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PULMONARY STRONGYLOIDIASIS - DIAGNOSED BY SPUTUM CYTOLOGY

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ABSTRACT

Strongyloides stercoralis is a unique parasite. It can complete its life cycle entirely within the human host and set up autoinfection cycle. As long as there is an intact immune system, the host can control the parasitic burden, and the organism may persist for years. When cell-mediated immunity becomes impaired, the parasite burden will grow, disseminate, and cause hyper infection. We report a case of pulmonary strongyloidiasis in a Chronic obstructive pulmonary disease patient who presented with acute exacerbation of breathlessness and productive cough. Sputum cytology and stool wet smears showed Strongyloides stercoralis larvae. Clinical improvement was achieved with Albendazole and Ivermectin treatment. This case emphasizes the importance of maintaining a high index of suspicion for the diagnosis of pulmonary strongyloidiasis in non endemic areas and also the possibility of presentation even without peripheral blood eosinophilia.

Key Words: Cell mediated immunity, Chronic obstructive pulmonary disease, Larvae, Breathlessness.

INTRODUCTION:-

Strongyloides stercoralis is a helminthic parasite that causes chronic infections of lungs and gastrointestinal tract. The parasite has the unique ability to complete its life cycle in human host and causes hyper infection or disseminated disease with high mortality if the host's immunity is suppressed, especially with corticosteroids¹. In patients with abnormalities of cell-mediated immunity, hyper infection syndrome may occur producing pulmonary infection that may manifest as asthma, chronic bronchitis, hemoptysis, eosinophilia and pulmonary infiltrates². Patient populations with a high prevalence of chronic lung disease, many of these symptoms are nonspecific and the diagnosis is often missed³. Sputum cytology is a useful test for demonstration of strongyloidiasis larvae.

CASE HISTORY:-

A 68 year old male farmer presented with one week history of exacerbation of breathlessness and productive cough .He was a known case of chronic obstructive pulmonary disease since 5years, and on irregular treatment. He gave history of recurrent exacerbations of his symptoms for which he used to take steroid injections and a course of oral steroids. He was a chronic smoker and alcoholic with history of Diabetes mellitus and hypertension. On admission he was afebrile, respiratory rate 30/mt, BP180/100mm of Hg. On general examination there was clubbing. His respiratory system showed decreased intensity of breath sounds on right side with bilateral rhonchi and basal crepitations.

He had normal blood counts, no eosinophilia and ESR was 77mm/hour. He was negative for HIV and HBsAg. CT chest showed emphysematous changes, multiple ill-defined centrilobular nodules scattered in both lung fields. He was treated with antibiotics, bronchodilators and steroids on the present occasion without any symptomatic relief.

Sputum Gram staining, culture and AFB were negative. All sputum cytology samples were negative for malignant cells; but showed broad coiled worm like structures with rounded and pointed ends (Figure 1,2,3). Wet smears of stool

specimens contained motile larvae of strongyloides (Figure 4,5). The patient was treated with Albendazole 400mg and Ivermectin 12mg tablets for 10days and his symptoms subsided. He was on follow up.

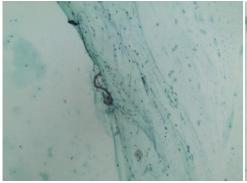




Figure 1.sputum smear shows larval form of strongyloides stercoralis, papanicolaou stain 10x

Figure 2. sputum smear shows larval form of strongyloides stercoralis, papanicolaou stain 40x

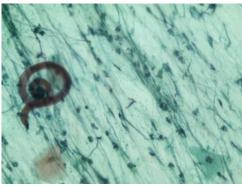




Figure 3. sputum smear shows larval form of strongyloides stercoralis, papanicolaou stain 40x

