Bangladesh Crit Care J September 2022; 10 (2): 149-153

Case Report

Cardiac ascites presenting as abdominal compartment syndrome – A case report

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DOI: https://doi.org/10.3329/bccj.v10i2.62210

ABSTRACT:

Ascites is an infrequent presentation of poorly controlled congestive cardiac failure. Tense ascites due to heart failure is more infrequent. Whenever there is tense ascites, intra-abdominal hypertension should always be considered. The most deleterious effect of intra-abdominal hypertension is abdominal compartment syndrome. It is a condition that is frequently overlooked and underdiagnosed. Abdominal compartment syndrome is a medical as well as surgical emergency which should be promptly diagnosed and adequately managed; otherwise, it may lead to multiple organ failure and death. Here we present a patient with congestive cardiac failure leading to huge ascites, intra-abdominal hypertension and abdominal compartment syndrome.

Keywords: *Congestive cardiac failure, Cardiac ascites, intra-abdominal hypertension, abdominal compartment syndrome.*

Introduction:

Congestive cardiac failure represents 5% of all causes of ascites.[1] Huge ascites leads to raised intra-abdominal pressure which in turn may lead to abdominal compartment syndrome (ACS). According to recent literature, the frequency of abdominal compartment syndrome in trauma ICU admissions is anywhere from 5-15% and much less in medically ill non trauma patients.[1] As it is an uncommon condition and presentation is variable, it high index of suspicion is necessary for early diagnosis. Intra-abdominal hypertension causing ACS can impair the function of almost all the organs of the body. When left untreated, it may lead to multiple organ failure and death.

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Case presentation:

A 40 years gentleman got admitted to a tertiary care hospital, intensive care unit (ICU), through emergency room (ER) with the complaints of progressively increasing shortness of breath for 7 days, gradually increasing abdominal distention and progressive decrease of urine output for 5 days with complete cessation of urine for 2 days. His previous medical conditions include history of recurrent heart failure, severe pulmonary hypertension, and severe tricuspid regurgitation. He had a metallic mitral valve replacement with tricuspid valve annuloplasty 8 years back. On admission, patient was fully conscious, oriented and severely dyspnoeic requiring high flow oxygen. He also had tachycardia and blood pressure was 90/60 mm of Hg. He had bilateral pitting pedal oedema without any rash. Abdomen was hugely distended with bulging flanks. Liver and spleen were not palpable. Heart sounds were distant, with a laterally displaced point of maximum impulse. Lung volumes were low with no crackles or wheezing. No tremor or neurological deficit was observed. Patient had no urine output even after urinary catheterization.

Remarkable laboratory data are shown in table 1.

