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Case Report

Disseminated Strongyloidiasis in a Patient with Membranoproliferative Glomerulonephritis- Case Report

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Abstract

Strongyloides stercoralis (SS) is a unique nematode with an auto infective cycle, so that it completes its life cycle within the human host and can live there for many years. In immunocompromised patients, infection can cause *Strongyloides* hyperinfection syndrome (S.H.S) that is associated with serious morbidity and mortality. As various infections are one of the leading causes of membranoproliferative glomerulonephritis (MPGN), we should consider subclinical strongyloidiasis as a possible underlying disease, especially in endemic areas. Here we describe a case of strongyloidiasis following immunosuppressive therapy for MPGN, the diagnosis of which was made, only a few hours before death, by stomach biopsy.

Introduction

Strongyloidiasis results from infection with *Strongyloides Stercoralis* (SS).The parasite can affect gastrointestinal, pulmonary, and dermatologic systems. Manifestations of the infection can range from asymptomatic eosinophilia in the immunocompetent host to disseminated disease when immunosuppression is implemented.

There are several case reports of disseminated strongyloidiasis among patients who

were receiving corticosteroid due to membranoprolifative glomerulonephritis (MPGN) or other pathologies of nephrotic syndrome (1-5).

Here we report a patient with *Strongyloides* hyperinfection syndrome (SHS) after a few months of immunosuppressive therapy for MPGN disease.

Case presentation

A 64 yr old man presented with vomiting and fever, from 2 weeks before admission to Hasheminejad Kidney Center (HKC) in Tehran, Iran in 2008. He was well until 6 months before admission, when he was referred from a southern province of Iran (Khuzestan) to another nephrology center due to nephrotic range proteinuria. Kidney biopsy had been performed at that time and glomerular membranoproliferative pattern was seen with crescent formation in 5 out of 13 glomeruli. Two glomeruli also showed fibrinoid necrosis. Tubular atrophy and proportional interstitial fibrosis was seen in 20% of submitted tissue surface. The diagnosis of diffuse proliferative and necrotizing glomerulonephritis with crescent formation was made with a suggestion of the background disease of Membranoproliferative Glomerulonephritis (Fig.1).

He was treated with prednisolone, 60 mg daily that was gradually tapered to 15 mg per day, and cyclophosphamide 75 mg daily. He was receiving both drugs when he was admitted to HKC. He was originally a resident of Khuzestan Province. In his past medical history, there was no positive finding of respiratory or gastrointestinal diseases.

On his first physical examination, the patient showed no abnormal finding except for a low-grade fever (37.8 °C). He appeared ill but had no respiratory distress, skin rash or lymphadenopathy.

Laboratory data and imaging studies are presented in Table 1.

Table 1: Laboratory data of the patient

CBC	**WBC (eosinophil count:2%)**	**$2*10^3/\mu l$**
	Hg	8 g/dl
	PLT	$75*10^3/\mu l$
Blood Biochemistry	BUN	109 mg/dl
	Cr	6.5 mg/dl
	K	6.7 mmol/L
	Na	136 mmol/L
Liver Enzymes	SGOT	Normal
	SGPT	Normal
U/A	blood	1+
	protein	1+
	WBC	2-3
Serologic Tests	ANA	Normal
	ANCA	Negative
	Complements(C3,C4,CH50)	Normal
Viral Markers	HBS Ag	Negative
	HCV Ab	Negative
Imaging		
	Chest X Ray	Normal
	Abdonimopelvic sonography	Normal

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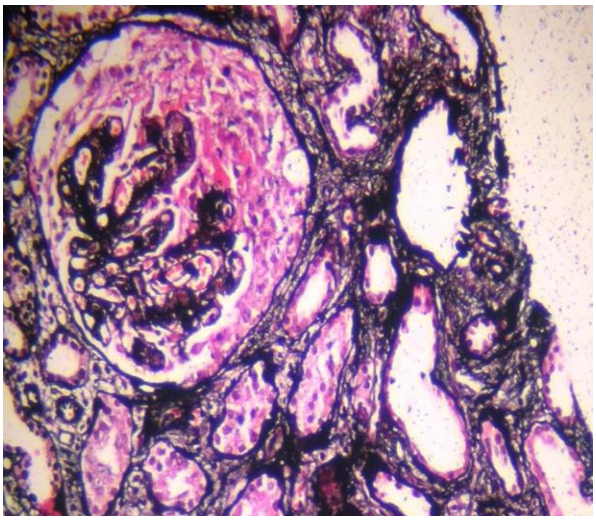

