid manipulation of the fornix. The surgeon should also preserve thalamostriate vein and internal cerebral vein. The septum pellucidum should be fenestrated and postoperatively a ventriculostomy drain should be placed to avoid hydrocephalus.

Sub temporal Approach

Indications- tumours extending to the infratentorial space, retrochiasmatic, or extend into the temporal fossa.¹³⁵

Preoperative MR venography is useful to assure that the vein of Labbé is not tethered anteriorly.

Endoscopic approach

The transnasal was brought into modern medical practices by Harvey Cushing and Oskar Hirsch in the early 1900s.¹³⁶

With technological development endoscopic skull base surgery became a simple, safe, and

minimally invasive technique in 1990s.¹³⁷ With recent addition in technology like navigation, endoscopic approaches have also expanded.¹³⁸

Advantages of endoscope:

- 1) improved visualization because of wide angled views
- 2) Close up magnification.
- 3) avoiding use of intraoperative fluoroscopy.
- 4) dynamic movement of endoscopic vision.
- 5) cosmetically accepted
- 6) less invasive than the conventional surgery.¹³⁹
- 7) improve clinical outcome with less postoperative complications.
- 8) Adequate decompression of optic pathway and oculomotor nerve.
- 9) greater chances to preserve vascular supply of optic chiasm and nerves. These findings are in accordance with reports of the visual outcome in the literature. Frank et al.¹⁴⁰ reported 94.7% improvement in vision, 3.8% no change, and 1.2% worsening. Tabaei et al.¹⁴¹ showed 92% improvement with no worsening, and Dehdashti et al.¹⁴² reported 91% improvement and 9% no changes of visual functions.

Since the pioneering work of Jho¹⁴³ the endoscopic approach is used for many suprasellar tumours like meningiomas.

Advantages of endoscopic approach for meningiomas include:

- a) minimal brain retraction
- b) Minimal handling of optic apparatus
- c) early identification of stalk and pituitary gland.
- d) Increased removal of involved bone and dura
- e) the tumour vascularity can be reduced in the early part of the operation
- f) 270° bony decompression achieved if optic canal is involved by tumour
- g) early extradural optic canal decompression in case of extensive hyperostosis.¹⁴⁴

70%–80% of patients have vision improvement after surgery if they have preoperative deterioration of vision. 7%–12% of patients experience worsening of vision after surgery.¹⁴⁵ The rates of postoperative visual improvement ranges from 85.7%–96.6% and that of post-operative deterioration 0%–3.6% in endoscopic approach. But in transcranial approach it is lower 44.4%–90.6% and 3.6%–24.2%, respectively.¹⁴⁶ Koutourousiou et al.¹⁴⁴ reported visual improvement in transnasal surgery to be around 94%. The lower rate of improvement in vision (74%) in transcranial approach shows us that there are limitations in achieving adequate optic nerve decompression via this approach.¹⁴⁷ Early optic canal decompression with endoscopic approach

could be the reason for higher rate of improvement.¹⁴⁸

Endoscopic approach for craniopharyngioma:

Recent advances in skull base has provided for safer gross total or near-total resection (72.7-90%). Previously transcranial approach was considered gold standard in the treatment of craniopharyngiomas.

Disadvantages of transcranial approach in craniopharyngioma-

- 1) If located in the retrochiasmatic region could be hidden from the view
- 2) Brain retraction, manipulation neurovascular structures
- 3) Poor visualization of the undersurface of the chiasm
- 4) Critical plane between capsule of the tumour with the stalk and hypothalamus is poorly seen.
- 5) perioperative mortality rate risk of 0%–20%¹⁴⁹ and pituitary-related endocrinopathy risk of up to 100%.¹⁵⁰ Hypothalamic obesity is seen in 70% of patients.¹⁵⁰

Recently the extended EEA has been advocated for suprasellar–retrochiasmatic craniopharyngiomas.¹⁵¹

Advantages are:

- 1) Direct visualization of the undersurface of the important neuroendocrine structures¹⁵².
- 2) to avoid or minimize visual and hypothalamic complications.¹⁵³
- 3) better visual outcomes, higher GTR rate, and stable quality of life, with comparable complication rates.¹⁵¹

Major limitations are:

- 1) Lateral extending tumours into sylvian fissure and superior extension into interhemispheric fissure.
- 2) direct microvascular repair, if vessel injury occurs during dissection, is difficult
- 3) The problem of CSF leakage,

Contraindications of Endoscopic Transsphenoidal Approach

- 1) Tumours extending lateral and posterior to carotids and orbit are difficult.
- 2) Encasement of A2 segment of anterior cerebral artery is relative contraindication
- 3) Hypothalamic hamartomas and germ cell tumours are examples not suitable for this approach.
- 4) Sinonasal infection is the relative contraindication.
- 5) surgeons with lack of appropriate experience and instruments.

Complications

The location of suprasellar region at base of brain and the important anatomical associations can cause serious morbidity and mortality.¹⁵⁴ These are:

1) Hypopituitarism- more common after transcranial approach for pituitary adenomas than transsphenoidal resections. Pituitary adenomas push normal pituitary tissue posterosuperiorly. Therefore, to prevent hypopituitarism one should resect the tumour from below. Normal gland is identified by the colour, striated appearance due to capillary network, consistency of the normal gland.

2) Stalk- manipulation causes Diabetes insipidus. It is less common after transsphenoidal approach. Tumour most commonly associated with DI is craniopharyngiomas.

3) Visual deterioration – It may occur in an immediate or delayed manner because normal optic nerves tolerate manipulation to a greater extent than compromised nerves. Factors associated with postoperative visual loss are a) prior irradiation b) previous surgery c) pre-existing deficit d) technical difficulties with surgery, d) diabetes mellitus.¹⁵⁵ The etiopathogenesis of visual loss is disruption of blood supply to optic chiasm or nerves. Therefore, it is necessary to understand the microvascular anatomy of optic nerves and chiasm. Meticulous microdissection techniques especially important to prevent postoperative visual deterioration. Perioperative steroid cover with 4mg q.i.d. of dexamethasone prophylactically is helpful.

4) Hypothalamic injury- Etiopathogenesis- 1) direct surgical injury 2) haemorrhage or ischemia, 3) previous surgery or radiation therapy. It is rare and carries high mortality. Clinically it manifests a) acutely with diabetes insipidus, b) somnolence or autonomic dysfunction c) specifically difficulties with temperature regulation d) chronically with morbid obesity, e) memory loss, d) insatiable hunger or thirst e) if severe causes decreased sensorium.¹⁵⁶ To minimize this one needs to have gentle surgical technique, less traction on tumour capsule and pituitary stalk, and less retraction on the brain.

5) Extraocular palsy- Third nerve palsy can occur in surgery for these tumours as it gets splayed over the surface of tumour posterolateral to the ICA

Review of literature

Heli H Vasani, et al¹⁵⁷ in their study of 125 patients of which 86 patients had visual impairment and 78 patients met their inclusion criteria. 42(53.84%) patients were males and 36(46.15%) patients were females (sex ratio:1.166) mean age at surgery was 50.02(46.77-53.27), all tumours were macroadenomas (>10mm). They had noted that Pre-operative tumour volume was $11.00 \pm 8.88 \text{ cm}^3$ ($p < 0.001$). Post-operative tumour volume was $4.87 \pm 3.88 \text{ cm}^3$ ($p < 0.001$). More than

70% reduction of the tumour volume was achieved in 68% of the cases and near total tumour resection (>90% tumour volume reduction) was achieved in 28% of the total cases. Visual improvement was seen in 116 of 156 eyes (58 of 78 patients- 74.35%), unchanged in 32 eyes (16 patients-20.51%) and worse in 8 eyes (4 patients -5.12%). Visual field improved in 120 of 156 eyes (60 patients), unchanged in 36 eyes (18 patients), worse in none. In their study factors associated with visual outcomes were:

1. Preoperative visual status-Eyes that had VA better than 6/48 showed better results postoperatively -89% of these eyes reported improvement while only 60% eyes with VA less than 6/60 showed good results postoperatively.
2. Age-younger patients had better results postoperatively, this was not related to the fact that old patients had poor eye status as young patients with worse preoperative visual status also showed better outcome than older patients
3. Duration-patients with duration of visual symptoms < 6 months fared better (88% of these patients achieved improved VA) than patients with visual symptoms for > 6 months (68% of these patients achieved improvement post operatively).
4. Preoperative visual field cut without loss of acuity showed better prognosis.

They finally concluded that endoscopic approach has shown significantly higher gross total resection rate when compared with microscopy, particularly for pituitary tumours with cavernous sinus invasion and fewer recurrences as well as better visual outcomes.

Desanka GRKOVIĆ et al¹⁵⁸ in their study had aimed to analyse visual function pre and postoperatively of meningiomas of optochiasmatic region, and to determine which factors, including the age of patient, duration of symptoms, visual function damage, and size of the tumour, affect postoperative visual function outcome. 43 out of 54 (79.6%) had visual dysfunction, women were 32 (74.4%) and men were 11 (25.5%). The average age was 53.3 years (ranging between 36 to 71 years). The most common symptom was reduction in acuity. 41 (95.3%) had visual acuity deterioration. Monocular visual acuity reduction was seen in 37.2%. Binocular visual acuity reduction was seen in 25 (58%) 43 patients. They noted that patients who had tumour size < 30 mm, 55% had improvement in visual acuity, 22% remained the same and 5% had an deterioration in visual acuity; in tumours with size between 31-70mm 37.5% of patients had improvement, vision remained same in 36.8% and 6.2% had deterioration in acuity. For patients with tumour >70 mm 50% of patients had deterioration in vision with mortality nearly 50%. Patients with symptoms < 6 months acuity improved in 61.1% patients. For patients with symptoms between 7 months- 2 years improvement was there in 50%. Visual acuity was better

in 10 patients having with symptoms > two years, no improvement was there in 60%, and deterioration was seen in 30% of cases. In 38 eyes with mild or moderate preoperative visual impairment, 81.6% showed improved or stable vision after surgery, compared to 58.6% of patients with severe reduction of preoperative vision. They concluded that visual acuity improvement was there in 50% patients 6 months post-surgery. The important factors that affect postoperative visual function improvement were preoperative visual acuity > 0.2 on Snellen chart, duration of symptom < 2 years, and size < 30 mm.

Nipat Aui-aree et al¹⁵⁹ had aimed to study ophthalmic presentations identify factors that influence improved visual outcomes in suprasellar tumour. They found out that 55% were male and 45% female. The median age was 38 years (range 10-82 years). The most common presentation was blurred vision (92.5%). 62% (23/37 patients) had uniocular complaints. 51 eyes had mean acuity 1.27 logMAR (range 0.2-3). Mean duration was 15.7 months (3 days-7 years). Most common field defect was hemianopia which was unilateral or bilateral. Most common lesion was pituitary adenoma. Mean visual acuity after surgery was improved to 0.71 logMAR value (range 0-3). Post-treatment visual improvement was best seen in pituitary adenoma, but there was no statistically significant difference between improvement and non-improvement groups in any of the variables examined. Main limitation of this study was an inadequate sample size. Another limitation was short follow-up period (4-6 weeks).

Farrukh Zulfiqar et al¹⁶⁰ had analysed postoperative visual changes in patients with suprasellar lesions with pre-existing visual deficit after surgery. A total 107 patients were operated. Transsphenoidal (43) or transcranial (64) surgeries were included. Pituitary adenomas were seen in 66 patients, 24 patients had craniopharyngiomas, 13 meningiomas, 3 chordomas, 1 had epidermoid cyst. About 25 had presented with uniocular visual deficit and 82 presented with binocular. They found that postoperatively 46% eyes improved, 34.4% and 19.6% remained same and deteriorated, respectively. Patients who underwent transsphenoidal technique had significant improvement 65%, and who underwent transcranial had 37.5% improvement (p-value=0.005). Pituitary adenomas showed greater visual improvement of 65% (p-value=0.000), patients with craniopharyngiomas showed improvement in 33.5% and meningiomas in 7.6%. About 52 patients (48.6%) showed improvement in vision. Visual acuity in remaining 55 (51.4%) did not improve. Limitations in their study was relatively short follow-up (4 weeks). They concluded that patients experience benefit in vision after surgery. Patients who had pituitary adenoma and undergo transsphenoidal surgery showed greater improvement.

Ashish Suri et al³ had studied the visual outcome after surgery in patients who had suprasellar tumours and presented with preoperative blindness in 1 or both eyes. There were total 79 patients (51 males, 28 females, age- 5–70 years). It included 37 pituitary adenoma, 19 craniopharyngioma, 18 meningioma, and 5 other tumours. About 61 patients had uniocular deficit. 18 patients presented with binocular visual deficit. Duration of visual deficit ranged from 3 days to 7 years. Patients underwent transcranial or transsphenoidal surgery. Visual improvement was seen in 23 (29%). Visual improvement was seen in 15 (24.6%) patients with uniocular deficit and 8 (44.4%) patients with binocular deficit. Analysis showed male sex, shorter duration of symptoms, apoplexy, sellar extension, soft consistency, haemorrhage in tumour, and histopathology (pituitary adenoma) had impacted significantly on the outcome. Multivariate analysis showed duration of blindness < 12 weeks, apoplexy, sellar extension have significant effect on outcome.

Frank G et al¹⁴⁰ in their analysis on endoscopic approach to pituitary tumours had found that vision improved in 90 patients (58.4%). No variations in the visual field were observed in 6 patients (3.8%), while in 2 patients (1.2%), we had a worsened visual capacity. They did not study the factors affecting the outcome and their study did not include detailed analysis on vision involvement.

Ashraf jallu et al¹ in his study on suprasellar meningioma had studied 70 cases. Mean age was 45.6 years and 87% were female. Deterioration of vision was main complaint in 85.7% and duration of symptoms were 4-12 months. Thirty-one [44.2%] of the 70 patients were found to have no light perception in one or both eyes. Only 10 patients (14.2%) had a total preservation of vision. Total excision of tumour was possible in 64.6% of cases, A total of 11 patients showed improvement in visual function, but most of them had presented with visual function better than counting fingers at 1 metre. However, their study lacked the association of factors, duration of follow up and small sample size.

Ahmed galal et al⁵ had studied visual outcomes and associated factors over 4 years in patients surgically treated for suprasellar meningioma. 21 patients were included in this retrospective study. Mean age was 43 years. All patients had visual acuity loss and visual field defects. Symptom duration ranged from 2 to 36 months. Tumour removal was complete in 17 patients, and subtotal resection was performed in four patients. The follow-up duration ranged from 24 to 48 months. 12 patients (60%) had achieved visual improvement, whereas vision was unchanged in eight patients (40%). None of the patients had visual deterioration during their follow-up. A univariate analysis of clinical and surgical parameters thought to be related to visual outcome

showed that the duration of symptoms, preoperative visual status, tumour size and adherence to the internal carotid arteries and/or anterior cerebral artery had a significant impact on visual outcome.

Mario monterio et al⁶ had investigated clinical and MRI findings that were predictive of visual loss in patients with pituitary adenomas and visual recovery after surgery. Visual loss was found in 47 eyes; 35 had optic atrophy (subtle in 9, moderate in 14, and severe in 12). complaint of visual decline was present in 47 eyes (78.3%). Of the 47 eyes visual decline was present for more than 2 years in 17 eyes (36.2%), between 6 months and 2 years in 25 (53.2%) and for less than 6 months in 5 (10.6%). Fundus examination was normal in 25 eyes (41.7%) and optic atrophy in the others. Acuity was worse than 20/30 in 24 eyes (40%) and 20/30 or better in 36 (60%). All 60 eyes had VF loss before treatment, most prominently in the upper temporal quadrant. The chiasm was 17.3 (SD 6.2, range 10-34) mm above reference line on sagittal and 21.8 (SD 8.3, range 12-39) mm on coronal images. Tumour size correlated with the severity of field defect. Field improvement occurred in 80% of eyes after treatment. The degree of optic atrophy, visual loss, and tumour size were significantly associated with improvement after treatment. They concluded that the best predictive factor for visual loss was tumour size, and factors related to visual recovery were the degree of optic atrophy, the severity of VF defect, and the tumour size.

Dutta et al¹⁶¹ examined the visual outcome after resection of pituitary adenomas in patients having preoperative visual defects. They collected information on visual outcome, visual acuity, and visual fields at discharge, at 3 months, and at 12 months. They found that bitemporal hemianopia was most common type of field defect (47.6). A large majority of the operations were transsphenoidal (96.9%). The authors found that postoperative visual outcome was directly proportional to the preoperative VA, and that patients with better preoperative Visual Acuity had better visual outcome. 82% of the patients with less severe visual deficits had improvement in visual status, whereas only 10% of the patients with no perception of light improved. They also noted that patients had earlier recovery of vision if the duration of symptoms was shorter. However, the ultimate outcome was good at the end of 1 year irrespective of the duration of symptoms. They also found that 93.2% of the patients had better vision at 1 year

D Zevgaridis et al¹⁶² in their study on 62 patients with suprasellar meningioma noted that visual deterioration was the first symptom. Median age was 54 years, mean duration was 7 months, mean follow up was 5.2 years. They found that vision improved in 39 (65%) patients, in 11 (18%) it

was unchanged, and worse in 10 (17%). Visual prognosis was favourably affected by age under 54 years ($p < 0,025$), duration of symptoms of less than seven months ($p < 0,037$), and the presence of an intact arachnoid membrane around the lesion ($p < 0,001$). Severe preoperative loss of visual acuity ($< 0,02$) appeared to be an unfavourable prognostic factor ($p < 0,047$).

