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Fulminant Necrotizing Fasciitis and Toxic Shock Syndrome Caused by *Streptococcus agalactiae*

Streptococcus agalactiae'nın Neden Olduğu Fulminan Nekrotizan Fasiit ve Toksik Şok Sendromu

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SUMMARY

Necrotizing fasciitis is a rare and life-threatening soft tissue infection that spreads rapidly and involves the skin, subcutaneous tissue, fascia, and muscle layer. The treatment is possible by initiating appropriate antibiotherapy for the clinically suspected cause and by performing surgical intervention quickly and aggressively. However, it should be known that necrotizing fasciitis is a disease that is difficult to manage despite all interventions, effective treatment protocols, and patient care. This article presents the case of a 60-year-old patient with diabetes mellitus who died of toxic shock syndrome with fulminant necrotizing fasciitis caused by Streptococcus agalactiae.

Key Words: *Necrotizing fasciitis; Toxic shock syndrome; Streptococcus agalactiae*

ÖZET

***Streptococcus agalactiae*'nın Neden Olduğu Fulminan Nekrotizan Fasiit ve Toksik Şok Sendromu**

Nekrotizan fasiit deri, deri altı doku, fasiya ve kas tabakasını tutan, hızla yayılım göstererek yaşamı tehdit eden nadir bir yumuşak doku infeksiyonudur. Tedavi, klinik olarak şüphelenilen etkene yönelik uygun antibiyoterapi başlanması ve cerrahi müdahalenin hızlı ve agresif olarak yapılmasıyla mümkün olabilmektedir. Ancak bilinmelidir ki, nekrotizan fasiit tüm müdahalelere, etkili tedavi protokolleri ve hasta bakımına rağmen yönetimi zor olan bir hastalıktır. Bu yazıda Streptococcus agalactiae'nın sebep olduğu fulminan nekrotizan fasiit ile birlikte toksik şok sendromu sonucu ölen 60 yaşında diabetes mellitus hastası bir olguyu sunduk.

Anahtar Kelimeler: *Nekrotizan fasiit; Toksik şok sendromu; Streptococcus agalactiae*

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INTRODUCTION

Necrotizing fasciitis (NF) is a rare, progressive, and lethal bacterial infection characterized by fascia and subcutaneous necrosis. Rapidly spread necrosis in tissues often leads to systemic sepsis, toxic shock syndrome, and multi-organ failure[1-5]. Most NF infections are polymicrobial. Monomicrobial infections, most commonly seen with Streptococcus pyogenes, constitute 1/3 of the cases. Despite early diagnosis, adequate debridement, and appropriate antibiotic treatment of NF, mortality is still high (30-70%), and virulence of causative pathogen is the main determinant of the progression of the disease[6]. This article presents a rare case of fulminant necrotizing fasciitis with toxic shock syndrome caused by *Streptococcus agalactiae*, which has been reported in the literature.

CASE REPORT

A 60-year-old male patient was admitted to our hospital from an external center for further examination and treatment with a pre-diagnosis of septic shock and multi-organ failure. The patient had been admitted to hospital two days prior due to nail dropping and had tetanus vaccination. One day later, in the morning, he was admitted to another hospital due to bad smell and purge discharge at the same foot, where he was dressed, prescribed oral antibiotics and discharged. In the evening of the same day, he was taken to a private hospital due to bruising on the foot, blurred consciousness and a blood sugar level of 500 mg/dL (N: 70-110). After examination and initial treatment, the patient was transferred to our hospital. He had been diagnosed with type 2 diabetes mellitus for 20 years and received insulin treatment. Physical examination revealed that the patient's condition was bad. He had no consciousness and no spontaneous respiration, so he was intubated. Tension arterial was 70/50 mmHg, pulse was 112 min, fever was 38°C, SpO_2 was 89%, and finger blood sugar was 380 mg/dL. Light reflex -/-, pupillary fix dilated. There was necrotic and ischemic appearance on 1/3 of the middle and distal left cruris (Figure 1A) and there was ecchymosis and common subcutaneous emphysema spread from the left cruris to proximal fossa, thigh, scrotum, pelvis, abdomen front wall, both flank regions, thorax front wall, both shoulder, neck, and face regions.

