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# Percutaneous Transcatheter Embolization for Recurrent Cerebral Embolism Due to Pulmonary Arteriovenous Malformation

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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**Patient:** Male, 67-year-old
**Final Diagnosis:** Pulmonary arteriovenous malformations
**Symptoms:** Acute dizziness • left limb paraesthesia • visual disturbances • recurrent transient dyspnea • sudden right-sided hemiparesis • severe vision loss
**Clinical Procedure:** —
**Specialty:** Neurology

**Objective:** Rare disease

**Background:** Pulmonary arteriovenous malformations (PAVMs) are rare structural vascular abnormalities that create communications between pulmonary arteries and veins, resulting in right-to-left shunts. PAVMs are often asymptomatic; however, patients may exhibit a range of clinical symptoms, including dyspnea, hemoptysis, chest pain, and cyanosis. Common serious complications include cerebrovascular events (eg, paradoxical embolism resulting in stroke or transient ischemic attack) and infectious complications (eg, brain abscess). PAVMs do not always require treatment; management should be guided by factors such as feeding artery size and patient symptoms. When intervention is indicated, percutaneous transcatheter embolization is the primary treatment and demonstrates efficacy in most cases. This report describes a case of recurrent cerebral embolism attributed to an undiagnosed PAVM, which was treated with percutaneous transcatheter embolization.

**Case Report:** A 67-year-old man lacking prior medical history or medication use experienced recurrent cerebral embolism within 3 months despite therapeutic anticoagulation. The paradoxical embolism was attributed to a PAVM diagnosed by contrast-enhanced chest computed tomography. Subsequent transcatheter embolization of the PAVM was successfully performed. No recurrence was observed at the 4-year follow-up.

**Conclusions:** In cases of unexplained cerebral embolism, PAVMs should be considered a potential underlying etiology. Hemorrhagic transformation requires careful risk assessment but is not an absolute contraindication to percutaneous transcatheter embolization. Timely diagnosis and embolization are essential to prevent disabling recurrent events.

**Keywords:** Arteriovenous Malformations • Cerebral Embolism • Embolization, Therapeutic • Paradoxical Embolism • Pulmonology

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## Introduction

Pulmonary arteriovenous malformations (PAVMs) are abnormal, direct, low-resistance connections between pulmonary arteries and veins that bypass the capillary bed [1]. PAVMs are infrequently encountered in clinical practice. The estimated annual incidence ranges from 2 to 3 cases per 100 000 population, whereas screening studies suggest a prevalence of approximately 1 in 2600 individuals [2,3]. Approximately 80% to 90% of cases are associated with hereditary hemorrhagic telangiectasia (HHT), although sporadic cases also occur [4]. HHT, also referred to as Osler-Weber-Rendu syndrome, is an autosomal dominant disorder with a prevalence of 1 in 5000 to 8000 individuals. Mutations in the *ENG*, *ACVRL1*, and *SMAD4* genes have been identified as the underlying cause of HHT [5,6].

Clinical manifestations of PAVMs range from asymptomatic to various symptoms, including dyspnea, hemoptysis, chest pain, and cyanosis. The primary clinical complication of PAVMs is paradoxical embolization, in which thrombi, bacteria, or air from the venous circulation bypass normal pulmonary capillary filtration and gain direct access to the systemic arterial system. This mechanism substantially increases the risk of severe neurologic events, including ischemic stroke and transient ischemic attack, as well as systemic abscesses [7,8]. Other potential complications include pulmonary hemorrhage and pulmonary hypertension. PAVMs represent an important but frequently overlooked, treatable cause of cryptogenic stroke, particularly in younger individuals [9]. Noninvasive diagnostic modalities for PAVMs include chest radiography, contrast-enhanced computed tomography (CT), and contrast echocardiography; pulmonary angiography remains the diagnostic gold standard [1]. Underdiagnosis is common due to nonspecific symptoms and the absence of routine screening.

