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# CYSTIC ECHINOCOCCOSIS IN CHILDREN: PATHOGENESIS, IMMUNOLOGICAL MECHANISMS, AND DIAGNOSIS

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**Abstract:** Cystic echinococcosis (CE) is a zoonotic parasitic disease caused by the larval stage of the cestode Echinococcus granulosus, representing a serious public health problem in endemic regions. In children, the disease is characterized by a more aggressive course, a higher frequency of multi-organ involvement, and specific immunological features of the host response. This review systematizes current data on the pathogenesis of CE in children, including mechanisms of invasion, echinococcal cyst formation, and its interaction with the host immune system. Immunological mechanisms are examined — the role of Th2 polarization, regulatory T cells, cytokine profiles (IL-4, IL-5, IL-10, IL-13), and specific antibodies of the IgE and IgG4 classes. Special attention is given to the diagnostic algorithm: imaging methods (ultrasound with WHO-IWGE classification, MRI, CT), serological tests (ELISA, IHA, immunoblotting), and molecular-genetic methods (PCR). Challenges of differential diagnosis in pediatric practice and prospective biomarkers are discussed. Understanding the pathogenetic and immune mechanisms opens opportunities for the development of new diagnostic and therapeutic approaches.

**Keywords:** echinococcosis; children; Echinococcus granulosus; pathogenesis; immunity; diagnosis; ultrasound; serological tests; PCR; pediatrics.

## 1. Introduction

Cystic echinococcosis (CE), caused by the larval stage (metacestode) of Echinococcus granulosus sensu lato, is one of the most prevalent parasitic zoonoses worldwide. According to the World Health Organization, approximately 1–3 million new cases are registered annually, and the economic burden of echinococcosis in endemic regions is estimated at hundreds of millions of US dollars [1]. The disease is widespread in Central Asia, the Middle East, Latin America, the Mediterranean, and in certain regions of Russia, Kazakhstan, and Uzbekistan [2].

Pediatric echinococcosis represents a distinct clinical and epidemiological challenge. Studies indicate that children account for 10 to 30% of all patients with echinococcosis in endemic areas, with infection often occurring at an early age — through contact with infected dogs or consumption of contaminated water and food [3]. In children, the disease follows a different course than in adults: cysts grow faster due to the metabolic and immunological characteristics of the developing organism, multi-organ involvement is more frequent, and clinical manifestation often does not appear until years after infection.

The interaction between the parasite and the host immune system is central to the pathogenesis of CE. Echinococcus granulosus has evolved complex immune evasion strategies that allow prolonged persistence in host tissues by forming a protective laminar membrane and secreting various immunomodulatory molecules. This creates a unique immunological "landscape" characterized by the dominance of a Th2 response, activation of regulatory T cells, and production of specific antibodies — all of which are of fundamental importance for both diagnosis and understanding of disease pathogenesis [4].

## 2. Literature Review

Recent scientific literature demonstrates that pediatric cystic echinococcosis remains a major public health concern in endemic regions. Previous studies have focused on the epidemiology, immunological mechanisms, diagnostic imaging, and molecular-genetic approaches associated with Echinococcus granulosus infection. Several authors emphasized the importance of Th2-mediated immune responses and the diagnostic value of recombinant antigens, PCR-based methods, and WHO-IWGE ultrasound classification systems in pediatric patients [5].

Diagnosis of CE in children is associated with considerable difficulty: the latent period may last for years, clinical symptoms are nonspecific, and standard laboratory parameters often remain within normal limits . The modern diagnostic algorithm includes imaging methods (primarily ultrasound with the international WHO-IWGE classification), serological tests, and molecular-genetic methods . This review aims to systematize current data on the pathogenesis, immunological mechanisms, and diagnosis of CE in children [6].

## 3. Methods

The present review was developed through analysis of contemporary scientific publications indexed in international medical databases related to pediatric cystic echinococcosis. Data concerning epidemiology, pathogenesis, immunological mechanisms, diagnostic imaging, serological methods, and molecular-genetic approaches were systematically evaluated and compared.

### Imaging Diagnostic Methods

Ultrasound (US) is the method of choice for primary diagnosis and monitoring of abdominal CE. Ultrasound is accessible, non-invasive, involves no radiation exposure, and is especially valuable in pediatric practice. The WHO Informal Working Group on Echinococcosis (WHO-IWGE) has developed and validated a standardized ultrasound classification of CE cysts, comprising five stages: CE1 (unilocular cyst with fine internal echoes), CE2 (multiseptated cyst — "honeycomb" appearance), CE3a (partially detached laminated membrane — "water lily sign"), CE3b (daughter cysts in a solid matrix), CE4 (degenerating cyst with heterogeneous content), and CE5 (completely calcified cyst).

