A 44-year-old Male Filipino with Spontaneous Acute Subdural Hematoma and Subarachnoid Hemorrhage Caused by a Dural Arteriovenous Fistula of the Occipital Lobe: A Case Report

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Abstract

Introduction. Spontaneous acute subdural hematoma (ASDH) is rare and comprises 2.6% of all ASDH. In one recent study, only 178 spontaneous ASDH were documented. However, only 1 case was attributed to dural arteriovenous fistula (dAVF). Vascular malformations cause less than 10% of subarachnoid hemorrhage (SAH). Spontaneous ASDH and SAH occurring together are extremely rare. Literature is scarce on cases with dAVF of the occipital lobe as a cause of simultaneous spontaneous ASDH and SAH.

Objective. This paper aims to present a case of a spontaneous acute subdural hematoma and subarachnoid hemorrhage caused by a dural arteriovenous fistula of the occipital lobe, along with its clinical presentation, diagnosis, and treatment.

Case Summary. A 44-year-old Filipino male with no history of trauma presented with severe headache, vomiting, and decreasing sensorium – CT scan revealed acute parenchymal bleed in the left occipital lobe with subarachnoid extension and subdural hematoma in the left fronto-parieto-temporal convexity along the tentorium cerebelli and posterior interhemispheric falx. Due to the location of the lesion seen on the CT scan and the gender distribution, Arteriovenous malformation (AVM) was initially considered, thus proceeded to computed tomography angiogram (CTA) to establish the diagnosis of vascular anomaly, however, revealed dAVF instead. Four-vessel angiogram was done to assess the tributaries of the dAVF and confirmed the diagnosis. Complete obliteration of dAVF of the occipital lobe was done with Onyx Embolization in one session.

Conclusion. This is the first case of Borden type II, Cognard type IIa+IIb dAVF, as reported in this institution. Although extremely rare as a cause of SAH and ASDH, dAVF should be considered a differential diagnosis in patients with no identifiable common cause of the new onset of severe headache and poor neurologic status.

Key Words: spontaneous acute subdural hematoma, subarachnoid hemorrhage, intracranial dural arteriovenous fistula

Introduction

Vascular malformations comprise a collection of disorders characterized by abnormal blood vessel growth. It can be divided into a low flow and high flow malformations based on flow hemodynamics. Low flow malformations are composed of capillaries, veins, lymphatics, or combinations. High flow malformation is characterized as direct communication between an artery and venous vessels without an intervening capillary bed (i.e., arteriovenous malformation and arteriovenous fistula).[1]

Arteriovenous malformation (AVM) are vascular abnormalities consisting of fistulous connections of arteries and veins without normal intervening capillary

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beds. AVM is primarily congenital due to a fetal or embryonic event caused by a lack of development of intervening capillary beds.[1] On the other hand, Intracranial dural arteriovenous fistulas (dAVF) are rare, acquired, direct, pathologic shunts between dural arteries and dural venous sinuses meningeal veins, or cortical veins with no transitional network.[2] Data on the incidence of dAVFs in the Philippines are lacking, but numerous international sources claim that dAVFs represent 10%-15% of all intracranial arteriovenous malformations.[3-5] According to Cedro & Villarosa (2012), the estimated incidence rate of intracranial AVM in the United States, which is similar to the average worldwide incidence, is around 0.04-0.52 percent.[6] However, according to Daroff (2016), intracranial dAVFs might be underestimated due to cases with spontaneous regression or lack of symptoms.[7]

Spontaneous acute subdural hematoma (ASDH) refers to hemorrhage located beneath the dura, which presents within 1-2 days after the onset of bleeding in the absence of provoking factors such as diffuse cerebral atrophy, head trauma, or anticoagulation. According to McBride (2015), spontaneous ASDH comprises 2.6% of all ASDH.[8,9] Rupture of the cortical branch of the middle cerebral artery is the most common cause of spontaneous ASDH. Accordingly, 50% of patients with ASDH present with coma at the time of injury, while 12-38% have lucid intervals after the injury followed by progressive neurologic decline. ASDH that arises in the posterior fossa acts as a space-occupying lesion producing symptoms of elevated intracranial pressure, including headache, vomiting, and anisocoria.[8] Only 178 cases of spontaneous ASDH were documented in a review of literature done by Shekarchizadeh in 2014. Of those, only 1 case was attributed to the presence of dAVF.[10]

