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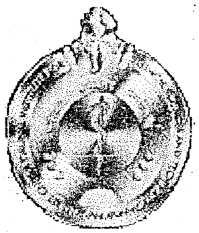


PROJECT REPORT

Name : **Dr Mathew Abraham.**
Programme : **M. Ch Neurosurgery.**
Month and year of Submission : **November 2002**

Certificate

I, Dr Mathew Abraham, Resident in Neurosurgery, SCTIMST here by declare that I have actually carried out this project - A Retrospective Statistical Study on parasagittal meningiomas.

Thiruvananthapuram
9/11/2002

Dr Mathew Abraham

Forwarded- He has carried out the minimum requirements for the project.

Thiruvananthapuram

Prof. R.N Bhattacharya
Head of the Department
Of Neurosurgery

PROJECT REPORT

Title of the project:

Retrospective Statistical Study on Parasagittal Meningiomas

Name : Dr Mathew Abraham.

Programme : M.Ch. Neurosurgery.

Month & Year of Submission : November – 2002.

Acknowledgement

I owe my sincere and profound gratitude to Prof RN Bhattacharya Professor and Head of Department of Neurosurgery for his constant guidance, support and constructive criticism during the period of study.

I would like to thank Prof Suresh Nair for guidance and for providing the necessary outlook required for a retrospective study. His depth of knowledge and rich experience were precious resources to bank on.

I am deeply indebted to Dr Ravi Mohan Rao for helping me out in several technical aspects of my study. Dr Rajesh B J deserves special thanks for his constant guidance, encouragement and practical advice.

The Medical Records of SCTIMST are certainly well maintained. The capable and helpful Medical Records Department of the Institute is certainly an asset to the scientific world. I am extremely grateful to all the staff and students in Medical Records Department, and I am most convinced that without their help and toil this study would not have been possible.

I would also like to thank scores of persons who have directly or indirectly helped me in completing this study.

Dr Mathew Abraham.

Contents

PREFACE

Meningiomas form 10-15% of all intracranial neoplasms. It is generally accepted that anatomical incidence of meningiomas roughly parallels the distribution of arachnoid villi. The highest distribution of arachnoid villi is in the parasagittal region. The incidence of meningiomas is also highest in this region. In the experience of Russell and Rubinstein the incidence of parasagittal meningiomas is approximately 50% and majority of these favour the middle third parasagittal region. Though an extremely common lesion in Neurosurgical practice parasagittal meningiomas pose, considerable uncertainties, regarding symptomatology, surgical options, operative morbidity, chance of recurrence and need for alternate management options. The intimate relation with the superficial venous drainage of the brain warrants parasagittal meningiomas to be considered different from meningiomas originating from other sites in all the above mentioned aspects.

The results from different series has varied considerably enough to prevent an individual centre from developing any clear cut guidelines from the management point of view, taking into consideration the innumerable variables involved. This study attempts to analyze this complex matter from the experience of a specialized tertiary care centre, serving a large population in a developing country.

Introduction

A large number of patients with meningiomas were operated in our Institute since it was formed in the late seventies. Parasagittal meningiomas form a substantial percentage of them. We have had a reasonably good follow up of majority of these patients over several years. To understand the clinicopathological course of this slowly progressive disease prolonged follow up is a must. Cases operated up to 1997 have been taken into consideration for the study for this reason.

Records of all patients are kept in our Medical records library since the onset of the Institution and are recoverable easily for each follow up the patient undergoes, and is also available for ready reference. This has been utilized for this retrospective statistical study.

The clinical profile of patients at presentation, a search for aetiology, imagological findings, operative correlation, extend of resection, histopathological study, operative consequences, and available follow up , has all been included in the study. Inevitable subjective variation is unavoidable in studies based on prior written records of different observers. Efforts are made to keep the study objective to the maximum extend possible.

The study is expected to improve our understanding of clinicopathological, management and prognostic aspects of parasagittal meningiomas in this part of the world, and to set realistic goals in the future management of patients with this complex clinical problem.

Review of the current literature is also made for a better outlook and for the purpose of comparison.

Aims and Objectives

- To study the clinical presentation of parasagittal meningiomas.
- To study the imagological findings in cases of parasagittal meningiomas.
- Study the operative findings and extend of resection and factors influencing the same.
- Study immediate and delayed consequences directly related to operative intervention, especially to resection grade.
- Study the histopathology and to understand the clinicopathological correlation.
- To study the recurrence rate and its correlation with extend of resection, and histopathological nature of lesion.
- To study the prognostic factors and ideal management options including adjuvant treatment.

Materials and Methods

Case records of patients who had undergone surgery for parasagittal meningiomas in SCTIMST since the beginning of the institution were studied. The inclusion criteria were unequivocal evidence of attachment of the lesion with the superior sagittal sinus, as observed during surgery. Cushing and Eisenhardt definition 'one that fills the parasagittal angle, with no brain tissue between the tumour and the superior sagittal sinus' was used as the reference. Only patients whose files gave clear records of, preoperative clinical status, details of imaging, operative findings, postoperative condition, and histopathological findings were included in the study.

The study was totally retrospective and based on written records only. Information from the records were collected by a single observer, and recorded on to a pre prepared performa.

Since the natural history of the illness spans over several years, only patients operated for the first time at least 5 years earlier were included in the study. Hence the last patient included was operated in January 1997. 144 patients met the criteria to be included for the study. Every attempt was made to follow-up these patients up to March of 2002. So the period of follow-up had varied from over 20 years to five years.

The performa used for the study had 94 variables. The variables spanned the clinical profile, search into aetiology, details of imaging, operative findings, immediate and delayed operative consequences, histopathology and maximum available clinical and imagological follow up. Data entry was done using the MS excel work sheet and analysis was done with the help of SPSS – statistical programme.

Review of Literature

"There is today nothing in the whole realm of surgery more gratifying than the successful removal of a meningiomas with subsequent perfect functional recovery, especially should a correct pathological diagnosis has been previously made. The difficulties are admittedly great, sometimes insurmountable, and though the disappointments still are many, another generation of neurological surgeons will unquestionably see them largely overcome."

Harvey Cushing, 1922.

History

Nomenclature:

Meningiomas carried a series of confusing names – fungoid tumour, cylindroma, endothelioma, fibroma, meningiothelioma etc to name a few. In 1863 Virchow had noted the presence of granules in these tumours and called it psammomas (sand like). In 1922 Harvey Cushing decided to end the existing confusion in nomenclature proposed the name 'Meningothelioma' and later changed it to "Meningioma". This tissue name had stood the test of time and was ideal for a tumour which occurs in various locations and has such a varied histological picture.

Cell of origin:

Pacchioni in 1705 had described arachnoid granulations (pacchionian granulations). Rainey 1846 had suggested the arachnoid origin of these granulations.

John Cleland who was Professor of Anatomy at Glasgow in 1864 described "villous tumours of the arachnoid". He suspected that meningiomas originated in the pacchionian granulations.

Martin Schmidt of Strasbourg after a microscopic study in 1902 concluded that the cells of meningiomas resembled the cell clusters capping the arachnoid villi – the arachnoid cap cells. This was confirmed later (1915) by Cushing and Weed LH.

Operative efforts:

Laurence Heister in 1743 was perhaps the first to attempt surgical treatment of a meningioma. The patient was a 34 year old Prussian soldier and Heister applied caustic of lime on the tumour. The patient had later died of possible infection.

Sanobi Pecchiolo Professor of operative medicine Siena University (1830 -1851), had published series that contained description of removal of a large meningioma from the right sinciput, by taking out a triangular bone flap. The operative site was covered with cambric soaked in sweet almond oil. The patient had survived and followed up for over 2 years.

In 1879 Sir William Macewan had successfully removed an olfactory groove meningioma in a 14 year old girl. The patient had survived and was in good condition.

Francesco Durante of Mazzoni Clinic Rome removed a meningioma from the base of skull of a patient in 1883, the outcome is not known. In 1885 he removed an apple sized (weighing 70gms) olfactory groove meningioma from a 35 year old woman. The patient had survived and required a resurgery for recurrence in 1896 (after 11 years). The patient was in good health in 1905.

William W.Keen in 1887 was credited with the distinction of being the first American surgeon to successfully remove a meningioma. The patient was a 26 year old man.

In 1893 GI Pribytkovin Moscow had removed a meningioma of the sphenoid wing region from a 33 year old patient with a past history of head injury. The patient had died a few hours later.

In 1897 B.Kozlovzki removed a meningioma from the left parasagittal region measuring 16.5cm X 6.5cm. The patient had postoperative aphasia and right hemi paresis from which he gradually recovered.

In 1905 Harvey Cushing operated on US Army general Leonard Wood, who had a prior history of head injury and removed the epidural part of a right parasagittal meningioma. In 1909 he removed the right parasagittal meningioma in 2 sittings 4 days apart. The patient was discharged in good health in about a month. In 1920 Leonard Wood was the Republican favorite to succeed Woodrow Wilson as the US President. In 1927 Wood again presented with left sided spasticity and was reoperated by Cushing. He died in the post operative period due to ventricular hemorrhage.

Cushing had discussed 85 cases of meningiomas in his Cavendish Lecture in 1922 and this was later published in Brain. (4)

Mac Carty in 1961 has rightly said "If we were to designate an intracranial neoplasm that has had the most effect on the development of neurological surgery, very likely the intracranial meningioma would be prominently considered."

Aetiology of Meningiomas:

Cells of the body have been divided into permanent, stable and labile cells. Permanent cells do no multiply during the life span of the individual. Neurons and muscles are examples of the same. Stable cells do not divide normally but multiply when there is need such as cell damage. Hepatocytes are typical stable cells. Labile cells are in a continuous process of division in order to replace those lost in day to day wear and tear.

Meningeal cells are considered to be stable cells and normally have a low turnover. Factors that increase the cellular turnover predispose to greater chance for chromosomal mutations. Meningiomas have been linked to multiple environmental and genetic factors. But no single factor can be accounted for a majority of meningiomas. Infact the causes may be multifactorial or different tumours may be related to different factors.

Possible aetiological agents include head trauma, irradiation – both high dose and low dose, viruses and genetic disorders like Neurofibromatosis type II.

Trauma:

Cushing's famous patient General Leonard Wood had history of injury adjacent to the site where the meningioma had developed later. Cushing and Eisenhardt had later reviewed 295 cases of intracranial meningiomas and found 93 cases (32%) in which there were a history of head injury. Based on this Cushing had concluded that head trauma was a significant aetiological factor.

Several small series were later published linking the relation between trauma and meningioma. But in some cases doubts were expressed whether the head trauma had focused the attention of the patient and the physician to hitherto unnoticed symptoms and signs.

Several case control studies have however failed to find an association between head trauma and meningiomas. Choi et al in a case control study interviewed 24 patients and matched controls for a history of prior trauma. They had defined brain injury as "fractured skull, unconsciousness or bleeding from the head which lead to hospitalization". They found no significant difference between cases and controls. Annegers et al(2) followed in a prospective study 2953 patients with history of head trauma for a total of 29859 person years and found no increase in incidence of any intracranial tumour in the affected population.

Hence confusion still exists regarding the importance of trauma as an aetiological factor. Although head trauma may be a cofactor it is now generally believed that trauma may not be a significant aetiological factor, at least from the angle of follow up of head injured patients.

Radiation:

Radiation is an important mutagen capable causing breaks in single and double stranded DNA. Specific criteria have be set forward to identify human tumours as radiation induced .These are (1) tumour appearing in the field of radiation,(2) a long latent period existing between the time of radiation and the appearance of the tumour and (3) a histological distinction between the tumour and any previous neoplasm.

On these criteria tumours were identified both after low dose radiation for benign conditions and also after high dose radiation for prior head or neck neoplasm.

Low dose radiation was given to nearly 17000 children migrating to Israel between 1949 and 1960 for tinea capitis. In 1974 Modan et al retrospective evaluated these children and found a 4 times higher incidence of meningiomas in them when compared to controls. The latent period ranged from 16 to 21 years. Another study by Shore et al also showed a similar result. Further studies by Soffer et al, Rubinstein et al have brought out several important aspects of radiation induced meningiomas. They are more likely to arise on the convexity(80%) ,more likely to be multiple and have a much higher chance of being malignant and have a higher cellularity, cellular pleomorphism and have a high frequency of giant cells.

High dose radiations given for malignant tumours most frequently induce malignant fibrosarcomas. There are several reports of meningiomas after high dose radiation particularly in children receiving radiation for medulloblastomas. The nature and ultimate course of these lesions is not clear due to natural history of the primary tumours.

Viruses:

Viruses are found to be capable of causing several CNS tumours in animal models. A model of meningioma is nonexistent at the moment. Recently small portions of viral DNA have been identified in many human tumours including meningiomas particularly SV 40 Viral DNA.The Inoue-Melnick virus a DNA virus has been identified in many human meningioma specimens. Hence strong evidence exists to link DNA tumor viruses with human meningiomas, but a definite role for viruses in oncogenesis of meningiomas is not yet established.

Genetic factors:

Cytogenetic studies have identified several chromosomal abnormalities in meningioma cells. The most consistent of these is loss of one of the chromatids at 22. Neurofibromatosis Type II an autosomal dominant is associated with very high incidence of meningiomas. The association of bilateral acoustic neurinoma and meningiomas in these patients show that there is a common mechanism in the development of both tumours. RFLP (restriction fragment length polymorphism – a stably inherited variation in the chromosomal structure) mapping of chromosome 22 has shown deletions in DNA map. The lost gene may be exposing a hitherto inactive oncogene to cause multiple lesions. An alternate explanation is the possibility of loss of a tumour suppressor gene from chromosome 22. Similar changes in Chromosome 22 have been demonstrated in sporadic cases of meningioma both within the meningioma and in other stable cells.

Currently loss of DNA from Chromosome 22 can be demonstrated in 40% of meningiomas. It is likely that the remaining 605 may have alterations in the Chromosome 22 which may be point mutations or small aberrations too small for detection with RFLP analysis. Tumourogenesis in both familial and sporadic cases may well be explained with certainty once our understanding of the human genome is reaches completion.

