

Case Report

Rare recurrent gluteal erysipelas associated with pressure ulcers in elderly patients: A report of two cases

Takashi Nagase ^{a*}, Takafumi Kadono ^b^a Kunitachi Aoyagien Geriatric Health Service Facility, Tokyo, Japan, ^b Department of Dermatology, St. Marianna University School of Medicine, Kanagawa, Japan

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ABSTRACT

Erysipelas is an acute infection of the upper dermis layer of the skin. Contrary to the usual cellulitis, erythema of erysipelas has a very conspicuous irregular shape with clear demarcation from the surrounding normal skin. Recurrence is frequently observed. Gluteal erysipelas is very rare, reported to be found in only 0.6% of all patients with erysipelas. No previous report was found regarding gluteal erysipelas associated with pressure ulcers. We herein report two elderly cases of recurrent gluteal erysipelas with pressure ulcers. Strikingly, erythema was distributed far from the wounds, and the contours of erythema in the recurrent erysipelas were quite similar to those of the original erysipelas in both cases. Possible involvement of microcirculatory impairment of lymphatic vessels due to previous inflammation was discussed.

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1. Introduction

Erysipelas is an acute bacterial infection of the upper dermis of the skin and is especially common in elderly patients, infants, and immunocompromised patients.^{1,2} The clinical features of erysipelas differ from those of common cellulitis, an infection of the deeper soft tissues such as the subcutaneous fat and muscles.³ In patients with erysipelas, the erythema is clearly demarcated from the surrounding skin. Recurrence is frequently observed.⁴ Erysipelas is mostly found in the extremities and face; erysipelas of the buttock is very rarely seen and not well described.⁵ We herein report two cases of gluteal erysipelas associated with pressure ulcers.

2. Case reports

2.1. Case 1

An 88-year-old bedridden woman with a major cognitive disorder was admitted to our facility after having undergone right hip arthroplasty. One month later, a deep sacral pressure ulcer (Stage 4

pressure injury⁶) developed. The wound measured 8 × 7 cm within a few weeks with undermining of a few centimeters. The ulcer then gradually shrunk in size by pressure release with an appropriate support surface, vigorous positional changes, surgical debridement, general and topical administration of antibiotics, and topical treatment with basic fibroblast growth factor.⁷ Three months after onset, the wound had decreased to 3.0 × 0.5 cm and was well covered by granulation tissue, and no infectious or inflammatory symptoms were observed.

A large, warm skin rash measuring approximately 15–20 cm in diameter on the right buttock was noted 3.5 months after the onset of the pressure ulcer (Fig. 1A). The area of erythema was edematous and indurated, and the border of the erythema was conspicuous, irregular, and sharply demarcated from the surrounding skin, similar to the appearance of urticaria. The patient's body temperature was 38.1 °C, and her white blood cell count was 11,400/μl. The pressure ulcer showed no severe infectious or inflammatory symptoms. The erythema completely disappeared after intravenous administration of cefazolin sodium for 2 days.

The pressure ulcer was mostly healed, leaving a 1.0 × 0.3 cm epithelialized scar fissure 13 months after onset with no massive infection. We observed recurrence of the erythema seven times during this period. Intriguingly, the conspicuous irregular shape of the erythema was strikingly similar among the different recurrences (Fig. 1A–C). The patient's white blood cell count was 13,000/μl, and her C-reactive protein concentration was 9.5 mg/l at

* Correspondence to: Takashi Nagase, Kunitachi Aoyagien Geriatric Health Service Facility, 3-5-1 Aoyagi, Kunitachi-City, Tokyo 186-0013, Japan. Fax: +81 42 595 7627.

E-mail address: tnagase@fb3.so-net.ne.jp (T. Nagase).



Fig. 1. Photographs of Case 1. The contours of erysipelas were strikingly similar among the first recurrence (A: 4 months after onset), third recurrence (B: 6 months after onset) and fifth recurrence (C: 10 months after onset).

the time of the sixth recurrence. At each recurrence, the erythema was uneventfully treated by intravenous cefazolin sodium or oral cefdinir for 3–5 days. We clinically diagnosed these episodes as erysipelas because of the pattern of the recurrences and the characteristic appearance of the erythema.

