

Original article

Association between reperfusion and shrinkage percentage of aneurysmal sac after embolization of pulmonary arteriovenous malformation: Evaluation based on contrast-enhanced thin-section CT images

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Abstract

Purpose: To investigate an association between reperfusion and percentage of shrinkage of the aneurysmal sac after embolization of pulmonary arteriovenous malformation (PAVM) and to determine the cut-off value of shrinkage percentage for indicating reperfusion.

Materials and Methods: Twenty-two PAVMs with completely embolized feeding arteries with coils were examined. The percentage of sac shrinkage and the presence of reperfusion were evaluated on computed tomography (CT) before and 1, 3, and 12 months after embolization. The percentages of sac shrinkage were compared between occlusion group and reperfusion group. The receiver operating characteristic (ROC) curve was generated to determine the diagnostic efficiency of reperfusion of PAVM by using shrinkage percentages.

Results: Reperfusion was seen in 14, 13, and 11 lesions at 1, 3, and 12 months, respectively. The mean percentage of sac shrinkage was significantly different between the 2 groups at 3 and 12 months. The area under ROC curve was 0.991 at 3 months and 0.934 at 12 months. All of 9 lesions with <60% sac shrinkage at 12 months showed reperfusion.

Conclusion: The percentage of sac shrinkage was closely associated with reperfusion after embolization of PAVMs at 3 and 12 months. A shrinkage percentage of <60% at 12 months indicated reperfusion.

Key words: pulmonary arteriovenous malformation, reperfusion, contrast-enhanced CT

Introduction

Pulmonary arteriovenous malformation (PAVM) is an abnormal connection between the pulmonary arterial circulation and the pulmonary venous circulation with no intervening capillary bed. PAVM may cause life-threatening complications, including stroke, cerebral abscess, and hemoptysis [1–3]. Since the first report of successful transcatheter embolization of a PAVM in 1977 [4], it has been accepted as standard treatment.

However, embolization occasionally results in incomplete occlusion of the shunt (i.e., reperfusion). Criteria for determining embolization success (i.e., absence of reperfusion) on radiological images have varied with different studies. One study determined success by visual assessment, which was indicated by complete or near-complete (linear scar only) resolution of the lesion on computed tomography (CT) or chest radiograph [5–8]; another indicated success by >30% reduction of lesion size [9]; and others indicated success by >70% reduction [5, 6]. Visual assessment is often subjective and depends on the modality (CT versus chest radiograph) and slice thickness of CT images.

Measurement of size reduction is objective, but which cut-off level accurately indicates the absence of reperfusion has not been fully investigated. Thus, we sought to retrospectively investigate an association between reperfusion and the shrinkage percentage of the aneurysmal sac after embolization using dynamic contrast-enhanced thin-section CT images. Subsequently, the cut-off value of shrinkage percentage for indicating reperfusion was determined.

Materials and methods

Study Population

Our Institutional Review Board (IRB) approved this retrospective study with a waiver

of obtaining informed consent. Between August 1999 and July 2011, we had treated 89 PAVMs in 42 patients with transcatheter coil embolization. Inclusion criteria were as follows: (a) the feeding artery of the lesion was completely embolized with coils; and (b) follow-up dynamic enhanced CT with thin-section reconstructions of the lesion of interest was performed at 1, 3, and 12 months. Exclusion criteria were as follows: (a) the sac or the draining vein of the lesion was embolized; (b) any of the follow-up CT examinations at 1, 3, and 12 months was unavailable; (c) follow-up CT was performed without contrast; and (d) thin-section reconstructed CT images were unavailable for the lesion of interest. Consequently, twenty-two lesions in 16 patients were included in this study. Characteristics of the study population are summarized in Table 1. Briefly, there were 5 men and 11 women; mean age was 52.6 ± 19.1 years. Seven patients were diagnosed with hereditary hemorrhagic telangiectasia. The mean diameter of the feeding arteries was 3.7 ± 1.1 mm. The type of PAVM was simple in 17 and complex in 5 lesions, according to the classification by White et al [10].

Embolization Technique

Criteria for embolization of PAVMs in our institution were lesions with a feeding artery larger than 3 mm in diameter [11–13] and/or those that caused clinical signs and symptoms. The endpoint of the embolization was complete occlusion of all feeding arteries. Through a femoral vein sheath, pulmonary arteriography was performed to assess the lesion using a 4- or 5-F catheter. Then, the catheter or a microcatheter through the catheter was advanced into the feeding artery of the lesion until its tip was immediately in front of the sac. Immediately before embolization, 3000 IU of heparin was administered intravenously for prophylactic anticoagulation, followed by additional administration of 1000 IU per hour. Any other anti-platelet or anti-coagulant drugs were

not administered before, during, or after the procedure. First, one or a few detachable bare microcoils were placed to prevent coil migration. Then, the feeding artery was embolized using pushable fibered microcoils alone or a combination of pushable fibered microcoils and 0.035-inch coils. After embolization, pulmonary arteriography was performed to confirm complete occlusion of the feeding artery.

Follow-up Examination

Embolization outcomes were evaluated with non-contrast followed by dynamic contrast-enhanced CT scanning using a single-detector or a multi-detector row scanner (Aquilion multi, Aquilion16, and Aquilion64; Toshiba Medical Systems Corporation, Tokyo, Japan, Discovery CT750 HD; GE Healthcare, Milwaukee, WI, USA, SOMATOM Definition Flash; Siemens Medical Systems, Erlangen, Germany) 1, 3, and 12 months after embolization. The scanner settings were 120 kV with the tube current set at 300 mA or automatically set by the exposure control system, depending on type of CT scanner. The region scanned extended from the cervicothoracic junction to the upper part of the abdomen. Images were acquired during deep inspiration.

