CLINICAL PICTURE: A 40 years old male patient found in deep coma on the toilet in the bathroom.

RADIOLOGICAL FINDINGS:

Figure 1. Anterior cerebral artery aneurysm. Precontrast CT scan showing interfrontal subarachnoid hemorrhage, breaking into the third and lateral ventricles, with a CSF- blood levels in the occipital horns of the lateral ventricle. The hematoma is seen dissecting upward into the septum pellucidum and distending the potential cavum between the layers of the septum pellucidum. Notice the bilateral anterior cerebral artery medial frontal infarctions more on the right side, mostly due to the associated vasospasm. Notice marked effacement of the sulci and gyri, mostly due to diffuse vasogenic cerebral edema.
**Figure 2.** Anterior cerebral artery aneurysm. Notice the interfrontal location of cerebral hemorrhage, The associated callosal hematoma, the hydrocephalic changes, the septal hematoma, the diffuse brain edema and the bilateral medial frontal anterior communicating artery infarctions.

**Figure 3.** A, ruptured anterior communicating aneurysm inducing interfrontal and intraventricular haematoma, B, ruptured anterior communicating aneurysm inducing callosal haematoma. Notice the bilateral medial frontal anterior cerebral artery infarction, mostly due to vasospasm and the CSF-blood level in the occipital horns of the lateral ventricle.

**Figure 4.** Ruptured anterior communicating aneurysm inducing interfrontal and intraventricular haematoma. Notice the bilateral medial frontal anterior cerebral artery infarction, mostly due to vasospasm, and the septal hematoma.

**DIAGNOSIS:**

**DIAGNOSIS:** RUPTURED ANTERIOR COMMUNICATING ARTERY ANEURYSM WITH SUBARACHNOID HEMORRHAGE AND BILATERAL ANTERIOR COMMUNICATION ARTERY INFARCTIONS

**DISCUSSION**

- Complications of cerebral aneurysms
The volume of blood that extravasates when an intracranial aneurysm ruptures is relatively small in comparison with the hemorrhages from aneurysms in other parts of the body. The acute appearance of blood in the subarachnoid space is not, in itself, a threat to life. On the other hand, death may rapidly ensue when there is trauma to important centers in the brain by a rapidly dissecting hemorrhage. Complications more often occur later owing to the secondary compression and displacement effects of a hematoma, the prolonged occurrence of cerebral ischemia leading to infarction or the development of both hematoma and infarction. Infarction, through the development of associated edema, may produce a mass effect which at times can induces herniation.

**Hematoma**

The rupture of approximately two-thirds of cerebral aneurysms results in the formation of hematomas which may be large or small. In less than one-half of patients in whom an intracerebral hematoma develops, the lesion is caused by a direct dissection of blood into the cerebral substance from the ruptured aneurysm. In the majority of the cases there is first an extravasation into the subarachnoid space with subsequent or indirect dissection into the cerebral substance. In a small number of patients (2%) the rupture of an aneurysm may be accompanied by tearing of the arachnoid, with direct hemorrhage into the subdural space or dissection of subarachnoid blood between the arachnoid and dura mater. Many patients with ruptured aneurysms have hematomas that remain confined to the subarachnoid space, where they produce the effects of a localized extracerebral mass.

![Image](image1.png)

**Figure 5. Massive subarachnoid hemorrhage**

Certain patterns of subarachnoid hematoma formation and of intracerebral dissection are found with aneurysms in specific locations. These patterns have been worked out pathologically and can be recognized by angiography, as described below. However, it is now more satisfactory to diagnose and follow intracerebral hematomas by the atraumatic technique of computerized tomography, once the cause of bleeding has been established.

![Image](image2.png)

**Figure 6. Ruptured aneurysm with subarachnoid hemorrhage.**

Aneurysms of the anterior communicating artery are not only responsible for subarachnoid hemorrhage more often than lesions at any other single site, but the rupture results in an intracerebral hematoma more often than with lesions at other sites. Inferior frontal or olfactory hemorrhages may dissect upward, and break into a frontal horn of the ventricular system.
Figure 7. Subarachnoid hemorrhage

At other times an anterior communicating artery aneurysm may rupture into the subarachnoid space between the medial surfaces of the frontal lobes and form an interfrontal subarachnoid hematoma. Such hematomas may dissect upward into the septum pellucidum, often distending the potential cavi

The anterior cerebral artery aneurysms are the most likely to rupture

Anterior communicating artery aneurysms are also the most common lesions to produce injury of the hypothalamus. Less frequently, extension of an interfrontal hematoma may pass around the corpus callosum to form a hematoma in the callosal sulcus or in the intercingulate region. Dissection into the corpus callosum itself may take place and occasionally an intracerebral hematoma developing primarily in the frontal lobe, or extending into it from the subarachnoid space, may dissect laterally into the external capsule. Aneurysms of an anterior cerebral artery distal to the circle of Willis produce hematomas in the proximity of the lesion.

