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Research Article

Echinococcus Granulosus Infection and Detection in Human and Animals

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Abstract

Cystic echinococcosis The disease is commonly seen in rural areas where the local population is in close contact with livestock and dogs. The larval stage of Echinococcus granulosus, known as metacestodes, is the cause of cystic echinococcosis (CE), a zoonosis. Despite being classified as a neglected illness by the World Health Organization (WHO), CE is the second most common foodborne parasite disease and, given its potential morbidity and zonal endemicity, continues to be a significant public health concern. One important WHO goal is the prevention and control of CE, particularly from a One Health standpoint. , since the illness impacts the food chain in addition to humans and animals. There is continuing research regarding the precise geographic prevalence of the disease, as there are relatively little recent epidemiological data available due to the fact that not all nations have a CE surveillance strategy, reporting system, or particular management protocols. We have examined and gathered data from national guidelines and numerous medical databases to provide fresh insights on the topic. After certain inclusion and exclusion criteria were applied, only 52 of the 751 research articles that were initially found were included in the analysis. Featured are noteworthy global initiatives that have made important contributions and had a good effect. The available statistics were compared to WHO guidelines on the matter, highlighting the steps that have been taken and those that remain to effectively restrict the disease's spread.

Key words: granulosus echinococcus; zoonosis; cystic echinococcosis

1.Introduction

According to recent investigations, the genus Echinococcus has a well-established taxonomy with 8–10 species, 6 of which are considered harmful to humans: Echinococcus canadensis, Echinococcus ortleppi, Echinococcus vogelii, Echinococcus multilocularis, Echinococcus granulosus sensu stricto, and Echinococcus oligarthr (Knapp et al., 2022) Global zoonotic disease cystic echinococcosis (CE) is brought on by the larval and metacestode stages of Echinococcus granulosus. The zoonotic cestodes of the species complex Echinococcus granulosus sensu lato (Taeniidae: Cestoda) are the cause of cystic echinococcosis (CE), also referred to as hydatid disease or hydatidosis. E. granulosus can have a domestic or sylvatic life cycle, with a carnivore and a herbivore usually involved. People may unintentionally become dead-end hosts and get CE. According to the Foodborne Disease Burden Epidemiology Reference Group of the World Health Organization (WHO), CE costs 184,000 disability-adjusted life years worldwide per year in humans. An estimated $2 billion is lost annually in producing animals due to decreases in carcass weight, milk production, fecundity, and wool/hide production (Tamarozzi et al., 2020). To finish its life cycle, Echinococcus granulosus needs two hosts: a canid definitive host and an intermediate host. E. granulosus eggs excreted in the feces of infected canine hosts are what give humans and other intermediate hosts the illness. The parasite's metacestode develop in intermediate hosts, This is called a hydatid cyst. Humans are not infected by the cysts; only canid definitive hosts are. In the United States, dogs (WHO, 2015) According to estimates, the prevalence of E. granulosus s.l. in Iran is 4.2% in humans, 15.6% in intermediate hosts, and 23.6% in definitive hosts. This illness is endemic in Iran, where many instances of infection are documented each year. Thus, the goal of the current investigation was to ascertain the genotypes and prevalence of E. granulosus s.l. isolated from domestic and stray dogs in the northeastern Iranian

province of Khorasan Razavi (Hejazi et al., 2024). Echinococcus granulosus infections in cats are rare because cats do not belong to the parasite life cycle that is represented by carnivorous and herbivorous animals. However, it can accidentally when consuming food or water tainted with the worm's larvae, particularly when the final host is present (Al-Ardi, 2024).

