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Case Series

Pathology Section

Clinico-histopathological Features of Pyoderma Gangrenosum: A Case Series

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ABSTRACT

Pyoderma Gangrenosum (PG) is a rare, ulcerative, non infectious neutrophilic inflammatory dermatosis and often associated with underlying systemic disorder. The incidence of PG is estimated to be 0.63 per 1,00,000 with the median age at presentation of 59 years. Among its clinical variants, classical PG is the most common. The diagnosis of PG can be difficult. This case series was an attempt to review the new trends for the diagnosis of PG and also to compare them with previous diagnostic criteria. Previously histopathological criteria were included as minor criteria in Su WP et al., classification. More recently proposed diagnostic criteria by Maverkis E et al., include histopathology of skin biopsy from edge of ulcer showing neutrophilic infiltration as a major criteria. Aim of the study is to correlate and stamp the clinically suspected cases of PG with help of histopathology. This retrospective observational case series study was conducted in a tertiary care hospital from May 2018 to April 2020 consisting of 19 cases (15 years to 68 years of age range; 13 males and 6 females). Detailed history, clinical examination and blood investigations were done in all suspected cases of PG followed by histopathological examination of skin biopsy. In 17 cases, lesions were located in lower limb, one case each in buttocks and lower abdomen. The classical, ulcerative form found in 12 cases (63.15%), vegetative form in 3 cases (15.78%), plaque and bullae form were in 2 cases each (10.52%). Pathergy test was positive in 11 cases (57.89%). Histopathological examination showed neutrophilic infiltration in all 19 cases (100%), vasculitis in 11 patients (57.89%), lymphoplasmacytic infiltrate in 6 patients (31.57%), pseudoepitheliomatous hyperplasia in 5 patients (26.31%), mixed inflammatory infiltrate in 4 patients (21%), Epidermal ulceration in 4 patients (21%) and mitosis is seen in 3 patients (15.78%). Histopathology is considered as a main tool which helps clinicians to stamp suspected cases of PG.

Keywords: Leukocytoclastic vasculitis, Neutrophilic infiltration, Panniculitis, Ulcer

INTRODUCTION

Pyoderma Gangrenosum (PG), first described by Brocq in 1916 and named by Brunsting et al., in 1930 as is mentioned in study by Bhat RM et al., [1]. It is a reactive, non infectious neutrophilic inflammatory dermatosis that typically presents as rapidly progressive, painful, necrotic ulcer with ragged undermined edge with erythematous border. The incidence of PG is estimated to be 0.63 per 1,00,000 with the median age at presentation of 59 years [2]. It has a predilection for lower extremities although it can present at any site of body. PG can occur at any age, but generally presents in the second to fifth decade of life. The lesion may be precipitated by minor trauma, a phenomenon known as 'Pathergy' [3]. Pathophysiology of PG is not well understood but loss of innate immune regulation and altered neutrophil chemotaxis are believed to be involved to some extent [4]. Underlying systemic conditions like inflammatory bowel diseases, rheumatoid arthritis and some haematological malignancies are found in 50% cases of PG [5]. Classical PG is most common form (approximately 85%) but other clinical variants include pustular, bullous, vegetative and superficial granulomatous [1].

Aim of skin biopsy was to exclude other causes of cutaneous ulceration such as infection, vasculitis (Wegener granulomatosis, Chrugstrauss syndrome, Behcet disease), malignancies (cutaneous lymphoma, leukaemia, basal cell carcinoma, squamous cell carcinoma), as histopathology is a main tool according to improved Maverakis criteria to diagnose suspected cases of PG [6], It will also help the clinician to confirm the diagnosis of PG and expand the available literature.

CASE SERIES

The present case series with retrospective observational design was conducted in the Department of Pathology at Ahmedabad Municipal Corporation's Medical Education Trust Medical College, Sheth Lallubhai Gordhandas Municipal Hospital, Ahmedabad, Gujarat, India, over a period of two years from May 2018 to April 2020. This study got approval from Institutional Review Board, AMC MET Medical College on 29th December 2020. All biopsies from skin lesions which were clinically suspicious of PG and confirmed histopathologically were included in the study. Inadequate biopsy material was excluded from study.