In study by K K Gnanalingam et al¹⁶³ on 41 patients with pituitary adenoma found that 36/41 patients (88%) presented with a visual disturbance. Mean duration of symptoms was 94 weeks; 12 (29%) had optic atrophy at presentation. Impairment of VF was greatest in the upper temporal quadrant, followed by the lower temporal. Visual Field recovery was progressive and apparent even at the five year follow up ($p < 0,001$). Visual Field returned to normal in 35% of eyes, improved in 60%, and remained unchanged in 5%. Patients whose Visual field returned to normal had a shorter duration of symptoms (16 (5) v 137 (56) weeks; $p < 0,05$), better preoperative visual acuity ($p < 0,05$), and a smaller degree of impairment in preoperative lateral quadrant VF ($p < 0,01$) than those whose VF only improved. On multivariate analysis, the only predictive factor for VF recovery was the degree of impairment in VF preoperatively. They concluded that Transsphenoidal surgery for pituitary macroadenoma results in a progressive recovery of VF in 95% of patients. The extent of the VF recovery is mainly dependent on the preoperative VF deficit, which emphasises the need for early intervention in these patients. The main limitation was small sample size, retrospective study.

Ricardo Komotar et al¹⁵³ in his metaanalysis on craniopharyngiomas found 88 studies, involving 3470 patients, were included. Patients treated via an endoscopic or transsphenoidal approach had a greater rate of visual disturbance at presentation ($P = 0,003$) There were significantly greater rates of gross total resection in the endoscopic cohort and transsphenoidal cohort than in the open cohort ($P = 0,003$). The endoscopic cohort had a significantly greater rate of gross total resection (66.9% vs. 48.3%; $P < 0,003$) and improved visual outcome (56.2% vs. 33.1%; $P < 0,003$) compared with the open cohort. They concluded that endoscopic approach is safe and effective for the removal of craniopharyngiomas and has good visual outcomes.

Tabaee A et al noted in their study that mean duration of surgery was 177 min and was longer in patients with larger tumour size ($P = 0,03$) and presentation with visual symptoms ($P = 0,02$) in univariate analyses. The median duration of hospitalization was 3 days and was longer in patients with larger tumours ($P = 0,0005$). Gross tumour removal was achieved in 89%. Tumour size correlated with extent of tumour removal. High rates of hormonal control (90%) and improvement in visual symptoms (92%) were noted. Of the 24 patients with visual symptoms prior to surgery,

22 (92%) reported either complete resolution or significant improvement after the procedure. No patients experienced worsened visual acuity, visual fields, or new onset diplopia.

Illustrative cases

Case 1:

56year female patient known case of hypothyroidism presented with complaints of diminution of vision in left eye for 6 months. She also complained of headache associated with vomiting. On examination she was conscious alert with normal higher mental function. Left eye visual acuity was 3/36 and right eye was 6/6. Pupils examination showed left RAPD and right was reactive. Fundus examination revealed pale disc in left eye. Extra-ocular movements were full. Visual fields were suggestive of right eye normal field and left eye could not be done due to poor acuity. No other cranial nerve deficits. Bulk and tone of muscles normal in all 4 limbs. Power grade 5/5 in all 4 limbs. Reflexes were normal. Plantars flexor. No sensory deficits. No cerebellar signs. Systemic examination unremarkable. On investigations blood parameters, hormone profile was normal. MRI done showed extra axial lesion 30x 30 x 32mm with wide based attachment over roof of sphenoid compressing bilateral basifrontal lobes, superiorly compressing optic chiasma and hypothalamic region suggestive of suprasellar (planum sphenoidale) meningioma. She underwent left Pterional craniotomy and gross total excision. Postoperatively on follow up on 6 months her visual acuity and visual field in left eye improved significantly. Histopathology report was meningothelial meningioma.

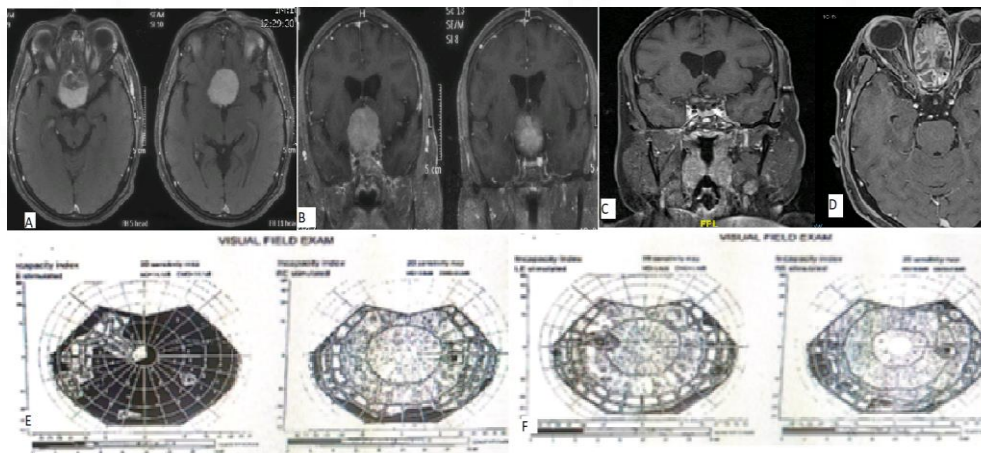


Figure 7 - Preoperative and postoperative MRI of brain and visual field testing. A- Preoperative axial T1 contrast MRI showing suprasellar meningioma arising from planum sphenoidale. B-

Coronal T1 contrast MRI showing same. C- Post operative coronal T1 contrast MRI at 6 months showing no residual lesion. D- Axial T1 contrast MRI showing the same. E- Preoperative visual field showing severe visual deficit in left eye and right eye was normal. F- post operative visual field showing improvement in visual field in left eye and right eye was normal.

Case 2-

53yr old male patient without any co-morbidities presented with complaints of headache and decreased vision in both side fields and frequent bumping of objects that are present in side field. On examination patient was conscious, alert with stable vital signs. Higher mental functions were normal. Visual acuity- 6/9 in right eye, 6/36 in left eye, Visual fields- bitemporal hemianopia +. Fundus- normal Extraocular movements- full. Pupils bilaterally equal and reactive to light. No other cranial nerve involvement, No motor/sensory deficits. No cerebellar signs. Systemic examination unremarkable. Blood parameters were normal, hormone profile showed low cortisol and T4. MRI brain showed Sellar suprasellar lesion of size 4.6 x 3.1 x 3.9 cm. Gland, chiasma, stalk not separately seen. T2 hyperintense, T1 isointense lesion with contrast enhancement. He underwent endoscopic transnasal transsphenoidal gross total excision. Post operatively on follow up on 6 months his field defect normalized. Histopathology was suggestive of non-functioning pituitary adenoma.

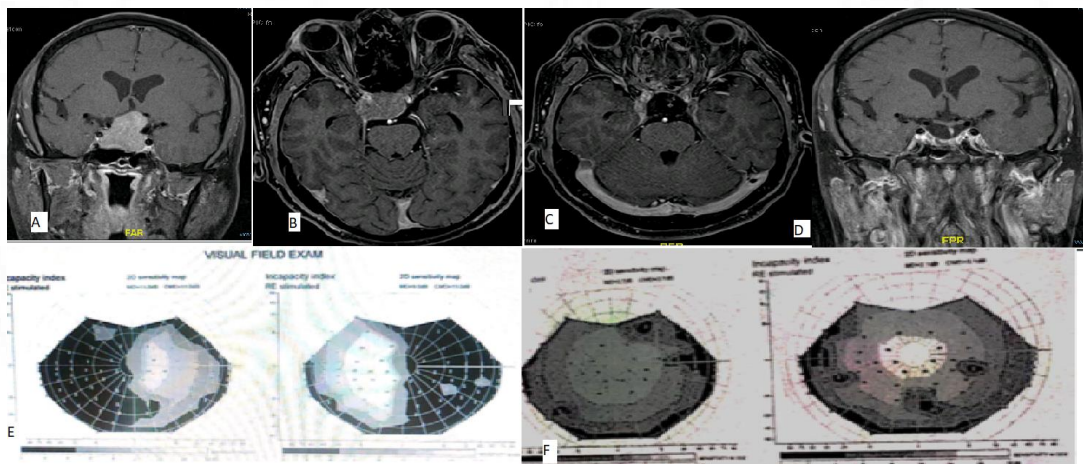


Figure 8 - Preoperative and postoperative MRI images of brain with visual field testing. A- Coronal T1 contrast MRI showing contrast enhancing pituitary macroadenoma. B- Axial T1 contrast showing pituitary macroadenoma. C- Post operative axial T1 contrast MRI at 6 months showing no residual lesion. D- Post operative coronal T1 contrast MRI showing at 6 months no residual tumor. E- Preoperative visual field testing showing bitemporal hemianopia. F- Postoperative visual field

testing at 6 months showing complete resolution of bitemporal defect.

Case 3-

This 45-year-old patient, without any comorbidity presented with headache x 3 years and diminution of vision in left eye x 2 months. On examination patient was conscious, alert with stable vital signs. Higher Mental Function was normal. Extra-ocular movements full. VA-6/12 right eye, Hand movement in left eye. Fundus - papilledema in right eye, primary optic atrophy in left eye. Pupils bilaterally equal and reactive Extraocular movement was normal. No other cranial nerve deficits. Bulk and tone of muscles was normal in all 4 limbs. Power grade was 5/5 in all 4 limbs. Reflexes were normal. Plantars were flexor. No sensory deficits. No cerebellar signs. Systemic examination unremarkable. Blood parameters and hormone profile were essentially normal. MRI done showed well defined peripherally enhancing lobulated cystic lesion in suprasellar cistern measuring approximately 29x30x20mm. Pituitary gland was seen separately from the lesion. It was hyperintense on T2 and FLIR and heterogenous on T1 with fluid level within. No diffusion restriction. Blooming seen within the lesion. Superiorly, the lesion was displacing the optic nerves and chiasm, laterally it was abutting the supraclinoid ICAs bilaterally. She underwent right pericoronal parasagittal craniotomy interhemispheric approach and near total excision. Histopathology was suggestive of adamantinomatous craniopharyngioma. Post operatively on follow up at 6 months overall there was no improvement in vision of left eye and right eye vision deteriorated to 6/36 and temporal field defect was seen.

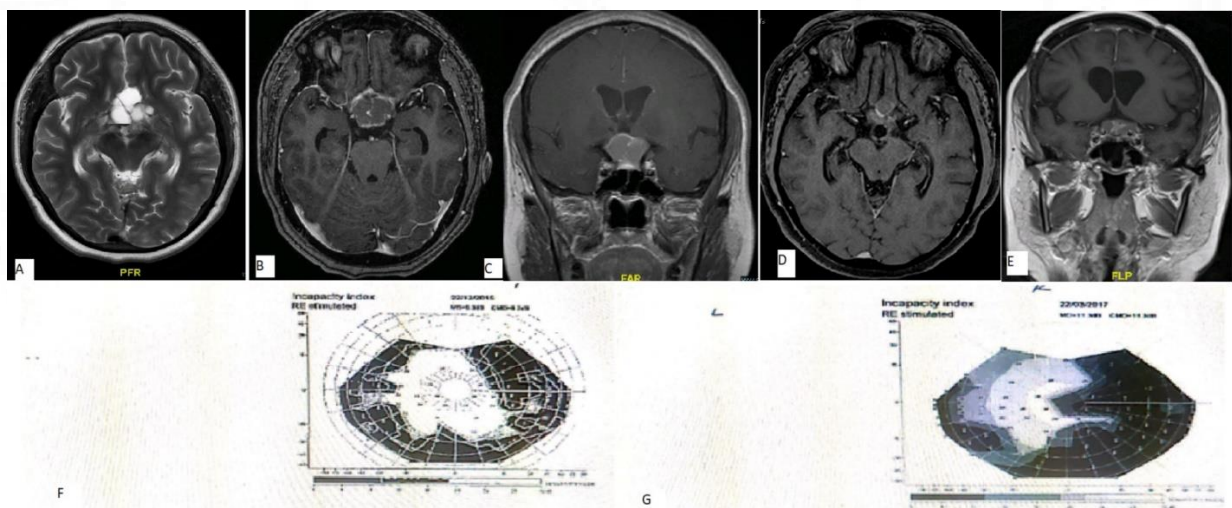


Figure 9 - Preoperative and postoperative MRI of brain and visual field testing. A- T2 axial MRI showing solid cystic lesion in suprasellar area. B- Axial T1 contrast showing enhancing lesion in suprasellar region. C- Coronal T1 contrast showing the same. D- Post operative axial T1 contrast MRI showing residual lesion in suprasellar area. E- post operative contrast MRI showing the same.

F- Preoperative visual field testing showing poor visual field in left eye and peripheral constriction in right eye. G- Postoperative visual field testing showing same poor visual field in left eye and presence of temporal hemianopia in right eye.



AIM AND OBJECTIVES OF THE STUDY

To study: Post-operative visual status in patients with suprasellar tumours with pre-existing preoperative corrected visual deficit after surgical resection.

Objectives:

1. To study the clinical profile among patients with preoperative visual deficits and underwent surgical excision of suprasellar tumour.
2. To evaluate the visual profile on admission, at discharge and on follow up till six months of surgery and operative procedures.
3. To study the predictive factors on visual outcome after surgery.

MATERIALS AND METHODS

Design:

Retrospective hospital based observational study of patients with suprasellar tumours, in the Neurosurgery department of Sree Chitra Institute of Medical Sciences and Technology (SCTIMST). A formal clearance was obtained after the evaluation of the study by Institutional Ethics Committee (IEC).

Study Period: 5 years (January 2015 and December 2019)

Funding: There were neither external sources of funding nor conflicts of interest in the current study.

Ethical Committee Clearance - The stud protocol was presented to the SCTIMST ethics committee and scrutinized at various angles regarding aim of the study and the safety of the patients involved in it. The study was started after the formal clearance from the IEC.

Methodology

Patients with suprasellar tumours with either unioocular or binocular blindness (no perception of light) at admission, who underwent surgical removal of the tumour in the department of Neurosurgery between January 2015 and December 2019, with 6 months follow up data were included in the study. The clinical and radiological details at the time of admission, at the time of discharge from hospital and follow-up details after discharge were retrospectively analysed from the hospital records. Data regarding age; gender; duration of symptoms; mode of presentation; and visual acuity and visual field status at admission, discharge, and follow-up were collected from the files. Visual acuity was recorded using the Snellen's chart. Visual field charting was performed using Goldman perimetry. Blindness was diagnosed when the patient had corrected visual acuity less than 3/60 as per WHO classification (annexure A) in one or both eyes. Imaging investigations done at that time included CT scanning, MR imaging, or both. The patients underwent either transcranial or transsphenoidal surgery depending on the tumour type, tumour location, and imaging findings. A few patients had postoperative complications such as

hydrocephalus or cerebrospinal fluid leak, which merited another procedure which was also evaluated. Visual improvement after surgery was defined as when the patient improved to perception of light or better vision or better visual field at discharge from the hospital and on follow up. The multifactorial effect of variables such as age, sex, duration of visual decline, duration of blindness, extent of resection, and histopathology was studied on the visual outcome.

Inclusion criteria

- Patients with suprasellar tumours of age >15 years and <60 years, of either gender with either unocular or binocular visual field deficit and having corrected preoperative visual acuity less than 6/6 (i.e. from 6/9 to No light perception),

No gender, class, caste, ethnic or racial considerations was used as inclusion or exclusion criteria.

Exclusion criteria

- Patients having suprasellar tumours without any visual deficit
- Patients with inadequate data
- Unfit patients for ocular examination.

Investigations

Mandatory preoperative endocrinologic assessment and imaging investigations were done including CT and MRI of the brain. Preoperative MRI was done for evaluation of the size of the pituitary adenoma and a CT scan was also done to preoperatively study nasal fossa, sinus, and skull base anatomy and for surgical navigation guidance. The measurement of tumour diameters - width, length and height, and the formula $0.5 \sim \text{width} \times \text{length} \times \text{height}$ also provided an adequate estimation of the tumour volume which was symmetrical. Tumour which is irregular in shape volume can be calculated based on MRI measurements, using an ellipsoid model (volume = $\frac{4}{3} \times \pi \times \frac{1}{2} d_1 \times \frac{1}{2} d_2 \times \frac{1}{2} d_3$) where d1, d2 and d3 are the maximum tumour diameter in the orthogonal spatial axes.⁷

Ophthalmological examination

Visual acuity

Visual acuity is an indicator of retinal function and is an appreciation of form sense. Patient was asked to read Snellen's test types at 6 m line and visual acuity was recorded, if one can't see top line they were asked to move towards the chart and distance at which he they were able to read and was recorded as 5/60,4/60,3/60,2/60. If they were not able to read at 1m,one was asked to

count finger and if they cannot then they were asked to appreciate hand movements and if not able to appreciate then perception of light was tested.

Visual field

The normal visual field is superiorly 50°, nasally 60°, inferiorly 70° and temporally 90°. Perimetry with red colour object is particularly useful in chiasmal compression. Perimetry is the procedure of choice. Automated perimetry test visual field by static method which involves presenting stimulus at a fixed position for a present duration with varying luminance. All the patients had a visual evaluation preoperatively and on discharge, one month, 3 months and 6 months postoperatively including measurement of visual acuity (VA) and/or visual field (VF)

A change in vision for each effected eye was noted as Improved (I)/ Unchanged (U)/ Deteriorated (D), after comparing preoperative VA and visual field with VA and visual field on discharge, 1 month, 3 months and 6 months follow up. The final visual outcome i.e. Improved or Not improved was assigned to each patient, if VA or visual field of either eye improved or if it remained unchanged/ deteriorated, respectively.

