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## Revision of facts on pulmonary arteriovenous malformations – experience with embolization techniques

### Przegląd aktualnej wiedzy dotyczącej malformacji płucnych – doświadczenia własne w embolizacji przetok tętniczo-żylnych

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#### Keywords

arteriovenous malformations, pulmonary arteriovenous fistulas, therapeutic embolization, AMPLATZER occluder, coil

#### Słowa kluczowe

malformacje tętniczo-żylnych, płucne przetoki tętniczo-żylnych, embolizacja, okluder AMPLATZER, spirale

#### Conflict of interest Konflikt interesów

None

Brak konfliktu interesów

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#### Summary

**Introduction.** Pulmonary arteriovenous malformations are pathological direct connections between arterial and venous circulation, bypassing capillary networks. The consequences of the occurring right-to-left shunt comprise systemic hypoxemia and loss of pulmonary vascular filtration properties, which pose a risk of serious life-threatening neurologic complications because of paradoxical embolism. 60-90% of PAVMs appear to be a manifestation of the underlying Hemorrhagic Hereditary Telangiectasia. Treatment is introduced as protective measures against life-threatening hemorrhagic or neurologic symptoms, and presently transcatheter embolization proved to be a method of choice.

**Aim.** The aim of this study was to discuss the efficacy and technical details of the selected current approaches to PAVM treatment with respect to the angioarchitecture of the fistulae, based on the literature review and clinical experience.

**Material and methods.** We performed a retrospective investigation of 5 patients with radiologically confirmed presence of pulmonary arteriovenous malformations. In each case, transcatheter embolization of the feeding artery with use of AMPLATZER vascular plug and/or coils and micro coils was executed. Technical success and presence of procedural and periprocedural complications were assessed with respect to technique of embolization and angioarchitecture of lesions. Pre- and post-procedural selective pulmonary angiographies were implemented to visualize lesions and assess technical success, respectively.

**Results.** Technical success was achieved in all cases. No procedural or periprocedural complications were observed.

**Conclusions.** Embolization of the feeding artery is a highly-effective treatment option in case of pulmonary vascular malformations. AMPLATZER vascular occluder is a convenient device allowing for almost immediate occlusion of the supplying vessel to the PAVM. However, limited selection of device characteristics (i.e. diameter) favors coil embolization in case of feeding artery size less than 3 mm.

#### Streszczenie

**Wstęp.** Przetoki tętniczo-żylnych krążenia płucnego stanowią bezpośrednie połączenia pomiędzy naczyniami tętniczymi i żylnymi, z pominięciem naczyń mikrokrążenia. Powstający w konsekwencji przeciek prawo-lewy może prowadzić do hipoksemii systemowej oraz utraty właściwości filtracyjnych płucnego łożyska naczyniowego, co z kolei stwarza zagrożenie pojawienia się zagrażających życiu komplikacji neurologicznych, wywołanych obecnością zatorów paradoksalnych. Spośród płucnych malformacji tętniczo-żylnych około 60-90% pojawia się w przebiegu dziedzicznej teleangiektazji krwotocznej. Celem leczenia przetok jest zapobieganie potencjalnie śmiertelnym powikłaniom natury krwotocznej lub neurologicznej, a metody embolizacji naczyniowej stanowią metodę z wyboru.

**Cel pracy.** Celem niniejszego opracowania było przedstawienie aspektów technicznych i skuteczności wybranych, aktualnych metod leczenia przetok tętniczo-żylnych krążenia płucnego w odniesieniu do ich angioarchitektury, w oparciu o przegląd dostępnej literatury i doświadczenia własne.

**Materiał i metody.** Przeprowadzono retrospektywną ocenę 5 przypadków klinicznych pacjentów z radiologicznie potwierdzonym rozpoznaniem malformacji tętniczo-żylnych w łożysku płucnym. W każdym z powyższych przypadków zastosowano leczenie w formie embolizacji naczynia doprowadzającego z wykorzystaniem zatyczek AMPLATZER i/lub spiral oraz mikrospiral. Ocenę sukcesu technicznego przeprowadzonych interwencji oraz

ewentualną obecność powikłań zabiegowych i okołozabiegowych oceniono w odniesieniu do zastosowanej techniki zabiegu oraz struktury naczyniowej malformacji. Angiografia przed- i pozabiegowa zostały wykonane u wszystkich pacjentów, aby odpowiednio: uwiarygodnić patologiczne zmiany oraz ocenić stopień powodzenia procedury.

