Rupture during Treatment of Recently Ruptured Aneurysms with Guglielmi Electrodetachable Coils

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BACKGROUND AND PURPOSE: We describe four cases of aneurysmal rupture during embolization with Guglielmi electrodetachable coils (GDCs) in an attempt to identify those aneurysms whose rupture during embolization represents a life-threatening risk; our emphasis is on emergency management, in particular, ventriculostomy.

METHODS: Medical records were reviewed retrospectively for 91 aneurysms treated with GDCs 0 to 21 days after subarachnoid hemorrhage. Rupture was ascertained by the presence of extravascular effusion of contrast medium.

RESULTS: Of the perforated aneurysms, two involved the anterior communicating artery, one the posterior inferior communicating artery, and one the basilar artery. Only two patients, whose aneurysms were located in the posterior fossa, had major complications (arterial hyperpressure, mydriasis, angiographically documented circulatory arrest or slowing). One of these patients died and the other improved after emergency ventriculostomy.

CONCLUSION: Aneurysmal perforation during embolization may be accompanied by severe intracranial hypertension, which causes either a decrease or arrest of cerebral perfusion, the duration of which determines clinical outcome. Emergency ventriculostomy (which should be performed in the angiographic suite) is an effective means to reduce intracranial pressure. Recognition of aneurysms associated with a high risk of mortality by rupture in the course of embolization (recently ruptured small aneurysms, posterior fossa aneurysms, associated ventricular dilatation, massive cisternal hemorrhage) and use of proper logistics should ensure the effective management of this devastating complication.

Perforation of an intracranial aneurysm that has bled is a potentially fatal complication of embolization. Four such cases occurred in our institution during treatment with Guglielmi electrodetachable coils (GDCs) between January 1994 and September 1997. Two patients had no serious consequences; one patient died, and the fourth was in critical condition until emergency ventricular derivation was performed.

Major subarachnoid hemorrhage (SAH) may complicate rupture and be responsible for severe intracranial hypertension. In such cases, emergency ventriculostomy is effective in rapidly reducing intracranial pressure, thereby restoring brain perfusion.

Methods

Between January 1994 and September 1997, 91 recently ruptured intracranial aneurysms were treated in our institution with the use of GDCs. Treatment was performed 0 to 21 days after SAH. In 68 cases (75%), embolization was done between 0 and 4 days, with the patient under general anesthesia. The microcatheter used was either a Tracker 10 or, more often, a Tracker 18; the microguidewire used was either a Dasher 10 or 14 (Target, Louvres Cédex, France), depending on the size of the microcatheter, or else a Transcent EX 14 guidewire (Boston Scientific, St Quentin, France). In three cases, a balloon catheter (Solstice, Micro Interventional System, Malmaison, France) was positioned across the neck of the aneurysm to stabilize the placement of coils. Clinical and technical data concerning the ruptured aneurysm were reviewed from medical records and are summarized in Table 1.

Aneurysmal size (summarized in Table 2) was determined by the largest diameter from the neck to the dome of the cavity. Measurements were performed during embolization directly on the films after calibration using a 20-mm-diameter coin placed on the part of the skull nearest the amplifier.

TABLE 1: Clinical, angiographic, and therapeutic options, plus results and follow-up in four patients with aneurysmal rupture

	Case			
	1	2	3	4
Age (y)/Sex	46/M	63/M	44/M	49/F
Location/size (mm)	ACoA/3	ACoA/3	PICA/3	Basilar artery
WFNS grade	IV	I	II	I
Heparin therapy	Yes	Yes	Yes	Yes
Hydrocephalus	CSF drainage	No	No	Yes, moderate ventricular enlargement; no previous CSF drainage
Time (d) to embolization/SAH	4	1	8	2
Arterial pressure after aneurysmal rupture	160/90	190/90	270/190	200/100
Mydriasis	No	No	Yes	Yes
Cerebral circulation (angiography)	Unchanged	Slowed	Arrested	Arrested
Mechanism of rupture	First coil*	Tip of microcatheter	First coil	First coil
First coil size†	[2/8]10	[3/12]10	[3/12]10	[4/10]10
Treatment‡	Protamine sulfate injection	Protamine sulfate	Protamine sulfate CSF drainage, effective 45 min after rupture	Protamine sulfate CSF drainage performed on angiographic table
Outcome	Good	Good	Coma>>>Death	Good
Follow-up	1	2	5	1

Note.—ACoA indicates anterior communicating artery; PICA, posterior inferior communicating artery.

