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Clinical Record

Intracranial hypertension secondary to sigmoid sinus compression by group A streptococcal epidural abscess

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Abstract

Objective: We present an extremely rare case of severe intracranial hypertension secondary to sigmoid sinus compression by a group A streptococcal epidural abscess.

Method: Case report and review of the world literature.

Results: A five-year-old boy was treated for acute otitis media and group A streptococcal bacteraemia, but subsequently developed severe intracranial hypertension. Computed tomography revealed that, although the sigmoid sinuses were not thrombosed, the patient had a dominant right sigmoid sinus that was almost completely compressed by a small epidural abscess. After surgical decompression of the epidural abscess, with aggressive debridement of the granulation tissue from the sigmoid sinus wall, the patient awoke from general anaesthesia with complete resolution of his symptoms and signs of intracranial hypertension. He suffered no sequelae over the subsequent six months' follow up.

Conclusion: This is the first reported case of intracranial hypertension due to an epidural abscess causing sigmoid sinus compression without thrombosis. This case illustrates the fact that, even in the absence of thrombosis of the sigmoid sinus, a small epidural abscess may require urgent surgical treatment.

Key words: Sigmoid Sinus; Epidural Abscess; Acute Otitis Media; Group A Streptococcus

Introduction

Sigmoid sinus thrombosis is a well known complication of mastoiditis. We present a very unusual case of a child with mastoiditis who, despite initial therapy, developed intracranial hypertension secondary to compression (without thrombosis) of a dominant sigmoid sinus.

Case report

A five-year-boy with a history of recurrent acute otitis media presented to the British Columbia Children's Hospital with purulent left otorrhoea, which occurred after five days of fever and bilateral otalgia and three days of headaches, nausea and vomiting. He was treated with intravenous (IV) ceftriaxone and rehydration. Two days later, after blood and left ear pus cultures had grown group A streptococci, antibiotic therapy was switched to IV penicillin G.

The next day, the patient's fever and vomiting had resolved, but his headaches persisted and his right tympanic membrane was still red and bulging. After an otolaryngology consultation, antibiotic therapy was switched to IV cefotaxime and clindamycin.

A computed tomography (CT) scan revealed no mastoid bone destruction, but demonstrated a small epidural fluid collection mildly compressing the right sigmoid sinus (Figure 1). That evening, bilateral myringotomies were performed, pus was aspirated and tympanostomy tubes were inserted. Ciprodex ear drops (Alcon Canada Inc., Mississauga, Ontario, Canada) were instilled twice daily.

The group A streptococci that had been isolated were found to be sensitive to all the systemic antibiotics that had been given. However, over the next five days, although the patient remained afebrile and his acute otitis media resolved otoscopically, his headaches worsened. A second CT scan revealed mild enlargement of the epidural fluid collection, causing more compression of the right sigmoid sinus (Figure 2). No venous thrombosis was evident. It was noted that the right jugular vein was the dominant outflow tract of the brain, and that the right transverse sinus was much larger than the left transverse sinus (Figure 3). Aspirin was commenced to prevent thrombosis.

That night, the patient's headaches worsened dramatically, his nausea and vomiting returned, and he complained of diplopia. The next day, the patient was found to have a mild right VIth nerve palsy and marked papilloedema. Lumbar puncture was performed; the opening pressure was found to be 36 cm H_2O. Acetazolamide and (prophylactic) heparin were commenced. The cerebrospinal fluid (CSF) cell count, protein concentration and glucose concentration were normal, and the CSF culture was eventually reported as negative.

The next day, the patient's neurological symptoms had not improved. Dexamethasone was commenced. It was postulated that the patient's intracranial hypertension was secondary to the compression of his dominant right sigmoid sinus.

