

# INTRODUCTION

Perinatal asphyxia is one of the most devastating complications associated with the process of birth. Medical complications that arise from this condition affect not only the brain, but also many other organ systems critical for the maintenance of life. An increased understanding of the multifactorial nature of this disease will help all health care workers to improve care for patients with this condition (*Frank et al., 2005*).

Hypoxic injury of liver cells is seen after a reduction in Systemic blood flow, hypotension and/or cardiac failure (*Henrion, et al., 2003*). Hypoxic hepatitis, also known as shock liver, is a disorder seen after hypoxic liver injury in adults (*Fuchs, et al., 1998*). It is defined as an early, sharp and transient increase of serum transaminases "*alanine aminotransferase (ALAT), and aspartate aminotransferase (ASAT)*" seen after acute cardiac or circulatory failure in the absence of viral hepatitis (*Karlsson, et al., 2006*).

An increase in liver enzymes after birth asphyxia is well known and frequently seen at the NICU. However, few previous studies have addressed the temporal pattern of hepatic enzymes in clinical neonatal asphyxia is known, it would be possible to distinguish enzyme elevations caused by other hepatic conditions (*Lachmann et al., 1993*).

The time course of the enzyme activity pattern in hypoxic hepatitis is similar between patients. A normal or slightly increased *S-ASAT* and/or *S-ALAT* close to the triggering event are followed by a peak concentration of aminotransferases and lactate dehydrogenase (*LDH*) in serum within 24 to 72 hs after the insult. After the peak, aminotranseferases levels return to near normal within 10 days (*Seeto et al., 2000*).

The prognosis of hypoxic hepatitis itself is good, and it rarely progresses into fulminant hepatic failure (*Hankins et al., 2000*). Severe birth asphyxia frequently induces multiorgan failure with cardiovascular, renal, cerebral and hepatic damage (*Shah et al., 2000*).