2.2. Case 2

An 84-year-old bedridden woman was admitted to our facility because of a major cognitive disorder and history of cerebral

infarction and angina pectoris. She also had a history of repeated development of small superficial pressure ulcers (Stage 2 pressure injury⁶) in the coccygeal region a few times a year; they were healed by standard wound care each time. There was no history of major surgery.

Sudden development of a large area of erythema was observed when the patient developed a new coccygeal pressure ulcer. The size of the ulcer was 3 × 3 cm, and the size of the area of erythema was approximately 30 × 15 cm. The erythema was located in the lumbar/upper buttock region distant from the ulcer (Fig. 2A). As in

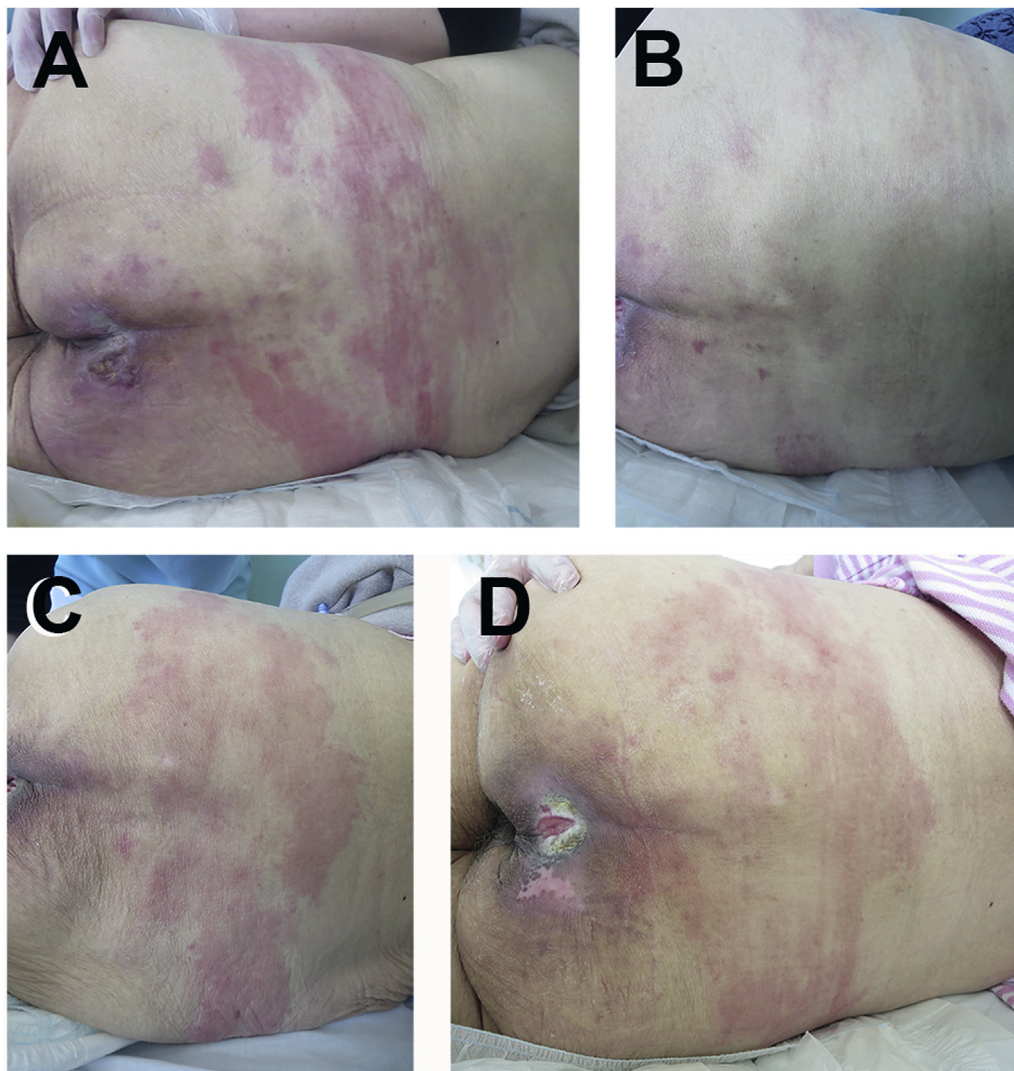


Fig. 2. Photographs of Case 2. The original erysipelas was located far from the pressure ulcer (A). It was healed three days later by oral antibiotics (B). As in Case 1, the appearance of the first recurrence (C: 1 month after onset) and second recurrence (D: 4 months after onset) were again quite similar to the original erysipelas.

the previous case, the border of the erythema was slightly elevated and clearly demarcated. The erythema disappeared after oral administration of cefdinir for 3 days (Fig. 2B). Two recurrences of the erythema with very similar contours were noted during the following 4 months (Fig. 2A–D), and they were healed by 3 days of oral antibiotics. The pressure ulcer was healed at 5 months.

