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Case No. 151: 19-year-old student from Albania with emergency admission due to shock

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Presentation of case

Keywords Hydatid cyst rupture · *Echinococcus granulosus* · anaphylactic shock

Dr. S. Horn: On the day of admission, the previously healthy 19-year-old student from Albania felt sick and vomited three times. She experienced a hot flash, flickering in the visual field, and a macular rash on her abdomen, followed by loss of consciousness. The emergency physician gave her a Glasgow Coma Score (GCS) of 3. On route to the hospital, she became hypotensive. When the electrocardiography showed tachycardic supraventricular arrhythmia and fibrillation, cardioversion was performed. She was then hemodynamically stable on admission to the intensive care unit but subsequently developed fever and laboratory evidence of inflamma-

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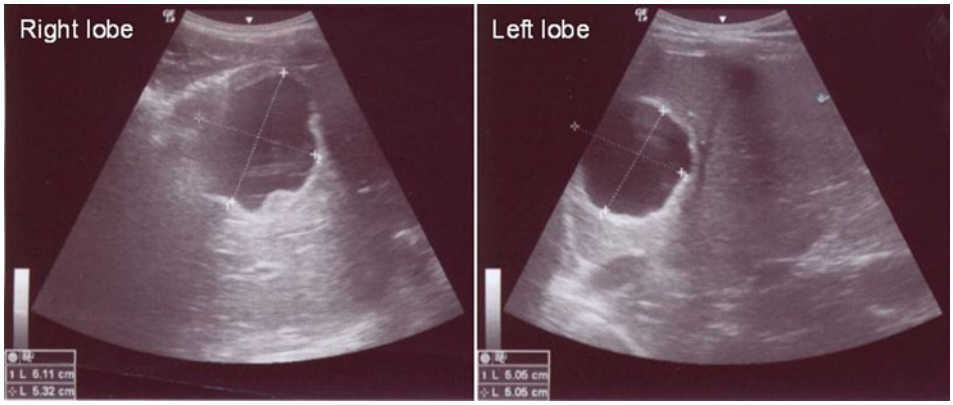

Fig. 1 Ultrasonography of the liver revealing cysts in the right lobe (diameter 6 cm) and the left lobe (diameter 5 cm)

tion. Intravenous antibiotic therapy (ampicillin and sulbactam) was begun. A drug screen of her urine was positive for methamphetamine, but she later denied any drug abuse and could not imagine how she could have tested positive for the drug. Selected laboratory results: day of admission: leukocytes 17.8 G/l (normal 4.4 -11.3); 70% neutrophils, 2% eosinophils; C-reactive protein (CRP) 8 mg/l (normal 0 -5). Day 4 after admission: leukocytes 9.0 G/l; 67% neutrophils, 17% eosinophils (absolute eosinophil count 1.6 G/l, normal up to 0.7); CRP 42 mg/l. The patient was extubated on the second day and transferred to a regular ward on the fourth day. Abdominal ultrasonography revealed a cystic lesion (diameter 6 cm) in the right lobe of the liver and another cystic lesion (diameter 5 cm) in the left lobe (Fig. 1). A dermatologist diagnosed urticaria, presumably as a drug reaction.

A diagnostic procedure was performed.

Differential diagnosis

Dr. C. Madl: The patient under discussion is a 19-year-old previously healthy student from Albania who was in shock on admission. In this case, shock is the leading symptom that needs to be analyzed. The initial symptoms included hypotension, tachycardia, vomiting, a hot flash, flickering in the visual field, a macular rash on the abdomen, and loss of consciousness. All these point to a positive shock index. There are, however, three remarkable facts in this case history: (1) tachycardic supraventricular arrhythmia and atrial fibrillation in such a young and previously healthy person is very unusual. Even with such a person in shock, I would expect sinus tachycardia with a heart rate of 140–170/min but not tachycardic supraventricular arrhythmia requiring cardioversion. This indicates very severe shock or may be due to another so far unknown and unidentified cardiac disease. (2) The patient had a GCS of 3 prior to any intervention by the emergency physician (e.g. administration of sedatives for intubation). GCS 3 is frequently seen in patients with cardiopulmonary arrest, severe intoxication, brainstem ischemia, or severe intracranial bleeding with increased cranial pressure. Loss of consciousness is a symptom of shock, but GCS 3 is a remarkably high score in this case. (3) A macular rash on the patient's abdomen is an atypical symptom in shock, but it may be a clue to the ultimate diagnosis.

