Conquering a Giant High-Flow Pulmonary Arteriovenous Malformation Under Real-World Constrains Using Combined Amplatzer Vascular Plug I and Coil Embolization

Anak Agung Ngurah Bagus Satya Sueningrat, MD[1]*, Bramadi Nugroho, MD[2], Dita Karini Ratnaningsih, MD[2], Anantia Sari Utami, MD[2]

[1]*Department of Radiology, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada, Yogyakarta, Indonesia*

[2]*Department of Radiology, Dr. Soeradji Tirtonegoro General Hospital, Klaten, Indonesia*

***Correspondence:** Sueningrat, Anak Agung Ngurah Bagus Satya Sueningrat; Department of Radiology, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada, Yogyakarta, Indonesia

dr.agungsatya@gmail.com

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AUTHORS' CONTRIBUTIONS

The authors confirm contribution to the paper as follows: Anak Agung Ngurah Bagus Satya Sueningrat contributed to the original draft preparation as well as review and editing of the manuscript. Bramadi Nugroho, Dita Karini Ratnaningsih, and Anantia Sari Utami contributed to manuscript review and supervision. All authors critically reviewed and approved the final version of the manuscript.

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DISCLOSURES

The authors have no financial or competing interests to disclose.

CONSENT

Non-written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

HUMAN AND ANIMAL RIGHTS

No experiments were performed on human or animal experiments.

ABSTRACT

Background: Pulmonary arteriovenous malformations are uncommon pulmonary vascular abnormalities characterized by direct arteriovenous communications, resulting in intrapulmonary right-to-left shunting. Although often asymptomatic until early adulthood, pulmonary arteriovenous malformations may cause serious complications, including hypoxemia and neurological events such as cerebral abscess and ischemic stroke. Giant high-flow Pulmonary arteriovenous malformations pose additional therapeutic challenges due to their complex hemodynamics.

Objective: To demonstrate the feasibility and effectiveness of a combined vascular plug I and coil embolization strategy for treating a giant high-flow pulmonary arteriovenous malformation under real-world device availability and cost constraints.

Case Report: A 34-year-old male presented with severe headache, chronic exertional dyspnea, and difficulty gaining weight. Brain imaging revealed a cerebral abscess with associated chronic cerebral infarction. Contrast-enhanced thoracic computed tomography demonstrated a giant high-flow PAVM in the apical segment of the right upper lobe supplied by a markedly dilated feeding artery, along with pneumonia in the same segment suspected to represent pulmonary tuberculosis. Although Amplatzer Vascular Plug II was initially recommended, embolization was performed using Amplatzer Vascular Plug I combined with adjunctive coils due to financial limitations, achieving complete occlusion.

Discussion: True pulmonary arteriovenous malformations are inherently high-flow vascular malformations due to direct arteriovenous shunting and loss of pulmonary capillary filtration, explaining the neurological presentation. Concomitant pneumonia likely exacerbated hypoxemia through ventilation–perfusion mismatch. In giant high-flow lesions, combined plug-and-coil embolization provides effective flow control and durable occlusion, while normalization of pulmonary hemodynamics may contribute to improvement of coexisting pulmonary infection.

Conclusion: Giant high-flow Pulmonary arteriovenous malformations represent a complex endovascular challenge. In this case, combined Amplatzer Vascular Plug I and coil embolization resulted in favorable clinical and radiological outcomes, supporting individualized, multimodal endovascular strategies under real-world constraints.

Keyword: Pulmonary arteriovenous malformation, High-flow vascular malformation, Amplatzer vascular plug, Coil embolization, Cerebral abscess.

CASE REPORT

CASE REPORT

Case Presentation

A 34-year-old male presented to the emergency department with a chief complaint of severe headache. The patient also reported long-standing symptoms of exertional dyspnea that interfered with daily activities, as well as difficulty gaining weight, and frequent nosebleed which had been present for several years. By performing anamnesis, physical examination and imaging 2 from the 4 Curaçao criteria are met which are epistaxis and visceral lesions, in which the patients could be diagnose with possible hereditary hemorrhagic telangiectasis (HHT).

Image Findings

A non-contrast multislice computed tomography (MSCT) scan of the head performed at presentation revealed a hypodense lesion with extensive surrounding edema in the right parieto-occipital lobe, raising suspicion for cerebral metastasis or astrocytoma. Subsequent contrast-enhanced magnetic resonance imaging (MRI) of the brain performed two days later demonstrated imaging features consistent with a cerebral abscess located in the right temporoparietal region, measuring AP 1.5 × LL 1.4 × CC 1.1 cm, accompanied by surrounding vasogenic edema. Additionally, chronic infarction was noted in the anterior horn of the left caudate nucleus. (Figure 1).

