Original Article

Role of Salt Intake in the Management of Cirrhotic Ascites

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Background: The purpose of study was to determine the role of salt intake in the management of ascites. Ascites is the most common complication of cirrhosis. Dilutional hyponatremia is present in most of patients with ascites. It is due to excess in antidiuretic hormone which occurs due to decrease in effective arterial volume. Water free salt intake tends to maintain the circulatory volume by drawing the fluid from third space, hence inhibiting ADH. It also increases the response of diuretics by increasing the filtered load of sodium. On the other hand, strict salt restriction and diuretics worsen the hyponatremia and make ascites refractory to treatment.

Objective: To determine the role of salt intake in the management of ascites in cirrhotic patients.

Methods: It was an interventional experimental study. This study was conducted in Services Hospital, Lahore. Sixty cirrhotic patients with ascites were randomized into two groups to undergo treatment with diuretics and salt restriction during the day. One group (A) was advised salt intake at night with over night fluid restriction, and the other group observed 24 hour salt restriction. The comparison was done in respect to average reduction in weight and abdominal girth over 9 days.

Results: The average reduction in weight (p=0.011) and abdominal girth (p=0.003) was significantly more in patients taking salt at night. Moreover these patients also maintained their serum sodium levels (p=0.00001) and majority of them did not need increase in the dose of diuretics or therapeutic ascitic tap.

Conclusion: Correction of dilutional hyponatremia by water free salt intake to improve the response of diuretics, gives a safe and effective option for the management of ascites.

Keywords: Cirrhosis, Ascites, Diuretics, Sodium.

Introduction

Cirrhosis represents the end stage of any chronic liver disease.[1] It is most frequently caused by viral infections like hepatitis C and alcoholism. It was the 12th leading cause of death in the United States in 2000, accounting for more than 25,000 deaths.[2]

Cirrhosis leads to two major syndromes of portal hypertension and hepatic insufficiency.[3]

Peripheral and splanchnic vasodilatation with the resulting hyperdynamic circulatory state is typical of cirrhosis and portal hypertension. Cirrhosis can remain compensated for many years prior to the development of a decompensating event.[4]

Decompensated cirrhosis is marked by the development of many complications. These include jaundice, variceal hemorrhage, ascites or encephalopathy.[5]

Ascites is the most common complication of cirrhosis. It is associated with a poor quality of life, increased risks of infections and renal failure, and a poor long-term outcome.[6,7] Approximately 50% of patients with compensated cirrhosis will develop ascites over 10 years period. This occurrence is an important mile-stone in the history of endstage liver disease, because only 50% of patients will survive 2-5 years after its onset.[8,9]

The chief factor contributing to ascites is splanchnic vasodilatation.[10] Increased hepatic resistance to portal flow leads to portal hypertension. This causes collateral vein formation and shunting of blood to the systemic circulation. In the advanced stages of cirrhosis, the effective arterial blood volume decreases due to splanchnic vasodilatation.[11]

To maintain the arterial pressure, homeostatic activation of vasoconstrictor, antinatriuretic and antidiuretic factors occurs resulting in salt and fluid retention. Later on, there is marked impairement in renal excretion of free water due to increased levels of ADH and renal vasoconstriction. All these changes contribute to dilutional hyponatremia and hepatorenal syndrome.[12]

In mild to moderate ascites, the mainstay of treatment has been sodium restriction and intake of diuretics. 80-90% of the patients responds to the combination initially.[13]

Material and Methods

The study was conducted in Medical Unit-III, Services Hospital, Lahore from August 2004 to January 2005 over a period of 6 months. It was an interventional experimental study. Sixty cirrhotic patients with ascites were selected on the basis of selection criteria. They were randomized into two groups. Purposive selection with random allocation to both groups was done.

Inclusion Criteria

1. Age group of 12-60 years.
2. Cirrhosis.
3. Mild to moderate ascites.
4. Serum sodium > 130mmol/L.
5. Serum creatinine <1.4mg/dl.
6. Normal kidneys on ultrasound.

Exclusion Criteria

1. Diabetes mellitus (as per history).
2. Hypertension (as per history).
3. Proteinuria (>++ on urine dipstick testing).
4. Serum bilirubin >3.0mg/dl.
5. Serum albumin <2.8g/dl.
6. Prothrombin time prolongation >16 seconds.
7. Hepatic encephalopathy.
8. Subacute bacterial peritonitis.
9. Exudative ascites.
10. Chronic renal failure (serum creatinine more than 1.5mg/dl).
11. Congestive cardiac failure (elevated jugular venous pressure on clinical examination).

Procedure

The patients were selected on the basis of inclusion and exclusion criteria. The procedure was explained in detail to the patient and written consent was taken. Detailed history regarding duration of cirrhosis, duration of ascites, fever, abdominal pain, abdominal tenderness, intake of diuretics, and salt restriction was taken. General physical examination and detailed systemic examination was carried out, to establish the diagnosis of cirrhosis and ascites. Palmar erythema, spider naevi, splenomegaly, jugular venous pulse, edema, shifting dullness and fluid thrill was looked for in each patient.

Ultrasound abdomen was done to confirm cirrhosis and presence of ascites.

Patients were asked to get their serum sodium, serum creatinine, serum albumin, prothrombin time, serum bilirubin and urine for proteins checked. These investigations were used to include or exclude them from the study.

Group A

Half of the patients were given 5gms of salt with 2 boiled eggs at 9.00pm. They were advised strict fluid restriction from 9:00 pm till 6:00 am.

Group B

Rest of the thirty patients took only 2 boiled eggs at night with no salt intake and fluid restriction.

Group A was given water free salt at night while group B was advised strict salt restriction.

