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## The Study of Hepatic Parameters in Cardiac Dysfunction.

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### ABSTRACT

Heart diseases may present with symptoms of decreased exercise tolerance due to dyspnea and/or fatigue related to impaired cardiac output or may present with a syndrome of fluid retention from elevated filling pressure. Any cause of right ventricular dysfunction can be associated with severe hepatic congestion. Subjects with hepatic congestion are usually asymptomatic and this picture may be suggested only by altered liver function tests (LFT). Liver clinical parameters are varied with the slight abnormal functioning of right ventricle; hence the detailed study was undertaken to look in to the hepatic parameter in cardiac dysfunction a study the prognostic importance of liver function abnormalities in cardiac failure (CF).

**Keywords:** LFT, Serum proteins, CCF.

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## INTRODUCTION

Liver is the biggest organ in human body with a mass of nearly 1.5 kg. Liver has a massive functional reserve and regenerating capacity. It plays a major role in maintaining the normal physiology and metabolic homeostasis of human body. It is also referred to as the custodian of milieu interior. Hence hepatic diseases can have a major impact on the homeostasis of body. Similarly disorders in other systems can adversely affect the liver as well [1,2].

Cardiac failure both chronic and acute and can cause hepatic dysfunction. Twenty five percent of total cardiac output goes to the liver, hence any decrease in cardiac output leads to decreased liver perfusion. By means of vasoregulatory mechanisms and enhanced extraction of oxygen liver can tolerate variations in blood flow. But hepatic damage occurs when the threshold levels are crossed. In right heart decompensation raised backpressure causes congestion of the sinusoids and hepatocyte hypoxia. Left heart failure produces decreased cardiac output further causing decreased blood flow to the liver producing hypoxia. Both this mechanisms leads eventually into centrilobular liver cell necrosis. The most susceptible region of the liver lobule to hypoxic insult is the zone three of acini because of the peculiar arrangement of blood flow in the liver [2,3].

In the present study, the consequence of congestive cardiac failure on liver function were studied in a group of 60 patients and was analysed and compared with various parameters. Differing causes of heart failure of various duration were included in the study. Follow up of the cases were done for a period of one week and measurements were repeated at the end of one week. Changes in the values were recorded and an attempt is done to find the prevalence of liver function abnormalities in these subjects, its correlation with various parameters and prognostic significance of liver function on cardiac failure.

A proper knowledge and understanding of structure and functions of liver, liver parameters, etiologies and different forms of heart failure and their presentation, as well as the mechanism and pathology of liver in cardiac failure is inevitable before assessing the liver function abnormalities in congestive cardiac failure.

## MATERIALS AND METHODS

This study was conducted in the General Medical wards and Intensive care unit of Medical College teaching Hospital.

### Period of Study

November – 2011 to November – 2013.

### Design of Study

This study is a Single Centre Cross-Sectional and Analytical Study. A total of 60 subjects were included in this study.

### Methodology

### Subject Selection

#### *Inclusion Criteria*

- Congestive Cardiac failure in all age groups of varying etiologies

#### *Exclusion Criteria*

- History of alcoholism.
- Past history of jaundice.
- Recent intake of hepatotoxic and cholestatic drugs.

- Presence of viral markers
- Blood transfusion.

All subjects included in this study were subjected to thorough clinical examination. All were subjected to laboratory investigations as per the proforma.

The following liver biochemical tests were carried out in this study:

- Serum bilirubin
- Serum transaminases
- Serum alkaline phosphatase
- Serum proteins
- Prothrombin time

### **Serum Bilirubin**

Estimation of serum bilirubin was done by the Wandenburg reaction. In this test the bile pigments are diazotized by the sulphalinic acid and the products are estimated calorimetrically. This reaction can also differentiate indirect and direct fractions due to the differing solubilities of these fractions. The water soluble direct fraction produces the direct VB reaction when carried out in an aqueous medium. In ethanol the intramolecular hydrogen bonds of indirect bilirubin are fragmented and both direct and indirect fragments show reaction giving the total bilirubin value. The direct bilirubin is subtracted from the total bilirubin to get the indirect bilirubin value.

### **Serum Enzyme assays**

#### **Serum AST and ALT**

In this study AST and ALT was measured by enzymatic substrate method. AST and ALT substrates along with dinitro phenyl hydrantel were used. The enzyme catalyses the exchange of gamma amino group of alanine to the gamma keto group of glutamate forming oxalo-acetic acid and pyruvic acid.