Table 1 Differential diagnoses of shock

	Cause
Hypovolemic shock	Loss of blood, dehydration
Cardiogenic shock	LVF - Left ventricular failure, arrhythmia, or mechanical, cardiac (*e.g., vitium, aneurysm, tumor*), or extracardial pathology (*e.g., pneumothorax*)
Distributive (vasodilatory) shock	Sepsis (infection, cave: purpura fulminans), SIRS - systemic inflammatory response syndrome, toxic shock syndrome (due to infection with *Staphylococcus*), anaphylactic shock, shock due to intoxication/toxin (transfusion, heavy metals, drugs), endocrine causes of shock (Addison's disease, thyreotoxic crisis, myxedema), neurogenic shock (spinal trauma), post-CPR - cardiopulmonary resuscitation

As shock is the leading symptom in this case, we have to consider its differential diagnosis (Table 1). Hypovolemic shock can be excluded in our patient because there is no history of blood loss or dehydration (the history of three episodes of vomiting cannot have led to dehydration severe enough to induce hypovolemic shock). As the medical history does not mention any cardiac or pulmonary disease, cardiogenic shock is very unlikely, but still cannot be excluded completely, as distributive or vasodilatory shock can be induced in many different ways (Table 1). In any case, we can now exclude all other causes except septic shock due to infection with meningococcus (the reported macular rash on the abdomen could have been purpura fulminans), anaphylactic shock, and shock due to intoxication.

This leaves us with these four possible differential diagnoses to consider: shock due to mechanical cardiac pathology would have been diagnosed quickly by echocardiography and seems very unlikely. Moreover, I would also exclude shock due to infection with meningococcus. If the macular rush on the abdomen had been purpura fulminans, which is typical for infection with meningococcus, the patient would have also had neurological deficits, but none were reported. This reduces my differential diagnosis to just two: anaphylactic shock and shock due to intoxication, probably drug abuse.

Drug screening of the patient's urine was positive for methamphetamine, but the patient later denied any drug abuse, and she could not explain how she could have tested positive. She seemed trustworthy, so the positive

result for methamphetamine has to be questioned; the test could have been false positive. Kelly [1] found that in five of nine patients who were on the H_2-receptor antagonist ranitidine, test results for amphetamine and methamphetamine were falsely positive due to cross-reactivity of ranitidine in the EMIT™ monoclonal assay that was used to detect amphetamine/methamphetamine. Administration of ranitidine in a patient with shock is common, so it could be that our patient received ranitidine from the emergency physician, and the protocol of the emergency team needs to be reviewed. The patient could also have used nose drops or a spray containing antihistamines [2] that could also have led to a false-positive test. Additionally, false-positive test results for methamphetamine have also been reported due to consumption of certain spices [3]. Who knows what kind of spices she may have used cooking Albanian dishes? Clinically, there is another important discrepancy: patients with methamphetamine intoxication usually present with hypertension, while our patient had severe hypotension. This all suggests a false-positive result for methamphetamine, and therefore, narrows my differential diagnosis to anaphylactic shock.

In summary, there were no symptoms or other indications of pre-existing disease before the acute emergency situation that developed spontaneously and within a very short time. Symptoms included vomiting, a hot flash, flickering in the visual field, a macular rash on the abdomen, and ultimately, loss of consciousness, all typical signs of anaphylactic shock. As the clinical status of the patient improved within a few days and was completely reversible, central nervous system causes such as intracranial hemorrhage can be ruled out.

Now we have to consider causes for anaphylaxis, which is a potentially life-threatening systemic state of hypersensitivity. Anaphylactic shock can be induced by allergic hypersensitivity; IgE is often, but not necessarily, involved. Here, however, on the basis of the history, we can exclude allergies to medications, vaccines, food, and insect venoms that could lead to anaphylaxis. This leaves us with only one diagnosis: anaphylactic shock due to antigen-induced hypersensitivity, but what was the responsible antigen? Blood work showed 17 % eosinophils in the differential blood count and an elevated absolute count of eosinophils. The two large cystic lesions in the liver found on ultrasonography are an extremely important finding that leads to the assumption that the patient went into anaphylactic shock due to antigen-induced hypersensitivity in the context of echinococcosis.

Echinococcus granulosus has a worldwide geographical distribution, with the highest prevalence in parts of Eurasia (especially Mediterranean countries), North and East Africa, Australia, and South America [4, 5]. Our patient now lives in Austria but came from Albania, a Mediterranean country between Greece and Montenegro, and regularly visits her home country, which is known to have a high prevalence of echinococcosis; furthermore, a rash like hers is also frequently seen in echinococcosis.

Our patient's serious complication of anaphylactic shock with cystic echinococcosis must have been due to rupture of one of the liver cysts with a subsequent acute hypersensitivity reaction. Other complications of such ruptured cysts have been reported to include peritonitis, pulmonary hydatidosis, cardiac manifestations (including cardiac tamponade), cerebral vascular spasm, ocular cystic lesions, nephrotic syndrome, liver abscess, and myositis.

