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Observation of Changes in Renal Function Tests and Serum Electrolytes in Cirrhosis of Liver in a Tertiary Care Centre of Jharkhand

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Abstract:Introduction: *Cirrhosis can result from almost all form of liver injury e.g. by viral hepatitis, alcoholism, and hepatitis due to drugs. The mortality of patient with renal failure in cirrhosis is high. Previous studies, have also reported high incidence of morbidity and mortality (Davenport et al1993), Kalso-Castellob while studying the fluid and electrolyte disturbances in terminal stage of cirrhosis cases, demonstrated that electrolyte disturbance in cirrhosis cases can be life threatening and must be recognized quickly and treated.*
Materials and Methods: *The patient of cirrhosis admitted in different units of Medicine Department, were included in this study. The renal function as monitored by recording daily urine output, routine examination of urine, plasma and urine creatinine, estimation of sodium, potassium and chloride and GFR, blood urea etc. Finally the data obtained was statistically analysed, presented in the groups and table and revised in the context of current available literature. Forty healthy people of both sexes (male and female) of same age groups and socio-economic status were selected as controls.*
Results: *compare the liver functions in study and control group. In study group I, the mean level of serum bilirubin was 17.68 ± 4.74 mg%, SGPT 239.62 ± 72.30 IU/L, prothrombin time 47.5±6.65 sec., serum albumin 2.93 ± 0.55 mg% whereas in control group the value recorded were serum bilirubin 0.65 ± 0.13 mg%, SGPT 28.05 ± 4.87 IU/L, prothrombin time 13.85 ± 0.5 sec., serum albumin 4.03 ± 0.21 gm%. Hence it was evident that in study group I liver functions parameter were more significantly raised ($p < 0.01$) while prothrombin time was raised significantly. However the serum albumin level was low as compared to control group. Similar observation of liver function tests were recorded in group II patients. Allen. J. Arieff et al (1973) in their study showed almost similar data for the liver function test in cases of advance liver disease.*
Conclusion: *To conclude the above work it was observed that amongst the cases of cirrhosis undertaken in study, more than 50% of cases develops renal failure which is itself a vulnerable sign for the moribund cases of cirrhosis. Amongst the cases of renal failure about 28% cases develop hepatorenal syndrome as evidenced by renal failure index less than 1. Again it was observed that about 8% of cases was in pre-renal uraemia category which was probably due to excess of gastrointestinal bleeding as was observed in those cases only.*
Key Words: *Cirrhosis, pre-renal uraemia, hepatitis, GFR.*

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I. Introduction

Cirrhosis can result from almost all form of liver injury e.g. by viral hepatitis, alcoholism, and hepatitis due to drugs. The mortality of patient with renal failure is high. Previous studies, have also reported high incidence of morbidity and mortality (Davenport et al1993), Kalso-Castellob while studying the fluid and electrolyte disturbances in terminal stage of cirrhosis cases, demonstrated that electrolyte disturbance in cirrhosis cases can be life threating and must be recognized quickly and treated.

The present study was conducted in the different medical units of the Department of Medicine in Jharkhand. Cases of cirrhosis of liver, were selected. Those suffering from cirrhosis due to alcoholic liver disease included in study group I, and those suffering from miscellaneous causes were put in study group II.Control group consisted of healthy individual of both sexes and of similar age group.

In each case detailed clinical examination, liver function test like serum bilirubin, prothrombin time, ALT and renal function like blood urea, serum creatinine, GFR, serum sodium, serum potassium and serum chloride was estimated and analyzed statistically.

In study group I and II the liver function test like serum bilirubin, ALT and prothrombin time as compared to control group were found to be significantly elevated where as serum albumin was significantly reduced.