#### **Serum ALP**

A variety of assays have been developed to measure alkaline phosphatase using different substrates. The substrate used was aminoantipyrine solution, alkaline phosphate buffer, potassium ferricyanide and substrate. Increased levels of ALP show biliary tract dysfunction. Slight to moderate elevation in ALP activity can occur in congestive cardiac failure. The raised levels signify rise in the synthesis of ALP by liver cells and biliary epithelium.

#### **Serum Proteins**

Widespread hepatic damage leads to reduction in serum levels of fibrinogen, prothrombin, albumin and other proteins which are produced exclusively by the liver cells. The estimation of serum proteins signifies hepatic synthetic function. The most major protein synthesized by the liver is albumin. Normal level ranges from 3.5-5.5 mg/dl. It has a long half-life i.e., 14-20 days and daily turn-over is less than 5%. Hence it is not an accurate indicator of acute hepatic damage.

Serum globulins include alpha beta globulins as well as immunoglobulins. Normal value is 2-3.5 mg/dl. Hyperglobulinemia occurs secondary to activation of the reticulo-endothelial system in response to the antigens presented passing through the liver.

Serum proteins are estimated by biuret method. Substances which contain 2 or more peptide bonds and CO-NH<sub>2</sub> groups give a purple or blue coloured precipitate with alkaline copper solution. Different proteins give differing amount of colour which helps in distinguishing them by this reaction.

### Prothrombin Time

Clotting factors like fibrinogen, factor 2, 5, 7, 9, 10 are synthesized in the liver. Any dysfunction of the clotting factors can be determined by one stage prothrombin time. This method calculates the conversion rate of prothrombin to thrombin in the presence of calcium and thromboplastin. This reaction requires properly functioning Vitamin K dependent clotting factors. These clotting factors have a short half-life, hence prothrombin time can be taken as an earlier marker of hepatic injury and its prolongation in both acute and chronic hepatic damage signifies worse prognosis.

**Table 1: Normal Values of Liver Function Tests**

SL. NO.	LIVER FUNCTION TESTS	NORMAL VALUES
1.	Serum BILIRUBIN	0.3 – 1.2mg/dl
2.	Serum AST/SGOT	0 – 40 I.U
3.	Serum ALT/SGPT	0 – 35 I.U
4.	Serum ALP	20 – 140 I.U
5.	Serum TOTAL PROTEINS	6 – 8.5 g/dl
6.	Serum ALBUMIN	3.5 – 5.5 g/dl
7.	Serum GLOBULIN	2.5 – 4.5 g/dl
8.	PROTHROMBIN TIME	12 – 14 sec (control) Abnormal if >1.5 times

### RESULTS

**Table 2: Age Distribution of the Cases**

Age group of the cases in years	Frequency of cases in each age group	Percentage
26 – 35	7	11.7
36 – 45	9	15.0
46 – 55	19	31.7
56 – 65	21	35.0
66 – 75	4	6.7
<b>TOTAL</b>	<b>60</b>	<b>100.0</b>

### Age Distribution

Out of 60 subjects studied 19 cases belonged to the age group 46 – 55 years and 21 cases belonged to the age group of 56 – 65 yrs showing the rising prevalence of cardiac disease with advancing age.

**Table 3: Etiology of Cardiac Failure**

Etiology of failure	Frequency of cases	Percentage
Coronary artery disease	23	38.3
CorPulmonale	14	23.3
Rheumatic heart disease	10	16.7
Cardiomyopathy	6	10.0
Hypertensive heart disease	7	11.7
<b>TOTAL</b>	<b>60</b>	<b>100.0</b>

### Etiology of Cardiac Failure

Maximum number of cases (38.3%) came under the category of Coronary Artery Disease followed by Corpulmonale (23.5%). This shows that Coronary artery disease is emerging has the single most common cause of cardiac failure. The incidence of Rheumatic Heart Disease which was previously considered as leading cause of heart failure has declined.

**Table 5: Duration of Cardiac Failure**

Duration of cardiac failure	Frequency in each group	Percentage
< 1	1	1.7
1 – 5	35	58.3
>5	24	40.0
TOTAL	60	100.0

Of the 60 subjects studied, the duration of illness is between 1 – 5 years in 35 cases, less than 1 year in 1 case and more than 5 years in 24 cases.

