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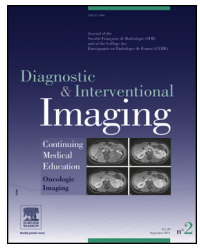

CONTINUING EDUCATION PROGRAM: FOCUS...

Complications and follow up of subarachnoid hemorrhages

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Abstract Complications of subarachnoid hemorrhage are the major life threatening and functional components of the follow up of a ruptured aneurysm. Knowing how to identify these is a key challenge. They vary in type throughout the postoperative follow up period. The aim of this article is firstly to list the main complications of the acute phase (rebleeding, acute hydrocephalus, acute ischemic injury and non-neurological complications), the subacute phase (vasospasm) and the chronic phase of subarachnoid hemorrhages: (chronic hydrocephalus and cognitive disorders) and to describe their major clinical and radiological features. Secondly, we describe the long-term follow up strategy for patients who have suffered a subarachnoid hemorrhage and have been treated endovascularly or by surgery. This follow up involves a combination of clinical consultations, cerebral MRI and at least one review angiogram.

Subarachnoid hemorrhage due to aneurysm rupture occurs in 5 to 10 per 100,000 people annually [1,2], mostly in young patients [3]. It is a serious disease, the complications of which are its major life threatening and functional components and require management in a multidisciplinary strategy. Classically, the clinical course can be divided into three groups: one third recovery, one third recovery of complications and one third fatal. Knowing how to identify these complications is therefore a key challenge. We describe initially the different possible complications following a subarachnoid hemorrhage due to aneurysm rupture and then consider the long-term management plan for patients who have suffered a ruptured aneurysm and have been treated endovascularly or by surgery.

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Complications

The complications of subarachnoid hemorrhage account for the severity of aneurysmal disease and negatively impacts on both survival and functional prognosis. They are classically described by the time they develop compared to the initial aneurysm rupture. Complications are distinguished between the acute phase (between D0 and D3), subacute phase (between D3 and D30) and late phase (after D30).

Acute complications (D0—D3)

Rebleeding

Rebleeding is the most serious acute complication [4,5] (Fig. 1) and generally occurs in the first three days after the initial bleed, with an estimated risk of up to 9 to 17% in the initial hours [3,6]. This is often associated with a poorer prognosis and higher Fisher grade [7] (Table 1). Occlusion of a ruptured intracranial aneurysm is therefore a treatment emergency in the initial 12 to 24 hours in order to reduce this major risk of rebleeding [8]. The ISAT study in 2002 showed coiling to be effective compared to surgery for ruptured aneurysms, and had an estimated risk of dependency and death of 23.7% compared to 30.6% respectively ($P = 0.0019$) [9]. Subarachnoid hemorrhage initially classified as grade 4 of the Fisher classification is a risk factor for rebleeding [10].

Table 1 Modified Fisher classification.

Grade	Criteria
0	No SAH or VH
1	Thin SAH, no VH in the lateral ventricles
2	Thin SAH, VH in the lateral ventricles
3	Thick SAH, no VH in the lateral ventricles
4	Thick SAH, VH in the lateral ventricles

SAH: subarachnoid hemorrhage; VH: ventricular hemorrhage.

