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Rupture during Treatment of Recently Ruptured Aneurysms with Guglielmi Electrodetachable Coils

Frédéric Ricolfi, Caroline Le Guerinel, Jerry Blustajn, Catherine Combes, Pierre Brugieres, Eliane Melon, André Gaston

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 hypertension, 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.
Diagnosis of rupture was made in four cases upon angio-
graphic visualization of extravascular effusion of contrast me-
dium, regardless of hemodynamic changes (arterial hyperten-
sion). 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 ante-
rior 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 oc-
curred 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 at-
ttempt). In case 4, a basilar artery aneurysm with a
large neck (3 mm) relative to the diameter of the
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 con-
sequences of perforation are expressed clinically in
varying degrees of severity. In three patients, perfo-
ration was accompanied by a severe increase in arte-
rial 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 circu-
latory 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 emboliza-
tion has dramatically changed the management of
recently ruptured aneurysms, making treatment less
invasive. However, aneurysmal rupture may occur
during the endovascular approach that could repre-
sent a devastating complication. It is our opinion that
any interventional neuroradiologist treating aneu-
rysms during the acute phase of SAH may be con-
fronted with this complication, whatever his skill level
and regardless of foreseeable technical improve-

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

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

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.

**TABLE 2: Aneurysmal size**

<table>
<thead>
<tr>
<th>Size</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 mm or less</td>
<td>29 (32)</td>
</tr>
<tr>
<td>5–6 mm</td>
<td>31 (34)</td>
</tr>
<tr>
<td>7–10 mm</td>
<td>19 (21)</td>
</tr>
<tr>
<td>More than 10 mm</td>
<td>12 (13)</td>
</tr>
<tr>
<td>Total</td>
<td>91 (100)</td>
</tr>
</tbody>
</table>
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-
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 clotted (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.

**References**