**Table 4: Case Distribution According To New York Heart Association Class of Heart Failure**

NYHA class of heart failure	Frequency of cases in each class	Percentage
Class I	17	28.3
Class II	22	36.7
Class III	12	20.0
Class IV	9	15.0
TOTAL	60	100.0

Of the total 60 subjects 17 cases belonged to class 1, 22 cases belonged to class 2, 12 belonged to class 3 and 9 belonged to class 4. This suggests the improvement in the control of failure symptoms with the currently available therapy.

**Table 6: Presence of Clinical Jaundice in the Cases**

Presence of Jaundice	Frequency	Percentage
Present	20	33.3
Absent	40	66.7
TOTAL	60	100.0

Clinical Jaundice was detected in 20 cases which constitute 33.3% and the rest were found to be normal.

**Table 7: Presence of Hepatomegaly In The Cases**

Presence of Hepatomegaly	Frequency	Percentage
Present	25	41.7
Absent	35	58.3
TOTAL	60	100.0

Out of 60 cases studied clinically hepatomegaly was found in 25 cases which forms 41.7% .

**Table 8: Presence of Ascites in the Cases**

Presence of Ascites	Frequency	Percentage
Present	7	11.7
Absent	53	88.3
TOTAL	60	100.0

Ascites was clinically made out in 11.7% of cases

**Table 9: Ultrasound Abdomen Showing Congestive Hepatomegaly in the Cases**

USG Abdomen with congestive liver	Frequency	Percentage
Present	23	38.3
Absent	37	61.7
TOTAL	60	100.0

Ultrasound scanning of the abdomen was done in all the 60 cases to identify congestive liver. Of these 23 cases showed abnormality in USG Abdomen (38.3%).

**Table 10: Liver Biochemical Abnormalities Noted in the Study Group**

Serum Bilirubin	Frequency	Percentage
Abnormal	34	56.7
Normal	26	43.3
TOTAL	60	100.0

Liver Parameters	Abnormal		Normal	
	Frequency	Percent	Frequency	Percent
Serum Bilirubin	34	56.7	26	43.3
Serum AST	32	53.3	28	46.7
Serum ALT	37	61.7	23	38.3
Serum ALP	8	13.3	52	86.7
Serum Total Protein	12	20.0	48	80.0
Serum Albumin	23	38.3	37	61.7
Serum Globulin	4	6.7	56	93.3
Prothrombin Time	21	35.0	39	65.0

Serum Bilirubin were found to be abnormal in 56.7% of the total congestive cardiac failure patients included in this study. Serum AST was found to be abnormal in 53.3% of the subjects and serum ALT was abnormal in 61.7% of the cases whereas serum ALP was found to be abnormal only in 13.3% of cases thus suggesting a hepatocellular pattern of liver enzyme elevation. Serum albumin was found to be decreased in 38.3% of the subjects and serum globulin was abnormal in 6.7% of cases. Prolongation of prothrombin time was observed in 35% of cases.

**Table 11: Prevalence of Liver Function Abnormality in the Study Group**

Liver Function Tests	Frequency	Percentage
Abnormal	34	56.7
Normal	26	43.3
TOTAL	60	100.0

**Table 12: Showing Comparison of Mean Bilirubin Values With NYHA Class Of Cardiac Failure**

NYHA Class	Frequency	Mean Bilirubin	SD	F	p value
Class I	17	1.1	.2	58.555	<0.001
Class II	22	1.5	.4		
Class III	12	2.3	.5		
Class IV	9	2.9	.5		
Total	60	1.7	.8		

P value less than 0.001 is significant thus suggesting a progressive increase in mean serum bilirubin values with worsening of heart failure class. Class 4 patients showed marked liver function abnormality.

**Table 13: Showing Comparison of Mean AST Values with NYHA Class of Heart Failure**

NYHA Class	Frequency	Mean AST Values	SD	F	p value
Class I	17	36.9	3.5	5.171	0.003
Class II	22	39.9	5.4		
Class III	12	87.5	41.7		
Class IV	9	188.8	271.8		
Total	60	70.9	115.0		

This table shows a sequential rise in serum AST levels with advancement of cardiac failure. p value is 0.003 which is significant.

