Sphenoethmoidal Sinusitis Complicated By Cavernous Sinus Thrombosis And Pontocerebellar Infarction

Robert L. Macdonald, J. Max Findlay and Charles H. Tator

ABSTRACT: An unusual case of sphenoethmoidal sinusitis complicated by cavernous sinus thrombosis, meningitis and pontine and cerebellar infarction is described. The patient presented with advanced intracranial complications which in retrospect caused delay in recognition and treatment of the underlying sphenoethmoidal sinusitis. Surgical drainage of the sinusitis was ultimately required. The pathogenesis of these complications is discussed, and the topic of sphenoid sinusitis reviewed in order to emphasize the numerous neurological manifestations of this disease.

RÉSUMÉ: Sinusite sphéno-ethmoïdale compliquée d'une thrombose du sinus caverneux et d'infarctus pontocérébelleux Nous rapportons un cas inusité de sinusite sphéno-ethmoïdale compliquée d'une thrombose du sinus caverneux, de méningite et d'infarctus du pons et du cervelet. Le patient s'est présenté avec des complications intracrâniennes à un stade avancé, ce qui, en rétrospective, a retardé l'identification et le traitement de la sinusite sphéno-ethmoïdale sous-jacente. Il s'est avéré nécessaire, en fin de compte, de recourir au drainage chirurgical de la sinusite. Nous discutons de la pathogénèse de ces complications et nous revoyons la sinusite sphénoïdale dans le but de souligner les nombreuses manifestations neurologiques reliées à cette entité.

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Patients with sphenoid sinusitis often present with headache and facial pain which may lead to referral to a neurologist or neurosurgeon. A high index of suspicion is required to establish the correct diagnosis before intracranial complications ensue. Conversely, patients who present with symptoms of cavernous sinus thrombosis, meningitis or other intracranial complications may experience delay in diagnosis because the clinician's attention may be directed away from the primary focus of infection - the sphenoid sinus. We describe a patient with acute sphenoethmoidal sinusitis who presented with cavernous sinus thrombosis, meningitis and focal infarction of the cerebellum and brainstem. We were unable to identify any reported cases of sphenoid sinusitis complicated by infarction in posterior fossa structures, which in our patient, appeared to be due to progressive venous thrombosis. However, the possibility exists that such cases occurred in more extensive, usually fatal, cases encountered in the preantibiotic era.[1] Sphenoid sinusitis and its intracranial complications are discussed.

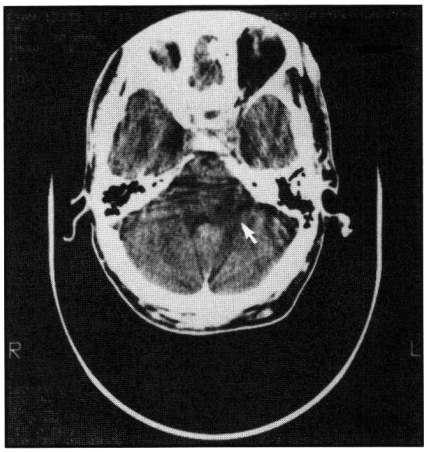

Figure 1 — Contrast enhanced CT scan the day of admission showing hypodensity in the structures near the left cerebellopontine angle with compression and displacement of the fourth ventricle (arrow).

CASE REPORT

A 65-year-old man presented with a five day history of bifrontal headache, nausea and vomiting. On the day of admission, he awoke with fever, bilateral conjunctival redness and swelling, and weakness of the left facial muscles. On examination, he appeared acutely ill and his temperature was 39.7°C. He was complaining of severe headache and had marked neck stiffness, but was alert and oriented. Facial erythema, periorbital edema, chemosis and bilateral exophthalmos were present. The pupils were equal and reactive and visual acuity was 20/30 in the right eye and 20/100 in the left eye with evidence of a dense left cataract. Extraocular movements in all directions were impaired with a

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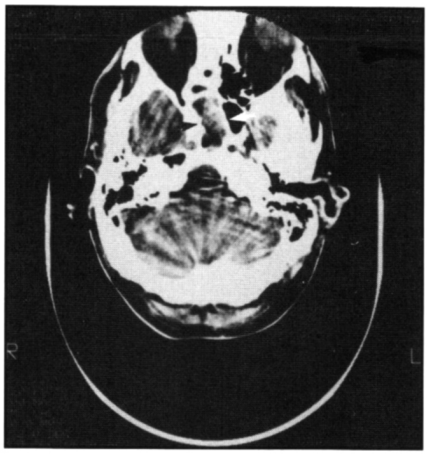

Figure 2 — CT scan one day after admission showing opacification of the sphenoid sinus (arrows).

suggestion of a left abducens palsy, and there was left beating nystagmus on left lateral gaze. Left trigeminal V1 and V2 hypesthesia with a decreased left corneal reflex, left peripheral facial palsy and left sensorineural hearing loss were noted. There was left upper extremity hypotonia, intention tremor and dysdiadochokinesis. The right plantar response was extensor. The remainder of the neurologic and general physical examination was normal.