After non-contrast CT scanning, a total of 80–100 mL of contrast medium was administered intravenously at a rate of 3–5 mL/s and CT images were acquired 33 s and 90 s after initiation of contrast administration. From each dataset, in addition to contiguous 5-mm transverse CT images of the whole thorax, contiguous thin-section (2–3-mm thick) transverse CT images of the area of interest were reconstructed with both mediastinal and lung windows.

Evaluation of Follow-up CT

Reperfusion of the aneurysmal sac was evaluated on the thin-section transverse CT images with the mediastinal window. Reperfusion was deemed present when the

aneurysmal sac was enhanced. The sac was considered to show enhancement when the CT number of the sac on contrast images increased by more than 100 Hounsfield units, comparing with that on non-contrast images. The maximum short-axis diameter of the sac was measured on the thin-section transverse CT images with the lung window. Aneurysmal sac shrinkage was measured using a reduction percentage of the diameter of the sac, comparing with that before embolization.

The lesions were divided into 2 groups according to the presence or absence of reperfusion at each follow-up time point (1, 3, and 12 months). Sac shrinkage percentage was compared between the 2 groups (reperfusion group versus occlusion group) with Student's *t*-test. Receiver operating characteristic (ROC) curves were generated to determine the diagnostic efficiency by using percentages of sac shrinkage to predict reperfusion. A *P* value < 0.05 was considered statistically significant. The statistical analyses were performed using commercially available software (SPSS software[®], IBM, Chicago IL, USA).

Results

Embolization

The feeding artery was embolized with microcoils alone for 15 lesions and a combination of microcoils and 0.035-inch coils for 7 lesions. The mean volume of coils deposited was $143 \pm 125 \text{ mm}^3$ (Table 1). Pulmonary arteriography immediately after embolization showed complete occlusion of the feeding artery in all lesions. Nevertheless, aneurysmal sac of 8 lesions was slowly opacified during the venous phase. This opacification was suggested to result from venous inflow into the sac from adjacent lung perfused by normal pulmonary arterial branches [14]. No complication

occurred in any of the embolization procedures.

Reperfusion and Sac Shrinkage

Cases of occlusion and reperfusion are shown in Fig. 1 and 2, respectively. Reperfusion and shrinkage percentage of the aneurysmal sac at each follow-up point in all cases are summarized in Table 1. Reperfusion was seen in 14, 13, and 11 lesions at 1, 3, and 12 months, respectively. In all cases of reperfusion, the sac was enhanced on CT images in both of the two phases. Of the 14 lesions with reperfusion at 1 month, 3 lesions were spontaneously occluded at 3 months, while 2 of the 8 lesions with occlusion at 1 month showed reperfusion at 3 months. Of the 13 lesions with reperfusion at 3 months, 2 lesions were spontaneously occluded at 12 months. Mean ($\pm$ SD) shrinkage percentage of the aneurysmal sac for all 22 lesions was $39.7 \pm 16.5\%$ (range, 11.4–80.1%), $52.8 \pm 17.9\%$ (range, 24.3–90.0%), and $56.1 \pm 21.7\%$ (range, 18.4–95.0%) at 1, 3, and 12 months, respectively. There were no lesions that showed partial or mural thrombosis in the venous sac on any follow-up CT images.

Among the 8 lesions that showed retrograde opacification of aneurysmal sac on pulmonary arteriography immediately after embolization, 3 lesions were unenhanced on CT at 1 month. Of the other 5 lesions, 1 became unenhanced at 3 month and another did at 12 months.

The ROC curve using sac shrinkage percentages and a dot plot of shrinkage percentages in the reperfusion group and the occlusion group at 1 month are shown in Fig. 3. At 1 month, the mean shrinkage of the aneurysmal sac was $45.6 \pm 18.0\%$ in the occlusion group ($n = 8$) and $36.3 \pm 15.1\%$ in the reperfusion group ($n = 14$); the difference was not significant ($P = 0.21$). The area under the ROC curve was 0.598 (95% CI, 0.346–0.850).

The ROC curve using sac shrinkage percentages and a dot plot of shrinkage percentages in the reperfusion group and the occlusion group at 3 months are shown in Fig. 4. At 3 months, the mean shrinkage was $41.4 \pm 12.1\%$ in the reperfusion group ($n = 13$) and $66.3 \pm 10.4\%$ in the occlusion group ($n = 9$); the difference was significant ($P < 0.001$). The area under the ROC curve was 0.991 (95% CI, 0.964–1.000). The sensitivity for reperfusion was 100% (13/13) and the specificity was 89% (8/9) when the cut-off value of the shrinkage was set at 60%; the sensitivity was 62% (8/13) and the specificity was 100% (9/9) when the cut-off value of shrinkage was set at 50% (Table 2).

ROC curve using sac shrinkage percentages and a dot plot of shrinkage percentages in the reperfusion group and the occlusion group at 12 months are shown in Fig. 5. At 12 months, the mean shrinkage of the aneurysmal sac was $39.7 \pm 16.3\%$ in the reperfusion group ($n = 11$) and $72.5 \pm 11.3\%$ in the occlusion group ($n = 11$); the difference was significant ($P < 0.001$). The area under the ROC curve was 0.934 (95% CI, 0.831–1.000). Sensitivity was 100% (11/11) and specificity was 55% (6/11) when the cut-off value of the shrinkage was set at 70%; sensitivity was 82% (9/11) and specificity was 100% (11/11) when the cut-off value of the shrinkage was set at 60% (Table 3).