Relevant clinical information (e.g., age, sex, site of lesions, associated diseases, pathergy test results and clinical types of PG) was obtained from case reports of all the included patients [Table/Fig-1]. Skin pathergy test was done in all patients on volar aspects of forearm. Skin prick was done using 16 gauge needle, inserted at 45 degree and read after 48 hours. Development of papule or pustule consider as a positive test [7]. All the biopsies suspected as PG by Dermatology Department were processed, cut and stained by Haematoxylin and Eosin (H&E). Then they were evaluated histopathologically.

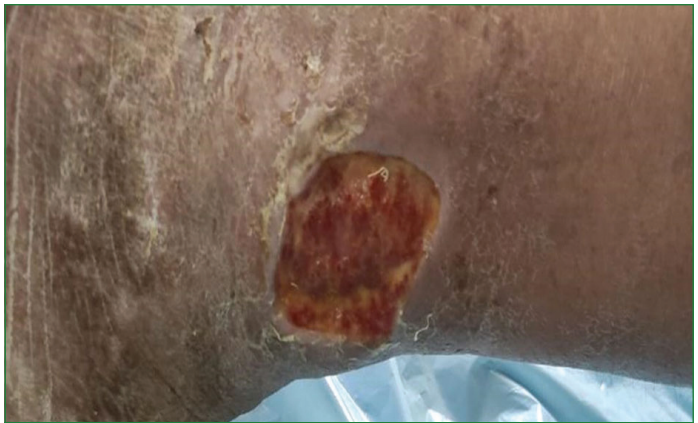
Case no.	Age (years)/ Gender	Co-morbidities associated	Pathergy test	Type of PG	Location	Histology
1	68/M	Diabetes mellitus, Hypertension	Negative	Ulcerative	Right leg	Infiltrate of neutrophils, leucocytoclastic vasculitis.
2	27/M	NA	Positive	Vegetative	Left leg	Pseudoepitheliomatous hyperplasia, neutrophilic abscess in epidermis, mixed inflammatory cell infiltrate in dermis, vasculitis.

3	28/F	Inflammatory arthritis	Negative	Ulcerative	Both back of heel	Infiltrate of neutrophils in epidermis and mild lymphocyte mediated vasculitis.
4	28/M	NA	Negative	Ulcerative	Left medial malleolus of left foot	Infiltrate of neutrophils in epidermis, leucocytoclastic and lymphocyte mediated vasculitis.
5	45/M	NA	Negative	Vegetative	Right knee	Infiltrate of neutrophils in epidermis, lymphocytic vasculitis.
6	40/F	Inflammatory arthritis	Negative	Nodules and plaque	Left great toe	Neutrophilic infiltrate in epidermis with superficial dermal suppurative inflammation admixed with plasma cells and eosinophils, pseudoepitheliomatous hyperplasia, occasional mitosis .
7	53/M	NA	Positive	Ulcerative	Right leg	Pseudoepitheliomatous hyperplasia, neutrophilic infiltrate in epidermis, lymphoplasmacytic infiltrate in dermis.
8	15/M	NA	Positive	Ulcerative	Right leg	Neutrophilic infiltrate in epidermis, vasculitis.
9	15/F	NA	Positive	Haemorrhagic bullae	Dorsum of left foot	Acanthotic squamous epithelium with intraepidermal bullae containing neutrophilic leucocytes.
10	22/M	NA	Positive	Ulcerative	Right lower leg	Neutrophilic infiltrate in epidermis, neutrophilic, eosinophilic, lymphoplasmacytic infiltrate and histiocytes in dermis.
11	45/F	NA	Negative	Ulcerative	Left side lower abdomen	Neutrophilic infiltrate in epidermis with ulceration, lymphocytic vasculitis.
12	20/M	Ulcerative colitis	Negative	Ulcerative	Right leg	Epidermis with focal ulceration and neutrophilic infiltration, ulcer base has granulation tissue with mixed inflammatory infiltrate, lymphocytic vasculitis.
13	52/M	Diabetes mellitus	Positive	Vesicle pustule	Left leg	Neutrophilic infiltrate in epidermis, polymorphonuclear leucocytic infiltrate in dermis, occasional mitosis is seen.
14	17/M	NA	Positive	Ulcerative	Right leg	Neutrophilic exudates in epidermis, Lymphocytic vasculitis.
15	70/F	Herpes zoster	Positive	Ulcerative	Lateral side of foot	Epidermis with ulceration and neutrophilic infiltration, lymphoplasmacytic infiltrate in dermis, proliferation of blood vessels.
16	50/M	Hypertension	Positive	Ulcerative	Buttocks	Infiltrate of neutrophils in epidermis, vasculitis.
17	48/M	Diabetes mellitus, Hypothyroidism	Positive	Ulcerative	Right thigh	Neutrophilic infiltrate in epidermis with ulceration, lymphocytic vasculitis.
18	19/M	Ulcerative Colitis	Positive	Vegetative	Right leg	Neutrophilic infiltrate in epidermis, pseudoepitheliomatous hyperplasia, lymphoplasmacytic infiltrate in dermis, occasional mitosis is seen.
19	19/F	NA	Negative	Plaque	Leg	Neutrophilic infiltrate in epidermis, pseudoepitheliomatous hyperplasia.

