Blindness following Rupture of Hepatic Hydatid Cyst: A Case Report

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ABSTRACT

A 19 year-old woman admitted to Emergency Department with hypotension, sudden loss of vision and acute abdominal pain. Ultrasound and computed tomography demonstrated an occipital infarct in brain and ruptured intraperitoneal cyst of hydatid liver disease. Urgent laparotomy was performed and it included aspiration of cyst contents, peritoneal washing and drainage. Her vision loss improved by 15 hours postoperatively but generalized seizures were started. Weakness in all extremities was present. Cranial MRI demonstrated ischemia in the areas of middle, posterior and anterior cerebral arteries. She was discharged from the hospital with severe neurological deficits (unable to walk, not able to eat herself). Neurological deficits were improved with physiotherapy after two years. There was no recurrence of hydatid cysts in the follow-up of three years. We assumed that anaphylaxis after intraperitoneal rupture of hydatid liver cyst resulted with hypotension and reduced cerebral perfusion, caused the acute vision loss and other neurological symptoms. This unusual presentation of intraperitoneal rupture should be kept in mind particularly in endemic areas of hydatid disease.

Key words: hydatid disease, blindness, acute abdomen, perforation, stroke, allergic reaction

INTRODUCTION

Hydatid disease has a world wide distribution and it has endemic areas in Asia, Australia, Middle East, Southern Europe, Africa and South America. The most affected organ of hydatid disease is liver. Most cases are asymptomatic and symptoms usually occur after complications [1]. We present a case of intraperitoneal rupture of hydatid liver cyst, in which the patient was admitted with abdominal findings and blindness.

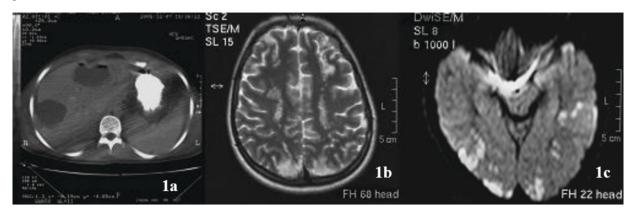
CASE PRESENTATION

A 19 year-old woman admitted to emergency department with sudden loss of vision for eight hours. She had a history of abdominal pain and distension lasting for 24 hours. There was no history of trauma or previously known disease.

She was irritable but had no dyspnea, stridor or wheezing. Blood pressure was 60/40 mmHg, heart rate was 130/min and respiratory rate was 28/min. The body temperature was normal. There was abdominal distention, muscular guarding and rebound tenderness. Other system examinations were normal. Ophthalmoscopy at the emergency room demonstrated no specific findings. She was urgently resuscitated with crystalloids/colloids and her blood pressure reached to 110/80 mmHg and heart rate decreased to 107/minute. There was mild leukocytosis (11.700/mm³) and eosinophilia (2.2%). Other laboratory parameters were within normal ranges. Abdominal ultrasound revealed intra-abdominal free fluid and three cysts in the liver. Perforated hepatic hydatid cyst was demonstrated with abdominal computed tomography (Fig. 1a). There was no evidence of hydatidosis in cranial and thorax computed tomography.

Urgent laparotomy was necessary for free perforation of hydatid liver cyst. Hydatid daughter cysts and six liter of free

Figure 1. (1a) Intra-abdominal free fluid and three cysts in the liver. The cyst in segment VI had an irregular wall and the inner layer was detached from its capsule. (1b, 1c) Cranial MRI and diffusion MRI revealed bilateral symmetrical ischemia in the areas of middle, posterior and anterior cerebral arteries.



fluid were aspirated at laparotomy. There were three cysts in the liver at segments II, VI and VII with measuring 8.5cm, 5 cm and 6cm, respectively. The cyst located in segment VI was found as ruptured but the others were intact. All the cysts were evacuated and washed with protoscolicidal (cetrimid 0.05%-chlorhexidine 0.005% combination) for 10 minutes. Cysts were partially excised and controlled for cysto-biliary communications. Cysts and the abdominal cavity were drained.