In order to identify the potential source of infection and to further evaluate the patient's chronic respiratory symptoms, a contrast-enhanced MSCT scan of the thorax was performed. The examination revealed a giant pulmonary arteriovenous malformation located in the apical segment of the right upper lobe, supplied by a markedly dilated feeding artery arising from the right superior pulmonary artery measuring 0.9 cm in diameter and draining into the right apical segmental pulmonary vein measuring 0.6 cm in diameter. The AVM nidus measured approximately 4.08 × 3.28 × 3.15 cm (anteroposterior × transverse × craniocaudal), consistent with a giant high-flow pulmonary arteriovenous malformation. In addition, pneumonia involving the apical segment of the right upper lobe was identified and clinically suspected to represent pulmonary tuberculosis, which may have acted as an aggravating factor contributing to the patient's respiratory symptoms (Figure 2).

Management

Endovascular embolization was planned, however, the procedure was delayed for approximately two months due to insurance and financial constraints. Although the use of an Amplatzer Vascular Plug II (AVP II) was proposed based on the high-flow characteristics of the lesion, only an Amplatzer Vascular Plug I (AVP I) was approved by the insurance provider. Consequently, embolization was performed using a combined strategy of AVP I and coil deployment to minimize the risk of device migration.

The procedure was performed under fluoroscopic guidance using a double-access, double-puncture technique via the bilateral femoral veins. A 260-cm guidewire and a 6-Fr JR 3.5 guiding catheter were utilized. Pulmonary angiography confirmed the presence of an arteriovenous malformation supplied by the right superior pulmonary arterial branch. A 12 × 8 mm AVP I was deployed within the feeding artery, followed by adjunctive coil embolization using Optima Complex-18 coils (9 × 6 mm; 30 cm) delivered posterior to the vascular plug via a Renegade STC microcatheter. The coils were deployed to reduce antegrade flow toward the vascular plug and to minimize the risk of device migration. Due to residual flow observed on control angiography, an additional Optima Complex-18 coil of identical size was subsequently placed. Final angiography demonstrated complete occlusion of the feeding artery, and the procedure was considered technically successful (Figure 3).

Follow-up

At two-month follow-up, contrast-enhanced thoracic imaging demonstrated a reduction in the AVM nidus size to 3.26 × 2.98 × 2.92 cm (anteroposterior × transverse × craniocaudal), with stable positioning of the embolic materials and no evidence of recanalization (Figure 4). Clinically, the patient reported marked improvement, including resolution of dyspnea, improved exercise tolerance, and gradual weight gain, allowing him to resume work without limitation. In addition, respiratory symptoms related to the previously identified apical pneumonia suspected to represent pulmonary tuberculosis showed clinical improvement. Although follow-up imaging demonstrated increased consolidation density, the overall extent of the infiltrative area was markedly reduced compared with prior studies, suggesting interval improvement of the inflammatory process (Figure 5).

DISCUSSION

Pulmonary arteriovenous malformations (PAVMs) are very rare pulmonary vascular abnormalities defined by a direct connection between pulmonary arteries and veins, creating a right-to-left shunt that bypasses the capillary bed and can lead to hypoxemia and complications such as paradoxical embolism, stroke, or brain abscess. Many patients remain asymptomatic, making imaging—particularly contrast-enhanced CT—essential for diagnosis and treatment planning, with transcatheter embolization as the preferred therapy.

Etiology & Demographics

PAVMs allows blood to bypass the capillary network and resulting in intrapulmonary right-to-left shunting. The estimated prevalence is approximately 1 in 2,630 individuals, and most cases are not clinically recognized until early adulthood and significant size PAVMs were observable more frequently in females (49.8 %) than in males (44.8 %) population [1,2]. Patients may present with respiratory manifestations such as dyspnea and hemoptysis, while extra-pulmonary complications

predominantly involve the central nervous system, including stroke and brain abscess, emphasizing the clinical significance of timely diagnosis and management [1,3,4].

More than 80% of PAVMs occur in association with hereditary hemorrhagic telangiectasia (HHT), whereas the remaining cases arise from idiopathic, traumatic, or postsurgical etiologies, including congenital heart disease palliation such as the Fontan procedure [1]. From a hemodynamic standpoint, PAVMs constitute high-flow, low-resistance vascular shunts due to the absence of an intervening capillary bed, a feature that contributes to both their clinical consequences and technical complexity during endovascular treatment [4,5].

According to the ISSVA classification, true PAVMs are categorized as high-flow vascular malformations due to the presence of a direct arterial-to-venous communication that bypasses the capillary bed, resulting in a low-resistance, high-velocity shunt [6,7]. In true PAVMs, the high-flow nature is primarily determined by the presence of a direct arteriovenous shunt bypassing the capillary bed, while the diameter of the feeding artery influences the magnitude of shunt flow rather than the flow classification itself [7]. The diameter of feeding arteries are consider large if it is over 3 mm [8]. This concept is particularly relevant to the present case, in which a markedly dilated feeding artery was identified, reflecting a substantial shunt burden and explaining the technical challenges encountered during endovascular treatment.