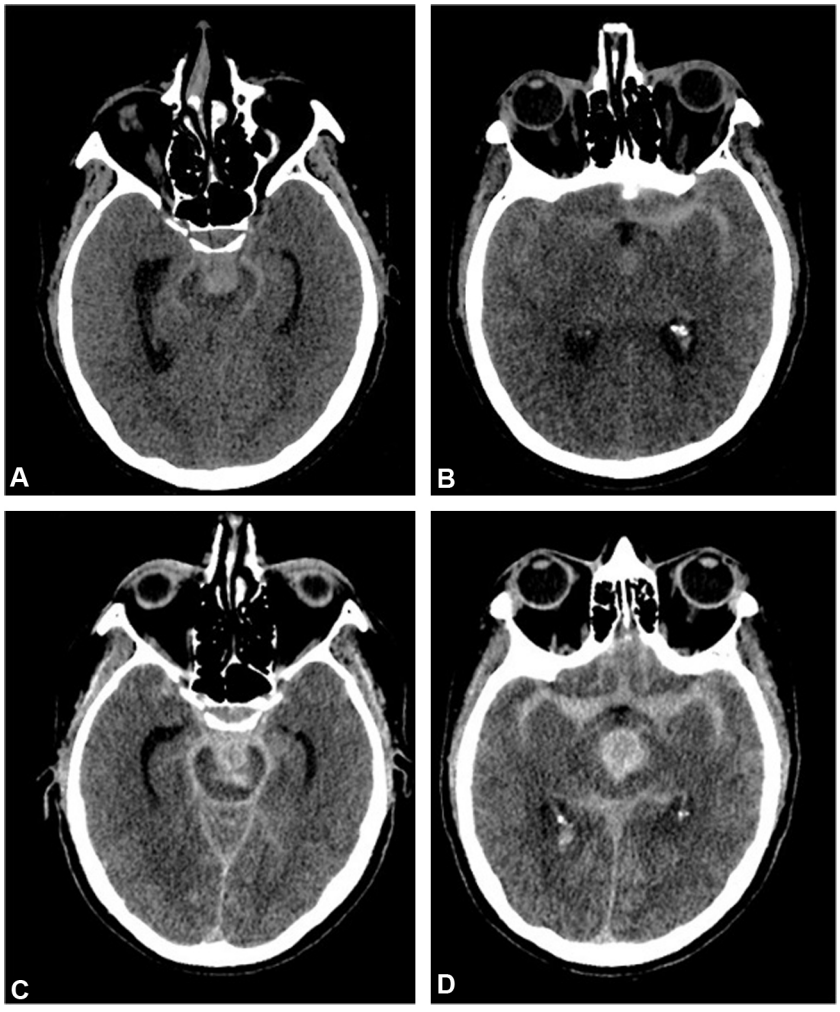

Figure 1. Sixty-year-old female patient who developed sudden-onset headaches which then resolved untreated followed by worsening of her symptoms and development of reduced consciousness; Glasgow score 14. Initial unenhanced CT showing spontaneously hyperattenuating area, predominantly in the perimesencaphlic cisternae (A) and motor cortex sulci (B) suggestive of a Fisher grade 2 subarachnoid hemorrhage due to rupture of a apical basilar trunk aneurysm (not illustrated). Minimal dilatation of the temporal horns (A). Clinical deterioration at H1 with increased abnormalities of consciousness. Cerebral CT at H1 showing an increase in unenhanced subarachnoid hyperdensity suggestive of a rebleed with development of a mesencephalic intraparenchymal component (C, D). The patient died on D7.

Insertion of an external ventricular shunt increases this risk [11].

Acute hydrocephalus

Acute hydrocephalus is defined as a sudden dilatation of the ventricular system due to obstruction to the flow of cerebrospinal fluid caused by blood degradation products when the ventricle is breached, and is seen in up to 20% of patients [10]. The risk is proportional to the extent of the initial bleed although subarachnoid hemorrhage with a low initial Fisher grade can also be complicated by acute hydrocephalus. This worsens the prognosis by increasing the incidence of raised intracranial pressure in addition to the cerebral edema caused by the subarachnoid hemorrhage itself [12]. It is therefore extremely important to identify ventricular dilatation from the point of early treatment onwards. It begins with dilatation of the temporal horns, which is easy to identify in the absence of subcortical atrophy. Before endovascular treatment to exclude the aneurysm, an external ventricular shunt can be inserted on an urgent basis by the neurosurgeons [13] (Fig. 2). Classically this is performed routinely for Fisher grade 3 or 4 subarachnoid hemorrhages and is considered depending on clinical symptoms for Fisher grades 1 or 2. Its purpose is to release the raised intracranial pressure and prevent the risk of neurological deterioration. A shunt catheter clamp test is performed on D15 before it is possibly removed.

Acute ischemic lesions

Acute massive cerebral edema is a rare complication, of poorly understood pathophysiology, which may result in diffuse cortical ischemic injury leading to death within hours after the subarachnoid hemorrhage [14—16]. This classically affects young people regardless of the severity of the initial bleed and involves a sudden-onset diffuse cerebral edema with abrupt accelerated phase raised intracranial pressure. It can be demonstrated by inserting an intracranial pressure sensor. Cerebral MRI with diffusion-weighted images can

