CASE REPORT

Non-pseudomonal ecthyma gangrenosum caused by methicillin-resistant *Staphylococcus aureus* (MRSA) in a chronic alcoholic patient

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SUMMARY

We report for the first time a case of non-pseudomonal ecthyma gangrenosum (EG) caused by methicillin-resistant *Staphylococcus aureus* in a chronic alcoholic without an associated immunocompromised state or disease. EG is an infection of the skin, commonly associated with *Pseudomonas aeruginosa* bacteraemia, though other organisms are also known to be causally related. It usually occurs in patients who are terminally ill and the immunocompromised. In our case, chronic alcoholism with suppression of innate immunity may have led to the development of the condition.

BACKGROUND

Ecthyma gangrenosum (EG) was initially described in association with *Pseudomonas* septicaemia 1897.^{1,2} Subsequently other organisms have been reported to cause EG.³ EG (non-pseudomonal) in a non-immunocompromised patient is rare and a possible association with chronic alcohol intake has not been reported before.³ Hence, we report a case of EG caused by methicillin-resistant *Staphylococcus aureus* (MSRA) in a chronic alcoholic with no history of an associated immunocompromised state or disease.

CASE PRESENTATION

A 47-year-old man was admitted with complaints of multiple ulcers over both the thighs for the past 1 week. He was treated with antibiotics from a local general practitioner. He was a chronic alcoholic for the past 33 years and had last consumed alcohol 2 days earlier. He consumed an average of 200 g of alcohol per day for the past 2 years. He was found on the road side under the influence of alcohol by the family members 1 week earlier, when they noticed multiple small fluid filled, blood stained blisters over both his thighs. The blisters broke down leaving behind larger expanding ulcers which were covered by dark slough. He complained of pain and burning at the site. There was no history of fever. Detailed history did not suggest the presence of a septic foci or an immunocompromised state.

Clinical examination

On admission, he was febrile with a temperature of 38.5°C, pulse rate was 110 per minute and the blood pressure was 130/70 mm Hg. Oxygenation

measured by a finger pulse oximeter was adequate. Lungs was clear, Glasgow coma score was 15 and other systemic examination was normal. Five ulcers of 2×2 to 4×5 cm were present over the medial aspect of both the thighs and the right heel. The individual ulcers were tender to touch, had well-defined margins and sloping edges; the floor was covered with thick black eschar and the base was mobile. The ulcers were surrounded by an erythematous halo (figure 1). The regional lymph nodes were enlarged. Examination of other systems were normal.

INVESTIGATIONS

Investigations showed no evidence of diabetes, HIV, syphilis, Hepatitis B or malignancy. X-ray chest PA was normal. Liver function tests showed serum gamma glutamyl transaminases (γGT) of 100 U/L and albumin of 2 g/100 mL. Rest of the liver function tests including alanine transaminase, aspartate transaminase, bilirubin, alkaline phosphatase and prothrombin time was within normal limits. Haemoglobin was 12 g per 100 mL, total leucocyte counts were 12 000 with polymorphonuclear cells 80%. Blood culture sent on admission was negative for microorganisms. Pus and tissue culture from the ulcer revealed MRSA.

Histopathology

Biopsy from the ulcer edge revealed gross epidermal necrosis with hypoplastic stratum corneum, been separated from the epidermis by a cleft (figure 2). There was irregular elongation of stratum spinosum

Figure 1 Clinical photograph showing multiple ulcers over the left thigh covered with black eschar. Erythematous halo seen in the surrounding skin.

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Figure 2 Photomicrograph at scanning view shows gross epidermal necrosis. Presence of a cleft in the subcorneal level.

with intact basal layer. The dermis showed sheets of inflammatory cells comprising neutrophils, lymphocytes and histiocytes (figure 3). The capillaries appear thrombosed, extravasation of red blood cells (RBC) was present (figure 4). The findings were compatible with a necrotising gangrenous infection. Tissue gram stain was not performed.

TREATMENT

Based on the clinical findings, laboratory data and histopathology, a diagnosis of EG was made. Patient was started on intravenous ceftriaxone empirically and later changed to imipenam and amikacin based on the culture report. Surgical debridement was done as often as required.