A diagnostic test for IgG specific for *E. granulosus* or analysis of antigens of *E. granulosus* should be performed to confirm or rule out this diagnosis. A computed tomographic (CT) scan of the abdomen would be useful to evaluate the state of the cystic lesions and would possibly confirm a previous rupture. An anthelmintic such as albendazol should be started immediately, and cystectomy should be considered.

Dr. C. Madl's diagnosis

Anaphylactic shock due to antigen-induced hypersensitivity in the context of infection with *E. granulosus* (cystic echinococcosis), probably induced by rupture of a hydatid cyst in the liver.

Discussion of diagnosis

Dr. K. Harnoncourt: Over the years I have often seen patients with cystic echinococcosis. Hydatid cysts of *E. granulosus* develop as unilocular taut, circular, fluid collections with a clearly thickened cyst wall. Presenting clinical features are highly variable and depend not only on the organ involved but also on the size of the cyst, its position within the organ, the mass effect within the organ, surrounding structures, and complications relating to cyst rupture and secondary infection. Ultrasonography in this case revealed two cystic lesions, but only one had the typical shape of *E. granulosus* cysts; the other one was not taut and circular and did not show the typical thickened cyst wall. This lends strong support to the assumption that this cyst had ruptured and so caused anaphylaxis as diagnosed by Dr. Madl.

Dr. S. Horn: Based on the symptoms, ultrasound images and routine laboratory tests showing inflammation, we also assumed that our patient had an infection with *E. granulosus.* Unfortunately, routine laboratory data are not specific for echinococcosis. Leukocytosis may suggest infection (of the cyst), and eosinophilia and hypergammaglobinemia are present in 25–30 % of all patients infected with *E. granulosus* [6]. We thus ordered a specific immunological test for circulating antibodies (IgG) against echinococcus; *E. granulosus* and *E. multilocularis* antigens in serum were also assessed. The results were positive for both antibodies against *E. multilocularis*

and *E. granulosus* antigen. Western blot was positive for *E. granulosus* but negative for *E. multilocularis*. This test is considered the definitive diagnostic procedure in such cases.

Dr. D. Schiller (by e-mail): My diagnosis is anaphylactic shock caused by rupture of an echinococcal cyst. Such a case was reported in a 25-year-old asylum seeker from Georgia [7].

Dr. R. Maderthaner: Computed axial tomography of the abdomen (biphase, native, and contrast enhanced; radiation exposure: Dose Length Product: 485 mGy·cm) was performed to obtain a sharper image. CT has high sensitivity and specificity for hepatic *E. granulosus* cysts. Intravenous administration of contrast material is not necessary unless complications such as infection and communication with the biliary tree are suspected [8]. CT showed two cystic lesions due to *E. granulosus*. A large cyst in segment VII had a diameter of 7.7 cm and a small daughter cyst (diameter 1.4 cm). There was also an inflammatory, reactive, hypodense, subcapsular, intrahepatic zone adjacent to the cyst. A second cyst was apparent in the left lobe anteriorly (segments II and III, with a maximal diameter of 5.1 cm). The subcapsular lesions presented as slightly calcified (Fig. 2). There was no extrahepatic evidence of cyst rupture, such as free peritoneal fluid or signs of peritonitis. However, it has to be considered that the CT scan was performed 6 days after the initial emergency. Due to the presence of an inflammatory infiltrate in segment VII of the liver, a previous rupture is possible.

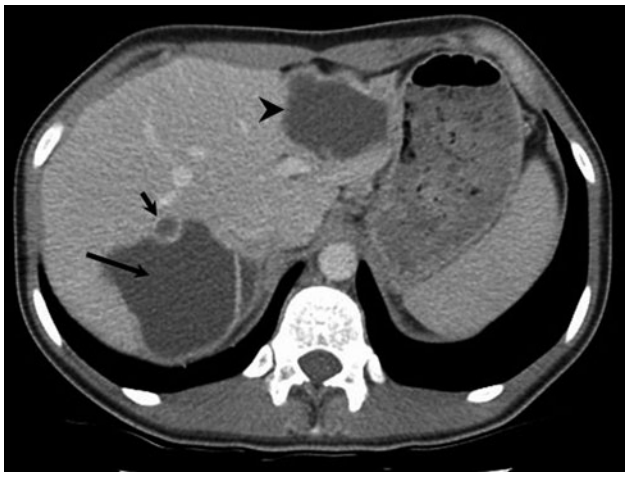

Fig. 2 Abdominal CT scan showing two subcapsular hepatic echinococcal cysts. One is located in segment VII, with a maximal diameter of 7.7 cm (*long arrow)* and intracystic septation and with a small daughter cyst (*short arrow*). An inflammatory, reactive, and hypodense area can be seen between the cyst and the liver surface, possibly the site of rupture 6 days earlier. The second cyst is located in segments II and III (*arrowhead*)

Dr. P. Kornprat: We performed a pericystectomy in segment III of the left lobe and in segments VI, VII, and VIII of the right lobe of the liver (Fig. 3). Due to the size and location of the cyst (extending to the central hepatic vein and vena cava), pericystectomy in the right lobe of the liver was complex and difficult but nonetheless proceeded without complications. Both cysts were resected in toto without rupture. Total cystectomy as in this patient is the ideal procedure to reduce complication and relapse rates in cystic echinococcosis [9].

