

Pseudo-Alcoholic Cirrhosis: An Interesting Case Of Amiodarone Induced Hepatotoxicity.

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Citation

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Abstract

Amiodarone is a commonly used antiarrhythmic drug in clinical practice, despite its notoriety for causing serious adverse effects. Long-term therapy, even at low doses poses great risks to the patient. We report a case of amiodarone induced hepatotoxicity.

BACKGROUND

Amiodarone is a commonly used antiarrhythmic drug in clinical practice, despite its notoriety for causing serious adverse effects. Long-term therapy, even at low doses poses great risks to the patient.

CASE REPORT

A 77-year-old female presented with increasing abdominal distension, weakness, nausea and vomiting, lethargy and confusion. Medical history was pertinent for atrial fibrillation, treated with amiodarone 200 mg daily for four years. Patient was known to have mildly elevated liver transaminases (less than 2 times of upper limit of normal) that were monitored by semi-annual transaminases.

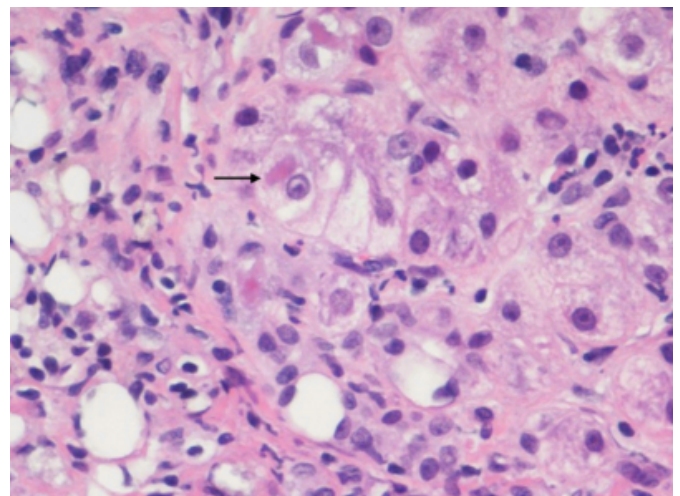
Physical exam was remarkable for fluctuating levels of mental status, asterixis, irregular heart rhythm, abdominal distension with a positive fluid thrill. Lab studies showed Hb-11.5, platelets-112, AST-192, ALT-162, ALK PHOS-122, reversal of albumin-globulin ratio, and prolonged prothrombin time. Abdominal paracentesis revealed transudative ascites with a high serum-ascites albumin gradient. Further work up revealed negative serology for ANA, anti-smith antibody, hepatitis A, B and C virus panel, normal alpha-1 antitrypsin and ferritin levels.

A transjugular needle biopsy of the liver revealed mixed inflammation with neutrophilic satellitosis, numerous Mallory hyaline bodies (figure 1), and foam cells likely representing phospholipidosis (figure 2). The wedge hepatic pressure was 40 mmHg, free hepatic pressure was 23 mmHg, and gradient was 17 mmHg consistent with portal hypertension. The hepatic and portal venous structures were

patent.

Figure 1

Figure 1: Tranjugular Liver biopsy showing hepatitis with Mallory's hyaline (arrow) and minimal macrovesicular steatosis.



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DISCUSSION

Liver enzyme abnormalities are an all-too-common finding in patients on amiodarone therapy in about 15-80% of patients¹. About 1-3% develop severe hepatotoxicity with cirrhosis developing in 0.6%¹. Though it is not indicated to withdraw or withhold treatment with amiodarone in patients with abnormal liver enzymes if it is elevated less than 3 times upper limit of normal, periodic liver enzyme surveillance is recommended to monitor progressive damage. This illustration clearly demonstrates the rare but

serious effects that low dose amiodarone has on the liver and the need for close monitoring of liver transaminases before and during therapy². It is not clear if there are certain genetic polymorphisms that predispose to amiodarone toxicity. The presence of phospholipidosis in liver biopsy is useful in diagnosis of amiodarone toxicity in patients who otherwise do not have an explanation for liver cirrhosis³.

References

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