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Pathophysiology And Epidemiology of Echinococcal Infection: A Review

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Abstract

Cystic echinococcosis (CE) is a cosmopolitan zoonosis representing serious health and economic risks to humans and livestock. The disease is commonly an indolent, asymptomatic and chronic infection by hydatid cyst. Causing both human and veterinary medical costs, as well as constituting loss of production in endemic areas. Prophylactic vaccination has been regarded as one of the most effective approaches for reducing echinococcosis. During the last few decades, much effort has been made to find and characterize protective antigens ofEchinococcus granulosus as well as investigating their immunization potential in different animal models. Therefore, the current study was conducted to systematically review and identify the best candidate antigens for vaccine formulation against cystic echinococcosis. Hum Echinococcosis in man is caused by larval stages of Taenia echinococcus—parasites whose adult form occurs in the intestines of dogs, wild canids and other carnivores (definitive hosts). The domestic and wild herbivores are the intermediate stages of life cycle. Humans encounter the parasite as an incidental intermediate host by ingestion of parasite eggs; typically, when consuming food, water or soil contaminated with feces from infected canids. The two most significant species from a medical point of view are E. granulosus, which causes cystic echinococcosis and is globally widespread (primarily in sheep- and cattle-raising areas), and E. multilocularis, which is responsible for alveolar echinococcosis; the latter occurs mainly in the northern hemisphere. Hexacanth embryos, upon ingestion, penetrate the wall of small intestine and reach the portal circulation from where they are carried to liver predominantly. From there, they can migrate to the lungs, brain or other tissues as cystic lesions. The clinical presentation depends primarily on size, number of cysts and the topography. CNS involvement by E. granulosus is uncommon and accounts for approximately 2% of cases. When it does develop, cysts are uniformly well-defined, round(ovoid) and intraparenchymal, often achieving considerable size with resultant mass effect producing neurologic symptoms or compressing surrounding brain structures is not unusual.

Keywords: Echinococcosis, Echinococcus granulosus, Echinococcus multilocularis

Introduction

Echinococcosis is a highly significant worldwide zoonosis caused by cestodes of Echinococcus genus. It is of both medical (human and animal) concern, and is considered a significant public health and economic problem in endemic areas. Echinococcosis, a neglected tropical disease according to World Health Organization (WHO) criteria, has been classified as such due to the chronic course of this debilitating infection and the challenges associated with diagnosis and its socioeconomic importance especially in pastoralist/remote habitats (Torgerson et al., 2020; Wen et al., 2019). Two species have medical significance: Echinococcus granulosus s.l., the causative organism of cystic echinococcosis (CE), and Echinococcus multilocularis, which causes alveolar echinococcosis (AE) (Romig et al. Each forms slowly growing larval cysts in the liver and lungs. Undiagnosed, these cysts can cause significant morbidity and mortality (Casulli et al., 2022).

One classic One Health issue in which the transmission is particularly related with the interactions between animals, humans and A:01environmental factors is echinococcosis. Definitive hosts are dogs, fox and other carnivorous; livestock and wild herbivores are intermediate hosts. Humans become an accidental intermediate host by ingesting parasite eggs shed in the feces of infected canids (Deplazes et al., 2017). After ingestion, eggs hatch to release oncospheres in the intestine that penetrate the gut mucosa and migrate through the blood stream to organs such as liver, lung or brain where they will grow into metacestodes or hydatid cysts (Cardona & Carmena, 2019). Intensity of transmission is largely determined by the ecology, agricultural practices and human behavior. Traditional husbandry systems, lack of control over slaughterhouses and intimate relationships between man and dog in rural areas appear to favor the maintenance of the life cycle of the bat (Possenti et al., 2016; Nakao et al., 2020).

From both a biological and an immunopathological perspective, CE and AE present with differing mechanisms of tissue injury and immune modulation. Unilocular cysts with thick laminated membranes grow slowly, exerting pressure on surrounding tissues and contributing to fibrosis occurs in CE (Vuitton et al., 2020). AE on the other hand exhibits invasive and proliferative properties simulating those of a malignant tumor, due to exogenous budding of vesicles that invade organs where from they have potential to metastasize itself in extra-hepatic tissues (Eckert & Deplazes, 2019). Echinococcus species escape from host immunity using intricate mechanisms such as releasing immunoregulatory molecules, reducing dendritic cell activity and inducing regulatory T cells, thus maintaining chronic infection and preventing the destruction of host tissues (Díaz et al., 2022; Gottstein et al., 2017).

