Oral Wooden Stick Injury Complicated by Meningitis and Brain Abscess

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Meningitis is rarely seen following oral injury. We describe a 3-year-old boy developing meningitis and brain abscess following a penetrating oral wooden stick injury. There was no cerebrospinal fluid rhinorrhea noted. A cerebrospinal fluid culture yielded viridans streptococcus. Brain magnetic resonance imaging and computed tomography revealed a multiloculated ring-enhancing mass. This patient underwent surgical drainage and completed 8-week antibiotic therapy. The patient demonstrated a late and dismal complication of a penetrating oral injury. At 2-year follow-up the patient was in good condition. A penetrating oral wooden stick inury should be regarded as potentially serious. *(Chang Gung Med J 2002;25:266-70)*

Key words: trauma, meningitis, brain abscess, viridans streptococci.

In the past two decades, two most-common causes of the intracranial collection of pus were ear, and nasa-oral cavity infections and trauma.[1] Development of brain abscess in patients with head injury and penetration of the dura is well established.[2] However, to our knowledge, brain abscess following a minor penetrating oral injury has never been reported. There has been only one reported case of meningitis complicated by a chopstick-penetration injury into the nasal cavity.[3] We describe a case of a 3-year-old boy who developed meningitis and brain abscess following an oral penetrating injury.

CASE REPORT

A 3-year-old boy fell down and sustained a penetrating oral injury by a wooden stick. The wooden stick was 25 cm long and had a sharpened end for affixing to a corn on the cob. The stick penetrated the nasal cavity between the upper incisior and upper lip; it was immediately removed by his father at home. His father stated that the depth of penetration of the wooden stick from the upper lip was around 10 cm. It was estimated that the stick penetrated between 3 and 5 cm into the nasal cavity. Mouth and nose bleeding occurred but stopped after intervention by an otolaryngologist at a local clinic. No cerebrospinal fluid (CSF) rhinorrhea was found. He had a fever of up to 39¢J the day following the trauma and was referred to our hospital 3 days later. On admission, the patient's consciousness was clear, but with irritable crying and a headache. Vital signs were: a temperature of 38¢J, heart rate of 76 beats/min, respiratory rate of 34/min, and blood pressure of 100/60 mmHg. He was normocephalic, but his neck was stiff. His cardiopulmonary exam was normal when at rest. The abdomen was soft, and flat, with no palpable masses. His extremities were normal with full range of motion. On neurologic

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examination, he was alert and speech was fluent. Pupils were 3 mm and symmetrical. Light reflex and cranial nerve examination were intact. Muscle power, muscle tone, and deep tendon reflex were all within normal range. An otolaryngologist was consulted, and no wound was found in the nasal cavity or sinus. Investigations revealed hemoglobin of 87 g/L, white blood cell count of 27,100/mm^3 (90% polymorphonuclear neutrophils, 5% lymphocytes, and 5% monocytes), and platelet count of 175,000/mm^3. C-reactive protein (CRP) was 188,000 μg/L. Lumbar puncture yielded cloudy CSF which showed 1690 leukocytes/mm^3, of which 73% were neutrophils, 15% were lymphocytes, and 12% were monocytes. Protein level was 0.85 g/L, and glucose was 2.6 mmol/L. Blood glucose was 9.4 mmol/L. Antigens screened by latex agglutination of the CSF were negative for *Haemophilus influenzae, Neisseria meningitidis, Streptococcus pneumoniae*, and group B streptococci. The Gram-stained smear was negative. CSF and blood cultures grew no bacteria. A plain skull roentgenogram was unremarkable. There was no CSF rhinorrhea throughout the course. The antibiotics ampicillin (800 mg, 4 times daily) and cefotaxime (800 mg, 4 times daily), were administered parenterally for 12 days. The patient became afebrile after 4 days of hospitalization. CRP was 48,500 μg/L on day 4 and 5000 μg/L on day 10. He was discharged in a stable condition without meningeal signs after a 12-day hospital course. However, the patient began to complain of headaches and vomiting 16 days after discharge (30 days after injury). In addition, neck stiffness was again noted. Physical examination revealed an acute ill-looking appearance but with clear consciousness. No focal neurologic sign was found, and he had a stable gait when walking. Lumbar puncture at the second admission showed turbid CSF with 700 leukocytes/mm^3, of which 78% were neutrophils, 8% were lymphocytes, and 14% were monocytes. Protein level was 2.36 g/L, and glucose was 0.1 mmol/L. The Gram stain revealed no bacteria. The CSF culture from the second lumbar puncture grew alpha-hemolytic viridans streptococcus which was sensitive to penicillin. Brain magnetic resonance imaging (MRI) without and with contrast showed a multiloculated abscess in the left deep frontal area involving the septum pellucidum with obvious perifocal edema noted. Post-contrast study showed multiloculated enhancement of the lesions (Fig. 1). Brain computed tomography (CT) showed similar pictures. Brain abscesses were diagnosed. No

