J Infect Chemother 22 (2016) 335–338

Contents lists available at ScienceDirect

Journal of Infection and Chemotherapy

journal homepage: http://www.elsevier.com/locate/jic

Case report

Necrotizing soft tissue infection caused by *Serratia marcescens*: A case report and literature review

Hideharu Hagiya [a, *], Masahiro Ojima [b], Takeshi Yoshida [b], Takahiro Matsui [c], Eiichi Morii [c], Kazuaki Sato [c], Shinichiro Tahara [c], Hisao Yoshida [a], Kazunori Tomono [a]

[a] Division of Infection Control and Prevention, Osaka University Hospital, Japan
[b] Department of Traumatology and Acute Critical Medicine, Osaka University Hospital, Japan
[c] Department of Pathology, Osaka University Hospital, Japan

ARTICLE INFO

Article history:
Received 14 October 2015
Received in revised form
11 November 2015
Accepted 24 November 2015
Available online 14 January 2016

Keywords:
Necrotizing soft tissue infection
Necrotizing fasciitis
Serratia marcescens
Sepsis

ABSTRACT

A 64-year-old man with advanced liver cirrhosis was transferred to an emergency center due to septic shock and markedly inflamed left leg. Under a clinical diagnosis of necrotizing soft tissue infection (NSTI), the patient undertook intensive therapy but died 25 h after arrival. The pathogenic organism, *Serratia marcescens*, was later isolated from blood and soft tissue cultures. NSTI is very rarely associated with *S. marcescens*. A literature review showed that only 16 such cases, including our case, have been reported to date. Our case is the first evidence of an *S. marcescens* NSTI in a patient with liver cirrhosis. *S. marcescens* NSTI has an extremely high mortality rate; total mortality and mortality in cases involving the extremities were 75% (12 of 16 cases) and 83.3% (10 of 12 cases), respectively. Physicians need to be aware that *S. marcescens* can induce fatal infections in community patients.

1. Introduction

Serratia marcescens is a gram-negative aerobic bacillus belonging to the family Enterobacteriaceae. The pathogen ubiquitously exists in hospitals and causes blood stream, respiratory, and urinary tract infections [1,2]. Soft tissue is rarely infected with *S. marcescens* [3], and the involvement of necrotizing soft tissue infection (NSTI) that yields a fulminant course and high mortality is extremely rare. To our knowledge, only 15 such cases have been described in the English literature to date [4–17]. We herein describe a fatal case of NSTI caused by *S. marcescens* along with a literature review.

2. Case report

A 64-year-old man with a 10-year history of liver cirrhosis due to a hepatitis C virus infection was transferred to our emergency center because of a swollen left leg accompanied by shock. A week prior to the transfer, he had burned his left calf while welding. A blister had formed, but he did not get medical attention and left the burn injury untreated. One day before admission, the left leg started to swell and the patient's general condition rapidly deteriorated.

On arrival, the vital signs were as follows: blood pressure, 80/50 mmHg; heart rate, 94 bpm; oxygen saturation, 93% (oxygen reservoir, 15 L/min); respiratory rate, 31/min; and body temperature, 35.7 °C. The abdomen was remarkably distended, and the left leg was extremely swollen with accompanying blisters and discolored skin (Fig. 1A). Laboratory examination revealed leukopenia, elevated serum C-reactive protein, liver and renal dysfunction, hyperammonemia, disseminated intravascular coagulopathy, significant lactic acidosis and positivity for the anti-hepatitis C virus antibody (Table 1). The laboratory risk indication for necrotizing fasciitis score was 4 [normal range; less than 5]. Systemic computed tomography demonstrated a contracted liver with multiple nodules and massive ascites. Edematous changes were observed inside the inner aspect of the thigh, but an abnormal air pattern was not apparent. An explorative incision was immediately performed, and tissue degeneration was confirmed (Fig. 1B). Gram staining of the tissue showed the presence of gram-negative rods, and a combination of benzylpenicillin (penicillin G), meropenem, and

* Corresponding author. Division of Infection Control and Prevention, Osaka University Hospital, 2-15 Yamadaoka, Suita, Osaka 565-0871, Japan. Tel.: +81 6 6879 5093; fax: +81 6 6879 5094.
E-mail address: highgear@hp-infect.med.osaka-u.ac.jp (H. Hagiya).

http://dx.doi.org/10.1016/j.jiac.2015.11.013
1341-321X/

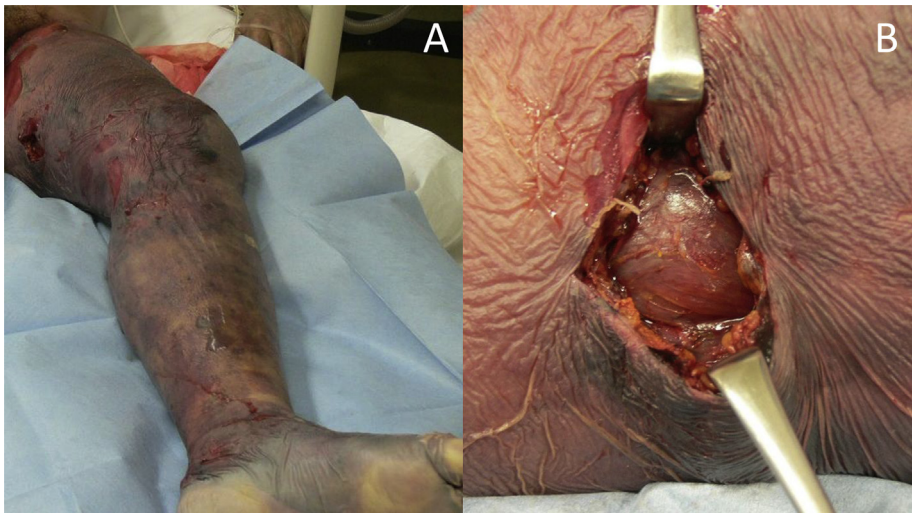

Fig. 1. A. A photograph showing the patient's necrotized left leg. B. A photograph showing ischemic tissue exposed by an explorative incision at the medial thigh.

