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Massive Inferior Vena Cava Thrombosis and Pulmonary Embolism Secondary to Hepatic Hydatid Cyst: A Fatal Case

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 68-year-old
Final Diagnosis: Hydatid cyst • pulmonary embolism • thrombosis of the vein
Symptoms: Abdomen distension • dyspena • fever • hypotension • metabolic acidosis • sepsis
Clinical Procedure: —
Specialty: Critical Care Medicine • Gastroenterology and Hepatology • Pulmonology

Objective: Unusual clinical course

Background: Hydatid disease, caused by *Echinococcus granulosus,* is an endemic parasitic infection that predominantly affects the liver. Although slow-growing and asymptomatic, large hepatic cysts lead to catastrophic vascular complications, including inferior vena cava (IVC) thrombosis and pulmonary embolism (PE). Although rare, life-threatening events require prompt recognition and multidisciplinary management to optimize outcomes.

Case Report: A 68-year-old woman with paroxysmal atrial fibrillation presented with right upper-quadrant pain, fever, and poor oral intake for several weeks. Examination revealed abdominal distension, hepatomegaly, and mottled, cold lower limbs. Laboratory findings showed severe metabolic acidosis and leukocytosis, indicating significant systemic involvement and possible sepsis. Computed tomography (CT) angiography demonstrated a pulmonary embolism, a giant (29 × 18 cm) right hepatic cyst exerting a mass effect, and extensive thrombosis extending from the infrarenal IVC to both external iliac veins. Echocardiography showed extrinsic cardiac compression without signs of tamponade but with functional compromise related to mass effect. Management included anticoagulation, albendazole, broad-spectrum antibiotics, vasopressors, and continuous renal replacement therapy. Ultrasound-guided percutaneous drainage of the cyst yielded 4 L of purulent fluid, with transient improvement in limb perfusion suggesting partial decompression of venous outflow obstruction. An IVC filter was inserted, but despite intensive therapy, the patient developed refractory shock and died due to progressive hemodynamic collapse and multiorgan failure.

Conclusions: Massive hepatic hydatid cysts can precipitate IVC thrombosis and PE through mechanical compression or infection-related inflammation. Early diagnosis, multidisciplinary coordination, and timely surgical or percutaneous decompression are vital to prevent death.

Keywords: echinococcosis • pulmonary embolism • vena cava, inferior

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952041

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Introduction

Hydatid disease, caused by *Echinococcus granulosus*, is an endemic parasitic infection that predominantly affects the liver. Although usually slow-growing and asymptomatic, large hepatic cysts can lead to catastrophic vascular complications such as inferior vena cava (IVC) thrombosis and pulmonary embolism (PE) [1].

The life cycle of *Echinococcus granulosus* involves a definitive host, typically a dog or other canid, which harbors the adult tapeworm in its small intestine [2,3]. Eggs are shed in the feces and ingested by intermediate hosts, such as sheep, cattle, or, accidentally, humans. Upon ingestion, the eggs hatch in the human small intestine, releasing oncospheres that traverse the intestinal wall to enter the portal venous system [4]. The liver acts as the primary physiological filter for these larvae, explaining why the right lobe of the liver is the site of infection in approximately 70% to 80% of cases [5].

The natural clinical history of hepatic cystic echinococcosis is characterized by an indolent course. The parasite develops into a fluid-filled cyst that grows at a rate of approximately 1 cm per year [6]. This slow growth often allows the host to remain asymptomatic for a long time, until the cyst reaches a massive size or a complication occurs [7]. Cysts are considered "giant" when they exceed 10 cm in diameter; these lesions are significantly more likely to cause mass effect-related complications in adjacent organs, including the gallbladder, stomach, and major vascular structures like the portal vein and the IVC [8].

The most common genotypes among canids are *Echinococcus granulosus* sensu stricto (G1-G3) and the G6/7 cluster of *Echinococcus canadensis*. The global prevalence of echinococcosis in canids reaches up to nearly 34% worldwide, with the highest prevalence reported in South America and the lowest in Europe, with a rate of nearly 66% and 19%, respectively [9]. In Africa, the overall prevalence in 14 countries was nearly 17%, with the highest prevalence in North Africa, with a rate of nearly 26% [10].

