Introduction

Hepatic dysfunction is a common finding in critically ill patients and is associated with poor outcome ***(Kramer et al., 2007)***. Serum aminotransferases serve as an indicator of hepatocellular damage and liver cell necrosis. Up to the mid-1990s viral and drug-induced hepatitis were considered the most frequent underlying diseases contributing to acutely and massively raised aminotransferase levels ***(Kaplan, 1993)***. However, several studies changed the clinical perception of acute hepatic injury: they concordantly identified hypoxic hepatitis **(HH)**, also known as ischemic hepatitis or shock liver, as the most frequent cause of notably raised aminotransferases in hospital and in the intensive care unit **(ICU)** ***(Whitehead et al., 1999)***.

HH is characterized by centrilobular liver cell necrosis as consequence of hypoperfusion with subsequent ischemia and passive congestion of the liver, severe systemic arterial hypoxemia, and/or impaired hepatic oxygen extraction ***(Ebert ,2006).*** The main underlying conditions are low cardiac output (**CO)** and sepsis ***(Fuhrmann et al. ,2009)***, although a recent prospective study has demonstrated that more than one underlying event causing HH was present in 74% of all patients ***(Fuhrmann et al. ,2009)*** .

So hypoxic hepatitis can be defined as a liver injury characterized by a centrilobular liver cell necrosis with a rapid increase in serum aminotransferases ***(Raurich et al. ,2009)***. A rapidly resolving elevation of serum enzyme activities, a profound fall in prothrombin activity, and an altered renal function form a triad of biochemical abnormalities that suggest a diagnosis of hypoxic hepatitis ***(Fuhrmann et al. , 2009)***.

The following clinical settings were compatible with the diagnosis of hypoxic hepatitis: acute heart failure, septic shock, trauma/hemorrhagic shock, obstructive shock, cardiac arrest, exacerbated chronic obstructive pulmonary disease, and arterial hypoxemia ***(Fuhrmann et al. , 2009)***.

❖ incidence :

The incidence of HH varies depending upon the underlying setting. According to the literature, HH was a rare finding on normal wards, with a reported incidence of less than 1% ***(Fuchs et al., 1998)***. A much higher proportion of HH can be expected in patients undergoing cardiovascular surgery and in cardiac care units, where the incidence increases up to 22% in patients with decreased cardiac output ***(Henrion et al., 1994)***.

In a recently performed a prospective epidemiological study the incidence of HH was more than 11% in these critically ill patients ***(Fuhrmann et al., 2008)*** .

The intensive care unit mortality of patients with HH was about 55% ***(Birrer et al., 2007)***. Also a 30-day mortality rate of 52% has been reported ***(Henrion et al. ,2003)*** . In the two studies the one-year survival rate was only 25% ***(Henrion et al. ,2003 , Birrer et al., 2007)***.

The aim of the work

The purpose of this review is to provide an update on the epidemiology, Pathophysiology, diagnostic and therapeutic options of HH.