Blistering erysipelas: not a rare entity

Chong F Y, Thirumoorthy T

ABSTRACT

Introduction: Soft tissue infections are common, but erysipelas, especially its blistering feature, is an under-recognised entity. There have been few reports of blistering erysipelas. We aim to describe the clinical characteristics, management and the risk factors for erysipelas in 20 patients admitted in a tertiary hospital in Singapore.

Methods: A chart review of all cases of erysipelas, diagnosed by experienced dermatologists and admitted to the Singapore General Hospital during the period January 2006 to August 2006, was conducted.

Results: There were 20 patients (11 male, nine female) with an average age of 62.2 (range 31–86) years. The most commonly-involved site was the leg (75 percent), followed by the arm (15 percent) and face (ten percent). The clinical characteristics were well dermarcated (50 percent), erythema (100 percent) and oedema (85 percent), and bullae and vesicles formation (80 percent). Most presented with no pain (40 percent) and minimal signs of systemic toxicity. There was no positive blood culture, but the swab on the blistering erysipelas yielded positive cultures in 67 percent. The most common predisposing factor was disruption in the skin barrier (65 percent), followed by venous insufficiency (20 percent) and lymphoedema (25 percent). All patients received empirical antibiotics, most commonly penicillin and cloxacillin (65 percent), for an average duration of 20.65 (10–41) days, and with local care, there was complete resolution.

Conclusion: In our experience, erysipelas is a clinically distinct entity and commonly presents with bullae or vesicles. It has favourable prognosis, and rarely develops any complication with timely and appropriate therapy.

Keywords: bullae, blistering erysipelas, cellulitis, erysipelas, skin infection

Singapore Med J 2008;49(10):809-813

Dermatology Unit, Singapore General Hospital, Outram Road, Singapore 169608

Chong F Y, MBBS, MMed, MRCP
Medical Officer

Thirumoorthy T, MBBS, FRCP, FAMS
Senior Consultant

Correspondence to:
Dr Chong Fei Yoong
Tel: (614) 1303 8877
Email: feiyon@yahoo.com

INTRODUCTION

Soft tissues infections are common and have a spectrum of severity depending on the depth of infection. Aetiological diagnosis is usually difficult on presentation. Clinical assessment is therefore crucial in determining the depth and severity of the infection, possible outcomes and the type of management. Erysipelas, which in Greek means red skin, is a superficial bacterial infection that affects the upper dermis and superficial lymphatics, and is characterised by fiery red and tender plaque with well-demarcated edges, commonly caused by *Streptococcus pyogenes*[1-3](Fig.1). Erysipelas was previously described mainly on the face. However, to date, it is mostly localised to the legs.[1,4] It is usually associated with lymphangitis and lymphadenopathy with mild systemic manifestation.[1,2] Bullae or vesicles may complicate about 5% of erysipelas.[1,2,5] A case control study in seven hospital centres in France found that lymphoedema, venous insufficiency, being overweight and disruption of the skin barrier such as ulcer, wound and toe web intertrigo, were independent risk factors in erysipelas.[6]

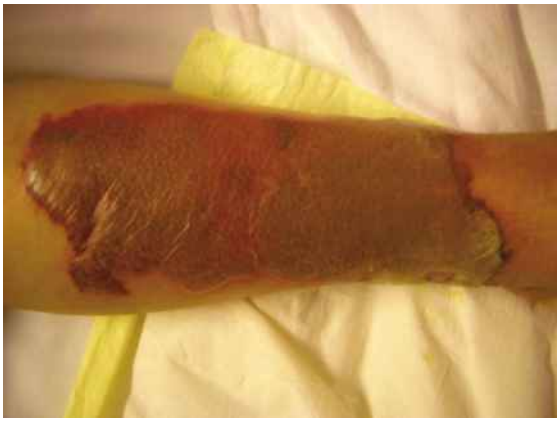

Fig. 1 Photograph shows erysipelas, seen as fiery red, oedematous skin with well-demarcated edges and bullae formation.

While cellulitis is an infection affecting the lower dermis and subcutaneous soft tissue, necrotising fasciitis is a deep-seated infection of the subcutaneous tissue with rapidly-progressive destruction of fat and fascia.[1,2] Clinically, cellulitis, which is characterised by ill-defined erythema and oedema, is warm and painful.[1-3] Necrotising fasciitis is initially characterised by erythema and oedema, then followed by bullae formation which rapidly become haemorrhagic with skin necrosis. Since necrotising fasciitis carries a mortality of 50%–70% and may mandate prompt surgical intervention, early recognition of this infection is crucial.[1,2] The Infectious Diseases Society of America (IDSA) guidelines in 2005 outlined some

Table I. Patients' and clinical characteristics on admission.

