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Etiology of ascites in adults living in Rohilkhand region, India: A hospital-based study

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Abstract
The study was conducted in a tertiary care hospital of Rohilkhand region located in the Northern India, over a period of 8 months from January 2016 to August 2016. All patients diagnosed as ascites on the basis of history, physical examination, ultrasonography, and age >18 years admitted in medicine department of Rohilkhand Medical College & hospital (RMCH), Bareilly (U.P) were included in the study. Patients of peritonitis and unwilling to participate in the study were excluded. The patients included in the study were evaluated by detailed history and physical examination. Ascitic fluid paracentesis and virologic testing for Hepatitis B and C were done in all the patients. Ascitic fluid was analyzed for cytology, biochemistry, gram staining, acid fast bacillus staining, malignant cells, culture, and sensitivity and for adenosine deaminase (ADA). Serum-ascites albumin gradient (SAAG) was estimated in all patients. Ultrasound abdomen was done in all patients followed by computed tomography if the ultrasound was inconclusive or if there was evidence of any intra abdominal malignancy. Specific etiology oriented investigations were carried out. Other investigations such as antinuclear antibodies, an antibody against liver-kidney-microsomes, anti smooth muscle antibodies, serum ceruloplasmin, urinary copper levels and slit lamp examination for Kayser–Fleisher ring were done if indicated. Cirrhosis was diagnosed on basis of physical examination and ultrasonography. Tubercular ascites was diagnosed on the basis of lymphocytic predominance on cytology, low SAAG (<1.1), high protein (>2.5), ADA more than 40 IU/L. The data, thus collected, was analyzed on Microsoft Excel sheet 2010 and percentages were calculated.
Cirrhosis was found to be the most common cause of ascites (60.78% patients), followed by tuberculosis (15.68% patients). Although cirrhosis was the leading cause of ascites, causes other than cirrhosis were present in around 37% of the patients. Among cirrhotics Hepatitis b was the leading cause of cirrhosis (41.93% patients). India accounts for a large proportion of the worldwide HBV burden. India harbors 10-15% of the entire pool of HBV carriers of the world. It has been estimated that India has around 40 million HBV carriers. About 15–25% of HBsAg carriers are likely to suffer from cirrhosis and liver cancer. While horizontal transmission in childhood appears to be a major route of transmission, the role of vertical transmission is probably underestimated. Blood transfusion and unsafe therapeutic injections continue to be important modes of transmission of HBV. There should be a focus on screening for hepatitis b in high risk individuals including, intravenous drug users, persons who receive blood transfusions, acupuncture, tattooing, unsafe injection practices, health care workers at risk of occupational exposure, etc. Identification and if necessary treatment of the HBV infected persons would play a role in decreasing the spread of the disease. Focus on primary prevention by hepatitis B vaccination is recommended. The advantages of this study is that the causes of ascites in Rohilkhand region were known which can help us in directing treatment decision, predicting the outcome and in formulating the future preventive strategy.

Keywords: Etiology, adults living, ultrasonography, ultrasound abdomen

1. Introduction
The word ascites is of Greek origin (askos) and means bag or sac. The word is a noun and describes pathologic fluid accumulation in the peritoneal cavity. Although the diagnosis of ascites may be suspected on the basis of the history and physical examination, final confirmation is based on successful abdominal paracentesis or detection of ascites on imaging. Determination of the cause of ascites is based on the results of the history, physical examination, and ascitic fluid analysis. Abdominal paracentesis is the most rapid and cost effective method of diagnosing the cause of ascites [1] Treatment decisions are directed according to the etiological profile.

Cirrhosis is the leading cause of ascites in both developed and developing countries [2-4]. In India cirrhosis of liver is the most common cause of ascites followed by tuberculosis [5] Epidemiological data on the etiological aspects of ascites are lacking from this region. Therefore, this study was planned and conducted in a tertiary care hospital located in the Rohilkhand region.

1.1 Pathogenesis of ascites formation

A detailed description of the pathogenesis of ascites formation is beyond the scope of this article but more detailed reviews are available [6-8]. There are two key factors involved in the pathogenesis of ascites formation-namely, sodium and water retention, and portal (sinusoidal) hypertension.

