MODERN MEDICINE AND PRACTICE
https://inovatus.es/index.php/ejmmp
EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE
Vol. 6 No. 5 (May - 2026) EJMMP ISSN: 2795-921X

Article

ASCARIASIS: CONTEMPORARY PERSPECTIVES ON EPIDEMIOLOGY,PATHOGENESIS, CLINICAL PRESENTATION, DIAGNOSIS, AND MANAGEMENT

Munisa Furkatovna Aslamova
Samarkand State Medical University, Samarkand, Republic of Uzbekistan

Abstract: Ascariasis is among the most prevalent parasitic infections in humans, caused by the nematode Ascaris lumbricoides. According to the World Health Organization, approximately 800 million people are currently infected worldwide, and the annual burden attributed to this disease runs into millions of disability-adjusted life years (DALYs). The condition imposes a particularly heavy toll on children, in whom severe infections are associated with nutritional deficiency, impaired physical and cognitive development, and diminished academic performance. This article provides a systematic review of current knowledge on the epidemiology, etiology, pathogenesis, clinical features, diagnosis, treatment, and prevention of ascariasis, drawing on up-to-date WHO guidelines and recent scientific literature. Special attention is given to disease distribution in the Republic of Uzbekistan and the broader Central Asian region, as well as to contemporary strategies for antiparasitic control programs.

Keyword: Ascariasis, Ascaris Lumbricoides, Soil-Transmitted Helminths, Nematodes, Helminth Pathogenesis, Löffler's Syndrome, Stool Microscopy, Albendazole, Mebendazole, Pyrantel, Mass Drug Administration, Neglected Tropical Diseases, Helminth Prevention, Uzbekistan

Introduction

Parasitic diseases occupy a distinct place in the global burden of infectious and non-infectious illness. Among them, soil-transmitted helminthiases represent some of the most widespread human infections, contributing substantially to the worldwide disease burden. This group of conditions — caused by nematodes that require a period of development in soil — includes ascariasis (*Ascaris lumbricoides*), trichuriasis (*Trichuris trichiura*), and hookworm infections (*Ancylostoma duodenale*, *Necator americanus*) [1].

Ascaris lumbricoides is often described as the "queen" of soil-transmitted helminths: it is the most prevalent of all human helminthiases and the dominant geohelminth in the majority of endemic countries. The causative organism is the largest parasitic worm known to infect the human intestine. Adult females reach 20–45 cm in length, while males measure 15–25 cm. A single female can produce up to 200,000 eggs per day, generating extensive environmental contamination even when only a small number of mature worms are present in the host [2].

The global reach of ascariasis, its predilection for children, and its potential to cause severe and life-threatening complications — including intestinal obstruction, biliary ascariasis, and perforative peritonitis — have led to its classification as a priority Neglected Tropical Disease (NTD), combating which is one of WHO's core mandates.

In Central Asia, including the Republic of Uzbekistan, ascariasis remains a significant public health concern. This is due to favorable climatic conditions for egg survival in soil, the widespread use of organic fertilizers

in agriculture, and persistent deficiencies in water supply and sanitation infrastructure in certain regions. According to the Republican Center for Sanitary-Epidemiological Surveillance, ascariasis is consistently recorded across several oblasts, with the highest rates in Samarkand, Fergana, Andijan, Namangan, and Surkhandarya regions [3].

The purpose of this review is to synthesize and critically appraise current evidence on ascariasis — from the molecular mechanisms of pathogenesis to the practical dimensions of diagnosis, treatment, and prevention — with a specific emphasis on applicability within the Central Asian context.

Materials and Methods

Etiology and biology of the causative organism

The causative agent of ascariasis — *Ascaris lumbricoides* Linnaeus, 1758 — belongs to the phylum Nematoda, class Secernentea, order Ascaridida, family Ascarididae. The parasite is gonochoristic (having separate sexes) and oviparous. Sexual dimorphism is pronounced: females (20–45 cm × 3–6 mm) are considerably larger than males (15–25 cm × 2–4 mm). The posterior end of the male is ventrally curved and bears copulatory spicules.