**Table 14: Showing Comparison Of Different Liver Parameters With Nyha Class Of Heart Failure**

Liver function tests	Nyha Class	Frequency in each class	Mean values	S D	F	p value
Serum ALT	Class I	17	32.4	4.0	5.933	.001
	Class II	22	34.9	6.0		
	Class III	12	81.0	34.5		
	Class IV	9	142.0	186.2		
	Total	60	59.5	80.7		
Serum ALP	Class I	16	44.3	10.8	.336	.799
	Class II	22	39.5	15.2		
	Class III	12	42.0	18.6		
	Class IV	9	39.4	20.2		
	Total	59	41.3	15.5		
Serum Total Protein	Class I	17	6.4	.2	20.476	.000
	Class II	22	6.3	.3		
	Class III	12	5.9	.2		
	Class IV	9	5.6	.4		
	Total	60	6.2	.4		
Serum Albumin	Class I	17	3.5	.1	16.646	.000
	Class II	22	3.4	.2		
	Class III	12	3.1	.2		
	Class IV	9	2.9	.3		
	Total	60	3.3	.3		
Prothrombin Time	Class I	17	13.8	.9	15.76	.000
	Class II	22	14.3	2.9		
	Class III	12	18.3	3.6		
	Class IV	9	20.2	3.5		
	Total	60	15.8	3.7		

Serum ALP did not show a progressive rise in mean levels with advancing heart failure.p value is .799 which is not significant. All other liver function tests showed a significant change in the mean values with worsening in heart failure class. p values were significant.

**Table 15: Comparison of Serum Bilirubin with Etiology of Cardiac Failure**

Etiology	SERUM BILIRUBIN				Total	
	Abnormal		Normal		Frequency	Percent
	Frequency	Percent	Frequency	Percent		
CAHD	14	60.9	9	39.1	23	100.0
CP	8	57.1	6	42.9	14	100.0
RHD	7	70.0	3	30.0	10	100.0
CM	4	66.7	2	33.3	6	100.0
HHD	1	14.3	6	85.7	7	100.0
Total	34	56.7	26	43.3	60	100.0

$X^2 = 6.255$        $df = 4$        $p = 0.181$ , p value insignificant

Serum bilirubin did not show much correlation with the etiology of cardiac failure.

**Table 16: Comparison of Serum Bilirubin with Duration Of Cardiac Failure**

Duration of illness	SERUM BILIRUBIN				Total	
	Abnormal		Normal		Frequency	Percent
	Frequency	Percent	Frequency	Percent		
<1	0	.0	1	100.0	1	100.0
1-5	18	51.4	17	48.6	35	100.0
>5	16	66.7	8	33.3	24	100.0
Total	34	56.7	26	43.3	60	100.0

$X^2 = 2.676$   $df = 2$        $p = 0.262$ , p value is not significant

No correlation was made out between serum bilirubin and duration of cardiac failure.

**Table 17: Showing Comparison of Mean Bilirubin with Outcome Of Cardiac Failure**

Outcome	Frequency	Mean Bilirubin	SD	T	p value
Dead	4	2.95	.68	3.603	.001
Alive	56	1.65	.70		

p value is 0.001 which is significant. Elevated serum bilirubin can be considered as a bad prognostic indicator of cardiac failure.

**Table 18: Showing Comparison between Values on First Day of Study and Seventh Day of Study**

Liver Function Test	DAY 1		DAY 7		McNemar Test P value
	Frequency	Percent	Frequency	Percent	
Serum Bilirubin	34	56.7	14	25.0	<0.001
Serum AST	32	53.3	16	28.6	0.002
Serum ALT	37	61.7	19	33.9	<0.001
Serum ALP	8	13.3	7	12.5	1.000
Serum TP	12	20.0	8	14.3	1.000
Serum Albumin	23	38.3	27	48.2	0.424
Prothrombin Time	21	35.0	20	35.7	0.508

p values are significant for Serum Bilirubin, Serum AST and Serum ALT which showed considerable improvement in values with control of failure symptoms and on followup at the end of 1 week. Serum total protein, serum albumin and prothrombin time did not show significant change at the end of 1 week.

### DISCUSSION

Large number of studies has been conducted in evaluating hepatic function in congestive heart failure. Umpteen numbers of studies are still going on in this arena of liver function.

In this study, liver biochemical abnormalities and clinical features in cardiac failure because of differing etiologies in 60 patients were recorded analysed and compared within and correlated with numerous research papers. The principal intention of this study is to relate indian picture with international scenario.

