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COMPREHENSIVE ANALYSIS OF ASCITES: UNRAVELING OPTIMAL MANAGEMENT STRATEGIES FOR ENHANCED TREATMENT OUTCOMES"

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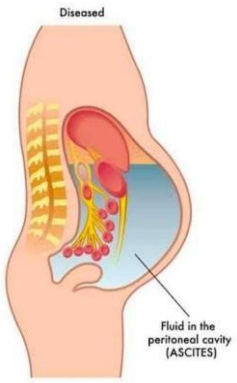
INTRODUCTION

Ascites is characterized as the amassing of overabundance liquid in the peritoneal hole. Liquid gathers when it enters the peritoneal pit from the mesenteries, the peritoneum and hepatic surface at a rate more noteworthy than can be gotten back to the flow through the vessels and lymphatics **(Bruce, 1935).**

Ascites frequently have been characterized into three grades, comprising Grade 1 which shows gentle ascites just perceivable by ultrasound assessment, Grade 2 which is moderate ascites and Grade 3 which shows the extreme ascites with checked stomach distension **(Moore, 2003).** Ascites possibly happens when portal hypertension has occurred and is basically connected with a failure to discharge a sufficient measure of sodium into pee, prompting a positive sodium balance **(Ripoll et al., 2007).**

An enormous group of proof proposes that renal sodium maintenance in patients with cirrhosis is optional to blood vessel splanchnic vasodilation. This causes a diminishing in successful blood vessel blood volume with enactment of blood vessel and cardiopulmonary volume receptors, and homeostatic initiation of vasoconstrictor and sodium-holding frameworks (i.e., the thoughtful sensory system and the renin- angiotensin-aldosterone framework). Renal sodium maintenance prompts development of the extracellular liquid volume and arrangement of ascites and edema **(Schrier et al., 1998).** The improvement of ascites is related with an unfortunate forecast and disabled personal satisfaction in patients with cirrhosis **(Tandon and Garcia Tsao, 2008)**. The control of ascites ought to be embraced at whatever point it becomes important to ease stomach distress, mitigate windedness, further

develop hunger, forestall unconstrained bacterial peritonitis (SBP), or lessen the gamble of stomach wall hernia. In a nutshell, the primary objective is to work on the personal satisfaction with the essential of keeping a stable hemodynamic condition **(Runyon, 1986).**



REVIEW OF LITERATURE

Ascites is a pathologic accumulation of peritoneal fluid commonly observed in decompensated cirrhotic states. Its causes are multi-factorial, but principally involve significant volume and hormonal dysregulation in the setting of portal hypertension **(Moore and Van, 2013).** The sign of ascites is a significant milestone in the progression of cirrhosis. It is the most considered normal reason for clinic confirmations and subsequently latest expenses **(Ginés et al., (1987).** It predicts expanded 1-year mortality **(Planas et al., 2006).** and functions as a marker of risk-stratification for orthotopic liver transplantation (OLT) **(Arroyo et al., 1976).**

Ascites can be of three severities: grade I, wherein it is analyzed by abdominal ultrasound, which requires roughly 100 mL of liquid inside the peritoneum (review that ordinary volume is around 25-50 mL); grade II, suggesting no less than 1000 mL of peritoneal liquid, which can be distinguished with actual assessment through the exemplary test discoveries of sagging flanks, shifting dullness, fluid wave, and the more difficult and seldom used Puddle's sign; grade III, appeared as a grossly expanded abdomen, suggesting liters of ascitic liquid. This last grade can evoke a serious type of uneasiness, and might be depicted as a tense ascites **(Runyon et al., 2010).**

