

Case Report

Posterior communicating artery aneurysm rupture mimicking apoplexy

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Abstract

Background: Cerebral aneurysm rupture can lead to devastating neurological complications and present a complex problem to treat. We report a unique case of a ruptured posterior communicating artery (PCoA) aneurysm presenting with sudden and complete vision loss.

Case Description: A 39-year-old man presented with the acute onset of severe headache and complete bilateral vision loss. The patient described headaches for several months prior to presentation. However, prior to the day of presentation, he had no visual disturbance. A CT angiogram (CTA) and magnetic resonance imaging (MRI) of the brain revealed a 1.6-cm, non-contrast enhancing suprasellar mass, eccentric to the left side, consistent with hemorrhagic mass. There was no obvious aneurysm or vascular malformation. The sella tursica was normal in appearance. The patient was taken for an immediate endoscopic endonasal transtuberulum approach for optic nerve decompression. Hematoma without an associated tumor was encountered and partially evacuated before aborting with resultant partial improvement in vision. A subsequent cerebral angiogram revealed an irregularly shaped, postero-laterally pointing, 2.5-mm left PCoA aneurysm. The patient was then taken for open clipping of the ruptured aneurysm. A large, fibrinous capsule was found over the superolateral aspect of the aneurysm. The ruptured aneurysm was secured with clips and the surrounding hematoma was evacuated.

Conclusion: In the immediate postoperative period, the patient regained vision in the nasal field of his right eye. This case illustrates a unique presentation of a ruptured PCoA aneurysm, and thus must be considered in the differential diagnosis of a suprasellar hemorrhage resulting in visual loss in absence of a recognizable associated tumor.

Key Words: Apoplexy, posterior communicating artery aneurysm, ruptured cerebral aneurysm

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INTRODUCTION

Rupture of a cerebral aneurysm can lead to devastating

consequences for a patient. Treatment of the aneurysm as well as the sick patient is very challenging. The majority of cerebral aneurysms are found in the anterior

circulation, and specifically, the posterior communicating artery (PCoA) is one of the most common locations. Classically, these aneurysms point postero-laterally, and rupture spills subarachnoid hemorrhage into the sylvian fissure, the basal cisterns, and the intrahemispheric fissure. There have also been reported cases of subdural hematoma resulting from PCoA aneurysm rupture.^[10] Patients can present with a deficits ranging from slight headache to coma to death. PCoA aneurysms can also be associated with ipsilateral oculomotor nerve palsy. We report a unique case of a ruptured PCoA aneurysm mimicking pituitary apoplexy, presenting with sudden and complete vision loss.

CASE REPORT

A 39-year-old man presented to the emergency room after awakening with a severe headache and complete blindness in both eyes. Further questioning revealed that the patient had been complaining of intermittent headaches for approximately 1 month. He did not have any visual disturbance with the headaches. Furthermore, the patient stated that he had used both alcohol and cocaine the night prior. In addition to substance use, he also had a medical history significant for hypertension. The patient did not take any medications.

The findings of physical and neurological examinations were significant for bilateral complete vision loss without light perception. His pupils were symmetric, but did not react to light. He had full range in extra-ocular muscles and no ptosis. He was awake, oriented and conversant, and had no noticeable motor or sensory deficit.

A non-contrast computed tomography (CT) scan of the head revealed a 1.6-cm hyperdense suprasellar mass compressing the optic chiasm, eccentric to the left



Figure 1: Non-contrast, axial CT image of the head demonstrating a 1.6-cm hyperdense suprasellar mass, and a small amount of subarachnoid hemorrhage extending into the sylvian and intrahemispheric fissures

side, and a small amount of subarachnoid hemorrhage extending into the sylvian and intrahemispheric fissures [Figure 1]. A CT angiogram (CTA) did not show an associated aneurysm or vascular malformation [Figure 2]. Magnetic resonance imaging (MRI) of the brain demonstrated the same isointense, non-contrast enhancing mass, consistent with hematoma [Figure 3]. Again, no vascular abnormalities were noted. There was no evidence of diffuse subarachnoid hemorrhage on imaging and no xanthochromia on lumbar puncture. The sella turcica appeared normal.

The patient was taken to the operating room for an urgent endoscopic endonasal transtuberculum approach for optic nerve decompression. Acute hematoma was encountered in the suprasellar cistern. This was partially evacuated, but without signs of a tumor, no further exploration or complete evacuation was performed at that time due to concern for vascular pathology.

The patient then underwent a four-vessel cerebral angiogram which revealed an irregularly shaped, postero-laterally pointing, 2.5-mm left PCoA aneurysm, with the suggestion of a larger surrounding pseudoaneurysm [Figure 4].

Treatment options were discussed with the patient and his family, and he was taken for immediate clipping of the aneurysm. Intraoperatively, there was a much thickened fibrin capsule over the optico-carotid cistern obscuring the carotid artery, and brisk arterial bleeding was encountered upon opening this pseudocapsule. The small ruptured aneurysm, adherent to the tentorium, was identified and secured with a single bayoneted clip and the remainder of the suprasellar hematoma was evacuated.

In the immediate postoperative period, the patient regained vision in his right eye nasal field, with 20/30 acuity.



Figure 2: Coronal CT angiogram image without evidence of aneurysm or vascular malformation

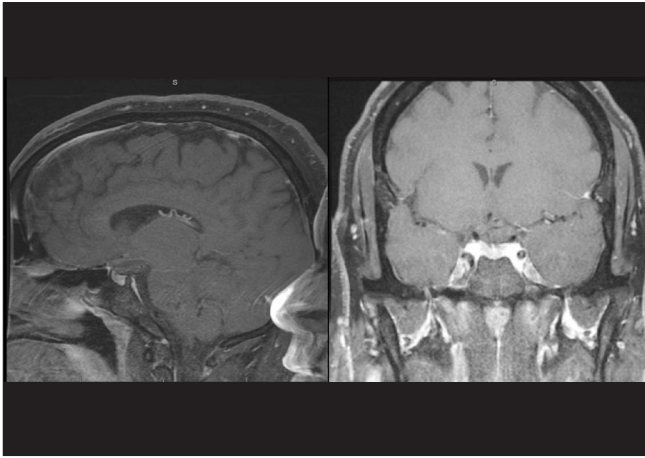


Figure 3: T1, post-contrast sagittal and coronal MR images of the brain demonstrating an isointense, non-contrast enhancing mass, consistent with hematoma

DISCUSSION

Cerebral aneurysm rupture usually presents with a constellation of symptoms including severe headache, nausea, vomiting, lethargy, and in some cases, coma or death. Rarely, an isolated acute change in vision is the predominant symptom. Cases of acute visual loss caused by cerebral aneurysms have been reported previously in the literature. Anterior communicating artery (ACoA) rupture,^[7,26] ACoA aneurysm enlargement,^[3] anterior cerebral artery (ACA) aneurysm rupture,^[2] carotid-ophthalmic aneurysm enlargement,^[11] and mycotic cavernous pseudoaneurysm enlargement^[19] have resulted in sudden monocular blindness. Carotid dissection has also caused sudden monocular loss of vision.^[13,15,16] Furthermore, ACoA artery aneurysm rupture and thrombosis has resulted in the acute onset of bitemporal hemianopsia.^[1,21]

Cerebral vascular events have also been reported to mimic pituitary apoplexy. These include internal carotid artery (ICA) aneurysm rupture,^[20,23] ACoA artery aneurysm rupture,^[22] and ICA dissection.^[18] Bilateral ICA unruptured aneurysms^[25] and thrombosis of an intracavernous aneurysm^[14] have also been reported to mimic apoplexy. Finally, there have been cases of ruptured aneurysms with associated pituitary tumors that have caused apoplexy.^[6,12,17]

