etanercept, she experienced aggravation of the psoriasis and psoriatic arthritis but refused other treatments except nonsteroidal anti-inflammatory drugs and topical betamethasone/calcipotriol.

Tumor necrosis factor (TNF)-alfa receptor blockers are great advances in the treatment of psoriasis and psoriatic arthritis. The immune regulation of TNF-alfa has been highly controversial regarding its risk in carcinogenesis. Kimball et al[4] recently reported that there was little difference in rates of lymphomas among different treatment methods such as nonbiologic systemics, etanercept, other TNF-alfa receptor blockers, and phototherapy. To our knowledge, no reports have shown an increased risk of lymphoma in patients using etanercept.[5] However, in patients with chronic inflammatory disease and a history of long-term use of immunosuppressive agents such as MTX or cyclosporine, the synergetic effect of TNF-alfa inhibitors and conventional immune modulators may increase the risk of developing malignancy. As TNF-alfa inhibitor use becomes more popular, this case suggests the need for close observation to identify the potential long-term risks associated with chronic immune modulation.

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Funding sources: None.

Conflicts of interest: None declared.

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http://dx.doi.org/10.1016/j.jaad.2014.05.068

Peripheral bands in the setting of drug hypersensitivity syndrome

To the Editor: Immature neutrophils in the peripheral circulation (bandemia) are an infrequently reported finding in drug hypersensitivity syndrome (DHS).[1-3] We report a case of DHS with bandemia that resolved after withdrawal of the offending medication and initiation of prednisolone.

A previously healthy, school-aged girl was admitted to the hospital with a 2-day history of fever, facial edema, cervical lymphadenopathy, and a morbilliform eruption that started on the face and progressed caudally. The rash and fever developed after 6 days of trimethoprim-sulfamethoxazole (TMP-SMX) therapy for an uncomplicated urinary tract infection. She denied ocular pain, dysphagia, odynophagia, dysuria, and painful skin. Physical examination revealed an ill-appearing African American child with a temperature of 39.9°C, mild tachycardia, and tachypnea. A blanching morbilliform eruption was noted on her face, trunk, and extremities with significant facial edema and subcentimeter, nontender cervical lymphadenopathy. Mucosal surfaces were spared. Laboratory values are listed in Table I. Blood culture revealed negative findings, as did repeated urinalysis and urine culture. Human herpesvirus-6 quantitative polymerase chain reaction was positive at 300 copies/μL. Epstein-Barr virus serologies showed no evidence of current or previous infection.

Although the patient's eosinophils were within normal reference range upon admission, the constellation of clinical and laboratory findings (morbilliform eruption with facial edema, fever, elevated liver enzymes and creatinine, and atypical lymphocytosis after administration of TMP-SMX) was suggestive of DHS. TMP-SMX was immediately discontinued, and prednisolone (1 mg/kg/d) was initiated, in addition to acetaminophen and maintenance intravenous fluids. After 1 day of therapy, her eruption had improved considerably, with normalization of her vital signs. Her repeated complete blood cell count on hospital day 2 demonstrated reduction of peripheral bands to 3%, normalization of serum

Table I. Key laboratory values on admission

Laboratory component (normal value)	Values on admission	Values 1 d after discontinuation of 16-d corticosteroid taper	Values after a total 31 d of corticosteroid treatment
White blood cell count (4.27-11.40 $\times$ $10^3/\mu L$)	4.9	8.1	11.6
Bands (0%)	16	N/A	N/A
Eosinophils (0.0%-4.0%)	2.0	0.0	1.0
Atypical lymphocytes (2%-3%)	8.7	N/A	N/A
Platelets (199-367 $\times$ $10^3/\mu L$)	138	639	215
Hemoglobin (11.5-15.5 g/dL)	10.6	11.9	12.9
Urea nitrogen (5-17 mg/dL)	7	15	15
Creatinine (0.1-0.5 mg/dL)	0.8	0.5	0.60
Alkaline phosphatase (175-420 U/L)	111	174	151
Alanine aminotransferase (10-35 U/L)	44	194	42
Aspartate aminotransferase (15-40 U/L)	67	297	18
Gamma-glutamyltransferase (11-21 U/L)	25	30	17
C-reactive protein (0.0-0.9 mg/dL)	8.1	N/A	N/A

N/A, Not available.

creatinine, and improvement in liver function test results. Within 1 day of discontinuing the 16-day taper of prednisolone, however, her rash and fever recurred, and laboratory evaluation revealed elevated liver enzymes (Table I). Prednisolone (1 mg/kg/d) was reinitiated with subsequent normalization of laboratory values (Table I). The prednisolone was tapered after an additional 3 weeks of therapy with no evidence of recurrence.

Although the presence of bandemia is frequently seen in systemic infection as a result of bone-marrow activation, it is rarely reported in DHS.[1-3] Our patient's rapid resolution with discontinuation of TMP-SMX and initiation of prednisolone strongly suggests that the bandemia was caused by DHS rather than infection.

Various mechanisms causing blood dyscrasias in DHS include cytotoxic antibody-mediated destruction, reversible inhibition of stem cell activity, cessation of myeloid maturation, and induction of cytokine-activated macrophages leading to the development of hemophagocytosis syndrome.[3,4] The complex inflammatory response and the resultant cytokine storm, notably tumor necrosis factor-alfa among others, can stimulate the bone marrow to release immature neutrophils.[5]

Recognition of the constellation of clinical findings suggestive of DHS is vital particularly when bandemia is present, as failure to stop the offending medication can lead to exacerbation of the underlying hypersensitivity, with resultant multisystem organ failure. Bands are most commonly associated with infection, but this report confirms that peripheral bands can also be associated with DHS.

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Funding sources: None.

Conflicts of interest: None declared.

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http://dx.doi.org/10.1016/j.jaad.2014.06.007