

Unruptured Posterior Communicating Artery Aneurysm Masquerading as Migraine: Report of Two Cases

Pages with reference to book, From 172 To 174

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Introduction

Ruptured intracranial aneurysm is a major public health problem afflicting approximately 28,000 Norih Americans each year and resulting in death or disability in 18,000 of these patients¹, Vast improvements have been made in the operative results for ruptured aneurysms during the past 3 decades, but these have not reflected in a proportional improvement in overall management results. In spite of all these surgical advances, the overall mortality and morbidity of ruptured aneurysm continues to be very high². The reason why the improvement is minimal in the overall management result is that more than one-third of the patients with ruptured aneurysms are dead or disabled before they reach the hospital or become available for treatment¹, Nothing can at present be done to save these patients who die quickly after aneurysmal rupture³. The factors responsible for this fatal outcome in majority of these patients included: ignorance of warning symptoms, initial misdiagnoses and late referral¹. For any future improvements in the overall outcome of the patients with aneurysmal subarachnoid hemorrhage, one has to address to the subtle warning signs of the unruptured aneurysms. Recognition of atypical migraine as an important warning symptom of unruptured aneurysm may lead to their treatment in the pre-rupture stage, thus improving the overall results.

Case Report

Case 1

A 35-year old woman presented with a history of severe left orbital and hemicranial pain for 6 weeks. The pain was continuous and refractory to medical treatment. Left third nerve palsy was noted 2 days prior to admission. The patient had a history of migraine diagnosed 5 years previously, for which she had been treated with caffergot and propanadol. Examination revealed an alert patient in excellent health but bitterly complaining of left-sided headache. Except for the left third nerve palsy, the neurological examination was normal. An axial CT scan through the suprasellar region showed a small lobulated contrast-enhancing structure adjacent to the posterior clinoid process (Figure 1).

