CASE REPORT

Acute expansion of an asymptomatic posterior communicating artery aneurysm resulting in oculomotor nerve palsy

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SUMMARY

Acute expansion of an intracranial aneurysm is an extremely rare event. In this report, we describe the unusual radiographic documentation of acute expansion of a posterior communicating artery (PCOM) aneurysm from 4 to 8 mm, resulting in complete oculomotor nerve palsy. A 40-year-old man was admitted to our institution with a 3-week history of persistent occipital headaches but was otherwise neurologically intact. CT angiography demonstrated a 4 mm left PCOM aneurysm. Digital subtraction angiography performed several hours following presentation demonstrated an expansion in aneurysm size to 8 mm with new-onset complete oculomotor nerve palsy. The aneurysm was successfully obliterated by clip ligation. Although growth in aneurysm size is often a progressive phenomenon, acute expansions of aneurysms can occur.

BACKGROUND

Intracranial aneurysms can be a major source of mortality and morbidity, especially when presenting as a ruptured aneurysm with associated subarachnoid haemorrhage (SAH). Posterior communicating artery (PCOM) aneurysms represent one of the most commonly encountered intracranial aneurysms within the basal cisterns. The most common symptomatic presentation of PCOM aneurysms is oculomotor nerve palsy (ONP),^{1 2} which can develop secondary to either progressive expansion in aneurysm size or, less commonly, to acute rupture of an aneurysm. While aneurysms can progressively expand in size over time as part of their natural history, acute expansion in size is an extremely rare phenomenon. To the best of our knowledge, there is no radiographic documentation of acute expansion of intracranial aneurysm in the current literature. We hereby present a unique case of radiographic documentation of the acute expansion of a PCOM aneurysm with subsequent ONP.

CASE PRESENTATION

A 40-year-old man with a history of untreated hypertension as well as migraines in adolescence, presented to our institution with a 3-week history of persistent occipital headaches. His headaches were described as progressive in nature, and were exacerbated by straining or sneezing. On neurological examination, the patient was intact. Non-contrast CT was negative for SAH or any acute intracranial abnormality (figure 1). Lumbar puncture was negative for xanthochromia.

Figure 1 Axial non-contrast CT scans of the head. (A) There is no evidence of acute hydrocephalus. (B) There is no evidence of subarachnoid haemorrhage within the basal cisterns.

However, CT angiography (CTA) revealed a 4 mm left PCOM aneurysm (figure 2). Several hours following presentation, the patient underwent digital

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subtraction angiography (DSA), which demonstrated interval aneurysm expansion to 8 mm in conjunction with development of complete left-sided ONP (figure 3). All of these events occurred within 24 h of initial presentation.

TREATMENT

Given the acute change in neurological status as well as radiographic findings, the patient was emergently taken to surgery. A large aneurysm sac was appreciated juxtaposed to the ipsilateral oculomotor nerve. The aneurysm was obliterated by clip ligation. Postclip DSA confirmed aneurysm obliteration (figure 4).

OUTCOME AND FOLLOW-UP

Postoperative course was uneventful, and the patient was discharged 2 weeks after admission with mild improvement in ophthalmoplegia and ptosis.

Figure 2 CT angiography images of the head. (A) Axial view demonstrating a 4 mm aneurysm (arrow) within the left posterior communicating artery/terminus region. (B) Sagittal view of aneurysm (arrow).

Figure 3 Cerebral digital subtraction angiography showing 8 mm, irregularly shaped saccular aneurysm (arrow) at the origin of left posterior communicating artery.

DISCUSSION

Our index case is unique both in terms of the radiographic and clinical presentation, as well as the unprecedented acute expansion phenomenon of the intracranial aneurysm with respect to the current literature. Acute expansion with a doubling in size of the aneurysm less than 24 h from initial presentation is quite remarkable. The aneurysm that initially was 4 mm on CTA doubled in size to 8 mm on DSA. Furthermore, there was clinically correlated sequela of a new-onset compressive ONP in an otherwise asymptomatic patient at presentation. Even more illustrative is the fact that this case defies our traditional notion of gradual growth and expansion of intracranial aneurysms.

