Acute Popliteal Artery Embolism Due To Ruptured Mediastinal Hydatid Cyst Into Thoracic Aorta – A Case Report

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Abstract

A 40 year old female presented with the clinical features suggestive of acute arterial occlusion of the right leg for one day. Compression Ultrasonography revealed a linear membrane like defect in the right popliteal artery suggestive of an embolus. Patient was subjected to emergency catheter embolectomy revealing a laminated hydatid membrane within the artery. The patient was evaluated postoperatively and CECT chest documented a primary mediastinal hydatid cyst eroding into descending thoracic aorta. The mediastinal lesion was later treated with total cyst excision. In endemic areas, it is important to consider hydatid cysts in the differential diagnosis of an acute arterial occlusion.

INTRODUCTION

Hydatid disease is a zoonosis caused by flat worm Echinococcus, most commonly Echinococcus granulosus. Infected dogs release eggs through their faeces and the eggs infect humans through food and water. The most common locations of hydatid cysts are the liver (65-75%) and lungs (10-25%). If the larvae are not trapped in either liver or lungs, or if they by-pass the liver by travelling via lymphatics, it may lodge itself in any part of the body including the peritoneal cavity (8-18%), spleen (2-3%), kidneys (1-4%), uterus and adnexa (0.5-1%), retroperitoneum (0.5-1%), pancreas (0.5-0.8%), subcutaneous (1-2%), others (0.1-3%) (1). Extra pulmonary but intra-thoracic hydatid cysts are very rare, its reported incidence being between 0.5-0.8 percent. Intrathoracic extra pulmonary locations are generally the mediastinum, pleura, pericardium and chest wall (2). A mediastinal site is exceptional (0.1% of all localisations) (3) and can result in arterial occlusions in the aorta, iliac arteries, femoral arteries, popliteal arteries and even the myocardial arteries (4,5).

CASE REPORT

A 40-year old female presented in accident and emergency department with the complaint of pain in the right leg of day duration. The pain was sudden in onset, moderately severe, continuous in nature, localized to the whole leg and associated with its bluish discoloration. There was no such history in the past and she had been previously healthy. On examination the patient was hemodynamically stable. The right leg was tender and cold. The dorsalis pedis and posterior tibial arterial pulsations were absent on palpation. Homans sign was negative. Sensations, joint movements and deep tendon reflexes were normal. Her abdominal and chest examinations were unremarkable. Compression Ultrasonography of the right leg showed a linear membrane like filling defect, with free distal end, in the right popliteal artery suggestive of an embolus (Fig 1). The patient was taken for urgent catheter embolectomy, which revealed a shiny laminated membrane of hydatid cyst within the artery. The patient was evaluated for localisation of primary site of hydatid disease post operatively. ELISA (enzyme linked immunosorbent assay) for hydatid disease was positive. Abdominal ultrasonography was unremarkable. Compression Ultrasonography of the right leg showed a linear membrane like filling defect, with free distal end, in the right popliteal artery suggestive of an embolus (Fig 1). The patient was taken for urgent catheter embolectomy, which revealed a shiny laminated membrane of hydatid cyst within the artery. The patient was evaluated for localisation of primary site of hydatid disease post operatively. ELISA (enzyme linked immunosorbent assay) for hydatid disease was positive. Abdominal ultrasonography was unremarkable. Chest X-ray showed a small left-sided pleural effusion. A multislice CECT scan of the chest documented a primary mediastinal hydatid cyst (cystic laminated structure) eroding into the descending thoracic aorta with contrast leak from aorta into the mediastinal cyst noted (Fig 2,3). No other abnormality was detected in the abdomen or chest on CT scan.
Mediastinal hydatid cyst was treated by cystostomy and total pericystectomy through a median sternotomy approach and the patient was put on prophylactic albendazole (10 mg/kg/day) for three cycles of 21 days each with a gap of one week between each cycle to avoid recurrence. Postoperative period was uneventful and the patient was discharged after 10 days.

**Figure 1**
Figure 1. Compression ultrasonography of right leg showing linear membrane like filling defect in the popliteal artery, with free distal end, suggestive of an embolus. The artery is otherwise within normal limits.

**Figure 2**
Figure 2. CECT Chest documented a primary mediastinal hydatid cyst (cystic laminated structure) eroding into the descending thoracic aorta with contrast leak from aorta into the mediastinal cyst.

**DISCUSSION**
Hydatid disease is seen endemically among sheep raising communities. The disease still continues to be a serious problem in countries like Australia, New Zealand, Middle East, Africa, India, South America, Turkey and Southern Europe (6). Mediastinal hydatid cysts are usually isolated and primitive. The parasite localizes in the region after passing the hepatic and pulmonary filter, probably via an arterial branch of the thoracic aorta or via lymphatics. While there are no specific symptoms, patients may present with chest pain, cough, and dyspnoea. In addition, haemoptysis when the cyst involves the pulmonary parenchyma, superior vena cava syndrome when the cyst is large, intrathoracic phrenic or laryngeal recurrent neurological symptoms, vertebral destruction, and possible Bernard-Horner’s syndrome have been reported. Most patients are asymptomatic; the lesion being discovered incidentally on a routine chest X-Ray (3).

Other complications of mediastinal hydatid cyst can be very serious and include: rupture into the mediastinum, pleural cavity, and right ventricle, cysto-aortic fistula with the possibilities of multiple systemic and vascular embolizations, multi-organ failure, and death (7), infection, compression...
of vital structures (3.8) and life-threatening pulmonary embolism in case of rupture into the right ventricle. Complications caused by hydatid cysts if vascular invasion develops include anaphylactic shock, haemorrhage, systemic emboli and arterial occlusion (9). Our patient presented with acute popliteal arterial occlusion by hydatid laminated membrane secondary to erosion of primary mediastinal hydatid into the descending thoracic aorta. The contrast-enhanced computed tomographic features represent a pathognomonic sign of a communicating rupture of an echinococcal cyst into the aorta (10).

In conclusion, hydatid disease should be considered in the differential diagnosis of acute arterial occlusion, especially in the areas where the disease is endemic. The patient should be investigated to rule out hydatid cyst in other parts of body, especially in relation to large vessels and when there is no clear cause or risk factor for arterial occlusion such as in our patient.

Lastly one should also keep in mind the possibilities of local recurrence of the disease and development of hydatidosis at the primary or other sites. The patient has to be kept on regular follow up paying attention to these possibilities (11).

References

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