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Isolated thrombocytopenia due to transient methimazole toxicity in acute ischemic liver failure

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Background: Methimazole is an anti-thyroid medication in the thioamide group that includes propylthiouracil and carbimazole. These medications are well known causes of agranulocytosis (0.1-0.5%). Other blood dyscrasias such as thrombocytopenia and aplastic anemia are rare. Liver dysfunction can interfere with hepatic metabolism of these drugs. We report a case of thrombocytopenia secondary to transiently-reduced hepatic metabolism of methimazole in a patient with temporary ischemic liver damage from cardiogenic shock.

Case Description: A 91-year-old male with history of ischemic cardiomyopathy, ESRD, and hyperthyroidism presented to the ED with vague abdominal pain. Medications included methimazole 5 mg every other day, aspirin, clopidogrel, erythropoietin with HD, and midodrine, which were all continued throughout the hospitalization. On exam, he was hypotensive with cold extremities and crackles. Echocardiogram revealed a decrease in ejection fraction from 50-55% to 5-10%. He was diagnosed with cardiogenic shock and started on dobutamine infusion. Prior to initiation of dobutamine, he had a transient episode of complete AV block causing marked hypotension. The following morning, his liver enzymes were markedly elevated, with AST 1009, ALT 1398, alkaline phosphatase 129, Total-bilirubin 1.69, INR 1.4. There were no physical exam findings of liver disease, and right-upper-quadrant ultrasound was unremarkable. These values gradually returned to normal over the course of a week. Initial platelet count was 131,000-dropping to 91,000 on the first day after hepatic injury. After a HIT score calculation of four, heparin prophylaxis was discontinued. A nadir of 26,000 was reached, with a gradual return to 133,000 that paralleled the recovery of liver enzymes. HIT antibodies were eventually negative.

Conclusion: This case highlights the importance of hepatic metabolism of methimazole and the potential for toxicity occurring secondary to acute liver injury. It is important to be cognizant of not only the well-described agranulocytosis, but also rare idiosyncratic reactions like thrombocytopenia.

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MYC/BCL2 double hit high grade B-cell lymphoma

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Double-hit lymphoma (DHL) has been defined by 2008 WHO as a B-cell lymphoma with MYC/8q24 rearrangement in combination with a translocation involving another gene, such as *BCL2* or *BCL6*. The most common form of DHL has translocations involving MYC and BCL2, also known as MYC/BCL2 DHL. In the past few years, numerous case series of MYC/BCL2 DHL have been reported in the literature. Most cases of MYC/BCL2 DHL morphologically resemble diffuse large B-cell lymphoma (DLBCL) or high grade B-cell lymphoma, not otherwise specified (previous name in 2008 WHO: B cell lymphoma, unclassifiable, with features intermediate between DLBCL and Burkitt lymphoma). These tumors have a germinal center B-cell immuno phenotype but an aggressive clinical course characterized by a high proliferation rate, advanced-stage disease, extra nodal involvement, high International Prognostic Index score and high serum lactate dehydrogenase levels. All tumors have a complex karyotype. Despite a variety of therapeutic approaches that have been used to date, patients with DHL have a poor prognosis. Here, we will discuss the clinicopathologic, immunophenotypic, cytogenetic and prognostic features of MYC/BCL2 DHL and some remaining issues.

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