Due to its efficacy and safety, percutaneous transcatheter embolization has become the first-line therapy for PAVMs with feeding arteries at least 2 mm in diameter, as well as PAVMs in symptomatic patients [10-12]. Relative to surgical resection (lobectomy), embolization is less invasive, associated with shorter recovery time, and preserves more lung parenchyma. This minimally invasive procedure aims to occlude the feeding vessel, thereby eliminating the right-to-left shunt, alleviating hypoxemia, and, most importantly, reducing the long-term risk of paradoxical embolism. Complete occlusion is associated with a substantial reduction in recurrent cerebrovascular events [12,13].

This report describes a 67-year-old man with recurrent paradoxical embolism due to an untreated PAVM who was successfully treated via percutaneous transcatheter embolization; he experienced no recurrence during follow-up. This case highlights the importance of including PAVMs in the differential diagnosis of paradoxical embolism and underscores the definitive role of percutaneous transcatheter embolization.

**Figure 1.** Axial brain computed tomography scan obtained at initial presentation. A mixed-density area is evident in the right temporal and occipital lobes, including a hypodense region (cerebral infarction) and small hyperdense foci suggestive of hemorrhagic transformation.

## Case Report

### Initial Presentation and Diagnostic Workup

A 67-year-old male farmer with no clinically significant medical history was hospitalized after 9 hours of acute dizziness, left-sided limb paresthesia, and visual disturbances. He had been in a convalescent state for the preceding 3 months, with restricted mobility due to a cow-attack-induced chest injury. Physical examination revealed left homonymous hemianopia, with a National Institutes of Health Stroke Scale (NIHSS) score of 2. Routine blood test results were unremarkable. CT of the brain demonstrated acute infarction with hemorrhagic transformation in the right temporal and occipital lobes, a region supplied by the posterior cerebral artery (**Figure 1**).

The patient reported recurrent transient dyspnea over the preceding month, which worsened in the upright position. Contrast-enhanced chest CT revealed a well-circumscribed nodular lesion in the left lower lobe. Upon contrast administration, the lesion showed internal enhancement with contrast filling, consistent with a PAVM (**Figure 2**). Venous ultrasonography confirmed deep venous thrombosis (DVT) in the left leg. Transthoracic echocardiography, carotid and vertebral





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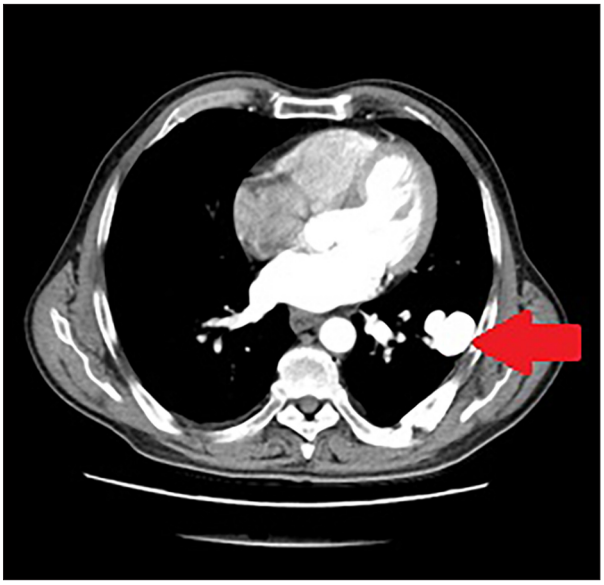

**Figure 2.** Contrast-enhanced chest computed tomography scan. The image demonstrates a well-circumscribed, enhancing nodular lesion in the left lower lobe, consistent with a pulmonary arteriovenous malformation. Prominent vascular structures contiguous with the lesion represent the feeding artery and draining vein.

