Liver Injury and Ascites in Dengue

AA Bamanikar*

Sir,

The original article “The study of Hepatic dysfunction in Dengue” in the issue no.7 of July 2013 vol 61 invokes the rapid response to discuss the significance of liver enzymes in dengue fever and unusual features specifically mentioned in conclusions. Normal blood test for liver function tests include: ALT, AST, ALP, albumin, total protein, bilirubin, GGT, PT. However, it must be clarified that they can be normal in patients with serious liver disease and abnormal in patients with diseases that do not affect liver. The liver carries out thousands of biochemical functions, most of which cannot be easily measured by blood tests. Laboratory tests can measure only limited number of these functions. In fact, many tests such as aminotransferases or alkaline phosphates do not measure liver function at all.1 The title gives an impression that this study is only assessing hepatic dysfunction; though the parameters studied cannot be considered as strong evidence in favour of the same. The impact of co-infection with hepatitis viruses or concomitant hepatotoxic drugs was not assessed in this retrospective study. The aetiology of elevated aminotransferase levels during acute dengue illness is unclear since AST is expressed in the heart, skeletal muscle, red blood cells, kidneys, brain, and liver, while ALT is secreted primarily by the liver.2-3 Because dengue infection can cause acute damage to these non-hepatic tissue types that express AST, raised aminotransferase levels may not be entirely due to severe liver involvement. It is therefore possible that the patients with high AST levels were also more likely to be classified as severe dengue under the 2009 criteria due to the common pathways to non-hepatic tissue damage, even though there is no association with poorer outcome. In this study not every patient with elevated AST or ALT was comprehensively evaluated for other aetiologies of viral and non-viral hepatitis.

The finding of high AST in 100% cases cannot be strong evidence to conclude that all dengue patients had liver dysfunction; since the cases included are not screened for alcohol, drug induced or other viral infections related rise of transaminases and other co morbid conditions. Elevation AST more than ALT is well reported finding in almost all published data and is usually a transient phenomenon.

The study reports that 60% dengue cases had ascites; however, there is no data provided on clinical and investigations in this subgroup of patients. In table 2 the comparison between DHF and DF parameter ascites is not included. Ascites in Dengue fever is manifestation of endothelial damage leading to leakage of fluid, which an indicator of severity of dengue fever.4 The data does not reveal if ascites was present in all DF or DHF. It would be interesting to know the distribution of these cases of ascites among DF and DHF category. Analysis of fluid and exclusion of other likely causes would have been more emphatic to accept that these are due to mild form of dengue.

References


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Reply from Author

Vaibhav Shukla

Sir,

We appreciate the critical appraisal of Dr AA Bamanikar with regard to our article “A study of hepatic function in dengue” and would like to clarify the following points:

1. Dr Bamanikar has quoted Harrison’s textbook in saying that “in fact many tests such as aminotranferases and alkaline phosphate do not measure liver function at all”. But he seems to have missed the immediate next line in Harrison in the same chapter which says “rather they detect liver cell damage or interference with bile flow”. In the same chapter the authors say that aminotransferases are sensitive indicators of liver cell injury. So to say that aminotransferase measurement is not indicative of liver disease/dysfunction is inappropriate.

2. AST rise can be due to sources other than liver, but 91% of our patients also had concomittent rise of ALT which is primarily secreted by the liver. So when there is a simultaneous rise of AST and ALT one should consider liver as the source rather than other causes.

3. None of our patients were on any hepatotoxic drugs at the time of admission. We did not look for a viral aetiology for hepatitis as we were dealing with confirmed cases of dengue who had an average serum bilirubin 0.9 mg% and aminotranferases in the range of 100-300 IU, which is an unlikely presentation of acute viral hepatitis. The same chapter in Harrison says that in both viral and drug induced hepatitis serum bilirubin is raised and aminotranferases are often more than 500 IU and ALT is more than AST which is not what we found in our patients. With regard to alcoholic liver disease, most of our patients were young (mean age 28 years) and did not give history of regular, chronic consumption of alcohol. Besides hyperbilirubinaemia is common in alcoholic liver disease and there is modest increase in alkaline phosphate levels as well.

4. With regard to ascites, it was present in all patients of DHF and 20 patients out 48 in DF. As already mentioned in the article since the ascites was minimal to mild in most cases analysis of fluid was not done.

Our study was based on certain findings which we observed in dengue patients in one particular year. There are other tests which assess liver dysfunction, but we presented the data of certain parameters which we had observed and which are important for assessment of liver disease.

References

1. Pratt DS, Kaplan MM Evaluation of Liver Function, Harrison’s principles of internal medicine 18th edition editors longo et al vol 2 chapter 302, pages 2527-31
2. Mailliard ME Sorrell MF Alcoholic liver disease, Harrison’s principles of internal medicine 18th edition editors longo et al vol 2 chapter 307, pages 2590-2607