Literature review

Echinococcus spp. are Taeniidae cestode parasites. There are now eight species recognized in the genus Echinococcus, and only one genotypic cluster (E. canadensis) within it. The genus is worldwide in distribution, with the exception of Antarctica, and these parasites use a predator-prey interaction to spread. Certain Echinococcus species are spread by predator-prey relationships involving domestic animals, whereas other species rely on wildlife lifecycles, although domestic animals may also play a role in transmission (Romig et al., 2017). While all species in the genus have been identified as zoonotic, two species— Senu lato (s.l.) E. granulosus and E. multilocularis represent a serious threat to public health because they are known to cause alveolar echinococcosis (AE) and cystic echinococcosis (CE), respectively. These parasites are two of the most common zoonoses of medical significance, causing sickness in both humans and animals and causing major health and economic issues (Tamarozzi et al., 2020). The parasitic zoonotic Human cystic echinococcosis (CE) is caused by Echinococcus granulosus. Infection occurs when hosts eat Echinococcus eggs, which then develop into the larval (metacestode) stage. Canids are the only animals that the adult-stage parasite can parasitize in terms of host. The hydatid metacestode stage of the parasite is found in domestic ruminants, including sheep, cattle, and camels. The fecal–oral pathway is the means by which transmission from definitive to intermediate hosts happens. Humans are unintentionally dead-end intermediate hosts for the parasite since they are unable to support its life cycle biologically (Tamarozzi et al., 2020).

1.1 Transmission and life cycle

Echinococcosis is spread by a variety of hosts in endemic areas, including intermediate (cattle, pig, sheep, etc.) and definitive (domestic dogs, lions, etc.). Numerous routes of transmission have been identified, including the fecal-oral route, which involves consuming contaminated water, eating raw produce without washing it, and coming into contact with contaminated soil. Other routes of transmission include coming into contact with dogs and livestock (especially ruminants), either directly or by coming into contact with contaminated fur. The fact that the mode of transmission of echinococcosis varies geographically and is influenced by host availability, social and cultural norms, public health awareness, and environmental factors should be especially noted while discussing the disease. Although it has long been believed that direct contact with canine hosts is the primary means of transmission to humans, the association is not strong (Chaâbane-Banaoues et al., 2015). There are endemic areas where there is little to no contact with dogs despite their presence, or where the prevalence of the disease and the number of infected dogs are unrelated. This has led to the conclusion that, even while direct contact isn't usually the cause, soil pollution is probably the main method that the high environmental quantity of Echinococcus eggs contributes to transmission. The eggs go through their larval stage after transmission and become metacestodes. The organs of the intermediary hosts are then where the metacestodes develop; they especially like the liver and lungs. The cycle is completed when adult worms form as a result of dogs consuming these diseased organs Determining the exact moment and mode of transmission in a patient is challenging due to the various routes of transmission and the inability to precisely ascertain the time of infection based on the metacestode itself (Tamarozzi et al., 2020).

