

Fig. 3. Photomicrographs of the tumour specimen showing characteristic small clusters (Zellballen) separated by delicate fibrovascular stroma. The cells had a finely granular basophilic cytoplasm (left; haematoxylin and eosin stain; original magnification $\times 100$) and were positive for chromogranin staining (right; chromogranin stain; original magnification $\times 200$). This figure is available in colour on Science Direct.

surgical excision is the only curative therapy for pheochromocytoma, but should be performed as an elective procedure after careful pre-operative preparation.

Pheochromocytoma is a rare cause of secondary hypertension. However, its diagnosis is important, as the hypertension is usually cured with resection of the tumor, whereas it is potentially lethal if left untreated and is associated with long-term morbidity. Pheochromocytoma must be considered in the differential diagnosis of hypertensive patients with signs of sympathetic hyperactivity. The patient presented here highlights the importance of careful investigation to determine the remediable causes of hypertension in children and young adults before initiating antihypertensive therapy.

References

1. Hernandez FC, Sanchez M, Alvarez A, et al. A five-year report on experience in the detection of pheochromocytoma. *Clin Biochem* 2000;**33**:649–55.
2. Chuang HL, Hsu WH, Hsueh C, Lin JN, Scott RM. Spontaneous intracerebral hemorrhage caused by pheochromocytoma in a child. *Pediatric Neurosurg* 2002;**36**:48–51.
3. Scardigli K, Biller J, Brooks MH, Cespedes LE, Posniak HV. Pontine hemorrhage in a patient with pheochromocytoma. *Arch Intern Med* 1985;**145**:343–4.
4. Moritani H, Sakamoto M, Yoshida Y, Nasu H, Nemoto R, Nakamura I. Pheochromocytoma of the urinary bladder revealed with cerebral hemorrhage. *Intern Med* 2001;**40**:638–42.
5. Fonseca V, Bouloux PM. Pheochromocytoma and paraganglioma. *Baillieres Clin Endocrinol Metab* 1993;**7**:509–44.

doi:10.1016/j.jocn.2005.04.025

Giant unruptured anterior communicating artery aneurysm presenting with seizure

Ferda Çağavi ^{a,*}, Murat Kalaycı ^a, Aysun Ünal ^b, Hüseyin Tuğrul Atasoy ^b, Zeynep Çağavi ^c, Bektaş Açıkgöz ^a

^a Department of Neurosurgery, Zonguldak Karaelmas University, Medical Faculty, Zonguldak 67600, Turkey

^b Department of Neurology, Zonguldak Karaelmas University, Zonguldak, Turkey

^c Department of Radiology, Zonguldak Karaelmas University, Zonguldak, Turkey

Received 19 January 2005; accepted 18 April 2005

* Corresponding author. Tel.: +90 372 2610169/4136; fax: +90 372 2610155.

E-mail address: cagavif@yahoo.com (F. Çağavi).

Abstract

Giant aneurysms of the anterior communicating artery (ACoMA) are rare. The clinical presentation of giant ACoMA aneurysms is usually associated with the mass effect of the space-occupying lesion or with subarachnoid haemorrhage. A giant ACoMA aneurysm presenting with a seizure has only been reported twice previously. We report a 70-year-old female patient, in whom a single seizure was the only symptom of a giant ACoMA aneurysm, with no neurological deficit. The diagnosis of unruptured giant ACoMA aneurysm was made with cranial CT, MRI and angiography. The patient refused surgical intervention, was treated with anti-epileptic therapy and has been asymptomatic for 7 months. We suggest that elderly patients presenting with a first seizure need detailed evaluation and giant aneurysms, which may be confused with other intracranial space-occupying lesions, need to be considered in the differential diagnosis.

© 2006 Elsevier Ltd. All rights reserved.

Keywords: Anterior communicating artery aneurysm; Giant aneurysm; Seizure

1. Introduction

Aneurysms larger than 2.5 cm in diameter are classified as giant aneurysms. Giant aneurysms account for approximately 5% (range, 3–13%) of all intracranial aneurysms¹ and the frequency of giant aneurysms is higher in women (female:male, 3:1).² Giant aneurysms generally present with neurological symptoms due to mass effect or with subarachnoid haemorrhage.^{3–5} Mass effect occurs in 50% of patients.⁵ Cranial nerve palsies are the most common clinical finding and have been reported in 43–47% of cases.^{3,5} Seizures are rare.^{2,5} In the series reported by Bull,⁶ four of 22 giant intracranial aneurysms presented initially with epilepsy. Solomon et al.⁷ reported epilepsy in six of 49 giant unruptured intracranial aneurysms located outside the cavernous sinus.