Patient Outcome

The mean follow-up period of the patients was 28 ± 12 months (range, 12–51 months). All patients were alive and none developed symptoms (e.g., hemoptysis or dyspnea) or events (e.g., brain ischemia, abscess) related to the pulmonary AVMs.

Discussion

The literature includes different modalities using various criteria for post-embolization PAVM evaluation. The modalities include arterial blood partial pressure [6, 15], oxygen shunt testing [8], contrast echocardiography [8], chest radiography [6, 8, 15], unenhanced CT [5-8, 15], enhanced CT [9, 16], and pulmonary angiography [5-8, 15, 16]. The criteria for embolization success include complete or near-complete resolution (only linear scar visible) of the lesion on chest radiograph or CT [5-8], >30% reduction of the sac size [9], >70% reduction of the sac size [5, 6], <3% shunt fraction by lung perfusion scintigraphy [15], stable arterial blood gas levels (variance within 10% of value after embolization) [6, 15], and no perfusion of the lesion on enhanced CT [9] or pulmonary angiogram [5-8, 15, 16].

We believe that chest radiography, arterial blood partial pressure, contrast echocardiography, and lung perfusion scintigraphy are not sensitive enough to document minor reperfusion. Although pulmonary angiography is the most reliable modality for evaluation of reperfusion, its invasiveness makes it difficult to perform routinely for follow-up of the lesions. Thus, we used enhanced CT scanning for evaluation. Because the ability to detect reperfusion may depend on reconstructed image thickness, especially for small lesions, this study included only the cases in which thin-slice reconstructed images were available.

The proportions of reperfusion in our study were higher, compared with those in previous large series: 3% (11/393 lesions) by Pollak et al. [7], 7% (22/306 lesions) by Prasad et al. [8], and 8% (25/296 lesions) by Mager et al. [15]. In those studies, however, the presence of reperfusion was surveyed mainly by unenhanced CT or chest radiography [7], by oxygen shunt testing, contrast echocardiography, and unenhanced CT [8], or by chest radiography and/or arterial blood gas [16]. Only when reperfusion

was suspected by screening, pulmonary angiography was performed for confirmation. Sagara et al. [16] followed up patients with enhanced CT or pulmonary angiography. They showed 57% (8/14) reperfusion, which is similar to our result. We suggest that percentage of reperfusion depends highly on how intensively it was surveyed by using an examination with a contrast.

The degree of shrinkage of the sac was not significantly different between the reperfusion group and the occlusion group at 1 month, which suggests that 1 month is too soon to make a significant difference in degree of sac shrinkage by the presence of reperfusion. ROC analyses showed a close relationship between reperfusion and sac shrinkage at 3 and 12 months. At 3 months, >60% shrinkage of the sac always indicated occlusion and <50% shrinkage always indicated reperfusion; at 12 months, >70% shrinkage percentage always indicated occlusion, while <60% shrinkage percentage always indicated reperfusion. These results are similar to the findings reported by Milic et al. [5]: all PAVMs with documented reperfusion at angiography exhibited residual sac and/or draining vein on CT and all PAVMs without documented reperfusion at angiography demonstrated at least 70% reduction of the sac size. Thus, we suggest that the cut-off value of sac shrinkage indicating occlusion at 12 months should be 70%. In contrast, the cut-off value of sac shrinkage indicating reperfusion should be 50% at 3 months and 60% at 12 months.

Notably, 3 of the 14 reperfused lesions at 1 month were spontaneously occluded at 3 months, while 2 of the 8 occluded lesions at 1 month showed reperfusion at 3 months. These results indicate that the embolized PAVMs are hemodynamically unstable and thus efficacy of the embolization cannot be determined at 1 month. There are 4 mechanisms of reperfusion [5, 7]: (i) recanalization [6], (ii) accessory vessel

development [6, 17], (iii) bronchial arterial supply [16], and (iv) pulmonary-artery-to-pulmonary-artery collateral development [18]. Previous studies showed that the most common (33–100%) mechanism of reperfusion is recanalization of the embolized artery [5-8, 15-17, 19]. Treatment strategies for reperfusion are controversial. Some hold that any reperfusion should be treated because some patients with reperfused PAVMs developed neurologic events [5, 6]; others suggest that reperfusion with a feeding artery <3 mm is too small to be occluded [9]. We agreed with the latter suggestion and performed no repeat embolization for reperfusion, because the feeding arteries of all reperfused lesions were <3 mm in diameter. In our study, none of the patients with reperfused AVMs developed symptoms or signs related to reperfusion during follow-up.

Our study has several limitations. This study was retrospective. The relatively strict inclusion criteria resulted in a small sample size. Reperfusion was not angiographically confirmed. Thus, the rate of reperfusion in our study might have been underestimated, and without angiography, the mechanism of reperfusion could not be determined. In 8 lesions showing retrograde opacification of aneurysmal sac on arteriography immediately after embolization, enhancement of the sac on CT at any follow-up time point might have resulted from residual retrograde opacification rather than reperfusion. The relatively short follow-up period (up to 1 year after embolization) is another limitation. However, we suggest that presence of reperfusion is unlikely to change after 1 year.

Conclusions

The percentage of shrinkage of the aneurysmal sac was closely associated with reperfusion at 3 and 12 months. Sac shrinkage of <50% at 3 months and of <60% at 12

months indicated reperfusion.