The eggs of *A. lumbricoides* are highly resistant to environmental stressors. Fertilized eggs (50–70 × 40–50 μm) possess a multilayered shell consisting of an outer mammillated protein coat, an intermediate glycoprotein membrane, and an inner lipid layer — the latter accounting for their exceptional resistance to chemical and physical agents. Eggs withstand most common disinfectants, including chlorine at concentrations typically used for water treatment. However, they are rapidly inactivated by heating to 60°C and are killed instantaneously by boiling.

From an epidemiological standpoint, it is important to distinguish between fertilized and unfertilized eggs: only fertilized eggs that have undergone maturation in soil are capable of causing infection. Under optimal conditions (temperature 25–30°C, adequate moisture, and oxygen availability), eggs develop into the infective stage within 9–15 days. Development is arrested below 12°C and above 40°C, and also under desiccating conditions. In temperate climates, eggs can remain viable in soil for 5–7 years; in tropical settings, viability persists for several years, while hot, arid conditions accelerate inactivation.

It is also worth noting *Ascaris suum*, the swine ascarid, which is morphologically nearly identical to the human species. Human infection with *A. suum* has been documented; however, larvae typically fail to mature in the human host, though they may provoke a pulmonary syndrome (visceral larva migrans).

Life cycle of ascaris lumbricoides

The life cycle of *A. lumbricoides* is direct, requiring no intermediate host, and proceeds through the following sequential stages:

- **Egg excretion into the environment.** Mature female worms residing in the small intestine deposit eggs that are passed with feces into the external environment. A single female produces between 100,000 and 250,000 eggs per day. At this stage, the eggs are not yet infective.
- **Egg maturation in soil.** In soil, under favorable conditions, an infective second-stage larva (L2) develops within the egg. Depending on temperature and humidity, this process takes 9 to 40 days.
- **Ingestion of infective eggs.** Humans become infected by consuming water or food — particularly raw vegetables, fruits, and herbs — contaminated with embryonated eggs, or through inadequate hand hygiene. In the stomach and duodenum, digestive secretions dissolve the egg shell, releasing the larva.
- **Tissue migration (larval phase).** L2 larvae penetrate the small intestinal wall, enter the bloodstream, and are carried hematogenously to the liver (via the portal vein), then to the right side of the heart and into the pulmonary capillaries. In the lungs, larvae (now L3) rupture the capillary wall, enter the alveolar space, molt to L4, ascend the bronchioles and bronchi to the trachea, are coughed up, and swallowed. The migratory phase spans approximately 14–21 days.
- **Intestinal phase.** Upon re-entering the small intestine, L4 larvae undergo a final molt and mature into adult worms. Females begin laying eggs 60–75 days after initial infection. The lifespan of adult ascarids is 12–18 months, after which they die and are expelled with feces.

Results

Epidemiology

Ascariasis has a ubiquitous global distribution, with the highest burden in tropical and subtropical regions. According to a systematic review by Pullan et al. (2014), approximately 819 million people worldwide are infected with *A. lumbricoides*, of whom some 221 million experience significant clinical consequences. The global disease burden attributable to ascariasis is estimated at 1.31 million DALYs annually [4].

The geographic distribution of ascariasis is uneven. The highest prevalence is recorded in South and Southeast Asia (India, Bangladesh, Myanmar, Indonesia, Vietnam), sub-Saharan Africa (Nigeria, Ethiopia, the Democratic Republic of Congo), and Latin America (Brazil, Peru, Colombia), where infection rates in rural communities can reach 40–80%.

In the Central Asian region, ascariasis has historically been a leading soil-transmitted helminthiasis. The Republic of Uzbekistan, situated in a continental climate zone with long, hot summers, provides favorable conditions for egg survival in soil — particularly in irrigated areas with high soil moisture. The highest infection rates are recorded in rural communities of the Samarkand and Fergana regions and in Surkhandarya Oblast [5]. Official data from the Ministry of Health of the Republic of Uzbekistan document thousands of cases annually, though true prevalence substantially exceeds reported figures due to the high proportion of asymptomatic infections and limited diagnostic coverage.

