Original Article

Study of SGOT, SGPT, LDH and Cardiac Troponin–I Level and Their Pearsons Coefficient Correlation Variations in Sickle Cell Disease

Chandrakant G. Kamble[1], Suresh Sharma[2], Bhausaheb V. Jagdale[3], Jagdish D. Powar[4]

[1]Associate Professor, Department of Biochemistry, Government Medical College, Sriganganagar, Rajasthan, India. [2]Associate Professor, Department of Anatomy, Government Medical College, Jhunjhunu, Rajasthan, India. [3]Professor Department of Physiology, S.M.B.T. Institute of Medical Sciences & Research Centre, Nashik, Maharashtra, India. [4]Assistant Professor, Department of Community Medicine, S.M.B.T. Institute of Medical Sciences & Research Centre, Nashik, Maharashtra, India

Abstract

Background: A point mutation (GAG→GTG) in the beta-globin chain causes glutamic acid (βs 6 Glu→Val) to replace valine in sickle cell disease (HbS), a blood-related genetic condition that is passed from parents. SGOT, SGPT, LDH, Troponin markers and Pearson's coefficient correlation were altered in SCD. **Material and Methods:** This work was carried out at the Center for Scientific Research and Development (CSRD) at People's University in Bhopal and the Department of Biochemistry at PCMSRC. Kit and colorimetric techniques were used to estimate the enzymes. **Results:** This study used SPSS software version 24 to calculate the mean standard deviation of cases compared to controls, which revealed a significant difference. The mean ± SD cases vs controls of SGOT, SGPT, LDH and Troponin –I were found 69.98 ± 69.31; 25.17± 5.25; 65.28 ± 60.07; 22.72 ± 5.47; 256.66 ± 69.56; 38.19 ± 20.04 and 0.0484 ± 0.0914 respectively. A statistically significant P<0.000 was discovered. **Conclusion:** SGOT, SGPT, LDH, and Troponin-I were significantly elevated and showed a positive correlation in sickle cell disease, suggesting that they may be another clinical biomarker for the diagnosis of SCD. SGOT, SGPT, LDH, and Troponin-I showed a positive association according to Pearson's association.

Keywords: SGOT, SGPT, LDH, Cardiac Troponin–I, Pearsons's coefficient, Sickle Cell Disease.

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Introduction

An inherited hemoglobin abnormality called sickle cell disease is brought on by both parents' faulty HbS genes. The disease is caused due to a single point mutation in the 2β-globin chain of the globins tetramer. Globin is the protein part which is composed of two pairs of globins chains and two subunits of 2α-globins chains and 2β-globin chains, that is denoted by α2β2 in each with its heme group. The disease is caused by the single point mutation, where glutamate is replaced by the valine at the 6th position in β-globin chains.[1] According to molecular or genetic research, sickle cell disease is a group of blood-related genetic disorders that are inherited from parents. It is distinguished by the presence of HbS, a single point mutation (GAG →GTG) at the sixth codon of the β-globin (HBB) gene, where glutamic acid (βs 6 Glu→Val) replaces valine at the beta-globin chain.[2,3]

In this study Serum Aspartate aminotransferase, Alanine amino transferase &, LDH, and I markers have been studied as promising molecules for better understanding of the sickle cell disease. SGOT, SGPT and LDH are the cardiac markers, both have equal sensitivity and specific in cardiac injury compared to other cardiac markers like troponin T and I, Along with sickle cell disease these markers are also elevated in conditions like trauma to the skeletal muscles and in liver diseases.[4-7]

Serum SGOT and SGPT: These markers are diagnostically significant liver enzymes called as Serum Glutamic Oxaloacetate Transaminase (SGOT/Sr.AST) and Serum Glutamic Pyruvic Transaminase (SGPT/Sr. ALT) respectively. Serum SGOT, SGPT, LDH and I are diagnostically significant liver enzymes all the three four are clinically important enzymes and these were found altered in the different hepatic and cardiac (stroke) abnormal conditions.[8-11]

Materials and Methods

The study's location and ethical approval: The study was carried out at People's University's Center for Scientific Research and Development Department (CSRD) Bhopal, and the Department of Biochemistry, People's College of Medical Sciences and Research Centre (PCMS & RC). The Departmental Doctorate Committee approved the study protocol. (Ref. No: PCMS/ BIOCHEM/ 2016/1447), Research Advisory Committee (Ref. No.: PCMS/ OD/ 2016/ 2566), Institutional Ethical Committee (Ref. No: PCMS/OD/2016/2551), and University Doctoral

Address for correspondence: Dr. Bhausaheb V. Jagdale, Professor Department of Physiology, S.M.B.T. Institute of Medical Sciences & Research Centre, Nashik, Maharashtra, India.
E-mail: bhausaheb.jagdale@gmail.com

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Study Design: This is a descriptive, hospital-based case control study.

