Bilateral Sudden Deafness as the First Manifestation of *Streptococcus Suis* Capsular Type 2 Meningitis

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Streptococcus suis meningitis is an occupational hazard of pork handlers. Hearing loss is one sequela of this illness. We report one butcher with *Streptococcus suis* capsular type 2 meningitis who initially presented with bilateral profound sensorineural hearing loss; meningitis was diagnosed by cerebrospinal fluid analysis. The organism was isolated and showed susceptibility to penicillin. After treatment with intravenous penicillin, all symptoms of meningitis improved except for his bilateral deafness. The clinical course and histological cause of this case are reported herein. **(Mid Taiwan J Med 2005;10:155-8)**

Key words

meningitis, *Streptococcus suis*, sudden deafness

INTRODUCTION

Streptococcus suis (*S. suis*) is a zoonotic bacterium. It was first identified as a cause of septicemic infections in pigs by de Moor in 1959 [1]. The organism causes septicemia, meningitis, polyserositis, arthritis, pneumonia and endocarditis in piglets. Human infection by *S. suis* was first reported by Perch et al in 1968 and almost all of the reported cases have occurred in pig farmers or pig meat handlers [2]. We report a case in which a patient initially presented with bilateral sudden deafness but whose condition was finally diagnosed as *S. suis* capsular type 2 meningitis.

CASE REPORT

A previously healthy 38-year-old male butcher presented with a half-day history of sudden onset bilateral hearing loss, accompanied by persistent low-pitched tinnitus. No history of trauma, dizziness, nausea, vomiting, headache, fever or chills was noted. Physical examination revealed a fully conscious male with bilateral intact eardrums. His temperature was 36.1˚C, blood pressure 87/58 mmHg, and pulse rate 87 per minute. Neurological examination results were normal other than bilateral sensorineural hearing loss. Blood test showed a white blood count of 8.79 $\times$ 10^9/L with 90.4% neutrophils, 7.98% lymphocytes and 1.49% monocytes, and a platelets count of 12,100/mm^3. Bilateral profound sensorineural hearing loss was confirmed by pure tone audiometry. Brain computed tomography (CT) demonstrated no brain abnormalities. He was admitted and then treated for bilateral sudden deafness.

On the second day after admission, he complained of severe headache and progressive disorientation. In addition, fever, chills, neck stiffness, positive Kernig sign and Brudzinski sign were noted. Meningitis was confirmed by lumbar puncture, which revealed turbid cerebrospinal fluid (CSF) containing 800 leucocytes/μL (93% of which were segmented)

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and Gram-positive cocci arranged in pairs and short chains. Pneumococcal meningitis was suspected and vancomycin 500 mg intravenous drip (IVD) q6h with cefotaxime 2.0 g IVD q6h was started. Two days later, CSF culture yielded growth of streptococci; treatment was changed to penicillin 3×10^6 U IVD q6h. Further analysis of the streptococci by API 20 Strep (BioMerieux, Lyon, France) identification strip, a commercially available laboratory kit with high stability and good reproducibility between different laboratories, revealed a strain of *S. suis* capsular type 2 [3], that was sensitive to penicillin. Fever subsided on the eighth hospital day. Owing to persistent hearing impairment, the patient underwent an auditory brainstem response which revealed poor wave formation in both ears and indicated retrocochlear lesions. He was discharged in good clinical condition except for the deafness after a 2-week treatment course. At 1 year follow-up visit, he still suffered from bilateral deafness.

DISCUSSION

Three main theories exist to explain idiopathic sudden deafness: viral infection, vascular compromise, and intracochlear membrane rupture. The treatment regimens for sudden deafness are diverse because of its uncertain etiology. The therapies include vasodilators, rheologic agents, anti-inflammatory agents, and antiviral agents. Our patient's condition is notable in that his initial complaint was bilateral sudden deafness. Corticosteroid therapy was contemplated until meningitis was confirmed by CSF analysis. Since 47% to 64% [4] of patients with *S. suis* meningitis develop permanent deafness, physicians should be alert to the entity.

S. suis is a commensal organism in the tonsils of young weaned pigs. By 1991, 28 capsular types had been characterized, with type 2 being the most pathogenic among pigs, as well as being the predominate serotype described in human infections [5]. *S. suis* is thought to enter the upper respiratory tract of both pigs and humans and has been found in the tonsils of asymptomatic pigs. In humans, the organism may gain access through cuts and abrasions. Most infected subjects have had occupational exposure to pigs in slaughter houses, as pig farmers, butchers, pork meat transporters, meat inspectors, and employees in food-processing industries concerned with pork preparation [6].

Human infection by *S. suis* was first reported in 1968. Most of the patients presented with meningitis, followed by septicemia, arthritis, endocarditis and endophthalmitis. One of the most serious complications of *S. suis* meningitis is deafness which is likely to remain permanent. Unilateral or, more commonly, bilateral hearing loss has been documented in 47% of the European and 64% of the Asian cases. The hearing loss is often severe and may be accompanied by vertigo. It either improves slightly or not at all even after treatment of the meningitis. The hearing loss is likely to be due to suppurative labyrinthitis caused by the invasion of the perilymph via the cochlear aqueduct by *S. suis* [7-9]. In 1995, Osborne proposed that hearing loss is associated with bacterial invasion of and damage to the organ of Corti which results in multiple lesions within the cochlea. Lesions within the cochlea will almost certainly result in permanent hearing loss [8]. Saumil reported that spiral ganglion cells in one patient were severely degenerated, indicating a retrocochlear site of hearing loss in addition to the cochlea [9]. Some authors have also proposed that a specific exotoxin produced by *S. suis* may be responsible for the hearing loss [8,10]. Perceptive deafness followed by meningitis may be caused by other infections (e.g. typhoid, syphilis and Cryptococcus meningitis), inflammatory conditions such as polyarteritis nodosa, and Cogan's syndrome [11]. However, the hearing loss is rarely as prominent in those conditions as it is in *S. suis* capsular type 2 infection.

Usually, infection by *S. suis* has a favorable outcome when treated by appropriate therapy because *S. suis* is generally susceptible to penicillin. In our patient, symptoms and signs of meningitis improved after therapy with penicillin for 8 days. However, he continued to suffer from

bilateral perceptive deafness.

In conclusion, *S. suis* is a zoonotic bacterium that tends to affect individuals with an occupational exposure to pigs or pork. *S. suis* is remarkably invasive even in healthy adults. Severe sequelae of permanent perceptive deafness are characteristic of *S. suis* meningitis. When evaluating patients with sudden deafness, history of occupation, lifestyle and habits should be taken to avoid misdiagnosis of sudden deafness caused by *S. suis* meningitis.

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以雙側突發性耳聾為初始表現的 *Streptococcus suis* Capsular Type 2 腦膜炎

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Streptococcus suis 腦膜炎爲處理豬肉人員的職業傷害之一，而永久的神經性聽力障礙爲這種腦膜炎的常見後遺症之一。一位年輕的屠夫因爲雙側突發性耳聾而求診，經由腦脊髓液分析後證實感染 *Streptococcus suis* capsular type 2 腦膜炎。其病原菌被分離後發現使用 penicillin 並無抗藥性。病人在接受靜脈注射 penicillin 後，腦膜炎的症狀幾乎都改善了，卻造成永久的神經性聽力障礙。（中台灣醫誌 **2005;10:155-8**）

關鍵詞

腦膜炎，*Streptococcus suis*，突發性耳聾

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