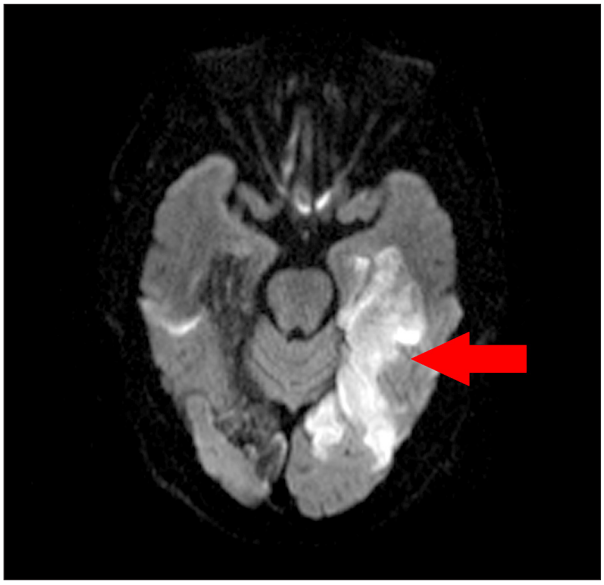

**Figure 3.** Axial diffusion-weighted magnetic resonance imaging scan obtained at the time of the recurrent event. New areas of restricted diffusion (high signal intensity) are present in the left temporal and occipital lobes, indicating acute infarction. These lesions are nearly symmetric to the prior right-sided infarcts shown in Figure 1.

artery ultrasonography, and magnetic resonance angiography of the intracranial vessels showed no structural abnormalities. A proposed agitated saline bubble study to confirm a right-to-left shunt was declined by the patient's family. Based on comprehensive evaluation, paradoxical embolism was diagnosed as the cause of stroke; the embolus likely originated from left lower limb DVT and passed through the PAVM.

### Initial Management and Clinical Course

Given the acute stage of cerebral infarction and the presence of hemorrhagic transformation, the multidisciplinary team elected to initiate medical management rather than immediate transcatheter embolization of the PAVM. This decision reflected the need for perioperative anticoagulation, the associated bleeding risk, and the recommendation to defer elective intervention until at least 3 months after stabilization of the cerebral infarction. The patient was discharged on warfarin with a target International Normalized Ratio (INR) of 2 to 3. The time in therapeutic range was 62.5% during the 3 months preceding recurrence. Recorded INR values ranged from 2.08 to 2.65, indicating generally stable anticoagulation.

### Recurrent Event and Definitive Treatment

Within 3 months of the initial stroke, the patient was readmitted with sudden right-sided hemiparesis and severe vision loss (NIHSS score of 7). Diffusion-weighted magnetic resonance imaging revealed a new acute cerebral infarction in the left temporal and occipital lobes (within the posterior cerebral artery territory), almost symmetric to the previous lesions (**Figure 3**). Stroke recurrence despite therapeutic anticoagulation prompted reevaluation of the treatment strategy.

The interventional radiology team conducted a detailed discussion and obtained informed consent from the patient's family, given that the patient had experienced considerable cognitive decline (Mini-Mental State Examination score of 16). Pulmonary angiography confirmed a substantial PAVM in the left lower lobe with a feeding vessel measuring 4.2 mm. The lesion was successfully embolized using 9 coils (**Figures 4, 5**).

### Outcome and Follow-up

The patient continued anticoagulation after the procedure. Serial ultrasonography demonstrated resolution of the left lower extremity DVT after 1 year, and anticoagulation therapy was subsequently discontinued. At the most recent follow-up (4 years post-embolization), there had been no recurrence of stroke or other thromboembolic events, and the patient's transient dyspnea had completely resolved. However, the family reported that the patient has persistent cognitive deficits (eg, memory impairment and impaired navigation) and visual field defects, resulting in partial functional dependence.







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**Figure 4.** Digital subtraction angiography image of the left pulmonary artery before embolization. Selective injection of the feeding artery demonstrates a large pulmonary arteriovenous malformation with a dilated aneurysmal sac and early filling of a draining vein.