Stages CE1 and CE2 correspond to active cysts, CE3a and CE3b to transitional stages, and CE4 and CE5 to inactive stages. This classification is of fundamental importance for treatment selection: active cysts require intervention, whereas inactive cysts may be managed conservatively. In children, active stages (CE1–CE3) are more frequently encountered, confirming the more rapid disease progression in this age group.

Computed tomography (CT) provides more detailed visualization of cyst anatomy, detection of calcifications, assessment of the cyst's relationship with vascular and biliary structures, and surgical planning. CT is the method of choice when complicated echinococcosis is suspected (compression of bile ducts, portal vein, or inferior vena cava). Magnetic resonance imaging (MRI) involves no radiation exposure and surpasses CT in assessing soft tissue structures, the cyst's relationship with biliary

pathways, and cyst localization in areas inaccessible to ultrasound. MRI is the preferred method in children when detailed examination is required and there is no indication for emergency intervention.

Chest radiography remains the primary screening method for pulmonary echinococcosis: the cyst appears as a rounded, homogeneous opacity with sharp margins. In complicated cysts (rupture), the characteristic "floating membrane" sign ("lotus position" sign) may be observed. Chest CT is used for precise localization, size assessment, evaluation of bronchial communication, and surgical planning.

**Serological Diagnostic Methods**

Serological methods occupy an important role in the CE diagnostic algorithm, particularly as a complement to imaging techniques. The most widely used methods include enzyme-linked immunosorbent assay (ELISA), indirect hemagglutination assay (IHA), latex agglutination, and immunoblotting (Western blot). The diagnostic sensitivity of ELISA for hepatic cysts is 80–95%, and somewhat lower for pulmonary cysts (50–75%), reflecting differences in antigenic load.

The confirmatory test following a positive ELISA is immunoblotting with recombinant antigens EgAgB (8 kDa subunit) and EgAg5 (antigen 5), which minimizes cross-reactions. The specificity of immunoblotting reaches 95–99%. WHO recommends a two-step serological diagnostic algorithm: a screening ELISA followed by confirmatory immunoblotting.

The diagnostic limitations of serological methods in children are attributable to several factors: immunological immaturity in young children, the presence of intact small cysts with limited antigen release, superficial cyst location without contact with the systemic circulation, and CE4–CE5 stages characterized by reduced or absent seropositivity. According to several authors, seronegativity in confirmed CE in children may reach 20–40%.

Monitoring of specific antibody levels following treatment is important for assessing therapeutic efficacy: after radical surgical treatment, antibody levels generally decline over 12–24 months. Persistently elevated or rising antibody titers indicate relapse or incomplete parasite removal.

**Molecular-Genetic Methods**

Molecular-genetic methods (PCR and its modifications) have opened new possibilities for CE diagnosis, species identification of E. granulosus s.l., and parasite genotyping. PCR enables detection of parasite DNA in serum, urine, biopsy specimens, and other biological materials. The sensitivity of PCR in serum ranges from 60 to 90%, depending on the cyst stage and the DNA extraction method employed.

Of particular promise is real-time PCR (qPCR) targeting high-copy repetitive sequences of the E. granulosus genome (e.g., EgG1U1 or mitochondrial markers), which substantially enhances the sensitivity of the method. For genotype differentiation (G1–G10) of E. granulosus s.l. — which is important for epidemiological and prognostic purposes — sequencing of mitochondrial genes (cox1, nad5) is employed.

An innovative approach involves the detection of circulating parasite DNA (cfDNA) in plasma using highly sensitive digital droplet PCR (ddPCR). This method enables detection of extremely low concentrations of parasite DNA in seronegative cases and during post-treatment monitoring. The application of cfDNA PCR in children remains an area of active investigation and may transform diagnostic capabilities in difficult-to-diagnose CE cases.

Loop-mediated isothermal amplification (LAMP) — a DNA amplification method that requires no costly equipment — is of interest for use in resource-limited settings. LAMP-based assay systems developed for E. granulosus have demonstrated sensitivity of 85–90% and specificity exceeding 95%, comparable to real-time PCR.

