Nigerian Medical Journal
NIGERIA MEDICAL ASSOCIATION
Print ISSN 0300-1652, E-ISSN 2229-774X

Case Report

Gastrointestinal Ascariasis: Unusual Presentation in 2 Cases

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Abstract

Ascariasis is a helminthic infection caused by the nematode *Ascaris lumbricoides* and remains the most prevalent helminth infection worldwide. Although typically asymptomatic or limited to intestinal symptoms, intestinal ascariasis can present unusually as demonstrated in these two cases.

The first case involves a 73-year-old male who was being treated for non-steroidal anti-inflammatory drug (NSAID) - induced upper gastrointestinal bleeding (UGIB) and was incidentally found to harbor Ascaris worms in the duodenum during endoscopy. The second case describes a 63-year-old male with decompensated liver cirrhosis who began vomiting and passing large quantities of Ascaris worms while hospitalized. Both patients responded excellently to anthelminthic therapy.

The first case highlights the need to consider ascariasis in the differential diagnosis of upper gastrointestinal bleeding, either as a primary cause or in conjunction with other causes of upper GI bleeding. The second case underscores the importance of considering biliary ascariasis or ascariasis co-infection in patients with liver cirrhosis.

Ascariasis continues to pose a significant public health challenge. Effective preventive strategies such as improved sanitation, enhanced personal hygiene, and routine deworming programs are crucial for reducing the disease burden and averting potentially severe complications.

Keywords: Ascariasis, Upper Gastrointestinal bleeding (UGIB), Liver cirrhosis

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How to Cite Duruewuru PU, Chukwurah SN, Nnemelu PO, Nduaguba ST, Nwafor NE, Ogbonna EK, Ilika VC. Gastrointestinal Ascariasis: Unusual Presentation in 2 Cases. Niger Med J 2025; 66 (6): 2447-2456 https://doi.org/10.71480/nmj.v66i6.1074

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Introduction

Ascariasis is a helminthic infection in humans caused by the nematode *Ascaris lumbricoides.* The primary habitat of the adult worms is the jejunum, which, when it is infected, is known as intestinal ascariasis.[1] It is one of the major soil-transmitted helminths (STHs), along with *Trichuris trichiura* and hookworms (*Necator americanus* and *Ancylostoma duodenale*). Collectively, these parasites are responsible for infecting over 1.5 billion people globally—representing nearly one-quarter of the global population. [2] The global prevalence of Ascariasis is estimated at 11.01%.[3] Local studies done showed that *Ascaris lumbricoides* was the most prevalent helminth in the Southwestern (21%) and South-southern (13%) parts of Nigeria, while hookworm was more prevalent in south eastern Nigeria (19%).[4]

Adult Ascaris worms reside in the small intestine, where female worms release hundreds of thousands of eggs daily. Once these eggs mature in the environment and become infective, they are ingested by humans. The larvae hatch in the gut, migrate to the lungs via the bloodstream, ascend the respiratory tract, and are eventually swallowed back into the intestines, where they mature into adult worms. These infections are primarily transmitted through the ingestion of water or food contaminated with Ascaris eggs. They are most prevalent in tropical and subtropical regions, particularly in areas with poor sanitation and hygiene.[3] Other predisposing factors for ascariasis include age—particularly in children under the age of five—poverty, and host genetic susceptibility.[6,7]

The majority of patients with intestinal ascariasis are asymptomatic and usually have a low worm burden. Those with heavy infestations can present with symptoms that include anorexia, nausea, vomiting, bloating, abdominal discomfort, recurrent abdominal pain, abdominal distension, and diarrhea.[2,6] They can also present with complications, which include intestinal obstruction, biliary colic, recurrent pyogenic cholangitis, cholecystitis, Löeffler syndrome, obstructive jaundice, cholelithiasis, pancreatitis, and malnutrition.[6]

Upper gastrointestinal bleeding is an uncommon manifestation of Ascaris lumbricoides infection; however, in rare instances, it may occur due to complications or atypical presentations.[8,9]
We present two cases of intestinal ascariasis with unusual presentations

Case Reports

Case 1

A 73-year-old retired civil servant presented to the accident and emergency department with vomiting of blood for 24 hours. Vomitus contained both fresh and altered blood with clots. He had about 5 episodes with a total estimated blood loss of about 700mls. There was associated dizziness, generalized body weakness, and melena. He had a positive history of chronic NSAID use for arthritis of the left knee. He had never been previously diagnosed with peptic ulcer disease. There was no history suggestive of chronic liver disease, and he does not take alcohol or tobacco in any form.