The data were entered in SPSS (Statistical programme for social sciences) version 17. Data was checked again to avoid any error. Frequencies with percentages were computed for categorical variables like sex, ocular involvement, operative procedure, tumour type and final visual outcome (Improved or Not improved), whereas mean \pm S.D was utilized for numerical variables like age and tumour size. Stratification was done with regards to age, sex, ocular involvement, operative procedure, and tumour type to control the effect on these outcome variables. Through Chi-square test, p-value ≤ 0.05 was taken significant. Multivariate logistic regression analysis was done to see the effect of the variables on the outcome and p value < 0.05 was taken significant

RESULTS

Age

Table 2 - Age distribution

Age (Years)	Number	Percentage
≥ 15 – 30	48	11.0
31 – 45	213	49.0
46 - 60	174	40.0
Total	435	100.0

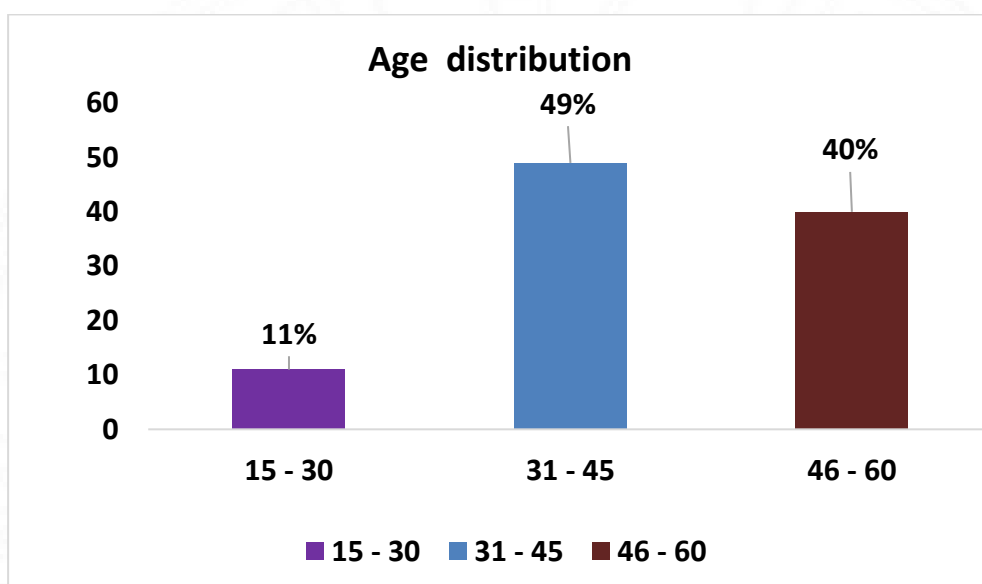


Figure 10- Showing age distribution among the participants

The maximum number of patients were in the age group of 31-45 years 213 patients out of 435 (49%) followed by 46-60 years 174 patients out of 435 (40%) and minimum in 15-30 years of age 48 out of 435. (11%)

Gender

Table 3 - Gender distribution

Gender	Number	Percentage
Female	216	49.7
Male	219	50.3
Total	435	100.0

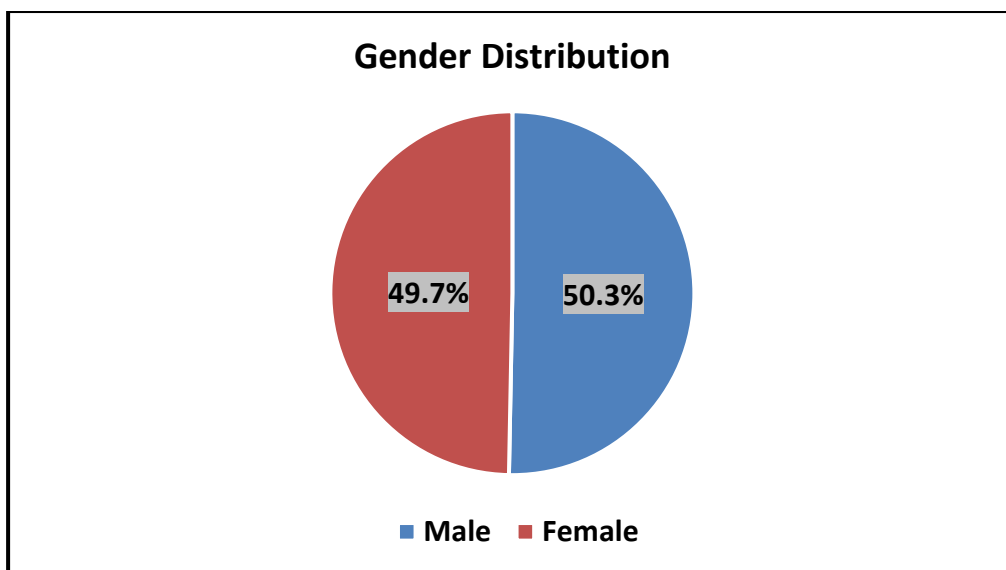


Figure 11 - Gender distribution

There were 219 males and 216 females (male/ female ratio 1:1.01).

Visual deficit

Table 4 - Visual deficit (Unilateral and bilateral affection)

Eyes	Number affected	Percentage
One eye	62	14.26
Both eyes	373	85.74
Total	435	100

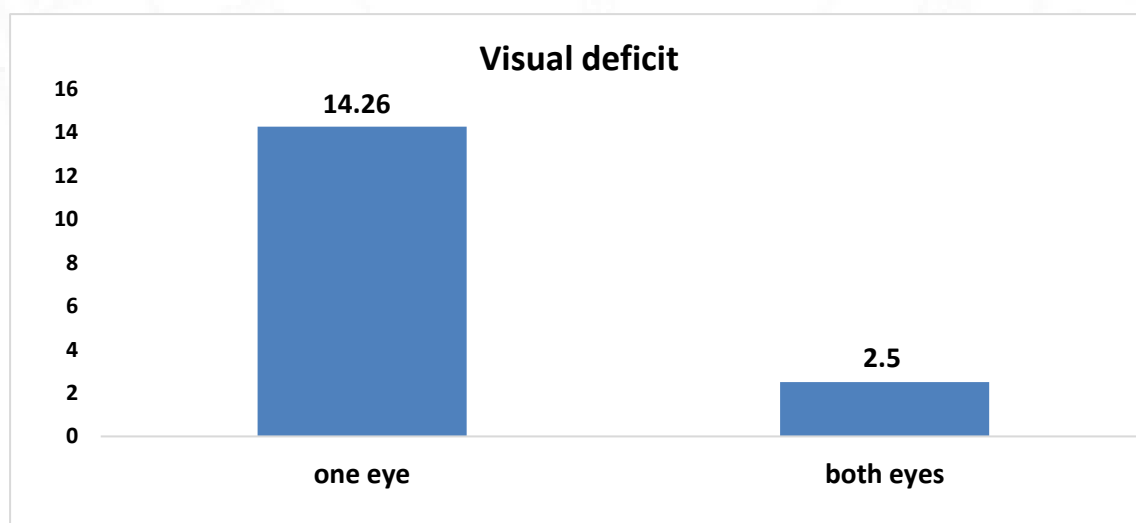


Figure 12 - Visual deficit (Unilateral and bilateral affection)

Visual decline was present in all patients, leading to reduced visual acuity or visual field in 1 or both eyes. Of 435 patients, 373 patients (85.74%) had binocular visual decline and the remaining 62(14.26%) had unioocular visual decline.

Visual acuity deficit

Table 5 - Severity of decline in visual acuity in both eyes

WHO Classification	Right eye	Left eye	Total
Mild/normal	288(66.2)	257(59.1)	545(63)
Moderate	85(19.5)	110(25.3)	195(22)
Severe	8(1.8)	3(0.7)	11(1)
Blindness	54(12.4)	65(14.9)	119(14)
Total	435	435	870

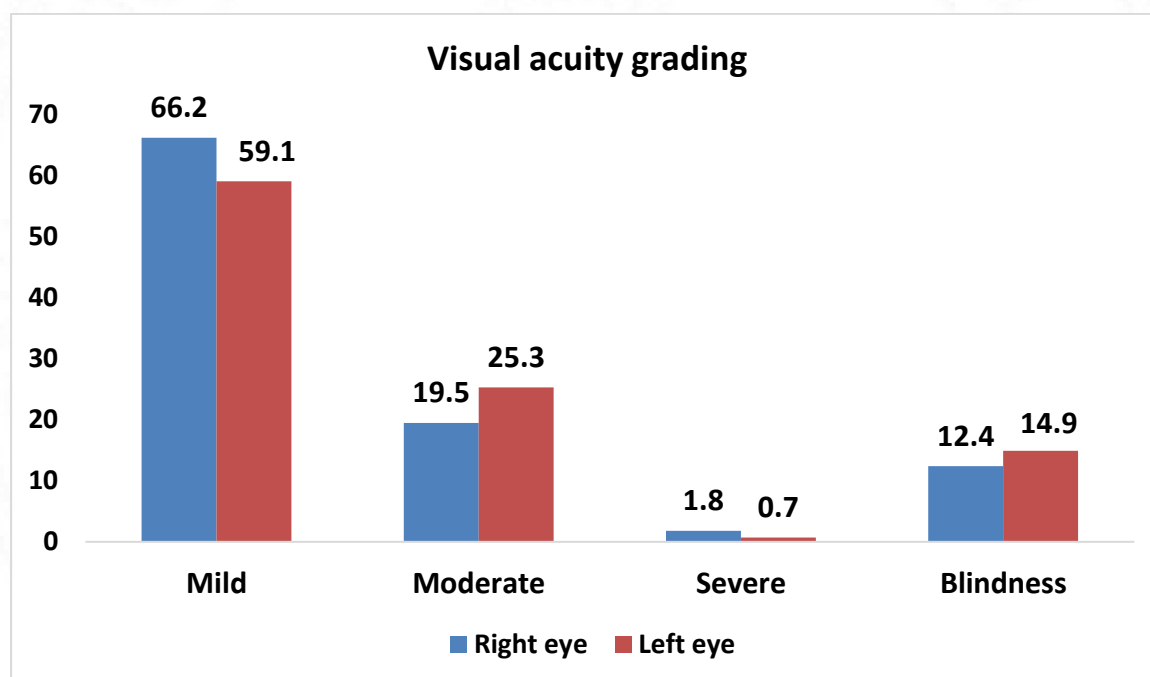


Figure 13 - Severity of decline in visual acuity in both eyes

According to WHO classification of visual decline in visual acuity, 435 patients (870 eyes) were classified into 4 groups. Normal/mild visual impairment included eyes with corrected visual acuity not worse than 6/18 on Snellen chart which was 545 eyes out of 870 eyes (63%). Moderate visual impairment was defined as visual impairment not worse than 6/60 on Snellen chart which was seen in total 195 eyes out of 870 eyes (22%). Severe visual impairment was defined as visual

acuity not worse than 3/60 which included 11 eyes out of 870 eyes (1%). Blindness was defined as visual acuity worse than 3/60 and included finger counting, hand movements and perception of light to no perception of light. This included 119 eyes out of 870 eyes (14%).

Visual Field deficit

Table 6 - Types of visual field defect in both eyes

Field defect	Number	Percentage
Enlargement of blind spot or altitudinal defect	50	11.5
Quadrantanopia Unilateral or bilateral	18	4.1
Hemianopia Unilateral or bilateral	252	57.9
Others	115	26.4
Total	435	100.0

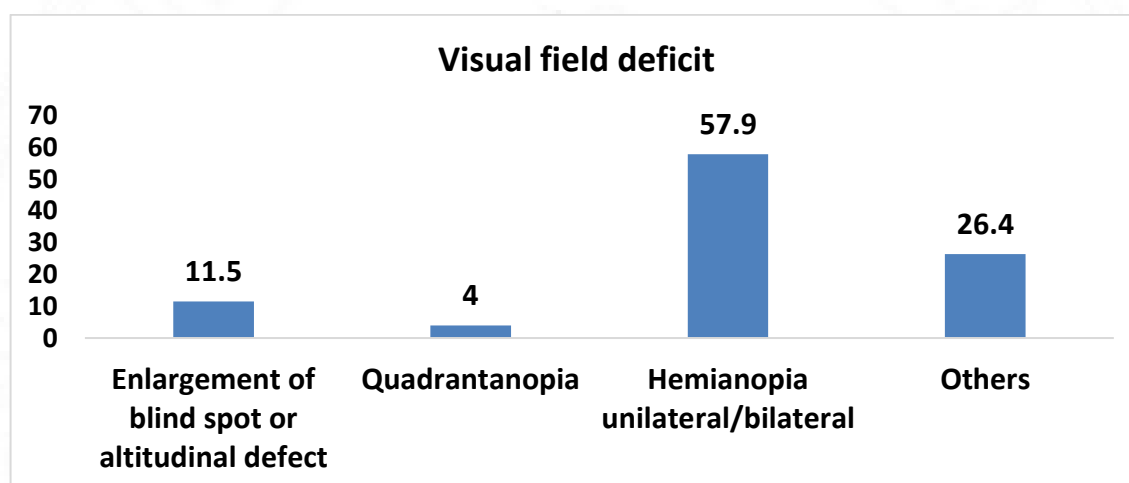


Figure 14 - Types of visual field defect in both eyes

Enlargement of blind spot or altitudinal defect was seen in 50 patients out of 435 (11.5%). Unilateral or bilateral quadrant defect included superior or inferior quadrant defect in temporal or nasal side in both eyes and was seen in 18 patients out of 435 (4.1%). The most common field defect was hemianopia and included unilateral or bilateral hemifield defect on nasal or temporal or both sides in both eyes and was seen in 252 patients out of 435 (57.9%). Out of these bitemporal hemianopia was the most common seen in 195 patients. Others included all patients with 3 quadrant field deficit, blindness, severe peripheral constriction of visual fields and patients in whom visual could not be tested because of poor visual acuity. This included 115 patients out of 435 (26.4%).

Associated symptoms

Table 7 - Associated symptoms in the study population

Symptoms	No. of patients	Percentage
Headache	265	60.91
Pituitary dysfunction	106	24.36
Seizures	18	4.13
ICP	20	4.59
Extraocular palsy	38	8.7
Others	4	0.9

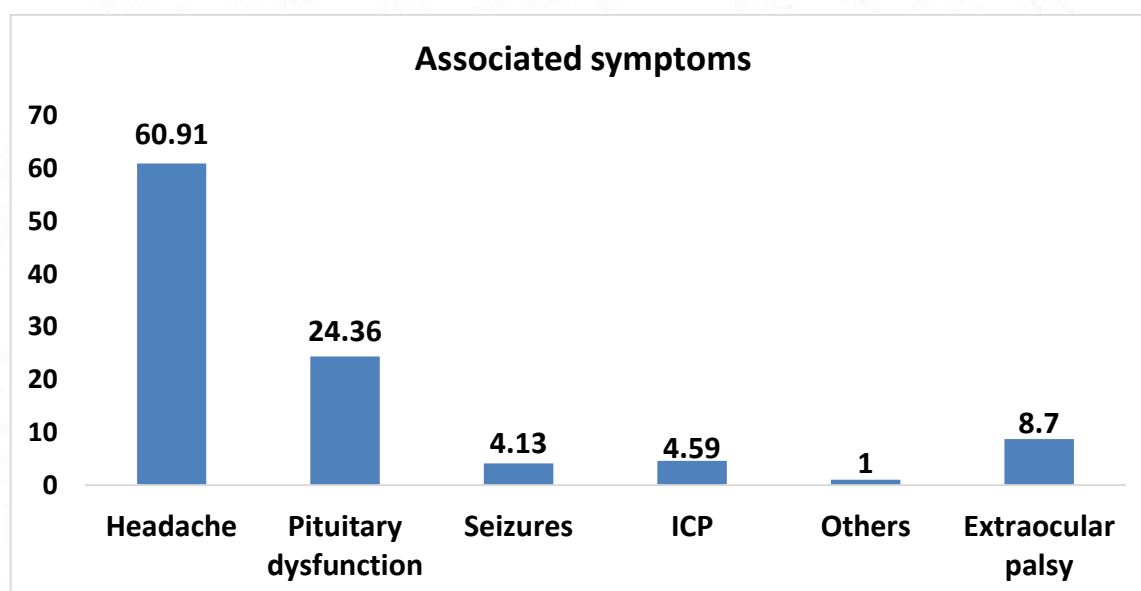


Figure 15 - Associated symptoms in the study population

Of 435 patients, 265 (60.91%) patients had headache associated with visual deficit. 106 patients (24.36%) had associated pituitary dysfunction. 18 patients (4.13%) presented with history of seizures associated with visual dysfunction. 20 patients (4.59%) had symptoms of raised intracranial pressure. About 4 patients had history of memory or behavioural disturbances associated with visual deficit. 38 patients (8.7 %) had associated extraocular palsy.

Fundus

Table 8 - Fundus examination findings

Fundus finding	Number of eyes	Percentage
Optic atrophy	121	13.9
Papilledema	147	16.89
Temporal pallor	358	41.14
Normal	244	28.04

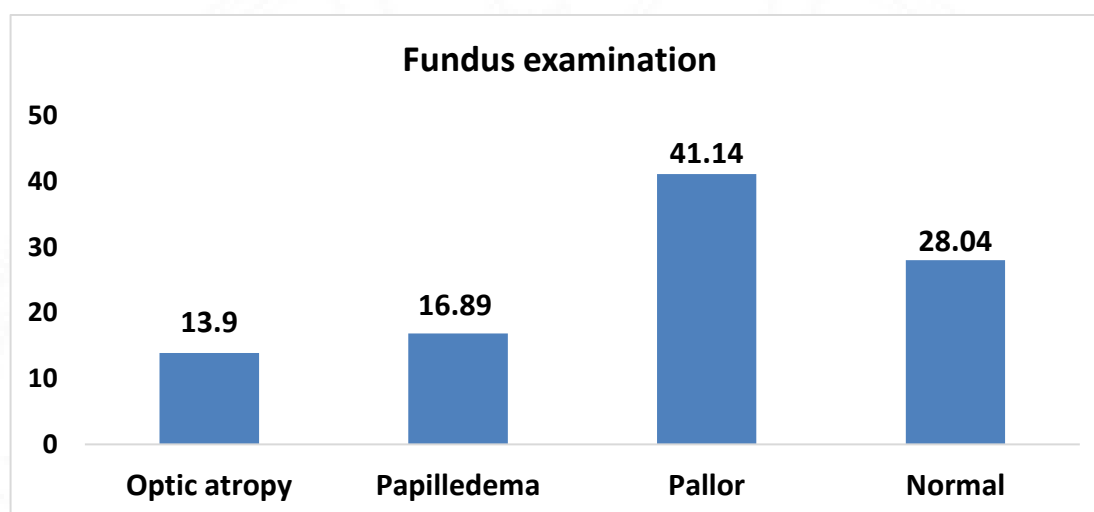


Figure 16 - Fundus examination findings

Out of 870 eyes, 121 eyes (13.9%) had evidence of optic atrophy on funduscopy examinations in the affected eye at presentation; 147 eyes (16.89%) had papilledema, 358 eyes (41.14%) had Temporal pallor and normal fundus was seen in 244 eyes (28.04%).

