Brain Abscess Secondary to Pulmonary Arteriovenous Malformation: A Rare Complication

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AUTHORS' CONTRIBUTIONS

Van Luong Tran and Van Hoang Nguyen contributed to data acquisition and image analysis. Thi Hao Nguyen contributed to clinical management and data interpretation. Huu An Nguyen contributed to study conception, manuscript drafting, and final revision. Dang Luu Vu supervised the study and critically revised the manuscript. All authors read and approved the final manuscript.

DISCLOSURES

The authors declare that they have no financial or competing interests to disclose.

CONSENT

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

HUMAN AND ANIMAL RIGHTS

This study did not involve any experiments on humans or animals. All procedures were conducted in accordance with institutional ethical standards and the principles of the Declaration of Helsinki (as revised in 2000).

ABSTRACT

Pulmonary arteriovenous malformations are rare vascular anomalies characterized by abnormal connections between a pulmonary artery and vein, bypassing the capillary bed. Most patients remain asymptomatic, but right-to-left shunts allow septic or non-septic emboli to enter systemic circulation, leading to serious central nervous system complications. We report a case of a middle-aged woman with a brain abscess of initially unknown origin, in whom a solitary pulmonary arteriovenous malformation was incidentally detected on thoracic computed tomography. Although brain abscesses secondary to pulmonary arteriovenous malformation are very uncommon, this case underlines the importance of considering pulmonary arteriovenous malformation in patients with cryptogenic brain abscess.

CASE REPORT

BACKGROUND

Brain abscess is an uncommon but severe complication of pulmonary arteriovenous malformations (pAVMs) caused by paradoxical septic embolization. Because pAVMs may be clinically silent and occur without hereditary hemorrhagic telangiectasia, the etiology of infection can be overlooked. This case underscores the key role of imaging in detecting an occult pAVM in a patient with cryptogenic brain abscess and highlights the importance of definitive endovascular treatment to prevent recurrent neurological complications.

CASE REPORT

Clinical manifestation

A 55-year-old female presented to our hospital with complains of headache, nausea, vomiting, and fever for five days. Neurological examinations were unremarkable without neurological deficit. Laboratory tests were within normal limits except for mild polycythemia. Lumbar puncture revealed protein and white blood cell count (predominantly neutrophils), while cerebrospinal fluid (CSF) direct smear, CSF culture, and blood culture were negative. Brain magnetic resonance imaging (MRI) was indicated due to a suspicion of intracranial infection.

Imaging findings

Brain MRI was conducted with the protocol of axial T2 TSE, sagittal T1 TSE, axial DWI (b=0 and b=1000) with the apparent diffusion coefficient (ADC) map, and 3D T1 post-injection of Gadolinium. It revealed a focal lesion in the cortico-subcortical region of right occipital lobe. This lesion presented a center in hyposignal T1 (Figure 1A), hypersignal T2 (Figure 1B), restricted diffusion on diffusion weighted imaging (DWI) and the apparent diffusion coefficient (ADC) map (Figure 1C and 1D), without enhancement post-injection. It's capsule appeared as isosignal T1 (Figure 1A), hypo-hyper T2W (Figure 1B), with thin and smooth ring enhancement on post-contrast T1-weighted imaging (Figure 1E). There was also a remarkable perilesional edema in hyposignal T1, hypersignal T2 (Figured 1A,1 B). This lesion measuring approximately 2.8 x 4.7 x 2.9 cm (transverse x anteroposterior x craniocaudal) on 3D T1 post-contrast (Figure 1E). We also noted a leptomeningeal enhancement in the right temporo-occipital region (Figure 1E). There were no signs of thrombophlebitis or facial sinusitis associated (images not shown). Based on these findings and her clinical symptoms, a pyogenic brain abscess was the most likely diagnosis. The main differential diagnosis of brain abscess in this case was a cerebral metastases particularly necrotic adenocarcinoma.