Incidence and Location of Parasagittal meningiomas:

By far the incidence of meningiomas in general is considered to be between 11 and 15 % of all intracranial tumours. It has varied from 11.3% in Cushing's series to about 18 % in the series by Olivecrona. Large surveys like the Zulch study (1986) and the Mahaley et al (1989) survey have shown an incidence percentage of 16.6 and 23.1 percentage respectively.

The incidence of Parasagittal meningiomas in most studies has been approximately between 20 and 32 percent of all meningiomas. In fact it has paralleled the incidence of convexity meningiomas. In Olivecronas series of over 902 cases the incidence of convexity meningiomas were half that of parasagittal meningiomas. (17% and 34% respectively).

In his paper "The parasagittal Meningeal Fibroblastomas" Elsberg (1931) had confirmed that lesions occur more often in front of than behind the rolandic area. Parasagittal meningiomas occur with greatest frequency in the middle third of the Superior Sagittal Sinus, with about half of all lesions occurring in that location. An explanation is the highest incidence of pacchionian bodies in the location. The case of falcine meningiomas is different in that half of them arise in the anterior third of the falx, where infact half the bulk of the falx lies. In Cushing's series the incidence of anterior to middle to posterior third lesions were 22, 37 and 6 respectively. In Olivecronas series it was 57, 111, and 31in the same order.

Pathology of Meningiomas

Meningiomas are currently believed to take origin from the arachnoid cap cells. This was first proposed by Cleland and Robin. It had remained in obscurity for 3 decades in the background of prior theories proposed by Virchow that the cell of origin was the meningothelial or meningoblastic cell. It was revived by Schmidt.

Meningiomas show tremendous histological diversity. This diversity of cells can be explained in 2 ways. (1) While arachnoid cells are the major components of a majority of tumours other cellular components of the pia arachnoid and dura such as fibroblasts, endothelial cells also take part in the formation of the tumour mass. (2) Alternatively the arachnoid cells may be pleuripotent and capable of forming vessels and fibroblasts, in varying proportions following neoplastic transformation. Another explanation for the diversity of cells formed is that the meninges is formed by the fusion of the cells derived from the embryonic mesoderm and the neural crest cells with a potential of multiple functional properties. (Janet et al).

Common and uncommon sites: the incidence parallels the incidence of arachnoid villi. In order of descending frequency the arachnoid villous population is present in: 1. Parasagittal, 2. Cavernous, 3.tuberculum sellae, 4. Lamina cribrosa, 5. foramen magnum and 6. trocular zones.

Uncommon sites for meningiomas in the neuraxis include the spinal canal (with a 3 times higher incidence in females), within the ventricles, orbital, intraosseous, pineal, and extracalvarial.

Ectopic sites include nasal cavity, paranasal sinus, the parotid gland, lung, adrenal gland, and mediastunum. Migration of arachnoid cells along the spinal and cranial nerves is an explanation for these abnormal sites.

Macroscopic features of Meningiomas:

The most characteristic features are the well defined borders and the broad base on the dural surface. They seldom cause pial breeches and tend to invade dura and dural sinus in preference.

The cut surface is pale and transparent with a whorled appearance when fixed in formalin. A gritty consistency is a common feature and is related to the presence of psammoma bodies.

Despite a very high vascularity, hemorrhage into the tumour is very rare with only 43 cases reported in a review in J Neurol Neurosurgery and Psychiatry 1980 by Helle TL.

Massive calcification may be rarely seen. Usually the nidus of calcification is psammoma body. Microscopic calcification of the lesion is much more common.

Hyperostosing en plaque meningioma is mainly a disease of the bone with the meningioma cells invading the haversian canals initiating reactional hyperostosis. Bone destruction is relatively unusual and is related to aggressive and malignant meningiomas and is encountered particularly in haemangiopericytomas. Convexity meningiomas are frequently associated with an endostosis. The endostosis is minimal when considering the size of the lesion. The bone appears hard with no obvious involvement by the tumour.

Classification of meningiomas on the basis of histopathological features.

In 1993 WHO classified tumours of the meninges into 2 major groups. (1) Tumours arising from Meningothelial or arachnoid cap cells otherwise called meningiomas and (2) tumours arising from mesenchymal non-meningothelial cells.

The WHO Classification of Tumours of the Meninges (1993);

1. Tumours of Meningothelial cells.
A. Meningioma
    1. Meningothelial
    2. Fibrous (fibroblastic)
    3. Transitional (mixed).
    4. Psammomatous.
    5. Angiomatous.
    6. Microcystic.
    7. Secretory.
    8. Clear cell.
    9. Chordoid.
    10. Lymphoplasmocyte rich.
    11. Metaplastic.
B. Atypical Meningiomas.
C. Papillary meningioma.
D.Anaplastic (malignant) meningioma.

The last WHO classification put forward in 1979 was modified considerably in the present classification. New subtypes have been included namely microcystic, secretory, clear cell, chordoid, lymphoplasmocyte rich and metaplastic. Papillary type has been moved to its own category due to its aggressive behavior. The term angioblastic has been eliminated and is now called hemangiopericytoma. The 1993 classification recognizes 3 forms meningioma that were included to reflect three levels of biological behavior: benign, atypical and anaplastic (malignant).

Typical Histological features of classic meningiomas.

Meningothelial:

Otherwise called syncytial or endotheliomatous, they resemble their cell of origin. The cells are arranged in sheets or layers with no clear borders seen for individual cells. Hence the name syncytial. The nuclei are oval with delicate chromatin and small inconspicuous nucleoli. A distinctive and useful identifying feature is the presence of intranuclear cytoplasmic pseudo inclusions, where a portion of the cytoplasm invaginates into the nucleus. The central clearing gives the Orphan Annie appearance. Meningothelial meningiomas do not show the clear whorled pattern that has almost become synonymous with the histological picture of meningiomas.

Fibroblastic:

This has the appearance of multi –laminated sheets of interlacing elongated spindle cells, in a background of collagen and reticulin fibers. Poorly defined whorls are seen. The nuclei have the features of the prior described form, even though the cells may appear elongated. Focal pseudopalisading of nuclei may be seen in this form of lesion.

Transitional:

This is a mixture of both fibroblastic and meningiotheliomatous types. The characteristic feature is the formation of cellular whorls that are separated by elongated spindle cells with focal deposition of collagen. The whorls may be formed centering on blood vessels. Psammoma bodies may be seen in this type of lesions.

Psammomatous:

Numerous sand like calcified concentric structures containing varying amounts of collagen, calcium apatite and iron, is the characteristic feature of this type of meningiomas.The psammomas are found in highest number in transitional meningiomas.

Angiomatous:

These meningiomas are hyper vascular and the characteristic feature is the presence of numerous vascular channels within. The pattern is also indicative of a more aggressive pattern. But a significant number of vascular channels are hylinised and resemble those seen in benign sclerosing haemangiomas.

Microcystic:

Masson had proposed the name of humid meningiomas for these lesions. They are typically soft suckable and glistening. Numerous microscopic cysts formed by many small intracellular and intercellular cystic spaces.

Xanthomatous:

They contain intra cytoplasmic vacuoles containing lipids. These are positive for lipid stains. This is different from the formation of foam cells within the meningioma which is a degenerative change. Very often the xanthomatous variant is seen in focal regions in the meningioma.

Myxoid:

This variant tends to occur in fibroblastic meningiomas. Stellate or multipolar cells are seen to be distributed within a myxoid matrix which stains with PAS.

Lymphoplasmocyte rich:

Lymphofollicular formation and infiltration by inflammatory cells is seen in this form of meningiomas. There is often an increase in the amount of globulin level in the CSF of these patients. Mononuclear infiltration is also seen in the more aggressive variants of meningiomas.

Papillary meningiomas:

Now recognized as distinct entity papillary formation is the hallmark. They have a more aggressive behavior and border on malignant meningiomas. They have an increased frequency in younger patients (50%). There is an incidence of 30% metastasis to distant sites. They have high cellularity and have numerous mitotic figures.

Malignant meningiomas:

Distant metastasis is an unequivocal evidence of malignancy. But often invasion of brain parenchyma associated with aggressive histological features like increased mitosis, anaplasia and necrosis is taken as a marker for these types of meningiomas. They have a significantly higher incidence among parasagittally located meningiomas.

The Superior Sagittal Sinus: Bonnal and Brotchi classification (3)

The Superior sagittal sinus extends from the Crista galli to the trocula and is an endothelium lined space triangular in cross section between dural layers which forms all the 3 walls. It is the main draining site for the superficial veins of the supratentorial compartment. Alternate routes of drainage are through the superficial middle cerebral vein and the vein of Labbe.

Anterior to the coronal suture the Superior Sagittal Sinus is small and can be divided with no significant morbidity. Posterior to this the compromise of a patent sinus may result in severe and often unpredictable increase in morbidity and mortality. Hence the preservation of the patency of the sinus is utmost importance.

Akira Hakuba modification of the Bonnal and Brotchi classification of parasagittal meningiomas into 8 types is useful in classifying the extend of sinus involvement.

Type I Meningioma attached only to the outer surface of the lateral wall of the sinus.
Type II Part of the tumour lies in the corner of the sinus.
Type III The meningioma invades one whole wall of the sinus.
Type IV The meningioma invades 2 sinus walls.
Type V Meningioma invading 1 wall and grows largely within the lumen with significant stenosis of the sinus lumen.
Type VI Meningioma invades all 3 walls and the lumen is very stenotic.
Type VII All walls are invaded and the lumen is obliterated.
Type VIII A bilateral meningioma invading 3 sinus walls with complete obstruction of the sinus.

Clinical presentation of Parasagittal Meningiomas

In 1922 Cushing summarized the clinical presentation of parasagittal meningiomas as follows:

"When a parasagittal tumour is surmounted by a dome of hyperostosis, its situation and identity are easily recognized. Also when Jacksonian seizures originating in a foot are followed in the course of years by a spastic paralysis which begins in the corresponding leg, one may feel reasonably assured that a parasagittal meningioma will be disclosed at operation, provided the mesial edge of the hemisphere can be brought into view.

When on the other hand, the parasagittal tumours arise well in front of the motor cortex or what is less common, posterior to it, they may be exceedingly difficult to localize by symptoms alone."

Parasagittal lesions are often very slow growing and would have reached a large size before causing symptoms. Due to the strategic location, Jacksonian seizures and raised ICP due to obstruction to the superior sagittal sinus may present early even before the lesion has reached any significant size to cause mass effect or distortion on its own. The classical features of parasagittal meningiomas with their approximate incidence are as follows.

Presentation	Tumour location			
	Ant(50)	Central(89)	Post (14)	Total(153)
Focal fits	1(2%)	29(33%)	3(21%)	33(22%)
Generalized fits	13 (26%)	10 (11%)	-	23 (15%)
Headache	18(36%)	9(10%)	5(36%)	32(21%)
Mental Symptoms	18(36%)	6(7%)	3(21%)	27 (18%)
Monoplegia (leg)	2(4%)	23 (26%)	-	25(16%)
Visual symptoms	1(2%)	4(5%)	3(21%)	8(5%)
Swelling	6(12%)	2(2%)	-	8(5%)
Dysphasia	-	4(4%)	-	4(3%)
Focal ischemia	-	2(2%)	-	2(1%)
Vertigo	-	1(1%)	-	1(1%)
Hemiplegia	-	6(7%)	2(14%)	8(5%)
Monoplegia (arm)	1%	1(1%)	-	2(1%)
Multiple symptoms	7(14%)	11(12%)	3(21%)	21(14%)

1. Focal seizures may be an early symptom seen in about 33% of patients.
2. Generalized Seizures; more frequent in anteriorly located tumours they are seen about 23% of patients.
3. Raised ICP features; most frequent in posteirorly located tumours and is the presenting symptom in one third of patients.
4. Behavioral abnormality is seen in about 27% of patients.
5. Monoplegia (LL) 27%of patients present with contralateral monoplegia.
6. Hemiplegia is relatively rare with an incidence of 8 %.
7. Visual symptoms are seen in about 8% of patients and are due to raised ICP or interference of the visual pathway.
8. Bony swelling is seen about 8 % of patients.

Imaging of Parasagittal Meningiomas:

Imaging in parasagittal meningiomas serves for diagnosis and preoperative evaluation. The location, extend, size, vascularity, patency of the superior sagittal sinus are to be established by CT (and or MRI) and angiography.

Plain Radiography:

There are 3 hallmarks of a meningioma on plain X-ray, namely hyperostosis, increased vascular marking relative to the other side, and calcification.

Hyperostosis is the most common findings and is seen in 38-61% of all meningiomas. Extend beyond the midline is seen in parasagittal meningiomas. The convex appearance of the hyperostosed bone is a feature of importance and is termed blistering.

The second most common finding is the vascular marking seen due to the enlargement of the branches of the middle meningeal artery.

Calcification is the least common and most specific of plain film findings in meningiomas. The reported incidence is 3 -18% CV Mosby 1971 Nonspecific punctuate irregular ad curvilinear peripheral calcification may be seen. The more characteristic pattern is fine diffuse nodular (cloud like) or dense ossific calcification.

CT imaging in Parasagittal meningiomas:

Parasagittal meningiomas share all the typical features of meningiomas in general. They are iso to hyper dense to the contiguous brain matter, homogenous and sharply marginated.20 to 25% of lesions may be calcified. Calcification is present in a number of ways. Tiny punctuate calcification is very common and may contribute to the hyper density of the tumour. Large dense calcification of the entire tumour is less commonly seen. Dense calcification when enhancement patterns cannot be seen is sometimes seen.

75% of meningiomas are hyperdense. About 20 % lesions may be isodense. Hypodense lesions are found in approximately 1-3% of lesions. Xanthomatous and lipoblastic tumours have low negative attenuation values.

