Case Report

LOWER DOSES VENLAFAXINE-ASSOCIATED TOXIC HEPATITIS IN A PATIENT WITH CHRONIC HEPATITIS

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ABSTRACT:

Toxic hepatitis is observed with high doses of Venlafaxine. But toxic hepatitis has not been yet reported at lower doses of Venlafaxine such as 37.5 mg per day. In this case report, a case of Venlafaxine-associated toxic hepatitis with lower doses in patient with history of chronic hepatitis is presented. We suggest that liver function should be regularly monitored in patients with history of chronic hepatitis receiving Venlafaxine even at lower doses and even when their liver enzymes are normal.

KEY WORDS: Venlafaxine, Chronic hepatitis, toxic hepatitis.

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INTRODUCTION

Venlafaxine is a novel antidepressant drug that is absorbed from the gastrointestinal tract and metabolised in the liver by cytochrome P450 isoenzyme CYP 2D6. Venlafaxine -

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associated adverse effects, related to gastrointestinal tract and autonomic nervous systems have been reported. These adverse effects have been observed with higher doses of Venlafaxine by Kaplan and Sadock¹. These observations have also been made by Horsmans et al.² and Cardona et al.³. However, toxic hepatitis has not been yet reported at lower doses of venlafaxine such as 37.5 mg per day. We present a case of acute toxic hepatitis superimposed on chronic hepatitis with low-dose Venlafaxine.

The Case

On 26 June 2002, a 30 -year-old woman was admitted to our clinic with complaints of weakness and nausea. Routine laboratory analysis presented a high serum aminotransferase, AST: 369U/L (0-38), ALT:689 U/L (0-41). Her medical history revealed utilization of interferon - α 9 million units, three times weekly for six months with the completion of therapy two months earlier. At the time completion of interferon therapy aminotransferase levels were normal. Additionally, serological tests of HCV, HDV and HBV DNA

PCR were negative. On 12 May, she had been started, Venlafaxine (Efexor, Wyeth, Istanbul) with dosage of 37.5 mg/d due to a depression episode.

Venlafaxine therapy was stopped because of elevated liver enzymes after admission of the patient to the clinic. Serological markers of HDV, HCV, HAV, HEV and HBV DNA were negative. Abdominal ultrasonography, α – fetoprotein, autoantibodies were normal and no intake of alcohol and other hepatotoxic drugs were present. The patient gradually improved and hepatic function tests returned to normal within three weeks. Anti-HCV and HCV RNA were negative still after six weeks. We excluded all other possible causes of acute liver injury, and symptoms and biochemical markers of liver injury returned to normal after three weeks of discontinuation of venlafaxine therapy.

CONCLUSION

We agree with Horsmans², Cardona³ and colleagues who suggest that liver function should be regularly monitored in patients receiving venlafaxine. Additionally, it is well justified to identify lower doses of venlafaxine as the culprit in acute liver toxic injury of patient's with history chronic hepatitis even when their liver enzymes are normal.

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