Clinical & Imaging Findings

Pulmonary arteriovenous malformations predispose patients to cerebral abscess formation due to the loss of the pulmonary capillary filtration mechanism, allowing microorganisms and septic emboli to bypass the lungs and enter the cerebral circulation through a right-to-left shunt. This abnormal shunting also permits non-septic emboli to reach the systemic circulation, thereby increasing the risk of ischemic cerebrovascular events, as demonstrated by the presence of chronic cerebral infarction in this patient. Previous studies have reported that brain abscess may represent the initial clinical manifestation of previously undiagnosed PAVMs, particularly in young and immunocompetent individuals without an identifiable primary source of infection, highlighting the central role of paradoxical embolization in both infectious and ischemic neurological complications [2,7,9]. In the present case, concomitant pneumonia suspected to represent pulmonary tuberculosis likely acted as an additional aggravating factor by increasing the pulmonary infectious burden and further impairing gas exchange. Active pulmonary infection may exacerbate hypoxemia and increase the risk of systemic dissemination of microorganisms in patients with unfiltered right-to-left shunts, thereby compounding the clinical impact of PAVMs [10].

On contrast-enhanced CT, PAVMs are characterized by a direct connection between a dilated feeding artery and draining vein through a vascular nidus, often with early venous opacification, while angiography confirms their high-flow nature with rapid arteriovenous shunting and early venous filling, consistent with ISSVA-defined high-flow vascular malformations [7,11]. The location of PAVMs usually located in pleura of lower lobe [12]. These imaging hallmarks were clearly demonstrated in the present case and played a crucial role in procedural planning and device selection.

Treatment & Prognosis

Transcatheter embolization has emerged as the preferred therapeutic approach for PAVMs, owing to its high efficacy and favorable safety profile. A range of embolic agents, including detachable occlusion balloons, coils, and vascular plugs, has been employed, particularly for large or complex lesions [4]. Nevertheless, the high-flow nature of these malformations may predispose to procedural challenges such as incomplete occlusion and device migration, highlighting the importance of careful device selection and, in selected cases, the use of combined embolization techniques to achieve durable treatment outcomes [4,5].

Coil embolization has historically been the cornerstone of PAVM treatment due to its flexibility, deliverability through microcatheters, and ability to achieve dense packing within the feeding artery or venous sac. However, coil-only embolization is associated with several limitations, particularly in high-flow or large-caliber feeding arteries, including a higher risk of recanalization, incomplete occlusion, and coil migration, especially when packing density is insufficient or when coils are deployed too proximally [4,5]. The limitations become more pronounced in giant or high-flow PAVMs, where rapid shunt flow may prevent stable thrombus formation.

Vascular plugs, particularly the AVP, are widely favored for the treatment of large or high-flow PAVMs owing to their enhanced stability, controlled deployment, and lower rates of recanalization when compared with coil embolization alone. Despite these advantages, plug-only embolization may be suboptimal in extremely high-flow lesions, as it requires suitable landing zones and may be complicated by delayed thrombosis, residual shunting, or device migration when flow control is inadequate or device undersizing occurs [4]. Among available devices, the AVP II is generally preferred over AVP I in high-flow settings due to its multilayer, trilobed nitinol architecture, which provides multiple occlusive planes and greater thrombogenicity, resulting in faster and more dependable vessel occlusion than the single-layer cylindrical configuration of AVP I (Table 1) [13].

In the present case, although AVP II was considered the optimal device, its use was restricted by availability and cost considerations. To address the inherent limitations of AVP I in high-flow conditions, a combined embolization strategy incorporating adjunctive coil deployment was implemented. The coils served to reduce antegrade flow, enhance thrombosis, and provide additional anchoring for the vascular plug, thereby

improving device stability and promoting complete and durable occlusion. This individualized, multimodal approach is consistent with existing evidence supporting combined embolization techniques when ideal device selection is not feasible, particularly in complex or giant high-flow PAVMs [4,13].

Double puncture technique is preferred so we could maintain vascular access after plug deployment by using 1 access route for delivery system carrying the Amplatzer Vascular Plug I (AVP I), while the second access is dedicated to the microcatheter for coil deployment which allows additional coils to be placed without removing or disturbing the plug delivery system. Another reason for using double puncture technique is to facilitate plug and coil combination technique which allows both embolic components to be deployed efficiently with greater control, to allow for additional embolization when necessary, and to enhance procedural safety.

Following successful endovascular treatment, the prognosis of PAVMs is generally favorable, with significant reduction in right-to-left shunt, improvement in oxygenation, and substantial decrease in the risk of neurological complications, including cerebral abscess and ischemic stroke. Previous studies have demonstrated that effective embolization of PAVMs markedly reduces the incidence of paradoxical embolization and prevents recurrent central nervous system events, particularly when complete occlusion of the feeding artery is achieved [4,7]. In the present case, successful exclusion of the high-flow shunt using a combined vascular plug and coil strategy resulted in both radiological and clinical improvement, as evidenced by a significant reduction in nidus size on follow-up imaging and stable positioning of embolic materials without evidence of recanalization.