Echinococcosis is distributed worldwide in almost all continents, with the highest prevalence observed in Central Asia, Middle-East and western China; Africa and South America (Torgerson et al., 2020). It has been estimated that more than one million individuals are infected at the same time, though reporting bias is still large in resource poor areas (Casulli, 2021). CE is geographically widespread, in contrast to AE which is primarily limited to the Northern Hemisphere, including Europe, Russia and some regions of China (Romig et al., 2017). Poverty, poor veterinary infrastructure and environmental contamination continue to play an important role in transmission (Craig et al., 2017). Recent information suggests epidemiological changes in Europe and Asia, with an expansion of wildlife reservoirs and changing climatic conditions promoting the northern distribution range of E. multilocularis (Deplazes et al., 2017; Conraths et al., 2017).

On a clinical basis CE can be asymptomatic for many years, since the cysts gradually enlarge and develop symptoms only when they rupture or compress neighboring structures. Complications include secondary bacterial infection, rupture-induced anaphylaxis or dissemination (Tappe et al., 2019). However,...), however, it most frequently appears as progressive hepatic lesions that can be mistaken for tumor and even extend to lung or brain often making an early diagnosis difficult (Wen et al., 2019). Diagnosis is mostly based on imaging techniques (ultrasound, CT and MRI) along with serological and molecular tests (Larrieu et al., 2019). But in low-resource endemic regions, the challenge is of under-capacity in laboratories and human resource constraints that lead to suboptimal screening. An accurate diagnosis and activity staging of the cyst are crucial in defining the most suitable treatment approach (Vuitton et al., 2020).

The treatment of echinococcosis is based on cyst type, size, location and viability. The main treatment modalities are surgery, percutaneous treatment (PAIR—

puncture, aspiration, injection, and re-aspiration), long-term benzimidazole chemotherapy and a preventive attitude for stable cysts (Velasco-Tirado et al., 2018). For both CE and AE (with antigen confirmation), the recommended drugs of choice are albendazole, though treatment duration is sometimes prolonged (months to years). In experimental studies, it has been demonstrated that the combination of albendazole and praziquantel is useful to accelerate parasite clearance (Walker J et al., 2022). Regarding AE, the only curative option is a radical surgical excision (Hogea et al., 2024), whereas patients that do not benefit of complete resection must receive lifelong antiparasitic treatment. The latter is the focus of ongoing research investigating innovative drug formulations, nanoparticles delivery systems and host-mediated treatments to enhance drug efficacy while shortening treatment courses (Kern et al., 2023).

Comprehensive and prudently planned One Health approach involving veterinary, human and environmental measures is essential in controlling this devastating disease. Programs for regular deworming of dogs with praziquantel, the implementation of safe disposal measures for infected offal and vaccination of livestock (e.g., the EG95 vaccine for sheep) are all beneficial strategies to reduce transmission (Borhani et al., 2024). Education about hygiene, hand washing and safe handling of animals is important for reducing exposure, particularly in rural areas (Craig et al., 2017). The importance of integrated surveillance, community-based engagement and ecosystem-based control was recently underscored in One Health activities designed to move towards sustained reduction in disease (Larrieu et al., 2019). Echinococcosis remains a global public health issue. Chronicity, diagnostic challenges and the strict influence of socioeconomic factors make control measures hard to implement (especially when domestic and sylvatic transmission overlap) (Casulli, 2021).

Epidemiology of Echinococcosis

Echinococcosis remains an important public health problem globally, being broadly distributed but with a widely varying prevalence across different geographical regions. The endemic areas of contaminations are over one hundred countries, especially in populations with pastoralist and agriculturalists lifestyles. Described as a neglected zoonotic disease, echinococcosis makes a significant contribution to the global burden of disease and the total disability adjusted life years (DALYs) for parasitic diseases (Torgerson et al., 2020). Cystic (due to Echinococcus granulosus s.l) and alveolar echinococcosis (AE, due to Echinoccocus multilocularis) are the most important forms of human echinococcosis. The less frequent species, E. vogeli and E. oligarthrus cause polycistic echinococcosis found predominantly in Central and South America (Romig et al., 2017).

Despite the fact that echinococcosis is widely spread worldwide, it is negligently reported. It is estimated that there are over one million infected people globally, most commonly and concentrated in China, Central Asia, the Middle East, South America and East Africa (Casulli, 2021). More than 95% of human cases worldwide are CE, rather than AE, but the latter present a much higher fatality rate when untreated (Wen et al., 2019). Apart from the losses in human health, echinococcosis has serious economic consequences in endemic areas because of loss to livestock, quarantine and condemnation of infected organs, increased veterinary costs—pressures on rural economies of subsistence animal husbandry systems (Craig et al.