The intermediate host in Saudi Arabia varies among hosts according to site, season, animal age, and sex; the prevalence of infection is 32.9% in camels, 8.3% in cattle, 12.6% in sheep, and 6.56% in goats. It was reported that sheep and goats are the most important intermediate hosts of *Echinococcus granulosus* due to higher percentages of fertile cysts (47.67% in sheep and 23.99% in goats) [11].

Hepatic hydatid cysts result from infection with *Echinococcus granulosus* and can cause serious complications when expanding lesions compress adjacent structures such as the IVC [2]. Thrombosis can result from mechanical obstruction, inflammation, or direct fistulization into the venous system [5,11-13]. Patients with IVC thrombosis secondary to hydatid cysts often present with features of venous obstruction or embolic complications. Early recognition is crucial because these events are rare but potentially fatal [12,13].

Case Report

A 68-year-old woman with a history of paroxysmal atrial fibrillation presented to the emergency department with 3 months of right-upper-quadrant abdominal pain and recent worsening fever. She denied respiratory, urinary, or neurological symptoms. While in the waiting area, she suddenly collapsed, gasping for air. Her oxygen saturation dropped to 75% despite high-flow oxygen, and her Glasgow Coma Scale (GCS) score was 8/15. She was transferred to the resuscitation unit and intubated.

On examination, the abdomen was distended with palpable hepatomegaly. Air entry was decreased bilaterally. Both lower limbs were cold; the right leg was swollen with purple mottling. Venous blood gas analysis revealed severe metabolic acidosis. Laboratory tests are shown in **Tables 1 and 2**.

Table 1. Arterial blood gases on admission.

Initial Venous blood gas	Patient value	Reference range
pH	7.09	7.35-7.45
PCO_2 (mm Hg)	45	35-45 mm Hg
PO_2 (mm Hg)	40	35-40 mm Hg
HCO_3^- (mmol/L)	13.7	24-28 mmol/L
Hemoglobin (g/dL)	14	13-18 g/dL
Sodium, Na^+ (mmol/L)	124	135-145 mmol/L
Potassium, K^+ (mmol/L)	9.4	3.5-5.0 mmol/L
Chloride, Cl^- (mmol/L)	98	95-107 mmol/L
BEecf (mmol/L)	-16.1	-3 to +3 mmol/L

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Table 2. Laboratory test results on admission.

Hematology, coagulation, inflammatory markers, and chemistry	Patient value	Reference range
Hemoglobin (g/dL)	13.6	12-16 g/dL
WBC (× 10^9/L)	22.30	4.0-11.0 × 10^9/L
RBC (× 10^{12}/L)	4.10	4.1-5.1 × 10^{12}/L
NEUT (× 10^9/L)	17.84	2.0-7.5 × 10^9/L
MCV (fL)	81.7	80-96 Fl
MCH (pg)	25.8	27-33 pg
MCHC (g/L)	316	320-360 g/L
RDW (%)	18.2	11.5-14.5%
Platelets (× 10^9/L)	323	150-400 × 10^9/L
INR	1.49	0.8-1.2
PT (seconds)	15.90	11-14 s
PTT (seconds)	28.50	25-35 s
CRP (mg/L)	94.40	< 5 mg/L
ESR (mm/hr)	119	0-20 mm/hr
PCT (ng/mL)	0.32	< 0.05 ng/mL
Lactic acid (mmol/L)	5.91	0.5-2.2 mmol/L
Troponin I (ng/L)	30.8	< 14 ng/L
Uric acid (µmol/L)	449	150-360 µmol/L
Total bilirubin (µmol/L)	24.7	5-21 µmol/L
ALP (U/L)	1249	40-129 U/L
Phosphorus (mmol/L)	2.06	0.8-1.5 mmol/L
Adj Ca (mmol/L)	2.45	—
Potassium (mmol/L)	5.4	3.5-5.0 mmol/L
Albumin (g/L)	28	35-50 g/L
Magnesium (mmol/L)	0.89	0.7-1.0 mmol/L
Sodium (mmol/L)	129	135-145 mmol/L
Chloride (mmol/L)	101	98-107 mmol/L
Creatinine (µmol/L)	100	62-106 µmol/L
eGFR (mL/min/1.73 m^2)	51	> 90 mL/min/1.73 m^2
CO_2 (bicarbonate) (mmol/L)	11	22-28 mmol/L
Glucose (mmol/L)	11.5	< 11.1 mmol/L
Serum urea nitrogen (mmol/L)	7.5	2.5-7.1 mmol/L
AST (U/L)	94	10-40 U/L
ALT (U/L)	156	7-56 U/L
GGT (U/L)	856	8-61 U/L
D-dimer (mg/L)	6.12	< 0.5 mg/L
Fibrinogen (g/L)	2.97	2.0-4.0 g/L

