Corticosteroids and monocytosis
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Abstract
Although the association between steroid administration and neutrophilia is well known, the association with monocytosis is not as common and the mechanism less clear. This report illustrates the association and provides an update of postulated mechanisms and clinical significance.

Case report
An 83-year-old man was admitted from a rest home for fatigue, retching, nausea, and weight loss of 18 kg in the previous 6 months. He was on prednisone 5 mg oral daily and methotrexate 15 mg oral weekly for polyarteritis nodosa and rheumatoid arthritis. Other medical problems included obesity, hypertension, ex-smoker with 50 pack year history and stopped age 70, COPD, dermatitis, osteoporosis, chronic renal impairment and dementia. Other medications at admission included alendronate 70 mg weekly, omeprazole 20 mg daily, folic acid, and paracetamol as required.

He had no known drug allergies. He was not taking any other over-the-counter medications or complementary medications. Gastroscopy showed severe non-reflux oesophagitis consistent with chemical ulcer secondary to alendronate. There was no evidence of malignancy on CT chest abdomen pelvis.

A severe red itchy rash developed on the patient’s back during the admission. On dermatology review this was thought to be acantholytic dermatosis (Grover disease). Skin biopsy was consistent with either a fixed drug reaction or, more likely, urticated dermatitis. Prednisone 40 mg oral daily was started to control the rash. The patient developed a neutrophilia with accompanying monocytosis up to 10.05 × 10^9/L—see Figure 1.

There was no fever or other evidence of systemic infection, including multiple negative peripheral blood cultures. Blood film examination showed mild granulocyte left shift and neutrophil hypersegmentation, which could be explained by methotrexate, but not toxic change. The monocytes had normal morphology and there were no dysplastic features or abnormal cells.

The skin rash resolved with the oral steroid and following tapering of steroid therapy to his admission dose, total white cell count, neutrophil count and monocyte count returned to baseline.

The patient continued topical steroids and methotrexate. After discharge his rash remained well controlled.
Discussion

The association between corticosteroid administration and neutrophilia is well documented, although accounts vary on the degree and mechanism of the effect. The consensus is that corticosteroid increases neutrophils through increased bone marrow production and release, redistribution of the marginated and circulating neutrophil pools and reduced neutrophil movement into inflamed tissues. The degree of granulocyte left shift and the presence of toxic granulation on blood film examination may distinguish between neutrophilia caused by infection and corticosteroids.

Although monocytosis may accompany the neutrophilia in corticosteroid use, this is less well documented and the mechanism is less clear. Corticosteroid has been shown to facilitate Interleukin-1 and Granulocyte-Monocyte Colony Stimulating Factor (GM-CSF) in inducing Colony Forming Unit – Granulocytes-Monocytes (CFU-GM) proliferation, which can increase monocyte production. Monocyte number, however, has been observed to drop following administration of high dose corticosteroids, in contrast to the granulocytosis and overall leukocytosis. Monocyte production and release was also suppressed in mice injected with hydrocortisone.

The production and kinetics of the monocyte population, in both directions, are probably influenced by corticosteroid administration and also by the disease processes themselves.
In our patient’s case, the rapid increase in leukocyte numbers with delayed but parallel increase in monocytes, their resolution on corticosteroid withdrawal, and the negative investigation findings for infection, suggest the monocytosis is secondary to prednisone use.

Recognition of this glucocorticoid side effect is useful to avoid unnecessary investigation and anxiety.

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**References:**