Clinically, the patient experienced resolution of exertional dyspnea, improved exercise tolerance, and gradual weight gain, reflecting restoration of pulmonary capillary filtration and improved systemic oxygen delivery. Concurrently, respiratory symptoms related to the previously identified pneumonia suspected to represent pulmonary tuberculosis improved clinically, which may be partially attributed to normalization of pulmonary hemodynamics and improved ventilation–perfusion matching following exclusion of the high-flow shunt [9]. If the tuberculosis was identified as the underlying cause of pneumonia, the patient will be referred to the infectious disease/ pulmonology service and initiated on standard anti-tuberculous therapy according to national treatment guidelines. More importantly, no recurrent neurological symptoms were observed during follow-up, suggesting effective prevention of further paradoxical septic or non-septic embolic events. The presence of a chronic cerebral infarction likely represents prior embolic events occurring before diagnosis and treatment, whereas the absence of new ischemic or infectious lesions after embolization supports the protective role of timely endovascular intervention. These findings are consistent with existing literature indicating that early and complete embolization of high-flow PAVMs offers durable clinical benefit and favourable long-term prognosis when appropriate device selection and embolization strategy are employed [4].

Differential Diagnoses

The differential diagnosis of giant high-flow PAVMs include multiple of vascular and parenchymal abnormality such as pulmonary varix, which represents focal venous dilatation without a feeding artery, pulmonary artery aneurysm (PAA) which maintains connection with the pulmonary arterial system and lacks a draining vein, pulmonary sequestration which is a congenital disease that can resemble a vascular mass but is characterized by systemic arterial supply and infected lung parenchyma, hypervascular tumors or metastases, such as renal cell carcinoma or carcinoid tumors, may mimic PAVMs due to strong enhancement but typically demonstrate a solid component without direct artery-to-vein communication, and pulmonary hemangiomas are rare benign vascular tumors that lack a clear feeding artery and draining vein configuration. Contrast-enhanced CT is the primary imaging modality for evaluation. Careful evaluation of vascular anatomy especially identification of feeding arteries and draining veins is important for accurate diagnosis. Misdiagnosis may lead to inappropriate interventions, emphasizing the importance of a systematic imaging approach [14,15].

CONCLUSION

Giant high-flow pulmonary arteriovenous malformations constitute a complex endovascular challenge due to their high-velocity shunt physiology and associated risk of neurological complications. In this case, a tailored embolization strategy using a combination of AVP I and adjunctive coils provided effective flow control, stable device anchoring, and durable occlusion, resulting in significant radiological regression and marked clinical improvement. Beyond shunt exclusion, normalization of pulmonary hemodynamics following embolization was associated with clinical and radiological improvement of concomitant pneumonia suspected to represent pulmonary tuberculosis, highlighting the broader systemic and pulmonary benefits of timely intervention. This experience underscores the importance of individualized, multimodal endovascular approaches to achieve optimal outcomes in complex high-flow PAVMs, particularly when ideal device selection is constrained by real-world considerations.

TEACHING POINT

In the management of giant high-flow pulmonary arteriovenous malformations, a combined embolization strategy using Amplatzer Vascular Plug I and adjunctive coil deployment is an effective and pragmatic way which provides rapid flow reduction and a stable scaffold, while coils facilitate distal occlusion and prevent recanalization. This combined technique enhances procedural safety, minimizes the risk of device migration in high-flow shunts, and improves long-term occlusion outcomes.

QUESTIONS

Question 1: What is the primary hemodynamic characteristic of a giant pulmonary arteriovenous malformation **(PAVM)?**

A. Left-to-right shunt

B. Right-to-left shunt [4] **(applies)**

C. Bidirectional shunt

D. No significant shunting

Question 2: What is the main advantage of using the Amplatzer Vascular Plug I in high-flow PAVMs?

A. It promotes rapid endothelial proliferation

B. It reduces flow and provides a stable occlusion scaffold [4,5] **(applies)**

C. It dissolves thrombus within the malformation

D. It enhances arterial wall elasticity

Question 3: Why are coils often used in combination with vascular plugs in high-flow PAVM embolization?

A. To increase blood flow through the lesion

B. To provide diagnostic imaging contrast

C. To achieve distal occlusion and reduce recanalization risk [4] **(applies)**

D. To replace the need for vascular plugs entirely

Question 4: What is a key challenge when treating giant high-flow PAVMs in real-world settings?

A. Lack of imaging modalities

B. Excessive patient sedation requirements

C. Risk of device migration due to high flow dynamics [4,5] **(applies)**

D. Inability to access the pulmonary artery

Question 5: What is the primary reason for combining vascular plug and coil embolization in giant high-flow PAVMs?

