

Hemorrhagic Rupture Of Simple Hepatic Cyst: A Case Study

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Abstract

Symptomatic hepatic cysts are infrequent and their presentation with rupture leading to an hemoperitonium is even more uncommon. This case report illustrates the challenges associated with the diagnosis and management of a ruptured simple hepatic cyst.

We report the case of 72-year-old female, a known case of liver cirrhosis, hypertension, diabetes mellitus, and cholelithiasis who presented to our center with features of acute abdomen and was diagnosed with the ruptured simple hepatic cyst. Deroofing and marsupialization of the cyst was performed.

This case report emphasizes the significance of early recognition and a multidisciplinary approach in managing ruptured simple hepatic cysts. Heightened clinical awareness, coupled with advanced imaging techniques, is crucial for accurate diagnosis and timely intervention.

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Introduction

Simple hepatic cysts are rare lesions that are mostly asymptomatic. According to imaging-based research, the estimated prevalence is higher, ranging from 2.5% to 18%.¹ Less than 10% to 15% of people with simple hepatic cysts have symptoms severe enough to require medical intervention for the cyst.² Symptomatic hepatic cyst is more common in women compared to men by the ratio of 10:1, presenting with symptoms such as right upper quadrant pain, nausea, vomiting, dyspnea.³ Ultrasound of Abdomen helps in confirming the presence of a simple hepatic cyst, which has sensitivity and specificity of 90%.⁴ Sometimes, simple hepatic cysts lead to uncommon consequences apart from presentation including rupture of the cyst, torsion, secondary infection, and obstructive jaundice.^{5,6}

Here we report a case of a 72-year-old lady who was admitted to our hospital with a symptomatic simple hepatic

cyst which spontaneously ruptured after admission. The patient was managed surgically where deroofing and marsupialization of the cyst wall was done.

Case report

A 72-year-old lady presented in the emergency department with the complaints of generalized abdominal pain with multiple episodes of vomiting for five hours. She also had generalized weakness and abdominal distension for the same duration. She was a known case of liver cirrhosis, hypertension, diabetes mellitus, and cholelithiasis. She denied a history of melena, hematemesis, and altered sensorium. On arrival, she was anemic, her blood pressure was 80/60 mm of Hg, her pulse was 112 bpm and her urine output was less than 10ml in an hour. Abdominal examination revealed a grossly distended abdomen with tenderness in the right upper quadrant.

Her Hemoglobin was 10.4 g/dl, total lymphocyte count (TLC) was 15,100 and platelets were 266,000. Urea and creatinine were 54 and 1.5 mg/dl respectively. ABG showed features of respiratory acidosis. Liver function tests were normal. She had normal serum lipase but elevated serum amylase. Laboratory findings are listed in **Table 1**. Her echocardiogram revealed reduced Left Ventricular Systolic Function (LVSF) with a reduced Ejection Fraction of 35%. Abdominal ultrasound showed a loculated thick-walled cystic lesion (79 x 48 x 47 cm) with thick septation in the left lobe of the liver with mild ascites and cholelithiasis (24.9 mm).

Table 1. Laboratory findings.

Laboratory tests	Result	Unit	Reference range
Total Leukocytes Count (TLC)	15.1	$10^3/\mu\text{L}$	4–11
Neutrophil	58	%	40–80
Lymphocyte	35	%	20–40
Hemoglobin	10.4	g/dl	13–17
Platelet Count	266	$10^3/\mu\text{L}$	150–450
Urea	54	mg/dl	17–43
Creatinine	1.5	mg/dl	0.7–1.3
Sodium	137	mEq/L	135–145
Potassium	4.2	mEq/L	3.5–5.2
Bilirubin Total	0.2	Mg/dl	0.4–1
Bilirubin Direct	0.1	mg/dl	<0.4
Alkaline Phosphatase (ALP)	66	U/L	32–128
Alanine Transferase (ALT)	12	U/L	5–40
Aspartate Transferase (AST)	20	U/L	5–35
Serum Albumin	3.5	g/dl	3.4–5.4
Prothrombin time (PT)/ International Normalized Ratio	13/1.75	seconds	11–13.5
Amylase	116	IU/L	<80
Lipase	61	U/L	<60
Hepatitis B Surface antigen	Negative		
Hepatitis C Antibody	Negative		
Human Immunodeficiency Virus	Negative		

She was admitted to the Intensive care unit and initial resuscitation was done but her clinical condition did not improve. Her blood pressure was 50/30 pulse was 120 bpm her GCS was 3, her hemoglobin dropped to 4gm/dl and an Ultrasound Abdomen revealed the disappearance of the previously identified hepatic cyst, and on aspiration, hemorrhagic content was revealed. The patient was intubated and a massive transfusion protocol was initiated and taken to the Operative room and emergent laparotomy was done.