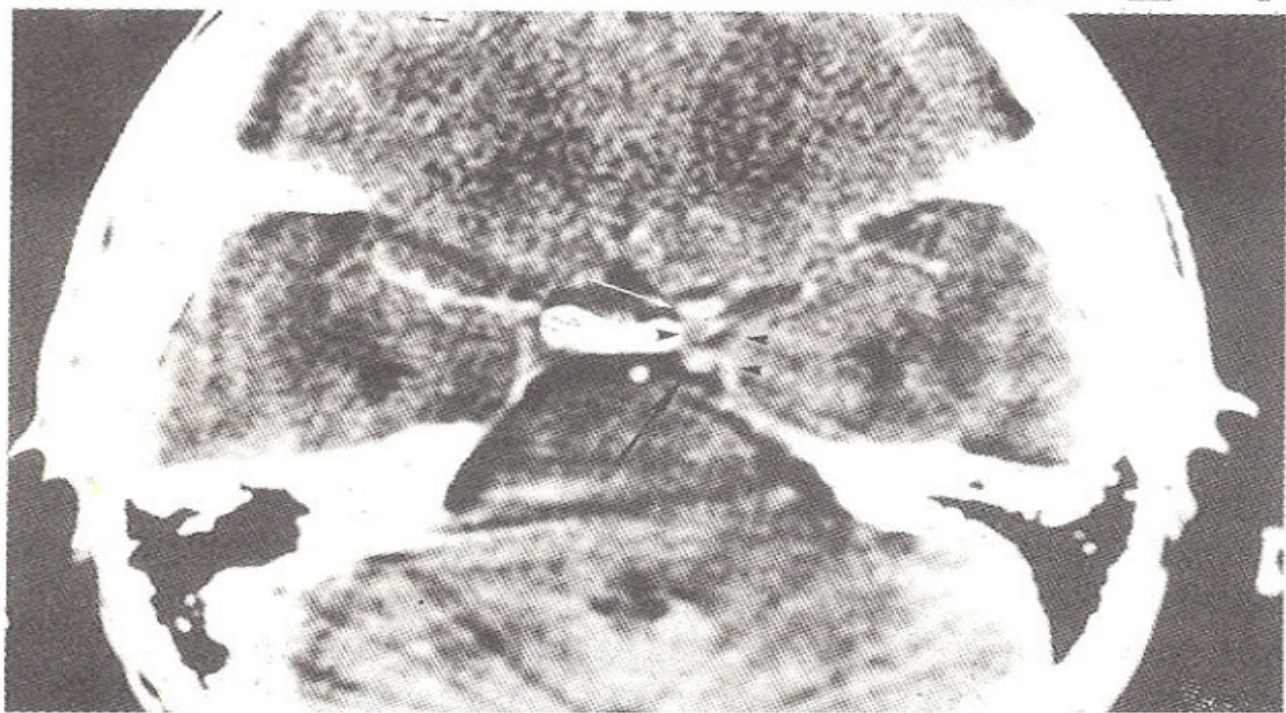
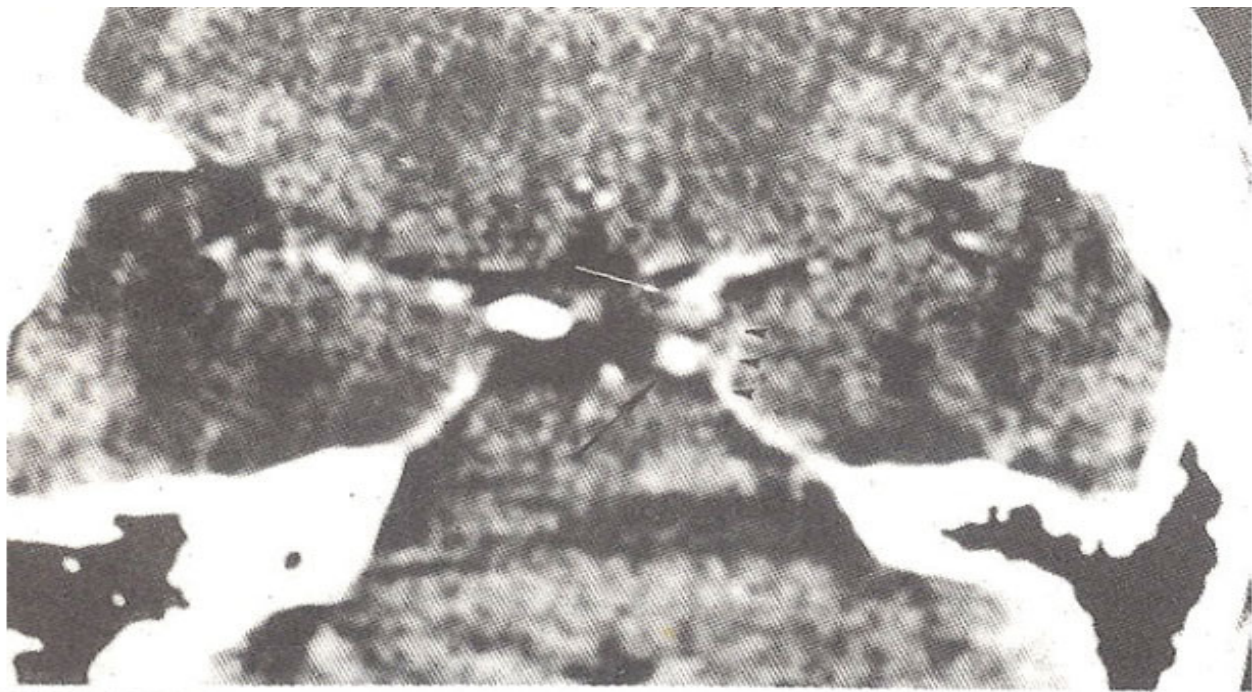


Figure 1. Case 1: Axial CT scans through the suprasellar region showing lobulated contrast-enhancing aneurysm (arrow). The structure appears to be arising from the back of the internal carotid artery (white arrow). In B, note the impaction of the aneurysm between the left posterior clinoid process (arrowhead) and tentorial edge (arrowheads) both covered with pain-sensitive dura.

