

Levocetirizine induced hepatotoxicity in a patient with chronic urticaria

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Dear Editor

Levocetirizine is a third-generation non-sedative antihistaminic agent and has developed from the second-generation antihistaminic drug: cetirizine. It may have side effects such as sleepiness, headache, dry mouth, lightheadedness, vision problems, palpitations and fatigue.¹ There are only few cases reporting cetirizine induced liver toxicity.² However, we could not find any published data about levocetirizine associated hepatotoxicity in the literature. Herein, we present the first case of levocetirizine induced hepatotoxicity.

A 64-year-old man was consulted to our department from the outpatient clinic of dermatology department for elevated liver enzymes. He has been taking levocetirizine 5 mg twice a daily for two weeks because of chronic urticaria. His past medical history was unremarkable, there were no administrations of any other medications, nutritional supplements or herbal products and he denied using alcohol. At the time of routine check-up tests, before the treatment, liver enzymes were in normal limits. Physical examination was normal except for skin rashes. Biochemical parameters performed on admission were as follows: alanine transaminase (ALT): 115 IU/mL (normal range 0-32 IU/mL), aspartate transaminase (AST): 113 IU/mL (normal range 0-32 IU/mL), alkaline phosphatase: 440 IU/mL (normal range 0-270 IU/mL), γ -glutamyltranspeptidase (GGT): 112 IU/mL (normal range 0-38 IU/mL). Albumin, bilirubin and pro-

thrombin time were within the normal ranges. Serological markers for acute viral hepatitis were negative for hepatitis A, hepatitis B, herpes simplex viruses, Epstein-Barr virus, and cytomegalovirus. Anti-HCV, anti-HIV, HBV-DNA, and HCV RNA were all negative. In addition, laboratory tests for autoimmune hepatitis, hemochromatosis, thyroid diseases or Wilson's disease were also unremarkable. Abdominal ultrasonography revealed no evidence of extrahepatic obstruction, biliary ductal disease, hepatic parenchymal abnormalities, or cholelithiasis. After cessation of levocetirizine, liver enzymes were within the normal limits in the day 20 (AST: 25 IU/mL, ALT: 31 IU/mL, ALP: 242 IU/mL, GGT: 35 IU/mL). The patient continued to complain itching and two months later, we restarted levocetirizine again. However, three days after re-administration of the drug, liver enzymes increased again (AST: 55 IU/mL, ALT: 60 IU/mL, GGT: 124 IU/mL, ALP: 312 IU/mL). Therefore, we stopped levocetirizine and liver enzymes gradually normalized in four weeks. In the light of finding no evidence of other liver disease and normalization of liver enzymes after withdrawal of the drug pointed us the diagnosis of levocetirizine-induced hepatotoxicity.

Drug-induced hepatotoxicity is a frequent cause of liver injury and accounts for < 5% of the cases with jaundice or acute hepatitis. However, patients with drug-induced liver injury can be presented similarly with other causes of hepatobiliary disease, it is important to maintain a high index of clinical suspicion in making a diagnosis. The predominant clinical presentation of drug-induced liver injury is acute hepatitis and/or cholestasis, although idiosyncratic reactions often occur on a background of a higher rate of mild asymptomatic liver injury.³ This case of liver injury was considered as an idiosyncratic drug reaction. Levocetirizine is generally well-tolerated drug, which has consistently

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demonstrated high response rates and a favorable side effect profile. Although, both elevated transaminases and cholestatic liver enzymes due to levocetirizine are described in the drug's prescribing information but to our knowledge, this is the first report of hepatotoxicity caused by levocetirizine in the literature. In conclusion, however, levocetirizine is an effective drug for the treatment of chronic urticaria, physicians should be aware of hepatotoxicity due to levocetirizine.

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