

**IMAGING OF INTRACRANIAL DURAL ARTERIOVENOUS
FISTULA – COMPARISON OF ADVANCED MAGNETIC
RESONANCE ANGIOGRAPHIC SEQUENCES WITH DIGITAL
SUBTRACTION ANGIOGRAPHY**



THESIS

SUBMITTED IN PARTIAL FULFILLMENT FOR DEGREE OF

**DM NEUROIMAGING AND INTERVENTIONAL
NEURORADIOLOGY**

(2019-2021)

OF

**THE SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL
SCIENCES AND TECHNOLOGY, TRIVANDRUM, INDIA**

DR. ARUN PRASAD B


**DEPARTMENT OF IMAGING SCIENCES & INTERVENTIONAL RADIOLOGY
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND
TECHNOLOGY, TRIVANDRUM, INDIA**


**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL
SCIENCES AND TECHNOLOGY, TRIVANDRUM, INDIA**





CERTIFICATE


This is to certify that the work incorporated in this thesis titled "Imaging of Intracranial Dural Arteriovenous Fistula – Comparison of Advanced Magnetic Resonance Angiographic Sequences With Digital Subtraction Angiography." for the degree of DM NEUROIMAGING AND INTERVENTIONAL NEURORADIOLOGY has been carried out by Dr. Arun Prasad B under our supervision and guidance. The work done in connection with this thesis has been carried out by the candidate himself and is genuine.


Dr. Santhosh Kumar K
Additional Professor
Principal Guide


Dr. Bejoy Thomas
Professor
Co-Guide


Dr. Kesavadas C
Professor
Co-Guide


Dr. Jayadevan ER
Additional Professor
Co- Guide


Dr Bejoy Thomas
Professor & HOD
Department of Imaging Sciences and Interventional Radiology,
SCTIMST, Thiruvananthapuram.

DECLARATION

I hereby declare that this thesis titled “Imaging of Intracranial Dural Arteriovenous Fistula – Comparison of Advanced Magnetic Resonance Angiographic Sequences With Digital Subtraction Angiography.” has been prepared by me under the supervision and guidance of Dr.Santhosh Kumar K (Additional Professor) , Dr Jayadevan E R (Additional Professor), Dr Bejoy Thomas (Professor), Dr.Kesavadas C (Professor), Department of Imaging Sciences and Interventional Radiology, Sree Chitra Institute for Medical Sciences and Technology, Trivandrum.

Date: 29/07/2021

Place: Thiruvananthapuram



(Dr Arun Prasad B)

ACKNOWLEDGEMENT

- ✚ *I hereby express my sincere gratitude to all my teachers & guides especially Dr.Santhosh Kumar K., Dr Jayadevan E R, Dr Bejoy Thomas and Dr. Kesavadas C, of Department of Imaging Sciences and Interventional Radiology, for their constant unwavering support, insightful criticism, expert supervision and immense patience throughout this study.*

- ✚ *I would specially like to acknowledge my gratitude to my past and present colleagues and the technologists of the Department of IS and IR and the advanced radiology technology trainees of the department esp Col(Dr) Manoj Gopinath, Dr Sabarish, Dr Santhakumar, Wg Cdr(Dr) Gurpreet, Dr Sathish, Surg Cdr(Dr), Somnath Pan, Dr Vinayagamani, Mr.Alex, Mr Mahesh, Mrs sheeba, Mrs Meena, Ms Merlin, Mr Vikas, Mr Joy, Mr Johnson, Mr, Ashiq, Mr Fawaz, Ms Suneera, Ms Ajimi, Ms Sumeeja, Mr Aromal, Mr Abhiraj, Ms Shivani and Mr Jithin for their valuable assistance at all times during the course of this study.*

- ✚ *I would also like to extend my heartfelt gratitude to my wife Er S Bhavadharini and my son A Mithurvan and my parents and other family members for being the pillar of support in all my efforts. I could not have achieved without their prayers, love and support.*

- ✚ *I also express my sincere gratitude to all my patients & their relatives who had extended their full cooperation without any qualms all through the study.*

CONTENTS

Sl No	Section	Page No.
1	Introduction	1
2	Aims & Objectives	5
3	Review of Literature	7
4	Materials and Methods	35
5	Results	44
6	Discussion	59
7	Conclusion	70
8	References	72
9	Representative Cases	87
10	Annexures	108
11	Abbreviations	120
11	Plagiarism Report	121



INTRODUCTION



INTRODUCTION

Intracranial dural arterio venous fistula (dAVF) is a rare intracranial vascular malformation constituted by pathological connections between dural arteries and dural venous sinuses, meningeal veins or cortical venous channels (1). These vascular malformations comprise 10 to 15 % of all the intracranial vascular malformations (2). Various etiologies have been proposed in the etiopathogenesis of dAVF. However, there is common association of intracranial venous sinus occlusion or stenosis (3) & (4).

dAVF has got varied clinical presentations. The presentation may be benign or aggressive. Benign presentation of dAVF is usually with nonspecific symptoms like headache, proptosis, tinnitus, cognitive disturbances and visual symptoms like decrease in visual acuity whereas aggressive presentation usually is associated with intracerebral hemorrhage, focal neurological deficits and seizures. The site of the fistula and pattern of venous drainage are the decisive factors in determining the clinical symptoms (5). As the clinical features are very varied and non-specific, diagnosis by neuroimaging has become indispensable.

The pathogenetic course of the disease process shows recruitment of newer vessels as part of the disease progression and this aspect was visualized in patients who have been partially embolized. Hence post procedural follow up of these patients is imperative and cannot be overemphasised. Digital subtraction angiography(DSA) has always been the gold standard for diagnosis of this entity as well as for follow up of these patients post treatment(6). However, it has got its own disadvantages in the form of radiation exposure, contrast related adverse effects and minimally invasive nature of the procedure with its inherent rare complications.

Numerous studies have been published on cranial dural arteriovenous fistulas (dAVF) and brain arteriovenous malformations (BAVM) which had compared the non-invasive MR angiographic

sequences with DSA for diagnosis and evaluation of these vascular malformations. Newer sequences like SWAN (Susceptibility weighted angiography) and ASL (Arterial spin labeling) have been studied in the diagnosis and analysis of cranial vascular lesions with arteriovenous shunting which includes dAVF. There is variable diagnostic accuracy shown by these modalities with only a few prospective studies available for validation of these modalities (7).

Comparison of three-dimensional time of flight (3D-TOF) angiography with angiography on evaluation of dAVF shows moderate intermodality agreement (8). One of the studies involving SWI has shown that this sequence has detected the fistulous point and the reflux into cortical veins in cases of dAVF with reasonable accuracy (9). All the non-invasive imaging sequences which have been used have their own strengths and limitations. A novel MR angiographic sequence has been utilised for the angiographic characterisation of Brain arteriovenous malformations and also in the follow up of intracranial aneurysms with reliable accuracy in agreement with DSA. It is an arterial spin labelling based magnetic resonance angiographic sequence, Silent Magnetic resonance angiography (Silent- MRA), available in 3.0 Tesla Magnetic Resonance scanners. It provides good background suppression along with non-contrast magnetic resonance angiographic images on intracranial arteries (10) (11) (12). At present, there are no studies which had analysed the reliability and accuracy of silent MRA in diagnosis and evaluation of dAVF in comparison with other noninvasive MRA sequences and angiography. This study purports to systematically evaluate utility of this novel magnetic resonance angiography technique in diagnosis, characterization, classification of dAVF and also to compare the accuracy of this technique with other sequences like ASL, SWAN, 3D TOF along with gold standard DSA in diagnosis of dAVF.



AIMS & OBJECTIVES



AIMS & OBJECTIVES

1. To compare the diagnostic performance of Silent MRA with other MR sequences (3D TOF, SWAN, Arterial spin labelling), in the diagnosis and characterization the intracranial dural arteriovenous fistulas with Digital Subtraction Angiography (DSA) as gold standard.
2. To find out the best possible combination of advanced MR sequences for diagnosis, classification and characterization of dAVF.



REVIEW OF LITERATURE



REVIEW OF LITERATURE

Epidemiology:

Intracranial dural arteriovenous fistula are anomalous communications between dural arteries and dural venous sinuses, meningeal veins, cortical veins. The incidence of this rare intracranial vascular malformation is around 0.16/100,000 per year which is lesser than the incidence of AVM and this incidence is higher in Asian population which is 0.29/100,000/year. (13) & (14)

The mean age of presentation is around 50 to 60 years and the incidence in paediatric populations is very less and if present, the etiology could be congenital. dAVF comprise of approx 5 to 6% in supratentorial and 30 to 35% in all infratentorial vascular malformations. (15).

Historical analysis of dAVF:

The knowledge about the pathology, etiopathogenesis and natural history of the disease has grown over the years ever since the disease was first described by 1873 by Rizzoli wherein he described a dural based arteriovenous aneurysm in a 9-year-old girl who presented with symptoms of seizures and pulsatile tinnitus which was later found to be an abnormal communication between transverse dural sinus and transdural occipital artery (15).

Later on, after 58 years of its discovery, Sachs in the year 1931, followed by Tonnis in the year 1936 were the pioneers in describing the angiographic appearance of dAVF. They found that fistulous connections exist between dural meningeal arteries and dural sinuses. (15) & (16). The occurrence of spontaneous dural AVF was first described by Verbiest and Fincher in the year 1951 followed by description of high flow dural Arteriovenous shunt in a 3-year-old boy by Van de werf in 1964. (17).

Han Newton in the 1960s and later on Rene Djindjian in the 1970s were the pioneers in angiographic characterization of these dural arteriovenous fistulae. They studied these fistulae extensively through selective injections of external carotid arteries and helped in understanding the vascular supply of these dural lesions which emanated not only from external carotid arteries but also from pial branches from ICA, vertebral arteries. Based on the arterial supply Newton also grouped the lesions into pial, mixed pial- dural and dural lesions. Newton also emphasized the role of contralateral ECA injections by demonstrating the bilateral ECA supply to the lesions (1). Later on, the Lasjaunias, extensively studied the angioarchitecture of these dural arteriovenous fistulae and the role of cortical venous drainage in determining the progression of the disease. His work also gave insight into the access for endovascular management of these fistulae along with the associated risks and complications (18). Contributions by Davies et al and Van Dijk et al in the natural history of the disease helped in subgrouping of the disease based upon the morphology and clinical presentations, thus, helping in robust patient selection with varied therapeutic options for each subgroup. (19), (20), (21). Numerous landmark articles by several authors have helped in understanding the natural history and progression of the disease, thus paving the way for better diagnosis management of the disease.

Etiopathogenesis:

The exact etiology and pathogenesis of the formation of these fistulae is unclear. The earlier school of thought was that of a congenital benign lesion due to venous sinus anomaly. Later in the 1970s, Djindjian and Castaigne postulated the acquired etiology where in there is formation of numerous microfistulae which could be either embryological remnants or developing de novo and neoangiogenesis between the meningeal arteries and dural sinuses as a sequel to the venous sinus occlusion. The cause for the venous sinus occlusion could be congenital in patients with

hereditary prothrombotic states like factor V mutation, Protein C&S deficiency or it could be acquired like surgery, trauma, post-partum status, dehydration, tumors, hormonal disorders and infection. However, the variability of development of the dAVF in these wide spectra of etiological factors pertaining to dural sinus occlusion may be due to the varied host response in a prothrombotic environment. (22), (15).

The central point in the pathogenesis is venous occlusion. Increased venous pressure and venous congestion occurs with venous sinus occlusion, leading to local ischemia and hypoxia with recruitment of angiogenic factors like vascular endothelial growth factor (VEGF) and Basic fibroblast growth factor (bFGF) which in turn triggers the formation of neoangiogenic channels between the osteodural, meningeal veins and meningeal arteries. These microfistulae can be dormant embryological fistulae which are opening up after the trigger or they can arise de novo after the occlusion of sinus. This has been supported by the fact that many immunohistochemical studies have shown expression of these factors in the dAVF. (22), (23). Further increase in venous pressure will lead to neointimal proliferation and venous reflux into the cortical veins with further remodelling of these cortical veins. These remodeled veins are fragile and lead to formation of aggressive type of fistula with intracranial hemorrhage as a sequela. With increasing venous hypertension, local ischemia occurs, which again starts the vicious cycle of newer vessel recruitment and progression of the disease. (15). Various articles have been published enumerating further causes for the development of these fistulae. Emissary vein playing a part in the formation of dAVF has been emphasized by Miyachi et al. The role of congenital dural shunts with hemodynamic changes leading to progression of the disease has been brought out by Nagm et al. (24,25)

Classification:

Many classification systems were proposed for subgrouping this disease which aid in the therapeutic decision and management of these patients. One of the earlier classifications attempted to classify the fistulas based on the location into cavernous, transverse sinus and anterior fossa fistulas. (26) Then, Aminoff et al in 1973, proposed a classification of the pathology based on the location of the fistula and subgrouping them into anteroinferior, superior and posterior groups. (16) Various other groups have also classified the dAVF into subgroups based on the location, of which Moret et al proposed a newer classification which is based on the relationship of the fistula with regards to the tissue type in the dural location.

Table 1: dAVF classifications based on location of the fistula

Name of the classification	Grades/types	Description
Picard	1	Lateral sinus
	2	Cavernous sinus
	3	Superior sagittal sinus
	4	Other locations : tentorial, falcine, convexity
Awad	1	Transverse- sigmoid sinus
	2	Cavernous sinus
	3	Tentorial
	4	Convexity

Name of the classification	Grades/types	Description
	5	Anterior Falx
	6	Sylvian
	7	Others including marginal sinus
Mironov	1	Dural sinus
	2	Cavernous sinus
	3	Galenic
	4	Base of skull venous plexus
	5	Cortical
Moret	1	Osteo-dural
	2	Duro- dural
	3	Duro-arachnoidal
	4	Duro-pial
Geibprasert	1	Anterior epidural including cavernous region
	2	Dorsal epidural
	3	Lateral epidural

These topographical classifications were not correlating with the clinical course and lack in aiding of the therapeutic decisions. Hence, classification systems based on the venous drainage came into vogue. The key factor in these classification systems is the draining vein pattern.

Castaigne et al in 1976, proposed a model based on venous drainage of cortical veins. They classified the fistula into three groups group 1 draining into sinus directly or through meningeal vein and group 2 drainage into a large venous sac and group 3 with drainage into cortical veins. (27). The first classification based on angioarchitecture with focus on the pattern of venous drainage was proposed by Djindjian and Merland in 1978(Table 2)

Table 2: Classification of dAVF by Djindjian and Merland

Type of classification	Nomenclature
I	Fistula draining into sinus or meningeal vein
II	Fistula draining into sinus with cortical venous reflux
III	Fistula with direct cortical venous reflux
IV	Fistula with venous dilatation

In 1993, Lalwani et al proposed a classification based on the draining vein pattern and classified these fistulas into four grades with grade 3 and 4 fistulas showing aggressive symptoms due to cortical venous drainage (28). In 1995 Cognard et al modified the classification of Djindjian with emphasis on cortical venous drainage and proposed a classification which is linked to the clinical course, prognostication and management of the patients (3). In the same year Borden and Schucart proposed a classification which combined the spinal as well dural arteriovenous fistulas and they described the lesions as dural arteriovenous malformations to emphasize upon the fact that multiple fistulas can occur in the same patient.

The various anatomical classifications after the initial classification by Djindjian et al which were based on the venous drainage pattern are shown in Table 3

Table 3: Various anatomical classification systems of DAVF

Name of the classification	Grades	Description
Lalwani et al	1	Antegrade flow into the sinus without venous restriction and cortical venous drainage
	2	Antegrade and retrograde flow into the sinus without venous restriction and cortical venous drainage
	3	Retrograde flow into the sinus with cortical venous drainage
	4	Only cortical venous drainage
Borden and Schucart et al	1	Flow into the sinus directly or via meningeal veins
	2	Flow into the sinus and subarachnoid veins
	3	Flow into the subarachnoid veins only

Name of the classification	Grades	Description
Cognard et al	1	Antegrade flow into the sinus without cortical venous reflux
	2a	Retrograde flow into the sinus without cortical venous reflux
	2b	Antegrade flow into the sinus with cortical venous reflux
	2a+b	Retrograde flow into the sinus with cortical venous reflux
	3	Direct cortical venous reflux without venous ectasia
	4	Cortical venous reflux with venous ectasia (>5mm)
	5	Reflux into the spinal perimedullary veins

Over the years. Both the Cognard and Borden systems of classification have been widely accepted in view of their aid in therapeutic decisions based upon the clinical features as well as radiological angioarchitecture. A new classification which takes into consideration the directness and exclusivity of the leptomeningeal venous drainage and also the venous strain caused by the leptomeningeal venous drainage was proposed. Lesions were classified as DES (Directness,

Exclusivity and Strain) and adding the letter "n" for non/no direct Leptomeningeal venous drainage (LVD), no exclusive LVD and No venous strain due to venous congestion.

The classification has 8 possible subtypes as illustrated in figure 1

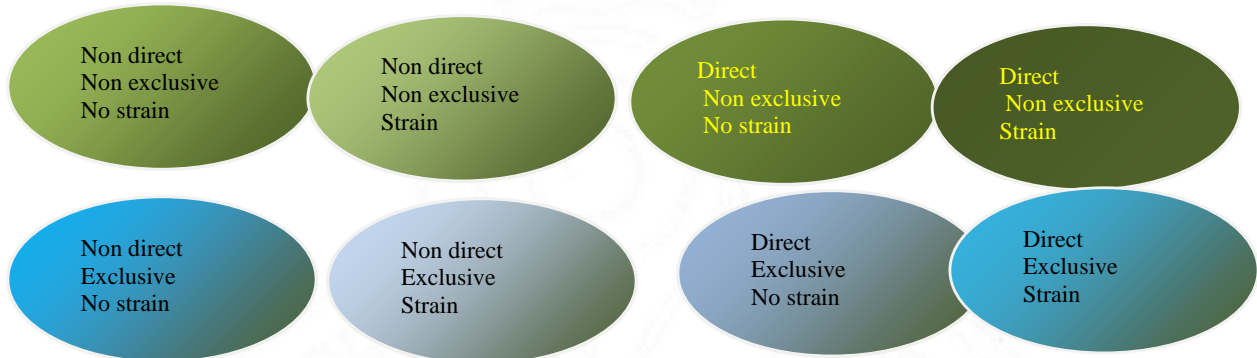


Figure 1: Baltsavias subtypes with the ovoid bubbles in green tint show nonexclusive LVD and in blue tint show exclusive LVD. The subtypes in right belong to Bridging vein shunts and the subtypes in left belong to dural sinus shunts (29).

This classification takes into account the aggressive lesions and gives insight into the leptomeningeal venous drainage. Direct LVD gives the location of the shunt and exclusive LVD indicates the anatomical cum functional nature of the shunting veins and strain gives an idea about the venous congestion associated with LVD. Thus, this classification is more robust and clearer with regards to the aggressive nature of the fistula.

Clinical features

dAVF usually presents in the age group of 50 to 60 years with variable clinical symptoms. Clinical symptoms may be related to the fistula, its venous drainage and also based upon the presence or absence of cortical venous drainage. It can present with life threatening hemorrhage as well as focal neurological deficits. Broadly clinical symptoms are grouped as benign and aggressive. (15), (30). The benign symptoms are seen in patients without cortical venous drainage. These include headache, tinnitus and ocular symptoms like proptosis, chemosis,

diplopia and decreased visual acuity. The aggressive symptoms are usually seen in patients with cortical venous drainage and these symptoms include hemorrhage, non-hemorrhagic focal deficits, dementia, seizures. The fistulas located in the anterior cranial fossa, cavernous regions present predominantly with ocular symptoms due to reflux into the superior ophthalmic vein from the cavernous sinus and fistulas located in the region of sigmoid sinus and jugular bulb regions present with pulsatile tinnitus due to increased flow within the venous channels in the vicinity of the middle and inner ear apparatus. The fistulas located in the superior sagittal sinus location present with seizures or symptoms related to raised intracranial tension, hemorrhage and hydrocephalus like headache with vomiting, papilloedema due to venous congestion and venous hypertension. (15). Grade 5 fistulas with spinal perimedullary venous drainage present with symptoms related to myelopathy. Hemorrhage is related to the aggressive pattern of venous drainage especially with cortical venous reflux and it can be intraparenchymal, subarachnoid and even subdural in location. It is due to the rupture of the thin fragile arterialized veins or hemorrhagic transformation of the cortical venous congestion. Non hemorrhagic neurological deficits like, cognitive disturbances, seizures, parkinsonism, cranial nerve palsies, dementia, ataxia. These symptoms are attributable to the venous hypertension and ischemia due to the high-pressure flow within the cortical veins. Brainstem dAVFs usually present with cranial nerve palsies and quadriparesis (4), (31),(32).

Natural History and course of the Disease:

Natural history of the disease is very valuable in therapeutic decision making and also in prognostication of the patient's condition. The aggressive features of the dAVF leading on to the

intracranial hemorrhage is one of the most dreaded complication of dAVF with risk to life and poorer clinical outcome. (33), (34), (35).

In 1994, Brown et al, in one of the earliest studies on natural history of dAVF, done on a group of 54 patients, noted a 1.8% risk of bleed per year and also found that fistulas in petrosal, straight sinus and cortical venous reflux and venous ectasia were found to be with higher risk of bleed. (36).

Davies et al in 1997, extensively worked on the natural history of dAVFs and they published their work as two part series separately for benign and aggressive lesions respectively. (19), (20). In their first article on benign lesions, they studied a cohort of 55 patients and found that the common site was transverse and cavernous sinuses (52 cases) and 27% of cases had a traumatic past history. Conservative management was provided for 58% of these cases. There was no worsening in both the conservative and treated patients' groups. Hence, they concluded that Borden Grade 1 lesions, without cortical venous reflux, can be managed conservatively and should be treated palliatively for any disabling symptoms or neurological deficits. In their second cohort of 46 patients with aggressive symptoms and Borden 2 and 3 fistulae, the outcome or course of the disease was not good. 30% of patients were managed conservatively and they showed 10% NHFND and 19% of hemorrhage and death. Hence it was concluded that patients with untreated Borden 2 and 3 lesions along with residual cortical venous reflux have to be treated as their outcome is really poor.

In a study by Soderman et al, for a cohort of 89 patients with Borden 2 and 3 dAVF, it was seen that Borden 2 lesions were common in transverse sigmoid sinus region whereas Borden type 3 fistulae were seen mostly located in the tentorium. Hemorrhage was seen in 32 patients and the annual incidence was 6% and it was noted more in patients with prior history of bleed (37)

In another study by Gross et al, in a cohort of 56 patients, 34% were of Borden type 1, 17% were of Borden type 2 and 49% were of Borden type 3 fistulae. It was found that most of the Type 1 fistulae had minimal risk of hemorrhage and were only followed up and they also had a 13 % rate of spontaneous occlusion whereas patients with cortical venous reflux and aggressive types (type 2 and 3) showed a 30% risk of bleed as well as 30% risk of non-hemorrhagic functional neurological deficits (NHFND). The annual risk of hemorrhage in this cohort was 6% with an overall mortality of 3% and only 3% of this cohort had spontaneous occlusion of fistula. The risk of bleed was much higher in patients with venous ectasia, to the tune of 21%, thus warranting urgent and complete treatment in these patients at the earliest. (6,38).

Van Dijk in 2002, in a cohort of 118 patients with cortical venous reflux found that these patients had an annual mortality rate of 10%, and 15 % rate of any neurological event with 8.1% risk of hemorrhage and 6.9% risk of non 20 hemorrhagic events. This further reinforced the aggressive management decision for all dAVFs with CVR irrespective of site and presentation. (21).

In comparison with other vascular malformations, like intracranial aneurysms with an annual risk of rupture for <7 mm of $\approx <1\%$ per year³⁹ and pial arteriovenous malformations with an annual risk of rupture $\approx 1\%$ to 4% per year), dAVFs with CVR have an increased risk of hemorrhage and they constitute the most dangerous lesions among the cerebrovascular malformations. (4).

Shah et al In their study have found that there was an annual rate of 0.8% in upstaging of dAVFs without CVR to dAVFs with CVR. (39). After endovascular management, recurrence was noted in almost 14% of cases, which was predominantly due to incomplete percolation and incomplete occlusion of fistula especially to the venous side of the fistula. Hence it emphasizes the fact that

these patients have to kept on regular follow up for early detection of recurrence and further planning of treatment with a curative intent. (40) (41).

DIAGNOSIS AND EVALUATION OF DAVF:

Intracranial dAVF are rare vascular malformations characterized by anomalous connections between dural arteries and meningeal veins, dural sinuses or cortical veins without any intervening nidus. The location is usually in the wall of the sinus or in its vicinity. Time and again, it has been proven that presence of cortical venous reflux increases the risk of hemorrhage and there is progression of disease from benign to aggressive type. Hence, the importance of early diagnosis and treatment cannot be overemphasized. As the presentation of the disease is very variable and non-specific, there is often a delay in the diagnosis of the disease (42).

Among the imaging modalities utilized for evaluation of dAVF, still the gold standard imaging for both diagnosis and follow up of these patients is DSA. CT and MRI including CT angiography and MR angiography, were the initially and commonly used modalities for initial diagnosis and evaluation of dAVF.

DIGITAL SUBTRACTION ANGIOGRAPHY (DSA):

DSA is the investigative modality of choice for diagnosis as well as angioarchitectural evaluation of dAVF with high temporal, spatial and contrast resolution among other imaging modalities. The information obtained from DSA is very critical in further planning of neuro interventional procedure. It is also the modality of choice for post treatment follow up of these patients.

In a case of dAVF, DSA study comprises of full six vessel angiography of bilateral ICAs, ECAs and vertebral arteries. Super Selective injections of ECA branches and 3D rotational angiogram

from the main involved feeding arterial pedicle have to be obtained for further treatment planning. In DSA, it is essential that the detailed delineation of fistula angioarchitecture should be obtained. The location and number of fistulae, anatomy, course of the ECAs and their dural branches, pial branches if any, the venous drainage pattern, presence or absence of CVR, any presence and degree of dural sinus stenosis/ occlusion, and venous ectasia have to be evaluated. Each of these findings, is very essential in prognostication as well as in the further management of these patients. (43).