Sample size estimation: The sample size was calculated taking into account Madhya Pradesh's 15–30% sickle cell disease prevalence (mean 23%).

The sample size was estimated according to the formula: $n = 4PQ/ L^2$

Where, P = prevalence of SCD, Q = 100-P, L = Alpha error

n = 4X23X77 / 8X8; So, N = 7084/64 = 110.68 =111.

Selection of Study Subjects: 222 human subjects were intended to participate in the study. The first test group included of 111 SCD patients and 111 age-matched healthy participants.

Inclusion criteria: Cases: Test group will be the diagnosed cases of SCD (SS and AS) and controls will be the: Age and sex matched healthy individuals as control.

Exclusion criteria: Acute illness like URTI / fever etc. Concomitant illnesses like IHD / DM / HTN Sickle cell anemia cases with acute crisis History of recent blood transfusion duration of less than 3 months will be excluded from the study.

Collection of Blood Samples: Blood samples were obtained from People's Hospital's inpatient and outpatient departments, Rajeev Gandhi Hospital, Civil Hospital Bairagarh, Civil and Hospital Baranpur districts of Madhya Pradesh. The subjects were enrolled in the study after the confirmation by the physician. These patients will be compared with healthy normal controls based on age, sex, dietary conditions, and their lifestyles. Under aseptic precautions and after due consent from the study subject, 8-10 ml venous blood was collected in plain bulb and EDTA bulbs. Following a 30-minute clotting period at room temperature, the blood samples were centrifuged for 10 minutes at 3000 rpm to separate the serum sample. The separated sample was then promptly kept at -20 0C in a deep freezer until further analysis.

Statistical Analysis: The z test, chi-square test, and Pearson's correlation coefficient were used for both descriptive and inferential statistical analysis. Graph Pad Prisam and SPSS 17-0 were the tools utilized for the analysis. The significance level is determined by the P value of less than 0.05. in the outcomes ($p< 0.05$).

Results

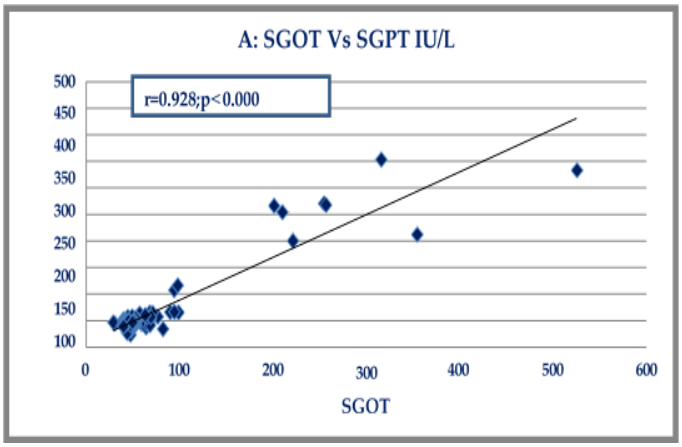
Table 1: Comparisons of SGOT, SGPT and LDH and Cardiac Troponin-I sin Patients & Controls Groups in Sickle Cell Disease.

