

## **Ciprofloxacin induced pyoderma gangrenosum**

Sir,

Ciprofloxacin has been recognized to cause various cutaneous adverse reactions like fixed drug eruption, erythema multiforme, Stevens Johnson syndrome, toxic epidermal necrolysis, hypersensitivity leukocytoclastic vasculitis, bullous pemphigoid

and photosensitivity. Pyoderma gangrenosum (PG) is a rare, noninfectious neutrophilic dermatosis usually associated with an underlying disease, most commonly inflammatory bowel disease, rheumatic, haematological disease and/or malignancy. Recently, there have been reports of drug induced PG by compounds such as propylthiouracil,<sup>[1]</sup> pegfilgastrim,<sup>[2]</sup> and gefinib.<sup>[3]</sup> We report a case of PG secondary to ciprofloxacin.

A 22 year old male presented with joint pain and purpuric rash on the trunk and both upper and lower limbs of 3 days duration. He has a history of fever for which he was started with tablet ciprofloxacin 500 mg twice daily on day 1. Two days after taking the drug he developed joint pain of the lower limbs and rash on the trunk and limbs. The rash started as purpura and progressed to form hemorrhagic blisters, which ruptured to form ulcers over a span of 7 days. On examination, he had palpable purpura and hemorrhagic blisters and ulcers over the upper and lower limbs [Figure 1]. Ulcers were of varying sizes ranging from 0.5 x 0.5 to 7 x 5 cm with undermined edges and hemorrhagic crusts [Figure 2]. Ciprofloxacin was stopped on the fourth day after which he did not develop new lesions. Blood investigations revealed a low platelet count of 45,000/ $\mu$ l (normal 150-400x10<sup>3</sup>/ $\mu$ l), elevated renal function tests (urea – 193 mg/dl, creatinine – 2.23 mg/dl) and liver function tests (bilirubin total – 2.3, serum glutamic pyruvic transaminase

(SGPT) - 107, serum glutamic oxaloacetic transaminase (SGOT) 51, alkaline phosphatase (ALP) – 113). Urine routine and ultrasound abdomen revealed normal results. Platelet count and renal function test results returned to normal levels 3 days after stopping ciprofloxacin. Histopathological evaluation of a specimen from the ulcer showed perivascular infiltrate of neutrophils and few lymphocytes in the superficial and deep dermis [Figure 3]. ANA, double stranded DNA (ds DNA) and rheumatoid factor tests were negative. Immunofluorescence from the pupuric lesions was negative. A diagnosis of ciprofloxacin induced pyoderma gangrenosum was made based on the clinical, histopathological and laboratory findings. The patient was treated with oral steroids prednisolone 30 mg / day tapered over a period of two months and salazopyrine 500 mg thrice daily x 2 months. Ulcers healed completely in two months [Figure 4] with no incidence of recurrence during the follow up period of one year.

Pyoderma gangrenosum is a rare noninfectious neutrophilic dermatosis. It starts as sterile pustules that rapidly progress to painful ulcers of variable depth and size with undermined violaceous borders. In many cases PG is associated with an underlying disease, most commonly inflammatory bowel disease, rheumatic, hematological disease and/or malignancy. Recently there have been reports of drug induced PG by



**Figure 1:** Purpura and hemorrhagic blisters and ulcers on the right leg

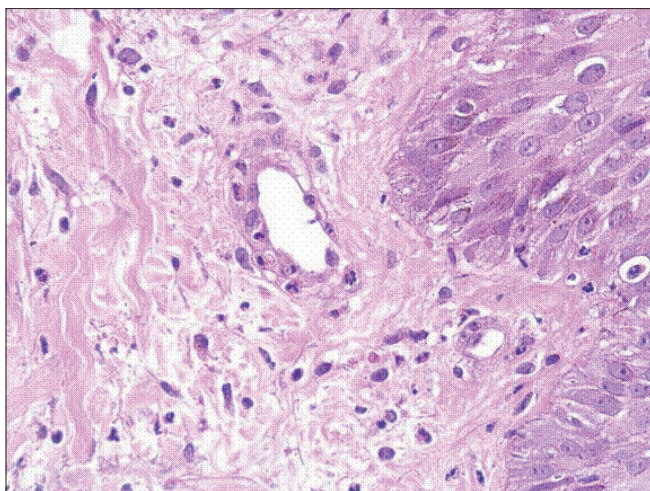


**Figure 2:** Ulcer with undermined edges and hemorrhagic crusts on left elbow

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**Figure 3:** Histopathological evaluation of a specimen from the ulcer showing perivascular infiltrate of neutrophils and few lymphocytes in the superficial and deep dermis under H and E, ×40



**Figure 4:** Healed ulcer over the both the elbows after two months of stopping ciprofloxacin and medical intervention.

propylthiouracil,<sup>[1]</sup> pegfilgrastim – a granulocyte-stimulating factor,<sup>[2]</sup> and gefitinib – an inhibitor of epidermal growth factor receptor.<sup>[3]</sup> Diagnosis of PG is based on history of an underlying disease, typical clinical presentation, histopathology, and exclusion of other diseases that would lead to a similar appearance. Our patient presented with renal dysfunction, thrombocytopenia, purpuric rash and pyoderma gangrenosum on the limbs following ciprofloxacin. Renal dysfunction and thrombocytopenia improved after withdrawing ciprofloxacin. He was treated with oral steroids and salazopyrine for PG. The temporal correlation with the drug, clinical appearance of the ulcers, histopathological findings, exclusion of other conditions and absence of new lesions after stopping ciprofloxacin led to a diagnosis of PG secondary to ciprofloxacin. We present our case as there are not many reports of PG secondary to ciprofloxacin mentioned in literature.

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