The normal cerebral venous drainage along with circulation time is also evaluated in DSA. All these details help in further classification of the dAVF based on the Cognard or Borden classification model. Venous congestion due to the disease could be identified by presence of tortuous, engorged leptomeningeal veins called as pseudophlebitic pattern in the venous phase. This feature has a role in prediction of aggressive nature of the fistula even without the presence of CVR. (44)

DSA has its own disadvantages, being a minimally invasive procedure with inherent risk of complications pertaining to puncture site hematoma, pseudoaneurysms and retroperitoneal hematoma as well as neurological complications like stroke. Repeated follow up studies pose the risk of radiation hazard. In a study by Kaufman et al, the neurological complication rate of DSA is approximately 2% and a mortality rate of approximately 0.06%. (45)

COMPUTED TOMOGRAPHY

Non contrast CT (NCCT) has been found to have poor specificity and sensitivity for the diagnosis of dAVF, even though it may be the first imaging modality to be employed in any neurological condition especially presenting with headache. Various indirect findings attributed

to dAVF include presence of hemorrhage, diffuse cerebral edema, hyperdense curvilinear sulcal vascular structures pertaining to dilated venous channels, bony resorption and prominent osseous foramina due to pressure effect by long standing dilated tortuous venous sinuses. The hemorrhage seen in NCCT may be remote from the site of shunt as it is often due to the venous varices. (46)

COMPUTED TOMOGRAPHY ANGIOGRAPHY

CTA has the potential to illustrate the vascular details of dAVF along with the parenchymal details of the brain parenchyma. Meckel et al demonstrated the role of helical MDCT angiography in arterial phase in identifying not only the fistulous point but also in demonstrating the retrograde flow and CVR based on differential density within these structures. They suggested that this arterialization of cerebral veins must be looked for in the appropriate clinical scenario. (47).

Beijer et al in a small case series demonstrated the value of 4D CTA in evaluation DAVF and showed correct identification of fistulous site and pattern of venous drainage but acknowledged the lower spatial and temporal resolution compared to DSA. (48)

Williams et al in their study of 11 patient, found that 91.1% of dAVF were accurately identified and categorized as per Borden classification. Larger feeding arteries could be identified in all these cases, while the feeding arteries from ICA or VA were often missed. Though CTA was found to be useful to formulate the treatment strategy, accurate depiction of venous drainage patterns, was not attempted. (49).

With CT and CTA, the exposure to iodinated contrast and repeated radiation exposure remains a cause for concern and besides none of the studies have conclusively demonstrated sensitivity or

specificity comparable to DSA. Thus, their role in management of dAVF is limited.

MAGNETIC RESONANCE IMAGING

MRI is a non-invasive imaging modality in diagnosis and follow up of dAVF patients. In any patient presenting with nonspecific symptoms of headache and vague neurological symptoms, MRI has been used as the first imaging modality for ruling out intracranial pathology. In one of the studies by Dietz et al, for evaluation of pulsatile tinnitus with MRI, the most frequent pathology was DAVF. Features like prominent extracranial flow voids along with trans osseous flow voids /vessels, patency of dural sinuses and stenosis of transverse sinuses were noted in MRI and helped in the diagnosis of dAVF. (50)

In a study by Kitajima et al, the role of contrast enhanced MRI in identification of cortical venous drainage was evaluated. It was concluded that Contrast Enhanced MRI (CEMRI) performed better than non-contrast scans in identification of CVR and also concluded that 3D MPRAGE sequences are superior to T1 contrast enhanced sequences due to the higher spatial resolution and the detailed evaluation of vessels in subarachnoid spaces. (51)

Kwon et al also established the pivotal role of cortical venous reflux with aggressive features. The study correlated poor outcome with the presence of tortuous leptomeningeal and medullary vascular structures on MRI. It was concluded that MRA can be an adjunctive tool to DSA for dAVF diagnosis and evaluation. (52)

The role of 3T MRI in evaluation of dAVF was assessed by Bink et al. The study analysed for the presence and site of fistula, venous drainage and feeders and also assessed whether management planning is feasible with MRI. Although the sensitivity for diagnosis was 85%, the study concluded that DSA is still required for management planning. (53)

In a comparative analysis of CT and MRI in dAVF, Lin et al in 2016, concluded that even though CT and MRI showed high sensitivity and accuracy, MRI was better than CT. The study concluded that MRI did not help in increasing the accuracy of the modality. (46)

MAGNETIC RESONANCE ANGIOGRAPHY

Both non-contrast MR angiography as well as contrast MR angiography have been proved to be effective in diagnosis and evaluation of dAVF as compared to DSA in various studies. In the study by Meckel et al in 2007, the role of time resolved 3D contrast enhanced MRA in evaluation of dAVF in a short series of 14 patients was assessed. The location of fistula, grading and post treatment follow up for shunt obliteration was assessed. The side, location of fistula and also post treatment occlusion were assessed accurately in all cases, However, the grading of fistula showed only 80% accuracy. They concluded that this sequence can be utilized as a follow up method to DSA and not as a replacement for DSA. (54).

Farb et al in 2009, studied the role of time resolved contrast enhanced MRA at 3T for dAVF evaluation. TRICKS (Time resolved imaging in contrast kinetics) model was compared to DSA in their series of 40 patients with 20 fistulae. It showed 93% accuracy in identification and grading of fistula. it was concluded that time resolved MRA can be an useful modality for initial diagnosis as well as continued surveillance of dAVF. (55)

Nishimura et al in their study analysed that 4D time resolved CE-MRA showed 87 to 89% sensitivity in identifying the main arterial feeders with moderate interrater agreement. The accuracy of classification as per Borden system was 89% and for the site of the fistula, it was 95%. However, detailed description of the feeders and veins to the fistula was not carried out in this study. (56)

Ertl et al in their study done in 2016, showed that CEMRA had 100% accuracy or detection of dAVF along with identification of the cortical veins. In this study, dominant feeders were only evaluated with moderate intermodality agreement. However, veins draining the fistula were not assessed in detail. The study showed that CEMRA can be useful for planning therapeutic strategy in 95.4% of cases, though the prediction of accessible route for intervention only modest (59%). (57)

Dissaux et al, in a dual center study of 44 patients, assessed the role of 4D MR angiography in 3T, in the follow up of treated dAVF patients, found that 4D MRA had 63.6 % sensitivity and 96.6 % specificity with good intermodality agreement (0.6 Kappa) for detection of residual/recurrent fistula. (58)

Azuma et al compared 3D non contrast TOF MRA at 3T with DSA in evaluation of dAVF in 26 patients. The study showed good interobserver and intermodality agreement for fistula site, arterial feeder and venous drainage. It was concluded that still DSA is required for treatment planning and risk stratification of dAVF. (8)

Cheng et al in their study of 41 patients with intracranial vascular malformations, compared MR contrast enhanced time resolved angiography with keyhole technique (4D-TRAK MRA) and Time of flight MR angiography (3D TOF MRA) for accuracy in diagnosis, evaluation of arterial feeder and draining veins. The diagnostic accuracy of 4D-TRAK MRA (92.7%) was more than 3D TOF MRA (63.4%) with conclusion that 4D-TRAK MRA is advantageous than 3D TOF MRA in the evaluation of these lesions. (59)

SUSCEPTIBILITY WEIGHTED IMAGING (SWI):

SWI has been extensively studied in the evaluation of various cerebral vascular malformations and stroke. (60) (61)

Saini et al in 2009 reported the use of SWI in identification of cranial dAVF. This study noted the prominent hypointense vessels in a patient with dAVF. The site of fistula as well as the partial reversibility of this venous prominence after embolization of the shunt was accurately demonstrated. The study concluded that these findings were caused by prolonged circulation time in dAVF with venous congestion leading to greater oxygen extraction, increased desaturation and increase in deoxyhemoglobin concentration within the venous channels. (62)

Noguchi et al analysed the role of SWI in small series of 10 patients in identifying retrograde cortical venous drainage in intracranial dAVF and compared with dynamic susceptibility contrast (DSC) perfusion. DSC sequence was more sensitive for cortical veins and SWI was more sensitive for deeper medullary veins and the combination of these sequences improved the diagnostic accuracy of retrograde venous drainage. (63)

Gasparetto et al in a small series of patients found about the presence of presence of cortical venous reflux drainage in transverse sinus dAVF patient with progressive dementia. This study concluded that SWI may be incorporated in routine protocol for evaluation of all suspected dAVF cases. (64).

Guillon et al studied the role of SWI in evaluation of the fistulous point, identification of CVR and pseudophlebitic pattern in small series of 6 patients. They concluded that the fistulous point can be identified as focal hyperintensity along the wall of the dural venous sinus or cortical vein

and venous congestion can be identified by the tortuous, prominent increased venous channels. (65)

Nakagawa et al in 2013, compared SWI with DSA in pre and post treatment follow up. It was found that the pre-treatment SWI hyperintensity in the cortical vein was found to resolve in post treatment SWI images. It was concluded that SWI can be a viable non-invasive follow up of dAVF. (66)

Hodel et al, in 2016, analysed both SWI and ASL in evaluation of intracranial vascular shunts. In this series, 10 dAVFs were identified with excellent correlation with DSA in diagnosis and site of fistula (67)

In a larger series of 26 patients, Jain et al analyzed the role of SWI in pretreatment evaluation of intracranial dAVF. The location of fistula, presence of CVR and venous ectasia were assessed and it was found that the SWI had higher accuracy rates as compared to conventional MRI and can be utilized in preprocedural evaluation of DAVF (9).

DIFFUSION WEIGHTED IMAGING

Sato et al in 2011, evaluated the role of DWI in analyzing the effects of cortical venous reflux on the brain parenchyma. The study hypothesized that CVR can lead to adverse hemodynamic changes and this can be identified by presence of lower ADC valued in these affected regions and this change improved post treatment. This novel objective criteria for assessment of severity of disease as well as post treatment follow up of patients can be a part of routine protocol of dAVF evaluation. (68)

ARTERIAL SPIN LABELLING

Arterial spin labelling perfusion study, an adiabatic inversion pulse labels the protons in vessels in a predetermined proximal plane. When the labelled protons traverse the imaging plane, signal will be generated on subtraction of labelled images from the control images obtained without labelling pulse. Due to signal decay over time and achievement of spin equilibrium, no signal is seen beyond the level of capillaries. However, in intracranial shunts, this signal directly goes into the venous side and can be identified as hyperintense signals in the location of expected dural sinus or cortical veins. (7) (69).

Diebler et al found that vascular malformations can lead to focal hyperperfusion states and also conclude that ASL can be used for identification of vascular malformations with high success rates. (70)

Wolf et al in a small series of 7 patients with intracranial vascular malformations, analysed the role of ASL and found that there was increased signal intensity noted in the nidus as well as in the draining veins and adjacent parenchyma with decreased perfusion in deeper regions like deep gray nuclei caused by steal effect. (71)

Robson et al proposed an unique time resolved vessel selective MRA using ASL with variable time delays and vessel selective ASL pulses with a temporal resolution of 200msec. The study concluded that this ASL based technique could supplant DSA without using any exogenous contrast agent. (72)

Sakamoto et al was one of the first study to report the utility of ASL in a case of cavernous sinus dAVF. The hyperintense ASL signal in bilateral SOVs resolved on post treatment imaging and hence they conclude that ASL can be useful in follow up of these cases with shunts. (73)

Noguchi et al analyzed the role of ASL in a small series of patients with dAVF for evaluation of venous drainage from the shunt site and also the grading of dAVF with 100% accuracy in identifying venous sinus signal in Borden 1 and Borden 2 cases and also cortical venous signal in Borden 3 cases. ASL was poor in detection of CVR in Borden 2 cases (74). Iryo et al in 2013 analysed the role of 4D ASL MRA in 3T in intracranial dAVF in a small series of 9 patients and their findings were compared to DSA. The site of fistula, feeders and venous drainage to the fistula were evaluated and it was observed that good to excellent agreement between modalities could be obtained. (51).

In a review article on ASL artefacts with 3D pseudocontinuous ASL analysed the presence of venous ASL signal in vascular shunts and proposed that venous hyperintense signals in a shunting lesion of the brain. necessitates further intensive evaluation to confirm the underlying shunt. (75).

Sunwoo et al analysed the degree of AV shunting in 40 cases of intracranial vascular malformations and found that the presence of venous signal in all these shunting lesions and the venous signal intensity correlated well with the extent of shunting which in turn had very good correlation with the shunt volume on DSA evaluation. (76).

Amukotuwa et al in their study of 34 patients with intracranial dAVF evaluated 3D pseudocontinuous ASL sequences for venous drainage and grading of fistula. It was found that high grade dAVF can be identified with high accuracy and suggested inclusion in the routine protocol of MRI screening of dAVF patients. (77).

Yamamoto et al in 2016 analyzed small group of 13 patients with dAVF and compared conventional MRI and ASL technique at 3T for detection of dAVF and also compared pre and

post treatment ASL images. It was concluded that this sequence can be very useful for identifying the presence of shunt and in post procedure follow up of patients in spite of poor spatial resolution. (78).

In a review of ASL artefacts, Jagannohan et al, analysed the venous ASL signal in intracranial high flow malformations and also proposed that the smearing or blurring artefact occurring in cortical venous reflux or retrograde venous flow can be due to the stagnation of tagged spins and can be utilized in prediction of grading of DAVF especially high grade fistulas. (79).

SILENT MR ANGIOGRAPHY

Silent MR angiography is a novel sequence which is based on ASL technique with a preparatory pulse followed by data acquisition by 3D Radial sampling with ultrashort TE of 0.016 msec. The algorithm employs a long radiofrequency inversion pulse for tagging the blood protons and once the tagging is done, the tagged blood protons are captured by radial acquisition with ultra-short TE as low as 0.016 milliseconds.

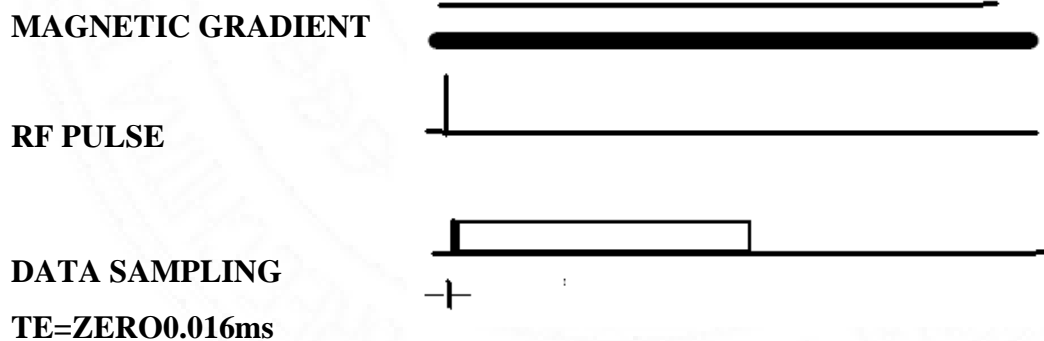


Figure 2: Silent MRA Pulse sequence diagram

The acquisition is then followed by a control dataset with a labelling pulse applied above the head to minimize the magnetic transfer effects and these two datasets are subtracted to obtain only the vascular tree with excellent background suppression. The ultra-short TE eliminates the susceptibility artefacts. This helps in various clinical situations. (10)

An initial clinical report on utilization of silent MRA for vascular shunts was published by Moon et al. This study reports the utility of silent MRA in diagnosis of brain arteriovenous malformations. The most important factor is the ultra-short TE which minimizes the phase dispersion of tagged blood proton signals in the acquired voxel and also decreases the susceptibility artefacts. The ASL preparatory pulse helps in visualization of minimal blood flow signals also. (80)

Takano et al in their study done in 2017, analysed the utility of silent MRA in follow up of basilar top aneurysms treated by stent assisted coiling in 07 patients in comparison with 3D TOF MRA. They found that flow within the stents was visible in silent MRA with a higher score than TOF MRA along with accurate depiction of neck remnants in comparison with DSA. (81).

Oishi et al in 2019, studied the utility of silent MRA and TOF MRA in assessment of parent artery and embolization status of aneurysm after flow diverter placement. They evaluated 78 aneurysms and found that silent MRA scored higher than TOF MRA in assessment of parent artery along with significant better scores in embolization status of aneurysm, post flow diverter placement. They concluded that silent MRA is better for visualization of blood flow inside flow diverter devices and also helpful in assessment of the embolization status of aneurysms. (82)

In the study published by Ryu et al, the role of silent MRA as a follow up imaging for treated intracranial aneurysms was analysed. It was found that silent MRA scored better than TOF MRA in assessing the flow at treated site with substantial interobserver agreement. The diagnostic performance of silent MRA was superior to TOF MRA with higher AUC 0.962 versus 0.843. (12)

Arai et al analysed silent MRA in evaluation of cerebral arteriovenous malformations. They found out that silent MRA scored better than TOF MRA in identification of the nidus and drainage significantly. The accuracy rates of grading the AVM by silent MRA was around 79% whereas the accuracy rate of TOF MRA was 38%. Hence the study concluded that silent MRA is very useful in evaluating the AVMs with better accuracy rates for detection of micro-AVM than TOF MRA. (11).

Tomura et al in their study analysed the efficacy of silent MRA in visualizing the cerebral arteries in patients with Moya Moya disease. Even though signal to noise ratio and contrast noise ratio was higher in TOF MRA, silent MRA scored better than TOF MRA in grading of the moya moya vessels. They concluded that silent-MRA was better than TOF MRA for visualization of moya-moya vessels and performed equally in depiction of steno occlusive lesions in cerebral arteries in patients with moya moya disease. (83).

POSITRON EMISSION TOMOGRAPHY

Iwama et al analysed the metabolic changes in patients with dAVF by evaluating a small group of 10 patients in both pre and post treatment states and estimated the relative cerebral blood flow(rCBF), relative cerebral blood volume(rCBV) and relative oxygen extraction fraction(rOEF) in these cases. The study showed that the rCBF was low and rOEF was high in

those areas of cortical venous reflux from the fistula prior to intervention and the same normalized after the treatment.(84)

TREATMENT

Benign intracranial dAVF are generally managed conservatively; early definitive treatment is indicated for aggressive lesions. (85) & (86).

Satomi et al evaluated the outcomes of conservative management of benign DAVF in 119 patients in 2003. They observed 73 patients of which 43 underwent palliative embolization and 1 patient underwent surgery due to intractable symptoms. It was noted that up-staging to aggressive lesion was observed only in 2 % of cases whereas in 98% of the cases the disease was stable and hence the study concluded that good clinical and imaging follow up is a suitable option for benign presentation of dAVF. (87).

In 2014, Rammos et al analysed the results of endovascular management of dAVF. They found that endovascular approach is the modality of choice for treatment of dAVF. The study also concluded that the transarterial route was the commonest approach especially due to the increase in use of liquid embolic agents. Transvenous route was used for fistulae in midline and skull base and also for dAVFs with isolated sinus (88)

In the study by Gross et al in 2016, one of the largest series of cases, they had analysed in depth various management issues pertaining to dAVF especially the occlusion rates, recurrence rates, further clinical course after endovascular treatment of dAVF. With the advent of liquid embolic agents, the paradigm has been shifted to endovascular treatment of dAVF with significant improvement of occlusion rates. In analysis of 251 patients, it was noted that the initial occlusion rate was 70% with a recurrence rate of 3% and a permanent complication rate of 3%. (89)

In the study by Serulle et al, conservative management was suggested for benign Cognard lesions (1 and 2a lesions) with spontaneous occlusion seen in cases with stagnant flow especially with use of carotid compression techniques. Hence, imaging follow up is warranted in these patients with benign lesions to detect worsening of lesion grade over time. (42).

COMPLICATIONS

Embolization of intracranial dAVF has some risk of peri as well as post procedural complications. In the outcome analysis by Lv et al, an overall complication rate of 12% was reported. There was occurrence of bradycardia due to trigemino-cardiac reflex in 2 patients with tentorial dAVFs and cranial neuropathy was noted after embolization of cavernous sinus dAVFs. Intraparenchymal infarcts due to occlusion of arteries and veins are usually rare. Procedure related complications like stuck catheter and onyx migration can be improved by endovascular technique refinements. (90)

The series by Baltasvias et al with a cohort of 170 patients reported an occlusion rate of more than 60%. There was nil mortality with approximately 2% risk of permanent as well as temporary neurological deficits with occurrence of temporary self-limiting diplopia in 3 patients following embolization. (91)



MATERIALS AND METHODS



MATERIALS AND METHODS

Institutional ethics committee (IEC) approval was obtained prior to the study. The study was conducted as a retrospective and prospective study from May 2016 to May 2021. All patients with intracranial dural arteriovenous Fistulas who have presented since May 2016 till date of IEC approval, who have undergone DSA and MR imaging including Silent, TOF MRA, ASL, SWAN as part of their imaging protocol were taken as part of the retrospective arm of the study. Patients with suspected intracranial dural arteriovenous fistula who presented to the SCTIMST neurosurgery, neurology and interventional radiology clinics by direct referrals or through consultations from other departments from the date of IEC approval till Jun 2021 were included in prospective cohort. Consent waiver approval for the retrospective arm of the study was obtained from the IEC. Informed written consent was obtained from each and every patient/guardian after explanation of all aspects of the study as per consent form and details were noted as per proforma. (Appx A and Appx B). Patients who need DSA and MRI for assessing the completion of treatment on follow up were also included.

Inclusion criteria:

- All consecutive patients with dAVF presenting to SCTIMST neurosurgery, neurology or radiology clinics – outpatient as well as inpatient.
- Patients who have been diagnosed with dAVF and undergone MR imaging including silent MRA along with DSA.

Exclusion criteria

- Patient or relatives declining consent in the prospective study.

- Claustrophobic patients, patients with 3Tesla incompatible metallic implants, pacemakers or cochlear implants.
- Contraindication for iodinated contrast (DSA).
- Brain vascular lesions other than dAVF will be excluded.

Study protocol:

1. Retrospective patients since May 2016 till date of IEC approval, who have satisfied the inclusion criteria and Consecutive patients with intracranial dural arterio venous Fistula were considered for inclusion.
2. Consecutive patients who undergo Digital subtraction angiography underwent Magnetic resonance imaging as per the stated protocol.
3. The digital subtraction angiography was done in Interventional Radiology Suite at SCTIMST on General Electric Innova biplane 3131 and Magnetic resonance was done on General Electric Discovery 750E 3.0 Tesla machine.
4. The MRI protocol for the Study included the following sequences,
 - (a)**3DTOF MR angiogram** (TR/TE, 19/2.9 msec; flip angle, 15°; field of view, 200×200 mm; matrix, 416×192; section thickness, 1.2 mm; NEX, 1; band width, ±41.7 kHz; acquisition time, 3 min 31 s.)
 - (b)**Silent MR angiogram** (TR/TE, 1116.4/0.016 msec; flip angle, 5°; field of view, 180×180 mm; matrix, 150×150; section thickness, 1.2 mm; number of excitations (NEX), 1.5; band width, ±20 kHz; acquisition time, 7 min 40 s.)
 - (c)**SWAN angiogram** :TR/TE, 42/28 msec; flip angle, 15°; field of view, 220×220 mm; matrix, 288×384; section thickness, 2.4 mm; NEX, 1; band width, ±31.2 kHz; acquisition time, 4 min 5 s.

(d)**Stack-of-spirals 3D-fast-spin echo pseudocontinuous ASL sequence:** TR/TE/PLD 4852/10.7/2025ms, FOV 240x240mm, NEX 3, spiral readout of eight arms x 512 samples, 30 x 4.0 mm axial sections with whole brain coverage, and time duration of 4 min 22 s.

(e). Fusion with 3D FLAIR sequences was performed using the proprietary READY view software (GE Healthcare, Milwaukee, USA).

DSA Protocol :

All DSA studies were done on biplane flat panel unit (Innova 3131, GE, Milwaukee, USA). All diagnostic angiograms were done under local anesthesia after premedication administration. The right common femoral artery was punctured, and a 6F or 5F short femoral sheath was secured. Heparin was used as anticoagulant in the dose 50U/Kg body weight. The angiograms were done using 5F vertebral glide or Judkins right coronary catheter with 0.035 Terumo guidewire. Iohexol or Iodixanol was used as contrast agents for the studies. Angiograms of bilateral CCAs, ICAs, ECAs and vertebral arteries were obtained. After identification of shunt, selective angiograms of ECA branches commonly Internal maxillary artery (IMA), occipital arteries and ascending pharyngeal arteries were also obtained. After conclusion of the diagnostic study the femoral short sheath was removed and hemostasis achieved by manual compression.

The patients underwent magnetic resonance imaging as per the stated routine protocol. Digital subtraction angiography was obtained as per standard guidelines followed in the institution. For the purpose of the study magnetic resonance imaging and digital subtraction angiography images were obtained from picture archiving and communication system (PACS), anonymized and stored separately in numbered folders.

The anonymized images in the separate folders were analyzed by two neuroradiologists (SK and AP with 15 and 02 years of experience) independently. The 3D-TOF MRA image datasets, Silent MRA data sets, SWAN data sets, ASL data sets, DSA images were separately provided to the participating radiologists. The images were read with a time gap of at least 2 weeks to exclude memory bias.

The data sets of minimum diagnostic quality were included. Then the 3D-TOF, Silent MRA and the DSA were evaluated independently for following parameters: the location of the dural arteriovenous fistula, grading as per Cognard & Borden systems of grading, feeding arteries(overall as well as prominent >1.5 mm diameter feeding arteries), accessible feeder, venous drainage including immediate venous drainage in the vicinity of the fistula as well as the overall venous egress from the fistula, cortical reflux and pseudophlebitic pattern of vessels (in SWAN and DSA). ASL and SWAN images were evaluated for location of the fistula, grading of the fistula and cortical venous reflux and compared with DSA for the same variables. Proforma for the evaluation of patient is attached as appendix C. The quality of the silent, TOF MRA images was also evaluated and was classified as good, average and poor. After collection of data, statistical analysis was done to generate the inferences.

DEFINITIONS :

Prominent arterial feeders: Feeders > 1.5 mm in diameter supplying the fistula.

Immediate venous drainage: Venous drainage in the vicinity of the fistulous site.