Figure 3 Photomicrograph at low power (100x) shows intact basal layer with sheets of inflammatory cells in the dermis. Thrombosis of superficial vessels seen.

Figure 4 Photomicrograph at high power (400x) shows the infiltrate in dermis comprising neutrophils, lymphocytes and histiocytes with massive extravasation of red blood cells.

OUTCOME AND FOLLOW-UP

Ulcers healed completely at the end of 4 weeks (figure 5).

DISCUSSION

Apart from *Pseudomonas*, EG can be caused by many bacteria, fungi and viruses including *Escherichia coli*, *Staphylococcus aureus*, *Streptococcus pyogenes*, *Citrobacter freundii*, *Neisseria*

Figure 5 Healed wound 6 weeks old.

gonorrhoea, *Yersinia pestis*, *Morganella morganii*, *Aeromonas hydrophila*, *Klebsiella pneumoniae*, *Candida albicans* and herpes simplex.^{4–6} EG has been rarely reported in healthy individuals with no history of an associated immunocompromised state.^{7,8} Indeed, in a recent review of all published cases of EG (167 cases) over a 45-year period from 1976 to 2014, only 24 cases of non-pseudomonal EG were reported in either healthy (7 cases) or immunocompetent (17 cases) individuals.³ Diseases and conditions commonly associated with EG are HIV, malignancy, burns, organ transplant and a history of immunosuppressive medications.³

EG can occur anywhere in the body, especially in moist areas like the groin, buttocks and perineum and in the head, face neck region.³ The characteristic lesion of EG and histopathology has been described well in literature.^{3,4,8,9} Briefly, EG starts as an erythematous macule that rapidly develops into a gangrenous lesion with central necrosis and a surrounding halo after going through a haemorrhagic bullous stage. Histological features show necrosis and gangrene. They include separation of the stratum corneum, infiltration of inflammatory cells, vascular necrosis caused by bacterial infiltration into the media and adventitia, thrombosed vessels and extravasation of RBCs.^{8,9} The diagnosis is clinical. Gram stain, blood culture, urine culture, skin biopsy and tissue smear/culture are all helpful in confirming the diagnosis of EG. Moreover, investigations to diagnose immune suppressive states like HIV infection are important.

Antibiotics like third-generation cephalosporin, penicillin, aminoglycosides, piperacillin, aztreonam or fluoroquinolones are empirically started until culture reports. Surgical debridement of the necrotic ulcers is done. Nutritional and underlying immune suppressive states have to be managed simultaneously. Conditions of acute skin necrosis that may mimic EG include necrosis induced by drugs and medications like warfarin, cocaine and vasoactive drugs, necrotising fasciitis, skin lesions in people with diabetes, disseminated intravascular coagulation and familial Mediterranean fever.^{3,10}

It has been hypothesised that EG can originate in one of the two ways: either through the blood stream following generalised bacteraemia or by local inoculation.¹¹ In our patient, the infection must have travelled via the blood stream because of multiple lesions on both sides of the body. Bacteraemia is reported in 50% of cases of EG.³ In our case, the absence of growth of bacteria in blood may be due to antibiotics that he had received prior to the blood culture test. Association of the disease with chronic alcoholism has not been reported. Chronic alcoholism suppress the innate immune system predisposing to increased susceptibility to cutaneous and systemic infections.¹² This probably explains the cause of EG in our patient who had no other immune-depressed condition. He had raised serum γ GT and low albumin, which are common in patients who are chronic alcoholics.¹² The

Learning points

- ▶ Ecthyma gangrenosum (EG) can occur in people with chronic alcoholism.
- ▶ EG can occur in patients who do not manifest an overt immunocompromised state.
- ▶ People with chronic alcoholism may have low innate immunity, and this may predispose them to develop EG.

mechanism by which alcohol modifies the immune system is not clear. It has been suggested that alcohol negatively influences the immune system in a mechanism similar to the HIV virus.¹³ It was shown that chronic alcoholics have increased endotoxin translocation from a weakened gut mucosa and a blunted monocyte response to infection.^{3,12}

Contributors SRG and VM treated the patient surgically and followed up until cure, took clinical photographs and wrote the manuscript.

Competing interests None declared.

Patient consent Obtained.

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