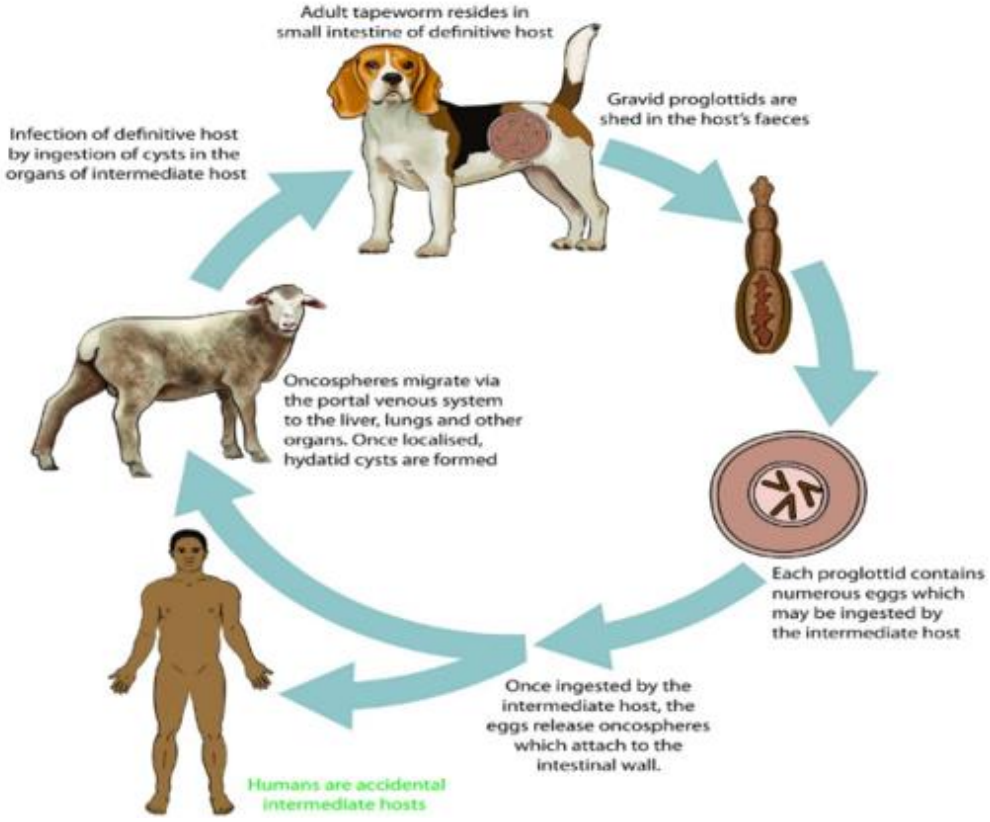

Figure 1: Life cycle of the Echinococcus granulosus.

The eggs release oncospheres, which adhere to the intestinal wall after being consumed by the intermediate host. Through the portal venous system, the oncospheres go from the colon to different organs where they form cysts and daughter cysts. 1-4 The liver accounts for 70% of cases,with the lungs coming in second with 20%. The kidneys, heart, and spleen, peritoneum, CNS , and bones are among the infrequent locations.The growth rate of hidatid cysts is sluggish, ranging from 1 to 10 mm annually; liver cysts grow more slowly than lung cysts (Tsoulfas et al., 2020). The CE cyst is a circular cystic lesion that is characterized by an exterior, acellular, laminated layer called the ectocyst and an interior, germinal layer called the endocyst. Little vesicles known as brood capsules are produced by the inner germinal layer; these capsules divide asexually to produce several protoscolices (Figure 2). The pericyst is a granulomatous, adventitial layer that surrounds the echinococcal laminated layer. This layer, which is frequently observed in imaging investigations, is created by the immunological system of the host to ward off the cystic infection (Calame et al., 2022).