A. To increase pulmonary arterial pressure

B. To enhance imaging visibility only

C. To improve flow control and ensure durable occlusion [4,5] **(applies)**

D. To eliminate the need for angiography

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FIGURES

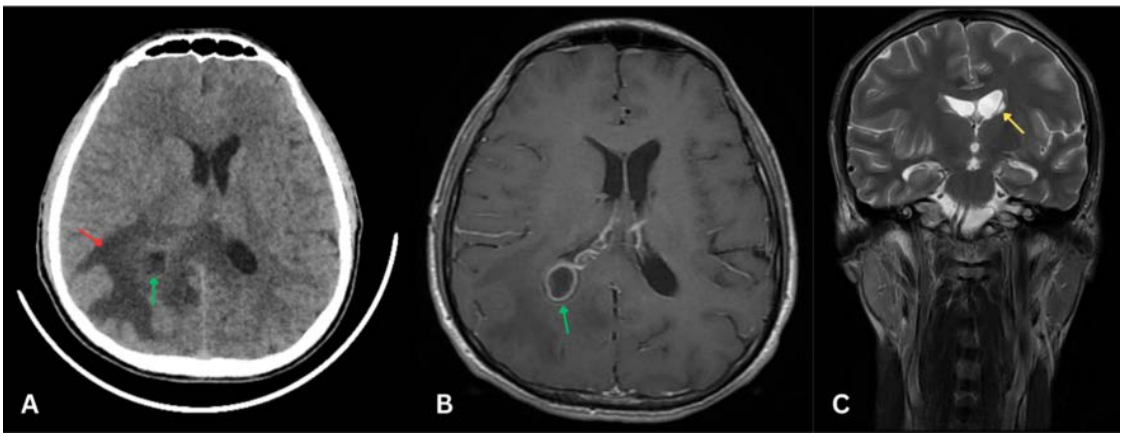


Figure 1: 34 years old male with severe headache.

FINDINGS: cerebral abscess, rim enhancing lesions and chronic infarct.

TECHNIQUE: Axial and coronal contrast enhanced brain MSCT scan.

Figure A shows a non-contrast multislice computed tomography (MSCT) scan of the brain demonstrating a suspicious hypodense lesion consistent with a cerebral abscess (green arrow), accompanied by surrounding vasogenic edema (red arrow).

Figure B shows contrast-enhanced T1-weighted brain MRI revealing a rim-enhancing lesion, a characteristic imaging feature of a cerebral abscess (green arrow).

Figure C shows a T2-weighted MRI sequence demonstrating a chronic infarction involving the left caudate nucleus.

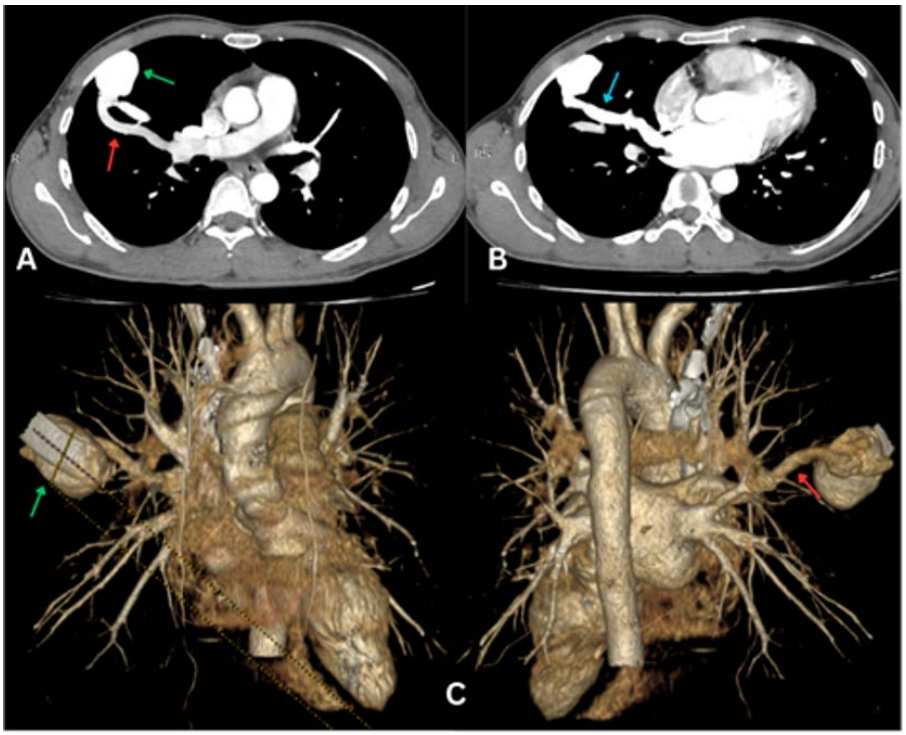


Figure 2: 34 years old male with PAVMs.

FINDINGS: giant high-flow pulmonary arteriovenous malformation in the apical segment of the right upper lobe and draining into the right apical segmental pulmonary vein and demontrastion of the AVM nidus and its feeding artery.

TECHNIQUE: Axial angiography of thorax MSCT scan and three-dimensional CT angiography reconstruction.

Figures A and B show contrast-enhanced MSCT of the thorax demonstrating a giant high-flow pulmonary arteriovenous malformation (4.08 × 3.28 × 3.15 cm; green arrow) in the apical segment of the right upper lobe, supplied by a dilated feeding artery arising from the right superior pulmonary artery (0.9 cm; red arrow) and draining into the right apical segmental pulmonary vein (0.6 cm; blue arrow).