Subarachnoid hemorrhage (SAH) is bleeding within the subarachnoid space, which lies between the arachnoid and pia mater and is normally filled with cerebrospinal fluid. Most cases of SAH are caused by rupture of an intracranial aneurysm, but vascular malformations cause only less than 10% of SAH.[11] The clinical presentation of non-aneurysmal SAH often mimics aneurysmal SAH, a sudden, severe headache classically described as the "worst headache of my life" in 97% of cases.[12]

Patients with d`AVFs may present with a variety of symptoms ranging from clinically asymptomatic to fatal hemorrhage, depending on the location and venous drainage of the AVF.[13] Although dAVF can occur anywhere within the dura mater, only 35% of cases are located in the posterior fossa.[7] Furthermore, only 7-8% of cases drain to the superior sagittal sinus while the rest drains elsewhere.[14] In such cases, headache is the most common presentation (50%), followed by intracranial central nerve deficits (29%) and intracranial hemorrhage(23%).[13] Daniels et al. reported that among patients who present with intracranial hemorrhage, the most common pattern of bleeding was intraparenchymal (71%), followed by intraventricular (32%), subarachnoid (21%), and subdural (18%).[14] Spontaneous ASDH accompanied by SAH due to ruptured cortical artery (e.g., occipital) is extremely rare, and there is a scarcity of such reported cases.[10]

Although the pathogenesis is not completely understood, the most commonly accepted theory for the formation of dAVFs is the occlusion or thrombosis of a dural sinus resulting in venous hypertension. The long-term increase in venous pressure results in retrograde flow, which leads to (1) opening of microscopic vascular connections within the dura leading to direct shunting between the dural arteries and veins or (2) the production of endothelial growth factors that induce the formation of aberrant arteriovenous shunts or both.[14]

Multiple diagnostic imaging modalities aid in the proper diagnosis of dAVF. CT scan of the head is often employed as the initial imaging of choice due to its speed, widespread availability, and relative simplicity. CT angiogram (CTA) is indicated to evaluate intracranial bleeding when there is no history of trauma or obvious cause and to rule out the possibility of an underlying vascular lesion. However, immediate decompressive surgery is indicated before CTA in patients presenting with rapidly neurological deterioration.[10] This is supported by observational studies reviewed by McBride (2015), suggesting that surgery within 2-4 hours after the onset of neurologic deterioration in patients with SDH is associated with lower mortality (30-47%) than delayed surgery (80-90%).[8] If the patient presents with a stable neurological condition, CTA is indicated before surgery.[10] Four-vessel angiogram remains the most accurate method of diagnosis because it assesses the tributaries and lesional angioarchitecture and venous outflow pattern of the dAVF, which will dictate the best strategy.[7]

Treatment largely depends on the classification of the fistula and the age and co-morbidities of the patients. The presence of the symptoms directly attributable to the fistula should also be considered. Endovascular therapy has become the first-line treatment for most dAVFs. Other treatment options include observation, compression therapy, open neurosurgery, and stereotactic radiosurgery. Lesions with a benign venous drainage pattern (Borden I or Cognard I/IIa) can be managed conservatively owing to their exceedingly low risk of hemorrhage or neurological deficit. Endovascular therapy has become the first-choice therapy for most patients with higher-grade fistulas, using transarterial, transvenous, or combined approaches. The goal of endovascular therapy for dAVFs is to use embolic material to occlude the fistula pouch and proximal venous drainage.[14] Currently, transarterial embolization with Onyx, which consists of ethylene-vinyl alcohol copolymer dissolved in dimethyl sulfoxide (DMSO), is the agent of choice for the majority of dAVFs. Onyx embolization can obtain a complete obliteration in one session or sufficient size reduction for subsequent surgery or radiosurgery.[21] After treatment, patients are often followed with contrast-enhanced MRA and need to have repeat angiograms only if there is a suggestion of a rare recurrence.[14]