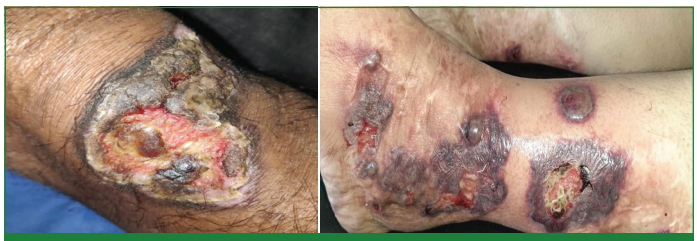
[Table/Fig-1]: Clinical and histopathological characteristics of patients of Pyoderma Gangrenosum (PG).
M: Male; F: Female; NA: Not applicable

Total 19 cases were identified by clinical findings and histopathological features consistent with PG together with haematological, biochemical and microbiological investigations to rule out the other causes of cutaneous ulceration.

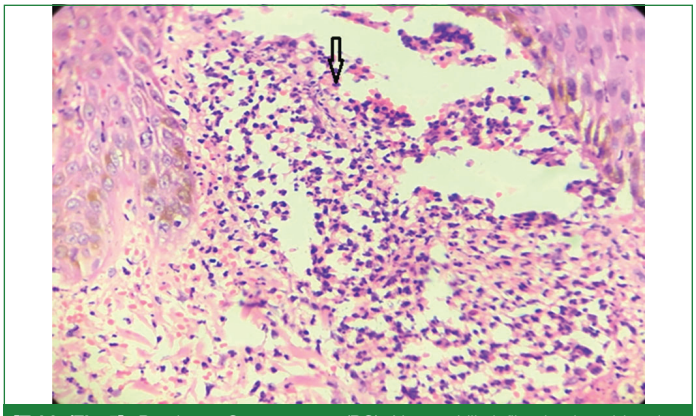
- Out of 19 patients 13 were males and 6 were females; sex ratio M:F=2.2:1. Majority of patients in this case series were between 15 to 50 years.
- The classical, ulcerative form [Table/Fig-2] was found in 12 cases (63.15%), vegetative form [Table/Fig-3] in 3 cases (15.78%), plaque and bullae form [Table/Fig-4] were in 2 cases each (10.52%).
- Lesions were mainly located in lower limbs in 17 cases, buttocks and lower abdomen in one case each. Pathergy test was positive in 11 cases (57.89%).
- Histopathological examination showed a neutrophilic infiltrate [Table/Fig-5,6] in all 19 patients (100%), vasculitis in 11 patients (57.89%), lymphoplasmacytic infiltrate in 6 patients (31.57%), pseudoepitheliomatous hyperplasia in 5 patients (26.31%), mixed inflammatory infiltrate in 4 patients (21%), Epidermal ulceration in 4 patients (21%) and mitosis is seen in 3 patients (15.78%) [Table/Fig-7].
- Associated disease with PG were found in 9 cases (47.36%), out of which diabetes mellitus in 3 cases (15.78%), Inflammatory arthritis, ulcerative colitis and hypertension in 2 cases each



[Table/Fig-2]: Ulcerative Pyoderma Gangrenosum (PG) (Gross picture)- A solitary 3.5×2.5 cm sized well-defined ulcer with red granulation tissue at the base, indurated margin, not fixed to underlying tissue, present over medial aspect of distal one-third of the right leg.



