SHORT COMMUNICATIONS

Serum Sickness Secondary to Fluoxetine

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A 15-year-old female with past medical history significant for generalized anxiety disorder, PTSD, and depression was admitted to the hospital for suicidal ideation. Her psychiatric symptoms were not responsive to sertraline, prompting a cross-taper with fluoxetine 20mg. Three days later, the patient developed an itchy cutaneous eruption associated with new-onset joint pain and swelling in her hands, wrists, and shoulders. Skin examination was notable for blanchable, confluent, polycyclic, erythematous patches and plaques consistent with urticaria on the chest, abdomen, back (Figure 1), upper extremities (Figure 2), and thighs, although sparing face and neck. Lymph node examination was normal. Laboratory testing revealed a normal complete blood count (CBC), comprehensive metabolic panel (CMP), and erythrocyte sedimentation rate (ESR), but an elevated C-reactive protein (CRP) (45 mg/L), low-normal complement C3 (89 mg/dL), and low complement C4 (5 mg/dL). The cutaneous eruption and joint complaints resolved within a week after discontinuation of fluoxetine, and in response to treatment with topical triamcinolone 0.1% ointment. Repeat laboratory testing within two weeks showed that C3 and C4 complement levels had returned to normal (128 and 31 mg/dL, respectively). Given the temporal association with the administration of a new drug and the concurrence of dermatitis, arthralgias, and hypocomplementemia, this was most consistent with a case of serum sickness.

Figure 1. Confluent, polycyclic, blanchable erythematous plaques over the back.
DISCUSSION

Drug-related cutaneous eruptions are common, affecting approximately 2-3% of hospitalized patients, and varying in severity from benign to life-threatening. Serum sickness is a rare, systemic, immunologic condition, often presenting with fever, rash, and joint pain. It is caused by the deposition of immune complexes in blood vessels and other tissues, resulting in complement activation and consumption and ensuing inflammation. A serum sickness-like reaction (SSLR) presents very similarly and spontaneously resolves with withdrawal of the culprit drug. While the mechanism for SSLRs is not well understood, it is thought to be distinct from a true serum sickness and typically does not present with hypocomplementemia, vasculitis, lymphadenopathy, or hepatic and renal dysfunction. A number of drug classes including antibiotics, beta-blockers, and antidepressants have been implicated in SSLRs, with only 3 reported cases associated with fluoxetine. Given the benign clinical course, absence of organ dysfunction, and onset of rash within few days of drug onset, our patient was initially diagnosed with a serum sickness-like reaction secondary to fluoxetine. However, the presence of hypocomplementemia in our patient is highly suggestive of a true serum sickness rather than SSLR, as this is often the distinguishing factor between the two conditions. Furthermore, the resolution of hypocomplementemia within weeks of discontinuation of the culprit drug (fluoxetine) supports immune complex deposition as the underlying etiology. Providers should be aware that rarely serum sickness may occur with administration of non-biologic drugs as was the case with our patient. Serum sickness with fluoxetine, although rare, has been reported. Although our patient was young and relatively healthy, such a reaction has the potential for severe consequences especially in patients with immunosuppression, autoimmune disease, or other significant comorbidities.

Figure 2. Blanchable erythematous patches over the bilateral forearms and dorsal hands.

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