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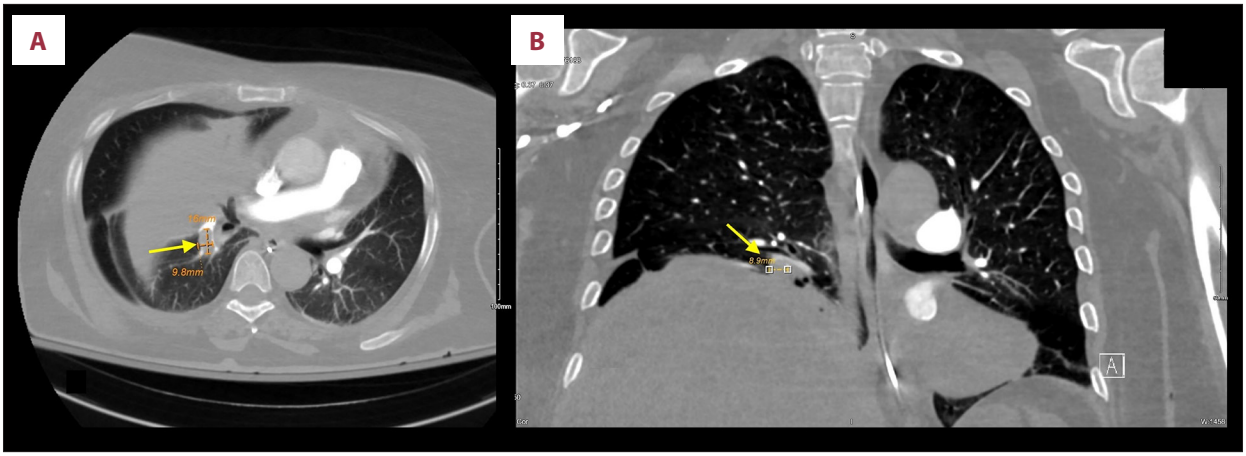

Figure 1. CT pulmonary angiography showing segmental and subsegmental pulmonary emboli (yellow arrows) and a giant right hepatic cyst measuring 29 × 18 cm, exerting right-hemithoracic mass effect with leftward mediastinal displacement. (**A**) Axial image. (**B**) Coronal image

