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HYPERBILIRUBINEMIA IN PATIENTS WITH LONG BONE FRACTURES

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ABSTRACT

BACKGROUND: Hyperbilirubinemia is defined as excess of bilirubin in the blood, occurring as a result of liver or biliary tract dysfunction or with excessive destruction of red blood cells.

AIMS AND OBJECTIVES: To evaluate the levels of serum bilirubin in patients with long bone fractures.

MATERIALS AND METHODS: 200 participants were enrolled in the study. 100 patients with long bone fracture were taken as study group and 100 age and sex matched healthy participants were taken as control group. The serum samples of the patients were used for measuring various parameters. Total and direct bilirubin were measured by Photometric test using 2, 4 dichloroaniline. Alkaline phosphatase, Alanine transaminase and Aspartate transaminase enzyme levels were measured by Optimized UV test according to International Federation of clinical chemistry and Laboratory medicine.

RESULTS: We found an increase in levels of Bilirubin, SGOT and SGPT in patients with long bone fractures compared to the control group.

CONCLUSION: The study concluded that patients with long bone fractures developed hyperbilirubinemia and hepatic dysfunction. Hence it is important to evaluate bilirubin levels early so that patients with long bone fractures can be prevented from developing sepsis or other liver complications.

KEY WORDS: Bilirubin**,** Fractures, Hyperbilirubinemia, Liver dysfunction

INTRODUCTION

Hyperbilirubinemia is commonly observed in patients with trauma, even in the absence of preexisting hepatobiliary disease[1]. Bilirubin is the ultimate breakdown product of haemoglobin and serves as a diagnostic marker of liver and blood disorders[2].

In clinical practice, serum bilirubin levels are the most commonly used criteria to establish the presence of hepatic dysfunction. The disturbance of liver function can be caused by several factors, including massive transfusion, hematoma in tissues, shock, heart failure, anoxia, infection and perhaps medications[3]. It is important to know about hepatic dysfunction in trauma patients because it is associated with greater consumption of hospital resources, frequent development of complications and increased mortality and morbidity[4]. It is a known fact that in patients with abdominal trauma the bilirubin levels are elevated, but very few studies are found with bilirubin levels in patients with long bone fracture. Hence we planned to study the bilirubin levels in patients with long bone fractures.

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MATERIALS AND METHODS

The study group comprised of 100 patients with long bone fractures in the age group of 20-60 years admitted in Sanjay Gandhi Institute of Trauma and Orthopedics, Bangalore. The diagnosis of long bone

fractures was done by senior orthopedic surgeons and confirmed by radiological images. As a control group 100 age and sex matched healthy participants were taken. The study was conducted from February 2015 to June 2015 at department of Biochemistry, Sanjay Gandhi Institute of Trauma and Orthopedics, Bangalore. The research protocol has been examined and approved by institutional ethics committee.

After obtaining informed written consent, 10ml venous blood sample was collected from the participants under aseptic conditions on admission. The serum samples were used for measuring various parameters. Total and direct bilirubin was measured by Photometric test using 2, 4 dichloroaniline[2]. Alkaline phosphatase[5], Alanine transaminase[5] and Aspartate transaminase[5] enzyme levels were measured by Optimized UV test according to International Federation of clinical chemistry and Laboratory medicine.

Exclusion criteria

Patients with known hepatic diseases, hemolytic anemia's, intra and extra hepatic biliary obstruction, known alcoholics, patients on drugs causing hepatitis and cholestasis, were excluded from the study.

Statistical analysis

The results are expressed as mean ± SEM. The results are further subjected to students't' test, differences between means are considered significant at $p< 0.05$.

RESULTS

The study found that there was an increase in levels of Bilirubin, SGOT and SGPT in patients with long bone fractures when compared with the control group as shown in Table no 1.

DISCUSSION

Hyperbilirubinemia is commonly observed in patients with trauma. The causes contributing to the development of hyperbilirubinemia can be divided into prehepatic, hepatic and post hepatic causes[2]. The disturbances in liver function can be caused by several factors, including massive transfusion, hematoma in tissues, shock, heart failure, anoxia, infection and medications[6].

This study found that the serum levels of enzymes SGOT and SGPT were increased in patients with trauma (Table 1). Champion HR[7] et.al also showed similar findings. The increase in the enzyme levels indicates mild hepatic dysfunction. This might be attributed to decreased perfusion of the liver following trauma induced blood loss, resulting in mild hepatic dysfunction.

In this study we found that in patients with long bone fractures the bilirubin levels were increased (Table 1). These findings are in agreement with Nakatani T[8] et al. It is considered that impairment in the most energy requiring process of bilirubin metabolism, excretion of conjugated bilirubin from cytosol to capillary bile duct, due to post-traumatic hepatic mitochondrial dysfunction, followed by the reabsorption of conjugated bilirubin into the blood stream, would be one of the factors responsible for post traumatic hyperbilirubinemia.

It is also found in some animal studies that large intravenous loads of unconjugated bilirubin can cause extensive hepatocyte canalicular membrane damage and intrahepatic cholestasis[9]. This might be the reason for increased conjugated bilirubin levels in trauma patients

The post traumatic mild liver dysfunction might also be the cause for increase in the bilirubin levels in these patients. It is found that hyperbilirubinemia

Table no 1: Biochemical parameters in Cases and Control participants.

Sl. No	Parameters	Cases(n=100)	Controls(n=100)	p value
1	Age (years)	38.8 ± 1.523	40.83 ± 1.43	0.337**
2	Total Bilirubin (mg/dl)	1.610 ±0.07	0.56 ± 0.01	<0.0001*
3	Indirect Bilirubin (mg/dl)	1.09 ± 0.06	0.40 ± 0.01	<0.0001*
4	Direct Bilirubin (mg/dl)	0.49 ± 0.02	0.16 ± 0.01	<0.0001*
5	SGOT (IU/L)	64.47 ± 2.40	17.54 ± 0.41	<0.0001*
6	SGPT (IU/L)	55.36 ± 6.334	17.70 ± 0.41	<0.0001*
7	ALP (IU/L)	104.2 ± 6.334	65.54 ± 1.52	<0.0001*

*p<0.05 – Significant, **p>0.05 – Non Significant, n = number of participants

is usually associated with bacterial infections and sepsis as concluded by K.J.Labori[10] et al. Bilirubin itself could be causally related to sepsis development, because of its anti oxidative properties, bilirubin impairs the bactericidal activity of neutrophils and reduces bacterial killing rates in a dose – dependent manner[11]. Hence it is important to monitor the bilirubin levels in trauma patients.

There are studies which show that liver dysfunction is associated with a poor outcome independently of other organ dysfunctions[12]. Since strategies to support liver function are minimal, a timely and accurate identification of factors promoting liver dysfunction may lead to prevention or attenuation of its consequences.

It is known that in patients with abdominal trauma the bilirubin levels are elevated, but in this study we observed that even in patients with long bone fractures hyperbilirubinemia is seen. Thus early measures should be taken to detect hyperbilirubinemia and also take adequate measures to prevent liver dysfunction. These measures can prevent morbidity in these patients.

The study concluded that patients with long bone fractures developed hyperbilirubinemia and hepatic dysfunction. Hence it is important to evaluate bilirubin levels early so that patients with long bone fractures can be prevented from developing sepsis or other liver complications.

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