INTERHEMISPHERIC SUBDURAL HEMATOMA FROM RUPTURED ANEURYSM: CASE REPORT

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Sirs: Interhemispheric subdural haematoma (ISH) is a relatively rare subtype of acute subdural hematoma (ASDH) and it is almost invariably due to head trauma [1]. Here we describe a case of a non traumatic ISH from ruptured aneurysm without bleeding into the subarachnoid space.

A 62-year-old hypertensive woman started complaining with a flu-like syndrome together with a dull widespread headache. Three days later her headache dramatically worsened. Pain was localized on the left side of the head, spreading both to the occipital region and to the eyebrow and increasing with strain and Valsalva manoeuvre. Mild nausea and an episode of vomit followed. Four days later the patient experienced diplopia and left eyelid drop. She was admitted to our Department nine days after the onset of the symptoms. Her past neurological history was unrevealing and there was no history of head trauma, blood clot disturbances or anticoagulant therapy. There were no concomitant medications. Her blood pressure was 169/95 mmHg. The neurologic examination revealed a complete left third nerve palsy, but no other signs were observed. No abnormalities were observed in routine blood counts, blood chemistry and coagulation studies. Cranial CT scan revealed a spontaneous bleeding along the cerebral falx, over the left tentorium and posterior to the clivus, confirmed by MRI. Both exams did not reveal any evidence of a component of subarachnoid or intraparenchymal haemorrhage (Figure 1). Cerebrospinal fluid (CSF) examination was normal but for the presence of 10 white cells/mm$^3$ (normal value <5). The angiographic sequences of MRI revealed a left internal carotid artery wide-based aneurysm located in the supra-clinoid portion of carotid syphon, close to the posterior communicating artery, in the so-called internal carotid – posterior communicating artery junction (IC-PC). The aneurysm projected infero-posterolaterally and was confirmed by an angiographic study. The patient underwent an endovascular embolization treatment by means of Guglielmi
detachable coils. After embolization, a cranial CT scan showed a reduction of the ISH. The left oculomotor nerve palsy remained unchanged during the following weeks. When last examined, six months after the onset of the symptoms, the patient had a normal neurologic examination and the left third nerve palsy has completely recovered.

Non traumatic cerebral aneurysm rupture usually involves the subarachnoid space, thus causing a subarachnoid haemorrhage (SAH). The concomitant involvement of the subdural space is much less frequent [3]. Three mechanisms for the occurrence of subdural bleeding have been hypothesized. First, previous small bleedings allow adhesion of the aneurysm to the adjacent arachnoid membrane and the final rupture occurs into the subdural space. Second, a high pressure haemorrhage may lead to pia-arachnoid rupture, thus causing extravasation of blood into a subdural or parenchymal location. Third, a rapid accumulation of blood distends the subarachnoid space causing a tear of the arachnoid membrane, even far from the site of aneurysm rupture [2,3]. In a very few cases the aneurysm rupture involves exclusively the subdural space, thus causing a pure ASDH, which is usually located at the convexity [5]. Among the above mentioned hypotheses, only the first one can account for a pure ASDH, without either SAH or parenchymal haemorrhage. ISH is a quite rare subtype of ASDH, which occurs almost invariably after head trauma. To the best of our knowledge, only four cases of spontaneous ISH in the absence of SAH have been previously reported. Our case is the fifth and the first one in the European literature (Table). Our patient underwent embolization of the aneurysm, while the others had the aneurysm clipped. Focal neurological deficits, which may address to a localization of the aneurysm, could be found only in two patients (#4 and ours): in both cases a third cranial nerve palsy occurred, which is known to be related to IC-PC aneurysms [6]. Three patients had an aneurysm at the distal anterior cerebral artery (ACA), while the other two (including
ours) had an aneurysm at the IC-PC junction. Blood from distal ACA aneurysms caused an ISH, but also reached the convexity, resulting in a more severe clinical picture (consciousness disturbances) which required the hematoma evacuation. On the other hand, in the two patients with ISH from ruptured IC-PC aneurysms, blood drained also over the tentorium cerebelli. These continuity differences could be helpful in determining the aneurysm location. In summary, our report indicates that, although uncommon, rupture of an intracranial aneurysm should be kept in mind as a cause of spontaneous ISH. Importantly, this can be the case even in the absence of subarachnoid bleeding and of a history of head injury.

References


Figure legend:

A,B: TC scan, axial slices showing hyperdense retroclival tissue extending along the left tentorium and the interhemispheric region. No subarachnoid hemorrhage is seen.
C: RM scan, coronal slice, showing hyperintensity, due to the presence of methemoglobin, of the falx and left tentorium. No subarachnoid bleeding is observed.
D: RM scan, sagittal median slice, analogous hyperintensity as in C along the cerebral falx
E: RM scan, axial 3DTOF angiographic sequence, shows an aneurysm in the supraclinoid part of left internal carotid artery, projecting infero-postero-laterally (white arrow)
F: Digital angiography after endovascular coiling of the aneurysm
Table (supplementary material)

<table>
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<tr>
<th>Case #</th>
<th>Reference</th>
<th>Age/Sex</th>
<th>Symptoms/signs</th>
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<th>Aneurysm site</th>
<th>Treatment</th>
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<td>Headache, left eyelid ptosis</td>
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