Surgical procedures for hepatic echinococcus cysts are either limited or radical. Limited methods aim at sterilization and evacuation of cyst content, including the hydatid membrane (hydatidectomy) and partial removal of the cyst. Radical methods aim at complete removal of the cyst with or without hepatic resection [10]. Surgical options include pericystectomy, partial hepatectomy or lobectomy, and open cystectomy (with or without omentoplasty) [11]. The most common technique is total or partial cystectomy [10]. Generally, the aim of surgery is total removal of the cyst, avoiding spillage of the contents and subsequent secondary spread.

Although several surgical techniques are available, there is no consensus as to the best option. Open issues are the role of cyst aspiration and external drainage, hepatic resection, management of the residual cavity, cyst recurrence after surgery, and complications and mortality related to reoperation in recurrent disease [10, 12, 13]. More radical interventions are associated

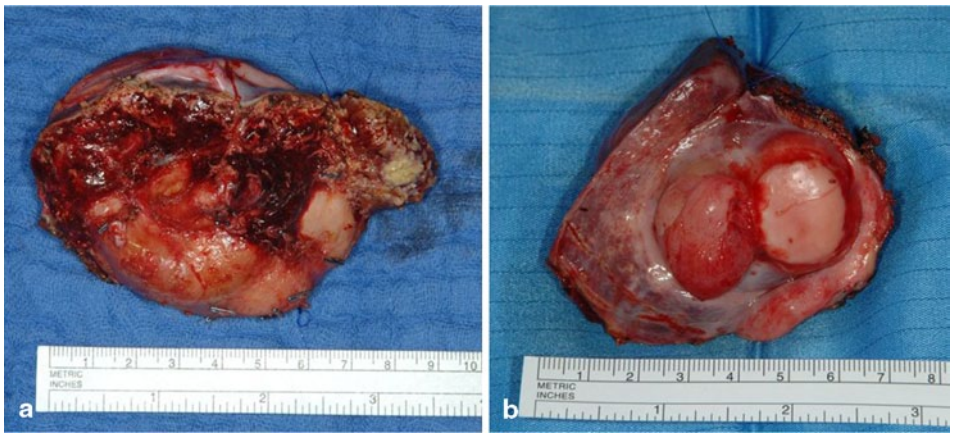

Fig. 3 Operative specimens of *E. granulosus* removed in toto by pericystectomy from the right lobe (segments VI, VII, VIII) (**a**) and the left lobe of the liver (segment III) (**b**)

with higher complication rates and an increased operative risk (morbidity, 32 % [14]; mortality, 0.5–8 % [5, 15]), but show a lower relapse rate [5]. Although immediate cure is claimed for surgical treatment, this is not always achieved, and relapse rates between 2 and 25 % have been reported [16]. Recurrence is usually due to either inadequate cyst removal or previously undetected cysts or spillage. Peri- and postoperative administration of an anthelmintic minimizes the chance of recurrence [5].

In uncomplicated hepatic cysts, surgery is increasingly replaced by other therapeutic strategies such as percutaneous treatment, chemotherapy, and a "watch and wait" approach, but surgery remains the mainstay for treatment of large cysts, those that are superficial and likely to rupture, infected cysts, and those in vital anatomical locations or exerting a substantial mass effect, all in all any complicated cyst. Surgery may be impractical in patients with multiple cysts in several organs [11].

Dr. A. Aigelsreiter: Neither of the resected cysts had ruptured, and, after being fixed with formalin, had a diameter of 5 cm. Within the fibrous host-derived capsule of the cyst resected from the right lobe of the liver, we found collapsed daughter cysts that were also seen on CT scan. Young *E. granulosus* cysts typically present as unilocular cysts, but with time, internal septations and daughter cysts can form. Cyst growth rates vary, ranging from 1 to 5 cm in diameter per year [5].

Hydatid cysts are usually surrounded by a host-produced reactive granulomatous adventitia [5]. In this case, the massive inflammatory (granulomatous) infiltration observed between liver tissue and fibrous capsule strongly suggests a previous rupture of the cyst that then sealed itself off again (Fig. 4). Histologically, the cysts presented as typical hydatid *E. granulosus* cysts consisting of two parasite-derived layers: an inner nucleated germinal layer and an outer acellular laminated layer surrounded by a host-derived fibrous capsule. Brood capsules bud internally from the germinal membrane and produce protoscoleces by asexual division (Figs. 4 and 5). This histology is typical for *E. granulosus*. In contrast, if cysts form in *E. multilocularis* infection, the inner nucleated germinal layer and protoscoleces are missing. This diagnosis of *E. granulosis* cysts is also consistent with the serological findings.

Dr. G.J. Krejs: Dr. Fickert was the patient's attending physician in the intensive care unit and recommended the therapy. Do you have any comments on the case?

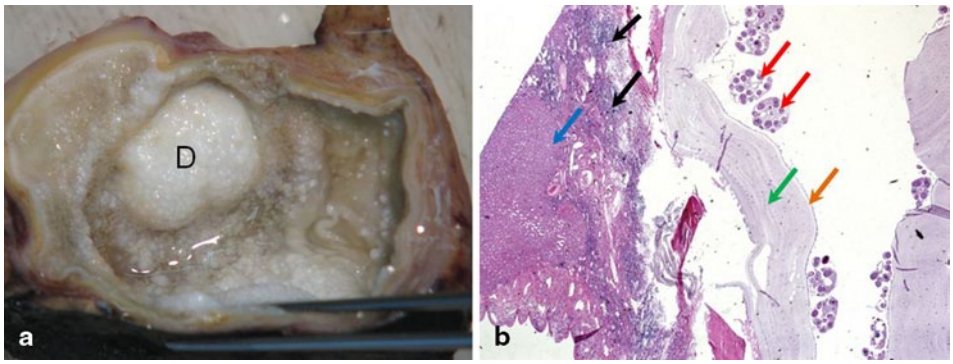

Fig. 4 Hepatic hydatid *E. granulosus* cyst. **a** Macroscopic aspect of opened hydatid cyst from the right lobe of the liver after fixation with formalin. The host-derived fibrous capsule with a white, soft, inner membrane is visible. A daughter cyst (D) impinges on the large cyst. **b** Histology: there is an inflammatory infiltrate (*black arrows*) from the right lobe between the liver parenchyma (*blue arrow*) and the fibrous capsule of the hydatid cyst. The cyst wall is composed of an acellular laminated external layer (*green arrow*) and a thin, germinal nucleated inner layer (*orange arrow*). The cyst contains brood capsules with viable protoscoleces inside (*red arrows*; *hematoxylin–eosin, ×40*)

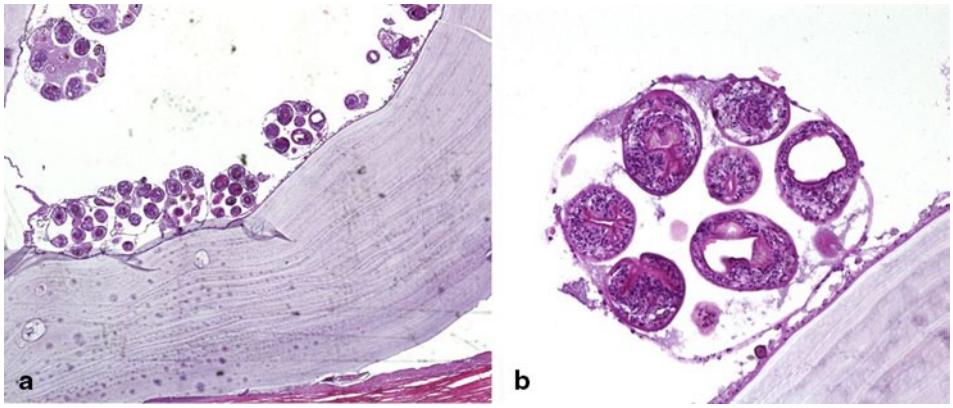

Fig. 5 Hydatid *E. granulosus* cyst wall consisting of two parasite-derived layers: an inner nucleated germinal layer and an outer acellular laminated layer surrounded by a host-derived fibrous capsule. Protoscoleces bud from the germinal membrane (**a**; *hematoxylin–eosin, ×100*) A brood capsule containing protoscoleces (**b**; *hematoxylin–eosin, ×600*)

Table 2 Number of cases of echinococcosis reported in Austria. (*Data provided by the Austrian Ministry of Health* [17])

Year	*E. granulosus*	*E. multilocularis*
2012	5	3
2011	5	3
2010	18	3
2009	4	2

Dr. P. Fickert: As an emergency and intensive care physician and also a gastroenterologist and hepatologist who cared for this patient, I have two general remarks: (1) GCS 3 and hypotension are not consistent with methamphetamine abuse, and there is usually a hypertensive crisis with methamphetamine intoxication, as Dr. Madl already pointed out. (2) Physicians should be aware of the importance of pre- and perioperative anthelmintic therapy for patients with echinococcosis who undergo resection of a cyst. If there are complications during surgery, such as intraoperative rupture of the cyst, the patient is already under chemotherapeutic coverage and the risk for secondary spread of echinococcosis is minimized.

