

were also positive for IgM antibodies to *Orientia tsutsugamushi* by Scrub typhus detect IgM ELISA kit (Inbios, USA). Other additional important clinical findings included thrombocytopenia, anemia and low serum albumin.

#### DISCUSSION

Scrub typhus usually presents with fever, rash and complications involving respiratory, cardiac or central nervous system. Inoculation of *O. tsutsugamushi* through the bite of chigger is often painless and unnoticed [9]. Scrub typhus is common in rural areas. Out of five cases presented, 4 lived in Kuccha house and went for open field defecation which predisposes them to chigger bite. Appropriate history, and finding of eschar are often pathognomonic but can be missed by inexperienced observers. Lack of knowledge among physicians can lead to under diagnosis and improper treatment. Routine laboratory tests are normal; elevated transaminases and hypoalbuminemia can be used as pointer to investigate for rickettsial diseases. In resource poor countries, initial Weil felix test followed by ELISA based test for *O. tsutsugamushi* and *Rickettsia conorii* can make proper diagnosis. Although Indirect immunofluorescence assay (IFA) or Indirect Immuno-peroxidase test (IIP) and polymerase chain reaction (PCR) based tests are considered gold standard in confirmation of rickettsial diseases, they can only be performed in sophisticated laboratories, which was not possible in our case. We made the diagnosis based on clinical symptomatology along with two different tests (weil felix and IgM ELISA) and prompt response and recovery in response to doxycycline. Further studies are required to estimate the exact magnitude of disease in Sikkim.

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## Community-acquired *Streptococcus Viridans* Pneumonia in a Healthy Child

FANG-YIH LIAW, \*CHIH-CHIEN WANG, YAW-WEN CHANG AND \*SHYI-JOU CHEN

From the Department of Family and Community Health, and \*Department of Pediatrics, Tri-Service General Hospital, Taipei, Taiwan.

Correspondence to: Dr Shyi-Jou Chen, Department of Pediatrics, Tri-Service General Hospital, No. 325, Cheng-Kung Rd., Sec. 2, Nei-hu 114, Taipei, Taiwan. [pedneuchen@hotmail.com](mailto:pedneuchen@hotmail.com)

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*Streptococcus viridans* is usually considered to be nonpathogenic in healthy patients. Some strains become penicillin-resistant and cause life-threatening infections in immunocompromised patients. We report an immunocompetent boy who had community-acquired *S. viridans* pneumonia that was resistant to penicillin. Clinicians should note local patterns of virulence and antibiotic resistance in *S. viridans* and adjust treatment strategies accordingly.

**Key words:** Community-acquired pneumonia, Drug resistance, Immunocompetent,

**V**iridans streptococci are usually regarded as low-virulence alpha-hemolytic streptococci that commonly colonize the human oropharynx, gastrointestinal tract, and female genitalia. However, infection can result in bacteremia and may disseminate to distant sites in an immunocompromised patient.

*Streptococcus pneumoniae* is the most commonly identified pathogen in community-acquired pneumonia. *S. Viridans* is not commonly related to community-acquired pneumonia. Most *S. viridans* strains are sensitive to penicillin; nevertheless, the rate of penicillin resistance has increased steadily worldwide. Herein, we report the case of an immunocompetent 4-year-old boy who presented with cough, intermittent fever, and dyspnea attributed to penicillin-intermediate-resistant *S. viridans*-related community-acquired pneumonia.

#### CASE REPORT

A 4-year-old boy presented with intermittent fever and productive cough for 2 weeks. He was given medications to alleviate the symptoms; however, high fever and shortness of breath were noted 4 days later. The parents stated that the boy had no history of aspiration, dental caries, or dental extraction. He had previously received a four-dose pneumococcal 7-valent vaccination. There was no history suggestive of food or drug allergy, chest pain, or oliguria.

On initial examination, the patient had fever with a temperature of 39°C, a heart rate of 143/min, a respiratory rate of 28/min, blood pressure of 101/65 mmHg, and oxygen saturation on room air of 98%. Coarse breathing sounds combined with rhonchi over the right lung field were audible, and no heart murmur was revealed by physical examination. No skin rash was found over any part of the body. The remainder of the physical examination was unremarkable.