Figure 8. Anterior communicating artery aneurysm hemorrhage. Axial CT scan shows typical subarachnoid hemorrhage from rupture of anterior communicating artery aneurysm. Hemorrhage is present in the septal region (arrows). B, Ruptured anterior communicating aneurysm causing intraventricular and interfrontal haemorrhage

The more proximal anterior cerebral aneurysms most often rupture into the frontal lobe substance, whereas the more peripheral aneurysms produce hematomas in the callosal sulcus or in the intercingulate fissure.

Aneurysms extending upward and forward from the bifurcation of the internal carotid artery often are imbedded in the frontal lobe and rupture directly into its substance; thence the hematoma may burst into a frontal horn of a lateral ventricle. Aneurysms at the bifurcation that extend backward and upward may rupture into the hypothalamic nuclei or through the lamina terminalis into the third ventricle.
Aneurysms of the cerebral segment of the internal carotid artery, which usually arise at the site of origin of the posterior communicating artery and extend backward, most often rupture into the anterior temporal lobe substance, thence into the temporal horn. At other times, a subarachnoid hematoma may develop above the uncus and dissect along the choroidal fissure into a temporal horn. The hemorrhage may also extend beneath the uncus. Bleeding from the fundus of a forward pointing aneurysm may result in a subarachnoid collection beneath the frontal lobes.

Middle cerebral artery aneurysms often result in the formation of a hematoma deep in the Sylvian fissure over the central lobe or island of Reil. Such Sylvian hematomas then may dissect into the external capsule There also may be direct rupture into the external capsule or into the frontal or temporal lobe. A hematoma may dissect forward from the external capsule or from the frontal horn of a lateral ventricle. At other times there may be dissection backward from the external capsule or temporal lobe with rupture into the atrium of a lateral ventricle.

Correlating well with the pathologic changes described above, the frontal carotid angiogram may display following features denoting hematoma formation from rupture of an anterior communicating artery aneurysm, an aneurysm of the carotid bifurcation, or an aneurysm of the ending portion of the anterior cerebral artery: (a) elevation of the proximal transverse limb of an anterior cerebral artery when a hematoma occurs in the subfrontal region; (b) lateral bowing of the proximal ending portions of the anterior cerebral arteries caused by a frontal intracerebral hematoma (in the case of anterior communicating artery aneurysms that point toward the opposite hemisphere from which they arise, there may be cross-frontal rupture and ipsilateral bowing of the anterior cerebral arteries); (c) widening of the space between the anterior cerebral arteries owing to separation by an interfrontal hematoma in the subarachnoid space.

The corresponding lateral angiogram may reveal, as a result of rupture of an aneurysm of the anterior communicating artery, a lesion of the carotid bifurcation or a forward pointing aneurysm of the supraclinoid portion of the internal carotid artery, (a) backward bowing of the cerebral segment of the internal carotid artery or "closure" of the carotid siphon caused by a subfrontal or inferior intrafrontal hematoma; (b) elevation of the forward extending segment of the anterior cerebral artery, corresponding to the change shown in the proximal transverse limb in the frontal view; (c) depression of the pericallosal artery if there has been dissection of a subarachnoid hematoma over the corpus callosum into the callosal sulcus or intercingulate area. If an aneurysm situated distally on an anterior cerebral artery ruptures into the corpus callosum, or if a subarachnoid hematoma dissect into this structure, the arc of the pericallosal artery may be widened or there may be a localized elevation of this vessel. Differentiation from the wide anterior cerebral sweep of hydrocephalus may be made if there is a normally inclined thalamostriate vein. If there is a subarachnoid hematoma between the corpus callosum and cingulate gyri or in the intercingulate fissure, the pericallosal arteries may be depressed.

