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Case report

Streptococcal toxic shock syndrome from necrotizing soft-tissue infection of the breast caused by a mucoid type strain

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ABSTRACT

Streptococcal toxic shock syndrome is a severe infectious disease. We report a Japanese case of Streptococcal toxic shock syndrome caused by a highly mucoid strain of *Streptococcus pyogenes*. A 31-year old female with shock vital sign presented at a tertiary medical center. Her left breast was necrotizing and *S. pyogenes* was detected by Immunochromatographic rapid diagnostic kits. Intensive care, including administration of antibiotics and skin debridement, was performed. After 53 days in our hospital, she was discharged. The blood cultures and skin swab cultures all grew *S. pyogenes* which displayed a highly mucoid morphology on culture media. In her course of the disease, the *Streptococcus* strain had infected two other family members. All of the strains possessed the T1 and M1 antigens, as well as the *emm1.0* gene. As for fever genes, the strains were all positive for *speA*, *speB*, and *speF*, but negative for *speC*. All of the strains exhibited and the same pattern in PFGE with the SfiI restriction enzyme. The strain might have spread in the local area by the data from the Japanese Infectious Disease Surveillance Center. Immunochromatographic rapid diagnostic kits are very useful for detecting *S. pyogenes*. However, they can not be used to diagnose severe streptococcul disease by highly mucoid strain alone. Careful observation of patients and colony morphology are useful methods for diagnosing severe streptococcal disease by highly mucoid strain.

1. Introduction

Streptococcal toxic shock syndrome (STSS) is a severe infectious disease, originally caused by *Streptococcus pyogenes* infection. STSS is not restricted to *S. pyogenes* infection recently, therefore it is any streptococcal infection associated with the sudden onset of shock and organ failure. We encountered a case of STSS caused by a highly mucoid strain of *S. pyogenes* complicated with necrotizing soft-tissue infection of the breast.

2. Case report

A 31-year-old female (patient A) presented on October 13 2010 with high fever, left breast pain, and pharyngalgia. She had no past history of serious illness, surgery, or hospitalization. She had two children, and her second baby (age: 2 months) was being breastfed at the time. She visited a hospital on October 15 and was diagnosed with purulent mastitis. Her left breast was hot, swollen, and tender, but did not display necrosis. Oral antibiotics and antifebriles were administered, and she was managed as an outpatient. She returned to the hospital the next day. At this point, she was febrile, and necrosis was observed on the skin of her left breast. A rapid immunochromatography-based diagnostic kit for the detection of group A *Streptococcus* (SEKISUI, Japan) was used, and her breast produced a positive result. Thus, blood cultures and skin swab cultures were taken. As a result of the abovementioned positive

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result, she was admitted to hospital and given intravenous antibiotics. On October 17th, she went into shock (systolic blood pressure less than 90 mmHg) and was transferred to our emergency medical center.

On arrival at the medical center, her vital signs were as follows: conscious level, GCS15; respiratory rate, 30 breaths/min; blood pressure, 121/48 mmHg (with 0.5 mg/h noradrenalin); heart rate, 135 beats/min; axillary temperature, 38.3 °C. Her left breast, especially around her nipple, displayed white colored necrosis on the lateral side and erythema on the medial side (Fig. 1). A laboratory evaluation revealed the following results: hemoglobin level, 11.9 g/dl; platelet count, 8.5×10^5/μl; white blood cell count, 2800/μl (neutrophils, 78.0%); C-reactive protein, 37.4 mg/dl; albumin, 2.8 g/dl; aspartate amino transaminase (AST), 129 IU/L; alanine amino transaminase (ALT), 61 IU/L; blood urea nitrogen (BUN), 34.5 mg/dl; creatinine, 1.06 mg/dl; Na, 131 mmol/L; Cl, 96.8 mmol/L; K, 4.3 mmol/L; prothrombin time/international normalized ratio (PT/INR), 1.02; activated partial thromboplastin time (aPTT), 31.0 s (control, 31.5 s). The patient's antistreptolysin O (ASO) and antistreptokinase (ASK) levels were measured at various time points: ASO: 45 IU/ml, ASK: <80 times on October 18th; ASO: 2520 IU/ml, ASK: <1280 times on November 6th; ASO: 1735 IU/ml, ASK: 320 times on December 7th. A chest CT scan revealed left breast swelling, bilateral pleural effusion, and regions of consolidation. Chest MRI did not detect any abscesses or necrotizing lesions in the mammary glands or within the soft tissue. *S. pyogenes* was cultured from her left breast swab sample, but was not cultured from her blood, as was found at the previous hospital. In addition, *S. pyogenes* was not detected in a throat swab obtained at admission to our hospital. We diagnosed her as streptococcal toxic shock syndrome (STSS) based on case definition for the STSS [1]: hypotension, erythematous macular and soft-tissue necrosis in this case. After admission, intravenous antibiotics and immunoglobulins were administered. Skin debridement was performed on October 19th, and *S. pyogenes* was cultured from the debridement sample. After suffering an extended period of high fever, her condition gradually improved. We were able to preserve her left breast without breastectomy. On November 16th, a skin graft transfer was performed, and the wound was closed. After 53 days in our hospital, she was discharged on December 8th with motor weakness in her left arm and thrombus in her femoral vein. Rehabilitation was continued for the weakness in her left arm, and warfarin was used to treat the thrombus. These symptoms had been cured by the end of the following March.