Variables	Group	N	Mean	SD	't' value	'p' value	Significance level
SGOT (IU/lit)	Patients	111	69.98	69.31	6.79	<0.000	Highly significant
	Control	111	25.17	5.25			
SGPT (IU/lit)	Patients	111	65.28	60.07	7.43	<0.000	Highly significant
	Control	111	22.72	5.47			
Sr.LDH IU/Lit.	Patients	111	256.66	69.56	17.24	< 0.000	Highly significant
	Control	111	138.19	20.04			
Sr.Cardiac Troponin-I ng/dl	Patients	111	0.0686	0.0484	2.0608	< 0.040	Significant
	Control	111	0.0484	0.0914			

Discussion

Sickle cell disease is a communal hereditary hemoglobinopathy and a multi-system disease, associated with episodes of acute illness, progressive organ damage. It is also known as red cell disorder with multiple chronic complications. The complications occur due to multiple factors such as intrahepatic sinusoidal sickling, increased bilirubin level where in there is a need for the blood transfusion which can further cause infections like hepatitis or excess iron deposition. This difference in patho-physiological outcomes results in to several healthcare challenges.[12-13]

Aspartate amino transferase (SGOT) and other variables Pearson's Correlation coefficient in sickle cell disease: Sr. Aspartate amino transferase (SGOT) Vs Alanine amino transferase (SGPT): Graph A; presented the positive correlation between SGOT and GPT. The other studies also supporting with the same correlation that is Ahn et al (2005) Akuyam et al (2007) Mohamed et al (1993) also noted that SGOT, SGPT and Sr. ALP were significantly increased and it was a common finding in patients with sickle cell anemia with crises.[14-16]

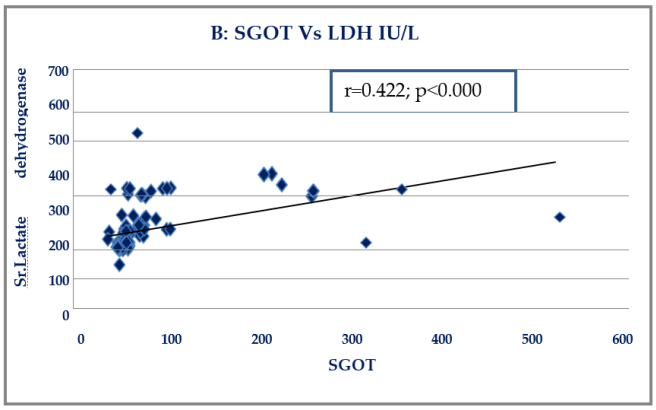


Aspartate aminotransferase (SGOT) Vs Lactate dehydrogenase (LDH):

Graph 'B' describes the positive and significant correlation between SGOT and LDH, here r = 0.422; p <0.000. Similarly, Brody et al (1975) and Johnson et al (1985) noted that there was a positive correlation between AST and LDH indicating some involving role of erythrocyte AST from hemolysis to the serum levels of AST.[17,18]

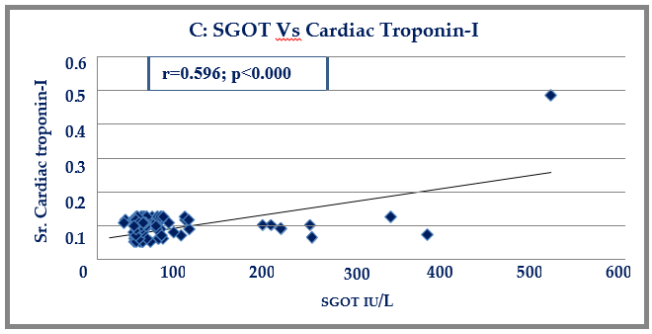
Aspartate aminotransferase (SGOT) Vs Lactate

dehydrogenase (LDH):



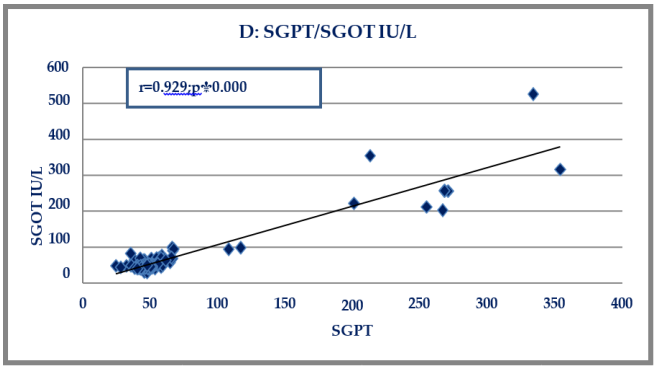
Sr. Aspartate amino transferase (SGOT) Vs Cardiac Troponin–I :

Gladwin et al (2004) studied and concluded that cardiac transaminases along with troponin-I were positively correlated in sickle cell disease.[19] r = 0.596 and p <0.000.