References

1. Gossage JR, Kanj G. Pulmonary arteriovenous malformations. A state of the art review. *Am J Respir Crit Care Med*. 1998;158:643-61.
2. Swanson KL, Prakash UB, Stanson AW. Pulmonary arteriovenous fistulas: Mayo Clinic experience, 1982-1997. *Mayo Clin Proc*. 1999;74:671-80.
3. Shovlin CL, Letarte M. Hereditary haemorrhagic telangiectasia and pulmonary arteriovenous malformations: issues in clinical management and review of pathogenic mechanisms. *Thorax*. 1999;54:714 -29.
4. Porstmann W. Therapeutic embolization of arteriovenous pulmonary fistula by catheter technique. In: Kelop O, ed. *Current concepts in pediatric radiology*. Berlin: Springer, 1977;23-31.
5. Milic A, Chan RP, Cohen JH, Faughnan ME. Reperfusion of pulmonary arteriovenous malformations after embolotherapy. *J Vasc Interv Radiol*. 2005;16:1675-83.
6. Lee DW, White RI Jr, Egglin TK, Pollak JS, Fayad PB, Wirth JA, et al. Embolotherapy of large pulmonary arteriovenous malformations: long-term results. *Ann Thorac Surg*. 1997;64:930-40.
7. Pollak JS, Saluja S, Thabet A, Henderson KJ, Denbow N, White RI Jr. Clinical and anatomic outcomes after embolotherapy of pulmonary arteriovenous malformations. *J Vasc Interv Radiol*. 2006;17:35-44.
8. Prasad V, Chan RP, Faughnan ME. Embolotherapy of pulmonary arteriovenous malformations: efficacy of platinum versus stainless steel coils. *J Vasc Interv Radiol*. 2004;15:153-60.
9. Remy-Jardin M, Dumont P, Brillet PY, Dupuis P, Duhamel A, Remy J. Pulmonary

- arteriovenous malformations treated with embolotherapy: helical CT evaluation of long-term effectiveness after 2-21-year follow-up. *Radiology*. 2006;239:576-85.
10. White RI Jr, Pollak JS, Wirth JA. Pulmonary arteriovenous malformations: diagnosis and transcatheter embolotherapy. *J Vasc Interv Radiol*. 1996;7:787-804.
11. Hewes RC, Auster M, White RI Jr. Cerebral embolism--first manifestation of pulmonary arteriovenous malformation in patients with hereditary hemorrhagic telangiectasia. *Cardiovasc Intervent Radiol*. 1985;8:151-5.
12. White RI Jr, Lynch-Nyhan A, Terry P, Buescher PC, Farnlett EJ, Charnas L, et al. Pulmonary arteriovenous malformations: techniques and long-term outcome of embolotherapy. *Radiology*. 1988;169:663-9.
13. Moussouttas M, Fayad P, Rosenblatt M, Hashimoto M, Pollak J, Henderson K, et al. Pulmonary arteriovenous malformations: cerebral ischemia and neurologic manifestations. *Neurology*. 2000;55:959-64.
14. Pugash RA. Pulmonary arteriovenous malformations: overview and transcatheter embolotherapy. *Can Assoc Radiol J*. 2001;52:92-102.
15. Mager JJ, Overtom TT, Blauw H, Lammers JW, Westermann CJ. Embolotherapy of pulmonary arteriovenous malformations: long-term results in 112 patients. *J Vasc Interv Radiol*. 2004;15:451-6.
16. Sagara K, Miyazono N, Inoue H, Ueno K, Nishida H, Nakajo M. Recanalization after coil embolotherapy of pulmonary arteriovenous malformations: study of long-term outcome and mechanism for recanalization. *AJR Am J Roentgenol*. 1998;170:727-30.
17. Saluja S, Sitko I, Lee DW, Pollak J, White RI Jr. Embolotherapy of pulmonary arteriovenous malformations with detachable balloons: long-term durability and efficacy. *J Vasc Interv Radiol*. 1999;10:883-9.

18. Faughnan ME, Thabet A, Mei-Zahav M, Colombo M, Maclusky I, Hyland RH, et al. Pulmonary arteriovenous malformations in children: outcomes of transcatheter embolotherapy. *J Pediatr*. 2004;145:826-31.
19. Andersen PE, Kjeldsen AD. Clinical and Radiological Long-Term Follow-up After Embolization of Pulmonary Arteriovenous Malformations. *Cardiovasc Intervent Radiol*. 2006;29:70-4.

Figure legends

Fig. 1 A case of successfully occluded pulmonary arteriovenous malformation after embolization.

a. CT image before embolization shows an aneurysmal sac (arrow) with maximum short-axis diameter of 24.7 mm.

b. CT image 1 month after embolization shows an aneurysmal sac (arrow) with maximum short-axis diameter of 18.2 mm (26.3% shrinkage) without perfusion. Enhancement demonstrated at the periphery of the sac indicates an adjacent pulmonary vessel (arrowhead).

c. CT image 3 months after embolization shows an aneurysmal sac (arrow) with maximum short-axis diameter of 7.3 mm (70.4% shrinkage) without perfusion. Arrowhead indicates the deposited coils.

d. CT image 12 months after embolization shows an aneurysmal sac (arrow) with maximum short-axis diameter of 6.7 mm (72.9% shrinkage) without perfusion. Arrowhead indicates the deposited coils.