Contrast enhancement of intense nature is compulsory criteria for all meningiomas .However approximately 10% of meningiomas may have only mild to moderate or patterned enhancement (Osborn). It is typically homogenous. Inhomogenous enhancement occurs when there are cystic changes, necrosis or metaplastic changes. Bright enhancement may be seen for Angiomatous variant of meningiomas. The cause of enhancement is not the demonstrable hypervascularity evident on DSA but due to a partially deficient barrier causing the iodinated contrast material infiltrating into the interstitial spaces.

Meningiomas of the parasagittal region are broadly based on the SSS and have a wide almost obtuse angle with the same. This indicates the primary attachment to the sinus. By definition the there must be no brain matter between the SSS and the lesion.

Dural enhancement pattern in the form a tail is not a reliable radiological sign but tends to indicate the meningeal origin and the site of primary attachment.

A coronal section is most helpful for parasagittal meningiomas and helps in differentiating them form falcine meningiomas. Growth into the sinus is sometimes seen in the form of a solid cord, but CT is a poor indicator of the patency of the sinus.

CT is a sensitive indicator of hyperostosis and bony erosion. When viewed in a wide bony window hyperostosed new bone has characteristic features. The new bone is less homogenous and has a thickened cortex and irregular margins.

Odema around a Parasagittal meningioma shows a different pattern from other meningiomas. Slow growing benign lesion causes the least odema. Much smaller lesions may cause extensive odema. A possible explanation is the obstruction to the SSS. Several studies have not established this with certainty (Smith et al Neurosurgery 1981.)

Magnetic Resonance Imaging in Parasagittal meningiomas:

MRI of the brain is the most reliable and useful imaging modalities as far as Parasagittal meningiomas are concerned.

The main advantages of the MRI are, the exact differentiation of the tumour brain interface, and making out the exact configuration of the lesion. The arterial feeders can be visualized with some degree of certainty. Most importantly the extend of involvement of the Superior sagittal sinus can be made out as reliably as a DSA from an MR Venogram.

Many features will help in determining the histological type of the meningioma. Focal calcification is a feature of fibroblastic and transitional meningiomas, but is not seen in syncytial meningiomas. Psammomatous calcification occurs in psammomatous meningiomas.

Fibroblastic, transitional and mixed tumours are less intense than syncytial and angioblastic tumours. Elster A S et al Radiology 1989.

The requirement of extensive dural excision and arachnoid excision may be indicated in sensitive MRI studies.

In vivo grading of Meningiomas by Positron Emission Tomography:

Although meningiomas are considered benign very often one sees a meningioma that behaves in an aggressive manner. Recurrences are very common and occasionally a metastasis may be generated.

Numerous criteria to predict a meningiomas aggressive behavior and propensity for recurrence have been proposed. Among these, location, neuroradiological findings, and type and extend of surgical resection have been thoroughly evaluated. But unfortunately none have yielded absolutely reliable indices. Often only the passage of time affords the clinician the opportunity to determine the natural history of a particular lesion.

Imagological findings have to some degree predicted the behavior of meningiomas. Positron emission tomography has been recently used to grade meningiomas.PET – FDG (F2 flurodeoxy glucose) can assess the metabolism of lesion. In meningiomas glucose metabolic rate was correlated with histopathologic findings, tumour recurrence and tumour growth. (Domenic J. De Michele and Giovanni Dichiro). Lesions with long CT doubling time possessed low metabolic rates while metabolically active lesions displayed a short doubling time. Another practical utility of metabolic study is the differentiation between radiation necrosis and tumour recurrence. Assessment of the postoperative tumour bed where post surgical changes obscure the interpretation of CT and MRI PET is an investigation of value.

Treatment of Parasagittal Meningiomas: Surgical

The treatment of parasagittal meningiomas is surgical. From a surgical point of view lesions are divided into those of the anterior 1/3rd and those of the posterior 2/3rd.

Technique:

Position: Surgery may be done in different positions – sitting, supine oblique or prone. The basic strategy is to keep the lesion at a higher level that the cardiac atria and to avoid unnecessary torsion to the neck veins. A Doppler monitor must be attached to the chest wall and an intra arterial line should be maintained. This is essential to detect any air embolism and to aspirate off the air in case of such an event.

Craniotomy: Ordinarily for the exposure of a unilateral parasagittal meningioma, a scalp flap is designed with a wide base anteriorly, laterally or posteirorly, with the medial limb just to the other side of the superior sagittal sinus. However if the preoperative angiogram is showing the scalp veins are acting as important draining pathways with SSS occluded then an alternate path of exposure may be chosen.

A free flap is created with burr holes on either side of the sinus. Cutting with a craniotome over the sinus more posterior than the anterior third should be avoided in parasagittal meningiomas. Dura is periodically tacked to the bone after creating drill holes or to the pericranium with 3 0' nonabsorbable suture materials.

The dura is then cut around the tumour except at the medial surface with at least 1cm margin with the tumour.This devascularises the tumour. Lesions which are large or which have an iceberg shape with portion of the tumour covered by the cortex internal decompression must be performed prior to complete dissection from the cortex. This can be done with various means

like bipolar cautery with suction, cutting loop cautery, CUSA, laser vapouriser. This reduces the bulk of the tumour and facilitates separation of the tumour from the brain without undue retraction of the brain.

Although the strategy is to minimize the damage to the cortex some damage can be expected. The surgeon must make every effort to preserve the bridging cortical veins.

In the words of Elsberg

'The large cortical veins on the cerebral convexities perforate the pia arachnoid near the median line just before they enter the longitudinal sinus. The anterior veins opening at right angles, the posterior veins open quiet obliquely from behind forward into the sinus. The veins in the latter category carry away considerable blood from the motor cortex and the interruption of the blood stream in them may be followed by a diminution in the motor power of the limbs on the opposite side of the body. During an operation there is only one way of adequately exposing the vascular structures, and that is by removal of the central core of the growth , so that the remaining shell will fall away from the midline to some extent so as to allow the veins to stand out as separate structures.'

The final portion of the dissection is the removal of the tumour from the attachment to the superior sagittal sinus. If easily separable the surgeon can remove it and can coagulate the attachment so as to destroy any residual tumour cells.

Aggressive surgical approach with reconstruction of the dural sinus involved in meningiomas: (Akira Hakuba) (8)

If the tumour has grown into the lateral aspect of the superior sagittal sinus, but the sinus still retains the patency, the surgeon has 3 choices.1.To leave this portion of the tumour in place, recognizing that as it continues to grow it will lead to the development of collateral circulation, and will eventually totally occlude the SSS after which the sinus along with the tumour can be removed much more safely. 2. To resect the portion of the involved tumour and to close it primarily or the use a graft. Or 3. To excise the involved portion of the sinus.

It is common wisdom that the third option is possible for the anterior third tumours. Excision of the sinus beyond this point is fraught with the high risk of bilateral venous infarct of the motor and sensory areas. The second option requires meticulous suturing technique with a continuous watch for the development of air embolism.

In tumours encroaching into the angle of the sinus the approach is excision the involved portion of the sinus with a simultaneous clamping and suturing of the sinus.

When the tumour involves the lateral wall of the sinus the treatment options can be summarized as Logue has put it.

'Small area of attachment less than 2 cm in diameter can be treated in an elderly patient simply by through coagulation. In a young patient it is safer to excise the whole of the involved wall and to replace with a graft.

This is best done by utilizing the pericranium from the bone flap. The sinus is separated and displaced downwards away from the overlying bone. The affected portion of the sinus wall is overlaid with a graft which is trimmed to overlap the area by 2 -3 mm. The graft is covered with a cottonoid patty 2 mm narrower than the graft, so as to expose the graft edge for suturing. The anterior end of the graft is sutured on to the sinus wall and the graft and cottonoid are turned back on themselves so as to expose the tumour, and then a incision is made 2mm from the latter's anterior edge.

A suture is inserted through the sinus wall on either side, and the graft propelled forward by the finger or forceps to occlude the opening. A further 2 mm incision is made above and below the neoplasm and the graft is again pushed forward and sutured at each edge. This is continued step by step until the area is completely excised.'

Repair of the partially involved posterior third of the superior sagittal sinus (Type III) using an external shunt. : A silicone rubber shunt tube 18cm in length 3.5mm in diameter and 5mm outer diameter with a balloon on either side was inserted proximal and distal to the posteirorly set tumour and the circulation is maintained until repair of the excised portion of the tumour is done with help of saphenous vein graft. After the grafting is complete the openings made for the silicon rubber tube is also closed.

Repair of a significantly stenotic sinus (Type V or VI Bonnal and Brotchi): In cases with the superior sagittal sinus grossly invaded by a tumour, total excision of the intrasinus portion of the tumour and repair of the sinus and its bridging veins with autogenous external jugular vein graft can be done. The involved portion of the sinus is trapped and excised. Intravenous barbiturates to obtain an isoelectric EEG are started for cerebral protection. Jugular vein graft is sutured in continuity with the SSS or posteirorly to either Transverse sinus in case the trocular herophili is involved. Interrupted sutures with 6'0 prolene and partially continuous sutures of 8'0 prolene are used. Dural graft is sutured to the lateral margin of the vein and dura closed water tight. Individual cortical veins may be anastamosed on an end to side manner to the graft if found to be essential.

Reconstruction of the completely blocked sinus with collateral circulation around it.: Excision of the obstructed segment of the sinus would be feasible without causing significant morbidity or mortality if the involvement of the sinus is either anterior to the entries of the main rolandic vein or veins and also occasionally posterior to entry of the vein of Trolard. Angiogram is not entirely reliable for the demonstration of a completely occluded sinus; it must be concluded at surgery by close inspection. When collateral circulation exists it mainly runs longitudinally along the falx and sometimes downwards to the inferior sagittal sinus. It also courses through the sagittal bridging veins, in the front and behind the tumour. The craniotomy hence has to be wide enough, crossing the midline for at least 3 cm and anteriorly and posteirorly to view the possible collateral circulation.

All possible cortical veins, possibly acting as collateral circulation has to be carefully preserved during excision of the tumour, with particular care for the bilateral rolandic veins which may pass through the tumour. A portion of the external jugular vein is harvested. Longitudinal division and suturing together of either halves gives a vein with double girth. Any venous branch is of great importance and is an important pathway for anastamosis particularly for end to end anastamosis of the rolandic branches. End to end anastamosis is then done.

It is generally agreed that the patent superior sagittal sinus can be safely ligated or resected en bloc only in the anterior third of the sinus. In the management of meningiomas involving the posterior sinus cerebral odema and/ or infarction causing death or severe deficits almost always occurs after excision of a still functioning sinus or occlusion of its tributary veins. So total excision of the intrasinus portion of a meningioma with a patent sinus posterior to the coronal suture must always involve reconstruction of the sinus and bridging veins.

Avoiding excessive blood loss and the possibility of air embolism from the cut or opened SSS of the posterior aspect is affected with the help of digital pressure at both ends of the sinus. (Brotchi and Logue). This method is not uniformly successful in the posterior third of the sinus (Akuba) since there will be unacceptable bleeding and brain swelling and use of venous shunts during repair has been advised. Major bleeding is easily controlled by inflating the balloons on each end of the sinus.

When dealing with lesions in the posterior sinus totally occluded, with dominant collateral circulation already present, the involved sinus may be trapped and cerebral protection may be offered with barbiturates and careful positioning of the patient with the head elevated by 30 degrees well above the atrium. Under no circumstance any possible collateral vein must be sacrificed during excision of the lesion. Reconstruction of the sinus is still desirable as the excision might alter the collateral flow in an unpredictable manner.

The ideal graft (Hakuba) is the external jugular vein. It would have side arms simulating sagittal bridging veins. The vein is pliable, flexible and easily sutured in the same contour and size as the sinus. Because it does not have valves there is less risk of subsequent thrombosis in a slow flow system than with the saphenous vein.

A more conservative surgical approach: (Robert G Ojemann)

The resection or reconstruction of the sinus is undertaken with considerable risk, particularly when the sinus is still open and tumour is invading it. The morbidity and mortality of the procedure may not be acceptable to many. A thin layer of tumour maybe left on the sinus to avoid damaging the sinus.

Ojemann "I have not seen an indication to undertake the risks of resecting the middle or posterior third of the sagittal sinus when it is open. When a plaque of tumour is left the patients are carefully followed with MRI. Several have shown no re-growth. If re-growth occurs with significant mass effect, another radical subtotal removal can be done. However the sinus may gradually be occluded by the tumour and when this occurs the patient can be cured with a subsequent operation. The place of radio surgery when re-growth is seen on MRI remains to be determined. The one circumstance in which resection and graft of an open sagittal sinus might be indicated is when the patient has a malignant meningioma."

Alternate management options for meningiomas:

The mainstay of treatment of meningiomas is surgery. Total excision can be considered curative as meningiomas are generally believed to be benign. But unfortunately total removal is not always possible, and also meningiomas are sometimes malignant by histology. If an operated patient is at a risk of recurrence the surgeon may follow the patient with a preparedness of re-operating or may consider postoperative radiation to diminish the chance of recurrence.

External beam radiotherapy:

The currently advised dose of radiation is 4500 to 6000 cGy with an average dose of 5000 to 5500 cGy have been used. The daily dose fractions range from 180cGy to 200 cGy with the total dose delivered over a 5 – 6 week period. As meningiomas are slow to recur, a time gap of 1-3 months should be allowed for proper dural and wound healing.

When postoperative radiation is given following first surgery, a recurrence rate of 22% and 39% 5 and 10 year respectively (Bloom) appears to be better than 30% to 75% recurrence rate after subtotal removal.

The indication for radiation for recurrent meningiomas appears to be more clear cut. In a study by Taylor et al in 1988 on 30 patients, there was a recurrence rate of only 10% versus 70% reoperated with out radiation.

In patients with inoperable meningiomas radiation therapy was found to arrest the progression of 36% of 12 patients at 4 years. 44% of patients treated showed subjective improvement without definite imaging evidence of improvement.

Although the true efficacy of radiation therapy has not been fully evaluated in the case of malignant meningiomas, postoperative radiation therapy is to be considered in all cases of malignant meningiomas and is preferable in case of atypical meningiomas.