Case Report

A 44-year-old male patient with no known co-morbidities, no family history of cancer, hematologic disorder, or neurologic disorders, and no vices was admitted because of a sudden onset of severe headache unrelieved with over-the-counter pain medication. There was no history of trauma or fall. At the emergency room, the patient had episodes of vomiting and was noted to have decreasing sensorium. Pertinent findings include elevated blood pressure, anisocoria (Left fixed dilated 5mm, Right 3mm), and Glasgow coma score of 5 (E1 M3 V1), indicating no spontaneous eye-opening, in decorticate position, no verbal response, deep tendon reflex had a brisk response (+2). Complete blood count and bleeding parameters were within normal range. A stat CT scan of the brain was taken, which revealed acute parenchymal bleed of approximately 5cc volume in the left occipital lobe with subarachnoid extension and subdural hematoma in the left fronto-parieto-temporal convexity along the tentorium cerebelli and posterior interhemispheric falx with mass effect (*Figure 1 A to C*). Hunt and Hess grading system is an index of surgical risk for the severity of intracranial hemorrhage. The patient was in a state of coma while having a decorticate positioning. This classifies his SAH as Hunt and Hess grade 5. Fisher scale groups patients based on the hemorrhage pattern seen on the initial head CT scan and is an index of vasospasm risk. [23] Our patient had subarachnoid extension measuring less than 1mm; thus, we classify our patient as belonging to Fisher group 2.[15]

CT also noted compression of the lateral and 3rd ventricle, causing a midline shift to the right. He then underwent emergency left decompressive hemicraniectomy and evacuation of hematoma within 4 hours from onset of neurologic deterioration.

Intraoperative findings revealed thick subdural hematoma at the left frontotemporal and cortical bleed at the left occipital area. Laboratories such as CBC and bleeding parameters are within normal limits. At this point, we considered the possibility of a ruptured intracranial AVM for the following supporting reasons: (1) it is more common in men,[11] (2) it is most commonly discovered between the 3rd or 4th decade of life, although they can present at any age,[14] (3) supratentorial compartment is the most common location,[14] and (4) an absence of other common causes of ASDH and SAH, left occipital lobe.

CTA of the brain was performed to establish the diagnosis of AVM. Instead, it showed a well-defined lobulated dAVF in the left occipital lobe measuring approximately 1.0 x 1.8 x 1.0 cm supplied by a prominent terminal of the left posterior cerebral artery with prominent draining veins to the superior sagittal and left transverse sinuses with more prominent perilesional edema (*Figure 2*).

A four-vessel angiogram was taken. It showed dAVF at the level left occipital artery above the transverse sinus (*Figure 3*). According to Daroff (2016), this was done because a four-vessel angiogram is considered the most accurate method to critically assess the lesional angioarchitecture and venous outflow pattern.[7]

Onyx Glue Embolization was performed to obliterate the left occipital artery feeders. Onyx embolization was then carried out with good dAVF penetration. Post embolization angiogram showed complete obliteration of left occipital dAVF (*Figure 4*).

