CASE REPORT

Pseudomonas aeruginosa intrapetrous internal carotid artery mycotic aneurysm—a complication of mastoiditis: first reported case

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SUMMARY

Mycotic aneurysms of the intrapetrous carotid artery are a rare complication of adjacent middle ear infections that occur by direct invasion of arterial adventitia. We report the first case of a *Pseudomonas aeruginosa* mycotic intrapetrous carotid aneurysm arising from mastoiditis, confirmed with middle ear cultures, presenting with high-grade bacteraemia and otorrhagia in a diabetic man. Infection was related to elective myringotomy tube placement and was initially treated with empiric antibiotics. Diagnosis required careful evaluation of imaging studies, particularly MR angiography. Resolution required aggressive debridement and carefully selected long-term intravenous antibiotics, appropriate for carefully determined antibiotic sensitivity of his pathogen, but no neurosurgical intervention. He has had no evidence of recurrence over the subsequent year. We offer our experience to highlight the dangers of invasive *P aeruginosa* middle ear infections in patients with diabetes to other practitioners and to encourage earlier, more aggressive intervention.

BACKGROUND

Serious pseudomonas ear infections, including malignant external otitis, are potentially invasive and life-threatening events, primarily affecting patients with diabetes. Otitis in any host may be complicated by the presence of foreign bodies including myringotomy tubes. However, extension of infection, with invasion of deep arterial structures creating a mycotic aneurysm, represents a life-threatening complication that has not been reported. Our patient developed a deeply invasive, previously unreported, severe complication of *Pseudomonas* otitis associated with a myringotomy tube, that was easy to overlook and required special imaging studies to detect.

CASE PRESENTATION

A 79-year-old Caucasian man with diabetes mellitus and end-stage renal failure, on haemodialysis via a native vessel arteriovenous fistula, presented with left otalgia and otorrhea. He had bilateral myringotomy tubes placed 1 year earlier to accommodate hyperbaric oxygen therapy employed as adjunctive therapy for a *Staphylococcus aureus* left foot osteomyelitis. His otorrhea was initially treated with empiric oral antibiotics. However, later culture of his ear drainage revealed multidrug resistant (MDR) *P aeruginosa*. A trial of ofloxacin otic drops did not

improve the otorrhea. One month after onset, a CT scan of the head and sinuses revealed opacification of the left mastoid air cells (figure 1A). The patient's symptoms improved after a course of intravenous meropenem. However, he developed worsening otalgia and recurrent otorrhea within a month of completing meropenem. Repeat culture of the drainage again grew MDR *P aeruginosa*, this time, resistant to carbapenems. The left myringotomy tube was removed with release of about 2 mL of bloody effusion, and he was treated with piperacillin/tazobactam for 8 weeks. His C reactive protein and erythrocyte sedimentation rate normalised and all symptoms subsided with completion of the course of intravenous antibiotics.

However, the patient returned 2 months later with recurrent headaches and new left otorrhagia. CT scan of the head demonstrated more extensive opacification of the left mastoid air cells and an air-fluid level in left external auditory canal. In addition, deossification of a portion of the left internal carotid artery (ICA) was noted (figure 1B), indicating loss of integrity of the arterial wall, which prompted urgent MR angiography (MRA). MRA revealed a 10×6 mm pseudoaneurysm arising from the intrapetrous portion of the left ICA (figure 2) and mastoid opacification consistent with haemorrhage. Although afebrile, he had pronounced rigors. Four sets of blood cultures were drawn and all grew MDR *P aeruginosa*. CT of the sinuses was repeated, demonstrating gas in the left parapharyngeal space and enlargement of the aneurysm.

TREATMENT

Neurosurgical consultants deferred performing a coil/stenting procedure because of bacteraemia. He was started on ceftazidime (minimum inhibitory concentration (MIC)=8 µg/mL) and amikacin (MIC≤16 µg/mL). Within 2 days of admission, he developed left-sided multifocal embolic cerebral infarcts, in the distribution of his left ICA. Once his blood cultures became sterilised, he underwent mastoidectomy, but had no additional surgical or endovascular intervention for the pseudoaneurysm.

OUTCOME AND FOLLOW-UP

He completed 8 weeks of intravenous ceftazidime and amikacin and remained clinically stable over the subsequent year, with no further otalgia, otorrhagia or further otic findings.

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Figure 1 CT scan of the head. Original study (A) compared with later study (B) demonstrates progressive opacification of the left mastoid air cells and deossification of a portion of the left internal carotid artery (arrow).