Figure C shows a three-dimensional CT angiography reconstruction demonstrating the AVM nidus (green arrow) and its feeding artery (red arrow).

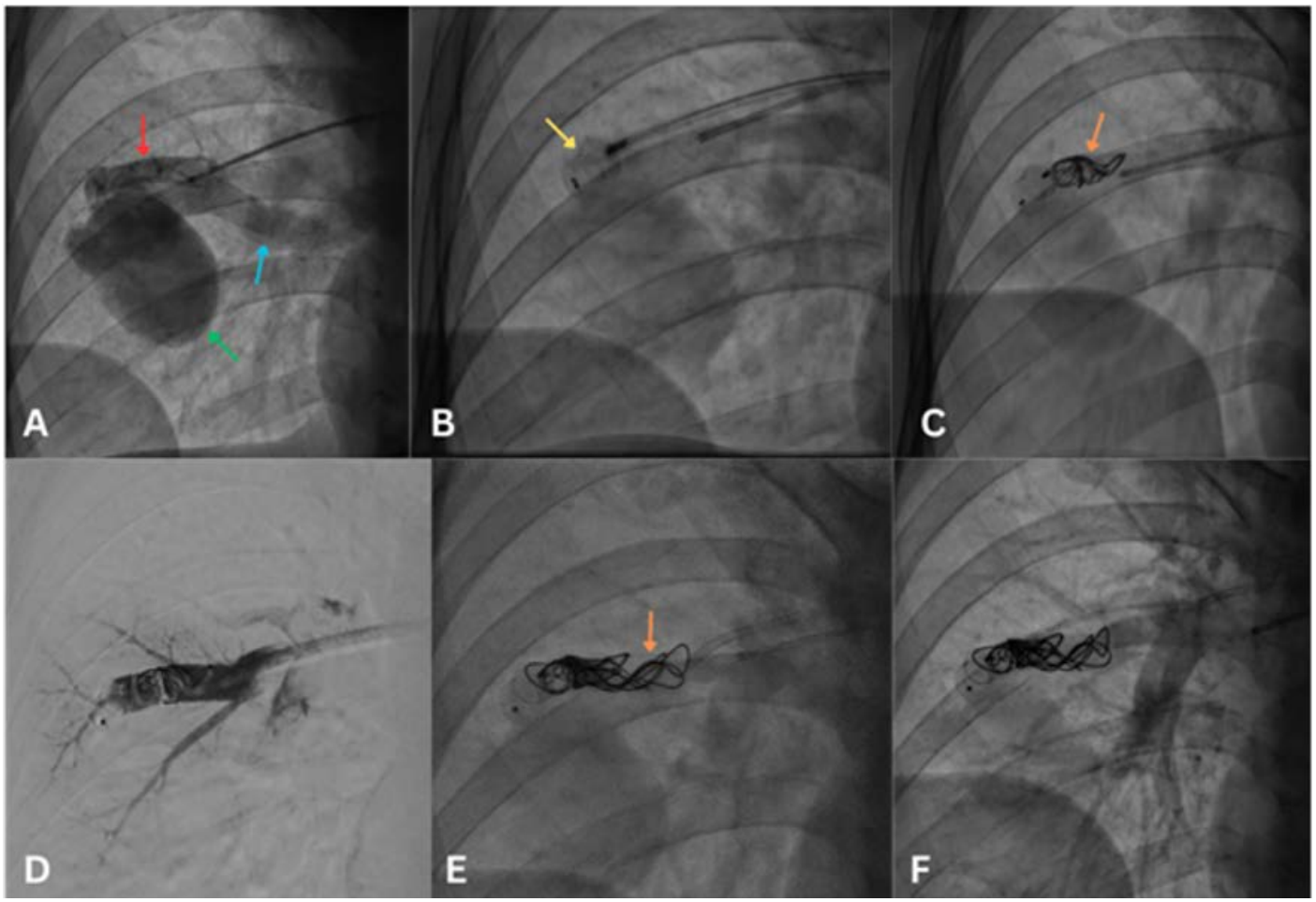


Figure 3: 34 years old male with PAVMs.

FINDINGS: PAVM with feeding artery and venous drainage, deployment of AVP I, coil embolization, persistent high flow opacifications of feeding artery and follow up angiography.

TECHNIQUE: Pulmonary angiography.

Figure A shows pulmonary angiography demonstrating a PAVM (green arrow) with a feeding artery arising from the right superior pulmonary artery (red arrow) and venous drainage into the right apical segmental pulmonary vein (blue arrow).

Figure B shows deployment of an AVP I within the feeding artery (yellow arrow).

Figure C demonstrates subsequent coil embolization (orange arrow).

Figure D shows persistent high-flow opacification of the feeding artery.

Figure E demonstrates additional coil embolization (orange arrow) placed to act as a flow-reducing buffer and minimize the risk of vascular plug migration.