#### Age and Sex Distribution of the Cases

Out of 60 patients studied 19 of them belonged to 46-55 age group and 21 of them belonged to above 55 age group. This shows that cardiac disease and progression into failure is more common in the elderly age group. Out of 60 subjects selected randomly 41 were males and 19 were females which show the rise in cardiac disease and failure in the male population when compared to the opposite gender which is in correlation with the international scenario. Demographic and Clinical Characteristics of Patients Admitted with Heart Failure in the Euroheart Survey and ADHERE Programmes in the US has shown increased incidence of heart failure in the elderly age group and the male population [1,5]. The median age at first presentation in most recent studies has been in the mid-70s, with a higher incidence in men than in women of all ages.

#### Etiology and Duration of Cardiac Failure

In this study out of the 60 patients studied 23 patients (38.3%) suffered from coronary artery disease, 14 patients from corpulamonale, 10 from rheumatic heart disease, 6 from cardiomyopathy, 7 from hypertensive heart disease. In this study most common cause for heart failure was found to be coronary artery disease which is in correlation with the international scene. The incidence of rheumatic heart disease as the reason for heart failure has declined which is considered as a changing trend in the etiology of heart failure. Cowie et al and Fox et al has shown in there studies the solitary most common cause of heart failure in the developed world is coronary heart disease [1,4,5] Kannel et al, McMurray et al and levy et al in there studies suggest valvular heart disease and hypertension have come down as the principal reason for heart failure[5].

Out of 60 subjects studied the duration of cardiac failure was greater than 5 years in 24 cases (40%), was in the range of 1-5 years in 38 cases (58.3) and less than 1 year in 1 case (1.7%).

### **NYHA Class**

Out of the 60 patients studied 17 belong to class 1,22 belong to class 2,12 belong to class 3,9 belong to class 4. Most of the patients come under lower classes than higher classes which suggests improved quality of life with cardiac failure medications

### **Jaundice**

Out of the 60 patients studied 20 subjects which is nearly 33% had jaundice. Other causes for jaundice like liver damaging drugs and alcohol related injury were excluded by taking proper history from the subjects. Serological tests were carried out to rule out viral causes for liver damage. In many papers that the serum bilirubin was frequently above the upper limit of normal range. Cogger et al [6] has described jaundice clinically 33% of the cases.

### **Hepatomegaly**

In this study hepatomegaly was found to be present in twenty five out of sixty subjects which is nearly 41.7%. the enlargement in liver ranged from 1 cm to 8 cm in these subjects. Dunn et al and white et al has demonstrated liver enlargement in more than 90% cases in their studies [7].

### **Ascites**

Out of 60 subjects only seven persons showed clinically detectable ascites which is 11.7%. Norman et al demonstrated ascites and oedema in 15% of cases of cardiac failure studied [8]

### **Ultrasound Abdomen**

USG Abdomen was carried out in all the 60 subjects. 23 (38.3%) of them showed changes of congestive hepatomegaly in the abdominal scan.

### **Hyperbilirubinaemia**

Elevated serum bilirubin values were detected in 34 out of 60 patients which is 56.7%. In this study most of them had bilirubin values less than 3mg/dl. Serum bilirubin values were less than 3mg/dl in 55 cases and more than 3mg/dl in 5 cases. Those with values more than 3 showed features of severe congestive cardiac failure. it was seen that with control of cardiac failure the serum bilirubin levels returned to basal values in more than 50% of the cases In this study no specific correlation ( $p$  value not significant) was found between etiology and mean serum bilirubin values though subjects with rheumatic heart disease showed a slight increase in incidence of jaundice followed by coronary heart disease [9,14]. Hypertensive heart disease patients showed least rise in serum bilirubin values. Similarly no significant correlation was found between duration of cardiac failure and mean serum bilirubin values. Felder at al has shown elevate bilirubin levels in 52% of the cases in his study [13] Kubo et al have reported that bilirubin values rarely increase more than 5mg% and are usually less than 3 mg% [10] In this study it was noted that as the NYHA class of heart failure advanced the mean serum bilirubin level also progressively increased which suggests higher classes of heart failure were associated with higher degree of liver dysfunction. This was supported by R M Ross et al in their study [11].

### **Serum Aminotransferases**

In the present study 32 subjects (53.3%) showed increased AST levels and 37 subjects (61.7%) showed raised ALT levels. The rise in AST levels ranged from 40-910 IU which correlates with the findings of Richman et al who observed striking rise in transaminase in cardiac failure secondary to corpulmonale or tricuspid regurgitation or hypotension and shock. Piersol et al [12] have observed that rise in serum ALT is noted in 5-

50% of patients and it is seen more in acute heart failure than chronic heart failure. In this study it has been observed that mean AST levels increase progressively as the NYHA class of cardiac failure progresses.