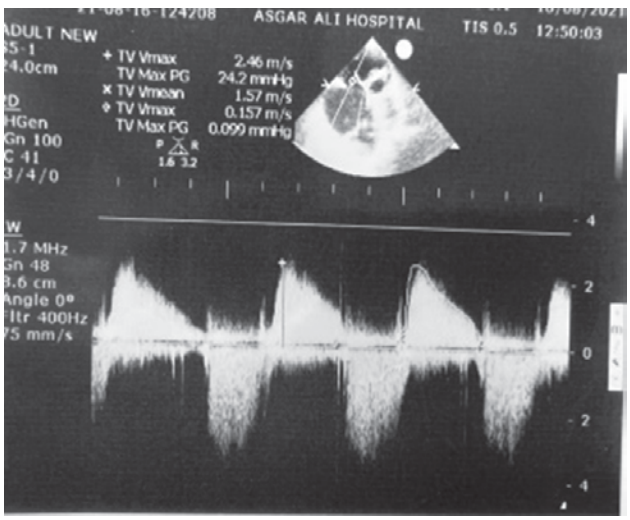
Table 1: Remarkable lab parameters

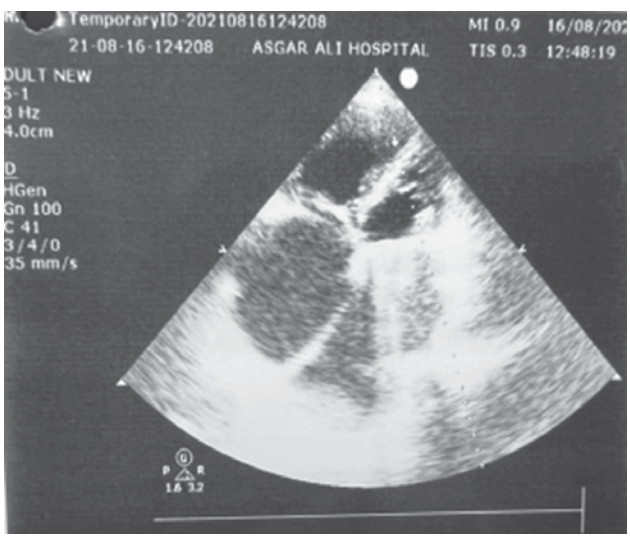
Variable	On admission	After 1st Paracentesis	After 2nd Paracentesis	Day 6	Day 7
S.Creatinine (mg/dl)	4.6	3.8	3.0	1.6	1.2
S.Urea (mg/dl)	111	101	86	44	30
HCO_3 (mmol/L)	18	21	24	28	30

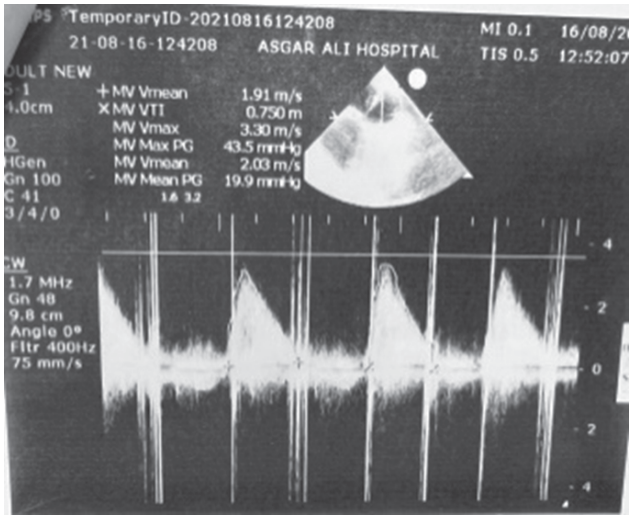
Received : June 19, 2022; Accepted : August 10, 2022

S. Albumin (gm/dL)	3.5
Troponin I	0.02
ALT	158
S. Bilirubin	0.9

His transthoracic echocardiogram showed normally functioning prosthetic mitral valve in situ with no transvalvular and paravalvular leakage, significant tricuspid stenosis with severe tricuspid regurgitation, severe pulmonary hypertension (PASP 90 mm of Hg), dilated right atrium and right ventricle. There was no regional wall motion abnormality at rest with normal left ventricular systolic function (EF 65%). Inferior vena cava was found dilated and non-collapsing (Fig 1).







