

## Strongyloidiasis: A Neglected Disease but Highly Fatal

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### Abstract:

Strongyloidiasis is commonly refer to helminthic infection by a small size parasite belongs to intestinal nematodes named *Strongyloid stercoralis* which is most common helminthic parasite of humans and most common in tropic and subtropic arias. The disease is world wide distribution and estimated 300 to 600 million individuals around Africa, Asia, South and central America. Bad sanitation, poor hygiene, rural regions and bad socioeconomically status, these are important factor for transmitted the infection. The transmission mainly occurs via attach with fecal contaminated soil. Life cycle of parasite consists of 3 stages they are adult, larvae and egg stage. larvae(L3) serve as infective stage and enter host via penetration of skin. Pathogenicity of parasite occurs due to damage of intestinal mucosa by adult worm also larva migration though body of final host may be cause of many symptoms such as cutaneous, pulmonary, and intestinal symptoms. The microscopic examination of stool, sputum and duodenal content are considered a good method for detect rhabditiform larvae and occasionally filariform larvae. Strongyloidiasis can be treated by both albendazole and ivermectin which are good medications against parasite. long duration of disease or chronic infection is very common and lead to a wide range of clinical manifestations. Ther is two defining features of *Strongyloid stercoralis* first one the autoinfection which lead to long duration of infection and may lead to hyper infection and finally distribution of parasite through the body and cause disseminated disease usually occurs among immunosuppressive patients. with more than sixty percentage fatality. Prevent and control of strongyloidiasis may be comprised by reduce contamination of soil with stool of humans, sanitary disposal of human feces, wearing shoes and gloves is necessary when contact with infective soil, treatment of all infected cases are effective ways to prevent occurrence of strongyloidiasis.

**Key words:** Strongyloidiasis, disseminated disease, *Strongyloid stercoralis*, hyper infection, immunosuppressive patients.

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# INTRODUCTION

Strongyloidiasis is a soil transmitted disease caused by helminthic parasite called *strongyloid stercoralis*. The common name of parasite is thread worm because of uterus are assume intertwined shape that giving appearance as a twisted thread. [1]. *Strongyloides stercoralis* is cosmopolitan and widely distributed in tropic and subtropic regions worldwide. Transmission has been reported during the summer months among temperate zones. Human helminthic infection strongyloidiasis occurs via attach with contaminated soil and estimated three hundred to six hundred million individuals infected in Africa, Asia, South and central America, and the Pacific. Strongyloidiasis, is intestinal parasitic illness of man, A soil-transmitted parasite which estimated to infect three thousands to six thousands million people and considered endemic in Europe, Africa, Southeast Asia, Japan, and South America. Strongyloidiasis is endemic and more common in undeveloped countries among regions with bad sanitation, poor hygiene, bad socioeconomic conditions, unorganized and ineffective health care facilities, another causes that increased the transmission of infection include moist soil, open defecation, human walking barefoot on field also play in contaminated soil environment and bad habit of human such as open defecation. The distribution of *Strongyloid stercoralis* were reported more common in tropical and subtropical areas [2]. The adult worm resides in small intestine and can cause intestinal manifestations may be acute symptoms including epigastric discomfort, vomiting, nausea, diarrhea and bloating or the disease remain for a long period as chronic illness with symptoms like as vague abdominal pain, occasional nausea and vomiting, burning, cramp, intermittent diarrhea alternate with constipation, worse after eating and in heavy infection may cause weight loss of patient. [3]. The adult worm (female only) inhabit the mucosa of duodenum and jejunum and the parasite may persist for many years because of autoinfection transmission. Although more than fifteen percentage of patients with chronic infection are asymptomatic [4], it may cause abdominal distension and abdominal pain, diarrhea and constipation. In rare cases but highly specific manifestation is a dermatologic symptoms known as larva racing or larva currens. Ivermectin considered a drug of choice for treatment, due to the drug has low side effect, Albendazole is also effective in treatment of strongyloidiasis. Mebendazole has a much higher failure rate in clinical practice than Albendazole and Ivermectin drug [5].