Characteristics	No. (%) patients
Age > 60 years	12 (60)
BMI > 25 kg/m^2	7 (58)*
Gender	
Male	11 (55)
Female	9 (45)
Fever (T > 38°C)	7 (35)
SBP < 90mmHg	1 (5)
HR >100 bpm	8 (40)
No pain (pain score 0)	8 (40)
Site of infection	
Lower limb	15 (75)
Arm	3 (15)
Face	2 (10)
Clinical signs	
Erythema	20 (100)
Swelling	17 (85)
Sharp demarcation	10 (50)
Bullae/vesicles	16 (80)
Skin necrosis	0
Associated lymphangitis and/or local adenopathy	6 (30)

* based on n = 12 (2 patients did not have their heights measured)

Table II. List of associated comorbidities.

Underlying diseases	No. (%) patients
None	2 (10)
Diabetes mellitus	6 (30)
Malignancies	4 (20)
Cardiovascular diseases	4 (20)
Cerebrovascular diseases	2 (10)
Immunosuppression	0
Others	16 (80)

Others include asthma, hypertension, hyperlipidaemia, obstructive sleep apnoea, peptic ulcer disease, hepatitis B infection, sinusitis.

Table III. Investigations done in patients with erysipelas.

Investigations	No.(%) patients
Imaging modalities	
Radiographs	5 (75)
Ultrasonography	9 (45)
Magnetic resonance imaging	4 (20)
Microbiological	
Positive blood cultures	0
Positive cutaneous swabs	6 (67)*

* n = 9

clues to diagnosis of potentially severe deep infection. These includes pain disproportionate to the physical findings, violaceous bullae, cutaneous haemorrhage, skin sloughing, skin anaesthesia, rapid progression and gas in the tissue. The presence of signs of systemic toxicity, such as temperature (T) > 38°C or < 36°C, heart rate (HR) > 100 beats per minute (bpm) and hypotension (systolic blood pressure (SBP) < 90 mmHg or 20 mmHg below baseline) may also be indicative of a severe soft tissue infection.[1,2]

However, it is interesting to note that erysipelas, a superficial dermis infection, may also share some of the features above, particularly bullae formation, as in blistering erysipelas. Therefore, erysipelas may be misdiagnosed as necrotising fasciitis with an unfavourable impact on patients, especially by unnecessary investigations and aggressive treatment. In this retrospective review of all cases of erysipelas referred to Dermatology Unit in Singapore General Hospital (SGH) over eight months, we aimed to conduct a descriptive study of the clinical characteristics of erysipelas and the risk factors for its development. We also evaluated the investigations conducted, microbiological characteristics and treatment of erysipelas.

METHODS

Medical records of patients who were admitted and referred to Dermatology Unit in SGH, during the period January 1, 2006 to August 31, 2006, with the diagnosis of erysipelas, were reviewed. We conducted an analysis on the patients' characteristics (age, gender, ethnic group and body mass index [BMI]), signs of systemic toxicity (fever, T > 38°C, SBP < 90 mmHg, HR > 100 bpm, pain score), site of infection and clinical signs (erythema, swelling, sharp demarcation of erythema, presence of bullae or vesicles, skin necrosis and associated lymphangitis and lymphadenopathy).

The diagnosis of erysipelas was made or confirmed by two dermatologists with a composite experience of over 50 years and one visiting specialist in dermatology with at least two years' experience (post-training). The diagnosis was based on skin criteria (erythematous, tender plaque with well-demarcated edges), and evidence of infection such as fever or raised white cell counts. T, HR, blood pressure on presentation to the hospital, and the highest pain score in the first three days of hospitalisation were recorded. The pain score was obtained using a numerical rating scale from zero to ten, where zero is no pain and ten the worst possible pain. Records of clinical signs were based on the description by the primary physician and consulting dermatologist.

Predisposing factors to the development of erysipelas, such as disruption of skin barriers (ulcer, toe web intertrigo, tinea pedis, underlying dermatosis, skin trauma), venous insufficiency and presence of lymphoedema, were evaluated. Other data, such as duration of hospitalisation and fever, antibiotics treatment and duration, presence of comorbidities, use of imaging studies (radiographs, ultrasonography, magnetic resonance imaging) and

Table IV. Positive wound culture results.

Organisms	Sensitivities
Staphylococcus aureus	Penicillin, ampicillin, cloxacillin, cephalothin, clindamycin.
Pseudomonas spp.	Ampicillin, ceftriaxone, cefepime, gentamicin, ciprofloxacin, aztreonam, cotrimoxazole, piperacillin/tazobactam.
Group A streptococcus	Penicillin, ampicillin, clindamycin, erythromycin.
MRSA	Vancomycin.
Pseudomonas aeruginosa	Piperacillin/tazobactam, cefepime, aztreonam.
Enterococcus spp.	Vancomycin

microbiological investigations (blood cultures, wound cultures), were recorded and analysed. Finally, we also noted the complications of the disease and outcome of the treatment.