Presence of fistula:

SWAN - Hyperintensity within a venous structure either dural venous sinus or cortical vein; on magnitude images; where the signal was noted to be brighter than the normal expected bright signal within the normal veins distant from the involved site.

ASL- Bright signal within a venous structure, either dural venous sinus or cortical vein.
Exclusion of ASL related artefacts was mandatory.

Silent and TOF MRA: Abnormal cluster of vessels converging onto the wall of the sinus with early venous drainage in the sinus.

Fistulous point (FP):

Silent and TOF MRA: the point where there is convergence of the cluster of feeders in the wall of the sinus or directly into the cortical vein.

SWAN – The site which shows within the venous drainage pathway either dural venous sinus or cortical vein, with signal intensity similar to that of the arterial signal, at circle of Willis.

ASL- Site where ASL signal was first noted within the venous drainage pathway either dural venous sinus or cortical vein, with signal intensity similar to that of the arterial signal, at circle of Willis

Cortical venous reflux:

Silent and TOF MRA: abnormal retrograde reflux into the cortical vein, adjacent to the fistulous point.

SWAN - Hyperintensity seen within a cortical vein adjacent to the identified fistulous point where the signal intensity was noted to be more than that within normal veins distant from the involved site.

ASL- Bright signal within a cortical vein adjacent to the previously identified fistulous point.

Pseudophlebitic pattern:

SWAN - Pseudophlebitic pattern was defined as presence of multiple, dilated, tortuous, prominent venous channels either cortical, medullary or both; compared to normal brain parenchyma.

ASL, Silent and TOF MRA- Pseudophlebitic pattern was not commented upon.

Grading of fistula:

Silent and TOF MRA : abnormal venous drainage antegrade to the site of fistula without any identification of cortical vein is considered as Cognard 1, abnormal venous drainage retrograde to the site of fistula without any identification of cortical vein is considered as Cognard 2a, abnormal venous drainage antegrade to the site of fistula with any identification of cortical vein is considered as Cognard 2b, abnormal venous drainage retrograde to the site of fistula with any identification of cortical vein is considered as Cognard 2a+b, abnormal venous drainage seen directly into the cortical veins without any antegrade or retrograde venous drainage is considered as Cognard grade3, abnormal direct cortical venous drainage with ectatic venous sacs was considered as Cognard grade 4, abnormal direct venous drainage into the spinal perimedullary veins was considered as Cognard grade 5. Fistulas of Cognard grade 1 and 2a were grouped as Borden grade 1, fistulas with Cognard grading of 2b and 2a+b were grouped under Borden grade 2 and Fistulas with Cognard grading 3, 4 and 5 were grouped under Borden grading 3.

SWAN and ASL:

On SWAN and ASL, abnormal hyperintensity within the dural sinus adjacent to fistulous point without any abnormal signal in adjacent cortical veins was taken as Borden Grade I, abnormal hyperintensity seen within both the dural sinus as well as adjacent cortical vein was taken as Borden grade II and abnormal signal only within the adjacent cortical vein without any abnormal signal within the dural sinus was taken as Borden Grade III fistula.

On SWAN and ASL, abnormal hyperintensity only downstream within the dural sinus adjacent to the FP was taken as Cognard Grade 1. Abnormal signal only upstream from the FP, within the dural sinus, was taken as Cognard Grade 2a. Abnormal signal only downstream from FP with

signal within adjacent cortical veins was taken as Cognard Grade 2b. Abnormal signal both upstream as well as downstream from FP with signal within adjacent cortical vein was taken as Cognard Grade 2a+b. Abnormal signal only within cortical veins adjacent to FP without upstream or downstream signal within dural sinus was taken as Cognard Grade 3. These features of Cognard Grade 3 when associated with a visible ectatic venous sac was graded Cognard Grade 4. The visualization of abnormal signal within perimedullary veins around the spinal cord led to Cognard Grading 5.

Benign grading was considered with Borden Grade 1 and Cognard Grade 1 and 2a
Aggressive grading was considered with Borden Grade II and III and with Cognard 2b, 2a+b, 3, 4 and 5.

Accessible feeder: The feeder which is relatively straight, hypertrophied and easily accessible was designated as accessible feeder for endovascular management in silent and TOF MRA.

STATISTICAL ANALYSIS

The demographic details of the patients, clinical presentations were tabulated. Male: female ratio, median age, ratio of aggressive and non-aggressive fistulas was calculated. Two neuroradiologists interpreted the silent MRA, TOF MRA, SWAN and ASL findings by consensus method, after 2 weeks interval to avoid memory bias. The DSA findings were taken as gold standard. The above analysis was done in Microsoft Excel format.

Sensitivity, specificity, positive predictive value and negative predictive value for the arterial feeders was obtained for silent MRA and TOF MRA. Sensitivity, positive predictive value and accuracy was obtained for site, grading, accessible feeder, venous drainage (immediate as well as overall venous drainage) for silent and TOF MRA. Sensitivity, positive predictive value, accuracy was also obtained for ASL, SWAN with regard to site, grading of fistula and pseudophlebitic pattern in SWAN. Spearman's rank correlation analysis was done for correlation of the arterial feeders and draining veins with DSA for each patient. Chi-square test was also done for corroborating the relation between image quality in both silent and TOF MRA for diagnostic accuracy. McNemar test was done for testing the level of significance of difference in sensitivity between silent and TOF MRA for arterial feeders and venous drainage.

Intermodality agreement and Kappa values were obtained for Silent MRA, TOF MRA, ASL and SWAN for all the variables analysed. Kappa values ≤ 0 was taken as indicating no agreement and 0.01–0.20 as none to slight, 0.21–0.40 as fair, 0.41–0.60 as moderate, 0.61–0.80 as good, and 0.81–1.00 as excellent agreement.

Statistical analysis was performed using R software, version 4.0.2, 2020 and p value < 0.05 was considered significant.



RESULTS



RESULTS

Demographic details:

A total of 40 patients with intracranial dAVF were included in this study of which 18 patients were from the prospective arm and 20 patients were from the retrospective arm of the study. Two patients had not undergone TOF MRA. The male to female ratio was 5:1 and median age was 46 years (12-77).

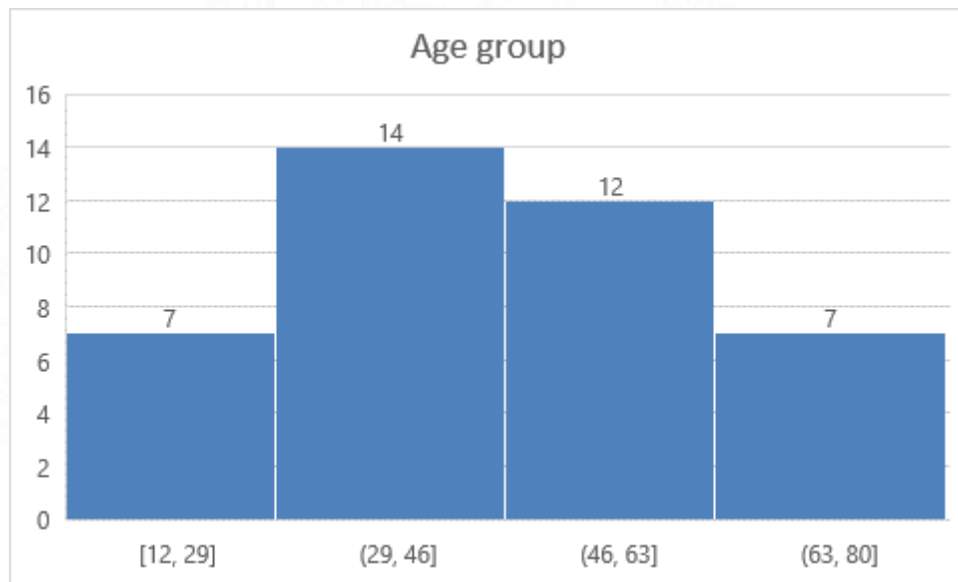


Figure 3: Bar diagram showing age wise distribution of patients.

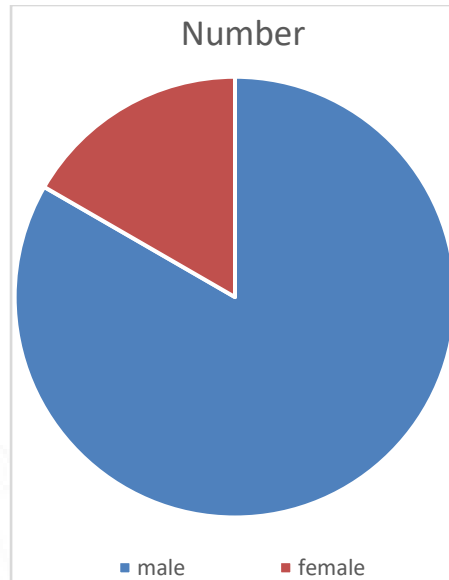


Figure 4: Pie diagram showing sex distribution of patients.

Clinical presentation :

Predominant symptom was headache (73%) followed by tinnitus (31%) and ocular/orbital symptoms (31 %) such as diplopia, proptosis, blurring of vision and ocular congestion, seizures (26% each) and memory disturbances (17%). Three patients (7%) presented with stroke like symptoms of hemiparesis and aphasia. Nine patients (22.5%) presented with intracranial hemorrhage and 31 patients (77.5%) had non hemorrhagic clinical presentations. Out of 40 patients, one patient was for follow up and retreatment of the residual fistula. Six patients (14%) had prior history of cerebral venous thrombosis and 1 patient had history of head trauma.

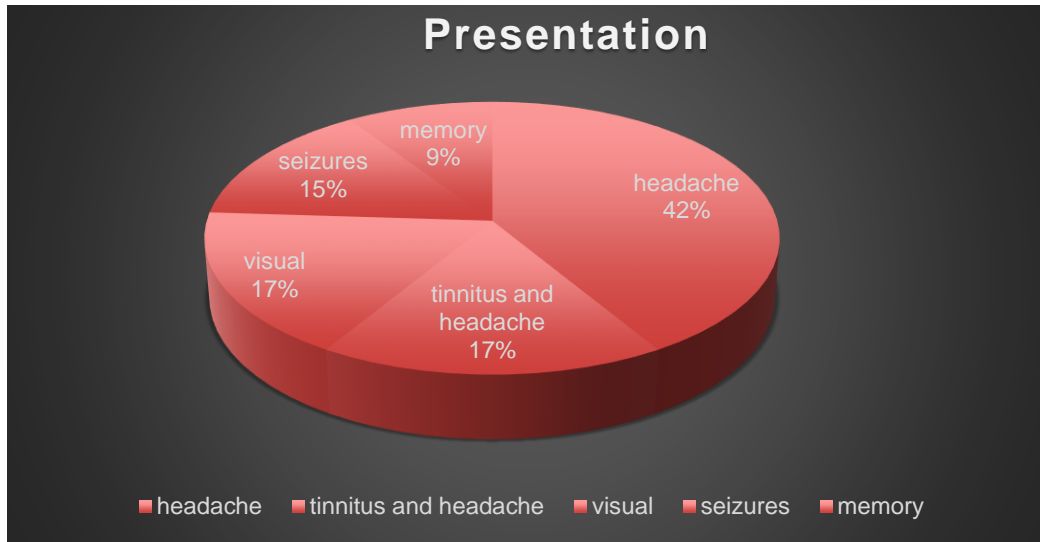


Figure 5: Pie diagram showing distribution of clinical presentation among the patients.

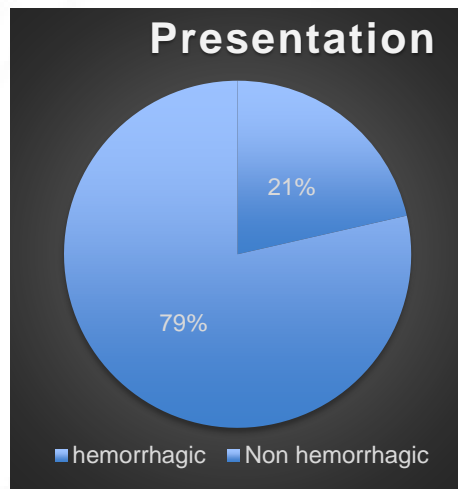


Figure 6: Pie diagram showing distribution of patients in two groups : Hemorrhagic and Non Hemorrhagic.

Angiographic characteristics:

Localization and grading of DAVF

Out of 40 patients, multisite fistulas were seen in 08 patients (19%). The frequency of the fistula location is as under: Transverse sinus- sigmoid sinus junction - 12 (23.6%), Transverse sinus – torcula - 8 (14.5%), superior sagittal sinus in 6 (11.3%), midline tentorial location in 5 (9%), 4 in

convexity (7.2%), 3 in jugular foramen(5.4%) & transverse sinus(5.4%), 2 each in torcula, sigmoid sinus- jugular bulb junction, sphenoid wing and hypoglossal canal(3.6%), 01 each in parasagittal, basifrontal regions, pterion and superior petrosal sinus locations (1.8%).

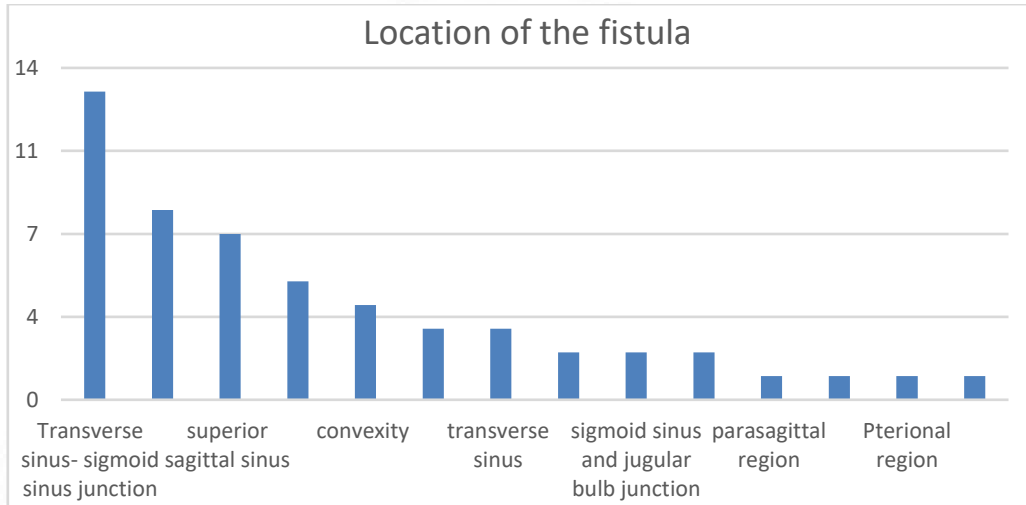


Figure 7: Frequency polygon showing location of fistulas

Grading of Fistula :

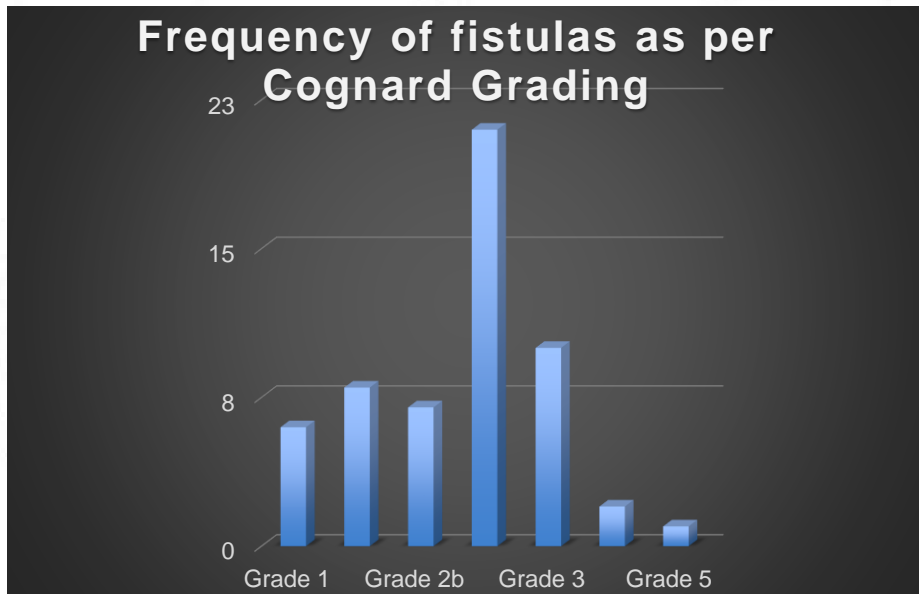


Figure 8: Frequency Polygon showing distribution of fistulas as per Cognard Grading

Silent and TOF MRA:

Both silent and TOF MRA showed 96.4% sensitivity and 100 % specificity when compared to DSA for identification of the site of fistula. For grading as per Cognard system, silent MRA showed 96% sensitivity and 100% specificity where as TOF MRA showed 94 % sensitivity and 100 % specificity. For intermodality agreement kappa value for fistula site and grading was 0.88. Both silent MRA and TOF MRA had 2 missed cases of convexity fistulas. The probable reasons for non-visualization of the fistulas in silent MRA were very small calibre of the arterial feeders supplying these convexity fistulas combined with probable loss of signals in convexity locations due to admixture of spins from normal venous drainage. The reasons for non-visualization of the fistulas and also failure to demonstrate cortical venous reflux in a grade 3 fistula in convexity region in TOF MRA could be due to saturation of flow related enhancement of the TOF signals in end planes combined with flow parallel to the imaging plane.

SWAN and ASL:

ASL: 53 fistulas were compared with DSA for site of the fistulas and for grading of the fistula as per Cognard Grading. We could not identify 4 fistulas on ASL which were located at convexity, transverse sinus and sigmoid sinus junction, medial tentorial location and hypoglossal canal respectively. The possible reasons could be due to saturation of signals at convexity location, loss of signal due to bone interface with susceptibility artefacts especially at the location of hypoglossal canal, part of the sinus being utilized for normal venous drainage at the transverse sinus, sigmoid sinus junction and medial tentorial location resulting in mixing of the spins leading to saturation of the signals at the site of these fistulas.

SWAN: 53 fistulas were compared with DSA for site of the fistulas and for grading of the fistula as per Cognard Grading. We could not identify 3 fistulas on SWAN on comparison with

DSA with two fistulas of convexity locations and another one at transverse sinus and sigmoid sinus junction. The possible reasons could be saturation of signals at convexity location and part of the sinus (right transverse sinus) being utilized for normal venous drainage at the transverse sinus resulting in mixing of the spins leading to saturation of the signals at the site of these fistulas.

Table 4 : Results of statistical analysis pertaining to ASL and SWAN.

Site & Grade of the fistula (Cognard and Borden)	ASL			SWAN		
	Value	95% CI		Value	95% CI	
Sensitivity	93%	83.00% 98.05%	to	95 %	84.88% 98.86%	to
Specificity	100.00%	2.50% 100.00%	to	100.00%	2.50% 100.00%	to
Negative Likelihood Ratio	0.07	0.03 to 0.18		0.05	0.02 to 0.16	
Positive Predictive Value	100.00%			100.00%		
Negative Predictive Value	99.94%	99.84% 99.98%	to	99.95%	99.85% 99.98%	to
Accuracy	99.94%			99.95%		

Arterial feeders

Silent MRA was able to detect 258 of 296 (sensitivity of **87%** (83 to 93% - 95% CI)) total arterial feeders to the fistulous site with an accuracy of 78%. In 8 patients, all the feeders to the fistula were identifiable. The silent MRA showed good correlation with DSA findings ($\rho = 0.94$ and $p < 0.001$). 12 % of smaller feeders were not detected by silent MRA either due to the smaller caliber of these feeders, their location in the convexities or due to poor image quality. With a cut off 1.5 mm diameter, 96.4% of the prominent arterial feeders (162 out of 168 feeders) were correctly identified by silent MRA, while 6 feeders could not be identified as they were misinterpreted as veins or non-dominant feeders. The sensitivity of TOF MRA for all arterial feeders was 79% (73.6 to 83.2% 95% CI) with an accuracy of 70%. TOF MRA could detect 233 of 296 feeders supplying the fistulous sites. In case of prominent arterial feeders, TOF MRA was able to detect 149 of 168 feeders with a sensitivity of 89%. 19 prominent arterial feeders were missed either due to loss of signal due to phase dispersion or due to inadequate coverage due to axial acquisition. For the purpose of calculating specificity of the MR angiographic sequences we had taken 16 commonly encountered arterial feeding trunks namely bilateral middle meningeal trunk, superficial temporal trunk, occipital trunk, posterior auricular, ascending pharyngeal, vertebral, dural trunks from ICA and ophthalmic arterial trunks. We assessed silent MRA, TOF MRA and DSA for feeders from these arterial trunks to the fistulas. Silent MRA showed 90 % sensitivity (85.5 to 93.6% -95% CI) and 99 % specificity (98.4 to 99.9% -95% CI) and 99 % negative predictive value; TOF MRA showed 76 % (69.8 to 81.2% - 95% CI) sensitivity and 99 % specificity (98.8 to 99.9% - 95% CI) and 99 % negative predictive value. The TOF MRA showed significant correlation with DSA findings ($\rho = 0.901$ and $p < 0.001$). McNemar test showed 4.54% difference between the two MRA sequences, which was statistically

significant (p value <0.0001). The kappa value for intermodality agreement for the arterial feeders are 0.91 and 0.87 for silent and TOF MRA respectively.

Table 5: Sensitivity, Specificity, Positive & Negative predictive value and Accuracy of Silent MRA

Parameter	Arterial feeder	Prominent Arterial feeder	Accessible feeder	Immediate Venous drainage	Overall venous drainage
Sensitivity	87%	96 %	96 %	92.6%	81.6%
Specificity	-	99.5%	-	-	-
Negative predictive value	-	99.1%	-	--	-
Positive Predictive Value	78 %	87%	86%	85%	73%
Accuracy	78 %	96%	96 %	92%	80%

Venous drainage

Silent MRA was able to detect 212 out of 259 draining veins from these fistulas with a sensitivity of 81.6% (76.6 % to 88% - 95% CI), positive predictive value of 73% in all 40 patients whereas TOF MRA could identify 179 out of 259 draining veins in 40 patients with a sensitivity of 67% (61.5 to 73.2% 95% CI) and positive predictive value of 60%. However, the sensitivity improved if only venous drainage which is adjacent to the fistula was taken into

consideration. The silent MRA identified 150 out of 162 immediate draining veins/sinuses with 92.6 % sensitivity (95% CI of 87.4 to 96.1%) where as TOF MRA showed could identify only 139 out of 162 immediate draining veins/sinuses with a sensitivity of 85.8% and 95% CI of 79.4 to 90.8%. The correlation coefficient for immediate venous drainage (r) was 0.92 for silent MRA and 0.87 for TOF MRA. The main reasons for reduced accuracy of immediate venous drainage in silent MRA was low quality of images (92%) along with utilization of veins for normal venous drainage (6%), and misdiagnosis as arteries (2%). The reasons of reduced accuracy in TOF MRA were poor quality (90%) apart from dispersion of flow related enhancement in extremes of slice selection plane especially for the fistulas located in convexity, superior sagittal sinus and jugular foramen/hypoglossal canal. McNemar test showed superiority of Silent MRA over TOF MRA with a paired proportions difference of 9.93% difference (p value of <0.0001).

The kappa value for intermodality agreement for the venous drainage were 0.88 and 0.84 for Silent and TOF MRA respectively.

Endovascular treatment prediction

Conservative management was considered for 14 patients (36%), whereas 26 patients (64%) were managed by endovascular treatment through transarterial embolization. In 25 out of 26 (96.1%) patients, the predicted endovascular therapeutic approach by silent MRA accurately correlated with the feeder identified by DSA whereas TOF MRA showed 85% accuracy in predicting the possible endovascular approach (22 of 26 patients). 88% of the accessed feeders (22 of 26) were middle meningeal artery branches and rest of the accessible feeders were from occipital artery (3 of 26), and ascending pharyngeal artery (1 of 26) Silent MRA could not predict the accessible feeder in one patient in which DSA revealed a small caliber MMA feeder to dAVF. TOF MRA could not predict the accessible feeder in 3 patients as these feeders were

not accurately identified due to loss of flow related enhancement probably caused by signal dissipation.

Image quality:

The quality of images was assessed in both silent as well as TOF MRA for prominent arterial feeders and immediate draining veins. The median score was 3 for both the sequences. For silent MRA with regard to arterial feeders, image quality did not significantly affect the interpretability (p value 0.118) whereas for TOF MRA image quality significantly affected the interpretability with p value of <0.00001. For immediate venous drainage, in silent MRA 92% (p value of 0.014) of the missed veins were due to poor image quality with a score of 2 or 1 whereas in TOF MRA 90% (p value of 0.001) of the missed veins had a score of 2 or 1.