We report a female patient who presented with a seizure and who was diagnosed with an unruptured giant anterior communicating artery (ACoMA) aneurysm.

2. Case report

A 70-year-old female patient was admitted to the Neurosurgery Department, Zonguldak Karaelmas University Hospital, after a single generalized seizure 1 week prior. She had no headache or other neurological symptoms. Her medical history was significant for coronary artery bypass surgery 1 year earlier, hypertension for 10 years and diabetes mellitus for 3 years. Neurological examination was normal. Routine laboratory tests were all within normal limits. Direct anteroposterior and lateral X-rays revealed an anterobasal round, calcified lesion (Fig. 1). A calcified mass in the suprasellar region was seen on CT scan (Fig. 2). There was no subarachnoid, subdural or intraparenchymal haemorrhage or hydrocephalus. A 40 × 33 × 35 mm hyperintense lesion with an isointense centre on T₁-weighted images and which was hypointense on T₂-weighted images was seen on MRI (Fig. 3). Angiography revealed a giant ACoMA aneurysm that was filling from the right (Fig. 4). The size of the aneurysm on angiography was smaller than that on MRI, thus the aneurysm was mostly thrombosed. Surgical inter-

vention or embolization was planned, but the patient refused treatment. Phenytoin, 300 mg orally, was used to prevent further seizures and the patient was discharged on the 7th day. She has been asymptomatic for 7 months.

3. Discussion

Giant aneurysms have a propensity to occur at certain locations. The internal carotid and middle cerebral arteries, including the intracavernous carotid, are the most common sites,^{4,5,8} but giant aneurysms arising from the ACoMA are rare, with a reported frequency ranging between 0% and 33.5%.^{1–5,8–10} In a series of 56 cases of giant aneurysm, Vorkapic et al. found no ACoMA aneurysms.⁵ Yasargil,¹ in a series of 30 cases, and Jaspán et al.,⁹ in a series of 18 cases, also reported no ACoMA aneurysms. Olsen et al. reported one patient with a giant aneurysm arising from the ACoMA in a series of 15 (7%).³ Rosta et al. reported 110 patients with giant aneurysm, of which 13% arose from the anterior cerebral artery complex; however, the frequency of giant aneurysms arising from the ACoMA was not specified.⁴ In other papers, the frequency of giant aneurysms arising from the ACoMA has been reported to be 12.5%² and 17%.⁸ Of 49 patients with unruptured giant aneurysm, Solomon et al. reported that six (12%) arose from the anterior cerebral artery.⁷ The highest frequency, 33.5%, was reported by Sahs et al.¹⁰ In childhood, the frequency of giant ACoMA aneurysms has been reported to be 10%.¹¹ It is accepted that ACoMA aneurysms have a higher risk of rupture.¹² Although the frequency of ACoMA localization is higher for small (<12 mm) or large (12–24 mm) intracranial aneurysms, giant ACoMA aneurysms are uncommon. This may be due to the higher rate of rupture of smaller ACoMA aneurysms.

Most aneurysms presenting with seizures are located on the middle cerebral artery.^{13–16} Of 151 patients with unruptured aneurysms, epilepsy occurred in 36% of patients with an aneurysm of the middle cerebral artery and in 8% of patients with an aneurysm of the posterior communicating cerebral artery.¹⁴ Morley and Barr described 28 patients with giant aneurysms: three had seizures and, in all those

