

CASE REPORT

Unruptured anterior communicating artery aneurysms presenting with seizure: Report of three cases and review of literature

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ABSTRACT

Aneurysms generally present with bleed and epileptogenic aneurysms are rare. Unruptured epileptogenic anterior communicating artery aneurysms are extremely rare and anecdotal. We present three patients with unruptured anterior communicating artery aneurysms who presented with seizures and were surgically managed. Seizure might be related to the large size, presence of thrombus, microbleeds and surrounding gliosis. We suggest that large thrombosed anterior communicating artery aneurysms should be considered in the differential diagnosis of patients presenting with late onset of seizure and having a suprasellar lesion on imaging. Surgical clipping offers a fair chance of seizure freedom in selected patients.

Key words: Aneurysms, epilepsy, unruptured

Introduction

Unruptured intracranial aneurysms (UIA) seldom present with seizures and are generally not considered in the differential diagnosis of late of onset seizures. Among epileptogenic unruptured incidental aneurysms, the common locations are middle cerebral artery and internal carotid artery- posterior communicating artery.^[1-3] Epileptogenicity is thought to be related to the size of the aneurysm and giant thrombosed aneurysms are predisposed to present with seizures.^[4,5] We present three cases of large unruptured anterior communicating artery aneurysms presenting with seizures and discuss the management issues.

Case Reports

The three reference cases constituted 0.45% of the total 662 intracranial aneurysms surgically managed in our department during the period January 2005 to December 2010. Rupture of aneurysms was excluded in all cases by history,

symptomatology, clinical examination, imaging, and intraoperative confirmation of the absence of cisternal clots or xanthochromic staining. Management recommendations were based on the current recommendation for unruptured aneurysms to prevent future hemorrhage.^[6]

Case I

This 61-year-old male, with no history of any comorbid factors, presented with history of focal twitching over the left half of face for the last six months. He gave no history of aura, generalization or involvement of limbs. General, systemic, and neurological examination was unremarkable and scalp electroencephalogram (EEG) did not show any epileptiform discharges. Magnetic Resonance Imaging (MRI) of brain showed a well defined globular lesion in the suprasellar area, 13 × 12 × 11 mm in size, hypointense on T1, T2 with peripheral hyperintensity on T2 [Figure 1]. Computerized tomographic angiogram (CTA) head revealed a saccular aneurysm arising from the anterior communicating artery of size 15 × 12 mm, with neck of 5 mm. Digital subtraction angiography (DSA) confirmed the presence of the saccular aneurysm measuring 10.8 × 6.8 mm directed antero-inferiorly with a neck of 5.2 mm [Figure 2]. Intraoperatively, the aneurysm was found to be large, thrombosed with a small flowing part. The aneurysm was surgically managed. The rim of cortex around the aneurysm appeared compressed but not gliotic, and hence was not removed. The patient is presently seizure free on 2 years follow-up on a single antiepileptic.

Case II

This 57-year-old right handed gentleman, hypertensive since 5 years, presented with one episode of generalized tonic clonic

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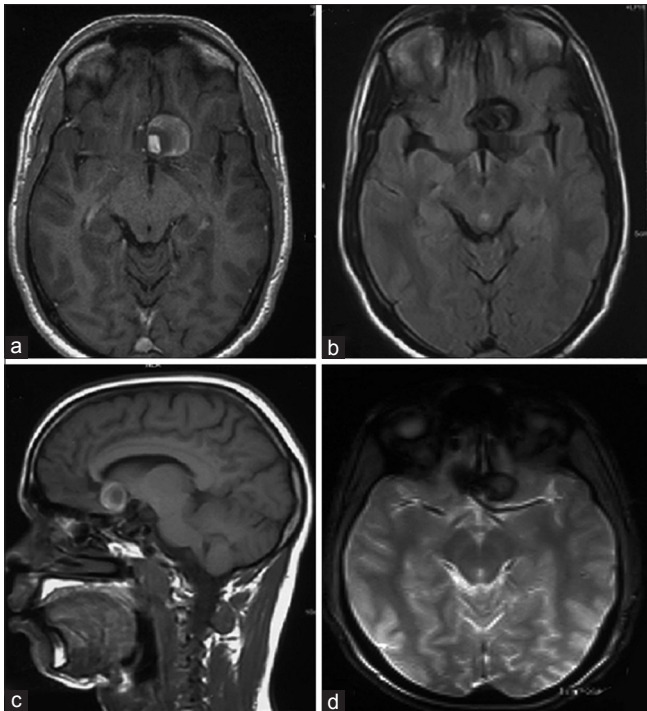