Duration of visual decline

Table 9 - Duration of visual decline before surgery

Duration	Number	Percentage
<6 months	178	40.9
6 – 12 months	208	47.8
>12 months	49	11.3
Total	435	100.0

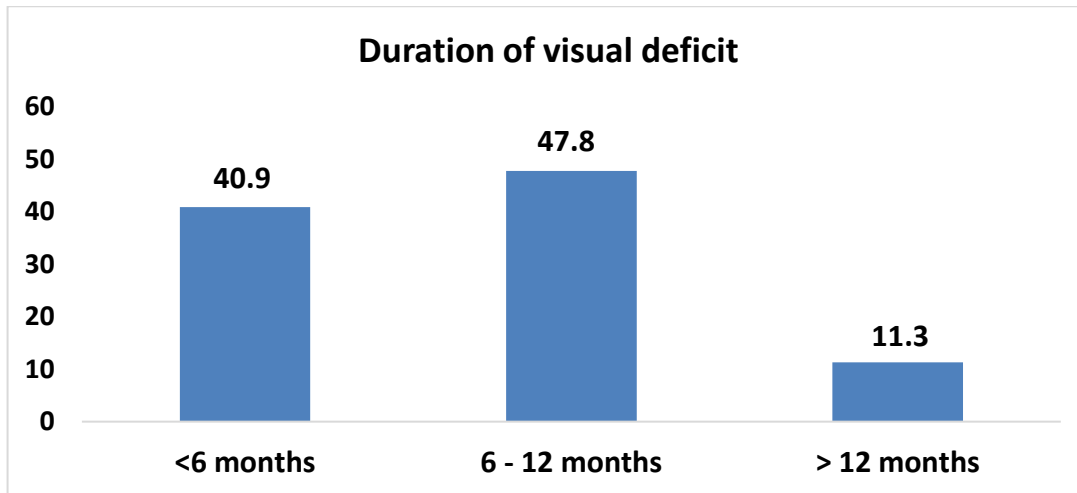


Figure 17 - Duration of visual decline before surgery

The duration of visual decline ranged from 1 month to 44 months (mean 10.29 ± 4.04 months). 133 patients (40.9%) had been having visual acuity decline for < 6 months and 277 (47.8%) had it for 6 months – 12 months and 11.3% had for more than 12 months before admission. The duration of blindness in cases of binocular blindness was based on the time of onset of blindness in the first eye.

Surgical approach

Table 10 - Types of surgical approaches

Surgical approach	Number	Percentage
Endoscopy	292	67.1
Transcranial	143	32.9
Total	435	100.0

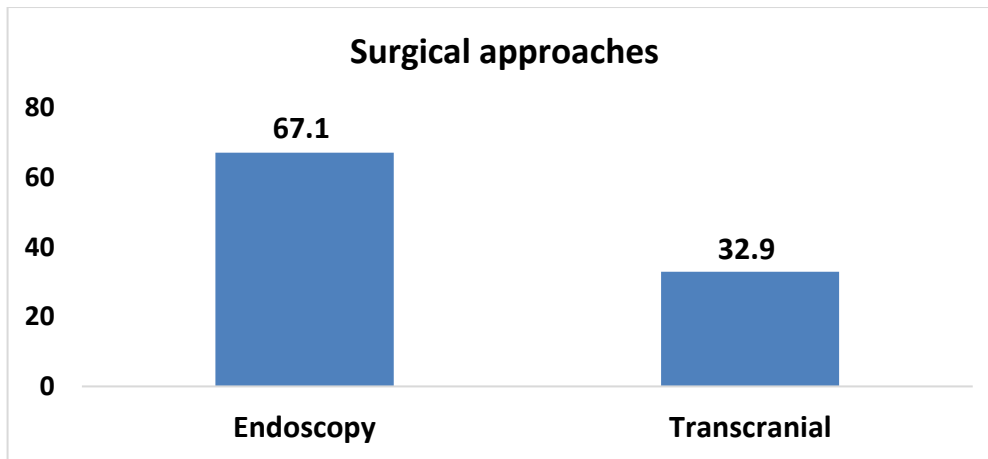


Figure 18 - Types of surgical approaches

All patients underwent surgical tumour decompression. The surgical approach depended on the location of the tumour, its extension, and the tumour characteristics noted on preoperative images. Transsphenoidal decompression of the tumour was performed in 292 (67.1%) patients and craniotomy was performed in 143 (32.9%) patients.

The craniotomies were Pterional in 119 patients, interhemispheric transcallosal interforncial approach in 21 and bifrontal approach was taken in 3 patients.

Endoscopic Extended transnasal transsphenoidal approach was taken in 32 patients

Extent of resection

Table 11 - Extent of resection of the tumour

Extent of tumour resection	Number	Percentage
Biopsy	4	0.9
Sub total	26	6
Near total	59	13.6
Gross total	346	79.5
Total	435	100.0

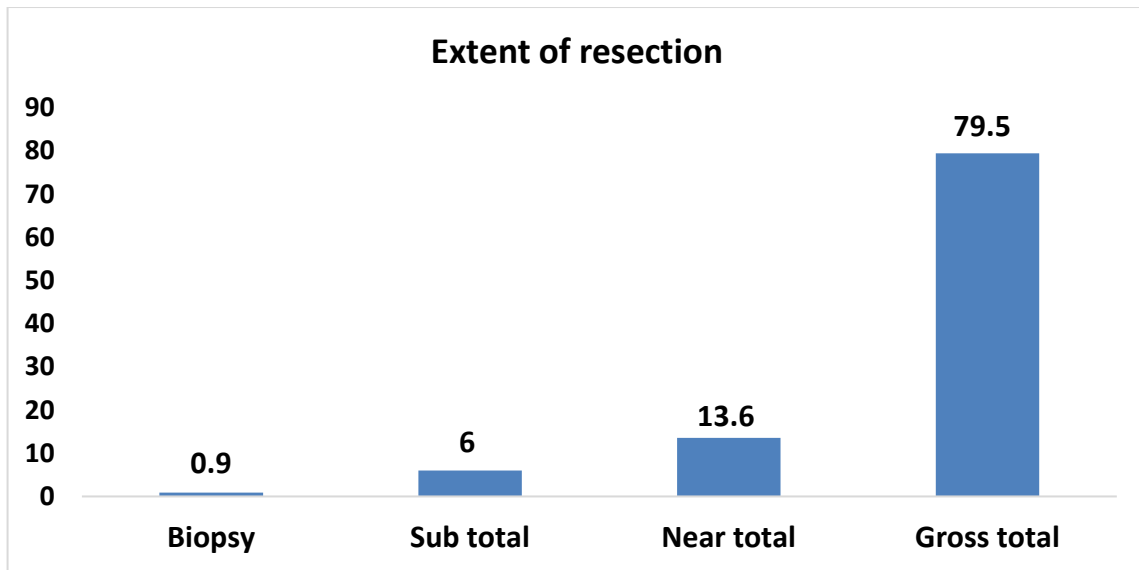


Figure 19 - Extent of resection of the tumour

Gross-total resection was performed in 346 (79.5%), near total in 59(13.6), subtotal in 26 (6%) and biopsy in 4(0.9%) patients

In 292 endoscopic approaches gross total excision was done in 273 (93.49%), near total in 13 (4.4%), subtotal in 6 (2.05%) and biopsy in 0 patients

In 143 transcranial approaches gross total excision was done in 103 (72.02%), near total in 24 (16.78%), subtotal in 12 (8.39%) and biopsy in 4 (2.79%) patients

Tumour histopathology

Table 12- Pathology of suprasellar tumours in the study population

Tumour pathology	Number	Percentage
Pituitary	284	65.3
Craniopharyngioma	74	17.0
Meningioma	61	14.0
Others	16	3.6
Total	435	100.0

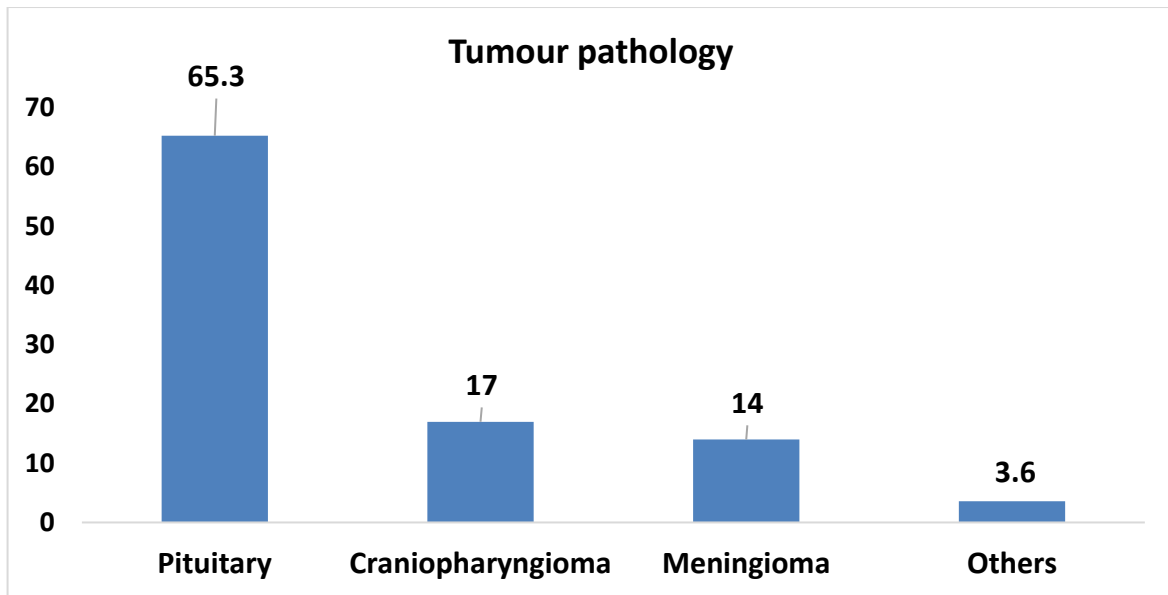


Figure 20 - Pathology of suprasellar tumours in the study population

Histopathological examination of tumour specimens revealed pituitary adenomas in 284 patients (65.3%), craniopharyngioma in 74(17%), and meningioma in 61patients (14%). The remaining patients had epidermoid cyst 2 (0.5), Ratke cyst 6 (1.4%), dermoid in 1 (0.2%), glioma in 2 (0.5 %), granulomatous hypophysitis in 1 (0.2), pineoblastoma in 1 (0.2%) and arachnoid cyst in 1 (0.2%) germ cell tumour in 2 (0.5%) patients

Visual Outcome

Table 13 - Improvement in visual deficit on discharge and follow up

Visual deficit	On discharge	1st month	3rd month	6th month
Improved	263(60.5)	313(72)	359(82.5)	397(91.3)
Worsened	36(8.3)	33(7.6)	15(3.4)	6(1.4)
Unchanged	136(31.3)	89(20.5)	61(14)	32(7.4)
Total	435	435	435	435

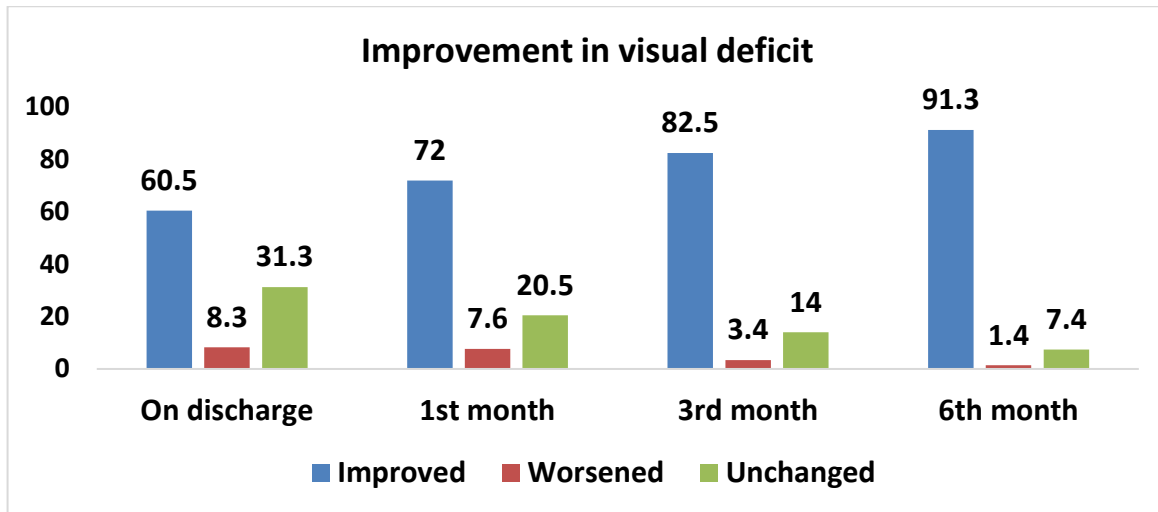


Figure 21 - Improvement in visual deficit on discharge and follow up

Outcome was analysed at discharge from the hospital and at follow-up at 4 weeks, 3 months, and 6 months. improvement in acuity was defined as perception of light to near-normal vision after surgery and improvement in visual field as documented by perimetry

Out of 435 patients on discharge visual deficit had improved in 263 (60.5%), remained same in 136 (31.3%) and worsened in 36 (8.3%)

Out of 435 patients on follow up at 1-month visual improvement was seen in 313 (72%). Out of the 36 patients who had worsened 3 improved and 33 (7.6%) remained the same. Out of 136 with unchanged vision 47 improved and 89 (20.5%) remained the same.

Out of 435 patients on follow up at 3 months visual improvement was seen in 359 (82.5%). Out of the 36 patients who had worsened initially 21 improved and 15 (3.4%) remained the same. Out of 136 with unchanged vision 75 improved and 61 (14%) remained the same.

Out of 435 patients on follow up at 6 months visual improvement was seen in 397 (91.3%). Out of the 36 patients who had worsened initially 30 improved and 6 (1.4%) remained the same. Out of 136 with unchanged vision 104 improved and 32 (7.4%) remained the same.

Visual field improvement

Table 14 - Improvement of visual field at 6 months

Visual field defect	6 months	
	Improved	Not Improved
Increased blind spot/altitudinal defect	38 (76%)	12 (24%)
Quadrantanopia Unilateral or bilateral	16 (88.9%)	2 (11.1%)
Hemianopia Unilateral or bilateral	239 (94.8%)	13 (5.2%)
Others	94 (81.7%)	21 (18.3%)
Total	387	48

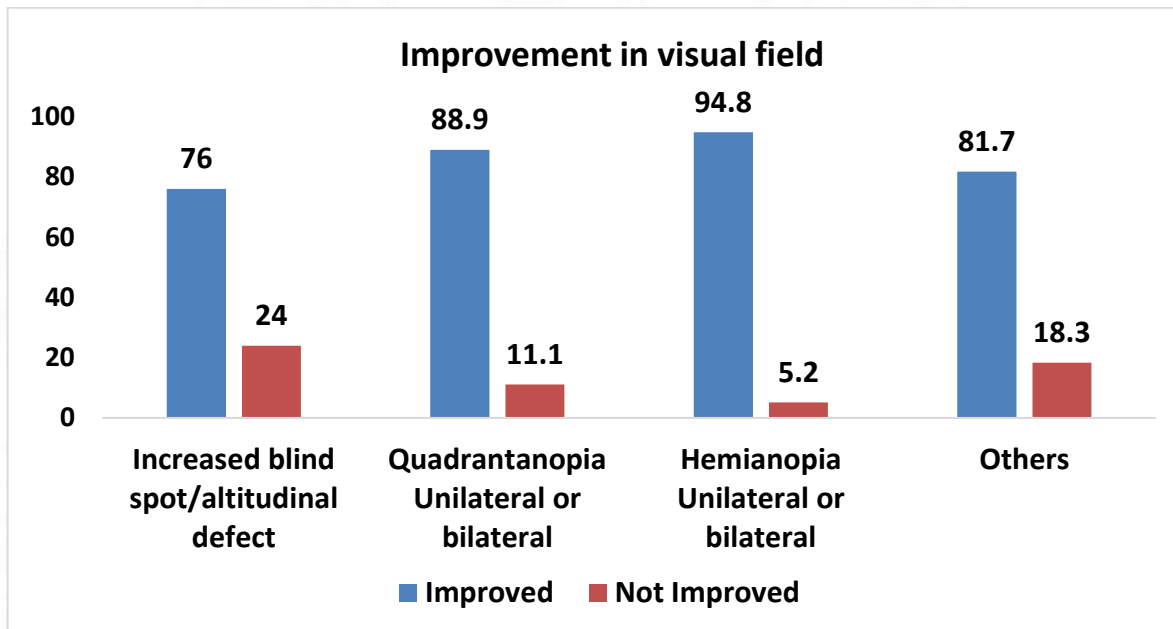


Figure 22 - Improvement of visual field at 6 months

94.8 % patients with hemifield defect had improved visual fields at 6 months of follow up followed by 88.9% improvement in quadrantanopia.