Epidemiologically, children are the most heavily affected group. The peak intensity of infection occurs at ages 2–10 years, driven by geophagy, soil play, underdeveloped hygiene practices, and attendance at preschool facilities. Although adults in highly endemic areas also carry significant worm burdens, infection intensity among them is generally lower [6].

The seasonality of ascariasis is moderate. In Uzbekistan, the peak transmission period falls in spring and summer (April–September), when soil temperature and humidity most favor egg development, agricultural activity intensifies, and consumption of raw, unwashed vegetables and herbs rises sharply. Nonetheless, detection of the disease through stool surveys is relatively uniform throughout the year, reflecting persistent, ongoing carriage [7].

Key risk factors driving the epidemiological burden of ascariasis include the absence or improper use of latrines, the application of untreated human feces as organic fertilizer, consumption of raw water from open sources, poor hand hygiene — especially among children — eating raw produce without adequate washing, and overcrowded living conditions.

Pathogenesis

The pathogenesis of ascariasis is determined by the stage of disease and reflects a combination of mechanical, toxic, sensitizing, and nutritional effects of the parasite on the host [8].

Migratory Phase

Mechanical tissue damage caused by migrating larvae is the initial step in pathogenesis. As larvae penetrate the small intestinal wall, they produce pinpoint hemorrhages and foci of inflammation. In the liver, they form transient micronecro-ses surrounded by eosinophilic infiltrates. The most pronounced pathological changes occur in the lungs: rupture of capillary walls by larvae produces small hemorrhages into the pulmonary parenchyma, and larval entry into the alveolar lumen triggers an accumulation of eosinophils, neutrophils, and macrophages — resulting in eosinophilic pneumonitis, or Löffler's syndrome [9].

The immune response of the host plays a central role in this phase. Excretory-secretory antigens of the larvae — primarily C-type lectin-binding proteins, glycoproteins, and proteases — activate a Th2-polarized immune response, stimulating IL-4, IL-5, and IL-13 production. This cascade leads to B-lymphocyte activation, IgE and IgG4 synthesis, and recruitment of mast cells, basophils, and eosinophils. Peripheral blood eosinophilia is the hallmark hematologic finding of the migratory phase. Pronounced

sensitization of the host accounts for the development of urticaria, angioedema, bronchospasm, and rhinoconjunctivitis.

Intestinal Phase

In the chronic intestinal phase, the predominant effects are mechanical, toxic, and nutritional, exerted by adult worms. Mechanical effects include irritation of intestinal mucosal receptors, disruption of peristalsis, reflex dyskinesia of the biliary tract, and — in heavy infections — intestinal obstruction.

Toxic effects are mediated through the excretory-secretory products of adult worms, including ascarone, ascaridol, histamine-like substances, and proteolytic enzymes. These compounds inhibit the host's digestive enzymes (trypsin, chymotrypsin, pepsin) and impair the absorption of proteins, fats, carbohydrates, and vitamins. Notably, impaired absorption of vitamins A, C, B2, and folic acid compounds the nutritional deficiency associated with the infection [10].

Nutritional effects result from direct consumption of host nutrients: *A. lumbricoides* feeds on semi-digested food contents, intestinal epithelium, and blood. In heavy infestations, parasites can consume significant quantities of protein (estimated at 4–8 g per worm per day), causing hypoproteinemia, hypoalbuminemia, and edema in children. Nutritional impairment is the key mechanism linking ascariasis to stunted growth, cognitive decline, and immune suppression.

The immunosuppressive effect of ascarids deserves specific attention. Excretory-secretory products modulate the host's immune responses, reducing the efficacy of vaccination against measles and cholera and increasing susceptibility to bacterial and viral co-infections. Evidence indicates that deworming children improves their vaccine immunogenicity [11].

