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CASE REPORT

Pyoderma gangrenosum preceding the diagnosis of ulcerative colitis: a case report

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Pyoderma gangrenosum (PG) is an inflammatory, neutrophilic dermatosis characterised by rapid progression, ulceration, necrosis, and cribriform scar formation. It may be associated with inflammatory, haematologic, and rheumatologic conditions. A 32-year-old male patient from Gurage Zone, Ethiopia, presented with long-standing skin ulceration over his lower limbs. After about eight and a half years, he developed symptoms of ulcerative colitis (UC). His complete blood count revealed anaemia (haemoglobin 9.5 g/dl) and thrombocytosis. The erythrocyte sedimentation rate (ESR) was also elevated. A colonoscopy revealed evidence of pancolitis with backwash ileitis. Histology from colonic biopsy revealed chronic inflammatory cells and crypt abscesses consistent with the UC diagnosis. Later, the patient was started on prednisolone (1 mg/kg/day) and azathioprine, improving the gastrointestinal (GI) symptoms without significant changes to the skin lesions. It is important to note that UC can occur before or after the onset of PG, and a low threshold should be maintained for investigating UC in patients with PG.

Keywords: pyoderma gangrenosum, ulcerative colitis

Introduction

PG is a reactive, non-infectious, inflammatory dermatosis, which is on the spectrum of neutrophilic dermatoses that present as an extremely painful erythematous lesion with rapid progression to a blistered or necrotic ulcer, often with a ragged violaceous/erythematous border. The most frequently affected sites are the lower extremities, though it can occur anywhere.[1] More than half of PG cases are associated with an underlying comorbid systemic disease, including inflammatory bowel disease (IBD), haematologic disorders, connective tissue disease, and rheumatoid arthritis.[2] Skin lesions may precede, follow, or occur alongside the associated disease.[3]

Case description

We present a 32-year-old male patient who was relatively healthy 10 years prior, at which time he presented with skin lesions over both lower extremities. These were initially pruritic with easy bruising and minimal purulent discharge over the anterior aspect of the shins. Three years after the initial presentation, the lesions expanded with significant pain. At that time, the patient visited a dermatologist at a government hospital, and the PG diagnosis was made after a skin biopsy. After outpatient follow-up sessions, he was referred to Tikur Anbessa Specialised Hospital.

Initially, the patient was treated with oral prednisolone at 40 mg/day, later tapered to a lower dose within three months (5 mg/day orally), and daily clobetasol ointment (0.05%), without significant improvement. The patient developed intermittent bloody diarrhoea with a frequency of 4–6 times per day, with associated tenesmus, easy fatigability, and dizziness for the past year and a half. He had no fever, chills, or rigours. He had no family history of a similar illness.

On physical examination, his blood pressure was 100/70 mmHg, with a pulse rate of 100 beats per minute, a respiratory rate of 20 breaths per minute, and an axillary temperature of 36.7 °C. His body mass index (BMI) was 17.6 kg/m^2, and his mid-upper arm circumference was 22 cm. He had pale conjunctivae and anicteric sclerae. Findings on the integumentary system were notable, with multiple hypopigmented lesions accompanied by areas of hyperpigmentation and scarring over the anterior aspect of both shins and the feet bilaterally, as well as scaly, dried, yellowish matter at the borders (Figure 1).

The laboratory evaluation of the patient showed a haemoglobin level of 9.5 g/dl, a mean corpuscular volume of 63.3 fl (low), and a platelet count of 734 000/µl (reactive thrombocytosis).

