Severe chronic iron deficiency anaemia secondary to Trichuris dysentery syndrome – A case report

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Abstract. Trichuris dysentery syndrome is caused by Trichuris trichiura which contributes to one of the most common helminthic infections in the world. It is associated with heavy colonic infection that manifests as mucoid diarrhoea, rectal bleeding, rectal prolapse, iron deficiency anaemia, and finger clubbing. Here, we report a case of trichuris dysentery syndrome complicated with severe chronic iron deficiency anaemia in a 4-year-old girl who required blood transfusion. The nematode was visualized on stool microscopic and colonoscopic examination. A longer duration of anti-helminthic treatment is required to achieve effective and better outcome.

INTRODUCTION
Trichuris trichiura is a soil-transmitted helminth which is found to be prevalent in the warm, moist, tropical and subtropical countries with poor sanitation. It is also common in areas where protein energy malnutrition and anaemia are prevalent as well as in areas where access to medical care and educational opportunities are often limited. The prevalence of T. trichiura is found to be high especially in children reaching up to 95% (Stephenson et al., 2000). The largest number of cases was found to be prevalent among school-age children (5-14 years) in Sub Saharan Africa, India, China, the rest of Asia and nearby islands, Latin America and Caribbean and Middle Eastern Crescent. A study done by Al-Mekhlafi et al. (2006), revealed that about 98.2% of orang asli communities in Selangor, Malaysia aged between 2 to 15 years have trichuriasis. In addition, several studies in Malaysia has demonstrated that trichuriasis are also prevalent in traditional villages ranging from 13% up to 99% and other areas such as estates (36-93%) and squatters (44-62%) (Norhayati et al., 2003).

Trichuris dysentery syndrome has been diagnosed in 6 children who were admitted initially with diarrhoea to Hospital Universiti Sains Malaysia, Kelantan. The diagnosis was confirmed by the presence of chronic diarrhoea, short stature, anaemia and T. trichiura by colonoscopy. As T. trichiura is prevalent in Kelantan, there is possibility of underdiagnosed cases in this area as the diagnosed cases need to have parental consent for colonoscopy which is not frequently obtained (Noorizan & Raj, 2001).

Human acquires infection with T. trichiura when embryonated eggs are ingested via contaminated food or water. The first-stage larvae hatch and mature in the distal small bowel. The larvae migrate to the caecum, where they finally mature into adult worms in about 2 to 3 months. The slender anterior end of the adult worm penetrates the mucosa and from there the burrowing activity takes place and lies embedded in the caecal
wall while the posterior end protrudes into
the lumen of the large intestine (Stephenson
et al., 2000). Eggs appear in faeces about 3
months after infection and undergo
development in soil.

*Trichuris trichiura* infections are mostly
asymptomatic. However, most people in
endemic areas are colonized by a small
number of worms (usually less than 15), and
the parasite is regarded as a commensal
organism rather than a pathogen (Bundy et
al., 1987). In light infection, their existence
is harmless to the host. However, when it
progresses to heavy infection, a specific
disease known as Trichuris dysentery
syndrome (TDS) develops (Cooper et al.,
1992) which commonly affects children
between 2 and 10 years of age (Ramsey,
1988). This syndrome is characterized by
mucoid diarrhoea, rectal bleeding, rectal
prolapse, iron deficiency anaemia, and finger
clubbing (Cooper & Bundy, 1988). Thus, this
shows that although most of the affected
people are asymptomatic, it may complicate
the host particularly children with severe
infection. The purpose of this case report is
to highlight the severe chronic anaemia as
one of the complications that had developed
as a result of long term *T. trichiura* infection
in a child.

Case report
A 4-year-old Malay girl presented with
a history of prolonged fever for 1 month
duration associated with mild shortness of
breath and lethargy for 1 week prior to
admission. According to her mother, she had
intermittent fever and loose bloody stools
about 2 to 3 times per day since the age of 2
years. However, her condition worsened
whereby she looked pale and less active for
the past 1 month prior to admission and
refused to eat since 1 week before admission.

There were multiple histories of
admissions to different hospitals due to
anaemia at the age of 1 year. The cause of
anaemia was most probably due to worm
infection; however, the degree of anaemia
was mild, not requiring blood transfusion.
She was given anti-helminthic during that
presentation. Unfortunately, the treatment
schedule was interrupted when she defaulted
follow-up. There was history of pica (eating
sand) and prolapse of rectum during
defecation since the age of 2.