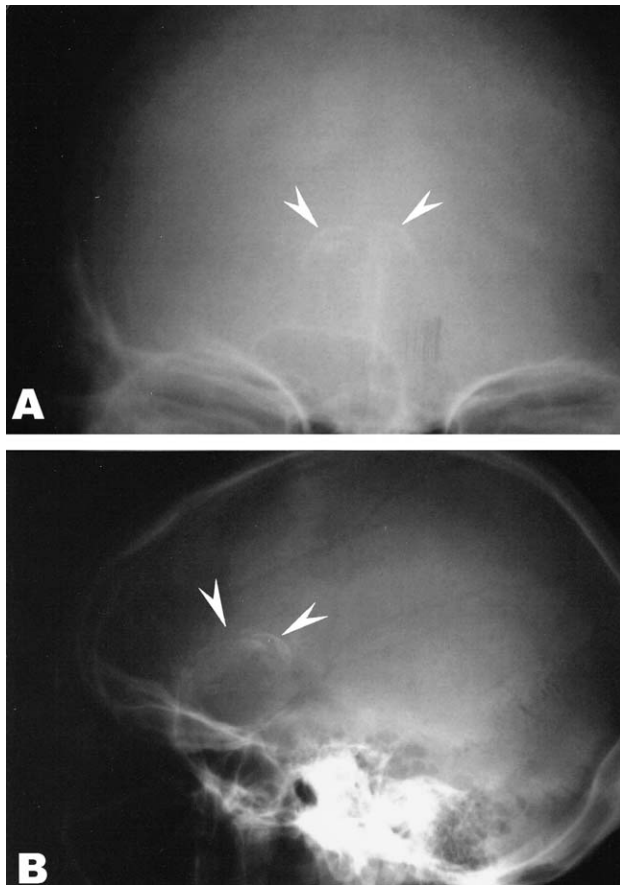


Fig. 1. Anteroposterior (A) and lateral (B) X-rays demonstrating a frontobasal calcified lesion (arrows).

patients, the aneurysm was located on the middle cerebral artery.¹⁵ In a report of 21 patients with giant aneurysms, Liang-fu and Da-jie reported epilepsy in two patients both



Fig. 2. Cranial CT scan without contrast enhancement revealed a calcified lesion in the suprasellar region.

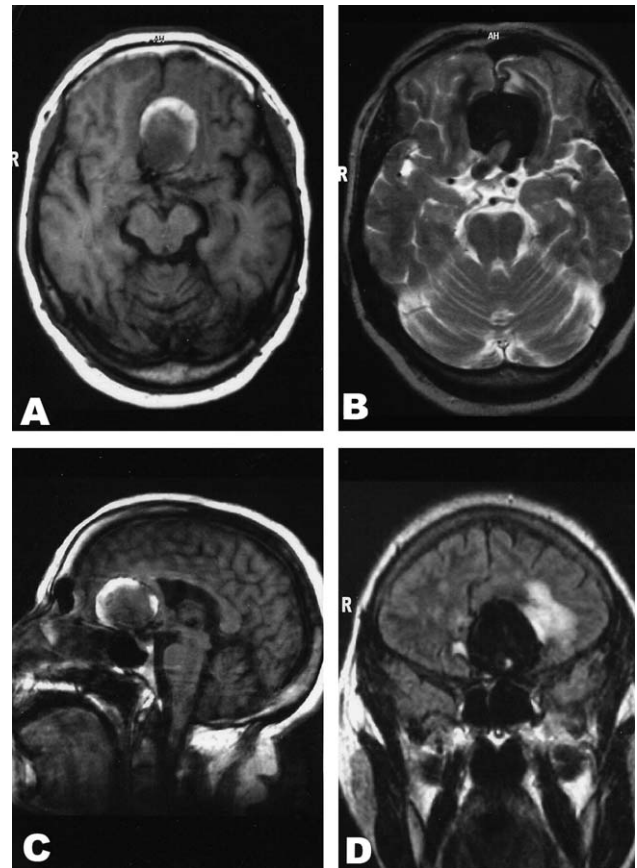


Fig. 3. Axial T₁-weighted (A), axial T₂-weighted (B), sagittal T₁-weighted (C) and coronal FLAIR (D) MRI of the brain demonstrating a giant saccular aneurysm.

of whom had aneurysms of the middle cerebral artery.¹³ Sengupta et al. reported six patients with seizures and unruptured aneurysms.¹⁶ In all cases, the aneurysms were located on the middle cerebral artery. Ellamushi et al. presented two patients, one with an aneurysm on the middle cerebral artery,¹⁷ and Sena et al. also reported an aneurysm of the middle cerebral artery with epileptic seizures.¹⁸

Giant AComA aneurysms have been reported to present with subarachnoid haemorrhage,¹⁹ visual loss,^{19–23} bitemporal hemianopsia,²⁴ dementia,¹⁹ anosmia,²⁵ headache,^{22,23,26,27} frontal lobe psychotic syndrome,²⁸ loss of consciousness,^{20,22} progressive neurological deficits,²⁹ aphasia³⁰ and blindness.³¹ The most commonly reported clinical presentations are subarachnoid haemorrhage, dementia and visual field deficits.¹⁹ In a series of 18 patients, Lownie et al. reported that eight (44%) presented with subarachnoid haemorrhage, six (33%) presented with dementia due to mass effect and three (17%) presented with clinical findings of optic nerve or chiasm compression. One asymptomatic patient was diagnosed when a calcified mass was found on X-ray.¹⁹ Giant aneurysms of the AComA rarely present with seizures. To our knowledge, there are only two prior reports of patients presenting with seizures in the literature;^{32,33} however, seizure was not the only symptom in these two

