CASE REPORT

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Sinusitis and ischemic stroke*

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SUMMARY

Acute sinusitis is a prevalent and generally uncomplicated infection that is normally resolved by medical therapy. However, severe neurological complications are known, and comprise of cerebral abscess, cavernous sinus thrombosis, meningitis, and epidural or subdural empyema. We report a case about a 10-year-old girl with a severe acute pansinusitis and ischemic stroke in the right lentiform nucleus and the anterior part of the right internal capsule. Possible explanations for this rare combination are discussed.

Key words: sphenoid, sinusitis, stroke, paediatric, vasospasm

INTRODUCTION

Sinusitis is a common and generally uncomplicated infectious disease, but an undiagnosed or non-sufficient treated aggressive sinusitis may lead to life threatening intracranial complications. The neurological disorders in patients with sinusitis most often reported are: cerebral abscess, cavernous sinus thrombosis, meningitis, and epidural or subdural empyema, causing neurological symptoms and findings such as hemiparesis, photophobia, cranial nerve palsies or seizures (Whitehead and De Souza, 1974; Macdonald et al., 1988; Brockbank and Brookes, 1991; Rosenfeld and Rowley, 1994; Swift and Gill, 1994; Singh et al., 1995; Bitsori et al., 2000; Oruckaptan et al., 2000; Perez Baretto et al., 2000). We report a case of a 10-year-old girl with an acute pansinusitis and ischemic stroke in the right lentiform nucleus and in the anterior part of the right internal capsule.

To our knowledge, simultaneous acute sinusitis and ischemic stroke have not been reported without additional meningitis. We discuss possible explanations for this combination.

CASE REPORT

A 10-year-old girl was admitted to a local hospital with fever and headaches 5 days after onset of symptoms. A general practitioner had started penicillin treatment the day before she was referred, because pneumonia was suspected. Upon admission, trismus, cervical microadenitis, as well as hypertrophy of the tonsils were found, but the girl had no fever. Blood tests showed moderate leucocytosis with neutrocytosis. In accordance with the findings, an acute tonsillitis was suggested. She was dismissed the day after admittance without antibiotic treatment.

Three days later the girl was re-admitted at the same hospital. She now had high fever. An upper respiratory infection was suspected in accordance with hypertrophy of the tonsils and trismus, which were noted again. As the general condition had clearly worsened, a subsequent examination by an ENT specialist was performed. This examination additionally revealed periorbital oedema with bilateral redness over the maxillary region, but the ENT specialist noted no trismus. An acute CT scan showed opacification of all paranasal sinuses confirming pansinusitis. She was treated with intravenous penicillin and gentamycin in conjunction with displacement therapy (Proetz suction). Culture of the pus showed *Staphylococcus aureus*, *Haemophilus parainfluenzae,* and non-haemolytic *Streptococcus.*

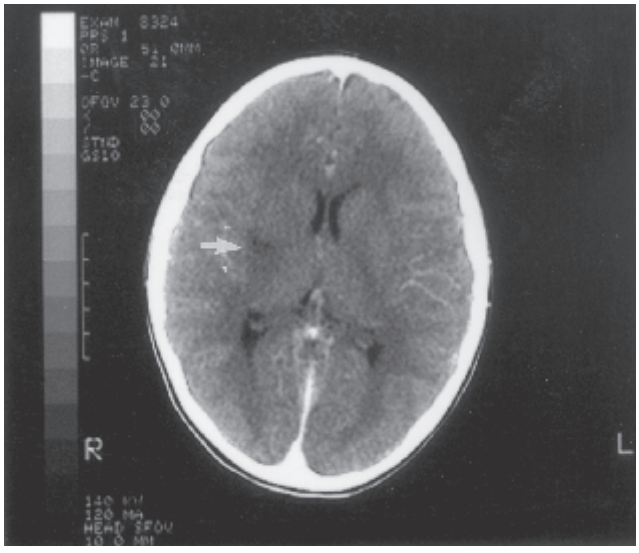

Figure 1. Enhanced CT, 9 days after onset of symptoms, demonstrating acute ischemic infarction in the right lentiform nucleus and anterior part of the right internal capsule (arrow).