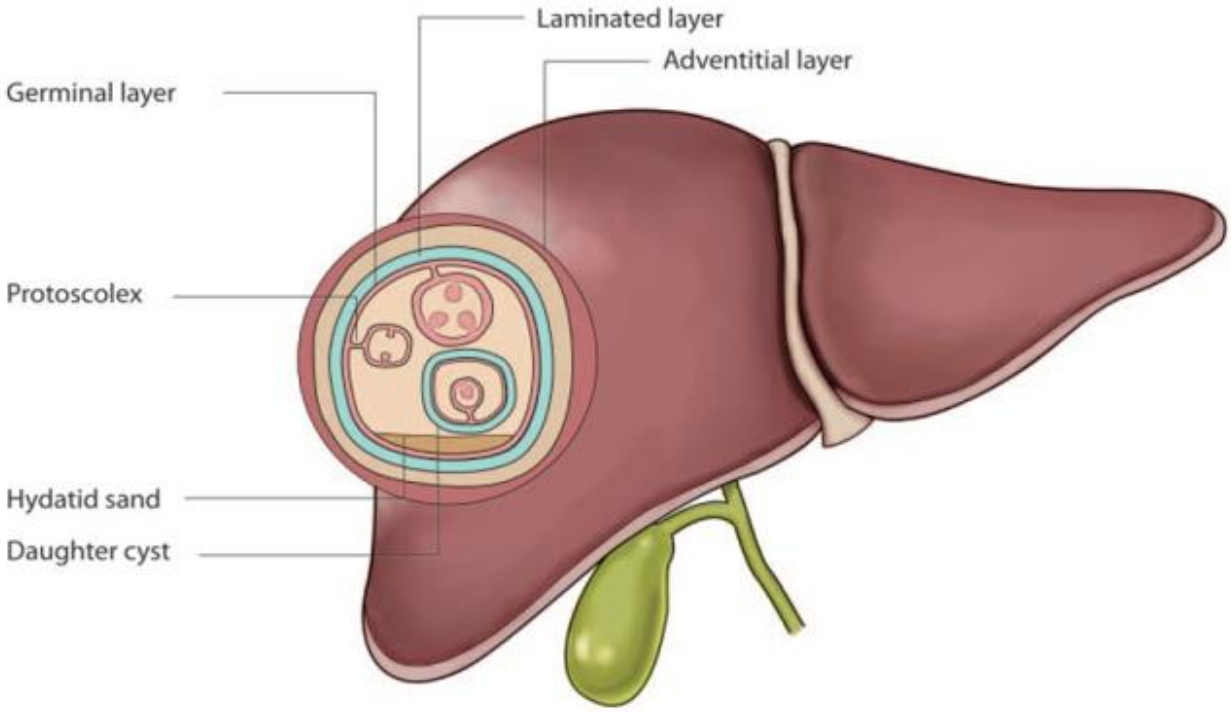

Figure 2: Illustration of the layers of a liver hydatid cyst, comprising the outside (laminated) acellular layer, the inner (germinal) nucleated layer, and the granulomatous layer (adventitial layer), which is created by the host immune system to fend off the cystic infection. Here, the daughter cyst is visible floating inside the primary cyst, and the protoscolex—the future head of the adult worm—is observed budding from the germinal layer. A sonographic finding that combines cystic fluid with protoscolices is called the hydatid sand (Tsoulfas et al., 2020).

1.2 Echinococcus granulosus in livestock

The hydatid cysts in livestock's bodies develop after they consume these eggs through food, water, or vegetables. Hydatidosis is caused by the worm's larvae (cysts) establishing themselves in several organs, including the liver and lungs, and occasionally the brain, heart, and spinal cord of the intermediate host, such as humans, even though infection of carnivores with the worm's mature stage does not pose a specific threat. But a ruptured cyst can also lead to more serious problems, such as internal injuries and trauma. The quantity, size, and location of the developed cysts determine the clinical signs and symptoms of hydatidosis in people and animals. In humans, the disease is relevant because it affects vital organs like the liver and lungs, but in domestic cattle and livestock, it is significant because of the financial loss . Hydatidosis is regarded as one of the main health and economic issues because of the significant financial losses it causes in the cattle and public health sectors (Hosseini-Safa et al., 2016).

1.3 Clinical prestation

In the early stages of the disease, patients may have nonspecific signs and symptoms or be completely asymptomatic. Patients with intra-abdominal CE often show symptoms much later in the course of the illness. This could be because the cysts are growing slowly or because the granulomatous adventitial layer was initially used by the immunological system of the host to seal off the infection with cysts. Cystic development is more rapid in AIDS-positive CE patients, suggesting that an immunological condition may contribute to the progression of the disease (Wen et al., 2019). Hepatic mass, nausea, vomiting, and right hypochondrial discomfort are examples of chronic presenting problems. If the biliary system is compressed by a nearby liver cyst, obstructive jaundice may result. 2,4 Abdominal distension or hepatomegaly may be observed on a physical examination. When a cyst is large (diameter greater than 10 cm) or occupies 70% of the organ volume, signs of liver involvement usually appear) (El Nakeeb et al., 2017). Patients with complex illnesses may also have spontaneous or sporadic cystic rupture following forceful abdominal trauma. Rupture is a potentially lethal complication, despite its uncommon manifestation. If burst cysts communicate with the biliary system, cholangiolitis may be present as a result of biliary tree obstruction. An immune response to a spontaneous intraperitoneal rupture may cause an allergic reaction, the most serious of which is anaphylactic shock. Additionally, live protoscolices may "seed" in the peritoneum as a result of cystic rupture, leading to secondary hydatidosis Rarely the breaking open of a liver cyst by the diaphragm may result in subsequent pulmonary involvement (Kern et al., 2017; Keong et al., 2018).

Diagnosis

To diagnose hepatic CE, a thorough clinical assessment of the patient is essential. This entails a comprehensive history that highlights any

interactions with dogs, wildlife, or trips to endemic regions, as well as a targeted physical examination. When a patient presents with questionable clinical findings, additional laboratory and radiographic tests may be necessary to confirm the diagnosis (Junghanss et al., 2008).

