Clinical Case Study

Hemolytic Anemia Following Attempted Suicide

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CASE DESCRIPTION

A 43-year-old African-American man with a history of hypertension, depression, and chronic alcohol abuse presented to the emergency service of an outside hospital complaining of chest pain. Laboratory testing indicated hepatocellular injury with aspartate aminotransferase (AST)[2] of 14000 U/L (reference interval 5–41 U/L) and alanine aminotransferase (ALT) of 6400 U/L (reference interval 8–45 U/L). Because laboratory indicators of liver function worsened, the patient was reinterviewed and confessed to having attempted suicide 3 days prior by ingesting about one-half gallon (approximately 4 L) of vodka and one-half bottle of extra-strength acetaminophen.

The patient was administered a bolus of N-acetylcysteine (NAC) at the outside hospital and referred to our institution because of concern for progression to fulminant hepatic failure. Laboratory tests were repeated and results were consistent with significant hepatocellular damage secondary to acetaminophen poisoning, including increased AST of 11000 U/L (reference interval 8–48 U/L), ALT of 6510 U/L (reference interval 7–55 U/L), total bilirubin of 395 μmol/L (23.1 mg/dL) [reference interval 2–17 μmol/L (0.1–1.0 mg/dL)], and direct-bilirubin of 207 μmol/L (12.1 mg/dL) [reference interval <5.0 μmol/L (<0.3 mg/dL)]. Treatment with intravenous infusion of NAC was continued, and nitroglycerin was administered for chest pain.

Within 24 h of admission the patient exhibited dyspnea with low hemoglobin (Hb) oxygen saturation (89%). Chest x-ray findings were unremarkable, whereas laboratory tests indicated a borderline-low Hb of 137 g/L (13.7 g/dL) [reference interval 135–175 g/L (13.5–17.5 g/dL)] with 4% methemoglobin (reference interval <1.5%). The patient received high-dose oxygen treatment but remained hypoxemic.

At 48 h the patient continued to have pronounced hyperbilirubinemia with evidence of hemolytic anemia, which included low Hb of 7.5 g/dL with 6% methemoglobin, marked reticulocytosis of 11.5% (reference interval 0.6%–1.83%), nondetectable haptoglobin [1.4 μmol/L (<14 mg/dL); reference interval 3–20 μmol/L (30–200 mg/dL)], increased mean corpuscular volume (MCV) (97.8 fL; reference interval 81.2–95.1 fL), and increased lactate dehydrogenase (LD) (6870 U/L; reference interval 122–222 U/L). Moreover, results of a direct Coombs test were negative, although a routine peripheral blood smear revealed the presence of "bite cells." An enzymopathy with a drug-induced acute episode of hemolytic anemia was suspected.

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Questions to Consider
• How does acetaminophen poisoning disrupt typical acetaminophen metabolism and what are the consequences?
• What are potential causes of hemolytic anemia?
• What laboratory tests should be performed to diagnose hemolytic anemia?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the December 2008 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol54/issue12/ and follow the link to the Clinical Case Study and Commentaries.

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