1.2 Role of portal hypertension

Portal hypertension increases the hydrostatic pressure within the hepatic sinusoids and favours transudation of fluid into the peritoneal cavity. However, patients with presinusoidal portal hypertension without cirrhosis rarely develop ascites. Thus patients do not develop ascites with isolated chronic extrahepatic portal venous occlusion or non-cirrhotic causes of portal hypertension such as congenital hepatic fibrosis, except following an insult to liver function such as gastrointestinal haemorrhage. Conversely, acute hepatic vein thrombosis, causing postsinusoidal portal hypertension, is usually associated with ascites. Portal hypertension occurs as a consequence of structural changes within the liver in cirrhosis and increased splanchnic blood flow. Progressive collagen deposition and formation of nodules alter the normal vascular architecture of the liver and increase resistance to portal flow. Sinusoids may become less distensible with the formation of collagen within the space of Disse. While this may give the impression of a static portal system, recent studies have suggested that activated hepatic stellate cells may dynamically regulate sinusoidal tone and thus portal pressure.

Sinusoidal endothelial cells form an extremely porous membrane which is almost completely permeable to macromolecules, including plasma proteins. In contrast, splanchnic capillaries have a pore size 50–100 times less than that of hepatic sinusoids. As a consequence, the trans-sinusoidal oncotic pressure gradient in the liver is virtually zero while it is 0.8–0.9 (80%–90% of maximum) in the splanchnic circulation [6]. Oncotic pressure gradients at such extreme ends of the spectrum minimise any effect the changes in plasma albumin concentration may have on transmicrovascular fluid exchange. Therefore, the old concept that ascites is formed secondary to decreased oncotic pressure is false, and plasma albumin concentrations have little influence on the rate of ascites formation. Portal hypertension is critical to the development of ascites, and ascites rarely develops in patients with a wedged hepatic venous portal gradient of <12 mm Hg-9 Conversely, insertion of a side to side porta-caval shunt to decrease portal pressure often causes resolution of ascites.

1.3 Pathophysiology of sodium and water retention

The classical explanations of sodium and water retention occurring due to “underfill” or “overfill” are oversimplified. Patients may exhibit features of either “underfill” or “overfill” depending on posture or severity of liver disease. One of the key events thought to be critical in the pathogenesis of renal dysfunction and sodium retention in cirrhosis is the development of systemic vasodilatation, which causes a decrease in effective arterial blood volume and a hyperdynamic circulation [10]. The mechanism responsible for these changes in vascular function is unknown but may involve increased vascular synthesis of nitric oxide, prostacyclin, as well as changes in plasma concentrations of glucagon, substance P, or calcitonin gene related peptide [8].

However, the haemodynamic changes vary with posture, and studies by Bernardi *et al* have shown marked changes in secretion of atrial natriuretic peptide with posture, as well as changes in systemic haemodynamics [11, 12]. In addition, data showing a decreased effective arterial volume in cirrhosis have been disputed [13]. It is agreed however that under supine conditions and in experimental animals, there is an increase in cardiac output and vasodilatation.

The development of renal vasoconstriction in cirrhosis is partly a homeostatic response involving increased renal sympathetic activity and activation of the renin-angiotensin system to maintain blood pressure during systemic vasodilatation [14]. Decreased renal blood flow decreases glomerular filtration rate and thus the delivery and fractional excretion of sodium. Cirrhosis is associated with enhanced reabsorption of sodium both at the proximal tubule and at the distal tubule [14]. Increased reabsorption of sodium in the distal tubule is due to increased circulating concentrations of aldosterone. However, some patients with ascites have normal plasma concentrations of aldosterone [15]. leading to the suggestion that sodium reabsorption in the distal tubule may be related to enhanced renal sensitivity to aldosterone or to other undefined mechanisms [16].

In compensated cirrhosis, sodium retention can occur in the absence of vasodilatation and effective hypovolemia. Sinusoidal portal hypertension can reduce renal blood flow even in the absence of haemodynamic changes in the systemic circulation, suggesting the existence of a hepatorenal reflex [17, 18]. Similarly, in addition to systemic vasodilation, the severity of liver disease and portal pressure also contribute to the abnormalities of sodium handling in cirrhosis [19].

2. Materials and Methods

This cross sectional observational study was conducted in a tertiary care hospital of Rohilkhand region located in the Northern India, after getting approval from the ethical committee, over a period of 8 months from January 2016 to August 2016.