Fig. 2 A case of reperfused pulmonary arteriovenous malformation after embolization.

a. CT image before embolization shows an aneurysmal sac (arrow) with maximum short-axis diameter of 9.5 mm.

b. CT image 1 month after embolization shows a perfused aneurysmal sac (arrow) with maximum short-axis diameter of 4.7 mm (50.5% shrinkage).

c. CT image 3 months after embolization shows a perfused aneurysmal sac (arrow) with maximum short-axis diameter of 4.7 mm (50.5% shrinkage).

d. CT image 12 months after embolization shows a perfused aneurysmal sac (arrow)

with maximum short-axis diameter of 5.1 mm (46.4% shrinkage).

Fig. 3 Receiver-operating characteristic (ROC) curves using sac shrinkage percentages (a) and a dot plot (b) at 1 month.

- a. The area under the ROC curve was 0.598 (95% CI, 0.346–0.850) at 1 month.
- b. Mean shrinkage of the aneurysmal sac was $36.3 \pm 15.1\%$ in the reperfusion group ($n = 14$) whereas it was $45.6 \pm 18.0\%$ in the occlusion group ($n = 8$); the difference was not significant ($P = 0.21$).

Fig. 4 Receiver-operating characteristic (ROC) curves using sac shrinkage percentages (a) and a dot plot (b) at 3 months.

- a. The area under the ROC curve was 0.991 (95% CI, 0.964–1.000) at 3 months.
- b. Mean shrinkage was $41.4 \pm 12.1\%$ in the reperfusion group ($n = 13$) whereas it was $66.3 \pm 10.4\%$ in the occlusion group ($n = 9$); the difference was significant ($P < 0.001$).

Fig. 5 Receiver-operating characteristic (ROC) curves using sac shrinkage percentages (a) and a dot plot (b) at 12 months.

- a. The area under the ROC curve was 0.934 (95% CI, 0.831–1.000) at 12 months.
- b. Mean shrinkage of the aneurysmal sac was $39.7 \pm 16.3\%$ in the reperfusion group ($n = 11$) whereas it was $72.5 \pm 11.3\%$ in the occlusion group ($n = 11$); the difference was significant ($P < 0.001$).

Table 1. Summary of 22 lesions in 16 patients

Lesion no.	Patient no.	Sex	Age (yrs)	Presence of HHT	Lesion Type	Diameter of feeding artery (mm)*	Volume of coils deposited (mm ³)**	Retrograde opacification via draining	Follow up results						
									1 mo		3 mo		12 mo		
									Reperfusion	Sac shrinkage (%)	Reperfusion	Sac shrinkage (%)	Reperfusion	Sac shrinkage (%)	Dimameter of feeding artery (mm)
1	1	F	58	yes	simple	4.6	87.0	yes	no	32.5	yes	54.5	no	61.2	1.6
2	1	F	58	yes	complex	2.6	42.7	no	yes	44.7	yes	41.3	yes	27.5	2.6
3	2	F	59	no	simple	3.8	101.7	yes	yes	38.9	no	63.9	no	70.8	2.6
4	3	F	33	yes	simple	4.5	59.1	no	no	32.3	no	54.6	no	60.0	2.0
5	4	F	64	no	simple	3.8	62.3	no	yes	50.5	yes	50.5	yes	46.3	1.9
6	5	F	60	no	complex	4.6	131.3	no	no	38.0	no	62.0	no	62.0	1.6
7	6	M	16	yes	simple	3.4	52.5	yes	no	47.5	yes	44.3	yes	27.9	2.0
8	6	M	16	yes	complex	5.8	232.9	yes	no	26.3	no	70.4	no	72.9	1.3
9	7	M	14	yes	simple	3.2	50.9	yes	yes	42.0	yes	43.2	yes	43.2	2.5
10	7	M	14	yes	simple	3.0	88.6	no	yes	36.7	yes	36.7	yes	32.2	2.8
11	7	M	14	yes	simple	2.1	39.4	no	yes	22.4	yes	24.5	yes	18.4	2.4
12	7	M	14	yes	simple	2.6	39.4	no	yes	48.4	yes	52.4	yes	54.2	2.0
13	7	M	14	yes	simple	1.7	32.8	no	yes	35.1	yes	25.6	yes	26.3	1.9
14	8	F	74	no	simple	4.4	281.5	yes	yes	64.5	no	73.8	no	74.6	1.9
15	9	M	60	no	complex	3.2	420.7	no	no	63.2	no	77.8	no	83.1	1.7
16	10	F	82	no	simple	5.5	358.5	no	yes	36.7	yes	50.3	yes	62.1	2.4
17	11	F	51	no	simple	2.0	25.2	no	yes	13.2	no	63.2	no	65.8	1.2
18	12	F	71	no	simple	5.1	286.5	yes	yes	45.2	yes	59.6	yes	68.2	1.3
19	13	M	50	yes	complex	3.7	289.1	no	no	80.1	no	90.0	no	95.0	1.4
20	14	F	62	no	simple	2.8	32.8	no	yes	11.4	yes	24.3	yes	30.4	1.4
21	15	M	40	no	simple	4.7	311.5	yes	yes	19.0	yes	31.0	no	66.5	1.5
22	16	F	48	no	simple	3.8	120.3	no	no	45.1	no	67.6	no	85.9	2.3

HHT hereditary hemorrhagic telangiectasia, M male, F female

*The largest diameter of the feeding arteries is shown in case of complex lesions.