Aneurysms of the internal carotid artery that project and rupture backward and ruptured aneurysms at the division of the middle cerebral artery may exhibit in the frontal angiogram, as a result of hematoma formation: (a) widening of the angle formed by the supraclinoid portion of the carotid artery and the proximal portion of the middle cerebral artery due to the presence of a subtemporal or anterior Sylvian hematoma; (b) displacement of the anterior choroidal artery by a hematoma lateral or medial to this structure or as a result of tentorial herniation; (c)
a deformity of the orderly columnar arrangement of the middle cerebral branch loops over the island of Reil if a subarachnoid hematoma of the Sylvian fissure is present or if there is a hematoma in the external capsule. Of particular importance is the effect of a subarachnoid hematoma on the medial or lateral lenticulostriate arteries, or the anterior perforating ganglionic arteries. Such hematomas produce obliteration of the perforating arteries at the point where they pass through the pia with the result that their subarachnoid course is seen for a few millimeters in the angiogram, followed by an abrupt termination of the shadows, often along an almost straight line.

In the lateral angiogram internal carotid artery aneurysms that rupture backward and bleeding lesions of the middle cerebral bifurcation that produce hematomas exhibit a loss of undulation, flattening, straightening, or bowing of the anterior choroidal artery, depending upon the exact location of the hematoma in relation to the vessel. When a subuncal hematoma is present, the anterior choroidal artery is elevated. A Sylvian hematoma will produce deformity of the orderly triangular arrangement of the middle cerebral arterial branches over the island of Reil. The looped arrangement is obliterated with straightening, stretching, and separation of the middle cerebral branches, if the hematoma is anterior. Rostral displacement and crowding of the branches may be shown if a posterior Sylvian hematoma is present. The deformity of the Sylvian triangle is accentuated by the presence of a hematoma in the external capsule.

Figure 11. A CT scan revealing a right frontotemporal hematoma secondary to a right MCA aneurysm rupture.

Figure 12. A CT scan revealing interhemispheric and bilateral (right–left) inferior frontal lobe hematomas secondary to an ACoA aneurysm rupture.
Aneurysms at the rostral end of the basilar artery may rupture directly into the third ventricle, the lesions often being imbedded in the structures forming the posterior portion of the ventricular floor. At other times a hematoma may form in the cisterna interpeduncularis. Such a subarachnoid hematoma may then dissect caudal into the midbrain and pons, following the course of perforating branches of the basilar and posterior cerebral arteries. A posterior inferior cerebellar aneurysm may produce a hemorrhage in the brainstem and in the subarachnoid cisterns. Some posterior inferior cerebellar aneurysms are peripheral in position and produce a cerebellar hematoma in the proximity of the lesion.

- **Site of Ruptured Aneurysm**

The most frequent site of aneurysm rupture in patients with an associated hematoma was the MCA (38% of patients) followed closely by the ACoA (36% of patients). The incidence of a hematoma was higher in patients with MCA (56%) and distal ACA aneurysms (50%), and it was lower in patients with ICA (24%) and PCoA aneurysms (15%). Intracerebral hematomas were very rare in patients with VBA aneurysms (1%).

- **Relationship Between Aneurysm Site and Hematoma Location**

The types of ICH can classified into seven groups according to CT findings on admission: 1) frontal; 2) temporal, 3) sylvian; 4) basal ganglia; 5) interhemispheric; 6) callosal; and 7) cerebellar.

The location of the ICH is related to the location of the ruptured aneurysm. In patients with MCA aneurysms, the sylvian fissure was by far the most common location for hematoma occurrence. Frontal and temporal lobe hematomas also occurred in patients with MCA aneurysms (Fig. 11). In patients with ACoA aneurysms, frontal lobe hematomas occurred most frequently along with several cases of interhemispheric clots (Fig. 12). Temporal lobe hematomas were most frequent in patients with PCoA aneurysms (Fig 13). In patients with distal ACA aneurysms, ICHs were most commonly frontal and callosal in a typical butterfly-type pattern (Fig. 14). In patients with ICA aneurysms hematomas were most often found in the temporal lobe but were also associated with basal ganglia hematomas (Fig. 15) and one sylvian clot. Only one patient experienced a hematoma (fourth ventricle/cerebellar) that occurred in conjunction with a VBA aneurysm.