In this report, however, we describe the unique case of a ruptured PCoA aneurysm mimicking pituitary apoplexy and presenting with sudden complete vision loss. This is especially unusual as PCoA aneurysms typically point laterally toward the tentorium or third nerve. The pseudocapsule formed in this case led to a “blowback” hematoma in the suprasellar space. The lack of sellar remodeling was a significant clue that an adenoma was not the source of hemorrhage. Hemorrhage into



Figure 4: Lateral cerebral angiogram image demonstrating an irregularly shaped, postero-laterally pointing, 2.5-mm left PCoA aneurysm, with the suggestion of a larger surrounding pseudoaneurysm

suprasellar tumors is rarely reported. This combined with the presence of pure hematoma and rapid recognition of no tumor on endonasal exploration was enough to prevent overly aggressive exploration and potential disaster. This diagnosis must be considered in the case of a suprasellar hemorrhage resulting in visual loss in the absence of a clearly recognizable associated tumor. Caution and good fortune during the endonasal exploration led to a good outcome in this case. Aneurysmal rupture during this case could have been catastrophic. Proximal control at the level of the paraclinoid or parasellar carotid artery could have been obtained from the transsphenoidal approach,^[8,9] but might not have been sufficient for control of a ruptured aneurysm.

Retrospectively, this patient should have received a cerebral angiogram as the CT scan did not reveal a noticeable pituitary mass. However, the CTA was negative and the presentation was thought to be clinically consistent with apoplexy. This was an unusual case and the CTA was not sufficient to rule out vascular pathology. CT angiography has a reported sensitivity between 91 and 100% depending on the aneurysm size,^[4,5,24] and formal angiography needs to be performed in a setting such as this where suspicion is high. Without a perfect match between clinical presentation and imaging, more initial investigation would have been useful and could have prevented a nearly catastrophic event. If presented with a similar unusual case in the future with a questionable vascular etiology, a cerebral angiogram is recommended, even with an unrevealing CTA.

CONCLUSIONS

Pituitary apoplexy and subarachnoid hemorrhage can have very similar presentations. One should always be considered in the differential of the other. Practically, any

aneurysm in the Circle of Willis can cause vision loss with rupture, including PCoA aneurysms. If suspicion is high, formal angiography is recommended prior to surgical exploration.