Epidemiologic features of echinococcosis are determined by intricate ecological, environmental and socioeconomic contexts which potentiate the transmission between different hosts. The disease continues to be hyperendemic in the Middle East and Eastern Mediterranean (Iraq, Iran, Turkey) because of close animal contacts among humans, dogs & livestock. Seroprevalence studies indicated high endemicity in rural populations and abattoir workers, suggesting on-going transmission due to domestic slaughter practices and poor discard of offal (Sadjjadi, 2019). In Central Asia, particularly in Kyrgyzstan and Kazakhstan, the disease is still considered hyperendemic since degradation of veterinary services after the fall of Soviet Union (Deplazes et al., 2017). CE and AE coexist in China, which is a highly endemic region around the world especially in Tibet, Xinjiang, Sichuan where both domestic and wildlife transmission cycles overlap (Nakao et al., 2020). By the same token, CE is endemic in pastoralist communities of Kenya, Uganda and Ethiopia as traditional ways raising livestock and interaction with dogs are norm (Magambo et al., 2022). In South America, especially in Argentina and the southern of Brazil and Peru, CE is still common, although long-term control programs have significantly

decreased it's incidence at some places (Larrieu & Frider, 2019).

In contrast, the geographical distribution of AE is mainly limited to the Northern Hemisphere. It is enzootic in western China, Mongolia eastward through Russia and into multiple EU countries (Conraths et al., 2017). However, in the last decades E. multilocularis has expanded its distribution in Europe due to ecological changes like increasing of red fox population and urban colonization of wild hosts (Hegglin et al., 2022). Surveillance summary data from Europe show a slow rise in AE cases and emergence of new infections in areas previously believed to be non-endemic (Romig et al., 2017). Sporadic AE cases have been reported in Canada and the northern United States, one of countries of North America continent and are associated to wildlife reservoirs as foxes and coyotes (Gesy et al., 2021).

The cycle of transmission of echinococcosis is based on a complex interplay involving definitive hosts (dogs, foxes and wolves), intermediate hosts (domesticated mammals or wild rodents) and inadverdent humans who act as accidental intermediate host. Humans are infected by ingestion of Echinococcus eggs passed in the feces of infected definitive hosts. The eggs are extremely resistant and can persist for long periods in soil, water or plants, maintaining the cycle of transmission (Vuitton et al., 2020). The domestic cycle occurs when dogs ingest the infected offal, consuming viable cysts and humans become infected by ingestion of food or unclean drinking water (or direct contact with contaminated animals). In sylvatic cycles, wild carnivores and small mammals are reservoirs of the parasite independent of domestic hosts, which complicates eradication programs (Craig et al., 2017).

Human risk factors are poor hygiene, contact with dogs, feeding of raw visceras to animals, home slaughter and absence of veterinary care (Possenti et al., 2016). Echinococcosis remains endemic in low and middle income settings with poor veterinary and public health infrastructures, and limited awareness. The morbidity of the disease reality tends to create a vicious circle of poverty whereby many infected persons are less productive, not to mention the economic drain on household income due to losses from livestock (Casulli 2021).

Adaptations to the environment and human activities also have an impact on transmission dynamics. Increased scale of deforestation, extension of agricultural areas and climate change have occurred on the host distribution area and these phenomena brought changes to ecinococcosis incidence in different geographic zones (Conraths et al., 2017). Some milder climate change conditions and the possible expansion of wildlife habitats led to a northward spread of E. multilocularis in Europe and China (Hegglin et al., 2022). Urbanization has additionally changed disease ecology, e.g., increasing numbers of stray dogs occurring around cities and less formal meat markets, which sustain the possibility of domestic transmission even in non-rural settings (Deplazes et al., 2017). The cross-border migration of infected animals, growth in world trade and human migration due to war or economic pressure has also helped spread parasites (Torgerson et al., 2020).

Surveillance is difficult because of under diagnosis, no standardized reporting of cases and persistence latent of chronic infection (Wen et al., 2019). Many countries endemic for these helminths have insufficient diagnostic ability, resulting in case detection delays and worse burden of disease estimates. Current global initiatives like the “Integrated Global Control of Echinococcosis” program by the WHO stress on upgrading of One-Health-based surveillance systems, improving diagnosis and promoting veterinary–human health linkages (Craig et al., 2017). Molecular diagnostics and genetic mapping of the Echinococcus species have further defined the transmission routes. Genetic studies reveal a significant diversity in E. granulosus with genotype G1 (sheep strain) being the most widespread and so far responsible for the majority of human infections (Nakao et al., 2020). This genetic diversity has significant consequences for virulence, host adaptation and vaccine design. However there are considerable gaps in the epidemiological information, which remain more significant for Africa and South America due to the scarce resources available for a complete surveillance and reporting.

(Magambo et al., 2022).