Complications of external beam irradiation include besides acute reactions like nausea and vomiting local injury and also neuronal injury with the optic nerves the retina and the brain stem being most vulnerable. If possible at least 3 months period must be allowed between surgery and radiotherapy in the case of meningiomas.

Gamma Knife radio-surgery in the management of parasagittal meningiomas:(Douglas konziolka)-

The use of gamma knife surgery as adjuvant therapy in parasagittal meningiomas is extensively practiced. A small portion of tumour left on the sinus or a small portion within a sinus may be targeted and results have been encouraging. There are no case control studies as yet still the residual tumours were found arrested after this form of treatment.

Hormonal manipulation in the case of Meningiomas:

The higher incidence of meningiomas in females point to the possible hormonal sensitivity of meningiomas.Donel in 1979 reported that meningiomas contained oestrogen receptor binding protein and suggested that incompletely resected meningioma patients may benefit from anti-estrogen therapy. In 1981 Poison et al reported progesterone receptors in meningiomas. Later receptors for epidermal growth factor (EGF) Fibroblast growth factor (FGF) and platelet derived growth factor (PGDF) were found in meningioma tissue. Cell culture studies did not offer any conclusive evidence for the effect of hormonal manipulation. A few clinical studies have also been undertaken In 1985 Markwalder et al reported 6 patients with meningiomas who were treated with tamoxifen for 8 to 12 months. No significant effect on the growth rate of meningiomas was reported.

Meningioma Recurrence:

The exact meaning of recurrence of meningiomas is itself not clear. When a portion of the tumour is left behind the tumour naturally regrows and presents itself either radiologically or clinically. This should be in real terms called re-growth rather than recurrence. But the extend of resection of the meningioma itself is subjective. Minimal tumour left behind in the confines of the superior sagittal sinus, or the microscopic amount of tumour left after shaving a lesion off the sinus wall are all in a true technical sense residual tumour. For purpose of evaluation one may take clinical recurrence or more correctly one should include only those lesions whose excision was macroscopically total or subtotal.

One has to differentiate between clinical and radiological recurrence of meningiomas. Chan and Thompson et al reported that the average detection of tumour recurrence was 5.7 years before the use CT and 2.9 years after the use of CT scanning.

Age and recurrence: The influence of age on tumour recurrence is not clear. The risk of recurrence is expected to be higher among younger patient due to the fact that they have a longer post operative course. In the Phillippon series the age of surgery for patients who had recurred was 42 while that of non recurred patients was 53 years. When patients less than 40 years were considered the risk of recurrence was 42% versus 15% in the whole group. Besides the delay in recurrence was also shorter - 48 months versus 64 months. In the same series relapses occur with the same frequency in both sexes. Between the age of 30 and 40 years 38% females experienced recurrence versus 28% of males.

Recurrence and tumour location:

Table: Recurrence in difference series according to the site of the lesion.

	Simpson			Jaaskelanen			Philippon			Baird		
Site of lesion	*Total*	*Rec*	*%*	*Total*	*Rec*	*%*	*Total*	*Rec*	*%*	*Total*	*Rec*	*%*
Convexity	64	3	4.7	65	9	5.45	154	8	5.2	115	13	11.3
Parasagittal	90	14	15.5	136	11	8	101	8	7.9	46	11	23.9
Falx				63	3	4.8	44	4	9.1	22	4	18.1
Olfactory				56	12	21	24	3	12.5			
Sphenoid	70	4	23.5	79	13	16.4	51	8	15.6	27	6	22.2
Suprasellar				64	2	3.1	22	2	9	31	2	6.5
Post fossa	16	4	25	18	2	11	44	3	6.8	5	0	
Ventricular.				7	0		8	0		6	1	

The difference may be due to the different degrees of completeness of excision.

Recurrence and quality of excision: As early as 1957 Simpson insisted on the relation between the quality of surgical excision and the rate of recurrence. In Simpson's analysis the recurrence was as follows. GrI – 9%, GrII -19%, Grade III – 29%, Grade IV 40%. This series is before the use of CT scanning for the diagnosis of recurrence. Matheisen et al had identified 16% symptomatic recurrence for Gr I excision and 24% for Grade II resection. This is compared with the recurrence rate of 93% and 80% at 10 years and 5 years for subtotal excision.

Recurrence and histology: In various studies among the different histological subgroups, meningothelial and transitional tumours represent approximately 75% of lesions. The rate of recurrence of these groups did not differ significantly from other groups. Opinion regarding fibroblastic meningioma is different with a lower rate of recurrence. Psammomatous meningiomas with a high density of calcification recur rarely, there was only

one recurrence among 19 cases in Kujas series. None of the atypical and anaplastic meningiomas in Jaaskelainen series had calcification while 9 out of 16 lesions of benign nature had calcification.

In the Baird and Gallagher series one out of three angioblastic meningiomas recurred. Atypical meningiomas have recurred in about 75% of cases in Jaakelainen series.

Cellular criteria and recurrence:

The presence of mitosis was found to be a predictor for recurrence with mitosis found in 8.3% of non recurrent cases against 19.6% in recurrent cases. (Boker et al) Nuclear pleomorphism was associated with tumour recurrence in Jaaskelainen et al series.

The relative importance of necrosis has been diversely appreciated. It has no significance in Jellinger and Slowik series. Jaaskelainen et al, Boker et al and Compton and Gautier – Smith has given significance for necrosis. In Boker series necrotic areas were found in 6.1 % of nonrecurring tumours versus 26% of recurring tumours.

Jaaskelainen et al had considered as a whole 6 histological parameters: loss of architecture, increased cellularity, nuclear polymorphism, mitotic figures, focal necrosis and brain infiltration. These features have been found to have significant association with tumour recurrence.

Some other features have also been proposed as predictive of recurrence. De la Monte et described sheet like arrangement of cells in a monolayer like pattern without formation of whorls, syncytial or small aggregations. Sheeting was observed in recurrent meningiomas (76%) versus 11% in nonrecurrent ones.

Radiological predictors of recurrence: Vassilouthis and Ambrose compared CT scans of 16 tumours having aggressive growth and 86 meningiomas which were benign. The main features of the first group were an absence of calcification, areas of hypodensity within the tumour and moderate enhancement. New et al also had similar findings and also emphasized the importance necrosis within the tumour. Irregular margins was a marker of

atypical meningiomas in Jaaskelainen with mushrooming of the tumour.The presence of odema corresponded well with the presence of histological features such as mitotic figures and hypercellularity. This does not exactly correlate with the tumour subtype.

An important observation is that many tumours with normal features do recur.

Results and Analysis

A total of 144 cases satisfied all the criteria to be included in the study. Of this 111 cases were first time operations while 3 cases were third recurrences.

Adequacy of follow up - was availability of clinical or radiological follow up details 5 years after the surgery.

Age of presentation:

The mean age at presentation of fresh cases was - 43.32 Yrs
The mean age at presentation of males was -43.06 Yrs
The mean age at presentation of females was -43.55 Yrs

Statistics Age		
N	Valid	111
	Missing	0
Mean		43.3153
Median		42.0000

Gender statistics:

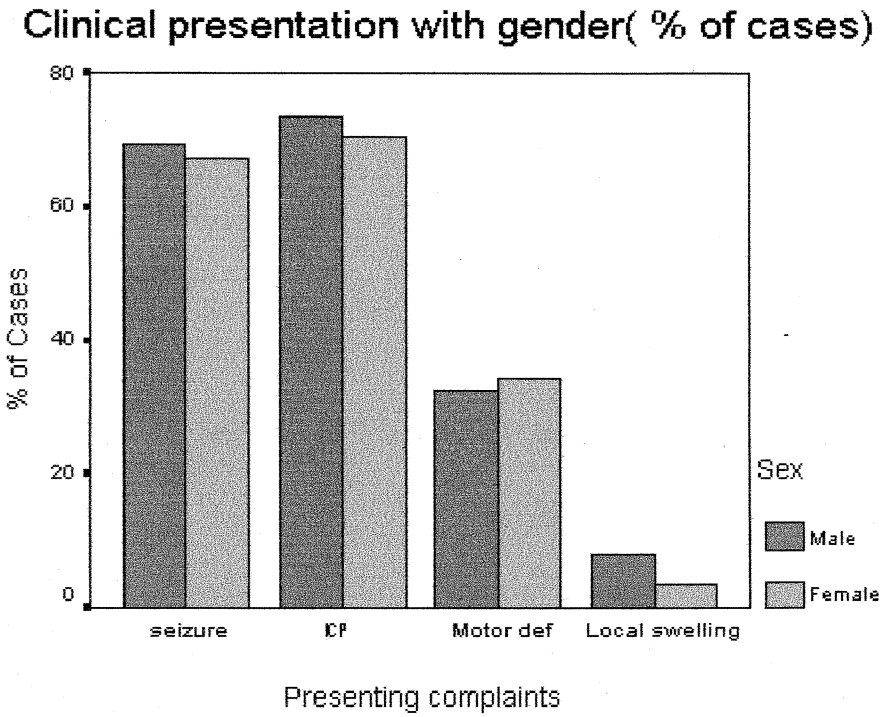
The male female ratio of the whole sample was - 1:1.1
The male and female age at presentation was not different.

	Sex	Mean	N	Std d
Total	Male	43.0566	53	14.3237
	Female	43.5517	58	12.2677
	Total	43.3153	111	13.2301

There was no significant difference on Gender basis on the following:

1. Clinical presentation.
2. Histological features.
3. Imageological findings.
4. Operative findings.
5. Post op Motor status.

Clinical presentation: of the study sample of fresh cases.



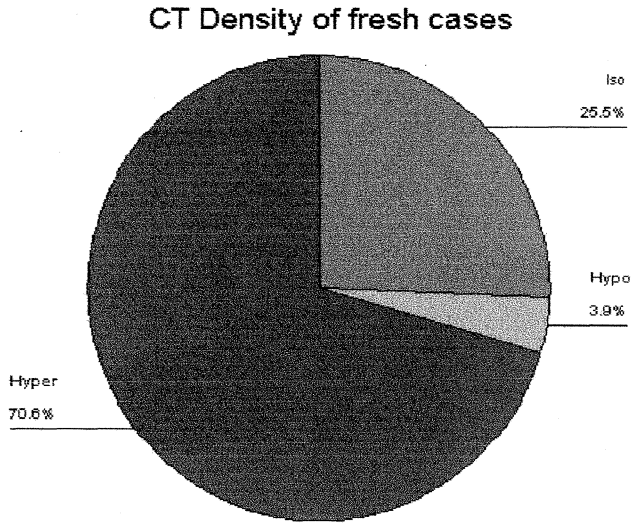
Seizure was the presenting complaint in – 67.6% of patients.
Features of Raised ICP in - 71.2% "
Motor deficits in - 35%
Local swelling in - 5.6%

No significant difference between male and female was present in the sample.

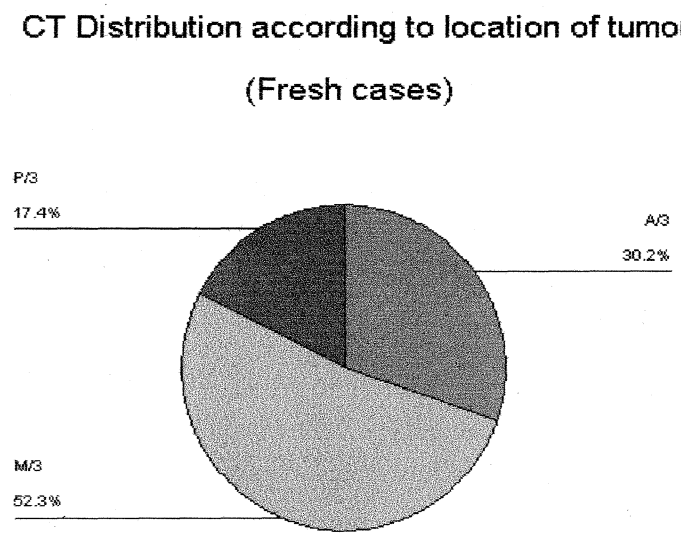
CT Characteristics of the sample:

The different CT characteristics - The density, location, presence of calcification, degree of enhancement, extend of odema were studied.

CT Density: 70.6% - Hyperdense, 25.5% -isodense, and 3.9%- hypodense.



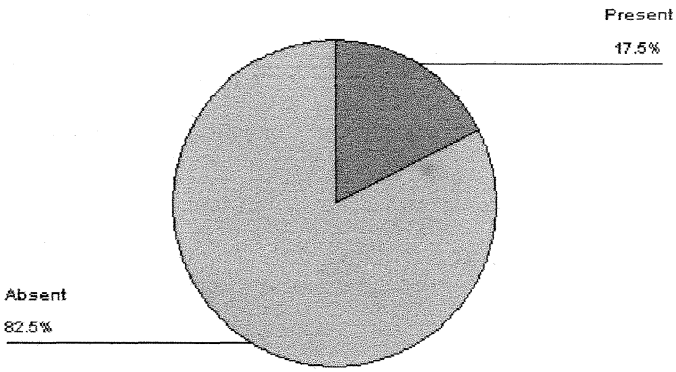
CT location of lesion: Ant3rd -30%, Mid 3rd - 52.3%, Post 1/3rd – 17.5%



Calcification from CT: CT calcification positive in 17.5% of cases.