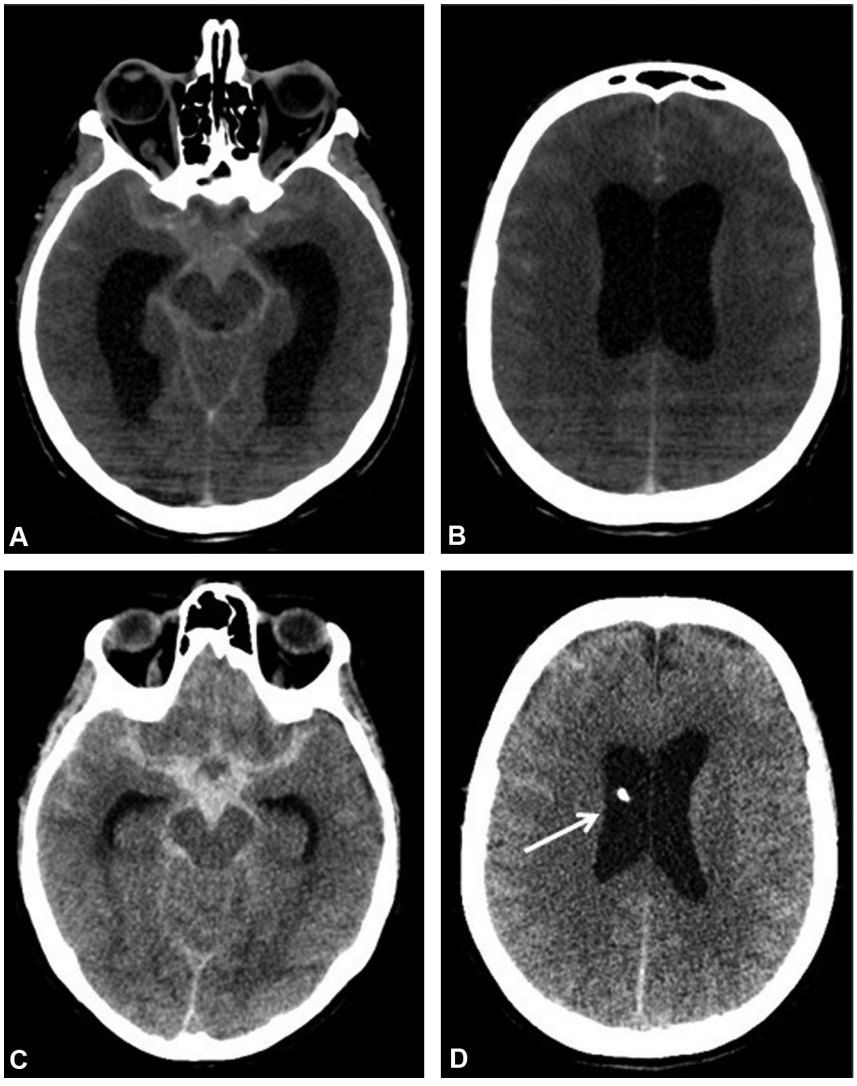

Figure 2. Fifty-one year old woman with sudden-onset headaches. Unenhanced cerebral CT shows hyperattenuating area in the motor cortex sulci and perimesencephalic cisternae with ventricular involvement and dilatation of the ventricular system suggestive of Fisher grade 4 subarachnoid hemorrhage complicated by acute hydrocephalus (A, B). Insertion of an external ventricular shunt cannula (D, arrow) before endovascular treatment reducing the ventricular volume seen on the immediate postoperative CT (C, D).

help in the diagnosis in a patient presenting with sudden-onset abnormalities of consciousness (Fig. 3). Treatment is with a combination of medical therapy for the edema and neurosurgery with insertion of an external ventricular shunt, but in most cases this is not sufficient to improve the prognosis.

Non-neurological complications

Some non-neurological complications have been described during the days following the subarachnoid hemorrhage. In particular these include diffuse electrocardiographic repolarization abnormalities [17], occasionally genuine findings of myocardial distress with raised troponins [18], and the Tako-Tsubo syndrome with acute respiratory injury (acute pulmonary edema, ARDS) [19]. Regular electrolyte monitoring is required as serum sodium and potassium abnormalities are common [20].

Subacute complications (D3—D30): vasospasm

Vasospasm is the most common complication following subarachnoid hemorrhage [21]. The risk of vasospasm occurs later than the risk of rebleeding, classically between D4 and D15, but cases of later vasospasm have also been reported [22]. This risk correlates with the extent of the initial bleed and with Fisher classification [23]. It preferentially affects young women. Degradation and lysis of extravascular blood clots within the cerebral fluid lead to the release of vasoactive mediators, which cause cerebral vasoconstriction leading to a fall in cerebral blood, flow [24]. Although an inconsistent finding, it may be followed by the development of a cerebral hypoperfusion area with a risk of cerebral infarction, which may be fatal. Clinically, a warning sign for this is development of a focal neurological deficit on D3 after a subarachnoid hemorrhage. Clinical symptoms are occasionally more silent and the development