Dr. R. Krause: Echinococcosis is a zoonosis caused by the adult or larval stages of tapeworms (cestodes) belonging to the genus *Echinococcus* (family Taeniidae). Humans are affected as intermediate hosts by larval and cystic stages of echinococcus. The two major species of medical and public health concern are *E. granulosus* and *E. multilocularis*, which cause cystic and alveolar echinococcosis, respectively. While *E. multilocularis* is endemic in the northern hemisphere (central part of Western Europe, parts of the near East, Russia, China, northern Japan, Alaska), *E. granulosus* has a worldwide geographic distribution, with the highest prevalence in the temperate zones, including the Mediterranean countries [4, 5].

In most developed countries, echinococcosis is a disease with very low incidence and prevalence and is found almost exclusively in migrants from endemic regions. In Austria (population 8.5 million), approximately 10 cases of echinococcosis (including infection with *E. granulosus* and *E. multilocularis*) per year are reported (Table 2). In neighboring Bavaria, with a population of 12.5 million, 36 cases of echinococcosis are diagnosed every year [18]. In Austria, game is not usually inspected by veterinarians, while inspection of meat from slaughtered domestic animals is obligatory. In 2011, hydatid cysts of echinococcus were identified in 3 of 5.5 million pigs, in 170 of 615,153 heads of cattle, and in 37 of 127,089 sheep [19]. In Austria, domestic dogs are generally recognized as free of tapeworm, though foxes throughout the country are infected with *E. multilocularis* [19].

Larval infection, i.e. hydatid disease or hydatidosis, is characterized by long-term growth of metacestode (hydatid) cysts (mainly in the liver and lungs) in the intermediate host. Human echinococcosis is caused by the larval stage of echinococcus. In this situation, the human is an aberrant intermediate host; transmission, i.e. infection, can be due to direct fecal–oral contact or indirect transfer of eggs, either through contaminated water and uncooked food or through flies and other arthropods as intermediary vectors [5]. Definitive hosts for the intestinal tapeworm are canines such as dogs and wolves, in which *E. granulosus* resides, attached to the mucosa in the small intestine. Gravid proglottides or eggs are shed in the feces. After ingestion by an intermediate host (e.g. sheep, goat, pig, cattle, horse), the eggs hatch in the small bowel and release oncospheres. Penetration through the mucosa leads to blood-borne delivery to the liver (80 %), lung (20 %), and rarely to other organs, where they develop into a hydatid cyst (metacestode larvae) producing protoscolices. The definitive host is infected by ingestion of cyst-containing organs of infected intermediate hosts. After digestion, protoscoleces evaginate, attach to the intestinal mucosa, develop into adult stages in 32–80 days, and start to shed gravid proglottides and eggs in the feces. As the life cycle of *E. granulosus* relies on carnivores eating infected herbivores, humans are usually a dead-end for the parasite [20, 11] (Fig. 6).

Asymptomatic hepatic cystic echinococcosis is common in endemic regions. Approximately 75 % of infected people may remain free of symptoms [21]. Treatment should be reserved for symptomatic cases or those in which cysts affect vital anatomical structures. Three decades ago, surgery was the only option available to treat cystic echinococcosis; today, advanced alternative treatment modalities increasingly supplement or even replace surgery as the preferred treatment.

The *P*uncture, *A*spiration, *I*njection, *R*easpiration (PAIR) technique was introduced in the mid 1980s [22] and developed as an attractive alternative to surgery. In PAIR, the cyst is punctured under ultrasound guidance, cyst fluid is aspirated, a protoscolicide (e.g. hypertonic saline or 95 % ethanol) is injected, and 15 min later, the cyst content is reaspirated. This technique should only be used in patients with anthelmintic coverage to minimize the risk of secondary cystic echinococcosis and should only be performed by experienced physicians who must be prepared to treat anaphylaxis if there is accidental spillage during the procedure. It must also be certain that there is no connection to the biliary tree, as ethanol may cause severe caustic cholangitis. Combined treatment (PAIR with albendazole) may yield better results than those of either chemotherapy or PAIR alone [23].

Only in a thrid of patients is administration of an anthelmintic, i.e., a benzimidazole compound such as albendazole and mebendazole, is effective as monotherapy. Small (diameter <7 mm), isolated cysts surrounded by minimal reactive adventitia respond best to chemotherapy, whereas complicated cysts with multiple segments or daughter cysts, with a thick adventitial surrounding reaction, or with a calcified capsule are relatively refractory to anthelmintic treatment [5].