She has 3 siblings. Other siblings were
healthy with no similar complaint. She is
staying in Bachok, Kelantan which is
probably an area with poor sanitation. She
came from a low socio-economic
background. Her mother is a housewife while
her father works as a vegetable seller.

There was no history of bleeding
tendencies, easy bruising or cyanosis. No
history of similar illness among other family
members.

Other systemic review was
unremarkable.

Clinically, she was pale, febrile (37.8°C)
and fretful. There was no finger clubbing or
other sign of iron deficiency anaemia such as
koilonychia and angular stomatitis. The
weight and height were 13 kg and 94 cm
respectively (below 3rd centile). There was
generalized crepitation heard over both
lungs. The heart sounds were normal and no
murmur was noted. She had hepatomegaly
with liver palpable about 5 cm below the
subcostal margin. Multiple shotty lymph
nodes were palpable over inguinal, axillary,
cervical and occipital regions. There was
no rectal bleeding or prolapse during
examination.

Chest x-ray revealed cardiomegaly and
bilateral pneumonic changes. However, on
echocardiogram examination, no structural
abnormality of the heart was noted.

Laboratory findings
Full blood count examination showed
leucytosis (total white cells of 30.32 X 10⁹/
L), anaemia (haemoglobin of 6.8 g/dL) and
thrombocytosis (platelet count of 415 X 10⁹/
L). Other blood count indices were low (red
blood cell count and haematocrit levels were
2.49 x 10⁹/L and 18.8% respectively).

Full blood picture revealed evidence of
microcytic hypochromic anaemia,
eosinophilia (4.5%), leucoerythroblastic and
dysplastic changes.

Liver function test, blood urea serum
electrolytes, coagulation profile, stool and
blood for cultures and blood film for malarial
parasite were unremarkable. Mycoplasma,
Epstein Barr virus and cytomegalovirus serological tests were unremarkable. Autoimmune screening for Coomb's test, anti dsDNA and anti-nuclear antibody (ANA) gave negative results. Stool microscopic examination showed T. trichiura ova with no other ova or parasite seen. Egg count was not done as it was not part of the routine stool microscopic procedure in this setting. Stool for occult blood was negative.

Serum iron and ferritin were low (32.12 ug/L and 12.40 ug/L respectively).

She was initially treated as bronchopneumonia and iron deficiency and was started with syrup augmentin 300 mg three times daily.

She was treated empirically with syrup albendazole 400 mg once daily for 3 days as the history was suggestive of worm infection and this was further supported by positive stool microscopic examination. Syrup multivitamin 2.5 mls once daily and syrup folate 2.5 mls once daily were given. She was given blood transfusion with 10ml/kg packed cells.

The patient had shown marked clinical improvement following these treatments and was discharged on day 11 of admission with the diagnosis of Trichuris dysentery syndrome.

On follow-up, colonoscopy was done to exclude other possible causes of anaemia which revealed numerous T. trichiura adult worms along the rectum and sigmoid colon. Syrup albendazole 400 mg once daily for 3 days was repeated to ensure adequate eradication of the worms. However, this patient had defaulted the subsequent follow-up. Thus, we are unable to monitor the disease progress.

DISCUSSION

TDS has been associated with massive T. trichiura infection mainly among children. In light infection, most of the patients are asymptomatic and the parasite usually harmless to the hosts. As it progresses to massive infection, it can cause complications with colonic hyperaemia, oedematous mucosa and multiple erosions as a result of inflammatory changes (Kim et al., 2003).

Furthermore in patients with greater worm burden, they may present with anaemia, diarrhoea, abdominal pain, weight loss, malnutrition, appendicitis, colonic obstruction, perforation or intestinal bleeding. Specifically in heavily infected children, they usually manifest as anaemia, chronic diarrhoea, stunting and finger clubbing (Stephenson et al., 2000).

Disease occurs either due to mechanical effects or allergic reaction. The diarrhoeal episodes are preceded by immunological response which is mediated by specific IgE antibody and mast cell degranulation. Other hypothesis suggested that it is induced by antigen-induced secretion of chloride (Mahmoud, 2000).

Non-specific immunity in terms of macrophage numbers in the mucosa as well as the production of macrophage-derived cytokine, tumour necrosis factor α (TNFα) are important factors (MacDonald et al., 1991). This process is induced by bacterial products such as lipopolysaccharides and peptidoglycans, leaking across the damaged colonic epithelium in TDS could activate hepatic macrophages to produce TNFα which lead to increase permeability and also increase local TNFα production (Breese et al., 1994).

