

Case Report of a Rupture of Arteriovenous Malformation by an Unusual Factor, Brain Abscess: A Pediatric Case

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Abstract

Introduction: Arteriovenous malformation is a rare pathology, often discovered accidentally in children. Generally, it presents as an intra-parenchymal hematoma following rupture of the affected vessels. The risk of rupture is linked to the volume of the malformation, its cortical location and venous drainage. In literature, few cases of rupture have been reported in cases of meningitis, but none are associated with a brain abscess. **Objective:** To report a case of an intraparenchymal hemorrhage due to rupture of an arteriovenous malformation with an associated brain abscess. **Observation:** The authors report the case of a 2-year-old child, admitted for impaired state of consciousness associated with a right hemiparesis and seizures in a febrile context. Clinical evaluation revealed a Blantyre score of 3, fever at 40°C, divergent strabismus of the right eye, a right pyramidal syndrome and meningeal irritative syndrome. Cerebral CT scan revealed a left fronto-parietal intra-parenchymal hematoma and a right occipital ring-enhanced lesion, suggesting a brain abscess. An MRI suggested a probable rupture of an arteriovenous malformation resulting in the intra-parenchymal hematoma. Management consisted of craniotomy to evacuate the intra-parenchymal hematoma, antibiotic therapy with vancomycin and ceftriaxone at meningeal doses. Histopathological analysis of the intraoperative sample revealed an arteriovenous malformation. The post-operative course was marked by regression of hyperthermia, persistence of spasticity, irritability and clonic movement of the right lower limb. These were managed with baclofen, phenobarbital, and ergotherapy. We observed a regression of spasticity and improved motor skills in the right limbs. At 6 months follow-up, child could interact with his social environment despite aphasia,

regression of spasticity and right hemiparesis. **Conclusion:** Rupture of arteriovenous malformations can be enhanced by neuro-meningeal infections and particularly brain abscesses. Cerebrovascular complications of these conditions mostly have unfavorable outcomes and neurological sequelae.

Keywords

Arteriovenous Malformation, Brain Abscess, Rupture, Evolution

1. Introduction

Arteriovenous malformations (AVMs) of the brain are congenital vascular lesions that can be observed at any age. AVMs are defined by the presence of arteriovenous shunting through a nidus of coiled and tortuous vascular connections between arteries to draining veins [1]. This direct arteriovenous shunting due to the lack of capillaries leads to hypertrophy in the arterial and venous components of the AVMs. On embryological basis, AVMs are due to either the persistence of a primitive arteriovenous connection, or the development of a new connection after a normal closure process. Even though the precise pathophysiologic events by which such malformations occur are unknown, it is hypothesized that most malformations occur during the third week of embryogenesis. They are the most common cause of spontaneous intraparenchymal hemorrhage in children. Pediatric AVMs could also present with recurrent seizures or headaches [2] [3]. The natural history of AVMs in children is not well studied or understood. The annual rupture rate was reported to be between 2% and 10% [3] [4]. Conflicting reports exist regarding the relationship between the pediatric AVM size and its risk of rupture [4]. Other risk factors include a previous history of hemorrhage, deep-seated or infratentorial AVMs, deep venous drainage, female sex, associated aneurysms, and diffuse AVM morphology [4]. In literature, few cases of rupture have been reported in cases of bacterial meningitis, but none are associated with a brain abscess.

2. Aim

The aim of this report is to present a case of an intraparenchymal hemorrhage due to the rupture of an arteriovenous malformation associated with a brain abscess.

3. Clinical Presentation

We present a case of a 2-year-old lad admitted at Chantal Biya Foundation following referral from a nearby health facility for the management of altered state of consciousness associated with right hemiparesis and generalized tonico-clonic seizures in a febrile context. This clinical picture evolved over a period of 3 weeks before admission. Patient presented with intermittent fever, headache associated with 2 episodes of emesis on the same day, which motivated mother to consult at

a nearby health facility where he received ceftriaxone (50 mg/kg per dose) for 5 days and artesunate injectable (3 mg/kg per dose) for the management of sepsis and malaria. This was followed by 3 days oral antimalarial medication on discharge. Evolution on treatment was marked by persistence of symptoms associated with progressive onset of right upper and lower limb weakness and asthenia noticed by his mother. This motivated a secondary consultation at same health facility where the infant was hospitalized and an undocumented treatment plan administered. The persistence of fever, right sided weakness and onset of abnormal right upper limb twitching movement followed by tonico-clonic generalized seizure prompted referral to Chantal Biya Foundation for better management. Concerning the past medical history, immunizations status was up till date for age with a good psychomotor development.