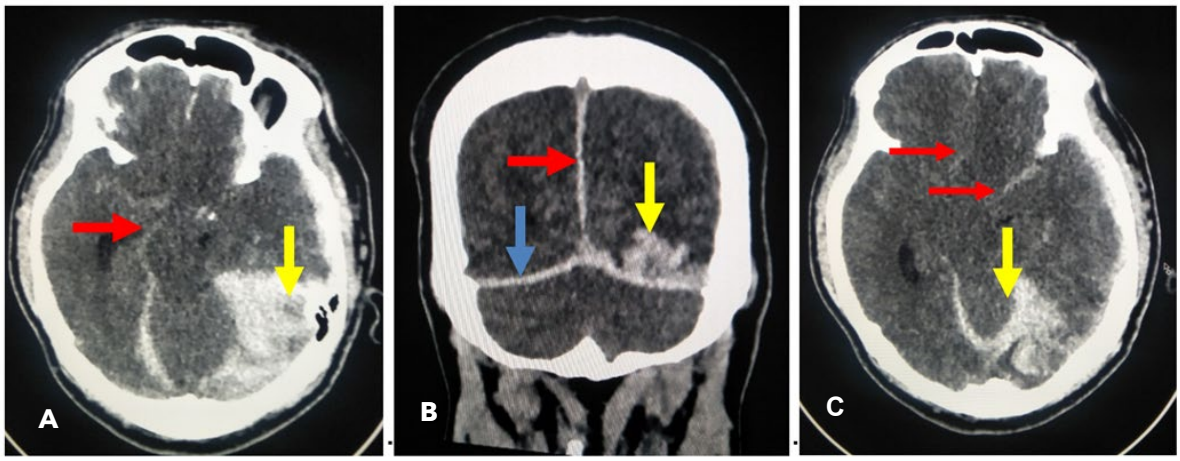

Figure 1. Non-contrast brain CT imaging at symptom onset. (A) Axial section showing hyperdensities in the visualized perimesencephalic cistern (red arrow) and image artifact (yellow arrow). (B) Coronal section showing acute parenchymal bleed (yellow arrow) and subdural hematoma along the tentorium cerebelli (blue arrow) and posterior interhemispheric falx (red arrow). (C) Axial section showing acute parenchymal bleed of approximately 5cc volume in the left occipital lobe (yellow arrow) with subarachnoid extensions (small red arrows).

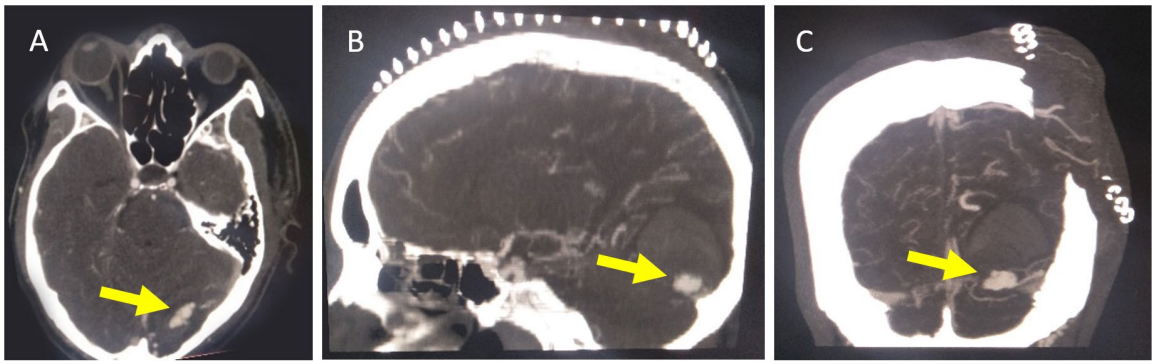

Figure 2. CTA post decompressive hemicraniectomy showing well-defined lobulated dAVF (yellow arrow) in the left occipital lobe. (A) Axial section (B) Sagittal section (C) Coronal section.

