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Case Report

Erysipelas lymphedema: Two case reports and literature review

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ABSTRACT

Erysipelas, a variant of cellulitis, is a superficial dermal bacterial infection. This superficial cutaneous infection may cause lymphangitis with resultant lymphedema in poorly treated cases. Reports of erysipelas preceding lymphedema are rare, rather many have reported the occurrence of erysipelas in patients with subclinical or overt lymphedema. Two case reports of erysipelas preceding lymphedema after appropriate parenteral antibiotics therapy and limb elevation are here presented. Following diagnosis, admission, and commencement of therapy, the second patient (Case 2) discontinued hospital treatment halfway for native treatment and returned after about 3 weeks. All patients recovered successfully but later presented with lymphedema after 8 weeks and 6 weeks, respectively. Both patients made a sustained functional recovery of their limbs with conservative management including elastic compression stocking and limb elevation. Erysipelas infection preceding lymphedema may develop when it occurs, prompt and appropriate treatment modalities for erysipelas infection may forestall the development of lymphedema.

Keywords: Erysipelas, Cellulitis, Lymphedema

INTRODUCTION

Erysipelas, previously called *Saint Anthony's fire,*[1,2] was coined from the Greek word "*Eruthros*" and the Latin word "*Pella*" meaning red and skin, respectively. Erysipelas is a superficial dermal infection usually caused by *Streptococcal* organisms, and the beta-hemolytic *Staphylococcus aureus*. Other documented microbes include *Enterobacteriaceae* occurring either singly or in combination with the *Streptococcus* organism. The microbes invade the dermis through a breach in the integrity of the skin, initiating an inflammatory reaction. The cutaneous manifestation precedes a prodromal phase which is characterized by malaise, chills, and high-grade fever. This inflammatory process leads to the destruction and disruption of vessels including the superficial lymphatic system.

Erysipelas also occurs in patients with an underlying comorbidity. Damstra *et al.*[3] hypothesized that in cases of erysipelas without an overt lymphedema or an obvious precipitating agent; then, a subclinical pre-existing congenital or acquired disturbance of the lymphatic system had existed.

Case 1

Mrs. AFI, a 50-year-old school teacher, presented to the accident and emergency department (A&E) with complaints of fever, malaise, general body weakness, and abdominal discomfort of 4 days duration.

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The onset of symptoms was sudden. The abdominal discomfort followed a maize meal. The meal was freshly prepared and not refrigerated. There were no colic, no diarrhea, and no nausea and vomiting. The patient had decreased appetite. She was a known hypertensive and no diabetic.

Examination revealed no significant findings except obesity (BMI – 39.5). A random blood sugar (RBS) test indicated glucose level of 8.1 mmo1/l. Blood film for malaria parasite was positive for malaria falciparum parasites. The patient was admitted into A&E ward for observation. Intravenous fluids, analgesics, and a course of antimalarial were given. The patient showed some improvement and was discharged home the next day.

Forty-eight hours later, the patient's condition became worse and was brought back to hospital. Now, she had additional complaint of painful and swollen right lower limb of 24 h duration.

At this point, the patient volunteered a history of sustaining minor injury on that right leg about 3 days before the first presentation.

A consult was sent to the burns, plastic, and reconstructive (BPU) surgeons for review and management. A clinical review by the BPU team revealed an obese patient with mild toxemia (temperature 38.2°C), tachypnea, and pallor but no cyanosis and jaundice.

Regional examination showed tender and diffusely swollen right leg from the foot to upper third of the leg with multiple bullae and associated tender right inguinal lymphadenopathies. A diagnosis of bullous erysipelas was made. The patient was admitted into the ward. The following investigations; full blood count (FBC), RBS, fluid aspirate from the bullae for microscopy, culture, and sensitivity (M/C/S), and urine analysis were requested. The patient was commenced on intravenous ceftriaxone, parenteral nonsteroidal anti-inflammatory drugs, with bed rest and elevation of the affected limb.

