

Warning leak of intracranial aneurysm masquerading as sinus node dysfunction: A case report

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Abstract

We describe the successful endovascular repair of an intracranial aneurysm causing subarachnoid hemorrhage in a 62-year-old man, who was initially diagnosed and treated as a case of symptomatic sinus bradycardia. The aim of this report and following discussion is to discuss the subtle warning signs of intracranial aneurysm that may masquerade as sinus node dysfunction.

Keywords

Ruptured intracranial aneurysm, sinus node dysfunction, endovascular repair

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Introduction

An aneurysm is an abnormal bulge or focal outpouching of arterial wall that causes substantial morbidity and mortality. Commonest histo-pathological abnormality responsible for aneurysmal bulging is decrease in muscular tunica media in arterial wall. Autopsy studies showed a prevalence rate of 1%–5% in adults.¹ Cerebral aneurysms occur both sporadically as well as familial with other systemic diseases like autosomal dominant polycystic kidney disease, Marfan syndrome, fibromuscular dysplasia, and Ehlers–Danlos syndrome.²

An unruptured cerebral aneurysm is mostly asymptomatic and detected incidentally on neuroimaging studies. An unruptured aneurysm causes symptoms by compressing neighboring neural parenchyma like cranial nerve palsies or brain stem dysfunction.³ An aneurysm may rupture and cause life-threatening subarachnoid hemorrhage (SAH). Ignorance of warning symptoms, initial misdiagnoses, and late referral may lead to fatal outcome in these patients.⁴ For better outcome of the patients with aneurysmal SAH, one has to address the subtle warning signs of the unruptured aneurysms. Recognition of these signs as an important warning symptom of unruptured aneurysm may lead to its treatment in the pre-rupture stage, thus improving the overall prognosis. SAH, if occurs, is more common in female (female:male=2:1),⁵ and peak incidence is seen in sixth decade of life.⁶ Treatment depends on mode and acuity of presentation. Incidentally

detected aneurysm requires observation or elective intervention to occlude the aneurysm, depending on the clinical profile of patient. For ruptured aneurysm, treatment requires control of bleeding and repairing the aneurysm. Endovascular treatment with coil embolization is now the first-line treatment in the management of ruptured aneurysms.⁷

Case

A 62-year-old Indian male presented to the emergency department with complaints of one episode of presyncope 2 days back. He also had history of syncope about 2 weeks prior to presyncope with trivial trauma to neck. He did not complain of any abdominal or chest symptoms, and there was no history of nausea, vomiting, headache, dizziness, blurring of vision, or focal neurological deficit. He first sought medical attention at secondary care center where he was detected to have sinus bradycardia (Figure 1(a)). He was diagnosed as a case of sick sinus syndrome and referred to our center for permanent pacemaker implantation. The past, personal, and family history was noncontributory. On general physical

