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European Journal of Obstetrics & Gynecology and Reproductive Biology 116 (2004) 117–119
EUROPEAN JOURNAL OF OBSTETRICS & GYNECOLOGY AND REPRODUCTIVE BIOLOGY
www.elsevier.com/locate/ejogrb

LETTER TO THE EDITOR—BRIEF COMMUNICATION

Pyoderma gangrenosum at the incision site following gynecologic surgery

Dear Editors,

Pyoderma gangrenosum is an uncommon necrotizing skin disorder and the diagnosis is made clinically because no specific histopathologic pattern is present [1]. Peak incidence is at age 30–50 years, with a slight female preponderance. In 50–75% cases an underlying condition is identified. The cause of pyoderma gangrenosum remains obscure; this condition was initially called pyoderma because it was thought to be a bacterial infection caused by streptococci. The cause is now recognized to be non-infectious, although secondary bacterial colonization may occur. Recent investigations emphasize an altered immune system with impaired cellular immunity and defective function of polymorphonuclear leukocytes [1,2].

A 27-old-year woman presented with a 6-month history of left lower quadrant pain. Her gynecologic and ultrasonographic examination revealed a large endometrioma of 10 cm and laparotomy was planned. Immediately before surgery povidone-iodine solution was applied to the abdominal skin. The abdomen was entered by a Pfannenstiel's incision and the cut edges of the subcutaneous tissue were protected by placement of moist towels at the incision edges. A self-retaining retractor with lateral blades was put in place. Cystectomy was performed for the endometrioma within the left ovary and peritoneal endometriotic implants were coagulated. Meticulous layer-by-layer anatomical closure was performed. The parietal peritoneum, fascia and skin were closed continuously with plain catgut, delayed absorbable polyglactin (Vicrly; Ethicon, USA) and subcuticular polypropilene (Prolene; Ethicon, USA), respectively. No suture was placed in the muscular and subcutaneous adipose tissue layers. The patient received prophylactic antibiotic therapy (Cefazolin 1 gr IV) and was discharged on day 3 postoperatively. At this stage, no peculiarity was noted at the incision site and the patient was called for follow-up 2 days later.

The next day (day 4) the patient presented with wound tenderness and low-grade fever. Examination of the incision revealed a 1-cm area of slight erythema and induration on the lower edge of the right margin. Her systemic examination was normal. Oral ampicillin-sulbactam was begun for presumed early wound infection and the patient was seen again 2 days later.

On day 6, the patient was febrile (39 °C) and the incision site was markedly tender. The well-defined erythematic region was red-purple and had expanded to a diameter of 3 cm. The incision edges of the right margin had separated and started draining purulent yellow-brown discharge. Cultures were obtained for causative pathogens. She was hospitalized for further evaluation. A regimen of broad-spectrum antibiotics (ampicillin and gentamicin) was started.

The lesion expanded rapidly despite broad-spectrum antibiotic therapy. On day 8, the right margin of the incision site was completely detached and the erythema had expanded to the upper edge of the incision. The wound was probed to facilitate drainage and surgical debridement was performed for suspected necrotizing wound infection. The wound was cleansed with wet-to-dry dressing three times daily.

On day 10, the blue-red lesion expanded in all directions in a superficial manner; the center of the lesion was necrotic, blue-purple in appearance while the periphery was erythematous. On day 12, the lesion appeared as a large skin ulcer with notable irregular ragged purple-red overhanging margins. The ulcer was very painful and was accompanied by malaise and fever. The patient received triple antibiotics (metronidazole, ampicillin and gentamicin) and wet-to-dry dressing for another 3 days. Finally, she was presumed to have pyoderma gangrenosum after dermatology and plastic surgery consultations. Small biopsies obtained from the center and undermined ulcer showed non-specific, diffuse, neutrophilic dermal infiltrate underneath the ulcer with scattered chronic inflammatory cells and areas of focal necrosis. Cultures of the biopsy material were negative for acid-fast bacilli, bacteria, and fungi. Based on the clinical appearance of the lesion and other findings, pyoderma gangrenosum was diagnosed.

