Case Report

A rare case of cerebral hemorrhage due to left fronto-parietal mixed pial-dural AV-malformation, Spleitzer Martin grade 5 - A case report

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Abstract

Cerebral arteriovenous malformation (AVM) is a complex network of vascular channels consisting of arterial feeders, a nidus and enlarged venous drainage. AVMs usually increase in size with time, but may rarely obliterate; spontaneous angiographic regression occurs in less than 1.5% of cerebral AVMs. Several causes of spontaneous regression have been postulated such us hemodynamic alterations due to hemorrhage, hypercoagulability, atherosclerosis, and thromboembolism from associated aneurysms. In this report we describe a case of cerebral hemorrhage due to left fronto-parietal AVM with predominant middle cerebral artery and anterior cerebral artery feeders, minor left external carotid artery feeders, mixed pial-dural AVM – Spleitzer Martin Grade 5.

Key words

Cerebral hemorrhage, Mixed pial-dural AV-malformation, Spleitzer Martin grading.

Introduction

Brain arteriovenous malformations (bAVMs) are very rare vascular lesions which present with spontaneous intracranial hemorrhage (ICH), seizures, or headache in young adults [1–3]. Most of patients are diagnosed with incidental

asymptomatic bAVMs after images of brain were obtained for other reasons [4]. The latest treatment options include conservative management, surgical resection, stereotactic radiosurgery (SRS), endovascular embolization, or combinations of these treatments (multimodal therapy). The primary goal of the treatment is to prevent hemorrhagic. The risks of these treatments must be weighed against the natural history risks [5].

Imaging Diagnosis and Evaluation

The definitive diagnosis of a bAVM is made by digital subtraction angiography (DSA), although many bAVMs can be diagnosed by computed tomography (CT) and MR imaging (MRI), including angiographic imaging (CTA and MRA) [5].

Spetzler-Martin grading:

Designed to assess the patient's risk of neurological deficit based on characteristics of the AVM itself. Based on this system, AVMs may be classified as grades 1 - 5.

AVM size	Adjacent eloquent complex	Draining veins
< 3 cm	Non – eloquent= 0	Superficial only = 0
3 – 6 cm	✪ Eloquent= 1	Deep veins = 1
> 6 CM		

- ✪ "Eloquent cortex" is area of cortex that, if removed will result in loss of sensory processing or linguistic ability, minor paralysis, or paralysis. The risk of post-surgical neurological deficit (difficulty with language, motor weakness, vision loss) increases with increasing grade [6].

Treatment Modalities

The definitive treatment of bAVMs is to complete elimination of the nidus and the arteriovenous shunt. Partial nidal obliteration will not reduce hemorrhage risk. There are three therapeutic tools to achieve these goals.

1) The first is microsurgical resection. This may be performed primarily or after endovascular embolization to reduce bleeding risks during surgery and to facilitate complete removal.
2) The second is SRS. This also is done primarily or after embolization to reduce nidal volumes and potentially to improve nidal obliteration rates.
3) The final method is endovascular embolization itself. Although this is most often used as a precursor to microsurgery or radiosurgery, in some cases it may be definitive therapy [5].

Incidence

Cerebral arteriovenous malformations (AVM) are a complex network of vascular channels consisting of arterial feeders, a nidus and an enlarged venous drainage. Their incidence is 1.1/1.3 per 100.000 persons-years [7, 8]. Inheritance remains uncertain [9], although some familiar cases have been described [10].

Clinical presentation

Diagnosis is established early in life, between second and fourth decade [8]. An intracerebral hemorrhage is the main mode of presentation [11, 12, 13, 14], with an incidence of 0.51 per 100.000 person years [7, 8].