clindamycin was administered to the patient. Under a diagnosis of septic shock due to NSTI of the left leg, leg amputation was discussed with the patient's family who refused it.

The patient underwent endotracheal intubation and was admitted to the emergency center. Despite massive fluid therapy with catecholamine support, lactic acidosis progressed and continuous renal replacement therapy was initiated. The skin color of the left leg further deteriorated and a lot of effusion exuded from the ruptured blisters. Finally, the patient died 25 h after admission in spite of intensive care.

Later, the blood culture became positive and *S. marcescens* was isolated. The gram-negative rod detected in the left leg tissues and blisters was also identified as *S. marcescens*. The pathogen was resistant to sulbactam/ampicillin, and cefazolin, but susceptible to piperacillin, ceftriaxone, ceftazidime, cefepime, meropenem, gentamicin, minocycline, levofloxacin, and sulfamethoxazole/trimethoprim. *Streptococcus pneumoniae* was also detected from the blood culture, but not from the other tissues. An autopsy showed advanced liver cirrhosis accompanying hepatocellular carcinoma, and the presence of *S. marcescens* in the necrotic tissues of the left leg.

3. Discussion

S. marcescens rarely causes NSTI and the clinical characteristics of the infection are still unclear. To get a better understanding, we conducted a literature review of previous reports of *S. marcescens*-induced NSTI from MEDLINE. The most recent review of the disease was performed by Rehman et al. in 2012 [15]. Thereafter, five additional cases have been described [12–14,16,17]. A summary of the previous cases including our case is shown in Table 2.

Although most cases of *S. marcescens* infections are nosocomial in origin, 13 cases (81.3%) were community-onset infections. Ten (76.9%) of 13 adult cases had underlying conditions such as diabetes mellitus [5,9,10], chronic kidney disease [6,10,15,16], malignancy [9,17], immunosuppressive drug use [6,13,15], chronic heart failure [10,16], nephrotic syndrome [6,13], and advanced age [16], although cases without such predisposing conditions existed [4,7,12]. We consider that these chronic underlying diseases, potentially causing immunocompromised states, were mainly responsible for the onset of community-onset *S. marcescens*-induced NSTI. Community-acquired infections associated with *S. marcescens* are increasingly reported [18,19]. In this era of advanced medicine, there are many people in community who live well along with underlying chronic diseases. Thus, we should bear in mind that, as well as *S. marcescens*, other Gram-negative pathogens can cause fulminant infections to those immunocompromised people in community.

Our patient had underlying advanced liver cirrhosis and malignancy, which was not observed in the other patients. Chronic liver dysfunction is a representative underlying disease for *Vibrio vulnificus* infections, and frequently causes fatal necrotizing infections as seen in our case [20]. The reasons for increased risk of fulminant *V. vulnificus* infections in patients with liver disease are as follows: (i) reduced clearance by Kupffer cells in hepatic sinusoids, (ii) direct bacterial invasion through the portosystemic shunt leading to systemic infections secondary to bacterial translocation, and (iii) bacterial overgrowth due to elevated serum iron concentration [21]. Although the relevance of liver cirrhosis and onset of *S. marcescens* infection has not been uncovered, our case presents the first evidence for chronic liver dysfunction as an underlying condition in *S. marcescens* NSTI.

Of the three pediatric cases, two occurred in the cervical regions of previously healthy children [8,11]. Both had suffered from

Table 1
Laboratory examinations on admission.

Complete blood count		**Biochemical examinations**	
WBC	1030/mm^3	AST	294 IU/L
Nt	55.3%	ALT	84 IU/L
Lym	37.9%	ALP	250 IU/L
Mono	3.9%	γGTP	111 IU/L
Eo	2.9%	T-bil	4.8 mg/dL
Baso	0%	D-bil	3.5 mg/dL
Hb	10.7 g/dL	BUN	27 mg/dL
Platelet	6×10^4/mm^3	Cre	2.03 mg/dL
Coagulopathy		TP	5.2 g/dL
PT-INR	2.83	Alb	2.6 g/dL
APTT	48 s	LDH	454 IU/L
FDP	19.4 μg/mL	CK	366 mg/dL
Fibrinogen	217 mg/dL	Na	139 mEq/L
Arterial blood gas		K	4.1 mEq/L
pH	7.161	Cl	105 mEq/L
PaCO2	41.2 mmHg	CRP	4.14 mg/dL
PaO2	82.2 mmHg	NH3	109 μg/dL
BE	−13.6 mEq/L	HBs-Ab	Negative
Lactate	9.9 mmol/L	HCV-Ab	Positive