## Scientific calcification of *S.stercoralis* [1]:

**Kingdom:** *Animalia*

**Subkingdom:** *Hielminthes*

**Phylum:** *Nematoda*

**Class :** *Chromadorea*

**Order:** *Rhabbitidea*

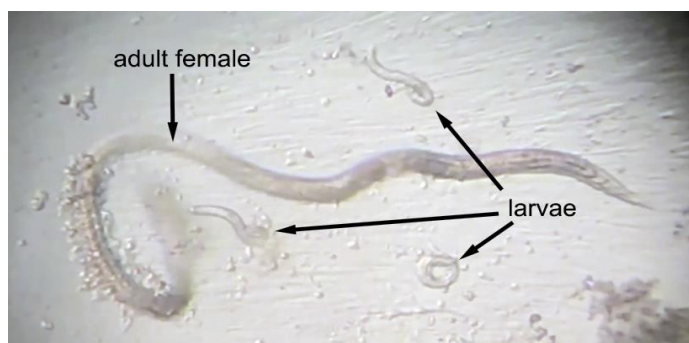
**Family:** *Strongylea*

**Genus:** *Strongyloid*

**Species:** *Stercoralis*

The female parasitic worm is tiny, thin, semi-transparent, colorless, slender in shape with tapered ends, thread like appearance. It resides inside the mucous epithelium of small intestine. They are measuring about 2.5 mm long and 0.05 mm wide, while the freely living female worm is smaller and thicker about one mm by eighty microns. It has complete digestive tract started by mouth surrounded by three small lips and cylindrical esophagus reside in the anterior third of the body, the intestine occupies the posterior third and the anus located ventrally. Cuticle is representing the outer surface of worm that aid in protect parasite from the immune response of host. The reproductive system contains two uterus, vagina and vulva. The double uterus leads to the vulva located at the junction of the middle and back thirds of the body. as in figure (1). Mature female worm produces up to fifty eggs per day. Life span of individual worm is live three to four months, but because it can cause autoinfection, the infection can remain for

years in infected person. Her uterus is tangled, giving the appearance of a twisted thread, hence its common name is thread worm nematode [5].



**Figure 1. Adult female**

#### **Male worm:**

It has a thread -like appearance and cylindrical in shape, shorter and wider than females and is about 0.6-1 millimeters long and about 40-50 microns wide as in figure -2-. Male similar female in shape but differ from female and easily distinguished by having two characters; the posterior end curved and possess spicules aid in copulation. Male do not have ability to invade mucosa of small intestine of host and not observed in human infection [6].



**Figure 2. Free living – non-parasitic- adult male.**

#### **Larval stages:**

##### **Rhabditiform larva (stage L1):**

It is the first stage of the larva that immediately hatches from eggs in the small intestine of host. It is the most common form of parasite found and pass with stool. It has actively motile and measuring about 200 to 300 micron in length by 16 microns breadth. It has a short buccal capsule represent the mouth, a relatively short double bulb muscular esophagus, and large genital primordium . It is liberate into the intestinal lumen and passes through the feces of host to external environment , in a warm, humid environment, it is undergo four molt to develop in to adult male and female worms that live free on soil[6]. It considered the diagnostic form found in stool of infected human .fig.3



**Figure 3. Rhabditiform larva (L1) of *S. stercoralis* in stool specimen of patient , Observed under microscope by using of wet mount method and stained by iodine.**

**Filaria larva (L3):**

It is the third stage of the larva and serve as the infective form for humans. The L1 larva molt twice to transform into L3 larva. It has long, slender, and thin body. It measuring about 360 microns in length and 10 microns in breath. It has a short buccal capsule as mouth with a long esophagus with a uniform width and a serrated tail has a notch. These L3 infective stage can be survive for a long period and can persist in the external environment until found a suitable host. Human infection occurs mainly through penetration of host skin. [5]



**Figure 4. Filaria larva (L3)**

**Egg stage:**

Eggs inside the uterus of the mature female. Uterus of gravid female having eight to ten eggs. They are oval in shape with blunt ends, transparent, it has thin shelled and measures about fifteen to sixty microns and a width of thirty to thirty-five microns as in figure (5). Once the eggs are laid, they immediately hatch to give a non-infective stage rhabditiform larva (L1) within the intestinal lumen of final host and find their way to the intestinal lumen where are pass with stool of host. Sometimes the eggs may pass with stool in case of severe diarrhea, it is not usually pass with stool while the L1 larva is commonly excreted with stool of patient and it was detected in the microscopic examination of stool as a diagnostic stage of parasite [3].