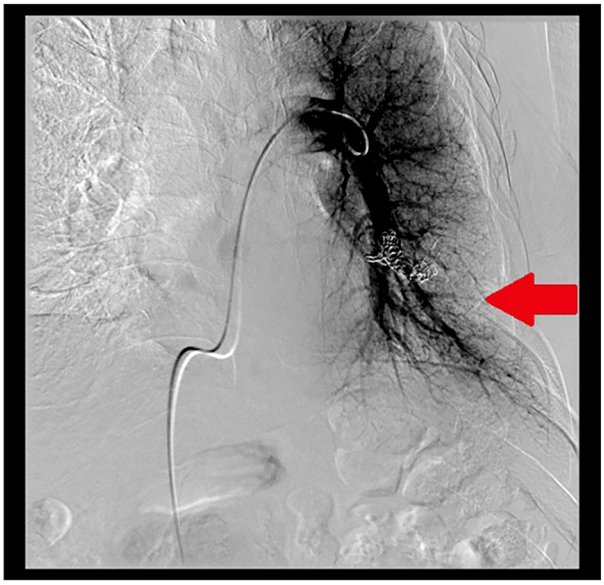

**Figure 5.** Post-embolization angiogram. The feeding artery is completely occluded by multiple coils (visible as radiopaque densities). No contrast filling of the pulmonary arteriovenous malformation sac or draining vein is evident, confirming complete occlusion.

## Discussion

This case provides 2 key clinical insights. First, it highlights the limitations of anticoagulation alone in preventing recurrent paradoxical embolism due to a PAVM. Second, it illustrates the complexity of clinical decision-making when a PAVM is diagnosed in the setting of hemorrhagic transformation of an acute stroke, underscoring the need for individualized timing of definitive treatment.

Before discussing these clinical insights in detail, it is important to consider the underlying etiology of the PAVM in our patient. PAVMs are most commonly associated with HHT [1,4]. Formal genetic testing was not performed due to financial constraints and family preference. In the present case, HHT was considered unlikely given the absence of clinical features defined by the Curaçao diagnostic criteria (no history of recurrent epistaxis, no mucocutaneous telangiectasia, no relevant family history, and no hepatic vascular malformations detected on ultrasound). Acquired PAVMs can occur in relation to factors such as chest trauma, cardiothoracic surgery, chronic liver disease, and infections [1]. The etiology of the PAVM in our patient remains uncertain. Although the onset of dyspnea coincided with chest trauma, a definitive causal relationship is difficult to establish because purely traumatic PAVMs are exceedingly rare in the literature [1,2]. It is possible that the patient had a preexisting, asymptomatic congenital PAVM that became symptomatic or was incidentally detected after trauma-related changes in hemodynamics or vascular integrity. However, the absence of pre-trauma imaging precludes confirmation. The most plausible explanation is therefore an idiopathic PAVM, with clinical manifestations potentially exacerbated by trauma.

PAVMs can be asymptomatic or present with a wide range of clinical manifestations, depending on factors such as size, number, type (simple vs complex), and blood flow [1,7]. Common symptoms include dyspnea, hemoptysis, chest pain, and cyanosis. Serious neurologic complications, which often constitute the initial presentation, include migraine, intracranial hemorrhage, transient ischemic attack, cerebral embolism, and brain abscess [4,7,8]. In our patient, the relatively large feeding artery likely contributed to clinically significant dyspnea and neurologic complications. PAVMs are more commonly located in the lower lobes, as observed in the present case. Dyspnea is often exacerbated in the upright position due to gravity-induced increases in blood flow to lower lobe PAVMs, which augment right-to-left shunting [1,2,4]. This mechanism likely explains the patient's postural dyspnea.

