PYODERMA GANGRENOSUM (A CASE REPORT)

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Pyoderma gangrenosum is a rare cutaneous disease of unknown aetiology begins with cutaneous abscesses that breakdown forming chronic destructive inflammatory ulcers.

Various names have been used for the same clinical condition, such as Burrowing Phagedenic ulcer of Boeq; Chronic undermining burrowing ulcer of Meleney; Post-operative serpiginous progressive ulcer of Cullen.

Ulcer usually starts as a pustule or an insect-bite or a tender nodule which breaks. Ulcer usually increases in size upto 10 cms., or more in diameter and may persist for a few weeks to many years. The ulcer may be single or multiple and usually occur on legs, thighs, buttocks or trunk. Males and females are equally affected, and most cases are often found in adults. Many a time healing occurs spontaneously leaving atrophic scarring.

Ulcer borders are slightly raised, oedematous and boggy, and usually have distinct bluish purple colour. Beyond the border, the erythema extends merging with normal skin. The centre of ulcer is covered by mucopurulent exudate which when removed reveals a clear granulomatous-base. Margins are polycyclic or serpigenous and rolled under and undermined. It spreads peripherally with tendency to heal centrally.

Histopathology is non-specific and not diagnostic. In the region of ulcer the

Dept. of Dermatology, Gandhi Medical College, Hyderabad Received for Publication on 3-6-1971 epidermis is absent. Upper dermis shows necrosis, permeated by acute inflammatory infiltrate. Further down, the infiltrate is chronic, consisting of lymphocytes, neutrophils, plasma-cells, histiocytes and fibroblasts. The blood vessels are increased with endothelial proliferation. The epidermis at the edge of ulcer shows pseudocarcinomatous hyper plasia with intra-epithelial abscess formation. The aetiology is completely unknown.

Many types of organisms including pathogenic-cocci, gram negative rods, which are normal resident flora, or common transients have been isolated from the ulcer. Bacterial allergy has been postulated and remains a possibility. Defective immune response may be an essential factor, as occasionally the cases are associated with hypoprotenemia, but in many, there may be no abnormality in serum proteins. The beneficial effect of internal administration of corticosteroids suggest that the disease may be due to antigen antibody reaction rather than due to infection.

The disease has been reported in association with ulcerative colitis or rheumatoid arthritis or lesions like lungabscess or empyema. They may run a course parallel to the activity of internal disorder if associated with. Malnutrition with hypogammaglobulinaemia may sometimes be associated with, but extensive and persistent lesions are found in healthy people also.

History:

R, a 35 year old Hindu male was admitted in the skin ward of Gandhi

Hospital, Secunderabad, Andhra Pradesh, on 7-11-1969 for multiple ulcers on the skin of 6 months duration. He gave past history of burns 8 years ago on left-axilla, left-side of chest, left-arm and right fore-arm. The right fore-arm was amputated due to severe burns but the remaining burns healed in three months time with scarring. Since three years he has been suffering from attacks of pain in Rt.hypochondrium and right iliac fossa which used to subside after some medication. There was no history of dysentry. 6 months ago a small "boil" developed on the right-hip which gradually increased in size and formed a large ulcer. After 3 weeks a similar "boil" developed on the left side of the chest which again turned into an ulcer.

Later similar ulcers developed on rightthigh, right-leg, left-leg, neck, back and buttocks.

On Examination:—He was an anxious looking debilitated young man with multiple necrotic ulcers on the skin. There were about eight ulcers varying in size from 1 cm., to 10 cms., with irregular, ragged bluish-red edges and the base covered with yellowish slough which when removed, there was profuse bleeding granulation. (Fig. 1 & 2). There were also few pustules at the margins of the ulcer. During his stay in the Hospital it was observed that the ulcers started as pustules every time. Systemic examination did not reveal any gross abnormality.

Investigations done:

Stools - No Ova - No cysts.
Urine for sugar & albumin - Negative. Total Leucocyte-count 10,182/cu.mm., Polymorphs - 72%. Lymphocytes - 28%.
Fasting Blood sugar - 75mgm.%.
Pus for fungus - Negative.
Total proteins - 6.5 grams%. Albumin - 4 gram%. Globulin - 2.5 gram%.
Pus for AFB – Negative M.L. Pus for Gram's stain – Negative.
Pus for culture - Staph coagulase positive grown. Sensitive to Furacin, Chloromycetin and Streptomycetin. Resistant to Penicillin Orisal and Tetracyclin.
V.D.R.L Negative. Blood group - B.
Pus for culture - Staph coagulase Positive grown. Sensitive to Streptomycetin, Chloromycetin, Polycyclin, Synthomycin, Rovamycin, Resistant to Penicillin & Orisal.
Occult Blood test - Positive.
X-ray abdomen - Plain - N.A.D.
X-ray of chest - N.A.D.
Pus for culture Deep Mycosis - Negative.



