Acute pseudohepatitis in a chronic substance abuser secondary to occult seat belt injury

ECS Lam MS· MD¹, Rhonda M Janzen MD², R Mark Meloche MD FRCPC², Paul J Trepanier MD FRCPC³, Eric M Yoshida MD FRCPC¹

Pseudohépatite aiguë chez un toxicomane chronique secondaire à une blessure infligée par une ceinture de sécurité

Résumé : Les causes d'une élévation massive des aminotransférases sériques (aspartate aminotransférase [AST] et alanine aminotransférase [ALT]) chez le toxicomane sont notamment l'hépatite virale et l'hépatotoxicité liée à la consommation de drogues. Un toxicomane adipeux de l'alcool et de la cocaïne s'est présenté au service des urgences d'un établissement de santé après un accident de la route. Il a été admis au service de médecine d'un centre de soins intensifs et, dans un état confus, a été hospitalisé au service de médecine d'un centre de soins intensifs. Le patient a été soumis à une tomographie par résolution de l’image. L'examen a révélé une fonction hépatique n’ont révélé qu’une séropositivité à l’égard de l’hépatite 

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A n acute elevation in serum aminotransferases (aspartate aminotransferase and alanine aminotransferase) in excess of 1000 U/L reflects significant hepatocellular injury, typically a consequence of acute viral hepatitis, toxic injury from medications and poisons (eg, acetaminophen overdose and isoniazid), or a hypotensive episode resulting in ischemic hepatitis (1). When investigating an acute hepatic flare of liver enzymes in the polysubstance-abusing patient, however, the clinician must also consider the psychoactive substances consumed and their route of administration as well as patterns of behaviour associated with the user's subculture. The use of injection drugs may be associated with transmission of acute, and occasionally fulminant, hepatitis B infection; coinfection or superinfection with the delta agent (2); and, rarely, acute hepatitis C viral (HCV) infection (3,4). The possibility of acute hepatitis A viral (HAV) infection in this subgroup also must also be considered because many injection drug users suffer from chronic viral hepatitis (5), which, in the case of HCV, has recently been reported to be associated with an increased risk of fulminant failure during acute HAV infection (6). HAV is also well recognized to be frequently associated with lower socioeconomic class (7), which substance abusers tend to occupy. The psychoactive substances may either directly or indirectly result in acute hepatocellular injury. Cocaine use is well recognized to be associated with hepatotoxicity (8) on the basis of segmental infarction (9). The drug better known as 'ecstasy', 3,4-methylenedioxymethamphetamine, likewise, has been reported to result in acute hepatotoxicity (10,11). Heavy alcohol consumption will result in the induction of the cytochrome P450 enzyme, CYP II E 1, which, together with concurrent glutathione depletion, may increase the likelihood of acute acetaminophen hepatotoxicity with even therapeutic doses – 'therapeutic misadventure' (12). With these considerations in mind, we report a chronic injection drug user who recently presented to the Vancouver Hospital and Health Sciences Centre with an acute flare in hepatocellular enzymes that was found to result from none of the indications mentioned above.

### CASE PRESENTATION

A patient with a long standing addiction to both injection heroin and cocaine, chronic alcoholism and a history of bipolar affective disorder had been an in-patient in the psychiatric unit four weeks before presentation to the author's centre. The patient was discharged with a prescription for lithium carbonate, chloral hydrate, torazepam and clonazepam. After discharge, the patient resumed use of cocaine and heroin, and was involved in a motor vehicle accident. The patient did not seek medical attention and denied any significant injury except for fatigue, anorexia and right upper quadrant abdominal discomfort. For these symptoms, the patient took acetaminophen. The day before admission, the patient was visited by the family physician who found the patient lethargic and unable to move out of bed. The patient was certified under the Mental Health Act of British Columbia (13) because of a concern for potential selfharm and was taken to hospital.

In the emergency room, the patient was noted to be diaphoretic, with dried blood under the fingernails but no evidence of bleeding, and alert but disoriented with respect to time and place. Chest examination was unremarkable; there was no bruing or tenderness, and auscultation was unremarkable. Physical examination did not reveal any stigmata of chronic liver disease or abdominal bruising. The liver was enlarged, 8 cm below the coastal margin at the midclavicular line and mildly tender to palpation. The rest of the examination was unremarkable, and a fecal occult blood test was negative. Laboratory investigations revealed a serum alanine aminotransferase level of 3820 U/L (normal less than 55 U/L), aspartate aminotransferase 4120 U/L (normal less than 38 U/L), total bilirubin 39 µmol/L (normal less than 22 µmol/L), gamma-glutamyltransferase 15 U/L (normal less than 50 U/L), alkaline phosphatase 137 U/L (normal less than 125 U/L), international normalized ratio 1.4 (normal 0.8 to 1.2), albumin 24 g/L (normal 35 to 45 g/L) and serum creatinine 469 µmol/L (normal greater than 110 µmol/L) (Table 1). A chest radiograph was unremarkable. Urinalysis revealed trace opiates. A drug and toxin screen was negative for ethanol and acetaminophen.

