Ascaris lumbricoides causing infarction of the mesenteric lymph nodes and intestinal gangrene in a child: a case report

Infarkte an mesenterischen Lymphknoten und intestinale Gangrän durch Ascaris lumbricoides bei einem Kind

Abstract

Ascaris lumbricoides or round worm infestation is quite common in the developing world. It affects all age groups but is more common in children. Most of the cases remain asymptomatic. The usual presentation is an intestinal obstruction. The physicians should be aware of this condition and consider it in the differential diagnosis when faced with such a case. The rare fatal complications include bleeding, perforation and gangrene.

Keywords: Ascaris lumbricoides, infarction, lymph nodes, gangrene

Zusammenfassung

In Entwicklungsländern ist der Befall mit Ascaris lumbricoides oder Spulwurm vielfach zu beobachten. Betroffen sind alle Altersklassen, Kinder jedoch am häufigsten. Die meisten Fälle bleiben symptomlos. Häufigste Komplikation ist Darmverschluss. Ärzte sollten an eine derartige Komplikation denken und sie bei der Differentialdiagnose berücksichtigen. Zu den seltenen und schwerwiegenderen Komplikationen gehören intestinale Blutungen, Darmperforation und Gangrän.

Schlüsselwörter: Ascari lumbricoides, Lymphknoten, Infarkte, Gangrän

Kincho Lhasong Bhutia[1]
Subhajeet Dey[1]
Varun Singh[1]
Amlan Gupta[2]

1 Department of General Surgery, Sikkim Manipal Institute of Medical Sciences, Sikkim Manipal University, Sikkim, India

2 Department of Pathology, Sikkim Manipal Institute of Medical Sciences, Sikkim Manipal University, Sikkim, India

Introduction

Ascaris or roundworm infestation of the gastrointestinal tract is quite common in the tropics, especially in children. The presentation ranges from being completely asymptomatic to presenting as an acute abdomen following intestinal obstructive symptoms and rare fatal complications like perforation and gangrene of intestines. Ascaris or other helminth infestation is rare in the developed world.

We present a case of mesenteric lymph node infarction and intestinal gangrene due to extensive infestation by Ascaris lumbricoides in a child aged 4 years. Surgical intervention was done in this patient and follow up at 6 months, revealed the child to be asymptomatic.

Case presentation

A 4 year old girl presented to the outpatient department with the complaints of abdominal pain and distension since last 6 days, as per her mother. She also had history of vomiting since last 3 days. The pain was gradual in onset, colicky in nature and initially was not associated with vomiting. This was followed by a gradual abdominal distension and an increase in the severity of pain and vomiting. The abdominal distension was more pronounced in the umbilical region. The vomiting was projectile in nature and the vomitus was greenish in colour and contained undigested food particles. The child was partially immunized. The mother also denied the child having any significant medical problems in the past. On physical examination she was poorly nourished with pallor, oral temperature was 37.7 °C, blood pressure was 98/60 mmHg, pulse regular, with a rate of 110 beats per minute, respiratory rate of 34 per minute. Respiratory system revealed bilateral air entry with no added sounds but slight respiratory effort, cardiovascular examination revealed normal S1 and S2 with no murmur, central nervous system revealed no neurological deficits. Abdominal examination revealed a distended abdomen of all quadrants but more pronounced in the umbilical region. Superficial dilated veins could be seen in the epigastric region. All the quadrants were moving equally with respiration and the hernial orifices were normal. On palpation, there was a diffuse tenderness with guarding. There was no organo-

megaly. On auscultation, hyperdynamic bowel sound were audible. Per rectal examination revealed stool with altered coloured blood and multiple live roundworms. At the time of admission, the laboratory investigation included a complete blood count which showed low haemoglobin as 4.5 g%, raised total leucocyte count as 14.2 x 10^3/µL, low red blood cell count as 1.61 x 10^6/µL, low hematocrit 13.1%. Differential leucocyte count were: neutrophils –32.6%, lymphocytes –7.9%, raised monocyte –56.1%, eosinophils –0.9% and raised basophil count of 2.5%. Kidney function test, random blood sugar were within normal limit. Sodium level was low 129 mmol/L, potassium level was 3.9 mmol/L and chloride level was 100 mmol/L.

Her chest X-ray revealed no abnormality. However, X-ray of the abdomen revealed several moderately distended bowel loops with few air fluid levels with no free gas under the diaphragm.