* The first coil filling partly the daughter cavity is supposed to be responsible for the rupture.

† [Coil diameter (mm)/coil length (cm)] coil caliber (inch ×1000).

‡ Additional coils were deposited in all four cases.

Diagnosis of rupture was made in four cases upon angiographic visualization of extravascular effusion of contrast medium, regardless of hemodynamic changes (arterial hypertension). In one case, rupture resulted in blood contamination of the externally derived CSF without hemodynamic changes. Arterial pressure values were obtained retrospectively before and after rupture from anesthesiology medical charts.

Results

Four patients (two with an aneurysm of the anterior communicating artery, one with an aneurysm of the posterior inferior communicating artery [PICA], and one with a basilar artery aneurysm) suffered aneurysmal rupture during embolization. Perforation occurred in the aneurysms whose largest diameter was 4 mm or less (4/29). One such aneurysm had a double pouch with a daughter cavity in which the first coil was partially deployed. Three perforations occurred in aneurysms treated between 0 and 4 days after SAH (in case 3, embolization was not performed until the eight day owing to a previous surgical attempt). In case 4, a basilar artery aneurysm with a large neck (3 mm) relative to the diameter of the

TABLE 2: Aneurysmal size

Size	No. (%)
4 mm or less	29 (32)
5–6 mm	31 (34)
7–10 mm	19 (21)
More than 10 mm	12 (13)
Total	91 (100)

cavity (4 mm), led to the placement of a balloon catheter within the basilar artery to maintain the coils in place.

In three cases, perforation was caused by pressure applied to the aneurysmal wall by the loops formed during deposition of the first coil. In another case, the advancing catheter perforated the aneurysm during withdrawal of the guidewire. The hemodynamic consequences of perforation are expressed clinically in varying degrees of severity. In three patients, perforation was accompanied by a severe increase in arterial pressure; two of these aneurysms were located in the posterior circulation (cases 3 and 4; Figs 1 and 2). In addition to arterial hypertension, cerebral circulatory arrest in association with mydriasis was observed. In the fourth patient (case 1), an external ventricular catheter was positioned before embolization and opened to maintain the level of CSF drainage at 20 cm. Perforation in this patient did not result in any hemodynamic changes.