Improvement in visual acuity and visual field on follow up

Table 15 - Improvement in visual acuity and visual field on follow up

Variables	Improvement at follow-up		
	1 month	3 months	6 months
Visual acuity	164(52.39)	224(62.39)	299(75.31)
Visual field	250(79.87)	301(83.8)	387(97.48)

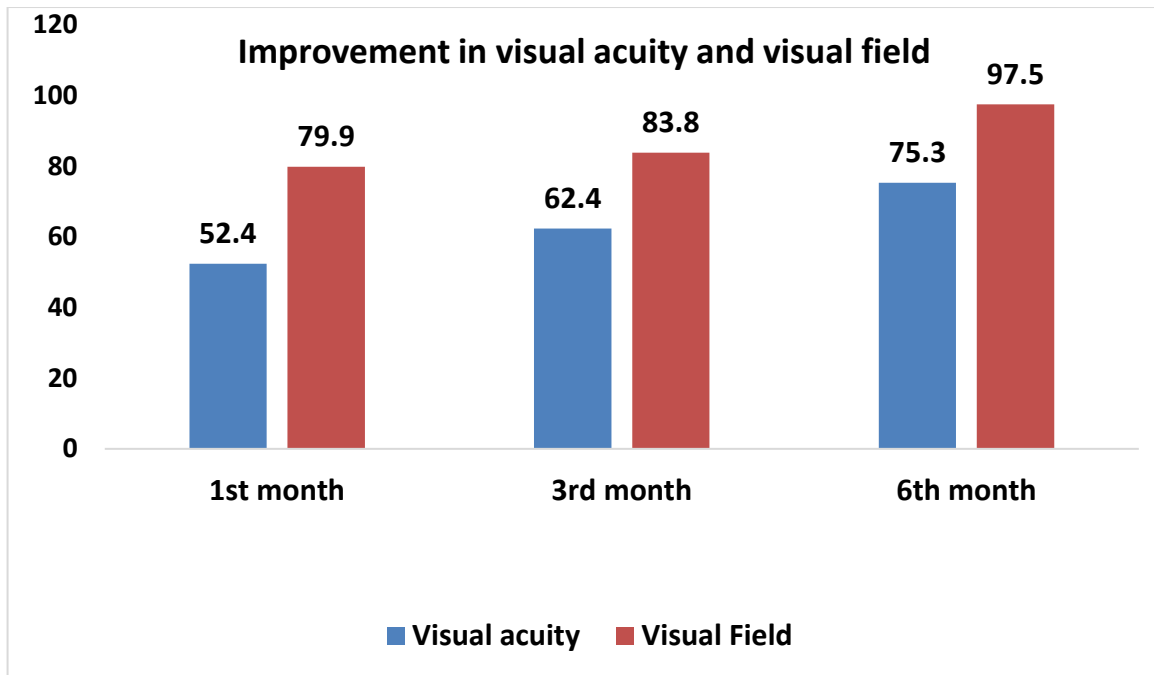


Figure 23 - Improvement in visual acuity and visual field on follow up

On follow up at 1 months among 313 improved patient's visual acuity improved in 52.39% and visual field in 79.87% of the total improvement seen

On follow up at 3 months among 359 improved patient's visual acuity improved in 62.39 % and visual field in 83.8% of the total improvement seen

On follow up at 6 months among 397 improved patient's visual acuity improved in 75.31 % and visual field in 97.48% of the total improvement seen

Visual improvement in study population with blindness

Table 16 - Visual improvement in study population with blindness

Side	No. of patients with blindness before surgery	Improvement at 6th month of follow-up	Percentage
Right	54	13	24.1
Left	65	32	49.2
Total	119	45	37.8

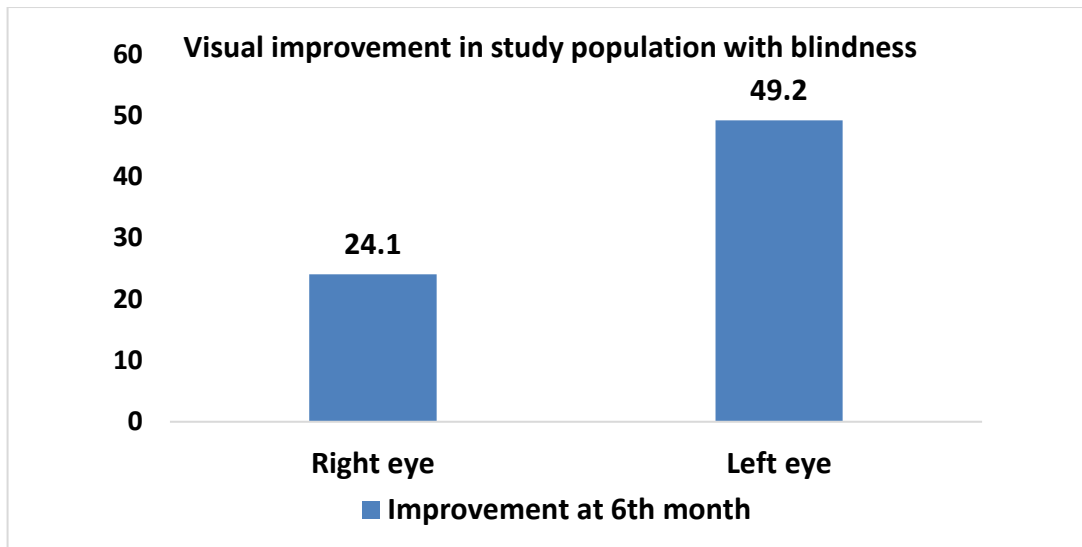


Figure 24 - Visual improvement in study population with blindness

Patients with blindness were seen in 119 eyes (14%)

Visual improvement was seen in 45 eyes (37.8%)

Complications

Table 17 - Complications after surgery for suprasellar tumours

Sl. No.	Complications	Yes (%)	No (%)
1.	CSF Leak		
a)	Endoscopic	52(17.8)	240(65)
b)	Transcranial	14 (9.7)	129 (34.9)
	Total	66	369
2.	Meningitis	12(2.8)	423(97.2)
3.	DVT	3(0.6)	432 (99.31)
4.	Seizures	11 (2.5)	424 (97.47)
5.	Wound infection	21(4.8)	414 (95.17)
6.	DI	123 (28.27%)	312 (71.72)
7.	Weakness	7(1.6)	428 (98.39)
8.	Haematoma	7(1.6)	428 (98.39)

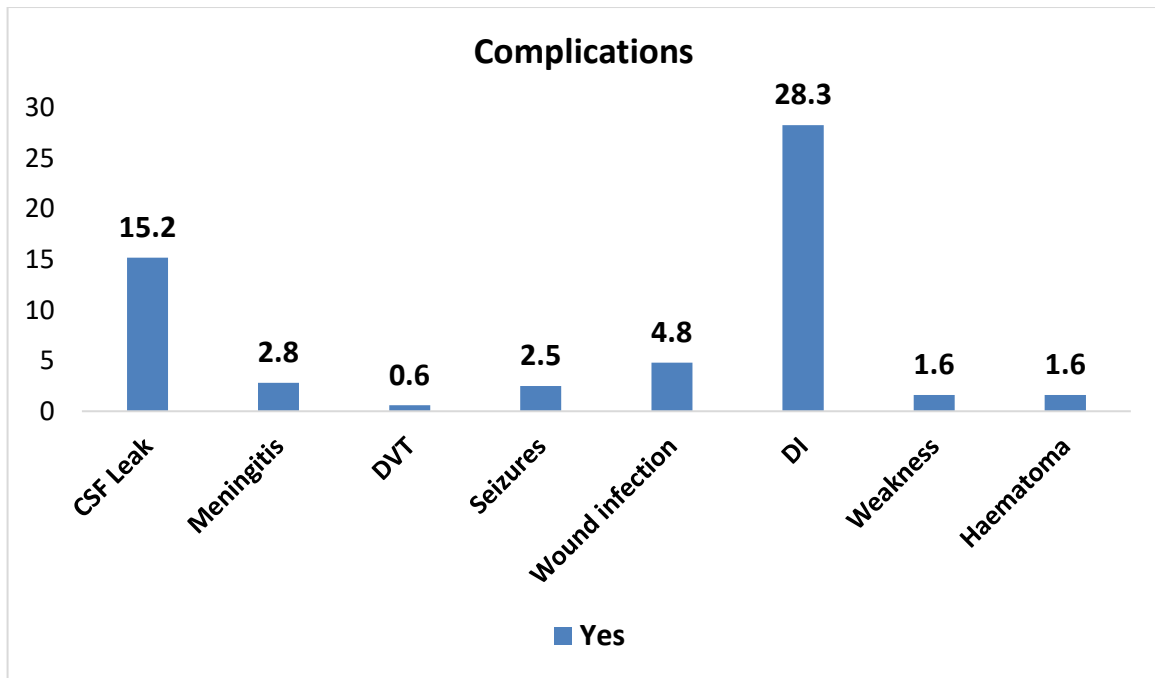


Figure 25 - Complications after surgery for suprasellar tumours

Of 435 patients in the study, CSF leak was present in 66 patients (15.2%) out of which 52 were seen in endoscopic and 14 (3.2) were seen in transcranial approach. Other complications like meningitis was seen in 12 patients (2.8%), seizures, wound infection in 21 patients (4.8%), hematoma including postoperative Extradural hematoma or operative site hematoma seen in 7 patients (1.6%), DVT seen in 3 patients (0.6%) and DI in 28.27%

Visual outcome in patients with recurrent disease

Table 18 - Visual outcome in patients with recurrent disease

Diagnosis	Vision improved	Vision not improved
Recurrent disease	24 (66.7)	12 (33.3)
Primary disease	373 (93.5)	26 (6.5)

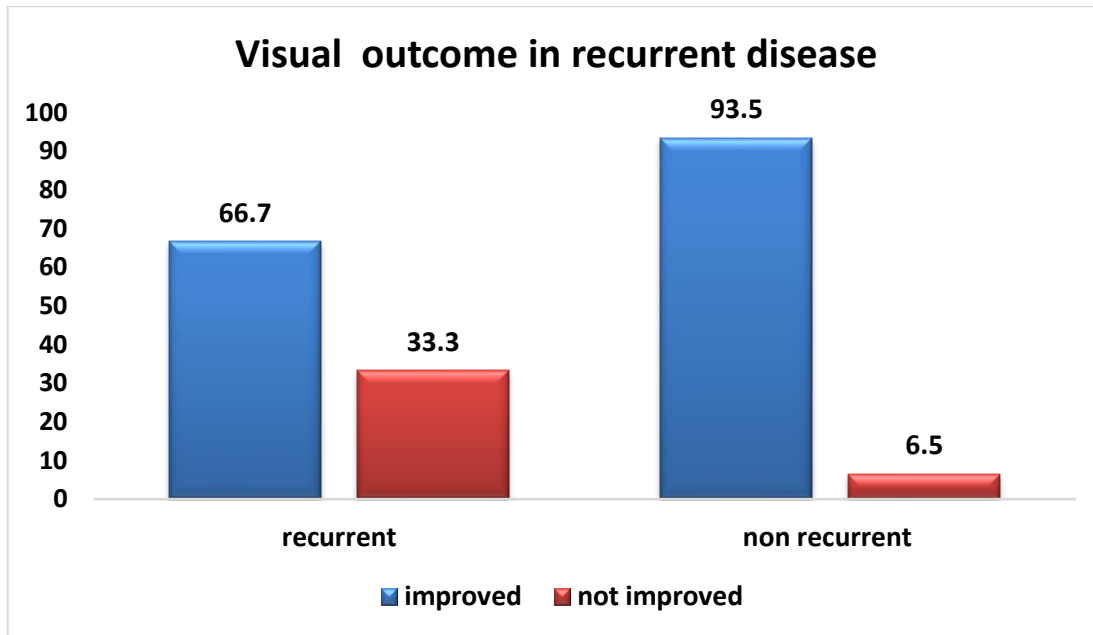


Figure 26 - Visual outcome in patients with recurrent disease

66.7 % patients having recurrent disease had improvement in their vision following surgery as compared to 33.3 % patients who had no improvement.

Factors Affecting Visual Outcome

Table 19 - Variables affecting the visual outcome and their associations

Patient Characteristics	6th month visual assessment		P value	Unadjusted OR (95% CI)	Adjusted OR (95% CI)
	Improved	Not improved			
Age(years)					
15 – 30	42(87.5)	6(12.5)	0.087	0.538 (0.095 – 3.004)	
31 – 45	160(92)	14(8)			
46 – 60	195(91.5)	18(8.5)			
Gender					
Female	196(907)	20(9.3)	0.790	0.883 (0.353- 2.211)	
Male	201(91.8)	18(8.2)			
Duration of symptoms					
<6 months	167(93.8)	11(6.2)	<0.001	0.136 (0.038 – 0.489)	0.096 (0.028 – 0.326)
6 – 12 months	195(93.8)	13(6.3)			

>12 months	35(71.4)	14(28.4)			
Tumour pathology					
Pituitary adenoma	256(89.8)	29(10.2)	0.048	27.033(1.693 – 431.743)	10.949(0.839 – 142.894)
Craniopharyngioma	69(93.2)	5(6.8)			
Meningioma	60(98.4)	1(1.6)			
Others	13(81.3)	3(18.8)			
Surgical Approach					
Endoscopy	274(93.8)	18(6.2)	0.007	0.038(0.008 – 0.186)	0.061(0.014 –0.269)
Transcranial	123(86)	20(14)			
Extent of resection					
Biopsy	0(0)	4(100)	<0.001	6.151(1.626 – 23.274)	4.636(1.328 – 16.185)
Subtotal	15(57.7)	11(42.3)			
Near total	52(88.1)	7(11.9)			
Gross total	330(95.4)	16(4.6)			
Diagnosis					
Recurrent	24 (66.7)	12(33.3)	<0.001	0.139 (0.0632-	0.039
Non recurrent	373 (93.5)	26 (6.5)			
Pre-Visual Acuity Right eye	Post VA Improvement Right Eye		P value	OR (95% CI)	
	Improved	Not improved			
Mild/Normal	236(81.9)	52(18.1)	<0.001	14.314(7.163- 28.604)	
Moderate	60(70.6)	25(29.4)			
Severe	4(50)	4(50)			
Blindness	13(24.1)	41(75.9)			
Pre-Visual Acuity Left eye	Post VA Improvement Left Eye		P value	OR (95% CI)	
	Improved	Not improved			
Mild/Normal	190(73.9)	67(26.1)	<0.001	0.725(0.600- 0.874)	
Moderate	88(80)	22(20)			
Severe	3(100)	0(0)			
Blindness	32(49.2)	33(50.8)			

Age of patient

The improvement in VA six months after surgery was observed in 87.5% of patients under 30 years of age, 92% in those of age between 31-45 years and 91.5 % in those over 46years of age. Chi square test showed $p=0.022$. Since $p>0.05$, the changes of visual deficit after surgery are not in correlation with the age of patient. Therefore, the age of patient did not significantly influence the postoperative visual outcome in our sample.

Gender

Visual improvement was seen 90.7% female patients as compared to 91.8 % male patients and as p value was 0.790 which was insignificant gender is not associated with better visual outcomes

Duration of visual symptoms

Visual improvement was confirmed in 93.8% of the patients having had the symptoms for less than 6 months before surgery. The visual improvement postoperatively was observed in 93.8% of the patients having had symptoms for 6 months to 12 months. Visual improvement was observed in 71.4% patients having had the symptoms for more than 12 months surgery Visual acuity was improved in 93.8% of patients with symptoms lasting less than 12 months. With Chi square test, $p=0.001$. Therefore, with $p<0.05$, we suggest that there is a significant direct influence of duration of symptoms before surgery on postoperative visual acuity in our patients.

Surgical approach

Visual improvement was observed in 274 patients (93.83%), who were operated via transsphenoidal technique. No improvement was observed in the remainder 17 patients (5.8%) and worsened in 1(0.3%). In the patients, who underwent transcranial approach, 123 patients (86.1%) had improvement in vision and in the rest 15 (4.2%) no improvement and 5 worsened (3.5%). Using Chi square test, we got p -value $<.001$. Therefore, the choice of approach significantly influenced the postoperative visual outcome in our sample.

Extent of resection

Out of 435 patients 330 (95.4%) patients who had gross total excision had significant improvement in visual outcomes whereas 16(4.6%) patients did not show vision improvement. Near total excision showed visual improvement in 52 patients (88.1%) patients and subtotal in 15 (57.7 %) patients. Using Chi square p value was <0.001 and therefore extent of resection has significant direct influence on visual outcomes.

Tumour pathology

273 patients with pituitary adenoma showed significant (p value < 0.001) visual improvement (96.12%) as compared to patients with 61 craniopharyngiomas (82.4%) and 51 meningiomas

(83.6%).

Degree of Preoperative Visual Reduction

It was found that 426 eyes (78.16%) out of 545 eyes with mild impairment preoperatively improved or remained stable 6 months after surgery. Out of 195 eyes with moderate preoperative VA impairment, 148 eyes (74.74%) showed improved postoperative VA. Out of 11 eyes with severe visual impairment 7 eyes (63.63 %) showed improvement and out of 119 blind eyes 45 eyes (37.8 %) showed improvement By Chi square test, we gain $p=0.001$. Since in this analysis $p<0.05$, we concluded that preoperative VA in suprasellar tumours was a significant prognostic factor, and directly contributed to better postoperative VA in the patients.

Tumour recurrence

66.7 % patients having recurrent disease presenting with visual deterioration improved following re-exploration. Primary disease with visual deterioration has a better outcome than redo surgeries Re-surgery if done early after visual deterioration and if we are able to achieve gross total or near total excision then it carries good prognosis for visual outcome.

On multiple logistic regression duration of visual deficit, extent of resection, tumour pathology, surgical approach, preoperative visual acuity, and recurrent disease were found to have statistically significant effect on post-operative visual outcome.

DISCUSSION

The most common presentation of suprasellar tumours is visual impairment.,² A large population among them are treated by Ophthalmologists initially and later referred to Neurosurgeon for definitive management causing delay in early detection and treatment.¹ Literature review shows results of surgery for suprasellar lesion based on the visual outcome but majority of them included only single type of tumour.¹⁶⁴ In our study, patients with different histopathologic tumours were included and the visual outcome was observed after decompressive surgery.

