Blastomycosis-like pyoderma: A diagnostic, as well as, therapeutic challenge

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Abstract
Blastomycosis-like pyoderma is a very rare, chronic inflammatory disorder, which is also known as pyoderma vegetans (PV). It is commonly seen in middle aged males. It is found to be associated with reduced immunity like primary immunodeficiency syndromes, nutritional deficiencies, HIV infection, diabetes mellitus, chronic myeloid leukemia and alcoholism. It tends to have a link with inflammatory bowel diseases. We report a 41-year-old male who presented with pus filled lesions over his left shoulder, axilla and left elbow for last 4 months. Initially, it was a painless pea-sized lesion, which gradually enlarged to form a larger lesion with central ulceration and peripheral extension. He had consulted a couple of doctors and took multiple courses of antibiotics, which had failed to comfort him of the lesions. Cutaneous examination revealed a well-defined large plaque, sized 16x14cm over the left shoulder extending to the axilla with few pustules, which had erythematous base with serous-purulent discharge. The plaque was nontender and ulcerated, with uneven surface, elevated edges and yellowish crust. There was a similar smaller (sized 5x3cm) plaque over the left elbow. Colonoscopy was not suggestive of either Crohn’s disease or ulcerative colitis. Histopathological examination revealed pseudoepitheliomatous hyperplasia and upper dermal abscesses with predominantly neutrophilic infiltrate. He was further started on prednisolone 40mg once a day in the morning hours after breakfast along with antibiotic for 10 more days. Corticosteroid was tapered by 10mg every 10 days and 10mg was given for a period of 1 month. Resolution of vegetating granulation tissue was observed within 10 days month and complete remission in 1 month. It is very essential to be familiar with PV, as correct diagnosis may reveal an underlying inflammatory bowel disease.

Key words
Blastomycosis-like pyoderma, pyoderma vegetans.

Introduction
Pyoderma vegetans (PV), also known as blastomycosis-like pyoderma is a very rare, chronic inflammatory disorder. It is commonly seen in middle-aged males. It is found to be associated in persons with reduced immunity like primary immunodeficiency, nutritional deficiencies, HIV infection, diabetes mellitus, chronic myeloid leukemia and alcoholism.1,2 It tends to have a link with inflammatory bowel diseases (Crohn’s disease and Ulcerative colitis) and cutaneous T-cell lymphoma, as well.3 Microbial infections, tattooing, foreign bodies and halogen ingestion are the factors which can precipitate this disorder. It is characterized by large exudative plaques which are verrucous and have well-defined and elevated borders. It has been reported in immunocompetent patients, too.1 The pathognomonic histopathological features in PV are pseudoepitheliomatous hyperplasia with intraepidermal and subepidermal neutrophilic microabscesses.4

Case Report
A 41-year-old man presented with pus filled lesions over his left shoulder, axilla and left
elbow for last 4 months. Initially, it was a painless pea-sized lesion, which gradually enlarged to form a larger lesion with central ulceration and peripheral extension. There was no history of trauma. He gave history of similar lesions over the back and right thigh 1 year back which had healed leaving brown colored flat lesions. He had consulted a couple of doctors and took multiple courses of antibiotics, which failed to comfort him of the lesions. He was a known alcoholic for last 20 years. There was no history of any cough or fever. His general and systemic examinations were found to be normal.

Cutaneous examination revealed a well-defined large plaque, sized 16×14cm over the left shoulder extending to the axilla with few pustules, which had erythematous base with serous-purulent discharge. The plaque was nontender and ulcerated, with uneven surface, elevated edges and yellowish crust (Figure 1a). There was a similar smaller (sized 3×5cm) plaque over the left elbow. Left axillary lymphadenopathy was appreciated. There were few hyperpigmented patches over the back and right thigh, suggestive of old healed lesions.

The clinical differential diagnoses included lupus vulgaris, chromoblastomycosis, cutaneous nocardiosis, atypical mycobacterial infection and pyoderma vegetans as diagnosis of exclusion. He was investigated for complete blood count, liver function tests, renal function tests, glucose levels which were within normal limits with hemoglobin of 8.6gm/dl. Mantoux test was negative. Colonoscopy was performed to check for any inflammatory bowel disease, however, there was nothing suggestive of either Crohn’s disease or ulcerative colitis. Histopathological examination revealed pseudoeoepitheliomatous hyperplasia and upper dermal abscesses with predominantly neutrophilic infiltrate (Figure 2a and 2b).
were no granulomas and special stains like periodic acid-Schiff and Ziehl-Neelsen were negative for fungal elements or acid-fast bacilli. Pus culture examination showed growth of Enterococci, Proteus mirabilis, and Escherichia coli repeatedly at several occasions. A diagnosis of pyoderma vegetans was ascertained, the patient fulfilled 5 of 6 criteria proposed by Su, Duncan and Perry. Patient had taken multiple courses of antibiotics from several dermatologists without much relief. We started him on hematinic syrup along with systemic antibiotics like clindamycin and rifampicin for 10 days and witnessed not a slightest improvement in the lesions. He was further started on prednisolone 40mg once a day in the morning hours after breakfast along with antibiotic for 10 more days. Corticosteroid was tapered by 10mg every 10 days and 10mg was given for a period of 1 month. We could not put him on dapsone for maintenance due to low levels of hemoglobin. Resolution of vegetating granulation tissue was observed within 10 days and complete remission in 1 month (Figure 1b and 1c).

Discussion

Pyoderma vegetans is a very rare disease. It is an unusual vegetative tissue proliferation in response to an infection. Su et al. proposed a diagnostic criteria for PV which are: (1) Isolation of a pathogenic microbe; (2) clinical presentation of large verrucous plaques with multiple pustules and elevated borders; (3) microscopic findings of pseudoepitheliomatous hyperplasia with neutrophil-rich microabscesses within the epidermis and dermis; (4) negative culture for deep fungi, atypical mycobacteria, and Mycobacterium tuberculosis; (5) negative fungal serology test result; and (6) bromide level in the blood within reference range. The common organisms isolated from tissue cultures include Staphylococcus aureus, Beta-hemolytic streptococci, P. mirabilis, E. coli, and Candida albicans and anaerobes like Corynebacterium. PV is considered a marker of inflammatory bowel disease; colonoscopy is to be performed to investigate for ulcerative colitis or Crohn’s disease. More commonly it occurs in immunocompromised individuals. Clinically, the differentials are lupus vulgaris, pyoderma gangrenosum, and deep fungal infection like chromoblastomycosis. Hence, tissue culture and histopathology are mandatory to come to a final diagnosis. Even knowing about the bacterial etiology, systemic antibiotics alone are no effective treatment of PV, the standard first-line therapy is systemic steroids at a dose of 0.5 to 1.0 mg/kg, which are tapered with time. The other treatment options include dapsone, azathioprine, cyclosporine and laser debridement.

Conclusion

It is very essential to be familiar with PV, as correct diagnosis may reveal an underlying inflammatory bowel disease and help us for prompt treatment.

References