Presence of Calcification

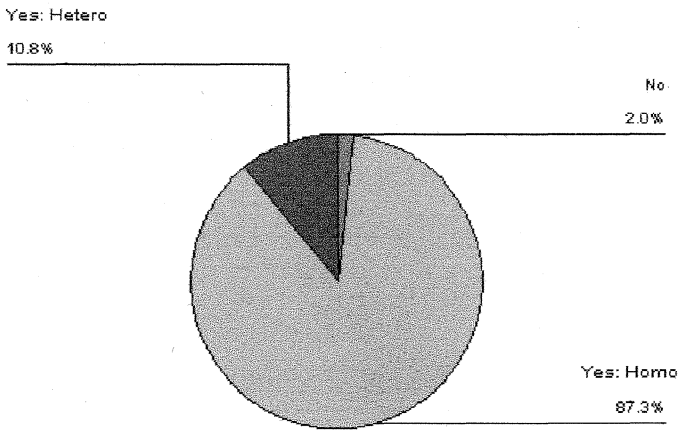
(Fresh cases)



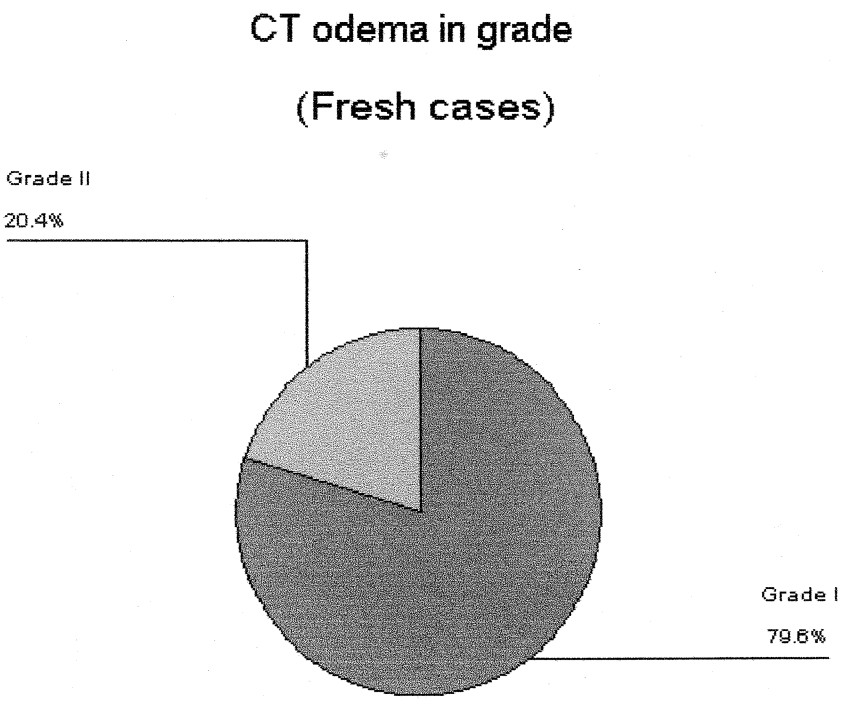
CT enhancement pattern: Homogenous – 87.3, Hetero – 10.8 Nil-2%

CT contrast enhancement

(Fresh cases)

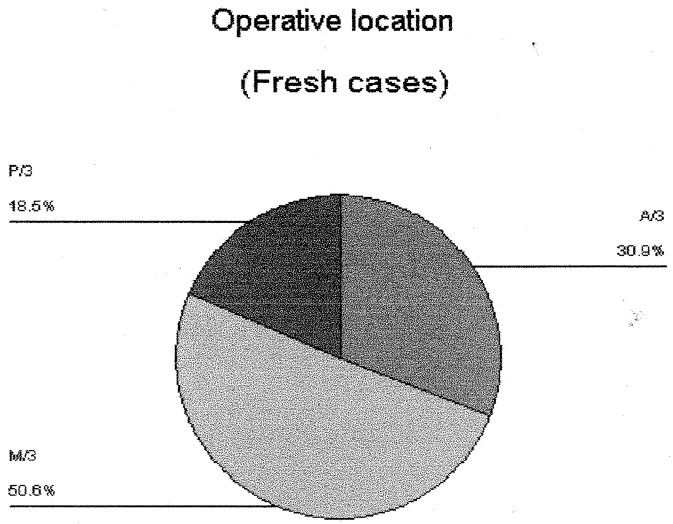


CT odema - Gr I odema (regional) - (79.6%), Gr II (holo- hemispheric) - (20.4%)

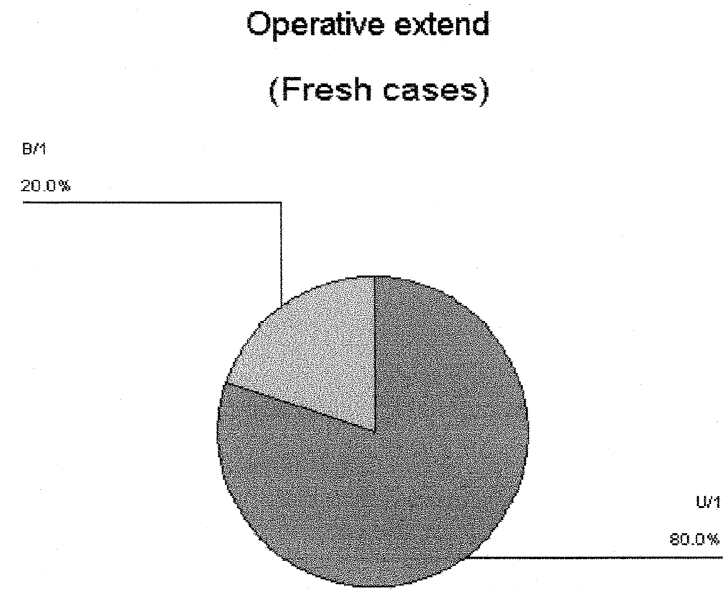


Characteristics of the study sample at surgery:

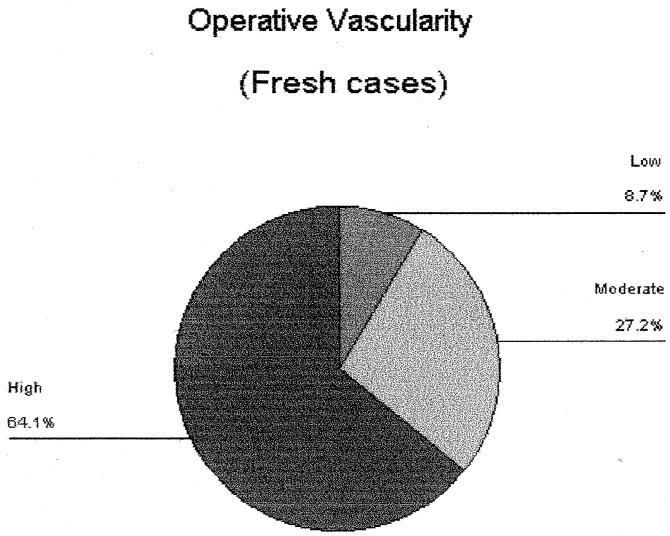
Operative location: Anterior 3rd 30.9%, Middle 3rd- 50.6%, Post 3rd – 18.5%



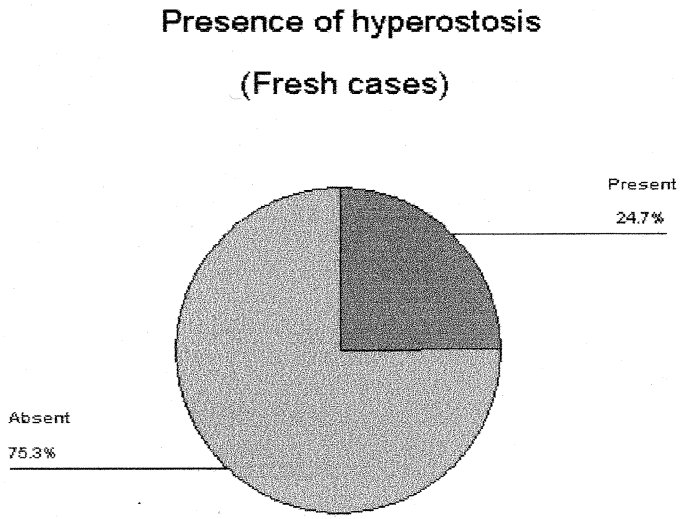
Operative extend: lesions extended bilaterally in 20% of cases.



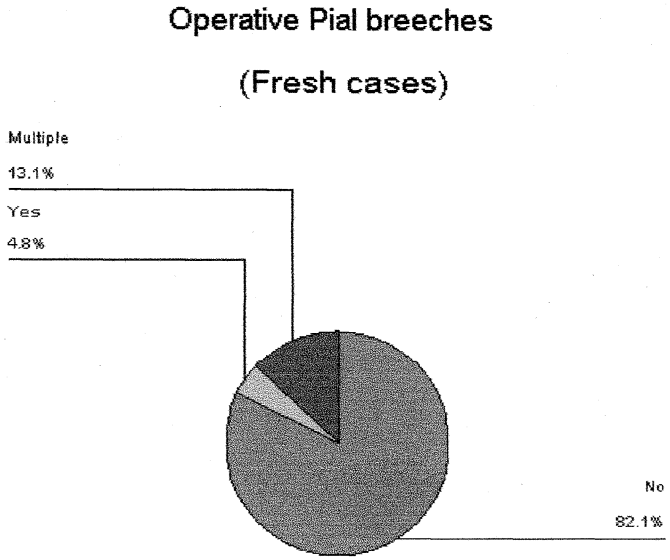
Vascularity of meningioma: Subjective categorization into- High (64.1%), Moderate (27.2%) and low (8.7%)



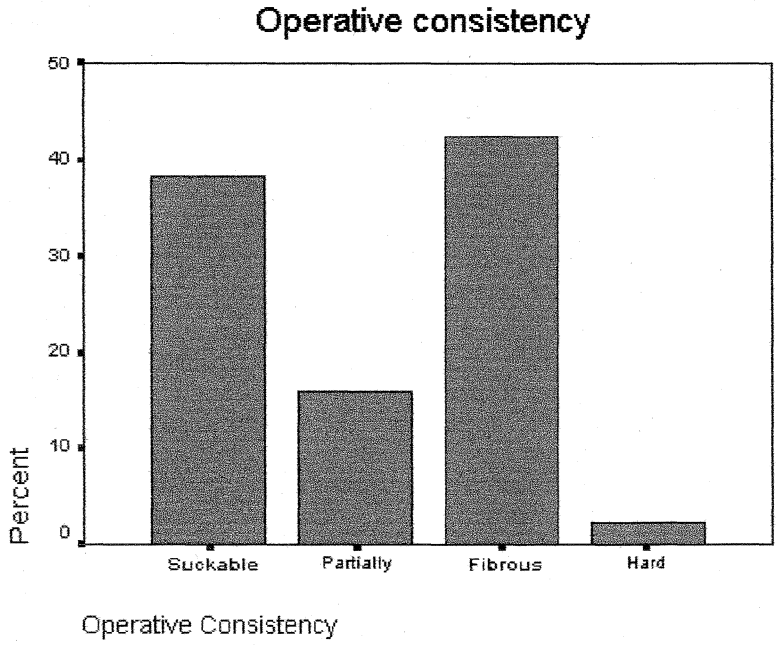
Operative documentation of hyperostosis: Present in about 1/4th of cases



Pial breeches: Present in 18% of cases is a marker of an aggressive lesion.

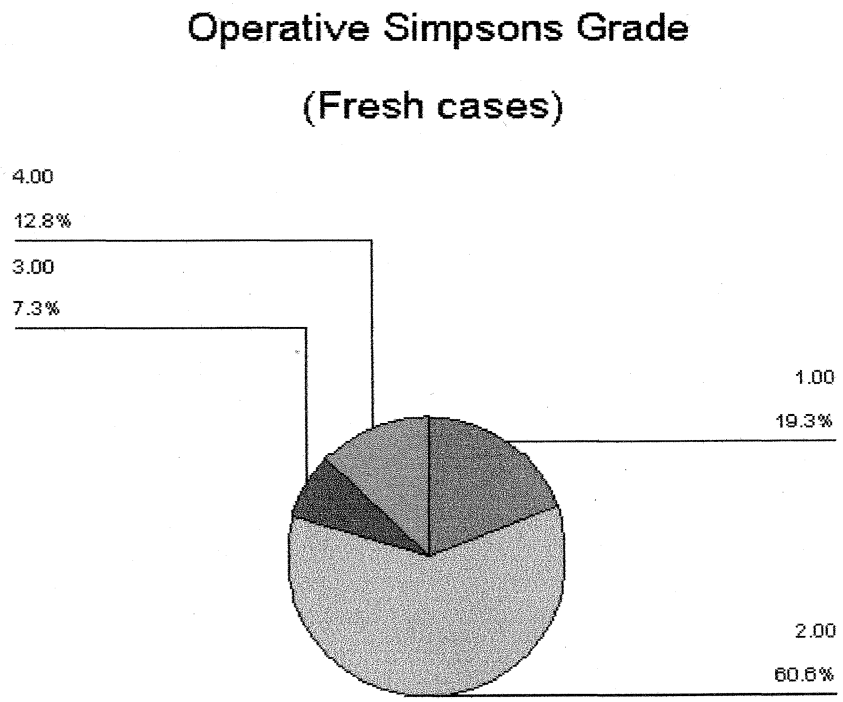


Consistency of the meningioma: was classified into suckable (38%), partially suckable (17%), fibrous (43%) and hard (2%).



