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Acute Liver Failure to Fluoxetine Therapy

Muhammad Tahir, MD

Department of Internal Medicine Sisters of Charity hospital, 2157 Main St, Buffalo, NY 14214, USA.
muhammadtahir090@gmail.com

***Corresponding Author:** Muhammad Tahir, MD, Department of Internal Medicine Sisters of Charity hospital, 2157 Main St, Buffalo, NY 14214, USA.*

Abstract

Fluoxetine is a commonly prescribed antidepressant. The hepatic failure reported secondary to this particular drug is very rare i.e. 0.5% of the patients who are taking this drug for long term therapy. The routine monitoring is not advised for someone on maintenance therapy. The exact mechanism is still unknown. We reported a case of hepatic failure in a 35 year old patient who was started on this drug for her persistent depression. The presenting complaint was jaundice with a cholestatic manifestations. The workup was unremarkable, other than liver enzymes dysfunction (ALT 1129 U/L, AST 1892 U/L). The hepatitis panel and imaging was negative. Ultimately biopsy was done which showed portal, periportal and lobular hepatitis. The working diagnosis of Drug-induced liver injury was made and patient showed improvement in her condition in 2-4 weeks. The patient was also followed at outpatient clinic and the liver enzymes were normalized.

Keywords: *drug-induced liver injury, hepatic failure, SSRI*

Introduction

The drug induced liver injury by antidepressant is being reported in the literature nowadays[1]. However fluoxetine induced hepatotoxicity has not been well recognized. Usually asymptomatic increase in liver enzymes have been reported in 0.5% of the patient who are taking this drug for long-term therapy[2]. The exact mechanism is still unknown however treatment approach is usually very conservative.

Here we discuss a case of hepatic failure secondary to short term Fluoxetine therapy which is very rarely reported in the literature.

Case report

35 year old female with the past medical history of major depression was admitted to hospital for the evaluation of jaundice which was reported acute in onset. Patient was recently switched to fluoxetine therapy from TCA for persistent depression. The liver dysfunction was also reported with ALT 1129, AST 1892, Alkaline phosphatase of 967, GGT 1364 and prothrombin time of 23.1 and total bilirubin of 11.4.The workup included negative CMV, EBV, negative ANA, Antimitochondrial antibody, Rheumatoid factor and acetaminophen level. Ferritin level reported was 83 with TIBC of 352. The blood alcohol level was negative. The serologies for hepatitis A, B and C was negative. The autoimmune workup was also unremarkable and patient denied any substance abuse or herbal intake. Imaging studies ruled out any mass, fibrosis and biliary pathology. MRI abdomen was also unremarkable. US Doppler of abdomen showed no abnormality. The patient was initially stabilized and then a liver biopsy was performed during the hospitalization which showed portal, periportal and lobular hepatitis. The case was discussed at different institutions at multidisciplinary level. The working diagnosis of Drug-induced liver injury was made and patient showed improvement in her condition in 2-4 weeks. The patient was also followed at outpatient clinic and the liver enzymes were normalized.

Discussion

The selective serotonin reuptake inhibitors (SSRIs) are some of the most commonly used prescription medications worldwide, yet reports of clinically apparent hepatic injury during their use are rare. Typically, the latency to onset is 1 to 6 months and the pattern of injury is either an acute hepatocellular or cholestatic hepatitis that is self-limited and rapidly reversed upon withdrawal of the agent[3]. When patients develop clinically apparent liver injury from an SSRI, it is not clear whether another member of this

group can be substituted. A structurally unrelated substitute along with careful monitoring is perhaps prudent if antidepressant therapy is considered necessary[4].

Learning point

Fluoxetine is a commonly prescribed antidepressant. Although routine monitoring of liver function is not recommended, physicians should remain alert to the possible relation of SSRI and liver dysfunction. The treatment approach is usually conservative however appropriate consultation or transfer is necessary if there is a risk of liver failure.

References

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