

Laboratory and Epidemiology Communications

Fatal Rapidly Progressing Streptococcal Toxic Shock-Like Syndrome; Case Report

Miki Yokota*, Tadao Hasegawa¹, Michio Ohta¹ and Tatunari Satake

*Nagoya Ekisaikai Hospital, Department of Clinicopathology,
Shonen-cho 4-66, Nakagawa-ku, Nagoya 454-0854 and*

*¹Nagoya University Graduate School of Medicine, Department of Molecular Bacteriology,
Tsurumai-cho 60, Showa-ku, Nagoya 466-8550, Aichi, Japan*

Communicated by Hiroshi Yoshikura

(Accepted May 26, 2000)

The clinical manifestations of group A streptococcal infection include pharyngitis, scarlet fever, necrotizing fasciitis, and toxic shock-like syndrome (Strep TSS) (1-2). Cases of Strep TSS have been reported with increasing frequency in the past several years in Japan as well as in Europe and North America (1, 3-5).

A 58-year-old man visited the emergency room of our hospital at 8:00 p.m. complaining of cough, sore throat, fever of 38°C, and joint pain that had persisted over the previous 3 days. Physical examination showed no remarkable signs and he was prescribed medication for a common cold. The next morning at 6:00 a.m., he returned to the emergency room again complaining of worsening of general fatigue and he was advised to visit the out-patient clinic of the hospital. While waiting for examination, he gradually lost his consciousness and fell into a state of cardiopulmonary arrest. As soon as cardiopulmonary resuscitation was performed, he was admitted to the intensive care unit. On admission, he was in deep coma and laboratory data after resuscitation were as follows; body temperature 38.2°C, heart rate 142/min, blood pressure 100/60 mm Hg, respiration rate 18/min, white blood cell count 2200/ μ l, hemoglobin 15.1 g/dl, platelet 81000/ μ l, total protein 6.8 g/dl, albumin 3.7 g/dl, C reactive protein 9.81 mg/dl, creatinin

kinase 657 IU/l, blood urea nitrogen 38.5 mg/dl, creatinin 2.6 mg/dl, aspartate aminotransferase 57 IU/l, alanine aminotransferase 29 IU/l, lactate dehydrogenase 1245 IU/l. His extremities were cold and no obvious dermal manifestations were seen. Chest X-ray revealed mild infiltration in the right lower lobe. He had had 3 years of medical histories of diabetes mellitus and atrial fibrillation, and tricuspid valve replacement had been performed a year before. His condition deteriorated despite maximum inotropic support and he died two-and-a-half hours after admission. A large amount of blood was sucked from his endotracheal tube. Autopsy was performed immediately after death. Macroscopic examination revealed bilateral pulmonary hemorrhage. All of the cardiac valves including the implanted porcine valve were intact. A small fresh clot was found inside the left pulmonary artery trunk. At the time of dissection, considering the clinical course and the macroscopic findings, pulmonary embolism was a likely diagnosis. However, we were left feeling slightly skeptical of this diagnosis because the size of the clot was too small and no clots were seen inside the other pulmonary artery trunk. Histologically, the pulmonary alveoli were filled with exudate, the walls of which were almost intact except for mild hemorrhage. However, no invasion of inflammatory cells was observed. As the Figure shows, many bacteria-like small round cells were observed along the walls of the alveoli. Later those small cells were found to be gram-positive cocci with

*Corresponding author: Fax +81-52-652-7783, E-mail:YOmikRAV@aol.com

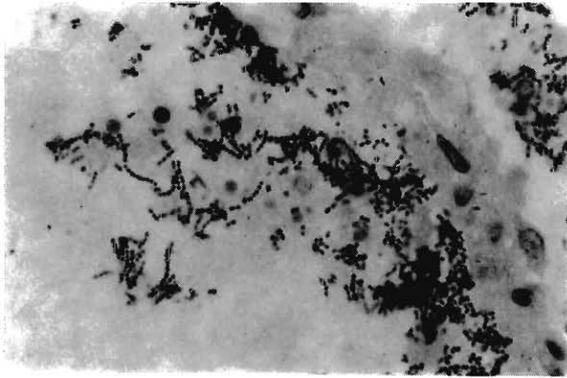


Figure. Histology. Pulmonary alveoli were filled with exudate and large numbers of gram-positive cocci were observed along the walls of alveoli (Gram stain; $\times 1,000$)

gram stain of the tissue. We then performed a culture of the frozen-serum stock of the patient. After 2 days of incubation, *Streptococcus pyogenes* was isolated at a high concentration. This strain produced streptococcal pyrogenic exotoxin (Spe) A, B, F, and M type 1 and T type 1 antigens.

This case appeared to fulfill the Centers for Disease Control and Prevention (CDC) criteria of Strep TSS (6). However, pulmonary hemorrhage without inflammatory reaction as seen in this case appears rare. The commonest respiratory tract complication reported in the literature was pneumonia (3). Influenza-like symptoms were reported in 20% (1). Eighty percent of the patients had clinical signs of soft tissue infection (1). Probably the progress of the disease in this patient was

too rapid to give rise to an inflammatory reaction in the lung. The diagnosis was established only postmortem by bacterial culture of frozen specimens. Some acute deaths with unknown etiology may have been due to infection of this kind.

REFERENCES

1. Stevens, D. L. (1999): The flesh-eating bacterium: what's next? *J. Infect. Dis.*, 179 (Suppl. 2), S366-S374.
2. Hoge, C. W., Schwartz, B., Talkington, D.F., Breiman, R. F., MacNeill, E. M. and Englender, S. J. (1993): The changing epidemiology of invasive group A streptococcal infections and the emergence of streptococcal toxic shock-like syndrome. *JAMA* 269, 384-389.
3. Davies, H. D., McGeer, A., Schwartz, B., Green, K., Cann, D., Simor, A. E., Low, D. E. and the Ontario Group A Streptococcal Study Group (1996): Invasive group A streptococcal infections in Ontario, Canada. *N. Engl. J. Med.*, 335, 547-554.
4. Yoshitaka, S. (1997): Group A streptococcal toxic shock-like syndrome. *Sogo Rinsho*, 5, 1578-1582 (in Japanese).
5. Kiska, D. L., Thiede, B., Caracciolo, J., Jordan, M., Johnson, D., Kaplan, E. L., Gruninger, R. P., Lohr, J. A., Gillingan, P. H. and Denny, F. W., Jr. (1997): Invasive group A streptococcal infections in North Carolina: epidemiology, clinical features, and genetic and serotype analysis of causative organisms. *J. Infect. Dis.*, 176, 992-1000.
6. Working Group on Severe Streptococcal Infections (1993): Defining the group A streptococcal toxic shock syndrome; rationale and consensus definition. *JAMA*, 269, 390-391.