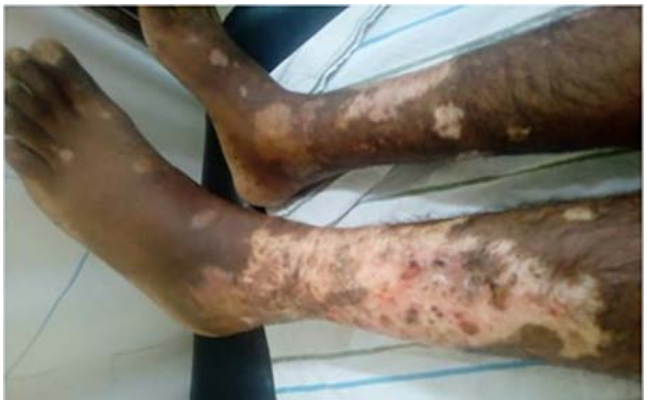

Figure 1: Characteristic cribriform scar with hypopigmentation on the anterior shin in a patient diagnosed with ulcerative colitis

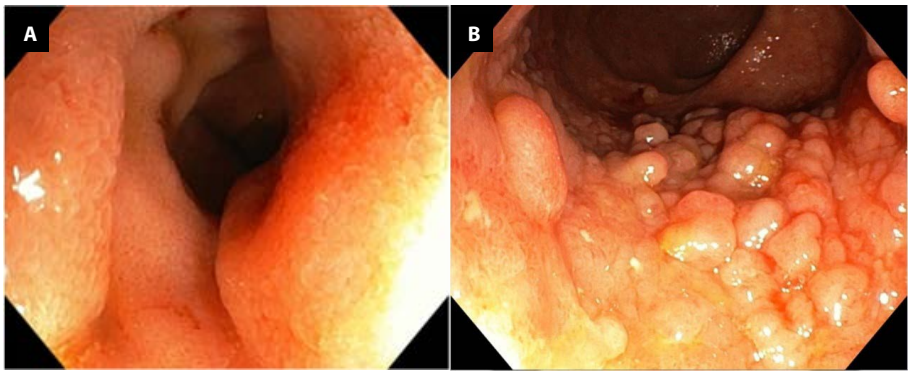

Figure 2: A) Oedematous terminal ileal mucosa B) Multiple pseudopolyps at the colon

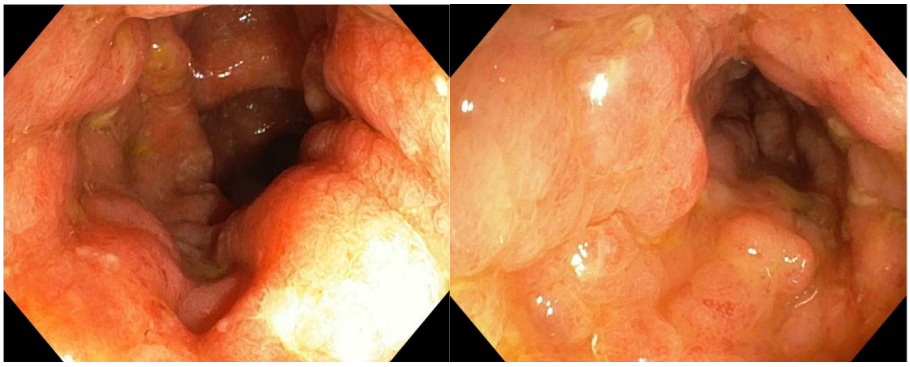

Figure 3: Marked erythema, absent vascular pattern, small ulcers, and pseudopolyps seen at the colon