**Wyniki.** We wszystkich przypadkach osiągnięto pełny sukces techniczny. Nie zaobserwowano wystąpienia powikłań podczas przeprowadzanych zabiegów ani powikłań okołozabiegowych.

**Wnioski.** Embolizacja tętnicy doprowadzającej jest wysoce efektywną metodą zaopatrywania płucnych przetek tętniczo-żylnych, a okludery AMPLATZER stanowią wygodne rozwiązanie, gwarantujące niemal natychmiastowe zamknięcia naczynia zaopatrującego malformację. Jednakże, ze względu na ograniczony wybór specyfikacji (zwłaszcza średnicy) ww. urządzeń, embolizacja z użyciem spiral jest podstawową metodą zaopatrywania tętnic doprowadzających o średnicy poniżej 3 mm.

## INTRODUCTION

Pulmonary arteriovenous malformations (PAVMs) are pathological direct connections between arterial and venous circulation, bypassing capillary networks that occur with estimated prevalence of 1 in 100,000 (1). The consequences of the occurring right-to-left shunt comprise not only impaired blood oxygenation mechanisms leading to systemic hypoxemia (more pronounced if diameter of the feeding artery exceeds 5 mm), but even more importantly, loss of pulmonary vascular filtration properties which pose a risk of serious life-threatening neurologic complications because of paradoxical embolism (2, 3).

### Occurrence

A considerable majority of PAVMs (i.e. 60-95%) appear to be a manifestation of the underlying Hereditary Hemorrhagic Telangiectasia (HHT), otherwise known as Osler-Weber-Rendu syndrome (2-5). Conversely 15 to 50% of patients diagnosed with HHT develop PAVMs over lifetime (6). In this autosomal dominant, often underdiagnosed condition, with estimated prevalence of 1 in 5000 individuals, vascular malformations are often multiple and occur not only in the setting of pulmonary circulation, but also within brain, gastrointestinal tract and liver. The diagnosis of HHT is typically based upon clinical appearance as approximately 90% of patient will meet Curaçao Criteria (tab. 1) by the age of 40 (3, 5). However, these require presence of epistaxis and/or teleangiectases – symptoms that rarely occur in children and young adults. Genetic testing is therefore the most suitable diagnostic tool in those groups of patients. Despite some particular genes involvement in development of certain HHT types (tab. 2), there are no common mutations that can be easily distinguished. A unique, private mutation is rather present among members of each of the affected families. The remaining 10% of PAVMs are acquired fistulae, mostly associated with development of hepatopulmonary syndrome in cirrhotic patients. However, their appearance may also be attributed to mitral valve stenosis, trauma, actinomycosis, tuberculosis and schistosomiasis. No differences were observed in terms of radiologic and histologic appearance between idiopathic and hereditary PAVMs (3, 5, 7).

**Tab. 1.** Curaçao Diagnostic Criteria for Hereditary Hemorrhagic Telangiectasia (HHT)

<b>Criteria</b>	epistaxis	spontaneous, recurrent nose bleeds
	teleangiectases	multiple at characteristic sites (lips, oral cavity, nose, fingers)
	visceral lesions	gastrointestinal telangiectasia, pulmonary arteriovenous malformation (AVM), hepatic/cerebral/spinal AVM
	family history	first degree relative with HHT according to these criteria
<b>Diagnosis</b>	definite	3 or more criteria are fulfilled
	possible/suspected	2 criteria are present
	unlikely	1 or none criteria are present

**Tab. 2.** Genes involved in Hereditary Hemorrhagic Telangiectasia

HHT 1	<i>ENG</i>	endoglin gene
HHT 2	<i>ACVRL1</i>	activin A receptor like type 1 gene
HHT with juvenile polyposis	<i>SMAD4</i>	SMAD family member 4 gene

### Angioarchitecture and classification

80-85% of PAVMs present with simple angioarchitecture. It means that there is only one feeding artery (FA) to the arteriovenous sac, and a single draining vein. In 15-20% cases multiple feeding and/or draining vessels are present – these PAVMs are referred to as complex malformations. In the above mentioned, the simple sac may be septet or multichanneled, or replaced with plexiform tangle of tortuous, dilated vessels (2, 5, 6, 8).