Discussion

In our institution, introduction of GDC embolization has dramatically changed the management of recently ruptured aneurysms, making treatment less invasive. However, aneurysmal rupture may occur during the endovascular approach that could represent a devastating complication. It is our opinion that any interventional neuroradiologist treating aneurysms during the acute phase of SAH may be confronted with this complication, whatever his skill level and regardless of foreseeable technical improve-
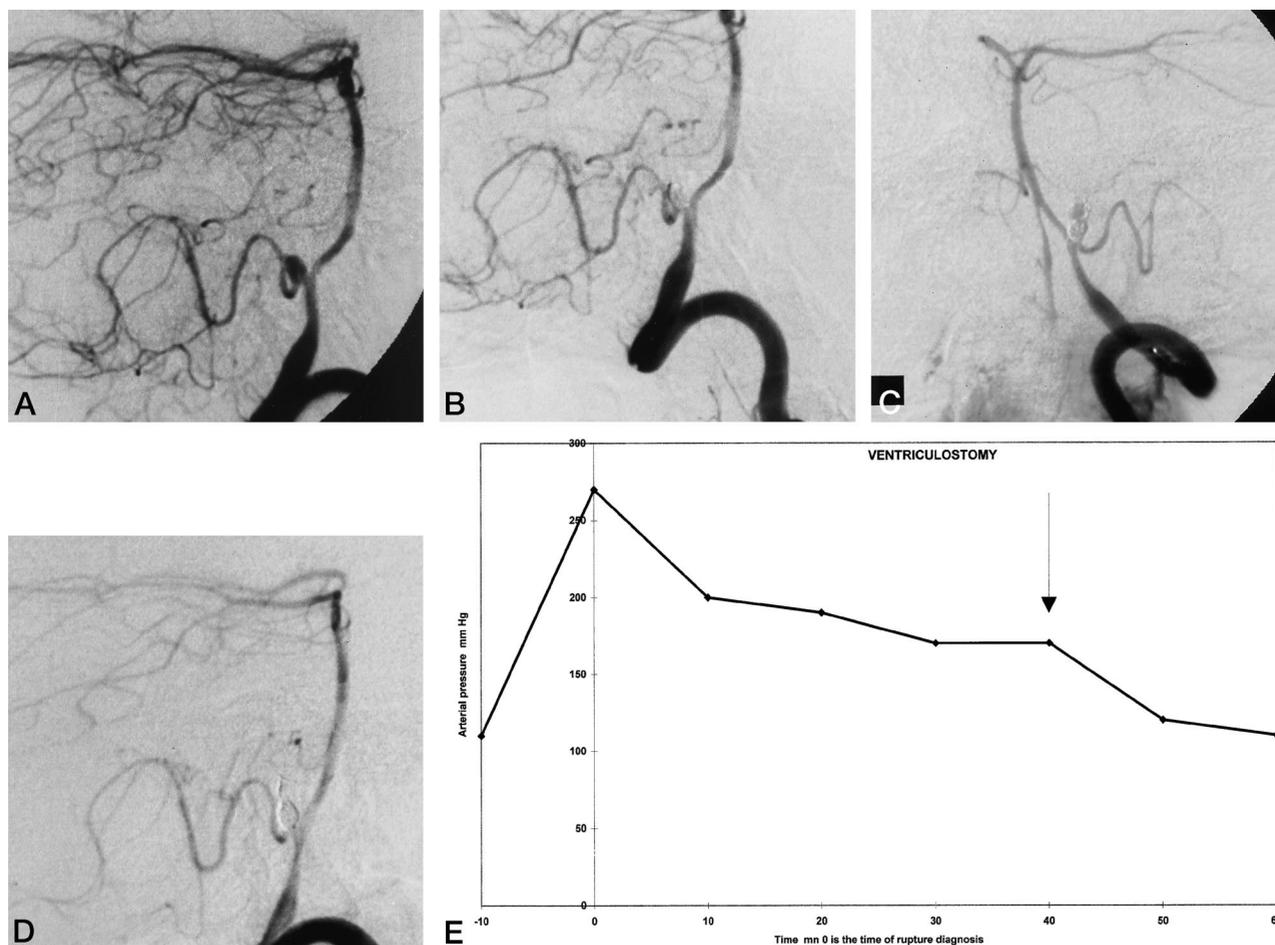
FIG 1.   Case 3: 44-year-old man admitted in WFNS grade I after rupture of a left PICA aneurysm. Surgical attempt was unsuccessful. Embolization was done 8 days after SAH via the femoral route. During coil detachment, a sudden rise in arterial pressure was observed. Extravasation of contrast medium as well as slowing and arrest of cerebral circulation confirmed aneurysmal rupture. Endovascular treatment was continued and resulted in complete occlusion. Ventricular drainage was effective 45 minutes after rupture. Ischemic lesions were detected on follow-up CT scans, and the patient eventually died on day 21.

A, Preembolization left vertebral angiogram, lateral view, shows a 3-mm PICA aneurysm.

B, Predetachment angiogram, lateral view, shows subtotal occlusion of aneurysmal cavity.

C, Immediate postrupture angiogram, left anterior oblique view, shows extravasation of contrast medium, indicating rupture. Note partial outpouching of coil.

D, Postembolization angiogram (after placement of additional coil) shows total occlusion of aneurysmal cavity and outpouched loops seen previously.

E, Evolution of arterial pressure during procedure. Significant decrease of arterial pressure was obtained 45 minutes after diagnosis of aneurysmal rupture.

ments. Our purpose here is to focus on the critical situation of acute intracranial hypertension and to discuss the role played by emergency ventriculostomy in our institution.

The reported percentage of aneurysmal ruptures that complicate embolization of an intracranial aneurysm varies among studies. Vinuela et al (1) reported 11 cases of rupture out of 403 hemorrhagic aneurysms treated in eight different centers, representing 2.7% of the treated cases. Six of the 11 patients with complications died as a result of aneurysmal perforation. According to Valavanis et al (2), reporting their experience with 128 cases of aneurysmal endovascular treatment, the only aneurysms that were perforated were those that had bled previously. In that series, the rate of perforation was 4.2% among the 69 aneurysms revealed by SAH, which is close to that observed in our series (4.4%).

