

Thrombosed pulmonary arteriovenous malformation in an elder hemodialysis patient: case report

Malformación arteriovenosa pulmonar trombosada en anciano con hemodiálisis: reporte de caso

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ABSTRACT. Acquired pulmonary arteriovenous malformations (PAVM) in adults are a rare finding. Its etiology is related to pneumopathies, infections or parasitosis. Its presence is associated with complications such as hemorrhage, endocarditis, embolism. We present the case of an PAVM observed in an elderly person that was successfully closed with a percutaneous device.

Keywords: Arteriovenous fistula, aged, renal replacement therapy.

RESUMEN. Las malformaciones arteriovenosas pulmonares (MAVP) adquiridas en el adulto son un hallazgo poco frecuente. Su etiología se relaciona a neumopatías, infecciones o parasitosis. Su presencia se asocia a complicaciones como hemorragia, endocarditis, embolia. Presentamos el caso de una MAVP observada en un anciano que fue tratada exitosamente con dispositivo percutáneo.

Palabras clave: Fístula arteriovenosa, anciano, terapia de reemplazo renal.

INTRODUCTION

PAVM are abnormal vascular communications between arteries and veins. They are usually diagnosed in youth subjects with severe chronic hypoxemia or in patients with hereditary hemorrhagic telangiectasis (Osler Weber Rendu syndrome). In older patients, its etiology is related to previous cavopulmonary fistulas in patients with complex congenital heart disease, chronic liver disease (hepatopulmonary syndrome), or pulmonary infections due to tuberculosis or actinomycosis. In most cases they are asymptomatic and are diagnosed as an incidental finding of imaging studies. When they are large or multiple, are associated with complications such as massive hemoptysis, cerebral abscess, stroke, spontaneous hemothorax, endocarditis and embolism.¹

CASE PRESENTATION

This is a 75-year-old woman with a history of type 2 diabetes mellitus, high blood pressure, dyslipidemia, hypothyroidism, obesity and chronic kidney disease on hemodialysis therapy with arteriovenous fistula in the upper left limb. The patient has presented chest pain during hemodialysis, associated with arterial hypotension and diaphoresis, in addition to occasional hemoptysis. Physical examination was unremarkable, except for low finger saturaometry (80%). No platypnea or orthodeoxia was demonstrated. Rest electrocardiogram was normal. Coronary computed coronary angiography was performed, and showed extensive coronary calcification but with non-significant obstructions, however on the right pulmonary base there is a round structure with central

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hypodensity, a feeding artery of 7 mm diameter and an efferent vein (Figure 1). Immunological studies of parasites or tuberculosis were negative. Invasive selective angiography was performed confirming the tomographic diagnosis. Occluder device (vascular plug) was placed successfully without evidence of residual

shunt. The patient's symptoms disappeared and is alive without complications after two years.

DISCUSSION

We present a rare case since the image could be confused with tumor or hydatid cyst,