Therefore, a chest X-rays and chest computed tomography (CT) was performed to rule out a pneumonia or a primary lesion. This exam revealed a serpiginous, well-circumscribed, non-calcified lesion connected with blood vessels of the right middle lobe (Figure 2A, 2C). Subsequently, CT angiography (CTA) demonstrated enhancement of the feeding artery, the aneurysmal part, and the draining vein of a pulmonary arteriovenous malformation (pAVM) (Figure 2B).

Management

A multidisciplinary team concluded that that the brain abscess was secondary to paradoxical septic embolization from an underlying pAVM. The patient therefore underwent targeted antibiotic therapy and treatment of the pAVP first, prior to surgical drainage of the cerebral abscess. Digital subtraction angiography (DSA) demonstrated a simple pAVM supplied by a single feeding artery with one draining vein in the right middle lobe (Figure 3A). Transcatheter coil embolization was performed using three coils (one 8/40 Ruby® coil (Penumbra Inc.,Alameda, CA, USA) and two 8/20 Interlock™ coils (Boston Scientific, Natick, MA, USA)) (Figure 3B). Final run control showed complete occlusion of the pAVM without complications (Figure 3B).

After embolization, the patient underwent neurosurgical drainage of the brain abscess. Purulent material was aspirated intraoperatively, but microbiological cultures were negative and no causative pathogen was identified.

Outcome and follow-up

At three-month follow-up, the patient had well recovered without neurological deficits. Follow-up pulmonary CTA confirmed complete embolization of the pAVM with coils in place (Figure 3C), and non-contrast brain CT demonstrated regression of the brain abscess (Figure 4).

DISCUSSION

Etiology & demographics

Pulmonary arteriovenous malformations (pAVMs) are rare pulmonary vascular anomalies characterized by direct communications between pulmonary arteries and veins, resulting in an intrapulmonary right-to-left shunt [1,2]. This shunt bypasses the normal filtering function of pulmonary capillaries, allowing paradoxical emboli, including septic emboli, to enter the systemic circulation. Consequently, patients with pAVMs are predisposed to neurological complications such as transient ischemic attacks, ischemic stroke, and brain abscesses [1,3].

Most pAVMs are associated with hereditary hemorrhagic telangiectasia (HHT), also known as Osler–Weber–Rendu syndrome. Approximately 70% of patients with pAVMs have underlying HHT, while about 15% of patients with HHT develop pAVMs [1,4]. HHT is an autosomal dominant disorder with an estimated prevalence of approximately 1 in 5000 individuals [4]. The Curaçao criteria are used for the clinical diagnosis of HHT include: (1) spontaneous recurrent epistaxis, (2) mucocutaneous telangiectasia, (3) visceral arteriovenous malformations, and (4) a first-degree relative with HHT [4]. The presence of three or more criteria confirms the diagnosis, two criteria suggest possible HHT, and fewer than two criteria make the diagnosis unlikely [4].

In the absence of clinical features suggestive of HHT, pAVMs are considered idiopathic. These idiopathic pAVMs are more often solitary and may lack the typical lower-lobe predominance observed in HHT-related lesions [2,5]. In our patient, no clinical or familial features of HHT were identified, consistent with an idiopathic solitary pAVM.

Clinical & imaging findings

pAVMs are frequently asymptomatic until adulthood. When present, pulmonary manifestations include exertional dyspnea, hypoxemia, secondary polycythemia, and less commonly hemoptysis [1,2]. Extra-pulmonary manifestations are predominantly neurological and result from paradoxical embolization through the right-to-left shunt [3]. Among these; brain abscess represents the most severe central nervous system complication and has been reported in approximately 5–10% of patients with pAVMs [6,7].

Mathis et al. described 26 cases of brain abscess associated with HHT, with infections predominantly caused by anaerobes organisms, particularly *Streptococcus* species, while staphylococcal infection was notably absent [6]. Clinically, patients often present with nonspecific symptoms such as fever, headache, nausea, and vomiting, which may delay diagnosis [7,8].