Table 6: Sensitivity, specificity, Positive & Negative predictive value and Accuracy of TOF MRA

Parameter	Arterial feeder	Prominent Arterial feeder	Accessible feeder	Immediate Venous drainage	Overall venous drainage
Sensitivity	79%	89 %	85 %	86 %	67 %
Specificity	-	99.8%	-	-	-
Negative predictive value	-	99.7%	-	--	-
Positive Predictive Value	70 %	75 %	79%	82%	60%
Accuracy	70 %	89%	85 %	85%	68%

Table 7: Intermodality agreement Kappa value

Parameter	Site of the fistula	Grading of the fistula	Overall Arterial feeder	Prominent Arterial feeder	Accessible feeder	Immediate Venous drainage	Overall venous drainage
Silent MRA	0.88	0.88	0.78	0.91	0.92	0.88	0.54
TOF MRA	0.88	0.88	0.6	0.87	0.88	0.84	0.51

Table 8: Analysis of silent and TOF MRA of dural intracranial arteriovenous fistula with cerebral angiography – Location

Site of the fistula	Silent MRA	TOF MRA	DSA
Site of fistula	96%	96%	
Transverse sinus and sigmoid sinus junction	12	12	12
Midline tentorium	5	5	5
Superior sagittal sinus Convexity	6	6	6
Torcula	2	2	4
TS – torcula junction	2	2	2
Superior petrosal sinus	8	8	8
Jugular foramen	1	1	1
Sigmoid sinus – jugular bulb	3	3	3
Sphenoid wing	2	2	2
Pterion	2	2	2
Transverse sinus	1	1	1
Hypoglossal canal	3	3	3
Parasagittal region	2	2	2
Basifrontal region	1	1	1

Table 9: Analysis of Silent and TOF MRA – Cognard classification

Classification Cognard	Silent MRA	TOF MRA	DSA
1	5	5	5
2a	8	9	8
2b	7	6	7
2a+b	20	20	20
3	8	8	10
4	2	2	2
5	1	1	1

Table 10: Analysis of Silent and TOF MRA – Borden classification

Classification Borden	Silent MRA	TOF MRA	DSA
1	13	14	13
2	27	26	27
3	11	11	13

Table 11: Analysis of Silent and TOF MRA – Arterial feeders and Venous drainage

Characteristic	Silent MRA	TOF MRA	DSA
Arterial feeders			
Arterial feeders >1.5mm	162(96.4%)	149(89%)	168
Overall arterial feeders	258(87.2%)	233(79%)	296
Venous drainage			
Immediate venous drainage	150(92%)	139(86%)	162
Overall venous drainage	212(82%)	179(69%)	259

Table 12: Analysis of Silent and TOF MRA – Therapeutic decision

Characteristic	Silent MRA	TOF MRA	DSA
Therapeutic decision			
Conservative	14	14	14
Endovascular Management	26	26	26
Endovascular access route			
Transarterial			26
Potential Accessible feeder	25(96%)	22(85%)	26
Middle meningeal artery	21	18	22
Occipital artery	3	3	3
Ascending pharyngeal artery	1	1	1

DISCUSSION



DISCUSSION

DAVF is relatively uncommon neurovascular malformation constituting about 10 to 15% of all intracranial AV malformations. With the advent of various advanced MRI sequences early diagnosis is possible in these patients. DSA is the gold standard in diagnosis of the disease as well as for evaluation. The recurrence chance of fistula is upto 14 % even after endovascular management and hence follow up is necessary, for which routinely angiography is indicated. (40). DSA however is a minimally invasive procedure that involves radiation concerns as well as significant procedural complications, albeit low. Thus, a noninvasive imaging modality that is equivalent to DSA in performance could be advantageous for fistula work up as well as follow up of these patients. predicting the recurrence of fistula.

In the present study, the predominant type of dAVF was Cognard aggressive types (2b-5) was seen in 33 patients (80%) of which Cognard type 2a+b was seen in 20 patients. 9 patients presented with ICH and features of raised ICP. The benign types (7 patients) (Cognard 1-2a) presented with either headache, symptoms of blurred vision or tinnitus. The male: female ratio was 5:1. These findings are in contradiction with the studies of Baltasvias et al and Oh et al. Baltasvias et al study had 67% benign presentation and 33% aggressive presentation in their cohort of 116 patients (91). Similarly, Oh et al too observed aggressive clinical presentation in 30% of their cases (92). The reason may be that since our institution is a tertiary care centre and most of the aggressive cases were selectively referred for endovascular management. The clinical presentation, natural history and prevalence of the disease corroborated with various studies. (6), (19). The predominant location of the fistula was transverse sinus- sigmoid sinus junction (12 fistulas) (31%) followed by transverse sinus (10 fistulas) (24%) in our study. Piippo et al had 67% of cases predominantly located in the transverse and sigmoid sinus regions in their

cohort of 227 patients (93). Baltasvias et al study had 22% of cases, in transverse sinus region excluding cavernous sinus with another 18% of cases seen in the transverse- sigmoid sinus region. (85). About three quarters of the fistulas were of Borden grade 2 or grade 3 in our study.

Fistulous site and Grading: Role of Advanced MRI sequences

In identification of fistulous point, all the sequences, including, ASL, SWAN, Silent MRA and TOF MRA performed well with excellent intermodality and interrater agreement. All these sequences are highly sensitive and specific for identification of the site of fistula. Silent MRA showed 96% sensitivity, TOF MRA showed 96 % sensitivity, ASL showed 93% sensitivity and SWAN showed 95 % sensitivity in predicting the site of fistula. All these sequences showed 100% specificity. For multisite dAVF, both silent and TOF MRA could identify 88 % of multisite fistulas whereas ASL and SWAN could identify 75% of fistulas. Silent and TOF MRA could not identify two fistulas located in the convexity as these fistulas were supplied by small calibre feeders coupled with spin saturation effect. ASL missed two convexity fistulas in addition to one fistula in transverse sinus and sigmoid sinus junction and medial tentorial region. SWAN missed two convexity fistulas in addition to one fistula in transverse sinus and sigmoid sinus junction and hypoglossal canal region. The ASL and SWAN sequences show limitation in identification of fistulous points in the convexity as well as near the bony interface in the skull base region. There is a limitation in identification of the multiple fistulas in both ASL and SWAN due to the variability in the flow pattern and grading of the fistulas.

Jain et al study showed that SWI could identify 75% of the fistulous points accurately with a sensitivity of 85%. This study also showed that SWI has its limitations in identification of the multiple fistulas as well as fistulas close to bony interface due to susceptibility artefacts. (9)

All the four sequences showed excellent sensitivity and specificity in predicting the grade of the fistula as per both Cognard and Borden systems of classification with sensitivities ranging from 92 to 96%. Identification of CVR is possible in most of the patients by all four sequences. In few cases where the cortical venous reflux was missed or the fistula could not be identified, the predominant reason being saturation of signals near the vertex as well as inadequate coverage by TOF MRA. Hence, in this aspect of inadequate coverage, silent MRA scores over TOF MRA, where in the acquisition is 3D coronal acquisition with ample FOV and with reduced acquisition time for similar FOV as compared to TOF MRA. This negates the aspect of inadequate coverage in TOF MRA.

Arterial spin labelling perfusion utilizes magnetically labelled protons for identification of identify vascular structures and in DAVF, as there is no intermediate parenchyma, the labelled protons show rapid venous shunting, thus appearing as hyperintense signals in venous structures. Thus, this principle was utilized by ASL sequence in detection of dAVF with presence of cortical venous reflex. ASL showed a high sensitivity of 93.6% and a specificity of 87.6 % in diagnosis of DAVF with cortical venous reflux. However, ASL perfusion technique was not helpful in detailed evaluation of other important variables like arterial feeders and venous drainage patterns. ASL showed >90% sensitivity and specificity in identifying CVR with interobserver Kappa value >0.8. The interobserver Kappa value in identifying the Borden grade was also noted to be >0.8. Hence it was proposed that ASL is of considerable use in diagnosis and grading of intracranial dAVF (94).

Noguchi et al demonstrated that ASL could identify the cortical venous hyperintense signal in all Borden III cases whereas it was identified in only 25% of Borden II cases (74). However, in our study ASL showed cortical venous signal in both Borden II and III cases with good sensitivity.

This is in agreement with Hodel et al study which showed a sensitivity of 98%, specificity of 97% and a high kappa value of 0.9 in a cohort of 63 shunting patients with 10 dAVF cases. (67)

Pseudophlebitic pattern

Pseudophlebitic pattern is the presence of dilated tortuous veins in the venous phase of angiography. These dilated veins are the result of chronic venous congestion and presence of this venous pattern is an indicator of aggressive nature of the fistula with a propensity for hemorrhage. (44,95)

Pseudophlebitic pattern (PPP) was identified in 22 patients (52%). There was 100% accuracy with excellent sensitivity and specificity in detecting the same by SWAN in comparison with DSA with excellent correlation. Our study also demonstrated this pattern in around 52% of patients, of which 41% of patients presented with aggressive features and this pattern was recognized by SWAN accurately. 09 patients (41%) with pseudophlebitic pattern presented with hemorrhage and aggressive clinical features. Willinsky et al had concluded that 42% of their cases had pseudophlebitic pattern in their cohort of 122 patients and around 73% of these patients showed aggressive clinical presentation as compared to the ones without PPP. Hence, the study concluded that this particular angiographic pattern was a prognostic indicator and helpful in arriving at a therapeutic decision of early endovascular intervention (44). In a recent study by Brinjikji et al, SWI was 47% sensitive in identifying the dilated transmedullary veins in patients with proven pseudophlebitic pattern on catheter angiography. (95)

Arterial feeders evaluation by Silent and TOF MRA

Both silent and TOF MRA were highly sensitive in the angioarchitectural evaluation of arterial feeders. These findings were correlated well with the DSA with good intermodality and interrater agreement. In the analysis of arterial feeders, silent MRA was relatively more sensitive than

TOF MRA (87% vs 79%) in identification of the arterial feeders. The sensitivity improved considerably when only prominent feeders were considered and also the difference in sensitivity between silent and TOF MRA also reduced (96% Vs 89%). Williams et al, using CTA, showed 91.1% accuracy in identification and classification of grading of dAVF. Prominent hypertrophied feeding arteries were identified in that study, while smaller calibre arteries and pial branches from ICA or VA were not identified. Also, venous drainage pattern was not studied, which is very essential in management decision and prognostication. Dynamic CT angiography incurs high radiation dose especially with regards to the repeated follow up of these patients. Hence temporal resolution provided by the dynamic CT does not provide significant advantage over the higher radiation dose incurred by the patients on follow up. (49)

Over the years many non-contrast as well as contrast enhanced MRA(CE-MRA) sequences have been extensively studied for evaluation of dAVF. In the study by Nishimura et al 4D time resolved CE-MRA showed 89% sensitivity in identification of main arterial feeders with moderate interrater agreement. However, detailed evaluation of the arteries or the venous drainage was not done in that study (56). Ertl et al, in their study utilizing CE MRA, found that CEMRA was 100% accurate in detection of dAVF and also in depiction of cortical veins. However, this study evaluated only the major feeders with moderate intermodality agreement whereas the draining veins were not submitted for detailed evaluation. The study also emphasized the role of CEMRA in planning of therapeutic decision in 95.4% of cases, though the route prediction for endovascular treatment was moderate (59%). (57)

In one of the recent study by Dissaux et al, 4D CE MRA has shown to have moderate sensitivity (63.6%) for detection of residual/recurrent fistulas in evaluation of follow up cases along with good intermodality agreement with DSA. (58).

Due to inherent risk of gadolinium deposition in repeated CEMRA studies for follow up studies, utility of other non-contrast MR angiographic techniques were studied for the evaluation of dAVF. In a study by Azuma et al, TOF MRA showed good intermodality agreement for site of fistula (96%), identification of prominent arterial feeders (88%) and classification of fistula by Borden method (88%) alongwith high interrater agreement. (8)

Flow sensitive alternating inversion recovery technique(FAIR), utilizing ASL principle was able to provide temporal hemodynamic information akin to DSA and this sequence showed 100% agreement with DSA for site of fistula and only 78% and 89% interobserver agreement for main feeder identification and venous drainage (51).The evaluation of arterial feeders was largely confined to major larger arteries only and the performance of these sequences for DSA like angioarchitecture evaluation of dAVF is largely unknown.

Silent MRA is a novel MRA sequence based on ASL, and it has been studied in the evaluation of arteriovenous malformations, or intracranial aneurysms. (11,81,96) As the TE in this sequence is ultra-short to the tune of 0.016 msec, there is negligible effect by susceptibility effects. This property was utilized in the evaluation of follow up of intracranial aneurysms treated by Stent assisted coiling and flow diverters. As it is a ASL based technique, rapid shunting occurring in dAVF enables the shunt site and early draining veins to appear as hyperintense structures as the magnetically labelled protons in blood vessels act a as a macrovascular tracer. This helps in accurate identification of the site of fistula and draining veins. (71)

In this study, the prominent arterial feeders were defined by an arbitrary cut off of 1.5 mm. This classification of main/prominent arterial feeders was not attempted in any of the prior studies. (49,51,56,57). The main reasons for missing of the prominent arterial feeders in the present study are mistaken identity of the artery as venous structure, as it was retrospectively found that these

arteries could be identified by thorough and detailed analysis for feeders, specific to the location of the fistula. Retracing of these structures to the main arterial branch confirmed the arterial origin. This will improve the accuracy of the scan for overall arterial feeder detection. However, the sensitivity of silent MRA was 87% for overall arterial feeders, which was mainly due to non-visualization of smaller calibre arterial feeders (23%). Other factors being, relatively poor spatial resolution, poor signals contributed by the relatively lower spin density in normal calibre feeders, suboptimal image quality. None of the previous studies have gone into the detailed evaluation of smaller calibre arterial feeders or pial feeders from ICA or VA. (8,49,57)

TOF MRA showed relatively lower sensitivity both for the prominent arterial feeders and overall arterial feeders (79% & 89%). The performance of the TOF MRA in depiction of these prominent feeders in comparison to silent MRA is non inferior as proved by Binary test. The reasons for the reduced sensitivity of TOF could be either due to the non-visualization of small arterial feeders or loss of flow related enhancement due to spin saturation in the extremes of the imaging plane in TOF MRA. Since TOF is an axial acquisition, inadequate coverage would have also contributed in non-identification of the feeders. As compared to TOF, silent MRA is a sagittal acquisition with wider field of view and coverage without the disadvantage of spin saturation in extremes of imaging plane. Other contributing factors for the non-visualization of feeders could be low spatial resolution, suboptimal image quality affecting optimal visualization, misrepresentation of small arteries as venous structures. Previous studies of MRA in dAVF have not provided any comparative analysis of these sequences. (8,51)

Analysis of Venous Drainage by Silent and TOF MRA

In the analysis of venous drainage, Silent MRA had better sensitivity than TOF MRA (82 % vs 69%) which was significant by McNemar test. However, the sensitivity improved with reduction

in the difference when immediate venous drainage was taken into consideration (92 % vs 86%). The image quality of the sequences for the analysis of arterial feeders and venous drainage was good. However, poor image quality was a decisive factor in the venous drainage analysis.

The distinction of immediate draining veins, which were located in the vicinity of shunt site and overall draining veins, was due to the variable re-routing pattern of the veins with difficulty in accurate evaluation of these veins. Prior studies have not reported any detailed analysis of the draining veins. (51,56,57)

Silent MRA had high sensitivity of 92 % for identifying the immediate venous drainage with accurate identification of the cortical veins in all the aggressive cases. This helps in venous mapping, as well as on the therapeutic decision making due to the accurate classification of the fistula. For overall drainage of veins, the sensitivity of silent MRA was only 82%, whereas for the TOF MRA it was only 69%. The main factor in the poor venous drainage depiction for silent MRA is the image quality apart from other contributing factors like thrombosis of veins, stagnant flow in the draining sinus, mistaken identity of the veins as arteries and vice versa. Non tagged spins from the normal venous drainage also affect the draining vein visualization by getting mixed up with the tagged spins with reduction in overall signal intensity. The relatively poor sensitivity for depiction of overall draining veins in TOF MRA is mainly due to the suboptimal image quality (90%). The orientation of the vessels to the imaging plane is also a decisive factor in determining the saturation of spins in the vessels which are being imaged. Horizontally oriented vessels and tortuous vessels have shown to be affected by the flow related saturation effects in TOF MRA thus contributing to the poorer depiction of veins by TOF MRA.

Assessment of accessible feeder and Therapeutic decision

Both silent and TOF MRA contributed equally in planning therapeutic intervention and identifying the potential arterial feeder that could be used for transarterial endovascular treatment. There was an excellent agreement with the endovascular intervention. However, silent MRA performed marginally better than TOF MRA (96% vs 85 %) in prediction of the accessible feeder which correlated with actual choice of endovascular intervention. The accessible feeder was determined by relatively straighter, larger, easily accessible course of the vessel to the fistulous site. As compared to previous studies, silent MRA was highly sensitive (96%) (25 of 26 cases) and accurate in prediction of the accessible feeder. TOF MRA also did not significantly lag behind silent MRA in prediction of the therapeutic approach. We could accurately predict showed 22 out of 26 feeders in TOF MRA (85%).

With respect to treatment decision, similar to the other imaging methods, it was possible to devise a therapeutic strategy using silent MRA, which had excellent concordance with DSA observations. (49,57). In CE-MRA, the identification of accessible feeder was accurate in only 60% of the cases (57). In our study, identification of accessible feeder was possible in 96% of cases in silent MRA and 85% in TOF MRA. Excellent suppression of background and increased contrast resolution of silent MRA along with the experience of the investigator were one of the decisive factors for the accuracy of silent and TOF MRA in prediction of therapeutic decision.

Strengths of the study

The main strengths of the study are

1. Uniform protocol of the study
2. Relatively larger cohort of patients for analysis using a combination of non-contrast MR sequences for evaluation of intracranial dAVF
3. Detailed analysis of various angioarchitectural aspects of dAVF, that were clearly defined.
4. Additionally, this study has also assessed the utility of observation from therapeutic perspective.

Limitations of the study

Few limitations of the study are

1. Presence of inherent bias in selection of patients
2. Lack of analysis on utilization of these sequences in the follow up of these patients
3. Smaller sample size
4. Cohort of patients were not entirely prospective.



CONCLUSION



CONCLUSION

This study is one of the largest studies to evaluate intracranial dAVF, analyzing the utility of non-contrast MRA angiographic sequences. It has demonstrated that all the four sequences namely silent MRA, TOF MRA, ASL and SWAN are excellent in diagnosis as well as in grading of the intracranial dAVF with 92 to 96% sensitivity and 100% specificity. Silent MRA has an advantage over TOF MRA in the diagnostic accuracy of arterial feeders and venous drainage with relatively higher sensitivity even though TOF MRA was not found to be inferior by statistical analysis. The accurate depiction of vessels in extreme ends of imaging plane and parallel to imaging plane has shown to be advantageous for silent MRA over TOF MRA in prediction of the therapeutic decision. Non-invasive sequence has significant accuracy in the evaluation of various angioarchitectural aspects, and hence could be routinely employed for dAVF evaluation. Silent MRA could be used a single sequence that optimally evaluates all the components. Role of these sequences in follow up of the treated patients need to be evaluated and its ability to supplant DSA needs to be studied. Future studies addressing these concerns are warranted.



REFERENCES



REFERENCES

1. Newton TH, Cronqvist S. Involvement of dural arteries in intracranial arteriovenous malformations. *Radiology* [Internet]. 1969;93(5):1071–8.
2. Gandhi D, Chen J, Pearl M, Huang J, Gemmete JJ, Kathuria S. Intracranial dural arteriovenous fistulas: classification, imaging findings, and treatment. *American J Neuroradiol*. 2012;33(6):1007–13.
3. Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, et al. Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. *Radiology*. 1995;194:671–680.
4. Reynolds M, Lanzino G, Zipfel G. Intracranial Dural Arteriovenous Fistulae. *Stroke*. 2017;48:1424-1431. DOI: 10.1161/STROKEAHA.116.012784.
5. Awad IA, Little JR, Akarawi WP, Ahl J. Intracranial dural arteriovenous malformations: factors predisposing to an aggressive neurological course. *J Neurosurg*. 1990;72(6):839–50.
6. Gross BA, Du R. The natural history of cerebral dural arteriovenous fistulae. *Neurosurgery*. 2012;71(3):594–602.
7. Alexander M, McTaggart R, Santarelli J, Fischbein N, Marks M, Zaharchuk G, et al. Multimodality evaluation of dural arteriovenous fistula with CT angiography, MR with arterial spin labeling, and digital subtraction angiography: Case Report. *J Neuroimaging*. 2014;24(5):520–3.
8. Azuma M, Hirai T, Shigematsu Y, Kitajima M, Kai Y, Yano S et al (2015) Evaluation of intracranial dural arteriovenous fistulas: comparison of unenhanced 3T 3D time-of-flight

- MR angiography with digital subtraction angiography. *Magn Reson Med Sci* 14:285–293.
9. Jain NK, Kannath SK, Kapilamoorthy TR, Thomas B. The application of susceptibility-weighted MRI in pre-interventional evaluation of intracranial dural arteriovenous fistulas. *Journal of NeuroInterventional Surgery*. 2017 May;9(5):502–7.
 10. Exhibit E, Suzuki M, Irie R, Takano N, Hori M, Kamagata K, et al. Basics and clinical application of Ultra short TE MRA combined with Arterial Spin Labeling algorithm – Silent MRA -. 2016;1–16.
 11. Arai, N., Akiyama, T., Fujiwara, K. et al. Silent MRA: arterial spin labeling magnetic resonant angiography with ultra-short time echo assessing cerebral arteriovenous malformation. *Neuroradiology* 62, 455–461 (2020).
 12. Ryu, Kyeong & Baek, Hye & Moon, Jin & Choi, Bo & Park, Sung & Ha, Ji & Park, Hyun & Kim, Seung & Kim, Jung-Soo & Cho, Soo & Carl, Michael. (2019). Usefulness of Noncontrast-Enhanced Silent Magnetic Resonance Angiography (MRA) for Treated Intracranial Aneurysm Follow-up in Comparison with Time-of-Flight MRA. *Neurosurgery*. 10.1093/neuros/nyz421.
 13. Brown Jr RD, Wiebers DO, Torner JC et al. (1996). Incidence and prevalence of intracranial vascular malformations in Olmsted County, Minnesota, 1965 to 1992. *Neurology* 46:949–952.
 14. Satomi J, Satoh K (2008). Epidemiology and etiology of dural arteriovenous fistula. *Brain Nerve* 60: 883–886.
 15. Chaichana KL, Coon AL, Tamargo RJ et al. (2012). Dural arteriovenous fistulas: epidemiology and clinical presentation. *Neurosurg Clin N Am* 23: 7–13.

16. Elhammady MS, Ambekar S, Heros RC. Epidemiology, clinical presentation, diagnostic evaluation, and prognosis of cerebral dural arteriovenous fistulas. *Handb Clin Neurol*. 2017;143:99-105. doi: 10.1016/B978-0-444-63640-9.00009-6.
17. Sarma D, ter Brugge K (2003). Management of intracranial dural arteriovenous shunts in adults. *Eur J Radiol* 46:206–220.
18. Lasjaunias P. Angioarchitecture and natural history of dural arteriovenous shunts. *Intervent Neuroradiol* 1997;3:313-7.
19. Davies MA, Saleh J, ter Brugge KG, Willinsky R, Wallace MC. The natural history and management of intracranial dural arteriovenous fistulae. Part I: benign lesions. *Intervent Neuroradiol* 1997;3:295 /302.
20. Davies MA, ter Brugge KG, Willinsky R, Wallace MC. The natural history and management of intracranial dural arteriovenous fistulae. Part II: aggressive lesions. *Intervent Neuroradiol* 1997;3:303 /11.
21. VanDijk M, ter Brugge K, Willinsky RA, Wallace C. The clinical course of cranial dural AV-fistulas with long-term persistent cortical venous reflux. *Stroke* 2002;33(5):1233-6.
22. Dipanka Sarma, Karel ter Brugge, Management of intracranial dural arteriovenous shunts in adults, *European Journal of Radiology*, Volume 46, Issue 3, 2003, Pages 206-220.
23. Uranishi R, Nakase H, Sakaki T (1999). Expression of angiogenic growth factors in dural arteriovenous fistula. *J Neurosurg* 91: 781–786.
24. S Miyachi E, Izumi T, Matsubara N, Naito T, Haraguchi K, Wakabayashi T. Mechanism of the formation of dural arteriovenous fistula: the role of the emissary vein. *Interv Neuroradiol*. 2011;17(2):195–202.

25. Nagm A, Horiuchi T, Kanaya K, Hongo K. Dural Arteriovenous Fistula Could Be Due to Hemodynamic Disturbance in Dural Physiological Shunts? Histopathological Study and a Case Report. *World Neurosurg* [Internet]. 2016;90:699.E11-699.E18.
26. Hayes GJ. External carotid-cavernous sinus fistulas. *J Neurosurg*. 1963 Aug;20:692-700. doi: 10.3171/jns.1963.20.8.0692.
27. Castaigne P, Bories J, Brunet P et al. (1976). Meningeal arterio-venous fistulas with cortical venous drainage. *Rev Neurol (Paris)* 132: 169–181.
28. Lalwani AK, Dowd CF, Halbach VV (1993) Grading venous restrictive disease in patients with dural arteriovenous fistulas of the transverse/sigmoid sinus. *J Neurosurg* 79:11–15.
29. Baltasvias G, Roth P, Valavanis A. Cranial dural arteriovenous shunts. Part 3. Classification based on the leptomenigeal venous drainage. *Neurosurg Rev*. 2015 Apr;38(2):273-81; discussion 281.
30. Kim MS, Han DH, Kwon OK, et al. Clinical characteristics of dural arteriovenous fistula. *J Clin Neurosci* 2002;9(2):147–55.
31. Hasumi T, Fukushima T, Haisa T, et al. Focal dural arteriovenous fistula (DAVF) presenting with progressive cognitive impairment including amnesia and alexia. *Intern Med* 2007;46(16):1317–20.
32. Hirono N, Yamadori A, Komiyama M. Dural arteriovenous fistula: a cause of hypoperfusion-induced intellectual impairment. *Eur Neurol* 1993;33(1):5–8. & Lee PH, Lee JS, Shin DH, et al. Parkinsonism as an initial manifestation of dural arteriovenous fistula. *Eur J Neurol* 2005;12(5):403–6.

33. Hernesniemi JA, Dashti R, Juvela S, Väärt K, Niemelä M, Laakso A. Natural history of brain arteriovenous malformations: A long-term follow-up study of risk of hemorrhage in 238 patients. *Neurosurgery*. 2008;63(5):823–9.
34. Jolink WMT, van Dijk JMC, van Asch CJJ, de Kort GAP, Algra A, Groen RJM, et al. Outcome after intracranial haemorrhage from dural arteriovenous fistulae; a systematic review and case-series. *J Neurol*. 2015;262(12):2678–83.
35. Satomi J, Ghaibeh AA, Moriguchi H, Nagahiro S. Predictability of the future development of aggressive behavior of cranial dural arteriovenous fistulas based on decision tree analysis. 2015;123(July):86–90.
36. Brown RD, Wiebers DO, Nichols DA. Intracranial dural arteriovenous fistulae: angiographic predictors of intracranial hemorrhage and clinical outcome in nonsurgical patients. Vol. 81, *Journal of neurosurgery*. 1994. p. 531–8.
37. Soderman M, Pavic L, Edner G, Holmin S, Andersson T. Natural history of dural arteriovenous shunts. *Stroke*. 2008;39(6):1735–933.
38. Bulters DO, Mathad N, Culliford D, Millar J, Sparrow OC. The natural history of cranial dural arteriovenous fistulae with cortical venous reflux - The significance of venous ectasia. *Neurosurgery*. 2011;70(2):312–9.
39. Shah MN, Botros JA, Pilgram TK, Moran CJ, Cross DT 3rd, Chicoine MR, et al. Borden-Shucart Type I dural arteriovenous fistulas: clinical course including risk of conversion to higher-grade fistulas. *J Neurosurg*. 2012;117:539–545. doi: 10.3171/2012.5.JNS111257.