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16 170	Serum Electrolytes - Sodium - 339 mgm%. Potassium - 19mgm%. Chlorides - 560 mgm%.
24 170	Vanden Burgh - Negative. Bilirubin - 0.8 mg%. T.T.T 3 Units. Serum Alk Posphatase - 8.6 K.A. Units per 100 ml. Serum Amylase - 8 Units/1 ml.
4- 4-70	Bleeding Time 2 min., 30 sec., Clotting Time 3 min. 45 sec.,
Biopsy:	(I.P. No. 10013) shows hyperkeratosis – collection of inflammatory cells in the corium. No epitheloid or Langhans giant cells seen. Diag: Non-specific dermatitis.
31- 1-70	Barium meal and Barium enema - No evidence of ulcerative colitis

Treatment given:

Date	Treatment given	Response
1–11–69 to 15–11–69	Local: 1% G.V. Lotion on superficial ulcers and glycerene Magsulphas dressing to deep ulcers. System:—Streptopenicillin daily B-complex tabs Vit C and Prepaline cap., orally. Inj., B-complex twice/week.	Condition same except ulcers appear cleaner but fresh ulcers appearing at margins.
15-11-69 to 22-11-69	Local: Same therapy. Systemic:—Supportive therapy same. Resteclin cap., 6th hourly. 300 cc., Blood-transfusion given.	No improvement in ulcers.
22-11-69 to 8-12-69	Restectin continued, Betnelan tab- 1-6th hourly. Local & supportive therapy - same.	Discharge from ulcers red- uced. Ulcer shows tendency to heal. Fresh ulcers did not appear.
8-12-69 to 14-12-69	Resteclin cap., stopped. Betnalan tabs., continued.	Pain of ulcers and discharge from ulcers increased.
14-12-69	Resteclin re-started.	
17-12-69 to 28-12-69	C/o Pain Rt. Hypochondrium Lumb ++. Tender? Amoebic Hepatitis. Nivaquin 1 tab. TDS for 10 days and Liver extract 2 cc., I.M. Betnalan tabs., and Resteclin cap. contd.	Pain of Hypochondrium- subsided.
27 1-70	Resteclin and Betnelan stopped after tapering the dose.	General Health improved. Ulcers healing but few les- ions appearing on old ones.

3- 2-70	300 cc., blood transfusion given (B group.)	Bleeding ulcers increased. Bleeding & clotting time normal.
6- 2-70	Pain Rt. Iliac fossa. Surgeon's opinion T.B. Caecum. Barium enema and Meal normal.	
9- 2-70 to 20- 2-70	Pain and discharge from ulcers increased. Ledermycin 300 mg. B.D. and Kenacort (4 mg.,) 1 B.D.	Ulcers increased in number and severity.
24- 3-70 to 1- 4-70	Transferred to Surgical ward for investigations—Pain Rt. Iliac fosagiven Streptomycin and INH.	No improvement of ulcers - Pain abdomen - subsided.
26- 4-70 to 25- 5-70	Transferred back. Resteclin 6th hrly. Prednisolon (5 mg) t. d. s. B.Complex 2 cc, (IM) and Durabolin 1 amp, I.M. Sterile vaseline dressing daily.	Ulcers almost healed. General condition improved. No fresh lesions.
	Blood transfusion -300 cc., on 6-4-70	
	Blood transfusion -200 cc., on 11-4-70	
	Blood transfusion -150 cc., on 25-4-70	

25-5-70 Patient was discharged with an advice to continue prednisolone 1 tabtwice daily, G.V. lotion for healing ulcers and Multivit tabs., and calcium tabs.

Patient was reviewed after 3 months. All the ulcers have healed completely with atrophic scars. General condition is good. Advised to continue prednisolone 1 tab., daily and Multivit and Calcium tablets. Patient is able to do his daily duties i.e., Ricksha pulling.

Acknowledgement:

Our thanks are due to Dr. N. R. V. Swami, Superintendent, Gandhi Hospital, Secunderabad-A.P. for permission to publish the case.

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