**Volume of coils deposited is calculated using diameter and length of the coils.

Table 2. Number of lesions with reperfusion according to sac shrinkage at 3 months

Reperfusion	Sac shrinkage (%)		
	<50%	50-60%	>60%
Yes	8	5	0
No	0	1	8

Table 3. Number of lesions with reperfusion according to sac shrinkage at 12 months

Reperfusion	Sac shrinkage (%)		
	<60%	60-70%	>70%
Yes	9	2	0
No	0	5	6

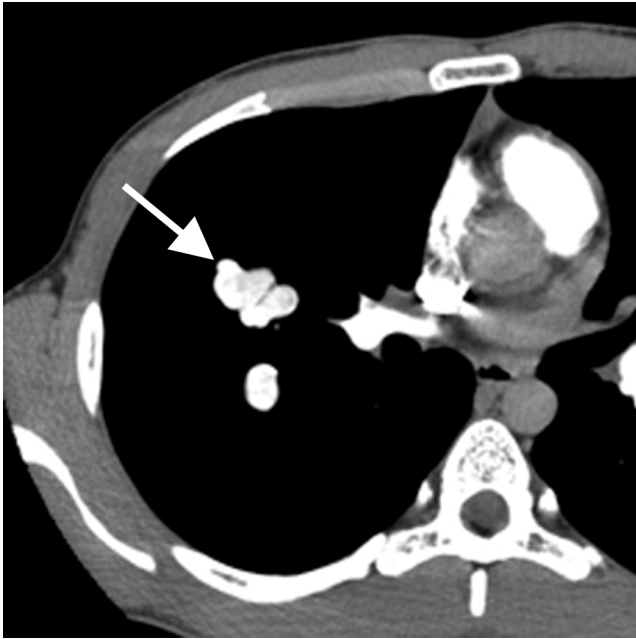


Fig1a

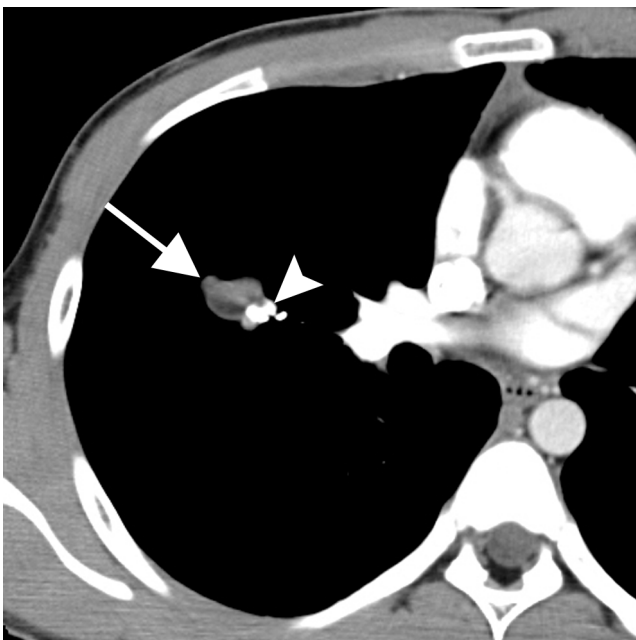


Fig1b



Fig1c

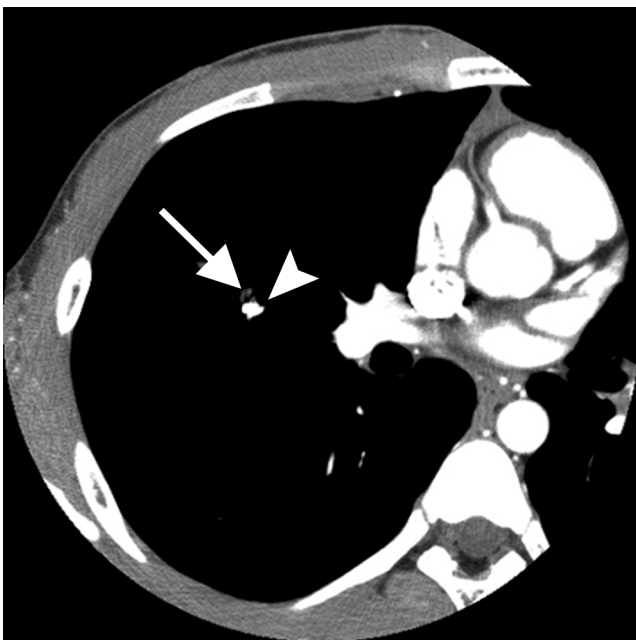


Fig1d

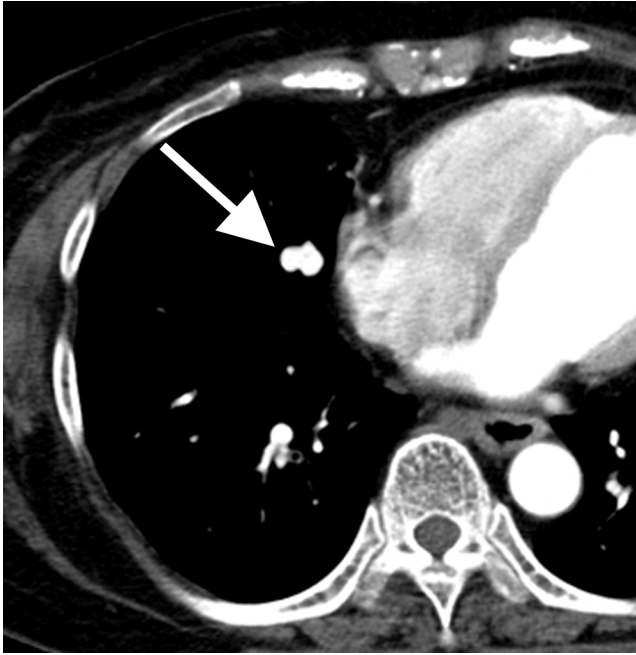


Fig2a

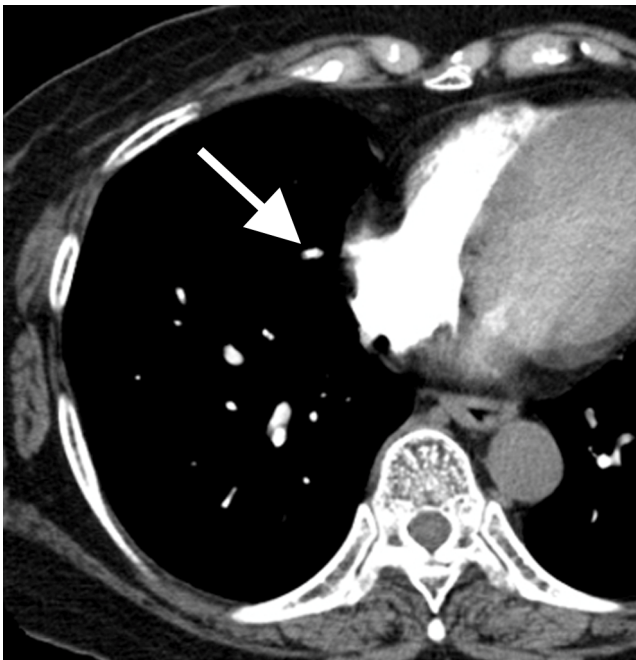


Fig2b

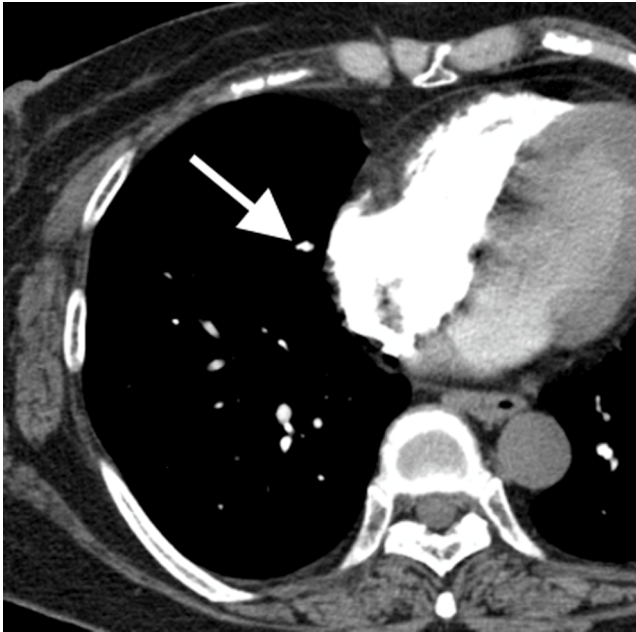


Fig2c