On magnetic resonance imaging (MRI), a brain abscess typically appears as a ring-enhancing lesion with central diffusion restriction on diffusion-weighted imaging (DWI), surrounded by vasogenic edema. These imaging features reliably distinguish abscesses from necrotic tumors or metastases [9,10]. Although the parietal lobe is the most frequently involved site, abscesses may occur in any cerebral region, including the occipital lobe [7].

In patients with cryptogenic brain abscess, thoracic imaging plays a crucial role in identifying an extracranial source of infection. Chest computed tomography (CT) may incidentally reveal a suspected pAVM, which should be further characterized with pulmonary CT Angiography (CTA). CTA is currently considered the imaging modality of choice for diagnosing pAVMs, as it accurately delineates the feeding artery, aneurysmal sac, and draining vein [11,12].

Treatment & prognosis

Treatment of pAVM is recommended even in asymptomatic patients due to the substantial risk of paradoxical embolic complications [4,13]. Endovascular embolization with detachable coils or vascular plugs is currently the standard of care, while surgical resection (segmentectomy or lobectomy) is reserved for lesions not amenable to endovascular treatment or in cases of embolization failure [13,14].

When a brain abscess is present, management requires a multidisciplinary approach combining prolonged intravenous antibiotic therapy with neurosurgical drainage when indicated [7,8]. Following successful embolization of pAVM and appropriate treatment of the intracranial infection, long-term outcomes are generally favorable, with a significant reduction in the risk of recurrent neurological events [5, 14].

Differential diagnoses

In patients presenting with a cryptogenic brain abscess, a pAVM should always be considered as a potential underlying source of septic emboli [1,3]. Other differential diagnoses for ring-enhancing intracranial lesions include high-grade glioma, metastatic disease, tuberculoma, and tumefactive demyelinating lesions [10]. However, marked diffusion restriction on DWI, together with clinical and laboratory findings, strongly favors the diagnosis of brain abscess [9].

TEACHING POINT

Pulmonary arteriovenous malformations produce a right-to-left shunt that allows septic emboli to bypass the pulmonary capillary filter, representing an important and frequently underrecognized cause of cryptogenic brain abscess. Brain MRI typically shows a ring-enhancing lesion with marked diffusion restriction, while pulmonary CT angiography demonstrates a direct artery-vein communication with an enhancing feeding artery, aneurysmal sac, and draining vein – key diagnostic features that enable timely and potentially curative endovascular treatment.

QUESTIONS

Question 1: Which mechanism best explains why pulmonary arteriovenous malformations can lead to brain abscess?

1. Increased pulmonary arterial pressure causing cerebral hypoperfusion.
2. Direct spread of pulmonary infection through lymphatic channels.
3. Bypass of the pulmonary capillary filter allowing septic emboli to enter the systemic circulation (applies).
4. Immune-mediated cerebral vasculitis.
5. Hematogenous spread from chronic sinusitis.

Explanation for question 1

Pulmonary arteriovenous malformations create a right-to-left shunt that bypasses the normal filtering function of pulmonary capillaries, allowing paradoxical emboli, including septic emboli, to reach the systemic circulation and the brain. This mechanism explains the occurrence of brain abscess in patients with pulmonary arteriovenous malformations. [Pulmonary arteriovenous malformations … resulting in an intrapulmonary right-to-left shunt … allowing paradoxical emboli, including septic emboli, to enter the systemic circulation.].

Question 2: Which imaging feature most reliably differentiates a brain abscess from a necrotic brain tumor on magnetic resonance imaging?

1. Presence of vasogenic edema.
2. Ring enhancement after contrast administration.
3. Central diffusion restriction on diffusion-weighted imaging (applies).
4. Leptomeningeal enhancement.
5. Hypointensity on T1-weighted images.