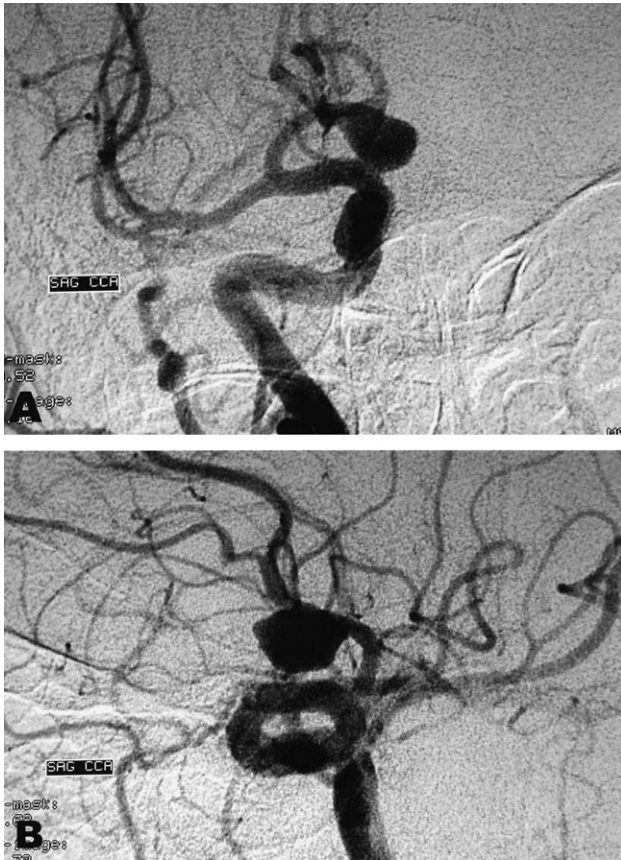


Fig. 4. Right anteroposterior (A) and lateral (B) carotid angiography showing an anterior communicating artery aneurysm.

patients. In one, right hemiparesis and expressive aphasia were accompanying signs,³³ whereas in the other, the accompanying sign was a visual field deficit.³² The case reported here is the only patient presenting with seizure without any accompanying neurological signs.

Although the mechanism for the seizures due to intracranial giant aneurysm is not known, some hypotheses may be put forward. Liang-fu and Da-jie suggested that in two of their patients, the calcified walls of the aneurysms may have acted as hamartomas, causing epilepsy.¹³ Another possibility is that aneurysms may leak without producing clinical manifestations of subarachnoid haemorrhage. These minor leakages may produce localized damage to the brain and produce an epileptogenic focus. Whittle et al. described four patients with complex partial seizures for several years, who were shown to have giant aneurysms of the middle cerebral artery.³⁴ None had a history of subarachnoid haemorrhage. Operative findings included compression of the mesiotemporal cortex and evidence of previous haemorrhage from the aneurysms. In two patients, clipping of the aneurysm with decompression of the adjacent temporal lobe cured the seizure disorder. In the other two patients, the aneurysms could only be wrapped and the seizures continued.³⁴ Recurrent subclinical bleeding, was also suggested by Sengupta et al.¹⁶ to have provoked localized brain damage, producing an epileptogenic focus. Ellamushi et al. concluded that the

aneurysm of the middle cerebral artery in one of their patients was too small to cause local pressure effects.¹⁷ On the basis of the presence of gliotic brain tissue surrounding the aneurysm, they suggested that subclinical haemorrhage may have been the mechanism of the seizures. In the second patient reported by Ellamushi et al., the aneurysm was giant, invaginating the mesial temporal lobe with no calcification in the wall and, in this case, they concluded that local pressure produced by the aneurysm on the temporal lobe may have been the cause of the seizures.¹⁷ Leibrok et al. reported a patient with seizures due to a thrombosed basilar artery apex aneurysm.³⁵ These authors postulated that emboli from this aneurysm travelled through the posterior cerebral artery and its branches to the temporal lobe, with resultant microinfarction and subsequent development of an epileptogenic focus. As surgery was not performed in the patient in this report, we cannot confirm gliosis around the aneurysm. The cause of the seizure may have been the mass effect of the calcified giant aneurysm.

