Original Article

ADVANCES IN BIORESEARCH
Volume 2, Issue 2, December 2011: 79 - 81
ISSN 0976-4585
Journal's URL: www.soeagra.com/abr.htm
[Accepted 18 December 2011]

Study of Certain Biochemical Parameters in Patients of Sickle Cell Anemia

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ABSTRACT
Sickle Cell is an inherited blood condition in people of Central India, Africa, Medditarean and Middle East region. It is caused by abnormalities in the production of haemglobin. Sickle Cell Disease occurs when people inherent abnormal genes responsible for haemoglobin production from their parents. Present study was carried out to know the relationship between pathogenicity of sickle cell anemia and renal and hepatic disorder. We found that out of 117 samples, Urea concentration was found above than normal range in 46 (48.20±0.80 mg/dl) samples, Creatinine in 39 (2.4±0.32mg/dl) samples, Bilirubin in 55 (1.6±0.2mg/dl) samples, SGPT activity was also found raised in 36 (48.0±2.4 IU/l) samples. Protein metabolism was also found altered. In 19 samples Total protein was found decreased (12.60±1.30 mg/dl) and Albumin was found decreased in 7 samples (6.34±0.24 mg/dl). Thus the present finding indicates a clear adverse effect on renal function and moderate adverse effect on hepatic function under stress of sickle cell anemia.
Key words – Sickle cell anemia, Urea, Creatinine, Bilirubin, SGPT, Total Protein, Albumin

INTRODUCTION

Sickle cell disorder is a hemoglobinopathy caused by point mutation in the β chain of the globin gene [1]. It is an autosomal recessive inheritance, and clinical severity varies widely from the sickle cell trait (heterozygous) to sickle cell anemia (homozygous) [2]. The mutant hemoglobin undergoes aberrant polymerization on deoxygenation, resulting in permanent distortion of the red blood cell (RBCs) into characteristic irreversible sickle cell [3]. Since the sickle cell gene is usually seen among people of African origin in North and South America , the Caribbean and Europe, it is commonly believed to be associated with African ancestry and Africa is supposed as place of origin of sickling. There have been at least three independent occurrences of Sickle cell gene in Africa known as *haplotypes* and named after the areas where they were first described viz. Benin, Central African Republic or Bantu and Senegal. However the fourth independent occurrence of the sickle cell mutation occurred in Asia and is shared by people in Eastern province of Saudi Arabia and Central India and is known as *Asian haplotype* [4]. In India the sickle cell gene has been reported in 73 % of studies among tribal people, 17 % among lower castes, 9 % among middle castes and 1% among higher castes [5]. Sickle cell traits present with varied clinical problems including increased urinary tract infection, gross hematuria, complication of hyphema, splenic infarction with altitude hypoxia or exercise, and life threatening complications of exercise, exertional heat illness (excretional rhabdmyolysis, heat stroke or renal failure) or idiotypic sudden death [6,7]. Based on prevailing symptoms of hepatic and renal disorder in the present study we considered to test creatinine, Urea, SGPT, Bilirubin, total protein and albumin from sickle cell anemia patients.

METHODOLOGY

For the present study 2 ml intravenous blood were collected from total 117 sickle patients by paramedical staff from different locations of Rajnandgaon District of Chhattisgarh India following ICMR and Institutional Ethical Committee norms and kind consent of donors. Immediately after collection of blood it was centrifuged at 3000 rpm for separation of serum for biochemical analysis. Following parameters viz. Urea (by Photometric method), Creatinine (by Two point reaction method),Bilirubin (by Jendrassic and Grof method)[8], SGPT (by End point reaction), Total protein (by Biurett method), Albumin (by Bromocresol green method) were analyzed using Autoanalyser (Merck) and commercial biochemical analysis kits by Merck.

RESULT AND DISCUSSIONS

The study showed that the concentration of urea in the blood of sickle patient was significantly raised in 46 samples beyond the normal limit (48.20±0.8 mg/dl; P< 0.05%) out of total 117 samples. Creatinine concentration was found significantly raised (2.4 ± 0.32 mg/dl; P< 0.05%) in 39 samples but in 9 samples it was found lowered (0.6±0.08 mg/dl; P>0.05%) than the normal concentration. Bilirubin, a product of liver metabolism, generally found between 0.2- 1 mg/dl in blood but in the present study we reported its raised conc. in 55 samples (1.6 ±0.2 mg/dl; P< 0.05%) out of 117. The finding attributes with liver function stress. SGPT activity was also found raised in 36 samples (48.0 ±2.4 IU/l; P< 0.05%) beyond normal limit. Increased activity of SGPT also attribute with liver and renal impairment.

Table I – Biochemical alterations in Sickle cell Anemia patients

S.No.	Parameters	Normal Range	Observed Value		
			High	Low	Normal
2.	Urea	10-40 µg/ml	36 (48.20 ±0.80)	-	81
3.	Creatinine	0.8-2 mg/dl	39 (2.4 ± 0.32)	9 (0.6 ± 0.08)	69
4.	Bilirubin	0.2 – 1ng/dl	55 (1.6 ±0.2)	-	62
5.	SGPT	5-40 IU/l	36 (48.0 ±2.4)	-	79
6.	Total Protein	15-40mg/dl	7 (56.20 ± 3.36)	19 (12.60 ± 1.30)	91
7.	Albumin	8-30mg/dl	6 (36.80 ± 0.86)	7 (6.34 ± 0.24)	104

Among sickled patients protein metabolism was also found disturbed. Two parameters were considered, total protein and albumin. Total protein was found raised in 7 samples (56.20 ± 3.36 mg/dl; P< 0.05%) and lowered in 19 samples (12.60 ± 1.30 mg/dl; P>0.05%)while the albumin conc. was found high only in 6 samples (36.80 ± 0.86 g/dl; P> 0.05%) and low in 7 samples (6.34 ± 0.24 mg/dl; P>0.05%) out of 117 samples and rest were found within normal limit.

Elevated levels of urea are generally observed in pre renal, renal and post renal condition. Creatinine is the end product of creatine metabolism. It is an anhydride of creatine found in muscle brain and blood in free from as well as in the form of creatinine phosphate. Creatinine is largely formed in muscle by the irreversible and non enzymatic removal of water from cratinine phosphate. Increase in serum creatinine is seen in any renal function impairment and its clearance is significantly reduced. The renal impairment may be due to intrinsic renal lesion, decreased perfusion of kidney or by obstruction of the lower urinary tract. Raised concentration in urea and creatinine suggest that pathogenicity of sickling might result renal impairment.

Bilirubin is a metabolic by-product of haemoglobin and serum total bilirubin is increased in hepatocellular damage (toxic hepatopathy , neoplasm), intra and extra hepatic biliary tract obstructions, intravascular and extravascular hemolysis etc. Disproportionate elevation of direct bilirubin is seen in cholestasis and late in the course of chronic liver disease. In the present study SGPT activity was also found elevated in 36 samples which suggest necrosis of hepatocytes or myocardial cells or erythrocytes or skeletal muscle cells. The decreased concentration of total protein and albumin is might be due to oxidative modification of glycoprotein carbohydrate moieties. Since the serum albumin and small fraction of globulin are synthesized in liver and generally in hepatocellular damage their concentration lowered down. So our finding suggests that under stress of sickle cell anemia hepatic impairment occurs. The conclusive finding is sickling may cause renal and hepatic damage in large scale in population.

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