Intraprocedural Rupture of Unruptured Cerebral Aneurysms During Coil Embolization: A Single-Center Experience

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■ OBJECTIVE: **Incidence of intraprocedural rupture (IPR) during endovascular coiling is reported to be 2%–5%. We reviewed a single-center experience of IPR during coil embolization of unruptured intracranial aneurysms.**

■ METHODS: **Between January 2011 and April 2016, 849 patients were treated with endovascular therapy for unruptured intracranial aneurysm. IPR was documented in 10 (1.18%) of these patients. We reviewed medical records to evaluate characteristics of the aneurysms, angiographic findings related to rupture, management, and outcomes.**

■ RESULTS: **Among the 10 patients, there were 4 internal carotid artery aneurysms, 3 anterior communicating artery aneurysms, 2 basilar tip aneurysms, and 1 middle cerebral artery aneurysm. The probable mechanism of IPR in 7 patients was focal coil mass distention. Two patients underwent rupture owing to injury by a microcatheter tip that was related to device-device interaction. In 1 patient who had no other clear etiology, increased intra-arterial pressure induced by contrast injection was suspected as a cause of rupture. In all cases, rapid occlusion at the point of suspected leakage was performed, and final angiography showed complete obliteration of the aneurysm. After the procedure, neurologic deterioration was demonstrated in 2 patients. The modified Rankin Scale score at 6-month follow-up was 0 in 7 of the patients.**

■ CONCLUSIONS: **Incidence of IPR during endovascular coiling of unruptured aneurysms is relatively low. Early detection followed by rapid occlusion of the aneurysm can lead to a benign clinical course in most cases.**

INTRODUCTION

The endovascular treatment of cerebral aneurysms undergoes continuous innovation. Neurologic interventional devices have evolved from earlier detachable coils and liquid embolic agents into the variety of different coil types and flow diverters currently available. Coil embolization is widely accepted to treat cerebral aneurysms,[1-3] and intraprocedural rupture (IPR) is one of the most serious complications.[4-7] Many studies have discussed the incidence of IPR without regard for whether or not the aneurysm was ruptured before treatment. Most of these studies considered IPR of previously ruptured aneurysms, whereas few studies to date have investigated IPR in cases of unruptured aneurysms. With consideration of the large number of unruptured cerebral aneurysm cases treated within our center, in this study, we focused on IPR during coil embolization of previously unruptured aneurysms, examining risk factors, mechanisms of rupture, and management.

MATERIALS AND METHODS

Between January 2011 and April 2016, 849 unruptured cerebral aneurysms were treated with endovascular coil embolization at our institution. Of these, 10 patients (1.18%) who had IPR during embolization were included in this analysis. Patient demographic data, aneurysm characteristics, procedural details, clinical outcomes, and follow-up imaging were obtained. Data were obtained from electronic medical records (progress notes, anesthesia records, procedural records, and radiologic reports), which were available for all cases. To evaluate aneurysm characteristics, digital subtraction angiography, including rotational angiography for three-dimensional (3D) reconstruction imaging, was performed

Key words
- Aneurysm
- Coiling
- Embolization
- Rupture

Abbreviations and Acronyms
3D: Three-dimensional
CT: Computed tomography
IPR: Intraprocedural rupture

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preoperatively in all patients using the Artis zee biplane angiographic system (Siemens Healthcare GmbH, Erlangen, Germany). Aneurysm size was measured on the 3D reconstruction image, and sizes were classified as very small (0–3 mm), small (3–7 mm), medium (7–10 mm), large (10–25 mm), and giant (>25 mm), according to maximal dimension. Treatment plans were developed by 2 neurointerventionalists and 3 neurosurgeons on the basis of these data. The same protocol for endovascular therapy was used in all patients, with all of them undergoing coil embolization under general anesthesia. All patients received dual antiplatelet medications (100 mg aspirin and 75 mg clopidogrel per day) for at least 5 days before the procedure, without a loading dose. After puncture of the common femoral artery, systemic heparinization was achieved with a target activated clotting time of 200–250 seconds, or 2–2.5× baseline. Aneurysm obliteration was performed by packing of the sac with a detachable coil. A single- or multiple-microcatheter technique was primarily considered, with stent or balloon assistance performed for wide-neck aneurysms. Cases of IPR were considered to be cases that showed extravasation of contrast material from the aneurysm sac on angiography during the procedure. Radiologic follow-up was performed using digital subtraction angiography, magnetic resonance angiography, or computed tomography (CT) angiography. Clinical outcomes were determined using a modified Rankin Scale at the last follow-up.