The child was initially managed conservatively with correction of fluid and electrolyte imbalance, nasogastric aspiration, broad spectrum antibiotic coverage, blood transfusion and analgesics. No antihelminthic drug was used at this stage. The likely explanation would be that the drugs would kill the Ascaris and that would likely form a bolus aggravating the obstruction. Hypertonic saline enemas which is used routinely in conservative management was also not used due to the risk of complications like perforation of small and large bowel, loss of fluids, dehydration and electrolyte disturbances. She underwent exploratory laparotomy under general anaesthesia the next morning, which revealed 1.5 litres of foul smelling hemorrhagic peritoneal fluid and about 25 cm of gangrenous small intestine upto approximately 15 cm proximal to the ileo ceacal junction (Figure 1a). The proximal segment of the bowel was filled with live roundworms forming a bolus, to such an extent that the outline of the worms could be made out through the stretched bowel wall. After resection of the involved bowel during surgery, the bolus of worms were found occluding the whole bowel lumen. The worms filled approximately a 1 litre kidney tray. A thorough exploration was done to exclude any other associated pathologies as well as to rule out other areas of questionable viability in rest of the gut. The gangrenous segment was resected after ligating the mesenteric vessels and every effort was made to prevent the further contamination of the peritoneal cavity. Following resection, milking of the proximal bowel was done, which revealed both live and dead roundworms (Figure 1b). After ensuring the viability of resected bowel ends, continuity of bowel was restored by a two layered end to end anastomosis. A thorough peritoneal lavage was given with warm normal saline. The whole of the resected bowel segment was sent for histopathological examination (Figure 1c). The patient received one unit of compatible matched whole blood during surgery. She was put on total parenteral nutrition along with broad spectrum antibiotics and care taken to maintain fluid and electrolyte balance. On the 6th post-operative day she received one dose of Albendazole (400 mg). She passed 4–6 worms in the following subsequent post-operative period. The child was discharged on 10th post-operative day. The dose of Albendazole was repeated at 6 weeks.

Follow up at 6 months revealed the child to be thriving well.

Histopathology of the resected specimen revealed intestinal wall with extensive transmural haemorrhagic infarct of the intestinal wall. The underlying connective tissue layer, including muscularis showed an intense infiltrate by neutrophils (Figure 2a). Sections also showed mesenteric fat with thrombosed blood vessels (Figure 2b) and the intestinal wall with the connective tissue below the muscularis showed oedema, fibrin deposits and eosinophilic infiltrate (Figure 2c). One of the most remarkable features was the section showing massively infarcted mesenteric lymph nodes with only a narrow peripheral rim of spared cortical tissue with lymphocytes. Almost the whole nodal parenchyma was necrotic, strongly eosinophilic with ghosts of lymphocytes and of other tissue components. The reticulin network was preserved. In the perinodal fibroadipose tissue, an abundant inflammatory exudate was composed of fibrin and polymorphonuclear leukocytes (Figure 3a). The other remarkable feature was the presence of infarction of only the germinal centre (Figure 3b). This probably can be explained by the centripetal nature of the anatomical blood supply of the mesenteric lymph nodes [1].

Discussion

Ascariasis or the common roundworm is cosmopolitan, having a world-wide distribution being specially prevalent in the tropics, such as China, India and south east Asia and Africa [2], [3]. It is relatively rare in the developed world. It is more common in the lower socioeconomic class, and areas with poor sanitation.

There exist several reports that Ascaris infection may account for 25% of hospital admission for intestinal obstruction in Kolkata or even up to 57% of all acute abdominal cases in Burma [4]. However, surveys undertaken by the Division of Helminthology, in the National Institute of Communicable Diseases, New Delhi during the period 1998–2000 to assess the problem of soil transmitted helminths only using WHO methodology of sampling and laboratory examination, found prevalence of Ascaris lumbricoides as 30.7% in Gangtok. Surveys were undertaken targeting children in the age group of 9–10 years. This condition is found in all age groups but is more common in children. The mode of infection is by swallowing fertilized eggs of Ascaris. The released larvae then migrate to the lungs. The larvae when coughed up by the host are once more swallowed back into the intestine to develop into adult worms. The size of an adult worm ranges from 15–40 cm in length and 3–5 mm in diameter [5], [6].

The clinical features due to the migrating larvae are pneumonia (Loeffler's syndrome), visceral damage, urticarial rash, eosinophilia, abscess, hepatosplenomegaly