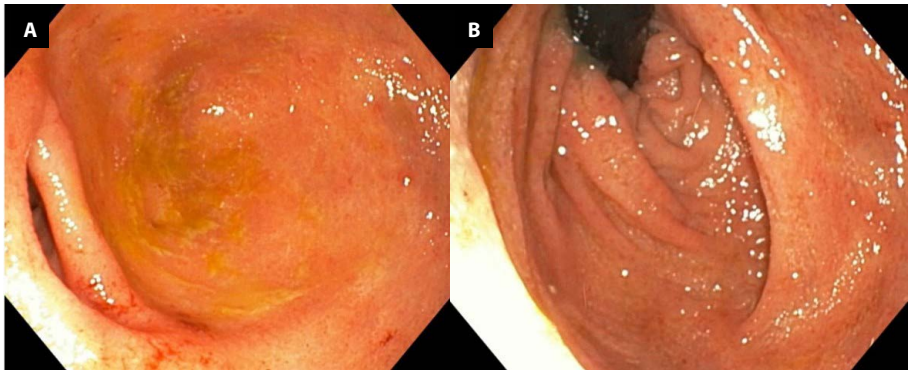

Figure 4: A) Oedematous cecal mucosa B) Rectal involvement

ESR was 80 mm/hr (elevated). The white blood cell count, liver enzymes, bilirubin, renal function, and serum electrolytes (Na, K, Cl) appeared normal. Serum albumin was 2.8 g/dl (low). Hepatitis B surface antigen, anti-hepatitis C virus antibody, and human immunodeficiency virus (HIV) tests were negative. Stool examination and culture were also negative.

The colonoscopy revealed involvement of the entire colon with diffuse mucosal oedema, loss of vascular pattern, granularity, and easily friable mucosa with inflammatory pseudopolyps (Figures 2, 3, and 4). Biopsies taken from all colonic segments showed colonic mucosal fragments with a polypoid configuration exhibiting surface ulceration and an expanded lamina propria with mixed inflammatory cells, including plasmacytosis, crypt abscesses, and fibrosis, confirming the UC diagnosis.

The patient was admitted and started on intravenous hydrocortisone 100 mg three times a day with significant improvement of the diarrhoea and related symptoms. Oral azathioprine 50 mg/day was given as maintenance treatment, later escalated to 150 mg/day orally, according to his weight. Iron replacement therapy was also given.

Discussion

Extraintestinal manifestations associated with IBD are usually related to an exacerbation of the underlying disease, although they may exhibit a course independent of the disease activity, sometimes preceding the diagnosis. Our patient had a skin manifestation long before he developed the clinical manifestations of IBD. About a third of patients with IBD develop extraintestinal manifestations, such as PG. PG is an uncommon inflammatory skin disorder with unclear pathogenesis and no specific serological or histological markers.[4] Consequently, PG diagnosis relies predominantly on clinical assessment.[4]

The incidence of PG is uncertain. It is estimated that 3–10 patients per million population per year experience this condition, with a peak incidence between the ages of 20 and 50 years.[5] Worldwide, the incidence and prevalence of UC have been gradually increasing. The highest incidences of UC were reported in northern Europe (24.3 per 100 000), Canada (19.2 per 100 000), and Australia (17.4 per 100 000).[6] PG can be associated with IBD, representing up to 1–3% of its extraintestinal dermatological manifestations. PG may occur alongside UC and Crohn's disease, with a similar prevalence.

The correlation between IBD activity and PG is still controversial; even if PG onset may anticipate IBD by years, the two conditions seem to progress independently, according to some studies.[7] PG often affects patients with an underlying disease, frequently an IBD, such as UC and Crohn's disease, or rheumatoid arthritis.[8] The legs are the most affected, similar to our case, but other parts of the skin and mucous membranes may also be involved. PG has been reported on other sites of the body, including breast, hand, trunk, head and neck, and peristomal skin.[9]

A faecal calprotectin is advised if there is a clinical suspicion of IBD.[1] Approximately 50% of PG cases are associated with systemic diseases (autoimmune, neoplastic, haematological) or secondary to drugs. To ascribe drugs as a cause, active investigation of these entities is mandatory.[10] Systemic corticosteroids, cyclosporine,

and tumour necrosis factor-alpha inhibitors have the strongest evidence supporting their efficacy and safety in PG.[11]

Conclusion

We presented a 32-year-old man with persistent skin ulcers on his lower limbs that were subsequently accompanied by symptoms of UC after eight and a half years. The diagnosis of UC was made through clinical, laboratory, colonoscopy, and histological findings. Proper evaluation of patients with PG for GI symptoms, such as diarrhoea, tenesmus, and bleeding, followed by colonoscopy and colonic biopsy, is vital for diagnosing underlying UC.

Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

This case report was approved by the Research Ethics Review Committee of the School of Medicine, Addis Ababa University. Prior to data collection, written informed consent was acquired from the patient.

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