After a neurosurgical consultation, the patient was taken to the operating theatre, under the care of both the otolaryngology and the neurosurgery teams, for right mastoidectomy

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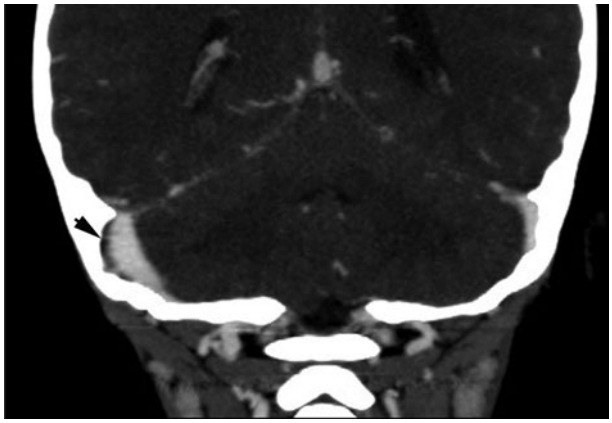

FIG. 1

Coronal computed tomography scan with contrast, demonstrating mild compression of the right sigmoid sinus by epidural fluid (arrow).

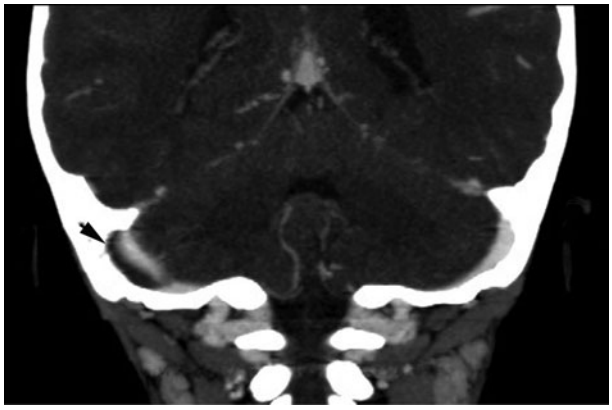

FIG. 2

Coronal computed tomography (CT) scan taken five days after the original CT, showing further compression of the right sigmoid sinus by the epidural abscess (arrow).

and sigmoid sinus decompression. The mastoid was filled with granulation tissue. Eventually, bright yellow pus under pressure was found in the epidural space, just medial to the posterior mastoid. After drainage of the pus, abundant granulation tissue was found. The bony window to the epidural space was enlarged to a diameter of 5 mm, to allow careful cup-forceps debridement of the granulation from the sigmoid sinus wall, which had become thickened. Intramural veins refilled rapidly after the release of light pressure, suggesting good flow within the sigmoid sinus. Inferiorly, some blue colour could be seen in the area of the sigmoid-transverse junction. The dural sinuses were not opened.

The patient awoke from general anaesthesia with complete resolution of his headache and nausea. Within an hour, he was eating solid food. The next day, CT confirmed complete re-expansion of his right sigmoid sinus (Figure 4). He was found to have no optic neuropathy or hearing loss. The dexamethasone and acetazolamide doses were tapered.

The patient's VIth nerve palsy eventually resolved as he completed a three-week course of ceftriaxone as an out-patient. Six months later, he received a second set of tympanostomy tubes for recurrent acute otitis media, but otherwise remained healthy.

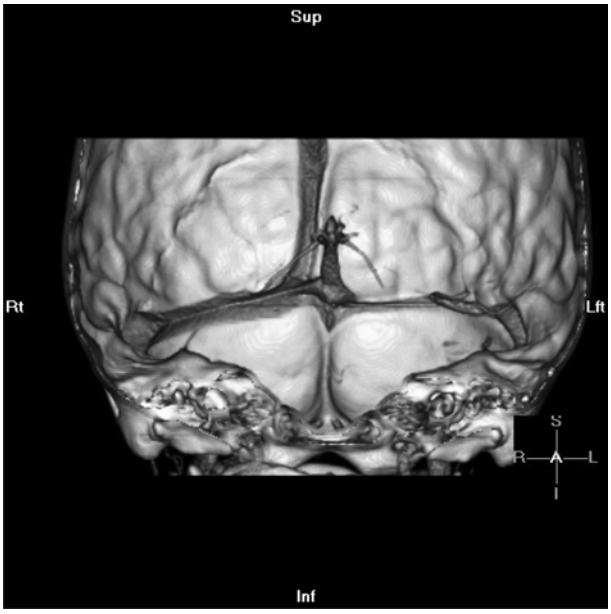

FIG. 3

Three-dimensional computed tomography reconstruction, demonstrating dominance of the right posterior intracranial venous outflow tract (the right transverse sinus is much larger than the left transverse sinus). Sup = superior; inf = inferior; rt = right; lft = left