PAVMs may be also classified as single and multiple focal or diffuse (up to 5% of patients). Whereas the former two definitions are quite clear and intuitive, the latter had been subject to discussion. Diffuse PAVMs were first characterized by Faughnan et al. as a condition in which all segmental branches of one or more lobes were diffusely involved by small malformations (3). However, this interpretation was questioned a few years later by Pierucci et al. who described the diffuse pattern of involvement in a more restrictive manner. The new definition simply required reporting on diffuse involvement of particular segments. This was due to the fact, that except for diffusely engaged segmental arteries, many patients simultaneously pre-

sented with normal segmental arteries in the same lobe. Such modification allows to distinguish between multiple focal and diffuse lesions, which has its implications in treatment approach and clinical outcomes (9).

### Clinical presentation and complications

PAVMs is a pathology with unpredictable natural history. Although the majority of lesions – simple in nature and of small size – remain asymptomatic for a long time, the associated mortality risk reaches 15%. A considerable risk of morbidity associated with neurologic complications persists as well. The peak of clinical manifestations of PAVMs occurs at four to six decade in as many as 70% of patients, who often require treatment at this point (4, 5, 10). Symptoms of systemic hypoxemia, including dyspnea, cyanosis, fatigue, exercise intolerance and eventual heart failure predominate, but do not pose the most danger to health and life of the affected individual (5). The greatest risk is associated with hemorrhagic consequences of PAVM rupture (i.e. hemoptysis, hemothorax) and/or neurologic manifestations of paradoxical emboli (i.e. transient ischemic attack (TIA), ischemic stroke or brain and systemic abscesses) (2, 5). It had been proven as well that pregnancy is a significant risk factor for development of complications (11, 12). As stated above, patients with diffuse pattern of PAVM involvement are more prone to occurrence of serious, life-threatening complications, especially when lesions are located bilaterally (9).

### Diagnosis and treatment

According to the latest international guidelines on diagnosis and management of HHT, the reference diagnostic tests for pulmonary malformations comprise unenhanced thin-cut computed tomography (CT) and angiography. However, it has been postulated that transthoracic contrast echocardiography (TTCE) with agitated saline may be a useful screening tool in suspected patients. The examination is considered positive in case of detection of air bubbles within left atrium due to existence of intrapulmonary shunt. All positive outcomes must be confirmed with reference CT (3, 7, 11).

The aforementioned high incidence of PAVMs among patients with HHT implies careful screening for the disease in individuals with PAVMs who have not been previously diagnosed with Osler-Weber-Rendu syndrome (3, 7, 11).

Treatment is introduced as protective measures against life-threatening hemorrhagic or neurologic symptoms (5, 13, 14). This implies that even asymptomatic lesions are managed in adult patients, contrary to children and individuals with diffused pattern of the disease – in these two cases treatment is delayed until symptoms occur. Because PAVMs are classified as high-flow malformations, the greater diameter of FA results in greater risk of dislodgement of thrombotic material. Therefore, it is generally accepted that all PAVMs with diameter of FA exceeding 3 mm are treated (2, 3, 9, 14). However, some of the authors argue that smaller

diameter of the supplying vessel fails to prevent from migration of bacteria and, as a result – cranial and systemic abscesses (3, 14).

Historically, all PAVMs were treated by surgical means, however, presently transcatheter embolization proved to be highly-effective, organ-sparing first-line treatment and surgery is reserved only to PAVMs amenable to embolization. In many sites, the procedure is carried out under local anesthesia, on outpatient basis (4, 10, 11). Several minimally invasive approaches to embolization have been proposed depending mostly on PAVM angioarchitecture, with feeding artery embolization serving as the gold standard. The embolic materials used nowadays include pushable and interlocking detachable coils (IDC), as well as AMPLATZER Vascular plugs. The mechanism of action and hemodynamic effects of the latter resemble mechanism of detachable balloons which were withdrawn from the market. The results of procedures carried out with AMPLATZER devices are very promising, with the reported rates of immediate technical success of 100%, low reperfusion rates of 0-7% and rare occurrence of serious complications (6, 10, 15).

