Huge hydatid cysts (HCs), especially those that are superficial and those in vital anatomic locations, are prone to abdominal trauma and rupture. Surgery has been the mainstay of therapy for large HCs. We report on a patient with a 32-cm diameter HC who presented with an abdominal mass originating from the right lobe of the liver. The mass grew exophytically, filled the abdominal cavity and extended down into the pelvis. The patient was taken into operation. Upon examination, a huge, thin-walled cyst with daughter vesiculae was found to be filling the entire right side of the abdomen, which grew exophytically. Direct HCs rupture when both the endo- and pericysts are torn, spilling cyst material into body cavities or adjacent hollow viscera. Occasionally, as in our case, a cyst may reach a huge diameter without any complications and activity limitations.

Background

Hydatid disease (HD) is prevalent and widespread in most sheep-raising countries in Asia, Australia, South America, the Far East, Southern Europe and in Mediterranean countries, including Turkey. Echinococcosis is a zoonosis caused by the larval stage of echinococcus granulosus (EG). EG is a cestod and is often manifested by a slowly growing cystic mass. The life cycle of echinococcosis and the natural course of HDs are well described. From contact with infected dogs or by ingesting contaminated vegetables and drinking polluted water, people become the incidental intermedi-ate host. The most commonly affected organ is the liver (75%), followed by the lungs (15%).

Surgery remains the mainstay of treatment for HD and aims to eliminate the parasite, promoting the rapid disappearance of any residual cavity and preventing complications and recurrence. Giant HC is life threatening but rare. It has potentially lethal complications, such as anaphylactic shock due to perforation; thus early diagnosis with definitive treatment is life-saving.

Case report

A thin (42 kg) woman, aged 19 years, was admitted to our general surgery clinic with complaints of dyspepsia, nausea and right upper quadrant pain radiating to the right groin for 5 years. Her symptoms did not limit her daily activities. Routine laboratory tests were normal and screening tests, such as indirect hemagglutination and enzyme-liked immunosorbent assay (ELISA), were positive. Abdomino-pelvic computed tomography and ultrasonography showed a 32 × 15-cm loculated cyst originating from the posterior segment of the right hepatic liver. The cyst filled the abdominal cavity, displacing the intestines and pancreas to the left and the right kidney posteriorly and extending down to the bladder (Fig. 1).

The patient was taken to operation with the diagnosis of huge HC. On examination, a huge, thin-walled cyst with daughter vesiculae was found to be filling the entire right side of the abdomen, which grew exophytically. Direct HCs rupture when both the endo- and pericysts are torn, spilling cyst material into body cavities or adjacent hollow viscera. Occasionally, as in our case, a cyst may reach a huge diameter without any complications and activity limitations.

Background

Hydatid disease (HD) is prevalent and widespread in most sheep-raising countries in Asia, Australia, South America, the Far East, Southern Europe and in Mediterranean countries, including Turkey. Echinococcosis is a zoonosis caused by the larval stage of echinococcus granulosus (EG). EG is a cestod and is often manifested by a slowly growing cystic mass. The life cycle of echinococcosis and the natural course of HDs are well described. From contact with infected dogs or by ingesting contaminated vegetables and drinking polluted water, people become the incidental intermedi-ate host. The most commonly affected organ is the liver (75%), followed by the lungs (15%).

Surgery remains the mainstay of treatment for HD and aims to eliminate the parasite, promoting the rapid disappearance of any residual cavity and preventing complications and recurrence. Giant HC is life threatening but rare. It has potentially lethal complications, such as anaphylactic shock due to perforation; thus early diagnosis with definitive treatment is life-saving.

Case report

A thin (42 kg) woman, aged 19 years, was admitted to our general surgery clinic with complaints of dyspepsia, nausea and right upper quadrant pain radiating to the right groin for 5 years. Her symptoms did not limit her daily activities. Routine laboratory tests were normal and screening tests, such as indirect hemagglutination and enzyme-liked immunosorbent assay (ELISA), were positive. Abdomino-pelvic computed tomography and ultrasonography showed a 32 × 15-cm loculated cyst originating from the posterior segment of the right hepatic liver. The cyst filled the abdominal cavity, displacing the intestines and pancreas to the left and the right kidney posteriorly and extending down to the bladder (Fig. 1).

The patient was taken to operation with the diagnosis of huge HC. On examination, a huge, thin-walled cyst with daughter vesiculae was found to be filling the entire right side of the abdomen, which grew exophytically. Direct HCs rupture when both the endo- and pericysts are torn, spilling cyst material into body cavities or adjacent hollow viscera. Occasionally, as in our case, a cyst may reach a huge diameter without any complications and activity limitations.
the entire right side of the abdomen, which grew exophytically and was pushing the stomach, intestine and pancreas to the left (Fig. 2). Pericystectomy was performed without cyst rupture (Fig. 3). Postoperative period was uneventful and the woman was discharged at postoperative day 5.