Explanation for question 2

Marked diffusion restriction within the central cavity of a ring-enhancing lesion is a key magnetic resonance imaging feature that distinguishes brain abscess from necrotic tumors or metastases, which typically do not show true diffusion restriction.

[On magnetic resonance imaging, a brain abscess typically appears as a ring-enhancing lesion with central diffusion restriction on diffusion-weighted imaging … These imaging features reliably distinguish abscesses from necrotic tumors or metastases.]

Question 3: Which statement regarding pulmonary arteriovenous malformations is correct?

1. Most pulmonary arteriovenous malformations are acquired secondary to pulmonary infections.
2. Pulmonary arteriovenous malformations usually present with hemoptysis in childhood.
3. Approximately 70% of patients with pulmonary arteriovenous malformations have underlying hereditary hemorrhagic telangiectasia (applies).
4. Idiopathic pulmonary arteriovenous malformations are typically multiple and bilateral.
5. Pulmonary arteriovenous malformations rarely cause

neurological complications

Explanation for question 3

Most pulmonary arteriovenous malformations are associated with hereditary hemorrhagic telangiectasia (HHT), while idiopathic pulmonary arteriovenous malformations are more often solitary. Neurological complications are well recognized in pulmonary arteriovenous malformations patients due to paradoxical embolization. [Approximately 70% of patients with pulmonary arteriovenous malformations have underlying hereditary hemorrhagic telangiectasia, while about 15% of patients with hereditary hemorrhagic telangiectasia develop pulmonary arteriovenous malformations.]

Question 4: Which imaging modality is currently considered the modality of choice for diagnosing pulmonary arteriovenous malformations?

1. Chest radiography.
2. Ventilation–perfusion scintigraphy.
3. Pulmonary computed tomography angiography (applies).
4. Magnetic resonance angiography.
5. Digital subtraction angiography as the first-line diagnostic tool.

Explanation for question 4

Pulmonary computed tomography angiography is the preferred imaging modality for diagnosing pulmonary arteriovenous malformations because it accurately delineates the feeding artery, aneurysmal sac, and draining vein. Digital subtraction angiography is mainly reserved for therapeutic planning and intervention. [Computed tomography angiography is currently considered the imaging modality of choice for diagnosing pulmonary arteriovenous malformations, as it accurately delineates the feeding artery, aneurysmal sac, and draining vein.]

Question 5: Which of the following statements regarding the management of pulmonary arteriovenous malformations is correct?

1. Treatment is indicated only in symptomatic patients.
2. Surgical resection is the first-line treatment in most cases.
3. Endovascular embolization is recommended even in asymptomatic patients due to embolic risk (applies).
4. Brain abscess should always be surgically drained before treating the pulmonary arteriovenous malformations.
5. Embolization does not reduce the risk of recurrent neurological events.

Explanation for question 5

Treatment of pulmonary arteriovenous malformations is recommended even in asymptomatic patients because of the significant risk of paradoxical embolic complications. Endovascular embolization is the standard of care and significantly reduces the risk of recurrent neurological events. [Treatment of pulmonary arteriovenous malformations is recommended even in asymptomatic patients due to the substantial risk of paradoxical embolic complications.]

REFERENCES

[1] Shovlin CL. Pulmonary arteriovenous malformations. *Am J Respir Crit Care Med.* 2019; 200(6): 648–658. PMID: 31411943.

[2] Cottin V, Chinet T, Lavolé A, et al. Pulmonary arteriovenous malformations in adults: A concise review. *Eur Respir Rev.* 2022; 31(165): 210190. PMID: 35675847.

[3] Andersen PE, Kjeldsen AD, Oxhøj H, et al. Neurologic manifestations of pulmonary arteriovenous malformations. *Stroke.* 2020; 51(7): 2117–2124. PMID: 32426901.

