

Occlusion of a Pulmonary Arteriovenous Fistula With an Amplatzer Vascular Plug

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Pulmonary arteriovenous malformations are rare anomalies that carry a considerable risk of serious complications such as cerebral thromboembolism or abscess and pulmonary hemorrhage. The first-line treatment of such malformations is detachable coil or balloon embolotherapy. However, coils and balloons may migrate and cause paradoxical embolism especially in malformations with large arteriovenous shunts. We report a case in which we used a new vascular occlusion device (Amplatzer vascular plug) to occlude a pulmonary arteriovenous fistula in a patient with Rendu–Osler–Weber syndrome.

Key words: *Pulmonary arteriovenous malformations. Amplatzer vascular plug. Embolotherapy. Rendu–Osler–Weber Syndrome. Hereditary hemorrhagic telangiectasia.*

Introduction

A pulmonary arteriovenous malformation is an abnormal communication between a pulmonary artery and pulmonary vein—a condition that can be congenital or acquired. First-line treatment for such malformations is embolotherapy owing to high success rates and low percentages of complications.¹

The Amplatzer vascular plug (AGA Medical, Golden Valley, Minnesota, USA) is a new self-expanding device for occluding fistulas in peripheral veins or arteries.

We report a case in which we used the Amplatzer vascular plug to successfully close a pulmonary arteriovenous fistula in a patient with Rendu–Osler–Weber syndrome.

Case Description

The patient was a 35-year-old man who experienced sudden loss of consciousness. He arrived at our hospital conscious, with

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Oclusión de una fístula arteriovenosa pulmonar con el tapón vascular Amplatzer

Las malformaciones arteriovenosas pulmonares son anomalías poco frecuentes del pulmón, que entrañan un riesgo considerable de complicaciones graves como la tromboembolia cerebral, el absceso cerebral y la hemorragia pulmonar. La embolización con coils o con balones desprendibles es el tratamiento de elección. Sin embargo, éstos pueden migrar y producir embolia paradójica, especialmente en las malformaciones con comunicaciones arteriovenosas grandes.

Presentamos un caso en el que utilizamos un nuevo dispositivo de oclusión vascular (tapón vascular Amplatzer) para ocluir una fístula arteriovenosa pulmonar en un paciente con síndrome de Rendu–Osler–Weber.

Palabras clave: *Malformaciones arteriovenosas pulmonares. Tapón vascular Amplatzer. Embolización. Síndrome de Rendu–Osler–Weber. Telangiectasia hemorrágica hereditaria.*

right hemiparesis and dysphasia. Magnetic resonance imaging revealed small ischemic lesions in the left perisylvian region of the cortex. Doppler ultrasound of the supraaortic trunks showed normal patterns and transesophageal echocardiography revealed a patent foramen ovale with an aneurysmal septum and a large right-to-left shunt at rest.

While hospitalized the patient suffered episodes of severe epistaxis that were controlled with anterior and posterior packs. Telangiectasia detected on the patient's tongue and lips and his report of telangiectasis in successive generations of the maternal side of his family led to a diagnosis of Rendu–Osler–Weber syndrome and to the decision to postpone closure of the foramen ovale until the epistaxis was controlled, given that antiplatelet therapy would be needed for several months after the occlusion procedure. Faced with this situation, prophylactic embolization of the internal maxillary arteries was performed using spherical particles 500 to 700 μm in diameter; there were no complications.

Computed tomography scans of the chest were also ordered, given the high rate of association of pulmonary arteriovenous malformation with Rendu–Osler–Weber syndrome. The scans revealed a nodular lesion suggestive of a pulmonary arteriovenous fistula in the lower right lobe that was later confirmed by pulmonary angiography. At the point of arteriovenous anastomosis a mobile aneurysmal dilatation was identified. It was interpreted as a thrombus.