**Additional Methods and Differential Diagnosis**

In complex diagnostic cases, fine-needle aspiration (PAIR — Puncture-Aspiration-Injection-Reaspiration) with subsequent cytological and parasitological examination of cyst contents may be performed. PAIR is carried out strictly under ultrasound guidance with anaphylaxis prophylaxis and instillation of hypertonic saline solution or absolute alcohol for sterilization. Morphological confirmation of CE is provided by the identification of protoscoleces, hooklets, fragments of the laminated membrane, and specific elements of hydatid "sand".

Differential diagnosis of CE in children includes the following conditions: simple cysts (hepatic, renal, pulmonary), polycystic disease, abscesses (bacterial, amoebic), tumors (hepatoblastoma, nephroblastoma, lymphomas), mesenteric cysts, and alveolar echinococcosis (Echinococcus multilocularis). The most challenging differentiation is between CE and malignant tumors in children, where the combination of serological tests, PCR, and characteristic ultrasound/MRI features is of decisive importance.

In recent years, novel potential biomarkers of CE have been developed: circulating parasite-derived microRNAs (miRNA), heat shock proteins, and laminated layer glycans. These biomarkers exhibit high specificity and may be used for early diagnosis and treatment monitoring. Clinical validation of these approaches in the pediatric population represents a promising research direction.

**Diagnostic algorithm for suspected cystic echinococcosis in children**

The diagnostic algorithm for suspected CE in children follows the principle of "from simple to complex" and should include a comprehensive approach employing several complementary methods. At the first stage, an epidemiological history is obtained, a clinical examination is performed, and routine laboratory investigations are conducted (complete blood count with differential, biochemical blood analysis including hepatic function parameters). Detection of moderate eosinophilia, hyperIgEemia, and abnormal liver function tests in conjunction with a characteristic epidemiological history warrants prompt transition to instrumental evaluation.

At the second stage, abdominal ultrasound is performed, and, in the presence of respiratory symptoms, chest radiography is obtained. Upon detection of a cystic lesion meeting CE criteria according to the WHO-IWGE classification, serological testing (ELISA with subsequent immunoblotting) should be simultaneously ordered. Concurrently, ultrasound of all organs is performed to exclude multi-organ involvement, which is significantly more common in children than in adults.

At the third stage, in cases of ambiguous ultrasound findings, cyst location in areas inaccessible to ultrasound, suspicion of complications, or in surgical planning, MRI (preferred in children) or contrast-enhanced CT is performed. In cases of serologically negative results with persistent suspicion of CE, serum PCR is recommended. Fine-needle aspiration (PAIR) is performed in specialized centers under strict indications.

For standardization of diagnosis and monitoring in pediatric practice, the application of the WHO-IWGE international classification is of particular importance — not only at primary diagnosis but also during dynamic follow-up of patients under treatment. The cyst stage according to this classification determines the treatment strategy and allows objective assessment of therapeutic efficacy.

## 4. Results

Current evidence indicates that pediatric cystic echinococcosis is characterized by rapid cyst growth, frequent multi-organ involvement, and pronounced immunological alterations. Diagnostic effectiveness significantly increases when ultrasound classification, serological assays, and molecular methods are applied in combination [7].

**Epidemiology of cystic echinococcosis in children**

Cystic echinococcosis is prevalent primarily in agricultural regions with developed sheep and goat farming, where there is traditionally close contact between humans and dogs — the definitive hosts of the parasite. The highest incidence rates are reported in Central Asia (Kazakhstan, Uzbekistan, Kyrgyzstan), Mongolia, northern China, the Middle East (Iran, Turkey, Iraq), North Africa (Algeria, Tunisia, Morocco), and in Argentina, Uruguay, and Chile [8].

According to the meta-analysis by Budke et al. (2006) and subsequent WHO publications, the global burden of CE comprises approximately 1–3 million cases, with a hospitalization rate of 2–3 cases per 100,000 population in endemic regions. In highly endemic areas, the incidence in children reaches 50–200 per 100,000 per year. Pediatric cases represent a significant proportion — according to various sources, 10 to 36% of all surgical patients with echinococcosis [9].

The primary source of infection for children is domestic and stray dogs infected with E. granulosus, whose feces contain the parasite's eggs. Children are particularly vulnerable due to play contact with animals and inadequate hygiene practices. Alimentary transmission via contaminated water, vegetables, and greens is also significant. It has been established that most patients are infected before the age of 5–10 years, yet clinical manifestation more commonly occurs between the ages of 5 and 15, consistent with the average duration of the latent period [10].