Laboratory investigations revealed a white blood cell count of 14.9 with 90% polymorphonuclear leukocytes. A computed tomographic (CT) scan of the head with and without contrast enhancement showed a nonenhancing low density area in the left dorsolateral pons and middle cerebellar peduncles with displacement of the fourth ventricle to the right (Figure 1). Lumbar puncture was performed and the cerebrospinal

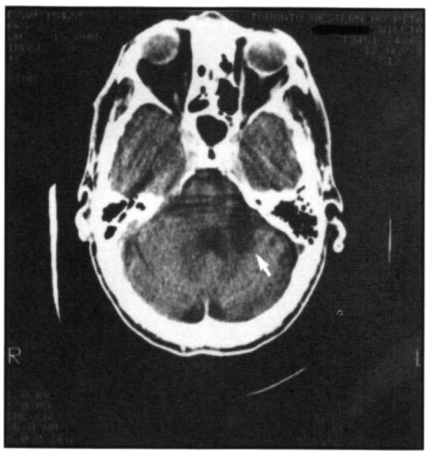

Figure 3 — CT scan 3 months post-operatively showing a well defined hypodensity in the left anterior superior cerebellum extending into the superior cerebellar peduncle and rostral lateral pons (arrows).

fluid (CSF) was cloudy with 3800 white blood cells (95% polymorphonuclear leukocytes) and 2200 red blood cells/cumm. CSF glucose was 1.8 mmol/L (serum 12.2 mmol/L) but gram stain failed to show bacteria. It was not until the following day that it was appreciated on a repeat contrast enhanced CT scan that the sphenoid and right posterior ethmoid sinuses were opacified (Figure 2); the lesion in the posterior fossa was unchanged. Cloxacillin 8 gm/d, Cefotaxime 8 gm/d, and Dexamethasone 12 mg/d were started intravenously, and an otolaryngologic consultation was obtained. The patient was taken to the operating room emergency for transseptal sphenoidotomy and right internal and external ethmoidotomies. Copious quantities of pus were obtained which grew *streptococcus intermedius*. Subsequently, blood and CSF cultures grew this same organism. Antibiotic treatment was changed to Metronidazole 1.5 gm/d and Penicillin G 12 million units/d intravenously and continued for two weeks. The patient defervesced rapidly and the chemosis and exophthalmos resolved over several days. A left tarsorrhaphy was performed for corneal protection.

Three months post-operatively, the patient had residual left facial numbness, left-sided deafness, and peripheral facial weakness. Neuro-ophthalmologic exam showed torsional nystagmus in lateral and down gaze bilaterally and hypometric saccadic eye movements to the left but the 6th nerve palsy had disappeared. Moderately severe left cerebellar signs consisting of incoordination of the arm and leg and dysdiadochokinesis persisted although the patient was able to ambulate without assistance. A CT scan without contrast showed a well defined hypodensity in the left cerebellum extending into the superior and middle cerebellar peduncles and dorsolateral pons (Figure 3). The fourth ventricle had returned to the midline.

Discussion

Sphenoid sinusitis is an uncommon clinical entity, and is usually accompanied by another paranasal sinus infection, as was the case in our patient where the infection also involved the ethmoid sinus.[2,3,4] Predisposing factors include a recent viral upper respiratory tract infection, trauma, congenital or acquired lesions affecting sinus drainage, immuno-suppressed states, and iatrogenic causes such as following transsphenoid surgery[5]. Acute cases are more likely to have an identifiable precipitating cause, although none was recognized in our patient.