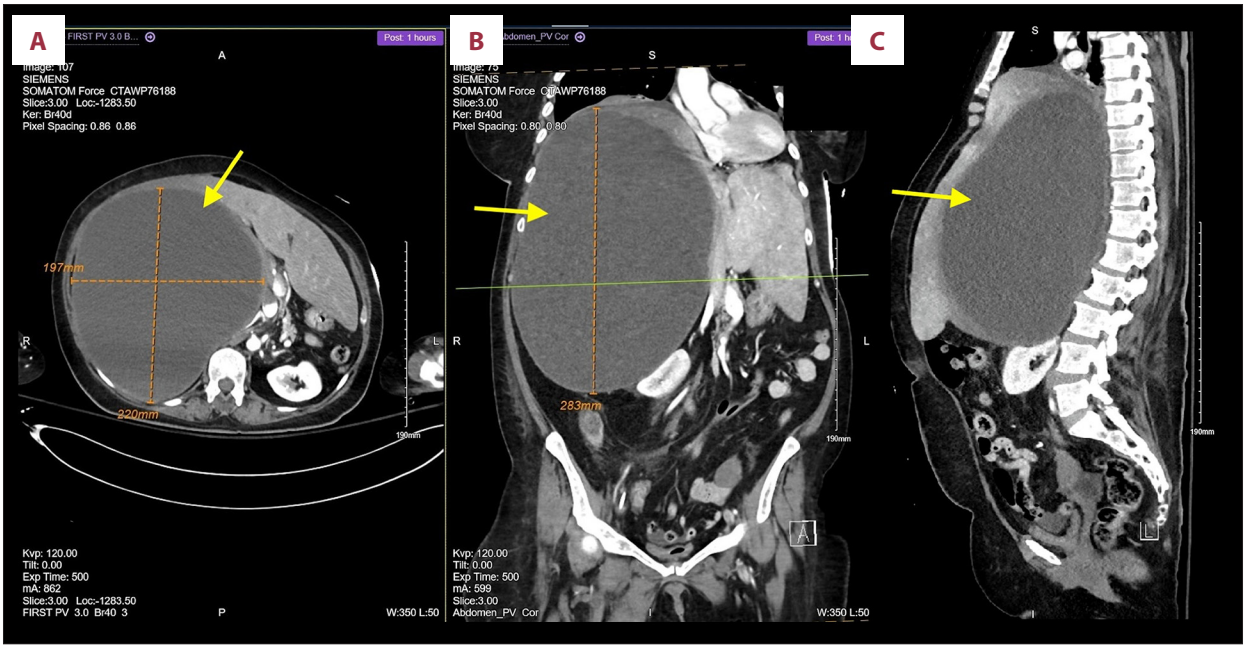

Figure 2. CT abdomen, showing a hydatid cyst with mass effect with IVC thrombosis in **A:** axial, **B:** coronal, and **C:** sagittal planes. The arrows point to the hydatid cyst in the liver.

Contrast-enhanced computed tomography (CT) pulmonary angiography demonstrated segmental and subsegmental PEs, a giant (29 × 18 cm) right-lobe hepatic cyst, and mass effect on the right hemithorax with leftward displacement (**Figure 1**). Abdominal CT revealed extensive thrombosis extending from the infrarenal IVC to both external iliac veins, more pronounced on the right- prothrombotic state (**Figure 2**). Echocardiography showed a small pericardial effusion and extrinsic right-atrial compression without tamponade or pulmonary hypertension. The diagnosis of *Echinococcus granulosus* infection was primarily supported by the characteristic CT imaging findings. Owing to the patient's critical emergency situation, there was insufficient time to perform the conventional laboratory serological tests before urgent intervention. Moreover, the surgical decision-making was substantially supported by the CT pulmonary angiography and CT abdomen. Abdominal ultrasound was not performed due to the emergency situation.

The pulmonary embolism response team initiated intravenous heparin, albendazole, and broad-spectrum antibiotics. Vascular surgery advised conservative management as Doppler studies demonstrated triphasic arterial flow without ischemia. The patient was admitted to the intensive care unit with worsening acidosis (arterial blood gas results are shown in **Table 3**).

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Table 3. Follow-up postoperative laboratory test results.

Arterial blood gas	Patient value	Reference range
pH	7.28	7.35-7.45
PCO_2 (mm Hg)	32	35-45 mm Hg
PO_2 (mm Hg)	233	80-100 mm Hg
HCO_3^- (mmol/L)	15	22-26 mmol/L
Hemoglobin (g/dL)	13	13-18 g/dL
Sodium, Na^+ (mmol/L)	125	135-145 mmol/L
Potassium, K^+ (mmol/L)	4.5	3.5-5.0 mmol/L
Chloride, Cl^- (mmol/L)	97	95-107 mmol/L
BEecf (mmol/L)	-11	-2 to +2 mmol/L

She received vasopressors, sedation, piperacillin–tazobactam, vancomycin, meropenem, albendazole, and continuous renal replacement therapy for acute kidney injury.