Among our patients, all the perforations occurred in small aneurysms (4-mm diameter or less). In the series reported by Vinuela et al (1), nine of the 11 perforated aneurysms measured between 4 and 10 mm in diameter, and two of the three perforated aneurysms reported by Valavanis et al (2) measured between 3 and 4 mm in diameter. This apparent fragility may have at least two explanations: first, the surface area of the initial rupture, which is obstructed by clot during hemorrhage, is proportionally greater for small aneurysms than for large aneurysms; and second, coils that measure 2 to 3 mm in diameter have a high degree of shape memory and may therefore have a tendency to cause damage to the weakened area of initial rupture. Aneurysmal wall perforations are most commonly caused by introduction of the microcatheter or the first coil. Aneurysmal rupture, however, may occur later in the course of emboliza-
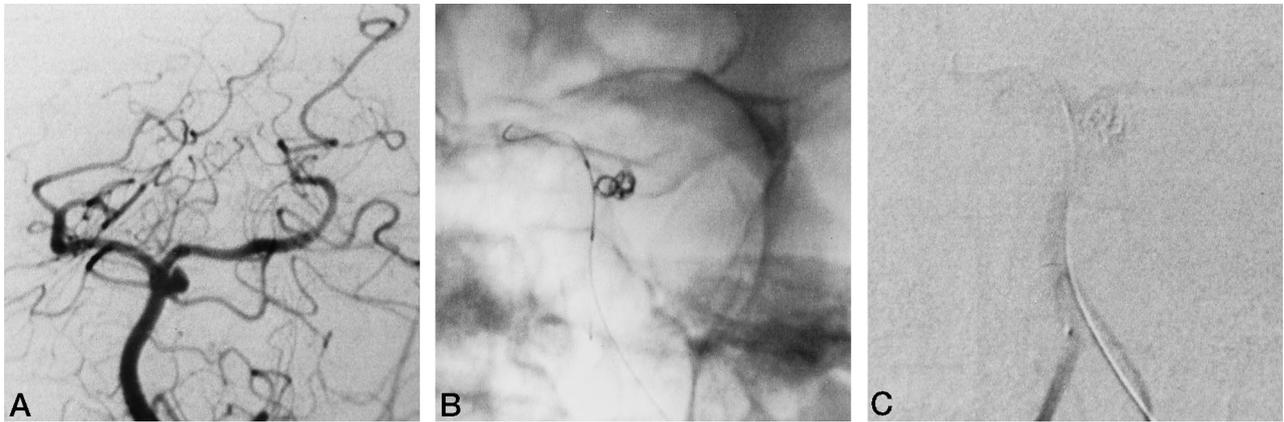
FIG 2. Case 4: 49-year-old woman in WFNS grade I underwent embolization 2 days after aneurysmal rupture.

A, Preembolization angiogram, left vertebral artery injection, shows a 4-mm saccular aneurysm with a 3-mm neck of the left lateral surface of the basilar artery between the superior cerebellar artery and the posterior cerebral artery.

B, Owing to the relatively large neck, a 3-mm-diameter balloon catheter was positioned via the right vertebral artery within the lumen of the basilar artery in front of the aneurysmal neck to allow stabilization of coils within the aneurysm. Catheter tip has already been positioned via the left vertebral artery in the center of the aneurysmal cavity. Outpouching of part of the coil is seen outside the aneurysmal cavity.

C, Angiogram shows rupture during temporary deflation of the balloon.

D, Nonsubtracted frame at end of embolization before balloon deflation shows dense mesh of coils within aneurysmal cavity.

E, Postembolization left vertebral angiogram shows lack of opacification of aneurysmal cavity, partial stenosis of basilar artery at the level of the aneurysm, and partial occlusion of superior cerebellar artery.