The early clinical presentation of sphenoid sinusitis can be relatively nonspecific, with symptoms such as headache, painful paresthesia in the distribution of the trigeminal nerve, and nasal discharge, but few physical findings.[4,6,7] Because of this, the correct diagnosis is often not made until complications ensue from intracranial spread of infection and even then the diagnosis follows only when special consideration is given to the sphenoid sinus.[6,7] In a review by Lew et al[7] of 30 cases of acute or chronic sphenoid sinusitis only six of the 15 patients with acute

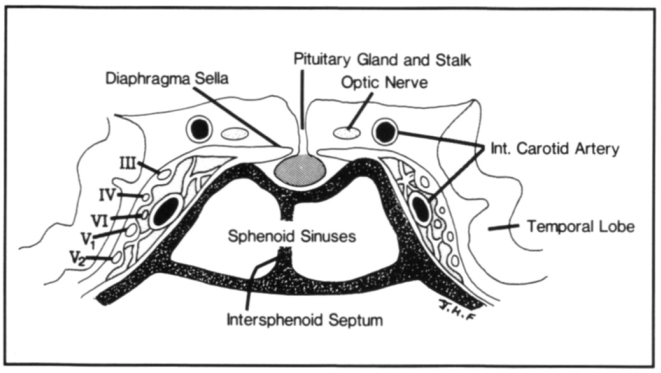

Figure 4 — The relationships of sphenoid sinus to vessels, nerves, and surrounding brain.

infections were correctly diagnosed on first arrival at the hospital, and six patients presented with intracranial complications that were initially mistakenly diagnosed as aseptic meningitis (four patients) or primary cavernous sinus thrombosis (two patients). In total, 14 of the 15 patients with acute sphenoid sinusitis in this series developed intracranial complications and four of these patients subsequently died, three without diagnosis ante-mortem of the underlying sphenoid sinusitis requiring surgical drainage. Our patient exemplifies this problem, presenting

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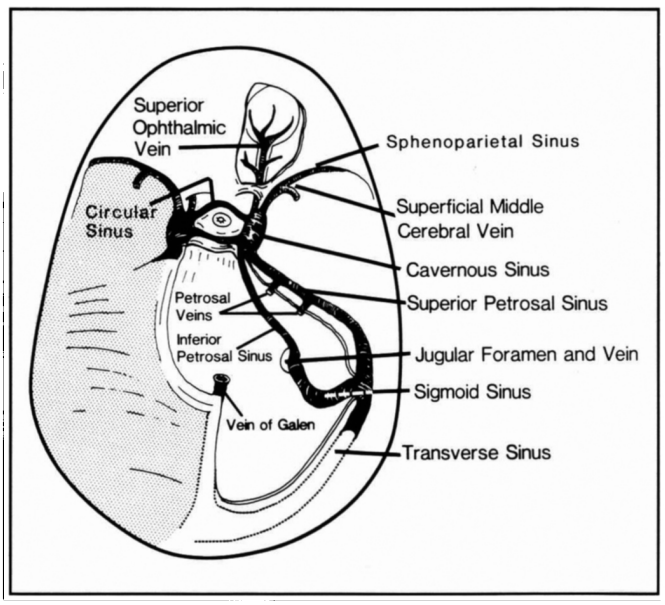

Figure 5 — The venous sinuses associated with the cavernous sinus.

with advanced intracranial spread of infection which initially distracted attention away from the seat of the infectious process in the sphenoid sinus. Indeed, it was not until the day following admission that the opacified sinus was identified on the CT scan, enabling correct therapeutic intervention. Clearly, a high index of suspicion is necessary to make the correct diagnosis of this disease and to avert intracranial spread.

The nature of these complications is governed by the unique neurovascular relationships of the sphenoid sinus (Figures 4 and 5).[8,9] Direct spread of infection through venous channels or deficiencies in the sinus wall into the adjacent cavernous sinuses can lead to cavernous sinus thrombophlebitis and thrombosis, which is usually bilateral.[10,11] Indeed, paranasal sinus infections are second only to midface infections as a cause of cavernous sinus thrombosis.[10,12] Congestion of the superior ophthalmic veins which drain directly into the cavernous sinuses leads to orbital swelling, exophthalmos, impairment of extraocular motility, chemosis and periorbital edema and erythema. Rarely is the venous pressure high enough to interfere with optic nerve perfusion and cause visual impairment. Our patient had cavernous sinus thrombosis, but his reduced visual acuity was preexistent and due to a cataract. Pressure and inflammation around the cranial nerves passing through the cavernous sinus (abducens nerve) or within its lateral wall (oculomotor, trochlear and the ophthalmic and maxillary divisions of the trigeminal nerve) can lead to palsies.[10,13] Our patient demonstrated left abducens and trigeminal nerve dysfunction. Also traversing the cavernous sinus is the cavernous segment of the internal carotid artery and its sympathetic plexus and spasm of this vessel has been reported as a complication of sphenoid sinusitis.[14]

