Clinical Quagmire of Transaminitis in a Patient with Shock

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Introduction:
- Amiodarone is a class III antiarrhythmic drug. Amiodarone-induced hepatotoxicity can occur secondary to an impairment of mitochondrial fatty acid oxidation.
- We present a case of intravenous amiodarone induced hepatotoxicity in a critically ill patient of septic shock secondary to Ludwig's angina, its identification and subsequent outcome.

Case description:
- 55 year old male being treated for septic shock due to Ludwig’s angina which resolved on day 0 developed atrial fibrillation with rapid ventricular rate of 120-130/min on day 1 of admission. Patient was started on IV Amiodarone.
- Patient’s ALT and AST on admission were 77 and 96 U/L respectively. Transaminases up trended within 24 hours of administration with ALT levels 403, 1958 U/L and AST levels of 605, 2350 U/L on day 1 and day 2 respectively. Amiodarone was stopped on day 3. Transaminases down trended to near normal limits subsequently.
- Initial rise in transaminases was attributed to septic shock but subsequent dramatic rise after shock resolution warranted attention.
- Amiodarone was believed to be the inciting agent as the uptrend and downtrend matched with the time of initiation and discontinuation of the drug.

Conclusion:
- Acute liver damage after intravenous amiodarone, possibly induced by the solubilizer polysorbate 80, is rare but potentially harmful. Prompt discontinuation of Amiodarone is warranted.
- Transaminitis secondary to IV Amiodarone occurs generally within 24 hours of administration. Patients with IV amiodarone induced transaminitis can still be given oral amiodarone. It should be noted though to avoid Amiodarone in pre-existing liver disease or transaminitis.
- Though rare cases acute liver failure has been mentioned in literature and hence use of intravenous Amiodarone should be exercised with caution especially in critically ill patients at risk of potential liver injury such as septic shock, cirrhosis.

References: