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Brief Reports

GROUP A *STREPTOCOCCAL* NECROTIZING FASCIITIS IN THE EMERGENCY DEPARTMENT

Jiun-Nong Lin, MD,*†‡ Lin-Li Chang, PHD,§ Chung-Hsu Lai, MD,*‡‖ Hsi-Hsun Lin, MD,‡ and Yen-Hsu Chen, MD, PHD*¶#

*Graduate Institute of Medicine, College of Medicine, Kaohsiung Medical University, Kaohsiung, Taiwan, †Department of Emergency and Critical Care Medicine, E-Da Hospital, I-Shou University, Kaohsiung, Taiwan, ‡Division of Infectious Diseases, Department of Internal Medicine, E-Da Hospital, I-Shou University, Kaohsiung, Taiwan, §Faculty of Medicine, Department of Microbiology, Kaohsiung Medical University, Kaohsiung, Taiwan, ‖Division of Infection Control Laboratory, E-Da Hospital, I-Shou University, Kaohsiung, Taiwan, ¶School of Medicine, College of Medicine, Kaohsiung Medical University, Kaohsiung, Taiwan, and #Division of Infectious Diseases, Department of Internal Medicine, Kaohsiung Medical University Hospital, Kaohsiung, Taiwan

Reprint Address: Yen-Hsu Chen, MD, PHD, Division of Infectious Diseases, Department of Internal Medicine, Kaohsiung Medical University Hospital, Kaohsiung, No. 100, Tzyou 1st Road, Kaohsiung 807, Taiwan

□ **Abstract—Background: Group A *Streptococcal* (GAS) necrotizing fasciitis is a critical emergency. Patients with necrotizing fasciitis principally present to emergency departments (EDs), but most studies are focused on hospitalized patients. Objective: An ED patient-based retrospective study was conducted to investigate the clinical characteristics, associated factors, and outcomes of GAS necrotizing fasciitis in the ED. Methods: Patients visiting the ED from January 2005 through December 2011 with the diagnosis of GAS necrotizing fasciitis were enrolled. All patients with the diagnosis of noninvasive skin and soft-tissue infections caused by GAS were included as the control group. Results: During the study period, 75 patients with GAS necrotizing fasciitis were identified. Males accounted for 84% of patients. The most prevalent underlying disease was diabetes mellitus (45.3%). Bullae were recognized in 37.3% of patients. One third of cases were complicated by bacteremia. Polymicrobial infections were found in 30.7% of patients. Overall mortality rate for GAS necrotizing fasciitis was 16%. Patients aged >60 years with diabetes mellitus, liver cirrhosis, and gout were considerably more likely to have GAS necrotizing fasciitis than noninvasive infections. Patients presenting with bacteremia, shock, duration of symptoms/signs <5 days, low white blood cell count, low platelet count, and prolonged prothrombin time were associated with increased mortality. Surgery is a significantly negative factor for mortality of patients with GAS necrotizing fasciitis (odds ratio = 0.16; 95% confidence interval 0.002–0.16; $p < 0.001$). Conclusions: A better understanding of the associated factors and initiation of adequate treatments will allow for improved survival after GAS necrotizing fasciitis.** © 2013 Elsevier Inc.

□ **Keywords—necrotizing fasciitis; group A *Streptococcus*; *Streptococcus pyogenes*; emergency department; associated factors**

INTRODUCTION

Necrotizing fasciitis, commonly known as flesh-eating disease, is a rapidly progressive and life-threatening skin and soft-tissue infection (SSTI). This severe infection is characterized by extensive necrosis of dermis, subcutaneous tissue, superficial fascia, deep fascia or muscle (1,2). Several micro-organisms contribute to the pathogenesis of necrotizing fasciitis, including aerobic, anaerobic, and mixed flora (1,2). Group A *Streptococcus* (GAS; *Streptococcus pyogenes*) is one of the important contributors responsible for necrotizing fasciitis (3,4). During the late 1980s, outbreaks of necrotizing fasciitis

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caused by GAS were reported. The Centers for Disease Control and Prevention estimates that approximately 500 to 1000 new cases of GAS necrotizing fasciitis occur each year in the United States (US), accounting for 6%−7% of all invasive GAS infections (5).