In the present study comprising 60 cases of cirrhosis, renal failure was observed in33 cases (55%). In present study, pre-renal uremia was present in 4 cases (8%), HRS in 14 cases (28%) and ATN in 6 cases (12%). In the present study it was found that renal failure index and fractional excretion of sodium remained to be the most sensitive parameter to differentiate between HRS and ATN, which is mentioned in text and that the severity of renal failure as shown by level of serum creatinine and GFR is related to severity of liver dysfunction as shown by level of serum albumin, bilirubin and prothrombin time.

In both the study group serum sodium and potassium level were found to be much low, when cirrhosis was accompanied with renal failure. Serum chloride level was significantly low in Group-I but insignificantly high in Group II as compared to control group.

Liver has got multiple functions hence its insufficiency in a cirrhosis patient gives rise to a complex syndrome which includes disturbance of nitrogen metabolism, bile formation, coagulation,disturbancesofnervous system, circulatory system, renal system and endocrine system. Liver disease largely affects the water and electrolyte equilibrium. It is known since the time of Austin Flint (1863) who made specific reference to kidney function in cases of cirrhosis of liver. He found that the patients with avid sodium retention exhibit oliguria with urinary output 300 cc to 500 cc.

Abnormal renal regulation of water and electrolytes occur commonly leading to impaired diuresis and dilutionalhyponatraemia (Goodyear et al,1960 and Rivera et al, 1961). Sherlock in her work 1956 found hyponatraemia in cases of cirrhosis with hepatic failure. In heropinion in such cases serum sodium below 130 mEq/L must be regarded as serious and below 125 mEq/L ominous. This hyponatraemia is notamenable to treatment and reflect impending cell death rather than bodysodium loss. Sherlock (1993) also mentioned hypokalaemia in cases of cirrhosis. The potassium deficiency usually arises from decrease dietary intake, vomiting, diarrhoea, high glucose feeding, secondary aldosteronismby diuretics. Fluid and electrolyte disturbance in cirrhosis can be lifethreatening and must he recognized quickly and treated. Similarly the mortality of the patients with hepatorenal syndrome remains high.

II. Material And Methods

The patient of cirrhosis admitted in different units of Medicine Department, were included in this study. The renal function as monitored by recording daily urine output, routine examination of urine, plasma and urine creatinine, estimation of sodium, potassium and chloride and GFR, blood urea ,ultra sound abdomen etc. Finally the data obtained was statistically analysed, presented in the groups and table and revised in the context of current available literature. forty healthy people of both sexes (male and female) of same age groups and socio-economic status were selected as controls.

The study group consists of sixty patients of cirrhosis of liver.

Exclusion criteria

- Person having suffered in past from any other coexisting disease known to produce its effect on urinary findings.
- Infants, children and adolescent were excluded and only adult cases were included to maintain the uniformity of research work.

The selections were made on the basis of detailed history, clinical examination. The diagnosis of all patients were made through clinical examination and investigations.