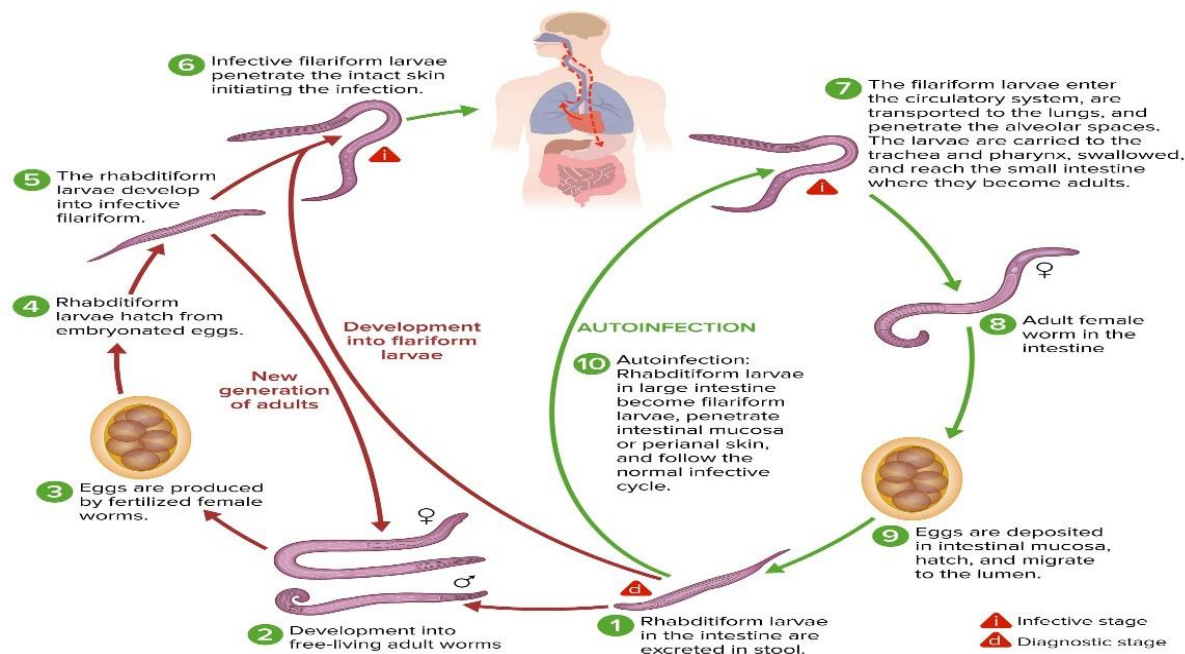
**Figure 5. Egg stage.**

**Life cycle of *S. stercoralis***

**Mode of transmission:**

- Direct contact with contaminated soil by human excreta is the main way of transmission.
- Skin penetration during barefoot walking on soil by the small filariform larvae L3 which has ability to release hydrolytic enzymes that facilitate the invade of host skin.
- Autoinfection: include two types, internal and external autoinfection.
- Transplantation of organs.
- Utero, trans mammary routes or zoonotic are very rare way of transmission.





**Figure 6. The life cycle of *S. stercoralis* it is heterogenic and more complex compared with other type nematode. It is unique within human nematodes because of it can alternation between a freely living soil life cycle and a parasitic life cycle, where it can persist for a long duration in the soil and feed on bacteria and passing through several generations. [10]**

**1. Freely living or soil life cycle:** the adult female worm produce embryonated eggs hatch inside the mucosal lining of small intestine of host and exit into the lumen of intestine to release rhabditiform larvae (L1) • larvae are pass in stool of infected host in a warm and humid soil.

