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A Rare Cause of Ascitis in A Child- A Case Report

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ABSTRACT

Introduction: Pancreatic ascites is a rare complication of acute pancreatitis, particularly in pediatric patients. While chronic pancreatitis is a well-known cause of ascites, acute pancreatitis-induced ascites remains underrecognized. This case report explores a pediatric case of pancreatic ascites secondary to acute pancreatitis, emphasizing the diagnostic and therapeutic challenges.

Case presentation: A seven-year-old male presented with generalized abdominal distension and pain, along with a history of fever, vomiting, and loose stools. Initial investigations suggested eosinophilic ascites, but despite treatment, the patient showed no clinical improvement. Elevated amylase and lipase levels in both serum and ascitic fluid led to a revised diagnosis of acute necrotizing pancreatitis with bacterial peritonitis. Imaging revealed moderate acute interstitial pancreatitis with gross ascites. The patient underwent multiple interventions, including peritoneal catheterization, ERCP (endoscopic retrograde cholangiopancreatography) with stenting, and exploratory laparotomy, ultimately leading to the drainage of a large intraabdominal abscess. The patient recovered with supportive care and was discharged after 1.5 months

Conclusion: Acute pancreatitis-induced pancreatic ascites is rare in children, and its diagnosis requires a high index of suspicion. Imaging studies, elevated amylase, and lipase levels are crucial for identifying the pancreatic origin of ascites. Treatment focuses on supportive care, but severe cases may require invasive interventions such as drainage, ERCP, and surgery. This case underscores the importance of early recognition and intervention to improve outcomes in pediatric patients with pancreatic ascites.

Keywords: *pediatric pancreatitis, pancreatic ascites, acute pancreatitis, eosinophilic ascites, peritoneal drainage, ERCP, case report.*

1. INTRODUCTION

Pancreatic ascites is an intraperitoneal pancreatic fluid collection and mostly results as a complication of chronic pancreatitis. Chronic pancreatitis leading to ascites is a well-studied entity. However, ascites secondary to acute pancreatitis in pediatrics is a lesser-known and studied entity. It is a rare cause of ascites and also an uncommon complication of pancreatitis in children. According to a study, pancreatic ascites accounted for 5% of all ascites cases in children [1]. Patients with severe acute pancreatitis are more likely to develop ascites. Pancreatic ascites and pancreatic pleural effusion are the least common complications of pancreatitis.

Reactionary pancreatic ascites can occur in the first week of acute pancreatitis due to peritoneal inflammation, which increases vascular permeability and leads to exudation of fluid from the intravascular space resulting in a "capillary leak" phenomenon. It is mostly mild to moderate ascites and does not cause symptoms. As the initial inflammatory storm subsides, ascites usually disappear but in some severe cases, it might persist. Diagnostic paracentesis should be avoided in such mild to moderate cases as it is self-limiting and paracentesis can cause intra-abdominal infection in the patient [2].

In the latter phase of acute pancreatitis, ascites is usually caused by pancreatic duct disruption in a setting of necrotizing pancreatitis, where necrosis of the ductal epithelium leads to disintegration of the duct. Limited studies have been conducted on ductal disruption in acute pancreatitis in pediatrics.

Other etiologies of pancreatic ascites are hypoalbuminemia-related pancreatic ascites, portal hypertension-related pancreatic ascites, intraperitoneal pseudocyst rupture, biliary pancreatitis, major pancreatic duct trauma, persistent internal fistula to the peritoneum, ampullary stenosis, cystic ductal duplications, and choledocholithiasis [3][4][5].

The severity of this condition varies widely and depends on the site, severity of ductal injury, and presence of infection. Mild cases resolve spontaneously, while surgical interventions are required for severe cases. Because the condition is rare, the diagnosis is often delayed in a significant number of cases. Thus, a high index of suspicion is required for prompt diagnosis and management.