Laboratory investigations

A useful supplement to radiographic studies in the diagnosis of liver hydatid disease is serology for CE. It does have certain drawbacks, though, such as serology's incapacity to differentiate between active and dormant cysts when the ultrasound results are unclear. Western blotting (WB), indirect hemagglutination assay (IHA), and enzyme-linked immunosorbent assay (ELISA) are three laboratory techniques that may be used to diagnose liver CE. For liver-only hydatid involvement, the gold standard serological test is thought to be the enzyme-linked immunosorbent assay (ELISA). E. granulosus-specific antigen is detected by immunoglobulin IgG (G) ELISA, which has a sensitivity of 93.5% and specificity of 89.7%., and 18 Nonetheless, cysts in their early stages wherein the endocyst contains E. granulosus antigens frequently have seronegative findings. The host's immune system's reaction to the parasite infection is then cut off from the antigens. When the cysts calcify in the later stages of the illness process, there is an identical lack of immune response that frequently yields a seronegative result (Keong et al., 2018; Calame et al., 2022). Eighty to one hundred percent sensitivity and the specificity of 88–96% are associated with WB serology for liver CE. The high cost of the test and the sharp decline in sensitivity rates in extra-hepatic illness are the drawbacks of WB.IHA testing has a 90% sensitivity rate, yet if the result is positive, it can stay that way for a number of years. Regular blood tests may reveal vague alterations. Liver function tests are abnormal in only around 40% of individuals. Alkaline phosphatase is frequently increased in cases of derangement, whether or not hyperbilirubinemia is present. In 25–40% of patients, a differential white cell count may reveal eosinophilia (Rashid et al., 2018; Tsoulfas et al., 2020).

Imaging

The preferred diagnostic method for liver CE is ultrasound imaging, which has a sensitivity of 90% to 95%. Liver CE can show up on ultrasonography as a solid mass or as a single, anechoic lens cyst with characteristics of a basic cyst, depending on the situation.20 Real-time imaging reveals a "falling snowflake pattern" as the patient shifts positions. This pattern is made by several echogenic foci that are created when cystic fluid and protoscolices are combined, commonly referred to as "hydatid sand (Kern et al., 2017). "Floating membranes" are the result of the endocyst's separation from the pericyst. Furthermore, a "wheel-spoke" form may result from a multivesicular mother cyst wit Hydatid cysts have been categorized by the World Health Organization Informal Working Groups about the WHO-IWGE Echinococosis according on sonographic characteristics. Cysts are categorized from CE1 to CE5 according to this categorization, which is based on many ultrasonography findings (Table 1). The CE1 and CE2 types correlate with the disease's "active" stage. According to Figure 3, CE3a and CE3b are in the "transitional" stage, whereas CE4 and CE5 are in the "inactive" stage. This method directs additional management in addition to aiding in the general classification of liver hydatid cysts. offspring cysts divided by radiating septae that depict the hydatid sand/matrix and the cyst walls (Fadel et al., 2019).