Figure 1: (a) T1w axial Post Contrast- MRI showing a partially thrombosed aneurysm having a laminated appearance with peripheral hyperintensity and central hypointensity. (b) Gradient echo axial- MRI images showing various intensity of blooming in a large anterior communicating artery aneurysm suggestive of different stages of blood products. (c) T1W sagittal plain MR images showing a suprasellar partially thrombosed aneurysm. (d) PDI-Intermediate weighted axial MR image, showing a partially thrombosed aneurysm with various blood products in the suprasellar region

seizure early in morning while getting up from bed. He had no significant past medical history including any history of seizures. General, systemic, and neurological examination was unremarkable, and scalp EEG did not show any epileptiform discharges. MRI and CT brain [Figure 3] showed a well defined hyper dense lesion of size 20×16 mm in the suprasellar region. DSA revealed a partially thrombosed, lobulated aneurysm, the flowing part having a size 7.19×6.66 mm arising from the anterior communicating artery, directed inferiorly, with a neck of 3.2 mm. Patient underwent thrombectomy and surgical clipping of the aneurysm. The brain surrounding the aneurysm appeared gliotic and was partially removed. The patient has remained seizure free on one year follow-up on antiepileptic medication.

Case III

This 35-year-old right handed gentleman, without any past history of medical illness, presented with history of two episodes of seizures in the last one year. His seizure semiology was suggestive of aura of epigastric pain followed by generalized tonic-clonic movement of all four limbs followed by loss of consciousness, without any post ictal confusion or focal neurological deficit. He gave no significant past history and his routine investigations results were

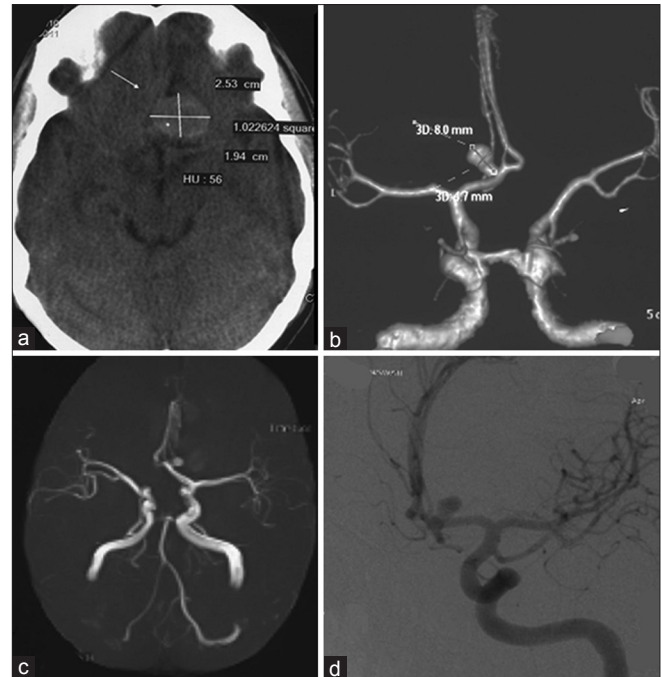


Figure 2: (a) CT brain Plain axial images showing a well defined hyperdense lesion in the suprasellar region. (b) CT angiogram (CTA) volume rendered images showing a saccular aneurysm arising from the anterior communicating artery directed superolaterally towards left. Right anterior cerebral artery is hypoplastic. (c) CTA-Maximum intensity projection showing a saccular aneurysm arising from the anterior communicating artery with above said findings. (d) 4 Vessel DSA Left Frontal Oblique- A saccular aneurysm arising from anterior communicating artery with superolateral projection

also normal. Scalp EEG did not show any epileptiform discharges. CT brain plain showed a hyperdense lesion anterior to the sella on left side from the midline. MRI brain revealed a 24×18 mm saccular aneurysm arising from anterior communicating artery which was hypointense on T1-T2 with peripheral hypointensity on T2 and with evidence of thrombus. CT angiography confirmed the presence of a partially thrombosed, multilobulated, anterior communicating artery aneurysm in the left A1-A2 junction, and directed superolaterally with a size of $11 \times 5 \times 6$ mm and neck of 4.5 mm [Figure 4]. He underwent left pterional craniotomy and clipping of aneurysm with removal of thrombus and excision of sac. The cortex surrounding the aneurysm did not look abnormal and was not removed. Presently, he is seizure free for the last 6-month on a single antiepileptic drug.

