

ESPS Peer-review Report

Name of Journal: World Journal of Gastroenterology

ESPS Manuscript NO: 10722

Title: N-Acetylcysteine treats IV Amiodarone Induced Liver Injury

Reviewer code: 00008590

Science editor: Ya-Juan Ma

Date sent for review: 2014-04-16 16:12

Date reviewed: 2014-04-18 16:56

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A (Excellent)	<input checked="" type="checkbox"/> Grade A: Priority Publishing	Google Search:	<input type="checkbox"/> Accept
<input checked="" type="checkbox"/> Grade B (Very good)	<input type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<input type="checkbox"/> High priority for publication
<input type="checkbox"/> Grade C (Good)	<input type="checkbox"/> Grade C: a great deal of language polishing	<input type="checkbox"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D (Fair)		BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E (Poor)	<input type="checkbox"/> Grade D: rejected	<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

COMMENTS TO AUTHORS

This is an interesting case reports with various clinical issues. However, a further case analysis is necessary. Major points: 1. I think the first flare was DILI, the second flare a combination of DILI and cardiac hepatopathy. Please include this interpretation more precisely into the revised version. At the first flare, ALT and AST were similar, compatible with DILI but not with cardiac hepatopathy where AST is always higher than ALT (quote relevant reports on this important issue, quote also: Henrion J. Hypoxic hepatitis. Liver Int 2012; 32: 1039-1052). At the second flare, AST is much higher than ALT (with ALT similar to the first flare), so both causes may apply. 2. Type of liver injury? Data should be submitted to causality assessment by CIOMS and to reexposure criteria (Teschke R, Wolff A, Frenzel C, Schwarzenboeck A, Schulze J, Eickhoff A. Drug and herb induced liver injury: Council for International Organizations of Medical Sciences scale for causality assessment. W J Hepatol 2014; 6: 17-32; Teschke R, Eickhoff A, Schulze J. Drug and herb induced liver injury in clinical and translational hepatology: Causality assessment methods, quo vadis? J Clin Translat Hepatol 2013; 1: 59-74. DOI: <http://dx.doi.org/10.14218/JCTH.2013.D002X>). Quote these reports and apply the actual schemes below. CIOMS scale for the hepatocellular type of injury in DILI cases Items for hepatocellular injury Score Result 1. Time to onset from the beginning of the drug • 5 – 90 days (rechallenge: 1 – 15 days) • < 5 or > 90 days (rechallenge: > 15 days) Alternative: Time to onset from cessation of the drug • ≤ 15 days (except for slowly metabolized chemicals: > 15 days) +2 +1 +1 2. Course of ALT after cessation of the drug Percentage difference between ALT peak and N • Decrease ≥ 50 % within 8 days • Decrease ≥ 50 % within 30 days • No information or continued drug use • Decrease ≥ 50 % after the 30th day • Decrease < 50 % after the 30th day

or recurrent increase +3 +2 0 0 -2 3. Risk factors • Alcohol use (drinks/d: > 2 for woman, > 3 for men) • Alcohol use (drinks/d: ≤ 2 for woman, ≤ 3 for men) • Age ≥ 55 years • Age < 55 years +1 0 +1 0 4. Concomitant drug(s) • None or no information • Concomitant drug with incompatible time to onset • Concomitant drug with compatible or suggestive time to onset • Concomitant drug known as hepatotoxin and with compatible or suggestive time to onset • Concomitant drug with evidence for its role in this case (positive rechallenge or validated test) 0 0 -1 -2 -3 5. Search for non drug causes Group I (6 causes) • Anti-HAV-IgM • HBsAg, anti-HBc-IgM, HBV-DNA • Anti-HCV, HCV-RNA • Hepatobiliary sonography / colour Doppler sonography of liver vessels / endosonography / CT / MRC • Alcoholism (AST/ ALT ≥ 2) • Acute recent hypotension history (particularly if underlying heart disease) Group II (6 causes) • Complications of underlying disease(s) such as sepsis, autoimmune hepatitis, chronic hepatitis B or C, primary biliary cirrhosis or sclerosing cholangitis, genetic liver diseases • Infection suggested by PCR and titer change for CMV (anti-CMV-IgM, anti-CMV-IgG) • EBV (anti-EBV-IgM, anti-EBV-IgG) • HEV (anti-HEV-IgM, anti-HEV-IgG) • HSV (anti-HSV-IgM, anti-HSV-IgG) • VZV (anti-VZV-IgM, anti-VZV-IgG) Evaluation of group I and II • All causes-groups I and II - reasonably ruled out • The 6 causes of group I ruled out • 5 or 4 causes of group I ruled out • Less than 4 causes of group I ruled out • Non drug or herb cause highly probable Tick if negative □ □ □ □ □ □ □ □ □ □ □ +2 +1 0 -2 -3 6. Previous information on hepatotoxicity of the drug • Reaction labelled in the product characteristics • Reaction publ

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A (Excellent)	<input checked="" type="checkbox"/> Grade A: Priority Publishing	Google Search:	<input type="checkbox"/> Accept
<input type="checkbox"/> Grade B (Very good)	<input type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<input type="checkbox"/> High priority for publication
<input checked="" type="checkbox"/> Grade C (Good)	<input type="checkbox"/> Grade C: a great deal of language polishing	<input type="checkbox"/> No records	<input type="checkbox"/> Rejection
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<input type="checkbox"/> Grade E (Poor)	<input type="checkbox"/> Grade D: rejected	<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

COMMENTS TO AUTHORS

1.-Authors should be careful with the abbreviations. a)the title and abstract should not contain them
2.-it's not clear when the bilirubin increased, which day? 3.-The author only write the use of diluent polysorbate 80 in discussion. This diluent should be noted in the description of case report. also, is the only diluent is used in iv administration of Amiodarone? The authors should clarify this point. 4.The authors write the levels of Lactic ac..but after they did not write if this levels is normalized and when. 5.-What is the probable mechanism of amiodarone toxicity ?. it is too vague to say "mitochondrial toxicity"