Respiratory support was provided to the patient with mechanical ventilator. The patient underwent a subclavian catheter. Blood samples of the patient were taken for hemogram and biochemical analysis. Blood and wound cultures were taken. Fluid resuscitation was initiated for the patient. Ceftriaxone 1 g IV and gentamycin 120 mg IV were administered to the patient as empirical antibiotics. Dopamine and adrenaline infusions were administered as inotropic support to keep the systolic blood pressure at 100 mmHg. Laboratory values of the patient were as follows: White blood cell (WBC) count: 3000/mm^3 (N: 4500-11.000), hemoglobin: 14.8 g/dL (N: 14.1-18.1), platelet: 100.000/mm^3 (N: 142.000-424.000), glucose: 431 mg/dL (N: 70-110), blood urea nitrogen (BUN): 54 mg/dL (N: 6-20), creatinine: 2.48 mg/dL (N: 0.67-1.3), sodium: 129 mmol/L (N: 135-145), potassium: 6.3 mmol/L (N: 3.5-5.1), chlorine: 94 mmol/L (N: 98-107), calcium: 7.7 mg/dL (N: 8-10.2), ALT: 149 U/L (N: 16-43), AST: 622 U/L (N: 0-40), and C-reactive protein (CRP): 32.5 mg/dL (N: 0-0.30). In unenhanced thoracic computed tomography (CT) examination, common air densities and subcutaneous emphysema under the skin and between the muscle planes were observed at both upper extremities and the right half of the thorax wall and common air densities were observed at the truncus pulmonalis and right ventricle level (Figure 1B). In unenhanced abdomen computed tomography (CT) examination, widespread air densities were observed, extending to the level of the pelvis and more prominent on the right side in both halves of the abdominal wall and linear air densities were observed between mesenteric fat planes and mesenteric vascular structures (Figure 1C). In pelvis and left lower extremity CT examination, common air densities and subcutaneous emphysema were observed throughout the subcutaneous tissue and muscle planes in all segments of the left lower extremity. Subcutaneous tissue and muscle planes had common air densities in the gluteal region and both thighs which are seen in the figure