No blood was seen in the subarachnoid space. Left carotid angiogram (Figure 2) showed a posterior communicating artery aneurysm on the internal carotid artery. A weak saccule projected posteriorly from the fundus of the aneurysm (Figure 2).

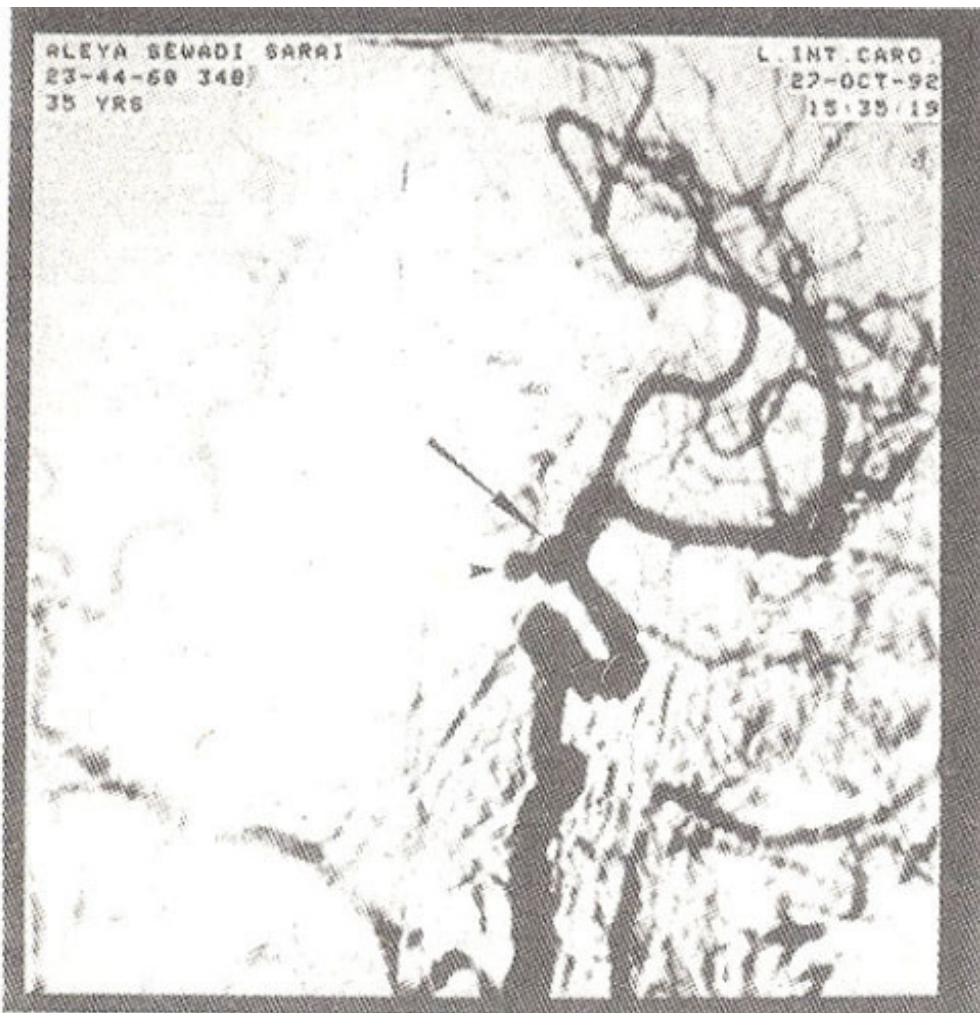


Figure 2. Case 1: Lateral projection of the arteriogram showing posteriorly projecting aneurysm (arrow). Note the weak posterior protrusion (arrowhead) which bled at operation and the enlargement of which probably coincided with the change in the character of headache.

At operation, an unruptured internal carotid artery aneurysm medial to the edge of the tentorium was clipped (Figure 3).