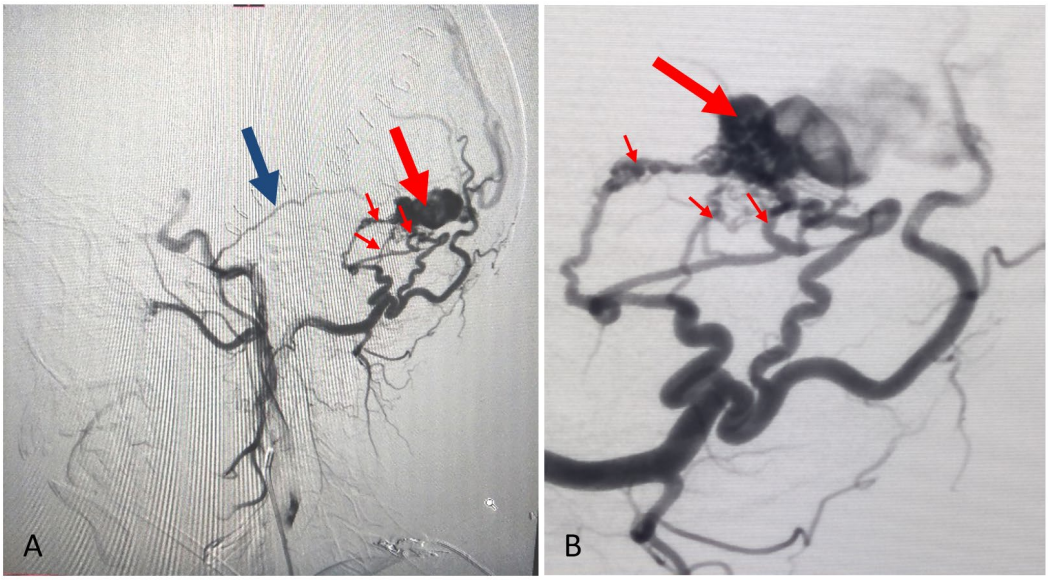

Figure 3. Sagittal section of the four-vessel angiogram. dAVF (big red arrow) with (A) arterial feeders were from multiple trans-osseous branches of the left occipital artery (small red arrows) and possible small arterial feeders from the left middle meningeal artery (blue arrow). (B) Magnified view.

For three months, the patient showed significant improvement with no recurrence of headache, positioning, or cranial nerve deficits. In addition, the patient had improved his quality of life and activities of daily living. Several methods have been used to classify patients according to their functional impairment, compare the effectiveness of therapies, and assess the prognosis of a patient. Among these scoring systems is the Eastern Cooperative Oncology Group (ECOG) performance status grade, which is used to assess how a patient's disease progresses and how it affects the daily living abilities of the patient. Using a scale of 0 to 5 – with 0 being fully active, able to carry on all pre-disease performance without restriction, and 5 being dead;[16] we classify the patient as having an ECOG grade of 0.

After three months, a repeat cranial CT scan showed encephalomalacia over the left occipital lobe indicating scarring of the old hemorrhagic site. (*Figure 5A*) Four-vessel angiogram was also taken, which confirmed complete obliteration of dAVF. (*Figure 5B*) The patient had cranioplasty the following day and was discharged improved.

Discussion

We were presented with a 44-year-old male patient who had sudden onset of severe headache, decreasing sensorium, and decorticate positioning, with no history of trauma or co-morbidities. The onset of new and severe headaches was key in the history of this case. As an initial screening procedure, CT was done, which revealed acute parenchymal bleed in the left occipital lobe suggestive of ASDH. Hemorrhage was also noted along the tentorium cerebelli, interhemispheric falx, and left fronto-parieto-temporal convexity, which is in

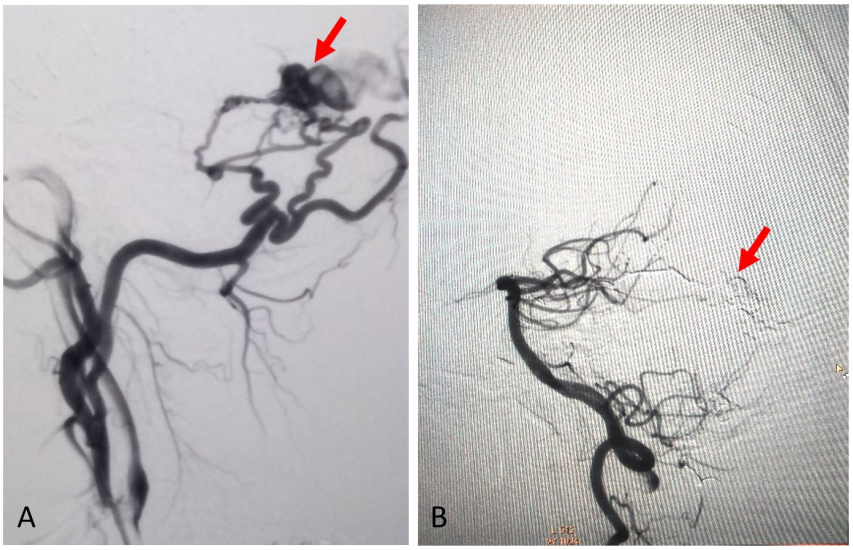

Figure 4. Sagittal section of the four-vessel angiogram during Onyx embolization. Pre embolization angiogram with dAVF visualized (red arrow) (A). Post embolization angiogram with obliterated dAVF (red arrow) (B).