A trauma incision was given (**Figure 1**) and on the opening of the peritoneum approximately two liters of blood was suctioned out and a clot of size 5 tetra pad was evacuated.



Figure 1. Intra operative findings of hemorrhagic rupture hepatic cyst.

Active bleeding was seen on the base and wall of the ruptured cyst with associated findings of cirrhosis of the liver. Hemostasis was achieved with suturing of the bleeding site with 4.0 polypropylene. Deroofing and marsupialization of the cyst wall was done (**Figure 2**), a biopsy was sent and a drain was kept on Morrison's pouch, and wound closure was done.

On 1st post-operative day, she was hemodynamically stable and was extubated. Her arterial blood gas analysis revealed elevated PCO_2 -55.5 and pH- 7.34 and managed with salbutamol nebulization and spirometry and was kept in Bi-PAP. On 2nd post-operative day, she developed features of AKI which were supported by a renal function test, and features of pulmonary edema which was managed with diuretics and glyceryl trinitrate (GTN). A repeat liver function test showed elevated SGPT and SGOT 2090/ 3870 which returned to normal on the 7th postoperative day. Her arterial blood gas analysis showed no improvement and a chest X-ray was done on the 3rd post-operative day revealing minimal right-sided pleural effusion with bilateral atelectasis which was treated by keeping her on Bi-PAP, chest physiotherapy, and spirometry. The patient was recovering over time. She developed arterial fibrillation on the 7th Postoperative day managed with verapamil then digoxin. On the 9th post-operative day drain was removed and she was shifted to the ward. And was discharged on the 12th post-operative day. Biopsy report revealed a simple hepatic cyst.

Discussion

Every cystic lesion is differentiated into true and false cyst based on epithelial lining. True hepatic cysts might be congenital, parasitic, malignant, or connected with the biliary canal, while false hepatic cyst are result of trauma, hemorrhage.⁵ Simple hepatic cyst is non-parasitic in nature and are congenital.



Figure 2. After obtaining hemostasis

With a frequency of 2.5–5% and a little female predominance in asymptomatic cases (female-male ratio, 1.5:1), the benign cyst is the most prevalent liver pathology. In general terms, simple hepatic cysts are congenital which is formed due to lack of any communication with intrahepatic biliary tree. The simple cyst usually contains clear fluid and varies in size ranging from few millimeters to centimeters.⁷ Congenital cyst, Caroli disease, biliary hamartomas and polycystic liver disease are common types of simple hepatic cyst. Simple hepatic cysts are usually detected incidentally during imaging.² Simple cysts that don't cause any symptoms don't need to be treated or monitored. It is recommended that cysts larger than 4 cm be monitored periodically using imaging modalities to guarantee stability. Only 15% of them exhibit symptoms, especially when they are greater than 4 cms.⁸

Simple hepatic cyst is common among women aged 30-70 years as coinciding with our case. Among symptomatic cases, sclerotherapy combined with aspiration is a useful

therapeutic approach⁹, which was not possible in our case. There were few available choices for treatment in this particular scenario and was challenged by time as patient was consistently hemodynamically unstable despite aggressive resuscitation and had low ejection fraction with left ventricular systolic dysfunction. There are only few documented case of successful management of hemorrhagic rupture of simple hepatic cyst.

Non-parasitic simple hepatic cysts can be effectively treated with open or laparoscopic cyst deroofing.¹⁰ For small hepatic cysts, laparoscopic deroofing offers a short hospital stay, complete symptom alleviation with little morbidity, low recurrence rates, and a tissue sample for histological confirmation of the lesion.¹¹ In our case we opted for open deroofing and marsupialization with drain placement in Morrison Pouch, as laparoscopic surgeries are not opted in ruptured hemorrhagic hepatic cyst and the patient was a known case of liver cirrhosis.

Prompt and timely management improved the outcome in our case.

Conclusion

Our case report concludes by highlighting the importance of identifying the uncommon presentation of symptomatic simple hepatic cysts, especially when associated with acute abdominal symptoms post rupture. Because this ailment is rare, misdiagnosis could be a problem, reinforcing the need for a high index of suspicion among healthcare providers. A multidisciplinary strategy combining clinical knowledge, radiological proficiency, and surgical intervention is required for the effective therapy of a ruptured simple hepatic cyst. In our case study, prompt diagnosis by imaging tests enabled quick decision-making, which in turn enabled a focused action plan. In order to stabilize the patient and treat the cystic rupture, the use of deroofing and marsupialization of cyst was beneficial.

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