DISCUSSION

This is the first reported case of an ICA mycotic aneurysm arising as a complication of *P aeruginosa* mastoiditis. Most previously reported mycotic ICA aneurysms did not specify the causative agent. In fact, we found 15 previously reported cases of intrapetrous ICA aneurysms and pseudoaneurysms arising from infection of adjacent tissues, but these cases do not delineate the pathogens in any instance.^{1–7} Thus, it is conceivable that *Pseudomonas* infections might have been among them, but might not have been recognised. Related reports highlight the extraordinary degree of carotid arterial damage that can be caused by *Pseudomonas*, including basilar skull infection and mastoiditis resulting in adjacent ICA pseudoaneurysms.^{5–7} The absence of blood culture data in these reports makes it unclear if the ICA pseudoaneurysms described were infective or primarily arose adjacent to inflamed tissue. In our patient, the infective aneurysm not only arose adjacent to infected mastoid tissue, but also the subsequent high-grade bacteraemia, that persisted despite antibiotics, as well as septic embolic phenomena within the distribution of the infected artery, provide strong confirmatory evidence for this diagnosis.

Aneurysms of ICA can develop secondary to congenital abnormalities, trauma or inflammation although they generally arise from the extracranial ICA within the cervical segment.² Aneurysms of the intrapetrous portion of the internal carotid are rare, but are reported sequelae of chronic otitis media or mastoiditis, as illustrated in this case. Although most infected aneurysms arise by haematogenous seeding, direct invasion of the adventitia of the artery from surrounding infection such as mastoiditis is a more likely cause in intrapetrous internal carotid aneurysms, due to proximity to the middle ear.⁸ Pharyngeal infections and cholesteatomas have also been implicated as causes of intrapetrous-infected aneurysms.⁸ As with all mycotic aneurysms, intrapetrous internal carotid mycotic aneurysms

should be treated with effective long-term antibiotic therapy. The role of additional intervention for these rare infections is unclear. For cases with diminished flow, enlarging or ruptured aneurysm, surgical or intravascular intervention may be necessary. Surgical resection and primary anastomosis have been used in the past; however, balloon occlusion is now favoured due to lower ischaemic complications and subsequent haemorrhages.^{2–5–7–9} For idiopathic or traumatic intrapetrous carotid aneurysms, coil embolisation or stent placement has been used successfully.¹⁰ Coil procedure has also been used in previously reported cases with surrounding infections,^{2–5} but is controversial as it involves leaving foreign material in the infected vessel. In fact, recently reported cases advise against revascularisation procedures that involve use of foreign material.³ Owing to high-grade bacteraemia and mastoiditis, coil embolisation was deferred in our patient.

We suspect that our patient's infection resulted from delay in removing the myringotomy tube and in surgical debridement. Although he was treated with intravenous antibiotics that were appropriate for the sensitivities of his MDR *P aeruginosa* isolate, the myringotomy tube was not removed until 2 months after initial onset of symptoms and 1 month after completion of the initial intravenous antibiotics. As in related reports, infection with *Pseudomonas* persisted, despite repeated courses of aggressive and appropriate antibiotic therapy.^{5–7} It is conceivable that the continued presence of the myringotomy tube, serving as a foreign body, and delay in debridement of infected mastoid tissue, contributed to persistence of the pathogen. Thus, our limited experience might serve to encourage more expedient intervention in future instances. Although this is the first reported case of its kind, we suspect that similar events may have occurred previously and have gone unrecognised. Particularly in diabetic patients, *Pseudomonas* ear infections can progress to severe infections, such as malignant otitis externa or

Figure 2 MR angiogram demonstrating a 10×6 mm. Pseudoaneurysm (arrows) arising from the intrapetrous portion of the left internal carotid artery, in axial (A) and coronal (B) views.

mastoiditis that are difficult to eradicate and potentially life threatening.^{11 12} Prompt intervention with appropriate antibiotics and surgical debridement are necessary and may prevent adverse events as occurred in this instance. Close monitoring of these patients after systemic antipseudomonal antibiotic treatment is also necessary in view of reported 15–20% recurrence rates.¹¹ We offer our experience to highlight the dangers of invasive *Paeruginosa* middle ear infections to other practitioners and to encourage earlier, more aggressive intervention.

Learning points

- ▶ Our experience highlights the dangers and complications of invasive *Pseudomonas* infections in patient with diabetes.
- ▶ This report also underscores potential complications of myringotomy tubes, dangers of delay in removal and dangers in delaying aggressive surgical debridement.
- ▶ Empiric antibiotic therapy without debridement or determination of antibiotic minimum inhibitory concentration to the pathogen resulted in emergence of multidrug-resistant *Pseudomonas* and progression of disease.
- ▶ There is no standard therapy for this entity. Our patient did well with aggressive surgical debridement and long-term antibiotics and did not require neurovascular intervention.
- ▶ The diagnosis required careful evaluation of imaging studies, particularly MR angiography.

Contributors Both authors have made substantial contributions to conception and acquisition of information, to drafting and revising the manuscript; and gave final approval of the edited manuscript for publication.

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