



Strongyloidiasis in Kidney Transplant Recipient

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Abstract

Strongyloidiasis caused by *Strongyloides stercoralis* is a Soil-Transmitted Helminth infection (STH) which is one of the most overlooked of helminthiases. *S. stercoralis* is an exception among other STHs as it can reproduce within the human host (autoinfection) causing long-standing chronic infections. Chronic low-intensity infections may remain asymptomatic. However, it can cause severe life-threatening conditions referred to as Strongyloides Hyperinfection Syndrome (SHS) and disseminated strongyloidiasis due to reactivation in immune compromised patients.

In this case, a recipient who had undergone cadaveric renal transplant was found positive for strongyloidiasis approximately one year after the transplantation. Though there was evidence suggestive of strongyloidiasis in the pre-transplant and post-transplant phases; high blood eosinophilia and intermittent gastrointestinal symptoms with skin lesions respectively, these pointers were missed on multiple occasions leading to delays in further investigations.

Immunocompromised states could facilitate autoinfection causing SHS and disseminated strongyloidiasis. In a transplant recipient, infection could be due to reactivation of chronic infection or donor derived infection or a primary acquired infection during the post-transplant phase. In the current case-scenario all three modes of infections were possible.

Proper screening of recipients and donors with high eosinophil counts for potential infections before transplantation is essential. The screening could be done coprological over several days and/or serologically.

Keywords: *Strongyloides stercoralis*; Immune compromised; Soil-transmitted infection; Post-renal transplant

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Introduction

Strongyloides stercoralis is a Soil-Transmitted Helminth Infection (STH) and it is one of the most overlooked helminth infection [1]. An estimated 30-100 million people worldwide have been infected with *S. stercoralis* [2,3]. Information on *S. stercoralis* is scanty, compared with other major STHs [3]. Low sensitivity of the most commonly used diagnostic techniques, such as direct faecal smear or Kato-Katz, is a major disadvantage in diagnosis of *S. stercoralis* [4].

S. stercoralis is an exception among other STHs; it can reproduce within human host (autoinfection) that can cause long standing chronic infection. Chronic low-intensity infections in particular, remain asymptomatic [5-7]. It can cause severe life-threatening infections known as Strongyloides Hyperinfection Syndrome (SHS) disseminated strongyloidiasis due to reactivation in immune compromised patients [5,8].

Case Presentation

A 59-year-old male with End-Stage Renal Disease (ESRD) secondary to long-standing diabetes and hypertension underwent deceased donor renal transplantation. Details of donor were not available.

He was a business man and his business was selling house hold plastic items house to house. His first two decade of life spent a slum area (Peliyagoda) with very poor hygienic conditions. Open defecation was very common in that area. Then he leaved that place and move to area with better hygienic area but he didn't has a permanent house until KT and he lived several rented houses.

He used various sources (tap and well) of drinking water and he was not used to drink boiled water before the KT.

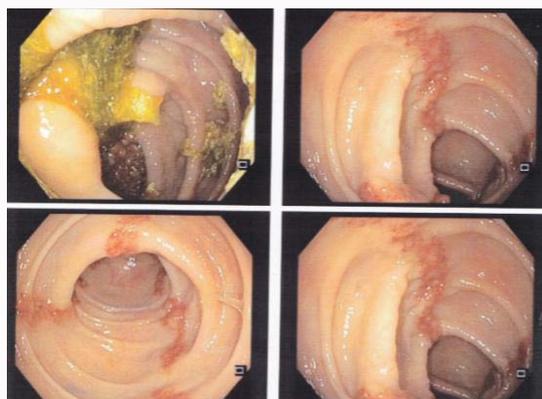


Figure 1: Histopathological report of LGIE biopsy indicated, sever colitis with heavy eosinophilia and suspicious of fungal spores.



Figure 2: Two serpiginous skin lesions were identified on bilateral hydrocele scrotum. There were no other skin lesions.

The pre-transplant laboratory investigations showed negative hepatitis B, hepatitis C, CMV, HIV, EBV. He was not investigated for Strongyloidiasis (stool for larvae or serum to detect *S. stercoralis* antigen or antibody). His pre-transplant white blood cell count was slightly elevated (11300 Cu mm) and eosinophilic count also elevated (20%). His pre KT hemoglobin level was 9.6 g/dl.