A larva that passes with stool of host may undergo two types of soil development they are:

- **Direct life cycle:** The rhabditiform larva pass in stool of host when reach the soil may either molt twice to becomes the infectious filaria larva (L3) within three to four days. And then the L3 larva acts as infective form and infects humans through penetrating of skin to initiate a new life cycle.
- **Indirect life cycle:** The rhabditiform larva pass in stool of human host when reach the moist and warm soil are develop into adult males and females that live freely within three to twenty-four hours. Adult worms (male and female) mate and the female produces embryonated eggs. The eggs are immediately hatches to give a next generation of rhabditiform larva or first stage larva (L1) that molt twice to develop into filariform larvae(L3) and become infective stage. the larvae penetrate human skin usually hands or foot and this led to the initiate parasitic cycle of human infection. [11]

**2. Parasitic cycle:** After penetrating the skin of final host by filariform larva , larvae (L3) reach the right side of the heart, either through veins or lymphatic vessels and then migrate to the lungs via pulmonary blood circulation and then the larvae penetrate the alveoli and then reach to the tracheal tree and then to the pharynx and finally swallows with sputum, they reach to GIT. In the proximal small intestine, the larvae molt twice to developed into mature female worms and produce eggs via parthenogenesis (males do not requires for fertilization). The adult female resides in submucosa of the small intestine of host and start by produce of eggs. the eggs appear during twenty-five day to one month after infection of human skin. The rhabditiform larvae release from egg immediately and enter into the lumen of small intestine and then pass with stool thus, the life cycle is repeated. or may cause autoinfection within the same host. [12]

**Autoinfection:**

*S. stercoralis* is capable of causing autoinfection in weakened individual that having low immune response toward infection. it is the more common of unusual feature of strongyloidiasis is concept of autoinfection. Rhabditiform larvae can developed to infective filariform larvae in the gut especially the colon of infected person. This infective filariform larva may invade mucosal lining of colon and cause internal autoinfection or may penetrate the skin of the perianal region and cause external autoinfection and reinfect the man and repeated the life cycle within the host • repeated process of parasite autoinfection may result in the continue of this parasite in the host for a long time and remain for many years and this condition lead to hyperinfection. [8]

### **Hyperinfection syndrome:**

*S. stercoralis* can causes autoinfection which lead to hyper infection syndrome that means a numerous number of rhabditiform larvae transvers to infective filariform larvae which has ability to penetrate the mucosa of colon of infected man and leading to increase burden of parasite and rapid repeated of its migration through the body and this cause of sever clinical manifestation of strongyloidiasis of infected man. This syndrome more frequently occurs in individuals with weak immune system due to anything result in decrease the immune response of human host against parasite infection like as malnourished status, debilitated children or old man , immunocompromised patients , human treated with corticosteroid therapy, decrease acidity of stomach, operation of transplantation of tissue and organs, lymphoma , leprosy , human with HIV infection all these factors are represent the main risk factors related with causing of severe hyper infection. [8]

### **Disseminated strongyloidiasis:**

The most severe form of hyper infection syndrome of infected man is developed to a disseminated infection, in this condition the larval stage of parasite distributed and migrate to unusual site of body and invade extraintestinal tissue and circulate through body of host, therefore the larvae spread in various organs such as the kidney, heart, liver, lymphatic system, central nervous system and urinary tract. In this case the translocation of intestinal bacteria may carid with migrating larva and this led to meningitis or sepsis and this causing of high mortality among infected human [9]

### **Epidemiology:**

Strongyloidiasis is one of neglected human illness caused by intestinal nematode *S. stercoralis* the disease has worldwide incidence and occurs mainly in temperate wither, humid climate, tropic and subtropic areas, there are thirty to one hundred million cases estimated to be affected by thread worm *S. stercoralis* . the infection is highly endemic in South America, Southeast Asia, Africa and most cases occurs in USA also in Europe. Based on stool tests, clinical manifestation and duodenal lavage biopsy tests, [13]. However, the estimated global burden of strongyloid *stercoralis* is arbitrary because of difficult diagnosis and lack of accurate methods for diagnosis. Insensitive and laborious stool tests under estimate the prevalence of illness, whereas a sensitive serological ELISA test using crude *S. stercoralis* somatic antigen overestimates the infection prevalence because of thes method of diagnosis give appositve result which confuse with other infection by nematode parasite with Strongyloidiasis in endemic regions . Accurate statistics analyses of global distribution of *S. stercoralis* remains uncertain until a gold testing technique for identified parasite is available. [14]