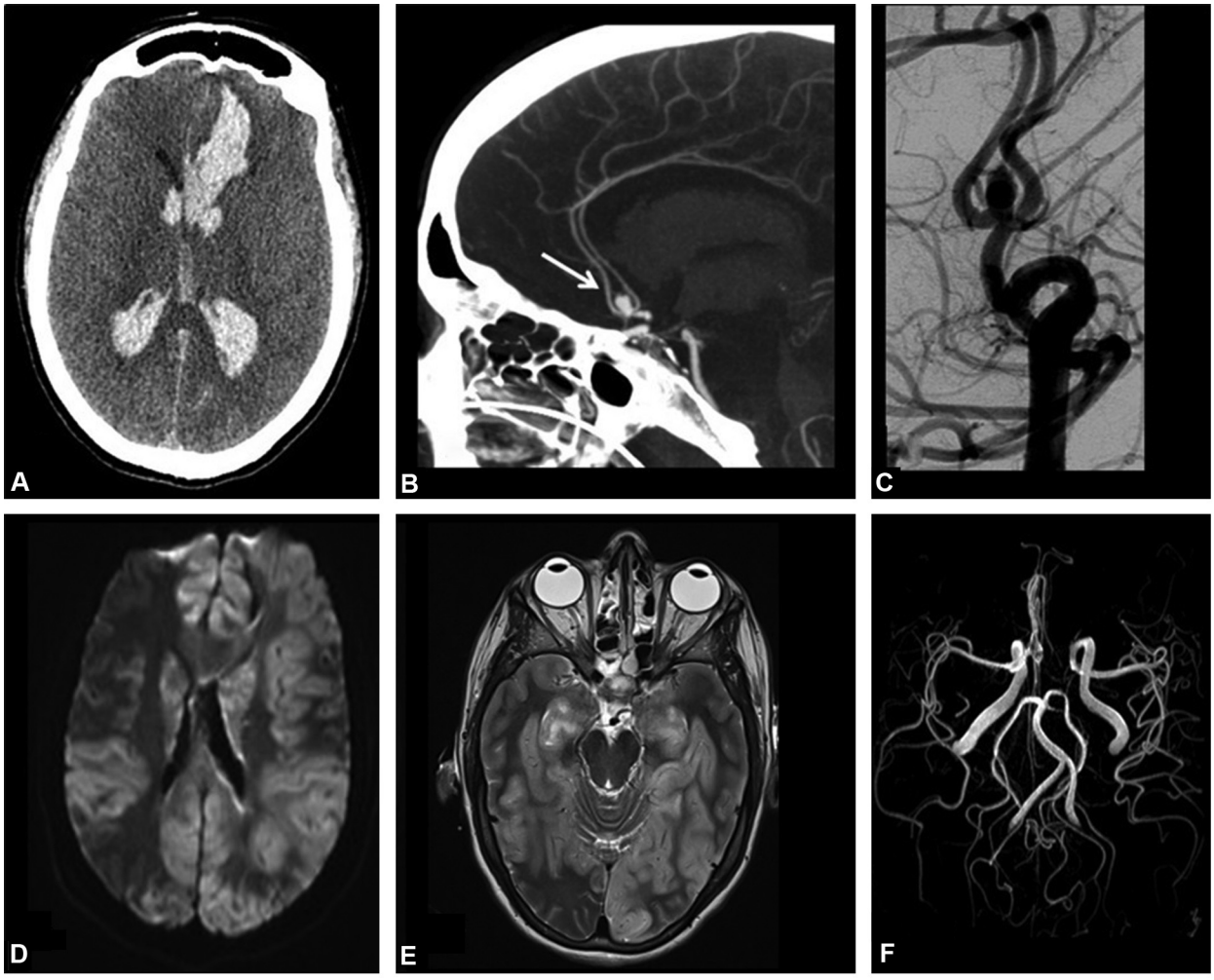

Figure 3. Thirty-six year old female patient with sudden-onset headaches, vomiting and abnormal consciousness level. Glasgow score 4. Bilateral meiosis. Unenhanced cerebral CT showing a hyperdensity in the cortical sulci with a left frontal intraparenchymal hematoma and ventricular flooding suggestive of a Fisher grade 4 subarachnoid hemorrhage (A) due to rupture of an anterior communicating artery aneurysm seen on the sagittal MIP reconstructions after enhancement (B, arrow) and cerebral arteriography (C). Abnormal transcranial Doppler ultrasound on D3. Cerebral MRI on D3 showing huge hyperintense b1000 cortico-subcortical lesions (D), and on T2 weighted imaging (E) with no appearances of vasospasm of the intracranial vessels on time of flight imaging (F) suggestive of diffuse cortical ischemic lesions. The patient died on D6.

of fever in the absence of other pointers towards infection, or of sweating, agitation or confusion should also suggest a diagnosis of vasospasm. The diagnosis should be confirmed radiologically if this is suspected clinically. Transcranial Doppler ultrasound is used first line and shows increased arterial circulatory systolic and mean velocities in the spastic artery [25]. The increase in middle cerebral artery circulation velocities is proportional to the severity of the vasospasm. A mean velocity of between 80 and 120 cm/s may represent mild vasospasm and with mean velocities of up to 130 cm/s arteriography usually shows moderate vasospasm. Using a cutoff of 130 cm/s, the specificity of transcranial Doppler ultrasound to detect spasm is 96%, with a positive predictive value of 87%. A mean velocity of over 200 cm/s is suggestive of severe vasospasm liable to cause cerebral ischemia. It is a rapid rise in velocities rather than the absolute value of the systolic peak, which is the poor prognostic indicator. A middle cerebral artery systolic/extracranial internal carotid artery systolic velocity ratio (the Lindegaard index) of over 3 is also used to assess vasospasm [26]. An increase in velocity due to vasospasm may be masked by concomitant raised intracranial pressure, although the increase in resistance indices should generally draw radiologists' attention and allow this to be taken account of in the interpretation [27]. Transcranial Doppler ultrasound however has certain limitations: the vessels in which the highest velocities are seen are not necessarily located in territories which correspond to the symptoms of ischemia; symptomatic ischemia is not always manifest in the arteries located close to the areas where the greatest bleed has occurred and distal cerebral arteries are difficult to examine. In view of these limitations, cerebral MRI [28] or cerebral CT angiography [29] with perfusion weighted images can confirm the actual diagnosis and exclude the differential diagnoses, which include acute hydrocephalus [30,31]. Images show arteries in spasm, which are tapered and responsible for a greater, or lesser area of cerebral hypoperfusion with raised mean transit time (MTT) and time to peak (TTP). This may be combined with greater or lesser areas of cerebral ischemia, which are often punctiform and diffuse and affect the distal arterial and junctional areas although the presence of arterial spasm is not necessarily synonymous with distal ischemic injury and vice versa. The treatment for vasospasm is initially pharmacological escalating therapy with intravenous nimodipine [25] and maintaining a high blood pressure [32]. Hypervolemia is still debated [33]. If a patient is resistant to pharmacological treatment, an endovascular approach can be offered involving repeated intra-arterial administration several days in succession close to the artery in spasm, nitrates (milrinone COROTROP®) [34] (Fig. 4) and possibly arterial balloon angioplasty. [35].

