A case of fatal gastrointestinal haemorrhage due to hyperinfection with *Strongyloides stercoralis*

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Abstract *Strongyloides stercoralis* is an intestinal nematode with a complex life cycle. It is usually asymptomatic in healthy host however it may be a life threatening condition in immunocompromised patients when hyperinfection is associated with disseminated disease. However, reports of gastric involvement are rare. This is a case report of fatal gastrointestinal hemorrhage due to hyperinfection with *S. stercoralis* in a Burmawi male patient after brief corticosteroid therapy.

Keywords *Strongyloides stercoralis* · Hyperinfection · Disseminated strongyloidiasis · Case report

Introduction

Strongyloides stercoralis can alternate between parasitic and free-living life cycles. In the free-living cycle, the rhabditiform larvae are shed in the stool on the soil, where they grow into adult worms that produce eggs and hatch to larvae. These larvae can either grow to adult worms or penetrate the human skin to start the parasitic life cycle. In humans, the larvae are transported in the blood stream to the lungs. Through the alveoli and bronchi the larvae then reach the pharynx, where they are swallowed into the stomach and the small intestine. The larvae finally grow into adult female worms in the small intestine. By parthenogenesis, the female worms produce the eggs and larvae that cause autoinfection (Agrawal et al. 2009). Patients with a compromised immune system may develop a rapidly fatal infection, commonly referred to as hyperinfection syndrome or disseminated Strongyloidiasis (Vitiello et al. 2011). Immunosuppressive states place patients at risk for the *Strongyloides* hyperinfection syndrome (SHS), whereby the organism rapidly proliferates and disseminates within the host. Left untreated, SHS is commonly fatal. Unfortunately, the non-specific presentation of strongyloidiasis and the hyperinfection syndrome may lead to delays in diagnosis and treatment Yung et al. (2014).

Case report

A 62 years old Burmawi male patient, living in Makkah, was admitted to the gastroenterology unit of Al Noor Specialist Hospital in December 2013, with complaints of progressive vomiting and 1 week constipation. The patient was generally ill, pale and severely dehydrated. On

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physical examination the patient was confused and had tachycardia.

Laboratory investigations revealed thrombocytopenia (platelet count ranged between 71 and 130/ μ L), normocytic normochromic anemia (Hb ranged between 6 and 8 g/dL). WBCs count was high on admission (14,000/ μ L) with 86 % of neutrophils, there was no eosinophilia (0.1 %). After that WBCs count was normal (ranged between 5000 and 11,000/ μ L). Stool examination was not performed at this time. Sputum culture was done which revealed growth of *Pseudomonas aeruginosa* and MRSA. He had elevated ESR, and CRP. Biochemical investigations showed elevated blood glucose level (150 mg/dL), hypoalbuminemia (2 gm/dL), liver enzymes and renal function test were normal. Malaria test, HbsAg, HCV Ab, HIV1&2 Ag/Ab were negative.

Both Abdominal U/S and CT of abdomen were done and revealed fusiform dilatation of the CBD throughout its whole course with no obvious mechanical cause of obstruction and no intrahepatic biliary duct dilatation. Also gall bladder was moderately distended without thickening of the wall neither stone inside it. Liver was enlarged in size with moderate to severe degree of fatty changes.

In upper gastrointestinal endoscopy: There was no evidence of bleeding. Esophagus showed multiple erythematous lesions, stomach showed pan gastritis; thickening of gastric mucosa and folds (multiple biopsies were taken). Duodenum showed severe erosive duodenitis in 1st and 2nd portions and increased thickening, ulceration of its folds (multiple biopsies were taken). It was repeated after 2 days and revealed active bleeding full stomach and duodenum D1, D2, D3 showed severe erosion, ulceration, necrosis and bleeding (Fig. 1).

Histopathology of gastric antral mucosa showed surface erosions, congestion and dense infiltration by inflammatory cells. Furthermore, it revealed numerous cross-sections of

adult worms, eggs and rhabditiform larvae of *S. stercoralis* in the glands and crypts (Fig. 2a, b). Duodenal mucosal tissue biopsies, was heavily infested by larvae and eggs of *S. stercoralis*. The diagnosis of hyperinfection by *S. stercoralis* was made in this elderly patient after demonstrating the parasite in his gastric and duodenal biopsies.

The patient developed so fever, blood culture was done. The result revealed growth of *Enterococcus faecalis*. Unfortunately, the patient died because of septic shock, bacteraemia, intestinal hemorrhage before starting the treatment of *S. stercoralis*.

So, we were facing a case of severe infestation and dissemination of *S. stercoralis* which gave a rather aggressive manifestation after corticosteroid therapy and led to patient mortality after approximately 10 days hospitalization.