Figure F shows follow-up angiography demonstrating marked reduction of flow within the feeding artery.

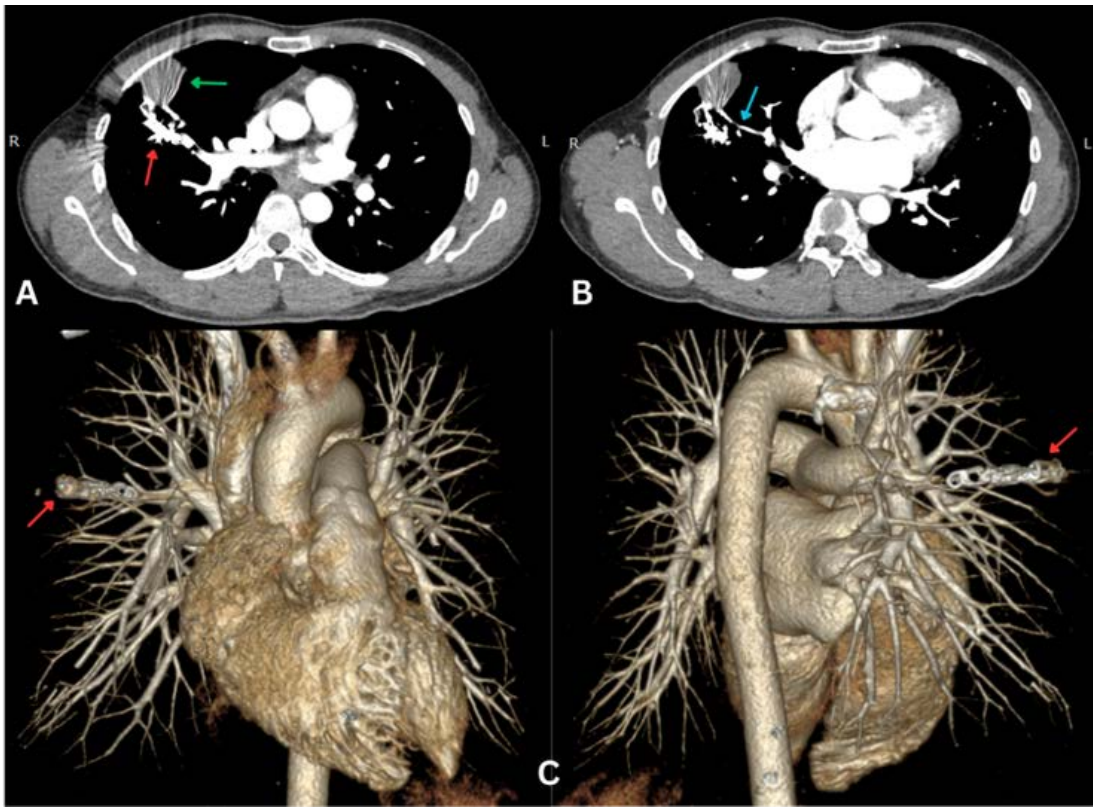


Figure 4: 34 years old male with PAVMs post embolization.

FINDINGS: Successful embolization with AVP I and coil well positioned within feeding artery.

TECHNIQUE: Axial angiography of thorax MSCT scan and three-dimensional CT angiography reconstruction.

Figure A shows the AVP I and coils remaining well positioned within the feeding artery, with no contrast opacification of the AVM nidus, indicating successful embolization. Figure B demonstrates a marked reduction in the caliber of the draining vein, with absence of minimal contrast filling.

Figure C shows a three-dimensional CT angiography (3D CTA) reconstruction confirming the position of the embolic materials.

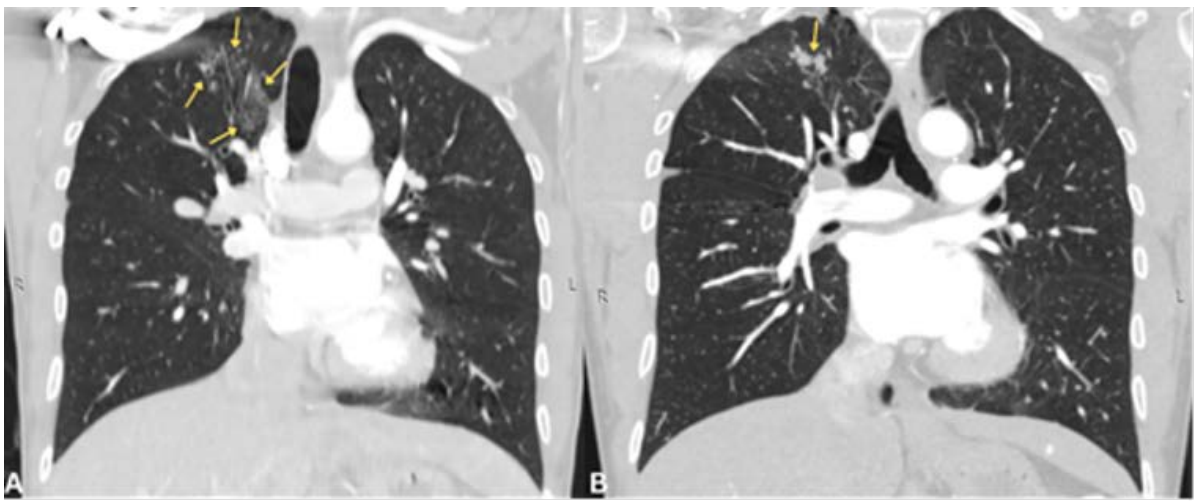


Figure 5: 34 years old male with PAVMs.