Simpson's Grade of excision:

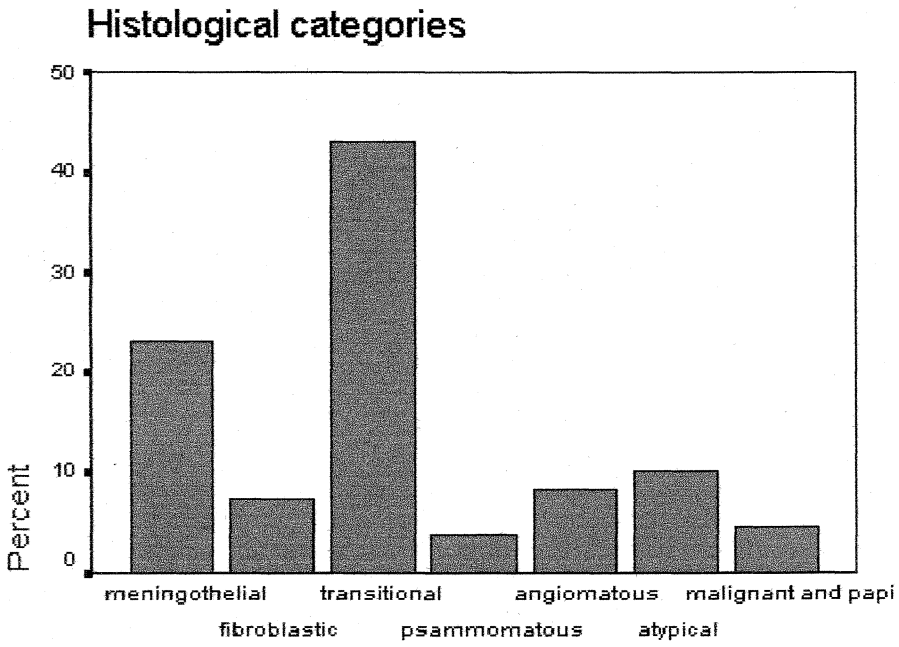
Based on the surgeons estimate of excision, the following was the pattern of extend of excision in the study group. GrI-19.3%, II-2%, III-7.3% and IV - 12.8%.



Histological Features of Meningiomas:

We had grouped Meningiomas based of histology into 7 categories.

1. Meningothelial- 23%.
2. Fibroblastic- 8%
3. Transitional-43.5%
4. Psammomatous-3.7%
5. Angiomatous – 7.4%
6. Atypical – 10.2%
7. Malignant and papillary – 4.2%



Histological categories were correlated with,

- Presenting complaints.
- CT characteristics such as density, contrast enhancement.
- Operative features such as consistency, extend of resection.

Those features which have shown significant correlation have been shown below.

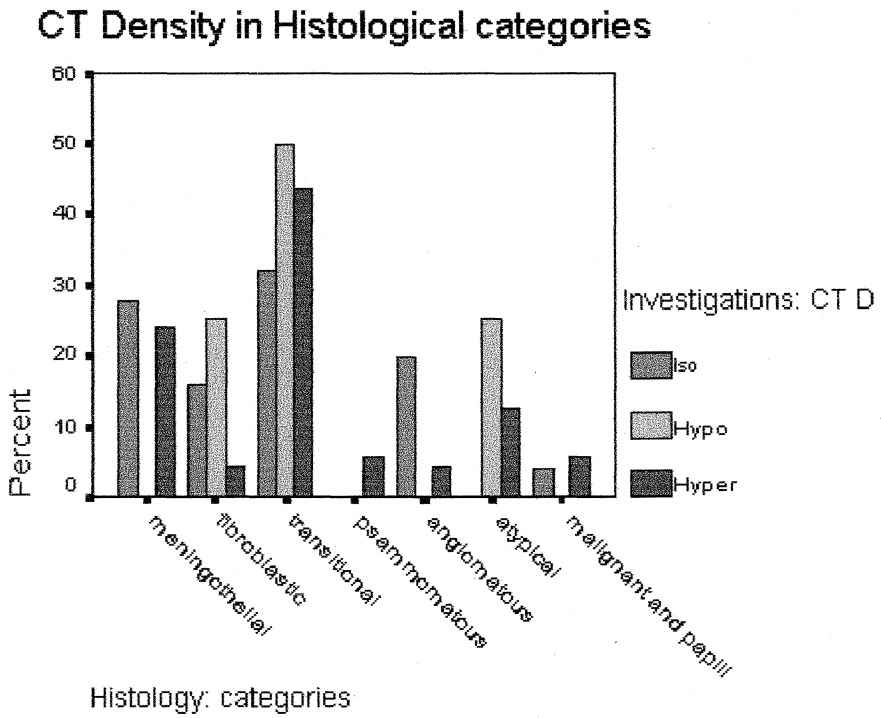
Histology & CT Density: Insignificant correlation seen with atypical lesions.

			Investigations: CT Density			Total
			Iso	Hypo	Hyper	
Histology: categories	meningothelial	Count	7	0	17	24
		Expected Count	6.0	1.0	17.0	24.0
		% within Investigations: CT Density	28.0%	.0%	23.9%	24.0%
	fibroblastic	Count	4	1	3	8
		Expected Count	2.0	.3	5.7	8.0
		% within Investigations: CT Density	16.0%	25.0%	4.2%	8.0%
	transitional	Count	8	2	31	41
		Expected Count	10.3	1.6	29.1	41.0
		% within Investigations: CT Density	32.0%	50.0%	43.7%	41.0%
	psammomatous	Count	0	0	4	4
		Expected Count	1.0	.2	2.8	4.0
		% within Investigations: CT Density	.0%	.0%	5.6%	4.0%
	angiomatous	Count	5	0	3	8
		Expected Count	2.0	.3	5.7	8.0
		% within Investigations: CT Density	20.0%	.0%	4.2%	8.0%
	atypical	Count	0	1	9	10
		Expected Count	2.5	.4	7.1	10.0
		% within Investigations: CT Density	.0%	25.0%	12.7%	10.0%
	malignant and papillary	Count	1	0	4	5
		Expected Count	1.3	.2	3.6	5.0
		% within Investigations: CT Density	4.0%	.0%	5.6%	5.0%

Total	**Count**	25	4	71	100
	Expected Count	25.0	4.0	71.0	100.0
	% within Investigations: CT Density	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	18.466(a)	12	.102
Likelihood Ratio	21.483	12	.044
Linear-by-Linear Association	.790	1	.374



Histology correlated with CT enhancement: CT enhancement had a significant correlation with a more aggressive histological pattern

Histology: categories * Investigations: CT-contrast enhancement

			Investigations: CT-contrast enhancement			Total
			No	Yes: Homo	Yes: Hetero	
Histology: categories	meningothelial	Count	0	19	3	22
		Expected Count	.4	19.1	2.4	22.0
		% within Investigations: CT-contrast enhancement	.0%	21.8%	27.3%	22.0%
	fibroblastic	Count	0	7	1	8
		Expected Count	.2	7.0	.9	8.0
		% within Investigations: CT-contrast enhancement	.0%	8.0%	9.1%	8.0%
	transitional	Count	0	43	1	44
		Expected Count	.9	38.3	4.8	44.0
		% within Investigations: CT-contrast enhancement	.0%	49.4%	9.1%	44.0%
	psammomatous	Count	0	2	1	3
		Expected Count	.1	2.6	.3	3.0
		% within Investigations: CT-contrast enhancement	.0%	2.3%	9.1%	3.0%
	angiomatous	Count	0	8	1	9
		Expected Count	.2	7.8	1.0	9.0
		% within Investigations: CT-contrast enhancement	.0%	9.2%	9.1%	9.0%

	atypical	Count	1	7	1	9
		Expected Count	.2	7.8	1.0	9.0
		% within Investigations: CT-contrast enhancement	50.0%	8.0%	9.1%	9.0%
	malignant and papillary	Count	1	1	3	5
		Expected Count	.1	4.4	.6	5.0
		% within Investigations: CT-contrast enhancement	50.0%	1.1%	27.3%	5.0%
Total		Count	2	87	11	100
		Expected Count	2.0	87.0	11.0	100.0
		% within Investigations: CT-contrast enhancement	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	32.429(a)	12	.001

CT Contrast enhancement when heterogenous or when absent correlated with a more aggressive lesion.

CT Calcification: Showed mild correlation with psammomatous meningioma but did not reach significance.

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	9.419(a)	6	.151
Likelihood Ratio	10.049	6	.123
Linear-by-Linear Association	.141	1	.707
N of Valid Cases	78		
a 10 cells (71.4%) have expected count less than 5. The minimum expected count is .36.			

CT Odema also showed no significant correlation with the Histopathological categories.

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	3.752(a)	6	.710
Likelihood Ratio	4.249	6	.643
Linear-by-Linear Association	.319	1	.572
N of Valid Cases	96		
a 8 cells (57.1%) have expected count less than 5. The minimum expected count is .59.			

Histology and Presenting Complaints:

Histology showed no correlation with the clinical presentation of the patient whether it was seizure, ICP, motor deficit or local swelling.

Recurrence Analysis:

The study group of fresh cases were divided into two based on CT recurrence at 5 years among all patients who had undergone adequate follow-up. (defined as – Clinical or CT follow-up at or after 5 years)

Statistics CT Brain recurrences (N of occurrences)		
N	Valid	105
	Missing	0

CT Brain recurrences (N of occurrences)					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	.00	66	62.9	62.9	62.9
	1.00	39	37.1	37.1	100.0
	Total	105	100.0	100.0	

66 patients formed the control group which never recurred while the 39 patients who had shown recurrence of the lesion formed the study group for this part of the analysis. The different aspects of the two groups were compared were.

1. Age at presentation and gender
2. Clinical presentation – as seizures, as raised intracranial tension, as mass effect or as local swelling.
3. CT findings – of CT density, contrast enhancement, CT odema, CT extend and location, and CT calcification.
4. Operative findings – site, extend, consistency and Simpson's Grade
5. Histological categories- 7 histological categories of meningiomas were seen to any significant number in our study. Their correlation with the chance of recurrence was studied.
6. Histological features- The description of mitotic figures, cellularity, necrosis and psammoma bodies were studied individually.

The entire case processing summery is shown below.

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
CT Brain recurrences (N of occurrences) * Sex	105	100.0%	0	.0%	105	100.0%
CT Brain recurrences (N of occurrences) * Presenting complaints: Seizure	105	100.0%	0	.0%	105	100.0%
CT Brain recurrences (N of occurrences) * Presenting complaints: Raised intracranial pressure	104	99.0%	1	1.0%	105	100.0%
CT Brain recurrences (N of occurrences) * Presenting complaints: Local swelling	102	97.1%	3	2.9%	105	100.0%
CT Brain recurrences (N of occurrences) * Presenting complaints: Motor Status	105	100.0%	0	.0%	105	100.0%
CT Brain recurrences (N of occurrences) * On Examination: Papillodema	105	100.0%	0	.0%	105	100.0%
CT Brain recurrences (N of occurrences) * On Examination: Sensory	105	100.0%	0	.0%	105	100.0%
CT Brain recurrences (N of occurrences) * On Examination: False localising signs	104	99.0%	1	1.0%	105	100.0%
CT Brain recurrences (N of occurrences) * On Examination: Extra cranial Swelling	103	98.1%	2	1.9%	105	100.0%
CT Brain recurrences (N of occurrences) * Investigations: CT Density	97	92.4%	8	7.6%	105	100.0%
CT Brain recurrences (N of occurrences) *	81	77.1%	24	22.9%	105	100.0%

Investigations: CT Localization						
CT Brain recurrences (N of occurrences) * Investigations: CT Extend	101	96.2%	4	3.8%	105	100.0%
CT Brain recurrences (N of occurrences) * Investigations: CT Hyperostosis	74	70.5%	31	29.5%	105	100.0%
CT Brain recurrences (N of occurrences) * Investigations: CT- calcification	76	72.4%	29	27.6%	105	100.0%
CT Brain recurrences (N of occurrences) * Investigations: CT-contrast enhancement	97	92.4%	8	7.6%	105	100.0%
CT Brain recurrences (N of occurrences) * Investigations: CT Odema	93	88.6%	12	11.4%	105	100.0%
CT Brain recurrences (N of occurrences) * Operative Extend	104	99.0%	1	1.0%	105	100.0%
CT Brain recurrences (N of occurrences) * Operative Location	77	73.3%	28	26.7%	105	100.0%
CT Brain recurrences (N of occurrences) * Operative Consistency	88	83.8%	17	16.2%	105	100.0%
CT Brain recurrences (N of occurrences) * Operative Vascularity	98	93.3%	7	6.7%	105	100.0%
CT Brain recurrences (N of occurrences) * Operative pial breeches	80	76.2%	25	23.8%	105	100.0%
CT Brain recurrences (N of occurrences) * Histology: Mitotic Figures	61	58.1%	44	41.9%	105	100.0%
CT Brain recurrences (N of occurrences) * Histology: Cellularity	67	63.8%	38	36.2%	105	100.0%
CT Brain recurrences (N of occurrences) * Histology: Necrosis	58	55.2%	47	44.8%	105	100.0%
CT Brain recurrences (N of occurrences) * Histology: Calcification	51	48.6%	54	51.4%	105	100.0%
CT Brain recurrences (N of occurrences) * Histology: Psammoma bodies	63	60.0%	42	40.0%	105	100.0%
CT Brain recurrences (N of occurrences) * Radiation	102	97.1%	3	2.9%	105	100.0%

Age at presentation: The age at presentation was younger by 3.5 years for patients who had recurrence. This together with a high standard deviation did not reach significance.

Group Statistics					
	CT Brain recurrences (N of occurrences)	N	Mean	Std. Deviation	Std. Error Mean
Age	.00	72	44.5417	12.2681	1.4458
	1.00	39	41.0513	14.7433	2.3608

Sex and recurrence: There was no difference in the chance of recurrence between the sexes or in the age of presentation within the sexes.

Cross tab					
			Sex		Total
			Male	Female	
CT Brain recurrences (N of occurrences)	.00	Count	31	35	66
		Expected Count	32.1	33.9	66.0
		% within Sex	60.8%	64.8%	62.9%
	1.00	Count	20	19	39
		Expected Count	18.9	20.1	39.0
		% within Sex	39.2%	35.2%	37.1%
Total		Count	51	54	105
		Expected Count	51.0	54.0	105.0
		% within Sex	100.0%	100.0%	100.0%

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.182(b)	1	.669		
Continuity Correction(a)	.051	1	.822		

Age and sex –in relation with recurrence of lesion showed no significant difference.

Report Age				
CT Brain recurrences (N of occurrences)	Sex	Mean	N	Std. Deviation
.00	Male	45.7879	33	12.6978
	Female	43.4872	39	11.9558
	Total	44.5417	72	12.2681
1.00	Male	38.5500	20	15.9884
	Female	43.6842	19	13.2206
	Total	41.0513	39	14.7433
Total	Male	43.0566	53	14.3237
	Female	43.5517	58	12.2677
	Total	43.3153	111	13.2301

Clinical presentation and chance of recurrence:

Highly significant correlation was obtained with recurrence and presentation with ICP as against presentation as seizure or motor deficit or local swelling.