In cases of sphenoid sinusitis, the associated venous thrombophlebitis can propagate from the cavernous sinus along feeding dural sinuses and their associated draining cortical and parenchymal veins.[1,7,15] Our patient had radiologic evidence of infarction in the left dorsolateral mid to lower pons and adjacent cerebellar peduncles and cerebellum, which caused persistent ipsilateral facial paralysis, deafness, torsional nystagmus with impaired saccadic eye movements and cerebellar deficits. The pathogenesis of the infarction is most likely related to thrombophlebitis and thrombosis involving the left superior and inferior petrosal sinuses and the bridging petrosal veins, which are in turn formed from the transverse pontine, cerebellopontine and middle cerebellar peduncle veins.[9] Although the distribution of the infarct does not conform to a defined arterial distribution in the posterior fossa,[16] we cannot exclude the possibility of arterial infarction secondary to an infectious vasculitis. A cerebral angiogram was not obtained.

In acute sphenoid sinusitis, the parameningeal inflammation of cavernous thrombophlebitis or overt bacterial migration into the cerebrospinal fluid from the cavernous sinus may lead to meningism or meningitis, respectively, with the latter occurring in our patient. Less commonly, bacterial transmission through venous channels may lead to subdural, intracerebral, and orbital abscesses.[17] In our patient, the discrete, nonenhancing hypodense lesion in the posterior fossa (Figure 1) was not consistent with cerebritis or an abscess.

Other complications of sphenoid sinusitis include anatomical variations of the above processes such as the superior orbital fissure syndrome (external ophthalmoplegia, exophthalmos, ophthalmic and maxillary nerve hypesthesia and Horner's syndrome) and orbital apex syndrome (superior orbital fissure syndrome accompanied by evidence of impaired optic nerve function) and orbital cellulitis.[2,6,17,18]

In the diagnosis of sphenoid sinusitis, uncomplicated cases frequently have normal plain radiographs of the paranasal sinuses.[4,7] Currently, CT scanning is the most accurate test for the diagnosis of paranasal sinus disease.[19] In addition, if cavernous sinus thrombosis or intracranial complication is suspected, emergent CT scanning is indicated. If cavernous sinus thrombosis is present, the CT scan will show lack of contrast enhancement in one or both cavernous sinuses.[20] Since cavernous sinus thrombosis is frequently secondary to paranasal sinusitis, it is imperative to examine all of the sinuses in these studies in order to detect an unsuspected sinus infection, such as in our patient. If the clinical and CT findings are consistent with cavernous sinus thrombosis, cerebral angiography, which has been the definitive diagnostic test for this disease, can be obviated. We are not aware of any reports of magnetic resonance imaging in cases of sphenoid sinusitis or its complications. Finally, lumbar puncture is indicated in the septic patient with sphenoid sinusitis because of the high incidence of coexistent meningitis.

The most commonly responsible organisms in sphenoid sinusitis are *Staphylococcus aureus, Streptococcus pneumonia* and other aerobic or anaerobic streptococci.[4,7] Multiple organisms which may include anaerobes are found in 25% of cases.[7]

If an opacified sphenoid sinus is demonstrated in a patient with a history suggestive of sinusitis, then immediate hospital admission is indicated following needle aspiration or open drainage of the sinus to obtain a bacteriologic diagnosis and allow drainage of the infection. Parenteral antibiotics should be started and should be directed against gram positive cocci and gram negative bacilli and should include a third generation cephalosporin such as cefotaxime. Although needle aspiration

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may be adequate in uncomplicated cases, open drainage should be performed in complicated cases (such as ours) or those with progression on antibiotics. There are multiple surgical approaches to the sphenoid sinus including intranasal sphenoidostomy and transseptal, transethmoidal and transantral sphenoidotomy[8,21] with the choice depending on a number of factors including the presence of associated infected sinuses. Systemic anticoagulation in cavernous sinus thrombosis is controversial, but is currently not recommended particularly in infectious cases.[22]

Outcome with aggressive management is improved over that of the preantibiotic era when a 50% mortality rate was recorded. Recent series still document a 10-27% mortality rate, with mortality clearly related to mistaken and delayed diagnosis preventing appropriate surgical treatment.[3,7] Morbidity from sphenoid sinusitis includes residual cranial nerve dysfunction secondary to orbital and intracranial complications. Visual deficits including blindness, and third, fourth, fifth and sixth cranial nerve palsies may be permanent in up to 50% of cases of cavernous sinus thrombosis and 20% of cases of sphenoid sinusitis[7,10] Chronic retro-orbital pain may also occur.[4,7]

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