40. Ambekar S, Gaynor BG, Peterson EC, Elhammady MS. Long-term angiographic results of endovascularly “cured” intracranial dural arteriovenous fistulas. *J Neurosurg* [Internet]. 2015;1–5.
41. Chung Y, Choi SK, Lee SH, Kim EJ. Case Report Spontaneous Aggressive Conversion of Venous Drainage Pattern in Dural Arteriovenous Fistula Treated with Onyx Embolization. 2016;396–401.
42. Serulle Y, Miller TR, Gandhi D. Dural Arteriovenous Fistulae: Imaging and Management. *Neuroimaging Clin N Am* [Internet]. 2016;26(2):247–58.
43. Van Rooij WJ, Sluzewski M, Beute GN. Intracranial dural fistulas with exclusive perimedullary drainage: the need for complete cerebral angiography for diagnosis and treatment planning. *AJNR Am J Neuroradiol*. 2007;28:348–351.
44. Willinsky RA, Goyal M, TerBrugge K, Montanera W. Tortuous, engorged pial veins in intracranial dural arteriovenous fistulas: Correlations with presentation, location, and MR findings in 122 patients. *Am J Neuroradiol*. 1999;20(6):1031–6.
45. Kaufmann TJ, Iii JH, Mandrekar JN, Schleck CD, Thielen KR, Kallmes DF. Complications of Diagnostic Cerebral Angiography : Evaluation Methods : Results : Conclusion : 2007;243(3).
46. Lin Y-H, Lin H-H, Liu H-M, Lee C-W, Chen Y-F. Diagnostic performance of CT and MRI on the detection of symptomatic intracranial dural arteriovenous fistula: a meta-analysis with indirect comparison. *Neuroradiology* [Internet]. 2016;753–63.

47. Meckel S, Lovblad KO, Abdo G, Ruiz DSM, Delavelle J, Radue EW, et al. Arterialization of cerebral veins on dynamic MDCT angiography: A possible sign of a dural arteriovenous fistula. *Am J Roentgenol*. 2005;184(4):1313–6
48. Beijer TR, Van Dijk EJ, De Vries J, Vermeer SE, Prokop M, Meijer FJA. 4D-CT angiography differentiating arteriovenous fistula subtypes. *Clin Neurol Neurosurg* [Internet]. 2013;115(8):1313–6. 45.
49. Willems PW, Brouwer PA, Barfett JJ, et al. Detection and classification of cranial dural arteriovenous fistulas using 4D-CT angiography: initial experience. *AJNR Am J Neuroradiol* 2011;32(1):49-53. DOI:10.3174/ajnr.A2248.
50. Dietz RR, Davis WL, Harnsberger HR, Jacobs JM, Blatter DD. MR imaging and MR angiography in the evaluation of pulsatile tinnitus. *AJNR Am J Neuroradiol*. 1994 May;15(5):879-89.
51. Iryo Y, Hirai T, Kai Y, Nakamura M, Shigematsu Y, Kitajima M, Azuma M, Komi M, Morita K, Yamashita Y. Intracranial dural arteriovenous fistulas: evaluation with 3-T four-dimensional MR angiography using arterial spin labeling. *Radiology*. 2014 Apr;271(1):193-9. doi: 10.1148/radiol.13122670.
52. Kwon BJ, Han MH, Kang H-S, Chang K-H. MR Imaging Findings of Intracranial Dural Arteriovenous Fistulas: Relations with Venous Drainage Patterns. *AJNR Am J Neuroradiol* [Internet]. 2005;26(10):2500–7.49.
53. Bink A, Berkefeld J, Wagner M, You SJ, Ackermann H, Lorenz MW, et al. Detection and grading of dAVF: Prospects and limitations of 3T MRI. *Eur Radiol*. 2012;22(2):429–38.

54. Meckel S, Maier M, Ruiz DSM, Yilmaz H, Scheffler K, Radue EW, et al. MR angiography of dural arteriovenous fistulas: diagnosis and follow-up after treatment using a timeresolved 3D contrast-enhanced technique. *AJNR Am J Neuroradiol* [Internet].2007;28(May):877–84.
55. Farb RI, Agid R, Willinsky RA, Johnstone DM, TerBrugge KG. Cranial dural arteriovenous fistula: Diagnosis and classification with time-resolved MR angiography at 3T. *Am J Neuroradiol*. 2009;30(8):1546–51.
56. Nishimura S, Hirai T, Sasao A, et al. Evaluation of dural arteriovenous fistulas with 4D contrast-enhanced MR angiography at 3T. *AJNR Am J Neuroradiol* 2010;31(1):80-85. DOI:10.3174/ajnr.A1898.
57. Ertl L, Brückmann H, Kunz M, et al. Assessment and treatment planning of lateral intracranial dural arteriovenous fistulas in 3 T MRI and DSA: A detailed analysis under consideration of time-resolved imaging of contrast kinetics (TRICKS) and CE-MRA sequences. *Eur Radiol* 2016;26(12):4284-4292. DOI:10.1007/s00330-016-4335-1.
58. Dissaux B, Eugène F, Ognard J, Gauvrit JY, Gentric JC, Ferré JC. Assessment of 4D MR Angiography at 3T Compared with DSA for the Follow-up of Embolized Brain Dural Arteriovenous Fistula: A Dual-Center Study. *AJNR Am J Neuroradiol*. 2021 Jan;42(2):340-346. doi: 10.3174/ajnr.A6903.
59. Cheng Y, Chen H, Wu C, Wu Y, Sun M, et al. Magnetic Resonance Angiography in the Diagnosis of Cerebral Arteriovenous Malformation and Dural Arteriovenous Fistulas: Comparison of Time-Resolved Magnetic Resonance Angiography and Three Dimensional Time-of-Flight Magnetic Resonance Angiography, *Iran J Radiol*. 2016 ; 13(2):e19814. doi: 10.5812/iranradiol.19814.

60. Santhosh K, Kesavadas C, Thomas B, Gupta AK, Thamburaj K, Kapilamoorthy TR. Susceptibility weighted imaging: a new tool in magnetic resonance imaging of stroke. Clin Radiol[Internet].2009;64(1):74–83.
61. Thomas B, Somasundaram S, Thamburaj K, Kesavadas C, GuptaAK, BodheyNK, et al. Clinical applications of susceptibility weighted MR imaging of the brain -A pictorial review. Neuroradiology. 2008;50(2):105–16.
62. Saini J, Thomas B, Bodhey NK, Periakaruppan A, Babulal JM. Susceptibility-weighted imaging in cranial dural arteriovenous fistulas. Am J Neuroradiol. 2009;30(1):2009.
63. Noguchi K, Kuwayama N, Kubo M, Kamisaki Y, Kameda K, Tomizawa G, et al. Intracranial dural arteriovenous fistula with retrograde cortical venous drainage: Use of susceptibility-weighted imaging in combination with dynamic susceptibility contrast imaging. Am J Neuroradiol. 2010;31(10):1903–10.
64. Gasparetto EL, Pires CE, Domingues RC. Susceptibility-weighted MR phase imaging can demonstrate retrograde leptomeningeal venous drainage in patients with dural arteriovenous fistula. Am J Neuroradiol. 2011;32(3):3174.
65. Letourneau-Guillon L, Krings T. Simultaneous arteriovenous shunting and venous congestion identification in dural arteriovenous fistulas using susceptibility-weighted imaging: Initial experience. Am J Neuroradiol. 2012;33(2):301–7.
66. Nakagawa I, Taoka T, Wada T, Nakagawa H, Sakamoto M, Kichikawa K, et al. The use of susceptibility-weighted imaging as an indicator of retrograde leptomeningeal venous

drainage and venous congestion with dural arteriovenous fistula: Diagnosis and follow-up after treatment. *Neurosurgery*. 2013;72(1):47–54.

67. Hodel J, Leclerc X, Kalsoum E, Zuber M, Tamazyan R, Benadjaoud MA, et al. Intracranial arteriovenous shunting: Detection with arterial spin-labeling and susceptibility-weighted imaging combined. *Am J Neuroradiol*. 2017;38(1):71–6.
68. Sato K, Shimizu H, Fujimura M, Inoue T, Matsumoto Y, Tominaga T. Compromise of brain tissue caused by cortical venous reflux of intracranial dural arteriovenous fistulas: Assessment with diffusion-weighted magnetic resonance imaging. *Stroke*. 2011;42(4):998–1003.
69. Wu H, Block WF, Turski PA, Mistretta CA, Rusinak DJ, Wu Y, et al. Noncontrast dynamic 3D intracranial MR angiography using pseudo-continuous arterial spin labeling (PCASL) and accelerated 3D radial acquisition. *J Magn Reson Imaging*. 2014;39(5):1320–6.
70. Deibler AR, Pollock JM, Kraft RA, Tan H, Burdette JH, Maldjian JA. Arterial spin-labeling in routine clinical practice, part 1: Technique and artifacts. *Am J Neuroradiol*. 2008;29(7):1228–34.
71. Wolf RL, Wang J, Detre JA, et al. Arteriovenous shunt visualization in arteriovenous malformations with arterial spin-labeling MR imaging. *AJNR Am J Neuroradiol* 2008;29(4):681-687. DOI:10.3174/ajnr.A0901

72. Robson PM, Dai W, Shankaranarayanan A, Rofsky NM, Alsop DC. Time-resolved vessel-selective digital subtraction MR angiography of the cerebral vasculature with arterial spin labeling. *Radiology* [Internet]. 2010;257(2):507–15.
73. Sakamoto S, Kiura Y, Okazaki T. Arterial Spin-labeling Imaging at 3-T in Dural Arteriovenous Fistulas of Cavernous Sinus before and after Endovascular Treatment. 2012;61(4):1–2.
74. Noguchi K. Intracranial Dural Arteriovenous Fistula: Preliminary Report of Arterial Spin Labeling. *Open J Med Imaging* [Internet]. 2013;3(1):1–6.
75. Amukotuwa SA, Yu C, Zaharchuk G. 3D Pseudocontinuous arterial spin labeling in routine clinical practice: A review of clinically significant artifacts. *J Magn Reson Imaging*. 2016;43(1):11–27.
76. Sunwoo L, Sohn CH, Lee JY, Yi KS, Yun TJ, Choi SH, et al. Evaluation of the degree of arteriovenous shunting in intracranial arteriovenous malformations using pseudocontinuous arterial spin labeling magnetic resonance imaging. *Neuroradiology*. 2015;57(8):775–82.
77. Amukotuwa SA, Heit JJ, Marks MP, Fischbein N, Bammer R. Detection of cortical venous drainage and determination of the borden type of dural arteriovenous fistula by means of 3D pseudocontinuous arterial spin-labeling MRI. *Am J Roentgenol*. 2016;207(1):163–9.
78. Yamamoto N, Satomi J, Yamamoto Y, Izumi Y, Nagahiro S, Kaji R. Usefulness of 3-Tesla magnetic resonance arterial spin-labeled imaging for diagnosis of cranial dural arteriovenous fistula. *J Neurol Sci* [Internet]. 2017;372(April 2013):428–32.

79. Jaganmohan D, Pan S, Kesavadas C, Thomas B. A pictorial review of brain arterial spin labelling artefacts and their potential remedies in clinical studies. *Neuroradiol J*. 2021 Jun;34(3):154-168. doi: 10.1177/1971400920977031.
80. Moon JI, Baek HJ, Ryu KH, Park H. A novel non-contrast-enhanced MRA using silent scan for evaluation of brain arteriovenous malformation: A case report and review of literature. *Medicine (Baltimore)*. 2017;96(45):e8616. doi:10.1097/MD.00000000000008616.
81. Takano N, Suzuki M, Irie R, Yamamoto M, Hamasaki N, Kamagata K, Kumamaru KK, Hori M, Oishi H, Aoki S. Usefulness of Non-Contrast-Enhanced MR Angiography Using a Silent Scan for Follow-Up after Y-Configuration Stent-Assisted Coil Embolization for Basilar Tip Aneurysms. *AJNR Am J Neuroradiol*. 2017 Mar;38(3):577-581. doi: 10.3174/ajnr.A5033.
82. Oishi H, Fujii T, Suzuki M, Takano N, Teranishi K, Yatomi K, Kitamura T, Yamamoto M, Aoki S, Arai H. Usefulness of Silent MR Angiography for Intracranial Aneurysms Treated with a Flow-Diverter Device. *AJNR Am J Neuroradiol*. 2019 May;40(5):808-814. doi: 10.3174/ajnr.A6047.
83. Tomura N, Kokubun M, Horiuchi K, Watanabe Z. Comparison of TOF-MRA and silent scan-MRA in depicting cerebral arteries in patients with Moyamoya disease. *Acta Radiol*. 2019 Oct;60(10):1321-1328. doi: 10.1177/0284185118824782.
84. Iwama T, Hashimoto N, Takagi Y, Tanaka M, Yamamoto S, Nishi S, et al. Hemodynamic and metabolic disturbances in patients with intracranial dural

arteriovenous fistulas: positron emission tomography evaluation before and after treatment. *J Neurosurg* [Internet].1997;86(5):806–11.

85. Baltsavias G, Valavanis A. Endovascular treatment of 170 consecutive cranial dural arteriovenous fistulae: Results and complications. *Neurosurg Rev.* 2014;37(1):63–71)
86. Woo HH, Masaryk TJ, Rasmussen PA. Treatment of dural arteriovenous malformations and fistulae. *Neurosurg Clin N Am.* 2005;16(2 SPEC. ISS.):381–93.
87. Satomi J, van Dijk JM, Terbrugge KG, Willinsky RA, Wallace MC. Benign cranial dural arteriovenous fistulas: outcome of conservative management based on the natural history of the lesion. *J Neurosurg.* 2002;97(4):767–70.
88. Rammos S, Bortolotti C, Lanzino G. Endovascular management of intracranial dural arteriovenous fistulae. *Neurosurg Clin N Am* [Internet]. 2014;25(3):539–49.
89. Gross BA, Albuquerque FC, Moon K, McDougall CG. Evolution of treatment and a detailed analysis of occlusion, recurrence, and clinical outcomes in an endovascular library of 260 dural arteriovenous fistulas. *J Neurosurg.* 2016;1–10.
90. Wang J, Wu H, Wang W, Zhao H, Dao R, Liu W, et al. Trigeminal Cardiac Reflex Caused by Onyx Embolization of Intracranial Dural Arteriovenous Fistula. 2016;26(3):325–30.
91. Baltsavias G, Spiessberger A, Hothorn T, Valavanis A. Cranial dural arteriovenous shunts. Part 4. Clinical presentation of the shunts with leptomeningeal venous drainage. *Neurosurg Rev.* 2015;38(2):283–91.

92. Oh JT, Chung SY, Lanzino G, Park KS, Kim SM, Park MS, et al. Intracranial dural arteriovenous fistulas: clinical characteristics and management based on location and hemodynamics. *J Cerebrovasc Endovasc Neurosurg* [Internet]. 2012;14(3):192–202.
93. Piippo A, Laakso A, Seppä K, Rinne J, Jääskeläinen JE, Hernesniemi J, et al. Early and long-term excess mortality in 227 patients with intracranial dural arteriovenous fistulas. *J Neurosurg* [Internet]. 2013;119(1):164–71.
94. Amukotuwa SA, Marks MP, Zaharchuk G, et al. Arterial Spin-Labeling Improves Detection of Intracranial Dural Arteriovenous Fistulas with MRI. *AJNR Am J Neuroradiol* 2018;39(4):669-677.
95. Brinjikji W, Cloft HJ, Lanzino G. Clinical Presentation and Imaging Findings of Patients with Dural Arteriovenous Fistulas with an Angiographic Pseudophlebitic Pattern. *AJNR Am J Neuroradiol*. 2020 Dec;41(12):2285-2291. doi: 10.3174/ajnr.A6811.
96. Irie R, Suzuki M, Yamamoto M, et al. Assessing Blood Flow in an Intracranial Stent: A Feasibility Study of MR Angiography Using a Silent Scan after Stent-Assisted Coil Embolization for Anterior Circulation Aneurysms. *AJNR Am J Neuroradiol* 2015;36(5):967-970. DOI:10.3174/ajnr.A4199.



ILLUSTRATIVE CASES



CASE 1

Case summary:

45 year old male presented with symptoms of left ear continuous subjective tinnitus of 04 months duration, followed by one episode of severe holocranial headache alongwith multiple episodes of vomiting. Imaging done outside hospital showed left cerebellar bleed with suspicion of DAVF. Then he was referred to SCTIMST after conservative management for left temporal bleed from which he recovered. On clinical examination, he was conscious, oriented with arterial bruit was heard over left retromastoid region. He had no other focal neurological deficits.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 2A+B dAVF at the left sigmoid sinus and jugular bulb region. ASL and SWAN showed hyperintense venous signals in left sigmoid sinus and jugular bulb region with antegrade as well as retrograde flow alongwith cortical venous reflux. SWAN also showed pseudophlebitic pattern in posterior fossa. Silent and TOF MRA showed arterial feeders from left occipital, ascending pharyngeal and left posterior meningeal arteries.

DSA:

6 vessel DSA showed Type 2A+B dAVF at left sigmoid sinus and jugular bulb region with arterial feeders from mastoid branch of left occipital artery, neuromeningeal trunk of left ascending pharyngeal artery and posterior meningeal artery from left vertebral artery. No prominent feeders were seen from left middle meningeal artery. Antegrade flow was seen into the left IJV and retrograde flow was seen into the left sigmoid sinus and transverse sinus. Cortical venous reflux was seen into the cerebellar hemispheric veins and left temporal veins.

Images:

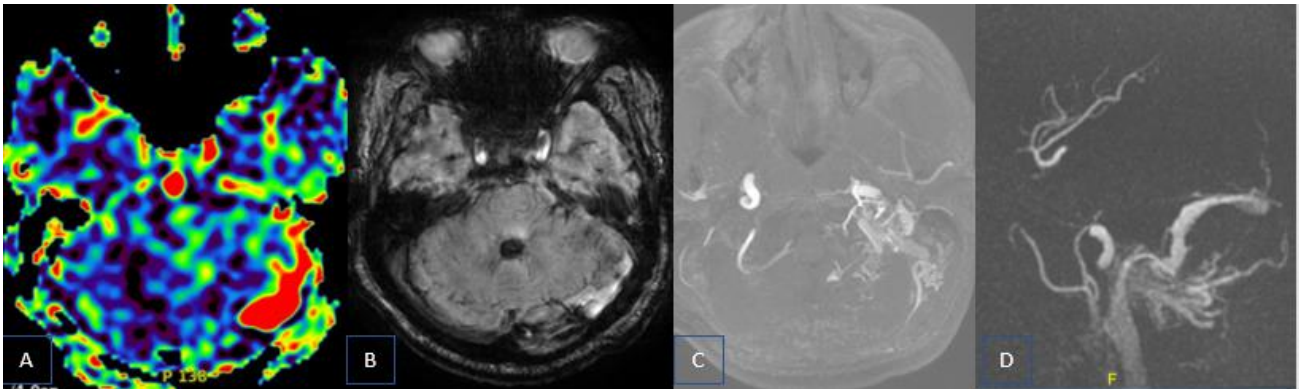


Figure 9: ASL (A) and SWAN(B) showing location of fistula with hyperintense signals at left sigmoid sinus, left jugular bulb and left transverse sinus. TOF MRA axial MIP images(C) and Silent MRA sagittal MIP images(D) showing location of fistula with venous drainage – both antegrade as well as retrograde alongwith cortical venous reflux

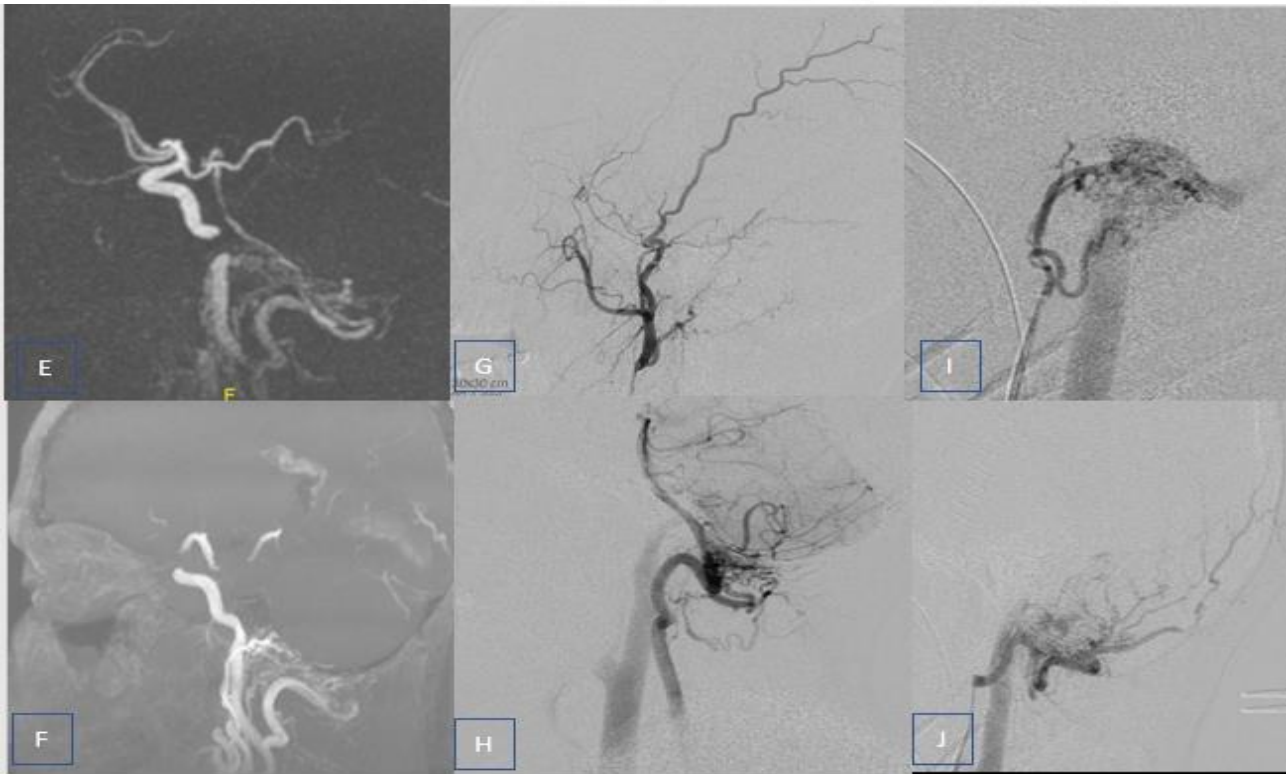


Figure 10: Silent MRA sagittal MIP(E) and TOF MRA sagittal MIP(F) images showing arterial feeders to the fistulous site. DSA images : Left IMA lateral injection showing absence of left MMA feeders to the fistula and selective angiograms showing arterial feeders from to the fistula from left posterior meningeal(H), left ascending pharyngeal(I) and left occipital arteries (J) with venous reflux. Pseudophlebotic pattern seen in left cerebellar hemisphere in left occipital artery selective angiogram.

CASE 2:

Case Summary:

39 year old male presented with symptoms of holocranial headache for 06 months duration. He also had symptoms of gradual onset visual blurring in the both eyes (Right > left) of 03 months duration. Ophthalmological evaluation revealed bilateral papilloedema and MRI was done which revealed suspicious vascular flow void in left pterional region. He was referred to SCTIMST for further evaluation.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 3 dAVF at the left pterion region. ASL and SWAN showed hyperintense venous signals in left pterion region with direct extension into the left frontal cortical vein. SWAN did not reveal any pseudophlebitic pattern. Silent and TOF MRA showed fistula located in left pterion and arterial feeders from left middle meningeal artery (MMA) and left ophthalmic artery with direct cortical venous reflux.

DSA:

6 vessel DSA showed Type 3 dAVF at left pterion region with network of arterial feeders from left MMA and small feeder from meningolacrimal trunk of left ophthalmic artery. Antegrade direct cortical venous reflux flow was seen into the left frontal cortical vein IJV with further egress into the superior sagittal sinus. No pseudophlebitic pattern is noted.