General rule is that all the devices used must be chosen with certain degree of oversizing (about 20%) with reference to FA diameter measured on CT scans and deployed within 1 cm or beyond from origin of any major arterial branch supplying normal lung tissue that arise from the FA. To prevent recanalization, dense, cross-sectional packing of the embolic devices is highly advised (10, 15, 16). In case of coil deployment, it is achieved by first creating a wire mesh with stiffer coils of greater radial force to prevent migration of smaller IDCs. Even though successive generations of AMPLATZER plugs are designed to fit FA of smaller diameter, in cases of tortuous, small-calibrated FA – coil embolization is preferred (7, 16).

Recent publications indicate a novel device – ArtVentive Endoluminal Occlusion System (EOS) – to possibly become a strong competitor for coils and AMPLATZER plugs. Initial experience proves ArtVentive EOS to reduce the time of radiation exposure, present with easier deployment technique as compared to AMPLATZER device and result in immediate occlusion of the vessel (6).

Another approach to PAVM management is the vascular sac embolization. This method proved to be effective in cases of high-flow malformations with large-diameter out-flow vessels or short feeders. The comparative study of efficacy of FA embolization (FAE) and venous sac embolization by Hayashi et al. showed reperfusion to occur only after FAE (17). The potential risks associated with this particular method, i.e. rupture of venous sac during coil deployment or dislodgement of thrombotic material formed during procedure may be easily overcome with use of soft, compliant IDCs and flow control with balloon, respectively (2, 10, 17). The main disadvantage if the procedure seems to be a large number of coils used, prolonging the procedure time and exposition to radiation, as well as its

unsuitability in cases of: diffuse or complex PAVMs, presence of clot within sac or sacs exceeding 3 cm diameter (10, 17).

**Follow-up**

Long-term, careful radiologic follow-up is necessary due to high recurrence rates, either to technical failure, reperfusion or growth of previously negligible PAVMs (10). Although angiography seems to be the most sensitive and specific means of radiologic control, it is reserved only for patients who return for successive stages of treatment, due to its invasive nature and radiation exposure (18). Therefore, the unenhanced thin-slice CT of the chest remains the most commonly applied method. However, certain authors report the method's low sensitivity and specificity (19). Control CT scans should be performed at 6-12 months after embolization and every 3-5 years thereafter (3, 5, 10). In case of diffuse PAVMs, which pose a great risk to affected patients, a yearly follow-up is recommended (9).

Recent publication by Kawai et al. presents a new method of PAVM follow-up, which constitutes time-resolved magnetic resonance angiography. Not only it proved to be effective, but also a promising tool which over time may become a new standard diagnostic measure requiring further investigation (18, 20).

**AIM**

The aim of this study was to discuss the efficacy and technical details of the selected current approaches to PAVM treatment with respect to the angioarchitecture of the fistulae, based on the literature review and clinical experience.

**MATERIAL AND METHODS**

We performed a retrospective investigation of 5 patients with radiologically confirmed presence of pulmonary arteriovenous malformations based on computed tomography, who were treated in our Department by feeding artery embolization between July 2010 and July 2016 (4 of the patients were treated between 2 Feb 2016 and 6 July 2016 – representing the most recent experiences).

AMPLATZER vascular plugs (AVP) II or 4 were employed in two patients, coil and micro coil embolization was performed in two patients, whereas combination of AVP4 and coil embolization was employed in the remaining one patient. Technical success (defined as occlusion of the feeding artery with no evidence of flow through the AVM) and presence of procedural and periprocedural complications were assessed with respect to technique of embolization and angioarchitecture of lesions.