The clinical evaluation on admission revealed an ill looking infant with Blantyre score of 3/5, fever of 40°C, divergent strabismus of the right eye, meningeal irritation syndrome (neck stiffness), and right pyramidal syndrome (brisk right reflexes bicipital, knee reflex, both right upper and lower limb hypertonia and right plantar reflex in extension). An initial diagnosis of meningoencephalitis was evoked, and paramedical examinations requested: full blood count showed pleocytosis with white blood cell count of 21,300; Haemoglobin at 11.5 g/dl; C-reactive protein at 96 mg/dl. Lumbar tap was not done since cerebral computed tomography (CT) scan was requested on the basis of focal neurologic signs.

Contrast head CT scan (**Figure 1**) revealed a left fronto-parietal intraparenchymal hematoma, associated with a right occipital ring enhanced lesion with perilesional grade one vasogenic edema in favor of a brain abscess. Given the location volume and geographical aspect, a cerebral magnetic resonance imaging (MRI) was requested. MRI revealed (**Figure 2**) a rupture of an AVM (left fronto-parietal intraparenchyma hematoma). Cardiac ultrasound done to investigate for endocarditis was negative. This was done in search of the primary foci. Hemoculture was equally sterile.

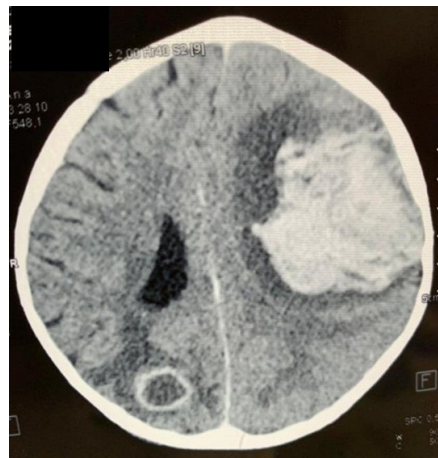


Figure 1. Contrast head CT scan with brain abscess and intraparenchymal hematoma.



Figure 2. MRI of the case.

Given the imaging results, neurosurgical consultation was sought and surgical indication was placed with the aim of hematoma evacuation and cerebral decompression. Preoperative evaluation was done and a left frontoparietal craniotomy was performed. After general anesthesia, patient was installed in the supine position head resting on a head pad and turned to the right at 45°. All pressure points were padded and soft band enrolled around the limbs. Aseptic scalp scrubbing plus sterile draping were done. A reversed question marked incision was made, hemostasis was done, and the scalp flap reflected. Bone flap was made with 6 burr holes. A stellate durotomy was done, finding hemosiderin cortical coloration, hematoma with fibrillary cortical vessels within the lesion and peripheral sclerosed cortical vessels engulfing part of the hematoma. Gest corticotomy, collection of samples for histological analysis, evacuation of the hematoma with suction plus bipolar coagulation were done. Duroplasty, bone flap repositioning, and closure of scalp under a non aspirative drainage system ended the intervention. Post-operative antibiotherapy consisted of vancomycine 40 mg/kg/24 hrs and ceftriaxone (100 mg/kg/24 hrs) at meningeal dosages for 21 days.

Early post-operative course was marked by regression of fever, improvement of irritability on day 5 post operatively, and persistent of spasticity with clonus of left lower limb and aphasia. The child was placed on baclofen 1 mg/kg, depakin 30 mg/kg and ergotherapy. Histological analysis was in favour of AVM. Patient was discharged on day 22nd following surgery after 21 days of oral course with cefixime 8 mg/kg, depakin 30 mg/kg, baclofen 1 mg/kg. After 4- and 8-week post-operative follow up, we still noted persistence of spasticity, aphasia and right hemiparesis. Regression of spasticity and marked improvement of right hemiparesis at 6 month follow up. At one year follow up, the child interacted with his social environment. We equally noticed a regression of spasticity and right hemiparesis with child being able to walk with assistance, but was still aphasic.

