Embolization of pulmonary AVMs of feeding arteries less than 3 mm: reports of two cases and an 8-year follow-up without embolization

Poul Erik Andersen¹ and Anette D Kjeldsen²

¹Department of Radiology (PEA); ²Department of Oto-Rhino-Laryngology (ADK), Odense University Hospital, Odense, Denmark Correspondence to: Poul Erik Andersen. Email: anders1@dadInet.dk

Abstract

Pulmonary arteriovenous malformations (PAVMs) with feeding arteries of 3 mm or more have been shown to be associated with paradoxical embolization and serious neurologic complications. Therefore it is generally recommended to occlude feeding arteries of this size, while smaller feeding arteries often are left untreated. However, neurologic complications have also been described in patients with small PAVMs, and it has not been possible to stratify risk by size of feeding artery and thus there is no evidence that 3 mm is the critical size of the feeding artery potentially giving complications. Further, it is well-known that with time PAVMs and their feeding arteries may enlarge. Also, embolization of small feeding arteries will minimize the risk of paradoxical emboli and reduce the need for CT follow-up controls in these patients. Two cases demonstrate the possibility to embolize small feeding arteries.

Keywords: Thorax, angiography, interventional, embolization, AVM

Submitted January 5, 2012; accepted for publication March 12, 2012

Pulmonary arteriovenous malformations (PAVMs) with feeding arteries greater than 3 mm in diameter have been shown to be associated with paradoxical embolization and serious neurologic complications (1-3) with decreased oxygenation of the blood and risk of rupture of the PAVMs. Serious neurologic complications, including TIA, stroke, and brain abscess, occur in 30-40% of patients with PAVMs (4-8). Therefore, it is generally recommended to occlude feeding arteries of \geq 3 mm to prevent paradoxical embolization, even in asymptomatic patients (1, 4, 6, 9–13), and as a consequence smaller feeding arteries are often left untreated. However, neurologic complications have also been described in patients with small PAVMs that were left untreated (9, 14, 15) and there is no evidence that 3 mm is the critical size of the feeding artery potentially giving complications, and it has not been possible to stratify risk by size of feeding artery (16). It has been debated for many years what size of feeding arteries should be treated (15), but the 3-mm guideline is still referred to in many articles describing PAVM treatment.

Further, it is well-known that with time PAVMs and their feeding arteries may enlarge (9, 10, 17), especially in growing children and after pregnancy.

Case 1

The first case report concerns a 40-year-old woman with hereditary hemorrhagic telangiectasia (HHT). Her father died from a probable cerebral attack. At screening of families with HHT the patient was discovered with PAVMs eight years previously. PaO_2 was then 9.7 kPa (72.8 mmHg), the contrast echocardiography showed a shunt of grade 4 (maximal), and the shunt was estimated to 24%. One big simple PAVM and two small PAVMs were visualized by pulmonary angiography (Fig. 1a and b). The big PAVM in right middle lobe was embolized with one detachable silicone balloon (Target Therapeutics, Fremont, CA, USA) (Fig. 2) while the two small ones were left untreated because of small feeders <3 mm.

At follow-up 3 months later the PaO_2 had increased to 13.0 kPa (97.5 mmHg), the oxygenation was 98%, the contrast echocardiography shunt had decreased to 3, and the shunt was estimated to 7%. The patient felt better performance. In the following years there were no occurring problems, especially no neurologic events, but the patient experienced periodic headaches.