3. Discussion

Erysipelas involves erythema with a characteristic well-demarcated contour because the inflammation and infection are localized in the upper dermis.^{1,2} The deeper tissues are affected by cellulitis, which induces redness and swelling in a more diffuse pattern without a clear border. Group A beta-hemolytic streptococcus has been believed to be the most common causal agent of erysipelas¹; however, this idea was recently critically reappraised, and other bacteria such as *Staphylococcus aureus* are now also considered to be possible mediators.² Bacteria invade the skin barriers through wounds, friction dermatitis between the toes, or folliculitis in patients with common erysipelas of the extremities or face. Diagnosis is usually based on clinical findings by an experienced dermatologist¹ because identification of bacteria requires a biopsy, which is invasive.

Gluteal erysipelas is extremely rare. According to Glatz et al,⁵ gluteal erysipelas was found in only 0.6% of all patients with erysipelas (9 of 1423 patients with erysipelas at all sites). Photographs of gluteal erysipelas in their report revealed a very conspicuous irregular border of the erythema, quite similar to our cases. They stressed that previous surgery, especially hip arthroplasty, may be an important predisposing risk factor for gluteal erysipelas as seen in Case 1 of the present report.⁵ Lymphedema after breast surgery is the most common risk factor for recurrent erysipelas in the upper extremities,⁴ suggesting that surgical impairment of the lymphatic microcirculation may also affect development of erysipelas in the buttock.⁵ It may not be directly mediated by remaining microorganisms in the scar tissue because one of the patients described by Glatz et al⁵ showed erysipelas even 20 years after hip surgery. An interesting hypothesis reported by other research groups is that a skin rash on the buttock can be caused by an allergic reaction to hip joint implants; however, this remains only speculative.⁸

A report by Glatz et al⁵ did not include an association of gluteal erysipelas with pressure ulcers, suggesting that our cases are quite rare. We consider that three interesting points should be addressed in our cases: 1) Erysipelas developed even when the ulcer underwent an uneventful healing process without massive infection. 2) Erysipelas was observed in a region remote from the ulcer. 3) The conspicuous irregular contour of the erysipelas was very similar among the recurrences. These facts suggest that the erysipelas seen in our cases may have been mediated by factors other than progression of the wound infection, as seen in common cellulitis. A possibility of physical environmental factors (e.g., allergic reaction to diapers) should be considered. Proske et al⁹ reported a case whose contact dermatitis of the hand caused recurrent erysipelas and secondary lymphedema. In this case, occupational exposure to

the allergen (the patient was a hairdresser) was as long as 8–10 years, suggesting that longer duration of dermatitis than our cases might be needed to develop erysipelas from contact dermatitis. As previously noted, we consider that irreversible impairment of the lymphatic microcirculation due to previous inflammation is a more important factor. If so, recurrence may occur even after the ulcer has completely healed.

As differential diagnoses of repeated gluteal rashes in bedridden patients, the following diseases may be considered; 1) acute diaper dermatitis, 2) other contact dermatitis, 3) Stage 1 pressure injury,⁶ 4) urticaria, 5) drug eruption and 6) candidiasis. Dermatitis should be excluded because epidermal damages were not observed. Pressure injury should be excluded because distribution of the skin rash was irrelevant to pressure. Urticaria should be excluded because the eruption persisted longer than 48 h, and the shapes of the eruptions were strikingly similar among the episodes. Drug eruption should be excluded because there was no history of irregular drug intake. Candidiasis should be excluded because there was no pustule, maceration nor desquamation.

Fortunately, intravenous or oral administration of antibiotics was very effective in our two patients. Some previous authors suggested the use of several years of prophylactic antibiotics to prevent recurrence.¹ However, we consider that the medical and nursing care provided by our geriatric health service does not require such a strict prophylaxis. Daily careful observation and immediate intervention at the time of recurrence were necessary and sufficient for our patients.

Conflicts of interest

There is no conflict of interests.

Acknowledgments

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