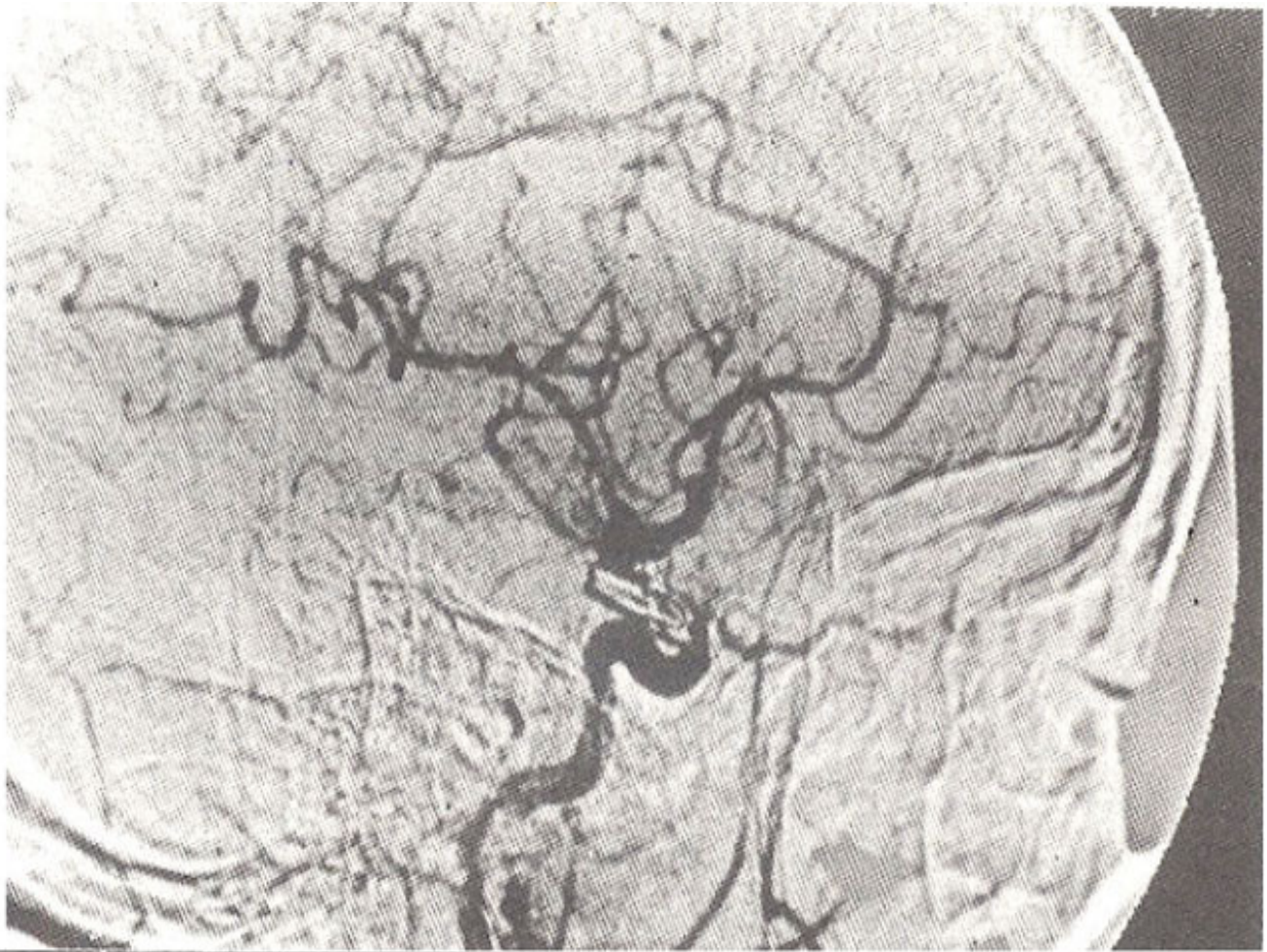


Figure 3. Case 1: Postoperative angiogram shows aneurysm clipped. Complete relief of longstanding migraine followed clipping of the aneurysm.