Figure 4. stool wet smear shows larval forms of strongyloides stercoralis 10x



Figure 5. stool wet smear showing larval form of strongyloides stercoralis 40x

DISCUSSION:-

Strongyloides stercoralis is an intestinal nematode occur world wide with high prevalence in tropical and subtropical areas⁴. It has got complex life cycle which alternates between free-living and parasitic cycles; has the potential to cause autoinfection and multiply within the host. The life cycle begins with the passing of rhabditiform larvae in stools. They can either develop into free-living adult worms or undergo direct development to become infective filariform larvae. Free-living adult worms mate and females produce fertilized, embryonated eggs. Rhabditiform larvae hatch from these eggs, and either develop into filariform larvae or into another generation of free-living adults. The parasitic cycle begins when the filariform larvae penetrate the human host skin. The circulatory system allows the larvae to travel to the lungs and penetrate the alveolar spaces, then transported to the pharynx, eventually swallowed, and reach the small intestine. In the small intestine, the larvae molt twice to become adult female worms. They produce eggs that become deposited in the intestinal mucosa. Once they hatch, the new rhabditiform larvae travel to the lumen and either passed in stool or cause autoinfection. In autoinfection, rhabditiform larvae develop into filariform larvae, which may penetrate the intestinal mucosa (internal autoinfection), or the skin of the perianal area (external autoinfection). In both cases, the larvae follow the normal life cycle, again heading to the lungs and eventually the small intestine⁵.

Autoinfection Associated with Antecedent Lung Disease

In patients with antecedent lung disease, delayed transit of filariform larvae through the lungs may result in moderate to severe pulmonary strongyloidiasis⁴. Another contributing factor could be the more frequent use of corticosteroids and immunosuppressive agents in patients with underlying lung disease, thereby accelerating the autoinfection and setting the stage for hyperinfection⁴. Typically, there are chronic symptoms of bronchospasm or bronchitis⁹. Examination of sputum may show adult worms, rhabditiform larvae, or ova in addition to filariform larvae⁶. Peripheral blood eosinophilia is common⁹.

Hyperinfection Syndrome

This reflects massive infection with *S.* stercoralis with widespread dissemination of larvae throughout the body⁶. In hyperinfection syndrome, classic life cycle is exaggerated(ie, the parasite burden and turnaround increase and accelerate) and is diagnosed when the larvae are recovered from extraintestinal sites. Disseminated disease is defined by the presence of parasites out-side of the traditional life cycle⁷. Peripheral blood eosinophilia is often absent; this may be due to suppression of eosinophils either by corticosteroids or by associated bacterial infection⁸.

Clinical diagnosis

The clinical diagnosis is difficult and often delayed due to non specificity of signs and symptoms. Inappropriate therapy with corticosteroids may then lead to dramatic worsening of patient's condition⁹. The finding of peripheral blood eosinophilia, in association with pneumonia, bronchospasm and abdominal pain or diarrhea, should strongly suggest the diagnosis of strongyloidiasis in patients who live in or have travelled to endemic areas⁴. Even in the absence of peripheral blood eosinophilia, strongyloidiasis should be considered if appropriate signs and symptoms are present in those known to be at risk for developing this infection¹⁰. Strongyloidiasis should also be suspected if peripheral blood eosinophilia develops in the setting of steroid therapy⁹.

The diagnosis is often confirmed by detection of larvae in the stool⁶, however, various reports¹⁰ indicated that examination of a single stool sample may show no larvae in up to 70% of cases. So, examination of at least 3 stool samples may be necessary ⁶. Duodenal aspirates or jejunal biopsy specimens show evidence of strongyloidiasis in up to 90% of cases⁴. It can also be detected by demonstration of larvae in sputum samples, transtracheal aspirates, bronchial washings and bronchoalveolar lavage¹⁰. Serologic tests are now available; ELISA is 84-88% sensitive, but may show cross-reactions to antigens indicative of ascariasis and other nematode infections ⁹.

CONCLUSION:-

Strongyloides infection can mimic a variety of disease conditions. So various authors suggested that steroid-treated patients with chronic lung disease or suspected malignant tumors be screened for strongyloides infection². Sputum cytology may be the most useful screening procedure⁵ and a bronchoalveolar lavage may be done in case the sputum is negative for larvae. Eosinophilia is not a prerequisite and this case illustrates a high index of suspicion is required even in non endemic areas for the diagnosis of strongyloidiasis, in patients without documented abnormalities of cell-mediated immunity who are not improving on conventional therapy, should alert the clinician to look for this uncommon pathogen and to treat it accordingly.

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