

Biochemical Changes in Burns

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Abstract:

Background: Burns are the second leading cause of death resulting from childhood injuries. It is estimated that there are approximately 3,000 pediatric deaths annually due to burns and probably three times as many disabling injuries. Liver is one of the major organs affected by thermal injury. Objective of the study was to measure liver enzymes and serum electrolytes.

Results: In the present study we found (highly significant) 23 times elevation in AST, 21 times in ALT, 4 times increase in ALP, 1.7 times decrease in sodium and 1.4 times increase in potassium levels.

Conclusion: An increase in edema formation in burns patients may lead to cell damage, with the release of the hepatic enzymes. Liver enzymes, such as aspartate aminotransferase (AST) and alanine amino transferase (ALT) are the most sensitive indicators of hepatocyte injury. Cellular injury or changes in cell membrane permeability, leak these enzymes into circulation. Low Na & high K⁺ occurs because of increased vascular permeability and cell edema. This enzymes might be the predictive markers of prognosis in burns patients.

Keywords: liver enzymes, electrolytes, burns

1. INTRODUCTION

Burn injuries represent an extremely stressful experience and constitute a major concern in pediatric age group with respect to morbidity and mortality. After a thermal injury, a variable degree of liver injury is present and it is usually related to the severity of the thermal injury. Thermal injury can cause liver damage by several mechanisms (hypoperfusion, proinflammatory cytokines, or other signals of cell death, formation of oedema, and fatty changes). Burn injuries are of major concern in children with respect to morbidity and mortality. It is the sixth leading cause of death among children less than 14 years, worldwide. Burn damages cell membrane, causes loss of cell integrity and membrane permeability which brings major changes in serum electrolytes. Liver, kidney and pancreas are among the most vulnerable organs in burn trauma, markers of which help to assess the pattern and severity of injury. Some studies report that significant abnormal laboratory values are uncommon even in severely injured burn patients, which needs to be clarified.

A few studies have reported the results of serum analyses that have assessed static liver dysfunction among burned patients. In a group with 30% TBSA and 15% mortality, hepatic dysfunction was detected in 3.5% [1]. Another study showed a 36% incidence of liver failure, the reported onset of which was in the second week after a lethal burn injury [2]. In a recent paper, enlargement of the liver and impaired protein synthesis have been shown to develop early among children with major burns, and the extent of that dysfunction correlated with the size of the burn. The activities of hepatic enzymes (serum aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were increased two-fourfold immediately after the burn, and took four to six weeks to return to their reference range, which suggested burn-induced liver damage [3].

Study by MGHalaet al suggests an elevated AST, ALT, ALP AND gamma GT –because of impaired hepatic functions in burns patients (4). Bhagwat VR et al measured hepatic enzymes serially on admission and reported AST and ALT showed elevation by day 5, and decline by day 10. ALP and amylase showed a rise by day 10. There was a correlation of burn size and enzyme levels (5). Anandani JH et al reported a low sodium & high potassium levels in burns patients (6).

1.1. Objective

- Measurement of biochemical parameters in pediatric burns patients at the end of one week so as to highlight the importance of analysis of biochemical parameters in burns.

2. METHODOLOGY

Research Design : Facility based cross sectional study

Setting of the study : **Indira Gandhi Medical College & Research Institute**

Indira Gandhi Government Post Graduate Institute

Population : Children (< 18 yrs) admitted in IGGPGI with burns and age and sex matched healthy controls

Sample size : 30 burns patients and 30 age and sex matched healthy controls

2.1. Sampling Criteria

2.1.1. Inclusion Criteria

Pediatric burn patients admitted in burns ward of IGGPGI due to thermal injury. Thirty patients with thermal burns involving 20% TBSA or more at the end of first week were included in the study.

2.1.2. Exclusion Criteria

1. Burns other than thermal cause
2. Those who are not willing

After obtaining the permission from the concerned authorities, both the institutional ethics committees, an informed consent from parents/caregivers was taken.

