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PYODERMA GANGRENOSUM

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Summary

A case of pyoderma gangrenosum and ulcerative colitis is reported. The case shows that it is mandatory to control the bowel disease to achieve healing of skin lesions.

Pyoderma gangrenosum is an association of the inflammatory bowel disease, ulcerative colitis. Though relatively less common than other skin manifestations of ulcerative colitis, pyoderma gangrenosum is the most serious and distressing complication. About 1-10% of patients with active ulcerative colitis might suffer from pyoderma gangrenosum[1].

A clear-cut parallel relationship of the skin and bowel disease was seen in our case. Healing of skin lesions occurred only after successful control of the colonic disease.

Case Report

A fifty year old female patient with painful ulcers on legs was referred to the Skin Department of the Kasturba Medical College Hospital from a nearby hospital where she had been admitted about two months earlier for treatment of loose motions with blood and mucous. While she was getting treatment for this she developed generalised itching and ulcers on legs. Diarrhoea was controlled to some extent but ulcers on legs did not resolve. Patient gave history of a similar attack of bloody diarrhoea two years before, which had subsided with native medicine. There were no skin lesions at that time.

Examination revealed that the patient was suffering from scabies. She also showed multiple painful, heavily crusted. oozy ulcers on both shins. She looked undernourished and had a III degree prolapse of uterus.

The abnormalities found on routine investigations were

1. Microcytic hypochromic anaemia (Hb — 6G%).
2. Hookworm infestation.
3. Raised E. S. R. (30mm in 1st hr.) and
4. Reversal of albumin - globulin ratio.

The hookworm infestation and scabies were successfully treated. Oral erythromycin along with potassium permanganate compresses for the ulcers was given. The ulcers showed no tendency to heal despite this and repeated blood transfusions which built up her Hb to 12G%. She developed a recurrence of bloody diarrhoea (10-14/day). The ulcers started spreading very rapidly. New lesions

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appeared after trivial trauma like a venepuncture. The initial lesions were erythematous nodules or pustules which broke down to ulcers that spread rapidly. The lips of the ulcers showed erythema and oedema (Fig. 1).

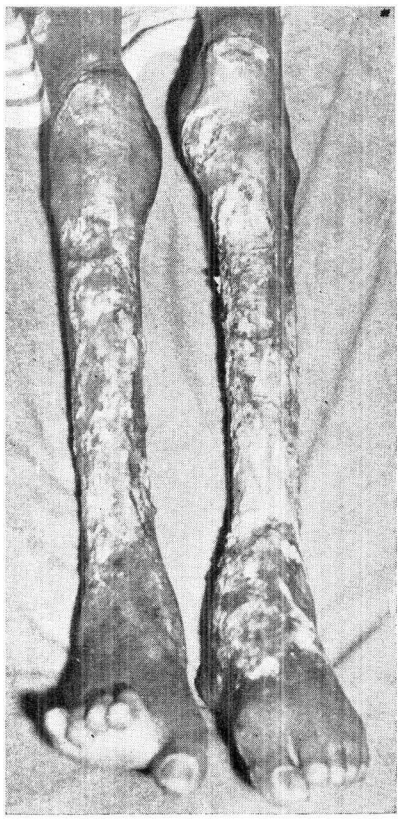

Fig. 1 Extensive ulceration on legs at the height of disease.

Stool culture grew E. coli and pus from the ulcers, Pseudomonas, Proteus, Staph aureus and E. Coli. Sulphaguanidine, aminosidine and gentamycin did not check the progress of the ulcers. New lesions appeared on thighs and forearms.

Sigmoidoscopy and rectal biopsy & biopsy from the ulcers on skin were performed. The histopathology was suggestive of ulcerative colitis. A barium enema revealed loss of haustrations and lead pipe appearance of colon; features of ulcerative colitis (Fig. 2,3).

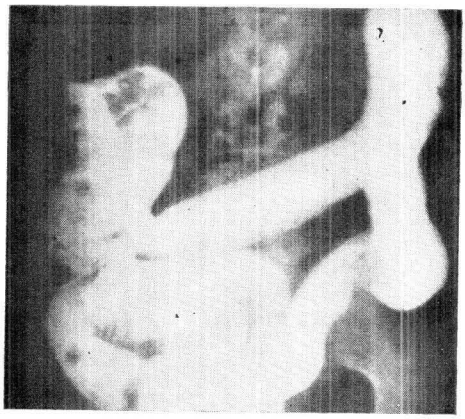

Fig. 2 Barium enema showing loss of haustrations with slight shortening of the colon. There is fuzziness along the contour of the transverse and the descending colon caused by ulcerations.