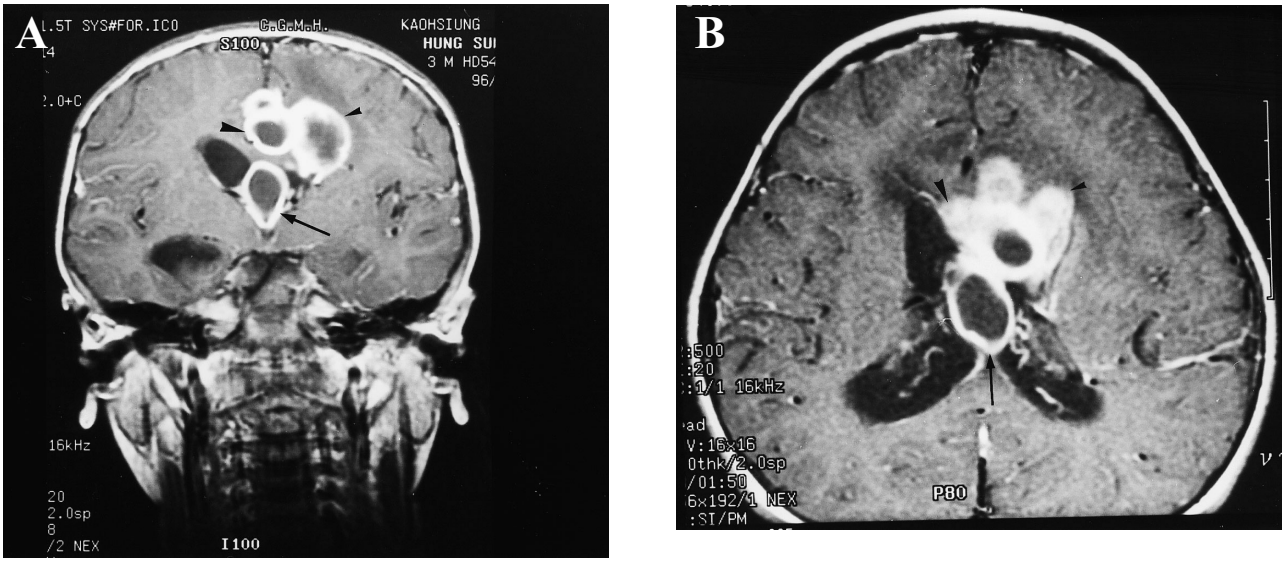

Fig. 1 (A) T1W1 coronal and (B) axial views of brain MRI after gadolinium-DTPA contrast administration showing multiloculated ring-enhancing masses at the corpus callosum (large arrowhead), septum pellucidum (arrow), and left frontal region (small arrowhead).

pneumocranium or any evidence of a fistula was found on brain CT and MRI. He underwent brain surgery with drainage of pus and a temporary ventriculostomy 1 week after the second hospitalization (37 days after injury). The pathology showed inflammatory cells present without foreign bodies. However, the pus culture yielded no growth. He was treated initially with cefotaxime, metronidazole, and penicillin, followed by intravenous penicillin therapy alone (1,600,000 unit, 4 times daily) for 6 weeks. Diminution of the abscess was shown by the serial follow-up brain CT. MRI at the end of therapy showed a residual abscess with dilated left occipital horn, while the electroencephalogram was negative before discharge. He was discharged in a stable condition and continued a further 2-week oral penicillin treatment. At 2-year follow-up, the patient was in good condition clinically.