First, occlusion of the pulmonary arteriovenous fistula (Figure 1) was performed with the patient under local anesthesia; that procedure was followed a week later by closure of the

foramen ovale. After right femoral vein puncture and placement of a 6F introducer sheath, a 5F pigtail catheter was advanced through the sheath to the level of the right pulmonary artery and an angiogram was taken showing the simple arteriovenous fistula (a feeding artery and a drainage vein) in the right lower lobe. The pigtail catheter was then exchanged for a 6F guiding catheter, which was advanced through the feeding artery to a point as close to the fistula as possible for distal occlusion. Then, a 10-mm Amplatzer plug for a 7-mm-diameter feeding artery was delivered through the catheter. When the proper position was reached, the guiding catheter was pulled back a few centimeters to give the device space at the occlusion site to expand; the plug was then released by turning the release cable counterclockwise.

Three minutes after release, an angiogram confirmed complete occlusion of the fistula. There were no complications during the procedure.

The patient was discharged 10 days after closure of the foramen ovale. The only remaining neurological sequela was slight facial asymmetry.

Discussion

Pulmonary arteriovenous malformations are infrequent and usually congenital. More than 70% occur in patients with Rendu–Osler–Weber syndrome or hereditary hemorrhagic telangiectasia,² which is a systemic autosomal dominant disorder that produces mucocutaneous telangiectases and pulmonary, cerebral, and hepatic arteriovenous malformations. The incidence of such malformations in the lung in these patients ranges from 15% to 33%.³

Clinical signs of pulmonary arteriovenous malformations depend on the number and size of the fistulas: when pulmonary involvement is extensive, dyspnea is the most common clinical symptom owing to right-to-left shunt, which produces hypoxemia—a condition that is usually well tolerated. However, more than half of patients can

develop serious complications, such as paradoxical embolization and rupture. To prevent such complications treatment is imperative. The central nervous system is the most common target of paradoxical embolisms, leading to cerebrovascular accidents or brain abscess owing to the unobstructed pass of thrombi and bacteria through the lesion.⁴ Swanson et al⁵ observed that 5 of 15 patients who were diagnosed with pulmonary arteriovenous malformations and went untreated had neurological complications during the subsequent 10 years.

Our patient had a single fistula and therefore experienced no shortness of breath or abnormal blood gas levels. The clinical presentation was cerebral embolism, attributed on first evaluation to a patent foramen ovale and aneurysmal septum. However, when the patient was diagnosed with Rendu–Osler–Weber syndrome and the fistula with thrombus within was detected, we considered that the cerebrovascular accident was more likely to be associated with a pulmonary arteriovenous malformation than with the patent foramen ovale.

Since the early 1990s embolotherapy has been gaining acceptance for the management of pulmonary arteriovenous malformations owing to high success rates and lack of complications. Embolotherapy can involve the use of coils, silicone balloons, polyvinyl alcohol particles, Histoacryl, and Gianturco–Grifka vascular occlusion devices. Although these supplies are effective, they have certain limitations. For example, many coils are sometimes required to occlude a single vessel, the percentage of reopening can be high (ranging from 5% to 57%),⁶ and migration and paradoxical embolization of coils may take place at unintended sites. Detachable balloons can deflate,⁷ and Gianturco–Grifka occlusion devices require large-caliber delivery catheters.⁸

The Amplatzer vascular plug (Figure 2) is a new cylindrical self-expanding device made of Nitinol wire