On examination, the patient was pale with bilateral pitting pedal edema. Pulse rate was 96b/m and blood pressure was 130/80mmHg. Abdominal examination revealed no abnormalities, and DRE showed the examining finger stained with melena.

Urgent Packed cell volume (PCV) done was 23%, and the eosinophil count was normal. His abdominal ultrasound scan showed no abnormal findings.

Impression was upper GI bleeding secondary to NSAID-induced gastritis.

Upper GI endoscopy revealed pangastritis with ascariasis infestation (2 live worms were noted). Following the procedure patient was commenced on Tabs Albendazole 400mg stat, triple therapy for H.pylori eradication, and cocodamol PRN. He was asked to stop NSAIDS and was discharged when he was stable. During subsequent follow-up visits, he was noted to be stable with no other episode of GI bleeding noted. Stool microscopy, culture, and sensitivity (MCS) performed after discharge revealed no parasites or eggs.

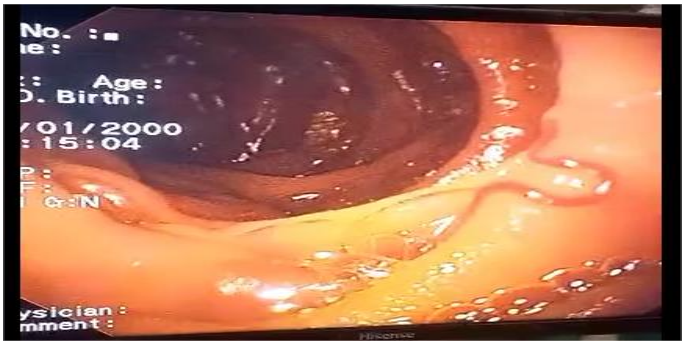
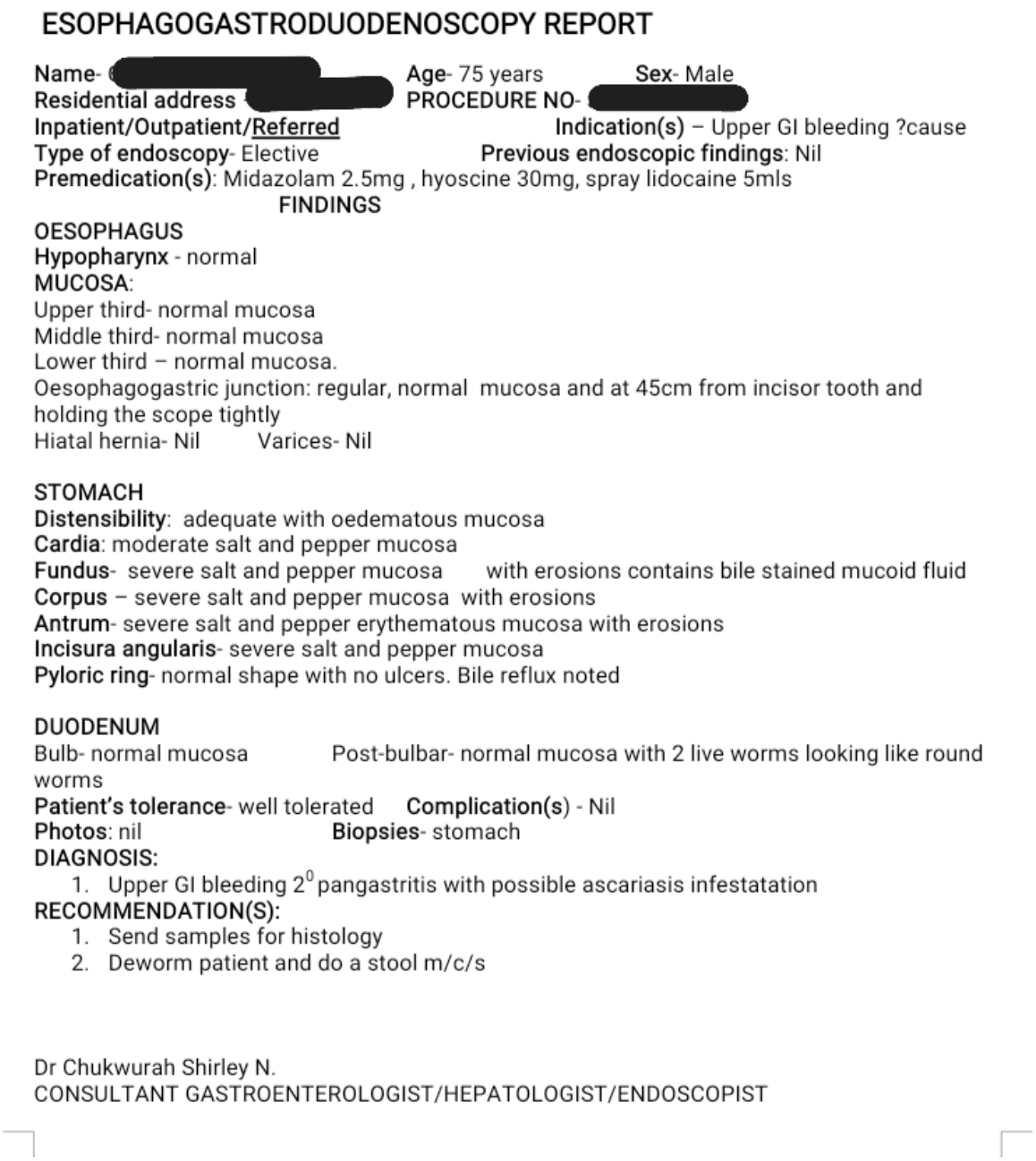