A review after 48 h of admission showed no remarkable improvement. Rather, the patient was more toxic, temperature now 39.4°C, with tachycardia and tachypnea. Worsening erysipelas infection noted. A wound biopsy was done and intravenous Augmentin 1.2 g 12 hourly, tablets low-dose vasopressin, multivitamins, and iron supplement were added to her prescription. By the 3rd day, the wound aspirate for M/C/S showed no microbial organism on Gram stain, yielded no bacterial growth after 48 h of incubation and the patient showed no clinical improvement.

By the 5th day, a tissue biopsy sent for M/C/S reported Gram-negative rods with moderate pus cells on microscopy. The culture yielded heavy growth of *Pseudomonas aeruginosa,* which was sensitive to levofloxacin and Gaxin but resistant to Clavamox, Graxone, and Zinacef.

The FBS level was now 5.5 mmol/L. The bullae were noted to be on the increase while some were discharging seropurulent exudate. The intravenous Augmentin was discontinued and replaced with intravenous Rocephin 1 g daily, intravenous gentamicin 80 mg 8 hourly, and intramuscular Pentazocine 30 mg 8 hourly.

The patient was reviewed 24 h later and she demonstrated remarkable improvement in clinical parameters. The temperature was on a downward trend.

All the bullae were deroofed, necrotic skin edges debrided by the bedside. The limb was irrigated with copious amount of 0.9% normal saline and raw surfaces of punctate wounds were dressed with 5% povidone-iodine soaked gauzes and crepe bandage applied from the knuckle of the toes up to the thigh. The limb was rested and elevated on multiple pillows.

On the 8th day, 3 days after the change of intravenous antibiotics, the patient demonstrated significant improvement. The patient was now eager to go home and requested for discharge. There was an evidence of reepithelialization at the bullae sites and the patient was discharged home the next week.

The patient was on a 2 weekly follow-up visits to the outpatient clinic. After the third visit, there was a complete reepithelialization with hyperpigmentation. However, the patient came down with another complaint. This was diffuse non painful swelling of the leg. An impression of lymphedema was made. This was successfully managed conservatively with rest, elevation, and use of elastic compression stocking.

Case 2

Mrs. GAR, a 52-year-old trader, presented at the A&E department with a complaint of painful swelling and blistering of the right leg of 3 days duration. The patient's problem started about a week before presentation with fever, malaise, neck pain, and abdominal pain. The patient went to a pharmacist who treated her for malaria and typhoid fever. About 4 days later, the patient noticed her condition was getting worse and that her right leg was swollen which made it difficult for to walk freely. She then went back to the same pharmacist who told her that she was burnt by evil fire. She was given some prescription including tablets and gentian violet to apply on the affected part of the limb. The patient adopted the treatment for days but to no avail. The patient then resolved to go to the A & E department of our hospital. The fever was high grade, persistent, associated with nausea, no vomiting. Examination revealed a moderately toxic patient with temperature as high as 39.5°C, respiratory rate 38 cycles/min, and pulse rate 108 b/min. The blood pressure was 140/95 mmHg.

Locoregional examination showed a diffusely inflamed right lower limb from the foot up to a middle third of the thigh, with bullae; some ruptured and others intact. The inguinal lymph nodes were enlarged and tender.

A diagnosis of fulminant erysipelas was made and the patient admitted into the female surgical ward.

Blood samples for FBC, blood culture, serum urea, electrolyte, and creatinine (E/U/Cr), RBS, and screening of retroviral disease were collected. The patient was immediately resuscitated with intravenous fluids and commenced on intravenous third-generation cephalosporin (Rocephin) and metronidazole. Analgesics, both suppositories and parenteral, were administered. Bedsides, deroofing of the bullae and proper irrigation with 0.9% normal saline was done. The wound was dressed with honey-soaked gauzes and application of crepe bandage. Following the result of the serum (E/u/Cr) levels, gentamycin was added to her treatment regime. After 1 week of treatment, there was a significant resolution of symptoms and signs except the blood pressure. The patient disappeared and reappeared 12 days after in our outpatient department pleading for readmission that she thought that it was "evil spell." Treatment was continued and the patient recovered before proper discharge for follow-up at the outpatient clinic.