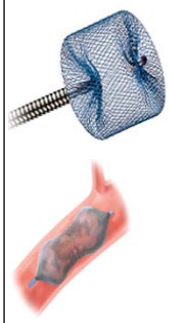
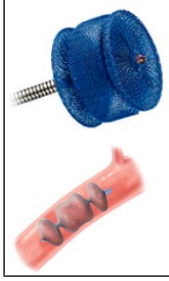
FINDINGS: Comparison between pre and post intervention.

TECHNIQUE: Coronal angiography of thorax MSCT.

Figure A shows contrast-enhanced thoracic CT performed prior to intervention demonstrating extensive ground-glass opacities in the apical segment of the right upper lobe, interpreted as pneumonia suspicious for pulmonary tuberculosis.

Figure B shows post-procedural CT angiography of the thorax demonstrating residual infiltrates in the same segment with a markedly reduced extent compared with pre-intervention imaging, suggesting interval improvement of the pneumonia following treatment of the pulmonary arteriovenous malformation.

Table 1: Comparison of Amplatzer Vascular Plug I and Amplatzer Vascular Plug II.

Version	Details	Comment
AVP I 	· Provides rapid occlusion with precise positioning in short landing zones. · Single-layered cylindrical disk. · Available in diameters ranging from 4 to 16 mm Requires 4–6F sheaths.	Not very thrombogenic in high flow situations, many times requiring placement. of other embolics Easy placement
AVP II 	· Provides faster occlusion and can adjust to the variable landing zones · Made out of a densely braided multilayer nitinol mesh with three components generating six barrier planes for acceleration of vascular occlusion. · Sizes: 3–22 mm. · Requires 4–7F sheaths.	Faster occlusion. Longer segment needed. The 3 lobes can be shortened by compression, allowing for better sealing and fitting into a shorter landing zone.

Summary Table

Aspect	Summary
Etiology	Mostly associated with hereditary hemorrhagic telangiectasia (HHT) (>80%); others include idiopathic, congenital, traumatic, or post-surgical causes (e.g., post-Fontan)
Incidence	Rare; estimated prevalence 1 in 2,630 individuals
Gender Ratio	More frequently in females (49.8 %) than in males (44.8 %) population
Age Predilection	Often asymptomatic until early adulthood; commonly diagnosed in young to middle-aged adults
Risk Factors	HHT (major), congenital heart disease, prior thoracic surgery, trauma; complications increased with high-flow shunt and large feeding artery
Treatment	First-line: transcatheter embolization (coils, vascular plugs, or combined approach); combined AVP I + coils effective in high-flow lesions
Prognosis	Generally favorable after successful embolization; improved oxygenation and reduced risk of stroke/brain abscess; low recurrence if complete occlusion achieved
Findings on Imaging	Chest X-ray: Well-defined round or lobulated opacity; may show vascular shadows extending to hilum CT Andiography: Direct connection between dilated feeding artery and draining vein with a nidus; early venous opacification; enlarged vessels Post Embolization CT Angiography: Absence of contrast filling in nidus; reduced size of lesion; stable position of coils/plug MRI Brain: Cerebral abscess or infarction due to emboli

Differential Table

Differential Diagnosis	Findings in CT Angiography
Pulmonary varix	Focal venous dilatation without a feeding artery,
Pulmonary artery aneurysm (PAA)	Maintains connection with the pulmonary arterial system and lacks a draining vein
Pulmonary sequestration which is a	Congenital disease that can resemble a vascular mass but is characterized by systemic arterial supply and infected lung parenchyma
Hypervascular tumors or metastases, such as renal cell carcinoma or carcinoid tumors	Solid component without direct artery-to-vein communication
Pulmonary hemangiomas	Lack a clear feeding artery and draining vein configuration.

KEYWORDS

Pulmonary arteriovenous malformation; High-flow vascular malformation; Amplatzer vascular plug, Coil embolization, Cerebral abscess.

ABBREVIATIONS

PAVMs = Pulmonary Arteriovenous Malformations (PAVMs)
CT = Computed Tomography
AVP I = Amplatzer Vascular Plug I
AVP II = Amplatzer Vascular Plug II
HHT = Hereditary Hemorrhagic Telangiectasia
MSCT = Multislice Computed Tomography
MRI = Magnetic Resonance Imaging
ISSVA = International Society for the Study of Vascular Anomalies