All patients who were diagnosed as ascites on the basis of history, physical examination, ultrasonography, and age >18 years admitted in medicine department of Rohilkhand Medical College & hospital (RMCH), Bareilly(U.P)were included in the study after getting the informed consent. Patients who had a secondary cause of peritonitis and unwilling to participate in the study were excluded. The patients included in the study were evaluated by detailed history and physical examination. Ascitic fluid paracentesis and virologic testing for Hepatitis B and C were done in all the patients. Ascitic fluid was analyzed for cytology, biochemistry, gram staining, acid fast bacillus staining, malignant cells, culture, and sensitivity and adenosine deaminase (ADA). Serum-ascites albumin gradient (SAAG) was estimated in all patients. Ultrasound abdomen was done in all patients followed by computed tomography if the

ultrasound was inconclusive or there was evidence of any intra-abdominal malignancy. Specific etiology oriented investigations were carried out. Other investigations such as antinuclear antibodies, an antibody against liver-kidney-microsomes, anti-smooth muscle antibodies, Serum ceruloplasmin, urinary copper levels and slit lamp examination for Kayser–Flesher ring were done if indicated. Cirrhosis was diagnosed on basis of physical examination and ultrasonography. Tubercular ascites was diagnosed on the basis of lymphocytic predominance on cytology, low SAAG (<1.1), high protein (>2.5), ADA more than 40 IU/L. The data, thus collected, was analyzed on Microsoft Excel sheet 2010 and percentages were calculated.

3. Results

Fifty one patients with ascites were included in the study. The age group of study population ranged from 22 years to 70 years and the mean age was 45.14 years. Twenty (39%) were females and thirty one (61%) were males. The male to female ratio was 1.55:1. Majority of patients, 28 (56%), were in the age group of 31–50 years. Chronic liver disease was the found in 31 patients. Among 31 patients with chronic liver disease, 13 patients had cirrhosis due to hepatitis B virus infection, 9 patients due to hepatitis C, whereas 9 patients were chronic alcoholic. Tuberculosis was the cause of ascites in 8 patients, whereas pancreatitis was the cause of ascites in 4 patients. Intra-abdominal malignancy and constrictive pericarditis were diagnosed in 3 patients each, whereas 2 patients had nephrotic syndrome. Among patients with cirrhosis, 15 patients were in Child Turcotte Pugh (CTP) class A, 6 were in CTP class B, while 10 were in CTP class C. 2 patients had evidence of spontaneous bacterial peritonitis, both of them were culture negative.

Gender	Total no of patients
Male	31
Female	20
Total patients	51

Age group	Total no of patients
21-30 years	10
31-40years	9
41-50years	20
51-60years	6
>60years	6
Total	51

Cause of ascites	Total no of patients
Chronic liver disease	31
Tuberculosis	8
Pancreatitis	4
Malignancy	3
Constrictive pericarditis	3
Nephrotic syndrome	2
Total	51

Causes of cirrhosis of liver	Total no of patients
Hepatitis b virus	13
Hepatitis c	9
Chronic alcoholism	9
Total	31

4. Discussion and Conclusion

Present study was conducted at a tertiary care hospital located in Rohilkhand region among patients of ascites who were admitted in the medicine department over a period of 8 months. Cirrhosis was found to be the most common cause of ascites (60.78% patients), followed by tuberculosis (15.68% patients). Although cirrhosis was the leading cause of ascites, causes other than cirrhosis were present in around 37% of the patients. Similar results have been found in the studies conducted by Kumar B *et al* [20], Adhikari P *et al* [21], and Khan FY *et al* [2].

Among cirrhotics Hepatitis b was the leading cause of cirrhosis (41.93% patients). India accounts for a large proportion of the worldwide HBV burden. India harbors 10–15% of the entire pool of HBV carriers of the world. It has been estimated that India has around 40 million HBV carriers. About 15-25% of HBsAg carriers are likely to suffer from cirrhosis and liver cancer. While horizontal transmission in childhood appears to be a major route of transmission, the role of vertical transmission is probably underestimated. Blood transfusion and unsafe therapeutic injections continue to be important modes of transmission of HBV [22] There should be a focus on screening for hepatitis b in high risk individuals including, intravenous drug users, persons who receive blood transfusions, acupuncture, tattooing, unsafe injection practices, health care workers at risk of occupational exposure, etc. Identification and if necessary treatment of the HBV infected persons would play a role in decreasing the spread of the disease. Focus on primary prevention by hepatitis B vaccination is recommended [8] The advantages of this study are that the causes of ascites in Rohilkhand region were known which can help us in directing treatment decision, predicting the outcome and in formulating the future preventive strategy.

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