After her admission to our hospital, her brother (patient B) developed pharyngitis and became febrile (he had a temperature of 40 °C) on October 24th. He went to hospital the next day and was hospitalized on October 26th. *S. pyogenes* was cultured from a throat swab obtained on admission; however, he did not display any symptoms of streptococcal toxic shock syndrome. Patient B did not live in same house as patient A, but he cared for patient A when she was admitted to the previous hospital. He was administered intravenous antibiotics and was discharged on November 1st.

We checked the health status of patient A's two children because she was anxious about her family members becoming infected, especially her children. Her children did not present with fever or any other severe infectious symptoms; however, the first child (patient C, aged 5) displayed nasal discharge. Thus, we obtained throat and nasal discharge swabs from both children. *S. pyogenes* was only cultured from the nasal discharge of patient C. We subjected the 4 strains obtained from patients A, B, and C to pulsed-field gel electrophoresis (PFGE) and checked their T antigen and M proteins. All of the strains possessed the T1 and M1 antigens, as well as the *emm1.0* gene. As for fever genes, the strains were all positive for *speA*, *speB*, and *speF*, but negative

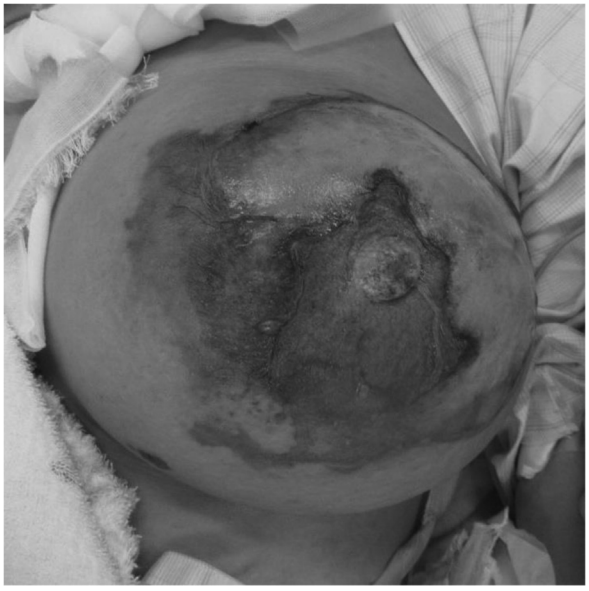

Fig. 1. Left breast of patient A on arrival at the emergency medical center. In this figure, the cranial to caudal direction runs from the top to the bottom of the image. Necrotizing tissue was present around the nipple.

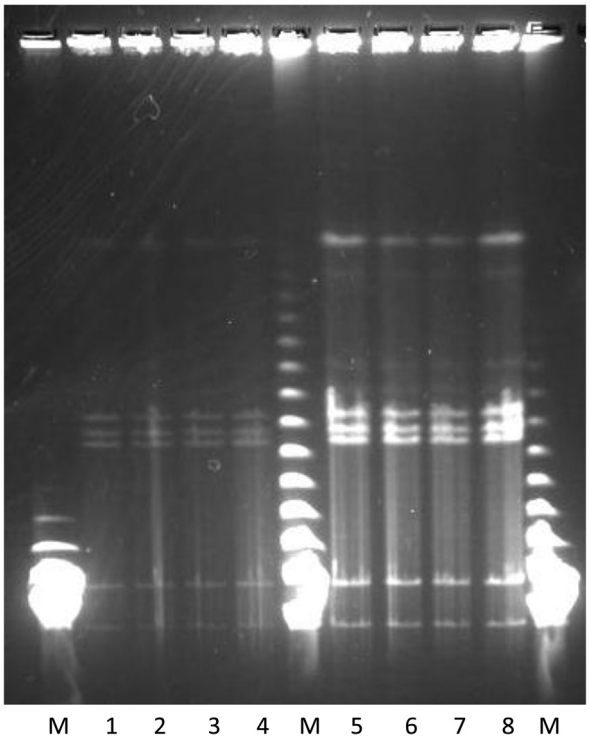

Fig. 2. Pulsed-field gel electrophoresis with the SfiI restriction enzyme . Lanes 1 and 5: Strain derived from the skin of patient A at the previous hospital. Lanes 2 and 6: Strain derived from the skin of patient A at the emergency medical center. Lanes 3 and 7: Strain derived from the pharynx of patient B. Lanes 4 and 8: Strain derived from the nose of patient C. M: lambda ladder marker (BIO-RAD) 1, 2, 3, and 4: after incubation with Todd-Hewitt broth at 30 °C for 3 hours. 5, 6, 7, and 8: after incubation with Todd-Hewitt broth at 30 °C for 24 hours.