Pulmonary Arteriovenous Malformations Leads To Embolic Cerebral Infarcts with Haemorrhagic Transformation

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Abstract

Pulmonary arteriovenous malformations (PAVM's) are rare vascular anomalies involving the pulmonary vessels. Patients with PAVM are commonly asymptomatic despite the presence of a right to left shunt. We report an interesting case of a patient with multiple PAVMs complicated with recurrent paradoxic brain embolism and leads to cerebral infarction.

Keywords: Pulmonary arteriovenous malformation; Stroke; CT; Embolism

Abbreviations

PAVM: Pulmonary Arteriovenous Malformation; CT: Computed Tomography; GCS: Glasgow Coma Scale

Introduction

Pulmonary arteriovenous malformation (PAVM) is defined as an abnormal communication between the pulmonary arteries and veins. It is a relatively rare finding and patients are commonly asymptomatic despite the presence of a right to left shunt [1,2]. However, it is well recognized as one of the many causes of paradoxical stroke [3]. Brain infarction associated with PAVMs often occur in patients with bigger feeding arteries (> 3mm) but not in small or tiny vessels [3]. We report a case of a patient with multiple PAVMs leads to paradoxical embolic cerebral infarct.

Case Presentation

An 85 year-old man, underlying rheumatoid arthritis presented with fall prior to hospital admission. On presentation he was noted to have left sided body weakness (power of both upper and lower limb 3/5), facial asymmetry and slurred speech. Patient was alert and GCS was full. His blood pressure was high on admission (170/84 mmHg). Initial CT brain showed an acute infarct in right centrum semiovale. He was then started on acetylsalicylic acid 100 mg once daily and admitted to ward for further management. However, in the ward, he developed worsening left sided body weakness. At day 3 of admission, there was worsening speech and no improvement of the weakness. GCS remained full. A repeated CT brain demonstrated evolving right basal ganglia with haemorrhagic transformation and acute infarct of the midbrain and pons (Figure 1). Aspirin was stopped immediately. Subsequently patient was monitored closely in the ward.

At day 13 of admission, he complained of dyspnea with SpO2 dropped to 90%. CXR showed few small well circumscribed round opacities at both lower lobes as shown in Figure 2. Immediately the medical team requested for a CT Pulmonary Artery (CTPA) scan to rule out pulmonary embolism. The CT scan showed multiple pulmonary arteriovenous malformations as evidenced by serpiginous feeding and draining vessels from the pulmonary vasculatures in right upper and bilateral lower lobes as shown in Figure 3 and Figure 4. There were also airspace consolidations in right middle and lower lobes. The radiological diagnosis was multiple pulmonary arteriovenous malformations associated with active lung infection. The earlier CT scans of brain showing cerebral infarcts were likely due to paradoxical emboli from PAVM. Subsequently he was treated for pneumonia requiring IV antibiotics and non-invasive oxygen treatment. Patient was offered for diagnostic pulmonary angiography and embolization under interventional radiological department. However, due to financial constraint, no further treatment was done. After 2 days, patient was discharged with Aspirin 100 mg daily and scheduled for follow up in the outpatient clinic.
Discussion

PAVM (Pulmonary Arteriovenous Malformations) is a vascular malformation between the pulmonary arteries and veins causing right-to-left shunt [2]. It is rare and usually detected as an incidental finding or on autopsies [2]. PAVMs are always associated with Hereditary Hemorrhagic Telangiectasia (HHT), also known as Osler–Weber–Rendu syndrome, which is an autosomal dominant syndrome [1]. Approximately 15%–35% of patients with HHT have associated with PAVMs and 50%–85% of PAVMs patients were diagnosed to have HHT [3].

Majority of patients (more than 50%) with PAVM have the abnormal vessels located in unilateral segments of lower lobe. One of the most important complications of PAVM is cerebral infarction where it occurred due to continuous right-to-left shunting [3]. Sometimes stroke is the first presentation of PAVM, in particular HHT patients with undiagnosed PAVM in [3]. Moussouttas et al. [4] has concluded that cerebral infarction are likely to occur in PAVM patients with feeding arteries of more than 3 mm in diameter. This finding was consistent with the current case scenario whereby the feeding artery is 3.5 mm in diameter. Therefore, PAVM embolization therapy was recommended for this patient [4]. However, procedure was not proceeding due to financial constraint.

Conclusion

PAVMs are relatively rare vascular anomaly with a strong association between PAVM and HHT [5]. 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 confirmative [6]. Our case history presents a rare cause of embolic stroke with haemorrhagic transformation likely from PAVMs. In those patients with cerebral infarction secondary to embolism from PAVM, long-term thrombolytic therapy may be an option for stroke prevention [2].

References

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