III. Results

Figure 1: Age Distribution in Study And Control Group

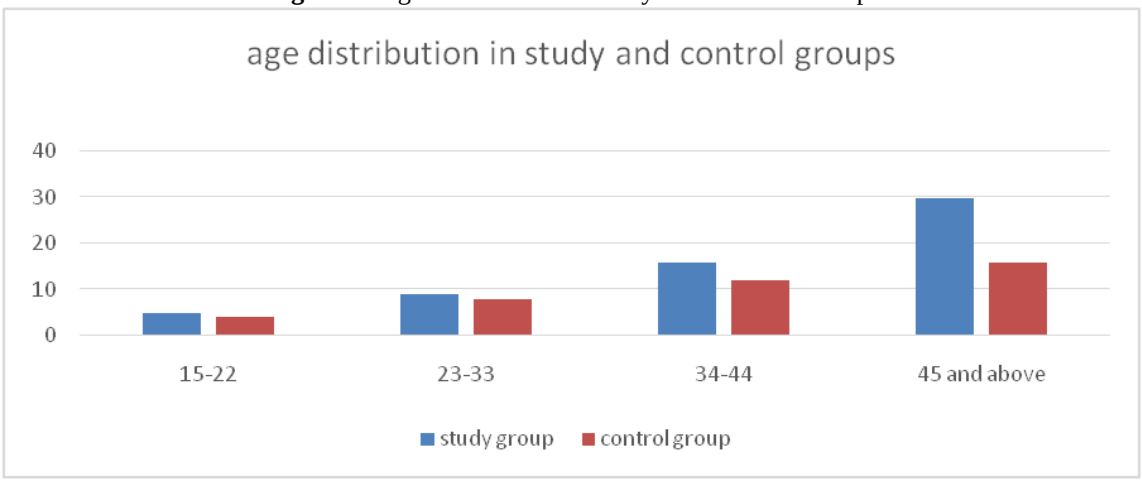


Table 1: Age Distribution In Study And Control Group

AGE IN YEARS	STUDY GROUP		CONTROL GROUP	
	Cases	percentage	Cases	percentage
15-22	**5**	**8**	**4**	**10**
22-32	**9**	**14**	**8**	**20**
33-44	**16**	**28**	**12**	**30**
45 AND ABOVE	**30**	**50**	**16**	**40**

Table I shows the age distribution in study and control group. The maximum and minimum number of patient in both group belongs to 45 or above and 15-22 years of age respectively.

Table 2: Sex Distribution Of Study And Control Group

SEX	STUDY GROUP		CONTROL GROUP
	GROUPI	**GROUPII**	
MALE	**30**	**16**	**30**
FEMALE	**10**	**4**	**10**

Figure 2: Showing Sex Distribution In Study And Control Group

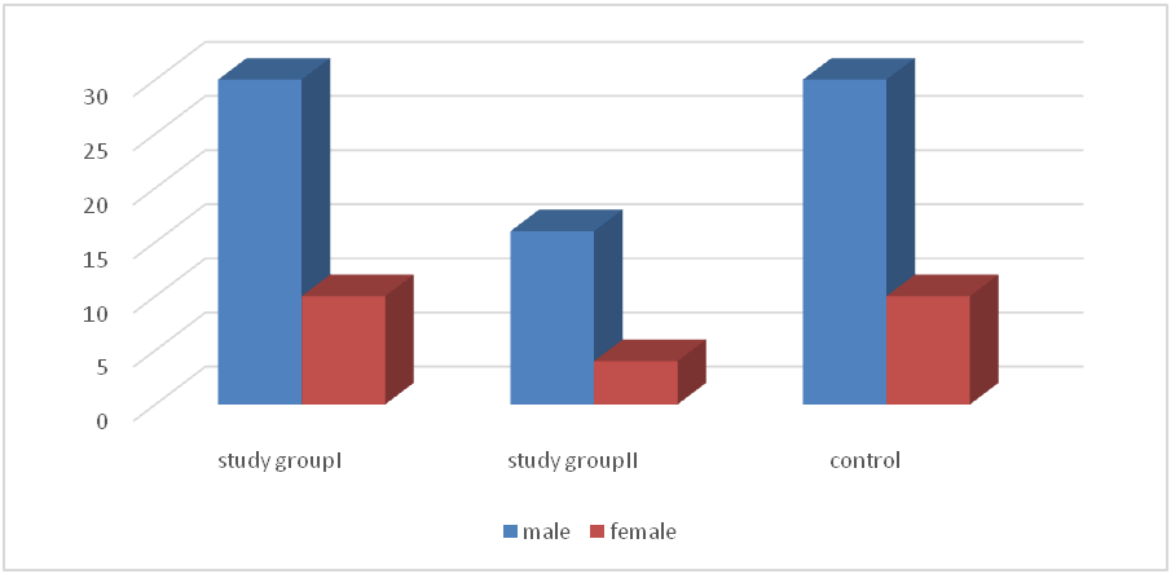


Table 3: Symptoms on Day Of Admission

SYMPTOMS	GROUPI	GROUPII
loss of appetite	40	10
General weakness	40	8
Vomiting	40	5
Yellow coloration of conjunctiva	45	10
Fever	12	2
Bowel irregularities	10	2
Swelling of feet	13	4
Swelling of abdomen	16	5
Headache	20	5
Git bleeding	15	3
Altered sleep rhythm	10	5
Disturbed sensorium	8	3
Unconciousness	10	5

Majority of the patients complained of loss of appetite , vomiting and general weakness. Jaundice was the predominant complain in group I. Other complains were as listed.