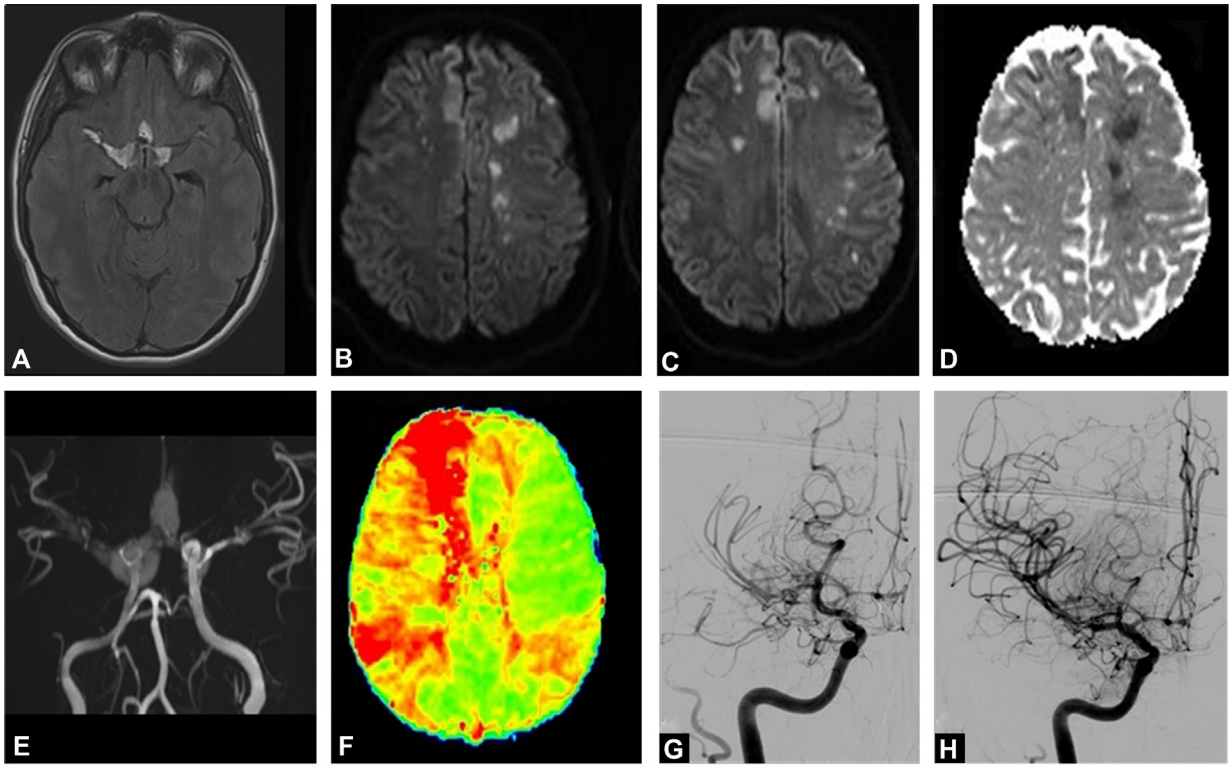

Figure 4. Twenty-nine year old woman presenting with a subarachnoid hemorrhage, initially Fisher grade 3 due to rupture of the anterior communicating artery treated endovascularly (not shown). On D10 she developed a left hemiplegia. Urgent cerebral MRI. Appearance of subarachnoid hemorrhage, hyperintense of FLAIR imaging in the motor cortex sulci (A). Development of bilateral junctional hyperintense b1000 punctiform ischemic lesions (B, C); restriction on ADC mapping (D). Pronounced spasm of the trunks of the carotid arteries and middle and anterior cerebral arteries seen on time of flight imaging of the circle of Willis (E) with large extensive increase in the mean transit time mostly affecting the middle and right anterior cerebral artery territories and the left posterior superficial middle cerebral artery (F). Confirmation of severe spasm of the carotid and middle and right anterior cerebral artery trunks on arteriography performed immediately afterwards (G) and a large improvement in cerebral parenchymography after intra-arterial injection of milrinone (H).

Chronic complication (after D30): chronic hydrocephalus

This complication occurs late after the initial subarachnoid hemorrhage. The classical symptoms are Adam and Hakims triad of walking difficulties, sphincter disturbances and cognitive disorders, disorientation and confusion. It is due to partitioning in the arachnoid space, which prevents normal reabsorption of cerebrospinal fluid and causes dilatation of the ventricular system. An unenhanced cerebral CT should be performed looking for ventricular dilatation if a patient develops neurological problems late after a subarachnoid hemorrhage [36]. Depending on age and clinical signs, the treatment of chronic hydrocephalus involves a depletional lumbar puncture or insertion of a ventricular peritoneal shunt by the neurosurgeons.

Studies have also shown that late cognitive or behavioral complications may develop. Twenty-five per cent of patients develop moderate depression after a subarachnoid hemorrhage and several years after the hemorrhage, approximately 50% of survivors report that they think that their personality has changed, generally for the worse, the cases reported being mostly weakness, excessive irritability, memory difficulties, daytime drowsiness and insomnia [37].

Long-term patient follow up

Which patients?

All patients should be followed up late after treatment for a subarachnoid hemorrhage. This is justified by the presence of a risk of recurrence of aneurysm in 15 to 30% of cases after endovascular treatment [38,39] (Figs. 5 and 6) with a rebleeding risk of 2 per 1000 at 1 year [9,40]. A new aneurysm may also develop in another vascular territory meaning that optimal examination of the 4 intracranial arterial systems is required [41,42].

How?

A consultation should be planned in order to identify any recurrence of neurological symptoms (headaches or weakness) and seek to identify the risk factors for recurrent aneurysm or de novo aneurysm formation, which are, mostly smoking and hypertension.