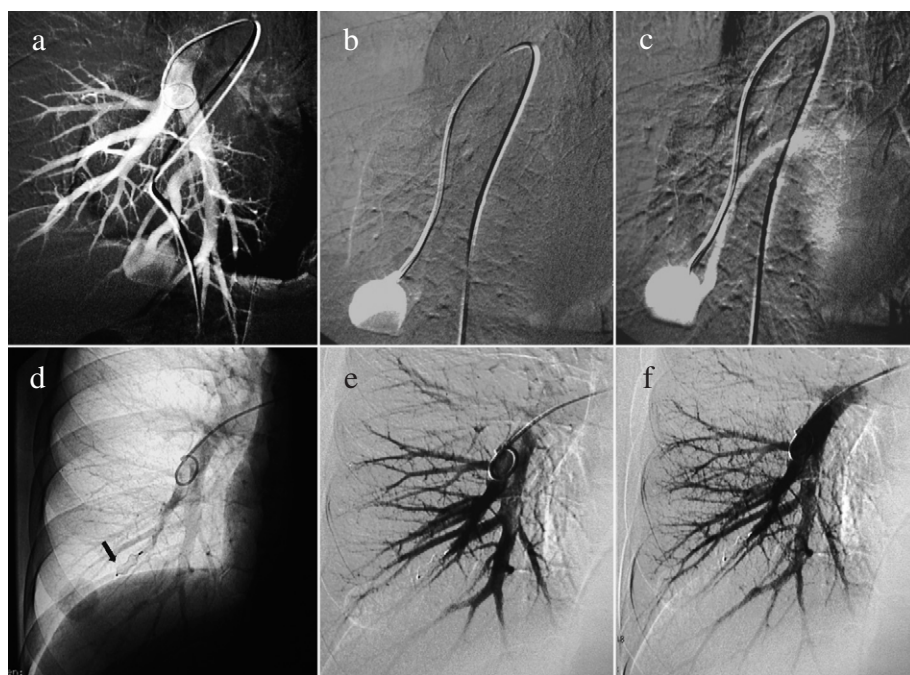


Figure 1. Selective angiography of the right pulmonary artery showing an arteriovenous malformation in the right lower lobe (a). After selective catheterization of the feeding artery and injection of a contrast medium through a 6F guiding catheter it was possible to visualize an aneurysmal dilatation with thrombus within (b) and a drainage vein between a pulmonary vein and the left atrium (c). Angiography without digital subtraction (d) shows the released Amplatzer vascular plug (arrow). Three minutes after release of the plug, complete occlusion of the fistula could be observed (e and f).

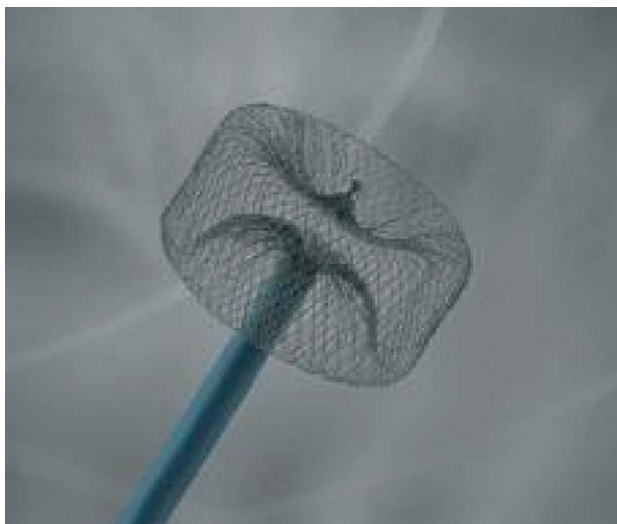


Figure 2. Photograph of the Amplatzer vascular plug.

mesh and indicated for arteriovenous embolizations in the peripheral vasculature. This plug enables more precise occlusion and positioning can be verified before release; if the position is unsatisfactory, the plug can be repositioned or withdrawn. The advantages of this device are that it reduces the risk of migration, releases easily, and enables complete occlusion with a single plug, thus cutting down on time and, in the case of large fistulas, reducing the cost as well. The only drawback is that the plug requires distal positioning through guiding catheters ranging from 5F to 8F, depending on the diameter of the vessel to be occluded.

The recommended plug size is 30% to 50% larger than the diameter of the vessel to be occluded. For our patient

this meant using a 10-mm plug since the diameter of the feeding artery was approximately 7 mm.

In conclusion, we consider the Amplatzer vascular plug to be an efficacious, easy-to-manage device for treating pulmonary arteriovenous malformation and that it can be applied in cases of abnormal shunts at other locations.⁹ Use of this plug reduces procedure time and risk.

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