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Scarlet Fever Caused by Community-associated Methicillin-resistant *Staphylococcus aureus*

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We describe a previously healthy 2½-year-old boy with staphylococcal scarlet fever associated with acute suppurative otitis media due to community-associated methicillin-resistant *Staphylococcus aureus*. The patient was successfully treated by spontaneous drainage in combination with trimethoprim-sulfamethoxazole therapy.

Key words: *Acute suppurative otitis media, Community-associated methicillin-resistant Staphylococcus aureus, Staphylococcal scarlet fever.*

Acute otitis media (AOM) is pervasive in children with high incidence rates reported both in developed and emerging nations [1]. Recently, strains of community-associated methicillin-resistant *S. aureus* (CA-MRSA) are increasingly found in skin and soft-tissue infections of children, usually linked to intravenous drug abuse, cystic fibrosis, chronic diseases and repeated antimicrobial therapy [2]. However, CA-MRSA otorrhea after tympanostomy tube insertion in well children has been reported

previously [3]. Here we describe a child with bilateral acute suppurative OM and staphylococcal scarlet fever (SSF) associated with CA-MRSA. To the best of our knowledge, this is the first pediatric case of such an association.

CASE REPORT

A previously healthy 2-year-and-6-month-old boy was seen in the emergency department because of the acute onset of turbid discharge with pus and bloody component over the left ear, and generalized

itching skin rashes followed by upper respiratory tract infection 4 days prior to admission. The family denied previous trauma or foreign body ingestion, and the boy had not been exposed to auricular, dental or oropharyngeal manipulation. Physical examination of the skin showed generalized pruritic scarlatiniform rash with positive blanching sign, but no enanthema, bullae or exfoliation. Oropharyngeal examination disclosed good dental hygiene, normal oral mucosa, and erythematous tonsils; no other abnormalities were noted. Other findings of physical examination were unremarkable.

Otorhinolaryngologists were consulted due to the abnormal discharge and poor visibility of both ear canals. After clearance, air-fluid level with pus formation over the right middle ear, and copious yellowish and bloody discharge through the perforated left eardrum were noted. The leukocyte count was 8,000/mm³ with 65.2% segmented neutrophils, C-reactive protein (CRP) level was 47 mg/l, and rapid antigen test for group A *Streptococcus* was negative; the remainder of the hematologic and blood chemistry findings were within normal reference ranges.

The diagnosis of bilateral acute suppurative OM and SSF was established and pus specimen, blood, and throat cultures were obtained. Cephazolin was empirically administered at a dosage of 100 mg/kg/day intravenously in three divided doses. Two days after admission, the initial pus cultures grew MRSA, susceptible in vitro to many antibiotics including gentamicin, trimethoprim-sulfamethoxazole, ciprofloxacin, fusidic acid, rifampin and vancomycin. The isolate was resistant to penicillin-G, cefazolin, oxacillin, clindamycin and erythromycin. Based on these findings, the antibiotic regimen was subsequently changed to oral trimethoprim-sulfamethoxazole. After 3 days of oral trimethoprim-sulfamethoxazole therapy, the patient's condition improved gradually and he was discharged home with a prescription of oral trimethoprim-sulfamethoxazole. Previous blood culture remained negative and throat culture revealed growth of normal pharyngeal flora. After completing a 2-week course of oral trimethoprim-sulfamethoxazole therapy, he was doing well at his 1-month follow-up evaluation with no evidence of recurrence.

Further molecular typing of the MRSA isolate revealed it carried the staphylococcal cassette chromosome *mec* (SCC*mec*) type V_T, possessed both Panton-Valentine Leukocidin (PVL) and staphylococcal enterotoxin B (SEB) genes, and multilocus sequencing typing (MLST) disclosed the genotype was sequence type (ST) 59. The presence of the *lukS-PV* and *lukF-PV* genes encoding PVL components and the *seb* encoding SEB were determined by polymerase chain reaction (PCR)-based method with the primer pair and thermocycler conditions [4]. SCC*mec* typing was performed using a multiplex PCR strategy with sets of region-specific primers [5]. MLST was performed by PCR amplification and sequencing of seven housekeeping genes using primers designed by Enright, *et al.* [6]. Each sequence was submitted to the MLST database website for assignment of the allelic profile and ST. These findings suggest that this case of bilateral acute suppurative OM and SSF was caused by ST 59, PVL- and SEB-positive CA-MRSA infection.

DISCUSSION

In recent times, there has been a steady increase in the number of cases of MRSA otorrhea [3], CA-MRSA has rarely been reported in discharging ears [2,3,7]. Previous cases of MRSA otorrhea were associated with bilateral myringotomy with tympanostomy tube insertion [3].

SSF is a rare disease first described in 1900 [8]. The diagnosis of SSF is traditionally made based on the consistent clinical manifestation of generalized scarlatiniform rash; no enanthem, bullae, or exfoliation; followed by desquamation similar to toxic shock syndrome and no evidence of streptococcal infection [8]. In a recent study of 49 children with SSF, cutaneous abscesses predominated (100%), and all responded to incision-and-drainage, manual expression of pustule contents, or spontaneous drainage, with fair outcomes despite treatment with ineffective antibiotics [9]. Review of literature suggests that CA-MRSA is very rarely associated with SSF originating from bilateral acute suppurative OM. Recovery of CA-MRSA from the pus specimen of our patient is strong evidence that this organism was the cause of both bilateral acute suppurative OM and SSF. Fortunately, our patient's

clinical condition improved after spontaneous drainage in combination with trimethoprim-sulfamethoxazole therapy.

Currently, the infecting strains of CA-MRSA in Taiwan are thought to have unique microbiologic characteristics such as resistance to multiple antibiotics (including clindamycin, erythromycin, tetracycline and chloramphenicol), different exotoxin gene profiles (*e.g.*, PVL and SEB), common pulsed-field gel electrophoresis patterns (which are different from those of the major pandemic clones of hospital-acquired MRSA), ST 59 genotype by MLST, and smaller SCC*mec* variants: SCC*mec* type V_T, or less frequently, type IV [5]. The microbiological characteristics of the CA-MRSA strain infecting our patient are consistent with the results of previous studies. The present case suggests that CA-MRSA should also be considered a potential cause of both suppurative OM and SSF in children.

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Congenital Myotonic Dystrophy with Asymptomatic Mother

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Myotonic dystrophy is an autosomal dominant neuromuscular disorder characterised by extreme pleiotropism and variability in disease expression. A congenital form is rare and is observed in infants born to symptomatic mothers with multisystem involvement. We report a case of a neonate with congenital myotonic dystrophy born to an asymptomatic mother.

Key Words: Asymptomatic mother, Congenital Myotonic dystrophy, Diaphragmatic weakness, Floppy infant.