As per the peripheral blood vessel vasodilatation speculation, portal hypertension causes an increment of vasodilators in the circulatory system and a subsequent decrease of compelling circulating volume. It brings about an enactment of sympathetic nervous system (SNS),

renin-angiotensin-aldosterone system (RAAS), nonosmotic discharge of vasopressin and a subsequent water and sodium maintenance that incites a liquid progress to the extravascular space, specifically, to the peritoneal cavity (**Møller et al**., **2001).** One more wellspring of vasodilators might be the gut, in which bacterial excess and intestinal dysbiosis may cause intestinal irritation and breakdown of the intestinal barrier. Bacterial items might animate the arrival of proinflammatory cytokines that increment splanchnic arterial vasodilatation **(Bernardi M et al., 2015).**

The diagnosis of ascites is usually carried out by patient history and physical assessment, and can be confirmed by abdominal ultrasound. The reason for ascites is recognized by set of history, physical assessment, laboratory tests, abdominal imaging, and ascitic fluid examination. Patients with ascites typically present with abdominal expansion, which may likewise be related with abdominal distention, early satiety, weight gain, and shortness of breath. Also, patients ordinarily have side effects and indications of the basic reason for ascites **(Runyon et al., 1992).** Since cirrhosis is the most widely recognized reason for ascites history and assessment physically ought to be coordinated for side effects and indications of cirrhosis as well as chance variables for advancement of cirrhosis. Patients with cirrhosis might have different side effects related with hepatic decompensation, for example, hepatic encephalopathy jaundice or gastrointestinal bleeding. Actual assessment of patients with ascites because of liver cirrhosis for the most part uncovers spider angioma, palmar erythema, jaundice, muscle wasting, gynecomastia, leukonychia, parotid extension, and abdominal wall collaterals. The liver and spleen might be substantial. Patients likewise should be explored for risk factors for cirrhosis including alcohol, viral hepatitis B and C, autoimmune system liver disease, and different reasons for cirrhosis. The people who come up short on obvious reason for cirrhosis ought to likewise be examined concerning lifetime body weight and diabetes as nonalcoholic steatohepatitis has been distinguished to be the reason for cirrhosis in a considerable lot of these patients **(Poonawala et al., 2000).**

The enactment of the renin-aldosterone-angiotensin-framework in patients with liver cirrhosis causes hyperaldosteronism and expanded reabsorption of sodium along the distal tubule. In this manner, aldosterone antagonists like spironolactone or its dynamic metabolite potassium canrenoate are viewed as the diuretics of choice. Patients with mild to moderate ascites are treated with a monotherapy of spironolactone. The beginning portion is 100-200 mg/d. A monotherapy with a loop diuretic like furosemide is less compelling contrasted with

spironolactone and isn't recommended. On the off chance that the reaction to 200 mg spironolactone inside the initial fourteen days isn't adequate, furosemide with an underlying portion of 20-40 mg/d is added. If important, the spironolactone portion is expanded stepwise up to 400 mg/d and the furosemide portion is expanded up to 160 mg/d **(Bernardi et al., 1985).**

It is for the most part prescribed to intake furosemide orally, since intravenous organization bears the risk of azotemia (**Daskalopoulos et al., 1987).** A mix treatment of spironolactone and furosemide abbreviates the reaction time to diuretic treatment and limits unfavorable impacts, for example, hyperkalemia (**Stanley et al., 1989).** A laid out plot for an underlying blend treatment is 100 mg spironolactone and 40 mg furosemide each day given in the morning **(Runyon et al., 1994).** A few examinations have assessed the more current loop diuretic torasemide in patients with liver cirrhosis and ascites. Torasemide was demonstrated to be all around as successful and protected as furosemide and is viewed as an option in the treatment of ascites **(Abecasis et al., 2001).** Amiloride is an option in contrast to spironolactone in patients with excruciating gynecomastia. Amiloride is given in a portion of 10-40 mg/d however is less compelling than potassium canrenoate **(Angeli et al., 1994).**

Refractory ascites happens in patients who don't respond to diuretic treatment, who have diuretic-prompted difficulties, or for whom ascites repeats quickly after helpful paracentesis. Once ascites becomes refractory, endurance diminishes to half at 1 year **(Singhal et al., 2012).**

AIMS AND OBJECTIVE

AIM- The primary aim of this analysis is to investigate ascites, focusing on unraveling the optimal management strategies for achieving enhanced treatment outcomes.