RESULTS

20 patients were diagnosed to have erysipelas and admitted to SGH during the study period. 11 were male (55%) and nine were female (45%). The average age was 62.2 (range 31–86) years. Among them, there were 12 Chinese, seven Malays and one Indian. The most common site of infection was the lower limb (15; 75%), followed by the arm (3; 15%), and then the face (2; 10%). 14 patients were weighed, with a median weight of 82 (range 40–150) kg. Only 12 out of the 14 patients had their heights measured, and thus, the average BMI was 30.9 (range 16.7–51.6) kg/m^2 (Table I).

On admission, most patients were haemodynamically stable, with an average blood pressure of 135.15/76.35 mmHg. One patient had a blood pressure of 80/90 mmHg and was clinically dehydrated. The blood pressure was stabilised with administration of fluid. The average HR on admission was 92.3 (range 58–126) bpm. The highest pain scores in the first three days of hospitalisation were documented, with a median pain score of 2.5 (range 0–8). Eight patients (40%) had no complaint of pain. Well-demarcated (50%) erythema (100%) and oedema (85%) were the unique clinical characteristics of erysipelas. It was interesting to note that 80% of our study population had bullae or vesicle formation (Table I). Diabetes mellitus was the most common comorbidity associated with erysipelas (Table II).

Radiographs were the most common investigation done (75%) to look for bony erosion or gas in the soft tissue. Ultrasonography was done on the affected lower limb to exclude concomitant deep venous thrombosis, but none was remarkable. Nine out of the 16 patients with bullae or vesicles had swabs done for bacterial culture. The swab was not done on intact skin. All patients had blood cultures done upon admission (Table III). Culture of punch biopsy specimens and needle aspirations of the inflamed skin were reported to yield an organism in 20%–30% of cases and varying from < 5% to 40%, respectively.[1,2] However, in our study, the culture of wound swab on blistering erysipelas yielded positive results in 67%. The microorganisms isolated were streptococcus (one), *Staphylococcus aureus* (one), methicillin-resistant *Staphylococcus aureus* (MRSA) (two), pseudomonas (two) and enterococcus (one) (Table IV).

The majority of the patients (65%) received intravenous crystalline penicillin and cloxacillin upon admission, in accordance to the hospital's antibiotics guidelines for cellulitis. However, other antibiotics, such as augmentin, clindamycin, levofloxacin, ciprofloxacin and ceftazidime, were used because of either allergy to penicillin or cloxacillin, or the treating physicians' choice. The average duration of intravenous and oral antibiotic treatment was 20.65 (10–41) days. The duration of intravenous antibiotics ranged from four to 36 (median 7.5) days, and was decided by the treating physicians. In most cases, intravenous antibiotics were ceased when there was evidence of clinical improvement. Only one patient did not respond to the initial empirical intravenous antibiotics. All patients with blistering erysipelas received local care, such as puncturing of bullae, potassium permanganate or saline compress, compression bandaging and elevation of the affected limb.

The average duration of fever was 1.5 days. 57% had no fever on admission. The average duration of hospitalisation was 10.2 (range 4–36) days. With respect to complications, one patient had myositis as evidenced by raised serum creatinine kinase, one had abscess formation, requiring incision and drainage, and one developed ulcer as a result of rupture of bullae. All our study patients were seen in the outpatient clinic after discharge and complete resolution of the infection was documented.

Our data showed that the most common predisposing factor to the development of erysipelas was disruption in the skin barrier (intertrigo, tinea pedis, eczema). Underlying dermatoses seen in our study included stasis eczema, asteatotic eczema, atopic eczema and psoriasis (Table V). These predisposing conditions were present

Table V. List of predisposing factors.

Factors	No. (%) patients
None	3 (15)
Disruption of skin barrier	
Ulcer	0
Intertrigo	2 (10)
Tinea pedis	6 (30)
Underlying dermatosis	9 (45)
Skin trauma	5 (25)
Venous insufficiency	4 (20)
Lymphoedema	5 (25)

in four out of seven patients with documented recurrent infection at the same site. Of the four patients, three had stasis eczema of the lower limb and tinea pedis, which were not treated adequately, and one had psoarisis of the arm. These patients were noted to be obese with BMI ranging from 35.7 to 51.6. No statistical method was conducted as this was more of a descriptive, rather than an analytical study.

DISCUSSION

Soft tissue infection is common and varies in severity. It is therefore often managed by doctors in various specialties, such as general physician, infectious disease physician, general practitioner, general surgeon, orthopaedic surgeon and dermatologist. In addition, many cases are managed as outpatients. This may be the reason why epidemiology of soft tissue infection is difficult to document and study.[4] This study described our experience of erysipelas in SGH. Most of our patients were elderly, with 60% aged more than 60 years (average age 62.2 years). Both genders were affected equally, with a slight male predominance (male: female ratio 11:9).