Clinical features

The clinical manifestations of ascariasis are determined by the phase of the infection, its intensity, the patient's age, and their immune status. In the majority of cases — particularly in light infections — the disease is subclinical or minimally symptomatic.

Migratory Phase (Early Ascariasis, Larval Phase)

This phase develops 2–4 weeks after infection and persists for 2–4 weeks. Clinically, it comprises several distinct syndromes.

Pulmonary syndrome (Löffler's syndrome) is the most characteristic manifestation of the migratory phase. Patients report a dry or minimally productive cough — often paroxysmal — exertional or resting dyspnea, chest discomfort, and low-grade fever, which may occasionally rise to 38–39°C. Auscultation reveals dry and moist rales with crepitation. Chest radiography demonstrates transient, "flitting" eosinophilic infiltrates of rounded or irregular shape with indistinct margins, ranging from 1 cm to several centimeters in diameter, typically bilateral and resolving spontaneously within 1–2 weeks without treatment. In severe cases, the presentation progresses to acute eosinophilic pneumonitis with respiratory failure.

Allergic syndrome appears concurrently with or slightly ahead of pulmonary symptoms, manifesting as generalized urticaria, angioedema, and allergic rhinoconjunctivitis. Exacerbation of atopic dermatitis and bronchial asthma may occur in predisposed individuals. These allergic reactions are driven by massive IgE production in response to larval antigens.

Hematologic findings in the migratory phase are characterized by pronounced eosinophilia (20–60%, occasionally reaching 70–80% of the differential count) and moderate leukocytosis, along with elevated total IgE. These abnormalities normalize as larval migration concludes.

Intestinal Phase (Chronic Ascariasis)

This phase develops 2–3 months after infection and persists throughout the lifespan of the adult worms (12–18 months). Light infections (fewer than 10 worms) are often asymptomatic or associated with minimal complaints. Moderate infections (10–100 worms) are dominated by gastrointestinal symptoms: abdominal pain, nausea, vomiting, anorexia, irregular bowel habits, and flatulence. Heavy infections

(more than 100 worms) lead to pronounced protein-energy malnutrition in children.

Complications

The most serious complication is ascariasis-related intestinal obstruction, an obstructive ileus caused by a mass of worms in the lumen of the ileum or cecum. Biliary ascariasis occurs when worms migrate through the sphincter of Oddi into the common bile duct, gallbladder, or intrahepatic bile ducts. Ascariasis-related pancreatitis develops when worms penetrate the pancreatic duct of Wirsung.

Discussion

Diagnosis

The diagnosis of ascariasis requires a comprehensive approach encompassing a thorough epidemiological and clinical history, physical examination, and targeted laboratory and imaging investigations.

Parasitological Methods

Stool examination is the cornerstone of diagnosis in the intestinal phase. Stool microscopy allows direct identification of *A. lumbricoides* eggs. The direct wet smear is the simplest and most widely used method in routine practice, with a sensitivity of approximately 30–40% per single examination, rising considerably with three consecutive tests. The Fülleborn flotation method, based on the flotation of helminth eggs in saturated sodium chloride solution, yields a sensitivity of 60–75%. The Kato-Katz technique is the WHO-recommended gold standard for quantitative stool microscopy, used in epidemiological surveys [12].

Laboratory Investigations

A complete blood count in the migratory phase shows a characteristic picture: eosinophilia (20–60%), moderate leukocytosis, and occasionally mild anemia. In the intestinal phase, the blood count may be normal or show mild eosinophilia (5–15%).

Serological methods — enzyme-linked immunosorbent assay (ELISA) for detection of specific anti–*A. lumbricoides* antibodies (IgG, IgM, IgE) — offer a sensitivity of 70–90% and a specificity of 85–95%.

Molecular methods (PCR-based diagnosis) can identify *A. lumbricoides* DNA in stool samples with high sensitivity (>95%) and specificity. At present, PCR is used primarily in research settings [13].