[4] Faughnan ME, Mager JJ, Hetts SW, et al. Second international guidelines for the diagnosis and management of hereditary hemorrhagic telangiectasia. *Ann Intern Med.* 2020; 173(12): 989–1001. PMID: 32894695.

[5] Buscarini E, Danesino C, Olivieri C, et al. Pulmonary arteriovenous malformations: Natural history and long-term outcomes. *Chest.* 2021; 159(4):1420–1429. PMID: 33129819.

[6] Mathis S, Dupuis-Girod S, Plauchu H, et al. Cerebral abscesses in hereditary hemorrhagic telangiectasia: A clinical and microbiological evaluation. *Clin Infect Dis.* 2018; 66(12): 1823–1829. PMID: 29309571.

[7] Brouwer MC, Tunkel AR, McKhann GM, van de Beek D. Brain abscess. *N Engl J Med.* 2014; 371(5): 447–456. PMID: 25075836.

[8] Muzumdar D, Jhawar S, Goel A. Brain abscess: An overview. *J Neurosurg Sci.* 2020; 64(3): 259–270. PMID: 32105291.

[9] Krings T, Geibprasert S. Diffusion-weighted imaging of brain abscesses. *Neuroimaging Clin N Am.* 2020; 30(1): 49–66. PMID: 31791646.

[10] Thurnher MM, Schindler E, Thurnher SA. Imaging of ring-enhancing brain lesions. *Radiol Clin North Am.* 2019; 57(6): 1075–1092. PMID: 31587999.

[11] Saboo SS, Chamarthy MR, Bhalla S, et al. Pulmonary arteriovenous malformations: Diagnosis and treatment. *Radiographics.* 2021; 41(4): 1021–1041. PMID: 34115022.

[12] Fitoz S, Atasoy C, Akyar S. Imaging findings of pulmonary arteriovenous malformations. *Diagn Interv Radiol.* 2021; 27(2): 245–253. PMID: 33403635.

[13] Trerotola SO, Pyeritz RE. PAVM embolization: An update. *Radiology.* 2019; 290(3): 720–731. PMID: 30620622.

[14] Mager JJ, Overtoom TT, Blauw H, et al. Long-term outcome after embolotherapy of pulmonary arteriovenous malformations. *J Vasc Interv Radiol.* 2023; 34(1): 45–54. PMID: 36209877.

