

Pulmonary Arteriovenous Malformations Complicated with Paradoxical Embolic Stroke

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Abstract

Pulmonary arteriovenous malformations (PAVM's) are rare vascular anomalies involving the pulmonary vessels. It is a rare finding and patients are commonly asymptomatic despite the presence of a right to left shunt. We report a case of a patient with multiple PAVMs causing recurrent paradoxical brain embolism resulting in stroke.

Keywords: Pulmonary arteriovenous malformation; stroke; Computed tomography; Embolism

Abbreviations: PAVM: Pulmonary arteriovenous malformation; CT: Computed tomography; GCS: Glasgow Coma Scale

1. Introduction

Pulmonary arteriovenous malformation (PAVM) is defined as an abnormal communication between the pulmonary arteries and veins. It is a rare finding and patients are commonly asymptomatic despite the presence of a right to left shunt [1, 2]. However, it is recognized as one of the many causes of stroke due to paradoxical brain embolism [3]. Brain infarction associated with PAVMs most likely occur in patients with feeding arteries of more than 3mm diameter and not in those of smaller size [3]. We report a case of a patient with multiple PAVMs causing recurrent paradoxical brain embolism.

2. Case Study

An 85 year old man with underlying rheumatoid arthritis not on any treatment, presented with an alleged fall in the morning prior to hospital admission. On presentation to the emergency department, there was a left sided body weakness with associated facial asymmetry and slurred speech. Patient was alert with full GCS on examination. His blood pressure was elevated on admission documented at 170/84 mmHg with power of the left upper and lower limbs were 3/5 and up going left plantar reflex. Initial CT brain revealed an acute infarct at the right centrum semiovale. He was then started on acetylsalicylic acid (Aspirin) 100 mg once daily and admitted to ward for further management. In the ward, he developed worsening left sided body weakness with power of the left upper and lower limbs of 1/5. He was subsequently started on subcutaneous Heparin 5000 U bd.

At day 3 of admission, there was worsening speech with no improvement of the left sided body weakness. However, his GCS remained full with stable vital signs. A repeated CT brain revealed an evolving right basal ganglia extending to the temporal lobe acute infarcts with haemorrhagic transformation and acute infarcts of the pons and midbrain as shown in Figure 1. Aspirin was stopped immediately and patient was monitored closely in the ward.

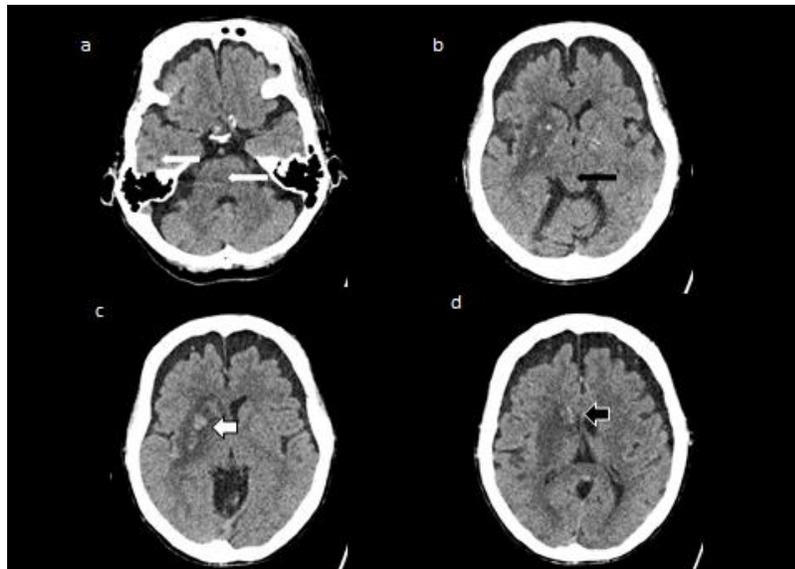


Figure 1: Plain CT brain in axial views showing (a) right temporal and pontine infarcts (long white arrows), (b) midbrain infarct (long black arrows), (c,d) right basal ganglia infarct with haemorrhagic transformation (short arrows).

At day 13 of admission, he developed sudden shortness of breath with SpO₂ dropped to 91%. Initial CXR showed small non-specific well circumscribed round opacities at both lower zones as shown in Figure 2. Upon further review of the patient, the medical team requested for a CT pulmonary artery scan to exclude pulmonary embolism. The CT scan showed multiple arteriovenous malformations in the lungs with serpiginous feeding and draining

vessels originating from the pulmonary arteries and veins at the right upper and both lower lobes as shown in Figure 3 and Figure 4. There were also consolidations noted at the right middle and lower lobes. The radiological impression was multiple pulmonary arteriovenous malformations with right with associated lung infective changes. The recent cerebral infarcts were likely due to paradoxical emboli from pulmonary arteriovenous malformations. He was subsequently treated for hospital acquired pneumonia requiring IV antibiotics and non-invasive oxygen therapy.

