Strongyloidiasis hyperinfection in a patient with membranoproliferative glomerulonephritis

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A 38-year-old female was diagnosed having membranoproliferative glomerulonephritis three months ago for which she was receiving 40-milligram prednisolone daily. She was admitted with 10 to 12 vomiting episodes per day for three days and epigastric pain. Laboratory investigations were: Haemoglobin- 1.023 mmol/L, total leukocyte count - 9.1 x 10⁹/L, neutrophils-80% (7.2 x 10⁹/L) lymphocytes-18% (1.6 x 10⁹/L), eosinophils - 2% (0.18 x 10⁹/L). Urine examination 4 + Proteinuria, 4-6 leukocytes and 6-8 red cells per high power field. Blood urea nitrogen was 20.3 mmol/L and serum creatinine was 129.7 mmol/L. Liver enzymes and serum electrolytes were normal. ELISA Tests for Human immunodeficiency virus and Hepatitis C virus were negative. Peripheral smear was negative for malarial parasite. Ultrasonography revealed minimal hepatosplenomegaly. Nephrology opinion indicated uraemic gastritis. On fourth post admission day she was irritable, non-cooperative and psychiatry opinion indicated adjustment disorder. Vomiting persisted and on the seventh day she had four to five diarrhoeal episodes, shivering, breathlessness and hypotension. Stool examination was not performed. The patient was given the following injectable drugs – Pantoprazole 40 mg od, Hydrocortisone 100 mg od, Ondensetron 4 mg tds, Ciprofloxacin 500 mg bd, Metrogyl 100 ml tds. The following drugs were given orally - Fluconazole 200 mg od, Frusemide 20 mg bd and liquid antacid 4 teaspoonsful tds. She died eight days after admission and partial autopsy of the abdomen was requested.

Pathologic findings: Kidneys were mildly shrunken,granular and showed membranoproliferative glomerulonephritis with many sclerosed glomeruli, tubular atrophy and interstitial fibrosis. Gastrointestinal tract revealed mild mucosal oedema and irregularity in small bowel and colon, while stomach was normal. However, on microscopy entire gastrointestinal tract revealed numerous larvae, few adult worms and ova of Strongyloides stercoralis [Figure 1]. The adult worms were located within the crypts and showed cuticle, thin muscle layer and internal organs. Larvae were coiled and seen within glands, lymphatics and in interstitial tissue. Moderate, mixed inflammatory infiltrate with few eosinophils and occasional granuloma was seen. The liver showed mild enlargement due to diffuse fatty change and spleen was normal. Lungs were red and rubbery due to intra-alveolar haemorrhages and few hyaline membranes. Larvae were more difficult to visualize in the lung than in the Gastrointestinal tract and were seen within alveoli, lymphatics and in the lumina of intra-parenchymal bronchi [Figure 2]. The other organs namely

Figure 1: Larval forms of Strongyloides stercoralis in colonic crypt. (H/E, 400x)

Figure 2: Strongyloides stercoralis larvae within bronchial lumen. (H/E, 400x)
liver, spleen, pancreas, adrenals and heart did not show the parasite. The cause of death was shock-lung syndrome following strongyloidiasis hyperinfection.

**Discussion**

*Strongyloides stercoralis* has a unique life cycle characterized by autoinfection, which permits recycling of larvae through duodenum, jejunum, air passages and lung, thereby allowing persistence of infection indefinitely in the host. Epigastric pain mimicking peptic ulcer, diarrhoea, cough, chest pain, dyspnoea and hemoptysis are common symptoms of strongyloidiasis. Autoinfection increases if immunity is depressed and this results in hyperinfection, which is characterized by increased larval population with exacerbation of above symptoms. Our case had membranoproliferative glomerulonephritis with chronic renal damage. Strongyloidiasis hyperinfection is a well-documented complication of chronic glomerular diseases as both heavy proteinuria and chronic renal failure lower the immune response. Additionally corticosteroid therapy in these patients predisposes to hyperinfection by suppressing lymphocyte activation and eosinophilia and also accelerating transformation of parasites to infective forms. Hyperinfection also complicates protein calorie malnutrition, renal transplantation, malignancy, HTLV-I infection and acquired immunodeficiency syndrome.

In strongyloidiasis hyperinfection, parasites are found not only in proximal small bowel and lung but also in stomach and colon. Mucosal irritation results in hyperemia and irregularity clinically presenting as epigastric pain, vomiting and diarrhoea. Parasites may be found in bronchi and their presence within alveoli results in shock lung with acute respiratory distress. Parasites are abundant and there may be mixed inflammation, granulomas and foreign body giant cells. Disseminated disease occurs when parasites spread to organs, which are not usually involved in their life cycle.

Strongyloidiasis is diagnosed by demonstration of larvae in stool, sputum, bronchial lavage and duodenal or proximal jejunal aspirates or biopsy. Repeated stool examinations with concentration techniques are essential. Eosinophilia is frequent but often absent in patients on corticosteroids. Sometimes diagnosis is made only at autopsy as in our case.

Strongyloidiasis hyperinfection is potentially fatal. Timely diagnosis is essential as it responds dramatically to thiabendazole therapy. It has been stressed, that symptoms of gastritis and diarrhoea in patients with lowered immunity should arouse suspicion of hyperinfection and prompt appropriate diagnostic measures. In fact, regular stool screening in patients on immunosuppressants is necessary to minimize risk of hyperinfection.

**References**