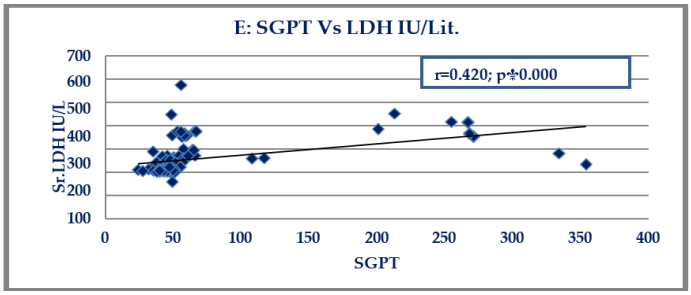
Sr. Alanineamino transferase (SGPT) Vs SGOT:

Scattered gram 'D' described that; there was the significant positive correlation between SGPT and SGOT. The other studies also supporting with the same correlation. Scientists Mohamed et al (1993) Ahn et al (2005) Akuyam et al (2007) also noted that serum SGPT, SGOT and Sr.ALP were significantly elevated and it was common finding in patients with sickle cell anemia with crises.[14-16]

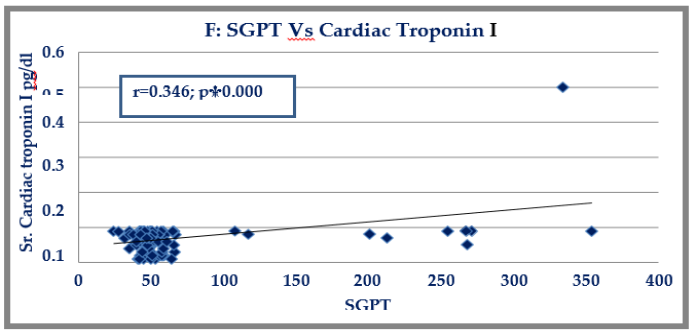


Sr. Alanine amino transferase (SGPT) Vs Lactate dehydrogenase (LDH): Scatter graph 'E' explaining the positive and significant correlation in between SGPT and LDH; r=0.420 and p<0.000 Similarly Brody et al (1975) and Johnson et al (1999) noted that, there is a positive correlation between ALT and LDH indicating some active role of erythrocyte ALT from hemolysis to these serum increased alanine amino transferase enzyme.[17,18]

E. Alanineamino transferase (SGPT) Vs Lactate dehydrogenase (LDH):



Sr. Alanineamino transferase (SGPT) Vs Cardiac Troponin – I: The scatter graph (F) indicates that there was a significant positive correlation between SGPT and in sr. cardiac troponin; r=0.346 and p<0.000. Similarly, scientist Gladwin et al (2004) cardiac transaminases along with troponin-I were positively correlated with sickle cell disease.[20] Mekontso-Dessap et al (2008) stated that the elevated sr. trponin-I is found in sickle cell disease with acute chest syndrome.[21]



SGOT, SGPT levels in sickle cell disease:

Since both SGOT and SGPT are found in liver cells, their activity has been utilized extensively in liver function tests to evaluate liver disorders. These enzymes are highly sensitive and dependable for the evaluation of liver function tests. These enzymes were released from the liver cells more often in sickle cell disease (SCD) because the sickling process produces organ damage to the liver cells.[6]

In the present study, it was observed that mean value of SGOT and SGPT were significantly higher in SCD patients as compared to healthy controls. [Table 1 and Graph –A]

Sickle cell illness is frequently associated with the multi-organ failure characteristic of the liver, which raises SGOT and SGPT levels. Because of the quick sickling of red blood cells in sickle cell disease (SCD), the transamination process was not finished, which prevented the enzymes from being used to convert keto acids and raised the blood's transaminase levels.[22]

There is a correlation between Richard and the outcomes of our investigation regarding liver function testing. According to S et

al. (2002), all SCD patients had statistically significant changes in the levels of liver enzymes (SGOT, SGPT) and other parameters when compared to controls ($p<0.0001$), and the levels of LFT parameters were significantly ($p<0.0001$) higher in cases than in controls.

