

Letter to Editor

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Apixaban Induced Hepatotoxicity in a Chronic Kidney Disease Patient with Left Ventricular Apical Clot

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ABSTRACT

Drug induced liver injury (DILI) is a common occurrence in clinical practice mostly seen with anti-tubercular drugs. Drug cessation is crucial part of management. But DILI secondary to direct oral anticoagulant (DOAC) is rare. Especially risk of hepatotoxicity with apixaban is lower than other DOACs. A 78 years old diabetic and hypertensive female with coronary artery disease admitted with acute on CKD stage-3 (serum creatinine 1.7) underwent echocardiography revealing left ventricular apical clot. Cardiology team added apixaban following which patient developed anorexia, nausea and abdominal discomfort. Liver function test revealed transaminitis with liver enzymes suggestive DILI. Patient was neither on any concomitant hepatotoxic drugs nor had a history of liver disease. Apixaban was discontinued and enoxaparin was started following which transaminitis improved and patient became better.

Dear Editor,

Drug induced liver injury (DILI) is a broad spectrum of manifestations ranging from asymptomatic liver enzyme elevation to severe hepatic failure. Drug cessation is crucial part of management. Chronic kidney disease (CKD) patients are prone for thrombo-embolic events and direct oral anticoagulants (DOAC) are used very often ^[1]. DILI secondary to DOACs is rare. Risk of hepatotoxicity with apixaban is lower than other DOACs ^[2,3]. A 78 years old diabetic and hypertensive female with coronary artery disease admitted with acute on CKD stage-3 (serum creatinine 1.7) underwent echocardiography revealing left ventricular apical clot. On adding apixaban 2.5 mg tablet patient developed anorexia, nausea and abdominal discomfort. Patient had transaminitis with alanine

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transaminase being 1402, aspartate transaminase being 1350, prothrombin time being 28 and total bilirubin was 5mg/dl; favouring diagnosis of DILI. Patient was neither on any concomitant hepatotoxic drugs nor had a history of liver disease. However, patient was investigated for causes of acute hepatitis and were found to be negative. Ultrasonography abdomen was negative for bile duct obstruction or chronic liver disease. Apixaban was discontinued; following which liver enzymes serum bilirubin, prothrombin time settled (within 3 days) and patient improved as well. After consulting cardiology and gastroenterology teams it was restarted. Liver function tests were repeated after 2 days which showed transaminitis with alanine transaminase being 1307, aspartate transaminase being 1103, prothrombin time being 25 and total bilirubin being 3.8. Apixaban was withdrawn from treatment following which transaminitis improved, liver function test normalized and patient became better. Enoxaparin was started for left ventricular apical clot. Few case reports on DILI due to DOACs are there in elderly patients not on concomitant hepatotoxic drugs and with favourable outcome after DOAC discontinuation^[3]. This occurrence is already an elephant in the room and requires further research for better understanding of incidence and risk factors for DILI secondary to DOACs and rescue measures needed in such cases.

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