Mild internal autoinfections may persist for a long time among persons with normal immune system without provoke clinical manifestations. More than fifty percentage of people infected with strongyloidiasis reported no symptoms. Mild clinical symptoms due to a acute stage of disease is rarely diagnosed. Acute infection may result in chronic illness with various clinical manifestations mostly related with GIT and in some cases include pulmonary symptoms [15]. Immunocompromised patient because of administration of steroid drugs in case tissue transplantation or chemotherapy therapies in case malignant tumors usually leads to chronic hyper autoinfection and then developed to disseminated strongyloidiasis. Disseminated autoinfection transports intestinal larvae into the blood stream during invasion the intestinal mucosa of host that may cause bacteremia or sepsis shock. *S. stercoralis* is high prevalent rate in people inhabit rural areas also it occurs in poor countries with hot climate and high humidity, inadequate water supply, low sanitary disposal of human excreta and poor personal hygiene. transmission commonly occurs in the tropics and subtropic zone

*Strongyloidiasis*. is spread not only in low level socioeconomic countries but also in good income regions. It is estimated that southeast asia and the pacific areas have the high rate of incidence reach fifteen percentage. *Strongyloidiasis* is related with malnutrition and retardation of growth in young ages patients. many studies report that the prevalence rate of strongyloidiasis tends to be the infection with this parasite was more common in older age individual compared to young ages.

Strongyloidiasis is usually underdiagnosed because of high number of patients has no clinical features and absent of sensitive methods for diagnosis [15, 14] The prevalence of *S.stercoralis* infection is associated very closely with autoimmune diseases and wasting, like as inflammatory intestinal disease, malignancy, encephalomyelitis, leukemia, tuberculosis, diabetes and chronic kidney disease [4] According to a report by the World Health Organization onseventeen neglected tropical illness that transmitted through contaminated soil, the parasite *S. stercoralis*, is a type of intestinal worm that is transmiited via soil, it has distinct characters that require different and moderate diagnostic methods compared from other helminths worm associated with soil-transmitted diseases, leading to recurrent cases of infection that are not easily identified. Additionally, *S.stercoralis* is not respond adequately to albendazole or mebendazole, that making it unaffected by large-scale of preventive treatment campaigns targeting other disease caused by contaminated soil STHs. [14,15,16]

### **Pathogenicity:**

#### **Effect of larval stage:**

The Infective stage filariform larva at the site of invasion in skin lead to macules and papules rash. In the human sensitized to parasite antigens, the effect of larva is allergic reaction appear as skin urticaria and sever itching, larva migrate to the lungs and provoke immune system to produce and accumulate large number of eosinophil at site of infection and cause Loeffler's syndrome, larvae movement through lungs tissue produces tissue damage and injury to the bronchiole and alveoli, this lead to pneumonitis or bronchopneumonia. It produces inflammatory exudates of immune cells such as macrophages, monocyte, eosinophil and epithelial cells and small hemorrhage around the migrating larvae. [17].

#### **Effect of adult female worm:**

the adult worms produce mild pathogenic effect of intestinal mucosa of the duodenum and jejunum like as oedema and cellular infiltration and may developed to atrophy of intestinal villi. In severe infection the parasite cause erosion of mucosa, ulcers and long period of infectious fibrosis is found in some cases. In autoinfection and hyperinfection, the inflammation of intestinal lining mucosa is severe. The parasite may involved colon and rectum and frequently mucosa becomes thickened and oedematose. [17]

### **Clinical manifestation: Clinical findings of disease may occur in three phases:**

#### **❖ Cutaneous manifestations :**

This phase of disease may be appear as dermatitis, erythema and itching of skin at the site of invasion of the filariform larva, particularly when large numbers of filariform larvae penetrate the skin. In those sensitized by prior infection, it may be occur as allergic reaction, Pruritis and urticaria, espicially surrounding the perianal skin and buttocks, The migrating larvae may lead to dermatological symptoms such as urticarial rash as red lines called larva currens (racing larvae) due to subcutaneous or intradermal invasion and movement of filariform larvae(L3) it can move rapid about ten centimeters / hour. The most common site that involved are buttock, thighs and perineum. Repeated cycle of autoinfection is responsible of causing the larva currens. Other dermatological symptoms of the disease include recurring rashes which appear as hives, rash skin or maculopapular lesions. This condition commonly represents important cutaneous clinical feature of autoinfection. Inrare cases but highly specific manifestation is a dermatologic symptoms known as larva racing or larva currens usually occurs in immunocompromised patients as cutaneous lesion[18].

