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ORIGINAL ARTICLE

Acute liver ischaemia after gastro-oesophageal variceal bleeding☆

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KEYWORDS
Ischaemia;
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Abstract
Introduction: Variceal upper gastrointestinal bleeding (UGIB) can trigger acute hypoxic hepatitis (AHH). The aim of this study was to analyse the incidence, associated risk factors and mortality of AHH after variceal UGIB.
Patients and methods: Retrospective study of cirrhotic patients with variceal UGIB, classified into two groups according to the development of AHH. AHH was diagnosed when AST and ALT reached levels 10 times above the upper limit of normal, after ruling out other causes of hepatitis. The standard initial treatment consisted of haemodynamic support, emergency endoscopy with rubber band ligation, somatostatin and antibiotics. In the case of failure of primary haemostasis, a transjugular intrahepatic portosystemic shunt (TIPS) was implanted. Both groups (AHH and non-AHH) were compared.
Results: Sixty-eight cirrhotic patients with variceal UGIB admitted to the gastroenterology department of Hospital Ramón y Cajal between January 2007 and March 2012 were analysed. Eleven of these patients (16.2%) developed AHH. Univariate analysis showed the following items as risk factors: diabetes (OR: 7.5; CI: 1.9–29), shock (OR: 8.5; CI: 2.06–34) and persistent bleeding (OR: 9.0, CI: 1.6–49, $p = 0.03$). However, multivariate analysis confirmed only diabetes (OR: 8.61; CI: 1.4–52.5) and shock (OR: 7.58; CI: 1.26–45.51) as risk factors. Mortality rate in the AHH group was 45%, compared to 10.5% in the non-HAA group ($p = 0.012$).

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Conclusions: AHH after variceal UGIB occurred in 16.2% of cirrhotic patients and was associated with a poorer prognosis, with a mortality rate of 45%. Our findings suggest that diabetes and shock are risk factors for the development of AHH. Early identification of at-risk patients could therefore help prevent AHH.

PALABRAS CLAVE
Isquemia;
Fallo hepático;
Hemorragia;
Varices: hipertensión portal

Isquemia hepática aguda tras hemorragia digestiva alta por varices esofagogástricas

Resumen
Introducción: La hemorragia digestiva alta por varices esofagogástricas (HDA por VEG) puede desencadenar una isquemia hepática aguda (IHA). El objetivo de este estudio fue analizar la incidencia de IHA tras una HDA por VEG, los factores de riesgo y su mortalidad.
Pacientes y métodos: Estudio retrospectivo sobre pacientes cirróticos con HDA por VEG. Se clasificaron en 2 grupos, determinados por el desarrollo o no de una IHA. Definimos IHA como AST y ALT por encima de 10 veces el valour basal, descartando otras causas de hepatitis aguda. El tratamiento inicial estándar fue soporte hemodinámico, endoscopia urgente con ligadura con bandas y/o escleroterapia, somatostatina y antibióticos. En caso de fracaso de estas medidas, se recurrió a la implantación de una derivación portosistémcica percutánea intrahepática (DPPI). Ambos grupos (IHA y no-IHA) fueron comparados.
Resultados: Durante un periodo de 5 años, se recogieron 68 pacientes con HDA por VEG. La incidencia de IHA fue del 16,2%. Tras el análisis univariante, los factores asociados con IHA fueron la diabetes mellitus (OR: 7,5; IC: 1,9-29), shock (OR: 8,5; IC: 2,06-34) y la persistencia de la hemorragia (OR: 9, IC: 1,6-49, p = 0,03). En el análisis multivariante solo mostraron significación estadística la diabetes mellitus (OR: 8,61; IC: 1,4-52,5) y el shock (OR: 7,58; IC: 1,26-45,51). La mortalidad del grupo de IHA fue mayor (45%) que en el grupo no-IHA (10,5%) (p = 0,012).
Conclusiones: La IHA tras una hemorragia digestiva por VEG en el paciente cirrótico ocurrió en el 16,2%, asociándose con un peor pronóstico y una mortalidad del 45%. Nuestros resultados sugieren que la diabetes mellitus y el shock hipovolémico son factores de riesgo para el desarrollo de IHA. La detección precoz de estos pacientes en riesgo podría por tanto ayudar a prevenir la IHA.

Introduction

Acute hepatic ischaemia, also called ischaemic or hypoxic hepatitis, is the result of hypoxia of the liver tissue, causing acute centrilobular necrosis.[1] Ischaemia-hypoperfusion of liver tissue is not the only mechanism involved, so many authors prefer the name acute hypoxic hepatitis (AHH) for this entity.[2] The liver is much more resistant than other organs to hypoperfusion due to its dual blood supply system, with 75% from the portal vein and 25% from the hepatic artery. However, AHH can occur in different conditions such as cardiovascular disease, respiratory failure or circulatory shock (sepsis, hypovolaemia). A prospective study conducted in an intensive care unit (ICU) over a 10-year period found that incidence of AHH could be as high as 0.9%.[3] The severity of this entity is determined by the underlying disease, and can reach a mortality rate of 72%.[4]

AHH is diagnosed when transient markedly elevated transaminases are observed within a compatible clinical picture such as those mentioned above, ruling out other causes of elevated enzymes.[5] This cut-off point has been debated, and an increase in transaminases of up to 10 times the upper limit of normal is considered reasonable for the diagnosis of AHH.[6]

Cirrhotic patients have impaired liver function, so it seems logical that they might be more susceptible to the development of AHH than patients with no previous liver disease. As a result of portal hypertension, cirrhotic patients can develop gastric and/or oesophageal varices (GEV) during the course of their disease. Gastrointestinal (GI) bleeding in these cases has a mortality of 15% per se, which can increase to 80% in the event of development of AHH.[7–9] According to Amitrano et al.,[9] incidence of AHH in cirrhotic patients following an upper gastrointestinal bleed (UGIB) varies from 1.5% to 12%.[9] Factors that have been related with higher mortality are high international normalised ratio (INR), sepsis-related organ failure assessment (SOFA) score, renal replacement therapy and septic shock.[10,11]

The aim of this study was to analyse the incidence and characteristics of AHH after a variceal UGIB, and to assess the associated factors.