Figure 3: showing symptoms on day of admission

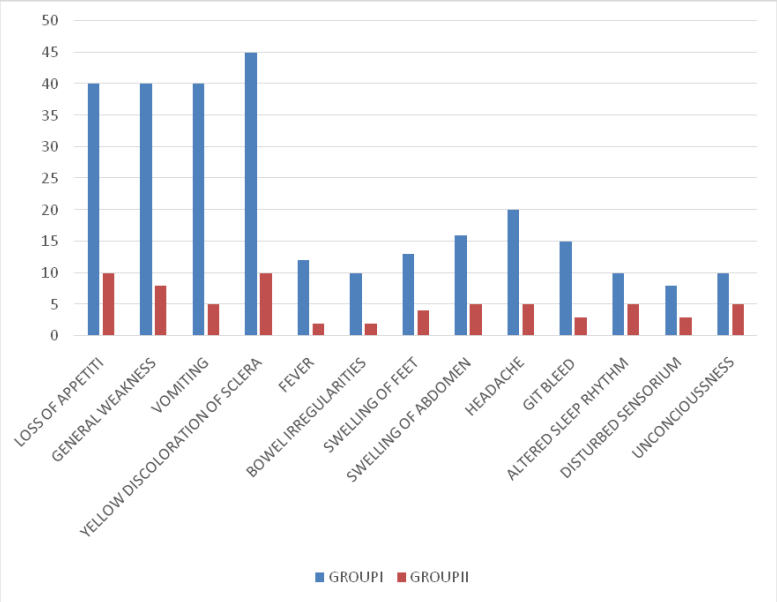


Figure 4: Showing liver function tests

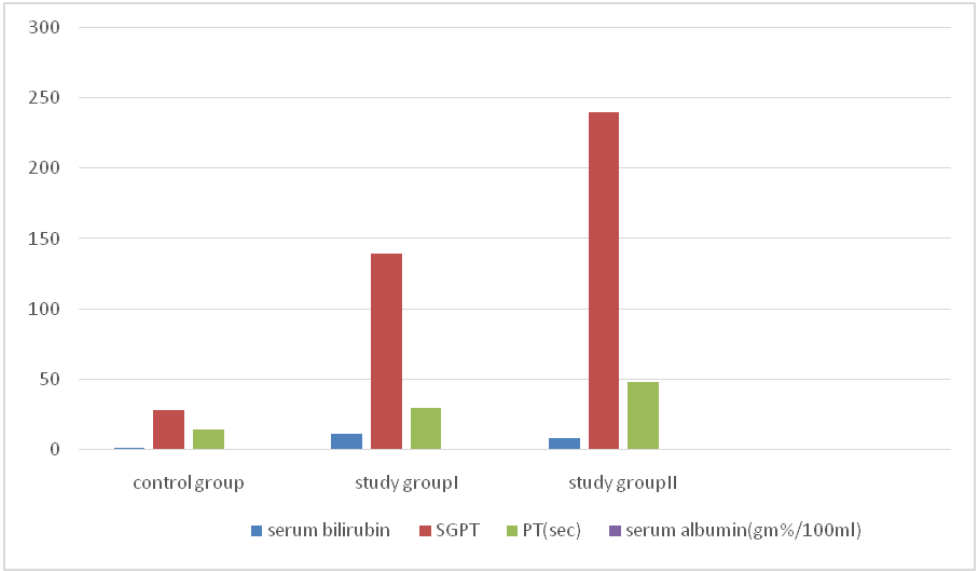


Table 4: Showing liver function tests

group	*range*	*mean*	*SD*	*S.E*	*t value*	*pvalue*
Control group (n=40)						
Serum bilirubin(mg%)	**.4-.8**	**0.65**	**0.13**			
SGPT	**20-35**	**28.05**	**4.87**			
Prothrombin time	**12-14**	**13.85**	**0.5**	-	-	-
Serum albumin(gm%/100ml)	**3.8-4.5**	**4.03**	**0.21**			
Study groupI(n=40)						
Serum bilirubin(mg%)	**7.4-14.5**	**17.68**	**2.86**	**0.9**	**11.27**	**<0.01**
SGPT	**90-305**	**239.62**	**59.2**	**18.75**	**5.93**	**<0.01**
Prothrombin time	**25-32**	**13.85**	**2.82**	**0.9**	**17.22**	**<0.01**
Serum albumin(gm%/100ml)	**2-3.9**	**2.93**	**0.38**	**0.14**	**8.5**	**<0.01**
Study group II(n=20)						
Serum bilirubin(mg%)	**10.2-25.6**	**7.68**		**0.75**	**22.70**	**<0.01**
SGPT	**120-350**	**239.62**	**4.74**	**11.48**	**18.42**	**<0.01**
Prothrombin time	**34-56**	**47.5**	**239.62**	**1.05**	**32.04**	**<0.01**
Serum albumin(g%/100ml)	**2.0-4.0**	**2.93**	**47.5**	**0.098**	**11.22**	**<0.01**
			2.93			

Table 4 tocompare the liver functions in study and control group. In study group I, the mean level of serum bilirubin was 17.68 ± 4.74 mg%, SGPT 239.62 ± 72.30 IU/L, prothrombin time 47.5±6.65 sec., serum albumin 2.93 ± 0.55 mg% whereas in control groupthe value recorded were serum bilirubin 0.65 ± 0.13 mg%, SGPT 28.05 ± 4.87 IU/L, prothrombin time 13.85 ± 0.5 sec., serum albumin 4.03 ± 0.21 gm%. Hence it was evident that in study group I liver functions parameter were more significantly raised ($p < 0.01$) while prothrombin time was raised significantly. However the serum albumin level was low as compared to control group. Similar observation of liver function tests were recorded in group II patients. Allen. J. Arieff et al (1973) in their study showedalmost similar data for the liver function test in cases of advance liver disease

Table 5: Chart showing incidence and types of renal failure in study and control group

group	**No of cases**	**percentage**
Whole study group	**60**	**100**
Renal failure	**33**	**55**
Pre renal failure	**7**	**11.66**
Hepatorenal failure	**14**	**23.33**
Acute tubular necrosis	**6**	**10**