Symptoms

More than 50% patients have ICH. Among AVM's, 20-25% have focal/gen seizures. They may have Localized headache due to increased blood supply around AVM. There may be weakness, speech or visual disturbances, depending on the location of bleed. AVMs usually increase in size with time, but may rarely obliterate [15]; recanalization after complete thrombosis can occur [16]. Spontaneous angiographic regression occurs in less than 1.5% of cerebral AVMs [17, 18].

Several causes of regression have been postulated, e.g. hemodynamic alterations due to spontaneous hemorrhage [19-24], surgery [25], the presence of a brain tumor [26], hypercoagulability [27, 28], atherosclerosis [15,

29, 30], and tromboembolism from associated aneurysms [29, 31].

Natural history

The most feared complication of brain arteriovenous malformations (bAVMs) is intracranial hemorrhage, and individuals harboring bAVMs are subjected to a life-long risk of hemorrhagic stroke [2, 3, 11, 14, 32, 33, 34].

Management of these lesions is complicated because the bAVMs form a very heterogeneous group of lesions. The variable locations, morphologies, and angioarchitectural characteristics of arteriovenous malformation (AVM), imparts a different risk of hemorrhage for each patient [35, 36, 37, 38, 39, 40, 41, 42], requiring individualized treatment decisions. Key among the goals of treating bAVMs is to reduce the risk of future hemorrhagic stroke. However, the risks associated with treating a given bAVM patient also vary [43, 44, 45, 46, 47, 48] and, consequently, must be weighed individually against the natural history of hemorrhage anticipated in that particular patient.. Reported yearly hemorrhage rates may be as low as 2% or as high as 32.6% [2, 3, 11, 32, 34, 49, 50].

The correct management of AVMs can therefore vary from simple observation to aggressive multimodality treatment aimed at total AVM obliteration. Because risks of treatment must be weighed against those of conservative management of bAVMs, one should have a better understanding of the natural history and factors predictive of hemorrhage in bAVM patients.

Case report

45 years, male laborer, admitted for two episodes of seizures within 24 hours. History of presenting illness - Patient was apparently asymptomatic a day earlier, then he had two episodes of seizures, history of tongue bite +. History of frothing from mouth +, history of deviation of angle of mouth to left, history of headache prior to seizure were present. History of loss of consciousness + lasting for 30 mins and Post-ictal confusion+, No history of limb weakness, No history of vomiting/ nausea/ diplopia, No history of dizziness/ vertigo, No history of ear pain/trauma to head or spine, No history of breathlessness/ cough/ syncope/ palpitations/ sweating, Next day one episode of GTCS, with deviation of head to right +, frothing +, with loss of consciousness, lasting for 30 mins , confusion +, No Aura, no weakness of limbs, no history of bladder or bowel disturbances, no history of head injury, no history of bleeding or clotting disorder. History of past illness – there was history of sudden onset burning sensation in the occipital region 4 years back, followed by profuse sweating and non-projectile vomiting. Admitted into local hospital, diagnosed hypertensive for the first time. Kept on anti-hypertenives, but patient non-compliant. N/K/C/O HTN, DM, Epilepsy, CAD, TB. There were no history of similar complaints in the family, Personal History was unremarkable.

General physical examination was unremarkable, Temp – Normal, Resp. Rate – 18/mt, PR 78/min regular in rhythm , Blood Pressure of 130/80 mmHg.

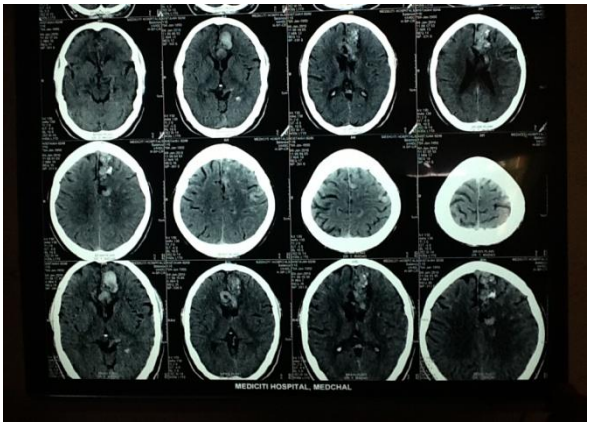
On CNS examination - Intellectual functions – normal, Speech – normal, Sensory system – normal, Cerebellar system – normal, Cranial nerves – normal, Motor system - Bulk in all limbs – normal, tone was normal, Power – 4/5 in all limbs, Superficial reflexes - present, Plantars – flexors. deep tendon reflexes - Biceps – rt - 3+, lt- 3+; Triceps - rt - 3+, lt- 3+; Knees - rt - 2+, lt- 2+; Ankles - rt - 3+, lt- 3+

Sensory system – Superficial sensations – touch, pain - present all over, Deep sensations – were normal Cerebellar system - No hypotonia, No Ataxia , no Swaying, No scanning speech, Finger Nose Finger Test – normal, Heel Knee Test: normal on both sides, Able to perform alternate rapid movements. No vasomotor changes, No trophic ulcers, Exam of other systems – CVS, RS and GIT was normal, and there was no thyromegaly.

On Investigation - Hb-13.6 g%, RBC count 7.2 mill/cumm, WBC count 9500 cells/cumm with Neutrophils - 80%, Lymphocytes - 16%, Eosinophils - 2%, Basophils - 0%, Monocytes - 2%, Platelet count – 3.51 lakhs/cu mm. ESR 1st hr - 12 mm, Blood Urea 15 mg%, Serum Creatinine - 0.6 mg, Complete Urine Examination (CUE) – normal, Random Blood Sugar - 120 mg/dl, Serum Electrolytes: Na – 133 m mol/L, K – 2.9 m Eq/L, CL – 92 mEq/L. LFT – Normal, Fasting Lipid Profile-WNL, ESR-10 mm/hr, , ECG-within normal limits. X-ray chest was normal.

Imaging studies – Figure - 1 CT scan: Acute hemorrhage in left parasagittal frontal lobe with adjacent multiple feeding vessels. **Figure 2A and 2B T2 Flair**: Multiple flow voids in left parasagittal frontal lobe. **Figure - 3 MR Angiogram TOF image**: Multiple vessels noted in parasagittal left frontal lobe with feeding arteries from ACA and MCA, **Figure - 4 DSA**- Feeding vessels from ACA and MCA & minor Left External Carotid Artery feeders, with mixed pial-dural AVM – Spleitzer Martin grade 5 and with AVM.

Figure – 1: CT scan: Acute hemorrhage in left parasagittal frontal lobe with adjacent multiple feeding vessels.



Diagnosis

Cerebral hemorrhage due to Lt. fronto-parietal AVM with predominant middle cerebral artery and anterior cerebral artery feeders, minor left external carotid artery feeders, mixed pial-dural AVM – Spleitzer Martin Grade 5.

Figure – 2A: T2 FLAIR: Multiple flow voids in left parasagittal frontal lobe.

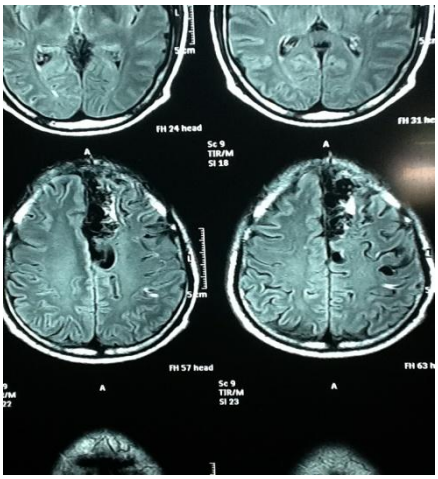


Figure - 2B: T2 FLAIR: Multiple flow voids in left parasagittal frontal lobe.