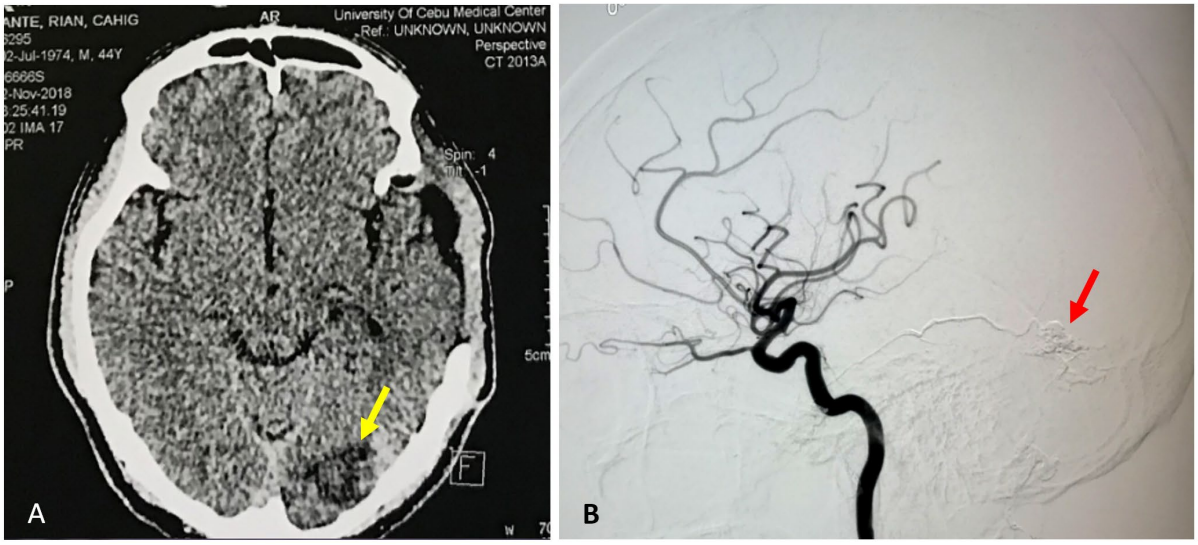

Figure 5. Repeat imaging after 3 months. (A) Axial sectional of CT scan showing Encephalomalacia (yellow arrow) at the left occipital lobe. (B) Four-vessel angiogram showing completely obliterated dAVF (red arrow).

consonance with SAH. Furthermore, CT also noted compression of the lateral and 3rd ventricle, which is reflected in obstructive hydrocephalus. The intention of the CT performed on this patient was to confirm the hemorrhage and its location and was not used to define dAVF. With the revelation of the CT results, evacuation of the hematoma ensued immediately and was successful. At this point, we considered the possibility of an intracranial AVM.

According to Sidawy (2019), in cases of high flow malformation such as AVM, CTA support the proper diagnosis of AVM thus was done.[1] Instead, it showed a lobulated, well-defined dAVF in the left occipital lobe supplied by prominent terminal branches of the left posterior cerebral artery with prominent draining veins to the superior sagittal and left transverse sinuses. Surrounding parenchymal bleed is also seen, which shows an interval increase in the volume measuring 17cc (previously 5cc). There was an absence of neither a ruptured of the cortical branch of the middle cerebral artery nor a ruptured intracranial aneurysm, thus effectively ruling out common causes of spontaneous ASDH and SAH, respectively. Although dAVF is classically diagnosed in the 5th to 6th decade of life[7], in contrast to our patient, several details of the case support the

Table I. Classification of Venous Drainage[13,19,20]

Classification	Merland-Cognard classification	Borden classification
Type I	Antegrade sinus drainage	Sinus or meningeal venous drainage
Type II	Insufficient antegrade sinus drainage	Sinus drainage with CVR
Type IIa	Retrograde sinus drainage only	---
Type IIb	Retrograde cortical venous reflux (CVR) only	---
Type IIa+IIb	Retrograde sinus drainage and CVR	---
Type III	CVR only without venous ectasia	CVR only
Type IV	CVR with venous ectasia	---
Type V	Spinal venous drainage	---

Table II. Frequency of Intracranial Hemorrhage and Aggressive Symptoms in Various Types of Venous Drainage [13,19,20]

Type	Intracranial hemorrhage (%)	Aggressive symptoms* (%)
Cognard type I-IIa, Borden type I	0	2
Cognard type II and IIa+IIb, Borden type II	11	39
Cognard type III-V, Borden type III	48	79

*Bruit, tinnitus, headache, visual symptoms, cranial nerve deficits, intracranial hemorrhage, and dementia.