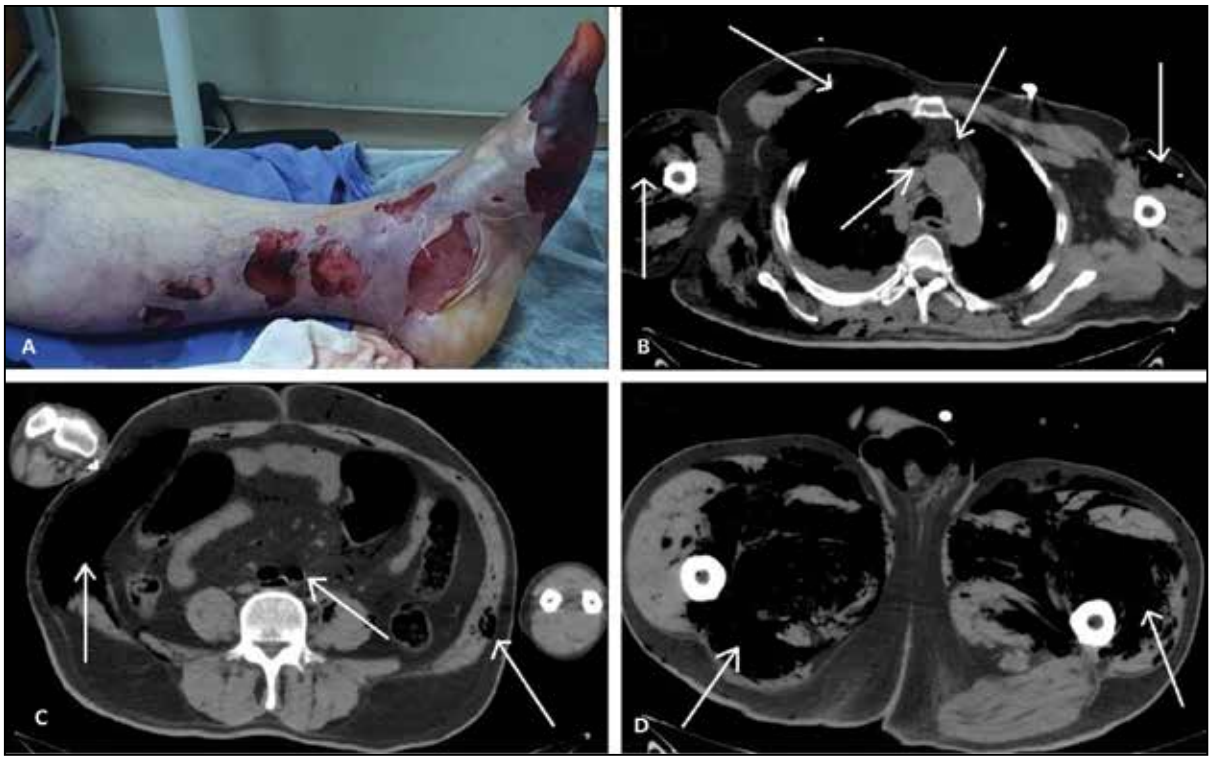

Figure 1. **A)** Necrotic and ischemic appearance in middle 1/3 and distal left cruris. **B)** In unenhanced thoracic computed tomography (CT) examination, common air densities and subcutaneous emphysema under the skin and between the muscle planes were observed at both upper extremities and the right half of the thorax wall and common air densities observed at truncus pulmonalis and right ventricle level (white arrows). **C)** In unenhanced abdomen computed tomography (CT) examination, widespread air densities were observed, extending to the level of the pelvis and more prominent on the right side in both halves of the abdominal wall and linear air densities were observed between mesenteric fat planes and mesenteric vascular structures (white arrows). **D)** In pelvis and left lower extremity computed tomography (CT) examination, common air densities and subcutaneous emphysema were observed throughout the subcutaneous tissue and muscle planes in all segments of the left lower extremity (white arrows).

(Figure 1D). Surgery was planned for the patient. Anesthesia, general surgery, and orthopedic consultation were required. However, the patient died due to resistant septic shock and multi-organ failure after approximately 1 hour in the emergency intensive care unit. *S. agalactiae* was isolated from blood cultures. *S. agalactiae* was sensitive to clindamycin and penicillin G. Wound culture was not appropriate.

DISCUSSION

NF is a serious bacterial infectious disease with high mortality and morbidity, which can be seen in all anatomical regions of the body, causing thrombosis and skin necrosis in subcutaneous vascular structures[2]. NF is present in all ages and both genders, but more frequently seen in males aged 50-60 years[2,3,6]. It can be seen that factors that harm skin integrity such as incisions, penetrant injuries, ulcerous skin lesions, burns, and abrasions are major causes of the disease, but also in rare cases, incarcerated hernia, insect bites and blunt traumas can also cause the disease. 20-30% of patients do not have skin lesions or skin associated injuries[7]. Although the disease can be seen in the entire body, the most common regions are the lower extremities, anogenital region, and abdominal wall.

The impression of the immune system increases the risk of NF. Diabetes mellitus is the most common co-morbidity in addition to other factors that facilitate NF development such as the use

of alcohol, smoking addiction, obesity, advanced age, corticosteroids, and parenteral drug use[8,9]. The cases in which there is no underlying disease constitute 50% of all cases[10]. NF showed early onset erysipelas and a cellulitis-like lesion showing pain, tenderness, and local temperature increase. The bullous lesions that develop in the advanced stage go up to skin necrosis depending on ischemia[11]. Clinically, NF can be confronted with all systemic signs and symptoms of high fever, weakness, general impairment, hypotension, and multi-organ failure. Diagnosis of NF should be based on clinical findings and anamnesis[12].

Although it is not necessary for diagnosis, computed tomography, which produces better results than normal radiography methods, is the most useful radiological imaging method that is able to reveal the etiology of the necrotizing process and subcutaneous and fascial edema, and show gas and fluid accumulation in advanced stages of the disease[13]. In this case, air densities and common subcutaneous emphysema were found in computerized tomography, which supported the diagnosis.

The cause of the disease in NF may be either a single microorganism or polymicrobial. The agent is mostly the virulent form of the group A streptococcus. The ability of *S. agalactiae* to cause invasive infections in adults is well documented[14,15]. Nevertheless, necrotizing fasciitis caused by *S. agalactiae*, which is probably the most severe and fatal form of invasive streptococcal infection, has rarely been described in the literature.

Toxic shock syndrome is a rapidly deteriorating clinical situation including shock, multi-organ failure, and destructive soft tissue infection. While it is well-known that group A streptococci induce this systemic problem, group B streptococci have only recently been described as a cause.

It is not clear why some patients with invasive *S. agalactiae* infection develop streptococcal septic shock-like syndrome and others develop necrotizing fasciitis or both. Although McGeer et al.[16] have recently shown that the risk factors for developing group A streptococcal toxic shock-like syndrome include patient age and toxin A genotype, an M-protein serotype and the presence of diabetes mellitus favor the development of necrotizing fasciitis. However, such associations are not clear in *S. agalactiae* infections.

In conclusion, early diagnosis and rapid progression of large surgical debridement are of vital importance for the effective treatment of necrotizing fasciitis, an infectious disease with high mortality and morbidity. This treatment should be combined with intensive antibiotherapy and other preventions in cases with systemic clinical findings.

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