The patient's immunosuppressive regimen consisted of tapering dose of prednisone, mycophenolate mofetil, and tacrolimus. Four months after the transplant, he presented with Shortness of Breath (SOB), Difficulty In Breathing (DIB), Loss of Appetite (LOA), Loss of Weight (LOW), loose motion (watery), abdominal pain, bloating and intermittent fever with chills. He had itchy skin rash over scrotum. He was investigated for above symptoms. His chest X-ray and 2D ECHO was normal. His urine culture was positive and *Escherichia coli* were isolated. Then he was treated for urinary tract infection and a meter dose inhaler (Foracort) was started. Then his clinical symptoms were subsided.

After eight months of KT he developed oral and gingival ulcers and he was managed as HSV infection. About ten months after the KT, his hemoglobin level was low. He was investigated for anemia. The blood picture revealed, mixed deficiency anemia and his stool was positive for occult blood. He was undergone upper GI and lower GI endoscopy. UGIE was normal but LGIE has noticed wide spread focal colitis. Histopathological report of LGIE biopsy indicated, sever colitis with heavy eosinophilia and suspicious of fungal spores (Figure 1).

Again, he developed fever and loose motion (watery) about one year after the KT. Then stool was examined for ova and larvae. It was positive for larvae and larvae were identified as rhabditiform larvae of

S. stercoralis. He was treated with 400 mg Albendazole twice a day for three days. Again, he was treated with Albendazole 400 mg bd 7 days and ivermectin 200 mg for 5 days. Repeat stool sample after one week of treatment, it was negative for larvae. But, he was treated with twice daily Albendazole for seven days and oral Ivermectin 200 mg twice a day for five days. While on second round of treatment SFR, stool PCR for *Strongyloides stercoralis* and cultures were done, at Faculty of Medicine, University of Kelaniya all was negative.

Two serpiginous skin lesions were identified on bilateral hydrocele scrotum. There were no other skin lesions (Figure 2).

Discussion

Strongyloidiasis is a Soil-Transmitted Helminth (STH) disease. But its life cycle has some specific features. It has free living cycle, multiplication at the environment, can produce infective stages. It shows autoinfection, which involves development of filariform larvae in (endo-autoinfection) or on (exo-autoinfection) the body. This may account for continued infection 30 to 40 years after the host has left the original area where the infection originated. The other specific feature is females can produce eggs by parthenogenesis.

In classic life cycle, *Strongyloides* travels from the skin to the lungs and then to the GI tract of its host. Hyperinfection represents an acceleration of the normal life cycle, leading to excessive worm burden within the traditional reproductive rout (accelerated autoinfection). Disseminated disease is defined by the presence of parasites outside of the traditional life cycle (i.e., in organs other than the skin, GI tract, or lungs).

Hyper-infection syndrome and disseminated strongyloidiasis mainly reported in patients with impaired cell-mediated immunity such as transplant patients, patients receiving steroids or immunosuppressants patients infected with HTLV type 1. In normal individuals, strongyloidiasis is mainly controlled by Th2-dependent mechanisms.

This immune compromise states facilitates, autoinfection and cause *Strongyloides* Hyperinfection Syndrome (SHS) and disseminated strongyloidiasis. In transplant recipient infection could be due to reactivation of chronic infection or donor derived infection or primary acquired infection [9].

In this case all the modes of infections are possible. Low sanitary standers are risk factor for transmission of strongyloidiasis [9]. The first two decades of this patient was in high risk area. His pre-transplant eosinophilia count was high. There after his eosinophil counts were within normal limits, it could be due to immune suppression.

Details of donor were not available. Screening of the donor for strongyloidiasis is highly impossible. Hence, donor derive infection cannot be excluded.

In renal transplant recipients, most of the reported strongyloidiasis cases were reported within first six months after transplantation. In this case, it was reported after one year of transmission, then primary infection cannot be excluded because, he was on higher doses of immunosuppression in the early stage of the transplantation but he didn't develop the infection.

Proper screening of potentially infected recipient and donors with high eosinophil counts before transplantation (coprological over several days and/or serologically) is essential.

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