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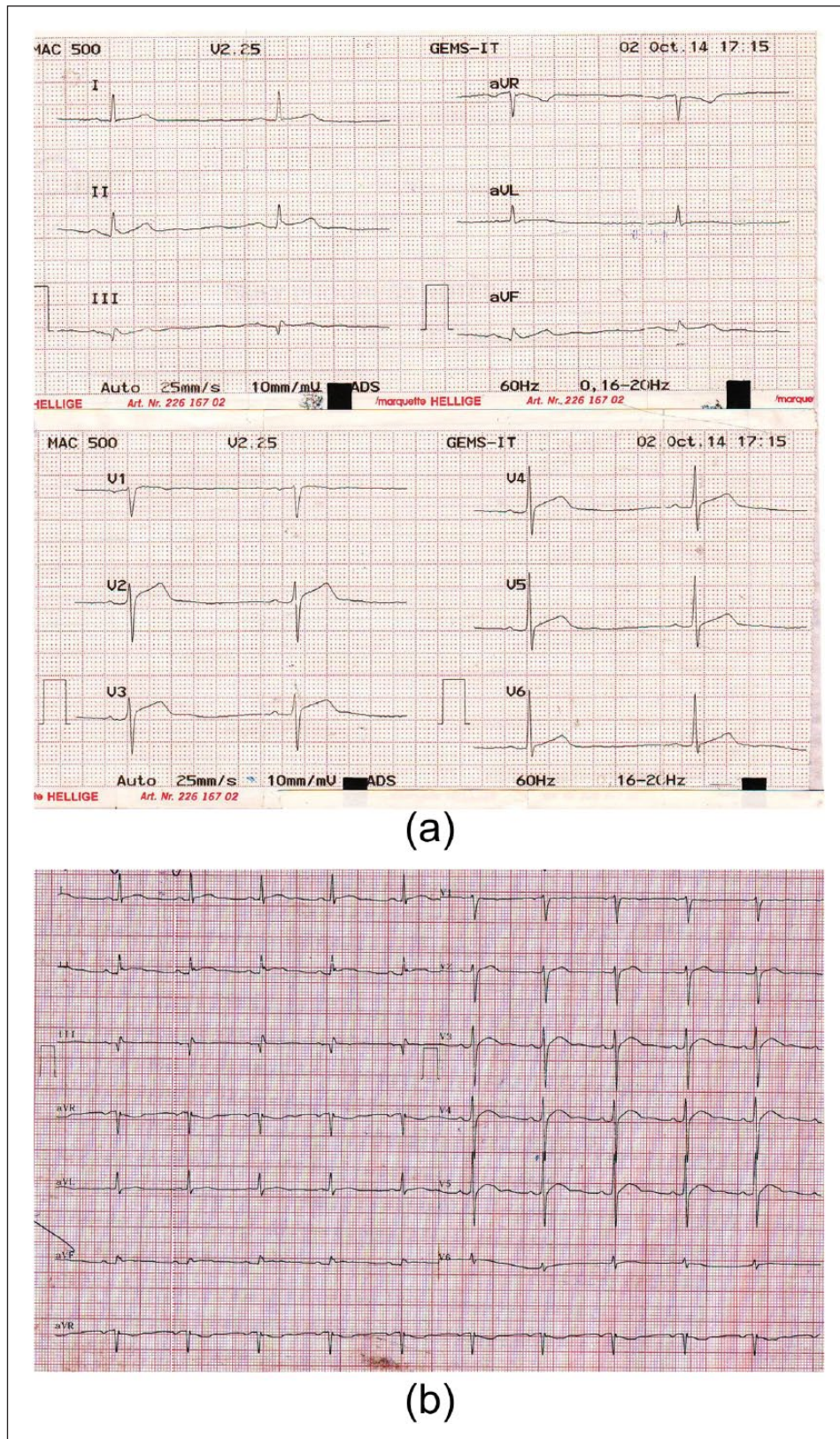


Figure I. (a) Admission electrocardiogram showing sinus bradycardia (sinus rate \approx 40 beats/min) with early repolarization in inferior limb leads II, III, aVF. (b) Pre-discharge electrocardiogram showing normalization of sinus bradycardia (sinus rate \approx 66 beats/min).