Discussion

This is the first reported case of intracranial hypertension due to an epidural abscess causing sigmoid sinus compression without thrombosis. In 1984, Schonsted-Madsen *et al.* reported a case of an 11-year-old patient who had intracranial hypertension secondary to a 'partly compressed and partly thrombosed' sigmoid sinus.[1] That patient did not undergo surgical decompression of the sigmoid sinus until two weeks after he developed bilateral VIth nerve palsies and papilloedema; fortunately, he still made a full recovery. Our patient had surgical decompression of his sigmoid sinus two days after he developed a VIth nerve palsy and papilloedema. Fortunately, he also made a full recovery.

- **This paper describes an extremely rare case of severe intracranial hypertension secondary to sigmoid sinus compression by a group A streptococcal epidural abscess, without thrombosis**
- **After surgical decompression of the epidural abscess together with aggressive debridement of granulation tissue from the sigmoid sinus wall, the patient made a full recovery**
- **Even in the absence of thrombosis of the sigmoid sinus, a small epidural abscess may require urgent surgical treatment**

Intracranial hypertension is a serious condition that can lead to severe vision loss.[2] Rapid diagnosis and treatment of intracranial hypertension is imperative. In the setting of recent mastoiditis, intracranial hypertension should be presumed to be secondary to epidural suppuration causing decreased flow within the sigmoid sinus due to thrombosis and/or compression. This is true even if the patient's fever and otoscopic signs of acute otitis have resolved, as the patient may have 'masked' mastoiditis, with obstruction of the aditus ad antrum.[3,4]

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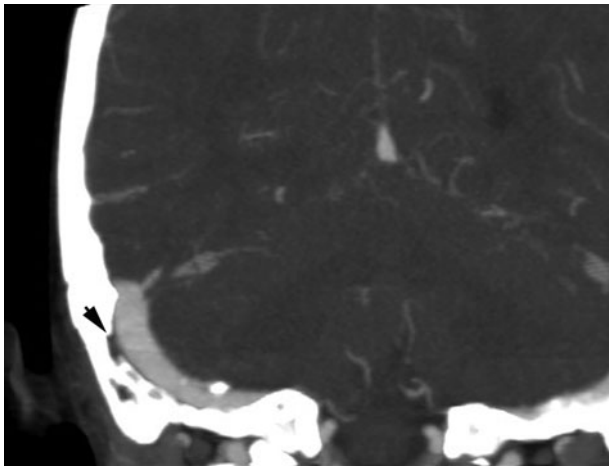

FIG. 4

Coronal computed tomography scan showing near-complete decompression of the right sigmoid sinus, 12 hours after trans-mastoid drainage of the epidural abscess and debridement of granulation tissue from the sigmoid sinus wall (arrow).

Progressive headache, vomiting and diplopia are the cardinal symptoms that suggest intracranial hypertension. They should prompt the medical team to consider further diagnostic tests, including fundoscopy, imaging and lumbar puncture. Computed tomography can be diagnostic and lead to correct and timely surgical management, by identifying epidural suppuration and characterising the impediment to venous drainage. Thickening of the sigmoid sinus wall can make it difficult to identify intra-operatively. Adequate decompression of the sinus may be suspected post-operatively due to the patient's signs and symptoms, and confirmed by further imaging studies.

Group A streptococci are uncommon pathogens in mastoiditis, but are capable of causing serious intratemporal and intracranial complications, including necrosis of the tympanic membrane and ossicles, semicircular canal fistula, epidural abscess, and meningitis.[5–8]

Conclusion

Intracranial hypertension may result from compression of a dominant sigmoid sinus without thrombosis. This can be caused by group A streptococci and may occur as 'masked' mastoiditis. Even in the absence of sigmoid sinus thrombosis, a small epidural abscess may require urgent surgical therapy.

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