RESULTS

Table 1 summarizes baseline characteristics and procedural details. All aneurysms were unruptured intracranial saccular aneurysms. There were 7 women and 3 men with a mean age of 58 years (range, 40–70 years). The aneurysms were incidentally detected in 9 cases (90%). In these cases, headache was the most common symptom leading to the radiologic diagnosis of aneurysm. The remaining case was a recurrent aneurysm that had previously been treated with coil embolization; the patient presented with visual loss secondary to compression by the recurring aneurysm sac. The shapes of the 9 de novo aneurysms were elongated ($n = 5$), round ($n = 2$), or lobulated ($n = 2$). Bleb or daughter sacs were also visible in 3 cases on 3D images obtained from rotational angiography. The mean aneurysm size was 5.1 mm (range, 3.5–15 mm); 8 aneurysms were small, 1 was medium, and 1 was large. The most common aneurysm location was the anterior communicating artery ($n = 3$), followed by the paraclinoid internal carotid artery ($n = 2$), posterior communicating artery ($n = 2$), basilar artery tip ($n = 2$), and middle cerebral artery bifurcation ($n = 1$).

The detection of IPR was based mainly on the angiographic findings. The main angiographic sign for the detection of rupture was protrusion of the coil loop from the aneurysm contour during coil insertion (7 cases) (Figure 1), with leakage of contrast material on the control angiogram (2 cases) and migration of the microcatheter tip over the aneurysm sac during the delivery of another microcatheter (1 case) also occurring. After first detection of the suspected rupture, a control angiogram was promptly obtained, and extravasation of contrast material was documented in all cases. The most common site of rupture was the aneurysmal neck. CT performed immediately after the procedures showed contrast material within the subarachnoid or cisternal space in all cases and combined intraventricular hemorrhage and hydrocephalus in 2 cases. Only 2 cases (20%) showed a simultaneous increase of arterial blood pressure when the rupture was detected.

All procedures were completed in 1 session and resulted in control of the leakage and complete obliteration of the aneurysm. After identifying IPR on the control angiogram, immediate heparin reversal by intravenous injection of prothrombin sulfate was performed in all cases. To control the leakage and achieve complete obliteration of the aneurysm, the coil packing was continued under close inspection in 8 patients. In some cases, balloon-assisted or stent-assisted techniques were performed. In 2 cases (cases 5 and 6), no additional endovascular procedure was performed, as a repeat angiogram obtained within several minutes showed no evidence of leakage, and the operator considered that spontaneous obliteration had been achieved. The mean time between the detection of the rupture and determination of the cessation of contrast leakage on the control angiogram was 16.1 minutes (range, 4–25 minutes). Postprocedural external ventricular drainage was required for the management of hydrocephalus and intraventricular hemorrhage that developed secondary to IPR in 2 cases.

The mean follow-up period was 22 months (range, 1–47 months), with only 3 patients (30%) having permanent neurologic deficits (modified Rankin Scale scores 3 and 2). Two of these patients had worsened neurologically after treatment: 1 patient with multiple infarctions and posthemorrhagic hydrocephalus developed hemiparesis (case 2), and 1 patient with posthemorrhagic hydrocephalus experienced mild cognitive dysfunction (case 7). The condition of another patient who had initially presented with visual loss secondary to a recurrent large ophthalmic artery aneurysm was the same at follow-up (case 5).

DISCUSSION

Incidence of IPR

The incidence of IPR during coil embolization varies across studies and has been estimated to be between 2% and 5%.[2,7-12] In the present study, the incidence of IPR was relatively low (1.18%). We believe this to be due to 3 factors. First, all the cases were unruptured aneurysms. In other studies, authors included IPR during embolization of both ruptured and unruptured aneurysms. In a meta-analysis, Cloft and Kallmes[3] calculated the occurrence percentage of aneurysmal IPRs and concluded that the incidence of IPR is higher in previously ruptured aneurysms than in unruptured ones (4.1% vs. 0.7%). They suggested that the mechanism of IPR of unruptured aneurysms was different from the mechanism of IPR of ruptured aneurysms. Unruptured aneurysms may necessitate de novo creation of a rent in the aneurysm wall, whereas IPR could occur without this requirement in the latter condition.[3] Second, there are continuous developments in coil embolization techniques and devices. One of the leading causes of IPR is the stiffness of the devices, which contributes to perforation of the aneurysmal wall. Soft and flexible coils and catheters could reduce IPR.[2,13] Third,