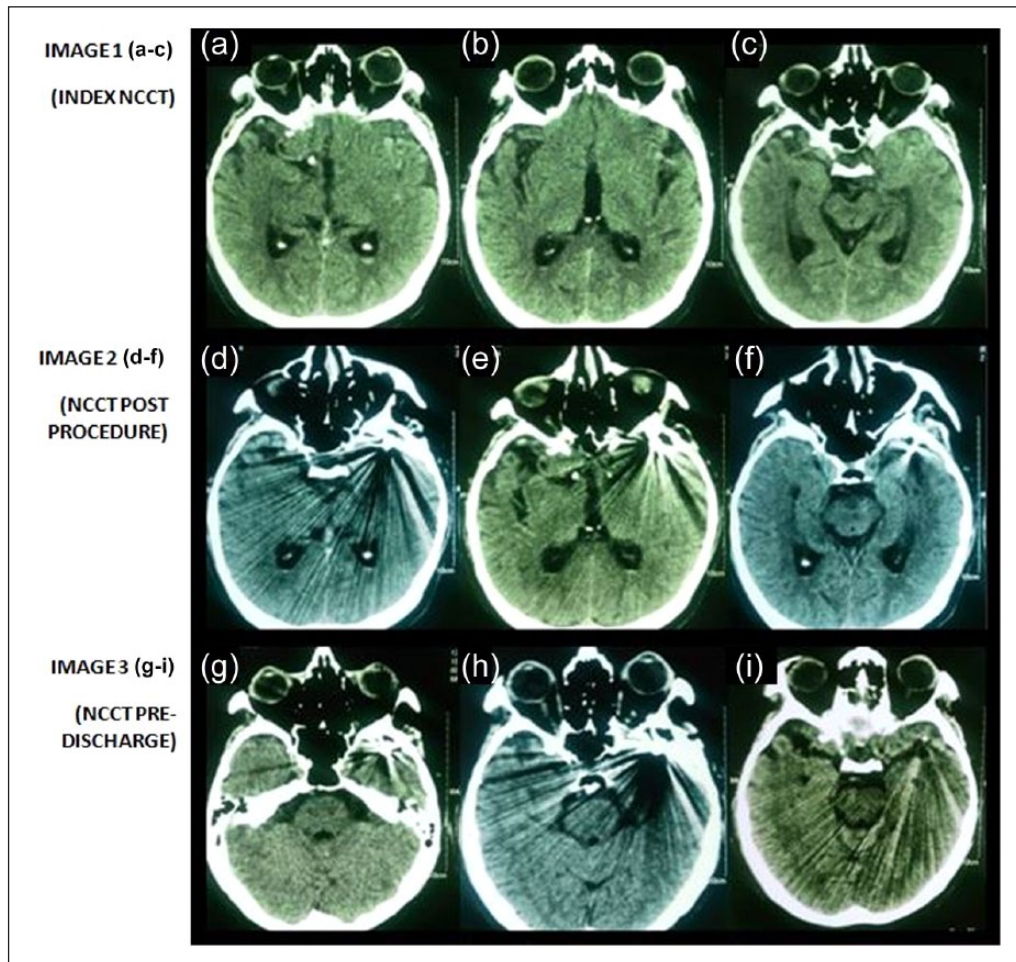


Figure 2. Image 1 (a–c) is index NCCT showing round to oval focal hyperdense lesion in the left sylvian fissure, perisylvian subarachnoid leak, and senile cerebral atrophy; image 2 (d–f) and image 3 (g–i) are post procedural NCCTs showing metal artifacts originating from the coils in left fronto-temporal region causing beam hardening artifacts.

examination, the patient was seen to be anxious. The heart rhythm was regular with a rate of 40 beats/min (the patient was not on any prior negative chronotropic drugs); the blood pressure was 140/90 mmHg and respiration was abdomino-thoracic with a rate of 20 breaths/min. The patient was conscious, well oriented, and there was no focal neurological deficit or neck rigidity. Findings from review of the systems, other than as reported above, were normal. Patient was admitted in the intensive cardiac care unit (ICCU) and kept on low-dose isoprenaline infusion that increased his heart rate to 50 beats/min. During his hospital stay, he was evaluated and his echocardiographic examination showed mild mitral and tricuspid regurgitation, grade 1 LV diastolic dysfunction, and normal LV systolic function (LV ejection fraction=65%). Next day morning, he complained of severe acute headache that was treated and responded to oral paracetamol. Immediate non-contrast computed tomography (NCCT) head examination was done for the evaluation of syncope and headache that confirmed focal subarachnoid leak extending to both the

ventricles and left temporal lobe. NCCT also diagnosed a well-defined round to oval focal hyperdense lesion in the left sylvian fissure with senile cerebral atrophy (Figure 2, image 1). A subsequent computed tomography (CT) cerebral angiography (Figure 3) for further evaluation of intracerebral aneurysm revealed narrow necked (2.8 mm) bilobed aneurysm ($10.9 \times 8.0 \times 6.4 \text{ mm}^3$) arising from the left middle cerebral artery (MCA) at the M2-M3 junction with the distal branches seen arising from fundus as well as the base of aneurysm. After the diagnosis of ruptured MCA aneurysm, neurology consultation was sought and he was started on cerebral decongestive therapy, antiepileptic drugs, and supportive medications. The patient was offered the choice of an endovascular or neurosurgical procedure.