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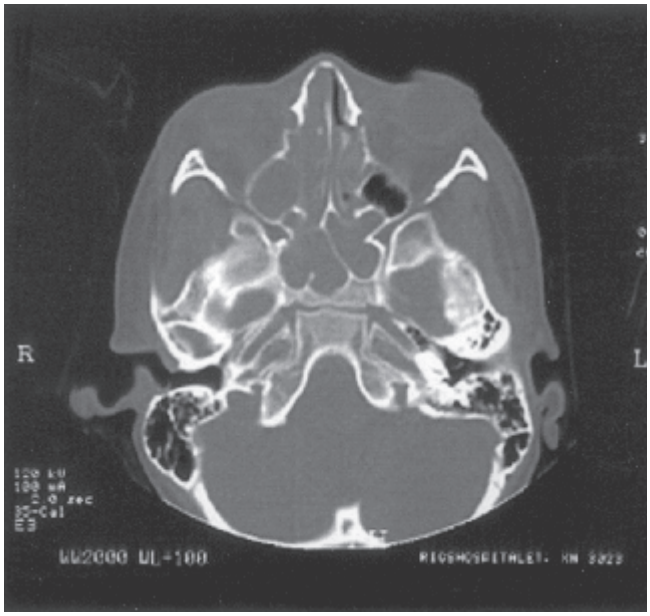

Figure 2. Preoperative CT demonstrating pansinusitis.

The next morning, a left-sided hemiparesis, left-sided central facial nerve palsy, right-sided abducens nerve palsy and somnolens were observed. A CT scan confirmed the pansinusitis as well as an infarction in the right lentiform nucleus and the anterior part of the right internal capsule (Figure 1). The girl was transferred to our hospital. A preoperative CT for surgical planning re-confirmed pansinusitis (Figure 2), and she was immediately operated on with endoscopic opening of the sinuses including the ethmoid and sphenoid sinuses. Pus was removed from several of the sinuses including the sphenoid sinuses. Culture of the pus showed non-haemolytic *Streptococcus*. A lumbar puncture excluded meningitis. The antibiotic treatment was changed to high doses of cefuroxime, netilmicin, and metronidazole. In addition, the patient was treated with solumedrol. In the following days she still suffered from a severe left sided hemiparesis, left-sided central facial nerve palsy and a right-sided abducens nerve palsy. A MRI performed 10 days after the onset of the neurological symptoms showed a secondary haemorrhage in the infarction in the right lentiform nucleus and anterior part of the internal capsule (Figure 3a). In addition, a MR angiography (MRA) showed narrow internal carotid arteries and narrowing of the first part of the right anterior and medial cerebral arteries (A1 and M1 part). The posterior cerebral arteries were normally calibrated (Figure 3b).

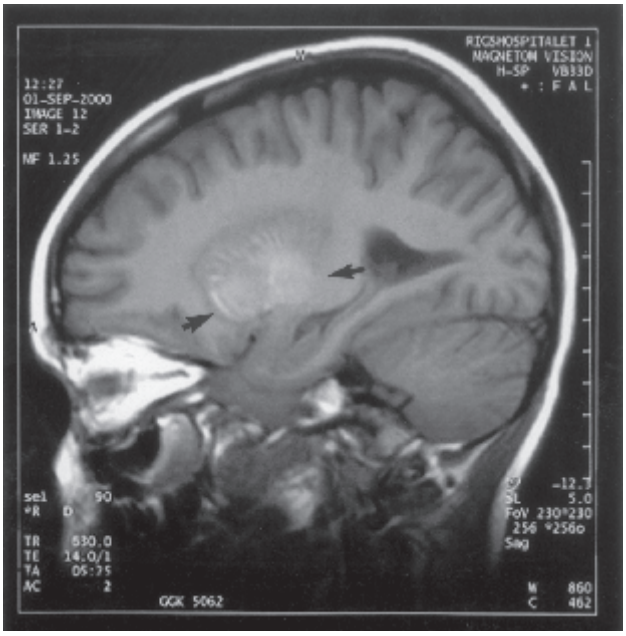

Figure 3a. MRI, saggital non-enhanced T1-weighted image 10 days after the onset of neurological symptoms, demonstrating secondary haemorrhage in the infarction (arrows).