FIGURES

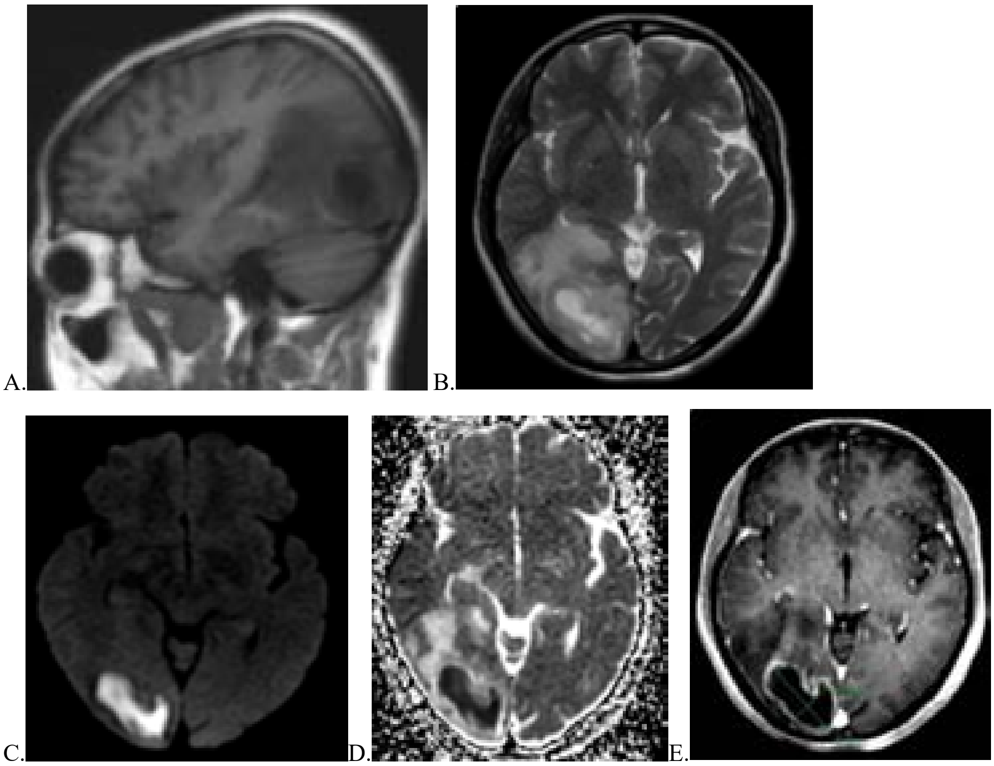

FIGURE 1: 55-year-old female with brain abscess secondary to pulmonary arteriovenous malformation.

FINDINGS: Brain magnetic resonance imaging demonstrates a well-defined intraparenchymal abscess located in the right occipital lobe, measuring approximately 28 × 47 × 29 mm. The lesion shows a central fluid-like component with hypointense signal on T1-weighted images (**Figure 1A**) and hyperintense signal on T2-weighted images (**Figure 1B**). There is marked diffusion restriction on diffusion-weighted imaging (DWI) with corresponding low signal on the apparent diffusion coefficient (ADC) map (**Figures 1C,1D**). The lesion is surrounded by vasogenic edema and demonstrates a thin, smooth, and strongly enhancing capsule on post-contrast 3D T1-weighted images (**Figure 1E**). **TECHNIQUE:** Contrast-enhanced brain MRI including sagittal T1-weighted imaging, axial T2-weighted imaging, axial diffusion-weighted imaging with ADC mapping, and post-contrast 3D T1-weighted sequences.

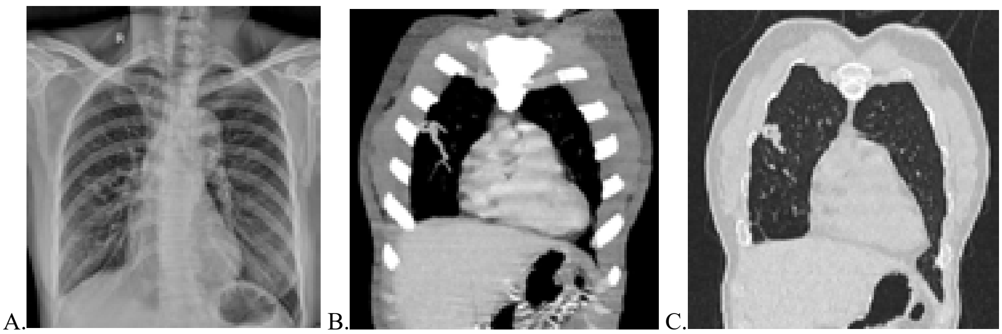

Figure 2: 55-year-old female with brain abscess and underlying pulmonary arteriovenous malformation

FINDINGS: Frontal chest radiograph reveals a tubular opacity projected over the anatomical region of the right middle lobe, raising suspicion for a vascular abnormality (**Figure 2A**). Pulmonary CT angiography demonstrates a direct-type pulmonary arteriovenous malformation located within the right middle lobe, characterized by a single feeding artery arising from the segmental branch of segment V of the right middle lobe pulmonary artery and a draining vein, without an intervening nidus (**Figure 2B**, coronal MPR pulmonary arterial phase). No associated parenchymal lung abnormality is identified (**Figure 2C**). **TECHNIQUE:** Pulmonary CT angiography performed using 100–120 kVp, 150–300 mAs, pitch 0.9–1.2, with thin-section axial acquisition (reconstructed slice thickness 0.625 mm) and multiplanar reconstructions (MPR). High-concentration iodinated contrast material (Ultravist 370 mgI/mL) was administered at a volume of 60–80 mL with an injection rate of 4.5 mL/s.