The patient was admitted to the general medical service with a diagnosis of acute liver disease and prerenal failure. Acetylcysteine by infusion was administered because the possibility of acetaminophen toxicity could not be excluded. Viral serology was ordered and was negative for HAV immunoglobulin M, and hepatitis B surface antigen and core antibody. Serology for HCV, which was negative seven months previously, was reactive. Renal function improved with intravenous fluids. Mental function also improved shortly after admission, and the patient denied significant acetaminophen use, alcohol consumption and illegal drug use other than habitual cocaine and heroin use. The only event that had occurred between the two hospital admissions was the

### TABLE 1

<table>
<thead>
<tr>
<th>Laboratory test</th>
<th>Baseline</th>
<th>On admission</th>
<th>Ten days postdischarge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine (µmol/L)</td>
<td>69</td>
<td>46</td>
<td>160</td>
</tr>
<tr>
<td>Total bilirubin (µmol/L)</td>
<td>NA</td>
<td>39</td>
<td>41</td>
</tr>
<tr>
<td>Alanine aminotransferase (U/L)</td>
<td>NA</td>
<td>3820</td>
<td>NA</td>
</tr>
<tr>
<td>Aspartate aminotransferase (U/L)</td>
<td>14</td>
<td>4120</td>
<td>111</td>
</tr>
<tr>
<td>Gamma-glutamyltransferase (U/L)</td>
<td>18</td>
<td>15</td>
<td>359</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>NA</td>
<td>137</td>
<td>209</td>
</tr>
<tr>
<td>International normalized ratio</td>
<td>NA</td>
<td>1.4</td>
<td>NA</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>NA</td>
<td>24</td>
<td>NA</td>
</tr>
</tbody>
</table>

*Baseline values from patient's previous psychiatric admission. NA Not available
motor vehicle accident, which occurred five days before presentation and from the history appeared minor, especially because the patient was wearing a seat belt.

The pattern of liver enzymes improved although cholestatic enzyme levels elevated later, with alkaline phosphatase level increasing to 209 U/L and 359 U/L. An abdominal ultrasound revealed an apparent complex intrahepatic mass. A computed tomogram (CT) of the abdomen revealed a small subcapsular hepatic hematoma and significant echodense changes in the right lobe, specifically in hepatic segments 5, 8 and a portion of segment 7 (Couinaud classification) (Figure 1). The hepatic vascular architecture appeared to be preserved without mass effect. The CT findings were consistent with a hepatic contusion injury. There was also decreased enhancement of the right kidney parenchyma consistent with a coincident renal contusion.

The patient remained clinically stable and was discharged after two weeks of hospitalization. Ten days postdischarge, (approximately four weeks after the motor vehicle accident), the patient experienced hematemesis but remained hemodynamically stable. Hemobilia was suspected; however, emergency esophagogastroduodenoscopy did not reveal any evidence of hemobilia or any other source of bleeding. An abdominal arteriogram did not reveal active bleeding or evidence of pseudoaneurysm. A follow-up abdominal CT scan revealed some resolution of the hepatic contusion, and the patient was discharged.

**DISCUSSION**

Blunt injury to the liver occurs in a reported 55% of severe and fatal motor vehicle accidents (14), with the most common source of injury arising from seat belt trauma or steering wheel impact (14,15). Following a contusive injury, there is typically a massive rise in serum aminotransferase levels (16). The massive elevation in serum aminotransferase levels may, in some cases, also reflect an ischemic hepatitis from hemorrhagic shock due to the effects of the multitraumatic injury (17,18). The hepatic injuries produced by abdominal trauma are typically hematomas that may be subcapsular or intraparenchymal, laceration of the capsule, parenchymal disruption to varying degrees and injury to the vascular structures including avulsion (19). A scale incorporating these features and their severity has been adopted by the American Association for the Surgery of Trauma Organ Injury Scaling Committee (19) to grade the extent of hepatic injury (Table 2), which has both prognostic and therapeutic implications. The most feared complications of hepatic contusion injuries are capsular rupture and sudden massive hemorrhage. Other complications include gastrointestinal bleeding from hemobilia; intraparenchymal hemorrhage into necrotic tissue, which is rarely exsanguinating; formation of bilomas; and biliary duct obstruction (20,21).

The approach to managing hepatic contusion injuries has changed over the past few decades. Previously, surgical exploration or diagnostic peritoneal lavage was considered mandatory for all patients and is clearly appropriate if there is hemodynamic compromise or high risk injury on diagnostic imaging. Definitive surgical management may include hepatectomy with ligation and/or repair of damaged vessels, resection, lobectomy, debridement and perihepatic packing.

**TABLE 2**

Grading of liver contusion injuries according to the American Association for the Surgery of Trauma Organ Injury Scaling Committee

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Hematoma</td>
<td>Subcapsular, nonexpanding, occupies less than 10% surface area</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Nonbleeding capsular tear, less than 1 cm in parenchymal depth</td>
</tr>
<tr>
<td>II</td>
<td>Hematoma</td>
<td>Subcapsular – nonexpanding 10% to 50% surface; intraparenchymal – less than 2 cm diameter</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, active bleeding, 1 to 3 cm parenchymal depth, 10 cm in size</td>
</tr>
<tr>
<td>III</td>
<td>Hematoma</td>
<td>Subcapsular – more than 50% surface area or expanding; rupture with bleeding; intraparenchymal – more than 2 cm or expanding</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>More than 3 cm parenchymal depth</td>
</tr>
<tr>
<td>IV</td>
<td>Hematoma</td>
<td>Intraparenchymal rupture – active bleeding</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Disruption involving 2.5% to 50% of lobe</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Parenchymal disruption more than 50% of lobe</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Venous-Pulmonary injuries (venacava, hepatic veins)</td>
</tr>
<tr>
<td>VI</td>
<td>Vascular</td>
<td>Avulsion</td>
</tr>
</tbody>
</table>

*Data adapted from reference 19*
grade injuries (ie, grades III to V) must be assessed on an in-

fluence the clinical presentation and management of vascular injuries specifically the absence of severestx screen because these findings can be falsely negative.

Our experience underscores the need for medical specialties to be aware of hepatic trauma as a cause of liver dysfunction and the need for diagnostic imaging in these cases. The current study also another entity – occult trauma to the list of differential diagnoses in the acute substance

REFERENCES