The frequency of calcification in giant aneurysms has been reported to be between 16% and 39%.^{5,9} Although total thrombosis of giant aneurysms is rarely reported (3%),⁴ the incidence of partial thrombosis ranges between 27% and 74%.^{3,4} Thrombosis in giant aneurysms of the AComA has been reported more frequently.¹⁹ Lownie et al. have recorded partial thrombosis in 14 cases (78%) in their series.¹⁹ Partial thrombosis presents as a medium–high signal intensity on T₁- and T₂-weighted MRI.³ Partial thrombosis was also present in our case and was well defined by hyperintensity on the T₁-weighted MRI.

In conclusion, the late occurrence of seizures in elderly patients with no other neurological findings may be a symptom of a giant AComA aneurysm. We suggest that elderly patients presenting with a first seizure need detailed evaluation and giant aneurysms, which may be confused with other intracranial space-occupying lesions, need to be considered in the differential diagnosis.

References

1. Yasargil MG. Clinical considerations, surgery of the intracranial aneurysms and results. *Microneurosurgery*, vol. 2. First ed. Stuttgart, New York: Georg Thieme Verlag; 1984.
2. Hosobuchi Y. Direct surgical treatment of giant intracranial aneurysms. *J Neurosurg* 1979;**51**:743–56.
3. Olsen WL, Brant-Zawadzki M, Hodes J, Norman D, Newton TH. Giant intracranial aneurysms: MR imaging. *Radiology* 1987;**163**:431–5.
4. Rosta L, Battaglia R, Pasqualin A, Beltramello A. Italian cooperative study on giant intracranial aneurysms: 2. Radiological data. *Acta Neurochir Suppl (Wien)* 1988;**42**:53–9.
5. Vorkapic P, Czech T, Pendl G, Ozturk E, Horacek A. Clinicoradiological spectrum of giant intracranial aneurysms. *Neurosurg Rev* 1991;**14**:271–4.
6. Bull J. Massive aneurysms at the base of the brain. *Brain* 1969;**92**:535–70.
7. Solomon RA, Fink ME, Pile-Spellman J. Surgical management of unruptured intracranial aneurysms. *J Neurosurg* 1994;**80**:440–6.
8. Creissard P, Godlewski J, Tadie M, Freger P, Thiebot J, Tayot J. Les anévrismes géants. *Neurochirurgie* 1980;**26**:309–53.