An imaging investigation is performed at the same time. CT, which is the primary investigation in the acute phase diagnosis of subarachnoid hemorrhage, has limited use in the post-treatment follow up aneurysms treated endovascularly because of the large metal artifacts due to coils,

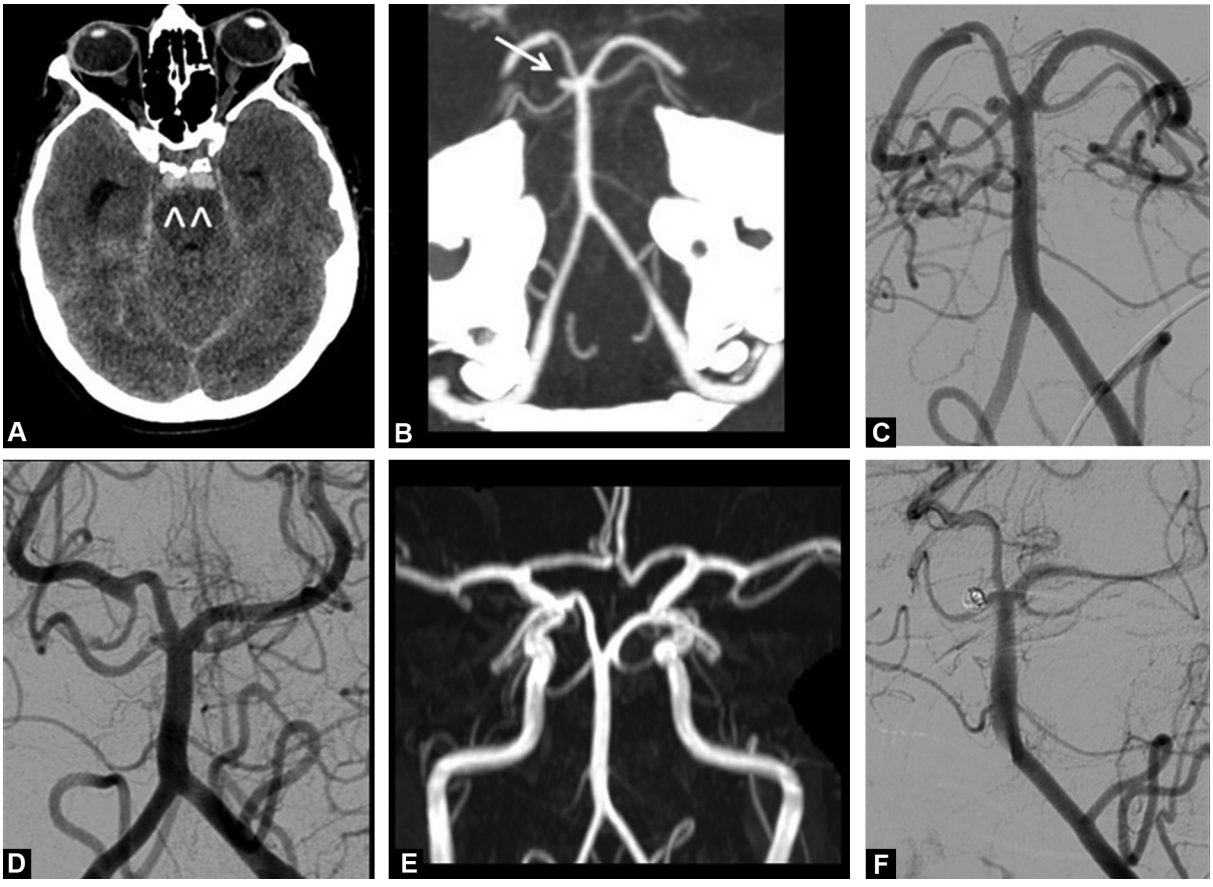

Figure 5. Sixty-one year old woman. Hyperdensity in the prepontine cisterne on the unenhanced CT (A, arrowheads) suggestive of a subarachnoid hemorrhage due to rupture of an aneurysm located at the origin of the right superior cerebellar artery (B, coronal MIP reconstruction after enhancement, arrow). Complete exclusion of the aneurysm after endovascular coiling (cerebral arteriography C, D). No recurrence of the aneurysm on time of flight imaging performed at 6 months (E) or on the routine cerebral angiogram at one year (F).