The patient's medical, social and family histories were non-contributory. Laboratory investigations revealed an elevated white blood cell count of $25 \times 10^3\ l^{-1}$ with a differential count of 86% polymorphonuclear leukocytes. The erythrocyte sedimentation rate was 120 mm/h. Since pyoderma gangrenosum can be a feature of various diseases, several tests and consultations were performed to investigate the associated diseases. The results of the following investigations were either normal or negative: blood biochemistry tests, liver complement levels, antinuclear antibody test, and serum and urinary protein electrophoresis.

Systemic steroid therapy with oral prednisolone 80 mg per day was initiated. Local wound care was performed with

0301-2115/$ – see front matter
doi:10.1016/j.ejogrb.2004.01.039

wet-to-dry dressing, antiseptic creams (silver sulphadiazine and zinc oxide) and the ulcer was covered with bio-occlusive semi-permeable dressing (Epiguard, Smith & Nephew), which were changed regularly.

A dramatic response occurred within days as noted by reduction of pain and erythema. Her lesions started to heal within a week; the surrounding erythema disappeared, expansion of the ulcer ceased and epithelization started from the periphery and around the incision site, where the initial lesion was observed (Fig. 1). The wound improved progressively and healed uneventfully leaving a large cribriform scar. The patient was discharged on prednisone therapy (60 mg b.i.d.) which was subsequently tapered off to maintenance levels and stopped at 3 weeks.

Pyoderma gangrenosum can be a feature, even a presenting one, of inflammatory bowel disease, chronic autoimmune liver disease, connective tissue diseases, solid cancers, monoclonal gammopathies and hematological or immunological malignancies. In 40% patients with pyoderma gangrenosum, no associated disease can be identified [1]. In our patient pyoderma gangrenosum was not associated with any condition or disease.

The ulceration of pyoderma gangrenosum is frequently characteristic. The earliest symptom may be pain in the area, followed by a small erythematous papule. Typically, the erythematous nodule or acneiform lesion appears on the calf or thigh, less commonly on the buttocks and face, and skin rapidly breaks down to form an ulcer, commonly enlarging to over 10 cm. The border is well defined and deep erythematous to violaceous in color. The diagnosis of pyoderma gangrenosum is based on the clinical appearance of dermatitis, and a poor response to antibiotics [1–4]. In the absence of associated systemic disease, diagnosis is more difficult but is based on the same variables. Although the histopathologic features are not diagnostic, a skin biopsy is necessary to rule out other causes of acute skin ulcerations, particularly infections and necrotizing vasculitis.

The rapid development of the lesions and the appearance of the ulcers, with their pus-covered centers and the ragged, undermined, violaceous borders, are hallmarks of the disease and distinguish it from soft tissue infection [1,2]. Vesicles and bullae may be present, but were absent in our patient. Lesions are most commonly found on the lower extremities but have been reported on the scalp, face, trunk, and arms. The ulcers are usually painful and can be accompanied by malaise and low-grade fever, and can heal leaving atrophic cribriform scars. The lesions may be single or multiple and may be precipitated by trauma (pathergy), as in our patient. Pathergy is a condition in which the application of a stimulus makes the organism unduly susceptible stimulus of a different kind. A history of pathergy is reported in 25% of patients [3].

Crucial to the successful management of pyoderma gangrenosum are correct diagnosis, identification, and treatment of the underlying disorder and appropriate selection of local and systemic therapies [4]. Good topical care is essential for ulcer healing and involves cleansing, gentle debridement and prevention of bacterial colonization. Local antibiotic preparations, if tolerated, can be beneficial. Intra-lesional injections of corticosteroid are recognized to help [5]. Reported topical agents that have been used successfully include topical cromolyn sodium and topical mechlorethamine hydrochloride [6,7]. Surgical procedures such as debridement and skin grafting are not recommended during the acute stage of pyoderma gangrenosum, especially in patients