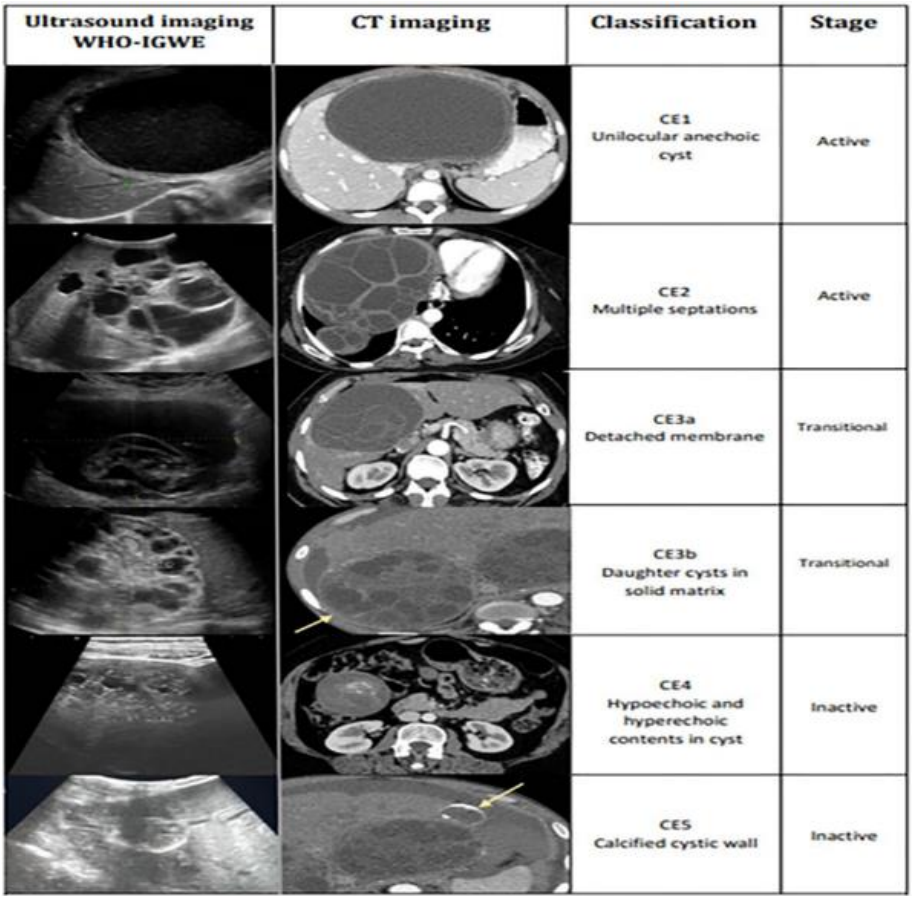

Figure 3: Imaging of liver cystic echinococcosis (Illuri et al., 2018).

Treatment

Routine care of hepatic patients involves considerations of the patient's symptoms, radiological stage, size and location of the cyst(s), presence of comorbidities, and treating physicians' ability (Velasco-Tirado et al., 2018). Medical therapy, a "watch and wait" strategy, percutaneous therapy, and surgical procedures are among the available management options (Tsoulfas et al., 2020).

Watch and wait' approach

With interval ultrasonography monitoring, inactive, degenerating CE4 and all CE5 cysts can be seen without the need for medication or surgery (Mönnink et al., 2021).

Surgery

Options for liver surgery There are two types of CE approaches: radical and conservative (Figure). The radical treatment has drawn criticism for being a severe kind of therapy associated with high morbidity for a relatively benign disease process. It includes total cystectomy and hepatic resection. Additionally, patients must typically have surgery in a specialized hepatobiliary unit due to the high technical difficulty of the procedure.These factors make the conservative strategy with a partial cystectomy more popular. Under albendazole cover, this entails removing the cyst's contents, sterilizing the cavity that remains, and partially resecting the cyst (Vagholkar et al., 2016; Tsoulfas et al., 2020). The hepatic cyst(s) are accessed and exposed by a suitable incision in the partial cystectomy technique. Abdominal swabs soaked in a scolicidal substance (20% hypertonic saline) are used to protect the operative field. In the unlikely case of a spill during the evacuation of cyst-content, this helps to prevent contamination. Next, a selected scolicidal substance is injected into the cyst after it has been pierced and aspirated. Similar to PAIR, if the cyst aspirates bile, There is biliary communication and avoidance of the scolicidal agent. The contents are re-aspirated after 15 minutes, and the cyst is then opened and the endocyst's contents are suctioned out, Subsequently, the cyst is exposed by removing the cyst wall external to the liver parenchyma. A cysto-biliary fistula can be sutured closed if it is discovered during surgery. After the contents of the cystic cavity are removed, the cavity must be completely destroyed using either omentoplasty or capitonnage (Mihmanli et al., 2020). An intraoperative picture of a partial cystectomy with biliary connections for a liver hydatid cyst (Deo et al., 2020)