Presentation as raised ICP: showed very good correlation with recurrence:

CT rec (adeq of flup 5 Yrs) * Presenting complaints: Raised intracranial pressure

Cross tab

			Presenting complaints: Raised intracranial pressure		Total
			Present	Absent	
CT rec (adeq of flup 5 Yrs)	Adeq fol up & Disease free	Count	18	14	32
		Expected Count	23.9	8.1	32.0
		% within Presenting complaints: Raised intracranial pressure	30.5%	70.0%	40.5%
	CT & cli evid for rec	Count	41	6	47
		Expected Count	35.1	11.9	47.0
		% within Presenting complaints: Raised intracranial pressure	69.5%	30.0%	59.5%
Total		Count	59	20	79
		Expected Count	59.0	20.0	79.0
		% within Presenting complaints: Raised intracranial pressure	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	9.666(b)	1	.002		
Continuity Correction(a)	8.097	1	.004		
Likelihood Ratio	9.634	1	.002		
Fisher's Exact Test				.003	.002
Linear-by-Linear Association	9.544	1	.002		
N of Valid Cases	79				

Presentation as seizure did not show any significant correlation with recurrence outcome.

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.074(b)	1	.786		
Continuity Correction(a)	.003	1	.956		
Likelihood Ratio	.074	1	.786		
Fisher's Exact Test				.832	.481
Linear-by-Linear Association	.073	1	.787		
N of Valid Cases	105				

Presentation as Local swelling also did not show significant correlation.

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.182(b)	1	.140		
Continuity Correction(a)	1.090	1	.296		
Likelihood Ratio	2.110	1	.146		
Fisher's Exact Test				.199	.149
Linear-by-Linear Association	2.161	1	.142		
N of Valid Cases	102				

Presentation as motor deficit did not reach significance in terms of outcome.

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.282(b)	1	.595		
Continuity Correction(a)	.102	1	.749		

CT recurrence with CT imaging features at presentation:

CT wise density, hyperostosis, enhancement, calcification, and odema did not show significant correlation with recurrence outcome.

CT density: into iso dense, hypodense and hyperdense did not reach significance.

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	.601(a)	2	.740
Likelihood Ratio	.598	2	.742
Linear-by-Linear Association	.239	1	.625
N of Valid Cases	97		
a 2 cells (33.3%) have expected count less than 5. The minimum expected count is 1.48.			

CT Hyperostosis : also did not reach statistical significance as shown below:

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	2.231(a)	2	.328
Likelihood Ratio	2.505	2	.286
Linear-by-Linear Association	.051	1	.821
N of Valid Cases	74		
a 2 cells (33.3%) have expected count less than 5. The minimum expected count is .36.			

CT enhancement (homo/hetero/no enhancement) did not correlate significantly with recurrence:

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	.974(a)	2	.614
Likelihood Ratio	.946	2	.623
Linear-by-Linear Association	.397	1	.528
N of Valid Cases	97		
a 3 cells (50.0%) have expected count less than 5. The minimum expected count is .74.			

CT scan wise calcification ; also was not statistically significant:

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	.994(b)	1	.319		
Continuity Correction(a)	.457	1	.499		
Likelihood Ratio	.961	1	.327		
Fisher's Exact Test				.349	.246
Linear-by-Linear Association	.981	1	.322		
N of Valid Cases	76				

CT scan measure of odema (into Grade I localized, GradeII holohemispheric) did not reach statistical significance regarding recurrence.

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.191(b)	1	.662		
Continuity Correction(a)	.025	1	.874		
Likelihood Ratio	.189	1	.664		
Fisher's Exact Test				.782	.431
Linear-by-Linear Association	.189	1	.664		
N of Valid Cases	93				

Operative findings and correlation with recurrence:

The operative location (Anterior , middle or posterior), operative extend (unilateral and bilateral lesions), operative consistency, vascularity and the extend of pial breeching were studied.

The operative location of lesion ie. Anterior third, middle third and posterior third had mild correlation with recurrence but not sufficient to reach significance.

Crosstab

			Operative Location			Total
			A/3	M/3	P/3	
CT Brain recurrences (N of occurrences)	.00	Count	14	26	6	46
		Expected Count	13.7	24.5	7.8	46.0
		% within Operative Location	60.9%	63.4%	46.2%	59.7%
	1.00	Count	9	15	7	31
		Expected Count	9.3	16.5	5.2	31.0
		% within Operative Location	39.1%	36.6%	53.8%	40.3%
Total		Count	23	41	13	77
		Expected Count	23.0	41.0	13.0	77.0
		% within Operative Location	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.240(a)	2	.538
Likelihood Ratio	1.220	2	.543
Linear-by-Linear Association	.485	1	.486
N of Valid Cases	77		

The Extend of lesion: (unilateral or bilateral) alone showed significance with recurrence.

Crosstab					
			Operative Extend		Total
			U/1	B/1	
CT Brain recurrences (N of occurrences)	.00	Count	56	9	65
		Expected Count	51.9	13.1	65.0
		% within Operative Extend	67.5%	42.9%	62.5%
	1.00	Count	27	12	39
		Expected Count	31.1	7.9	39.0
		% within Operative Extend	32.5%	57.1%	37.5%
Total		Count	83	21	104
		Expected Count	83.0	21.0	104.0
		% within Operative Extend	100.0%	100.0%	100.0%

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.332(b)	1	.037		
Continuity Correction(a)	3.345	1	.067		
Likelihood Ratio	4.210	1	.040		
Fisher's Exact Test				.046	.035
Linear-by-Linear Association	4.290	1	.038		
N of Valid Cases	104				
a Computed only for a 2x2 table					
b 0 cells (.0%) have expected count less than 5. The minimum expected count is 7.88.					

The consistency of the lesion at surgery (as suckable, partially suckable, amenable to cusa, fibrous and hard): had mild correlation with recurrence but not sufficient to reach significance.

Cross tab

			Operative Consistency					Total
			Suckable	Partially	CUSA	Fibrous	Hard	
CT Brain recurrences (N of occurrences)	.00	Count	22	8	1	22	0	53
		Expected Count	19.3	9.0	.6	22.9	1.2	53.0
		% within Operative Consistency	68.8%	53.3%	100.0%	57.9%	.0%	60.2%
	1.00	Count	10	7	0	16	2	35
		Expected Count	12.7	6.0	.4	15.1	.8	35.0
		% within Operative Consistency	31.3%	46.7%	.0%	42.1%	100.0%	39.8%
Total		Count	32	15	1	38	2	88
		Expected Count	32.0	15.0	1.0	38.0	2.0	88.0
		% within Operative Consistency	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	5.043(a)	4	.283
Likelihood Ratio	6.081	4	.193
Linear-by-Linear Association	1.417	1	.234
N of Valid Cases	88		

a 4 cells (40.0%) have expected count less than 5. The minimum expected count is .40.

The Operatively assessed vascularity (subjective – mild, moderate, high) showed mild but insignificant correlation with recurrence outcome.

			Operative Vascularity			Total
			Low	Moderate	High	
CT Brain recurrences (N of occurrences)	.00	Count	6	22	33	61
		Expected Count	5.6	17.4	38.0	61.0
		% within Operative Vascularity	66.7%	78.6%	54.1%	62.2%
	1.00	Count	3	6	28	37
		Expected Count	3.4	10.6	23.0	37.0
		% within Operative Vascularity	33.3%	21.4%	45.9%	37.8%
Total		Count	9	28	61	98
		Expected Count	9.0	28.0	61.0	98.0
		% within Operative Vascularity	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.973(a)	2	.083
Likelihood Ratio	5.212	2	.074
Linear-by-Linear Association	2.861	1	.091

Pial Breeching (single and multiple)correlated poorly with recurrence:

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.486(a)	2	.784
Likelihood Ratio	.492	2	.782
Linear-by-Linear Association	.132	1	.716
N of Valid Cases	80		

Operative Simpson's Grade and recurrence were studied in the sample. The correlation did not reach statistical significance. The recurrence after Grade I excision was 30% at 5 years when followed up with CT scans.

CT Brain recurrences (N of occurrences) * Operative Simpson's Grade Crosstabulation

			Operative Simpson's Grade				Total
			1.00	2.00	3.00	4.00	
CT Brain recurrences (N of occurrences)	.00	Count	14	37	4	9	64
		Expected Count	12.4	39.1	4.3	8.1	64.0
		% within Operative Simpson's Grade	70.0%	58.7%	57.1%	69.2%	62.1%
	1.00	Count	6	26	3	4	39
		Expected Count	7.6	23.9	2.7	4.9	39.0
		% within Operative Simpson's Grade	30.0%	41.3%	42.9%	30.8%	37.9%
Total		Count	20	63	7	13	103
		Expected Count	20.0	63.0	7.0	13.0	103.0
		% within Operative Simpson's Grade	100.0%	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.189(a)	3	.756
Likelihood Ratio	1.210	3	.751
Linear-by-Linear Association	.000	1	.986
N of Valid Cases	103		

a 3 cells (37.5%) have expected count less than 5. The minimum expected count is 2.65.

Histological Categories and recurrence:

Histological categories as mentioned by the pathologist as whole did not correlate well with recurrence. There was correlation between atypical meningiomas and recurrence reaching significance.

Crosstab

			Recurred Cases		Total
			Non-recurred	Recurred	
Histology: categories	meningothelial	Count	20	5	25
		Expected Count	17.9	7.1	25.0
		% within Recurred Cases	25.6%	16.1%	22.9%
	fibroblastic	Count	8	0	8
		Expected Count	5.7	2.3	8.0
		% within Recurred Cases	10.3%	.0%	7.3%
	transitional	Count	32	15	47
		Expected Count	33.6	13.4	47.0
		% within Recurred Cases	41.0%	48.4%	43.1%
	psammomatous	Count	3	1	4
		Expected Count	2.9	1.1	4.0
		% within Recurred Cases	3.8%	3.2%	3.7%
	angiomatous	Count	8	1	9
		Expected Count	6.4	2.6	9.0
		% within Recurred Cases	10.3%	3.2%	8.3%
	atypical	Count	3	8	11
		Expected Count	7.9	3.1	11.0
		% within Recurred Cases	3.8%	25.8%	10.1%
	malignant and papillary	Count	4	1	5
		Expected Count	3.6	1.4	5.0
		% within Recurred Cases	5.1%	3.2%	4.6%

Total	**Count**	78	31	109
	Expected Count	78.0	31.0	109.0
	% within Recurred Cases	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	16.461(a)	6	.011
Likelihood Ratio	17.602	6	.007
Linear-by-Linear Association	3.922	1	.048
N of Valid Cases	109		

a 7 cells (50.0%) have expected count less than 5. The minimum expected count is 1.14.

The high significance seen has been contributed by the high number of recurrent atypical meningiomas and the low chance of recurrence of psammomatous meningiomas.

Histopathological features and recurrence:

Presence of Mitotic figures, cellularity, necrosis and psammoma bodies as reported by the pathologist was correlated with recurrence.

Finding of Mitotic figures: showed no significant correlation with recurrence :

			Histology: Mitotic Figures		Total
			Present	Absent	
CT Brain recurrences (N of occurrences)	.00	Count	3	35	38
		Expected Count	4.4	33.6	38.0
		% within Histology: Mitotic Figures	42.9%	64.8%	62.3%
	1.00	Count	4	19	23
		Expected Count	2.6	20.4	23.0
		% within Histology: Mitotic Figures	57.1%	35.2%	37.7%
Total		Count	7	54	61
		Expected Count	7.0	54.0	61.0
		% within Histology: Mitotic Figures	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.272(b)	1	.259		
Continuity Correction(a)	.509	1	.476		
Likelihood Ratio	1.229	1	.268		
Fisher's Exact Test				.409	.235
Linear-by-Linear Association	1.251	1	.263		
N of Valid Cases	61				

Cellularity of the tissue correlated poorly with the likelihood of recurrence.

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.989(b)	1	.320		
Continuity Correction(a)	.474	1	.491		
Likelihood Ratio	.966	1	.326		
Fisher's Exact Test				.368	.243

Necrosis noted by the pathologist significantly correlated with recurrence.