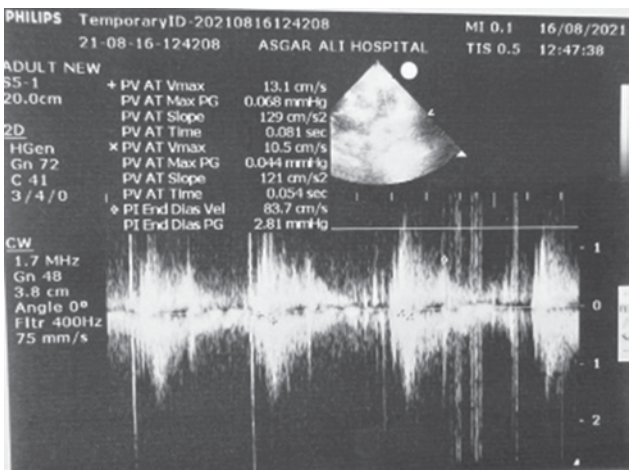
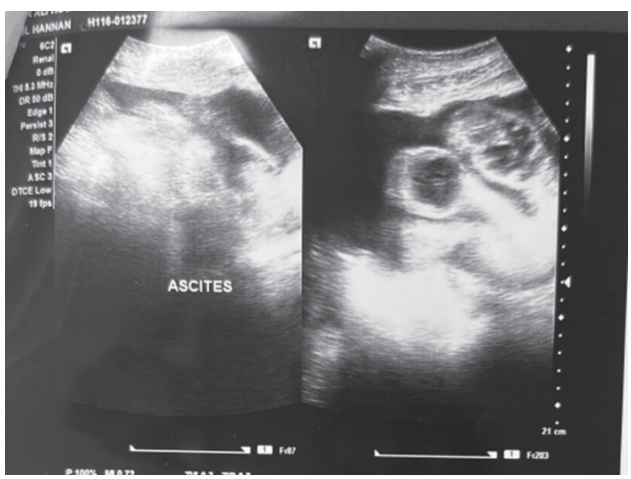
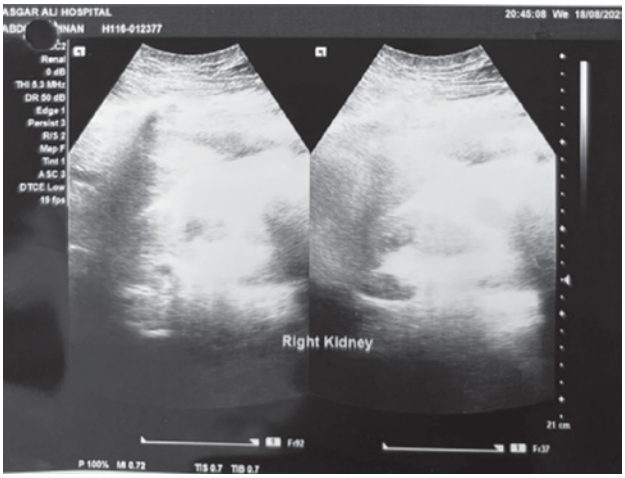


Fig 1: Transthoracic Echocardiogram

Ultrasonogram examination of whole abdomen showed huge ascites with evidence of acute kidney injury (Fig 2).





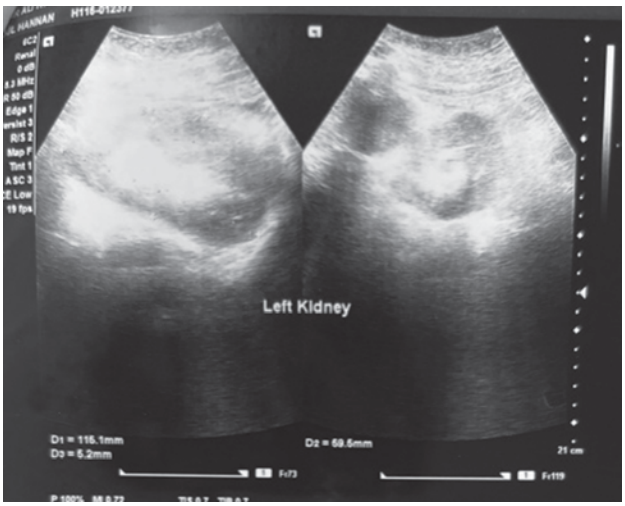


Fig 2: Ultrasound images showing huge ascites with features of AKI

Doppler study of upper abdomen showed no evidence of thrombus in portal or hepatic veins.