Imaging

Imaging modalities are employed primarily in complicated disease. Abdominal ultrasonography can detect worms in the biliary tree. Plain radiography with contrast is used in the evaluation of suspected intestinal obstruction. Esophagogastroduodenoscopy allows direct visualization and extraction of worms when they are present in the upper gastrointestinal tract.

Treatment

Management of ascariasis is comprehensive and encompasses etiotropic (anthelmintic) therapy, pathogenetic and supportive treatment, and management of complications [14].

Anthelmintic Therapy

Albendazole — a benzimidazole carbamate derivative — is the first-line agent according to WHO recommendations (2017). Its mechanism of action involves irreversible inhibition of tubulin polymerization in parasite cells. Dosing: adults and children over 2 years, 400 mg as a single oral dose. Efficacy against *A. lumbricoides*: 95–100%.

Mebendazole — also a benzimidazole derivative — is dosed at 100 mg twice daily for 3 days or 500 mg as a single dose. Single-dose efficacy: 95–100%. The drug is minimally absorbed from the gastrointestinal tract.

Pyrantel pamoate — a cholinomimetic agent that acts as a nicotinic acetylcholine receptor agonist in nematodes, causing depolarizing neuromuscular blockade. Dosing: 10 mg/kg as a single dose. Efficacy: 85–95%. Considered the agent of choice in pregnant women and infants.

Levamisole (Decaris) — an imidazothiazole compound that activates ganglionic receptors in nematodes, inducing spastic paralysis. Dosing: 150 mg as a single dose in adults; 2.5 mg/kg in children. Also possesses immunomodulatory properties.

Pathogenetic and Supportive Treatment

During the migratory phase, antihistamines are indicated; bronchodilators are given for bronchospasm; and

a short course of systemic corticosteroids may be warranted in severe eosinophilic pneumonitis. In patients with nutritional deficiency, a complete, nutrient-rich diet is prescribed, along with vitamin supplementation and iron preparations for associated anemia.

Management of Complicated Ascariasis

Intestinal obstruction may be managed conservatively in the early stages; surgical intervention is required when conservative measures fail. Biliary ascariasis is treated endoscopically via ERCP; surgical management with mandatory antibiotic coverage is indicated when endoscopic treatment is unsuccessful.

Prevention and control of ascariasis

The prevention and control strategy for ascariasis is multifaceted, encompassing chemoprophylaxis, environmental sanitation, health education, and epidemiological surveillance.

Mass Drug Administration (MDA) is the cornerstone of the WHO's global strategy for the control of soil-transmitted helminthiases. MDA programs involve annual preventive administration of albendazole (400 mg) or mebendazole (500 mg) to all pre-school and school-aged children (ages 1–14). WHO recommends annual MDA when community prevalence is 20–50%, and biannual MDA when prevalence exceeds 50%.

WASH programs (Water, Sanitation, and Hygiene) — interventions to improve water supply, sanitation, and hygiene practices — represent the most effective long-term tool for controlling soil-transmitted helminthiases. It is the implementation of WASH programs, rather than drug-based prophylaxis alone, that constitutes the decisive factor in the eventual elimination of these diseases.

Health education is an indispensable component of any helminthiasis prevention program. School- and preschool-based educational initiatives (the "Healthy Schools" approach) have proven particularly effective in changing the knowledge, attitudes, and practices of target populations [15].

Food safety measures recommended for the inactivation of helminth eggs on raw produce include: thorough washing under running water, treatment with a dilute acetic acid solution (1–2%), and heat processing. Standard chlorination at concentrations used in municipal water treatment is insufficient to kill ascarid eggs; boiled or adequately filtered water is recommended for drinking.

Conclusion

Ascariasis remains one of the most pressing challenges in medical parasitology and tropical medicine globally. Despite considerable progress in understanding the biology of the causative organism, the mechanisms of pathogenesis, and the development of effective therapeutic options, the disease continues to affect hundreds of millions of people worldwide.

