Case Report


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Although endovascular intervention is the first-line treatment of intracranial aneurysm, intraprocedural rupture or extravasation is still an endangering event. We describe two interesting cases of extravasation during embolotherapy for ruptured peripheral cerebral pseudoaneurysms. Two male patients were admitted after development of sudden headache with presentation of intracerebral and subarachnoid hemorrhage, respectively. Initial angiographic assessment failed to uncover any aneurysmal dilatation in both patients. Two weeks afterwards, catheter angiography revealed aneurysms each in the peripheral middle cerebral artery and anterior inferior cerebellar artery. Under a general anesthesia, endovascular embolization was attempted without systemic heparinization. In each case, sudden extravasation was noted around the aneurysm during manual injection of contrast after microcatheter navigation. Immediate computed tomographic scan showed a large amount of contrast collection within the brain, but they tolerated and made an unremarkable recovery thereafter. Intraprocedural extravasation is an endangering event and needs prompt management, however proximal plugging with coil deployment can be sufficient alternative, if one confronts with peripheral pseudoaneurysm. Peculiar angiographic features are deemed attributable to extremely fragile, porous vascular wall of the pseudoaneurysm. Accordingly, it should be noted that extreme caution being needed to handle such a friable vascular lesion.

KEY WORDS: Endovascular embolization · Extravasation · Pseudoaneurysm.

INTRODUCTION

Endovascular embolization has become the initial treatment method for intracranial aneurysm in majority of neurological institutes. As individual experiences accumulate, peri-procedural adverse effects with resultant sequelae can be minimized. However, complications including intraprocedural rupture and thrombo-embolic events, two most intimidating conditions, are existing threats345. We report two cases of extravasation during embolization therapy without neurological sequelae by imminent proximal occlusion with Guglielmi Detachable Coils (GDCs). We speculate that specific vascular anatomy of the pseudoaneurysm might result in otherwise unexplainable angiographic and clinical findings.

CASE REPORT

Case 1.
A 31-year-old, previously healthy man was admitted with a presentation of sudden headache during sleeping. On admission, he was alert and had no neurologic deficit, but sustained weight loss of 8 kg during the past 6 months. Pre-enhanced brain computed tomographic (CT) scan showed intracerebral hemorrhage and thin subdural bleeds on the left frontal area (Fig. 1A). Immediate CT angiography and transfemoral catheter angiography (TFCA) failed to reveal any lesion, and conservative treatment was then given. On day 15, a TFCA showed a globular shaped aneurysmal sac at the M3 of the middle cerebral artery (MCA) with peculiar angiographic features of delayed filling and emptying which designate a pseudoaneurysm (Fig. 1B, C).

On the next morning, endovascular embolization was performed under a general anesthesia without systemic heparinization. After placing 6-French Envoy guide-catheter (Cordis Corporation, Miami Lakes, FL) into the proximal
petrous carotid, an Excelsior 10 microcatheter with a Synchro microguidewire (Boston Scientific, Miami, FL) was navigated further into the junction of M2 and M3. Then, immediately following manual injection of the contrast medium (Visipaque, GE Healthcare Korea, Seoul), extravasation from the aneurysmal sac was noticed (Fig. 1D). At that time point, there was no alteration of vital signs and proximal M3 was immediately plugged with a GDC (2 × 30 mm, Ultrasoft), with confirming no distal flow to the GDC (Fig. 1E). He awoke soon afterwards and exhibited free of any neurologic deficit, but CT scan showed combination of a huge intracerebral collection of the contrast media with Hounsfield unit (HU) of 280, some blood and trapped air (Fig. 1F).

He made a full recovery in next several days, and meticulous evaluation of weight loss disclosed a grade III aortic regurgitation on echocardiography. He underwent an artificial valve replacement operation 1 month thereafter. Follow-up angiography showed no residual aneurysm one year later (Fig. 1G).

