ISCHEMIC HEPATITIS DURING CARDIAC ARREST: A CASE REPORT

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ABSTRACT

We describe a 74 year old patient who developed Ischemic Hepatitis following cardiac arrest from which she was successfully resuscitated. She was found to have a massive elevation of ALT at 6024 which returned to normal in one week.

Her cardiac arrest was thought to have been triggered by sepsis from a urinary tract infection. The rapid and massive rise in ALT with a normal bilirubin and early resolution within two weeks indicates that the underlying pathology was likely to be Ischemic Hepatitis.

CASE HISTORY

A 74 year old female presented with collapse and loss of consciousness, which occurred on standing up after urination. There were no associated visual disturbances, headaches, fits or post-ictal drowsiness. There were also no associated cardiovascular symptoms. Significant past medical history included a brain tumour diagnosed 30 years ago which had recently started to re-grow. A cysto-peritoneal shunt in situ was surgically inserted 5 years ago following episodes of dizziness, expressive dysphasia and excessive sleeping. There have been no previous episodes of loss of consciousness. The patient also had a history of hypothyroidism, and diverticulitis and a malignant melanoma which was removed 15 years ago. The patient has always been a non smoker and was a social drinker.

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On examination, the patient was afebrile, with a respiratory rate of 14, heart rate of 72bpm, and blood pressure of 149/74mmHg. The abdomen was soft and non tender with no organomegaly and normal bowel sounds. Cardiovascular and respiratory examinations were unremarkable and neurological examination revealed a resting tremor in the right upper limb and ptosis of the left eye.

The patient began to deteriorate over the next few days after admission. She was unable to sit upright, had bilateral upgoing plantars and her Glasgow Coma Scale dropped to 9/15. Soon after, she went into cardiac arrest and was resuscitated by chest compressions, bag mask ventilation, 1mg atropine IV and 1mg adrenaline IV. Within the next few hours, she began to desaturate and went into multi-organ failure. She was subsequently stabilised with a fluid challenge. Blood analysis was consistent with sepsis secondary to a urinary tract infection. Blood biochemistry demonstrated the following significant findings:

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
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<tbody>
<tr>
<td>ALT</td>
<td>6024 IU/L</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>15 IU/L</td>
</tr>
<tr>
<td>ALP</td>
<td>108 IU/L</td>
</tr>
<tr>
<td>GGT</td>
<td>161 IU/L</td>
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Cardiac Troponins and Creatinine Kinase were also raised, but this was suspected to be secondary to chest compressions.

A diagnosis of ischemic hepatitis in the setting of septic shock was made. The patient was accordingly managed by treating the underlying cause of septic shock with co-amoxiclav and meropenem resulting in complete recovery. Her ALT level returned to normal after one week.

**DISCUSSION**

Ischaemic hepatitis (IH) (also called hypoxic liver injury, hypoxic hepatitis and shock liver) is liver necrosis, primarily of the centrilobular hepatocytes secondary to a variety of causes(1). The diagnosis is usually based on biochemical findings due to the absence of symptoms and signs. IH is generally defined in the literature by the following important characteristics (incorporating Gibson and Dudley’s criteria (2, 3):

- Significantly elevated, transient (5-25 days) serum aminotransferase levels (Greater than 8 times the normal upper normal level) with or without lactate dehydrogenase elevation.
- Histological evidence of centrilobular necrosis.
Rapid resolution of pathology (within 7-10 days).
Exclusion of other hepatic insults.

Ischemia is not an essential criterion in the diagnosis of IH though insufficient hepatic perfusion is the most common cause (1). Other contributors include elevated metabolic requirements, liver toxins and hypoxia among others (2, 4, 5) summarized in Figure 1 (adapted from (3)).

The liver is relatively well protected against ischaemic insults, deriving its dual blood supply from the hepatic portal vein (80%) and hepatic artery (20%). The liver receives approximately 25% of the total cardiac output (6, 7). IH can occur in the setting of septic shock, which poses a significant cardiovascular imbalance. Although cardiac output is generally well preserved systemic circulation is often maintained through mesenteric blood flow compromise (1, 8). The hepatic sequelae results from hepatic artery vasoconstriction and intestinal vasoconstriction, mediated by the renin-angiotensin system, leading to reduced portal vein blood flow (9). The portion of the liver supplied by the hepatic artery is the acinus and divided into zones 1, 2 and 3 signifying reduced oxygenation as the blood moves from artery to central vein. IH is characterized by centrilobular necrosis as the central liver closest to the central vein in region 3 is the least well oxygenated (1, 8) hence most susceptible to damage. IH aetiology includes the following: Primary heart disease (78%), congestive heart failure (65%), myocardial infarction (17%), sepsis (15%) and chronic respiratory failure (12%) (3). Although IH is the most common diagnosis differential causes for transaminase elevations (typically >3000 U/L) include hepatic trauma, viral hepatitis, toxin or pharmacologically induced hepatitis (10). IH is differentially characterized by early increases in INR, creatinine and rapid transaminase recovery after removal of the causative factor. Finally the ALT:LDH ratio tends to be low due to the elevated LDH levels in IH (1, 10, 11).

Affected patients tend to be elderly, the majority of whom suffer from primary heart disease-right heart failure being the most common (12). Although most patients tend to suffer from a spectrum of diseases IH usually occurs secondary to an acute event e.g. myocardial infarction although interestingly most patients do not have a significant decrease in blood pressure (1, 12). Symptoms tend to be relatively non-specific and include weakness, shortness of breath and right upper quadrant pain (13, 14), patients are often asymptomatic. Serum transaminases increase due to hepatic damage and serum lactate can also be elevated due to inadequate removal by the dysfunctional liver (15). Ultrasound scans of the liver reveal hypoechoic areas whilst computerised tomography demonstrates hypodense regions, such abnormalities improve alongside recovery (16). Although mortality is high in patients with IH this tends to be from the underlying disorder as opposed to IH itself.
Figure 1: Ischaemic hepatitis aetiology and pathogenesis

REFERENCES