Case Report

Pyoderma gangrenosum triggered by insect bite: two case reports

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Abstract

Pyoderma gangrenosum is rare, probably autoimmune vasculitis and non-infective ulceration of skin. Two case reports are presented here, with interesting similarities and probable cause.

Key words

Pyoderma gangrenosum, insect bite, corticosteroids.

Introduction

Pyoderma gangrenosum (PG) is an autoimmune neutrophilic dermatosis without prominent vasculitic changes. It presents as a furuncle-like nodule or haemorrhagic bullae with blue centre or may resemble transient acantholytic dermatosis. The lesion enlarges and acquires the size of large ulcerating lesion as much as 10cm or more within 5 to 7 days. Edges of ulcer are often bluish in colour, raised, undermined and overhanging with surrounding erythema in early stage. Any area of body can be involved e.g. lower extremities, buttocks, abdomen, face. Dermatopathology is not pathognomonic and shows neutrophilic abscess formation and necrosis. Exact pathogenesis is unknown, though defective immune mechanisms i.e. T cell imbalance or failure of phagocytosis by monocytes are implicated in many cases.

PG may be associated with a number of diseases (Table 1). However, about 50% of cases are idiopathic.

Case 1

A 58-year-old female presented at dermatology outpatient department of District Headquarter Hospital, Tando Muhammad Khan with huge ulcerative lesion over extensor aspect of right forearm. Ulcer had erythematous base with purulent areas and ragged edges. It was painful and tender. Patient told the history of insect bite over forearm about 8 to 10 days back, after that a small nodule appeared which ulcerated and acquired present size (15cmx6cm) within 4 to 5 days.

Patient was fully conscious and well oriented. She was anemic with moderate fever (temperature 101.2°F). No other systemic abnormal finding was revealed.

Following investigations were done. Urine examination, blood counts, X-ray chest, X-ray right forearm, (to see deeper tissue involvement and gas in and around muscle), blood sugar, stool examination, rheumatoid arthritis factor, ANA, liver function tests, pus from ulcer for Gram stain and culture. All investigations except for the hemoglobin level of 7.5gm/dl were
Table 1 Pyoderma gangrenosum associated diseases [7]

I. Diseases of Gastrointestinal Tract
   i. Ulcerative Colitis
   ii. Crohn’s disease
   iii. Diverticulosis
   iv. Gastritis
   v. Gastric or duodenal ulcers
   vi. Intestinal polyps

II. Diseases of liver
   i. Chronic active hepatitis
   ii. Primary biliary cirrhosis
   iii. Sclerosing cholangitis

III. Arthropathies
   i. Rheumatoid arthritis
   ii. Ankylosing spondylitis
   iii. Osteoarthritis
   iv. Polychondritis

IV. Hematological disorders
   i. Leukemias
   ii. Myeloproliferative syndrome
   iii. Hyperglobulinemia
   iv. Thrombocythemia
   v. Splenomegaly
   vi. Myelodysplasia
   vii. Dysglobulinemia
   viii. Congenital
   ix. Monoclonal
   hypogammaglobulinaemia
   x. Myeloma
   xi. Lymphoma

V. Neoplasia
   i. Cancer of the colon, prostate, breast or bronchus
   ii. Carcinoid tumour

VI. Infectious disease
VII. Posttraumatic
VIII. Postoperative
IX. Miscellaneous
   i. Thyroid disease
   ii. Diabetes
   iii. Diseases of lung
   iv. Takayasu arteritis
   v. Lupus erythematosus
   vi. Sarcoidosis
   vii. Wegener’s disease
   viii. Mondor’s disease
   ix. Insect bite
   x. Postvaccinia
   xi. Disseminated intravascular coagulation
   xii. A fibrinogenemia
   xiii. Retinoid treatment of acne
   xiv. Hidradenitis suppurativa
   xv. Palmoplantar pustulosis
   xvi. Subcorneal pustulosis
   xvii. Transient acantholytic dermatosis
   xviii. Dermatitis herpetiformis
   xix. Erythema elevatum diutinum
   xx. Immunosuppression
   xxi. Acquired immune deficiency syndrome

Patient was admitted and treated with intravenous glucocorticoids. Healing started within 48 hours of therapy. Complete healing by scar formation occurred in three weeks. Patient was discharged and referred to tertiary care hospital for management of anaemia.

Case 2

A 65-year-old male presented at Dermatology outpatient department, District Headquarter Hospital, Tando Muhammad Khan with large ulcerating lesion over lateral aspect of left thigh. This patient also gave history of insect bite about 10 to 12 days ago after which a furuncule-like nodule appeared, enlarged and burst and achieved present size within 5 to 6 days. The lesion was accompanied by high grade fever. Rest of systemic inquiry was unremarkable.

Patient was conscious, well-oriented with body temperature of 99.4°F. Lesion was tender and painful. On lower thigh, there was a 12cmx5cm ulcer with a reddish base with purulent areas and ragged edges (Figure 2).

Investigations including urine examination, complete blood counts, X-ray chest, X-ray left thigh, blood sugar, RA factor, ANA, liver function tests, and pus for Gram staining and bacterial culture, were within normal range. Skin biopsy showed neutrophilic infiltration and necrosis.

Patient was prescribed oral prednisolone
60mg/day in three divided doses for 1st week then tapered off. Complete healing occurred within six weeks of treatment.

Discussion

Interesting similarity in two cases presenting on the same day prompted to report them. Both cases gave the history of insect bite, belonged to the same locality and closely resembled in clinical appearance, development and resolution of lesion.

Since the first association of PG with ulcerative colitis, many triggering causes have been described (Table 1). Insect bite, as in our two cases, has been reported an occasional cause.

Clinically, PG has a wide array of differential diagnosis. Ulcerative PG may resemble any ulcer of infective, vasculitic, ischemic or malignant origin. Similarly, the histopathology is not pathognomonic and may resemble dermatoses with predominant neutrophilic infiltrate. There are no characteristic laboratory indices to confirm the PG. All these make PG a diagnosis of exclusion.

The most important challenge is to find out the underlying cause which remains obscure in the majority. Like all other dermatoses of autoimmune origin, the disease responds to glucocorticoids, immunosuppressives and anti-inflammatory drugs.

References

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