REFERENCES

- Aoki N. Partially thrombosed aneurysm presenting as the sudden onset of bitemporal hemianopsia. *Neurosurgery* 1988;22:564-6.
- Bakker SL, Hasan D, Bijvoet HW. Compression of the visual pathway by anterior cerebral artery aneurysm. *Acta Neurol Scand* 1999;99:204-7.
- Caprioli J, Fagadau W, Lesser R. Acute monocular visual loss secondary to anterior communicating artery aneurysm in a patient with sickle cell disease. *Ann Ophthalmol* 1983;15:873-6.
- Chen W, Wang J, Xin W, Peng Y, Xu Q. Accuracy of 16-row multislice computed tomographic angiography for assessment of small cerebral aneurysms. *Neurosurgery* 2008;62:113-21; discussion 121-2.
- Chen W, Wang J, Xing W, Xu Q, Qiu J, Huang Q, et al. Accuracy of 16-row multislice computerized tomography angiography for assessment of intracranial aneurysms. *Surg Neurol* 2009;71:32-42.
- Chuang CC, Chen YL, Pai PC. A giant intracavernous carotid artery aneurysm embedded in pituitary macroadenoma presenting with pituitary apoplexy. *Cerebrovasc Dis* 2006;21:142-4.
- Cullen JF, Haining WM, Crombie AL. Cerebral aneurysms presenting with visual field defects. *Br J Ophthalmol* 1966;50:251-6.
- Germanwala AV, Zanation AM. Endoscopic endonasal approach for clipping of ruptured and unruptured paraclinoid cerebral aneurysms: Case report. *Neurosurgery* 2011;68 Suppl 1:S234-9.
- Kassam AB, Gardner PA, Mintz A, Snyderman CH, Carrau RL, Horowitz M. Endoscopic endonasal clipping of an unsecured superior hypophyseal artery aneurysm. Technical note. *J Neurosurg* 2007;107:1047-52.
- Kondziolka D, Bernstein M, Ter Brugge K, Schutz H. Acute subdural hematoma from ruptured posterior communicating artery aneurysm. *Neurosurgery* 1988;22:151-4.
- Kuzniecky R, Melmed C, Schipper H. Carotid-ophthalmic aneurysm: an uncommon cause of acute monocular blindness. *CMAJ* 1987;136:727-8.
- Laidlaw JD, Tress B, Gonzales MF, Wray AC, Ng WH, O'Brien JM. Coexistence of aneurysmal subarachnoid haemorrhage and pituitary apoplexy: Case report and review of the literature. *J Clin Neurosci* 2003;10:478-82.
- Lee SK, Kwon SU, Ahn H, Kim JS. Acute isolated monocular blindness and painless carotid artery dissection. *Neurology* 1999;53:1155-6.
- Locatelli M, Spagnoli D, Caroli M, Isalberti M, Branca V, Gaini SM, et al. A potential catastrophic trap: An unusually presenting sellar lesion. *Eur J Neurol* 2008;15:98-101.
- Lubin J, Capparella J, Vecchione M. Acute monocular blindness associated with spontaneous common carotid artery dissection. *Ann Emerg Med* 2001;38:332-5.
- Miranda M, Venegas P, Kagi M. [Blindness caused by an ischemic optic neuropathy by spontaneous carotid dissection. Report of a case]. *Rev Med Chil* 2003;131:1042-4.
- Okawara M, Yamaguchi H, Hayashi S, Matsumoto Y, Inoue Y, Okawara S. [A case of ruptured internal carotid artery aneurysm mimicking pituitary apoplexy]. *No Shinkei Geka* 2007;35:1169-74.
- Provenzale JM, Hacein-Bey L, Taveras JM. Internal carotid artery dissection associated with pituitary apoplexy: MR findings. *J Comput Assist Tomogr* 1995;19:150-2.
- Quisling SV, Mawn LA, Larson TC 3rd. Blindness associated with enlarging mycotic aneurysm after cavernous sinus thrombosis. *Ophthalmology* 2003;110:2036-9.
- Romano A, Chibbaro S, Marsella M, Ippolito S, Benericetti E. Carotid cavernous aneurysm presenting as pituitary apoplexy. *J Clin Neurosci* 2006;13:476-9.
- Schisano G. Large anterior communicating artery aneurysm with temporal hemianopsia. Case report. *J Neurosurg Sci* 1985;29:267-71.
- Shahlaie K, Olaya JE, Hartman J, Watson JC. Pituitary apoplexy associated with anterior communicating artery aneurysm and aberrant blood supply. *J Clin Neurosci* 2006;13:1057-62.
- Suzuki H, Muramatsu M, Murao K, Kawaguchi K, Shimizu T. Pituitary apoplexy caused by ruptured internal carotid artery aneurysm. *Stroke* 2001;32:567-9.
- Tipper G, U-King-Im JM, Price SJ, Trivedi RA, Cross JJ, Higgins NJ, et al. Detection and evaluation of intracranial aneurysms with 16-row multislice CT angiography. *Clin Radiol* 2005;60:565-72.
- Torres A, Dammers R, Krisht AF. Bilateral internal carotid artery aneurysm simulating pituitary apoplexy: Case report. *Neurosurgery* 2009;65:E1202.
- Umredkar AA, Singla N, Gupta SK. Ruptured anterior communicating artery aneurysm presenting with monocular blindness. *Neurol India* 2009;57:826-8.