It is generally accepted that the second bleeding of an aneurysm is more apt to result in serious complications, or even a terminal event, than is the first rupture. One important reason for the more serious prognosis is the fact that a second rupture usually occurs directly into the brain substance, owing to the fact that the first hemorrhage produces subarachnoid adhesions in the neighborhood of the aneurysm or the development of adhesions binding the sac of the aneurysm to the pia mater. In a high percentage of fatal intracerebral hemorrhages there is bleeding into the ventricular system. Under such circumstances, the ventricles may become rapidly distended with blood, which is evident on CT scan, MRI or in the venous phase of the angiogram, especially if there is impairment of decompression by adhesions about the foramina of the fourth ventricle and in the basal cisterns.

**Figure 13.** A CT scan revealing a left temporal lobe hematoma secondary to rupture of a left PCoA aneurysm rupture.
Table 1. Hemorrhage sites after aneurysmal rupture

<table>
<thead>
<tr>
<th>ARTERY</th>
<th>SITE OF HAEMORRHAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANTERIOR COMMUNICATING ARTERY ANEURYSM</td>
<td>1-Inferior frontal, olfactory hemorrhage that may dissect into the frontal lobes or hypothalamus</td>
</tr>
<tr>
<td></td>
<td>2-Interfrontal hemorrhage that may dissect into septum pellucidum, corpus callosum or intercingulate area</td>
</tr>
<tr>
<td>INTERNAL CAROTID ANEURYSM</td>
<td>Frontal lobe hemorrhage.</td>
</tr>
<tr>
<td>POSTERIOR COMMUNICATING ARTERY ANEURYSM</td>
<td>Anterior temporal lobe hemorrhage that may dissect into temporal horns</td>
</tr>
<tr>
<td>MCA ANEURYSM</td>
<td>Sylvian fissure haematoma that may dissect into the external capsule</td>
</tr>
<tr>
<td>BASILAR TIP ANEURYSM</td>
<td>Commonly rupture into the third ventricle, pons, midbrain or cisterna interpeduncularis</td>
</tr>
<tr>
<td>PICA ANEURYSM</td>
<td>Brain stem or cerebellar haematoma</td>
</tr>
</tbody>
</table>

Figure 14. A CT scan revealing a butterfly-pattern interhemispheric hematoma secondary to rupture of a distal ACA aneurysm

Figure 15. A CT scan revealing a temporal lobe/basal ganglia hematoma (with ventricular extension) secondary to an ICA aneurysm rupture.
Cerebral infarction is a more common fatal complication of the rupture of an intracranial aneurysm than intracerebral hematoma formation associated with ruptured aneurysms. It is commonly pale and ischemic, not hemorrhagic in type. Cerebral infarction occurs most often after the rupture of aneurysms (1) of the internal carotid artery where the posterior communicating vessel originates, (2) of the middle cerebral artery, (3) of the anterior communicating artery, and in that order of frequency. The order is just the reverse of that found for intracerebral hematomas. Aneurysms at the origin of the posterior communicating artery produce infarction over a wider area than other aneurysms, probably because they are more proximal on the carotid arterial vascular tree. The infarcted area is most often found in the distribution of the middle cerebral artery, which is the main continuation of the internal carotid. The rupture of aneurysms at the posterior communicating level also produces more ganglionic infarcts than aneurysms at other sites. Bilateral infarction occurs frequently after the rupture of aneurysms of the anterior communicating artery. Such infarction is usually in the cortical distribution of the anterior cerebral arteries and, although the survival rate is relatively high, many patients exhibit mental changes.

Table 2. Infarction sites after aneurysmal rupture

<table>
<thead>
<tr>
<th>Aneurysmal site</th>
<th>Infarction site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior communicating artery aneurysm</td>
<td>1. Aneurysms at the origin of the posterior communicating artery produce infarction over a wider area than other aneurysms, probably because they are more proximal on the carotid arterial vascular tree. The infarcted area is most often found in the distribution of the middle cerebral artery, which is the main continuation of the internal carotid.</td>
</tr>
<tr>
<td></td>
<td>2. The rupture of aneurysms at the posterior communicating level also produces more ganglionic infarcts than aneurysms at other sites.</td>
</tr>
<tr>
<td>Internal carotid artery aneurysm</td>
<td>Massive middle cerebral artery infarction.</td>
</tr>
<tr>
<td>Anterior communication artery aneurysm</td>
<td>Bilateral infarction occurs frequently after the rupture of aneurysms of the anterior communicating artery. Such infarction is usually in the cortical distribution of the anterior cerebral arteries.</td>
</tr>
</tbody>
</table>

There appear to be three overriding angiographic observations that can be made in the presence of cerebral infarction, or in anticipation of an impending infarction. Recognition of these changes may be of the utmost importance in making decisions concerning treatment.

