Acute Hepatic Failure and Oral Amiodarone
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Introduction:
Amiodarone is associated with serious complications such as lung toxicity, thyroid dysfunction, and QT-prolongation. Further, hepatic failure from the intravenous (IV) formulation of amiodarone is described in numerous case reports and case series. Although there are reports of elevated liver enzymes from the oral formulation of amiodarone, to our knowledge, none have definitively identified a case of acute hepatic failure from the oral formulation alone. In this case, we report an instance of acute hepatic failure after increasing the oral amiodarone dosage as well as rapid recovery following cessation of the medication.

Case report:
A 61-year-old male with a history of non-ischemic cardiomyopathy with reduced ejection fraction presented with heart failure exacerbation due to atrial fibrillation with rapid ventricular response. He had recently failed cardioversion for atrial fibrillation and was started on a higher dose of amiodarone (400 mg twice a day from 100 mg daily). In this admission, blood tests revealed elevated BNP (1285) and INR (3.9). Therefore, warfarin was held, and he was started on IV furosemide. His metoprolol was increased for a better rate control and he was continued on his amiodarone. The next day, his morning tests were significant for elevated liver enzymes (AST/ALT 476/434 from 25/36 one week prior), INR (6.5), and bilirubin (2.3). His kidney function has also deteriorated (BUN/Cr 30/3.0 from 21/2.2). Amiodarone was held after his heart rate dropped between 40s and 50s. His repeated laboratory tests revealed a lactate of 8.5 and potassium of 7.4. His systolic blood pressure has then dropped to 80s and 90s. His ALT, AST, INR, and creatinine were peaked to 6702, 3021, 8.2, and 5.1, respectively. He just required dobutamine briefly without any need for dialysis. Nonetheless, his liver and kidney conditions started showing rapid improvement.

Discussion:
Several case reports focused on IV amiodarone as a cause of acute hepatic failure while the cumulative oral dose is a classical cause of chronic hepatotoxicity. The solvent part of the IV formulation is believed to be the reason for the acute reaction since this solution is not found in the oral form [1]. However, our case sheds light on the possibility of acute hepatic failure following increasing the oral amiodarone dose. Although this patient had a brief episode of hypotension, the biochemical hepatic abnormalities happened before the hypotensive episode. This chronological sequence of events made us believe amiodarone was the drug involved in the hepatic injury.

Conclusion:
To our knowledge, the literature almost always links the IV form of amiodarone to acute hepatic failure. We report an increase in the oral dose, as opposed to the IV form, of amiodarone potentially causing acute hepatic failure.

References: