Mini Review
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New–onset ascites: a simplified diagnostic algorithm

Abstract

Normally, around 2 mL of fluid is present in the peritoneal cavity. Ascites is defined as a pathological (excess) fluid accumulation within the peritoneal cavity.[1] The commonest cause of new–onset ascites is liver cirrhosis (cirrhotic ascites) which is responsible for 85% of cases.[2] The remaining 15% of cases occur due to causes other than cirrhosis (non–cirrhotic ascites). While malignancy, heart failure, and renal causes predominate in Western countries, malignancy and tuberculosis predominate in developing countries.[2,3] Proper management of patients with new–onset ascites depends primarily on determining its cause which relies principally on proper analysis of ascitic fluid.[4] To simplify the approach to diagnosis of ascites etiology, this review suggests a two–step approach:

Step [I]: Serum ascites albumin gradient (SAAG) plus total protein concentration.

Step [II]: Choice of further tests will depend on the results of step [I] including, but are not limited to, cell count with differential, cholesterol, cytology, adenosine deaminase (ADA), and triglyceride.

Keywords: Ascites, cirrhosis, non–cirrhotic ascites, abdominal paracentesis, ascitic fluid analysis, etiology, diagnostic algorithm.

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Introduction

Normally, up to 30 mL of peritoneal fluid can be physiological, especially in menstruating females.[5] Transabdominal ultrasound can detect as low as 10 mL of fluid but shifting dullness requires about 1500 mL of fluid.[6] The volume of ascites is graded clinically into mild (only detectable on ultrasound), moderate (causing symmetrical abdominal distension), and large (causing marked abdominal distension).[7,8] Ascites can be divided into three etiological categories: Portal hypertensive (cirrhotic) ascites, non–portal hypertensive (non–cirrhotic) ascites and mixed ascites.[9] Mixed ascites refers to the presence of more than one cause, e.g., cirrhosis plus malignancy. The mandatory step to properly manage patients with new–onset ascites is to find the etiology. To do this, abdominal paracentesis is a rapid way to get fluid samples that can be submitted for appropriate ascitic fluid analysis.[6] Diagnostic abdominal paracentesis is a simple bedside or even Clinic procedure in which a needle is inserted into the peritoneal cavity to remove a small quantity of fluid for testing.[10] The procedure is considered relatively safe and serious complications are uncommon, but ascitic fluid leak is the most common complication following paracentesis occurring in about 5% of cases.[11] Bedside ultrasound is recommended to identify an area with sufficient fluid for aspiration. This helps decrease the incidence of unsuccessful aspiration and complications.[12] Sending at least 80 mL of ascites significantly increases the diagnostic yield of cytology, as does use of sterile blood culture bottles if infection is suspected.[6,13]

The role of SAAG in etiological diagnosis of ascites

Serum–ascites albumin gradient (SAAG) was first recommended by Hoefs et al. in 1981, and it is calculated by subtracting ascitic albumin concentration from serum albumin concentration.[14] If the SAAG is 1.1 g/dL or more, the patient has portal hypertensive ascites, with about 97 % accuracy.[15] Apart from cirrhotic ascites, other causes of high SAAG (≥1.1 g/dL) ascites include cardiac ascites as well as mixed ascites while causes of a low SAAG (<1.1 g/dL) include malignant ascites, tuberculous peritonitis, nephrotic syndrome, pancreatic ascites, and eosinophilic gastroenteritis. Therefore, SAAG is recommended as an initial ascitic fluid analysis according to clinical practice guidelines.[6–8,16–18]