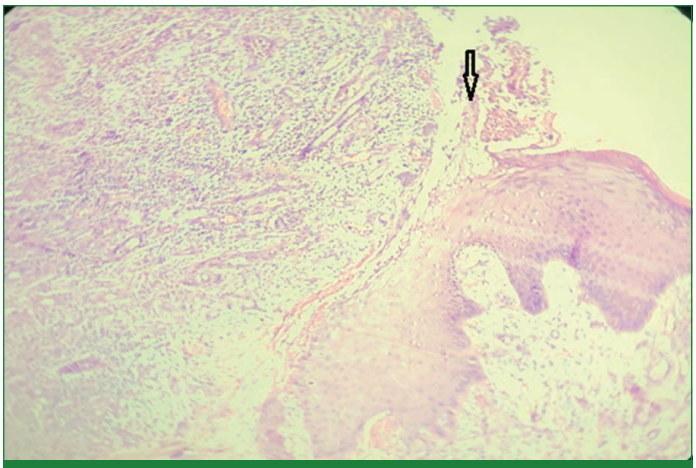
[Table/Fig-3]: Vegetative Pyoderma Gangrenosum (PG) (Gross picture)- Single 5.5x2 cm sized well-defined ulcer with vegetative growth, red granulation tissue over base and thickened margin presents over anterior aspect of left lower leg;
[Table/Fig-4]: Bullae with haemorrhagic form of Pyoderma Gangrenosum (PG) (Gross picture)-Multiple well-defined vesicles and bullae, filled with haemorrhagic fluid, with surrounding erythema and some of the bullae ruptured to form superficial ulcers with irregular border and thickened margins present over lateral aspect of left leg. (Images from left to right)



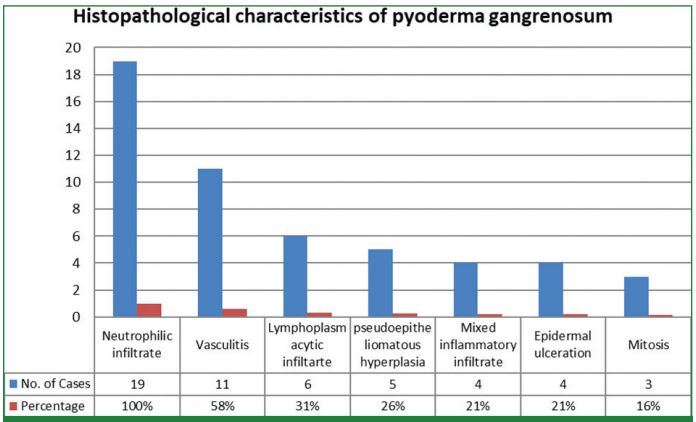
[Table/Fig-5]: Pyoderma Gangrenosum (PG) -Neutrophilic infiltration in epidermis (40x H&E Stain).

(10.52%), hypothyroidism and bacterial infection in 1 case each (5.2%). Some of the cases show more than one co-morbidity.

- One patient developed cutaneous squamous cell carcinoma after three years of disease onset.



[Table/Fig-6]: Pyoderma Gangrenosum (PG): Neutrophilic infiltration at the edge of the ulcer (10x H&E Stain).



[Table/Fig-7]: Histolopathological characteristics of Pyoderma Gangrenosum (PG).

DISCUSSION

Pyoderma Gangrenosum (PG) was first described by Brocq in 1916 as "phagedenismegeometrique" and later named by Brunsting et al., and they considered PG to be the dissemination of a distant focus of infection (i.e., the bowel in ulcerative colitis or lungs in empyema). Presently PG is considered a reactive inflammatory dermatosis [1,8].