100mg of spironolactone and 40mg of furosemide were given in the morning to both groups. Standard diet providing 2500 kcal/day and 2gm of salt was given throughout the day to both groups. Patients were assessed daily for 9 days. Their weight and abdominal girth at the level of umbilicus was checked daily. Serum sodium and creatinine was checked on day 3 and day 9.

Increase in the dose of diuretics or abdominal paracentesis was done, if there was failure of medical therapy or the clinical condition of the patient deteriorated. These patients were taken as end point in study and further follow up in terms of weight measurement and abdominal girth measurement was not done. However their initial data was included in analysis. All the data was collected by using a structured questionnaire.

Outcome Variables

At the end of study, all gathered data in questionnaire was tabulated. Following variables were analyzed at the end of study:

1. Mean Reduction in weight in 9 days.
2. Mean Reduction in abdominal girth in 9 days.
3. Sodium levels during the study.
4. Need for increase in dose of diuretics.
5. Need for therapeutic ascitic tap.

Results

Data analysis was computer based. It entered was in SPSS-10 and analyzed. Data was presented as means. The 2 means were tested using student t-test, to find out significance of data. During the study period, 60 cirrhotic patients with ascites presenting in medical outdoor and emergency department of Services Hospital, Lahore and fulfilling our inclusion criteria were randomized into two groups. Both groups were given standard treatment of salt restriction and diuretics during the day. Group-A was given 5gm of salt at night with overnight fluid restriction, while group-B was not given added salt.

The data analysis was computer based. Inter group comparison was done through student t-test.

Mean (±SD) weight reduction of the patients belonging to group-A in 9 days was 2.39±1.54kg. Patients in group-B showed mean (±SD) weight reduction of 1.43±1.27kg in 9 days. It was significantly more in group-A (p = 0.011) (**Table 1).**

Table-1: Mean reduction in weight of the subjects in 9 days.

Wt. Reduction in (in kg) in 9 days	Group-A n=30 No.	%	Group-B n=30 No.	%
0-1	8	26.66	13	43.33
1.1-2	2	6.66	8	26.66
2.1-3	7	23.33	6	20.00
3.1-4	10	33.33	2	6.66
4.1-5	3	10.00	1	3.33
Total	30	100.00	30	100.33
Mean± SD	2.39±1.54		1.43±1.27	

P value = 0.011 (significant)

Mean (±SD) reduction in the abdominal girth over 9 days was 2.01±1.26 inches in group-A and 1.07±1.05 inches in group-B. It was significantly more in group-A (p = 0.003) **(Table 2).**

Table-2: Mean reduction in abdominal girth of the subjects in 9 days.

Abdominal girth in (inches) in 9 days	Group-A n=30 No.	%	Group-B n=30 No.	%
0-1	7	23.33	15	50.00
1.1-2	7	23.33	8	26.66
2.1-3	10	33.33	6	16.66
3.1-4	6	20.00	2	6.66
Total	30	100.00	30	100.00
Mean± SD	201±1.26		1.07±1.05	

P value = 0.003 (significant)

The mean (±SD) serum sodium levels on day 9 of the study was 136.5±3.22mmol/L in group-A and 132.6±3.25mmol/L in group-B. Serum sodium levels were significantly maintained in group-A at the end of the study as compared to group-B (p value = 0.00001) **(Table 3).**

Table-3: Serum sodium of the subjects on day 9.

Serum Sodium (mmol/L) 9 days	Group-A n=30 No.	%	Group-B n=30 No.	%
125-130	0	0.00	7	56.66
131-135	12	40.00	9	30.00
136-140	14	46.66	4	13.33
141-145	4	13.33	0	0.00
146-150	0	0.00	0	0.00
Total	30	100.00	30	100.00
Mean± SD	136.5±3.22		132.6±3.25	

p value = 0.00001 (significant).

Discussion

The medical management of ascites in cirrhosis is still a matter of debate and varies from center to center. Dilutional hyponatremia is present in most of the patients with ascites due to high levels of anti-diuretic hormone and is worsened by the combination of salt restriction and diuretics. This leads to diuretic tolerance and the dose of diuretics has to be increased.[14] This worsens the hyponatremia and causes further shrinkage of circulatory volume, which results in the development of refractory ascites and hepato-renal syndrome.

This study was conducted in an attempt to demonstrate the role of sodium intake in the management of cirrhotic ascites. Salt given in the absence of water leads to correction of dilutional hyponatremia by relative increase in sodium, which then maintains the circulatory volume, by drawing the ascitic fluid in the vessels.

The increase in circulatory volume inhibits the secretion of ADH and aldosterone. This leads to decrease in secondary renal salt and water retention and also facilitates renal water excretion. Secondly, serum sodium levels increase the filtered load of sodium which increases the response of diuretics. So the aim was to demonstrate improved diuretic response, as a result of increased filtered load of sodium and maintained circulatory volume.

The results were compared with reference to daily reduction in weight, abdominal girth, correction of hyponatremia, prevention of hepato-renal syndrome and the use of invasive procedures.

The results of this study will help in bringing a change in the management plan of ascites in cirrhotic patients, thus reducing the associated complications, morbidity and mortality.

Conclusion

Dilutional hyponatremia is a frequent complication of cirrhosis. It is aggravated by salt restriction and diuretic therapy. This limits the usefulness of diuretics. No effective therapy exists for it. One of the options available is the intake of water free salt, so as to correct the dilutional hyponatremia. Correction of this hyponatremia by salt intake tends to improve the response of diuretics by increasing the circulatory volume, inhibiting the synthesis of antidiuretic hormone and increasing filtered sodium load. So patients can be maintained on low dose diuretics for longer duration, thus preventing their side effects.

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