Giant Aneurysm or Pituitary Macroadenoma: A Diagnostical Misconstrue


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Summary
We describe a case of a giant thrombosed intrasellar internal carotid artery aneurysm initially diagnosed as pituitary macroadenoma. The differentiating neuroimaging features were discussed.

Key Words: Giant aneurysm, Pituitary macroadenoma, MRI, Endovascular

Introduction
Giant intracranial aneurysms are defined as aneurysms greater than 2.5cm at their greatest diameter. Their overall frequency has been reported as approximately 5-13% of all intracranial aneurysms. Giant aneurysms frequently present with signs of an intracranial mass. These aneurysms can be mistaken with other mass lesions particularly when it is almost completely thrombosed as the usual appearance of patent residual lumen might not seen in CT or MR.

We present a case of thrombosed giant right internal carotid artery aneurysm initially diagnosed as pituitary macroadenoma. Patient underwent 2 neurosurgical procedures before cerebral angiogram done confirming the diagnosis of giant aneurysm. Patient later had successful endovascular treatment.

Case Report
A 53-year-old lady presented in February 2002 with progressive visual loss for the past 6 months. She had history of fainting attacks and headache 3 years ago and a computed tomogram of the brain (CT Brain) done at that time, reported as normal. She had normal neurological examination except for visual acuity where the right vision was 6/60 on the left 2/60. Funduscopy was normal. Following this, she had CT Brain done and reported as pituitary macroadenoma (Fig 1), subsequently she had transphenoidal exploration where prominent intracavernous veins was found, inconsistent with pituitary macroadenoma.

While the vision progressively worsens, magnetic resonance imaging (MRI) was done and reported as a sellar mass with suprasellar extension (Fig 1). Operative surgical decision based on the MRI findings includes aspiration of the mass. Following which, the patient underwent subfrontal approach craniotomy to remove the suprasellar mass. The surgeon noted heavy arterial bleeding during attempt of biopsy and decided to pursue with cerebral angiogram.

Cerebral angiogram revealed a lobulated thrombosed supraclinoid aneurysm of the right internal carotid artery (Fig 2). Not surprisingly, the histopathological result showed acellular homogenous eosinophilic material, probably fibrin, is in keeping with the aneurysm wall. She had good occlusion of the aneurysm after endovascular treatment with a total of 5 Guglielmi Detachable Coils (GDC).
CASE REPORT

Discussion

Although 90% of intracranial aneurysm present with subarachnoid haemorrhage (SAH), only 25% of giant intracranial aneurysm presented with SAH while majority have signs of intracranial mass. Aneurysms projecting into the sellar region account for 1% to 2% of all intracranial aneurysms and they usually originate from the infraclinoid segment of ICA.

Aneurysms of the sella region are commonly mistaken for pituitary adenomas, since they can have similar clinical, endocrinological and neurological symptoms. Mass like features of giant aneurysm and compression to adjacent cranial nerves or cerebrum can produce clinical and neurological presentation of space occupying lesion. Pituitary deficiency may result from compression of the hypothalamus or the pituitary stalk. In some cases rupture of the aneurysm may precipitate hypopituitarism. However, pituitary adenoma may be associated with an intrasellar aneurysm.

Pinto et al. reported about 8 of 21 giant aneurysms studied were initially diagnosed as meningioma, pituitary adenoma and glioma. Completely thrombosed aneurysms may give more diagnostic problem as angiography demonstrated only avascular mass. Reliable imaging techniques and high index of suspicion are essential in establishing the diagnosis as there are certain characteristics in non-invasive imaging, typical of giant aneurysms.

CT Brain may show laminated thrombus within the lumen, whether or not it has calcified. The differing

Fig. 1: CT Brain showed slightly hyperdense mass arising from sella with minimal heterogeneous enhancement after iv. Contrast. MRI images in T2W and post Gadolinium revealed, mixed signal intensities within the lesion representing various stages of clots. Peripheral enhancement due to fibrotic wall of the aneurysm. Note the relationship between the mass and signal void adjacent parent artery (arrow).

Fig. 2: Cerebral angiogram showed the supraclinoid right ICA giant aneurysm. Subsequent images showed successful endovascular treatment for the patient.
appearances of the main lesion would be consistent with the varying amount of thrombus around the wall and the size of the remaining patent lumen. Homogenous enhancement of the whole lesion occurs when no clot is present and the whole lumen is patent. Patchy enhancement occurs when layers of clot are present. The peripheral enhancement is believed to be the result of extravascular diffusion of contrast material into the vascularized fibrous tissue wall and is analogous to normal contrast enhancement of the dura mater. Majority has no cerebral oedema and with enhanced fibrous tissue wall of aneurysm, this may simulate meningoia. However, relationship with patent parent artery should suggest giant aneurysm. The presence of a curvilinear wall calcification and the absence of oedema support the diagnosis of a thrombotic giant aneurysm.

Magnetic Resonance Imaging (MRI) normally shows a well-defined lesion and is contiguous with the parent vessel. Thrombus in an aneurysm appears as an area of multilamellated, high signal intensity in T1-weighted images and usually has a dark rim in T2-weighted sections representing various stages of hemoglobin degradation. The dark rim is due to hemosiderin in the wall of the aneurysm or adjacent brain tissue from previous haemorrhages, which is more clearly seen in gradient echo.

Residual patent lumen within the lesion is another important feature of a giant aneurysm, which may be evaluated by MRA. 3D TOF MRA or Phase Contrast MRA can be used as a non-invasive method to evaluate the flow within giant aneurysm, however contrast enhanced dynamic 3D TOF MRA provides a more superior contrast between the patent flow and background. It also eliminates the T1 contamination artifact significantly and should be the sequence of choice in evaluating giant aneurysm5. Although cerebral angiogram remains the gold standard in evaluating the location, flow dynamics and extent of an aneurysm, it may underestimate the size, as the thrombosed part may not fill with contrast.

There are several treatment options mainly direct clipping of the aneurysm neck or endovascular treatment. The former is associated with open surgical treatment and overall management outcomes for giant aneurysms are poorer than for smaller lesions. Endovascular treatment is where platinum coils, which are soft, flexible and can induce thrombosis, placed into the aneurysm via microcatheter. The techniques evolved rapidly in recent years and increasingly becoming as an important treatment option for intracranial aneurysm. Endovascular option also noted to give good long-term clinical outcomes in treatment of giant intracranial aneurysms.

References