According to the World Health Organization (WHO) classification based on ultrasonography (Table 3), our patient's cysts could have been in a state of transition, i.e. probably starting to degenerate, and so could

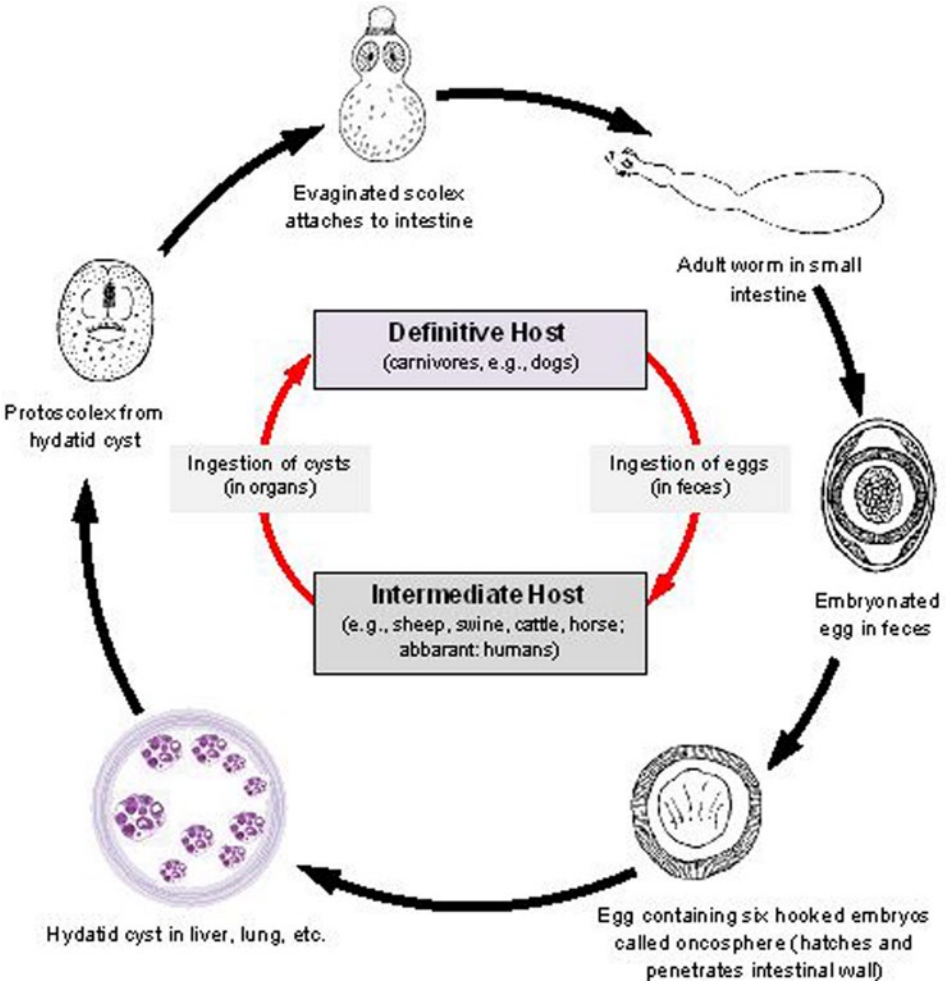

Fig. 6 Life cycle of *E. granulosus* [modified from 20]

Table 3 Classification of hepatic cystic echinococcosis lesions based on ultrasonography [24]

Type	Active	Fertile	Cyst wall	Remarks
CL[a]	Yes	No	Not visible	If cysts are due to cystic echinococcosis, early stage of development
CE1	Yes	Yes	Visible	Unilocular, anechoic or "snow flake" sign
CE2	Yes	Yes	Visible	Normally round or oval, features are pathognomonic
CE2a				Multiseptate and multivesicular, daughter cysts present
CE2b				Cyst septations may produce "wheel-like" structures
CE2c				Daughter cysts may produce a "rosette-like" or "honeycomb-like" structure
CE3	Transitional	Yes	Visible	Transitional stage, cyst is usually starting to degenerate. Degenerative signs: detachment and rupture of membranes, occasionally followed by production of daughter cysts, cysts' form may be less rounded due to decreased intracystic fluid pressure
CE3a				Anechoic content with detached laminated membrane from the cyst wall visible as floating membrane ("water-lily sign")
CE3b				Unilocular cyst that may contain daughter cysts (anechoic) and echoic areas (disrupted membranes/degenerating daughter cysts)
CE4[a]	Inactive	No	Not visible	
CE4a				Heterogeneous hypoechoic or dyshomogeneous degenerative contents, no visible daughter cysts
CE4b				"Ball of wool" sign indicative of degenerating membranes
CE5[a]	Inactive	No	Calcified	Thick, variably calcified wall producing a cone-shaped shadow, usually no viable protoscolices