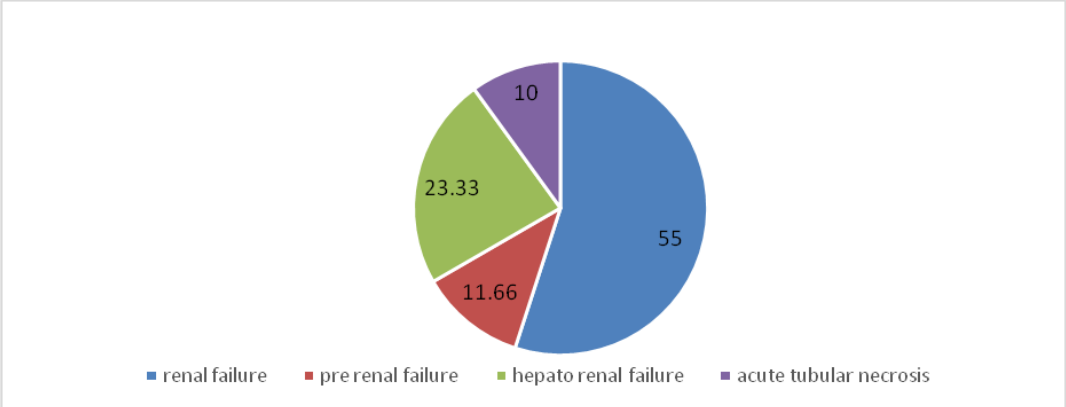
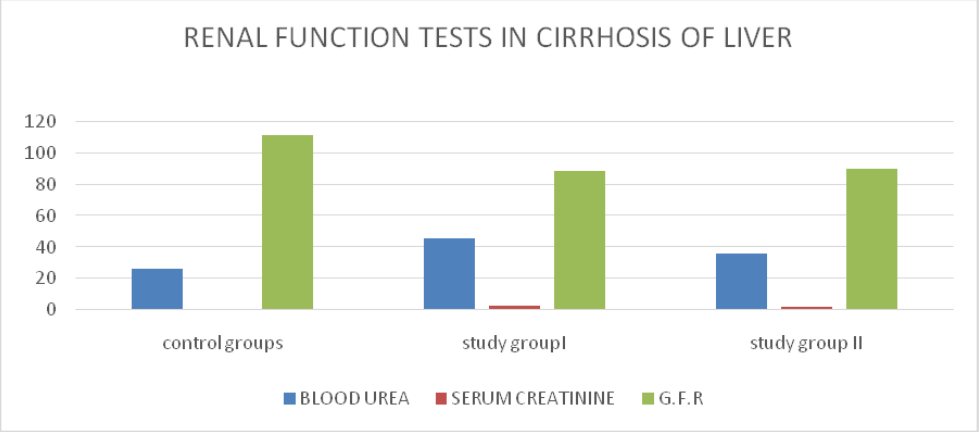


Chart showing renal function tests in cases of advance stage cirrhosis in both study and control groups

group	range	mean	SD	S.E	T value	P value
STUDY GROUPI						
Blood urea	20-106	45.95	20.68	3.38	5.78	<0.01
Ser.creatinine	1.2-4.4	2.68	0.83	0.137	14.23	<0.01
G.F.R	40-116	88.8	21.12	3.62	6.31	<0.01

STUDYGROUPII Blood urea Ser.creatinine G.F.R	22-58 0.8-3.3 68-114	35.8 2.06 90.1	11.03 0.66 19.76	3.60 0.21 6.40	2.61 6.3 3.36	<0.02 <0.01 <0.01
CONTROL GROUP Blood urea Se.creatinine G.F.R	20-40 0.5-1.0 100-116	26.4 0.73 111.65	4.0 0.18 6.32	-	-	-



Twenty (20) normal individual with regards to their renal function was studied. The mean value for blood urea, serum creatinine and GFR, were 26.4, 0.73, 111.65 respectively in control group.In study group comprising 60 cases of cirrhosis of liver renal failure was observed in 33 cases (55%).

In present study, pre-renal uremia was present in 7cases (11.66%), HRS in 14 cases (23.33%) and ATN in 6 case (10%). The result is in accordance with the observation of Ring-Larsen and Plazoo (1981) who shows HRS in 35% and ATN 17% cases.

To assess the renal function, blood urea, serum creatinine,GFR were measured in the study group and the mean level, werecompared with the mean level of the control group . Themean level of blood urea, in group I cases was 45.95 mg % (±SD20.68 mg%). This was found to be significantly higher than themean blood urea level of control group, which was 26.4 mg%, (± SD4.0 mg%). Similarly the mean serum creatinine level in group I was2.68 mg% (±SD 0.83 mg%) which was significantly higher than themean serum creatinine level to the control group (0.73 mg%) (± SD0.18 mg%). 't' being 14.23, p <0.01.

The mean GFR in group I was 88.8 ml/mm. (±SD 21.12) which was lower than the mean GFR of control group (111 .65ml/mm), SD ± 5.11). This was statistically significant ('t' = 6.31p<0.0l).In group II cases all the parameters studied to assess the renal function varied significantly from the normal.