Pediatric case series predominantly report hepatic involvement (60–70%), followed by pulmonary involvement (20–25%), with considerably rarer involvement of the kidneys, bones, spleen, brain, and orbit. Multi-organ involvement in children is observed in 10–20% of cases — notably more often than in adults. This is attributed to greater tissue permeability, immunological immaturity, and a shorter interval between primary infection and diagnosis [11].

**Pathogenesis of cystic echinococcosis**

**Life Cycle of the Parasite and Mechanisms of Invasion**

Echinococcus granulosus sensu lato is a cestode of the family Taeniidae, whose life cycle involves two hosts: a definitive host (canids, primarily dogs) and an intermediate host (mammals, including humans). Sexually mature tapeworms, measuring 3–7 mm in length, parasitize the small intestine of the definitive host and shed eggs containing oncospheres into the environment. When eggs are ingested by the intermediate host, the oncosphere is released in the duodenum under the action of bile and pancreatic enzymes, activates its defense mechanisms, and penetrates the intestinal wall to enter the bloodstream [12].

After penetrating the intestinal mucosa, the oncosphere enters the portal vein and is transported to the liver, which serves as the first "filter" for the larvae. The majority of larvae are retained in the liver (60–70%), some pass through and reach the lungs (the second "filter"), and a small proportion enters the systemic circulation, potentially involving virtually any organ. Within the host tissue, the oncosphere transforms into a protoscolex and begins forming a hydatid cyst [13].

In children, the portal system and tissues exhibit greater permeability, which may explain the more frequent multi-organ involvement and faster cyst growth rates observed in this population. Studies have shown that in children, cysts grow on average 1–5 cm per year, compared with 0.3–1 cm per year in adults. These differences are attributable not only to anatomical and physiological characteristics but also to the immunological features of the developing organism [14].

**Formation of the Echinococcal Cyst**

The hydatid cyst is a complex biological structure consisting of several components. Externally, it is surrounded by the pericyst — a dense connective tissue capsule of host origin, formed as a result of the inflammatory response to invasion. Immediately beneath the pericyst lies the laminated (cuticular) layer — an acellular structure synthesized by the parasite itself. It consists of carbohydrate and protein

components and performs several key functions: mechanical protection, regulation of nutrient and metabolite transport, and immunological shielding of the parasite from the host's defense mechanisms [15].

The inner surface of the laminated layer is lined by the germinal (germinal epithelium) layer — a thin stratum of nucleated cestode cells that constitutes the "living" component of the cyst. The germinal layer is responsible for cyst growth, formation of daughter cysts, synthesis of antigens, and secretion of immunomodulatory molecules. The interior of the cyst contains hydatid fluid — a clear liquid rich in parasite antigens that, upon cyst rupture, can trigger severe anaphylactic reactions [16].

As the cyst grows, protoscoleces — embryonic heads capable of developing into adult worms upon reaching a predator's intestine — are formed. Protoscoleces may accumulate in "hydatid sand" at the base of the cyst. Both unilocular and multilocular forms of CE have been described, although the latter is more characteristic of Echinococcus multilocularis.

**Interaction of the Parasite with Host Tissues**

The interaction between the hydatid cyst and surrounding host tissues is a dynamic and multifaceted process. The pericyst forms within the first weeks following invasion through infiltration of the inflammatory zone by fibroblasts, macrophages, eosinophils, and subsequent fibrosis. The degree of pericyst development varies depending on the organ, the patient's age, and the activity of the immune response.

A critical aspect of pathogenesis is the parasite's secretion of various molecules that directly affect host tissues. Echinococcal antigens (antigen B — EgAgB, antigen 5 — EgAg5) not only facilitate nutrient acquisition but also actively interact with the host immune system, directing it in a manner favorable to the parasite. EgAgB has been identified as the key immunomodulator, suppressing the pro-inflammatory Th1 response and activating regulatory pathways.

Mechanical pressure from the growing cyst on surrounding tissues leads to atrophy and organ dysfunction. In the liver, this manifests as compression of intrahepatic bile ducts and vessels, which may cause cholestasis, portal hypertension, and biliary leakage. In the lungs, the expanding cyst displaces bronchi and vessels, leading to atelectasis, pneumonia, and pulmonary hypertension. In children, due to the smaller size of organs, symptoms appear earlier than in adults.