IMAGES:

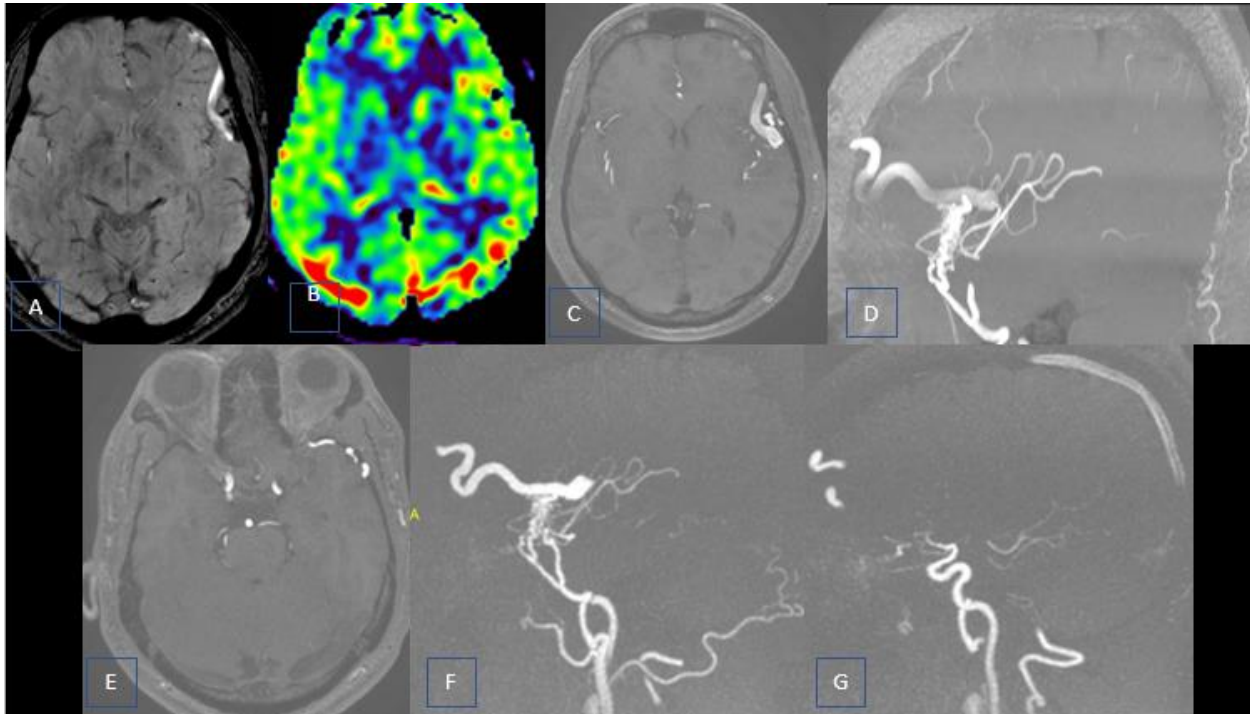


Figure 11: SWAN(A) and ASL (B) showing location of the fistula in left pterion with venous hyperintense signals along with venous reflux. Axial TOF MRA MIP(C), Sagittal TOF MRA MIP(D) and axial TOF MRA MIP(E) showing location of the fistula in left pterion and feeders to the fistula from left MMA, left ophthalmic artery and venous reflux into the cortical veins. Sagittal MIP images of silent MRA (F) and (G) showing location of the fistula as well as arterial feeders to the fistula from left MMA and left ophthalmic artery along with direct venous drainage to the frontal cortical vein.



Figure 13: DSA images from left ECA angiogram lateral view (H) showing left MMA feeders and direct cortical venous reflux. Selective left IMA angiogram lateral view (I) showing network of left MMA feeders with direct cortical venous reflux. Left ICA angiogram lateral view (J) showing arterial feeder from left ophthalmic artery.

CASE 3:

Case summary:

57 year old male presented with symptoms of redness in left eye for 03 months followed by swelling and increased lacrimation alongwith double vision in left gaze for 02 months. Ophthalmological evaluation revealed increase in left intraocular pressure to 22 mm hg and early papilloedema. Imaging done outside revealed prominent left superior ophthalmic vein. Then he was referred to SCTIMST with suspicion of left sided indirect caroticocavernous fistula.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 2A+B dAVF at the left hypoglossal canal region. ASL and SWAN showed hyperintense venous signals in left hypoglossal canal region with extension into the left cavernous sinus and left superior ophthalmic vein. SWAN did not reveal any pseudophlebitic pattern. Silent and TOF MRA showed fistula located in left hypoglossal canal and arterial feeders from left occipital, ascending pharyngeal and posterior auricular arteries with venous reflux into the left cavernous sinus, left SOV and IPS alongwith jugular bulb drainage.

DSA:

6 vessel DSA showed Type 2A+B dAVF at the left hypoglossal canal region with arterial feeders from left occipital, ascending pharyngeal and posterior auricular arteries with venous reflux into the left cavernous sinus, left SOV and IPS alongwith jugular bulb drainage. No pseudophlebitic pattern is noted.

IMAGES:

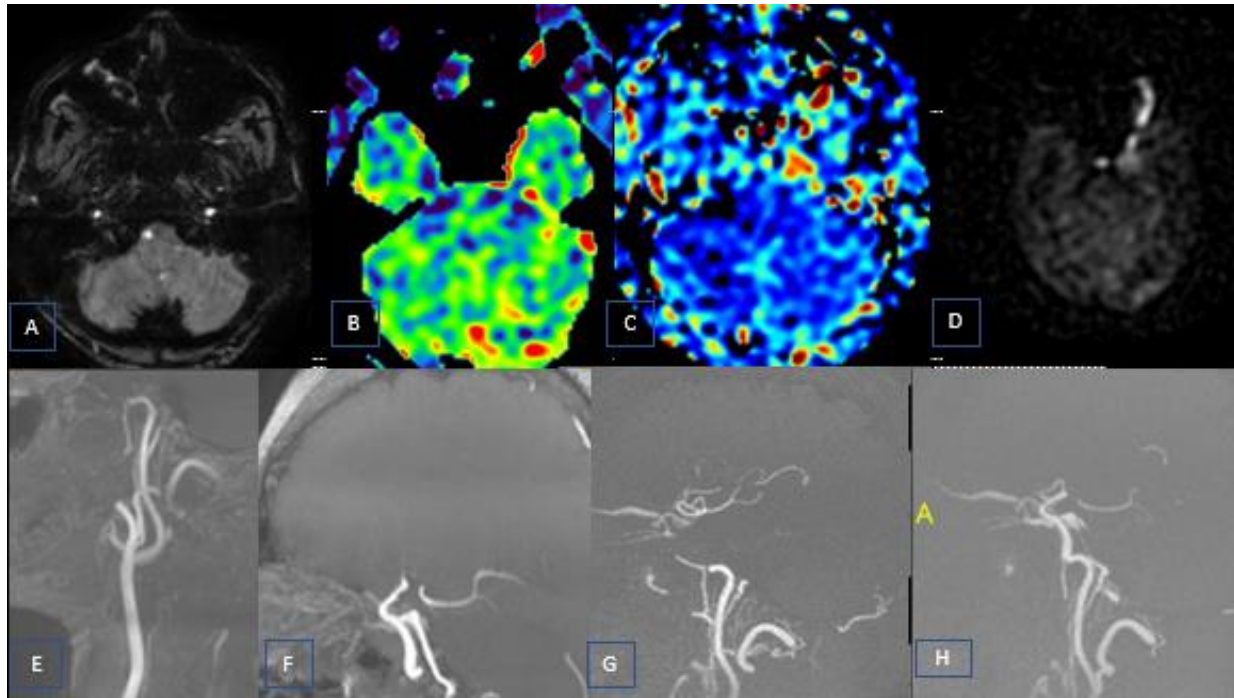


Figure 14: SWAN(A), ASL(B), multidelay ASL(C)images showing the venous hyperintense signals in left hypoglossal canal. ASL source images(D) showing the extension of venous hyperintense signals into the left cavernous sinus and left SOV. Sagittal MIP images of TOF MRA(E, F) showing the arterial feeders from left ascending pharyngeal and occipital arteries. Venous drainage into the left SOV is seen in F. Silent MRA Sagittal MIP images (G, H) showing arterial feeders from left occipital, ascending pharyngeal and posterior auricular arteries with H showing venous drainage into left cavernous sinus, left SOV,IPS better than TOF MRA.

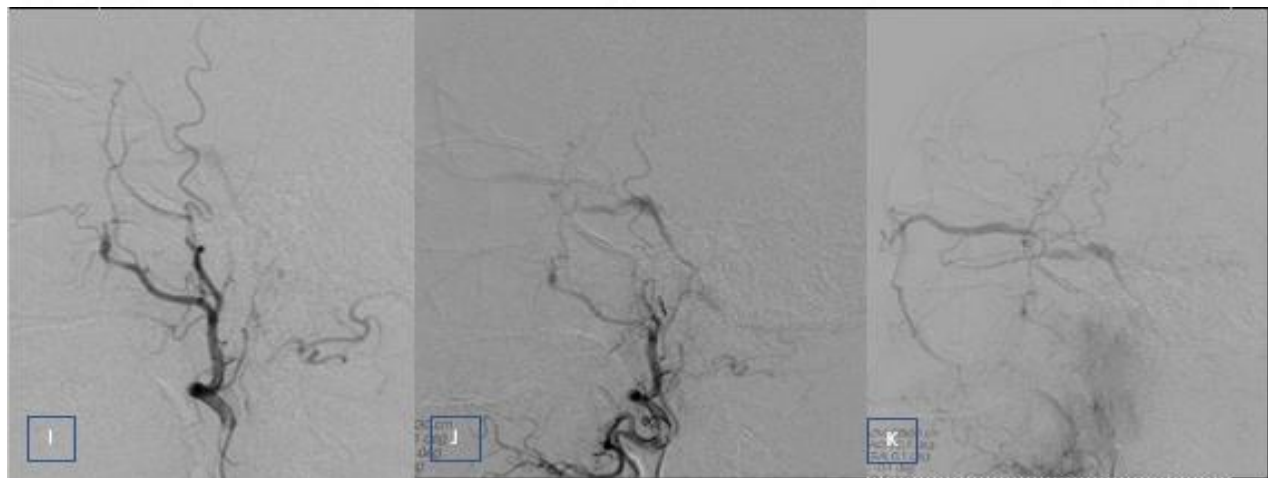


Figure 15: DSA left IMA angiogram lateral view (I) showing left occipital, ascending pharyngeal and posterior auricular arteries to the fistula at left hypoglossal canal. Late arterial and Early venous phase (J, K) showing venous drainage from the fistula into the cavernous sinus and left SOV and also into the left IPS.

CASE 4:

Case summary:

53 year old male presented with symptoms of sudden onset of severe holocranial headache and multiple episodes of vomiting. Prior history of moderate intensity headache was present for 05 months. Imaging done at outside hospital revealed left temporal bleed. He was managed conservatively. After improvement of the general condition, MRI was done which revealed tortuous vessels in in posterior fossa as well as in left temporal region. Then he was referred to SCTIMST with suspicion of dAVF. On examination at SCTIMST, he was conscious and oriented with fundus showing bilateral papilloedema. No other neurological deficits were noted.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 2A+B dAVF at the left transverse sinus and sigmoid sinus junction region. ASL and SWAN showed hyperintense venous signals in left transverse sinus and sigmoid sinus junction region with antegrade flow into left IJV and alongwith cortical venous reflux. SWAN also showed pseudophlebitic pattern in posterior fossa and left cerebral hemisphere. Silent and TOF MRA showed arterial feeders from left occipital, middle meningeal and left posterior meningeal arteries.

DSA:

6 vessel DSA showed Type 2A+B dAVF at left transverse sinus and sigmoid sinus junction region with arterial feeders from transosseous branches left occipital artery, petrous and petrosquamous branches of left middle meningeal artery and posterior meningeal branch from left vertebral artery. Antegrade flow was seen into the left IJV and cortical venous reflux was seen into the left temporal, parietal and occipital veins.

IMAGES:

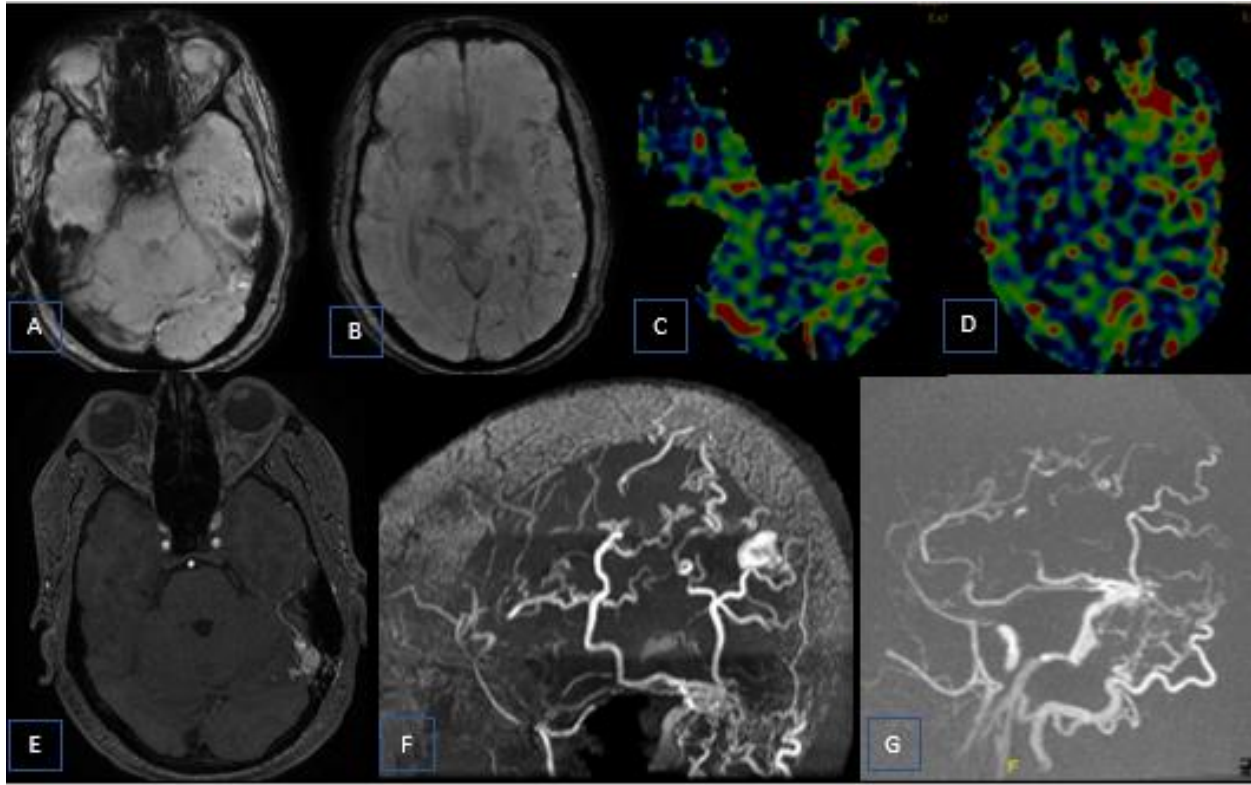


Figure 16: SWAN (A, B) and ASL (C, D) showing location of fistula in left transverse sinus and sigmoid sinus junction with venous hyperintense signals along with pseudophlebitic pattern in left side in SWAN(B). Axial MIP of TOF MRA (E) showing location of fistula in left transverse sinus and sigmoid sinus junction. Sagittal MIP TOF MRA images showing arterial feeders from left occipital artery, left MMA alongwith antegrade venous drainage and cortical venous reflux. Silent MRA Sagittal MIP images(G) images showing arterial feeders from left occipital artery, left MMA and left PMA with cortical venous reflux.

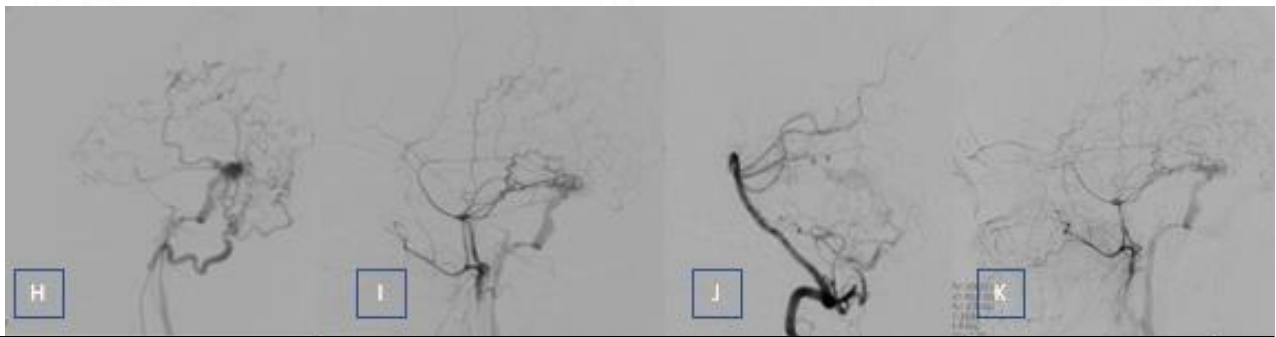


Figure 17: DSA images selective left occipital(H), left IMA(I), left Vertebral artery(J) showing location of fistula at left transverse sinus and sigmoid sinus junction with arterial feeders from left occipital, left MMA (petrous, petrosquamous branches), posterior meningeal branch of left Vertebral artery. Late arterial phase images showing (K)antegrade venous drainage with cortical venous reflux and pseudophlebitic pattern.

CASE 5:

Case summary:

62 year old female presented with symptoms of sudden onset headache, neckpain and vomiting followed by altered sensorium. Imaging done outside showed subarachnoid haemorrhage at the foramen magnum level and extending into the cervical canal. She was referred to SCTIMST. On examination she was disoriented with GCS of 13/15 with severe headache and neck pain. She was managed with Subarachnoid hemorrhage protocol in Neurointervention Centre ICU and was taken up for Imaging and DSA after improvement of the general condition and neurological status.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 5 dAVF at the level of foramen magnum. ASL and SWAN showed hyperintense venous signals in the foramen magnum region with anterior as well as posterior extension into the cervical canal. Silent and TOF MRA showed arterial feeders from bilateral posterior meningeal arteries with venous drainage into the spinal peri medullary veins.

DSA:

6 vessel DSA showed Type 5 dAVF at the level of foramen magnum with arterial feeders from bilateral posterior meningeal branches of vertebral arteries. Direct venous drainage was seen into anterior as well as posterior perimedullary veins.

IMAGES:

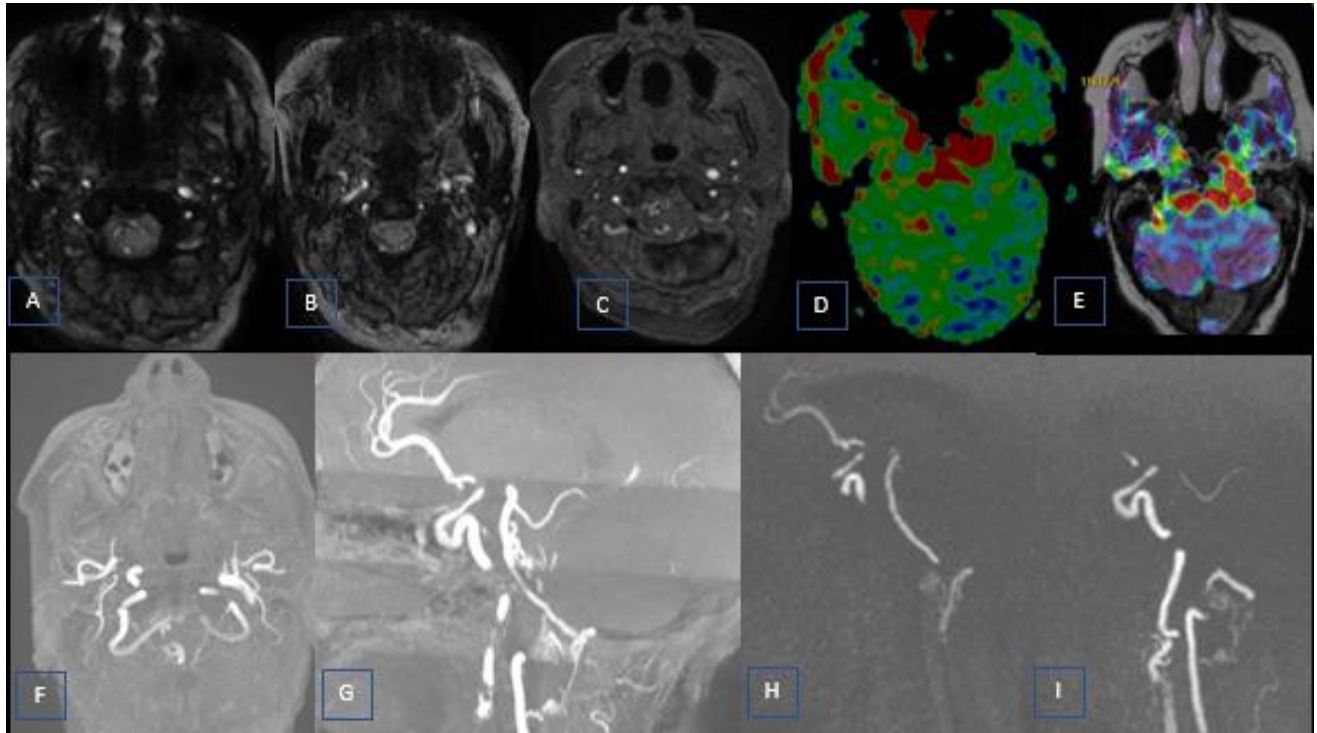


Figure 18: SWAN(A,B) images, Axial TOF MRA(C), ASL(D) and Fused ASL(E) images showing foramen magnum dAVF with drainage into the perimedullary veins. Axial TOF MRA MIP(F) and sagittal TOF MRA MIP(G) images showing arterial feeders from bilateral posterior meningeal arteries supplying the fistula. Silent MRA sagittal MIP images (H,I) showing arterial feeders from bilateral PMA and venous drainage into the anterior as well as posterior spinal perimedullary veins.

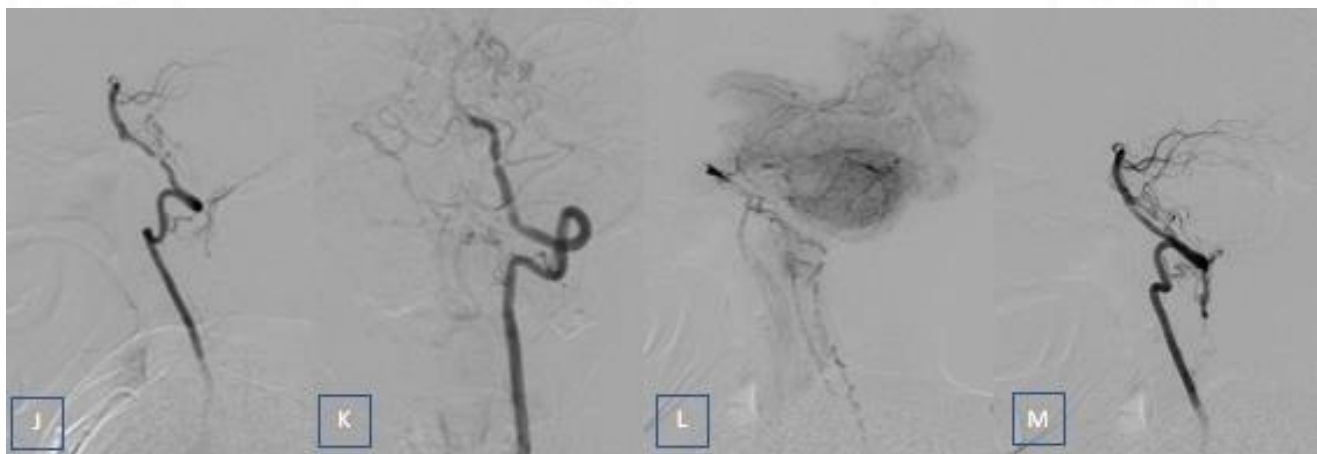


Figure 19: left Vertebral artery angiogram lateral and AP views(J,K) showing foramen magnum dAVF with posterior meningeal branch feeder. Capillary phase of left vertebral artery angiogram lateral view(L) showing venous drainage into the anterior as well as posterior spinal perimedullary veins. Right vertebral artery angiogram lateral view(M) showing feeder from right posterior meningeal artery.

CASE 6:

Case summary:

71 year old male presented with 6 months symptoms of tinnitus in right ear, pulsatile and subjective in nature and after two months he also developed redness in both eyes (right > left) with headache. Ophthalmological evaluation revealed no papilloedema. MRI done outside was reported as normal. In view of the persistent symptoms, he was referred to SCTIMST with suspicion of indirect caroticocavernous fistula. On examination he had bruit in the right retromastoid region and conjunctival congestion in both eyes (right > left). No other neurological deficits were noted.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 2A dAVF at the right hypoglossal canal and jugular bulb region. ASL and SWAN showed hyperintense venous signals in right hypoglossal canal and jugular bulb region with extension into the right sigmoid sinus and IPS. SWAN did not reveal any pseudophlebitic pattern. Silent and TOF MRA showed fistula located in right hypoglossal canal & jugular bulb region. Arterial feeders were noted from bilateral ascending pharyngeal and right occipital arteries with venous reflux into the right Inferior petrosal sinus(IPS) and left sigmoid sinus alongwith jugular bulb drainage.

DSA:

6 vessel DSA showed Type 2A dAVF at the right hypoglossal canal and jugular bulb region with arterial feeders from right occipital and bilateral ascending pharyngeal arteries with venous reflux into the right IPS, sigmoid sinus and subtle drainage into bilateral SOV alongwith jugular bulb drainage. No pseudophlebitic pattern is noted.