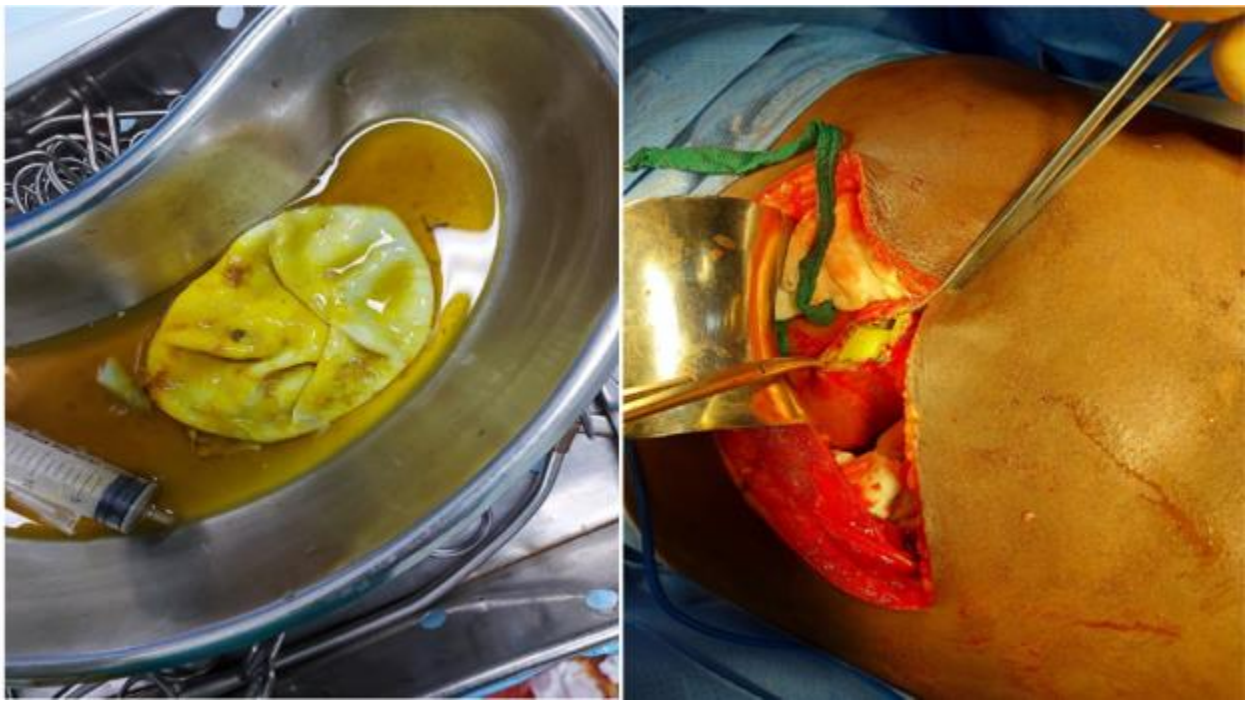

Figure 4: bile-stained floating membrane removed after a partial cystectomy is seen in an intraoperative picture (Tsoulfas et al., 2020).

Preventive strategies

Enacting legislation requiring the use of safe slaughter procedures and efficient canine deworming programs, as observed in New Zealand and Tasmania, can lead to the eradication of CE. By deworming their dogs, other nations including Uruguay, Chile, and Argentina saw a comparable decline in CE. The programs used supervised praziquantel deworming for dogs four to eight times a year (Craig et al., 2017). Vaccinating sheep with the EG95 vaccine is a more recent method to stop the spread of CE. As an extra intervention to lessen CE transmission, it is presently registered for usage in Argentina and China. For poorer nations where CE is typically endemic, this strategy is not feasible due to the high cost of the immunization. Although dog vaccination attempts have been documented, no quantifiable results have been demonstrated in terms of reducing the spread of CE (Wen et al., 2019). It is possible to take preventive actions to help lower the spread of CE. Public health initiatives including routine canine deworming programs, tightly enforced safe slaughter procedures reinforced by local laws, and public awareness campaigns regarding the illness process could help reduce the spread of the disease among endemic populations (Junghanss et al., 2008; Keong et al., 2018).

Conclusion

The true worldwide burden of CE is significantly underestimated since epidemiological data from endemic areas are scarce. Over two-thirds of cases of this disease process involve hepatic infection, making humans the unintentional intermediate hosts. Most symptoms and signs are non-specific, particularly in the early stages of the illness Consequently, especially in

endemic areas, doctors ought to have a low bar for considering the diagnosis in patients with positive serology and suggestive radiological features. The patient's symptoms and the radiographic stage determine the standard course of treatment for liver CE , the cyst's location and size, any problems, and the knowledge of the medical professionals. The WHO's well-established public health protocols must be put into practice in order to reduce the substantial yearly expenses related to CE. Even though the condition is most common in developing nations, increased migration and travel force medical professionals to include CE in their differential diagnosis for any worrisome liver cyst.

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