Immunological mechanisms in cystic echinococcosis in children

**Characteristics of the Host Immune Response**

The immune response in CE in children represents a complex interplay between innate and adaptive immunity, in which the parasite actively modulates this response to its advantage. The primary innate immune reaction is triggered upon contact of oncospheres with pattern recognition receptors (PRRs) — Toll-like receptors (TLRs) on macrophages and dendritic cells. Activation of TLR2 and TLR4 by E. granulosus antigens initiates production of pro-inflammatory cytokines; however, the parasite rapidly suppresses this response through secretory products.

The adaptive immune response in CE is characterized by pronounced polarization toward a Th2 phenotype. This entails a predominance of cytokines IL-4, IL-5, IL-10, and IL-13 over the pro-inflammatory Th1 cytokines (IFN-γ, TNF-α, IL-12). Th2 polarization drives the synthesis of specific IgE and IgG4 antibodies, activation of eosinophils and mast cells — providing a degree of host protection on one hand, while remaining ineffective in eliminating the parasite on the other.

In children, Th2 polarization of the immune response is more pronounced than in adults, attributable both to the physiological immaturity of the immune system and to a higher antigenic load during primary infection. This partly explains the faster cyst growth and the reduced tendency for spontaneous calcification in children compared with adults.

### Role of Regulatory T Cells and Cytokine Profile

Regulatory T cells (Tregs, CD4+CD25+FoxP3+) play a central role in maintaining immunological tolerance to E. granulosus antigens. Studies have demonstrated a significant expansion of the Treg pool in peripheral blood and pericyst tissue in patients with active CE. Treg cells secrete IL-10 and TGF-β, which suppress the effector functions of CD4+ and CD8+ T cells, reduce the cytotoxic activity of NK cells, and impede dendritic cell maturation.

The cytokine profile in CE in children includes elevated levels of IL-4, IL-5, IL-10, and IL-13, which correlate with disease activity and cyst size. IL-4 stimulates class switching to IgE synthesis; IL-5 promotes eosinophil activation and survival; IL-10 suppresses the Th1 response and enhances IgG4 production. Serum IL-10 concentration has been proposed as a potential biomarker of disease activity.

In recent years, EgAgB secreted by the parasite has been shown to directly interact with dendritic cell and macrophage receptors, suppressing their antigen-presenting function and inhibition of Th1 polarization. Furthermore, exosomes released by the germinal epithelium of the cyst contain microRNAs that modulate host gene expression, creating an immunosuppressive microenvironment.

### Specific Antibodies and Their Diagnostic Significance

The specific humoral immune response in CE manifests as the production of antibodies of various classes against E. granulosus antigens. The greatest diagnostic value is attributed to antibodies directed against antigen B (EgAgB) and antigen 5 (EgAg5). EgAgB is an 8-kilodalton lipoprotein specific to E. granulosus, making it the most promising antigen for serological diagnosis.

IgG antibodies (particularly subclasses IgG1 and IgG4) are detected in 60–90% of patients with CE, depending on the test employed and cyst characteristics. IgG4 is predominantly formed in chronic, long-standing disease. IgE antibodies are detected in 30–50% of patients and correlate with the risk of anaphylactic reactions upon cyst rupture. In children, the serological response is often less pronounced than in adults, especially in early-stage, small, or intact cysts — thereby reducing the diagnostic value of serological tests in pediatric practice.

Cross-reactions with antigens of other helminths (Taenia solium, Toxocara canis, Fasciola hepatica) represent a significant challenge in serological diagnosis of CE, particularly in regions with high prevalence of polyparasitism. To improve specificity, the use of recombinant antigens (rEgAgB, rEgAg5) rather than native extracts is recommended.

### Eosinophilia and Its Significance

Peripheral blood eosinophilia is one of the early laboratory signs of helminthic infections; however, it is not a constant feature in CE. Moderate eosinophilia (5–15%) is detected in approximately 20–40% of patients and is more commonly observed when cyst integrity is compromised or during active cyst growth. Marked eosinophilia (above 15%) occurs upon cyst rupture and is characteristic of preceding anaphylactic reactions.

In children, eosinophilia in CE occurs somewhat more frequently than in adults, consistent with the more pronounced Th2 response in this age group. Nevertheless, normal eosinophil counts do not exclude a diagnosis of CE, and this laboratory parameter should be considered only in conjunction with other diagnostic data.