In the present study , the level of urinary sodium in control group I cases was 30.65 meq/L (±SD 23.37). This was found to be significantly lower than the mean urinary sodium level of control group the value of which was 81.85 meq/L (±SD 19.83) 't' being 8.87 P< 0.01. Similarly the mean urinary sodium in group II subjects was 32.7 meq/L (±SD 25.23). which was significantly lower than the mean urinary sodium of the control group (81.85 meq/L, ± SD 19.83) 't' being 5.38 P < 0.01.

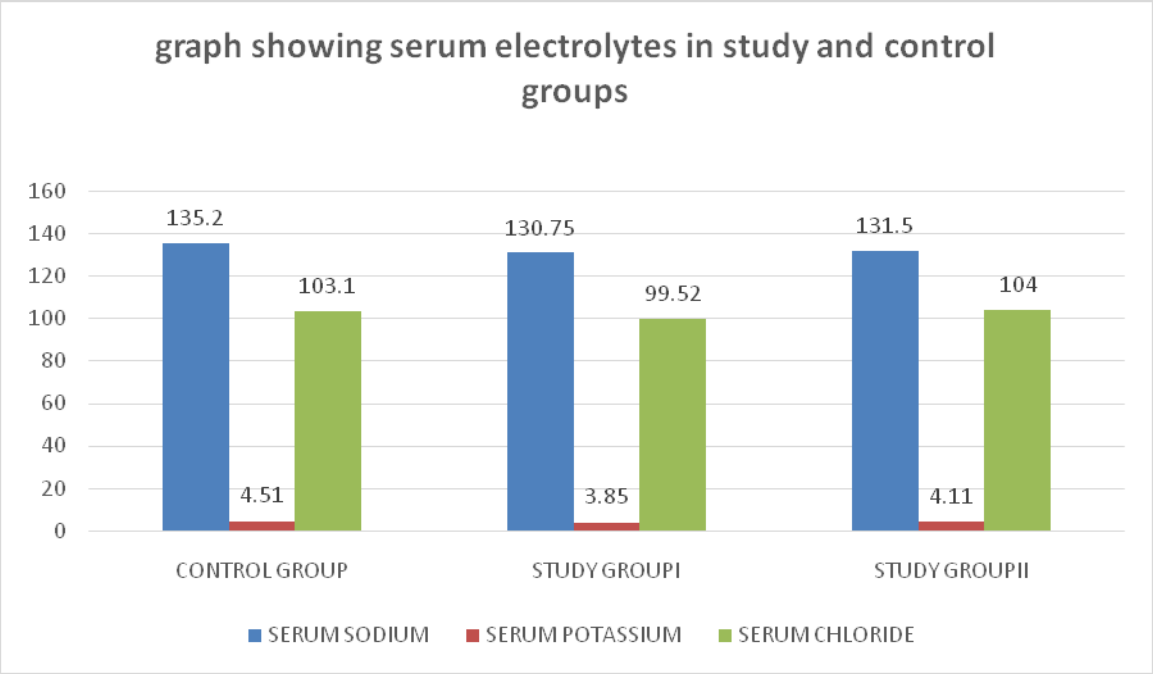
Hepatorenal syndrome of acute tubular necrosis are best differentiated by RFi and FENa (Espinal and Greger, 1980) Valuesof these parameters above one suggested ATN while values belowone is seen in HRS or pre-renal uraemia

In present study parameters Renal failure index(RFi) and Fractional excretion of filtered sodium (FENa) were usedto differentiate fulminant renal failure (FRF or HRS) and ATN. Itwas observed that in FRF both RFi and FENa were below one meandeviation(0.156 and 0.127 respectively) while in ATN these were above one mean 1.15 and 1.06 respectively.

