Navigating Filariasis in Adolescence: A Case Report of 16-Year-Old with Lymphatic Involvement

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ABSTRACT

Filariasis is a parasitic infection caused by thread-like nematodes transmitted to humans through mosquito bites. It primarily affects the lymphatic system leading to conditions like lymphatic filariasis (commonly known as elephantiasis), which can cause severe swelling in the limbs, genitalia, or other body parts. It affects 120 million people in 72 countries worldwide, mostly in the tropical and subtropical climates of Asia, Africa, Western Pacific, South America, and Caribbean. Half of the patients infected are in their 30s and 40s, and there is a 10:1 predilection for men and women. The global program to eliminate lymphatic filariasis is providing mass drug administration to eradicate this disease. We present here a case of 16-year-old female diagnosed to have filariasis due to Wuchereria bancrofti with acute upper respiratory infection.

Keywords: Filariasis, Wuchereria bancrofti, Diethylcarbamazine, mosquito bite.

INTRODUCTION

Filariasis parasites, such as Wuchereria bancrofti, Brugia malayi, and Brugia timori, complete their lifecycle across two hosts, the human (definite host) and mosquito (intermediate host). During bite, the mosquitoes introduce third-stage larvae onto human skin where they penetrate through the bite wound. The larvae migrate to the lymphatic system maturing into adult worms. These worms disrupt lymphatic function and produce microfilariae. Mosquitoes ingest again the microfilariae during subsequent bites. Inside the mosquito, the microfilaria develops into infective larvae completing the lifecycle. The treatment aims to eliminate adult worms and manage symptoms.

Case Presentation

A 16-year-old female (migrant) presented to the emergency room with a history of fever for three days associated with chills and rigors. She also complained of dry cough with history of throat pain, chest pain, and palpitations. She also complained of difficulty swallowing food. She claimed no previous history of cardiovascular disease, arrhythmia, or similar episodes in the past.

There was no significant family history and no known food and drug allergies. On presentation, the patient was conscious, oriented, and febrile. Her initial vitals revealed temperature of 100◦ F, blood pressure of 110/80 mmHg, a pulse of 118 bpm, a respiratory rate of 20 breaths per minute, and SpO2 at 98% on room air. There was no pallor, icterus, clubbing, cyanosis and edema. Systemic examination was done and as per P/A soft and bowel sounds positive. On examination of throat, there was enlargement of tonsils on the right side. The initial diagnosis made was an upper respiratory tract infection associated with tonsilitis. She was transferred to female medical ward and treatment continued further. CBC with peripheral smear, serum electrolytes, bone markers, ESR, CRP, fever profile, ECG, chest x-rays (figure -1) were ordered. She started on syrup Ascoril D 5 ml TDS and injection pantoprazole 40 mg initially and saltwater gargling.

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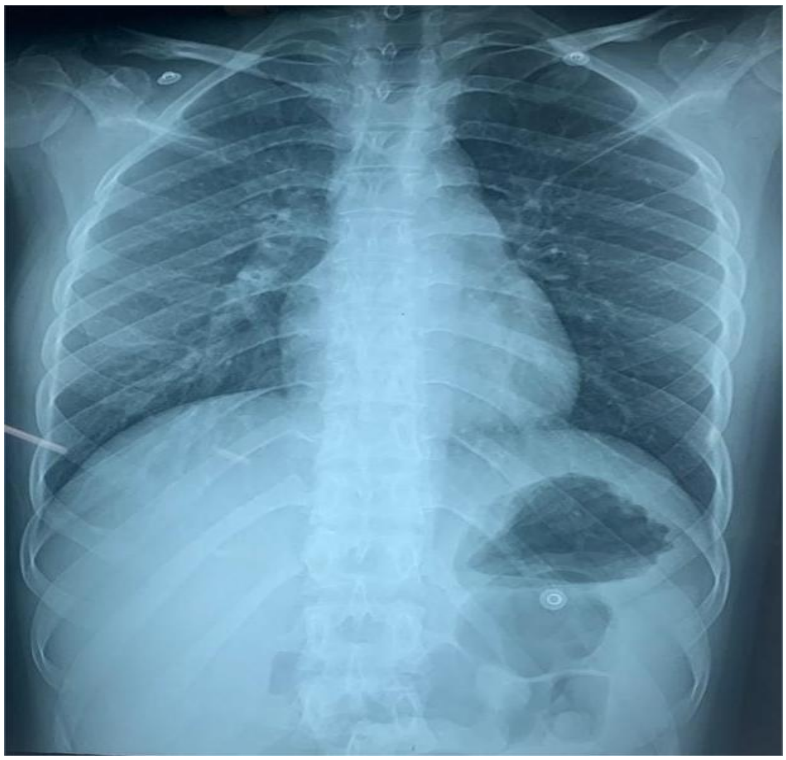