PG clinically can mimic other cutaneous ulcerative disease such as vaso-occlusive and venous disease, infections, vasculitis, lymphoma, leukaemia, other neutrophilic dermatosis such as atypical sweet's syndrome, Behcet's disease. The histopathological distinction of PG from other ulcerative diseases is by neutrophilic infiltrate at the edge of ulcer. According to previous diagnostic criteria proposed by Su WP et.al. and Von Den Driesch P, mainly depends on recognition of the evolving clinical features [Table/Fig-8,9] [9,10]. In Su WP et al., criteria, major criteria for diagnosis of PG is rapid progression of a painful necrotic cutaneous ulcer with irregular, violaceous, undermined border and exclusion of other causes of cutaneous ulceration that will lead to overdiagnosis as it is also present in other ulcerative disorder. But more recently Maverakis E et al., proposed a new criteria [Table/Fig-10], it include histopathology of skin biopsy from edge of ulcer showing neutrophilic infiltration as a major criteria which is yet to be widely adopted [6].

Histological findings can be variable among different variants of PG [Table/Fig-11] [11]. Initially, lesions of PG presents as a papulopustule that microscopically has a changes of folliculities involving a pilosebaceous unit with dense neutrophilic infiltrate. The perifollicular dermis frequently contains keratinous debris, hair shaft fragments,

Major criteria	Minor criteria
Occurrence of a primary sterile, chronic ulceration(s) typically with violaceous undermined borders.	Histology from the borders of the ulceration; neutrophil rich infiltration of the dermis with signs for vasculitis and deposits of immunoglobulins and/or complement factors in the vessels.
Exclusion of relevant differential diagnosis (pyoderma, arterial/ venous ulcers, ulcers of leukocytoclastic vasculitis).	Presence of relevant associated disease.
	Response to treatment with systemic immunosuppressive therapy. Little or no response to conventional external ulcer therapy.

[Table/Fig-8]: Diagnostic criteria (Von Den Driesch P 1997) [10].

Major criteria	Minor criteria
Rapid progression of painful, necrotic cutaneous ulcer with an irregular violaceous and undermined border	History suggestive of pathergy or clinical finding of cribriform scarring
Other causes of cutaneous ulceration have been excluded	Systemic disease associated with PG
	Histopathological findings (sterile dermal neutrophilia±mixed inflammation±lymphocytic vasculitis
	Treatment response (rapid response to systemic steroid therapy)

[Table/Fig-9]: Diagnostic criteria (Su WP et al., 2004) [9].

Major criteria
Biopsy of ulcer edge demonstrating neutrophilic infiltrate
Minor criteria
Exclusion of infection
Pathergy
History of inflammatory bowel disease or inflammatory arthritis
History of papule, pustule or vesicle ulcerating within 4 days of appearing
Peripheral erythema, undermining border and tenderness at ulceration site
Multiple ulcerations at least one on anterior lower leg
Cribriform or 'wrinkled paper' scar at healed ulcer sites
Decreased ulcer size within one month of initiating immunosuppressive medications

[Table/Fig-10]: Improved diagnostic tool for Pyoderma Gangrenosum (PG) (Maverkis criteria) [6].

Clinical types	Histopathological features
Ulcerative	Oedema, neutrophilia, secondary lymphocytic vasculitis
Bullous	Epidermal necrosis with neutrophilia, subepidermal bulla
Pustular	Epidermal and dermal neutrophilia
Vegetative	Neutrophilic and eosinophilic histiocytic mixed infiltrate, intra and subepidermal granuloma formation

[Table/Fig-11]: Histopathological characteristics of Pyoderma Gangrenosum (PG) [11].

squames and dense aggregation of neutrophils. Intraepidermal and subcorneal collection of neutrophils also seen. Frequently adjacent blood vessels show changes of leukocytoclastic vasculitis like neutrophils within and around disrupted blood vessels, extravasated red blood cells and necrosis of endothelial cells. Pustule evolves into noduloplaque that microscopically has changes of suppurative granulomatous dermatitis and panniculitis. Massive papillary dermal oedema with epidermal neutrophilic abscess and spongiosis is seen. Regressing lesion have a mononuclear cell infiltrates of lymphocytes and histiocytes and finally a fibroplasia [12].