Cross tabulation:

			Histology: Necrosis		Total
			Present	Absent	
CT Brain recurrences (N of occurrences)	.00	Count	2	35	37
		Expected Count	4.5	32.5	37.0
		% within Histology: Necrosis	28.6%	68.6%	63.8%
	1.00	Count	5	16	21
		Expected Count	2.5	18.5	21.0
		% within Histology: Necrosis	71.4%	31.4%	36.2%
Total		Count	7	51	58
		Expected Count	7.0	51.0	58.0
		% within Histology: Necrosis	100.0%	100.0%	100.0%

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.276(b)	1	.039		
Continuity Correction(a)	2.717	1	.099		

Likelihood Ratio	4.109	1	.043		
Fisher's Exact Test				.086	.052

Histological features of calcification correlated poorly with recurrence:

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	.125(b)	1	.723		
Continuity Correction(a)	.000	1	1.000		
Likelihood Ratio	.124	1	.725		
Fisher's Exact Test				.724	.501

Psammoma bodies also did not correlate well with recurrence:

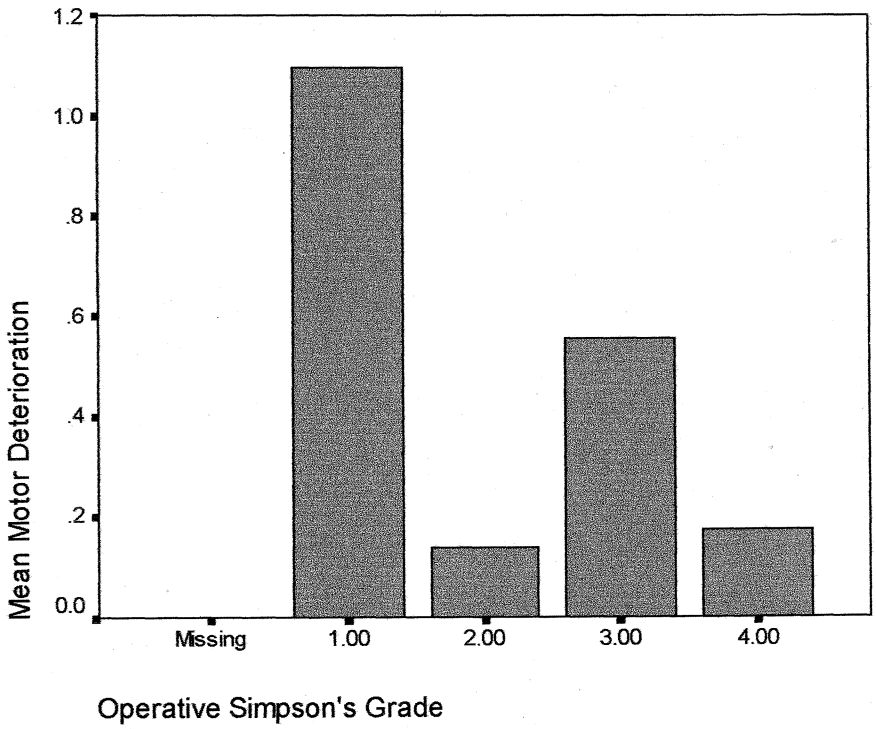
Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	2.741(b)	1	.098		
Continuity Correction(a)	1.949	1	.163		
Likelihood Ratio	2.763	1	.096		
Fisher's Exact Test				.123	.081
Linear-by-Linear Association	2.698	1	.100		
N of Valid Cases	63				

Outcome Study:

Outcome study was done with regards motor status of patients who had undergone excision of lesion. The motor outcome is matched with the Simpson's Grade of excision. On a linear scale fall in motor grade is considered positive motor deterioration while improvement in motor grade after surgery was negative motor deterioration.

Descriptives
MTRDETER

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
1.00	31	1.0968	2.1963	.3945	.2912	1.9024	-2.00	6.00
2.00	72	.1389	1.2368	.1458	-.1518	.4295	-5.00	5.00
3.00	9	.5556	1.1304	.3768	-.3133	1.4244	.00	3.00
4.00	17	.1765	1.2862	.3120	-.4849	.8378	-3.00	2.00
Total	129	.4031	1.5588	.1372	.1315	.6747	-5.00	6.00



DISCUSSION

This study has been conducted to analyze various aspects of the possible aetiology, clinical presentation, examination findings, gender related factors, and radiological features of parasagittal meningiomas .A comparison with available series in the literature is attempted. Factors of possible influence or correlation with, recurrence such as age, gender, clinical presentation, extend of lesion, radiological features, characteristics of the lesion at surgery, extend (grade) of surgery and histopathological characteristics have been studied and compared. The management outcome with regards various parameters has also been studied.

Aetiology: The Study failed to show any clear aetiological predictor for its origin. Other than in cases of phacamatosis most available series have not shown any significant aetiological correlation (Choi et al.Annegers et al) The often suspected association with trauma and radiation (Cushing and Eisenhardt, Modan et al) was not supported by the series. The sample of 144 may still be small to give to yield any definite trends.

The clinical presentation of parasagittal meningiomas is with one or several of the following features namely, seizures, raised ICP, local swelling, motor deficits or behavioral abnormalities.

Seizure; In most series the incidence of seizure had been 33 to 40%. In the study a much higher incidence of seizure as the presenting symptom - 67.6% has been found. There has been no appreciable difference between sexes in the incidence of the symptom. This would merit more detailed study regarding epileptogenesis of parasagittal lesions in our population.

Raised ICP: One third of patients present with raised ICP in most series. Again the incidence of ICP had been higher in our series 71.2%, with no male female difference.

Motor deficit: Approximately 28% of patients in most western series presented with motor deficit. The incidence in our series had been marginally higher with an incidence of 35.1 %.

Local swelling: 8% of patients had presented with localized swelling in western series while in our study group 5.6% had presented with this symptom.

Behavioral abnormality which is a common presentation with anteriorly located tumours (27%) had been a very rare presenting complaint in our study group. This reason could be the different cultural perspective of our population.

Multiple symptoms at presentation and higher incidence of raised ICP and seizures are possibly pointing to more advanced stage of disease at presentation in our population.

Age: The mean age of presentation of patients with parasagittal meningiomas was 43.3 years with a Std Deviation of 15 years. The median age of patients at presentation was 42 years. The influence of a few young age patients associated with phacomatosis, the large standard deviation and the small sample may make the age related study less accurate. The average age of presentation in western series is approximately 10 years more than what is seen in our series. There was no difference in the age at presentation among males and females in our study. Further aspects of age are discussed later.

Gender has been considered an important factor in the incidence of meningiomas. Most series have quoted a higher incidence of the condition in females. The ratio is approximately 1:2 in most series. The Manitoba study shows male female predominance only after the 5^{th} decade. Similarly there is no female predominance among blacks. A study from Chinese Taipei showed a ratio of 7:25. Our study has yielded a ratio of 1:1.1.This along with a younger age of presentation is consistent with the findings of the Manitoba study.

We have further analyzed the relation of clinical presentation CT features, such as odema, density, enhancement, distribution of tumour, operative findings and extend with gender. No significant correlation between sex and any of the above was found in our series.

CT findings in meningiomas including CT Density, CT contrast enhancement, calcification, and CT odema, were analyzed in our study. Findings of the study have corresponded well with other studies available in literature.

CT Density- Lesions were hyperdense in 70.6% of cases, Isodense in 25.5% of cases and hypodense in 3.9% of cases. This corresponds well with standard series (hyper 70- 75%, Iso -25%, Hypo 1-5%) available in literature (1).

CT Contrast enhancement – 87.3% of our lesions enhanced well and uniformly, 10.8% lesions had heterogenous enhancement while 2% had no enhancement. This also corresponds to the value of 90% uniform enhancement and 10-15% heterogenous enhancement seen in literature (Osborn).

CT Calcification: Was seen in 17.5% of patients as against 20 -25% seen in literature.

Operative findings in parasagittal meningiomas were analyzed.

Distribution: 30.9% anterior third, 50.1% middle third and 18% posterior third was the distribution by location. This is approximately same as in other series like Al Mefty ($M/3^{rd}$ - 58%, $A/3^{rd}$- 32% and $P/3^{rd}$ -10%), and Cushing ($A/3^{rd}$ – 25%, $M/3^{rd}$-65% and $P/3^{rd}$ -10%). This rules out any distribution bias in study sample.

Extend of tumour: Approximately 20% of tumours in our group were bilateral tumours. This indicates a high incidence of advanced lesions in the sample.

Other operative features such as consistency of tumour, vascularity and pial breeches were also analysed in the study. Correlation of these factors with recurrence and outcome is dealt with later.

Recurrence of lesion is perhaps the most significant event in the course of treatment and follow up of a patient operated for a parasagittal

meningioma. Predicting the long term outcome of a parasagittal meningioma is of prime importance in deciding the management of an individual case. The predictive importance of various factors such as age, gender, clinical features at presentation, findings at imaging, operative findings, location of tumour, histopathological category and characteristics and extend of resection of lesion were analysed.

Age as predictor for recurrence has been suggested by different studies. Recurrent meningioma patients were found to be 10 year younger (42yr versus 53yrs) in the series of Phillipon et al. In the study recurrent cases were younger by 3.61 years. (40.71 Versus 44.32) .This did not reach statistical significance.

Sex has not been reported as a predictor for recurrence in literature except in certain age intervals. In our study also no significant correlation has been obtained between gender and risk of recurrence.

Clinical features at presentation are not widely used as a predictor for recurrence in literature. In our study we found significant correlation between presentation as raised ICP and recurrence of lesion. (Pearson Chi-Square value – 7.808 df 1 Asymp. Sig (2 sided) .005). Similarly Papillodema – the best clinical sign of raised intracranial pressure also showed significant correlation with risk of recurrence (Pearson Chi – Square value 10.198(b) df 1 Asymp.sig (2 sided) .001).

Presentation as seizure, motor deficit, sensory deficit, localized swelling or with false localizing signs did not have significant correlation with recurrence.

CT findings have been used widely in literature as a predictor of outcome and recurrence. Of this the most important criteria were CT odema (exact quantification in thickness of odema) and mushrooming of the tumour (sign of brain invasion). In our retrospective study exact measures of CT odema and such imagological findings could not be obtained. Based of broader criteria we have studied CT density, CT enhancement,
CT odema- Grade, CT Calcification as possible predictors of outcome. No correlation with recurrence was obtained. A review of films is perhaps essential to give a more accurate picture.

Location of tumour whether anterior middle or posterior has mild correlation with recurrence in literature. This correlation has been indirectly through the extend of resection with the more posterior lesions fairing badly. In our study there was a marginally increased chance for posterior third lesions to recur. No statistical significance with location of tumour could be obtained.

Operative finding of pial breeches did not have any correlation with recurrence. Mild correlation was seen with vascularity of the meningioma with the more vascular ones more likely to recur. Operative consistency had a mild correlation with recurrence with harder lesions tending to recur more often. None of these operative findings could reach statistical significance in the study.

Operative Simpson's grade correlated poorly in our study on recurrence. Recurrence rates for different grades had been considerably higher than that described in well known series (Simpson, Al mefty, Ojemann). Grade I excisions of parasagittal meningiomas have shown recurrence of 30% in our series. Grade I excisions have been done for a disproportionate number (75% - 8 out of 12 cases followed up with CT for 5 years) of bilaterally extending lesions which points to a more aggressive disease process at the onset. Infact it is likely that the surgeon is forced to do the radical excision in these cases, but sticks to doing less radical procedures like Grade II excision for more operatively favourable lesions. This might account for loss of correlation between operative Simpson's Grade and recurrence.

Histological category, of lesion did not prove to have much correlation with recurrence except fibroblastic and psammomatous (Jaaskalainen, Kujas). Similar findings were present in our series also with only psammomatous meningiomas showing decreased chance of recurrence. Atypical and malignant meningiomas have increase chance to recur in all the available series.

Histological characteristics were found to have greater correlation with recurrence in most series. Mitotic figures (Boker et al), Cellularity

(Jaaskalainen -9) Necrosis (Gautier smith, Boker et al) Psammoma bodies were found to have correlation with recurrence. In our series only necrosis reached any degree of statistical significance.

Management outcome study was performed based on the motor grade at discharge (both deterioration and improvement considered on a linear scale). Motor deterioration was maximum when aggressive surgical measures (Gr I) was done – 1.1. It was least for GrII excision- 0.139, Grade III excision had .55 motor deterioration mainly due to lack of improvement of motor function.

A retrospective study spanning a long time interval has a lot of limitations. The need to rely on observations by multiple observers, periodic changes in patterns of recording and record maintenance, difficulty in recovering first hand imaging data, gradually changing follow-up protocols, changes in perspective due to technical advancements etc, are but some of them. A single observer had analysed all the available records to reduce inter observer bias, and every attempt to maintain maximal objectivity of observations has been made.

This study has gone only to broad clinical considerations. A more detailed study might bring out more correlations between variables. Accuracy and details of observations can be increased by direct outpatient interaction with the follow up patients. This would be the next stage of the study. The present study might dictate the required guidelines for such a venture. There is also vast scope of multiple regression studies with the available data to find new and hither to unsuspected factors influencing the clinical scenario in the management of parasagittal meningiomas.

Summary and Conclusions

This retrospective study on the entire clinical scenario of parasagittal meningiomas operated in SCTIMST Trivandrum, spanning almost the entire history of the Institute, was planned to cover all aspects of the disease from a practically possible point of view.

From an initial total sample size of 240 operated parasagittal meningiomas, nearly 100 cases were eliminated to obtain an accurate study group with all the initially planned variables.

In the basic patient profile, the study varied from known studies in literature in two aspects. 1. The mean and median age (43 yrs) was almost a decade younger than of most series. 2. The widely emphasized gender predominance of females was not seen in the study.

The clinical presentation of patients had a higher than usual incidence of seizures and features of raised ICP. Besides there was a greater incidence of multiple symptoms in each patient, pointing towards a more advanced stage of disease than in the west.

The radiological features of the disease corresponded accurately with all available series in literature. The operative findings and histopathological profile followed the same course.

Histopathological categories have shown a mildly significant correlation with the clinical presentation and outcome. The only notable finding was a marginally higher incidence of recurrence, among patients with atypical meningiomas. Histological features such as mitotic figures and cellularity did not show significance in predicting clinical behavior or recurrence. Necrosis was the only feature that reached statistical significance in terms of chance of recurrence in out study.

Analysis of the correlations of the recurrence yielded many interesting findings. Among the different forms of clinical presentations like seizure, features of raised ICP and its manifestation Papillodema, showed a very high correlation with recurrence.

The incidence of recurrence had been higher than that predicted by the Simpson's grade of excision. This disparity is possibly due to a disproportionate number of advanced cases undergoing Grade I excision, and grade II excisions were seemingly reserved for well preserved patients, with more surgically favourable lesions.

The outcome of patients with regards motor status has been significantly poorer for patients who underwent Grade I and Grade III resection when compared with patients who underwent Grade II resection. This read along with the lack of correlation between grade and recurrence outcome, suggests an optimal extend for surgical intervention.

The results of radical surgery with sinus reconstruction techniques have been extensively compared with more conservative approaches in the literature. Despite advancements in microsurgical and immunological techniques the controversy in the management of meningiomas attached to the SSS is far from being resolved. Neurosurgeons in developing countries are justified in having their own perception of this complex clinical problem, considering the possible wide variation in the availability and affordability of modern investigations, drugs, and strict follow-up protocols. It is hoped that our study might help surgeons to develop their own perspective, of this common yet complex pathology.

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