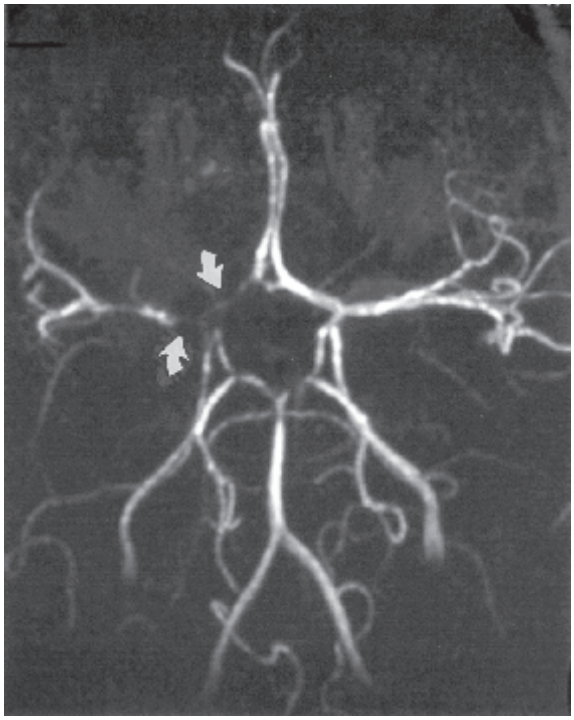

Figure 3b. MRA- TOF (time of flight), the same day as Figure 3a, demonstrating narrow internal carotid arteries, especially on the right side, and severe narrowing of the first part of the right anterior and medial cerebral arteries (A1 and M1, arrows).

The girl was further investigated for tromboembolic disposing factors without any significant results. The investigations included a full blood count, differential white cell count, erythrocyte sedimentation rate, coagulation factors, fibrinogen, plasminogen, protein C and S, plasma cholesterol, triglycerides, homocysteinuria, methylmelanolate, IgM and IgG cardiolipin antibodies, lupus anticoagulant, and DNA antibodies.

The girl was discharged from the paediatric department to a rehabilitation centre 6 weeks after the onset of symptoms. At that time she still suffered from a left-sided hemiparesis, but the paresis had decreased markedly over the 6-week period after her stroke. She was able to walk without assistance, and she no longer had abducens nerve palsy.

DISCUSSION

To our knowledge ischemic stroke has never been reported as a complication of sinusitis without additional meningitis. In a recently published article, four case reports with stroke and MR findings interpreted as sinusitis were presented (Perez Baretto et al., 2000). Apparently, sinusitis was diagnosed from MR findings alone without clinical or bacteriological confirmation. All of the patients presented had acute ischemic stroke secondary to tight stenosis or occlusion of the internal carotid artery. Our patient had a narrow first part of the right medial cerebral artery on the MRA (the M1). The ischemic stroke was in the area of the brain supplied by the right medial cerebral artery, so we believe the stroke was caused by cerebral ischemia.

The sinuses are, with exception of the maxillary sinuses, closely related to the cranial cavity and the sinuses are only separated from the cavity by a thin bone wall. The carotid artery is, in the cavernous sinus, very closely related to the sphenoid sinus, and we speculate whether this close relation can result in a direct perivascular inflammatory reaction resulting in a vascular thrombosis. Septic emboli does not seem to be a reasonable explanation for the wide and extensive arterial vessel changes we observed. We know our patient's right-sided cavernous sinus was affected in accordance with the observed right-sided abducens nerve palsy. Considering the narrow carotid arteries and the severe narrowing of the first part of the right anterior and medial cerebral artery shown on the MRA, it even seems more likely that a chemical or inflammatory reaction has caused vascular spasms as it is seen in patients with subarachnoid haemorrhage. The theory of cerebrovascular spasm in patients with sinusitis has been suggested once previously in the literature, where the authors found carotid artery spasm in a patient with severe sphenoid sinusitis and incipient cavernous sinus thrombosis (Whitehead and Souza, 1974). The time span from the onset of our patient's symptoms and the ischemic stroke was approximately 10 days. This is in accordance with findings in patients with subarachnoid haemorrhage (Mayberg, 1998). The reasons for vascular spasms seen in subarachnoid haemorrhage are still under debate (Mayberg, 1998; Dietrich and Dacey, 2000; Megyesi et al., 2000).

Our patient was extensively examined for underlying deficiencies or diseases previously reported as disposing for thrombosis without any significant results (Kirkham, 1999; Kirkham et al., 2000).

The presented case report emphasises the need for early and correct diagnosis and treatment of sinusitis, especially sphenoid sinusitis, since the complications can be life threatening with severe sequelae for the patient.

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