Recurrent bacterial meningitis caused by an occult basilar skull fracture

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Background: **We present a rare case of recurrent bacterial meningitis caused by an occult basilar skull fracture.**

Methods: **A 9-year-old boy presented with acute headache, fever and vomiting. He had recurrent meningitis episode one month earlier and a head trauma 6 months ago. Laboratory findings and lumbar puncture suggested an intracranial bacterial infection. Computerized tomography, magnetic resonance imaging and nasal endoscopy failed to find the presence of rhinorrhea. Spiral computed tomography was performed and a three-dimensional reconstruction of the bony cranium was done.**

Results: **A diagnosis of bone defect in the ethomoid was made. An endoscopic operation was performed to repair the defect successfully and the child was completely normal during a 4-month follow-up.**

Conclusions: **The skull base should be evaluated radiologically to find one or multiple bony defects in case of recurrent meningitis in absence of cerebrospinal fluid rhinorrhea. Compared to other neuroradiological technologies, three-dimensional computed tomography provides a better three-dimensional definition of the basilar skull fracture for both diagnosis and surgical planning. When the fracture is located in the anterior skull base, an endoscopic transnasal approach is considered the best option.**

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Key words: **bacterial meningitis;**
basilar skull fracture;
cerebrospinal fluid rhinorrhea;
spiral computed tomography

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Introduction

Intracranial infection, a raised erythrocyte sedimentation rate, an increased cerebrospinal fluid (CSF) leucocyte count and CSF protein concentration are often detected by laboratory examinations. Sometimes patients may be in a normal and recurrent status, but if not diagnosed and treated in time, they may die within hours. Rhinorrhea, otorrhea, otitis media, sinusitis, lumbar puncture, mondini dysplasia and ventricular drainage are known to be responsibe for recurrent bacterial meningitis. Also immunodeficiency is a potential cause for patients with agammaglobulinemia or terminal complement system defects.

Interestingly, anatomical communication between the subarachnoid space and skin or a non-sterile body cavity[1] may help comprehend the mechanism underlying recurrent pyogenic meningitis. In such cases, CSF rhinorrhea or otorrhea is a common symptom. However it is difficult to discern recurrent meningitis caused by skull base fragments in patients without rhinorrhea or otorrhea. Thus, localization of the lesion is more urgent. We present a pediatric patient suffering from recurrent meningitis without rhinorrhea or otorrhea.

Case report

A 9-year-old boy was admitted to the neurology department of our hospital because of headache, fever, nausea and vomiting. He had a meningitis episode one month before, a head trauma in a motorcycle accident 6 months ago, and recurrent sinus infection episodes in the past few years.

At the first admission one month before, the boy was febrile at 41ºC with chills. Neurological examination suggested neck stiffness and positive Kernig's sign. Blood tests showed a significant increase in leukocyte count ($20.16\times10^3/\mu L$) and C-reactive protein (CRP) level (110 mg/L), and proportion of neutrophils was 89%. CSF results were as follows: white blood cell (WBC) count $88/mm^3$, protein 0.65 g/L, and glucose 2.24 mmol/L. Microbial agents were not detected in blood cultures or CSF. Waking period recorded by electroencephalogram showed a 2-6Hz increase in θ and δ waves, suggesting part cerebral functional deprivation. However, cranial computed tomography (CT) was normal, and cranial

Case report

magnetic resonance imaging (MRI) revealed nothing special except for a poor display of pituitary from a possible empty sella. Immediately mannitol was used (120 ml every 8 hours, intravenously) to reduce the enhanced intracranial pressure when the patient was diagnosed with purulent meningitis. Also, antibiotic treatment with ceftizoxime (1.25 g every 12 hours, intravenously) and ceftazidime (1 g every 8 hours, intravenously) was started. Three weeks later the symptoms were alleviated completely, and blood and CSF tests were all normal. After 24 days the patient was discharged from the hospital and subsequently followed up.

