Ischemic hepatitis: A severe concern

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Dear Editor,

I read the review article by Waseem N and Chen PH (2016) about hypoxic hepatitis, also called ischemic hepatitis or shock liver [1]. The authors described the current concept, pathophysiology, clinical and anatomopathological features, diagnostic tools, and prevention and management of this ominous condition [1]. Ischemic hepatitis affects men at mean age of 64-70 years; the mortality rate is >50% [1-3]. The general incidence is 2 per 1000 patients, but in ICUs it may be up to 2.5 per 100 [1]. Diagnostic criteria include: cardiac, circulatory, or respiratory failure; dramatic transient rise of aminotransferase levels; and exclusion of other causes of liver cell necrosis [1-3]. The aminotransferase and lactate dehydrogenase levels typically peak within 24 hours, drop to nearly half values in 24 to 72 hours, and normalize between 1 to 2 weeks [1-3]. Liver biopsy can confirm the diagnosis in doubtful cases by demonstrating centrilobular necrosis [1-3]; however this procedure may be hazardous in presence of coagulopathy. Therapy directed to liver is unavailable, and one must correct predisposing factors [1-3].

The five case studies by Damasceno TA et al. (2016) also merit comments [2]. The authors described classical ischemic hepatitis following cardiopulmonary bypass, and suggested new researches about liver dysfunction associated with cardiac surgery [2]. All patients were males, 60-67 years old, and developed acute perioperative liver failure. As classically reported, the aminotransferase levels were 10-20 times the normal, lactate dehydrogenase and bilirubin were elevated, and coagulation factors were deficient [1-3]. Worthy of note was the characterization of five cases in the interval of ten months, because this condition has been considered of low incidence with scarce case reports [2].

In 2010, Brazilian authors described the case of a 69 year-old woman who died with ischemic hepatitis due to a ruptured hepatic aneurysm. The aneurysm was found during investigation of jaundice and abdominal pain [3]. Laboratory tests on Days 1, 6 and 10 showed: AST (U/L) 2,392, 17.4, and 52.7; ALT (U/L) 2,797, 18.0, and 139.0; total bilirubin (mg/dL) 3.0, 2.7, and 1.7 [3]. The giant aneurysm caused portal vein compression, but the extensive liver ischemic necrosis was related to a hepatic arterial thromboembolism resulting in centrilobular necrosis without inflammatory changes [3]. Complementary tests and total necropsy findings ruled out other possible etiologies [3].

These studies enhance the suspicion index about the scarcely reported condition.

Conflict of Interest

There is no financial interest or conflict of interest to disclaim.
REFERENCES