Figure 1: Endoscopic image showing live *Ascaris lumbricoides* in the duodenum

Preliminary Investigations	**Serum electrolytes, Urea, and Creatinine**	**Liver function test**	**Full Blood Count**	**Urinalysis**	**Others**
Anti-HCV: Non-reactive	Na- 140 (135-145) mmol/l	Total Bilirubin - 13.6 (0 – 21) umol/L	RBC = 2.5 x 10^6/uL (4-6.2)	**Urinalysis:** Appearance- Pale amber and turbid	**Total Protein:** 68 (66 – 83) g/l
HBSAg: Non-reactive	K- 4.2 (3.5 – 5.5) mmol/l	Direct Bilirubin- 1.9 (0 – 3.4) umol/L	HGB = 7.6 (11-17) g/dl	Ascorbic acid- Nil WBC- Nil	**Serum Albumin:** 55 (62 – 80) g/l
HIV Screening: Negative	Cl- 114 (96 – 106) mmol/l	ALT- 38 (1 - 45) IU/L	WBC = 4.02 x 10^9/L (4-12)	PH- 8.0 Glucose- Nil	

RBG: 6.7 (3.9 – 6.7) mmol/l	HC03- 21 (21 – 31) mmol/l	AST- 49 (0 - 40) IU/L	Neutrophils: 46.8% (50-80)	Protein- Nil Blood- +	
PCV: 0.23 (.38 - .53) L/L	Urea- 12.1 (2.8 – 7.2) mmol/l	ALP- 113 (0 – 270) IU/L	Lymphocytes: 37.6% (25-50)	Bilirubin- Nil Urobilinogen- Normal	
	Crea- 130 (70 – 115) umol/l		Monocytes: 9.9% (2-10)	Cast- Nil Nitrite- Negative	
			Eosinophils: 3 % (0-5)	Ketones – Nil RBC- >25	
			Platelets: 250 x 10^9/L (150-400)		

Figure 2- Investigation table



ESOPHAGOGASTRODUODENOSCOPY REPORT

Name- [redacted] Age- 75 years Sex- Male
Residential address [redacted] PROCEDURE NO- [redacted]
Inpatient/Outpatient/Referred Indication(s) – Upper GI bleeding ?cause
Type of endoscopy- Elective Previous endoscopic findings: Nil
Premedication(s): Midazolam 2.5mg , hyoscine 30mg, spray lidocaine 5mls

FINDINGS

OESOPHAGUS
Hypopharynx - normal
MUCOSA:
Upper third- normal mucosa
Middle third- normal mucosa
Lower third – normal mucosa.
Oesophagogastric junction: regular, normal mucosa and at 45cm from incisor tooth and holding the scope tightly
Hiatal hernia- Nil Varices- Nil

STOMACH
Distensibility: adequate with oedematous mucosa
Cardia: moderate salt and pepper mucosa
Fundus- severe salt and pepper mucosa with erosions contains bile stained mucoid fluid
Corpus – severe salt and pepper mucosa with erosions
Antrum- severe salt and pepper erythematous mucosa with erosions
Incisura angularis- severe salt and pepper mucosa
Pyloric ring- normal shape with no ulcers. Bile reflux noted