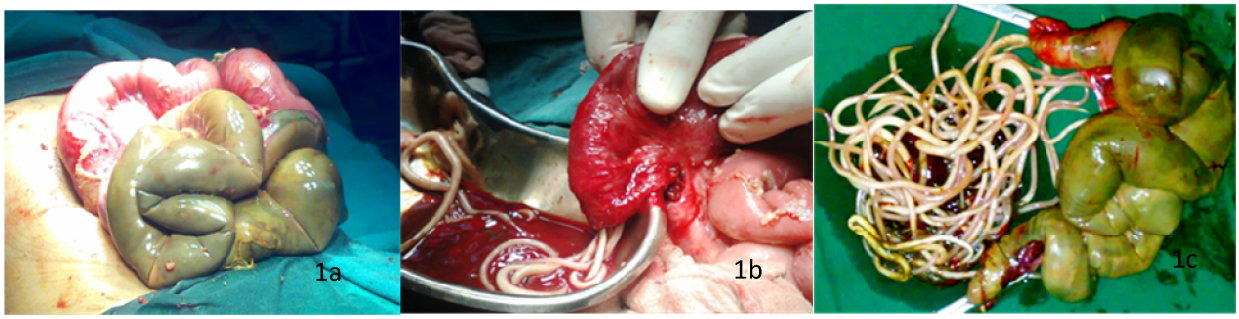

Figure 1: Fig. 1a shows the gross appearance of the gangrenous small bowel loops adjacent to the viable bowel loops after laporotomy, about 25 cm in length upto approximately 15 cm proximal to the ileo ceacal junction.
Fig. 1b shows milking of the proximal bowel being done following resection, which revealed both live and dead roundworms. Resection was done till the healthy bowel end denoted by bleeding from the cut bowel ends.
Fig. 1c shows the whole resected gangrenous segment of small bowel along with the dead and alive roundworms milched from both the proximal and the distal ends of the bowel during laporotomy.

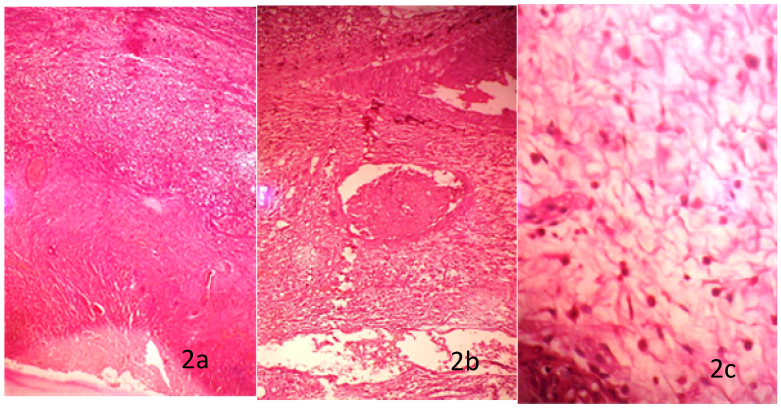

Figure 2: Fig. 2a shows extensive transmural haemorrhagic infarct of the intestinal wall with the underlying connective tissue layer, including muscularis showing an intense infiltration of neutrophils.
Fig. 2b shows sections showing mesenteric fat with thrombosed blood vessels.
Fig. 2c depicts the intestinal wall with the connective tissue below the muscularis showing oedema, fibrin deposits and eosinophilic infiltrate.

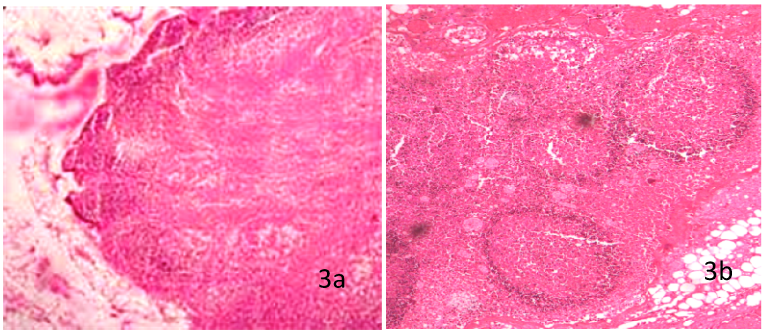

Figure 3: Fig. 3a shows sections showing massively infarcted mesenteric lymph nodes with only a narrow peripheral rim of spared cortical tissue with lymphocytes. Almost the whole nodal parenchyma is necrotic, strongly eosinophilic with ghosts of lymphocytes and of other tissue components. The reticulin network is preserved. In the perinodal fibroadipose tissue, is an abundant inflammatory exudate composed of fibrin and polymorphonuclear leukocytes.
Fig. 3b shows the presence of infarction of only the germinal centre. This probably can be explained by the centripetal nature of the anatomical blood supply of the mesenteric lymph nodes [1].

and toxicity. A heavily infested child may suffer from protein energy malnutrition along with deficiency of vitamin A which may cause night blindness [7], [8]. Sometimes the body fluid of Ascaris when absorbed is toxic and may give rise to typhoid like fever, urticaria, oedema of face, conjunctivitis and irritation of the upper respiratory tract [9], [10]. It is also known to cause intussusception, penetration through the ulcers of the alimentary canal, volvulus and even bolus intestinal obstruction when present in large numbers [11], [12]. The rare fatal complications are, massive gastrointestinal bleeding with ul-

ceration, perforation, and even gangrene of the bowel wall [13].