**Case 2.**

A 48-year-old, previously healthy man was brought in to the emergency room due to complaint of headache and dizziness. On admission, he was alert and fully oriented despite acute ill-looking appearance. He showed subtle diplopia on left lateral gaze and mild left hemifacial dysesthesia. There was no recognizable history of trauma or infection. A plain head CT scan showed diffuse, Fisher grade III subarachnoid hemorrhage (SAH) predominantly within the preoptic cistern (Fig. 2A). Both CT angiography and TFCA failed to uncover any vascular abnormality for bleeds, and he was treated conservatively. Two weeks post-ictus, CT scan showed resolved SAH with high-density lesion in the left preoptic cistern, presumably due to thrombosis. A TFCA exhibited a globular contrast filling in late arterial to venous phase at the left anterior inferior cerebellar artery (AICA), supplied via the right vertebral artery by crossing the midline (Fig. 2B, C). Pseudoaneurysm was suspected, and embolization was scheduled.

Under a general anesthesia, endovascular embolization was attempted with a 6-French Envoy guide-catheter placing in the proximal right vertebral artery. An Excelsior microcatheter with a Synchro microguidewire could reach the lesion, and during injection of contrast agent into the lesion within the superselected left AICA, sudden extravasation occurred around the aneurysm (Fig. 2D). At this time point, systolic pressure increased to 170 mmHg, and nitroprusside was rapidly infused. And meantime, GDC (2 × 30-mm, Ultrasoft) was promptly deployed into the arterial lumen proximal to the aneurysm, while maintaining a microcatheter position. Final working view confirmed GDC in

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**Fig. 1.** Radiographic images of a 31-year-old man with complaint of sudden headache during sleeping. A: Head computed tomographic (CT) scan shows intracerebral hemorrhage on the left frontal area. B, C: Late arterial phase, carotid angiograms show a pseudo-sac at frontal cortical branch of the middle cerebral artery in frontal and lateral projection. D: Following contrast injection, extravasation happens along the path of distal M3 and the aneurysm. E: Proximal M3 is plugged with a Guglielmi Detachable Coil (2 × 30-mm, Ultrasoft) and distal flow to the coil is not shown further. F: Postoperative CT scan shows combination of a large amount of the intracerebral collection of contrast agent (HU: 280), blood and air. G: Carotid angiogram shows no aneurysm at 1-year follow-up.
the appropriate position, and interstitial filling within the aneurysm was not shown distal to the plugging (Fig. 2E). He became fully conscious without limitation of limb motion, although a head CT scan showed collection of contrast in the left pons (Fig. 2F).

He made an unremarkable postoperative course, and TFCA on postoperative day showed no aneurysm filling. Six months postoperatively, TFCA exhibited no contrast filling from the origin of the left AICA with total disappearance of the aneurysm (Fig. 2G).

**DISCUSSION**

In this report, it is not certain that embolization angiography literally implies whether plain rupture of the aneurysm took place or simple contrast extravasation happened through the porous vessel wall. If we consider patient’s unusual angioarchitectures and clinical history of delayed diagnosis after repeat examination, pathologic subtype of the ruptured aneurysm must be false, rather than that of true type. Intracranial pseudoaneurysm is associated with apparent history of preceding inflammation, trauma, or invasive procedures, although we only disclosed the first case as secondary to the infective endocarditis. In general, intracranial infectious aneurysms should be treated conservatively without surgical or endovascular intervention, especially unruptured aneurysms. But, in our case, a ruptured aneurysm was located distally and being composed of extraluminal hematoma containing connective tissues following complete vessel wall injury, the hematoma is likely to recanalize, and this imperfect wall is formed through hematoma organization and fibrosis. Thus, as its name indicates, none of the normal vascular structures are present. Unremarkable post-extravasation neurologic state renders us to believe this phenomenon being inherent to the pseudoaneurysm. Pseudoaneurysm is clearly presented in the angiography, such as uncommon peripheral location, suspicious extramural thrombi, delayed filling and emptying of contrast agent, and stagnation of contrast with regard to the head position. High HU of 280 to 350 in the post-embolization CT scans as well as blood and some trapped air demonstrates this peculiar features as contrast extravasation.