Figure 1. Chest X-Ray PA view

Baseline laboratory investigations showed normal WBC and platelet count of 9950 and 2,17,000 and eosinophilia. Her electrolyte counts and renal function tests were normal. CRP negative. Peripheral smear showed microfilaria. ENT opinion was obtained. ENT reported normal oropharynx, right tonsil grade II hypertrophy with left tonsil grade I hypertrophy. PPW mild congestion. Neck right JD node size 0.5 X 0.5 cm palpable nontender. Advised to add Drezz mouth gargle 10 ml mixed in 100 ml warm water tid for 5 days. She was started on T. Diethylcarbamazine (DEC) 100 mg 1-0-1 for 21 days course, and T. Azee 500 mg 1-0-0 for 5 days, and T. Acetaminophen 500 mg 1-1-1. The patient has improved symptomatically and was discharged from the hospital. During the first month follow-up, she was given Ivermectin 150 microgram/kilogram once. Resolution of lymphadenopathy was confirmed by examination. The patient does not have any symptomatic complications as mentioned above and advised to turn up in case of any complaints.

DISCUSSION

Filarial nematodes belong to phylum nematoda, class secenentea, order Rhabditida, and family orchocercidae. They are usually differentiated by the location, habitat of the worms, geographical distribution, periodicity, or time of diagnosis of microfilaria of the adult worms, morphology, plus the type of vectors or the periodic biting preferences. Filarial worms require two hosts, human (definite host) and mosquitoes (intermediate host). The larvae stage circulates in the blood and is ingested by mosquitoes during feeding. Inside mosquito, larvae mature into infective third-stage larvae. When the mosquito bites a human, larvae enter into the bloodstream and migrate to lymphatic vessels. They reside in the lymphatic ducts, causing inflammation and obstruction, leading to conditions like elephantiasis. The worms trigger immune response characterized by CD4 (Th2) response, humoral response (antibodies: Ig1, Ig4, IgM, and IgE), and cytokine response (IL4, IL5, IL9, IL10, and IL13). Cellular responses involve the action of T regulatory cells and macrophages. Th2 is deemed generally protective for filarial infections.

Macrophage presentation with CD4 cells during filarial infections activates them to induce secretion of cytokines, to activate mast cells as well as IL5 for easinophilic and IL4 to induce plasma cell secretion of antibodies IgM, IgG, and IgE. Filarial worms have characteristic secreted products that can affect the immune functioning of host. Proteins found in filarial worms such as phosphorylcholine (ES 62) can prevent proliferation of CD4+ T cells and B cells. In this way, the release of activating cytokines such as IL4 may be hampered, and normal activation of antiparasitic mechanisms might not take place. Antibody dependent cytotoxicity will also

be affected. Antigen processing, presentation, and immune activation also affected with inhibition of proteins, and signaling compounds such as serpins, cystatins, indoleamine 2.3 dioxygenase genes, and Wnt signalling regulators. Products secreted by lymphatic filarial worms can also stimulate the expression of negative co-stimulatory molecules such as cytotoxic T-lymphocyte antigen-4 (CTLA-4) or programmed cell death (PD1) for T-cell downregulation.

Mass drug administration (MDA) is the cornerstone strategy to lymphatic filariasis that can cause debilitating conditions like elephantiasis and hydrocele. MDA involves annual or biannual administration of antifilarial drugs to entire at-risk population, regardless of infection status, to interrupt transmission. Depending on the region an co-endemic infections, the following combinations are used. DEC + Albendazole - standard two-drug regimen. DEC + Albendazole + Ivermectin - triple-drug therapy (IDA) used in selected districts to accelerate elimination. Albendazole alone in areas of co-endemic with loiasis. Transmission assessment surveys (TAS) are conducted after 5 rounds of MDA with >-65% coverage to determine if MDA can be stopped. Morbidity management includes hydrocele surgeries and home-based care of lymphoedema. Use of mosquito nets, insecticides, and environmental sanitation encouraged. Door-to-door drug delivery and awareness campaigns to ensure compliance.

CONCLUSION

Lymphatic filariasis, is a parasitic infection caused primarily by Wuchereria bancrofti transmitted through mosquito vectors. While the disease is endemic in tropical regions, its manifestation in adolescence particularly with overt lymphatic involvement is rarely uncommon and often underrecognized. In this case, the 16-year-old presented with localized lymphadenopathy and swelling, which raised differential diagnoses including tuberculous lymphadenitis, lymphoma, and congenital lymphatic malformation. Chest x-ray was taken to rule out tuberculosis. However, identification of microfilariae on cytological examination confirmed filariasis, highlighting the importance of considering parasitic infections in endemic zones, even in younger patients. Prompt treatment with diethylcarbamazine along with supportive care can reverse early lymphatic changes and prevent long-term morbidity. In this adolescence, early intervention likely contributed a favorable outcome.

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