Treatment was started immediately with high flow nasal oxygen. Patient's condition gradually deteriorated with fall of blood pressure requiring pressor support and increasing respiratory distress requiring non-invasive ventilation. Intra-abdominal pressure was measured by measuring bladder pressure which showed >25 mm of Hg. Patient was labelled as having grade IV intra-abdominal hypertension with abdominal compartment syndrome. Measures were taken for immediate paracentesis. Approximately 4L of straw colored fluid was tapped from peritoneal cavity under ultrasound guidance in two settings with intravenous albumin coverage. After the first paracentesis, intra-abdominal pressure decreased to 20 mm of Hg with immediate improvement of hemodynamic parameters, decrease of shortness of breath and oxygen requirement. Urine output was hourly 10-20 ml. After the second session of paracentesis, patient was completely weaned from pressor support, respiratory parameters improved significantly with minimum oxygen requirement. Urine output increased to 80 – 100 ml per hour. Ascitic fluid study came in favor of cardiac ascites with no feature of infection or malignancy.

Within a few days after the paracentesis, patient's renal function was restored to normal. Urine output was adequate. He was hemodynamically stable and had no respiratory distress or oxygen requirement. On day 7 of admission he was discharged in a stable condition.

Discussion:

Cardiac ascites have an overall incidence of around 5% of all causes of ascites. Chemical and cytological study of the ascitic fluid supported the diagnosis in our patient. When massive ascites develop in a relatively short period of time, intra-abdominal hypertension may develop.

Intra-abdominal hypertension (IAH) is defined by a sustained or repeated pathological elevation in intra-abdominal pressure (IAP) ≥ 12 mmHg.[2] IAP is the steady-state pressure concealed within the abdominal cavity. IAH is graded as follows, Grade I: IAP 12–15 mmHg, Grade II: IAP 16–20 mmHg, Grade III: IAP 21–25 mmHg and Grade IV: IAP > 25 mmHg.

ACS is defined as a sustained IAP > 20 mmHg,[2] that is associated with new organ dysfunction/failure. In our patient this new organ dysfunction was acute kidney injury.

Some of the important risk factors for intra-abdominal hypertension and abdominal compartment syndrome are outlined in table 2.

Table 2: Risk factors for intra-abdominal hypertension& ACS[2]

Risk factors for intra-abdominal hypertension and ACS

1. Diminished abdominal wall compliance
   a. Abdominal surgery
   b. Major trauma
   c. Major burn
   d. Prone positioning
2. Increased intra-luminal contents
   a. Gastroparesis/gastric distention/ileus
   b. Colonic pseudo-obstruction
3. Increased intra-abdominal contents
   a. Acute pancreatitis
   b. Hemoperitoneum/pneumoperitoneum or intra-peritoneal fluid
   c. collections
   d. Intra-abdominal infection/abscess
   e. Intra-abdominal or retroperitoneal tumors
   f. Peritoneal dialysis
4. Capillary leak/fluid resuscitation
   a. Massive fluid resuscitation or positive fluid balance
   b. Polytransfusion
5. Others
   a. Sepsis
   b. Mechanical ventilation with PEEP>10

c. Obesity or increased body mass index
d. Old age

Intra-abdominal hypertension (IAH) can impair the function of nearly every organ system, thereby causing ACS (Table 3).

Table 3: Complications of IAH and ACS

Organ system	Complications
Cardiovascular	• Impaired cardiac function • Reduced venous return[3]
Pulmonary	Mechanically ventilated patients with IAH have increased peak inspiratory and mean airway pressures, which can cause alveolar barotrauma[3]
Renal	• Renal vein and artery compression • Progressive reduction in both glomerular perfusion and urine output[3]
Gastrointestinal	• Decreased mesenteric blood flow[4] • Hypoperfusion, bowel ischemia, decreased intramucosal pH, and lactic acidosis
Hepatic	Liver's ability to remove lactic acid is impaired by increased intra-abdominal pressure[5]
Central nervous system	Elevated intracranial pressure[6]

Measurement of intra-abdominal pressure: Intra-abdominal pressure can be measured indirectly using intragastric, intracolonic, intravesical (bladder), or inferior vena cava catheters.[7] Measurement of bladder (ie, intravesical) pressure is the standard method to screen for intra-abdominal hypertension (IAH) and ACS. It is simple, minimally invasive, and accurate. It can be performed with supplies routinely available in the intensive care unit by the following steps:

- The drainage tube of the patient's Foley (bladder) catheter is clamped.
- Sterile saline (up to 25 mL) is instilled into the bladder via the aspiration port of the Foley catheter and the catheter is filled with fluid.
- An 18 gauge needle attached to a pressure transducer is inserted into the aspiration port. With some newer-style Foley catheters, this can be done using a needle-less connection system.
- The pressure is measured at end-expiration in the supine position after ensuring that abdominal muscle contractions are absent. The transducer should be zeroed at the level of the mid-axillary line.

Management: Management of intra-abdominal hypertension and abdominal compartment syndrome can be divided into two steps:

1. Grade I and Grade II intra-abdominal hypertension: Medical management (Table 4)
2. Grade III Grade IV intra-abdominal hypertension with ACS: Invasive management (Fig 3)

Table 4: Medical treatment options to reduce intra-abdominal pressure (IAP)[2]

Medical management of grade I and II intra-abdominal hypertension

1. Improve abdominal wall compliance
   a. Sedation and analgesia
   b. Neuromuscular blockade
   c. Avoid head of bed >30°
2. Evacuate intraluminal contents
   a. Nasogastric decompression
   b. Rectal decompression
   c. Gastro/coloprokinetic agent
3. Evacuate abdominal fluid collection
   a. Paracentesis
   b. Percutaneous drainage
4. Correct positive fluid balance
   a. Avoid excessive fluid resuscitation
   b. Diuretics
   c. Colloids
   d. Hemodialysis/ultrafiltration
5. Organ support
   a. Optimize ventilation, alveolar recruitment
   b. Optimize mean arterial pressure

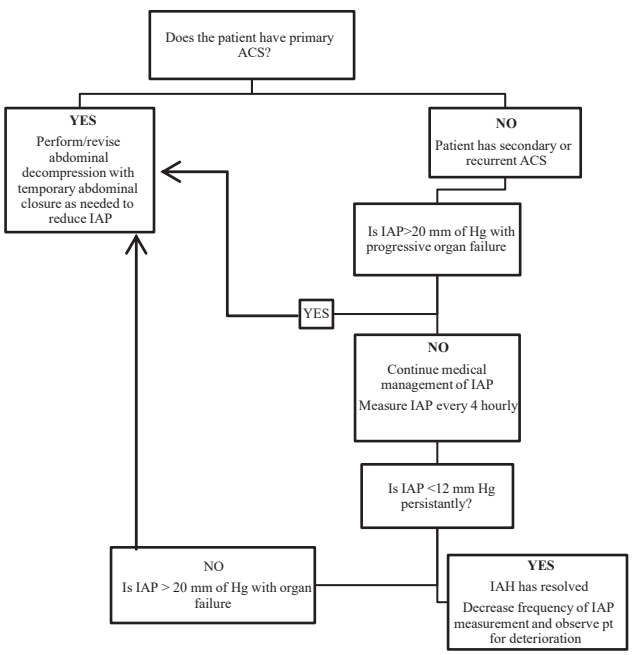


Figure 3: Management algorithm of Grade III & IV IAH with ACS

Conclusion:

Intra-abdominal hypertension and abdominal compartment syndrome is an under recognized condition in critically ill patients. Measurement of intra-abdominal pressure should be a part of routine monitoring in every critically ill patient and for those at high risk of developing the condition. Its low prevalence may be due to missed diagnosis. As ACS is a serious condition with significant mortality and morbidity, high index of suspicion, early diagnosis and prompt management are the key to halt its progression.

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