2.2. Sample Collection With Aseptic Precautions for Lab Investigations

Biochemical parameters: Electrolytes, Hepatic enzymes at 7th day. Aspartate amino transferase, alanine amino transferase and alkaline phosphatase were estimated by enzymatic method using auto analyzer (XL -640). Sodium and potassium were estimated by ion selective electrode method using Rosche electrolyte analyser.

2.3. Statistical Analysis

Student unpaired 't' test is applied. The data were expressed as mean ± standard deviation. The level of significance was taken at p < 0.05.

3. RESULTS

We found 23 times elevation in AST, 21 times in ALT, 4 times increase in ALP, 1.7 times decrease in sodium and 1.4 times increase in potassium levels.

Table I. Comparison of hepatic enzymes AND electrolytes between pediatric burns and normal children

Liver enzymes	Pediatric Burns	Normal controls
AST (U/L)	750±43*	32±10
ALT(U/L)	980±57*	47±12
ALP(U/L)	680±38*	167± 17
Electrolytes		
Na+(mmol/l)	115±7**	135±7
K+(mmol/l)	6.5±2.0**	4.5±1.0

* P < 0.0001

** P < 0.01

4. DISCUSSION

We have obtained an highly significantly elevated liver enzymes AST, ALT, ALP significantly low sodium and high potassium levels. Similar findings were reported by previous studies (4-6).

Burns lead to an increase in edema formation that in turn leads to cell damage, with the release of hepatic enzymes [7]. The three enzymes that achieve abnormal serum levels in hepatic diseases and during the aftermath of a severe injury are alkaline phosphatase, serum glutamic oxalacetic

transaminase (SGOT), and serum glutamic pyruvic transaminase (SGPT). Serum aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase (ALP) are elevated between 50 to 200% when compared with normal levels. It has been observed that serum AST, ALT, and ALP peaked during the first week post-burn and approached the normal range 3–5 weeks post-burn. If liver damage persists or sepsis occurs, enzymes stay elevated or increase again [7-9].

Low Na & high K⁺ occurs because of increased vascular permeability, oozing of plasma from injuries – increased osmotic pressure in burn tissue – shifting of fluid – cell edema. Potassium level increases because of massive tissue necrosis, breakdown of RBCs.

The liver has been shown to play a pivotal role after a thermal injury (10, 11). An increase in edema formation may lead to cell damage with the release of the hepatic enzymes (11). Reports showed that AST and ALT are increased and released into the serum for a period of 4 to 6 weeks, indicating that liver damage is present immediately after burn. Serum bilirubin level was only increased for 2 weeks after burn, indicating that bilirubin level during the post burn response is not an important marker as in other pathophysiological states, such as sepsis (12). In sepsis, an intrahepatic cholestasis occurs frequently without demonstrable extrahepatic obstruction. This phenomenon has been described in association with a number of processes, such as hypoxia, drug toxicity, or total parenteral nutrition (13). The mechanisms of intrahepatic cholestasis seem associated with an impairment of basolateral and canalicular hepatocyte transport of bile acids and organic anions (14). This is most likely caused by decreased transporter protein and RNA expression. Jeschke MG et al (15) and Bolder et al. (14, 16) have shown that decreased transporter expression is associated with decreased bile acid output, leading to increased intrahepatic bile concentration. Liver damage has been associated with increased hepatocyte cell death, which is caused by increased hepatocyte apoptosis and necrosis (17-20).

5. CONCLUSION

An increase in edema formation in burns patients may lead to cell damage, with the release of the hepatic enzymes. Liver enzymes, such as aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are the most sensitive indicators of hepatocyte injury. Both AST and ALT are normally present in low concentrations. However, with cellular injury or changes in cell membrane permeability, these enzymes leak into circulation. Of the two, the ALT is the more sensitive and specific test for hepatocyte injury. These enzymes may be predictors of prognosis in burns injury.

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