This manuscript was prepared following the CARE guidelines (https://www.care-statement.org)

2. CASE SUMMARY

A seven-year-old male child presented with complaints of fever, generalized abdominal distension, and abdominal pain for twenty days. The patient was alright twenty days before admission when he developed fever which was of insidious onset, low grade, and intermittent type. The abdominal distension was gradual and progressive which led to difficulty in breathing. The pain in the abdomen was generalized and dull aching associated with loose stools and vomiting which subsided within five days. He was evaluated for the above complaints. Ascitic tap was done and it revealed eosinophilia (40%). His AEC (absolute eosinophil count) was 5572 cells/µL (normal AEC level- 30-350 cells/µL). CECT(contrast-enhanced computed tomography) was suggestive of gross ascites with mild thickening of jejunal loops. Bone marrow showed erythroid hyperplasia with eosinophilia. A colonoscopy with biopsy and upper gastrointestinal tract endoscopy was done, which revealed 8 to 10 eosinophils per high-power field. Thus, a provisional diagnosis of eosinophilic ascites was made and the patient was started on oral steroids, hydroxyurea, albendazole, and ivermectin. Given no clinical response and an increase in abdominal pain, a repeat ascitic tap was planned. Repeat ascitic fluid showed neutrophilic leukocytosis for which antibiotics were given. Serum amylase and lipase levels were elevated. Thus, the diagnosis was revised to eosinophilic ascites with bacterial peritonitis with acute necrotizing pancreatitis. Octreotide infusion was started but there was no response and the patient was referred to our hospital for further management.

The patient presented to us on the 20th day of symptoms. There was no significant past history, and the birth history was uneventful. His weight was 20 kg, and his height was 122 cm. On general examination, vitals were stable, and blood pressure was within the 50th to 90th centiles. Pallor and pedal edema were present. Abdominal examination showed positive fluid thrill and scrotal edema was present. As seen in Table 1, the routine investigations showed neutrophilic leukocytosis, elevated inflammatory markers, serum amylase and lipase levels, and hypoalbuminemia.

Table 1:Laboratory results during the patient's admission.

LAB PARAMETER	VALUE	REFERENCE RANGE
Hemoglobin	10.8 g/dl	11.0-14.0 g/dl
Reticulocyte count	1.4%	0.5-2.5 %
Total Leukocyte count	27,860	5000-12000/µL
Absolute Neutrophil count	13,930	1500-8500/ µL
Absolute Lymphocyte count	7,243	1500-7000/ µL
Absolute Eosinophil count	5,572	0-650/ µL
Platelets	7,68,000	150000-410000/ µL
Total Bilirubin	0.60	0.22-1.20 mg/dL

Direct Bilirubin	0.40	Upto 0.5 mg/dL
Indirect Bilirubin	0.20	0.1-1.0 mg/dL
Aspartate aminotransferase	32	8-60 U/Lt
Alanine aminotransferase	20	7-55 U/Lt
Alkaline phosphatase	75	142-335 U/Lt
Total proteins	4.40	6.7-8.6 g/dL
Albumin	2.60	3.5-5.5 g/dL
Globulin	1.80	2-3.5 g/dL
Amylase	423	25-115 U/L
Lipase	1462	8-78 U/L
Prothrombin time	14.40	10.83- 13.17 secs
International normalised ratio	1.22	0.85-1.15
Activated partial thromboplastin time	31.10	21.75-28.70 secs
CRP	140	<6 mg/dL
ESR	24mm	<10mm
Procalcitonin	0.19	<0.08 ng/mL
Dengue NS1 antigen Ig M and Ig G	Negative	Negative
Rapid Malarial Test	Negative	Negative
Widal test	Negative	Negative
Lactate dehydrogenase	171	160-370 U/lT
Iron	10	50-150 µg/dL
Ferritin	227	21.81-274.66 ng/mL
Total iron binding capacity	180	250-450 µg/dL
Transferrin saturation	5.5%	20-50 %
Uric acid	4.50	2.4-5.4 mg/dL
Urea	16	17-49 mg/dL
Creatinine	0.40	0.19-0.49 mg/dL
Sodium	137	138-145 mmol/Lt
Potassium	3.7	3.5-5.1 mmol/Lt
Chloride	103	98-107 mmol/Lt
ANA blot	Negative	Negative
Serum IgE	275 KU/L	<400 KU/L
HIV/HbsAg	Non-reactive	Non-reactive

Gastric aspirate for acid fast bacilli	Negative	Negative
Gastric aspirate for CBNAAT	Not detected	Not detected.
2D echocardiography	Normal heart study	

(HIV- Human immunodeficiency virus; HbsAg- Hepatitis B surface antigen; CBNAAT- Cartridge-based nucleic acid amplification test)

Amylase and Lipase of ascitic fluid were grossly elevated as seen in Table 2.

