Persistently Increased Acetaminophen Concentrations in a Patient with Acute Liver Failure

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CASE

A 31-year-old woman was admitted into a regional hospital for abdominal pain, decreased appetite, malaise, confusion, and tea-colored urine. Investigations showed acute liver failure with a markedly decreased liver function characterized by greatly increased aminotransferases, bilirubin concentration, prothrombin time and international normalized ratio. There was no history of liver disease or intake of herbal medicines or over-the-counter medications. Her condition worsened 2 days later, and she was transferred to our hospital for further management and the possibility of liver transplantation. A physical examination revealed a jaundiced woman in a fair general condition and with a soft but tender right upper quadrant with no guarding or rebound tenderness of the abdomen. She went into a semicomatose state 1 day later. Routine laboratory testing of a blood sample obtained on her arrival in the hospital revealed the following results: bilirubin, 1210 μmol/L (reference interval, 7–19 μmol/L); alanine aminotransferase, 6170 U/L (reference interval, 5–31 U/L); aspartate aminotransferase, 5080 U/L (reference interval, 12–28 U/L); alkaline phosphatase, 150 U/L (reference interval, 34–104 U/L); ammonia, 171 μmol/L (reference interval, 0–33 μmol/L); lactate dehydrogenase, 6830 U/L (reference interval, 200–360 U/L); prothrombin time, 39.7 s (reference interval, 11.3–13.2 s); international normalized ratio, 3.3; acetaminophen, 121 μmol/L (therapeutic up to 100 μmol/L). Other results were unremarkable. A serologic evaluation was negative for hepatitis A and B. The plasma acetaminophen concentration prompted the clinical suspicion of drug overdose, but she denied taking acetaminophen. The patient's liver enzymes, prothrombin time, international normalized ratio, and acetaminophen concentrations were monitored on subsequent days. Her general condition and liver function gradually improved, but her plasma acetaminophen concentration remained >100 μmol/L. Failure of the liver to metabolize the drug was suspected, and liver transplantation was contemplated at that juncture.

Questions to Consider
• What are the common causes of acute liver failure?
• What is the usual pharmacokinetic pattern of acetaminophen after ingestion, and how does overdose cause liver injury?
• What methods are available to measure acetaminophen concentrations?
• What factors interfere with acetaminophen measurement?

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Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the January 2011 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol57/issue1 and follow the link to the Clinical Case Study and Commentaries.

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