4. Discussion

Literature on brain abscess and ruptured AVM is rare. Our patient was presented

with a clinical picture of fever, focal neurological sign, and seizures. He was managed 3 weeks after onset of symptoms. This could be explained by the fact that most parents in our context rush to health centers for initial management. But when the clinical course is above the managing capacity of these health centers and requires specific investigations, patients are then referred for better management to reference hospitals. This led to delay in appropriate investigations, diagnosis and management. Probably the onset of right hemiparesis could be in light with the hematoma secondary to the ruptured AVM at the left frontoparietal region. This clinical picture doesn't represent the classical picture of rupture AVM, but rather that of meningoencephalitis. The overall risk of hemorrhage from an untreated AVM in all age groups is estimated to be between 2 and 4% yearly [5]-[7]. Even though AVMs are rare in kids with an estimated frequency of 3% of all AVMs [8] [9], they tend to rupture more frequently than in adults [8] [10] [11]. They are considered to be the most frequent abnormality of intracranial circulation in childhood [12], and they are the most common cause of spontaneous intraparenchymal hemorrhage in children. Pediatric AVMs could also present with recurrent seizures or headaches [13] [14]. In a Dutch prospective cohort study involving 2306 patients with bacterial meningitis, intracerebral hemorrhage was identified in 2% patients [15]. The majority of the intracerebral hemorrhages were lobar (70%), followed by deep hemorrhages (9%), infratentorial hemorrhages (11%), and multifocal (micro) hemorrhages (9%) [15]. Intracerebral hemorrhage complicating bacterial meningitis was associated with high rates of unfavorable outcome (89%) and death (55%). Our patient presented with a lobar hematoma associated with brain infective process other than meningitis but instead an abscess as identified by the Dutch prospective study. The coexistence of brain abscess and ruptured AVM could be thought off as the abscess precipitating rupture of the AVM. The underlying mechanisms responsible for intracerebral hemorrhage is likely a result of a combination of factors, including inflammation of blood vessels, activation of coagulation pathways, and the inhibition of fibrinolysis, as suggested by prior research [16] [17]. These could render the AVM vessels more susceptible to rupture due to their increased fragility, or the coexistence of a septic embolic process which could precipitate. That is the existence of brain abscess at one location and ruptured AVM at another location.

The difficulty in isolating the causative germ could be due to the multiple antibiotic therapies administered in the health facility during the three weeks before our consultation. This can explain the delay in diagnosis and management, with negative blood cultures and stigmas of endocarditis. Lumbar tap and CSF analysis couldn't be obtained given the presence of the lobar hematoma associated with mass effect and midline shift, thus contradicting the procedure. Given the unusual nature and localization of the hematoma, a MRI was requested to enlighten us on the nature of this lesion. As for the management, surgical resection was indicated over medical management with the aim to decompress the brain and obtain a histological sample. It also has the potential benefit of allowing the immediate cure

of AVM. Moreover, in acute ruptured settings, surgery has the advantage of allowing the hematoma removal, thus improving outcome and neurological sequelae. The delay in diagnosis and surgical management could explain the post-operative neurological status of the patient. Continuous ergotherapy with physiotherapy will likely lead to regression of neurological deficits given the young age of the patient over time as brain development is a continuous process.

5. Conclusion

Brain abscess associated with ruptured AVM is rare. The physiologic modifications induced by these infectious can increase the risk of an AVM rupture. Cerebrovascular complications associated with this infection mostly have unfavorable outcome and neurological sequelae. Delayed diagnosis worsens the prognosis due to the association with brain abscess. Unfortunately, there are no established and effective management strategies to prevent or treat these specific complications in this population. However, we do recommend a neuroimaging in patients with clinical picture associating fever, seizures and focal neurologic symptoms or altered level of consciousness to identify the underlying cause. Early diagnosis and targeted surgical management accordingly improve the outcome and neurological status.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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