1. **Spasm.** Infarction occurs most often along the distribution of the artery bearing a ruptured aneurysm, and in many cases this is the result of prolonged spasm of the vessel. The possibility of infarction is always suggested when there is severe localized narrowing of the lumen, usually seen in the immediate vicinity of the ruptured aneurysm; it may also occur when there is only mild diffuse spasm.

2. **Atherosclerosis.** The existence of atherosclerosis may be an important contributing factor to the development of infarction as a complication of hemorrhage from an aneurysm. Infarction is more probable if blood flow in a cerebral vessel is reduced by stenosis of a large atheromatous plaque.

3. **Hematoma.** The more common angiographic changes denoting the presence of intracerebral or subarachnoid hematomas are described above. Subarachnoid hematomas apparently produce infarction more often by pressure on vessel while intracerebral hematomas cause early and marked spasm. Although it is possible to differentiate a subarachnoid from an intracerebral hematoma by CT scan or MRI, angiographic clues are often found, such as elevation of the anterior choroidal artery by a subunca hematoma, widening of the space between the anterior cerebral arteries by an interfrontal mass, or the production of a local deformity of the pericallosal artery by a supracallosal collection. The most common subarachnoid hematoma to produce infarction along the arborization of adjacent vessels occurs after rupture of an anterior communicating artery aneurysm. Other observations that can be made from angiograms do not appear to have nearly as great a significance as spasm, atherosclerosis, and hematoma formation.

Anomalies of the circle of Willis, the dominance of one side over the other in blood flow through the circle, and the overall circulation rate are not often of great value in anticipating or predicting the development of infarction. Computerized tomography is very useful in diagnosing cerebral infarction secondary to spasm. The infarction may or may not be associated with angiographic signs of infarction such as vascular occlusion or early filling veins, and...
only spasm may be present; but the computerized tomography scan [CT scan or MRI] demonstrate the typical findings of cerebral infarctions.

**Herniation**

The development of a herniation may be a critical event, regardless of where it occurs. Acute herniations follow most often the rapid development of massive intracerebral, intraventricular or subdural hematomas, or massive infarction associated with significant oedema.

The subfalcine herniations occurring with hematomas and infarction are similar to those commonly seen in association with tumors. The anterior cerebral arteries and their branches and the deep cerebral veins are the structures usually displaced to the greatest extent. In some cases hematomas may not only produce generalized or localized displacement of the internal cerebral and basal veins but may also deform the septal and Sylvian veins. Other cerebral hernias that may be seen in supratentorial tumours are not encountered in connection with rupture of aneurysms of the carotid system. In some instances, however, hematomas that form along the course of the vertebral-basilar system may cause an upward transtentorial herniation or a downward herniation at the foramen magnum.

![Figure 16. Subfalcine herniation (arrows). Subfalcial herniation is displacement of the cingulate gyrus from one hemisphere to the other, under the falx cerebri. Subfalcial herniation can compress the pericallosal arteries, causing an infarct in their distribution.](image)

**Hydrocephalus**

A sizable number of patients who have a subarachnoid hemorrhage develop hydrocephalus. The onset may be acute or gradual. Acute ventricular dilatation occurs when the initial hemorrhage extends directly into the ventricular lumen. Acute enlargement may also develop when an intracerebral hematoma dissects by pressure necrosis through the ventricular wall. Such a gross lesion usually produces coma and, whenever angiography, CT scan or MRI are performed on a seriously ill patient after a subarachnoid hemorrhage, evidence of ventricular enlargement should be sought. In some cases, unilateral dilatation can be observed, at other times the lateral ventricles may both be dilated but asymmetrical, whereas in still other cases symmetrical enlargement may be found. At times, a large third ventricle may be demonstrated.
In most cases the dilatation of the ventricular system develops gradually without dramatic symptoms. The changes often begin slowly after an interval of apparent clinical improvement. Patients who develop only mild ventricular enlargement may remain asymptomatic.

In most instances in which gradual hydrocephalus occurs, it begins within 1 month of the subarachnoid hemorrhage. In the patients who develop symptoms, the manifestations can be rather similar to normal pressure hydrocephalus; although there is great variability in the speed of development, the process is progressive when untreated and slow but steady dilation of the ventricles can be found over a period of time up to 3 years. The most prominent symptom of hydrocephalus is dementia, sometimes accompanied by gait disturbances and a spastic paraparesis. A surprisingly large number with more advanced hydrocephalus have epileptic seizures and develop hypertension when they had been normtensive before the subarachnoid hemorrhage. It is felt that arterial spasm and the occurrence of an intracerebral hematoma as well as the number of hemorrhages contributed significantly to the development of hydrocephalus.