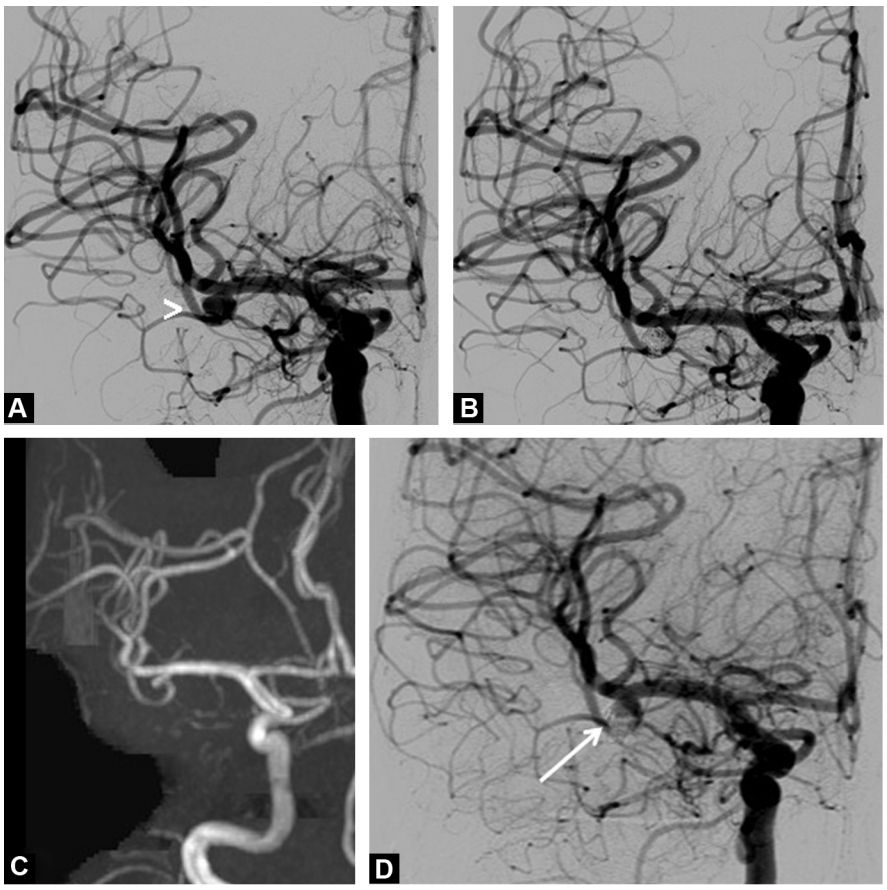

Figure 6. Forty-seven year old woman with a past history of subarachnoid hemorrhage due to rupture of an aneurysm of an inferior dividing branch of the right middle cerebral artery (A, arteriography, arrowhead) with complete exclusion after coiling (B). Residual aneurysm seen at 6 months on time of flight MRI (C) and cerebral arteriography (D, arrow).

which prevent a good vascular examination of the region of interest. Cerebral MRI is the recommended investigations to follow up embolized aneurysms as this method offers the best cost-effectiveness balance [43]. A short image acquisition protocol is used with a combination with FLAIR image looking for possible parenchymal complications together with a time of flight vascular image in the intracranial artery, which is less sensitive to coil-related artifacts and can be used to look for recurrence of the aneurysm in the treated area and the development of another remote aneurysm. In order to minimize artifacts, the technical settings for the image acquisition should be optimized (shortening the echo time, ET of under 3 ms) in order to improve detection of any residual circulation [44]. The investigation volume should also be appropriate, covering from the postero-inferior cerebellar arteries to the pericallosal arteries with high spatial resolution, preferably on a 3 T MRI instrument [45]. The sensitivity and specificity for detection of intracranial aneurysms under 5 mm in size are 98 and 94% respectively at 3 Tesla [46]. The routine use of gadolinium enhancement is still debated [47]. MRI of the aneurysm wall also appears to be an image of the future in order to identify unstable aneurysms [48]. Cerebral arteriography is performed 12—18 months after the initial bleed depending on the group. If recurrence of the aneurysm is suspected on MRI, cerebral arteriography is performed to assess the need for further endovascular treatment. The reference investigation for ruptured aneurysms treated by neurosurgical clipping is currently cerebral arteriography because of the large metal artifacts on CT and MRI.

When?

Most recurrences occur in the first year [39]. Depending on the center, patients should therefore be reviewed with a cerebral MRI at between 6 and 12 months and then with cerebral arteriography between 12 and 18 months [49]. The clinical and radiological follow up with cerebral MRI is then organized initially at 2-year intervals and then every 5 years.

Conclusion

The complications of subarachnoid hemorrhages are a major factor in the prognosis of patients who have suffered a ruptured aneurysm. In the acute phase, rebleeding is the most serious complication and requires emergency treatment of subarachnoid hemorrhages. Neurosurgical insertion of a ventricular shunt should be considered if acute concomitant hydrocephalus is present. Vasospasm occurs classically sub-acutely from D3 and should be diagnosed on an urgent basis with transcranial Doppler ultrasound or imaging with perfusion-weighted images if clinically suspected. Because of the risk of recurrence of the rebleeding or long-term

recurrence of the aneurysm all patients should be followed up.

Take-home messages

Complications

The complications of subarachnoid hemorrhage can be classified according to their time to onset:

- acute complications, from D0-D3:
  - rebleeding: the risk is present in the initial hours after the subarachnoid hemorrhage. This is the most serious complication and requires urgent exclusion of the aneurysm,
  - acute hydrocephalus: insertion of a ventricular shunt before endovascular treatment should be considered in order to prevent possible neurological deterioration,
  - acute ischemic injury: this should be diagnosed promptly, particularly if the patient's conscio-usness level deteriorates. It carries a poor prognosis,
  - non-neurological complications: cardiac, respiratory or electrolyte;
- subacute complications, from D3-D30:
  - vasospasm: described classically from D4. Vasospasm may result in ischemic injury. This may be suspected clinically and is confirmed by transcranial Doppler ultrasound and/or imaging with perfusion weighted images;
- chronic complications, after D30:
  - chronic hydrocephalus: an unenhanced cerebral CT should be performed in the event of late clinical deterioration,
  - late cognitive and behavioral problems have also been described.

Follow up

- All patients should be followed up because of the existent risk of rebleeding (2 per 1000 at one year) and recurrence of the aneurysm (15 to 30%). This follow up should be with a combination of a clinical consultation and cerebral MRI with at least one review angiogram regardless of type of treatment used.
- Cerebral CT should be avoided because of the coil-related artifacts.