The role of total protein concentration in etiological diagnosis of ascites

The best value that can be gained from testing ascitic protein is through its use in conjunction with SAAG. A high SAAG ascites (≥ 1.1 g/dL) indicates that the etiology is mostly portal hypertension. If this is associated with a low ascitic protein (<2.5 g/dL), it indicates liver cirrhosis, and cell count with differential would be an appropriate next test to differentiate sterile vs. infected cirrhotic ascites. If high SAAG ascites is associated with elevated ascitic protein, this is consistent with cardiac ascites, and echocardiogram would be helpful. A low SAAG ascites (<1.1 g/dL) indicates that the etiology is not portal hypertension. If this is associated with a low ascitic protein (<2.5 g/dL), it indicates a low protein overall (severe malnutrition or protein–losing disorder such as nephrotic syndrome), and 24–hour urine protein would be an appropriate next test. If low SAAG ascites is associated with elevated ascitic protein, think of potential ovarian malignancy and pelvic ultrasound would be indicated. Also, in low SAAG ascites associated with elevated ascitic protein, think of peritoneal tuberculosis, and ascitic adenosine deaminase would be justified.[19,20]

The simplified diagnostic algorithm

Based on these data, a simplified diagnostic algorithm is suggested (Figure 1). This figure shows that starting with SAAG, ascites can be divided into high vs. low SAAG. Each of these groups can be further divided into high vs. low ascitic protein. The next test will depend on the results of these two initial tests.

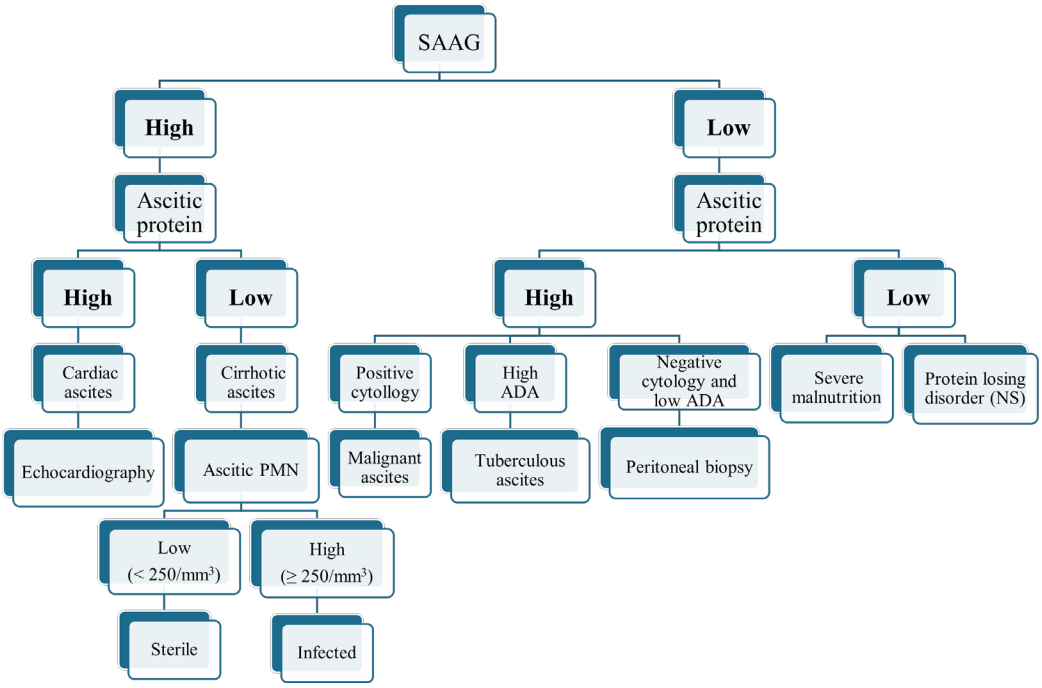

Figure 1 Simplified diagnostic algorithm for new-onset ascites.

Notes: SAAG, Serum ascitic albumin gradient; NS, Nephrotic syndrome; PMN, polymorphonuclear leucocytes; ADA, ascitic fluid adenosine deaminase.

Conclusion

This review suggests using this simplified diagnostic algorithm in cases presenting with new–onset ascites. Further studies implementing this algorithm are required to confirm its clinical utility.

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