The growth rate of intracranial aneurysms has been a subject of curiosity. It is generally expected that a subset of aneurysms

Figure 4 Cerebral digital subtraction angiography following clip ligation shows obliteration of aneurysm.

will grow in size over time as part of their natural history. In a series of 87 patients followed over a mean period of 19 years, Juvela *et al*³ observed that unruptured aneurysms increased in size ≥ 1 mm in 45% of patients. Furthermore, the investigators noted an increase in aneurysm size ≥ 3 mm in 36%.³ Cigarette smoking was noted to be a predominant precipitating factor for aneurysm growth.³ In another study, Miyazawa *et al*⁴ retrospectively assessed independent risk factors for aneurysm growth in a cohort of 130 patients with a total of 159 aneurysms. During their mean follow-up period of 29 months, they observed an aneurysm growth rate of 10.8%, where 16 of 159 aneurysms exhibited growth on magnetic resonance angiography (MRA).⁴ Middle cerebral artery location, multiple aneurysms, aneurysm size of 5 mm or larger and family history of SAH were identified as independent risk factors for aneurysm growth.⁴ In a more recent study, Sprengers *et al*⁵ performed MRA as part of a 5-year follow-up in patients whose ruptured aneurysms were coiled but who also had untreated aneurysms requiring radiographic follow-up. Of the 20 untreated aneurysms, only one aneurysm was reported to have grown slightly.⁵ In a similar study from Australia, So *et al*⁶ examined the frequency of growth and the characteristic factors predictive of growth in 285 unruptured aneurysms. With a mean follow-up duration of 21.8 months, the investigators observed growth in 33% of the aneurysms.⁶ Excessive alcohol consumption was noted to be the predominant contributory factor in that study. These series illustrate the relevance of close radiographic surveillance in patients with unsecured aneurysms. It is equally apparent from these studies that only a subset of aneurysms would increase in size over time and at a very modest expansion rate.

Moreover, within the literature there is no documentation of acute expansion of an aneurysm within the time frame of our index case, and no documentation of aneurysm growth in evolution. Anda *et al*⁷ reported rapid growth of an unruptured aneurysm in a single patient on DSA 40 days after the initial DSA. This finding was observed within a cohort of patients who had presented with SAH from a ruptured aneurysm, but who also had unruptured aneurysms that were not treated at the time of treatment for the ruptured aneurysm.⁷ The investigators hypothesised and concluded that factors such as systolic blood pressure above 200 mm Hg during the acute stage of SAH and vasospasm were statistically significant risk factors that influenced the growth of aneurysms.⁷ Although there was a significant change in the angiographic size of the aneurysm in that study, it is equally evident that such a change was extremely modest.

It is unclear why our index case exhibited such a profound increase in aneurysm size in an acute setting. Aneurysm rupture can explain the acute onset ONP, but aneurysms do not necessarily expand with rupture. On the contrary, aneurysms may maintain their size or experience deflation of aneurysm sac with rupture. Another possible confounding factor could relate to the difference in spatial resolution between CTA and DSA, resulting in slight size discrepancies, as the spatial precision of DSA is far greater than CTA. However, the significant doubling in size of the aneurysm in our index case cannot be entirely accounted for by differences in spatial resolutions between DSA and CTA. Moreover, CTA spatial resolution issues were previously

described for much smaller aneurysms.^{8–10} With recent refinements in CTA acquisition and processing methodology, CTA assessment of aneurysms is more comparable with DSA than in previous years.¹¹ Lastly, we appreciated new-onset ONP correlated with radiographic changes in aneurysm size, making it less likely that differences in acquisition techniques was contributory.

Our unique case is very instructive in that although growth in aneurysm size is often a progressive phenomenon, acute expansions of aneurysms can occur. Furthermore, while aneurysm size and orientation has been postulated as the underlying cause of ONP, we present the first documented case of growth in evolution captured both clinically and radiographically.

Learning points

- ▶ Patients with third nerve palsies should be evaluated for a posterior communicating artery (PCOM) aneurysm.
- ▶ Third nerve palsy in the context of a PCOM aneurysm could have been caused secondary to aneurysm enlargement, as exemplified in our case.
- ▶ Patients with a third nerve palsy associated with a PCOM aneurysm should be treated emergently, as aneurysm enlargement could be responsible for the third nerve palsy and thus, may predict imminent rupture.

Competing interests None.

Patient consent Obtained.

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