The patient's laboratory studies revealed a white blood cell count of  $8.53 \times 10^3$  cells/mm<sup>3</sup> with 60.5% neutrophils, 27.7% lymphocytes, and 11% monocytes; a hemoglobin of 13.3 g/dL; and a platelet count of 275000/mm<sup>3</sup>. C-reactive protein was 2.82 mg/dL. Mycoplasma IgM was undetectable from serum. An influenza rapid test was negative in a sample isolated from the nasopharynx. Initial chest radiography showed focal parenchymal consolidations over the right upper lobe.

We prescribed empiric antibiotics with cefuroxime and azithromycin at first. However, spikes fever persisted over 2 days. Two blood culture specimens grew *S. viridans* that were intermediately resistant to penicillin (MIC = 0.5 µg/mL) but sensitive to ceftriaxone and

vancomycin (MIC <0.5 µg/mL in both). Accordingly, the antibiotics were switched to ceftriaxone (600 mg every 6 hours; 100 mg/kg/day). Subsequently, we performed an echocardiogram that showed no regurgitation of the valves, no obvious vegetations, and normal left ventricular systolic function. The patient became afebrile and subjectively showed improvement within 24 hours of commencing intravenous ceftriaxone treatment. Blood culture, done 48 hours after beginning antibiotic treatment, was sterile. A recheck chest radiography 5 days after ceftriaxone therapy revealed only residual and small focal parenchymal consolidations over the right upper lobe. The species from our patient is identified as *S. mitis*. Therapy with ceftriaxone was changed to oral ceftibuten (200 mg daily), which was continued for 9 days. Consequently, the patient totally recovered without sequelae on follow-up, 1 month after discharge.

#### DISCUSSION

Viridans streptococci are often regarded as nonpathogenic bacteria; clinical isolation of *S. viridans* is usually attributed to specimen contamination. However, the organism is recognized as a cause of infective endocarditis in patients with valvular disease. In particular, it is commonly associated with dental caries followed by an episode of bacteremia and dissemination to remote sites. The lower respiratory tract is the most common site of infection [1]. In addition, *S. viridans* is recognized as an important cause of pneumonia and sepsis in neutropenic individuals [2]. Nevertheless, *S. viridans* is rarely reported to be the cause of pneumonia in immunocompetent patients, such as the case illustrated here.

In children with fever and neutropenia, Paganini and colleagues demonstrated an increasing incidence of viridans bacteremia paralleling the increasing use of histamine type 2 antagonists, the presence of oropharyngeal mucositis, prophylactic antibiotic treatment with cotrimoxazole or quinolones, severe neutropenia, and chemotherapy involving high doses of ARA-C [3]. Gingivitis and toothbrushes can play potential roles in viridans streptococcus bacteremia in immunodeficient patients [4]. However, no obvious predisposing factor has been identified in immunocompetent hosts with viridans bacteremia.

Gaudreau, *et al.* first reported the clinical relevance of bacteremia caused by *S. viridans* in 71 children in 1981 [5]. However, the report did not mention about the immune status of the children. In 1989, the first case series of primary pneumonia caused by *S. viridans* with bacteremia in healthy adults was reported and all of the cases were sensitive to penicillin [6].

In Brazil, Freitas, *et al.* reported that viridans streptococci rank as the second most frequent causal agent of community-acquired pneumonia in children under 5 years of age and that all of the affected patients appeared to be without underlying disease; in that study, other causes, such as degree of malnutrition or dental cavities, could not be excluded [7]. In our report, the patient did not have poor dental hygiene, malnutrition, or evidence of immune deficiency. Nevertheless, pneumonia was proved from chest radiography, and *S. viridans* bacteremia was identified from blood culture.

Inappropriate use of antibiotics may have resulted in the increased prevalence of antibiotic-resistant *S. viridans*. In recent decades, penicillin resistance has been progressively increasing in *S. viridans* [8]. Ioannidou, *et al.* analyzed 200 *S. viridans* cultures isolated from the oropharynxes of 96 healthy Greek children and documented different resistance rates to several antibiotics [9]. The prevalence of penicillin-resistant *S. viridans* varies worldwide, e.g., 37.8% in India [10] and 11% in the United Kingdom [11]. Furthermore, Smith, *et al.* pointed out that a significant proportion of penicillin-resistant *S. viridans* strains are susceptible to ceftriaxone [12].

The overuse of antibiotics may increase the incidence of infection by formerly nonpathogenic organisms such as viridans streptococci and may be a potential cause of bacteremia and pneumonia in the absence of other pathogens. Clinicians should note any increased local incidence of community-acquired pneumonia due to viridans streptococci and should select empiric antibiotic therapy based on the local pattern of antibiotic susceptibility among recent clinical isolates.

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