The core pillars of the contemporary ascariasis control strategy are: regular mass preventive chemotherapy with benzimidazole agents (albendazole, mebendazole), implementation of WASH programs, enforcement of food safety standards, and broad-based health communication. Only an integrated approach — combining medical, environmental, and educational interventions — can deliver sustainable control of ascariasis.

In the Republic of Uzbekistan, continued systematic epidemiological investigation is needed to accurately characterize the true burden of infection in the population, expand diagnostic coverage in organized child collectives, and strengthen the sanitary-epidemiological surveillance system for soil-transmitted helminthiases.

Priority areas for future research include: the development and clinical evaluation of new anthelmintic agents; the creation of a vaccine against *A. lumbricoides* (recombinant protein vaccine candidates are currently in Phase II–III clinical trials); elucidation of the molecular mechanisms underpinning resistance to benzimidazole compounds; and the development of highly sensitive point-of-care diagnostic tests for field use.

REFERENCES

[1] J. Bethony, S. Brooker, M. Albonico *et al.*, "Soil-transmitted helminth infections: ascariasis, trichuriasis, and hookworm," *The Lancet*, vol. 367, no. 9521, pp. 1521–1532, 2006.

[2] R. L. Pullan, J. L. Smith, R. Jasrasaria, and S. J. Brooker, "Global numbers of infection and disease

burden of soil-transmitted helminth infections in 2010," *Parasites & Vectors*, vol. 7, p. 37, 2014.

[3] J. E. Weatherhead and P. J. Hotez, "Worm infections in children," *Pediatrics in Review*, vol. 36, no. 8, pp. 341–354, 2015.

[4] P. M. Jourdan, P. H. L. Lamberton, A. Fenwick, and D. G. Addiss, "Soil-transmitted helminth infections," *The Lancet*, vol. 391, no. 10117, pp. 252–265, 2018.

[5] C. Dold and C. V. Holland, "Ascaris and ascariasis," *Microbes and Infection*, vol. 13, no. 7, pp. 632–637, 2011.

[6] J. Keiser and J. Utzinger, "Efficacy of current drugs against soil-transmitted helminth infections: systematic review and meta-analysis," *JAMA*, vol. 299, no. 16, pp. 1937–1948, 2008.

[7] N. Arizono, H. Hasegawa, Y. Kanehiro *et al.*, "Ascaris suum infection in humans," *Emerging Infectious Diseases*, vol. 16, no. 9, pp. 1492–1494, 2010.

[8] World Health Organization, *Preventive Chemotherapy to Control Soil-Transmitted Helminth Infections in At-Risk Population Groups*. Geneva, Switzerland: WHO, 2017.

[9] V. P. Sergiev, Yu. V. Lobzin, and S. S. Kozlov, *Parasitic Diseases of Humans (Protozoa and Helminthiases)*. St. Petersburg, Russia: Foliant, 2016.

[10] M. A. Mirzaeva and G. A. Khamraeva, "Prevalence of soil-transmitted helminthiases in children of Uzbekistan," *Medical Journal of Uzbekistan*, no. 3, pp. 45–50, 2020.

[11] P. J. Hotez, M. Alvarado, M. G. Basáñez *et al.*, "The global burden of disease study 2010," *PLoS Neglected Tropical Diseases*, vol. 8, no. 7, p. e2865, 2014.

[12] S. J. Brooker and R. L. Pullan, "Ascaris lumbricoides and ascariasis: a prospective study," *Transactions of the Royal Society of Tropical Medicine and Hygiene*, vol. 107, no. 11, pp. 662–671, 2013.

[13] W. Löffler, "Zur Differential-Diagnose der Lungeninfiltierungen," *Beiträge zur Klinischen Tuberkulose*, vol. 79, pp. 368–392, 1932.

[14] C. Catterick and N. Chitnis, "Models of elimination of soil-transmitted helminths," *Parasitology*, vol. 148, no. 6, pp. 656–670, 2021.

[15] P. Steinmann, G. Cringoli, F. Bruschi *et al.*, "Point-of-care diagnosis of soil-transmitted helminths," *Parasites & Vectors*, vol. 5, p. 173, 2012.