All the procedures were performed under local anesthesia, with the right femoral vein approach being preferred for access with Seldinger method. A pre-procedural selective pulmonary arteriography was performed via 5 Fr pig-tail catheter. After visualization of PAVMs, selective catheterization of the feeding arteries was performed in each case, with subsequent deployment of occluding device (coils and/or AVP) (figures 1 and 2a-c depict subsequent stages of embolization with AVPII).

In all cases a control angiography was performed to confirm vessel occlusion.

Patient and procedure details are found in table 3.

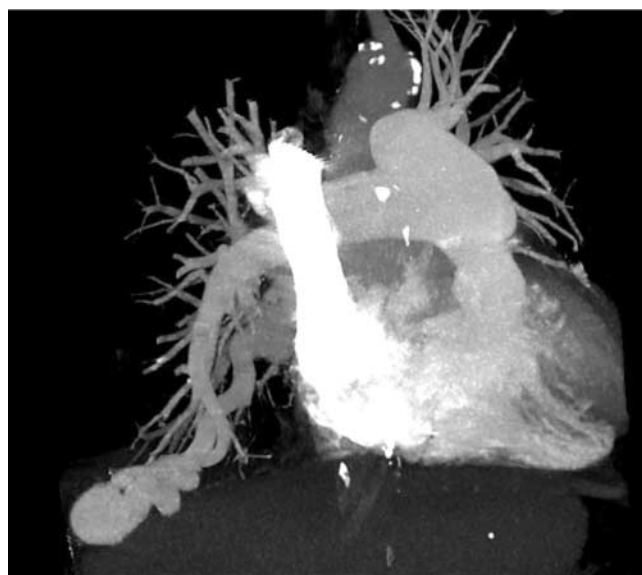


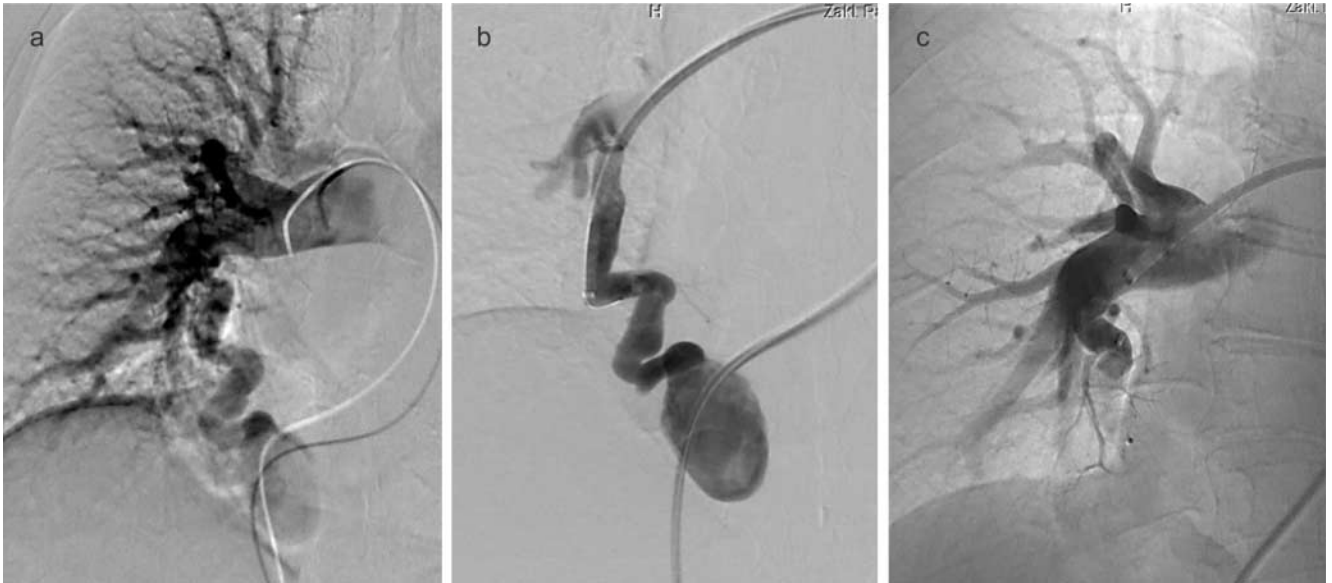
Fig. 1. 3D reconstruction of angio-CT in venous phase – a pulmonary arteriovenous aneurysmal malformation is visible