Our institution does not require ethics approval for reporting individual cases. After an informed consent for the procedure, the patient was taken up for endovascular repair under general anesthesia, and by means of standard endovascular techniques, the aneurysm was occluded with detachable coils.



Figure 3. CT cerebral angiography with 3D reconstruction revealed narrow necked bilobed aneurysm arising from the left MCA at the M2-M3 junction with the distal branches seen arising from fundus as well as the base of aneurysm.

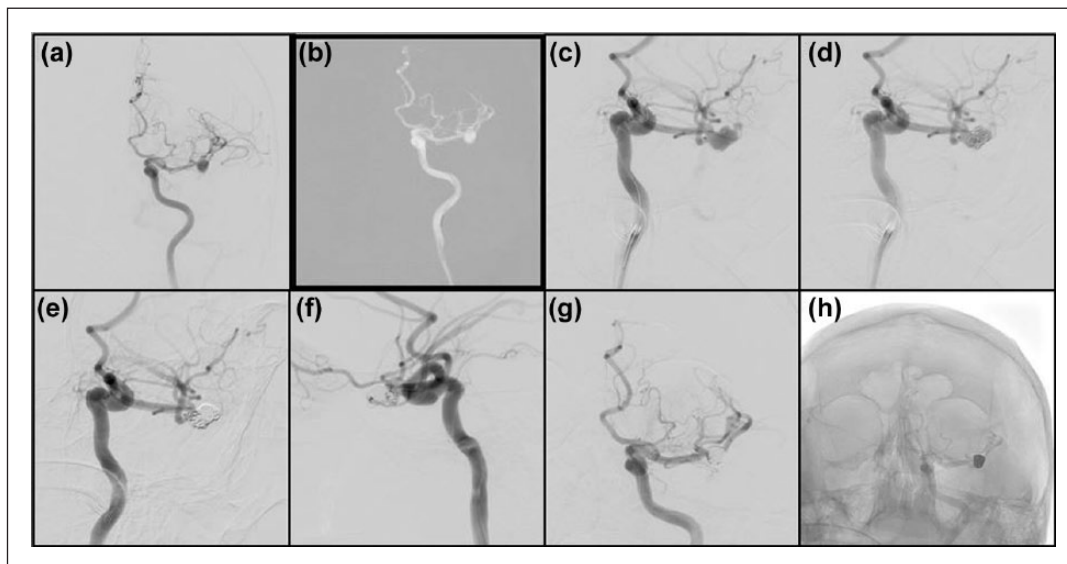


Figure 4. (a) Digital subtraction angiogram (DSA) showing aneurysm of left MCA. (b) Advancement of a guide-wire and micro-catheter in roadmap. (c and d) Deployment of platinum coils in the aneurysm. (e–h) DSA images in multiple projections showing complete obliteration of aneurysm without any leak.

A postembolization angiogram revealed complete obliteration of the aneurysm (Figure 4(a)–(h)). The patient was transferred to the ICCU for monitoring and recovered from the anesthesia with no neurological deficits. Patient was kept on oral nimodipine (120 mg/day in four equally divided doses) to prevent vasospasm of intracranial vessels.

Follow-up NCCT (Brain) performed 2 days after embolization revealed minimal intraventricular hemorrhage (IVH) with metal artifacts of the coils in the aneurysm (Figure 2, image 2). A final NCCT (Brain) and electrocardiogram done 1 day prior to discharge showed resolution of IVH (Figure 2, image 3) and normalization of cardiac rate and rhythm

(Figure 1(b)), respectively. The postprocedure stay was uncomplicated; his hemodynamics remained stable and he was discharged 8 days after endovascular repair. On 1-month and 6-month follow-up, the patient was asymptomatic; an informed consent for the publication of case report was obtained from the patient.