Pathophysiology of Echinococcosis

The pathophysiology of echinococcosis is an intricate product of biological and immunological events from the ingestion of parasite eggs to their larval form, host immune responses, tissues destruction, and clinical complications. Knowledge about these interrelated mechanisms is crucial for accurate diagnosis, grading of the disease, and planning appropriate therapeutic

measures. After Echinococcus eggs are ingested by an intermediate host (e.g. humans) they hatch in the small intestine and release oncospheres that penetrate the intestinal mucosa and bloodstream. These larvae migrate and establish infection in target organs with the liver, lung, brain, and other tissues being common sites of disease. Following Echinococcus granulosus infection, or cystic echinococcosis (CE), larvae establish metacestodes that evolve as fluid-filled cysts. Each cyst contains a brood sac with protoscoleces, a germinative membrane, an acellular laminated membrane and external adventitial layer metabolically inactive formed from the host's fibrotic reaction. In proteomic analyses hydatid fluids have been shown to carry proteins that are implicated in adhesion, development regulation and host–parasite signaling (Díaz et al., 2022).

Echinococcus multilocularis, the causative agent of AE, however, behaves invasively and does not occur as a distinct cyst. Its metacestodes multiply in an exogenous manner, giving rise to vesicular structures that actively invade hepatic and biliary tissues and hematogenously or lymphatically disseminate to various organs. As the parasitic load increases, AE lesions generally exhibit extensive necrosis, fibrosis and central necrotic cores (WHO, 2019; Strohaeker et al., 2022). The immune system of the host reacts through innate and adaptive means, however, Echinococcus species are equipped with sophisticated evasion mechanisms. The innate immune sensing includes macrophages, NK cells, dendritic cells and Toll like receptors (TLRs) that sense parasite antigens and induce inflammatory cascades (Bakhtiar et al., 2020).

Humoral response of the immune system in echinococcosis is usually theratectic and this may be respon sible for the IL-17 and CD8+ T cells -mediated humoral immune suppression where typical Th1 cytokines such as IFN-g-deficiency will cause an augmented IL-10 (Gonzalez G, et al.,2000).Th2-dominant responses including increase in the expression of connective tissue growth factor or being associated with high levels of IL-4 may interfere with optimal generation, adhesion, followed by blocking the cytotoxic killing effects during Echinococcus infection (Godschi A. M., 1999; Becher L, et al.,2013). The E. granulosus hydatid fluid component, antigen B (AgB), blocks chemotaxis and elastase release from neutrophils and up-regulates a non-protective Th2 response. Parasite-induced arginase further diminishes NO production and macrophage killing. In AE, there is a functional impairment of NK cells: the increased expression of NKG2A receptor is associated with decreased production of IFN-γ and granzyme B, leading to reduced early cytotoxicity in peri-hepatic lesions (Cao et al., 2020). The parasite further influences cell death and the regulation of immune cells. Dysregulated apoptosis, necrosis and autophagy in host cells surrounding cystic lesions have been described. This modulation of pathway-specific cell death is advantageous to the Trypanosoma parasite by minimizing inflammatory clearance. E. granulosus annexins proteins may change cytokines profile of PBMC and downregulate pro-inflammation activation (He et al., 2023).

The invasive behavior of the larval mass is, however, significantly more destructive in AE. The parasite destroys hepatic parenchyma, migrates into bile ducts, and disrupts vascular flow with resultant injury to biliary cells resulting in cholestasis and fibrosis. Stimulation of hepatic stellate cells promotes ECM synthesis and scarring In advanced cases, patients can develop portal hypertension, biliary obstruction, secondary cholangitis, hepatic failure. Advanced AE is often marked by the formation of collateral vessels in a compensatory response to major vascular occlusion (Jiang et al., 2023).

The hosts of Echinococcus In recent years, molecular studies have demonstrated that Echinococcus species actively interfere with host signaling pathways. Receptor tyrosine kinase (RTK) pathways have been suggested to participate in this host–parasite communication at the liver interface and thus represent potential therapeutic targets (. Also, the TGF-β/BMP pathway seems to be essential for parasite growth. In AE, the expression of host and parasite TGF-β/BMP ligands, as well as their cognate receptors are associated with brood capsule formation and protoscolex maturation. Parasite TGF-β-like molecules (e.g., EmACT) can mimic host's cytokines leading to the survival and tissue remodeling (Gao et al., 2024).

Conclusion

Echinococcosis remains a major global health and economic challenge, sustained by intricate ecological, social, and economic interactions. While advances in disease mapping and epidemiological understanding have been achieved, significant gaps persist in surveillance, control measures, and long-term

prevention. The complex transmission dynamics of this zoonosis emphasize the critical importance of implementing continuous and coordinated One-Health strategies that address the interconnectedness of humans, animals, and the environment. Future research should prioritize molecular monitoring, predictive modeling influenced by climatic and ecological changes, and the development of cost-effective, region-specific interventions. The epidemiological pattern of echinococcosis is largely influenced by the interdependence of ecological, cultural, and socioeconomic factors that shape relationships between people, livestock, and wildlife. Along with the improvements in diagnostic tools and disease management programs, echinococcosis remains deeply entrenched in disadvantaged rural populations where poverty, limited healthcare access, and close contact with animals perpetuate transmission.

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