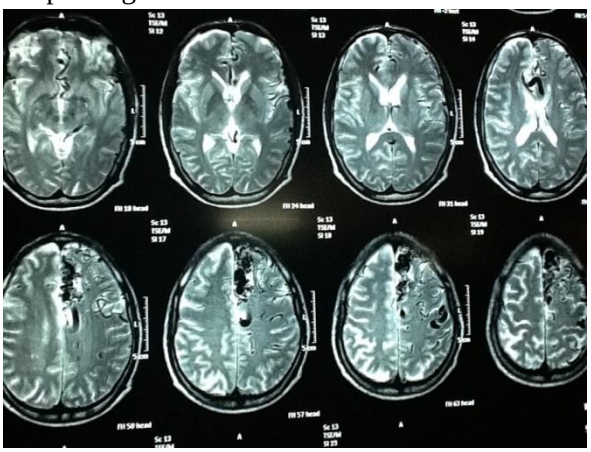


Figure – 3: MR angiogram TOF image: Multiple vessels noted in parasagittal left frontal lobe with feeding arteries from ACA and MCA.

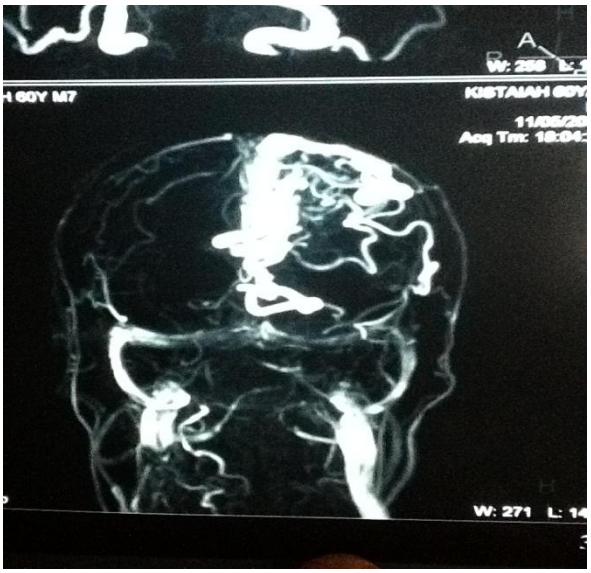
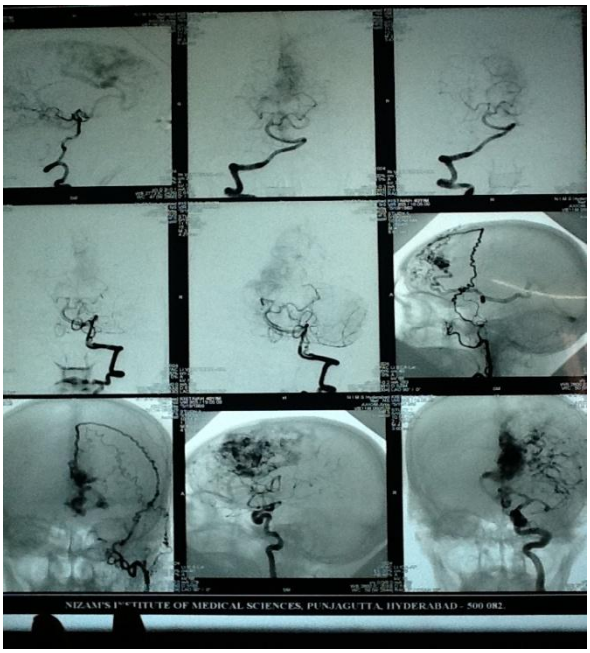


Figure – 4: DSA- Feeding vessels from ACA and MCA with AVM.