OBJECTIVE - To study optimal management strategies for ascites.

MATERIAL AND METHODS

Study design and site: Comprehensive Analysis of Ascites: Unraveling Optimal Management Strategies for Enhanced Treatment Outcomes"

Study population: Adult patients with ASCITES who were being treated in a tertiary hospital were included in the investigation's study population.

Study material: Data was collected from old case records of medical record department.

Data was collected from patients reporting to general medicine between November 2022 to April 2023**.**

Study Duration

6 Months Prospective and 2 Years Retrospective

Inclusion Criteria

Male or female subject aged between 18 -70yrs.

Subject with diagnosis of ASCITES (based on clinical, laboratory, endoscopic and ultra sonographic features.)

Subject who has been hospitalized for ASCITES

Exclusion Criteria

Below 18 years

Pregnant women and lactating women.

Patients on other medication (AYUSH)

Patients having no diagnostic evidence of cirrhosis.

STUDY PROCEDURES

Study was retrospective and prospective, of patients of ASCITES reporting at Ananta Institute of Medical Sciences and Research Centre. Data will be collected from case records of patients maintained in medical record department and also from general medicine department.

Data collection sheets were prepared which included the details of patient's, such as name, age, sex, including relevant history, examination details, diagnostic test (USG abdomen, endoscopy, CT SCAN, MRI) and laboratory investigation including level of serum SGOT, SGPT, serum total bilirubin, serum albumin, AG ratio, platelet count and PT- INR was collected and recorded.

Mentioned data was compiled and analyzed to record incidence and prevalence of liver cirrhosis during the duration mentioned. Analysis was also done to check the comorbidity occurring in such patients. It was further analysed to find out the treatment options.

ETHICAL CONSIDERATIONS

Before proceeding with collecting data, the appropriate academic ethics committee's consent was needed. All participants gave their informed consent, which protected their privacy and confidentiality throughout the study.

STATISTICAL ANALYSIS

The data collected and compiled was entered in MS EXCEL, MS. WORD, DESCRIPTIVE TEST, ANOVA TEST.

LABORATORY INVESTIGATIONS	NORMAL RANGE
Hb(g/dl)	11.5- 14.5 g/dl
TLC(10*3 cells/mm*3)	4.0- 11.0 (10*3 cells/mm*3)
PLA(per mlc)	150-400 10*3 cells/ul
BT(mg/dl)	0.2-1.3 mg/dl
BD/BI	0-0.3 / 0.0-1.1 mg/dl
SGPT(U/L)	13-41 U/L
SGOT(U/L)	5-35 U/L
Na(mmol/L)	136-145 mmol/L
K(mmol/L	3.5-5.1mmol/L
ALB(g/dl)	3.2-5.0 g/dl
GLO(g/dl)	2.3-3.6 g/dl
A/G	1.2-1.5
PT (secs)	12-4 secs
INR (secs)	0.85- 1.15 sec
S.Cr (mg/dl)	0.7-1.4 mg/dl

INVESTIGATIONS FOR ASCITES

1) SAAG (Serum Ascitic Albumin Gradient)

When SAAG > 1.1g/ dl: strongly suggest portal hypertension

When SAAG < 1.1g/ dl: non portal hypertensive

2) USG

Ultrasound can quantify the volume of ascites and aid in the decision for fluid drainage.