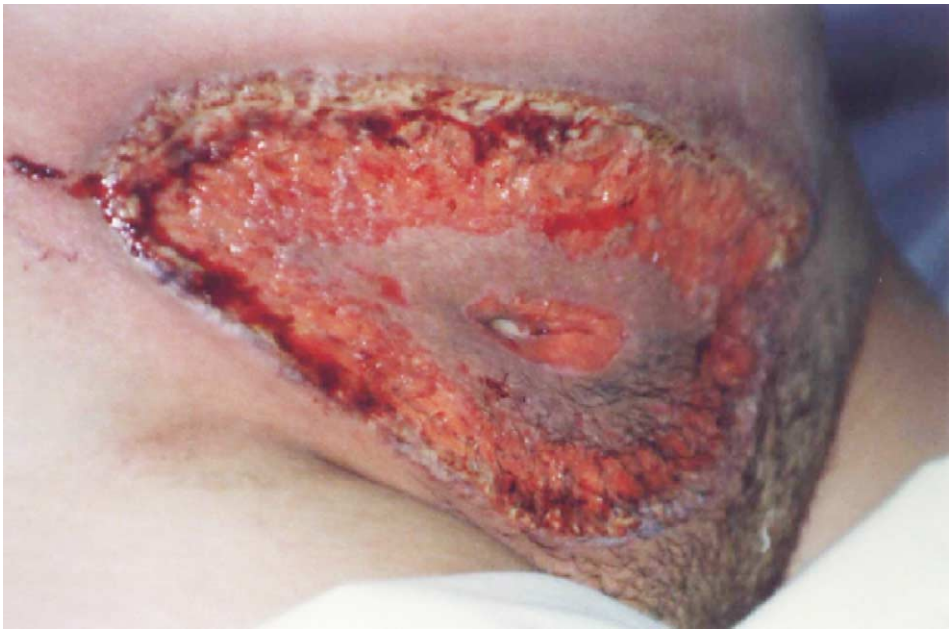

Fig. 1. Pyoderma gangrenosum at the incision site following gynecologic surgery.

who exhibit pathergy, because this could lead to further tissue destruction and progression [8]. Surgical debridement or grafting should be undertaken with great caution and only in patients who have no clinical evidence of active disease and are receiving appropriate immunosuppressive therapy.

High-dose parenteral corticosteroids are the treatment of choice for pyoderma gangrenosum [1,4,8]. Initial doses of prednisone of up to 80–120 mg per day may be given, with subsequent tapering of dosages to maintenance levels. Systemic treatment also includes therapies for the underlying disorder. High-dose systemic corticosteroids are commonly given with sulfasalazine, azathioprine, methotrexate, cyclophosphamide, chlorambucil and cyclosporin. Our patient had a rapid response to corticosteroid therapy with a reduction of pain and erythema within 12 h.

Acute pyoderma gangrenosum may simulate a wound infection when it develops postoperatively at the incision site. In gynecologic literature, we found only one case of pyoderma gangrenosum that developed at the incision site following cesarean delivery, which demonstrates that gynecologists are not familiar with this condition [9]. In our case, the condition mimicked a wound infection resulting in therapy delay and expansion of the lesion which consequently healed to form a very large cribriform scar despite appropriate therapy. The patient sought treatment for depression following this condition and was declined reconstructive surgery by plastic surgeons since it could be provoked by new surgery. When such a lesion appears following surgery, the characteristic morphologic appearance, negative cultures, skin biopsy consistent with the diagnosis, and failure to respond to antibiotics should point strongly to the diagnosis of pyoderma gangrenosum. Differential diagnosis with necrotizing fasciitis, a potentially lethal process, is essential. Necrotizing fasciitis is characterized by dishwater-like drainage, the edges of the incision is dusky and pale, and has a revitalized apparent. Tissue, especially the subcutaneous adipose, becomes mushy and easily fractured. A striking feature is the rapidity of progression that seems to advance before one's eyes. Necrotizing fasciitis is a surgical emergency and delay of diagnosis can lead to fatal progression of the disease.

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14 January 2004