Tab. 3. Patient and procedure characteristics

Age (years)	Sex	Pattern of PAVMs location	Number and location of embolized PAVMs	Angioarchitecture of embolized lesions	Device used	Follow-up
77	F	single unilateral	1 RLL	1 simple (aneurysmal)	AVPII	CT after 12 months
36	F	multiple bilateral*	1 LUL 4 LLL	1 complex 4 simple	Coils and micro coils	2nd stage of embolization after 3 months
24	F	multiple bilateral	1 LLL 2 RLL	3 simple	AVP4 and coils	CT after 12 months
22	M	multiple bilateral	1 RLL	1 simple	Coils and micro coils	
27	F	single unilateral	1 LLL	1 simple	AVP4	

F – female; M – male; PAVM – pulmonary arteriovenous malformation; LUL – left upper lobe; LLL – left lower lobe; RLL – right lower lobe; AVP – Amplatzer Vascular Plug; CT – computed tomography

\*Patient with confirmed diagnosis of Hereditary Hemorrhagic Telangiectasia



**Fig. 2a-c.** Successive steps of fistula embolization with use of vascular occluder: a) pre-procedural selective angiography of the right pulmonary artery visualized aneurysmal fistula; b) 5Fr catheter introduced selectively to the feeding artery; c) control angiography depicted complete occlusion of the feeding artery with 10 mm Amplatzer Vascular Plug II.

## RESULTS

Immediate technical success was achieved in the total of 5 patients and 11 PAVMs (100%). 10 of 11 PAVMs were localized within lower lobes, with predominance of the left side (7 PAVMs). No procedural or periprocedural complications were observed. Four patients were referred for a follow-up chest CT after 12 months. One patient (with confirmed HHT diagnosis) was referred for the second stage of embolization after 3 months, due to numerous, symptomatic bilateral PAVMs. In our case series neither the angioarchitecture and location of PAVMs, nor embolization method influenced the technical success rates.

## DISCUSSION

Transcatheter embolization of the feeding artery is a first-line treatment of pulmonary arteriovenous malformations. The effectiveness of the procedure may be assessed in terms of technical, radiologic and clinical success or failure. The immediate technical success is defined as the occlusion of FA visible on angiogram obtained just after embolization. Radiologic success evaluation is strongly connected to follow-up studies (4). Permanent success is assessed during consecutive CT scans, with generally accepted criteria as follows: disappearance or apparent reduction of the AVP remnant (30-70% shrinkage is accepted as the cut-off value depending on literature source) for unenhanced imaging, along with lack of enhancement of the AVP sac and adjacent draining vein – in contrast-enhanced tomography scans (2, 5, 10, 12, 18, 21). Alternatively, no residual perfusion on angiography was also considered acceptable in those followed with this modality (5). Finally, clinical success is equal to symptoms resolution (4).

According to literature, the overall immediate technical success of the procedure, regardless of the device used, amounts to 80-100% (5, 8, 10, 14, 15, 21).

Nonetheless, it should be emphasized that the statistical data concerning effectiveness of embolization techniques are based on mostly retrospective, observational case series, thus susceptible to bias (11).

Unfortunately, the encouraging immediate success rates do not always translate to long-term effectiveness, which as reported by Remy-Jardin et al. reaches 75%, as observed during 10-year follow-up with CT (10, 12). In addition, it has been shown in a study on 112 patients, that long-term success rates depend on whether all angiographically visualized PAVMs were embolized (14). Persistence rates vary between 25-58% depending on study, however usually does not exceed 8% (1, 10). In addition, follow-up evaluation with time-resolved MR angiography or pulmonary angiography revealed even higher reperfusion rates (18).

As shown by numerous studies, there are several mechanisms underlying reperfusion, with recanalization being responsible for 63-100% cases (10, 15, 16, 21). However, the overall incidence of recanalization amounts approximately 8-10% of all embolized PAVMs and seems to be higher in presence of complex angioarchitecture (14). Recanalization more often occurs after coil embolization than vascular plugs implementation (1). This occurrence is mainly technique-related and may develop due to the impaired thrombus formation when insufficient number of coils is deployed (resulting in poor coil packing) or the coil elongation occurs (4, 5, 18). Sometimes spontaneous recanalization is observed, which results from coil compaction (20).

