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Case Report

Horner Syndrome as Complication of Acute Sphenoid Sinusitis

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Horner syndrome · Sphenoid sinusitis · Cavernous thrombosis

Abstract
Horner syndrome is described in this case report as a rare complication of bacterial sphenoid sinusitis. A patient presented with miosis, ptosis, and ophthalmic nerve palsy with acute sphenoid sinusistis and cavernous sinus thrombosis on MRI. The impairment of sympathetic fiberscan can be explained through the direct septic effects of the sphenoid sinusitis and indirectly through thrombosis of the cavernous sinus at the level of the carotid plexus.

Introduction

A 62-year-old patient presented with increasing frontal headaches over the preceding 10 days, not relieved by ibuprofen. By the time of presentation, he had developed double vision and drooping of the right eyelid. He reported no other complaints, in particular no fever, rhinitis, or meningeal symptoms. His past medical history was unremarkable, especially with no acute or chronic sinusitis.

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Endoscopy showed mild edema of the mucosa of the ethmoid cells without any signs of purulent nasal discharge. Ptosis and miosis of the right eye (Fig. 1a) were found, consistent with Horner syndrome. Signs of paralysis of the oculomotor nerve included palsy of right eye movement when looking upwards (Fig. 1b), during adduction (Fig. 1c), and looking downwards. No further neurological deficits were found, and the visual acuity was normal.

Magnetic resonance imaging (MRI) of the brain revealed acute right sphenoidal sinusitis with subtle irregularities of the internal carotid artery in the cavernous segment in the 3D TOF MRA and TWIST. Furthermore, thrombosis of the cavernous sinus, ophthalmic vein, superior petrosal sinus, sigmoid sinus, internal jugular vein, and sphenoparietal sinus on the right side (Fig. 2) was seen in post-contrast acquired images. The additional computed tomography scan showed a permeative osseous erosion of the posterior and lateral walls of the right sphenoid sinus, while the carotid artery remained covered by bone (Fig. 2d).

Given the above-mentioned intracranial complications, surgical drainage of the right sphenoidal sinus was indicated. Endoscopic sinus surgery with neuronavigation, including endoscopic septoplasty, middle meatotomy, and drainage of the sphenoidal sinus was performed. Surgical access to the sphenoidal sinuses was exclusively paraseptal with consecutive enlargement of the natural ostium with drainage of purulent discharge of the right sphenoid sinus; the cavity was otherwise intact.

Penicillin-resistant *Staphylococcus aureus* was cultured from the intraoperative nasal swab; the histology showed chronic inflammatory changes of the tissue, with no signs of malignancy. Because of intracranial septic thrombosis the patient was treated empirically with intravenous ceftriaxone and metronidazole, followed by administration of oral sulfamethoxazole and trimethoprim for 39 days as advocated by the consultant for infectious diseases. Additionally, anticoagulation was started at the first postoperative day for a total duration of 6 months. All initial signs of ptosis, miosis, palsy of the oculomotor nerve, and double vision improved progressively postoperatively. Four weeks after surgery, the patient was symptom free apart from a mild headache (Fig. 1d–f). Upon endoscopic examination, the sphenoid sinuses were well accessible, with healed mucosa.

Our institutional review board (Kantonale Ethikkommission Bern) does not require formal approval for case reports. The patient granted written permission to publish the case and images.

Discussion

Rare intracerebral complications of bacterial sphenoid sinusitis, such as septic cavernous sinus thrombosis (CST) and internal carotid thrombosis, have previously been described [1]. However, to our best knowledge, there are no publications discussing Horner syndrome as a complication of bacterial sphenoid sinusitis.

Horner syndrome is described as the disruption of the sympathetic nerve supply to the face [2]. The postganglionic sympathetic fibers of the ciliary ganglion originate from the superior cervical ganglion. They reach the ciliary ganglion via the internal carotid plexus through the cavernous sinus and go to the dilator muscle of the iris, the orbital muscle, and the tarsal muscles. Denervation of the superior tarsal muscle causes ptosis. Sympathetic nerve supply is also responsible for the dilation of the pupil (mydriasis). When disrupted, parasympathetic supply is uninhibited, and constriction of the pupil (miosis) ensues. The reaction of the pupils to light and accommodation is normal as those systems do not depend on sympathetic nerve supply [2]. In the case described here, we would initially have expected mydriasis as a sign of

oculomotor palsy. However, we found miosis due to the disruption of the sympathetic fibers, which can be explained through the direct septic effects of the sphenoid sinusitis with osseous erosion of the posterior and lateral walls (Fig. 2d) and indirectly through thrombosis of the cavernous sinus at the level of the carotid plexus.

Apart from early surgical intervention, prompt antibiotic therapy plays an important role in the treatment of the above-mentioned complications. The role of anticoagulants in CST is controversial. Levin et al. [3] found no significant difference in mortality rates between patients treated with and without anticoagulation. Other authors argued that hemorrhage caused by anticoagulation is rare, and that early adjunctive anticoagulative treatment can be beneficial once hemorrhagic complications of CST are ruled out [4]. In this case, the patient recovered completely with a combination of surgical, antibiotic, and anticoagulative treatment.

Statement of Ethics

The patient gave his written informed consent for us to publish his case, including publication of images.

Disclosure Statement

The authors have no conflicts of interest to declare.