Particularly in young patients without vascular risk factors, stroke is increasingly recognized as frequently caused by paradoxical embolization; PAVMs represent an uncommon source [9,14]. More common causes of paradoxical embolization include patent foramen ovale [15]. In our patient, cardiogenic embolism was excluded, and no clinically significant stenosis or vascular dissection was identified in the posterior circulation. After interventional treatment, no further cerebral embolic





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events occurred. Therefore, it is likely that paradoxical embolization resulted from the PAVM, supported by the temporal association and the absence of post-treatment recurrence. Although precise epidemiological data are limited, PAVMs are associated with substantial morbidity and mortality due to complications such as stroke and brain abscess. Untreated PAVMs carry a mortality rate as high as 50%, compared with approximately 3% in treated patients [1]. Accordingly, accurate diagnosis and effective treatment are essential.

Through advances in embolization techniques and devices, percutaneous transcatheter embolization has become the preferred treatment for PAVMs, offering high success rates and low complication rates [10,12,13]. It is widely accepted that PAVMs with a feeding artery diameter of at least 2 mm, progressive enlargement, paradoxical embolization, symptomatic hypoxemia, or other serious complications warrant active intervention [1,11]. In the present case, the feeding artery was large, and neurological complications had already occurred, making embolization both necessary and appropriate.

The clinical course in the present case is consistent with previously reported cases of recurrent embolism due to PAVMs. Similar to the patient described by Alet et al, who experienced no further neurological events after successful PAVM embolization, our case reinforces the critical role of embolization in eliminating right-to-left shunts and preventing recurrence [9]. García-Alcántara et al reported 2 cases of ischemic stroke treated with embolization followed by antiplatelet therapy; no recurrence was documented during 24 months of follow-up, consistent with the 4-year recurrence-free outcome observed in our patient [16]. Collectively, these findings support the efficacy of percutaneous transcatheter embolization as first-line therapy for PAVM-related stroke.

However, the present case also exhibits distinct features that merit attention. First, both embolic events occurred in the posterior cerebral artery territory, a pattern less commonly emphasized in the literature on PAVM-related stroke. Although this may be coincidental and no specific vascular predisposition was identified, it underscores the need for heightened vigilance when strokes occur in atypical territories in the context of a right-to-left shunt. Second, the presence of hemorrhagic transformation on initial imaging led to a deliberate delay in intervention, during which the patient experienced a recurrent event despite therapeutic anticoagulation. This clinical dilemma – balancing the risk of recurrent embolism against the potential for exacerbating intracranial hemorrhage – has been infrequently addressed in prior reports and highlights the importance of timely definitive treatment when the acute bleeding risk has subsided. The present case also demonstrates that anticoagulation, although standard for DVT and often initiated empirically, may be insufficient to prevent paradoxical embolism in the presence of a high-flow structural shunt such as a PAVM.

It is important to note that clinical stability does not exclude residual shunting [17]. Although our patient remains asymptomatic, follow-up thoracic CT is recommended to confirm persistent occlusion. For untreated patients or those with small PAVMs, thoracic CT surveillance every 5 to 10 years is recommended to identify possible progression [18].

## Conclusions

This report reinforces that PAVM should be included in the differential diagnosis of cryptogenic stroke, particularly in cases of recurrence despite anticoagulation. Once diagnosed, timely and individualized assessment for definitive interventional treatment is essential. The presence of mild hemorrhagic transformation in acute stroke requires careful risk-benefit evaluation but should not be considered an absolute contraindication to percutaneous transcatheter embolization, which is critical to prevent disabling recurrent events.

### Institution Where Work Was Done

First Hospital of Qinhuangdao, Qinhuangdao, China

### Patient Consent

The patient was treated in accordance with the Declaration of Helsinki. The patient provided consent for all treatments and procedures, along with use of their clinical data and images for scientific publication.

### Disclosure

The authors used DeepSeek for language polishing during manuscript revision. The authors take full responsibility for the final content.

### Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.







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