The most common site of infection was the lower limb, followed by the arms, and then the face. We noted that erysipelas was not a well-recognised disease among hospital medical practitioners, with a majority of our study patients diagnosed as cellulitis (17; 85%) upon admission and the diagnosis was then revised to erysipelas by the consultant dermatologists. This study demonstrated the unique clinical signs of erysipelas (well-demarcated erythema and oedema) of which a diagnosis can be made confidently. Also unknown to many physicians, bullae and vesicles formation are not uncommon and occurred in 80% of our study patients.

It is often difficult to clinically differentiate between a life-threatening disease, necrotising fasciitis with bullae formation, and the more benign blistering erysipelas. This was evident when four of our study patients had magnetic resonance imaging done with the aim of excluding necrotising fasciitis. These four patients presented with erythema and oedematous skin which blistered. However, none of them nor did the rest of the patients with blisters demonstrate the signs, such as skin tenderness or necrosis, rapid progression or gas in the tissues, suggestive of deep infection as described in the 2005 IDSA guidelines for soft tissues infection.[1] Furthermore, pain was not a significant symptom among our patients with 40% denying any pain and the average of the highest pain score was only 2.9. With regard to signs of systemic toxicity, the majority of our patients showed only mild manifestation. Few patients displayed temperature of $>38°C$ (7; 35%), hypotension with SBP < 90 mmHg (1; 5%) and tachycardia of HR $>$ 100 bpm (8; 40%) on admission. There was no recorded episode of hypotension during the hospital stay in all patients.

The absence of radiographical abnormalities in our patients showed that radiographs may be of limited value in erysipelas. Unlike other forms of localised infection where microbiological diagnosis is important, cultures in erysipelas are usually negative and may not affect its treatment and favourable prognosis.[1,3,4] Since it is a localised infection and bacteraemia is rare,[1,3,4] it is of no surprise that all our patients had negative blood cultures. Routine blood culture collection may not be fruitful except in patients with diabetes mellitus, malignancy, neutropenia and immunodeficiency.[1] Our study showed that the yield of the isolating pathogen from an open skin lesion in blistering erysipelas is significant and may guide the choice and duration of the antibiotic regime. Though MRSA was isolated in two cases, they were considered as colonisers, rather than pathogens, as the skin condition improved without specific treatment for MRSA.

Conditions that lead to disruption in the skin barrier predisposed patients to erysipelas and probably recurrent infection, and thus should be aggressively treated.[1,3,4,6] Our study showed that obesity may play an independent risk factor for recurrent infection. A larger prospective case control study may help to elucidate this risk factor. Most of our patients received intravenous crystalline penicillin and cloxacillin, in accordance to our hospital's antibiotic guidelines, which is based on the SGH antibiogram in December 1998.[7] However, the 2005 IDSA guidelines recommended empiric treatment with first generation cephalosporin (intravenous cefazolin). Alternatively, clindamycin or new fluoroquinolone (levofloxacin) may be used in patients who cannot tolerate beta lactams. Intravenous vancomycin should be used if MRSA infection is suspected.[1,3] The SGH guidelines recommended treatment to extend three days more after acute inflammation has subsided,[7] while IDSA suggested five days of therapy in uncomplicated infection.[1] In our study, the duration of antibiotics therapy varied greatly, according to the judgment and experience of the

physicians.

Our experience suggested that local care of blistering erysipelas, such as puncturing of blisters and applying potassium permanganate or saline compress, may dry up the weeping area, thus quicken improvement.[3,5] We realised that de-roofing the blister may risk ulcer formation and delay healing. Compression bandaging and elevation of the affected area may promote gravity drainage of the oedema and inflammatory substances.[1,3,5] Being a retrospective study, it had a number of limitations. The patients' clinical presentation and progress may not be complete or accurately documented in the medical notes. The management of the study patients was not standardised, and was in accordance with the experience and knowledge of the primary physician or surgeon. Our study population was a selected patient sample and it was a hospital-based study. Therefore, it may be subjected to referral bias and may not represent a general population with erysipelas. The study of other forms of soft tissues infection, such as cellulitis and necrotising fasciitis, may be useful to better define the entity and natural history of erysipelas. Being a descriptive study, it lacked statistical analysis to better define the predisposing factors to the development of erysipelas and recurrent infections.

Despite the limitations, this study very well demonstrated the clinical features of erysipelas, highlighting especially the feature of blistering, which is common and easily confused with necrotising fasciitis by the non-dermatologist. Besides, the local management of skin in erysipelas may be of value in preventing the development of complication of skin ulceration and aiding in the resolution of the infection. Finally, this study reassured us, as treating physicians, that with empirical antibiotics, blistering erysipelas has a favourable prognosis with complete resolution in most cases and rarely develops complications.

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