Cysts' subclassification according to size: small, < 5 cm; medium, 5–10 cm; large, > 10 cm
[a]Further tests required to ascertain a diagnosis of cystic echinococcosis

Table 4 Treatment recommendation according to cyst stage [10, 25]

Type	Size	Preferred treatment	Alternative treatment
CE1	<5 cm	Albendazole alone	PAIR
	>5 cm	Albendazole + PAIR	PAIR
CE2	Any	Albendazole + either modified catheterization or surgery	Modified catheterization
CE3a	<5 cm	Albendazole alone	PAIR
	>5 cm	Albendazole + PAIR	PAIR
CE3b	Any	Albendazole + either modified catheterization or surgery	Modified catheterization
CE4 and CE5	Any	Regarded as inactive, and unless complicated, they should not be treated ("watch and wait")	

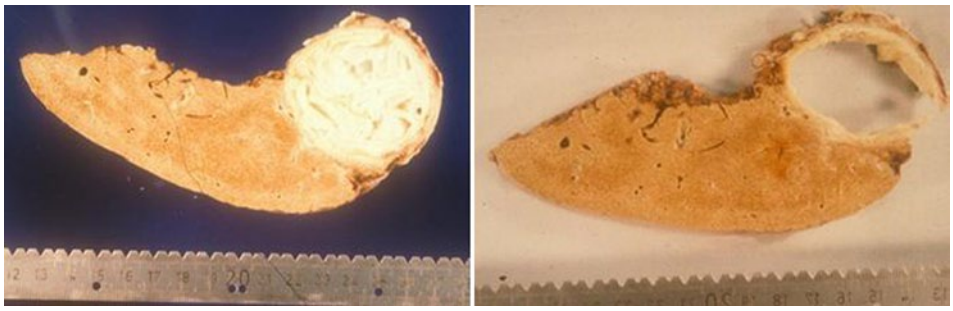

Fig. 7 Formalin-fixed resection specimen of a hydatid cyst in the liver, on the right side after the removal of the internal membranes. (Courtesy of Dr. S. Uranüs)

be classified as type CE3(a–b) (when degenerating, the inner nucleated germinal layer of the cyst detaches and forms a characteristic water-lily sign within the degenerating cyst). The WHO classification for treatment of cystic echinococcosis stratified by cyst stage for uncomplicated liver cysts confirms that surgical intervention was the right therapy for our patient (Table 4). Moreover, surgery maintains its place as the treatment of choice in patients with complicated cysts (e.g., rupture, cystobiliary fistulas, and compression of vital organs) [10, 26], as was present in this case.

In patients with echinococcosis, anthelmintic therapy is important and should start 1 week before surgery and last for at least 4 weeks postoperatively. As our patient's course indicates prior rupture of a cyst, I strongly recommend extending the anthelmintic therapy (400 mg albendazole b.i.d.) to 8 weeks to minimize the risk of secondary cystic echinococcosis. As adverse effects are frequently reported with this therapy, neutrophil and leukocyte counts as well as liver function should be monitored regularly.

Dr. B. Kränke (by e-mail): The high grade of eosinophilia present in the discussed patient should have alerted the consulting dermatologist to a parasite-triggered rash. Eosinophilia is usually not seen in allergic drug-induced skin reactions.

Dr. G. J. Krejs: Review of the protocol of the emergency physician providing the initial prehospital care shows that both an H_1- and H_2-receptor antagonists were given. Ranitidine can thus be considered to have caused the false-positive screening results for methamphetamine. In 48 years of patient care, I have never seen a cyst rupture in echinococcosis. Dr. H.J. Samec from St. Veit on the Glan saw a cyst rupture followed by a very prolonged recovery. Dr. H. Auer, the chief parasitologist in Vienna, comments in an e-mail that he has only heard of one case of rupture. Among 151 clinical-pathological conference (CPC) cases in the past 26 years in this institution, this is the second case of echinococcosis. A picture of the resected cyst of case no. 50 is shown in Fig. 7.

A CPC reported from the Massachusetts General Hospital presented another surgical approach in echinococcosis. A metal funnel with a rim perfused with liquid nitrogen is attached to the cyst, and freezing causes a tight connection. The cyst is then directly opened, and a protoscolicide is administered directly into the cyst before resection [27]. In a recent epidemiological study from the USA, analysis of death certificates showed that two to three persons die every year due to complications of echinococcal infection [28]. Typical magnetic resonance images were recently reported from Dallas, Texas [29]. Doctors all over the world should be made more aware of this disease. Two years after the initial presentation and surgery, our patient continues to be well and healthy, and CT only shows normal surgical changes but no recurrent cyst in the liver.

Final diagnosis

Anaphylactic shock due to rupture of a hydatid cyst in the liver (*E. granulosus*).

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Conflict of interest
All authors declare that there is no conflict of interest.

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