DUODENUM
Bulb- normal mucosa Post-bulbar- normal mucosa with 2 live worms looking like round worms
Patient's tolerance- well tolerated Complication(s) - Nil
Photos: nil Biopsies- stomach
DIAGNOSIS:
1. Upper GI bleeding 2^0 pangastritis with possible ascariasis infestatation

RECOMMENDATION(S):
1. Send samples for histology
2. Deworm patient and do a stool m/c/s

Dr Chukwurah Shirley N.
CONSULTANT GASTROENTEROLOGIST/HEPATOLOGIST/ENDOSCOPIST

Figure 3- Upper GI endoscopy Report

Case 2

A 63-year-old farmer who presented to the Accident and Emergency department on account of abdominal swelling of 6 months duration. He also had a positive history of jaundice, anorexia, easy satiety, and weight loss. There was no change in bowel habit. He had a positive history of significant alcohol intake; however, he does not take tobacco or herbal drugs. He is not known to have diabetes or hypertension.

On examination, the patient was chronically ill-looking, pale, and had bilateral pitting leg edema. The liver was shrunken with a span of 7cm, and massive ascites was demonstrated by a fluid thrill. Digital rectal examination findings were unremarkable.

Investigation results showed anemia (Hb-6.8g/dl) and a normal eosinophil count. Hepatitis B, C, and HIV screening were all non-reactive. Liver function test showed mild elevations in the ALT, AST, and ALP. Ultrasound scan revealed a shrunken liver with gross ascites.

He had hypoalbuminemia and hypoproteinemia, while alpha-fetoprotein was normal.

An impression of Decompensated Liver Cirrhosis secondary to Alcoholic liver disease was made. Child Pugh score was 9 (Class B).

Patient was subsequently commenced on Intravenous dextrose infusion with vitamin B complex, Frusemide, Spironolactone, Lactulose, and Norfloxacin.

On the fifth day of hospitalization, while receiving treatment, the patient began to vomit. About 2 episodes were observed, and the vomitus was noted to contain large amounts of adult worms. About the same time, he also began passing large quantities of worms per rectum, with approximately four episodes observed. Morphological examination of the expelled worms confirmed them to be *Ascaris lumbricoides.*

The patient was then commenced on Tabs Mebendazole 100mg twice daily for 3 days. Following the commencement of the medication, the vomiting and passage of worms stopped a few days later. Stool microscopy, culture, and sensitivity (MCS) performed revealed no parasites or eggs. The patient was subsequently discharged to be followed up at the clinic after he improved. However, the patient was lost to follow-up

Figure 4: The viable ascarids seen in the stool of the patient

Preliminary Investigations	**Serum electrolytes, Urea, and Creatinine**	**Liver function test**	**Full Blood Count**	**Urinalysis**	**Others**
Anti-HCV: Non-reactive	Na- 130 (135-145) mmol/l	Total Bilirubin - 11 (0 – 21) umol/L	RBC = 2.4 x 10^6/uL (4-6.2)	**Urinalysis:** Appearance- Pale amber and clear	**Total Protein:** 52 (66 – 83) g/l
HBSAg: Non-reactive	K- 2.7 (3.5 – 5.5) mmol/**l**	Direct Bilirubin- 3.8 (0 – 3.4) umol/L	HGB = 6.8 (11-17) g/dl	Ascorbic acid- Nil WBC- 0-1	**Serum Albumin:** 32 (62 – 80) g/l
HIV Screening: Negative	Cl- 90 (96 – 106) mmol/l	ALT- 46 (1 - 45) IU/L	WBC = 4.5 x 10^9/L (4-12)	PH- 5.0 Glucose- Nil	**PT**- 16.5 (11 -16) **INR**- 1.21 (0.8 – 1.2)
RBG: 4.2 (3.9 – 6.7) mmol/l	HC03- 27 (21 – 31) mmol/l	AST- 55 (0 - 40) IU/L	Neutrophils: 54 % (50-80)	Protein- Nil Blood- Nil	**Alpha Feto Protein**: 4.7 (0 – 10) mg/ml
PCV: 0.29 (.38 - .53) L/L	Urea- 2.8 (2.8 – 7.2) mmol/l	ALP- 282 (0 – 270) IU/L	Lymphocytes: 38 % (25-50)	Bilirubin- Nil Urobilinogen- Normal	**Ascitic Fluid MCS:** WBC- 0-1, RBC 3-5, Epithelial cell- Nil Culture yielded no growth

					after 24 hours of incubation
	Crea- 74 (70 – 115) umol/l		Monocytes: 1% (2-10)	Cast- Nil Nitrite- Negative	**Ascitic Fluid Albumin**: 23
			Eosinophils: 0.5% (0-5)	Ketones – Nil RBC- Nil	
			Platelets: 153 x 10^9/L (150-400)		