Regardless of conceptual vagueness, extravasation or intraprocedural rupture is one of the most dreadful complications during the endovascular treatment. It occurs in 2-5% for ruptured aneurysm, and high mortality is reported up to 50%. It is usually caused by increased intraluminal pressure after injection of contrast medium into the aneurysmal sac as seen in the present cases, or perforation of the aneurysm by coils and/or guidewire. It is possible to generate enormous pressure during injection with a 1 cc

![Fig. 2. Radiographic images of a 48-yr-old man presenting with headache and dizziness. A: Admission head computed tomographic (CT) scan shows diffuse subarachnoid hemorrhage and high-density lesion in the left preoptic cistern, designating thrombosis. B, C: Late arterial vertebral angiograms exhibit a globular pseudo-sac at the left-sided anterior inferior cerebellar artery (AICA), which are supplied from the right vertebral artery. D: Sudden extravasation occurs around the aneurysm, during contrast injection into the superselective AICA. E: A Guglielmi Detachable Coil (2×30 mm, UltraSoft) is promptly deployed into the proximal arterial lumen and filling within the aneurysm is hardly seen. F: Postoperatively, head CT scan shows collection of contrast medium (HU: 350). G: Postoperative six months, vertebral angiogram shows no contrast filling from the origin of the left AICA with total disappearance of the aneurysm.](image-url)
syringe through a microcatheter, particularly when the microcatheter is wedged in a small vessel such as shown in this case. Either small amount of pressure or volume alone is sufficient to cause an extremely friable cerebral artery to rupture. There is no vessel wall to support additional pressure during the microcatheter injection. It might be reasonable to consider some variation in technique that does not require forceful contrast injection into the fragile artery giving rise to a pseudoaneurysm. Once identified in read-out images, additional contrast injection might be omitted, only if decision to trapping is scheduled. Or, use of a microcatheter with a very small-diameter, not wedged in a vessel can be alternative, when glue or liquid coils can be selected. As for the rupture, flow diversion by coils toward weaker portions of the aneurysmal wall, smaller dome size (<4 mm), recent rerupture due to more fragile aneurysmal wall, and presence of daughter sac are another potential etiologies. In technical viewpoint, careless handling of the wire or catheter, aneurysm overpacking or coil oversizing, liberal use of stiffer 3-D GDCs, balloon-assisted coiling are also possible causes. Once hemorrhage occurs, there is transient arrest of angiographic flow with contrast extravasation and abrupt rise of systolic pressure. To gain control of rupture, heparin reversal with intravenous injection of protamine sulfate and further coil packing should be attempted quickly, if it occurs during early phase. When the aneurysm is perforated by the microcatheter, use of a second microcatheter to occlude a perforated aneurysmal wall allows the original microcatheter reposition and this generally results in good clinical outcome. In our case, prompt and adequate procedures made a desirable outcome and additional bleeding was reduced by non-heparinization. In a high-risk patient with daughter sac, small size, or tortuous anatomy, it is imperative to have an immediate extraventricular drainage or at least preparing it. However, it may not be sufficient to prevent poor clinical outcome. Vessel sacrifice is another option and it includes autologous blood patch, detachable balloons, glue, or GDCs. The GDCs are preferred to the others because they can cause less recanalization of the aneurysm, less chance of distal embolization, and more precise placement. Because brain ischemia is a major concern distal to the embolization on the parent vessel territory, it should be done with great caution after fully assessing pre-embolization angiography. Without actual occlusion at the site of rupture, it is possible that hemorrhage will continue through peripheral collateral circulation beyond a proximal occlusion. Obliteration of the aneurysm or pseudoaneurysm without further contrast filling should be the goal, regardless of treatment.

CONCLUSION

In summary, extravasation during embolization is a definite threat and needs swift management. But, when encountered in specific conditions of peripherally located pseudoaneurysm, proximal plugging with coils might be sufficient to prevent further disaster. If such a friable vascular lesion has to be treated, it is imperative to pay an extreme caution to avoid intraoperative mishaps. Moreover, several different microcatheter injection technique should be utilized to avoid rupture, when encountered in such a friable aneurysm.

References