Journal of Applied Medical Sciences, Vol. 14, No. 2, 2025, 1-7
ISSN: 2241-2328 (print version), 2241-2336 (online)
https://doi.org/10.47260/jams/1421
Scientific Press International Limited

Strange SAAG: Inappropriately Low Serum-Ascites Albumin Gradient in a 60-year-old Male with Heart Failure and Cirrhosis

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Abstract

The serum-ascites albumin gradient (SAAG) is a proven diagnostic tool for categorizing ascitic fluid, especially portal hypertension. Cases of low SAAG in the setting of portal hypertension are extremely rare. We present a case of a 60-year-old male with heart failure and liver cirrhosis who had a recurrently low SAAG despite having portal hypertension. Comprehensive workup for ascites should be performed when encountering ascites of unclear origin. Additionally, clinicians should consider other clinical clues beyond the SAAG when the diagnosis is unclear to reach the most plausible diagnosis.

JEL classification numbers: I10, Y5.
Keywords: Ascites, Cirrhosis, Heart failure, Portal hypertension, Albumin.

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Article Info: *Received:* May 13, 2025. *Revised*: May 28, 2025.
Published online: May 28, 2025.

1. Introduction

Ascites occurs when fluid builds up in the abdominal or peritoneal cavity. There are a multitude of etiologies of ascites and management depends on the underlying etiology. The serum ascites albumin gradient (SAAG) is a proven diagnostic tool for categorizing ascitic fluid. A SAAG greater than 1.1 g/dL indicates that ascitic fluid is from portal hypertension and a SAAG less than 1.1 g/dL indicates that ascitic fluid is from renal, infectious, or malignant etiologies. The accuracy of SAAG in diagnosing portal hypertension is extremely high at 97%, however, cases of low SAAG with portal hypertension have rarely been documented (Runyon, 2009). We present a case of a 60-year-old male with heart failure and liver cirrhosis who had a recurrently low SAAG despite having portal hypertension.

2. Case Presentation

A 60-year-old male with a past medical history of cirrhosis secondary to untreated hepatitis C, non-ischemic cardiomyopathy, and heart failure with reduced ejection fraction (10%) presented to the hospital for worsening abdominal pain and distention. He had multiple recent admissions for similar chief complaints. Upon initial presentation, he required four liters of oxygen via nasal cannula to maintain his oxygen saturations within normal limits, but he was otherwise hemodynamically stable. Physical examination revealed abdominal distention, fluid wave, and trace lower extremity pitting edema. Laboratory evaluation was significant for urea nitrogen of 23 mg/dL, creatinine of 1.61 mg/dL, aspartate aminotransferase of 58 IU/L, alanine aminotransferase of 26 IU/L, total bilirubin of 2.7 mg/dL and an N-terminal pro-B-type natriuretic peptide (BNP) of 12,211 pg/mL (Table 1).

Table 1: Pertinent serum laboratory evaluations with associated reference ranges

Pertinent serum laboratory evaluations	Patient values	Reference ranges
Urea nitrogen	23 mg/dL	6 - 22 mg/dL
Creatinine	1.61 mg/dL	0.51 - 0.96 mg/dL
Aspartate aminotransferase	58 IU/L	14 - 33 IU/L
Alanine aminotransferase	26 IU/L	10 - 42 IU/L
Total bilirubin	2.7 mg/dL	0.2 - 1.0 mg/dL
N-terminal pro-B-type natriuretic peptide	12,211 pg/mL	<125 pg/mL

A computed tomography chest with pulmonary angiography was done and showed no pulmonary embolism, but did demonstrate severe cardiomegaly with four-chamber dilation (Figure 1).

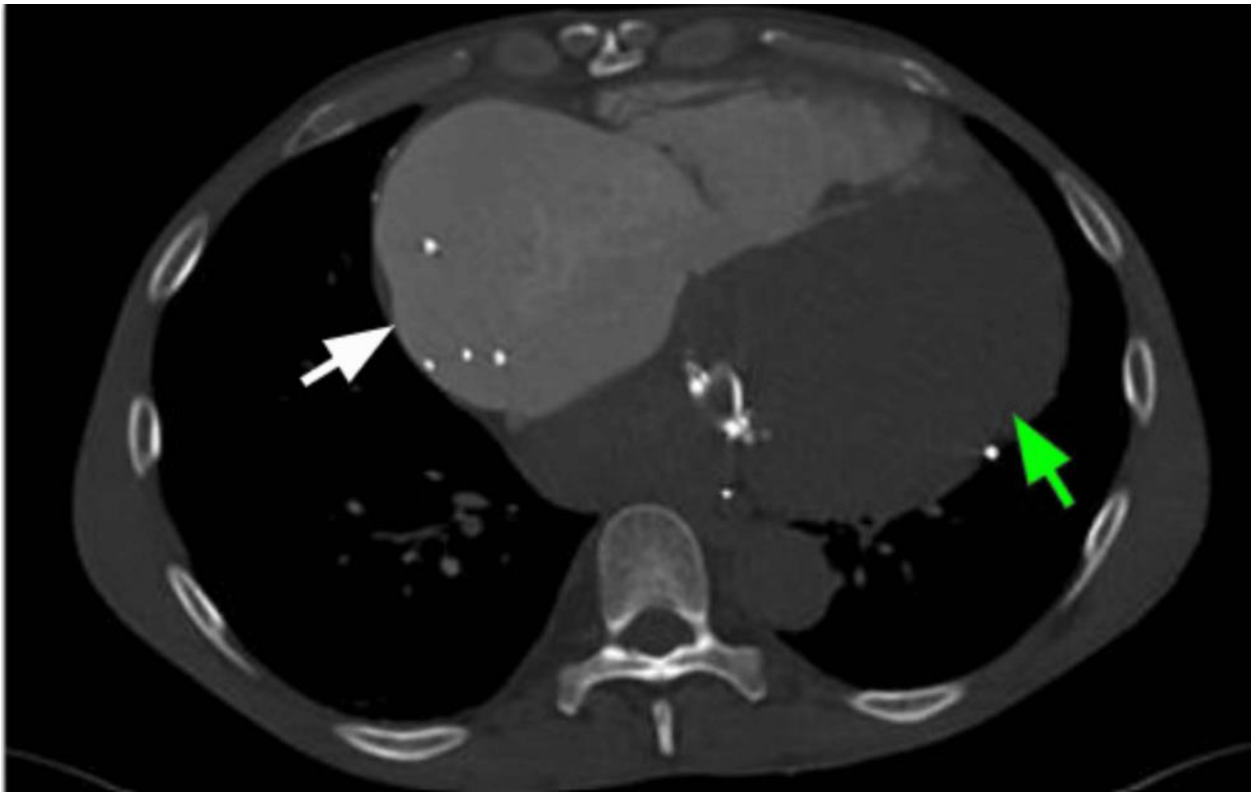

Figure 1: Computed Tomography Chest with Pulmonary Angiography demonstrating severe cardiomegaly, especially in the right atrium (white arrow) and left ventricle (green arrow)

A complete abdominal ultrasound showed pulsatility of portal venous flow with patent hepatic vasculature and no thrombus (Figure 2).

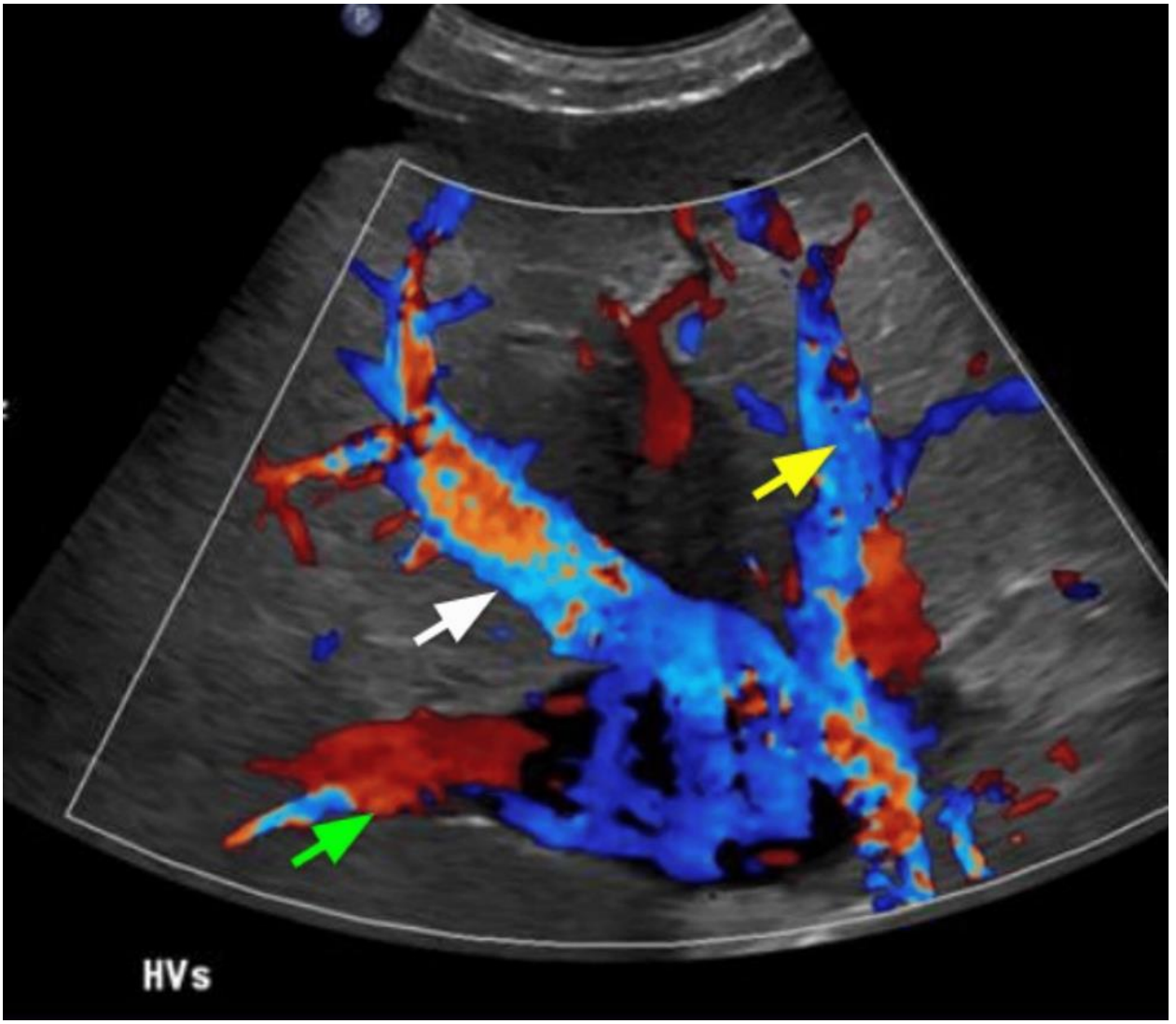

Figure 2: Abdominal Ultrasound with Doppler showing Middle Hepatic Vein (white arrow), Right Hepatic Vein (green arrow), and Left Hepatic Vein (yellow arrow) dilation with appropriate flow direction

A diagnostic and therapeutic paracentesis yielded 7 liters of brown-yellow fluid (Figure 3).

Figure 3: Evacuated Suction Bottles (1 Liter each) filled with ascitic fluid

Ascitic fluid analysis revealed albumin of 2.9 g/dL, protein of 3.5 g/dL, negative gram stain, negative adenosine deaminase, and normal levels of amylase, lactate dehydrogenase, triglycerides, lymphocytes, and neutrophils. Calculated with a serum albumin of 3.7 g/dL, his SAAG was 0.8 (Table 2).

Table 2: Serum-ascites albumin gradient values

Serum-ascites albumin gradient values	Patient values	Reference ranges
Serum albumin	3.7 g/dL	3.5 - 4.8 g/dL
Ascitic albumin	2.9 g/dL	0.31 - 1.77 g/dL
Ascitic protein	3.5 g/dL	None provided

Further workup for the etiology of his ascites included a urinalysis with trace proteinuria, a urine protein-creatinine ratio of 0.17, and an alpha-fetoprotein of 5.6 ng/mL. It was deemed that his ascites most likely was from a cardiac etiology, so he was started on intravenous diuresis, weaned to room air, and discharged in a stable condition with optimizations of his home medications.

3. Discussion

Heart failure leads to ascites and a high SAAG through increased central venous pressure, causing hepatic sinusoid congestion, portal hypertension, and subsequent fluid extravasation into the peritoneal space (Hernaez and Hamilton, 2016). This contributes to a high SAAG because the albumin is retained in the blood (due to its large molecular size and oncotic pressure) while fluid is pushed out (Hernaez and Hamilton, 2016).

Patients with portal hypertension have a low SAAG about 3.3% of the time however this can be in the presence of comorbid conditions such as nephrotic syndromes, malignancy, infections, or poor nutritional status (Arques and Ambrosi, 2011). When encountered, repeat paracentesis is often performed and yields high SAAG values, indicating that the initial paracentesis results were erroneous. In our patient, multiple paracentesis performed several months apart yielded similar results. Comprehensive testing for nephrotic syndrome (urinalysis and protein-creatinine ratio), pancreatitis (amylase), chylous (triglycerides), infections (ascitic fluid cell counts and cultures), malignancies (ascitic fluid cytology, abdominal imaging, serum AFP), thrombus (ruled out via doppler ultrasound) and malnutrition (serum albumin and body mass index) yielded normal results.

We propose that this patient had a low SAAG due to the severity of his heart failure. This is evidenced by his Doppler ultrasound showing pulsatility of portal venous flow (indicative of right heart failure), BNP, high ascitic protein levels, and absence of other etiologies of ascites (Elijaiek et al. 2019). The mechanism for this could be related to increased intravascular pressure ultimately causing albumin to leak into

the ascitic fluid, leading to a lower SAAG and higher ascitic protein level.
In one study, researchers identified that a BNP greater than 364 ng/L with an ascitic fluid total protein concentration greater than 2.5 g/dL suggests an underlying cardiac disease (Trongtorsak et al. 2022). Although this patient's SAAG was low, his ascitic protein level was consistent with cardiac ascites and his BNP was greater than the proposed cutoff. This could indicate the utility of using BNP when characterizing ascites of unclear origin.

4. Conclusion

SAAG remains a cost-effective and reliable tool for characterizing ascites, however, it can rarely be inconclusive. If ascites of unclear origin is encountered, a comprehensive workup for the etiology of ascites should be performed to properly categorize and manage the underlying condition. Workups including ascitic fluid testing and radiographic testing with Doppler ultrasound are low-risk methods to evaluate both common and uncommon causes of ascites. SAAG in addition to BNP may be useful to aid in the diagnosis of cardiac ascites. More studies on cardiac ascites' effects on SAAG need to be performed.

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