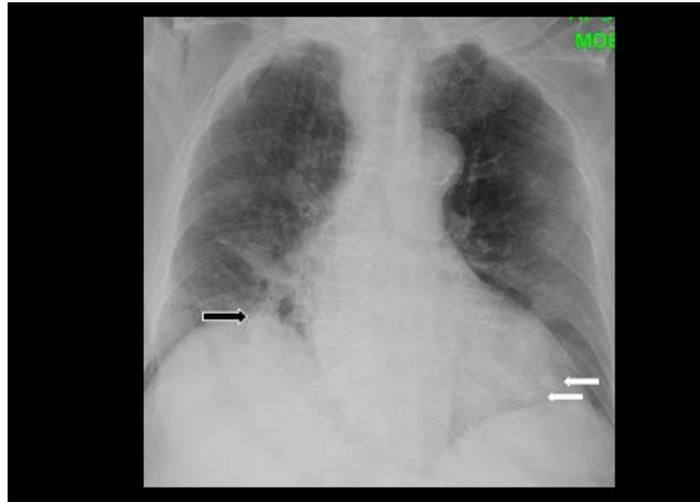


Figure 2: AP supine chest radiograph demonstrating well circumscribed round nodules at the right lower lobe (black arrow) and left lower lobe (white arrow).



Figure 3: CT scans of thorax (a,b) in axial views (c) in coronal view demonstrating a arteriovenous malformation in the right lower lobe connected to a serpiginous pulmonary vein (arrows).

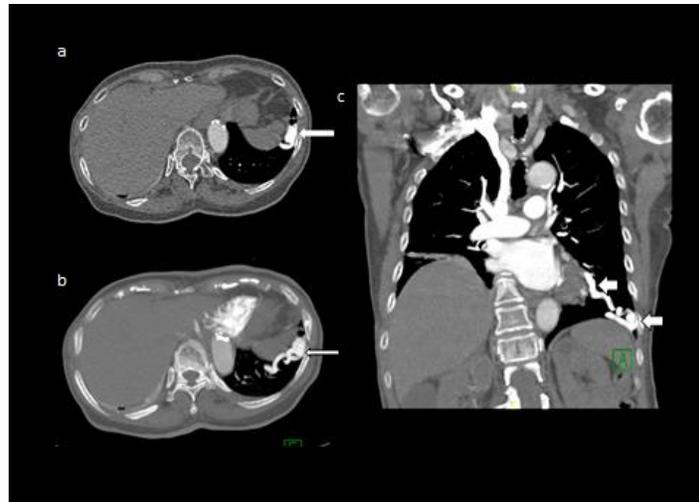


Figure 4: CT scans of thorax (a,b) in axial views (c) in coronal view demonstrating another similar well circumscribed arteriovenous malformation (long arrows) at the left lower lobe connected by a blood vessel with the feeding artery measuring 4.6 mm (short arrows).

No further intervention was done for the patient in regards to his pulmonary arteriovenous malformations due to financial constraint. Patient was then discharged well 2 days later to a nursing home with continuation of his tablet Aspirin 100 mg od and follow up in the outpatient clinic given. No further documentation regarding patient's visit to the outpatient clinic was obtained as patient had defaulted follow up.

3. Discussion

PAVM is a vascular anomaly between the pulmonary arteries and veins leading to continuous right-to-left shunting [2]. It is a rare condition and usually detected as an incidental finding or on autopsies [2]. PAVMs are commonly associated with hereditary haemorrhagic telangiectasia (HHT), also referred to as Osler–Weber–Rendu syndrome, an autosomal dominant syndrome [1]. Approximately 15–35% of HHT patients have concurrent PAVMs and 50–85% of PAVMs patients have underlying HHT [3]. The patient above did not fit the diagnostic criteria to suggest HHT.

Majority of patients have unilateral segments of pulmonary AVM usually located in the lower lobe in more than 50% of cases and out of these patients, 33% of them have more than one lesions, which may measure up to 10cm in diameter [1]. Brain infarction is one of the most important complications that may occur in PAVM patients due to continuous right-to-left shunting [3]. Stroke is usually the first presentation of PAVM and which includes undiagnosed PAVM in HHT patients [3]. Moussouttas et al. [4] has concluded that brain infarction are likely to occur in PAVM patients with feeding arteries of more than 3 mm in diameter and not for patients with feeding arteries of smaller size. Therefore, they recommended PAVM embolization therapy for patients with feeding artery larger 3mm in diameter [4].

4. Conclusion

PAVMs are an uncommon vascular anomaly with a strong link between PAVM and HHT [5]. The chest radiograph can often suggest the diagnosis of PAVM if it is large enough and contrast enhanced CT of the thorax or pulmonary angiography is usually confirmatory [6]. Our case history presents a rare cause of embolic stroke likely from PAVMs. A long-term treatment with thrombolytic therapy may be an option for secondary stroke prevention in those patients with underlying ischemic stroke secondary to embolism from PAVM [2].

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