Intrasellar Rupture of a Paraclinoid Aneurysm with Subarachnoid Hemorrhage: Usefulness of MR Imaging in Diagnosis

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Case Report

A 45-year-old man presented with good clinical grade subarachnoid hemorrhage. CT revealed blood in the basal cisterns and Sylvian fissures with symmetric distribution (Fig 1A,B). A digital subtraction angiography (Fig 1C, D) and a CT angiogram (CTA) demonstrated 2 aneurysms, a larger one arising from the right paraclinoid carotid and a smaller one at the right middle cerebral artery (MCA) bifurcation. The neck of the paraclinoid aneurysm could be seen at the level of the optic strut, with the sac pointing posteriorly and inferomedially, in close relationship with the sella turcica. (Fig 2). MR imaging was performed with the hope of being able to clarify if the paraclinoid aneurysm was intradural or extradural; results showed the aneurysmal neck to be intradural and its dome below the dural ring, extradural. In addition, an enlarged sella turcica was noted, with inferior displacement of the pituitary gland and a large amount of blood filling the sella and extending into the subarachnoid space (Fig 3). We concluded that the paraclinoid aneurysm had ruptured into the sella with subarachnoid extension. The paraclinoid aneurysm was successfully coiled.

Discussion

The establishment of either an intradural or extradural location of paraclinoid aneurysms has critical implications for treatment and prognosis. Extradural aneurysms have a good prognosis and very low risks for a subarachnoid hemorrhage and major neurologic complications. Treatment is reserved for symptomatic lesions or for those lesions with extension into the subarachnoid space. Intradural aneurysms carry a risk for subarachnoid hemorrhage and may require treatment. Most paraclinoid aneurysms can be localized by their direction of projection on angiograms. However, the complex anatomy of the juxtaocular ring area and individual variability in this region can sometimes make it difficult to define the relationship of the aneurysm to the dural ring, which differentiates extradural (below the dural ring) and intradural (above the dural ring) lesions.

Different techniques with several anatomic references have been used to differentiate intradural from extradural aneurysms. The simplest method uses the ophthalmic artery as the landmark for the transition of the extradural and intradural location. The major drawback of this method is that the origin of the ophthalmic artery is extradural in 10% of cases, leading to the erroneous assumption that an aneurysm is intradural when it is intracavernous. In our patient, the aneurysm arises distal to the ophthalmic artery, but its inferior direction places it below the plane of this artery (Fig 1D).

A reliable landmark for identification of the proximal dural ring, which defines the roof of cavernous sinus, is the optic strut. Lesions above this level are considered intradural, and below, intracavernous, extradural. In our case, the neck of the aneurysm is located at the exact level of the optic strut, and its fundus projects downward and medially, into the sella turcica. The aneurysm could be classified as a carotid cave aneurysm, arising between the 2 dural rings and growing into the cavernous sinus. The carotid cave is a pouch located in the posteromedial side of the distal dural ring, above the proximal ring, which may or may not have a communication with the subarachnoid space.

The diffuse subarachnoid hemorrhage demonstrated on CT could have been caused by either lesion, so it was important to determine if the proximal aneurysm was intradural or extradural. If the paraclinoid aneurysm was extradural, our treatment choice for the MCA aneurysm (Fig 1C) would have
been clipping (small size, relatively wide neck), but if it was intradural, our first choice of treatment would be coiling. CT and CTA alone were insufficient to define the location of the paraclinoid aneurysm and did not reveal the large intrasellar hemorrhage. In retrospect, after MR imaging revealed the intrasellar hemorrhage, intrasellar blood could be suspected on the plain CT head (Fig 1B).

MR imaging at 3T was used to visualize the dural ring8 and showed that the neck of the aneurysm was intradural and its dome, clearly extradural. More importantly, it revealed a large amount of blood inside an enlarged sella turcica, confirming that the paraclinoid aneurysm had bled. The large amount of intrasellar blood, the relatively small volume of blood in the subarachnoid space, and the empty sella syndrome lead to the hypothesis that the aneurysm had bled primarily into the sella and secondarily through an incompetent diaphragma sellae into the subarachnoid space. Absence of a bony septum in the posteromedial segment of the carotid cave9 allows direct contact of the pulsatile aneurysmal dome with the dura, which eventually could cause its progressive erosion and the intrasellar hemorrhage. Advanced MR imaging techniques have improved our ability to identify the ruptured lesion in cases of multiple intracranial aneurysms, where MR imaging may identify a parenchymal clot adjacent to an aneurysm and recently has been used to determine an intradural or extradural location in unruptured paraclinoid aneurysms.

Conclusion
We report the case of a patient with a unique presentation of a paraclinoid aneurysm with intrasellar hemorrhage. Lesions located in the paraclinoid carotid can be difficult to clearly define as intradural or extradural. In this case, localization of the paraclinoid lesion (intradural vs extradural) and identification of the aneurysm responsible for the subarachnoid hemorrhage were critical in choosing a treatment strategy. The presence of intrasellar blood and the relationship of the paraclinoid aneurysm to the dura elegantly demonstrated on MR
imaging were critical in choosing the target lesion for treatment.

References