PG may occur at any age but in this study maximum patients were found between 15 to 50 years (78.9%) with a male predominance [13], classical ulcerative form is most common (63.15%) but other clinical variants like plaque, bullae and vegetative are rare. Classical ulcerative form can occur at any site of the body but most

Author, year and type of study		Hurwitz RM and Haseman JH, 1991 (Case Series) [16]	MJ Ye and Ye JM, 2014 (Case Series) [17]	Chakiri R et al., 2020 (Case Series) [18]	Present study
Place of study		St. Vincent hospital and health care center, indianapolis, indiana	Western Hospital, Footscray, VIC 3011, Australia	University Hospital Hassan II, Fez, Morocco	AMC MET Medical College and L.G. Hospital, Ahmedabad, Gujarat, India
No. of patients		06	23	14	19
No. of male patients (%)		03 (50%)	16 (69.56%)	09 (64.28%)	13 (68.42%)
No. of female patients (%)		03 (50%)	07 (30.43%)	05 (35.71%)	06 (31.58%)
Mean age of subjects (years)		46	62.8	40	30.5
Site as lower limb (%)		04 (66.66%)	13 (56.52%)	12 (85.71%)	17 (89.4%)
Associated systemic disease (%)		05 (83.33%)	11 (47.8%)	06 (42.85%)	09 (47.36%)
Histological features	Neutrophilic infiltration (%)	06 (100%)	12 (80%)	14 (100%)	19 (100%)
	Vasculitis (%)	01 (16.66%)	01 (6.6%)	04 (28.57%)	11 (57.89%)
	Lympho-plasmacyticin filtration (%)	01 (16.66%)	06 (40%)	05 (35.71%)	06 (31.57%)

[Table/Fig-12]: Comparison of cases of Pyoderma Gangrenosum (PG) with previous published studies.
No.: Number

commonly it is found in lower extremities (83.33%). The reason for this specific location is not known [14]. Binus AM et al., found that almost one-third patients had co-morbid conditions such as diabetes and peripheral vascular disease, this two diseases might play role in development of PG and worsen the local healing [15]. Neutrophilic infiltration is present in all cases, which is comparable to other studies of Hurwitz RM and Haseman JH, and Chakiri R et al., [Table/Fig-12] [16-18]. Chakiri R et al., found that ulcers of classical subtype favor the lower extremities in upto 85.7% of patients and disease association in 42.85%, which is comparable to the present study. Associated disease at diagnosis of PG were found in 9 cases (47.36%) [18].

Investigations are necessary to determine treatable, systemic, associated diseases [19]. Aetiology of PG is not well understood; therefore, no specific therapy is available. Aim of treatment is to reduce pain and promote wound healing by reducing inflammation by anti-inflammatory and immunosuppressive agents to improve quality of life of patients of PG. Immunosuppressive therapy is the mainstay in the treatment of PG. Treatment of underlying disease aid in healing. Because of rarity of PG and ethical consideration, there is no placebo controlled trials in treatment of PG. Local therapy- wet compresses with saline and alginate dressings are useful. Topical agents such as tacrolimus, potent corticosteroids and cyclosporine are used [20]. Phenytoin solution 2% is beneficial. Systemic corticosteroids therapy is the most effective treatment of acute, rapidly progressive form. A high dose of prednisolone or pulse therapy with dexamethasone is useful in resistant disease [21]. Cyclosporine as an immunosuppressant is useful in steroid resistant PG. Sulfa drugs may be used as a steroid sparing agent. Recently, use of Tumor Necrosis Factor (TNF)-alpha blockers and other injectable biologics are successful. Infliximab and adalimumab are also effective in treatment of PG [22].

PG is a rare disorder and recruiting large number of patient is difficult. Thus, multicenter studies with large number of patients are required for confirmation of results of study. Patient recruitment was done only by Dermatology Department which was a selection bias. Thus, involvement of other specialists is also required.

CONCLUSION(S)

Pyoderma Gangrenosum (PG) is a unique disease in terms of clinical presentation and histopathology. It has variable course from its origin and during its progression. Ulcerative PG is the most common type of PG. More than half of the patients had associated systemic disease and positive pathergy test. PG should be regarded as a syndrome that presents with a spectrum of clinical and histopathological features that are clear, distinct and exhibit characteristic microscopic findings throughout its evolution. In Indian patients, Maverakis criteria should be used, in clinically suspected cases of PG with its histopathological characteristics and excluding other causes of infections.

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