Discussion

Strongyloides stercoralis is an intestinal nematode of humans that is endemic in tropical and sub-tropical areas, chronic infection by *S. stercoralis* is often asymptomatic or presents with non specific gastrointestinal signs and symptoms, although immunosuppression may produce potentially fatal autoinfection (Keiser and Nutman 2004). It is considered a life threatening condition in immunocompromised patients when hyperinfection is associated with disseminated disease Al-Safee et al. (2010).

The hyperinfection syndrome represents acceleration of the normal lifecycle of *S. stercoralis*, leading to excessive worm burden in the traditional passage route, even as, disseminated strongyloidiasis involves widespread dissemination outside the gut and lungs, involving the liver, brain, heart, and urinary tract Romero-Cabello et al. (2012).

Patients with the hyperinfection syndrome and disseminated disease often have catastrophic clinical manifestations such as, shock, disseminated intravascular coagulation (DIC), meningitis, renal and respiratory failure, and a mortality rate of 50–90 % Newberry et al. (2005) and Walker and Zunt (2005).

The diagnosis of strongyloidiasis by routine stool examination is very limited since the output of the parasitic larvae in the stool is low and thus 50–70 % of a single general stool examination is unrewarding (Anitha Sebastian et al. 2014; Fortun et al. 2008) and Patel and Khandekar (2006). Therefore, in our case report, stool examination was not performed.

The diagnosis of by *S. stercoralis* hyperinfection in this case was confirmed by recovery of the parasite in his gastric and duodenal biopsy. This was similar to a case of an elderly Omani patient in whom diagnosis was made only after demonstrating the parasite in gastric biopsy.

Fig. 1 Gastric endoscopy showing mucosal necrosis, erythema and bleeding 2nd and 3rd part duodenum

Fig. 2 **a** Biopsy specimen from gastric mucosa showing *Strongyloides stercoralis* numerous eggs (thick arrow) and adult worm (thin arrow), associated with an intense inflammatory reaction and

ulcerative mucosa (H & E power 100). **b** High power view from gastric mucosa showing numerous eggs of *Strongyloides stercoralis* (thick arrow) and larvae (thin Arrow) (H & E power 400)

(Gokale et al. (2010). This was similar to a 29-year-old black male immigrant from Africa presented with recurrent life-threatening upper gastrointestinal bleeding due to massive duodenal infection by *S. stercoralis* who was diagnosed by pathologic examination of a duodenal biopsy Bhatt et al. (1990).

S. stercoralis cases reported from Saudi Arabia are few and usually they are non-Saudi patients living in KSA. A study done by Al-Madani and Mahfouz (1995) who analyzed 5518 female housekeepers from different Asian countries. The overall prevalence reported was 0.6 %; 0.4 % in Filipinos, 0.5 % in Indonesians, 1.5 % in Sri Lankans, 2.6 % in Indians and 3.4 % in Thais, respectively.

A case report from Dammam of a Filipino male, having grade II astrocytoma was given dexamethasone and developed fever, epigastric pain and cough, shortness of breath and chest pain. Sputum Gram stain revealed *S. stercoralis* larvae Shormana and Al-Tawfiq (2009).

For unknown reasons, corticosteroids have a strong and specific association with the occurrence of hyperinfection syndrome, regardless of route or dose of steroid administration. Hyperinfection and disseminated infections may be lethal in immunosuppressed patients as transplant recipients and patients under corticosteroid treatment. Even short courses of steroids in immunocompetent patients have led to hyperinfection syndrome and death Ghosh and Ghosh (2007) and (Getaneh et al. 2010).

As larvae penetrate through the intestinal wall, they can carry enteric microorganisms with them into the bloodstream. Organisms reported to cause sepsis in such patients include *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Pseudomonas*, *E. faecalis*, coagulase negative staphylococci, *Streptococcus pneumonia* and *Candida*. Keiser and Nutman (2004).

In contrast to study in which enteric gram-negative rods produced the majority of extraintestinal infections Adedayo et al. (2002). Our case is in accordance to study that demonstrated that more than 70 % of bloodstream infections were caused by enteric gram-positive cocci (Al-Hasan et al. 2007). Recently, a study reported a case of *Strongyloides* hyperinfection with secondary bacteremia with *K. pneumonia*. (Abdelrahman et al. 2012). The strangest finding was the presence of *Clostridium perfringens* bacteraemia with disseminated infection. Shea and Chau (2013).

Conclusion

These cases presented with diverse manifestations that underline the diagnostic challenge of this grave illness. Up to our knowledge this is the first reported case of massive fatal upper GIT bleeding secondary to *S. stercoralis*. This study suggests that clinicians should look for underlying strongyloidiasis in patients with serious infections due to enteric organisms without a readily identifiable source, in the presence or absence of peripheral eosinophilia.

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