Freda et al¹⁶⁵, Kitthaweesin et al⁴, Suri A et al³ and Aui-aree et al² in their analysis found pituitary adenoma as the most common type of suprasellar tumour. Similarly, in our study pituitary adenoma was the commonest tumour seen in 284 patients (65.3%) followed by craniopharyngioma in 74(17%), and meningioma in 61 patients (14%).

In our study, the male to female ratio was (1.01) showing slight male predominance. This can be in contrary to ratio of 1.8:1 mentioned by Suri A³ in his series, and to ratio of 1:1.1 provided by Aui-aree.² Because both of these studies included multiple variety of suprasellar tumours, like ours, eliminating the association of a specific variety with a particular sex (e.g. meningiomas with female sex).

In our study, patient age ranged from 15-60 years (mean age 38.4 ± 7.46 years). Most patients were between 31-45 years (213patients,49%) whereas patients in between 46-60 years were (40%) and minimum in 15-30 years of age (11%). This shows that maximum burden of disease is on middle age group population which is the most important working class in developing countries. Visual deficit in this age group can cause significant socioeconomic burden and loss of working hours in this age group. This is not similar to the age of patients within the study of Suri A³ for visual outcome in patients with suprasellar tumours and preoperative blindness. In his study, maximum patients were in 4th and 5th decade of life. Kitthaweesin and Ployprasith⁴ in 2008 worked on ocular manifestations of suprasellar tumours 2008 and determined the mean age of 37.7 years and also the majority of patients were between 31- and 45-years age old.

In the present study, the patients with suprasellar tumours, presented mostly with impaired vision including decline in visual acuity and field in both eyes seen in 373 patients (85.75%) than unocular seen in 62 patients (14.25%), which is in contrary to the study of Aui-aree N et.al² on suprasellar tumours.

Visual function-anatomic relationships suggest that tumour extension 10 mm above the

diaphragm sellae is important for anterior visual pathway to get affected. Average size of tumour in our study was 30.8mm x 27mm x 26.8mm.

63% patients in our study had normal to mild impairment of visual acuity in accordance with WHO classification for visual impairment and 88.51 % had moderate to severe field defects showing that visual fields are more affected than acuity in suprasellar lesion .Normal or minimal visual field deficit was present in just 11.49% patients. This is supported by the study of Trautman et al who noted visual fields were affected more frequently than visual acuity.¹⁶⁶ In his series of suprasellar pituitary adenomas he found that visual fields were abnormal in 214 patients out of 285 patients as compared to visual acuity which was abnormal in 104 patients out of 285 patients. Typically, suprasellar lesions cause a bitemporal defect, which is explained by the anatomy of the visual pathways within the optic chiasm. The foremost common field defect in our study was hemianopia and included unilateral or bilateral hemifield defect on nasal or temporal or either sides in both eyes. Out of those bitemporal hemianopia was the most common seen in 195 patients. Truly atypical visual field defects encountered within the present study were peripheral constriction.

There are few reports within the literature from developed countries regarding blindness due to suprasellar lesion. The explanation may be lack of healthcare facilities and lack of neurosurgical centers in a rural location. In contrast to the believe that vision once lost due to compressive lesions is irreversible, Suri A and colleagues³ reported improvement of vision in 27.8% of eyes in their study. There are some case reports of recovery of vision from complete blindness after surgical resection of such lesions. In our study a total of 119 eyes (14%) were blind (defined as acuity worse than 3/60 in step with WHO classification) preoperatively, out of which 45 patients (37.8 %) improved which is better than operative series by various dedicated centers (Suri A et al³) and less than some reports like the one from Farrukh Zulfiqar etal¹⁶⁰ which showed 47 % improvement.

Our result points towards ability of optic nerves/chiasm to tolerate compression for long periods and chances of recovery after successful surgery.

Good results are achievable for blindness after surgery for traumatic second cranial nerve injuries.¹⁶⁷ However, the various origins of the tumours in our study and also the relatively subacute onset of blindness due to neoplasms compared with trauma make it difficult to match the results of the studies, but they both point to the actual fact that if blindness occurs due to compression of anterior visual pathways, it is reversible in a significant proportion of patients once managed early.

Suri A et al³ and associates in their work on detecting visual outcome after surgical decompression in patients with suprasellar tumours and preoperative blindness, reported visual improvement in 29% of patients. Higher percentage of improvement was noted in our study (91.3%) which included all patients with decreased vision rather than patients with blind eyes only, as seen in the report by Suri et al. Aui-aree N and colleagues² showed visual improvement in 30 out of 42 eyes (71%) after surgery in his series in Thailand. Bulters DO et al.⁷ reported, in his study in 2009 on optic nerve decompression for chronic compressive neuropathy, an improvement in the visual acuity in 46% of eyes. In his study 31% were unchanged and 23% showed deterioration. Menke E. and coworkers⁹ in their research got an improvement in 53 eyes (42.8%) after surgical decompression, while 51.6% eyes remained unchanged and 5.6% deteriorated. Frank et al.¹⁴⁰ reported 94.7% improvement in vision, 3.8% stabilization, and 1.2% worsening. Tabaee et al.¹⁴¹ showed 92% complete resolution in visual symptoms with no worsening, and Dehdashti et al.¹⁴² reported 91% normalization or improvement and 9% stabilization of the visual functions. The results of our study denoting improvement, unchanged and deterioration of 91.3%, 7.4% and 1.4% of eyes respectively are comparable to the series of Frank et al and to a major extent to Tabaee and Dehdashti et al.

Both transsphenoidal and transcranial approaches have shown improvement in acuity compared to preoperative deficit. Work on functional assessment before and after interventions on the optic chiasm by Menke E⁹ published an improvement of 41.2% and 44.6% after transsphenoidal and transcranial operations, respectively. Farrukh Zulfiqar et al¹⁶⁰ showed improvement was present in 65% of patients who underwent transsphenoidal operation, while improvement was 37.5% in patients who were operated with transcranial approach (p-value = 0.005). These results supported by our observation; within which visual improvement was present in 93.8% of patients who underwent transsphenoidal operation, while improvement was 86.01% in patients who were operated with transcranial approach (p-value <0.001). This can be explained by the very fact that in our study most of the transsphenoidal operations were done for the pituitary adenomas, that had the highest percentage of improvement (96.12%, p-value <0.001), comparable to other series in past. In series of Suri A et al³ patients had statistically significant favourable results in vision (52.3%) in those who had transsphenoidal resection compared with the vision in patients who underwent transcranial approach (10.9%).

The duration of visual symptoms (diminished vision and field defects) had a reciprocal relationship to the degree of improvement, no matter the degree of visual loss. Suri A et al³ had

shown that duration of symptoms was not statistically significant in improvement of post-operative visual deficit which is in contrary to our study. Nipat Aui-aree MD¹⁵⁹, et al found that there was no relation in the duration of visual deficit and post-operative improvement of visual deficit, but their sample size was small and follow up was up to 4 weeks. Kerrison JB, et al¹⁶⁸ noted that visual improvement with a longer follow-up (up to six months) as notable improvements in the recovery of visual function after decompression of anterior visual pathways is found up to 4 months following successful treatment. Kitthisak Kitthaweesin et al⁴ showed that patients, whose symptoms lasted less than twelve months, had the most improvement which is supported by our study.

Visual recovery occurs due to decompression effect of surgery and it occurs in three phases. In the first phase which is seen after one week of surgery, improvement is seen due to better nerve conduction ; second phase seen in about 6 months postoperatively as recovery occurs due to remyelination changes. Third phase of improvement which is seen after a year of surgery occurs as neovascularisation develops over time.¹⁶¹

In our study, nearly one fourth of the patients with suprasellar tumours who underwent surgery presented with preoperative uniocular/binocular visual deficit. In India, with a population of 1 billion, nearly 60–70% live in villages and small towns that are far from tertiary health care centres in metropolitan cities. Macroadenomas typically start compressing the crossing fibres of the chiasm first, creating the typical bitemporal hemianopsia. Visual Acuity changes are caused by compression of the macular fibres at a later stage of the disease.¹⁶²

A statistically significant difference exists between pre and postoperative Visual Acuity in surgically treated suprasellar tumours. In the present study, patients had significantly better Visual acuity 6 months after surgery in the suprasellar region. The influence of predictive factors on postoperative visual acuity outcomes have been done in previous studies but mostly on single pathology. Bivariate analysis revealed the following to be statistically significant favourable outcome factors: duration of blindness < 12 months, extent of resection, surgical approach, preoperative visual deficit, and tumour histopathology (pituitary adenoma compared with craniopharyngioma or meningioma). Post-operative visual acuity was not statistically related to age and sex of the patients. Younger patients show better improvement as compared to older patients. These results may be interpreted as follows:

a) Predominant reason for acuity reduction in patients with compressive optic neuropathies is

ischemia of nerve fibres.

b) Visual acuity efficiently recovers after surgery and with better microcirculation recovery in younger individuals than in older patients but that does not mean that visual deficit in older people have less potential to recover.¹⁶⁹

Our data is in accordance with other data, where importance of preoperative visual deficit status and the factors affecting it are important for postoperative visual deficit recovery. Desanka Grkovic et al¹⁵⁸ in his study of suprasellar meningioma had found that mild preoperative visual acuity loss had better recovery after surgery. Patients who have mild preoperative visual loss show lesser degree involvement of optic nerve and chiasma by the tumour intraoperatively.¹⁶⁹ Therefore postoperative chances for visual improvement after surgical decompression are better.

Slow, gradual progression of visual function defects, headache, neurological deficits, or neuropsychiatric changes in many patients contributes to late or delayed diagnosis with a loss of valuable time. Suprasellar tumours are detected most frequently at a time when vision in one eye is already significantly damaged, and tumour has reached large size. There is usually a transitory monocular visual reduction, with gradual peripheral field narrowing. These symptoms are often misleading and overseen both by patient and Ophthalmologist. On other hand, these signs, and symptoms, which are nonspecific vision-related may be misdiagnosed as retrobulbar neuritis or ischaemic optic neuropathy. Systemic corticosteroid therapy may improve vision and its related complaints, which often masks proper diagnosis. Therefore, every case with transient visual improvement, or if there is no response to corticosteroid therapy, must be taken as warning sign of compressive lesion of optic nerve or chiasmal region, and should warrant prompt neuroimaging. In our group of patients, who had symptoms for less than 12 months, a postoperative VA improvement was noted in 93.8% of the cases. 28.4% of those patients who had had symptoms for more than 12 months showed no improvement showing the importance of early diagnosis and surgery. Possible pathogenic mechanisms for nerve fibres leading to demyelination, degradation and degeneration of axons include a) direct compression of nerve fibres by tumour, b) obstruction of axoplasmatic transport, c) ischemia of nerve fibres due to pressure on fine feeding microvasculature. These processes are prolonged in cases with non-recognized symptoms. After surgical decompression, chronically ischemic partially demyelinated nerve fibres still have potential to recover and improve. But that can happen only if they have retained their significant biological recovery potential.¹⁷⁰ Atrophy of nerve fibres develops with prolonged, severe preoperative ischemia, advanced degeneration, and demyelination of nerve fibres. Recovery potential is irreversibly lost and there is no improvement of neurologic function even after surgical

decompression.¹⁷⁰ It is postulated that the critical preoperative period for postoperative recovery of visual function is 2 years. Time elapsed from first symptoms till the diagnosis of tumour depends on the tumour size, extent of surgical excision

In patients with severe impairment, surgical intervention is indicated because improvement of visual function occurs in most cases. Visual recovery can be noted within the first few days after surgery.¹⁷¹ Improvement in visual function can continue up to 1 year after surgery.¹⁷² Suri A et al³ in his study had found that extent of tumour resection (radical or gross-total excision) had a favourable trend with improvement rates twice (34.4%) those of subtotal excision in postoperative visual improvement. In our study 95.4% patients who had gross total excision had significant improvement in visual outcomes whereas 16 (4.6%) patients did not show vision improvement. Near total excision showed visual improvement in 88.1 % patients. In endoscopic approach gross total excision was done in 274(93.8%) as compared to 123 (86%) patients in transcranial. Although the data of de Paiva Neto et al.¹⁷² did not confirm that endoscopy increased rate of gross total excision, the endoscope has added benefit of providing panoramic view from within sella and access in a way not possible due to tunnel vision of microscope.

Prolonged compression on fine nerve fibres with ischemia leads to an irreversible loss of the function even with decompression surgery.

91.3% of the patients showed improvement in visual function including visual acuity and visual field. Visual field improvement was found in 88.9%. These scores encourage the practitioner to give good prognosis to patients with suprasellar lesions with visual impairment.

Gnanalingham et al.¹⁶³ had concluded that recovery of field deficit following endoscopic surgery in patients with pituitary tumours takes place over a long time. The greatest influence on this visual field recovery was the extent of deficit in visual field deficit preoperatively and is important clinically. Recovery of visual fields is progressive over several years and more than 50% takes place in the initial 3-6 months postoperatively. This is important from a prognostic point of view. This is supported by our study which showed visual field improvement by 88.9% within first 6 months.

Gnanalingham et al.¹⁶³ also stated that association between smaller deficit in preoperative visual field and greater postoperative recovery of visual field is not universal finding. This was also seen in our study as the patients with smaller visual field defect in group 1 (having enlarged blind spot or altitudinal field defect) had less improvement as compared to other groups. The reason for this is not clear. It can be due to referral bias, differences in patient population studied, including differences in preoperative deficits in VF and VA, the extent of tumour resection.

Our study supports data and conclusions found in world literature stating that degree of postoperative visual function recovery, with optimal surgical resection, has several dependent factors. These are the time elapsed from first symptom until tumour diagnosis and surgical treatment, degree of visual impairment preoperatively, recurrent disease, the extent of resection of tumour and pathology of tumour.

CONCLUSION

1. Patients with suprasellar tumours with pre-existing preoperative visual deficit experienced significant benefit in improvement of vision after surgery
2. Significant factors affecting the visual outcome were preoperative visual deficit, duration of blindness < 12 months, surgical approach, extent of resection, recurrent disease and tumour histopathology
3. Severe visual deficit or blindness should not be considered as a negative or deterrent factor while considering surgical decompression, as improvement is observed in a significant population of such patients also after surgery.
4. Recurrent tumours having visual deficit if operated early with a goal to achieve gross total resection carries good outcome and improvement in vision is observed in these patients. Therefore, history of previous surgery should not be considered as a negative or deterrent factor while considering surgical decompression for recurrent tumours in the suprasellar region.
5. Detailed evaluation of data on visual outcome can help counsel patients and their caretakers preoperatively to aid decision making and set expectations.

LIMITATIONS AND RECOMMENDATIONS

1. It is a retrospective study.
2. Evaluation of visual acuity and visual field separately may not be uniform and there is a need for uniform evaluation as these are the main factors affecting the visual outcome.
3. It is also recommended that although patients present in Ophthalmology clinics with unilateral or bilateral blurred vision for which the common diagnosis is refractive error or cataract, a complete and detailed ophthalmological examination should be performed to avoid delayed or misdiagnosis of treatable benign conditions i.e. suprasellar tumours which if undiagnosed for a long period can be even life-threatening.

REFERENCES

1. Jallu A, Kanaan I, Rahm B, Siqueira E: Suprasellar meningioma and blindness: a unique experience in Saudi Arabia. *Surg Neurol* 45:320–323, 1996
2. Aui-aree N, Phruanchroen C, Oearsakul T, Hirunpat S, Sangthong R. Three years experience of suprasellar tumours in neuro-ophthalmology clinic. *Medical journal of the Medical Association of Thailand*. 2010; 93(7):818.
3. Suri A, Narang KS, Sharma BS, Mahapatra AK. Visual outcome after surgery in patients with suprasellar tumours and preoperative blindness. 2008.
4. Kitthaweasin K, Ployprasith C. Ocular manifestations of suprasellar tumours. *Medical journal of the Medical Association of Thailand*. 2008; 91(5):711.
5. Galal A, Faisal A, Al-Werdany M, El Shehaby A, Lotfy T, Moharram H. Determinants of postoperative visual recovery in suprasellar meningiomas. *Acta neurochirurgica*. 2010; 152(1):69-77.
6. Monteiro MrLR, Zambon BK, Cunha LP. Predictive factors for the development of visual loss in patients with pituitary macroadenomas and for visual recovery after optic pathway decompression. *Canadian Journal of Ophthalmology/Journal Canadien d'Ophthalmologie*. 2010; 45(4):404-8.
7. Bulters DO, Shenouda E, Evans BT, Mathad N, Lang DA. Visual recovery following optic nerve decompression for chronic compressive neuropathy. *Acta neurochirurgica*. 2009; 151(4):325.
8. Chakrabarti I, Amar AP, Couldwell W, Weiss MH. Long-term neurological, visual, and endocrine outcomes following transnasal resection of craniopharyngioma. *Journal of neurosurgery*. 2005; 102(4):650-7.
9. Menke E, Osarovsky E, Reitner A, Matula C. Functional assessment before and after interventions on the optic chiasm system. *Wiener klinische Wochenschrift*. 2002; 114(1-2):33-7.
10. SL, Kline LB: Magnetic resonance imaging of the optic chiasm. *Surv Ophthalmol* 136:207-216, 1991
11. Kucharczyk W, Montanera WJ: The sella and parasellar region, in Atlas SW (ed), *Magnetic Resonance Imaging of the Brain and Spine*. New York, NY, Raven Press, 1991, pp 625-667
12. Dutton J J, Klingele TG, Burde RM, et al: Evaluation of the suprasellar cistern by computed tomography. *Ophthalmology* 89:1220-1225, 1982