Both mast cells and macrophages are capable of producing a wide array of pro-inflammatory cytokines which could have profound effects in the colon. Macrophages are usually the major source of TNFα in inflamed intestine, especially those macrophages which have recently been extravasated from the blood (Gordon et al., 1990; Galli et al., 1991). TNF also has been described to be associated with stunting in children (Cooper et al., 1990). Other cytokines such as IL-1 and 6, granulocyte-macrophage colony-stimulating factor, transforming growth factor β3, prostaglandins and leukotrienes have been documented in inflamed human intestine (Schreiber et al., 1992).

Trichuris infection can cause blood loss due to oozing of blood at the sites of
attachment where it is responsible for a daily blood loss of 0.005 ml per worm per day which only accounts for about 10-15% from the blood loss due to *N. americanus* and 2-3% of that attributed to *A. duodenale*. In infected children it may finally lead to anaemia. This is particularly seen in cases where the children also have co-infection with hookworm, malaria and/or a low intake of dietary iron. (Roche et al., 1957).

Furthermore, *Trichuris* infection can suppress the appetite, growth, physical fitness, physical activity, work capacity, cognitive development and school performance in malnourished populations.

In chronic *Trichuris* infections, children may suffer from chronic colitis. This form of chronic intestinal inflammation may lead to growth failure or profound growth stunting which can either occur by secondary effects or direct effects on metabolism. The secondary effect can be attributed to some mechanisms such as concomitant decrease in plasma insulin like growth factor-1 (IGF-1), increase in tumor necrosis factor-α (TNF-α) in the lamina propria of the colonic mucosa and peripheral blood (likely to decrease appetite and intake of all nutrients) and a decrease in collagen synthesis on nutrient balance (Cooper, 1990).

In growth stunting, this condition can be reversed by repeated treatment for the infection and oral iron supplement. However, this statement was commented by some researchers from Jamaica that the significant developmental and cognitive deficits are unlikely to disappear without increasing the positive psychological stimulation in the child’s environment (Stephenson et al., 2000).

Mebendazole and albendazole, both of which are on the WHO Essential Drugs List, are very effective against *T. trichiura* (Stephenson et al., 2000). Multiple doses are needed to attain complete parasitological cure in all cases. Mebendazole has been proven as the treatment of choice in trichuriasis. This drug is associated with 40-75% cure rates by single dosing of 500 mg. Standard dose regime of mebendazole of 100 mg twice daily for 3 days provides about 70% successful rate. Second course of mebendazole is indicated if patient is not cured within 3 to 4 weeks (Bartoloni et al., 1993).

Mebendazole acts by selectively and irreversibly blocking glucose uptake and other nutrients. This will result in worm death. Other alternative treatment is albendazole. The main drawback is that the efficacy or successful rate for trichuriasis is slightly lower than for mebendazole (Bartoloni et al., 1993). However, the ability to be given as a single dose regime makes the albendazole provides better adherence or compliance as compared to mebendazole. By extending the duration of the treatment up to 3 days may be helpful in achieving cure rates up to 80% (Ramalingam et al., 1983; Norhayati et al., 1997; Hall & Nahar, 1994). In some reported cases, patients might need a longer duration of treatment about 5 to 7 days to improve (Sirivichayakul et al., 2003).

However, as in this girl, the adult worms were not cleared by albendazole treatment that was given for 3 days duration. Repeated treatment was given on the next follow-up in view of the presence of adult worms during colonoscopic examination. The outcome of the treatment could not be determined as the patient defaulted follow-up. In this case, home visit by the community nurse would be of a great help to assist the continuity of our treatment.

The role of combination therapy has been studied by previous researcher. The use of albendazole in combination with ivermectin resulted in greater significant outcome in weight gains among infected children as compared to monotherapy with placebo, albendazole or ivermectin over 4 months therapy (Stephenson et al., 2000).

Control measures should be emphasized especially in endemic areas which focused particularly on personal hygiene, avoidance of pica, proper sanitation and faecal disposal and avoidance of night soil as fertilizer. Other siblings should be screened too in preventing and reducing transmission (Mohammad et al., 2009).

Pertaining to this case, other family members especially other siblings should be screened for *T. trichiura* infection. If other family members are found to be positive for the parasite, they need to be treated. The
patient was suggested to be followed-up regularly to ensure clearance of the worms and to monitor the long term complications that she may have developed.

In conclusion, close monitoring and follow-up of patients with TDS are crucial as the syndrome can result in long term complications and increased morbidity in patients especially children.

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REFERENCES