Figure 5- Investigation table

Discussion

These two cases highlight the unusual presentation of gastrointestinal ascariasis. The first case was a patient with upper GI bleeding thought to be due to NSAIDS use, but was incidentally found to have Ascaris worms. Two live worms were seen in the second part of the duodenum, and the endoscopy finding included gastric erosions as well as salt and pepper features of gastritis. Intestinal ascariasis does not typically cause upper GI bleeding, but when it does, the possible pathogenesis may be due to mechanical trauma of adult ascaris migration in the stomach, or it may be due to gastric mucosal irritation by the worm's secretion.[10] Our report is similar to a case reported in 1983 in a young Sri Lankan female presenting with upper GI bleeding, where several live worms were seen in the antrum with antral lesions on gastroscopy.[10]

The second case was that of a patient with decompensated liver cirrhosis who vomited and excreted large quantities of ascaris worms. Though there are paucity of similar cases in the literature, Ascariasis has been well known to cause hepato-biliary conditions in which worms migrate into the liver and bile ducts, potentially exacerbating liver damage in individuals with pre-existing conditions like cirrhosis.

This may lead to serious complications such as biliary bleeding, liver abscess formation, gallstone development, gallbladder rupture, and peritonitis. This could be seen as an infestation that can worsen liver disease in a patient with already impaired immunity. Abdominal CT Scan and/or Endoscopic retrograde cholangiopancreatography (ERCP) could have been offered to the patient if symptoms did not improve. The large burden of worms observed in this patient could be a result of possible background immunosuppression associated with chronic liver disease.[11]

Ascariasis is typically diagnosed by identifying characteristic eggs in stool samples using microscopy. In some cases, adult worms may be expelled in faeces, as seen in our second patient, or more rarely, migrate and exit through the mouth, nose, or rectum.[12]

Visual inspection and morphological examination of the worms are diagnostic. Abdominal CT scan, MRI, and Endoscopic retrograde cholangiopancreatography (ERCP) are frequently employed in cases of biliary ascariasis for diagnostic purposes. ERCP additionally has therapeutic value, as it enables the removal of *Ascaris* worms from the biliary system.[13]

Anthelminthic therapy is recommended for all individuals with ascariasis, regardless of symptom presence, to reduce the risk of complications associated with worm migration. Albendazole and Mebendazole are the first-line agents for treating children and non-pregnant adults, while Pyrantelpamoate is considered safe and effective for use during pregnancy. Surgical intervention may sometimes be required for complicated cases.[14]

Conclusion

This report describes two rare cases of ascariasis complicated by upper gastrointestinal bleeding and liver cirrhosis, in which adult Ascaris lumbricoides worms were found in the gastrointestinal tract, causing symptoms that were treated with Albendazole and Mebendazole, respectively.

The first case highlights the importance of considering helminthic infections in the differential diagnosis of gastrointestinal bleeding, as an atypical presentation, though uncommon but may occur. Ascariasis may also coexist with other causes of upper gastrointestinal bleeding, further complicating clinical assessment.

The second case reveals the need to consider biliary ascariasis as a possible differential in individuals with cirrhosis. In patients with chronic liver disease, ascariasis co-infection can occur due to immune suppression, which further worsens the clinical course.

Ascariasis remains prevalent in our environment; therefore, regular deworming should be strongly encouraged. Every patient encounter at a healthcare facility should be used as an opportunity to inquire about the patient's deworming history and administer treatment if it has not been done. This approach aligns with the World Health Organization’s recommendation of deworming two to three times annually for individuals living in endemic regions.

In addition, public health measures such as improved sanitation, safe waste disposal, and good personal hygiene are essential for reducing the burden of ascariasis and preventing its associated complications.

Declaration Of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial Support And Sponsorship

Nil.

Conflicts Of Interest

There are no conflicts of interest.

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