F, Arterial pressure curve: ventriculostomy was effective 15 minutes after diagnosis of rupture. Arterial pressure decrease was obtained soon after CSF derivation.
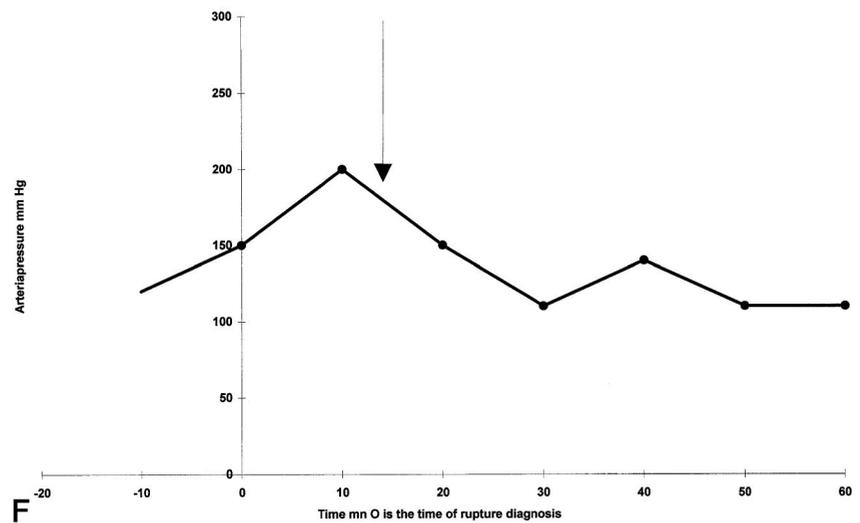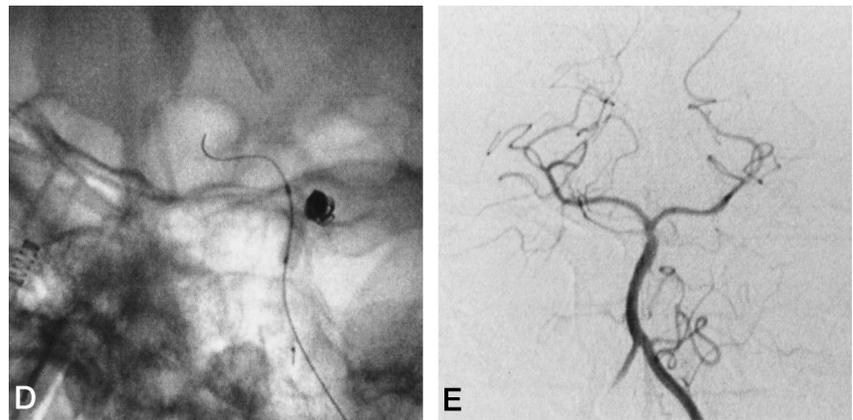
tion, as reported by Vinuela et al (1) in a case in which rupture occurred during introduction of the fifth coil. The proposed mechanism for such rupture is a weakening of the obstructive clot at the aneurysmal dome caused by the cage of coils. These authors believe that the risk of rupture justifies deferred packing of the aneurysmal neck at a later date. In our series, the characteristics of maximal aneurysmal fragility included recent rupture, small aneurysmal pouch, and presence of a daughter aneurysm.

Consequences of perforation may be minimal, and, in such cases, management simply involves continuing embolization and reversing heparin therapy by using protamine sulfate. Conversely, consequences of rupture may be dramatic, as they were in two of our patients who experienced cerebral circulatory arrest as a complication of aneurysmal perforation, with onset of bilateral mydriasis. The potential gravity of this complication has led us to analyze the pathophysiological data concerning aneurysmal perforation in the closed cranial vault and to try to define the type of emergency management that should be implemented during an acute increase of intracranial pressure caused by rehemorrhage.

The principal findings on the pathophysiology of events following SAH in humans have been provided

by the work of Grote and Hassler (3); in animal models, much of the data derive from the work of Mac Cormick et al (4), Brinker et al (5), Trojanowski (6), and Dorsch et al (7). Less than 1 minute after aneurysmal rupture, there is an acute increase in intracranial pressure, which reaches the level of the mean arterial pressure. This increase results in a major reduction in, or even an arrest of, cerebral blood flow, despite the reflex mechanism, leading to a secondary increase in systemic arterial pressure (Cushing reflex). This circulatory arrest (which occurs immediately, while the CSF surrounding the aneurysm is progressively replaced by clot) is one of the principal mechanisms of hemostasis and is associated with a decrease in or disappearance of the transparietal constraints applied to the rupture zone.