RESULT

Biochemical Parameters of Patients With Ascites

S. NO.	PARAMETERS	MEAN ± SD
1	HAEMOGLOBIN	9.73 ± 2.75
2	TOTAL LEUKOCYTE COUNT	9.94 ± 14.08
3	PLATELET COUNT	255.58 ± 824.40
4	BILURIBIN TOTAL	2.7 ± 4.34
5	SGPT	40.3 ± 46.08

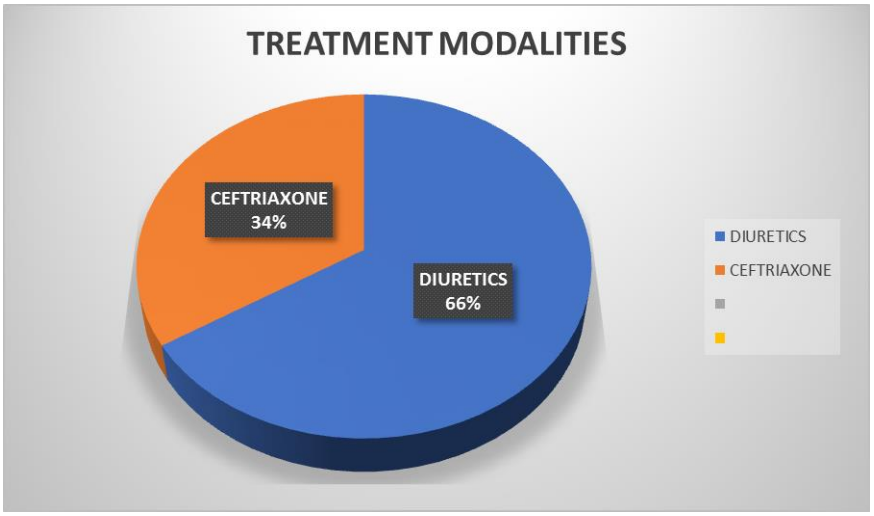
6	SGOT	64.38 ± 64.71
7	SODIUM	134.29 ± 9.00
8	POTASSIUM	4.53 ± 4.11
9	ALBUMIN	2.69 ± 0.92
10	GLOBULIN	3.45 ± 1.13
11	PT	18.77 ± 5.76
12	INR	1.58 ± 1.43
13	SERUM CREATININE	1.59 ± 2.51

TREATMENT MODALITIES

In our study of 100 patients with liver cirrhosis, we assessed the treatment modalities used for managing the condition. The distribution of treatment options among the patients is as follows:

1. **Diuretics**: Among the 100 patients, 72 individuals (72%) were prescribed diuretics as part of their treatment regimen. Diuretics are commonly used to manage ascites, a common complication of liver cirrhosis characterized by the accumulation of fluid in the peritoneal cavity.
2. **Ceftriaxone**: 37 patients (37%) received ceftriaxone, an antibiotic commonly used for the treatment of spontaneous bacterial peritonitis (SBP). SBP is an infection that occurs in individuals with ascites and can lead to serious complications if left untreated.

DRUGS	**NO. OF CASES**
Diuretics	**72**
Ceftriaxone	**37**

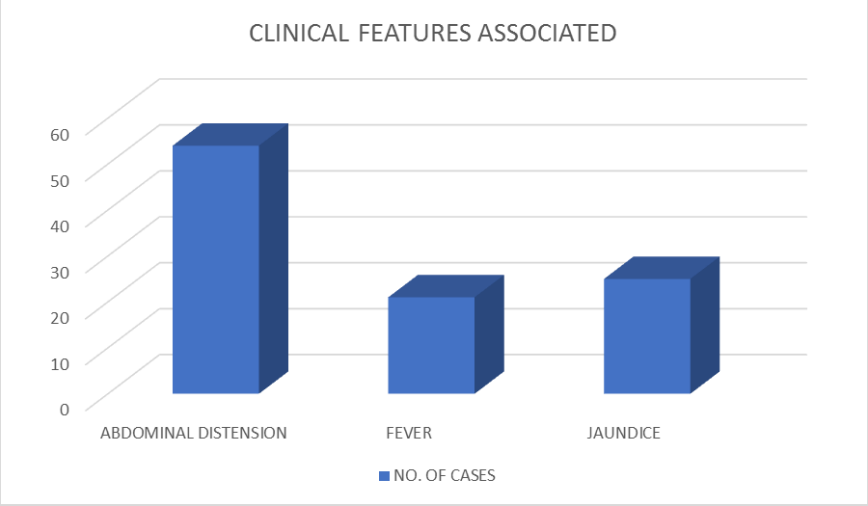


CLINICAL FEATURES ASSOCIATED

In a study involving 100 patients diagnosed with liver cirrhosis, the following symptoms were observed:

1. Abdominal Distension: The most prevalent symptom in the study was abdominal distension, observed in 54 patients. Abdominal distension refers to the abnormal swelling or enlargement of the abdomen.
2. Fever: Out of the 100 patients, 21 individuals exhibited fever as a symptom of liver cirrhosis.
3. Jaundice: A total of 25 patients in the study presented with jaundice, which is a yellowing of the skin and eyes due to impaired liver function

CLINICAL FEATURES	NO. OF CASES
ABDOMINAL DISTENSION	54
FEVER	21
JAUNDICE	25



PERCENTAGE OF DIURETIC USED

DYTOR +	**34**
LASIX	**34**
ALDACTONE	**36**
TORSEMIDE	**9**

CONCLUSION

In conclusion, liver cirrhosis is a progressive condition characterized by the development of fibrosis and nodular transformation of the liver tissue, resulting from persistent injury. It can be caused by various factors such as viral infections, toxins, genetic disorders, or autoimmune processes. The major complications associated with cirrhosis include varices, ascites, hepatic encephalopathy, portal hypertension. Ascites, the accumulation of excess fluid in the peritoneal cavity, is closely related to portal hypertension and is associated with sodium retention and fluid imbalance.

Diagnosis and management of ascites involves various clinical assessments, imaging techniques, and laboratory tests. Treatment strategies focus on reducing ascites, managing complications, and addressing the underlying cause of cirrhosis. Pharmacological interventions, endoscopic procedures, and liver transplantation may be considered depending on the severity of the condition.

Overall, understanding the management strategies in ascites is crucial for improving patient outcomes and enhancing their quality of life. Further research is needed to explore novel therapeutic approaches and improve the prognosis of individuals with this challenging condition.

REFERENCES

1. Bruce A. Ascites and spontaneous bacterial peritonitis Sleisinger and frodtran gastrointestinal disease pathology, diagnosis, management, 1935; 2.
2. Moore KP, Wong F, Gines P, Bernardi M, Ochs A, Salerno F, et al. The management of ascites in cirrhosis: report on the consensus conference of the International Ascites Club. Hepatology [Internet], 2003. [cited 2023 Jul 10].
3. Ripoll C, Groszmann R, Garcia-Tsao G, Grace N, Burroughs A, Planas R, et al. Hepatic venous pressure gradient predicts clinical decompensation in patients with compensated cirrhosis. Gastroenterology [Internet], 2007. [cited 2023 Jul 10].
4. Schrier RW, Arroyo V, Bernardi M, Epstein M, Henriksen JH, Rodés J. Peripheral arterial vasodilation hypothesis: a proposal for the initiation of renal sodium and water retention in cirrhosis. Hepatology [Internet], 1988. [cited 2023 Jul 10)
5. Tandon P, Garcia-Tsao G. Bacterial infections, sepsis, and multiorgan failure in cirrhosis. Semin Liver Dis [Internet], 2008. [cited 2023 Jul 10]
6. Runyon BA, Van Epps DE. Diuresis of cirrhotic ascites increases its opsonic activity and may help prevent spontaneous bacterial peritonitis. Hepatology [Internet], 1986. [cited 2023 Jul 10].
7. Moore CM, Van Thiel DH. Cirrhotic ascites review: Pathophysiology, diagnosis and management. World J Hepatol [Internet], 2013; [cited 2023 Jul 10]; 5(5): 251–63.