The value of UNa in ATN was 30-97 (57.8). In hepatorenal syndrome (HRS), study urine/plasma (U/P) creatinine was 34.5-90.5

Chart showing serum electrolyte levels various groups

GROUPS	RANGE	MEAN	SD	S.E	tVALUE	pVALUE
CONTROL GROUP						
SERUM SODIUM						
SERUM POTASSIUM	**132-144**	**135.2**	**3.21**			
SERUM CHLORIDE	**3.5-5.5**	**4.51**	**0.48**			
	95-107	**103.1**	**3.25**			
STUDY GROUPI						
SERUM SODIUM						
SERUM POTASSIUM	**120-140**	**130.75**	**6.25**	**1.22**	**3.64**	**<0.01**
SERUM CHLORIDE	**2.6-5.5**	**3.85**	**1.05**	**0.197**	**3.35**	**<0.01**
	84-123	**99.52**	**8.12**	**1.47**	**2.36**	**<0.05**
STUDYGROUPII						
SERUM SODIUM						
SERUM POTASSIUM	**128-136**	**131.5**	**2.37**	**1.03**	**3.59**	**<0.01**
SERUM CHLORIDE	**3.5-4.8**	**4.11**	**0.45**	**0.17**	**2.35**	**<0.05**
	98-110	**104**	**3.63**	**1.35**	**0.66**	**<0.05**



The serum electrolytes as obtained in the normal individual were compared with those in Group 1 and Group II.The mean serum sodium in Group I cases was found to be 130.75 meq/L with a ±SD of 6.25. This was found to be significantly lower than the mean serum sodium of the control group ('t' = 3.64; p <0.01). The mean serum ± SD of 2.37 which was significantly lower the control group ('t'=3.59, P< 0.01).

IV. Discussion

In the present study hyponatremia was found in both the group of patients. It has been observed in this study that vomiting and GIT bleeding may be contributory factor in causing hyponatremia.Vomiting was noted in all patient in the study group and GIT bleeding was also observed in few cases of the study group. Hyponatremia in cirrhosis may be caused due to diarrhoea, vomiting, deficient intake etc as suggested by Donald et al (1952) or it may bedue to dilutional effect or due to shift of sodium from extracellular to intra-cellular spaces (Arroya, 1 975),also to the use of diuretics.

As regards serum potassium, the mean serum potassium in Group I subjects was found to be 3.85 meq/L with ±SD of 1.05 which was significantly lower than the mean serum potassium level of the control group (t'=3.35, P<0.0l). The mean serum potassium in group II cases was 4. 11 meq/L with ±SD of 0.45 which was also lower than that of the control group (mean 4.5 1 meq/L, ±SD 0.48). The difference was also statistically significant ('t'-1.35; P<0.05). Group II patients also had statistically significant hypokalemia.

As regards serum chloride, the mean serum chloride level in group I cases was 99.2 meq/L with ± SD 8.12. This was found to be significantly lower than the mean serum chloride level of the control group ('t' 2.36;

P < 0.05). In group II cases the mean serum chloride level was 104 meq/L with ±SD 3.63 which was insignificantly higher than the control groups ('t'=0.66, P >0.05).

V. Conclusion

To conclude the above work it was observed that amongst the cases of cirrhosis undertaken in study, more than 50% of cases develops renal failure which is itself a vulnerable sign of prognostic significance. Amongst the cases of renal failure about 28% cases develop hepatorenal syndrome as evidenced by renal failure index less than 1. Again it was observed that about 8%of cases was in pre-renal uraemia category which was probably dueto excess of gastrointestinal bleeding as was observed in those cases only.

It is known that there is hyponatremia and hypokalemia in cases of cirrhosis, but with association of renal failure of severe grade the sodium, potassium level further reduces, probably due to redistribution of fluid in intra and extracellular compartment affecting the sodium, potassium and also chloride level.

Above observation elucidate that in addition to the renal parameters; electrolyte are also equally important in diagnosing severity of hepatorenal disease and its outcome. They may become a important prognostic indicators and help in defining the precarious internal metabolic harmony of a cirrhotic patient. This may be of immense help to those patients of cirrhosis of liver who have lost all hope of leading a fruitful life.

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