Discussion

AVMs are described as dynamic congenital anomaly [51], but the genetic polymorphisms involved in AVM development and risk of rupture remain controversial [52]. They originate from primary vestigial vessels and evolve during life [53] mainly by way of shear stress and blood pressure [54, 55]. The role of excessive angiogenesis and vascular remodelling has been described [49]. A number of possible mechanisms have been suggested by which venous hypertension may induce the formation of AVMs. Wilson [56, 57] suggested that diapedetic hemorrhage resulting from venous overload may trigger angiogenesis. Alternatively, a hemodynamic disturbance such as venous outflow restriction might open pre-existing arteriovenous connections, resulting in minute arteriovenous shunts that can enlarge over time. During evolution AVMs can bleed, obliterate or recanalize after a thrombotic event [16].

A number of possible mechanisms have been suggested by which venous hypertension may induce the formation of AVMs (Figure - 5). Wilson [45, 46] suggested that diapedetic hemorrhage resulting from venous overload may trigger angiogenesis. Alternatively, a hemodynamic disturbance such as venous outflow restriction might open pre-existing arteriovenous connections, resulting in minute arteriovenous shunts that can enlarge over time. During evolution AVMs can bleed, obliterate or recanalize after a thrombotic event [58].

Spontaneous and complete regression of an AVM is a rare phenomenon with a prevalence of 1–3% [17, 59, 60, 61]; 93 cases have been reported in the literature since 1949 [58]. Only four cases, above 65 years were described; this phenomenon is probably unusual in the older ages. AVMs are slightly prevalent in males with a 1.3 M/F ratio. In 10 patients the AVM were located in the posterior fossa, 2 AVMs in the brainstem; Forty out of ninety-three (42%) cases reported in Literature, experienced a primary intracerebral hemorrhage (ICH), 16 (17%) became symptomatic with a subarachnoid hemorrhage (SAH) and 11 (12%) with an intraventricular hemorrhage (IVH). In 3 other cases, the SAH was associated to ICH, while 5 patients had both IVH and ICH. In 1 case the AVM was diagnosed for the onset of hydrocephalus, in 6 cases for the onset of headache. In 17 cases seizures were the leading clinical symptoms. In 1 patient the diagnosis was performed after the onset of left homonymous quadrantanopsia and in 2 patients after the onset of a neurological deficit. In three patients, the cerebral AVM was an incidental finding and in another patient the Authors do not describe the exact clinical symptoms.

A three-category angioarchitectural radiological classification, validated as regards both angiographic description and endovascular treatment, on 99 intracranial arteriovenous lesions and gives a simple, clear terminology whatever the aetiology.