The patient also faulted outpatient follow-up visits. She reappeared after 6 weeks with lymphedema on the affected right lower limb. This was managed conservatively with good outcome.

DISCUSSION

Erysipelas is a bacterial infection involving the superficial dermis. Some authorities consider it a variant of cellulitis, which is an infection involving the deep dermis and subcutaneous tissue. Erysipelas also known as "*ignis sacer,*" holy fire, and Saint Anthony's fire[1,3,4] is an acute bacterial infection caused predominantly by beta-hemolytic *Streptococcus* (Groups A, C, and G) and *Staphylococcus aureus* organisms. Other microbes such as *Enterobacteriaceae* and *Pseudomonas* species as in our index patient are implicated. These occur either alone or in combination with *Streptococcus* organisms. The microbes penetrate through a compromised integrity of the skin, to provoke an inflammatory reaction.

Erysipelas affects all age brackets. However, the adults are mostly affected with a peak age bracket between 60 and 80 years. It may affect the face, head, and neck, the lower extremities in 70–80% of cases. Upper extremity infection occurs in 2–10% of cases while the face is affected in 6–20% of cases. The trunk and genitalia are less affected accounting for <2% of cases.[2,5] The infection starts with prodromal symptoms such as malaise, chills, aches, and high-grade fever before the cutaneous manifestation. The prodromal symptoms usually occur within 48–72 h before cutaneous manifestation. The cutaneous manifestations include burning sensation, diffuse swelling with tenderness, and bullae formation among others.

Erysipelas is more superficial than cellulitis. The distinguishing clinical features found in erysipelas being that the inflammatory swelling of erysipelas infection is more raised and demarcated at its border and may manifest with bullae formation. Other non-specific skin changes are figurate erythema, skin pigmentation, and complications of the infection, namely, lymphangitis, bacteremia, septic arthritis, glomerulonephritis, necrotizing fasciitis, and lymphedema.

Erysipelas may be a secondary complication of chronic lymphedema in 20–30% of cases.[4,6,7] The high protein content found in the interstitial fluid of lymphedematous area of the body serves as an ideal culture medium for the growth of invading bacteria.

There is a paucity of report on lymphedema arising as a complication of erysipelas. Damstra *et al*.[3] hypothesized that in such cases of erysipelas resulting in lymphedema that a subclinical pre-existing congenital or acquired physiological derangement of the lymphatic system was present before the onset of erysipelas.

Noteworthy is the opinion that persistent inflammatory reaction that precedes the erysipelas is a probable cause of the lymphatic destruction which then leads to the formation of lymphedema.[7-9] This hypothesis is laudable to uphold. However, it is also known that the lymphatic system is a high-volume transport system that clears protein and lipid, from the interstitial spaces to the vasculature through a differential pressure system. More so, the lymphatic vessels in the superficial and intermediate dermis lack valves and drain into the valved deep dermal and subdermal plexuses. It is the authors' opinion, therefore, that it is the post-inflammatory damage to the lymphatics of the superficial and intermediate dermis that accounts for the development of lymphedema preceding erysipelas infection. The specialist opinion explains why erysipelas infection predisposes to the development of lymphedema than cellulitis, which involves the deep dermal and subcutaneous tissues where the deep lymphatic system may be spared.

CONCLUSION

Erysipelas infection may complicate secondary lymphedema. However, clinical observation has shown that lymphedema may also develop as a complication of fulminant erysipelas infection.

The authors, therefore, recommend an aggressive management of fulminant erysipelas, especially those involving the lower extremities. The secondary and tertiary prevention strategies for lymphedema which include early management,

prophylaxis for bacterial and fungal infections, and limb exercises should be carried out. The above would curb or arrest the morbidities that may follow severe erysipelas.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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