Given persistent shock and concern for the infected cyst, bedside decompression was performed. Under ultrasound guidance, a 10-Fr catheter was inserted, draining 4 L of purulent material that cultured *Bacteroides thetaiotaomicron*. Following drainage, right-leg perfusion transiently improved. An IVC filter was inserted to prevent embolic events. Despite temporary hemodynamic stabilization, metabolic acidosis persisted, and continuous renal replacement therapy circuits repeatedly clotted. Anticoagulation was held because of a rising international normalized ratio. Re-accessing the cyst cavity was deemed unsafe. The patient developed refractory shock, severe hyperkalemia, bradycardia, and had cardiac arrest. Resuscitation was unsuccessful. Histopathological examination of the excised tissue confirmed the diagnosis of hepatic hydatid cyst of *Echinococcus granulosus*.

Discussion

This case highlights a rare but devastating complication of hepatic hydatid disease—IVC thrombosis with PE. The formation of a thrombus in the IVC is governed by the principles of Virchow's triad: stasis, endothelial injury, and hypercoagulability [15]. In this case, all 3 elements of Virchow's triad contributed to the development of IVC thrombosis [16]. First, stasis was present due to mechanical compression from a large cyst located on the retrohepatic segment of the IVC. Second, endothelial injury resulted from localized inflammation caused by an infected CE3b cyst. Third, hypercoagulability was driven by systemic sepsis, paroxysmal atrial fibrillation, and metabolic acidosis. These factors collectively created a profound prothrombotic state, culminating in the patient's presentation.

Current recommendations suggest that in the presence of extensive IVC thrombosis, the filter should be placed before any invasive manipulation of the cyst [15,17]. Decompressing a giant cyst relieves the tamponade effect on the IVC. While this is necessary for restoring venous return, sudden restoration of flow through a previously compressed vessel can mobilize unstable thrombotic material or fragments of the cyst wall, leading to catastrophic pulmonary embolism [18].

The pathophysiology likely involves extrinsic compression by the enlarging cyst, compounded by inflammatory changes and infection. Similar cases have been reported, but the combination of massive cyst size (29 × 18 cm), septic thrombosis, and embolic phenomena underscores the clinical severity [14]. Mahajan et al described acute IVC thrombosis as an unusual manifestation of hydatid cysts [19], while Ismail et al reported direct cyst–IVC fistulization [12]. Our patient's presentation was consistent with extrinsic compression rather than fistulization. Kantorová et al emphasized early recognition and surgical management in preventing fatal outcomes; however, our patient presented in extremis, precluding definitive surgery [13]. Management requires individualized multidisciplinary care. Surgical cyst excision is the standard treatment, but it may be infeasible in unstable patients. Percutaneous drainage can offer temporary decompression, as seen here, but carries risks of infection and recurrence [5,13]. Albendazole therapy and vigilant anticoagulation are essential adjuncts, but anticoagulation can be challenging in septic or coagulopathic patients.

Conclusions

Massive hepatic hydatid cysts can cause severe vascular complications, including IVC thrombosis and PE. Cyst complications arise because of the pressure effect of a large cyst on adjacent

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vascular structures, leading to mechanical obstruction and potentially triggering embolization. The potential life-threatening outcomes of a large hepatic hydatid cyst underscore the importance of vigilant clinical monitoring in affected patients.

Timely recognition of the vascular complications is essential. Early diagnosis enables prompt intervention, which may involve decompression procedures or definitive surgical management. Therefore, prompt action is critical to improving patient outcomes and survival. Multidisciplinary care is often required, and the approach should be tailored to the clinical status of the patient.

Department and Institution Where Work Was Done

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Statement

The authors confirm that this report is original and has never been published by any means in any journal or elsewhere. Grammarly was used for English language proofreading, and AI was used to prepare the abbreviation list required at the end of the manuscript. The authors declare that no AI tools were used to generate the scientific contents, interpret the findings, or construct the core text.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

Abbreviations

ALP, alkaline phosphatase; **ALT**, alanine aminotransferase; **AST**, aspartate aminotransferase; **BEecf**, base excess in extracellular fluid; **CE**, cystic echinococcosis; **CRP**, C-reactive protein; **CRRT**, continuous renal replacement therapy; **CT**, computed tomography; **GCS**, Glasgow Coma Scalel; **GGT**, gamma-glutamyl transferase; **HCO3–**, bicarbonate; **INR**, international normalized ratio, **IVC**, inferior vena cava; **MCH**, mean corpuscular hemoglobin; **MCHC**, mean corpuscular hemoglobin concentration; **MCV**, mean corpuscular volume; **PE**, pulmonary embolism; **PCT**, procalcitonin; **PT**, prothrombin time; **PTT**, partial thromboplastin time; **RBC**, red blood cell count; **RDW**, red cell distribution width; **WBC**, white blood cell count.