Discussion

HD is endemic in tropical and subtropical regions such as the Mediterranean basin including Turkey, South America, Near East, Southern Europe, Africa and Australia. In these areas, hydatidosis is known to be the source of serious human illness. E. granulosus is the causative agent of HD. E. granulosus is a cestode that grows in the small intestine of its definitive host, usually a dog. Eggs are eliminated in the feces and, when ingested, liberate their larvae in the duodenum of an intermediate host (cows, sheep). Humans are accidental hosts of this parasite, usually becoming infected through contact with infected dogs. The larvae cross the intestinal wall via the portal system and reach the liver, where they form cysts. Although cysts may develop in almost any part of the body, the location is mostly hepatic (75%) or pulmonary (15%); only 10% occur in the rest of the body.

Direct HC rupture occurs when both the endo- and pericysts are torn, spilling cyst material into body cavities or adjacent hollow viscera. Several reports describe cyst penetration into blood vessels or gastrointestinal tract lumen. Direct rupture may cause massive intraabdominal hemorrhage or biliary peritonitis. The incidence of HCs rupturing into the peritoneal cavity is unclear. Whether peritoneal spillage leads to a higher rate of recurrence remains unproven, although it may give rise to secondary cysts. Cyst rupture into the liver parenchyma occurs when cyst contents penetrate adjacent parenchyma without reaching the peritoneal cavity.

Rupture of the hepatic HC due to high intracystic pressure is one of the most serious complications. Cyst rupture into the biliary tract, which causes cholestasis, occurs in 5% to 17% of cases. Anaphylaxis and death secondary to ruptured abdominal hydatid cyst is well known. This case report demonstrates a huge HC which did not cause complication such as perforation for 5 years, despite having a very thin wall (2 mm) and being prone to trauma by its location, anterior to the visceral organs.

In conclusion, huge HCs can cause anaphylactic shock and death upon rupture. In this case, we present a patient with a cyst that reached a huge diameter without any complications and activity limitations. In similar patients, it is important to remain aware that minimal trauma may lead to cyst perforation with anaphylaxis.
Competing interests: None declared.

References

Urgent endovascular stenting of subclavian artery pseudoaneurysm caused by seatbelt injury

Hassan Adnan Bukhari, MD;† Roger Saadia, MD;† Brian William Hardy, MD†

Subclavian artery injury in blunt trauma is uncommon. The surgical repair of such an injury can be a major challenge. Because penetrating injuries are more common, many trauma surgeons have relatively little experience in dealing with blunt subclavian artery injury. This case is the first report of acute or immediate endovascular repair with pseudoaneurysm stenting of the subclavian artery, as a result of seatbelt injury, in the presence of clavicular fracture and neurological deficit.

**Case report**

A 45-year-old woman who was a restrained frontal passenger in a motor vehicle accident was transferred to our institution from a community hospital with a potential major thoracic vessel injury. She was alert, oriented and hemodynamically stable. Physical examination revealed a right supraclavicular hematoma. Her blood pressure and pulse were diminished on the right. Neurological examination revealed the presence of Horner’s sign on the right, with no other neurological deficit. A chest radiograph showed a left pneumothorax, a widened mediastinum with a right pleural cap and a displaced fractured right clavicle; the “scout view” of the computed tomography angiography (CTA) of the chest showed the same findings. CTA of the chest showed a right subclavian artery pseudoaneurysm measuring 22 × 17 mm with no extravasation. A chest tube was inserted for the pneumothorax. The patient remained hemodynamically stable. Angiography was done to evaluate the thoracic aorta and its branches. An arch aortogram demonstrated a normal thoracic aorta with a right subclavian artery pseudoaneurysm immediately distal to the origin of the right internal mammary and vertebral arteries (Fig. 1). Due to the close proximity of the aneurysm to the origin of the right vertebral artery, the right vertebral artery might have had to be sacrificed to treat the aneurysm by endovascular means. A subsequent left vertebral artery angiogram demonstrated a large patent left vertebral artery. At this point, it was decided to proceed with endovascular exclusion of the false aneurysm. With the use of a 38-mm Jostent, the pseudo-aneurysm was successfully excluded from the circulation with persistent flow identified in the right internal mammary and the right vertebral artery (Fig. 2). The pulses in both upper extremities became equal. After 2 months, the stent remained patent, and...