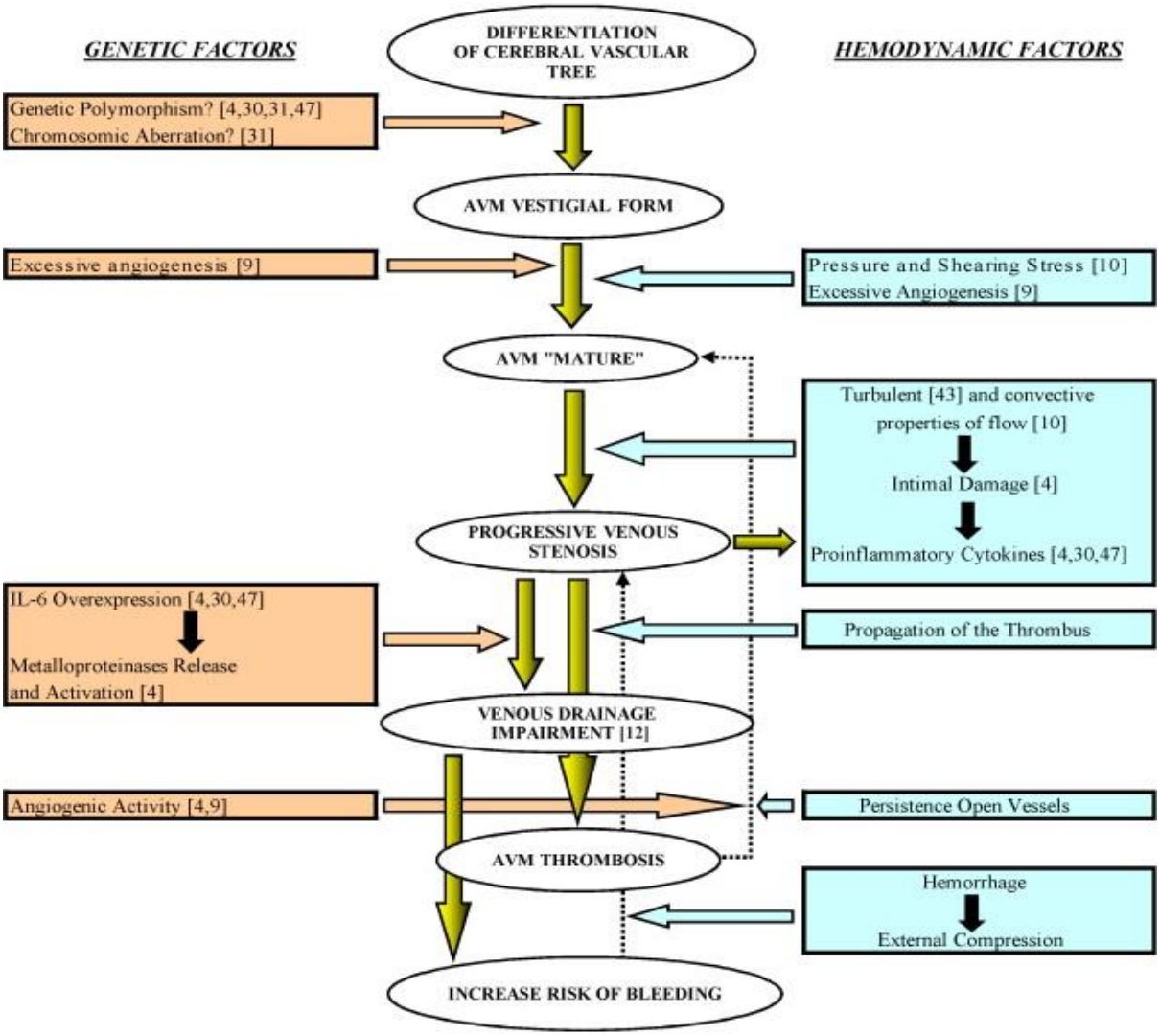
The angioarchitectural classification

They defined the lesion unit and termed it "fistula" in all cases. Indeed, fistula means channel and all arteriovenous lesions involve an abnormal channel between the arterial and venous blood flows. The fistula unit is described

in terms of the initial venous compartment, i.e. the first identifiable venous structure downstream from the shunt (e. g. the venous sinus for a dural AVF, the initial part of the draining vein for an AVM of the brain), and consists of the initial venous compartment and its afferents. The classification then takes into account the arterial side. Three types of fistulae can thus be distinguished [62].

1) **Arteriovenous fistulae** have at most three arterial feeders reaching a single draining vein which represents the initial venous compartment. A single arteriovenous channel is such a lesion.
2) **Arteriolovenous fistulae** display a plexiform arterial structure. Several arteries feed shunts in the wall of a single draining vein which constitutes the initial venous compartment.
3) **Arteriolovenulous fistulae** also have a plexiform arterial structure, but the shunts have a symmetrical arrangement: each afferent arteriole faces its efferent venule. The initial venous compartment is the collector of the venules.

Figure – 5: Possible mechanisms which venous hypertension may induce the formation of AVMs [58].