References:

1. Greco S, Cannella R, Giambelluca D, et al. Complications of hepatic echinococcosis: Multimodality imaging approach. Insights Imaging. 2019;10:113
2. Alabousi A, Patlas MN, Khalili K, Haider EA: Parasitic liver infections: Imaging findings and strategies for timely diagnosis. Curr Probl Diagn Radiol. 2020;49:447-51
3. Brunetti E, Tamarozzi F, Macpherson C, et al. Ultrasound and cystic echinococcosis. Ultrasound Int Open. 2018;4:E70-78
4. Frey H, Barth TF: Macroscopic and microscopic aspects of *Echinococcus multilocularis* and *Echinococcus granulosus* in humans: A guideline for pathologists. Visceral Medicine. 2025;42(2):85-91
5. Naser A, Alqader RS, Ghanem L, et al: Inferior vena cava thrombosis secondary to liver hydatid cyst: Case report. Clin Surg J. 2024;5:1-5
6. Staudacher M, Hotz JF, Ramharter M, Lagler H. Cystic echinococcosis (hydatid disease): Current insights into epidemiology, diagnosis, therapy, and prophylaxis. Br Med Bull. 2026;157:ldag008
7. Govindasamy A, Bhattarai PR, John J: Liver cystic echinococcosis: A parasitic review. Ther Adv Infect Dis. 2023;10:20499361231171478
8. Shaprinskiy V, Verba A, Formanchuk T, et al: Surgical treatment of echinococcosis of the liver and its complications. Wiad Lek. 2022;75:244-50
9. Shams M, Khazaei S, Naserifar R, et al. Global distribution of *Echinococcus granulosus* genotypes in domestic and wild canids: A systematic review and meta-analysis. Parasitology. 2022;149:1147-59
10. Karshima SN, Ahmed MI, Adamu NB, et al: Africa-wide meta-analysis on the prevalence and distribution of human cystic echinococcosis and canine *Echinococcus granulosus* infections. Parasit Vectors. 2022;15:357
11. Ibrahim MM: Study of cystic echinococcosis in slaughtered animals in Al Baha region, Saudi Arabia: Interaction between some biotic and abiotic factors. Acta Tropica. 2010;113:26-33
12. Ismail IB, Sghaier M, Boujmil K, et al: Hydatid cyst of the liver fistulized into the inferior vena cava. Int J Surg Case Rep. 2022;94:107060
13. Kantorová E, Vajdová V, Hnilicová P, et al: Cystic echinococcosis causing thrombosis of the inferior vena cava in a patient with multiple sclerosis treated with alemtuzumab. Ther Adv Neurol Disord. 2020;13:1756286420954081
14. Koçak S, Bumin C, Erdem E, et al: Unusual complication of hydatid cysts: Acute inferior vena caval thrombosis. Dig Surg. 1993;10:114-15
15. Musielak MC, Singh R, Hartman E, Bernstein J: Simple hepatic cyst causing inferior vena cava thrombus. Int J Surg Case Rep. 2014;5:339-41
16. Kushner A, West WP, Suheb MZK, Pillarisetty LS: Virchow triad. In: StatPearls [Internet]. StatPearls Publishing; 2024
17. Aloun A: Conventional and minimally invasive treatment of liver hydatid disease. In: Hydatid disease: Diagnosis, treatment and follow up strategies. Springer; 2025;105-14
18. Aili A, Peng L, Zhang J, et al: Hydatid pulmonary embolism: A case report and literature review. Am J Case Rep. 2021;22:e934157
19. Mahajan A, Verma M, Kapila S: Spontaneous rupture of hepatic hydatid cyst into the inferior vena cava and hepatic vein. Indian J Thorac Cardiovasc Surg. 2025;41:219-22

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