Clinical case

A 48-year-old woman presents with thunderclap headaches and no neurological deficit with a Glasgow score of 15. An unenhanced CT and then CT angiography of the circle of Willis is performed (Fig. 7).

Questions

1- List the abnormalities visible on the CT.
2- What is your diagnosis?

An external ventricular shunt is inserted before endovascular treatment which is carried out without complication, completely excluding the aneurysm.

On D12, the patient's neurological condition worsens and she develops altered consciousness and confusion. An unenhanced CT followed by CT angiography of the circle of Willis is performed (Fig. 8).

3- What is your detailed interpretation?
   a. Rebleed from the aneurysm
   b. Persistent hydrocephalus
   c. Vasospasm
   d. Ischemic injuries
4- In terms of the long-term management of this patient what should be proposed?
   a. As the aneurysm has been excluded no follow up is necessary for this patient.
   b. A repeat CT angiogram with the circle of Willis at 6 months is recommended.
   c. A repeat cerebral MRI with examination of the circle of Willis at 6 months is recommended.
   d. A repeat late angiogram should be performed

Answers

1- Unenhanced hyperdensity in the perimesencephalic cisternae and motor cortex sulci with exaggerated appearance of the temporal horns of the lateral ventricles on unenhanced CT (Fig. 7a and b). Saccular appearance at the apex of the basilar trunk on sagittal MIP reconstructions of the circle of Willis CT angiogram (Fig. 7c).
2- Fisher grade 2 subarachnoid hemorrhage due to rupture of an apical basilar trunk aneurysm complicated by early hydrocephalus.

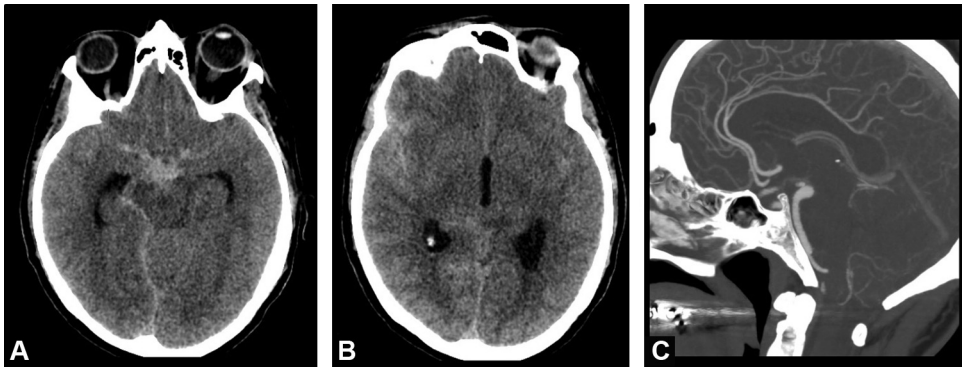

Figure 7. Initial unenhanced cerebral CT (A, B). Cerebral CT angiogram of the circle of Willis with sagittal MIP reconstruction (C).

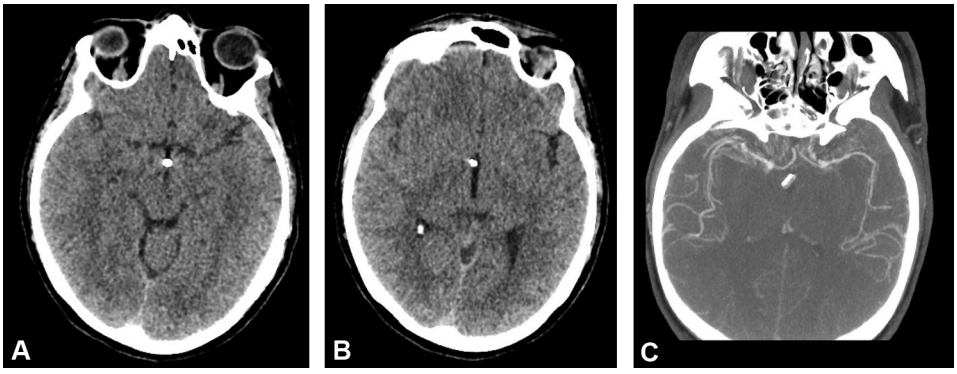

Figure 8. Unenhanced cerebral CT on D12 (A, B). Cerebral CT angiogram of the circle of Willis on axial MIP reconstruction (C).

3- Answer c. In this context of neurological deterioration in a patient on D12 after a subarachnoid hemorrhage (subacute phase), axial MIP reconstructions of a circle of Willis CT angiogram show a tortuous tapered appearance in the middle cerebral arteries (Fig. 8c), indicative of vasospasm.
   In addition:
   - regression of the unenhanced subarachnoid hyperdensities described before (Fig. 8a and b) and treated aneurysm excluded with no evidence of rebleeding;
   - reduction in ventricular volume with the shunt catheter in place, passing through the third ventricle (Fig. 8a and b), with no evidence of hydrocephalus;
   - no cortico-subcortical hypointensity: no evidence of an ischemic lesion.

4- Answers c and d. Because of the risk of regrowth and recurrence of an aneurysm this patient should be followed up. Examination of the circle of Willis by CT angiography may be impeded by the coils and cerebral MRI with investigation of circle of Willis should therefore be used in preference. A cerebral arteriogram should be performed routinely 12—18 months after the initial bleed.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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