13. Ono M, Ono M, Rhoton AL, et al: Microsurgical anatomy of the region of the tentorial incisura. *J Neurosurg* 60:365-399, 1984
14. Gentry LR, Smoker WRK, Turski PA, et al: Suprasellar arachnoid cysts: 1. CT recognition. *AJNR* 7:79-86, 1986
15. Wiggli U, Benz UF: Normal computed tomography anatomy of the suprasellar arachnoid space. *Radiology* 128:65-70, 1978
16. Rosner SS, Rhoton AL, Ono M, et al: Microsurgical anatomy of the anterior perforating arteries. *J Neurosurg* 61:468-485, 1984
17. Amundsen P, Newton TH: Subarachnoid cisterns, *in* Newton TH, Potts DG (eds): *Radiology of the Skull and Brain*, vol 4. Ventricles and Cisterns. Great Neck, NY, MediBooks, 1978, pp 3588-3711
18. Carpenter MB: *Core Text of Neuroanatomy* (ed 4). Baltimore, MD, Williams & Wilkins, 1991, p 4
19. Osborne AG: *Introduction to Cerebral Angiography*. Philadelphia, PA, Harper & Row, 1980, pp 143-165
20. Nagata S, Rhoton AL, Barry M: Microsurgical anatomy of the choroidal fissure. *Surg Neurol* 30:3-59, 1988
21. Yamamoto I, Rhoton AL, Peace DA: Microsurgery of the third ventricle: Part 1--microsurgical anatomy. *Neurosurgery* 8:334-356, 1981
22. Gibo H, Lenkey C, Rhoton AL: Microsurgical anatomy of the supraclinoid portion of the internal carotid artery. *J Neurosurg* 55:560-574, 1981
23. Yasargil MG, Kasdaglis K, Jain KK, et al: Anatomical observations of the subarachnoid cisterns of the brain during surgery. *J Neurosurg* 44:298-302, 1976
24. Pojunas KW: The sella and juxtaseellar region, *in* Daniels DL, Haugbton VM, Naidich TP (eds): *Cranial and Spinal Magnetic Resonance Imaging: An Atlas and Guide*. New York, NY, Raven, 1987, pp 121-158
25. Carpenter MB: *Core Text of Neuroanatomy* (ed 4). Baltimore, MD, Williams & Wilkins, 1991, pp 215-221
26. Doyle AJ: Optic chiasm position on MR images. *AJNR* 11:553-555, 1990
27. Hoyt WF: Correlative functional anatomy of the optic chiasm. *Clin Neurosurg* 17:189-208, 1969
28. Bergland RM, Ray BS, Torack RM: Anatomic variations in the pituitary gland and adjacent structures in 225 autopsy cases. *J Neurosurg* 28:93-99, 1968

29. Perlmutter D, Rhoton AL: Microsurgical anatomy of the anterior cerebral-anterior communicating-recurrent artery complex. *J Neurosurg* 45:259-272, 1976
30. Bergland RM, Ray BS: The arterial supply to the human optic chiasm. *J Neurosurg* 31:327-334, 1969
31. Woltshlaeger G, Wollschlaeger PB: The circle of Willis, in Newton TH, Potts DG (eds): *Radiology of the Skull and Brain*, vol 2, Angiography, book 2, Arteries. Great Neck, NY, MediBooks, 1978, pp 1171-1201
32. Daniels DL, Haughton VM, Williams AL, et al: Computed tomography of the optic chiasm. *Radiology* 137:123-127, 1980
33. Yamamoto Y, Satoh T, Asari S, et al: Normal anatomy of cerebral vessels by computed angiography in the axial transverse plane. *J Comput Assist Tomogr* 6:865-873, 1982
34. Aoki S, Sasaki Y, Machida T, et al: Cerebral aneurysms: Detection and delineation using 3-D-CT angiography. *AJNR* 13:1115-1120, 1992
35. Edelman RR, Mattle HP, Atkinson DJ, et al: MR angiography. *AJR* 154:937-946, 1990
36. Lewin JS, Laub G: Intracranial MR angiography: A direct comparison of three time-of-flight techniques. *AJNR* 12:1133-1139, 1991
37. Blatter DD, Parker DL, Robison RO: Cerebral MR angiography with multiple overlapping thin slab acquisition. Part I. Quantitative analysis of vessel visibility. *Radiology* 179:805-811, 1991
38. Pernicone JR, Siebert JE, Potchen EJ, et al: Three dimensional phase contrast MR angiography in the head and neck: Preliminary report. *AJNR* 11:457-466, 1990
39. Edelman RR, Ahn SS, Chien D, et al: Improved time-of-flight MR angiography of the brain with magnetization transfer contrast. *Radiology* 184:395-399, 1992
40. Carpenter MB: *Core Text of Neuroanatomy* (ed 4). Baltimore, MD, Williams & Wilkins, 1991, pp 297-324
41. Carpenter MB: *Core Text of Neuroanatomy* (ed 4). Baltimore, MD, Williams & Wilkins, 1991, p 375
42. Rhoton AL, Hardy DG, Chambers SM: Microsurgical anatomy and dissection of the sphenoid bone, cavernous sinus and sellar region. *Surg Neurol* 12:63-104, 1979
43. Loes DJ, Barloon TJ, Yuh WTC, et al: MR anatomy and pathology of the hypothalamus. *AJR* 156:579-585, 1991
44. Naheedy MH, Haag JR, Azar-Kia B, et al: MRI and CT of sellar and parasellar disorders. *Radiol Clin North Am* 25:819-847, 1987

45. Carpenter MB: Core Text of Neuroanatomy (ed 4). Baltimore, MD, Williams & Wilkins, 1991, p 20
46. Spickler EM, Hirsch WL, Hayman LA: Neurosecretory regions, in Hayman LA, Hinck VC (eds): Clinical Brain Imaging: Normal Structure and Functional Anatomy. St Louis, IL, Mosby, 1992, pp 417-426
47. Simmons GE, Suchnicki JE, Rak KM, et al: MR imaging of the pituitary stalk: Size, shape, and enhancement pattern. *AJR* 159:375-377, 1992
48. Peyster RG, Hoover ED, Adler LP: CT of the normal pituitary stalk. *AJNR* 5:45-47, 1984
49. Tien R, Newton TH, McDermott M, et al: Thickened pituitary stalk on MR images in patients with diabetes insipidus and Langerhans cell histiocytosis. *AJNR* 11:703- 708, 1990
50. Kivela T, Pelkonen R, Oja M, Heiskanen O: Diabetes insipidus and blindness caused by a suprasellar tumour: Peter Pauw's observations from the 16th century. *JAMA* 279:48-50, 1998
51. Vance ML. Hypopituitarism. *N Engl J Med* 1994;330(23): 1651-1662
52. Hart WM Jr. Acquired dyschromatopsias. *Surv Ophthalmol* 1987;32(1):10-31
53. Frisè L, Royt WF, Tengroth BM. Optociliary veins, disc pallor and visual loss. A triad of signs indicating sphenoidal meningioma. *Acta Ophthalmol (Copenh)* 1973;51(2):241-249
54. Newman SA, Miller NR. Optic tract syndrome. Neuro-ophthalmologic considerations. *Arch Ophthalmol* 1983;101(8):1241-1250
55. Bell RA, Thompson HS. Relative afferent pupillary defect in optic tract hemianopias. *Am J Ophthalmol* 1978;85(4):538-540
56. Adler FH, Austin G, Grant FC. Localizing value of visual fields in patients with early chiasmal lesions. *Arch Ophthalmol* 1948;40:579-600
57. Hoyt WF. Correlative functional anatomy of the optic chiasm. 1969. *Clin Neurosurg* 1970;17:189-208
58. Horton JC. Wilbrand's knee of the primate optic chiasm is an artefact of monocular enucleation. *Trans Am Ophthalmol Soc* 1997;95:579-609
59. Wilbrand HL. Schema des verlaufs der Schnervenfasern durch das Chiasma. *Z Augenheilkd* 1926;59:135-144
60. Traquair HM. An Introduction to Clinical Perimetry. CV Mosby; 1927
61. Huber A. Eye Signs and Symptoms in Brain Tumours. 3rd ed. CV Mosby; 1976
62. Fujimoto N, Saeki N, Miyauchi O, Adachi-Usami E. Criteria for early detection of temporal hemianopia in asymptomatic pituitary tumour. *Eye (Lond)* 2002;16(6):731-738
63. Sadun AA, Currie JN, Lessell S. Transient visual obscurations with elevated optic discs. *Ann*

Neurol 1984;16(4):489–494

64. Uhthoff W. Die Augenveränderungen bei denselben Erkrankungen des Gehirnes. In: Graefes-Saemisch Handbuch der gesamten Augenheilkunde, vol. 11. Wilhelm Engelmann; 1915:1143
65. Borchert MS. Principles and techniques of the examination of ocular motility and alignment. In: Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. Walsh & Hoyt's Clinical Neuro-Ophthalmology. 6th ed. Lippincott Williams & Wilkins; 2005:887–905
66. Cushing H: The chiasmal syndrome of primary optic atrophy and bitemporal field defects in adults with a normal sella turcica. Arch Ophthalmol 3:505–551, 1935
67. Gregorius FK, Helper RS, Stern WE: Loss and recovery of vision with suprasellar meningiomas. J Neurosurg 42:69–74, 1975
68. Schlezinger NS, Alpers BJ, Weiss BP: Suprasellar meningiomas associated with scotomatous field defects. Arch Ophthalmol 35: 624–642, 1946
69. Bergland R, Ray BS: The arterial supply of the human optic chiasm. J Neurosurg 31:327–334, 1969
70. McIlwaine GG, Carrim ZI, Lueck CJ, Chrisp TM: A mechanical therapy to account for bitemporal hemianopia from chiasmal compression. J Neuroophthalmol 25:40–43, 2005
71. Schoepf D, Zangemeister WH, Vines FS, et al. Correlation of ocular motor reading strategies to the status of adaptation in patients with hemianopic visual field defects. Ann N Y Acad Sci 1993;682:404–408
72. ErÅyahin Y, Yurtseven T, Å-zgiray E, Mutluer S. Craniopharyngiomas in children: Turkey experience. Child's Nervous System. 2005;21(8-9):766-72.
73. Zikel OM, Atkinson JL, Hurley DL: Prolactinoma manifesting with symptomatic hydrocephalus. Mayo Clin Proc 74: 475-477> 1999
74. Jennings MT, Gelman R, Hochberg F: Intracranial germ cell tumours: natural history and pathogenesis. J Neurosurg 63: 155-167,1985
75. Hartman ML, Crowe BJ, Biller BM, Ho KK, Clemmons DR, Chipman JJ; HyposCCS Advisory Board; U.S. HypoCCS Study Group. Which patients do not require a GH stimulation test for the diagnosis of adult GH deficiency? J Clin Endocrinol Metab 2002;87(2):477–485
76. Arnaldi G, Angeli A, Atkinson AB, et al. Diagnosis and complications of Cushing's syndrome: a consensus statement. J Clin Endocrinol Metab 2003;88(12):5593–5602
77. Frisén L. The foundations of visual field testing. In: Clinical Tests of Vision. Raven Press; 1990:55–77
78. Katz J, Tielsch JM, Quigley HA, Sommer A. Automated perimetry detects visual field loss

- before manual Goldmann perimetry. *Ophthalmology* 1995;102(1):21–26
79. Lowenfeld IE. *The Pupil*. Butterworth; 1999
80. Lam BL, Thompson HS, Corbett JJ. The prevalence of simple anisocoria. *Am J Ophthalmol* 1987;104:69–73
81. Levatin P. Pupillary escape in disease of the retina or optic nerve. *Arch Ophthalmol* 1959;62:768–779
82. Thompson HS, Corbett JJ, Cox TA. How to measure the relative afferent pupillary defect. *Surv Ophthalmol* 1981;26(1):39–42
83. Kennedy RF. Retrobulbar neuritis as an exact diagnostic sign of certain tumours and abscesses in the frontal lobes. *Am J Med Sci* 1911;142:355–368
84. Naidich TP, Pinto RS, Kushner MJ, et al: Evaluation of sellar and parasellar masses by computed tomography. *Radiology* 120:91-99, 1976
85. Kuuliala I: The normal suprasellar subarachnoid space in computed tomography. *Clin Radiol* 31:155-159, 1980
86. Webb SM, Ruscalleda J, Schwarzstein D, et al. Computerized tomography versus magnetic resonance imaging: a comparative study in hypothalamic-pituitary and parasellar pathology. *Clin Endocrinol (Oxf)* 1992;36(5):459–465
87. Davis PC, Hoffman JC Jr, Spencer T, Tindall GT, Braun IF. MR imaging of pituitary adenoma: CT, clinical, and surgical correlation. *AJR Am J Roentgenol* 1987;148(4):797–802
88. Patronas N, Bulakbasi N, Stratakis CA, et al. Spoiled gradient recalled acquisition in the steady state technique is superior to conventional postcontrast spin echo technique for magnetic resonance imaging detection of adrenocorticotropin-secreting pituitary tumours. *J Clin Endocrinol Metab* 2003;88(4):1565–1569
89. Kucharczyk W, Bishop JE, Plewes DB, Keller MA, George S. Detection of pituitary microadenomas: comparison of dynamic keyhole fast spin-echo, unenhanced, and conventional contrast-enhanced MR imaging. *AJR Am J Roentgenol* 1994;163(3):671–679
90. Tabarin A, Laurent F, Catargi B, et al. Comparative evaluation of conventional and dynamic magnetic resonance imaging of the pituitary gland for the diagnosis of Cushing's disease. *Clin Endocrinol (Oxf)* 1998;49(3):293–300
91. Pierallini A, Caramia F, Falcone C, et al. Pituitary macroadenomas: preoperative evaluation of consistency with diffusion-weighted MR imaging—initial experience. *Radiology* 2006;239(1):223–231
92. Elster AD. Imaging of the sella: anatomy and pathology. *Semin Ultrasound CT MR*

1993;14(3):182–194

93. Symons SP, Aviv RI, Montanera WJ, Kucharczyk W. The sella turcica and parasellar region. In: Atlas SW, ed. *Magnetic Resonance Imaging of the Brain and Spine*, vol. 2. Lippincott Williams & Wilkins; 2008:1120–1192
94. Pusey E, Kortman KE, Flannigan BD, Tsuruda J, Bradley WG (1987) MR of craniopharyngiomas: tumour delineation and characterisation. *AJNR* 8:439-444
95. Sartor K (1992) *MR imaging of the skull and brain*. Springer, Berlin Heidelberg New York, pp 379-451
96. Freeman MR, Kessler RM, Allen JH, Price AC (1987) Craniopharyngioma: CT and MR imaging in nine cases. *J Comput Assist Tomogr* 11:810~814
97. Kazner E, Wende S, Grumme T, Stochdorph O, Felix R, Claussen C (eds) (1989) *Computed tomography and magnetic resonance tomography of intracranial tumours*. Springer, Berlin Heidelberg New York
98. Young SC, Zimmerman RA, Nowell MA, Bilaniuk LT, Hackney DB, Grossman RI, Goldberg HI (1987) Giant cystic craniopharyngiomas. *Neuroradiology* 29:468-473
99. Hoff JT, Patterson Jr RH (1972) Craniopharyngiomas in children and adults. *J Neurosurg* 36:29-302
100. Bonneville JF, Cattin F, Dietemann JL (1986) *Computed tomography of the pituitary gland*. Springer, Berlin Heidelberg New York
101. Sartoretti-Schefer S, Wichmann W, Aguzzi A, Valavanis A. MR differentiation of adamantinous and squamous-papillary craniopharyngiomas. *AJNR Am J Neuroradiol* 1997;18(1):77–87
102. Petito CK, DeGirolami U, Earle KM (1976) Craniopharyngiomas: a clinical and pathological review. *Cancer* 37:1944-1952
103. Al-Mefty O, Hassounah M, Weaver R, Sakati N, Jinkins JR, Fox JL (1985) Microsurgery for giant craniopharyngiomas in children. *Neurosurgery* 17:585-595
104. Cabezudo JM, Vaquero J, Garcia de Sola, R, Leunda G, Nombela L, Bravo G (1981) Computed tomography with craniopharyngiomas: a review. *Surg Neurol* 15:422-427
105. Hofmann E, Becker T, Warmuth Metz M, Sørensen N, Luther I (1991) Suprasellar arachnoid cysts. *Klin Neuroradiol* 1:49-57
106. Sherman JL, Camponovo E, Citrin CM (1990) MR imaging of CSF-like chorioideal fissure and parenchymal cysts of the brain. *AJNR* 11:939-945
107. Krawchenko J, Collins GH (1979) Pathology of arachnoid cyst. *J Neurosurg* 50:224-228

108. Steffey D J, Filipp GJ de, Spera T, Gabrielsen TO (1988) MR imaging of primary epidermoid tumours. *J Comput Assist Tomogr* 12:438-440
109. Karnaze MG, Sartor K, Winthrop JD, Gado MH, Hodges III FJ (1986) Suprasellar lesions: evaluation with MR imaging. *Radiology* 161:77-82
110. Smith AS, Benson JE, Blaser SI, Mizushima A, Tarr RW, Bellon EM. Diagnosis of ruptured intracranial dermoid cyst: value of MR over CT. *AJNR Am J Neuroradiol* 1991;12(1):175–180
111. Kazner E, Wende S, Grumme T, Stochdorph O, Felix R, Claussen C (eds) (1989) Computed tomography and magnetic resonance tomography of intracranial tumours. Springer, Berlin Heidelberg New York
112. Razek AAKA, Castillo M. Imaging lesions of the cavernous sinus. *AJNR Am J Neuroradiol* 2009;30(3):444–452
113. Symons SP, Aviv RI, Montanera WJ, Kucharczyk W. The sella turcica and parasellar region. In: Atlas SW, ed. *Magnetic Resonance Imaging of the Brain and Spine*, vol. 2. Lippincott Williams & Wilkins; 2008:1120–1192
114. Kato T, Sawamura Y, Tada M, et al. Occult neurohypophyseal germinomas in patients presenting with central diabetes insipidus. *Neurosurg Focus* 1998;5(1):e6
115. Erdem E, Angtuaco EC, Van Hemert R, Park JS, Al-Mefty O. Comprehensive review of intracranial chordoma. *Radiographics* 2003;23(4):995–1009
116. Mancuso AA, Hanafee WN (1985) Computed tomography and magnetic resonance imaging of the head and neck. Williams and Wilkins, Baltimore, Maryland
117. Dean BI, Drayer BE Bird CR, Flom RA, Hodak JA, Coons SW, Carey RG (1990) Gliomas: classification with MR imaging. *Radiology* 174:411-415
118. Tien RD, Newton TH, McDermott MW, Dillon WR Kucharczyk J (1990) Thickened pituitary stalk on MR images in patients with diabetes insipidus and Langerhans cell histiocytosis. *AJNR* 11:703-708
119. Desai SB, Shah VC, Tavri OJ, Rao P (1991) MRI. More specific than CT in cranial tuberculomas. *Neuroradiology (Suppl)* 33: 216-218
120. Renn WH, Rhoton AL Jr: Microsurgical anatomy of the sellar region. *J Neurosurg* 1975;43: 288–298.
121. Bergland RM, Ray BS, Torack M: Anatomical variations in the pituitary gland and adjacent structures in 225 human autopsy cases. *J Neurosurg* 1968;28:93–99.
122. Couldwell WT. Transsphenoidal and transcranial surgery for pituitary adenomas. *J Neuro-Oncol.* 2004;69:237–56.