Notable operative findings included: (a) a tightly impacted aneurysm with a lobulated configuration, located medial to the tentorial edge; (b) a thin walled, angry-looking saccular protrusion from the fundus of the aneurysm that started leaking while arachnoid was being freed from the base of the aneurysm. Postoperative course and 3 year follow-up period was marked by complete resolution of the severe preoperative head pain and longstanding migrainous headaches. Recovery of the left oculomotor nerve palsy was complete 4 months after the operation.

Case 2

A 30-year old woman, who had suffered from paroxysmal left sided headaches for many years, was admitted because of increased severity of head and orbital pain that failed to respond to medical treatment for 3 weeks. Left third nerve palsy was noted a day before admission. CT scan of the brain was unremarkable but thin suprasellar cuts were not carried out. Examination revealed an alert patient complaining of severe left-sided head pain, but otherwise in good health. Third nerve palsy was present. Left carotid angiogram showed a posterior communicating artery aneurysm that projected posteriorly and had a lobulated configuration (Figure 4).

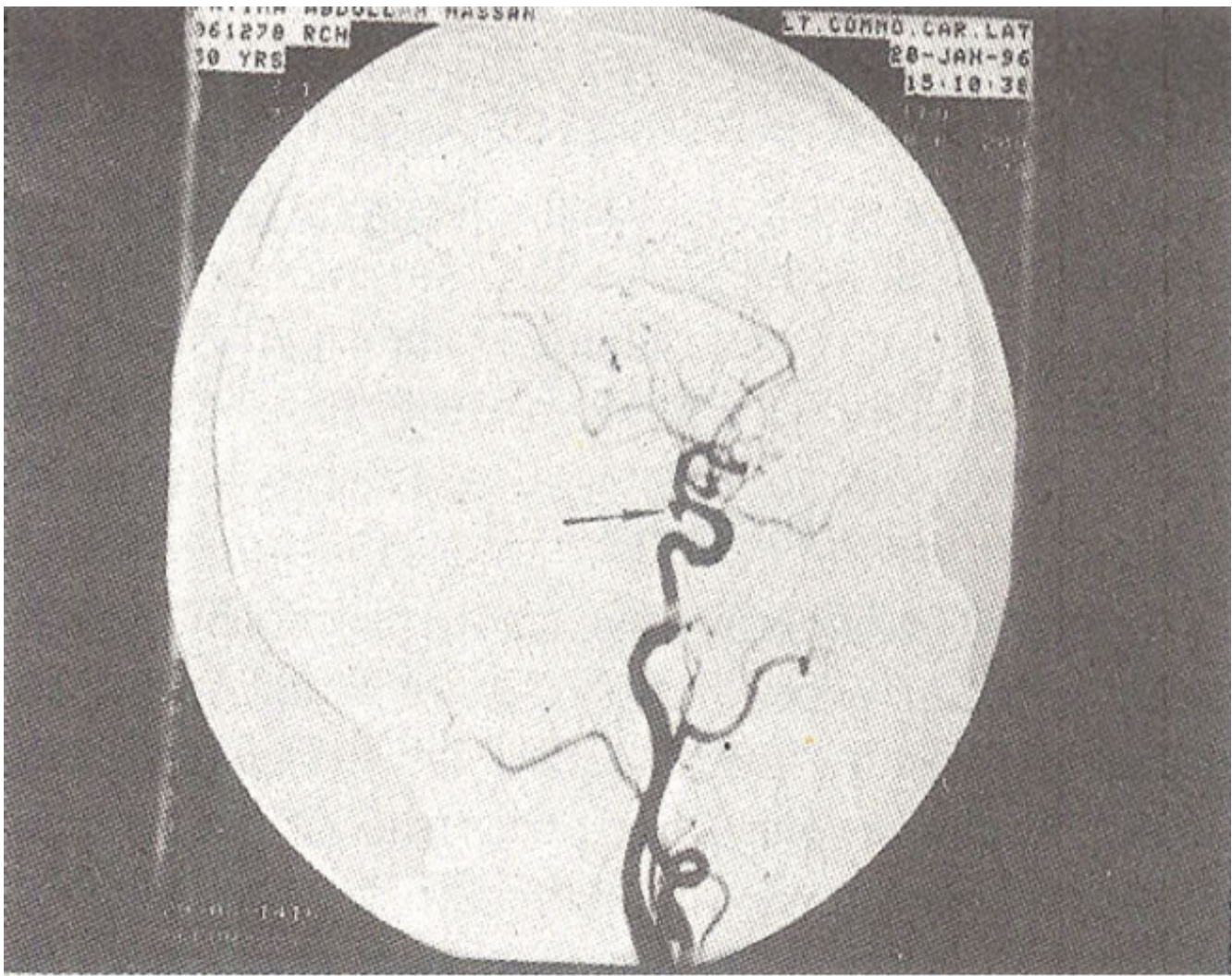


Figure 4. Case 2: Lateral projection of the arteriogram showing a small lobulated aneurysm projecting posteriorly (arrow).

Operative findings were similar to the first case. The dome of the aneurysm as well as a thin-walled pulsating lobule were located in a tight space medial to the tentorial edge. The weak, thin-walled saccule started leaking just before the application of the clip, in spite of minimal manipulation. Postoperatively, the patient did well and volunteered a complete relief of her unilateral headache. Return of third nerve function began soon after surgery with complete recovery by 3 months postoperatively.

Discussion

In typical migraine, a physical or surgically treatable cause is rarely found and angiography to rule out an intracranial vascular anomaly is seldom carried out. We, along with others⁴⁻⁹, feel that intracranial aneurysms may, at times, cause a migraine headache syndrome, especially with atypical features. These atypical features of migraine that should arouse suspicion include: beginning of migraine after the age of 40 years⁵, sudden change in the character of the migraine⁶, transient weakness of one arm and leg or oculomotor nerve^{5,9}. In our series of 50 patients who underwent aneurysm surgery, only two were diagnosed and pre-emptively operated in the pre-rupture stage on the basis of atypical migraine.