Although increased intracranial pressure is initially a protective mechanism, which contributes to the halting of SAH, its persistence is, on the other hand, deleterious in that it decreases or nullifies cerebral perfusion pressure and is rapidly complicated by ischemia. Normes (8), reporting on the results of a surgical series, clearly showed that clinical outcome depends on how quickly intracranial pressure returns to normal. Under the usual conditions of embolization, in the absence of direct intracranial pressure monitoring, the degree of intracranial hypertension is reflected by persistent circulatory arrest at angiography, mydriasis, and the rise in systemic arterial pressure. Management of aneurysmal perforation in the course of embolization includes continued coiling and the use of antagonists of heparin therapy through intravenous injection of protamine sulfate. Such measures may be sufficient to stop vascular effusion and to restore normal cerebral circulation. If these critical conditions persist, intracranial pressure must be decreased as quickly as possible. This can be achieved by rapid ventriculostomy. The effect on prognosis of a rapid return of intracranial pressure to normal values is suggested in three of our cases.

In case 1, in which external ventricular derivation was performed before treatment, systemic hemodynamic parameters remained stable and no neurovegetative signs were seen during aneurysmal rupture.

In case 3, the increase in systemic arterial pressure and mydriasis persisted until ventricular derivation was performed, which unfortunately was not possible until 45 minutes after rupture. The patient subsequently died of diffuse cerebral ischemia.

In case 4, the most recent case, in which the material conditions for quick ventricular derivation were united, we observed the same phenomenon (arterial hypertension and mydriasis) until derivation of ventricular fluid could be performed 15 minutes after aneurysmal rupture. Clinical outcome was favorable.

Several questions are worth considering: What types of aneurysms constitute a vital risk during rupture in the course of endovascular treatment? Can previously performed ventricular derivation be justified in some cases? and How can emergency ventricular derivation be performed in the course of severe aneurysmal rupture?

Which aneurysms are at risk of major intracranial hypertension during endovascular treatment? Our data as well as that of others concerning fatal ruptures (2, 9) show that aneurysms located in the posterior fossa and particularly those situated in the PICA have led to death through acute intracranial hypertension caused by aneurysmal rupture in the course of endovascular treatment. These findings are similar to those concerning the prognosis for aneurysms of the vertebrobasilar system, which is poorer than that for aneurysms of the anterior circulation (10). When perforation occurs during embolization of a posterior fossa aneurysm during the period of cisternal clotting (often associated with hydrocephalus), rebleeding complicates an already critical situation.

In which cases should preventive external ventricular derivation be considered? Previous external derivation of CSF was performed in one of our patients whose clinical outcome was favorable. CSF derivation following SAH may be indicated in case of acute hydrocephalus and is part of the normal management of patients in clinical grades IV and V of the WFNS (11). It constitutes an effective method for warding off intracranial hypertension, except in those cases in which severe clotting has occurred in the lateral ventricles. It has, however, two major drawbacks: the possibility of infection and a higher rate of rebleeding (12). Therefore, while its use is fully justified in cases of intracranial hypertension, it seems unreasonable to use this technique preventively, considering the possibility of complications and the small number of severe aneurysmal perforations. For small aneurysms located in the posterior fossa, we perform embolization as early as possible after ventriculostomy. When severe aneurysmal perforation occurs in the absence of ventriculostomy, the prognosis for clinical outcome worsens with each passing minute. In such cases, emergency CSF derivation must be performed immediately.

In our institution, and regardless of the diligence of the surgical team, transporting the patient to the operating room considerably delays CSF evacuation and the management of intracranial hypertension. After our experience with the patient described in case 3, we decided that external ventricular derivation should be performed in the angiographic suite (which was done for case 4 with a considerable time gain). Performing such a procedure rapidly and under safe conditions in a life-threatening context requires both managerial and logistic planning, including establishing easy access to the patient's head, with proper lighting; preparing for immediate trephination; having ventriculostomy materials available and ready for use in the angiographic suite; and making sure a neurosurgeon and a surgical aide are alerted and on stand-by. Catheters should be safely positioned so as to enable angiographic verification and to continue embolization once hemodynamic parameters have returned to normal after the ventriculostomy.

Considering that confirmation of rupture by CT does not constitute an emergency, we suggest that the

patient not be transported to the CT scanner until resuscitation is achieved.

Conclusion

Aneurysmal perforation in the course of embolization is a rare event. The clinical gravity is variable and can be limited to the demonstration of leakage of contrast medium within the subarachnoid spaces. Conversely, it may be complicated by severe intracranial hypertension. This is particularly true for small aneurysms located in the posterior fossa. In such cases, emergency ventriculostomy is effective in managing intracranial hypertension. Further data based on retrospective analyses of cases may allow a better assessment of the clinical spectrum of this complication, of the risk-related anatomy and location, and of the results of therapeutic techniques applied.

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