The mortality rate of necrotizing fasciitis is still high compared with other infections, despite advancements in medical treatment (6−9). The successful treatment of necrotizing fasciitis relies primarily on early diagnosis and initiation of appropriate management (1,3,6−10). For patients presenting with symptoms of SSTIs in the emergency department (ED), differentiating necrotizing fasciitis from noninvasive infections is of paramount importance. Unfortunately, the clinical symptoms and signs of necrotizing fasciitis, including severe pain, bullae, ecchymosis, skin sloughing, gaseous gangrene, and systemic toxicity, usually come to light in the late phase of the disease (10,11). It is therefore a great challenge for physicians to provide a correct diagnosis of necrotizing fasciitis in the ED.

Despite the critical nature of this catastrophic disease, the majority of the literature concerning GAS necrotizing fasciitis focused on hospitalized patients (6−8,12). However, most patients with necrotizing fasciitis present to a primary care provider or ED (13). Unfortunately, few ED patient-based studies discuss the manifestations of GAS necrotizing fasciitis. Therefore, we conducted an ED patient-based retrospective study to investigate the clinical manifestations, associated factors, treatments, laboratory findings, and outcomes of necrotizing fasciitis caused by GAS.

METHODS

Study Setting and Population

This study was conducted at an 1100-bed university-affiliated hospital that has approximately 60,000 ED visits per year and serves >2 million people in southern Taiwan. This study was approved by the Institutional Review Board.

Study Protocol

Patients visiting the ED from January 2005 through December 2011 with the diagnosis of necrotizing fasciitis caused by GAS were identified from the computer database. For comparison, all patients during the same period with the diagnosis of noninvasive SSTIs caused by GAS were included as the control group. The medical records of included patients were reviewed independently by two physicians who were experienced in the chart review. If any discrepancy was found, the medical records were inspected again by these two physicians together. Demographic information, underlying illnesses, clinical conditions, laboratory findings, treatments, and outcomes were collected from the medical records for analysis.

Case Definition

Patients were diagnosed as having necrotizing fasciitis if fascial edema and necrosis were recognized at surgery or necrosis of superficial fascia and polymorphonuclear leukocyte infiltration and edema of the reticular dermis, subcutaneous fat, and superficial fascia were detected in histopathology (6). If surgery was not performed, necrotizing fasciitis was defined as thickening and enhancement of the superficial or deep fascial layers, with or without air and fluid collections within the deep fascia in computed tomography or magnetic resonance imaging

Table 1. Demographic Characteristics, Underlying Illnesses, Clinical Conditions, Laboratory Findings, Treatments, and Outcomes of 75 Patients with Group A *Streptococcal* Necrotizing Fasciitis in the Emergency Department

Characteristics	
Sex, male, n (%)	63 (84)
Age (years), median (mean ± SD)	58 (57.6 ± 18.8)
Underlying illnesses, n (%)	
Diabetes mellitus	34 (45.3)
Liver cirrhosis	10 (13.3)
Gout	19 (25.3)
Alcoholism	15 (20)
Malignancy	3 (4)
Immunosuppressant	3 (4)
HIV	1 (1.3)
Sites of infections, n (%)	
Upper extremity	12 (16)
Lower extremity	56 (74.7)
Both upper and lower extremity	3 (4)
Trunk	4 (53)
Durations of illnesses (days), mean ± SD	4.4 ± 2.9
Clinical manifestations, n (%)	
Bullae formation	28 (37.3)
Shock	38 (50.7)
Bacteremia	25 (33.3)
Polymicrobial infection	23 (30.7)
Laboratory data, mean ± SD	
White blood cell, n = 75 ($\times 10^9$ cells/L)	18.45 ± 9.21
Hemoglobin, n = 75 (g/dL)	12.2 ± 1.9
Platelet, n = 75 ($\times 10^9$ cells/L)	165.8 ± 83.2
Serum sodium, n = 73 (mmol/L)	133 ± 4.9
Serum creatinine, n = 73 (mg/dL)	2.15 ± 1.17
Serum glucose, n = 66 (mg/dL)	194.5 ± 140.8
C-reactive protein, n = 55 (mg/L)	222.5 ± 120
INR of prothrombin time, n = 51	1.26 ± 0.37
Treatments, n (%)	
Inappropriate antibiotics	7 (9.3)
No surgery	7 (9.3)
Surgery	68 (90.7)
Fasciotomy/debridement	58 (77.3)
Amputation	10 (13.3)
Mortality, n (%)	12 (16)

HIV = human immunodeficiency virus; INR = international normalized ratio; SD = standard deviation.