Table 2: Ascitic fluid analysis

Ascitic fluid analysis	**On the day of admission**	**After one week of admission**
Appearance	Slightly turbid	Yellow
Coagulum	Absent	Absent
Deposit	Absent	Absent
Total leukocyte count	2,460 cells	2,000
Neutrophils	60%	80%
Lymphocytes	35%	10%
Macrophages	5%	10%
Proteins	2.90	3
Glucose	28 mg/dl	<5 mg/dl
Amylase	49,778	42,891
Lipase	67,320	13,579
Fluid for malignant cells	Negative	
Fluid for CBNAAT	Not detected	
Fluid culture	No growth	

Repeat CECT abdomen showed acute interstitial pancreatitis (of moderate severity). The radiological investigations have been tabulated as in Table 3.

Table 3: Radiological investigations of the patient.

Radiological investigations	**Impression**
Ultrasonography of abdomen	Visualised head of pancreas appears heterogenous in echotexture with peripancreatic fat stranding. Gross ascites with thick coarse septations.

Computed tonography of abdomen	Head and uncinate process of pancreas show ill-defined areas of heterogenous attenuation and hypoenhancement with marked adjacent peripancreatic fat stranding which is also seen along body of pancreas and extending along adjacent small bowel mesentery along with gross ascites and generalized anasarca as described. - Imaging findings are suggestive of acute interstitial pancreatitis. Modified CT severity index: 6/10 - Moderate severity.
Ultrasonography of scrotum	Minimal left sided hydrocele.

Antibiotics were upgraded, diuretics were started and octreotide infusion was stopped. Since the patient had high-grade fever spikes with tense ascites, and tenderness, a peritoneal pigtail catheterization was done. There was no clinical improvement following pigtail catheter placement, with a drain output of 400 to 500 milliliters per day. Thus, ERCP (Endoscopic retrograde cholangiopancreatography) with pancreatic stenting was planned. The patient was also started on pancreatic enzyme supplements. Despite, undergoing ERCP with stenting, the patient had fever spikes and abdominal tenderness, and an ultrasound of the abdomen suggested moderate ascites with septations. At this point exploratory laparotomy with peritoneal lavage was considered. On exploratory laparotomy, a large thick-walled, solitary intraabdominal abscess was detected extending from Morrison's pouch to the pelvis (of about 100 ml volume) with dense adhesions and inter-bowel loop adhesions. Intraoperatively, drainage of intraabdominal abscess with adhesiolysis was done and after thorough peritoneal drainage, a drain was also placed in the pelvis. Total parenteral nutrition with intravenous Polymyxin B was started and Teicoplanin, Fluconazole, and Colistin were continued. The patient recovered very well after this operative procedure. There was significant clinical improvement, with no fever spikes, ascites, or abdominal pain and tenderness. After a total duration of 1.5 months of hospital stay, the patient was successfully discharged. On follow-up, the patient is clinically better, gaining weight, and stent removal has been planned.

3. DISCUSSION

Acute pancreatitis is a rare cause of pancreatic ascitis. Reactionary ascites is usually mild and do not require any intervention, whereas severe ascites must be evaluated for etiology. In cases of intractable ascites, acute pancreatitis should be considered as a differential diagnosis [6].

The incidence of pancreatic ascites in pediatric population is extremely low when compared to adults [7].

Diagnosis is based on imaging studies, and elevated amylase and lipase levels in blood and or ascitic fluid confirm the pancreatic origin of ascites [8] .

Treatment primarily focuses on supportive care, including fluid management, nutritional support, and careful monitoring of electrolytes. More invasive treatments, such as drainage or surgical interventions, may be required in severe cases. Somatostatin or octreotide with diuretics may be used as it reduces pancreatic exocrine function and aids in healing the damaged duct [9].

The strength of my case report is that it emphasizes the importance of early recognition and intervention of a rare cause of ascites in children. The limitation is the limited availability of data on pancreatic ascites cases among pediatric age groups.

4. CONCLUSION

This case highlights the importance of considering pancreatic ascites as a potential complication of acute pancreatitis in children. Awareness of this potential outcome is important for early identification and intervention to optimize the outcome.

5. LIST OF ABBREVIATIONS

ERCP -Endoscopic retrograde cholangiopancreatography

AEC -absolute eosinophil count

CECT -contrast-enhanced computed tomography

HIV- Human Immunodeficiency Virus

HbsAg- Hepatitis B surface antigen

CBNAAT- Cartridge-based nucleic acid amplification test

6. STATEMENTS & DECLARATIONS

Ethics approval and Consent to participate – The Institutional Ethics Committee (Vidyapeeth Ethics Committee) approved the case report and informed consent was obtained from the parents.

Conflict of Interest: None to declare

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