Discussion

Seizures may occur as a secondary complication after subarachnoid hemorrhage.^[3,6] But epileptic seizure as the primary and sole presenting symptom of an unruptured intracranial aneurysm (UIA) is a rare occurrence. Currie *et al.* (0.2%), Morley and Barr (10.7%), Jomin *et al.* (8%), Raps *et al.* (2.7%) have all reported seizures in UIA with varying

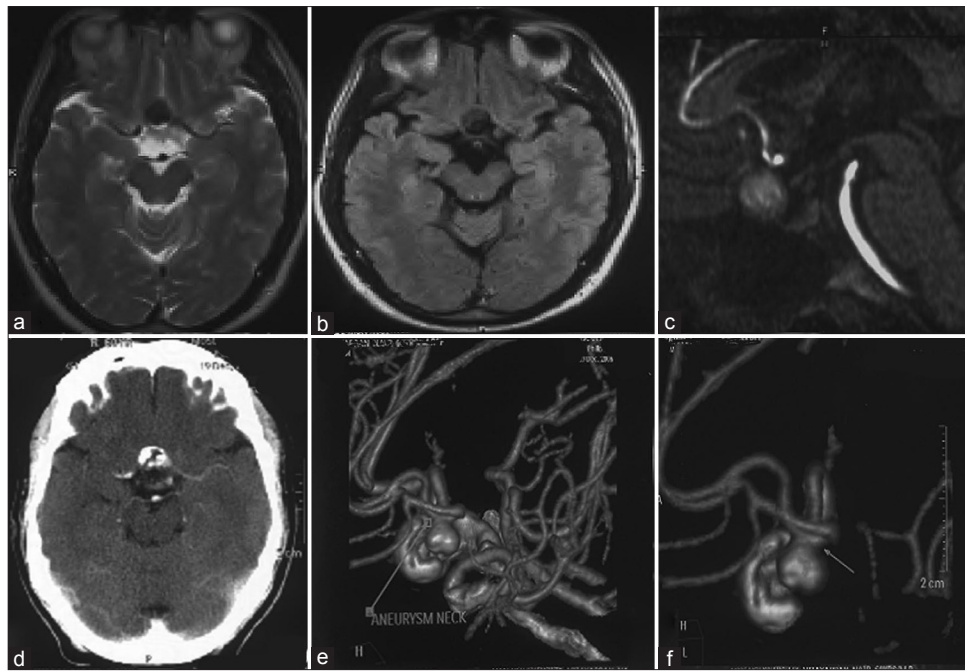


Figure 3: (a) T2axial MR images showing a well defined lesion in the suprasellar region with hypointense periphery and hyperintensity along the base. (b) FLAIR MR images showing the same lesion with peripheral hyperintensity and central hypointensity (blood products with lumen). (c) Sagittal MR post contrast images showing a globular suprasellar lesion suggestive of an aneurysm. (d) Brain Post contrast Axial images showing a hyperdense contrast enhancing lesion with central hypodensity in the suprasellar region. (e and f) D- CTA surface guided display image showing a saccular aneurysm with irregular wall and narrow neck directed anteroinferiorly arising from the anterior communicating artery