Infarction of the mesenteric lymph nodes due to intestinal helminths is unknown. Lymph nodes are rarely infarcted, due to the well developed vascularity and anastomosis. Reviews of the literature record only a limited number of cases [14], [15]. In one study reported [16], all the five patients were middle aged; two had a history of vascular disease, and three had undergone recent surgery in the area drained by the infarcted lymph nodes. Local trauma caused by fine needle aspiration biopsy of lymph nodes has been occasionally mentioned. There are also reports of lymph node infarction with acute febrile disease of unknown origin [17]. The most common cause of massive lymph node infarction is involvement by tumor, either primary lymphoma or metastatic carcinoma. Therefore, the differential diagnosis of lymph node infarction can be following:

- Kikuchi and Fujimoto disease,
- Tuberculosis, leprosy, lymphogranuloma venereum, and cat-scratch disease,
- Syphilis,
- Hypersensitivity reactions,
- Malignant lymphomas of Hodgkin and non Hodgkin types.

Only a limited number of intestinal gangrene cases due to torsion of bowel as a result of extensive intestinal ascariasis has been reported in the past [13], [18], [19]. But none of them has reported the associated infarction of the mesenteric lymph nodes.

The cause of the infarction of the mesenteric lymph nodes could be due to lymphatic stasis as a part of volvulus. Lymphatic stasis causing infarct has been studied in protein losing enteropathy [20]. However, no significant sinus dilatations were seen nor any obstructions by adult worms or larvae were seen in the lymphatic channels or the blood vessels. The causation could be purely physical torsion of the vessels. Volvulus causing coagulative necrosis of lymph nodes has been noted by Nursel [21] and Mahy [22].

The vascularity of the lymph node is different in mesentric lymph nodes. It is centripetal and therefore more prone to infarct [1].

Herman et al. [23] did report that following antigenic stimulation, the differential flow significantly increased compared with the resting node. The marked increase in blood flow, therefore, was largely due to increased shunt flow. They also noticed significant dilatation of the subcapsular vascular arcade. Interestingly, this was more pronounced in those areas of the lymph node where the perinodal granulocytic infiltrate was most apparent. The histology of the viable lymph nodes in our case did not reveal any significant granulocytic infiltrate and the sinuses were relatively free but not dilated as described by Herman et al. So this does not really explain the antigenic stimulus from dead larvae causing infarct. We had seen cytotoxicity by filarial worms in breast in the form of fat necrosis [24], but such histological features were not present in this case.

Adult worms may also enter the lumen of the appendix and may rarely cause appendicitis [25], [26]. Obstructive jaundice and acute pancreatitis have occurred when the worm has entered the biliary passage [27], [28].

The diagnosis can be made by the presence of adult worms or Ascaris eggs in the stool. Adult worms can also be demonstrated by ultrasound [5], [6].

In majority of the uncomplicated cases, Ascariasis can be treated successfully with drugs like Albendazole, Mebendazole or Pyrantel Pamoate [29], [30]. Most of the patients presenting with bolus obstruction respond to the conservative management. In such patients, antihelminthic should be given in the hospital only after the relief of obstruction [31]. If there is no further obstruction, the patient can be safely discharged home. The antihelminthic drug has to be repeated after 6 weeks to eradicate any worms that might have been in the larval phase at the time of admission.

The type of surgery depends on the findings during laparotomy. If the bowel is viable and the obstruction is at the level of ileum, milking of the worms to the caecum can be done carefully without causing trauma to the bowel wall. If the obstruction is at the level of the jejunum and if there are multiple masses, enterotomy should be done through a longitudinal incison with removal of worms by sponge holding forceps. The incison should be closed transversally with great care to avoid contamination of the peritoneal cavity by the worms or its eggs. In cases where the intestinal wall is thin for example volvulus, milking should not be attempted as this may cause serosal tears. Enterotomy is preferred in such cases. Breaking of the worms during milking should be avoided as this may release toxins. In cases presenting with bowel gangrene, perforation, or intussusception with non-viable bowel, resection with primary anastomosis may be required.

The preventive measures should be included in all health education program and should be directed at school children and their mothers [32], [33]. In Sikkim (India), regular deworming programmes are undertaken by the Department of Health Care, Human Service and Family Welfare, Government of Sikkim under the school health programme.

Conclusion

Thus we conclude that one of the rare causes of mesenteric lymph node infarction and intestinal gangrene is torsion of intestine as a result of extensive intestinal ascariasis. Majority of the cases can be managed conservatively but rare fatal complications like intestinal gangrene and perforation require urgent surgical intervention.

Notes

Competing interests

The authors declare that they have no competing interests.

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Corresponding author:
Dr. Kincho Lhasong Bhutia
Department of General Surgery, Sikkim Manipal Institute of Medical Sciences, Sikkim Manipal University, 5th Mile Tadong, 737102 Sikkim, India
klhasong@gmail.com

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