Postoperatively vision loss was improved by 15 hours but generalized seizures were started. There were bilateral extensor plantar reflexes and weakness of 2/5 on the Medical Research Council scale in all extremities. Bilateral symmetrical ischemia in the areas of middle, posterior and anterior cerebral arteries was demonstrated at both cranial MRI and diffusion MRI (Fig. 1b, 1c). EEG exhibited widespread background activity. She stayed in intensive care unit for seven days and during this period, echocardiography, and carotid and basillary artery Doppler ultrasound showed normal findings. Protein C, protein S, antithrombin III and antiphospholipid antibody levels were in normal ranges. She was followed in General Surgery Clinic and postoperative course was uneventful except neurological deficits. She was unable to walk and not able to eat herself. Neurologists suggested physiotherapy and acetylsalicylic acid 300mg/day for six months. Albendazol was administered 10mg/kg bid for 12 weeks. She was discharged from the surgery clinic with complete weakness of the extremities with no improvement on the 17th day. In the following two years, her neurological deficits were improved with physiotherapy except right gastrocunemius muscle shortening and hemianopsia in the left eye. Control cranial MRI demonstrated improvement with the previous infarcted areas except right occipital infarct and abdominal tomography demonstrated no recurrences after three years.

DISCUSSION

The hydatid cyst can rupture after trauma, or spontaneously as a result of increased intracystic pressure. It is a rare complication (1%-8%) and particularly superficial, large, and viable cysts with high pressure are prone to rupture [2]. Abdominal pain is the most common presenting symptom of intraperitoneal perforation, and in most cases nausea and vomiting accompany to pain. Spillage of hydatid fluid and protoscolices into the peritoneal cavity can cause allergic reactions such as urticaria, fever, hypotension or anaphylaxis [3]. Intraperitoneal perforated hydatid cysts have various clinical presentations between mild abdominal tenderness to fatal anaphylaxis [4-6]. Sometimes, neurological symptoms can be dominant [7]. Meyer and associates [7] reported a hepatic hydatid cyst rupture in a 5-year-old boy resulted with generalized seizures and anaphylaxis as the initial symptom. Our case had suffered from acute vision loss with abdominal findings as the first clinical findings. We assumed that hypotension and massive intraabdominal fluid collection occurred as a result of allergic reaction. The acute vision loss and other neurological symptoms can be attributed to abruptly reduced cerebral perfusion secondary to hypotension.

Because rupture of a hydatid cyst requires emergency intervention, rapid diagnosis is mandatory. Radiological evaluation with ultrasound or computed tomography should be performed immediately in the patients with suspected perforated intraperitoneal hydatid cyst [7-8]. Treatment against allergic reaction should be initiated and emergency surgery should be performed after diagnosis.

CONCLUSIONS

For the first time in the literature, acute vision loss was the prominent symptom of intraperitoneal hydatid cyst rupture. This unusual presentation of hepatic cyst rupture should be kept in mind particularly in endemic areas of hydatid disease.

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REFERENCES

- 1. Kayaalp C. Blumgart LH, Belghiti RJ, DeMatteo RP, Chapman WC, Büchler MW, Hann LE, D'Angleca M. Surgery of the Liver, Biliary Tract, and Pancreas. 4th ed. Philadelphia: Saunders Elsevier; c2007. Chapter, Hydatid cyst of the liver; p. 952-70.
- 2. Derici H, Tansug T, Reyhan E, Bozdag AD, Nazli O. Acute intraperitoneal rupture of hydatid cysts. World J Surg. 2006 Oct;30(10):1879-83.
- 3. Patel SS, Butt AA. Inadvertent rupture of an echinococcal cyst: case report and review of literature. Am J Med Sci. 2004 May;327(5):268-71.
- 4. Dirican A, Unal B, Ozgor D, Piskin T, Aydin C, Sumer F, Kayaalp C. Perforated Hepatic Hydatid Cyst into the Peritoneum with Mild Symptoms. Case Rep Gastroenterol 2008;2:439-43.

- 5. Kantarci M, Onbas O, Alper F, Celebi Y, Yigiter M, Okur A. Anaphylaxis due to a rupture of hydatid cyst: imaging findings of a 10-year-old boy. Emerg Radiol. 2003 Apr;10(1):49-50.
- 6. Kök AN, Yurtman T, Aydin NE. Sudden death due to ruptured hydatid cyst of the liver. J Forensic Sci. 1993 Jul;38(4):978-80.
- 7. Meyer PG, Bonneville C, Orliaguet GA, Dessemme P, Blakime P, Carli PA, Revillon Y. Grand mal seizures: an unusual and puzzling primary presentation of ruptured hepatic hydatid cyst. Paediatr Anaesth. 2006 Jun;16(6):676-9.
- 8. Gunay K, Taviloglu K, Berber E, Ertekin C. Traumatic rupture of hydatid cysts: a 12-year experience from an endemic region. J Trauma. 1999 Jan;46(1):164-7.