### **Serum Alkaline Phosphatase**

In this study out of the 60 subjects studied eight patients showed abnormal serum ALP (13.3%). Felder et al observed elevated serum ALP in ten – twenty percent of subjects studied [13] Jollifee also has recorded similar observations in their studies [15] Rise in serum ALP does not relate with the elevation in serum transaminases and bilirubin. ALP values return to basal level with control of failure symptoms. Serum ALP values showed no correlation with the NYHA Classes of cardiac failure. It was observed that mean ALP levels showed no progressive change with worsening of heart failure.

In the present study serum AST and ALT showed marked elevation than serum ALP thus suggesting a predominant hepatocellular pattern of hepatic damage.

### **Serum Proteins**

In this study 23 subjects showed hypoalbuminaemia out of 60 subjects ie 38.3% and 4 cases showed abnormal globulin. AG Reversal was noted in 4 cases. It was noted that with advancement in class of cardiac failure mean serum albumin levels showed a progressive fall with class IV patients showing the lowest mean serum albumin value with a significant p value. Lambert [16] in his study has reported decreased albumin in thirty – fifty percent of cases. Mild decrease in albumin levels were observed with values ranging from 2.5g/dl to 2.9g/dl.it was observed by Dunn et al that cases with marked fluid retention showed albumin values less than 1.5g/dl [17] Serum albumin values usually return to normal in a period of few months following control of cardiac failure. A rise in globulins and fall in albumin cases reversal of Albumin globulin ratio. It was found that unlike other parameters globulin values did not return to normal value following control of cardiac failure.

### **Prothrombin Time**

In this study 21 out of 60 patients showed prolongation in prothrombin time (35%) most of the values were 1.5 – 2 times the normal value. Following treatment prothrombin time repeated at the end of 1 week did not show any improvement. Mean prothrombin time also showed progressive increase with worsening of cardiac failure such that class IV cases showed highest mean prothrombin time. Delmann and Richmann [18] as described prolonged prothrombin time in nearly 85% of the subjects. It was reported that prothrombin time come to baseline values usually two – three weeks after control of cardiac failure.

### **Outcome**

Out of 60 cases studied 4 cases succumbed to the cardiac illness during the course of illness. The mean bilirubin value of these cases were 2.95 which was more when compared to other subjects which was 1.65 with a p value of .001 which is significant suggesting that elevated bilirubin levels can be taken as a bad prognostic marker in congestive cardiac failure.

### **CONCLUSIONS**

- The most common cause of congestive cardiac failure in patients presenting to Medical College Hospital was found to be Coronary artery disease.
- Elderly Male population was found to have an increased incidence of cardiac failure when compared to the opposite gender.
- Liver function abnormalities were found in 56.7% of the total congestive cardiac failure patients included in this study.
- Liver function abnormalities did not show any correlation with the aetiology and duration of cardiac failure though subjects with rheumatic heart disease showed a mild increase in incidence of abnormal liver function. Least incidence of liver function abnormalities were found in Hypertensive heart disease patients.

- Serum AST was found to be abnormal in 53.3% of the subjects and serum ALT was abnormal in 61.7% of the cases whereas serum ALP was found to be abnormal only in 13.3% of cases. This suggests a predominant hepatocellular pattern of liver injury than cholestatic pattern.
- Serum albumin was found to be decreased in 38.3% of the subjects and serum globulin was abnormal in 6.7% of cases and A/G reversal was noted in 4 cases. Prolongation of prothrombin time was observed in 35% of cases.
- Mean serum Bilirubin, AST and ALT values when compared with NYHA Class of heart failure showed a progressive increase with the advancement of heart failure. Mean serum ALP values did not show any correlation with NYHA Class of heart failure. Mean serum albumin values showed a progressive fall with advancement of cardiac failure. Prothrombin time also showed progressive prolongation with worsening of failure class. Thus Class IV heart failure patients showed a higher degree of predominant hepatocellular pattern of liver damage.
- With control of failure and on follow up after one week serum bilirubin and serum enzymes showed considerable improvement whereas serum albumin and prothrombin time did not show any significant change.
- Out of the 60 subjects studied 4 cases succumbed to the cardiac illness and rest of them showed improvement at the end of 1 week. On comparison of mean bilirubin value with the outcome it was observed that higher values were associated with a poor prognosis. Thus serum bilirubin was found to have prognostic significance in cardiac failure.

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