9. Jaspán T, Wilson M, O'Donnell H, Worthington BS, Holland IM. Magnetic resonance imaging with even-echo rephasing sequences in the assessment and management of giant intracranial aneurysms. *Br J Radiol* 1988;**61**:351–7.
10. Sahs AL, Perret GE, Locksley HB, Nishioka H. *Intracranial Aneurysms and Subarachnoid Haemorrhage. A Cooperative Study*. Philadelphia: Lippincott; 1969.
11. Aydin IH, Onder A, Kadioglu HH. Giant intracranial aneurysms in children and adolescents. *Zentralbl Neurochir* 1993;**54**:35–8.
12. Inagawa T, Hada H, Katoh Y. Unruptured intracranial aneurysms in elderly patients. *Surg Neurol* 1992;**38**:364–70.
13. Liang-fu Z, Da-jie J. Large and giant intracranial aneurysms: Diagnosis and surgical treatment. *Chin Med J* 1987;**100**:392–7.
14. Locksley HB. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations. *J Neurosurg* 1966;**25**:321–68.
15. Morley TP, Barr HW. Giant intracranial aneurysms: Diagnosis, course, and management. *Clin Neurosurg* 1969;**16**:73–94.
16. Sengupta RP, Saunders M, Clarke PR. Unruptured intracranial aneurysms: An unusual source of epilepsy. *Acta Neurochir (Wien)* 1978;**40**:45–53.
17. Ellamushi H, Thorne L, Kitchen N. Unruptured cerebral aneurysms causing seizure disorder (report of two cases). *Seizure* 1999;**8**: 310–2.
18. Sena JC, Reynier Y, Alliez B. Unruptured intracranial aneurysm presenting with epileptic seizure. *Arq Neuropsiquiatr* 2003;**61**: 663–7.
19. Lownie SP, Drake CG, Peerless SJ, Ferguson GG, Pelz DM. Clinical presentation and management of giant anterior communicating artery region aneurysms. *J Neurosurg* 2000;**92**:267–77.
20. Burde RM. Visual loss 'at stool'. *Surv Ophthalmol* 1984;**29**: 215–20.
21. Nakahara I, Taki W, Nishi S, Shibata O, Kikuchi H. Treatment of giant anterior communicating artery aneurysm via an endovascular approach using detachable balloons and occlusive coils. *AJNR Am J Neuroradiol* 1990;**11**:1195–7.
22. Sato T, Komatsu S, Sakurai Y, Suzuki J. A case of giant aneurysm at the anterior communicating artery treated by direct surgery. *No Shinkei Geka* 1980;**8**:93–8.
23. Yoshida S, Ezuka I, Ohtsuka A. Follow-up study of the giant aneurysm by CT scan after unilateral carotid ligation. *No Shinkei Geka* 1981;**9**:1083–6.
24. Hojer-Pedersen E, Haase J. Giant anterior communicating artery aneurysm with bitemporal hemianopsia: case report. *Neurosurgery* 1981;**8**:703–6.
25. Manconi M, Paolino E, Casetta I, Granieri E. Anosmia in a giant anterior communicating artery aneurysm. *Arch Neurol* 2001;**58**:1474–5.
26. Bas MB, Guerra N, Valsania V, Boccardo M. Giant intracranial aneurysm of the anterior communicating artery treated by direct surgical approach. Case report. *J Neurosurg Sci* 2000;**44**: 133–6.
27. Sjaastad O, Saunte C, Fredriksen TA, et al. Cluster headache-like headache, Hageman trait deficiency, retrobulbar neuritis and giant aneurysm. Autonomic function studies. *Cephalalgia* 1988;**8**: 111–20.
28. Bokemeyer C, Frank B, Brandis A, Weinrich W. Giant aneurysm causing frontal lobe syndrome. *J Neurol* 1990;**237**:47–50.
29. Schaller B, Lyrer P. Focal neurological deficits following spontaneous thrombosis of unruptured giant aneurysms. *Eur Neurol* 2002;**47**: 175–82.
30. Iwashita T, Tanaka Y, Koyama J, Kobayashi S, Hongo K, Kobayashi S. A giant thrombosed aneurysm of the anterior communicating artery which developed 18 years after clipping: A case report. *No Shinkei Geka* 2001;**29**:427–31.
31. Ito H, Shima T, Yamamoto S. Giant anterior communicating artery aneurysm. *No Shinkei Geka* 1975;**3**:171–6.
32. Gruber A, Killer M, Bavinski G, Richling B. Clinical and angiographic results of endosaccular coiling treatment of giant and very large intracranial aneurysms: A 7-year, single-center experience. *Neurosurgery* 1999;**45**:793–804.
33. Roitberg BZ, Cochran EJ, Thornton J, Charbel FT. Giant anterior communicating artery aneurysm infiltrated with a primary cerebral lymphoma: Case report. *Neurosurgery* 2000;**47**:458–62.
34. Whittle IR, Allsop JL, Halmagyi GM. Focal seizures: An unusual presentation of giant intracranial aneurysms. A report of four cases with comments on the natural history and treatment. *Surg Neurol* 1985;**24**:533–40.
35. Leibrock LG, Bennett DR, Bloch S. Complex partial seizures associated with unruptured thrombosed basilar artery apex aneurysm. *Surg Neurol* 1983;**19**:17–20.

doi:10.1016/j.jocn.2005.04.024

Multiple intracranial aneurysms presenting with epistaxis

S. Honeybul^{a,b,*}, S. Barker^a, C. Poitelea^a, A. Ditchfield^a

^a Wessex Neurological Centre, Southampton General Hospital, UK

^b Sir Charles Gairdner Hospital, 356 West Coast Highway, Scarborough, Perth, Western Australia, 6019, Australia

Received 6 April 2005; accepted 3 May 2005

Abstract

Although epistaxis secondary to rupture of a post-traumatic, false aneurysm is well documented, true intracavernous carotid artery aneurysms presenting with epistaxis are very rare. We present such a case in a patient with multiple aneurysms. The aneurysm was

* Corresponding author. Tel.: +61 8 9245 6483; fax: +61 8 9346 3824.
E-mail address: stephen.honeybul@health.wa.gov.au (S. Honeybul).