Other reperfusion mechanisms comprise missed or newly formed accessory artery, presence of bronchial or systemic collateral to the AVP sac (left-to-left shunt) and pulmonary artery-to-pulmonary artery collateral flow (4, 5, 15).

Additionally, coil embolization has two main technical limitations, i.e. 1) once deployed, the position of

device cannot be changed, and 2) sacrifice of vessels supplying normal lung tissue may be unavoidable due to use of common anchor and scaffold fixation techniques, which prevent device migration (1).

In spite of its simplicity and possibility of multiple repositioning before final deployment, AMPLATZER Vascular Plugs are not infallible as well (21-23). According to Fidelman et al. AVPs do not provide complete long-term occlusion of the feeding artery. Therefore, it had been proposed to decrease chances of reperfusion through additional deposition of coils, in the more proximal aspect of the FA (16). Furthermore, there are some serious difficulties related to reaching distally located, thin-walled PAVMs. However, this may be overcome by AVP4, which is characterized by lower profile (which makes them suitable to occlude < 6 mm vessels) and its delivery requires use of smaller sheaths and guiding catheters than previous AVP generations (2, 15).

At this point, it should be mentioned that enlargement of PAVMs (previously smaller than the threshold of CT detection) to a size requiring treatment is more common than reperfusion of the previously-treated ones. It occurs in approximately 18% of PAVMs – twice the collective number of reperfusion and persistence cases. It probably results from physiologic pressure due to redistribution of blood flow after embolization of other PAVMs,

rather than de novo growth (5, 22). Importantly, it has been proven that the clinically significant symptoms are mostly related to growing PAVMs and not recanalization of the previously embolized ones (5).

As suggested above, recanalization of previously embolized PAVMs may be avoided by precise technique of device deployment. Two main rules exist, which help to avoid this particular complication: 1) coils and vascular plugs should be located within 1 cm from the vascular sac; and 2) dense, cross-sectional occlusion should be obtained due to certain degree of device oversizing (10). Nevertheless, the need for diagnostic follow-up at regular intervals should be stressed as enlargement microscopic malformations, especially in patients with HHT often results in symptomatic recurrence of the disease.

## CONCLUSIONS

Embolization of the feeding artery is a highly-effective treatment option in case of pulmonary vascular malformations. AMPLATZER vascular occluder is a convenient device allowing for almost immediate occlusion of the supplying vessel to the PAVM. However, limited selection of device characteristics (i.e. diameter) favors coil embolization in case of feeding artery diameter less than 3 mm.