Richard S. et al. discovered that there were no appreciable changes in the levels of liver function enzymes in the steady state of sickle cell disease. This could be because sickling and hemolysis were not detected quickly in the patients.[22] In their research, Mahera et al. (2009) and Gardner et al. (2014) found that sickle cell anemia patients had significantly higher LFT parameters,[15] and Gardner K. et al. (2014) similarly found that SCD cases had a slightly higher LFT.[23]

Lactate dehydrogenase is an enzyme which is found in all tissues including blood, muscles, kidneys brain and pancreas, involved in the respiration process and released during the tissue damage which increased in SCD. The sickle cell findings indicated that, the Sr. LDH level was found to be moderately increased and significantly very high in cases as compared to controls, since $p < 0.000$.

Kato GJ et al., (2017) reported that increased the Sr. LDH level was due to the continuous sickling of the red blood cells, affects to raptured RBCs and released the LDH from red blood cells. The intra vascular hemolysis associated with vasoconstriction, platelet activation, endothelial damage, vascular complications, and tissue damage found in organ damage of liver and kidney, which leads to increase sr. LDH in sickle cell disease. During vaso-occlusion, LDH rises due to the lysis of red blood cells, and hemoglobin level falls below its normal.[24]

In order to maintain the actin-tropomyosin complex, cardiac troponin-I, a protein found in both skeletal muscle and the heart, binds to actin via thin myofilaments. In relaxed muscles, it stops myosin from attaching to actin. Sickle Cell Disease is associated with multiple cardiovascular abnormalities, in the diagnosis of SCD electro gram which is sometimes not reliable and nonspecific.[25,26]

In some complications where the conventional serum cardiac enzymes measurements LDH, CK and SGOT were not helpful in diagnosis of myocardial infarction in sickle cell disease,[27] Then the cardiac troponins are the other tools which may raise in diagnosis of sickle cell disease.

According to the results in Table No. 8, sickle cell disease cases had a significantly higher level of serum cardiac troponin-I than controls.

Increased level of serum cardiac troponin- I might be due to the organ damage property and sickling phenomena, induces endothelial damage, inflammation and erythrocyte – leukocyte adhesion to the endothelium may play a cardinal role in progressive large- vessel vasculopathy causes to the peripheral tissue damage in the cardiac muscle, which further leads to increase serum cardiac troponin- I in the sickle cell disease.[4]

Our findings were correlated with the other studies, which have been included in the discussion with their hypothetical support: Haywood et al (2009) showed that the serum cardiac troponin-I levels were elevated in SCD patients going through the acute chest syndrome (ACS).[4]

The above studies are correlated with Mekontso-Dessap et al hypothesis they explained that the Troponins C, T (cTnT), and Troponin I (cTnI) were found increased in skeletal and cardiac muscles, involved in muscle contraction.[27]

Troponin T and I both have equal sensitivity and specificity to cardiac injury compared to the other cardiac markers like SGOT, SGPT, LDH, and CPK. These markers are also elevated in conditions like trauma to the skeletal muscles, liver diseases, pancreatitis, intramuscular injection and cerebral vascular disease.[28]

The sickling and hemolysis of red blood cells in sickle cell disease may be the cause of the increase in the aforementioned parameters. The increased concentration of serum cardiac troponin-I level is also correlated with the below described studies.

Space et al (2000) Budhiraja et al (2004) stated that the cardiac injures were elevated in SCD individuals going through ACS, which also accompanied with high TRV and increased death rates.[7,29]

Haywood et al in (2009) and Aslam et al in (2009) also reported that the evaluation of serum cardiac troponin-I may provide information with the possibilities of myocardial injury in children with sickle cell anemia and also concluded that the serum troponin-I levels are the baseline indications of myocardial injury and in the patients of acute chest syndrome in sickle cell disease.[4,6]

In another hypothesis, Mansi et al (2009) reported that the serum troponins were the intra-cellular markers released during the episode of myocardial injury (90 to 94% sensitive and about 95% were found specifically increased in MI).[30]

The above studies are correlated to our research topic indicating that the serum cardiac troponin-I was found increased in the sickle cell disease.

Thus, serum cardiac troponin-I may be an additional diagnostic tool for sickle cell disease.

Conclusion

Troponin-I, SGOT, SGPT, and LDH were all markedly elevated and shown a positive connection in sickle cell disease, suggesting that they may be further clinical biomarkers for the diagnosis of SCD.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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