At the second admission, the patient was febrile at 39.2ºC. Decreased consciousness, neck stiffness and highly suspicious positive Kernig's sign were noted. Blood test suggested WBC 31.96×10^3/μl, neutrophil 95%, CRP 117 mg/L and erythrocyte sedimentation rate (ESR) 41 mm/h. Lumbar puncture showed increased WBC (69/mm^3, multinucleated cells 33%), protein (2.21 g/L), and decreased glucose (1.18 mmol/L) in turbid CSF. Gram stain was negative. No direct evidence revealed tuberculous or fungal infection. To exclude the potential immune disease, enzyme linked immunosorbent assay was used with antibodies against HIV, and the result was negative. In addition, immunoglobulin G, A, and E levels were all within normal limits.

Cranial CT and pinna CT showed nothing special. CT scan of paranasal sinuses suggested inflammation in the bilateral maxillary sinus and left ethmoid sinus. Cranial MRI series revealed abnormal signals in the left frontal sinus and ethmoid sinus (Fig. 1). An empty sella was also observed, which was insignificant in the diagnosis of a CSF leak. Initially, 20% mannitol (125 ml every 8 hours, intravenously) was applied. Meanwhile for uncertain pathogens, the patient was treated with ceftriaxone sodium (1 g every 12 hours, intravenously) and ceftazidime (1 g every 8 hours, intravenously) empirically. Within a two-week antibiotic therapy, the symptoms of meningeal signs were alleviated, blood and CSF tests suggested normal clinic parameters. However, occasional fever still emerged in the patient frequently.

Because of the concurrence of repeated sinus infection and recurrent meningitis, the patient was reevaluated for further therapy. No CSF rhinorrhea or otorrhea was found by otorhinolaryngology examination. Nasal endoscopy revealed bilateral congestive nasal mucosa, a swelling on the left inferior turbinate and middle turbinate, left-deviated nasal septum and sticky nasal discharge in the middle and common nasal meatus. However, all the results above were insufficient for CSF rhinorrhea. Fortunately, diagnostic bone fragment in the ethmoid was found by three-dimensional CT (Fig. 2). Then nasal endoscopy was performed again at day 23 and the findings showed significant hypertrophy of the left uncinate process, thickening of the left anterior ethmoid and frontal sinus mucosa, and a bone defect sized 1.0 × 0.8 cm^2 at the top of the left ethmoid. Endoscopic repair of the anterior cranial fossa was done at once. Briefly, we first resected the left uncinate, then opened the left anterior ethmoid and frontal sinus. Finally, we cleaned the lesions in the sinus. After that, anterior one third of the middle turbinate was resected and prepared for a graft, and the mucosa was incised to fit the edge of the turbinate bone. The graft was modified into an appropriate size and plugged into the defect. The edge of the graft was approximately adjacent to the nasal mucosa. Fibrin glue was placed on the graft and surrounding mucosa. In addition, gelatin sponge

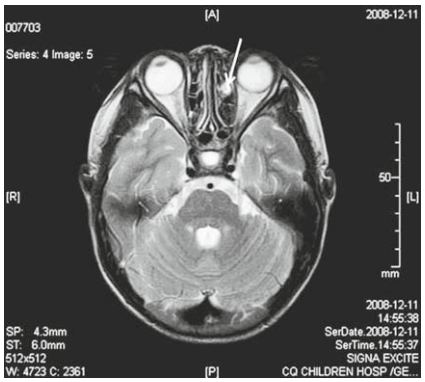

Fig. 1. T2-weighted axial magnetic resonance imaging shows abnormal signals in the left ethmoid sinus in the direction of arrow, which indicates a possible ethmoiditis.

