**Group A streptococcal toxic shock syndrome with severe necrotizing fasciitis following hysterectomy – a case report**

Abstract  In the last 10 years an increasing number of cases of group A streptococcal toxic shock syndrome have appeared in various clinical settings. The manifestation of this syndrome includes rapidly progressive multiorgan failure and soft-tissue necrosis.

This report presents a case of streptococcal toxic shock syndrome caused by *Streptococcus pyogenes* with severe necrotizing fasciitis of the abdominal wall following hysterectomy. Aggressive surgical intervention with debridement of all necrotic tissue necessitated resection of the complete abdominal wall (skin, subcutaneous tissue, muscle and peritoneum). The abdominal wall defect was covered with free myocutaneous flaps and split-skin grafts. Optimal treatment, including adequate antibiotic therapy and radical surgical intervention, is an indispensable prerequisite of successful outcome.

**Key words**  *Streptococcus pyogenes* · Septic shock · Toxic shock syndrome · Fasciitis · Surgical flaps · Hysterectomy

**Introduction**

*Streptococcus pyogenes* (group A streptococcus) are ubiquitous organisms, causing a wide spectrum of diseases, ranging from mild oropharyngeal and skin infections, scarlet fever, pneumonia or meningitis, to severe, life-threatening illnesses such as necrotizing fasciitis, myositis, septicemia, puerperal sepsis and toxic shock syndrome associated with multiorgan failure.

Invasive and often fatal infections due to group A streptococci have been increasing in frequency throughout the United States, Canada, Australia and Europe since the late 1980s [1, 2]. Streptococcal toxic shock syndrome (STSS) was first described by Cone et al. [3] in 1987. A large number of additional cases presenting signs of toxicity with early onset of shock and multiple organ failure have been reported in the last 10 years [1, 4].

The following report describes a case of group A streptococcal toxic shock syndrome with severe necrotizing fasciitis after hysterectomy. The successful outcome was made possible by early, ultra-aggressive surgical debridement with resection of the abdominal wall, accompanied by antibiotic therapy.
Fig. 1 Muscle swab with gram-positive cocci (Gram stain)

Case report

A 46-year-old woman underwent an abdominal hysterectomy following a 3-year history of uterine myoma (139 mm × 124 mm × 101 mm). Surgery was completed without difficulties and no prophylactic antibiotics were given. The initial postoperative course was uneventful. After 2 h in the recovery room the patient was transferred to her ward. In the evening her body temperature rose (39°C), early next morning (day 1 after hysterectomy) she appeared to be in a state of circulatory shock, hypotensive with a blood pressure of 80/35 mm Hg and tachycardic with a heart rate of 110/min.

No evidence of bleeding was found during the immediate abdominal examination in the operating room. There was ascites, which indicated the onset of peritonitis. Laboratory analysis showed the following values: leukocytes $1.9 \times 10^9/\ell$, platelets $94 \times 10^9/\ell$, thromboplastin time 76 s, AT III 21%, serum creatinine 201 µmol/l, lactate 10.8 mmol/l, base excess $-18$ mmol/l and pH 7.06 (arterial blood). Due to low central venous pressure and hypovolemia, the patient received volume substitution (1500 ml colloids, 4500 ml crystalloids). During the course of surgery, a small strip of the M. rectus abdominis revealed macroscopically visible signs of edematous swelling. This part of the rectus muscle near the skin incision in the lower abdomen was resected, a specimen of the abdominal muscle was taken for Gram stain, which showed abundant gram-positive cocci in chains (Fig. 1). Antibiotic therapy was initiated with intravenous penicillin G, imipenem and amoxicillin with clavulanic acid. After admission to the Intensive Care Unit (ICU) postoperatively, the patient was treated with vasopressors and remained on mechanical ventilation. She was placed on penicillin G, imipenem and clindamycin. In addition, she received hydrocortisone (0.18 mg/kg per h) as part of a protocol-guided treatment of septic shock [5, 6].

Within a few hours the clinical status deteriorated dramatically. Circulation had to be supported with noradrenalin and dopexamine. Laboratory evaluations showed not only renal impairment and coagulopathy, but also increasing signs of rhabdomyolysis (serum creatine kinase activity 263 U/l, myoglobin 1706 ng/ml). In addition to this, a rapidly spreading, foul-smelling abdominal wall erythema with palpable crepitation was observed, extending from the skin incision to the upper abdomen and the right and left flanks. The erythema darkened, changing from red to purple to blue. Five hours after the first examination she was returned urgently to the operating theater with a diagnosis of necrotizing fasciitis with rhabdomyonecrosis. By this time the whole abdominal wall (skin, subcutaneous tissue, fascia, muscle and peritoneum) had become infected by gas-forming microbial agents and was therefore completely resected, from the suprapubic area to the costal arch and flanks (Fig. 2). In the histological analysis samples of skin, subcutaneous tissue, fascia and muscle showed gram-positive cocci and partly expanded necroses with only a minor, acute inflammatory reaction. Muscle swabs yielded Staphylococcus pyogenes (M-type 1) sensitive to the antibiotic regimen. All blood cultures were negative. Extremely high levels of serum creatinkinase activity (554 U/l), myoglobin (2649 ng/ml) and lactate (11.2 mmol/l) were present after surgery. Disseminated intravascular coagulation (DIC) resulted in massive bleeding after the extensive necrosectomy. The patient developed a severe purpura as a cutaneous manifestation of DIC with thrombocytopenia. Packed red blood cells (22 units), platelets...