ORIGINAL RESEARCH

Assessment of cases of ascites

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ABSTRACT

Background: The identifiable and pathological accumulation of fluid in the peritoneal cavity is referred to as ascites.The present study was conducted to assess cases of ascites. **Materials & Methods:** 116 patients of ascites of both genders were selected. Measurements like icterus, parotid edema, and ascites symptoms were recorded, and tests like abdominal ultrasonography were carried out. Biochemistry, cytology, gram staining, acid-fast Bacillus staining, malignant cells, growth, and sensitivity were all examined in ascidic fluid. **Results:** Out of 116 patients, males were 90 and females were 76. The risk factors in patients were diabetes in 75, alcoholism in 40, and blood transfusion in 52 cases. Physical findings were gynecomastia in 42, icterus in 57, palpable liver in 31, pleural effusion in 52peripheral edema in 13, murmur in 28, elevated JVP in 24, and axillary hair loss in 30 patients. The difference was significant ($P< 0.05$). **Conclusion:** Common physical findings were gynecomastia, icterus, palpable liver, pleural effusion, peripheral edema, murmur, elevated JVP, and axillary hair loss.

Keywords: Ascites, Diabetes, palpable liver

INTRODUCTION

The identifiable and pathological accumulation of fluid in the peritoneal cavity is referred to as ascites. It is typically a clinical finding that a diagnostic paracentesis can confirm. Subclinical fluid volume (less than 1.5 liters) can be found on abdominal computed tomography or ultrasonography. Several anatomical, pathophysiological, and metabolic alterations lead to the development of ascites.[1] One can categorize the specific causes of ascites into two groups: noncirrhotic ascites, which are unrelated to portal hypertension, and cirrhotic ascites, which are connected with portal hypertension.As a result of sinusoidal portal hypertension, which affects capillary pressure, permeability, and the buildup of retained fluid in the abdominal cavity, ascites occur in people with liver cirrhosis.[2]

Transudation is the term for this fluid buildup mechanism. Because transudation does not primarily involve capillary damage, the transit of high molecular-weight compounds is restricted.[3] Ascites production is secondary to increased vascular permeability brought on by the inflammatory process, tumoral invasion, or mechanical damage to the peritoneum or intraperitoneal organs. Another method of ascites creation is known as exudation. Ascites is a common side effect of liver cirrhosis in many patients. Ascites in chronic liver disease can arise from a number of causes.[4] The kidneys play a major part in salt and water retention through intricate processes. The occurrence of distinctive circulatory anomalies in cirrhotic patients serves as the foundation for the "peripheral arterial vasodilatation hypothesis." Increased cardiac output, arterial hypotension, lower peripheral vascular resistance, and splanchnic vasodilatation are observed in these patients.[5]The present study was conducted to assess cases of ascites.

MATERIALS & METHODS

The present study comprised 116 patients of ascites of both genders. All were informed regarding the study and their written consent was obtained.

Data such as name, age, gender, etc. was recorded. Every patient underwent a general physical checkup. Measurements like icterus, parotid edema, and ascites symptoms were recorded, and tests like abdominal ultrasonography were carried out. Biochemistry, cytology, gram staining, acid-fast Bacillus staining, malignant cells, growth, and sensitivity were all examined in ascidic fluid. Adenosine deaminase (ADA) and the serum-ascites albumin gradient (SAAG).Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 116		
Gender	**Males**	**Females**
Number	90	76

Table I shows that out of 116 patients, males were 90and females were 76.

Table II Assessment of risk factors of ascites

Risk factors	Number	P value
Diabetes	75	0.92
Alcoholism	40	
Blood transfusion	52	

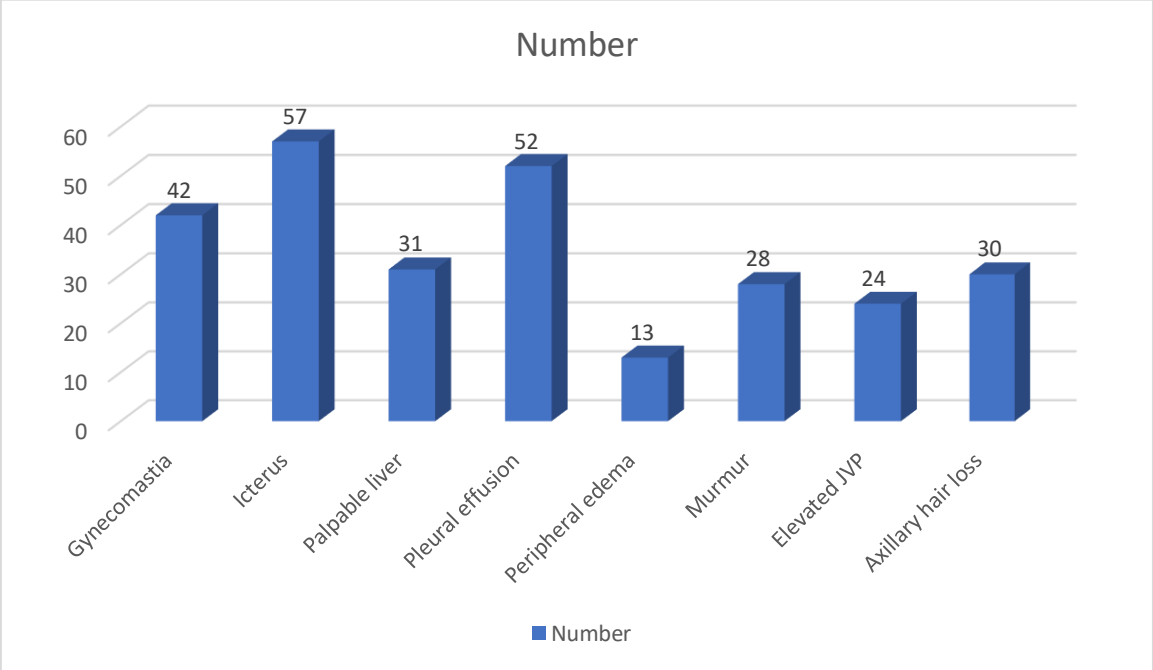
Table II show that risk factors in patients were diabetes in 75, alcoholism in 40 and blood transfusion in 52 cases. The difference was non- significant (P> 0.05).