Sulphasalazine (3g/day) for two weeks had no effect on the loose motions or on skin lesions. With a combination of prednisolone (45mg/day) and sulphasalazine both the diarrhoea and skin lesion

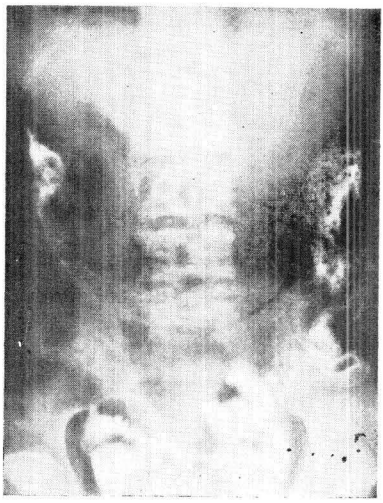

Fig. 3 The evacuation film shows numerous inflammatory polyps in the transverse and descending colon.

responded dramatically (Fig. 4). The ulcers healed with whitish artophic scars and patient passed normal stools 2–3 times a day. There was no recurrence

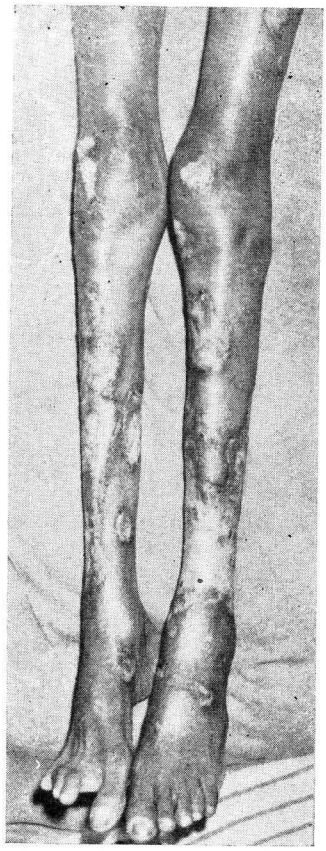

Fig. 4 Shows healed lesions with whitish atophic scars.

of ulcers on a maintenance dosage of 15 mg of oral prednisolone per day. Two months after healing occurred the atrophic scars developed into keloids (Fig. 5).

Comments :

Various skin manifestations are on record as associations of ulcerative colitis. It is observed that 3–13% of patients with this bowel disease have lesions[2] which include aphthae, maculopapular eruptions, purpura, erythema multiforme, erythema nodosum and pyoderma gangrenosum.

The initial lesion in pyoderma gangrenosum is a pustule or a reddish purple nodule which progresses to ulceration. Lower extremities and lower half of trunk are the most common sites of involvement though no area is exempt. Even trivial trauma can initiate lesions in the hypersensitive skin of the patients; hypersensitivity to iodides and bromides may also occur. Atrophic scarring results when the lesions finally heal with treatment of ulcerative colitis.

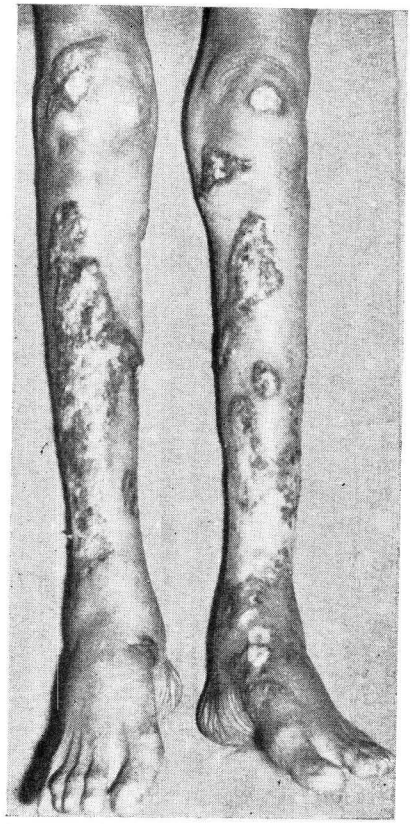

Fig 5 Keloidal response two months after complete healing of ulcers.

It is not invariable that all patients with pyoderma gangrenosum suffer from ulcerative colitis. But it is certainly necessary to have a thorough look at the gut macroscopically, microscopically and radiologically since over half of the patients with this type of progressive

ulcers on skin have ulcerative colitis. The mechanism by which pyoderma gangrenosum lesions are produced is obscure but a Shwartzman phenomenon has been proposed by Rostenberg[3]. The abundance of a mixture of micro-organisms in the ulcers does not incriminate them as the culprits and antibiotics directed towards eliminating these bacteria make little difference to progress of skin lesions as was seen in our case. Diagnosis of the bowel disease and appropriate therapy to control the same are mandatory to achieve healing of skin lesions. A combination of intralesional corticosteroids and oral diamino diphenyl sulphone is an addition[4] to the other established forms of therapy.

The development of Keloids from the scars of pyoderma gangrenosum lesions is perhaps very rare. This may be related to the tendency for Keloid formation in this patient's family as shown by large Keloids at BCG injection sites in two of her children.

Acknowledgement

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