Fig. 1 Case 1. A 31-year-old woman. Primary examination. (a) Right pulmonary angiography shows a big pulmonary arteriovenous malformation (PAVM) in the middle lobe and a small one in the lower lobe with a feeding artery of 1.6 mm (arrows). (b) Left pulmonary angiography shows a very small PAVM in the upper lobe with a feeding artery of 1.5 mm (arrow)

At follow-up 8 years later the CT scan showed that the big PAVM was occluded with a residual scar at the place of the former PAVM. At pulmonary angiography the balloon was invisible, but the embolized PAVM was without flow. The feeders of the other two PAVMs had since the initial pulmonary angiogram 8 years earlier grown from 1.6 mm to 2.7 mm (right lower lobe) (Fig. 3) and from 1.5 mm to 2.0 mm (left upper lobe) (Fig. 4a). The right sided PAVM was embolized using two vortexes – 35, 3–5 mm, 0.035 coils (Boston Scientific/Target Vascular, La Garenne-Colombes, Cedex, France) (Fig. 3b and c), and the left with one 3/30 mm coil (MR eye, non-ferro-magnetic standard embolization coil 0.035, Cook, Bjaeverskov, Denmark) (Fig. 4b).

Case 2

A 37-year-old woman, who was a member of a HHT family, had by screening been diagnosed with a PAVM in left lower lobe. At pulmonary angiography it appeared to be a complex PAVM (Fig. 5) with two feeding arteries: one was 4.4 mm in size (Fig. 6) and the other was 2 mm (Fig. 7). It was decided



Fig. 2 Case 1 after embolization of the big PAVM in the middle lobe with use of a detachable balloon (arrow)



Fig. 3 Case 1. Pulmonary angiography at 8-year follow-up. (a) The feeding artery to the PAVM in the right lower lobe had grown to 2.7 mm. (electronic measurement); (b, c) after embolization with two 3-5 mm vortex coils (Boston Scientific)



Fig. 4 Case 1 at 8-year follow-up. (a) The feeding artery to the PAVM in left upper lobe had grown to 2.0 mm (electronic measurement); (b) after embolization using one 3/30 mm coil (Cook, Denmark)

to embolize both, the larger one with an Amplatzer vascular plug 4 (AGA Medical Corporation/St Jude Medical Inc., St Paul, MN, USA) and the small one with deployment of one 3/50 mm MR eye coil (COOK, Bjaeverskov Denmark). The patient had a little pleurisy for two days after the



Fig. 5 Case 2. A 37-year-old woman with complex PAVM with two feeding arteries in left lower lobe

embolizaton but was well at 3-month follow-up and with a negative contrast echocardiography. Embolization of the bigger feeding artery only would have resulted in an insufficient embolization of this complex PAVM.

Discussion

The aim of treating PAVMs is primarily to prevent paradoxical embolization especially to the brain, but also to prevent pleuro/pulmonary hemorrhage and to improve hypoxemia. The risk of paradoxical embolization is most probably greatest in high-flow PAVMs with big feeding arteries \geq 3mm, but smaller feeding arteries also pose a risk (9, 14, 17). It makes no sense that septic emboli should not be able to enter the systemic circulation through smaller feeding vessels. It is not based on evidence that embolization of feeding arteries <3 mm will prevent paradoxical embolization but common sense and published data tell that this is a possibility. Modern technology with the introduction of microcatheter systems and (detachable) microcoils also makes it more easy and safe to embolize smaller PAVMs, also below 2 mm diameter (Fig. 8). The feeding arteries may grow, and occlusion will minimize the risk of paradoxical



Fig. 6 Case 2. The biggest feeding artery measured 4.4 mm in diameter (electronic measurement) and was embolized with an 8 mm Amplatzer vascular plug 4 through a 5F vertebral catheter



Fig. 7 Case 2. The small feeding artery measured 2 mm in diameter (electronic measurement) and was embolized with one MR eye 3/50 mm coil (Cook, Denmark)



Fig. 8 A 25-year-old man. 1.8 mm diameter feeding artery (electronic measurement) in right lower lobe embolized with one 3/30 mm and one 3/50 mm MR eye coils (Cook, Denmark)

emboli and reduce the need for follow-up controls to see if and when the PAVM reaches a size of 3 mm or more. Thus, the risk of lifelong recurrent exposure to ionizing radiation by CT is also reduced and embolization will also reduce the worry of the doctor and the patient. Therefore, embolization is suggested also of small feeders when technically feasible.