## BIBLIOGRAPHY

1. Tau N, Atar E, Mei-Zahav M et al.: Amplatzer Vascular Plugs Versus Coils for Embolization of Pulmonary Arteriovenous Malformations in Patients with Hereditary Hemorrhagic Telangiectasia. *Cardiovasc Intervent Radiol* 2016; 39: 1110-1114.
2. Hundt W, Kalinowski M, Kiessling A et al.: Novel approach to complex pulmonary arteriovenous malformation embolization using detachable coils and Amplatzer vascular plugs. *Eur J Radiol* 2012; 81: 732-738.
3. Faughnan ME, Palda VA, Garcia-Tsao G et al.: International guidelines for the diagnosis and management of hereditary haemorrhagic telangiectasia. *J Med Genet* 2011; 48: 73-87.
4. Tapping CR, Ettles DF, Robinson GJ: Long-Term Follow-Up of Treatment of Pulmonary Arteriovenous Malformations with AMPLATZER Vascular Plug and AMPLATZER Vascular Plug II Devices. *J Vasc Interv Radiol* 2011; 22: 1740-1766.
5. 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.
6. Corvino F, Silvestre M, Cervo A et al.: Endovascular occlusion of pulmonary arteriovenous malformations with the ArtVentive Endoluminal Occlusion SystemTM. *Diagn Interv Radiol* 2016; 22: 463-465.
7. Andersen PE, Kjeldsen AD: Interventional treatment of pulmonary arteriovenous malformations. *World J Radiol* 2010; 2: 339-344.
8. Ando K, Mochizuki A, Kurimoto N et al.: Coil Embolization for Pulmonary Arteriovenous Malformation as an Organ-sparing Therapy: Outcome of Long-term Follow-up. *Ann Thorac Cardiovasc Surg* 2011; 17: 118-123.
9. Pierucci P, Murphy J, Henderson KJ et al.: New definition and natural history of patients with diffuse pulmonary arteriovenous malformations: twenty-seven-year experience. *Chest* 2008; 133: 653-661.
10. Pollak JS, White RI Jr: Distal cross-sectional occlusion is the "key" to treating pulmonary arteriovenous malformations. *J Vasc Interv Radiol* 2012; 23: 1578-1580.
11. Hsu CC, Kwan GN, Thompson SA et al.: Embolisation for pulmonary arteriovenous malformation. *Cochrane Database Syst Rev* 2015; 1: CD008017.
12. Remy-Jardin M, Dumont P, Brillet PY et al.: Pulmonary Arteriovenous Malformations Treated with Embolotherapy: Helical CT Evaluation of Long-term Effectiveness after 2-21-Year Follow-up. *Radiology* 2006; 239: 576-585.
13. Shovlin CL, Jackson JE, Bamford KB et al.: Primary determinants of ischaemic stroke/brain abscess risks are independent of severity of pulmonary arteriovenous malformations in hereditary haemorrhagic telangiectasia. *Thorax* 2008; 63: 259-266.
14. Mager JJ, Overtoom TT, Blauw H et al.: Embolotherapy of Pulmonary Arteriovenous Malformations: Long-term Results in 112 Patients. *J Vasc Interv Radiol* 2004; 15: 451-456.
15. Rabellino M, Serra M, Peralta O et al.: Early experience with the AMPLATZER vascular plug IV for the occlusion of pulmonary arteriovenous malformations. *J Vasc Interv Radiol* 2014; 25: 1333-1337.
16. Fidelman N, Gordon RL, Bloom AI et al.: Reperfusion of pulmonary arteriovenous malformations after successful embolotherapy with vascular plugs. *J Vasc Interv Radiol* 2008; 19: 1246-1250.
17. Hayashi S, Baba Y, Senokuchi T, Nakajo M: Efficacy of venous sac embolization for pulmonary arteriovenous malformations: comparison with feeding artery embolization. *J Vasc Interv Radiol* 2012; 23: 1566-1577.
18. Shimohira M, Kawai T, Hashizume T et al.: Reperfusion Rates of Pulmonary Arteriovenous Malformations after Coil Embolization: Evaluation with Time-Resolved MR Angiography or Pulmonary Angiography. *J Vasc Interv Radiol* 2015; 26: 856-864.
19. Bélanger C, Chartrand-Lefebvre C, Soulez G et al.: Pulmonary arteriovenous malformation (PAVM) reperfusion after percutaneous embolization: Sensitivity and specificity of non-enhanced CT. *Eur J Radiol* 2016; 80: 150-157.
20. Kawai T, Shimohira M, Kan H et al.: Feasibility of Time-Resolved MR Angiography for Detecting Recanalization of Pulmonary Arteriovenous Malformations Treated with Embolization with Platinum Coils. *J Vasc Interv Radiol* 2014; 25: 1339-1347.
21. Abdel Aal AK, Ibrahim RM, Moustafa AS et al.: Persistence of pulmonary arteriovenous malformations after successful embolotherapy with Amplatzer vascular plug: long-term results. *Diagn Interv Radiol* 2016; 22: 358-364.
22. White RI Jr: Pulmonary Arteriovenous Malformations: How Do I Embolize? *Tech Vasc Interv Radiol* 2007; 10: 283-290.
23. Kucukay F, Özdemir M, Şenol E et al.: Large pulmonary arteriovenous malformations: long-term results of embolization with AMPLATZER vascular plugs. *J Vasc Interv Radiol* 2014; 25: 1327-1332.