Table III. Risk of Intracranial hemorrhage in Intracranial Dural arteriovenous fistula based on location[13]

Location	Risk
Cavernous Sinus (%)	Rare
Tranverse-Sigmoid Sinus (%)	15-28
Tentorium (%)	60-74
Superior Sagittal Sinus (%)	23
Anterior fossa (%)	44-45

diagnosis of dAVF. These include (1) the presentation of headache since it is the most common symptom for dAVFs with drainage to the superior sagittal sinus and transverse sinus, and (2) occipital location of dAVF in the patient since 35% of cases are located in that region. According to Hacking et al. (2018), findings that support the diagnosis of dAVF include: (1) abnormally enlarged and tortuous vessels in the subarachnoid space, corresponding to the dilated cortical vein; (2) an enlarged external carotid artery or enlarged trans-osseous vessels; and (3) abnormal dural venous sinuses including arterializations of contrast phase in the affected sinus due to arteriovenous shunting.[22] In our case, a large venous varix was noted above the left transverse sinus with multiple cortical venous outflows draining to the superior sagittal sinus. This further supports the diagnosis of dAVF and the previous descriptions of Hacking et. al.

Four-vessel angiogram is the gold standard in diagnosing and accurately classifying dAVF, allowing systematic evaluation of feeding vessels and demonstrating the presence and extent of retrograde venous drainage. The planning for a potential intervention is dependent on the findings of the angiogram.[7] Four-vessel angiogram was done, and in our case, the left external carotid artery injections showed a dAVF at the left occipital area above the left transverse sinus. The arterial feeders were from multiple trans-osseous branches of the left occipital artery and possible small arterial feeders from the left middle meningeal artery. A large venous varix was noted just above the left transverse sinus with multiple cortical venous refluxes draining to only the superior sagittal sinus – not draining to both the superior sagittal and left transverse sinuses as previously thought. [13]

The natural history of dAVFs strongly correlates with their pattern of venous drainage, in particular, the presence of reflux into pial veins. Therefore, the venous anatomy of the lesion is a central component of most dAVF classification schemes.[18] Several classification schemes have been proposed, but the most common are Borden and Cognard classification (*Table 1*). A classification system proposed by Borden et al. divides dAVFs into three types (I-III), and Merland-Cognard classification divides dAVFs into five types (I-V). Aggressive clinical presentation strongly correlates with higher Borden or Cognard types (*Table 2*).[13,19,20]

Based on the above classification, we classified our patient as Borden type II, Cognard type IIa+IIb. This is associated with an 11% occurrence of intracranial hemorrhage. According to Kiyosue, H. et al. (2004), drainage to the superior sagittal sinus posed an additional risk for the development of intracranial hemorrhage (23%) (*Table 3*). Other risk factors associated with increased risk of intracranial hemorrhage in patients with dAVF include male gender, history of smoking, alcoholic consumption, and is located at the tentorium, frontal-basal, foramen magnum, or cerebral convexity. Among these, only male gender was identified as an additional risk factor for our patient.[17]

Endovascular therapy has become the first-line treatment for most dAVFs. The goal of endovascular therapy for dAVFs is to use embolic material to occlude the fistula pouch and proximal venous drainage. Sadeh-Gonike, U. et al. (2017) reported complete initial occlusion in 82% and recurrence of dAVF in 2% of patients treated with Onyx embolization.[23] In our case, complete obliteration of dAVF of the occipital lobe was done by Onyx Embolization in one session. The patient was discharged in a fair condition and was scheduled for a repeat four-vessel angiogram after three months to confirm the complete obliteration of dAVF and monitor for recurrence.

Conclusion

This is the first case of Borden type II, Cognard type IIa+IIb dAVF reported in this institution. Although extremely rare as a cause of SAH and ASDH, dAVF should be considered a differential diagnosis in patients with no identifiable common cause of the new-onset severe headache and poor neurologic status. It should prompt a more specific imaging modality to establish the diagnosis since dAVF can easily be misdiagnosed as AVM, especially when many clinical signs support the latter's diagnosis. Onyx embolization is recommended to obliterate dAVF.

Conflict of Interest. The authors declared that they have no conflict of interest.

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