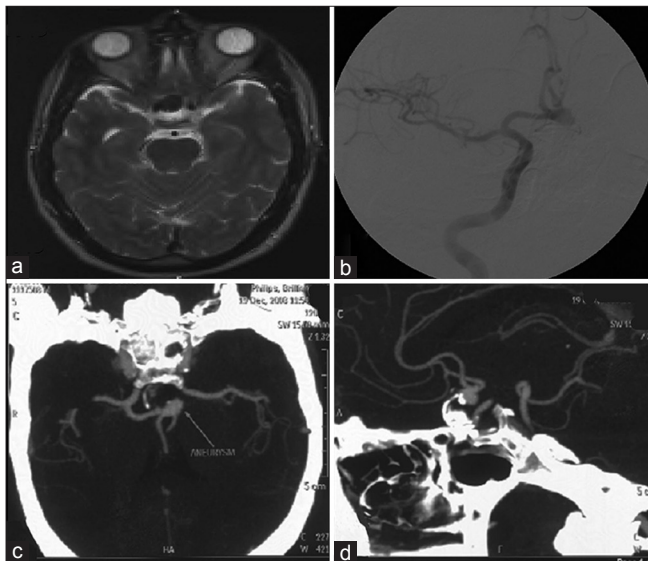


Figure 4: (a) T 2 Axial MR images showing a well defined hypointense lesion in the suprasellar region with minimal peripheral hyperintensity. (b) 4 Vessel DSA images showing a saccular anterior communicating artery aneurysm projecting inferiorly and laterally. (c) CT Angiogram - Maximum projection axial images showing a saccular aneurysm projecting inferiorly with a calcified portion seen peripherally. (d) CTA-sagittal images showing a saccular aneurysm directed postero-inferiorly-with a peripheral rim of calcification around the thrombus

incidence.^[7-11] Steiger *et al.*, too have provided a comprehensive analysis of all the reported cases of epileptogenic aneurysms adding nine of their own cases.^[1]

Site is a major determinant and Sahs *et al.* report that 8% of unruptured posterior communicating artery aneurysms and 36% of middle cerebral artery aneurysms present with seizures.^[12] Liang-fu and Da-jie *et al.*,^[13] reported epilepsy in two cases, both located at the middle cerebral artery and Sengupta *et al.* reported six cases of seizures in unruptured MCA aneurysms.^[14] Anterior communicating aneurysms presenting with seizures is a rare observation with very few reported cases.^[2,4] Our series of three patients probably is perhaps the largest series of patients with unruptured anterior communicating artery aneurysms presenting with seizures.

Size is another important predictor with large and giant aneurysms predisposed to manifest with seizures.^[1,3-5] In Solomon's series of 202 patients with unruptured cerebral aneurysms of size larger than 1 cm², 5% presented with seizures.^[11] In our series, all the three aneurysms were large, but none of them were giant.

Though various hypothesis have been proposed for the genesis of seizures in UIA, its mechanism is still not clear it. Steigers *et al.* postulate that epileptogenesis results from irritation of the mesial temporal structures by the aneurysm which can happen especially when the ICA is elongated, the tentorial edge is abnormally flat, or the aneurysm is large.^[2] Intermittent compression of underlying cortical tissue due to the pulsation of UIA is thought to be another potential cause for epileptogenicity.^[1] Large and giant aneurysms

cause direct cortical compression (mass effect) leading to the focal gliosis of cerebral tissue which may also result in epilepsy.^[2,5] We found a rim of gliotic cortex in only one of our patients. Sengupta *et al.* suggest that recurrent micro bleeds from the aneurysm with deposition of various blood products around lead to the focal destruction of the cerebral tissue and further development of epileptic focus.^[14] Focal ischemic lesions secondary to thrombotic emboli arising from aneurysm is one suggested etiology for the origin of seizure. Unruptured aneurysms are known to present with minor strokes and TIA especially when large and have intramural thrombus. Steiger also postulate that some of these minor strokes could in fact be minor seizures, and thus the incidence of epilepsy in patients with unruptured aneurysms may be underestimated.^[2,15]

The choice of clipping over coiling is a matter of debate and the management strategy depends on the experience of the treating surgeon. Surgery, theoretically has an advantage as aneurysm is physically removed or at least reduced thereby reducing the irritative focus. However, the added risk of seizures following any intracranial surgery negates this advantage. The need for removal of the perifocal gliosis as is done for epileptogenic cavernomas is again not proven. All the three patients are seizure free although we removed the gliotic brain in only one patient.

Conclusion

Large thrombosed unruptured anterior communicating artery aneurysms can occasionally present with seizures. Seizure might be related to the large size, presence of thrombus, microbleeds, and surrounding gliosis. Such aneurysms should be considered in the differential diagnosis of patients presenting with late onset of seizure and having a suprasellar lesion on imaging. Surgical clipping offers a fair chance of seizure freedom in selected patients.

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