DISCUSSION

In children, penetrating injuries to the brain are particularly insidious.[4] In this setting, brain MRI and CT scan are valuable for defining whether cranial penetration occurred.[5] In our patient, brain image study was not performed at the first admission because the patient had no CSF rhinorrhea and appeared to have made an uneventful recovery. Generally, imaging studies of the brain are needed only in patients at increased risk of cerebral herniation following acute meningitis.[6] To our knowledge, penetrating oral injury complicated with meningitis and brain abscess has never been reported. Neuroimages at the second admission in this case revealed no evidence of a fistula. In patients with craniofacial trauma, intracranial infections may occur via direct meningeal contamination, through areas of osteitis or osteomyelitis, or by retrograde thrombophlebitic spread via diploic or emissary veins into the intracranial compartment.[7] The presence of a CSF fistula is not essential to the development of meningitis, but rather providies direct access of pathogenic organisms to the meninges.[8] Penetrating injury with direct inoculation of the pathogenic organism into the bloodstream followed by intracranial involvement was the possible pathogenesis in this patient. Retained wood fragments, if present, can be an infectious nidus also; however the imaging study and the pathology report revealed that no wooden fragments had been retained.

Meningitis rarely occurs following head trauma, with a frequency of about 1% in all head trauma cases.[9] The most common causative organism of post-traumatic meningitis is *Streptococcus pneumoniae*.[10] In our patient, the organism was an alpha-hemolytic viridans streptococcus, which is part of the normal flora of the oral cavity. One epidemiologic report showed the incidence of bacterial meningitis caused by viridans streptococci in children in southern Taiwan was around 2.6%.[11] Neurosurgical procedures and head trauma are 2 major predisposing factors for childhood meningitis caused by viridans streptococci.[12]

Post-traumatic recurrent meningitis has been reported, usually in the presence of a skull fracture or persistent CSF rhinorrhea.[13] In patients with recent head trauma or a neurosurgical procedure and negative CSF culture data, empiric therapy is essential and should be directed to the most-likely pathogens: staphylococci, gram-negative bacilli, or *Streptococcus pneumoniae*.[14] Broad-spectrum antibiotics effective against both gram-positive and gram-negative organisms should be given. The duration of antibiotic therapy should be individualized on the basis of clinical and microbiologic responses.[14] This patient was unusual because he had no evidence of bone change, dura tear, or a fistula tract, but developed brain abscess later. We suggest that extended antibiotic therapy with close follow-up is manadatory for selected post-traumatic patients.

The occurrence of brain abscess following head injury is uncommon, with a reported incidence of around 3% to 16%.[2] In addition, the injury-infection interval is about 2 to 3 weeks.[15] Some minor puncture injuries may cause brain abscess,[5] especially in children, as was found in our patient.

Ikeda et al. reported a case of recurrent bacterial meningitis following chopstick injury with CSF rhinorrhea.[3] Louie et al. reported a 13-month-old child with brain abscess who, 2 weeks prior, had undergone rigid endoscopy for extraction of a coin from the esophagus.[16] In that case the presence of an esophageal coin was initially asymptomatic; the coin had been there for weeks prior to removal. Bautista-Casasnovas et al.[17] and Lui et al.[18] reported that brain abscesses can be secondary to esophageal

dilatations. Bautista-Casasnovas et al. hypothesized that the pathogen originated from the oropharynx and esophagus and was introduced into the bloodstream during esophageal dilatation.[17] We postulate that brain abscess and meningitis in our patient were secondary to penetration and infection of the oropharynx following the wooden stick injury

In conclusion, the patient in this report is unusual in that a penetrating oral wooden stick injury without CSF rhinorrhea or fracture caused subsequent meningitis and brain abscess. Wooden sticks are often used when preparing food in our country and nearby Asian countries. Thus penetrating oral wooden stick injuries are probably not uncommon, especially in children. Although symptoms may be clinically inconspicuous in the beginning, they should be regarded as potentially serious. Clinical follow-up and neuroimaging studies in such cases are mandatory.

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木籤造成口腔穿破性創傷併發腦膜炎及腦膿腫

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口腔外傷後引發腦膜炎是相當少見的。這名3歲大男童在一次意外的木籤穿刺口腔造成創傷之後第3天引發了腦膜炎而住院治療。改善出院後，卻在受傷後第30天（第1次出院後第16天）因頭痛及發燒回院求治而發現了腦膿腫。腦脊髓液培養發現草綠色鏈球菌，而腦部磁振照影和電腦斷層發現了多葉狀環狀腫塊。之後病人接受神經外科的引流手術以及持續8週的抗生素治療後獲得改善，而且在門診持續追蹤2年並無復發。病人因爲這一個口腔穿破傷口而產生嚴重的併發症。我們認爲這種因木籤在口內造成的傷害必須注意它潛在的危險性。(長庚醫誌 2002;25:266-70)

關鍵字：創傷，腦膜炎，腦膿腫，草綠色鏈球菌。

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