IMAGES:

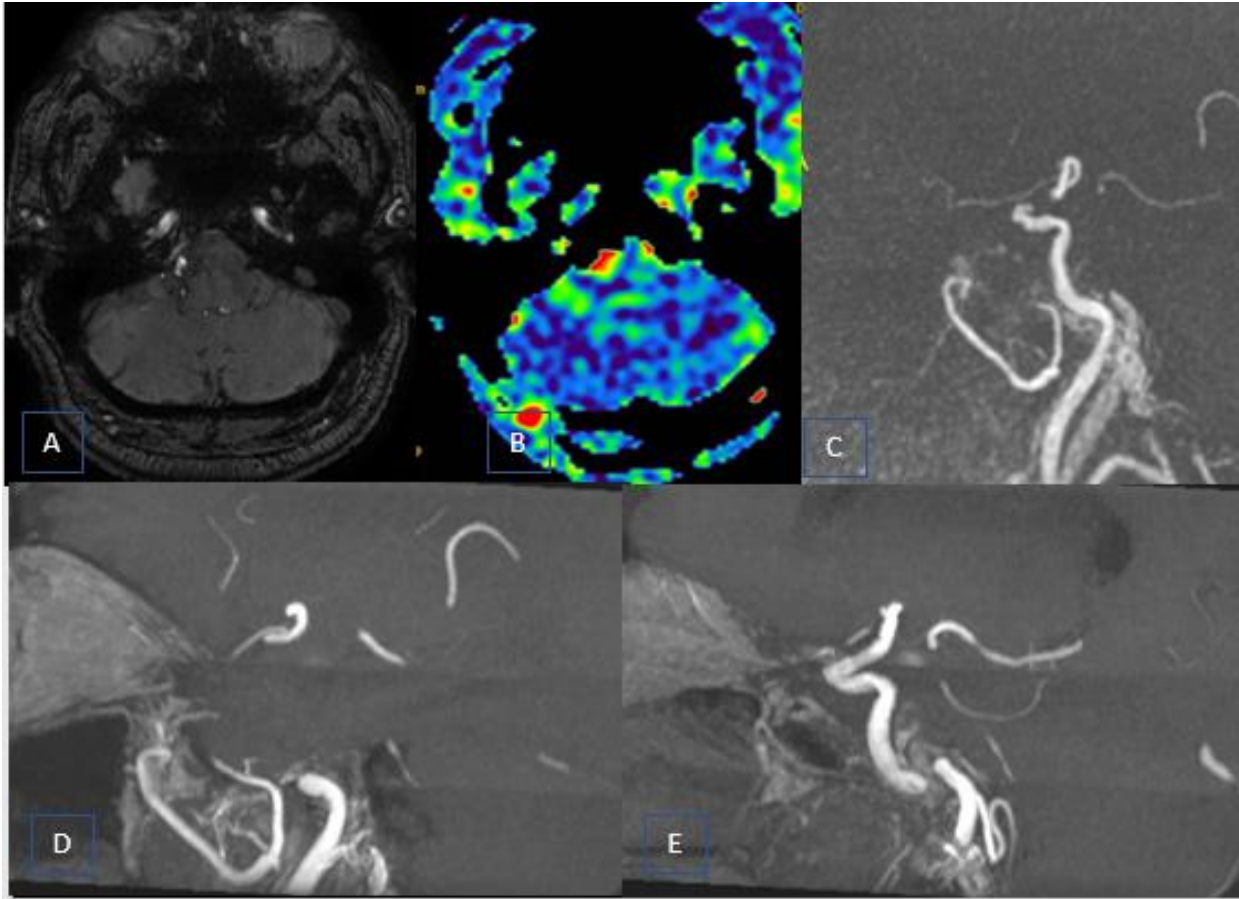


Figure 20: SWAN(A) and ASL(B) images showing location of the fistula with venous hyperintense signals in the right hypoglossal canal and jugular bulb region. Silent MRA sagittal MIP images(C) showing fistula at right hypoglossal canal & Jugulars bulb region with arterial feeders from right ascending pharyngeal and occipital arteries and venous drainage into the right IPS, Sigmoid sinus and IJV. TOF MRA sagittal MIP images(D, E) showing arterial feeders from right occipital and ascending pharyngeal arteries and venous drainage into the IPS and IJV.

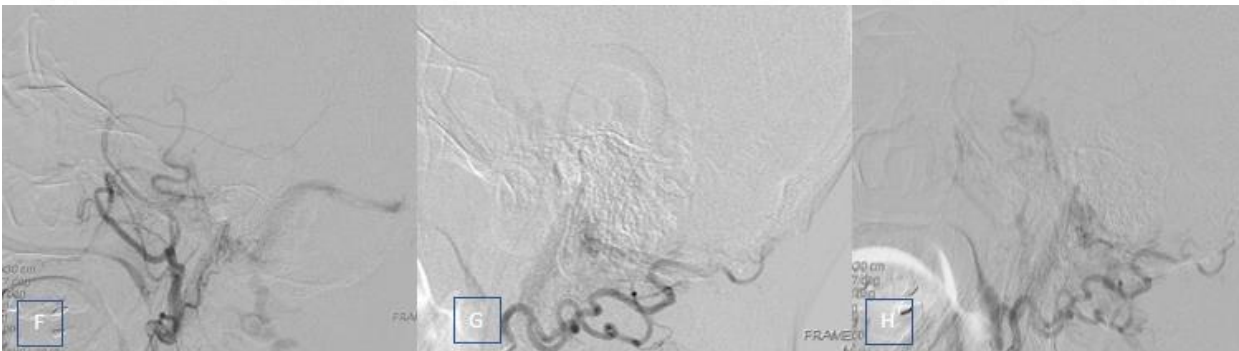


Figure 21: DSA Right ECA angiogram lateral view(F) showing feeders from ascending pharyngeal artery to the fistula with retrograde venous drainage into the sigmoid sinus and IPS. Right occipital artery selective angiogram (G) showing feeders from right occipital artery to the fistula with late arterial phase(H) showing venous drainage into the right IPS and Sigmoid sinus.

CASE 7:

Case summary:

40 year old male a known case of cerebral venous thrombosis on Tab Acitrom, presented with altered sensorium and irrelevant speech of 5 days duration. History of multiple episodes of vomiting and blurring of vision in both eyes was also present. Imaging done outside showed bilateral chronic transverse sinus thrombosis and suspected right transverse sinus dAVF. Then patient was referred to SCTIMST for further evaluation and management. On examination he was confused, bilateral gross diminution of visual acuity- no PL, bilateral papilloedema and elevated Intraocular pressure in right eye -22 mm hg. No other deficits were noted.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 2A+B dAVF at the right transverse sinus region. ASL and SWAN showed hyperintense venous signals in right transverse sinus and torcula region with extension into the straight sinus and vein of Galen. SWAN showed pseudophlebitic pattern. Silent and TOF MRA showed fistula located in right transverse sinus region. Arterial feeders were noted from right MMA, right ascending pharyngeal, right occipital artery and marginal tentorial branch of right ICA. Venous drainage was seen from the isolated right transverse sinus into the torcula, straight sinus, vein of Galen and posterior superior sagittal sinus alongwith cortical venous reflux.

DSA:

6 vessel DSA showed Cognard Type 2A+B dAVF at the right transverse sinus region with arterial feeders from right MMA – petrosquamous and petrous branches, right occipital, right ascending pharyngeal and right marginal tentorial arteries with venous reflux into the right torcula, straight sinus, vein of Galen, ICV and posterior superior sagittal sinus. Pseudophlebitic pattern is noted.

IMAGES:

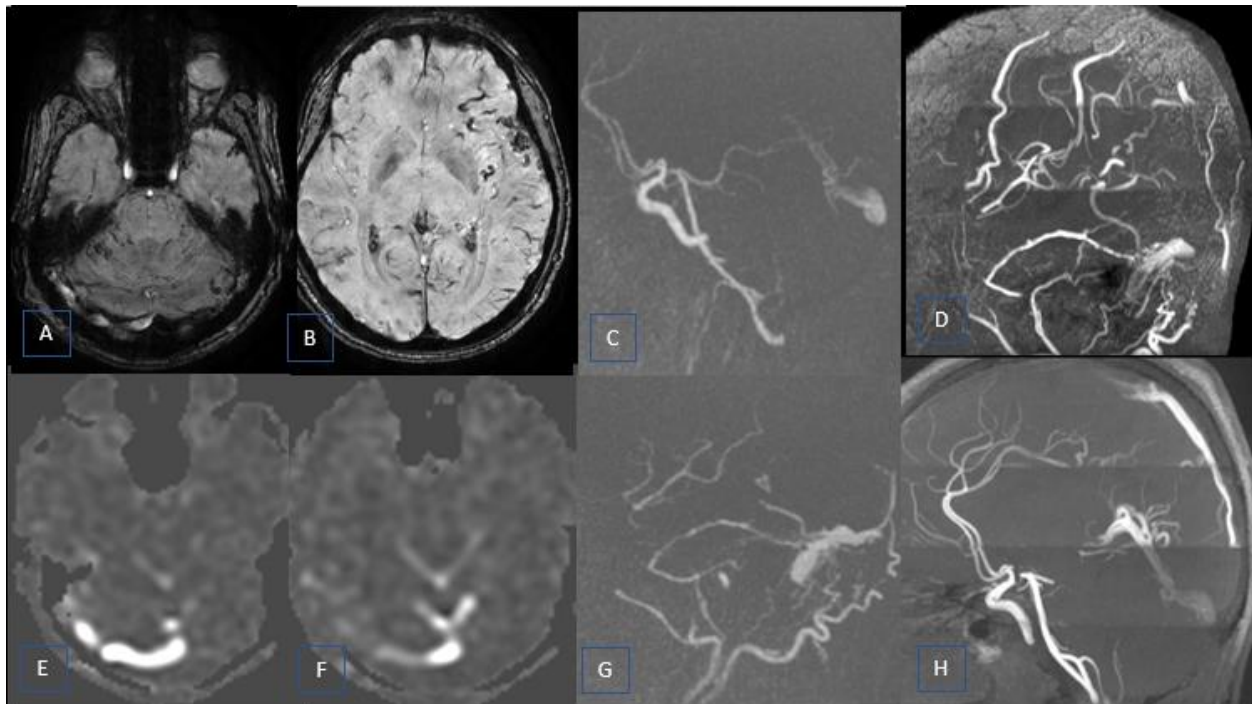


Figure 22: SWAN images(A,B) showing venous hyperintense signals in right transverse sinus with pseudophlebitic pattern(B). ASL source images (E,F) showing venous hyperintense signals in right transverse sinus with extension into torcula, straight sinus and cortical veins. Silent MRA(C, G)sagittal MIP images showing arterial feeders into the fistula from right MMA, occipital and ascending pharyngeal arteries with venous drainage into the torcula and straight sinus. TOF MRA(D,H) sagittal MIP images showing arterial feeders into the fistula from right MMA, occipital and ascending pharyngeal arteries with venous drainage into the torcula, straight sinus and posterior superior sagittal sinus.

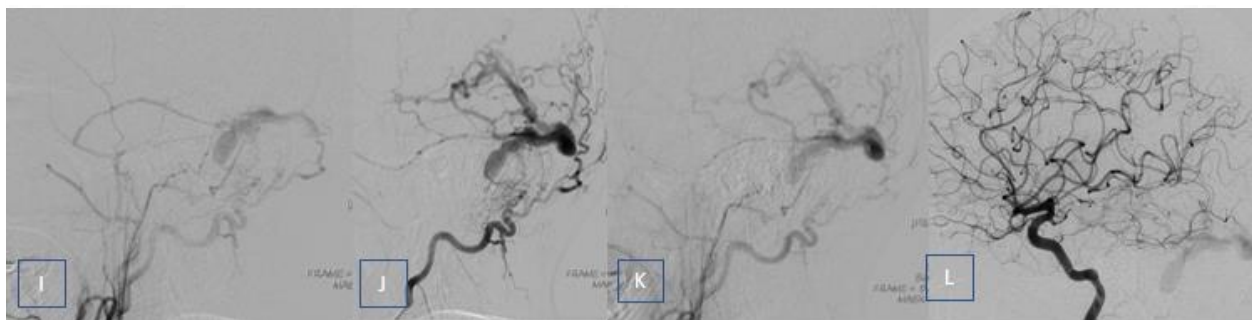


Figure 23: DSA right ECA angiogram (I,J) showing arterial feeders from right MMA, right occipital, posterior auricular and ascending pharyngeal arteries with venous drainage (J,K) into the torcula, straight sinus and Vein of Galen. Right ICA angiogram lateral view (L) showing marginal tentorial feeder supplying the fistula.

CASE 8:

Case summary:

43 year old male presented with two episodes of generalized tonic clonic seizures followed by altered sensorium and poor GCS. He was treated in ICU with ventilatory support. Imaging with angiography revealed right temporal bleed with suspected right transverse sinus DAVF. He was treated conservatively, Then he was referred to SCTIMST for further management. On examination, he was conscious with GCS of 15/15, with no focal deficits. No papilledema seen on fundus examination.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 2A+B dAVF at the right transverse sinus- sigmoid sinus junction region. ASL and SWAN showed hyperintense venous signals in right transverse sinus and sigmoid sinus junction region with extension into the torcula, straight sinus and superior sagittal sinus. SWAN showed no pseudophlebitic pattern. Silent and TOF MRA showed fistula located in right transverse sinus – sigmoid sinus region. Arterial feeders were noted from right MMA, right ascending pharyngeal, right posterior auricular, right occipital artery, right posterior meningeal artery and marginal tentorial branch of right ICA. Venous drainage was seen from the right transverse sinus into the torcula, straight sinus and posterior superior sagittal sinus alongwith cortical venous reflux.

DSA:

6 vessel DSA showed Cognard Type 2A+B dAVF at the right transverse sinus - sigmoid sinus region with arterial feeders from right MMA – petrosquamous branch, right ascending pharyngeal, right posterior auricular, bilateral occipital, right posterior meningeal artery and marginal tentorial branch of right ICA arteries with venous reflux into the right torcula, straight sinus and posterior superior sagittal sinus alongwith cortical venous reflux into occipital and parietal veins. Pseudophlebitic pattern is not seen.

IMAGES:

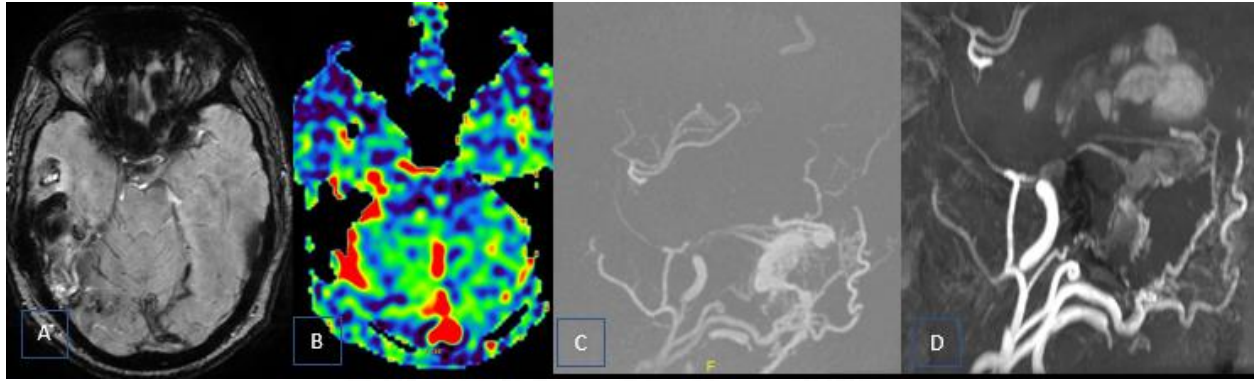


Figure 24: SWAN(A) and ASL (B) showing the fistula at right TS-SS junction with retrograde extension and cortical venous reflux. Silent MRA sagittal MIP images(C) showing arterial feeders from right MMA, Occipital, ascending pharyngeal and posterior auricular branches of ECA. Venous reflux into cortical veins is also seen. TOF MRA sagittal MIP images (D) showing arterial feeders from right occipital, ascending pharyngeal, MMA and posterior auricular branches of right ECA with cortical venous reflux. Focus of acute bleed in right temporal lobe is seen in SWAN and TOF images.

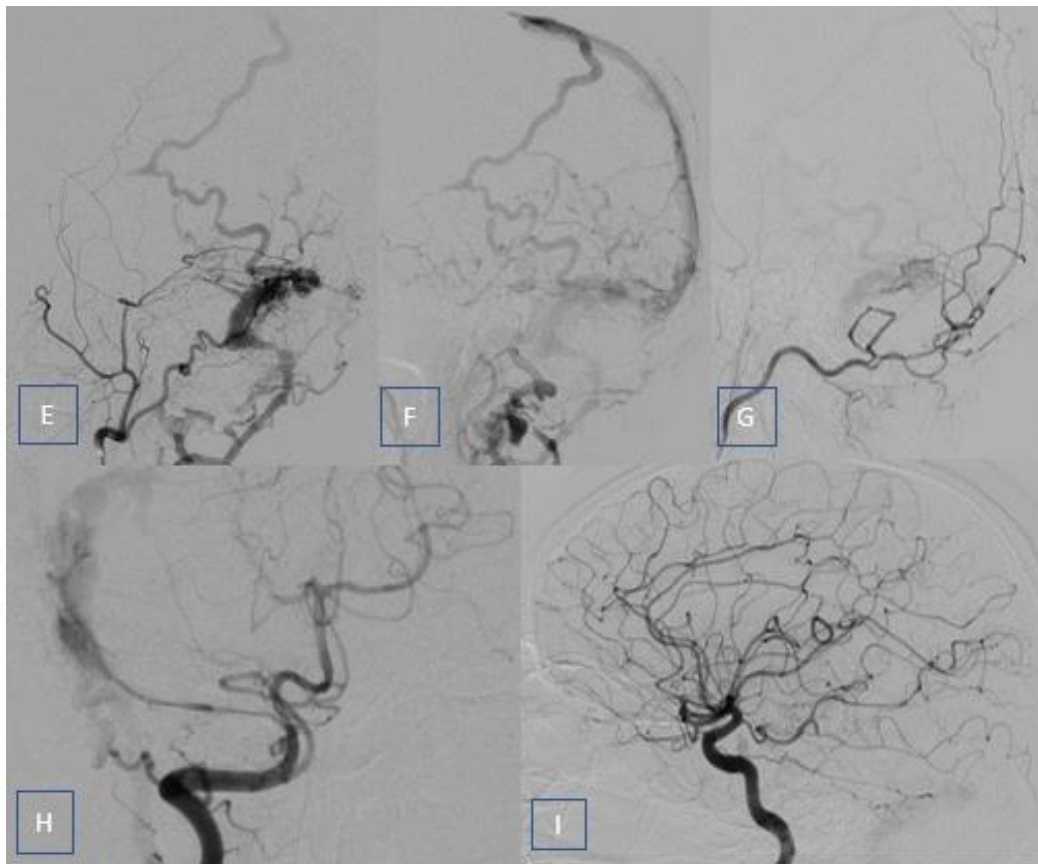


Figure 25: Right IMA selective angiogram lateral view(E) showing arterial feeders from right MMA, ascending pharyngeal, occipital and posterior auricular branches. Venous drainage(F) is seen retrogradely into the torcula, posterior Superior sagittal sinus and cortical veins. Left occipital artery(G), right posterior meningeal artery(H) and marginal tentorial branch of right ICA(I) are also seen supplying the fistula in respective selective angiograms.

CASE 9:

Case summary:

30 year old male presented with symptoms of headache and double vision since 09 months. He had one episode of sever headache with vomiting and altered sensorium for which he was admitted in local hospital. Imaging done outside revealed left temporal lobe hematoma and thrombosis of left transverse sinus and sigmoid sinus. Then he underwent left decompressive craniectomy. Follow up imaging revealed suspected right transverse sinus – sigmoid sinus junction dAVF. He also has intermittent blurring of vision and heaviness in left ear. Then he was referred to SCTIMST for further management. On examination, he was conscious with GCS of 15/15, Fundus showed bilateral minimal Disc swelling and IOP of 14 mm hg bilaterally. No deficits were noted.

MRI:

MRI with MR angiographic protocol revealed extensive Cognard Type 2A+B dAVF at the right transverse sinus- sigmoid sinus and left transverse sinus region. ASL and SWAN showed hyperintense venous signals in right transverse sinus, sigmoid sinus and left transverse sinus region with extension into the torcula, straight sinus, Vein of Galen and superior sagittal sinus. SWAN showed pseudophlebitic pattern. Silent and TOF MRA showed fistula located in right transverse sinus, sigmoid sinus and left transverse sinus region. Arterial feeders were noted from right MMA, right ascending pharyngeal, right posterior auricular, bilateral occipital artery and marginal tentorial branch of right ICA. Venous drainage was seen from the right transverse sinus into the torcula, straight sinus, Vein of Galen and posterior superior sagittal sinus alongwith cortical venous reflux.

DSA:

6 vessel DSA showed Cognard Type 2A+B dAVF at the right transverse sinus, sigmoid sinus and left transverse sinus region with arterial feeders from right MMA – petrous, petrosquamous branches, right ascending pharyngeal, right posterior auricular, bilateral occipital and marginal tentorial branch of right ICA arteries with venous reflux into the torcula, straight sinus and posterior superior sagittal sinus alongwith cortical venous reflux into temporal, occipital and parietal veins. Pseudophlebitic pattern is seen.

IMAGES:

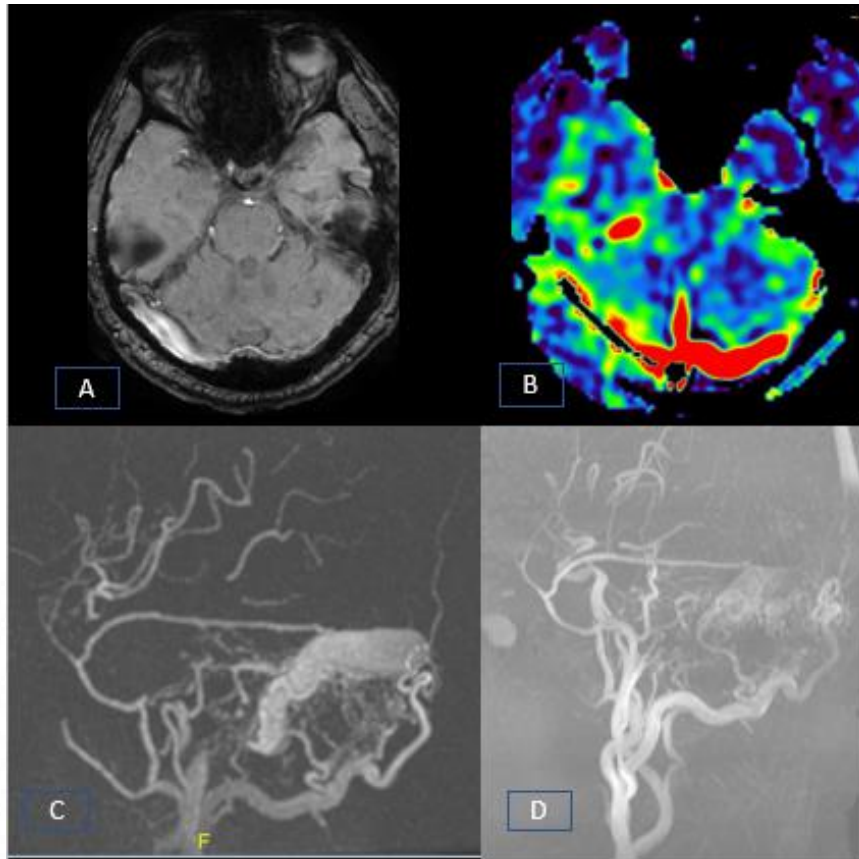


Figure 26: SWAN(A) and ASL (B) showing the location of fistula in right transverse sinus- sigmoid sinus and left transverse sinus regions with retrograde flow and cortical venous reflux. Silent and TOF MRA sagittal MIP images(C,D) showing arterial feeders from right MMA, posterior auricular, ascending pharyngeal, occipital, marginal tentorial branches to the fistula.

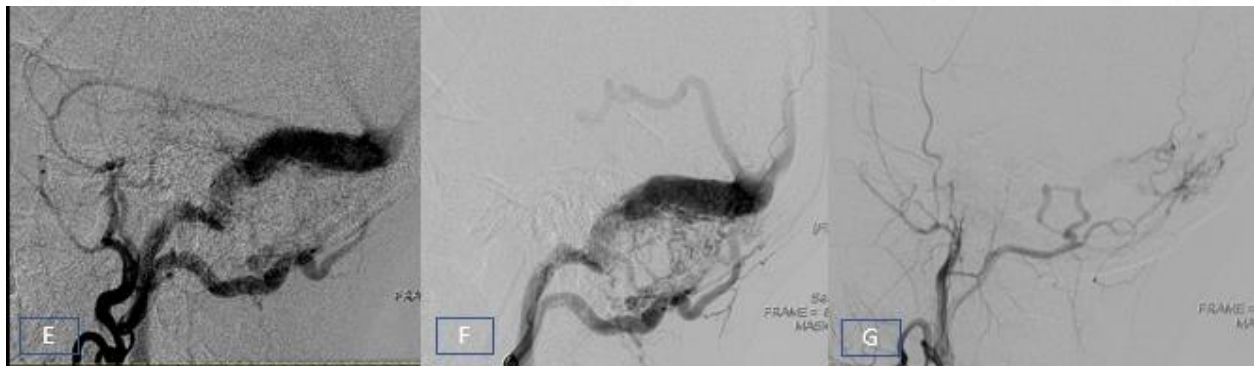


Figure 27: DSA right ECA angiogram lateral view (E) showing arterial feeders from left MMA – petrous and petrosquamous branches, right posterior auricular, right occipital and right ascending pharyngeal arteries. Right occipital artery selective angiogram(F) showing network of feeders from right occipital artery along with retrograde venous flow into the posterior superior sagittal sinus, straight sinus and vein of Galen. Left occipital artery selective angiogram (G) showing left occipital arterial feeder to the fistula,

CASE 10:

Case summary:

61 year old male presented with complaints of headache – severe intensity, not controlled with medications. MRI revealed few tortuous flow voids in basifrontal region. DSA was done outside which revealed dAVF in right frontal region. He also developed multiple episodes of focal right sided tonic clonic seizures. Then he was referred to sCTIMST for further management. On examination, he was confused, GCS of 10/15, Weakness of right upper and lower limbs were noted. Pupils were equal, normal and reactive.

MRI:

MRI with MR angiographic protocol revealed Cognard Type 4 dAVF at the right basifrontal region. ASL and SWAN showed subtle hyperintense venous signals in right basifrontal region with direct extension into the frontal cortical veins. ASL showed postictal hyperperfusion in left temporal and parietal lobes. SWAN showed no pseudophlebitic pattern. Silent and TOF MRA showed fistula located in right basifrontal region. Arterial feeders were noted from bilateral ophthalmic artery and bilateral terminal branches of internal maxillary artery and direct cortical venous reflux into frontal cortical veins with venous sacs.

DSA:

6 vessel DSA showed Cognard Type 4 dAVF at the right basifrontal region with arterial feeders from terminal branches of bilateral internal maxillary artery and recurrent meningeal branches of bilateral ophthalmic arteries. Direct cortical venous reflux into the basifrontal cortical veins noted with venous sacs. Pseudophlebitic pattern is not seen.