Table III Assessment of physical findings

Physical findings	Number	P value
Gynecomastia	42	0.05
Icterus	57	
Palpable liver	31	
Pleural effusion	52	
Peripheral edema	13	
Murmur	28	
Elevated JVP	24	
Axillary hair loss	30	

Table III, graph Ishow that physical findings were gynecomastiain 42, icterus in 57, palpable liver in 31, pleural effusion in 52peripheral edema in 13, murmur in 28, elevated JVP in 24, and axillary hair loss in 30 patients. The difference was significant (P< 0.05).

Graph I Assessment of physical findings



DISCUSSION

Ascite fluid might build up quickly or gradually, depending on what's causing it. Mild ascites may have no symptoms. A modest case of ascites might only result in weight gain and an expansion of the abdomen. Excessive fluid intake can cause pain in the abdomen, cause hernias to formespecially umbilical herniasand limit the patient's movement.[6] Liver disease may present with ascites initially. It is crucial to ascertain a patient's past medical history on risk factors for liver disease, such as drug or alcohol misuse, alcohol intake, blood transfusions, or

hepatitis. When ascites appears out of the blue in a previously stable cirrhosis patient, hepatoma should be suspected. In clinical practice, ascites differential diagnosis is still a challenge. Treatment decisions are made per etiologies.[7]Portosystemic shunting and/or decreased clearance of vasodilator drugs such as nitric oxide, endotoxins, prostacyclin, glucagon, and adenosine are potential causes of vasodilatation in ascites.This vasodilatation of the splanchnic and peripheral vessels is interpreted as a decrease in the effective plasma volume. Effective hypovolemia activates the sympathetic nervous system, renin-angiotensin-aldosterone system, and baroreceptor-mediated activation, which results in renal vasoconstriction and salt.[8]

When making a differential diagnosis, the initial assessment of the ascitic fluid's physical appearance can provide helpful information. Peritoneal fluid is typically transparent to pale yellow in color. Chylomicrons, which are lipoprotein particles made mostly of triglycerides, are a characteristic of milky ascites, also known as chylous ascites.[9]Chylous ascites have numerous recognized causes, such as cirrhosis, nephropathies, cardiopathies, malignancies, infections (tuberculosis and parasitic), trauma, inflammatory processes, and congenital abnormalities. Chylous ascites in adults is primarily caused by abdominal malignancy, but congenital lymphatic abnormalities are more likely to produce the condition in children.[10] It should be mentioned, too, that cloudy/turbid ascites, also known as pseudochylous ascites are linked to pancreatitis, peritonitis, and bacterial infections. Thus, to differentiate chylous ascites from other types of ascites, chylomicrons, and a high triglyceride concentration are both required.[11]

We found that out of 116 patients, males were 90 and females were 76. The risk factors in patients were diabetes in 75, alcoholism in 40, and blood transfusion in 52 cases. Huang et al[12]studied 146 cirrhosis patients diagnosed with a first episode of SBP from 2005 to 2006. Of these, 89 patients survived; the survivors were divided into two groups based on recurrence and non-recurrence of SBP, and clinical parameters, survival time and cause of death were analyzed.The in-hospital mortality was 39% (57/146). The SBP recurrence rate was 42.7% (38/89). The survival rate between patients with recurrent SBP and those without recurrence did not differ (P=0.092). Sepsis was the major cause of death in the recurrent SBP group, but not in the non-recurrent group. Serum albumin level before discharge and β-blocker use between the two groups differed significantly (P<0.0001). Using the cut-off point for serum albumin level before discharge of 2.85 g/dl as a predictor for the recurrence of SBP, the sensitivity was 70.2% and the specificity was 76.3%. Furthermore, long-term survival of the group with high albumin before discharge was better than that of the corresponding group with low albumin.

We found that physical findings were gynecomastia in 42, icterus in 57, palpable liver in 31, pleural effusion in 52 peripheral edema in 13, murmur in 28, elevated JVP in 24, and axillary hair loss in 30 patients.Ascitic glucose concentration is frequently much lower than usual in tuberculous ascites, according to Mansour-Ghanaei et al.[13] This makes it an indicator for distinguishing tuberculosis from other illnesses, like cirrhosis. This is in line with the recommendation made by Wilkins et al.[14]The ascitic/blood glucose ratio is a helpful test for distinguishing tuberculous peritonitis from ascites from other causes. Nevertheless, there was no discernible variation in the glucose values across patients with SAAG levels above or below 1.1 g/dL. Consequently, ascitic glucose analysis is not widely used in ordinary practice due to its low diagnostic sensitivity and specificity.

CONCLUSION

Authors found that physical findings were gynecomastia, icterus, palpable liver, pleural effusion, peripheral edema, murmur, elevated JVP, and axillary hair loss.

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