Author Contributions

All authors made substantial contributions to the study. C.K. examined the patient, participated in the treatment of the patient, and drafted the manuscript. L.A. performed the surgical treatment and critically revised the manuscript for important intellectual content. F.W. analyzed the radiological data and participated in the interpretation and revision of the manuscript. M.C. critically revised the manuscript for important intellectual content.

Disclosure Statement

The authors have no conflicts of interest to declare.

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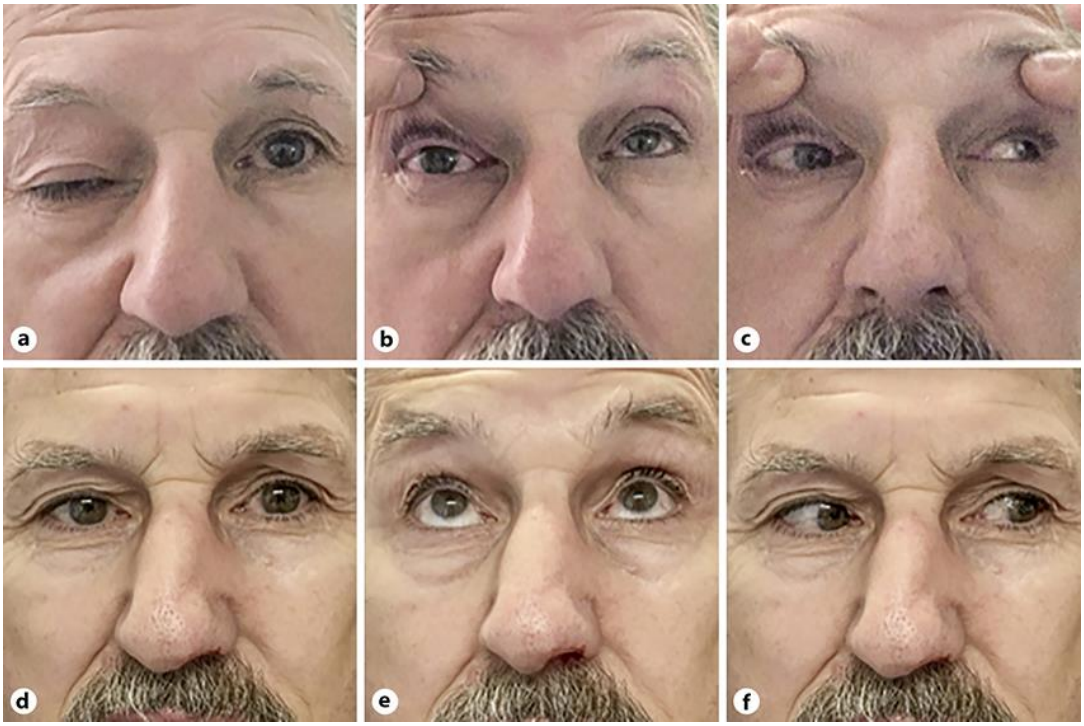

Fig. 1. **a** Preoperative ptosis. **b** Preoperative palsy looking upwards. **c** Preoperative palsy of adduction. **d–f** Postoperative status with normal eye movements.

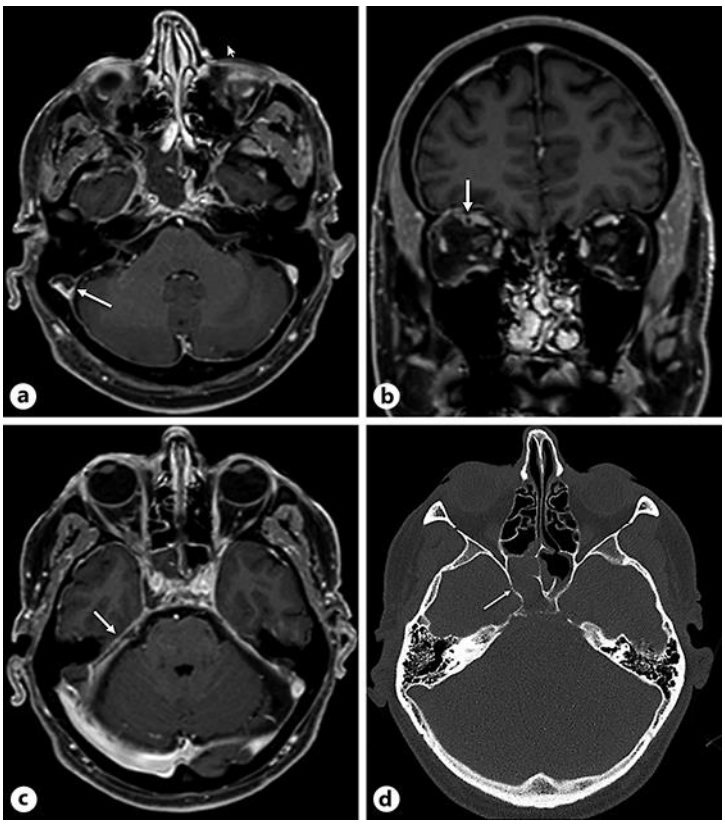

Fig. 2. MRI multiplanar reconstruction post-contrast. **a** Thrombosed sigmoid sinus. **b** Thrombosed ophthalmic vein. **c** Thrombosed sphenoparietal sinus. **d** CT with osseous erosion of the right sphenoid sinus.