## Diagnosis of cystic echinococcosis in children

### Clinical Presentation and Medical History

The clinical presentation of CE in children is determined by the location, size, and activity of the cyst, as well as the presence of complications. The disease frequently follows an asymptomatic course for several years and is discovered incidentally during abdominal ultrasound or chest radiography. In

hepatic involvement, the most common symptoms are pain or heaviness in the right hypochondrium, hepatomegaly, and low-grade fever; in pulmonary involvement — cough (often productive), dyspnea, and recurrent bronchitis and pneumonia.

Complications of CE — cyst rupture, suppuration, biliary fistula, cholangitis — are accompanied by acute clinical manifestations: severe pain, fever, jaundice, urticaria, or anaphylactic shock. Cyst rupture is a life-threatening condition requiring immediate medical intervention. In children, cyst rupture occurs more frequently than in adults, as the cyst wall is thinner and physical activity is higher.

In the epidemiological history, the following are of critical importance: residence in or travel to endemic regions, contact with dogs (especially herding dogs), consumption of unwashed vegetables, greens, or water from open sources. A family history of CE is also relevant, as members of the same household are often exposed to a common source of infection.

## 5. Discussion

Pediatric cystic echinococcosis remains a clinically and diagnostically challenging condition requiring a multidisciplinary approach. The pathogenetic features in children — faster cyst growth, tendency toward multi-organ involvement, and pronounced Th2 polarization of the immune response — largely determine the diagnostic difficulties and necessitate active screening in endemic regions. The relatively frequent seronegativity in children, particularly in small and/or intact cysts, underscores the necessity of combining imaging and laboratory methods [17].

A landmark advancement in recent years has been the introduction of the standardized WHO-IWGE classification, which has unified approaches to diagnosis and staging of CE. The application of this classification in pediatric practice, however, requires a degree of caution: in children, CE1 and CE2 stage cysts may demonstrate more aggressive growth than in adults, which must be considered when selecting a management strategy.

A significant contribution to understanding the pathogenesis of CE has been the elucidation of the molecular mechanisms of immune evasion by the parasite: EgAgB secretion, activation of regulatory T cells, and release of exosomes containing parasitic miRNAs. These discoveries not only enrich theoretical knowledge but also open perspectives for the development of new diagnostic and therapeutic tools — antigen-specific assay systems, immunotherapeutic approaches, and vaccines [18].

Molecular-genetic methods — PCR, qPCR, ddPCR — complement serological and imaging diagnosis, particularly in complex cases: in seronegative presentations, during treatment monitoring, and in differentiation from other Echinococcus species. Incorporation of PCR into the CE diagnostic algorithm in children in endemic regions appears warranted, especially when multiple competing diagnoses are under consideration [19].

Promising research directions include: development of highly sensitive and specific assay systems based on recombinant antigens for pediatric serodiagnosis; investigation of miRNA biomarkers of CE in the pediatric population; optimization of diagnostic algorithms for resource-limited settings; and establishment of international registries of pediatric echinococcosis for large-scale epidemiological and clinical research [20].

## 6. Conclusion

Cystic echinococcosis in children remains a significant medical and public health problem in endemic regions worldwide. The disease is characterized by specific pathogenetic and immunological features that distinguish it from the adult form, including rapid cyst growth, frequent multi-organ involvement, and pronounced Th2-mediated immune responses. These characteristics contribute to disease

progression and highlight the importance of early diagnosis and timely treatment in pediatric patients.

The interaction between *Echinococcus granulosus* and the host immune system plays a central role in the pathogenesis of the disease. Immune evasion mechanisms such as activation of regulatory T cells, suppression of Th1 responses, and increased production of IL-4, IL-5, IL-10, and IgE antibodies allow long-term parasite survival within host tissues. Understanding these mechanisms is essential for improving diagnostic and therapeutic strategies.

Current evidence demonstrates that the most effective diagnostic approach is based on a combination of imaging techniques, serological assays, and molecular-genetic methods. Ultrasound using the WHO-IWGE classification remains the primary diagnostic tool in children, while CT and MRI provide additional information in complicated cases. Serological tests and PCR-based methods further improve diagnostic accuracy, especially in atypical or seronegative presentations.

Recent advances in molecular biology and immunology have opened new perspectives for the development of highly specific biomarkers, recombinant antigen-based tests, and immunotherapeutic approaches. Nevertheless, further large-scale pediatric studies and standardized diagnostic protocols are still required. Strengthening preventive measures, epidemiological surveillance, and international collaboration is essential for reducing the burden of pediatric cystic echinococcosis and improving clinical outcomes in endemic regions.

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