#### **❖ Pulmonary manifestations:**

This condition happened when the larval stage of *S. stercoralis* emerge from the capillaries of lungs and enter into the alveoli, that lead to damage of parenchyma of lungs and bronchioles alveoli hemorrhages can occurs. pulmonary manifestations such as cough, wheezing, or shortness of breath or dyspnia may develop also

pulmonary infiltrates and accumulation of high number of eosinophil cells in lungs that refer to eosinophilia. Bronchopneumonia may be present which may developed to asthmatic symptoms or chronic bronchitis in some infected individual. Larval stage of *Strongyloides* may be appear in the sputum of patients in this phase [18].

#### ❖ Intestinal manifestations

It occurs with involvement of parasite worm to gastrointestinal tract. The adult worm (female only) inhabit the mucosa of duodenum and jejunum and the parasite may persist for decades because of autoinfection transmission. Although more than fifteen percentage of patients with chronic infection are asymptomatic but it may cause abdominal distension, abdominal pain, diarrhea and constipation. epigastric pain similar to that of peptic ulcer illness, bloating, anorexia and nausea. Other manifestations are reducing absorption of protein and paralysis of ileum. [19]

#### Complications:

It is rarely happened, more severe infections have been related with causing of protein malnutrition or nutrient malabsorption due to decrease of absorption of nutrition element in gastrointestinal tract because of severe damage of intestinal mucosa damage by action of parasite. In many chronic cases infected individuals has clinical symptoms which disappear with the pass or appearance of larval stage in the stool of patient, in spite of most have persistent disease developed to hyper infection syndrome and disseminated strongyloidiasis and this condition is very important complications especially in human with immunocompromise, HIV disease or any factor that decrease or make weakness of immune system. Pulmonary or neurological symptoms may occurs or the parasite may cause sepsis because the larvae bring pathogenic bacteria from intestinal lumen to other organs of the body through dissemination of larvae and lead to severe inflammation in various tissues. The degree of severity of manifestations in complication considered potentially risk factor of host lead to death [19].

#### Strongyloidiasis and host immune response:

The innate and adaptive immune response of infected man are playing an important role in maintaining chronic strongyloidiasis and preventing and spreading hyper-infection syndrome. Like as other helminthic infections, strongyloidiasis elicits a dominant immune response to T helper-2 lymphocytes with the production of interleukin, -E Abs, eosinophils cells, and mast cells which have important role in eliminated and destroy of parasitic worm. Antigens that release from parasite has ability to activate eosinophils through the innate immune response and then activation of eosinophils cells serve as antigen-presenting cells for encourage the creation of antigen-specific T helper -2 including interleukin -4 and this interleukin important in provoke activation of type B lymphocyte cells to categorically switch to produce additional of immunoglobulin -E and IgG Abs and further interleukin such as IL- 8 that attract granulocytes like as neutrophil cells to aid in killing and destroy larval stage of parasite [20]. The production of interleukin -E legalize the removes of mast cells and provoke more of eosinophil transition or migration to the site of infection. interleukin-5 woke as activator for eosinophil accumulation and activation more of eosinophil growth. About seventy-five percentage of individual with long period of infection has peripheral eosinophilia and elevated level of overall interleukin-E. Protective immunity of infectious larvae has been found to include specific Abs against *Strongyloides*, supplement activation and neutrophils in antibody-dependent and cell-dependent cytotoxicity type immune responses. persons with severe case have been appear to have significantly lower Abs levels and decrease in eosinophil levels when compared with symptomatic patient, thought that both antibodies and granulocytes are play an important role in control and protect host against parasitic infection [21]. The well-developed relationships between strongyloidiasis and the host immune system allows the parasite to remain for a long time in the host gastrointestinal tract. However, deficiency or disrupt of the host immune system with loss of granulocyte function, mixing-mediated antibodies production, or interleukin release T helper-2, reduces immune regulation of parasitic worm burden and prepares patients for hyper infection and dissemination strongyloidiasis. Any defect in the immune system of patient because of any factor like as malnutrition status, hematologic malignancy, patient with diabetes, gammaglobulinemia deficiency, administration of immunosuppressive drugs like as corticosteroids in case of autoimmune disease, any type of malignant tumor and operation of transplant of organs are related directly with elevated the risk of hyper-infection and dissemination of parasite in all internal organs of patient in short time or may be lead to death of patient, thus the immune system is very important for prevent the disease or occurrence of severe complications [22].