Therapeutic consequences of the angioarchitectural classification are important for endovascular management. Transvenous embolization is contraindicated in arteriolovenulous fistulas, the most common "AVMs of the brain". Occluding material

introduced by this route will not reach the shunts, due to the hemodynamics, and will block the venous drainage instead. This, if the arterial contribution is not disrupted, will result in hypertension in the shunts, rupture and haemorrhage. This is axiomatic in neurosurgery of AVMs of the brain: the venous drainage must be respected until arterial afferents are occluded [63, 64]. The same is true in interventional neuroradiology: accidental embolization of the draining vein without closure of the nidus can result in hemorrage [65]. Many vascular lesions can manifest with abnormal vessels in the brain at imaging and must be differentiated from one another due to their different natural histories and the various treatment strategies. For example, classic brain AVMs and pial arteriovenous fistulas (AVFs) should be managed according to the risk associated with the disease versus treatment-related risk, Developmental Venous Anomalies (DVA) are normal variants that never require treatment, and dural AVFs with cortical venous reflux always require treatment.

The imaging features of a nidus type brain AVM are consistent with its definition. The diagnostic criteria include (a) the presence of a nidus embedded within the brain parenchyma, identified at either cross-sectional imaging (eg, computed tomography [CT], magnetic resonance [MR] imaging) or conventional angiography; and (b) early venous drainage, which is best seen on dynamic studies, the standard of reference being conventional catheter angiography. Because DVAs rarely bleed, if a DVA encountered during investigation appears to be the cause of an intraparenchymal hemorrhage, an associated cavernoma must be sought and can best be seen with gradient-echo or blood oxygen level dependent sequences. CT and MR imaging findings include dilated cortical veins (pseudophlebitic pattern), which manifest as abnormal enhancing tubular structures or flow voids within the cortical sulci with no true nidus within the brain parenchyma [66].

In addition, specific angioarchitectural weak points must be included in an imaging report [67, 68, 69], since they may increase the risk of future hemorrhage. Intranidal aneurysms, venous ectasias [70], venous stenosis [68], deep venous drainage, single venous drainage, and posterior fossa locations all constitute angioarchitectural weak points.

Prognosis

Despite major advances in diagnostic and therapeutic resources, management decisions in the treatment of brain AVMs is still a challenge. Improved diagnostic methods, such as MRI, contribute to an increasing number of incidentally diagnosed AVMs, and better and new therapeutic advances allow for better treatment, but with potential morbidity. The main goal of the treatment of brain AVMs is to preserve neurological function mainly by preventing intracranial hemorrhage and its consequences. Therefore, understanding the natural history of brain AVMs, especially related to risk of future hemorrhage, is crucial [71].

There are Multiple factors for increased risk of hemorrhage: presentation with hemorrhage, presence of deep venous drainage, associated aneurysms, AVM location, size, male gender, venous outlet restriction, mean pressure and type of feeding arteries, and age [35, 36, 37, 38, 40, 42, 50, 70, 72, 73, 74, 75, 76, 77].

Conclusion

Brain arteriovenous malformations (bAVMs) are very rare vascular lesions which present with spontaneous intracranial hemorrhage (ICH), seizures, or headache in young adults. The latest treatment options include conservative management, surgical resection, stereotactic radiosurgery (SRS), endovascular embolization, or combinations of these treatments (multimodal therapy). The primary goal of the treatment is to prevent hemorrhagic. The risks of these treatments must be weighed against the natural history risks. Despite major advances in diagnostic and therapeutic resources, management decisions in the treatment of brain AVMs is still a challenge. In this case report, a

diagnosis of Cerebral hemorrhage due to Lt. fronto-parietal AV-malformation with predominant MCA & ACA feeders, minor LT ECA feeders, mixed (pial-dural) AVM – Spleitzer Martin grade 5 was made. Angiogram showed Lt. fronto-parietal AV-malformation with predominant MCA & ACA feeders, minor LT ECA feeders. s/o – mixed (pial-dural) AVM – Spleitzer Martin grade 5 was made. Cerebral hemorrhage due to mixed pial dural avm is very is rare; very rarely described this condition in vascular pathologies. Hence this case report.

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