Fig. 1: Cellular crescent with endocapillary proliferation of the glomeurlus under light microscopy in favor of MPGN pattern, core biopsy of the patient's kidney. This sample was obtained in Khuzestan and the blocks were reviewed in Pathology Unit, Hasheminejad Kidney Center (Jones staining). Original picture

Cyclophosphamie was discontinued, broad-spectrum antibiotic therapy was started and hemodialysis was performed. One day after admission, upper gastrointestinal (GI) endoscopy was performed, because of intractable vomiting which showed severe erythematous lesions in stomach mucosa. On third hospital day, his general condition deteriorated rapidly and the patient developed dyspnea. A chest CT scan demonstrated diffuse alveolar infiltrates. He was intubated due to worsening of dyspnea and massive hemoptysis. Histological examination of gastric biopsy specimens revealed numerous cross-sections of eggs and rhabditiform larvae of SS (Fig. 2).

Treatment was started with ivermectin immediately, however the patient died soon after, with massive alveolar hemorrhage and respiratory failure, on the fifth hospital day and only 12 hours after the diagnosis of SHS.

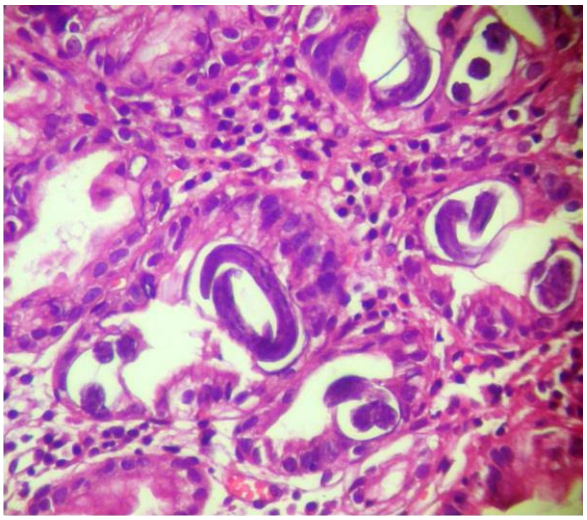

Fig. 2: Gastric mucosa of the patient showing inflammatory infiltration of lamina propria and eggs (A) and rhabditiform larvae (B) of *Strongyloides stercoralis* in the glands under light microscopy in a biopsy made by endoscopic procedure (H&E staining). Original picture

Discussion

Strongyloides stercoralis is an intestinal parasite affecting 100 million people worldwide. It is endemic in tropical and subtropical areas in the world (6-9). Diagnosis of latent infection is difficult due to limitations of current parasitological and serological methods (10-15). In Iran, it is an endemic parasite especially in southern and Northern provinces. Jalali et al. reported a prevalence of 1.4% in north of Iran, whereas Farahnak reported a prevalence of 6.9% in southern parts of Iran (6-7). In Kermanshah in 2004, out of 206 patients who were HIV positive, 2 (0.9%) had positive stool culture for SS (10).

In immunosuppressed hosts, SS may become invasive, causing SHS, which results from systemic dissemination of filariform larvae (8-9). Defects in cell-mediated immunity and corticosteroid use are considered the major risk factors for development of *Strongyloides* hyperinfection in immunocompromised hosts (17-18). Cruz and Rogers reported the first cases of SHS in 1966, as the occurrence of

fatal strongyloidiasis with immunosuppression (16-17). In Iran, there are few reports of SHS in patients who had acute or chronic lymphoblastic leukemia (26-27).

In a study, 103 previously described cases of presumed *Strongyloides* hyperinfection were reviewed. Among 89 patients, immunocompromised by therapy or disease, the mortality rate was 86% (18). In endemic areas, most cases are infected with this nematode a long time before manifestation of the hyperinfection syndrome (18).

In this case report, we present a case of steroid and cyclophosphamide resistant MPGN complicated by disseminated strongyloidiasis who died 7 months after beginning of proteinuria.

There are many reports of association of parasitic infections and glomerulonephritis, however S*trongyloides* associated glomerulonephritis has not been well-defined (19-21).

Although SS can involve any organ directly, there is evidence that suggest that immunological reactions can play a role in the pathogenesis of disease (22-24). Considering many reports of remission of nephrotic syndrome after treatment of *Strongyloides* infection with anthelmintic agents, the possibility of *Strogyloides* related glomerulopathy is strengthened.

Conclusion

Regarding long persistence of SS in the human host after initial exposure and its potential to progression to disseminated strongyloidiasis in immunosuppressed patient from one hand and its high mortality rate from another hand, patients with risk factors for SS who are candidate for immunosuppressive therapy should be screened for disease. We also recommend screening for SS in patients who have nephrotic syndrome and live in endemic areas.

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The authors declare that there is no conflict of interests.

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