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Clinical Image Open Access

Post Infantile Giant Cell Hepatitis (PIGCH) Induced by Habitual Therapeutic Dosing of Acetaminophen

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Clinical Image

A 36-year-old, information technology (IT) employee, presented to the National Liver Institute hospital, Menoufia University, Egypt, with jaundice and itching of 4 weeks duration. No history of herbal consumption, tobacco use or drinking habits. The benign medical and drug history of the patient has lately been intruded by daily repeated intake of acetaminophen. Admittedly, the patient used to take 4 grams of acetaminophen daily for the 6 weeks preceding the start of his illness. The patient claimed that he wanted to maintain his capacity all throughout the extended working hours. Also, he knows well the safe dosing of acetaminophen [1,2]. Possibilities of supratherapeutic dosing or single toxic dose intake were adamantly denied by the patient. The patient was advised to stop acetaminophen and received nonspecific medical care without any regression of his condition.

On presentation, he was conscious with acceptable vital signs and normal cardiopulmonary examination. Jaundice, itching marks, and hepatomegaly were clinically evident. Abdominal sonography and dedicated Doppler study showed bright hepatomegaly with normal hepatic vasculature and pancreaticobiliary system. Magnetic resonance cholangiopancreatography (MRCP) negated biliary obstruction and confirmed normal pancreaticobiliary system. Laboratory investigations revealed average blood picture, rising serum bilirubin (T/D: 40/31 mg/dl was the highest value), acute hepatocellular injury (transaminases exceeding thousands), and mildly elevated canalicular enzymes. Negative serology for viral hepatitis (HAV, HBV, HCV, HEV, CMV, EBV, and HIV), and autoimmune hepatic aetiologies along with the normal immunoglobulin serum levels: all were reported. Serum ceruplasmin was mildly elevated with normal 24-hour urinary copper.

Histopathological examination of liver tissue showed preserved lobular architecture with diffuse giant cell transformation reflecting the picture of post infantile giant cell hepatitis (PIGCH) [1,3]. Portal tracts showed mixed acute and chronic inflammatory infiltrate rich in neutrophils and eosinophils, assisting the diagnosis of hepatitic pattern of drug-induced liver injury [2]. Hepatic parenchyma showed mild interface hepatitis, intracellular and intracanalicular cholestasis but preserved bile ducts [1]; a picture matching the mildly elevated cholestatic enzymes. Calculation of Russel Uclaf Causality assessment method (RUCAM score) was probable for the diagnosis of drug-induced liver injury (DILI).

The findings in this case, are mostly consistent with acetaminophen-induced liver injury. Consequently, the diagnosis of PIGCH incited by the sustained maximal therapeutic dosing of acetaminophen was substantiated. Prednisolone 60 mg/day was started also phototherapy for disabling itching, to be followed by gradual clinical and biochemical improvement. Two weeks later, follow-up indicated the further significant reduction in bilirubin and both hepatocellular and cholestatic enzymes. Giant cell hepatitis; a unique pediatric disorder, is a rarely described illness in adults. Autoimmune disorders, lymphoma, and drugs are the evolving associates of the adult form of post infantile giant cell hepatitis (PIGCH). Although, acetaminophen is the safest among pain-reducing agents, its chronic therapeutic range usage is not free from hepatotoxicity in susceptible individuals (Figure 1).

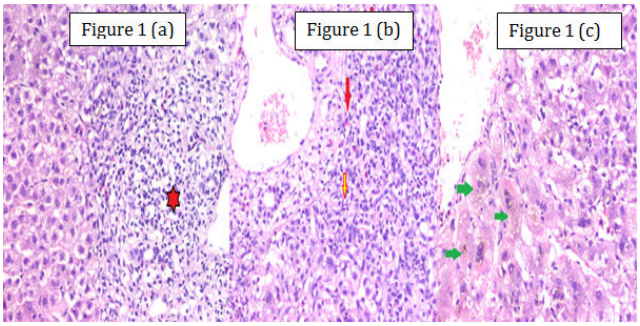

Figure 1: (a) and (b) Hepatic tissue showed expansion of the portal tracts (red star) with dense mixed acute and chronic inflammatory cellular infiltrate rich in eosinophils (red arrows) neutrophils (yellow vertical arrow), together with moderate/focal interface hepatitis (yellow horizontal arrow) (H&E 200X); (c) The hepatic parenchyma revealed perivenular giant cell transformation together with intracytoplasmic cholestasis (green arrow) (H&E 200X).

Discussion

The potentiating factors for acetaminophen-induced hepatotoxicity include; concomitant use of alcohol or other drugs, comorbid illnesses, advancing age, malnutrition as well as the individual's genetic makeup.

Polymorphisms exist in the cytochrome isoenzymes may contribute to increased oxidative metabolism of acetaminophen and increased incidence of hepatotoxicity in particular individuals.

In Gilbert's syndrome; glucuronidation capacity is decreased and more acetaminophen is shunted to the cytochrome P450 enzymes producing excess toxic metabolite.

References

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