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An Atypical Adverse Drug Event: Bactrim Induced Autoimmune Hemolytic Anemia

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INTRODUCTION

Autoimmune hemolytic anemia is an acquired disease of antibody induced red blood cell (RBC) destruction that occurs acutely and can be life-threatening. Warm thermal-based reactive autoantibodies are predominately involved, however, other etiologies including drug-induced immune hemolytic anemia (DIIHA) are possible. The prevalence of DIIHA is rare affecting 1 in 1 million, but makes up to 10% of all autoimmune hemolytic anemia cases and attributes to a 3.6% mortality rate in 33 months. There are over 150 known medications that cause this pathology, and though rare, should be considered during the workup of an acute hemolytic crisis.

CASE PRESENTATION

A 70-year-old female presented with symptoms of nausea, vomiting, abdominal pain and dizziness, as well as complaints of dark urine and dark stool. On physical exam, she exhibited scleral icterus and diffuse jaundice. She was afebrile and tachycardic, with other vitals within normal limits. Further evaluation revealed the addition of Bactrim and indomethacin as new medications. Timing of symptoms occurred after one day of initiating Bactrim and one week after the start of indomethacin. Pertinent laboratory data included a Hemoglobin of 5.6, total bilirubin greater than 35, Direct Bilirubin 25, LDH greater than 1000, and a low haptoglobin (<7). CT Abdomen/Pelvis revealed mild

splenomegaly and no infection or occult malignancies were suspected. Direct Anti-globin test (DAT) was positive for IgG, CD3; indicative of warm or drug-induced hemolytic anemia. She was stabilized promptly with blood transfusions followed by high dose steroids.

DISCUSSION

Our patient presented with a clinical picture consistent with intravascular hemolytic anemia likely secondary to trimethoprim-sulfamethoxazole and concern for Glucose-6-Phosphate-Dehydrogenase (G6PD) deficiency. Diagnostic efforts revealed the patient to be DAT positive, supporting the diagnosis of a drug-induced process or primary warm type hemolytic anemia. Mainstays of therapy predominately consists of prednisolone and transfusions with IVIG and plasmapheresis for severe or refractory cases. Supportive treatment with drug discontinuation, folic acid, anticoagulation, and medications to suppress the side effects of steroid therapy are also recommended. Given the severity of presentation with correlating mortality of drug-induced hemolytic anemias, decisions to prescribe potentially unnecessary medications should include thought of more than just the common adverse effects.