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Case report

Pleural empyema and streptococcal toxic shock syndrome due to *Streptococcus pyogenes* in a healthy Spanish traveler in Japan

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ABSTRACT

Group A *Streptococcus* (GAS, *Streptococcus pyogenes*) causes invasive infections including streptococcal toxic shock syndrome (STSS) and local infections. To our knowledge, this is the first report of a case of an invasive GAS infection with pneumonia and pleural empyema (PE) followed by STSS (disseminated intravascular coagulation [DIC] and acute renal insufficiency) in a healthy male adult. He received combined supportive therapies of PE drainage, anti-DIC agent, hemodialysis, and antimicrobials and eventually made a clinical recovery. GAS isolated from PE was found to have *emm1/speA* genes, suggestive of a pathogenic strain. Clinicians should be aware of the possibility of this disease entity (pneumonia, PE, and STSS) in healthy male adults as well as children and adult women.

Introduction

β-hemolytic Lancefield group A *Streptococcus* (GAS, *Streptococcus pyogenes*) is a well-known causative pathogen of upper respiratory tract and cutaneous infections and occasionally leads to streptococcal toxic shock syndrome (STSS). Although GAS is an uncommon pathogen of community-acquired pneumonia (CAP), its clinical course is fulminant and it comprises approximately 25% of CAP cases accompanied by STSS [1]. However, GAS is rarely isolated from pleural empyema (PE). We herein report a severe invasive GAS infection including CAP and PE followed by STSS with acral desquamation at peripheral sites of the hand and foot as typical manifestation of GAS STSS in a healthy Spanish traveler in Japan.

Case report

A 32-year-old, previously healthy man was admitted to our hospital with fever, cough, severe back pain, and oliguria. The patient was a Spanish tourist who worked in England and came to Japan 8 days before visiting our hospital. Four days before hospital admission, he had a cough and fever and developed oliguria 2 days before the visit.

On admission to the emergency room, the patient was afebrile, but he exhibited a blood pressure of 96/57 mmHg, respiratory rate of 40 breaths/min, and oxygen saturation of 90% while breathing under an oxygen mask (15 L/min). Chest respiratory rales were heard over the right middle and lower lung fields. Physical examinations were otherwise normal. His leukocyte cell count and C-reactive protein concentration were 3300/μL and 49.9 mg/dL, respectively. Serum levels of urea nitrogen, creatinine, and fibrinogen degradation products were 69 mg/dL, 5.4 mg/dL, and 40.3 μg/mL, respectively. Chest radiography revealed a poor lucent image of the whole right lung. Contrast-enhanced chest computed tomography showed a consolidation on the right upper and middle lobes including a large amount of right pleural effusion with pleural thickening. Thoracocentesis was performed, and cloudy yellowish pus was aspirated (Fig. 1), which was sent for bacterial culture along with two sets of blood cultures. Gram staining of the PE culture showed numerous Gram-positive cocci with many leukocytes.

Clinical diagnosis of sepsis with disseminated intravascular coagulation (DIC) and acute renal insufficiency due to CAP with PE was made based on these findings. A chest drainage tube was inserted, and intravenous administration of vancomycin and meropenem was initiated with the administration of an anti-DIC agent. Because of his acute renal insufficiency, hemodialysis was performed for 4 days. On hospital day 3, the PE culture revealed growth of GAS, while blood cultures were sterile. This isolate was stored at −80 °C until further evaluation. STTS

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Fig. 1. Aspirated (left side) and drainage (right side) pus from pleural empyema.

was subsequently diagnosed, and vancomycin and meropenem were replaced by combined intravenous administration of penicillin and clindamycin.

The patient eventually made a clinical recovery. The chest tube was removed on hospital day 8 and he was discharged on day 15. During hospitalization (on hospital day 9), acral desquamation erupted at peripheral sites of the hand and foot (Fig. 2**)**, which was considered as typical dermatologic manifestations of STSS.

Microbiological analyses

Phenotypic and genotypic features of the GAS isolate (named strain TA2) were determined and are summarized in Table 1. Phenotypic analyses included colonial morphology on sheep blood agar plate, identification percentage by numerical profile using the Rapid ID 32 Strep API system (SYSMEX bioMérieux Co., Ltd., Tokyo, Japan), and identification score value by matrix-assisted laser desorption/ionization-time of flight mass spectrometry using MALDI BioTyper™ (version 3.1) software (Bruker Daltonics GmbH, Leipzig, Germany). Antimicrobial susceptibility was determined using the broth microdilution method [2]. Genomic analyses compared the similarity (in%) of strain TA2 and type strain JCM 5674^T using 16S rRNA gene sequencing [3], and determined the *emm* type (subtype) with the full-length sequences [4], sequence type (ST) [5], *sic* allele [6], and macrolide/lincosamide (ML) resistance determinants, including *erm*(A), *erm*(B), and *mef*(A) [7]. We also examined the exotoxin gene profile (*speA-speB-speC-ssa-smeZ*) to assess the relationship between the profile and onset of STSS [8]. Briefly, all *emm* typing (sub-typing) was performed as described by the Centers for Disease Control and Prevention (http://www2a.cdc.gov/ncidod/biotech/strepblast.asp); full-length sequencing was performed with the same PCR primers. Multilocus sequence typing (MLST) to determine ST was performed by sequencing seven housekeeping genes (*gki*, *gtr*, *murI*, *mutS*, *recP*, *xpt*, and *yqiL*) according to the GAS pubMLST website (http://pubmlst.org/spyogenes/). The *sic* gene was amplified with primer pair SIC.1/SIC.2; sequencing was also performed with this primer pair. The *sic* allele number was determined and assigned by comparison to the reference allele. The three ML resistance genes and the five exotoxin genes (with 16S rRNA and *speB* as internal controls) were amplified by PCR and confirmed by the corresponding amplicon size on agarose gel electrophoresis. Genomic analyses revealed that the *emm* genotype/full-length sequence, ST, *sic* allele, exotoxin gene profile, and ML resistance determinant were *emm1*/identity similar to strain MGAS5005, ST28, *sic1.02*, *speA-speB-smeZ*, and *mef*(A), respectively.

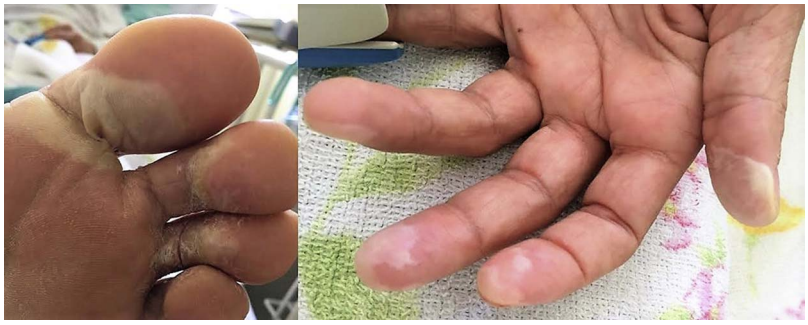

Fig. 2. Desquamation of the palms (right side) and soles (left side) on hospital day 9.