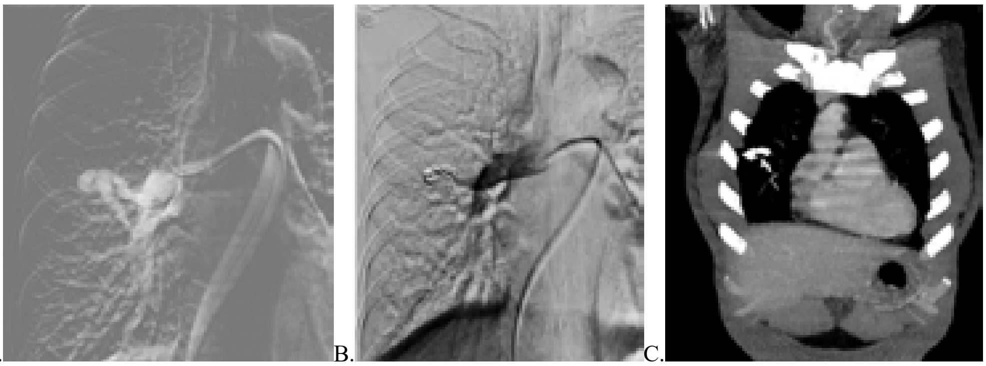

Figure 3: Endovascular treatment of pulmonary arteriovenous malformation in a 55-year-old female.

FINDINGS: Digital subtraction angiography demonstrates selective catheterization of the right middle lobe pulmonary artery using an 8F Neuromax guiding catheter with a 5F JB catheter (**Figure 3A**). Superselective catheterization of the segment V branch of the right middle lobe pulmonary artery was achieved using a 2.4F microcatheter, followed by embolization of the feeding artery with three detachable coils (one Ruby® coil 8 mm × 40 cm and two Interlock™ coils 8 mm × 20 cm). Post-embolization angiography confirms complete occlusion of the feeding artery (**Figure 3B**). Follow-up pulmonary CT angiography performed two months after embolization demonstrates complete exclusion of the pulmonary arteriovenous malformation (**Figure 3C**,**coronal MPR**).

TECHNIQUE: Pulmonary CT angiography performed using 100–120 kVp, 150–300 mAs, pitch 0.9–1.2, thin axial sections reconstructed at 0.625 mm, with multiplanar reconstructions. High-concentration iodinated contrast (Ultravist 370 mgI/mL) was injected at a volume of 60–80 mL and a flow rate of 4.5 mL/s.

Figure 4: Postoperative imaging findings following surgical drainage of brain abscess.

FINDINGS: One week after surgical evacuation of the brain abscess, microbiological culture of the aspirated purulent material showed no evidence of bacterial growth, consistent with a sterile brain abscess. Non-contrast brain CT demonstrates a postoperative defect in the right occipital lobe, consistent with the surgical cavity, associated with mild ex-vacuo dilatation of the ipsilateral occipital horn of the lateral ventricle. A right occipital craniotomy defect is also noted.

TECHNIQUE: Non-contrast brain CT performed with 120 kVp, 200–300 mAs, pitch 0.7–1.0, axial slice thickness 5 mm, with optional thin-section reconstruction (0.5 mm) and multiplanar reconstructions (MPR).

KEYWORDS

Pulmonary arteriovenous malformation, cryptogenic brain abscess, imaging, embolization, hereditary hemorrhagic telangiectasia. ..

ABBREVIATIONS

PAVM = Pulmonary Arteriovenous Malformation
MRI = Magnetic Resonance Imaging
CT = Computed Tomography
CTA = Computed Tomography Angiography
DSA = Digital Subtraction Angiography
HHT = Hereditary Hemorrhagic Telangiectasia