**Diagnosis:**

Stool examination: Microscopic examination of stool samples considered the good method for diagnosis of strongyloidiasis also simple and low cost for diagnosis. The larval stage is easily observed in stool examination but this method of diagnosis requires testing of several stool sample to obtain high sensitivity and it is usually used to detection of rhabditiform larvae and occasionally filariform larvae pass with stool of patient[4]

**CBC examination** ; *Strongyloidiasis* appear elevated in number of WBCs especially eosinophil cells as a result of migration of larval stage of parasite through the parenchyma of lungs and this condition refer to eosinophilia.

**Duodenal content**, biopsy specimens and possibly sputum in case of disseminated infections.

**Culture media**: Other methods used include culture of stool specimen in agar plates.

**Serological tests**: ELISA test.

**Imaging method**: CT scan computed tomography [20]

**Treatment:**

In clinical practice, the treatment of strongyloidiasis is more likely to fail due to autoinfection and weakened immunity of the host, therefore, it can be difficult. Since self-infection can occur within two to three weeks, the dose should be repeated in a period of two to three weeks to make treatment certain[22]. The most commonly used drugs to treat strongyloidiasis are benzimidazole such as albendazole, thiabendazole and ivermectin. Ivermectin is a very potent and broad-spectrum anthelmintic drug. It is a semi-synthetic derivative of a family of cyclic lactones called avermectins. It works by causing chloride ions to flow through the cell membrane of the parasite by activating specific ion channels that are sensitive to ivermectin. The resulting hyperpolarization leads to muscle paralysis. Oral ivermectin has been reported to have higher healing rates compared to other anthelmintics (albendazole), with a better profile of side effects. A chronic or acute cases of strongyloidiasis, ivermectin is widely use as drug of choice that administrated as a single dose, 200 milligram per kilogram of body weight, taken orally for one to two days also use of Albendazole, 400 milligram use orally twice daily for seven days. In case hyper infection or disseminated strongyloidiasis the drug of choice is Ivermectin, 200 µg per kg /day given orally until sputum or stool examination are negative from parasite after two weeks of treatment [23].

**Prevention:**

Control of reinfection with strongyloidiasis include a substantial problem because the parasite is abundant in soil therefore good sanitary disposal of human excreta is necessary to avoid contamination of soil with fecal material of infected man. Avoidance using of human excreta as fertilizer. Contaminated soil should be treatment although it is not highly advised mass treatment of all age group from 5 years to adult age with ivermectin 200 milligram per kilogram as single doses orally helps reduce transmission of parasite among population. Personal hygiene such as cleaning of hands before take meals. Health education these are very necessary factors for eliminate the infection and good measure to breaking the cycle of *S.stercoralis*[ 7 ].

**Conclusions:**

*Strongyloid stercoralis* is a small nematoda parasite which completes its life cycle within a single host and can survives for a long period with or without any GIT symptoms. The parasite remain several years of survival by evading from immune system of patient and this patient served as reservoirs host for transmit the infection to other healthy persons during close contact with toilet seat or via contaminated soil. *S. stercoralis* has simple life cycle, Thus, it appears a high rate of incidence especially in developed countries. A relationship between *S. stercoralis* infection and response of host immune system, patient with HTLV-1 infection or taken of corticosteroid therapies or organ transplantation, these factors facilitate rapid multiplication of parasite and may leads to cause disseminated strongyloidiasis. Many of fatal cases is associated with spread of parasite to various internal organs of the body. Thus, it is very important to early detection of *Strongyloidiasis* before the tissue transplantation or started treated by corticosteroid drugs. The serological tests are considered a useful and specific for detected strongyloidiasis

also aid in estimate the rate of parasite incidence. Early detection of strongyloidiasis and early use of ivermectin for treatment that reduce occurrence of complications. Distribution of disease related to control planning for eradication the parasite especially propped disposal of human's feces, good personal hygiene and health education about the disease aid to reduce of parasite distribution. Oher future studies of the different and new methods of resistance against parasite infection and programs for good prevention measure should be taken are important for decrease the infection in future.

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