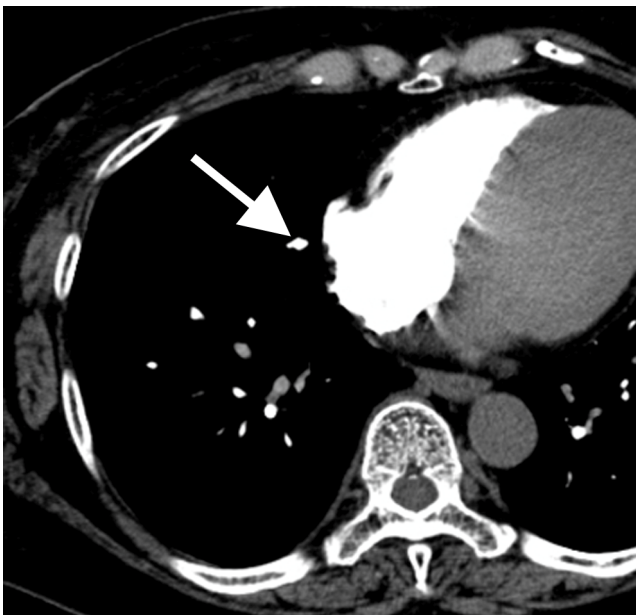


Fig2d

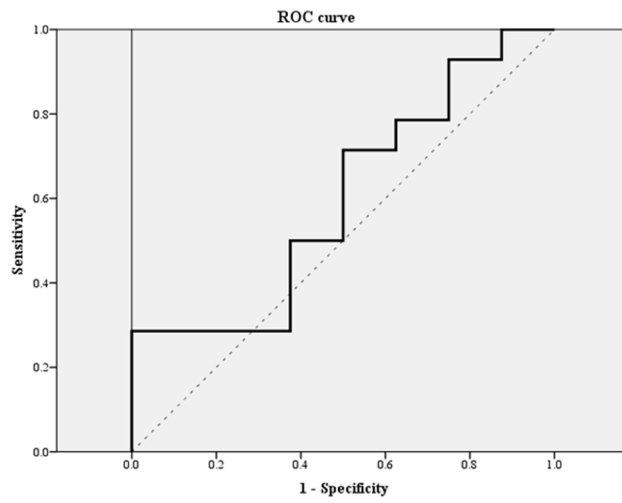


Fig3a

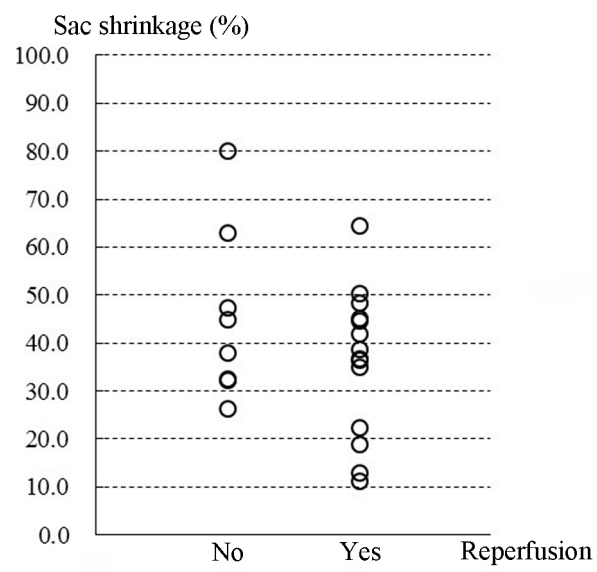


Fig3b

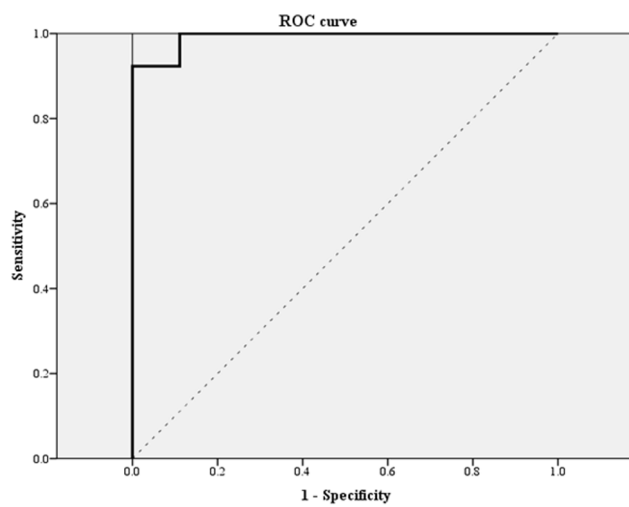


Fig4a

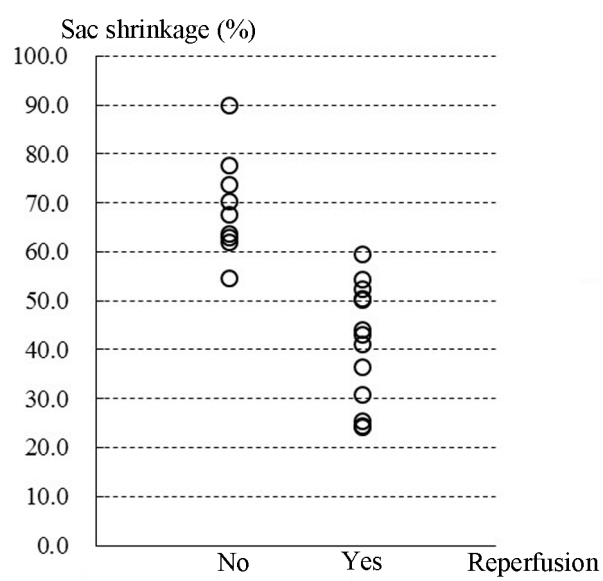


Fig4b

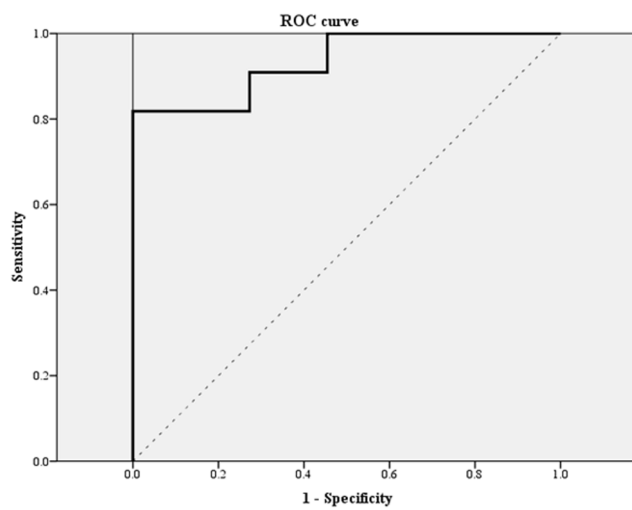


Fig5a

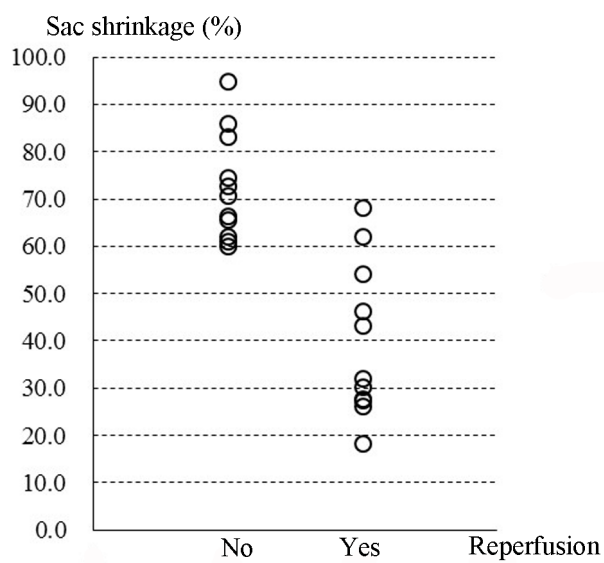


Fig5b