

SHORT REPORT

Severe anaemia owing to hookworm in a 12-day-old Nepalese infant

We report a 2.5-month-old infant with hookworm infection manifesting with black, tarry stools from 12 days of age. His mother also had hookworm ova in her stools. The usual incubation period for hookworm infection is 2 months. The very early manifestation in this newborn suggests transplacental infection.

Introduction

Hookworm is a common pathogen in many regions of the world. Hookworm infestation in infancy manifesting with black, tarry stools has been reported previously.¹ We report a case of infantile hookworm disease where symptoms started in the neonatal period.

Case Report

A baby boy was born to a primigravida mother by normal vaginal delivery in rural Nepal. The perinatal period was uneventful and he was exclusively breastfed. He began passing black, tarry stools from day 12 of life. No treatment was sought at this stage. He gradually became increasingly pale and was brought to St Stephen's Hospital, Delhi when he was 2.5 months old.

At admission he was markedly pale but was active and alert and weighed 4.5 kg. He had hepatosplenomegaly with his liver margin 5 cm below the right costal margin and spleen 2 cm below the left costal margin in its long axis. Haemoglobin was 2.1 g/dL. White blood cell count was $15.4 \times 10^9/L$ with

lymphocytes 70%, granulocytes 20% and eosinophils 10%, with an absolute eosinophil count of $1.5 \times 10^9/L$. Platelet count was $352 \times 10^9/L$. Reticulocyte count was 15% and red cell distribution width was 21.94% (normal range 11.6–16.5). Peripheral blood film showed eosinophilia with dimorphic red cell morphology suggestive of iron, vitamin B₁₂ and folate deficiency. Total serum protein was 53 g/L and serum albumin was 26 g/L. Prothrombin time was 11.6 sec (control 10.2 sec) and partial prothrombin time was 23.2 sec (control 28 sec). Technetium scan for Meckel's diverticulum was normal. Direct and indirect antiglobulin tests were negative. He was transfused with 10 ml/kg packed red cells on admission and again 2 days later. Despite the transfusions, his pallor persisted. Microscopic examination of stool showed many ova of hookworm and some smears showed live larvae inside eggs. The mother's stool also had eggs of hookworm and roundworm. Breast-milk was examined microscopically for larva of hookworm on one occasion, but was negative.

The infant was given mebendazole 50 mg twice daily for 3 consecutive days. On day 3, his stool colour changed from black to yellow. Repeat stool examination was negative for occult blood, ova or parasites. There was a consistent rise in haemoglobin over the next 15 days and it was 8.1 g/dL when the infant was discharged.

Discussion

Most cases of infantile hookworm disease have been reported from China² and there

have also been reports from Nepal.³⁻⁵ We were unable to identify the specific genus but most worms identified from this region have been *Ancylostoma duodenale*.¹

Hookworm larvae hatch from eggs passed in the stool of infected persons. The larvae usually penetrate the skin of people walking barefoot over contaminated soil. After penetrating the skin, larvae enter the bloodstream, are carried to the lungs, exit through alveolar walls, migrate up the tracheo-bronchial tree and are swallowed. It takes approximately 2 months for larvae to reach the intestine where they become sexually active and lay eggs.⁶ Our patient had manifestations of hookworm disease with tarry stools and anaemia as early as the 2nd week of life. Tarry stools were not detected after 3 days of antihelminthic treatment. A similar, dramatic change in the appearance of stools after antihelminthic treatment has been reported previously.⁷

Transplacental and transmammary routes of transmission of hookworm larvae have been postulated in the past,^{8,9} but no proof has been demonstrated. It is possible that hookworm larvae, crossing from the breast directly into the infant's gut, could have a shorter incubation period. Nwosu examined 12.4 L of colostrum from 316 nursing mothers to look for the infective larvae of hookworm but none were detected.⁸

The other possibility is transplacental infection which would result in a longer incubation period. It is conceivable that larvae that migrate from the pulmonary circulation into the alveoli can also enter the fetal circulation across the placental membrane. This form of transplacental transmission has been recorded for protozoa, namely toxoplasmosis¹⁰ and malaria.¹¹ Transplacental transmission of toxocara in mice has also been proven.¹²

Infants who acquire hookworm might otherwise acquire it through the standard route, after penetrating the skin. Infantile hookworm infection acquired from contaminated sand used in absorbent nappies in China and also from laying the child in soil

contaminated with hookworm has been reported previously.⁵ In this scenario, the incubation period needs to be 6-8 weeks. That the symptoms in our case appeared at 2 weeks of age argues against this possibility. We speculate that the neonate must have become infected transplacentally. There are anecdotal reports from China of hookworm infection at birth.¹ Other than that, the youngest case we found in the literature was aged 5 weeks and from Australia.¹³

The passage of altered blood by our patient made us suspect Meckel's diverticulum. Two cases of infantile hookworm operated upon for intestinal bleeding before the causative hookworm was detected have been reported.¹³ This latter report is mentioned above as probably the youngest case in the literature (5 weeks).

Our infant is perhaps the youngest with neonatal hookworm disease to be reported and manifestation within 2 weeks of birth supports transplacental spread. We recommend that hookworm be borne in mind in the differential diagnosis of neonates and infants with rectal bleeding and severe anaemia in hookworm-endemic areas.

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