Discussion

Intracranial aneurysm is a focal bulge or outpouching in a blood vessel in the brain. It often looks like a berry hanging on a stem. A brain aneurysm may leak or rupture, causing

SAH. Cardiac abnormalities documented after SAH are either part of Cushing reflex^{8,9} or hypothesized to certain neurotransmitter releases that affect the heart.

Our case was initially treated as sinus node dysfunction at peripheral center, and he was referred to our center for permanent pacemaker implantation. The preceding syncope and presyncope could be warning leak from the aneurysm. He underwent cerebral imaging after the emergence of headache which showed focal subarachnoid leak at left perisylvian fissure and confirmed sinus bradycardia as a manifestation of certain neurotransmitter releases that affect the heart. His sinus bradycardia ameliorated (Figure 1(b)) after the treatment of cerebral aneurysm by coil embolization.

Endovascular intervention with detachable platinum coils is now considered to be the first-line treatment for adult patients with ruptured intracranial aneurysms. The International Subarachnoid Aneurysm Trial¹⁰ confirmed that 1-year survival was significantly better following endovascular treatment than after neurosurgical intervention. The two most frequent complications of endovascular treatment of intracranial aneurysms are thromboembolic events and intraoperative rupture.^{11,12}

After reviewing the literature, we found that there is no such case report and sinus node dysfunction is rarest presentation of ruptured cerebral aneurysm. Our case demonstrates the need for appropriate evaluation of similar patients.

Conclusion

High clinical suspicion is required for the diagnosis of SAH in patients with unusual presentation. Timely intervention can improve outcomes in this frequently devastating condition.

Declaration of conflicting interests

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References

1. Wiebers DO, Whisnant JP, Huston J III, et al. Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. *Lancet* 2003; 362: 103–110.
2. Schievink WI. Intracranial aneurysms. *N Engl J Med* 1997; 336: 28–40.
3. Connolly ES and Solomon RA. Management of unruptured aneurysms. In: LeRoux PD, Winn HR and Newell DW (eds) *Management of cerebral aneurysms*. Philadelphia, PA: Saunders, 2004, pp. 271–285.
4. Kassel NE and Drake CO. Timing of aneurysm surgery. *Neurosurgery* 1982; 10: 514–519.
5. Stapf C and Mohr JP. Aneurysms and subarachnoid hemorrhage—epidemiology. In: Le Roux PD, Winn HR and Newell DW (eds) *Management of cerebral aneurysms*. Philadelphia, PA: Saunders, 2004, pp. 183–187.
6. Greenberg MS. SAH and aneurysms. In: Greenberg MS (ed.) *Handbook of neurosurgery*. 5th ed. New York: Thieme Medical, 2000, pp. 754–803.
7. Molyneux A, Kerr R and Stratton I. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. *Lancet* 2002; 360(9342): 1267–1274.
8. Hackett JG, Abboud FM, Mark AL, et al. Coronary vascular responses to stimulation of chemoreceptors and baroreceptors: evidence for reflex activation of vagal cholinergic innervation. *Circ Res* 1972; 31(1): 8–17.
9. Jones JV. Differentiation and investigation of primary versus secondary hypertension (Cushing reflex). *Am J Cardiol* 1989; 63(6): 10C–13C.
10. Ryttefors M, Enblad P, Kerr RSC, et al. International subarachnoid aneurysm trial of neurosurgical clipping versus endovascular coiling: subgroup analysis of 278 elderly patients. *Stroke* 2008; 39: 2720–2726.
11. Pierot L, Bonafé A and Bracard S; French Matrix Registry Investigators. Endovascular treatment of intracranial aneurysms with matrix detachable coils: immediate posttreatment results from a prospective multicenter registry. *AJNR Am J Neuroradiol* 2006; 27(8): 1693–1699.
12. Pierot L, Spelle L and Vitry F; ATENA Investigators. Immediate clinical outcome of patients harboring unruptured intracranial aneurysms treated by endovascular approach: results of the ATENA study. *Stroke* 2008; 39(9): 2497–2504.