123. Cope V. The pituitary fossa, and the methods of surgical approach thereto. *Br J Surg.* 1916;4:107–44.
124. Horsley V. On the technique of operations on the central nervous system. *Br Med J.* 1906;2:411–23.
125. Barrow DL, Tindall G, Tindall SC: Combined simultaneous transsphenoidal transcranial operative approach to selected sellar tumours. *Perspect Neurol Surg* 1992;3:49–57.
126. Alleyne CH Jr, Barrow DL, Oyesiku NM: Combined transsphenoidal and pterional craniotomy to giant pituitary tumours. *Surg Neurol* 2002;57:380–390.
127. Cooke SW, Smith Z, Kelly DF: Endonasal transsphenoidal removal of tuberculum sellae meningiomas: technical note. *Neurosurgery* 2004;55:239–244.
128. Silbermark M: Die intrakranielle Extirpation der Hypophyse. *Wien Klin Wochenschr* 1910;23: 467–468.
129. Heuer G: Surgical experiences with intracranial approach to chiasmal lesions. *Arch Surg* 1920;1:368–381.
130. Adson A: Hypophysial tumours through the intradural approach. *JAMA* 1918;71:721–726.
131. Fahlbusch R, Honegger J, Paulus W, Huk W, Buchfelder M: Surgical treatment of craniopharyngiomas: experience with 168 patients. *J Neurosurg* 1999;90:237–250.
132. Patterson R. Parasellar meningiomas. In: Apuzzo M, editor. *Brain surgery*, vol. 1. New York: Churchill Livingstone; 1993. p. 219–30.
133. Snow RB, Patterson RH Jr: Craniotomy for pituitary tumours; in Kaye AH, Black P (eds): *Operative Neurosurgery*. London, Churchill Livingstone, 2000, pp 709–714.
134. Steno J, Malacek M, Bizik I: Tumour-third ventricular relationships in supradiaphragmatic craniopharyngiomas: correlation of morphological, magnetic resonance imaging, and operative findings. *Neurosurgery* 2004;54:1051–1060.
135. Al-Mefty O. Approach selection in juxtaseellar surgery. In: Salzman M, editor. *Surgery of the cranial base*. Boston: Kluwer; 1989. p. 57–71.
136. Gandhi CD, Christiano LD, Eloy JA, Prestigiacomo CJ, Post KD. The historical evolution of transsphenoidal surgery: Facilitation by technological advances. *Neurosurg Focus* 2009;27:E8.
137. Cappabianca P, Alfieri A, de Divitiis E. Endoscopic endonasal transsphenoidal approach to the sella: Towards functional endoscopic pituitary surgery (FEPS). *Minim Invasive Neurosurg* 1998;41:66-73.
138. Alalade AF, Ogando-Rivas E, Boatey J, Souweidane MM, Anand VK, Greenfield JP, et al. Suprasellar and recurrent pediatric craniopharyngiomas: Expanding indications for the extended

- endoscopic transsphenoidal approach. *J Neurosurg Pediatr* 2018;21:72-80.
139. Zimmer LA, Theodosopoulos PV. Anterior skull base surgery: Open versus endoscopic. *Curr Opin Otolaryngol Head Neck Surg* 2009;17:75-8.
140. Frank G, Pasquini E, Farneti G, Mazzatenta D, Sciarretta V, Grasso V, et al. The endoscopic versus the traditional approach in pituitary surgery. *Neuroendocrinology* 2006;83:240-8
141. Tabaee A, Anand VK, Barrón Y, Hiltzik DH, Brown SM, Kacker A, et al. Predictors of short-term outcomes following endoscopic pituitary surgery. *Clin Neurol Neurosurg* 2009;111:119-22.
142. Dehdashti AR, Ganna A, Karabatsou K, Gentili F. Pure endoscopic endonasal approach for pituitary adenomas: Early surgical results in 200 patients and comparison with previous microsurgical series. *Neurosurgery* 2008;62:1006-15.
143. Jho HD. Endoscopic endonasal approach to the optic nerve: A technical note. *Minim Invasive Neurosurg* 2001;44:190-3
144. Koutourosiou M, Fernandez-Miranda JC, Stefko ST, Wang EW, Snyderman CH, Gardner PA. Endoscopic endonasal surgery for suprasellar meningiomas: Experience with 75 patients. *J Neurosurg* 2014;120:1326-39
145. Fatemi N, Dusick JR, de Paiva Neto MA, Kelly DF. The endonasal microscopic approach for pituitary adenomas and other parasellar tumours: A 10-year experience. *Neurosurgery* 2008;63:244-56
146. Makarenko S, Carreras EM, Akagami R. Craniotomy for perisellar meningiomas: Comparison of simple (appropriate for endoscopic approach) versus complex anatomy and surgical outcomes. *J Neurosurg* 2017;126:1191-200
147. Mahmoud M, Nader R, Al-Mefty O. Optic canal involvement in tuberculum sellae meningiomas: Influence on approach, recurrence, and visual recovery. *Neurosurgery* 2010;67:ons108-18
148. Attia M, Kandasamy J, Jakimovski D, Bedrosian J, Alimi M, Lee DL, et al. The importance and timing of optic canal exploration and decompression during endoscopic endonasal resection of tuberculum sella and planum sphenoidale meningiomas. *Neurosurgery* 2012;71:58-67.
149. Elliott RE, Jane JA Jr., Wisoff JH. Surgical management of craniopharyngiomas in children: Meta-analysis and comparison of transcranial and transsphenoidal approaches. *Neurosurgery* 2011;69:630-43
150. Bartels U, Laperriere N, Bouffet E, Drake J. Intracystic therapies for cystic craniopharyngioma in childhood. *Front Endocrinol (Lausanne)* 2012;3:39
151. Bal E, Öge K, Berker M. Endoscopic endonasal transsphenoidal surgery, A reliable method

for treating primary and recurrent/Residual craniopharyngiomas: Nine years of experience. *World Neurosurg* 2016;94:375-85.

152. Liu JK, Sevak IA, Carmel PW, Eloy JA. Microscopic versus endoscopic approaches for craniopharyngiomas: Choosing the optimal surgical corridor for maximizing extent of resection and complication avoidance using a personalized, tailored approach. *Neurosurg Focus* 2016;41:E5

153. Komotar RJ, Starke RM, Raper DM, Anand VK, Schwartz TH. Endoscopic endonasal compared with microscopic transsphenoidal and open transcranial resection of craniopharyngiomas. *World Neurosurg* 2012;77:329-41.

154. Zada G, Kelly D, Cohan P, Wang C, Swerdloff R: The endonasal transsphenoidal approach for pituitary adenomas and other sellar lesions: an assessment of efficacy, safety and patient impressions. *J Neurosurg* 2003;98:350–358.

155. Barrow DL, Tindall G: Loss of vision after transsphenoidal surgery. *Neurosurgery* 1990;27:60–68.

156. Landolt A: Transsphenoidal surgery of pituitary tumours: its pitfalls and complications; in de Villiers JC (ed): *Some Pitfalls and Problems in Neurosurgery*. Prog Neurol Surg. Basel, Karger, 1990, vol 13, p 1.

157. Vaidik Chauhan and Heli Vasani., et al. “Visual Outcomes and Factors Affecting the Outcome After Transsphenoidal Excision of Pituitary Macroadenoma”. *Acta Scientific Otolaryngology* 1.2 (2019): 17-21.

158. Grković D, et al. Suprasellar Meningiomas: Postoperative Visual Outcome *Med Pregl* 2016; LXIX (5-6): 146-152.

159. Nipat Aui-aree MD*, et al Three Years Experience of Suprasellar Tumours in Neuro-Ophthalmology Clinic *J Med Assoc Thai* 2010; 93 (7): 818-23

160. Zulfiqar F, Khan AA, Imran M, Ahmed SI, Bareeqa SB, Ali MF, Ghori SA, Shera MT. Suprasellar tumours; pre-operative and post-operative visual status in patients. A tertiary care experience from Karachi, Pakistan. *Professional Med J* 2018; 25(4):603-609. DOI:10.29309/TPMJ/18.4357

161. Dutta P., et al. “Visual outcome in 2000 eyes following microscopic transphenoidal surgery for pituitary adenomas: Protracted blindness should not be a deterrent”. *Neurology India* 64 (2016): 1247-53.

162. Zevgaridis D, Medele RJ, Muller RJ, Muller A. Meningioma of the sellar region presenting with visual impairment: impact of various prognostic factors on surgical outcome in 62 patients. *Acta Neurochir (Wien)*. 2001;143:471-6

163. Gnanalingham KK, Bhattacharjee S, Pennington R, Mendoza J, Ng N: The time course of visual field recovery following transsphenoidal surgery for pituitary adenomas: predictive factors for a good outcome. *J Neurol Neurosurg Psychiatry* 76:415-419, 2005.
164. Bristot ARR, Domenicucci M, Cantore G. Meningiomas of the tuberculum sellae: our experience in 69 cases surgically treated between 1973 and 1993. *Journal of neurosurgical sciences*. 1999; 43(4):253.
165. Freda PU, Wardlaw SL, Post KD. Unusual causes of sellar/parasellar masses in a large transsphenoidal surgical series. *The Journal of Clinical Endocrinology & Metabolism*. 1996; 81(10):3455-9.
166. Trautmann JC, Laws ER Jr. Visual status after transsphenoidal surgery at the Mayo Clinic, 1971- 1982. *Am J Ophthalmol* 1983; 96: 200-8.
167. Mahapatra AK, Tandon DA, Bhatia R: Prospective study of 100 patients with optic nerve injury. *Neurol India* 40:17–21, 1992
168. Kerrison JB., et al. *American Journal of Ophthalmology* 130 (2000): 813-820
169. Cottee LJ, Daniel C, Loh WS, Harrison BM, Burke W. Remyelination and recovery of conduction in cat optic nerve after demyelination by pressure. *Exp Neurol* 2003;184:865-77.
170. Lee JH, Jeun SS, Evans J. Surgical management of clinoidal meningiomas. *Neurosurgery*. 2001;48:1012-21.
171. Jakobsson KE, Petruson B, Lindblom B: Dynamics of visual improvement following chiasmal decompression: quantitative pre- and postoperative observations. *Acta Ophthalmol Scand* 80:512-516, 2002.
172. de Paiva Neto MA, Vandergrift A, Fatemi N, Gorgulho AA, Desalles AA, Cohan P, Wang C, Swerdloff R, Kelly DF: Endonasal transsphenoidal surgery and multimodality treatment for giant pituitary adenomas. *Clin Endocrinol (Oxf)* 72:512-519, 2010.

ANNEXURE A

The International Classification of Diseases 11 (2018) classifies vision impairment:

Mild – presenting visual acuity worse than 6/12 but not worse than 6/18

Moderate – presenting visual acuity worse than 6/18 but not worse than 6/60

Severe – presenting visual acuity worse than 6/60 but not worse than 3/60

Blindness – presenting visual acuity worse than 3/60

ANNEXURE B

PATIENT PROFORMA

Sree Chitra Tirunal Institute for Medical Sciences & Technology

“Visual outcomes in suprasellar tumors”

A. GENERAL INFORMATION

-Anonymized Patient ID:

-Age

-Gender

-Family history/Neurofibromatosis

B. CLINICAL DETAILS

GCS on admission:

Preoperative Vision profile :

- Visual acuity
- Visual field
- Colour vision
- Fundus examination
- Near vision
- OCT
- Ophthalmic evaluation if any

Karnofsky performance score:

Symptoms at presentation :

Size of tumour(in mm) :

Extent of tumour:

Radiological investigations :

B. INTRAOPERATIVE EVENTS-

- Surgical approach

Extent of tumour resection

-vascularity:

-consistency:

C. POST-OPERATIVE EVENTS

- Postoperative vision profile:

- Visual acuity
- Visual field
- Colour vision
- Near vision

-Duration of ICU stay (days):

-Duration of post-op hospital stay (days):

-Tumor pathology :

- Infection (surgical site infection):

-CSF leak:

-Pseudomeningocele (transcranial approach):

-Re-exploration / Decompression:

D. STATUS ON DISCHARGE

-Karnofsky performance score :

-visual acuity /visual field

E. STATUS ON FOLLOW UP ON 1, 3 , 6 MONTHS

-Karnofsky performance score:

-visual status

- Any other deficit

-Radiological follow up

ANNEXURE C

Institution Ethics Committee approval letter



श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)

Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

Institutional Ethics Committee (IEC Regn No. ECR/189/Inst/KL/2013/RR-16)

22.08.2019

SCT/IEC/1389/JULY-2019

Dr. Shah Shreykumar Pravinchandra
Resident, Department of Neurosurgery
SCTIMST, Thiruvananthapuram

Dear Dr. Shah Shreykumar Pravinchandra,

The Institutional Ethics Committee reviewed and discussed your application to conduct the study entitled "SUPRASELLAR TUMORS, PRE-OPERATIVE AND POST-OPERATIVE VISUAL STATUS EVALUATION IN A TERTIARY CARE CENTER (IEC/1389)" on 26th July, 2019.

The following documents were reviewed:

Original submission

1. Covering Letter addressed to the Chairperson, IEC, SCTIMST with checklist
2. TAC Approval Letter
3. IEC Application Form
4. Project Proposal
5. Proforma
6. Forwarding Letter from the HOD
7. Patient Information Sheet and Informed Consent Form in English and Malayalam
8. CV of Principal Investigator and Co-Principal Investigators

Revised submission

1. Covering Letter addressed to the Chairperson, IEC, SCTIMST with checklist
2. TAC Approval Letter
3. IEC Application Form
4. Project Proposal
5. Proforma
6. Forwarding Letter from the HOD
7. Patient Information Sheet and Informed Consent Form in English and Malayalam
8. CV of Principal Investigator and Co-Principal Investigators

Page 1 of 2

The following members of the Ethics Committee were present at the meeting held on 26th July, 2019 at Noshir H Wadia Conference Hall, AMCHSS, SCTIMST

SL. No.	Member Name	Highest Degree	Gender	Scientific /Non Scientific	Affiliation with Institution(s)
1.	Dr. Harikrishnan S	MD, DM (Cardiology) DNB (Cardiology)	Male	Clinician	Yes
2.	Dr. Kala Kesavan, P	MBBS, MD	Female	Basic Medical Scientist	No
3.	Smt. Sathi Nair	MA (English Literature)	Female	Lay Person	No
4.	Dr. Christina George	MD Psychiatry	Female	Clinician	No
5.	Dr. Mala Ramanathan	PhD	Female	Social Scientist (Member Secretary)	Yes

IEC Decision

The IEC approved the conduct of the study in the present form.

Remarks:

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

There was no member of the study team who participated in voting / decision making process. The ethics committee is organized and operated according to the requirements of Good Clinical Practice and the requirements of the Indian Council of Medical Research (ICMR).

Sincerely,



Mala Ramanathan
Member Secretary, IEC

ANNEXURE D

Plagiarism Report

Plagiarism Checker X Originality Report



Plagiarism Quantity: 6% Duplicate

Date	Sunday, August 23, 2020
Words	942 Plagiarized Words / Total 17079 Words
Sources	More than 86 Sources Identified.
Remarks	Low Plagiarism Detected - Your Document needs Optional Improvement.

TUMORS of suprasellar region are important because they lie in close proximity to visual apparatus and neuroendocrine structures. Coupled with their anatomical location is their delayed clinical presentation. These tumors have major modes of presentation in the form of visual symptoms (visual acuity decline, defect in the visual field and involvement of ocular movements), hormonal disturbances, or raised intracranial pressure. When their size increases they can cause hydrocephalus by obstructing the Foramen of Monro. The majority of these patients present with visual symptoms only, and are initially treated by ophthalmologists and then referred to neurosurgeons, which further accounts for the delay in initiation of proper treatment.¹

Blurring of vision is one of the most common complaint of visual pathway in suprasellar tumors.² This visual deterioration, due to optic nerve compression is common indication for surgery Tumors in this region consist of pituitary adenomas, meningiomas, craniopharyngiomas, epidermoid cysts, germinomas, chordomas, and so on. Commonest tumor is pituitary adenomas in suprasellar region followed by meningiomas and

Sources found:

Click on the highlighted sentence to see sources.

Internet Pages

- <1% <https://www.ncbi.nlm.nih.gov/pmc/article>
- <1% <https://issuu.com/doctorneuro2002/docs/a>
- <1% <https://www.scribd.com/document/62027088>
- <1% <https://www.researchgate.net/publication>
- <1% <https://www.neurosurgicalatlas.com/volum>
- <1% <https://doctor2017.jumedicine.com/wp-con>
- <1% <https://www.sciencedirect.com/science/ar>
- <1% <https://www.researchgate.net/publication>
- <1% <https://www.ncbi.nlm.nih.gov/pmc/article>
- <1% <https://www.researchgate.net/publication>
- <1% <https://www.researchgate.net/publication>
- <1% <https://www.healthtap.com/q/what-does-a>
- <1% <https://www.sciencedirect.com/topics/med>