It has been generally believed that the most important mechanism for the development of hydrocephalus is adhesions in the leptomeninges which cause an interference with the extracerebral circulation of cerebrospinal fluid. In some cases there may be arachnoiditis interfering with egress of fluid from the foramina of the fourth ventricle, but changes about these foramina more often result from infection. In some cases the subarachnoid blockage was not at the incisura but in other basal cisterns, the cisterns caudal to the block being irregularly dilated. It would also appear that mechanical blockage of cerebrospinal fluid circulation is not the sole cause of dilatation of the ventricular system but that cellular degenerative changes lead to a wasting of brain tissue because of anoxemic ischemia. Vasospasm is a prominent finding after subarachnoid hemorrhage in more than two-thirds of the patients who develop hydrocephalus. Such spasm may be prolonged and severe for many days, as noted earlier, causing a reduction in cerebral blood flow. Once hydrocephalus has become established it may increase further owing to elongation and stretching of the intracranial vessels reducing blood flow and the mechanical production of periventricular demyelination.

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**SUMMARY**

The term subarachnoid hemorrhage (SAH) refers to extravasation of blood into the subarachnoid space between the pial and arachnoid membranes. SAH comprises half of spontaneous atraumatic intracranial hemorrhages, the other half consist of bleeding that occurs within the brain parenchyma. Intracranial hemorrhage as a whole represents 20% of all strokes.
SAH is a devastating condition with high morbidity and mortality, and, in the United States, it is associated with an annual cost of $1.75 billion. SAH occurs in various clinical contexts, the most common being head trauma. However, the familiar medical use of the term SAH refers to nontraumatic (or spontaneous) hemorrhage, which usually occurs in the setting of a ruptured cerebral aneurysm or arteriovenous malformation (AVM). The scope of this chapter is confined to nontraumatic SAH.

Ancient Greek, Egyptian, and Arabic literature all have references to intracranial aneurysms. The first successful treatment of an intracranial aneurysm was reported in the early 19th century; however, such outcomes did not become routine until the Dandy era and the advent of modern neurosurgical techniques. Dandy performed the first successful clipping of an aneurysm in 1937 [13], using a vascular clip designed by Harvey Cushing. In the following years, advancements in microneurosurgical techniques, including the operating microscope, microsurgical instruments, better anesthesia, and improved management of SAH complications, have led to significant improvements in surgical outcomes.

Endovascular therapy for the treatment of intracranial aneurysms was pioneered in the mid 1970s by Serbinenko at the Moscow Institute of Neurosurgery. This initial approach, which attempted to achieve parent vessel occlusion using latex balloons, was moderately successful in a limited subset of cases. However, it never gained widespread applicability. Other balloon devices, including detachable silicon and latex balloons, subsequently were developed in the United States, Europe, and Japan. The success of balloon embolization has been tempered by the associated complications of deflation and aneurysmal rupture. Arguably, the most significant recent development in endovascular therapy occurred in 1991, when Guglielmi and colleagues at the University of California Los Angeles (UCLA) Medical Center developed the Guglielmi detachable coil system (GDC). [19,20]

The GDC is a radiopaque platinum coil that is delivered through a microcatheter into an aneurysm, which then is detached by electrolysis. GDCs gained approval by the Food and Drug Administration (FDA) in 1995 for treatment of aneurysms that have the potential for high surgical morbidity and mortality. In Europe, GDCs are used as a first-line intervention in lieu of surgical treatment for patients without contraindications to endovascular therapy. Other endovascular techniques under investigation include liquid embolic agents, intravascular laser treatments, and intravascular stents. As endovascular occlusive techniques evolve, it seems likely that they will play a larger role in the management of SAH.

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**Addendum**

- A new version of this PDF file (with a new case) is uploaded in my web site every week (every Saturday and remains available till Friday.)
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- To download the software version of the publication (crow.exe) follow the link: http://neurology.yassermetwally.com/crow.zip
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- For an archive of the previously reported cases go to www.yassermetwally.net, then under pages in the right panel, scroll down and click on the text entry "downloadable case records in PDF format"

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