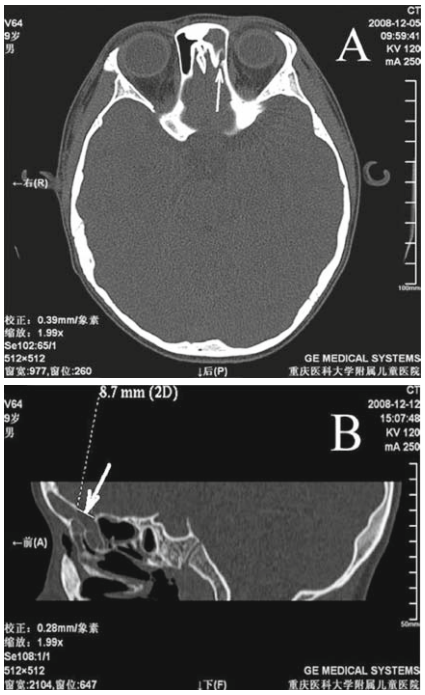

Fig. 2. Bone defect on posterior wall of frontal sinus in the direction of arrow on axial (**A**) cranial 3D-CT scan by a 64-slice spiral CT scanner. On sagittal (**B**) scan, the bone defect can be seen at the top of ethmoid with an anteroposterior diameter of 0.87 cm (in the direction of arrow).

was applied for further support. Finally, two pieces of iodoform gauzes were filled in the left middle nasal meatus. After the operation, the patient was asked to stay in a supine position on the bed. Antibiotics and mannitol were also prescribed. Nasal gauzes were removed 5 days after the operation. After 35 days of hospitalization, the boy completely recovered. The next 4-month follow-up showed that the patient was normal.

Discussion

Because the skull and dural mater are important cerebral barriers, any native or acquired defect in them may result in intracranial infection. The present case is unique because of occult basilar skull fracture associated with a recurrent meningitis history but no rhinorrhea. One pediatric patient with two different congenital skull base defects displaying similar symptoms has been reported,[2] in whom skull base defects were detected by careful skull base imaging. However, routine X-ray examination may fail to discern the above defects, then special radiological techniques may be helpful. 3D-CT offers a problem-solving option and the precise three-dimensional location of lesions after viewing axial images. It is particularly used in the emergency department to identify complex fractures and dislocations.[3] In our case, cephalic 3D-CT scan was made by a 64-slice CT scanner and a 3D reconstruction of the bony cranium was done, and finally the skull defect was found.

The management of recurrent bacterial meningitis consists of appropriate use of antibiotics and support therapy such as a supine position on the bed and avoidance of straining during an acute episode.[4] When the symptoms of meningeal signs are resolved, the anatomical or immunological defect is corrected if possible. The former may require surgical closure and endoscopic endonasal treatment is one of the most effective and feasible alternatives when the defect is located in the anterior skull base or sphenoid sinus.[5] Currently, more traumatic transcranial and extracranial surgical techniques have been replaced by nasal endoscopy in the repair of CSF rhinorrhea. However, the effectiveness of endonasal surgery is not satisfactory when the defect is on the posterior frontal sinus wall or is comparatively large. Various repairing materials have been used. Autogenous graft is the gold standard for skull repair in children because of its good integrated ability over time.[6-8] The plastic autogenous materials such as abdominal fat, rotated middle turbinate flap, nasal septum mucosal flap and muscle are commonly used in defect repair. The defect repair is made with a combination of plastic materials, and fibrin glue is often used for fixation of plastic materials.[9] If the defect is too large to be repaired by autogenous tissue only, synthetic materials such as hydroxyapatite cement, methylmethacrylate and mesh plates are needed. In our case, bone defect without CSF leak was at the top of the left ethmoid with a size of 1.0×0.8 cm^2, therefore we preferred the middle turbinate as an autogenous graft.

In summary, the skull base should be radiologically evaluated to find one or multiple bony defects in case of recurrent meningitis in absence of CSF rhinorrhea. High resolution three dimensional CT is crucial in detecting the hidden pathological changes that MRI or plain CT scans may miss. The treatment of basilar skull fracture is solely surgical. For a comparatively small defect in the anterior skull base, transnasal endoscopy might be a more effective approach. In addition, a long-term follow-up is also necessary since recurrent central nervous system infection is possible in patients with skull defect.

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