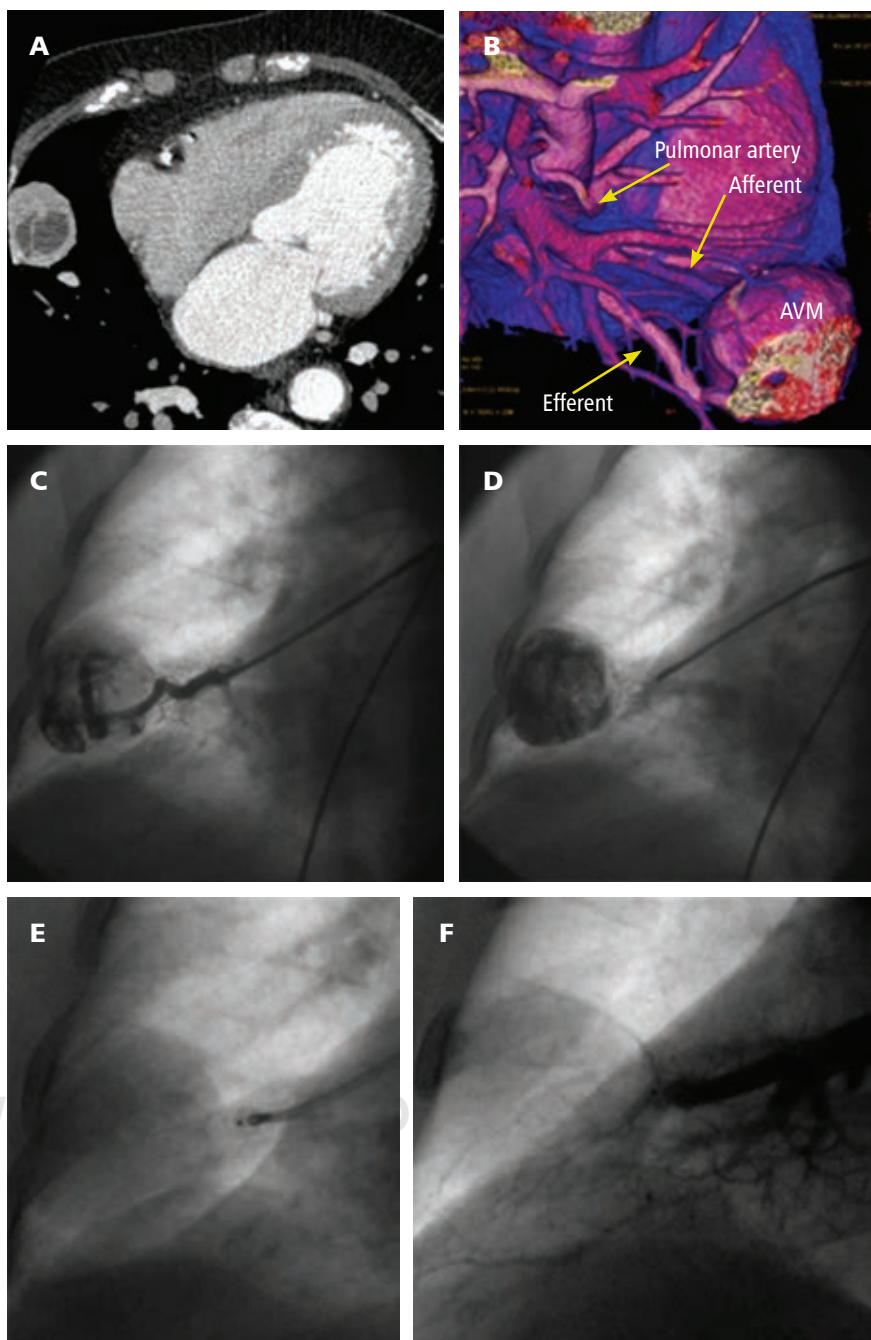


Figure 1:

A) Contrast tomography showing a rounded image with hypodense center in the right pulmonary base. **B)**

Volumetric reconstruction of the CT scan demonstrating the afferent vessel that feeds the malformation, as well as the efferent vessel. **C)** Selective pulmonary angiography demonstrating the feeding artery of the pulmonary arteriovenous malformation. **D)** Selective pulmonary angiography demonstrating the fullfill of the pulmonary arteriovenous malformation.

E) Embolization with vascular plug of the feeding artery of the pulmonary arteriovenous malformation. **F)** Selective pulmonary angiography, contrast shot at the end of the procedure, without evidence of residual malformation filling.

however the presence of the «feeding» vessels were key to the diagnosis. The presence of central hypodensity is very suggestive of thrombus which suggests previous contained ruptures. These ruptures could be related to the turbulent and accelerated flow produced in the peripheral fistula during hemodialysis. Most PAVMs are located in the lower pulmonary lobes, possibly due to the increased flow and pressure that occurs in this area. Nearly 90% have a single feeder vessel, called «simple».² Most complications are related to two mechanisms: hypoxemia or shunt. The first may cause cardiovascular complications, such as ischemic heart disease in patients with coronary artery disease. In our patient this could happen during ultrafiltration, the induced hypovolemia coupled with hypoxemia could lead to coronary hypoperfusion and ischemic symptoms. Etiology of our patient's fistula, it is still a mystery. There are reports of formation of this type of anomalies in patients with univentricular heart who underwent a cavopulmonary bypass (Glenn's procedure). In these subjects it is proposed that the redirection of the flow leads to a decrease in the clearance of inflammatory cytokines in the liver; at the same time, pulmonary hyperflow could lead to overexpression of growth factors that generate angiogenesis and the consequent malformation.³ Other authors have reported the development of pulmonary hypertension in patients with peripheral arteriovenous fistula for hemodialysis, possibly due to increased pulmonary flow.⁴ Our hypothesis considers in

our patient a similar phenomenon, secondary to the pulmonary hyperflow produced by the peripheral fistula. In cases where the feeding artery exceeds 2 or 3 mm, embolization treatment is recommended, which can be performed with coils, vascular plugs or balloons, with good outcomes.⁵ Some reports mention better outcomes with vascular plug.⁶

In conclusion, in patients with hemodialysis the presence of pulmonary arteriovenous malformations is very rare and can be complicated with rupture, so percutaneous embolization is a good option to avoid fatal outcomes.

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