REFERENCES

- 1 White RI Jr, Pollak JS, Wirth JA. Pulmonary aretriovenous malformations: diagnosis and transcatheter embolotherapy. J Vasc Interv Radiol 1996;7:787–804
- 2 White RI Jr, Pollak J. Pulmonary arteriovenous malformations: options for management. *Ann Thorac Surg* 1994;57:516-24
- 3 Wirth JA, Pollak JS, White RI Jr. Pulmonary arteriovenous malformations. In: Bone RC, Dantzker DR, George RB, eds. *Current Pulmonology and Critical Care Medicine*. Volume 17. Philadelphia, PA: Mosby, 1996
- 4 White RI Jr, Lynch-Nyhan A, Terry P, *et al*. Pulmonary arteriovenous malformations: techniques and long-term outcome of embolotherapy. *Radiology* 1988;**169**:663–9
- 5 Andersen PE, Kjeldsen AD. Clinical and radiological long term follow-up after embolization of pulmonary arteriovenous malformations. *Cardiovasc Interv Radiol* 2006;**29**:70–4

- 6 White RI Jr, Pollak JS. Pulmonary arteriovenous malformations: diagnosis with three-dimensional helial CT – a breakthrough without contrast media. *Radiology* 1994;**191**:613–4
- 7 Hewes RC, Auster M, White RI Jr. Cerebral embolism: first manifestation of pulmonary arteriovenous malformation in patients with hereditary hemorrhagic telangectasia. *Cardiovasc Interv Radiol* 1985;8:151-5
- 8 Kjeldsen AD, Oxhøj H, Andersen PE, *et al.* Prevalence of pulmonary arteriovenous malformations (PAVMs) and occurrence of neurological symptoms in patients with hereditary haemorrhagic telangiectasia (HHT). *J Int Med* 2000;**248**:255–62
- 9 Mager JJ, Overtoom TTC, Blauw H, *et al*. Embolotherapy of pulmonary arteriovenous malformations; long-term results in 112 patients. *J Vasc Interv Radiol* 2004;**15**:451–6
- 10 White RI Jr, Mitchell SE, Barth KH, *et al.* Angioarchitecture of pulmonary arteriovenous malformations: an important consideration before embolotherapy. *Am J Roentgenol* 1983;**140**:681–6
- 11 Lacombe P, Lagrange C, Beauchet A, *et al.* Diffuse pulmonary arteriovenous malformations in hereditary hemorrhagic telangiectasia: long-term results of embolization according to the extent of lung involvement. *Chest* 2009;**135**:1031–7
- 12 Pollak JS, Saluja S, Thabet A, *et al.* Clinical and anatomic outcomes after embolotherapy of pulmonary arteriovenous malformations. *J Vasc Interv Radiol* 2006;**17**:35–45
- 13 Gupta S, Faughnan ME, Bayomi AM. Embolization for pulmonary arteriovenous malformations in hereditary hemorrhagic telangiectasia: a decision analysis. *Chest* 2009;**136**:849–58

- 14 Todo K, Moriwaki H, Higashi M, et al. A small pulmonary arteriovenous malformation as a cause of recurrent brain embolism. Am J Neuroradiol 2004:**25**;428–30
- 15 Trerotola S, Bernhardt B, Pyeritz R. Outpatient single-session pulmonary arteriovenous malformation embolization. J Vasc Interv Radiol 2009;20:1287-91
- 16 Moussouttas M, Fayad P, Rosenblatt M, et al. Pulmonary arteriovenous malformations: cerebral ischemia and neurologic manifestations. *Neurology* 2000;55: 959–64
- 17 Trerotola SO, Pyeritz RE. PAVM Embolization: An Update. Am J Roentgenol 2010;195:837-45

 \odot 2012 The Foundation Acta Radiologica

This is an open-access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons. org/licenses/by-nc/2.0/), which permits non-commercial use, distribution and reproduction in any medium, provided the original work is properly cited.