IMAGES:

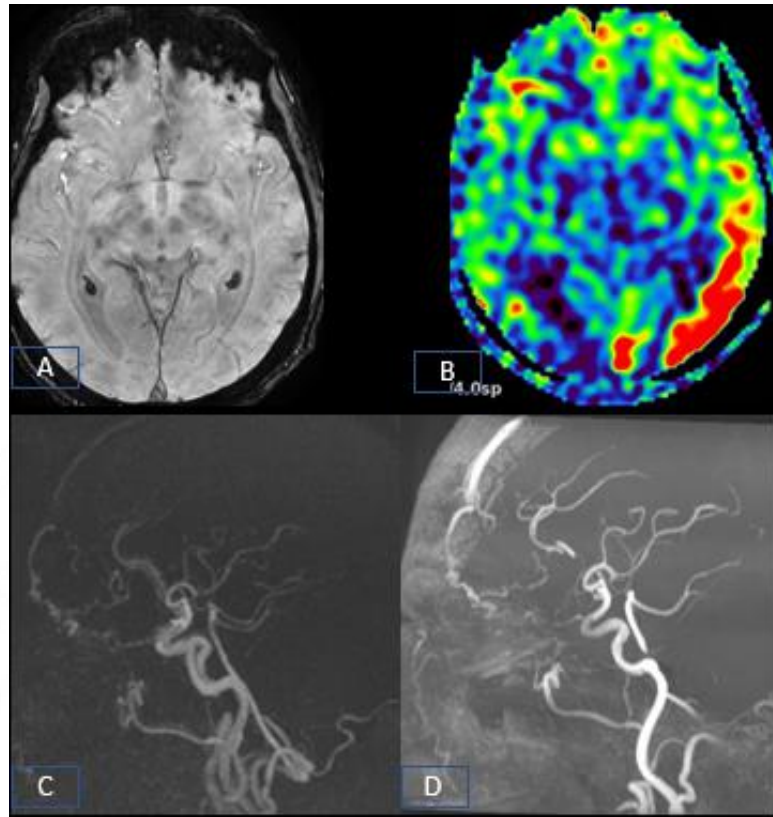


Figure 28: SWAN(A) and ASL (B) showing subtle hyperintensities in right basifrontal region with extension of the signal into the frontal cortical vein. Silent and TOF MRA sagittal MIP images (C, D) showing arterial feeders from bilateral ophthalmic and internal maxillary arteries with direct frontal cortical venous reflux alongwith venous sacs.

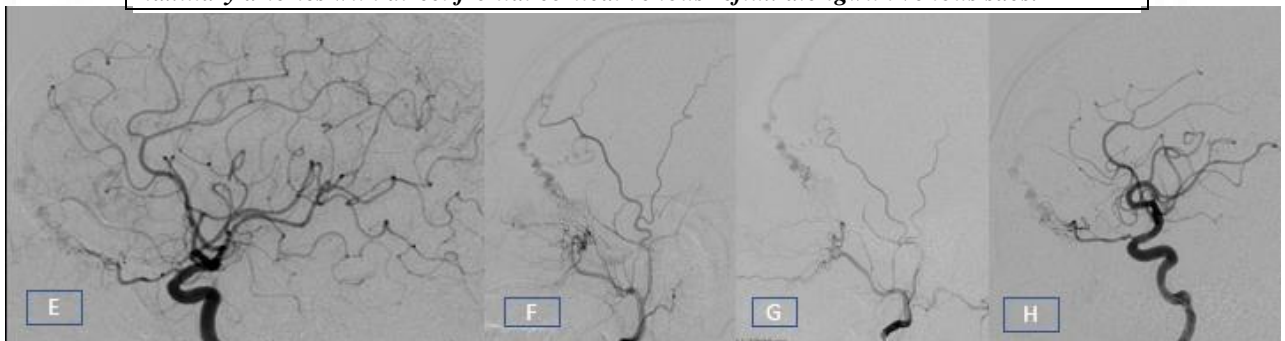


Figure 29: DSA Right and left ICA angiograms(E, H) showing arterial feeders from recurrent meningeal branches of ophthalmic arteries supplying the fistula. Right and left IMA selective angiograms lateral views(F,G) showing feeders from terminal branches of IMA supplying the fistula with direct cortical venous reflux into basifrontal cortical veins and venous sacs.



ANNEXURES



INFORMATION SHEET

TITLE OF THE STUDY: Imaging of Intracranial dural arteriovenous fistula – Comparison of advanced MRI angiographic sequences with Digital Subtraction Angiography**Study number:** _____**Participant's name:** _____**Date of Birth / Age (in years):** _____**Son/daughter of** _____

You have been informed that there is an abnormal communication between the arteries and veins (which is called Dural Arterio Venous Fistula) in your brain. For this you will have undergone or will be undergoing a digital subtraction angiography (DSA) test and Magnetic Resonance Imaging (MRI) as a part of clinical evaluation of your disease. This is to plan the treatment or for follow up your disease.

You are requested to participate in a study to evaluate the role of advanced Magnetic Resonance Imaging sequences in diagnosis of Dural arteriovenous fistulas. While participating in this study, only the imaging data from the MRI and DSA investigations you have undergone for your treatment purpose will be used. Participating in this study will in no way influence your treatment decisions. The benefit that you may incur from this study is, if this new MRI technique is found useful, during subsequent reviews/ follow up your further imaging follow up can be limited to just an MRI rather than performing an invasive evaluation like DSA.

What are DSA and MRI and do they have any harmful effects?

DSA (Digital subtraction angiography) test is an advanced imaging technique where the blood flow to your brain will be evaluated by injecting a dye into the arteries to the brain through a small tube which will be inserted through the artery in your thigh. X-Rays will be obtained during the procedure which will clearly show the abnormal connections between arteries and veins if they exist. You will not experience much pain as an injection will be given on your thigh prior to the procedure to make it numb. You will not feel any pain during the rest of the procedure. In rare cases some people may have allergic reaction to the dye. There is also a very small risk of injury to the blood vessel and slight chance of bleeding at site of puncture. This test is vital in diagnosis of your condition and is also the means of treatment if planned subsequently.

MRI is an advanced imaging technique, which uses certain waves and magnetic fields to image body part. It does not involve any ionizing radiation. There will be no administration of any type of drug or medicine during the study. Some patients may develop claustrophobia (Fear of closed spaces etc.) due to closed space and noise. This investigation is not to be done for patient with metallic implants, pacemakers. This MRI is being done as a part of clinical evaluation of your disease; however certain data from this study will be used for research purpose to compare with the DSA study which you have already undergone/ will undergo shortly.

If you take part what will you have to do?

This study will only analyse the results of the routinely ordered imaging investigations you will undergo during treatment and follow up of your illness. You will not be required to do anything apart from the regular follow up that will be advised to you.

Can you withdraw from this study after it starts?

Your participation in this study is entirely voluntary and you are also free to decide to withdraw permission to participate in this study. If you do so, this will not affect your usual treatment at this hospital in any way.

What will happen if you develop any study related injury?

This study only analyzes the results of your investigation and treatment details and thus we do not expect any injury to happen to you but if you do develop any side effects or problems due to the study, these will be treated at this institute by the experienced team of medical professionals. We are unable to provide any monetary compensation, however.

Will you have to pay for the study?

The study will only analyse the results of the investigations and treatment which you will undergo in natural process of your treatment for AVM at this institute and no extra cost will be borne by you for this particular study.

What happens after the study is over?

You may or may not benefit from this study. If the study is found useful then during subsequent reviews you can be evaluated just by non invasive MRI rather than undergo a more invasive DSA.

Will your personal details be kept confidential?

The results of this study may be published in a medical journal but you will not be identified by name in any publication or presentation of results. However, your medical notes may be reviewed by people associated with the study, without your additional permission, should you decide to participate in this study.

Will the study have any adverse effects on pregnancy if the patient is pregnant?

Both the DSA and MRI will be done only for a patient who require these studies as part of their treatment or follow up.

There is risk related to ionizing radiation in DSA study which can be harmful to the fetus. Regarding safety of MRI in pregnancy, to date there has been no indication that the use of clinical MRI during pregnancy has produced deleterious effects. However, these investigations will be done in a pregnant patient only in situation where the mother's life is in danger and she requires treatment or an immediate follow up study is warranted. For these tests to be done she has to give her informed written consent.

Moreover this study is a comparison of the imaging data from the DSA and MRI tests you have or is about to undergo as part of the treatment. You are not undergoing these tests for the study per se.

If you have any further questions, please ask Dr. Arun Prasad (tel: 9629974150) or email: docarun1980@sctimst.ac.in

IEC Member Secretary

Dr. Mala Ramanathan

Phone Number : 0471 2524234

INFORMATION SHEET – FOR GUARDIANS OF MINORS

TITLE OF THE STUDY: Imaging of IntraCranial dural arteriovenous fistula – Comparison of advanced MRI angiographic sequences with Digital Subtraction Angiography

Study number:

Participant's name: _____

Date of Birth / Age (in years): _____

Son/daughter of _____

You have been informed that there is an abnormal communication between the arteries and veins (Dural Arteriovenous fistula), within your child's brain. Your child will have undergone or will be undergoing a digital subtraction angiography (DSA) test and Magnetic Resonance Imaging (MRI) as a part of clinical evaluation of the disease to plan treatment or for follow up of disease.

You are requested to allow your child to participate in a study to evaluate the role of advanced Magnetic Resonance Imaging sequences in diagnosis of Dural arteriovenous fistulas. While participating in this study, only the imaging data from the MRI and DSA investigations your child has undergone for treatment purpose will be used. Participating in this study will in no way influence the treatment decisions. The benefit that your child may incur from this study is, if this new MRI technique is found useful, during subsequent reviews/ follow up further imaging can be limited to just an MRI rather than an invasive test like DSA.

What are DSA and MRI and do they have any harmful effects?

DSA (Digital subtraction angiography) test is an advanced imaging technique where the blood flow to brain is evaluated by injecting a dye into the arteries of the brain through a small tube inserted through an artery in the thigh. X-Rays obtained during the procedure will clearly show the any abnormal connections between arteries and veins. The patient will not experience much pain as an injection will be given on thigh prior to the procedure to make it numb. No pain will be felt during the rest of the procedure. In rare cases some people may have allergic reaction to the dye. There is also a very small risk of injury to the blood vessel and slight chance of bleeding at site of puncture. This test is vital in diagnosis of your condition and is also the means of treatment if subsequently planned.

MRI is an advanced imaging technique which uses certain waves and magnetic fields to image body part. It does not involve any ionizing radiation. There will be no administration of any type of drug or medicine during the study. Some patients may develop claustrophobia (Fear of closed spaces etc.) due to closed space and noise. This investigation will not be done in patients with metallic implants or pacemakers.

MRI and DSA will be done as a part of clinical evaluation of your child's disease. This study will require image data from the tests that your child will undergo as part of disease evaluation.

If you take part what will your child have to do?

This study will only analyse the results of the routinely ordered imaging investigations your child will undergo during treatment and follow up of your illness. Your child will not be required to do anything apart from the regular follow up that will be advised to you.

Can your child withdraw from this study after it starts?

Your child's participation in this study is entirely voluntary and you are also free to decide to withdraw permission to participate in this study. If you do so, this will not affect your child's usual treatment at this hospital in any way.

What will happen if your child develops any study related injury?

This study only analyses the results of investigation and treatment details and thus we do not expect any injury to happen to you but if you do develop any side effects or problems due to the study, these will be treated at this institute by the experienced team of medical professionals. We are unable to provide any monetary compensation, however.

Will you have to pay for the study?

The study will only analyse the results of the investigations and treatment which your child will undergo in natural process of your treatment for AVM at this institute and no extra cost will be borne by you for this particular study.

What happens after the study is over?

Your child may or may not benefit from this study. If the study is found useful then during subsequent reviews evaluation can be limited to just a non invasive MRI rather than more invasive DSA.

Will your child's personal details be kept confidential?

The results of this study may be published in a medical journal but your child will not be identified by name in any publication or presentation of results. However, the child's medical notes may be reviewed by people associated with the study, without your additional permission, should you decide to participate in this study.

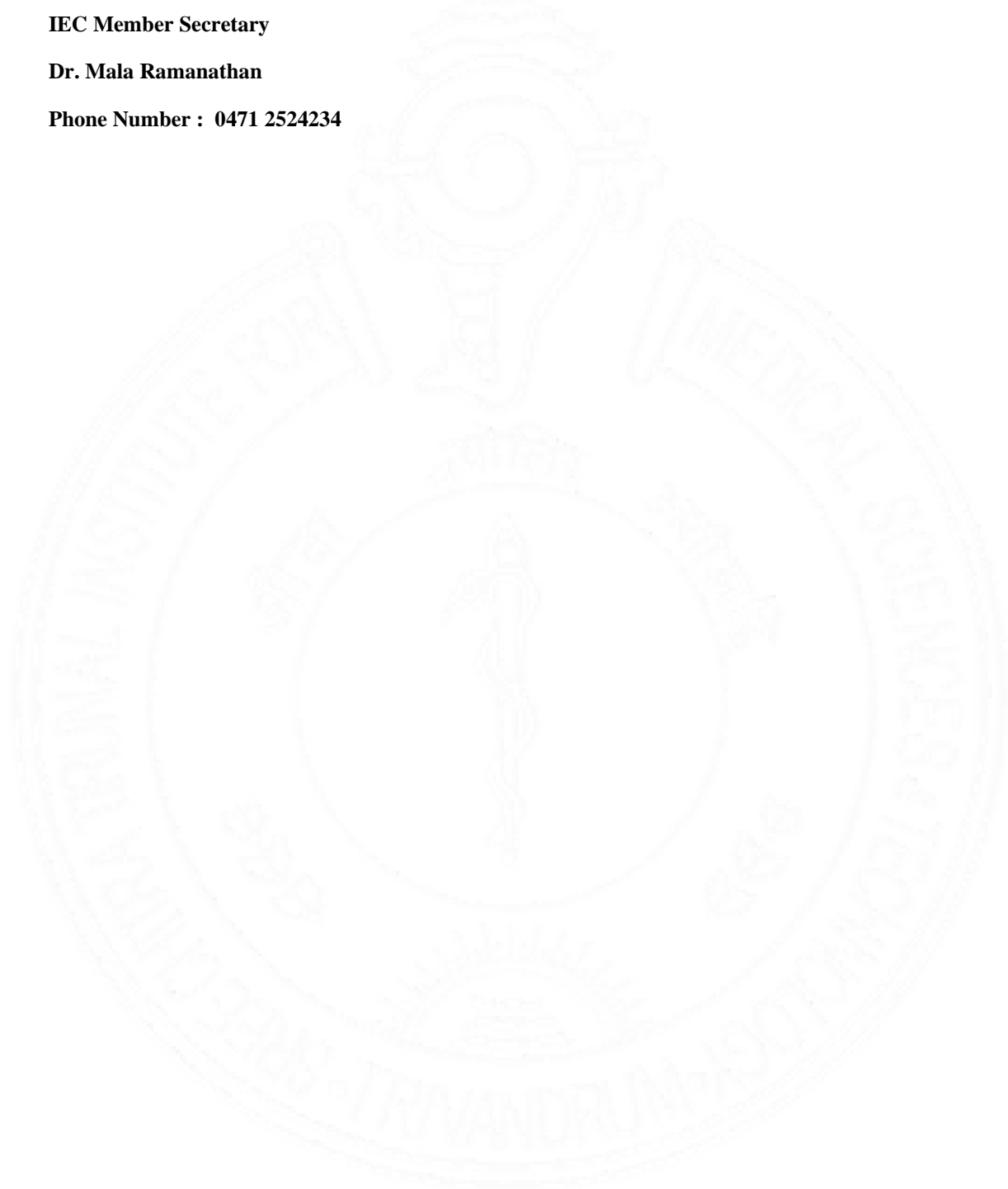
If you have any further questions, please ask

Dr. Arun Prasad (tel: 9629974150) or email: docarun1980@sctimst.ac.in

IEC Member Secretary

Dr. Mala Ramanathan

Phone Number : 0471 2524234



CONSENT FORM

TITLE OF THE STUDY: Imaging of IntraCranial dural arteriovenous fistula – Comparison of advanced MRI angiographic sequences with Digital Subtraction Angiography

Study number: _____

Participant's name: _____

Date of Birth / Age (in years): _____

Son/daughter of _____

(Please tick boxes) •

I declare that I have read the above information provided to me regarding the study – “**Imaging of IntraCranial dural arteriovenous fistula – Comparison of advanced MRI angiographic sequences with Digital Subtraction Angiography**” -- and have clarified any doubts that I had. []

I understand that my participation in this study is entirely voluntary and that I am free to withdraw the permission to continue my participation at any time without affecting my usual treatment or my legal rights. []

I understand that the study staff and institutional ethics committee members will not need my permission to look at my health records even if I withdraw from the trial. I agree to this access. []

I understand that my identity will not be revealed in any information released to third parties or published []

I voluntarily agree to take part in this study []

I have received a copy of this signed consent form []

Name: _____

Signature: _____

Date: _____

Name of witness: _____

Relation to participant: _____

Date: _____

(Person Obtaining Consent) I, _____ attest that the requirements for informed consent for the medical research project described in this form have been satisfied. I have discussed the research project with the participant and explained to him or her in nontechnical terms all of the information contained in this informed consent form, including any risks and adverse reactions that may reasonably be expected to occur. I further certify that I encouraged the participant to ask questions and that all questions asked were answered.

Name and Signature of Person Obtaining Consent

CONSENT FORM - MINORS

TITLE OF THE STUDY: Imaging of Intracranial dural arteriovenous Fistula – comparing advanced MRI angiographic sequences with Digital subtraction angiography.

Study number:

Participant's name: _____

Date of Birth / Age (in years): _____

Son/daughter of _____

(Please tick boxes) •

I declare that I have read the above information provided to me regarding the study – “**Imaging of Intracranial dural arteriovenous Fistula – comparing advanced MRI angiographic sequences with Digital subtraction angiography**” -- and have clarified any doubts that I had. []

I understand that my child's participation in this study is entirely voluntary and that I am free to withdraw the permission to continue participation at any time without affecting my child's usual treatment or legal rights. []

I understand that the study staff and institutional ethics committee members will not need my permission to look at my child's health records even if I withdraw from the trial. I agree to this access. []

I understand that my child's identity will not be revealed in any information released to third parties or published []

I voluntarily agree to take part in this study []

I have received a copy of this signed consent form []

Name: _____

Signature: _____

Date: _____

Name of witness: _____

Relation to participant: _____

Date: _____

(Person Obtaining Consent) I, _____ attest that the requirements for informed consent for the medical research project described in this form have been satisfied. I have discussed the research project with the participant and guardian and explained to him or her in nontechnical terms all of the information contained in this informed consent form, including any risks and adverse reactions that may reasonably be expected to occur. I further certify that I encouraged the participant to ask questions and that all questions asked were answered.

Name and Signature of Person Obtaining Consent

Study Title: Imaging of Intracranial dural arteriovenous fistula – Comparison of advanced MRI angiographic sequences with Digital Subtraction Angiography

Intracranial DAVF reporting Format for 3DTOF MRA; SILENT MRA; SWAN, ASL and DSA.

<p>a) Reallocated Anonymized Image identification number:</p> <p>b) Study / Sequence evaluated: MRI – (3DTOF MRA; SILENT MRA; SWAN, ASL)/DSA</p> <p>c) Investigator analyzing study: Date of Image analysis:</p> <p>d) Quality of image:</p>
--

CLINICAL:
Age:
Sex:
Chief complaints/duration of symptoms:
History of presenting complaints:
Hemorrhagic presentation/Non hemorrhagic presentation:
Seizures: Transient ischemic attacks (TIA): Neurovascular deficits:
Past history/treatment history:
EXAMINATION:
General examination:
System examination:
Neurological examination:

IMAGING FINDINGS:

MRI parameters:

1. Presence of SAH/ICH
2. Location:
3. Arterial Feeders:
4. Venous drainage: Sinus/cortical.....
5. Cortical vein reflux:(Yes/No):
6. Architecture

Cognard Classification:

Borden Classification:

7. Pseudophlebitic pattern in SWAN

DSA parameters:

1. Location
2. Architecture
Cognard Classification:
Borden Classification:
3. Feeding Artery
4. Venous drainage: Sinus /cortical
5. Cortical venous reflux: Present/absent
6. Pseudophlebitic pattern

A preliminary work of the thesis has been published in the month of May 2021 in the journal of Clinical radiology. The abstract of the published article is appended below.



Utility of silent magnetic resonance angiography in the evaluation and characterisation of intracranial dural arteriovenous fistula

A. Prasad Balasubramanian^a, S. Kumar Kannath^{a,*}, J. Enakshy Rajan^a, G. Singh^b, C. Kesavadas^a, B. Thomas^a

^a Department of Imaging Sciences and Interventional Radiology, Sree Chitra Tirunal Institute of Medical Sciences and Technology, Trivandrum, Kerala, India

^b Achutha Menon Centre for Health Science Studies, Trivandrum, Kerala, India

ARTICLE INFORMATION

Article history:

Received 22 January 2021

Accepted 5 May 2021

AIM: To evaluate the utility of silent magnetic resonance angiography (MRA) in the diagnosis, characterisation, and therapeutic planning of intracranial dural arteriovenous fistula (DAVF).

MATERIALS AND METHODS: Twenty consecutive patients with DAVF were enrolled prospectively and were evaluated using silent MRA and digital subtraction angiography (DSA) as a part of routine work-up. The diagnosis and location of fistula, Borden and Cognard classification, entire arterial feeders, and venous drainage were analysed. A therapeutic strategy was formulated, and the accessible route and vessel were predicted, which was confirmed on endovascular treatment.

RESULTS: Silent MRA was 100% sensitive and accurate for location and classification of fistulas. Silent MRA showed a sensitivity of 82% and 76.5% for entire arterial feeders and draining veins, which improved to a sensitivity of 90% and 94% when prominent feeders and immediate venous drainage was considered. Among the missed veins, thrombosed sinus, slow sinus flow, small calibre, reduced image quality were the causes. The therapeutic decision matched with DSA in all cases and silent MRA accurately identified the potential accessible feeder in 94% cases.

CONCLUSION: Silent MRA is a promising MR technique that can provide both diagnostic and therapeutic information similar to that obtained from DSA.

© 2021 The Royal College of Radiologists. Published by Elsevier Ltd. All rights reserved.

* Guarantor and correspondent: S. Kumar Kannath, Department of Imaging sciences and Interventional Radiology, Sree Chitra Tirunal Institute of Medical Sciences and Technology, India. Tel.: +91 471 2524117.

E-mail address: drsanthoshkannath@gmail.com (S.K. Kannath).

<https://doi.org/10.1016/j.crad.2021.05.008>

0009-9260/© 2021 The Royal College of Radiologists. Published by Elsevier Ltd. All rights reserved.

Please cite this article as: Balasubramanian AP et al., Utility of silent magnetic resonance angiography in the evaluation and characterisation of intracranial dural arteriovenous fistula, Clinical Radiology, <https://doi.org/10.1016/j.crad.2021.05.008>



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

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

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

SCT/IEC/1403/JULY-2019

30.08.2019

Dr. Arun Prasad B
Senior Resident, Department of IS & IR
SCTIMST, Thiruvananthapuram

Dear Dr. Arun Prasad,

The Institutional Ethics Committee reviewed and discussed your application to conduct the study entitled "IMAGING OF INTRACRANIAL DURAL ARTERIOVENOUS FISTULA – COMPARISON OF ADVANCED MRI ANGIOGRAPHIC SEQUENCES WITH DIGITAL SUBTRACTION ANGIOGRAPHY (IEC/1403)" on 26th July, 2019.

The following documents were reviewed:

Original submission

1. Covering Letter addressed to the Chairperson, IEC, SCTIMST dated 28.06.2019 with checklist
2. TAC Approval Letter
3. IEC Application Form
4. Project Proposal
5. Proforma
6. List of abbreviations
7. Information Sheet and Consent Form in English and Malayalam
8. Information Sheet for Guardians of minors and Consent Form for minors in English and Malayalam
9. Covering Letter from the Co-PI
10. CV of Principal Investigator and Co-Principal Investigators

Revised submission

1. Covering Letter addressed to the Member Secretary, IEC, SCTIMST dated 26.08.2019 with checklist
2. Copy of IEC Recommendations Letter dated 07.08.2019
3. TAC Approval Letter
4. IEC Application Form
5. Project Proposal
6. Proforma
7. List of abbreviations
8. Information Sheet and Consent Form in English and Malayalam
9. Assent Form in English and Malayalam
10. Information Sheet for Guardians of minors and Consent Form for minors in English and Malayalam
11. Covering Letter from the Co-PI
12. CV of Principal Investigator and Co-Principal Investigators

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

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

IEC Decision

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

Remarks:

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

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

Sincerely,



Mala Ramanathan
Member Secretary, IEC



ABBREVIATIONS



ABBREVIATIONS

dAVF	-	Dural Arteriovenous Fistula
CT	-	Computed Tomography
CTA	-	Computed Tomography Angiography
MRI	-	Magnetic Resonance Imaging
MRA	-	Magnetic Resonance Angiography
DSA	-	Digital Subtraction Angiography
SWI	-	Susceptibility Weighed Imaging
ASL	-	Arterial Spin labelling
SCTIMST	-	Sree Chitra Tirunal Institute for Medical Sciences and Technology
TOF	-	Time of Flight
CEMRA	-	Contrast enhanced magnetic resonance angiography
NEX	-	Number of excitations
TE	-	Echo time
TR	-	Repetition time
CVR	-	Cortical venous reflux
MMA	-	Middle Meningeal artery
IMA	-	Internal maxillary artery
ICA	-	Internal carotid artery
VA	-	Vertebral artery
SWAN	-	Susceptibility Weighted Angiography



Plagiarism Checker X - Report

Originality Assessment

Overall Similarity: **2%**

Date: Jul 13, 2021

Statistics: 203 words Plagiarized / 9671 Total words

Remarks: Low similarity detected, check your supervisor if changes are required.

Sources

- 1 https://www.researchgate.net/publication/38015067_Evaluation_of_Dural_Arteriovenous_Fistulas_with_4D_Contrast-Enhanced_MR_Angiography_at_3T
INTERNET
1%

- 2 https://www.researchgate.net/publication/26831392_Intracranial_dural_arteriovenous_fistulas_A_Review
INTERNET
1%

- 3 https://www.researchgate.net/publication/26245475_Cranial_Dural_Arteriovenous_Fistula_Diagnosis_and_Classification_with_Time-Resolved_MR_Angiography_at_3T
INTERNET
<1%

- 4 <http://dspace.sctimst.ac.in/jspui/bitstream/123456789/11027/1/6984.pdf>
INTERNET
<1%

- 5 <https://www.sciencedirect.com/science/article/pii/S0140673601409834>
INTERNET
<1%