However, careful questioning in the remaining 48 elicited a history of antecedent headache, distinct from that associated with the subarachnoid hemorrhage (SAB) in 19 (39%) patients, with orbital pain in nine (19%) patients. Similar migrainous history is common in patients with unruptured but symptomatic cerebral aneurysms¹⁰. Change in the nature and severity of longstanding headache, as noticed in our patients, may be caused by aneurysmal dilatation and enlargement leading to stretching or compression of the nerves or local pressure effect on the sensitive dura¹¹. The headaches may be periodic or even migrainous in nature^{8,11,12}. Walton⁹ believed that the most common site of an aneurysm associated with migraine headache is the internal carotid posterior communicating artery and in 150 cases of SAH, Magee¹² found 2% with antecedent history of ophthalmoplegic migraine. Local anatomical peculiarities of certain aneurysms provide the anatomico-physiological basis for the development of atypical migraine syndrome. For example, in both of our cases, the posterior communicating artery aneurysm was located in a very tight space medial to the tentorial edge where the fundus was in contact and pulsating against the sensitive basal dura as well as the third nerve. An enlarged thin walled lobule indicating recent dilatation was noticed in both the cases. The basal dura in this region is richly supplied by the sensory nerve endings from the first division of trigeminal nerve. The pain caused by the pressure/pulsation of the aneurysm against this dura is therefore referred to the distribution of the ophthalmic division of the trigeminal nerve and at times becomes truly hemicranial. Alteration in the character of the headache may be an ominous sign⁶ and may indicate an impending rupture as was confirmed by the operative findings of our cases. The possible mechanisms involved in the causation of headache by the unruptured aneurysms and change in character of headache prior to frank rupture are reviewed by others¹³ and include: (a) referred pain by traction or pressure of the aneurysm on the tentorial edge (first division trigeminal nerve supply); (b) distension of the aneurysmal sac stimulating periarterial sensory fibres. The true incidence of symptomatic unruptured aneurysms is not known¹⁴, but their number is not insignificant¹⁰. Prompt referral of this group of patients with suggestive symptoms including atypical migraine, to specialized centres for angiographic evaluation and, possible treatment in the pre-rupture stage is the only way in which significant advances in reducing the overall mortality and morbidity of this serious condition can be achieved.

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