Chapter

High-Frequency Ultrasound Imaging of the Intestine in Normal Subjects and Patients with Intestinal Parasites

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Abstract

High-frequency ultrasound imaging was used to evaluate the intestinal walls of the duodenum and colon in patients with intestinal parasitic infections. Ultrasound images were obtained from 100 consecutive patients with symptomatic intestinal parasitic diseases and 40 healthy controls. High-frequency annular array transducer of 7.5 MHz was used to obtain B-mode ultrasound gray-scale and color images of the duodenum and colon with and without water contrast. The diagnosis of parasitic infections was based on clinical presentation, serial stool microscopy, and finding of parasites in duodenal aspirates. We demonstrated normal duodenum and colon echoanatomy in control subjects. In patients with giardiasis, the lesions of the duodenum and colon were associated with increased dimensions and wall thickness compared to healthy controls ($p < 0.05$). The ultrasound features of giardial lesions were characterized by increased wall echogenicity, flattening or loss of duodenal folds, and/or colonic haustration, hyperechoic floating foci demonstrating chaotic motility, increased perilesional tissue echogenicity, and altered colonic peristalsis. In amebic lesions there were hyperechoic floating foci with bulk motility. There is loss of wall thickness at amebic ulcer sites or wall thickening at amebic granuloma. Helminths were visualized as large hyperechoic linear or curvilinear foci with serpentine or jolting motility. In conclusion, high-frequency B-mode ultrasound imaging with water contrast demonstrated details of duodenal and colonic echoanatomy in normal subjects and patients with giardiasis.

Keywords: parasites, diarrhea, tropical diseases, water-borne diseases, gastrointestinal tract

1. Introduction: clinical problem

Intestinal parasites infect over three billion people [1]. An intestinal parasite is an organism that lives in the intestine of the host and gets its food from its host.

There are two main classes of parasites that infect the human intestine: protozoa and helminthes [2].

1.1 Intestinal protozoa

Intestinal protozoa are microscopic unicellular organisms that could be free-living or parasitic within the intestine. It could be transmitted from human-to-human through fecal-oral route usually through contaminated food or water, or person-to-person contact [2].

The intestinal protozoa are classified according to motility as:

a. Sarcodina—*Amoeba* (e.g., Entamoeba)

b. Mastigophora—flagellates (e.g., *Giardia*)

c. Ciliophora—ciliates (e.g., *Balantidium*)

1.2 Intestinal helminthes

Intestinal helminthes are macroscopic large, multi-cellular organisms that are visible to the naked eye in adult stage in free-living or parasitic condition in the intestine [2].

There are three main groups of human intestinal helminthes:

1. Platyhelminths or flatworms include trematodes (flukes) and cestodes (tapeworms, e.g., *Taenia saginata*).

2. Thorny-headed worms (acanthocephalins) that infect the intestine in humans.

3. Roundworms include nematodes (e.g., *Ancylostoma duodenale* and *Ascaris lumbricoides*) in which adult forms infect the intestine.

2. Ultrasound classification of features of intestinal parasites

The use of ultrasound to study the features of intestinal parasites has been referred to as high-frequency ultrasound duodenography and colonography with and without water contrast [3, 4]. The ultrasound classification of the features of intestinal parasites is based on:

1. Ultrasonic reflector

2. Hyperechoicity

3. Motility

4. Changes in intestinal wall thickness.

2.1 Ultrasonic reflector

1. Directly visible parasites—e.g., Helminths

2. Indirectly visible parasites on "floaters" (formed by intestinal inclusions)—e.g., *Amoeba* and *Giardia*.

2.2 Hyperechoicity

1. Hyperechoic floating foci (HFF) showing small echogenic particles in floatation, e.g., *Giardia*.

2. Hyperechoic curvilinear foci show a large echogenic curved and/or linear independent objects, e.g., *Ascaris*.

2.3 Motility

1. Bulk motility by slow active movement of the large mass of the lesion at the same time in the same direction independent of gastrointestinal motility—e.g., *Amoeba*.

2. Chaotic motility by fast active movement of singular or small groups of the lesion in different directions independent of gastrointestinal motility, e.g., *Giardia* and *Balantinum*.

3. Jolting motility by fast jerks, e.g. *Ancylostoma*.

4. Serpentine motility by serpentine-like movement, e.g. *Ascariasis*.

2.4 Changes in intestinal wall thickness

1. Increased wall thickening of all layers by cystoskeletal dysfunction, e.g., *Giardia*.

2. Increased wall thickening by abscess formation, e.g., *Amoeba*.

3. No change in wall thickness, e.g., helminths.

4. Loss of tri-layer wall replaced by single layer due to cytoskeletal rearrangement caused by multiple parasitic lesions in immune-compromised patients (e.g., giardiasis in patients with HIV/AIDS).

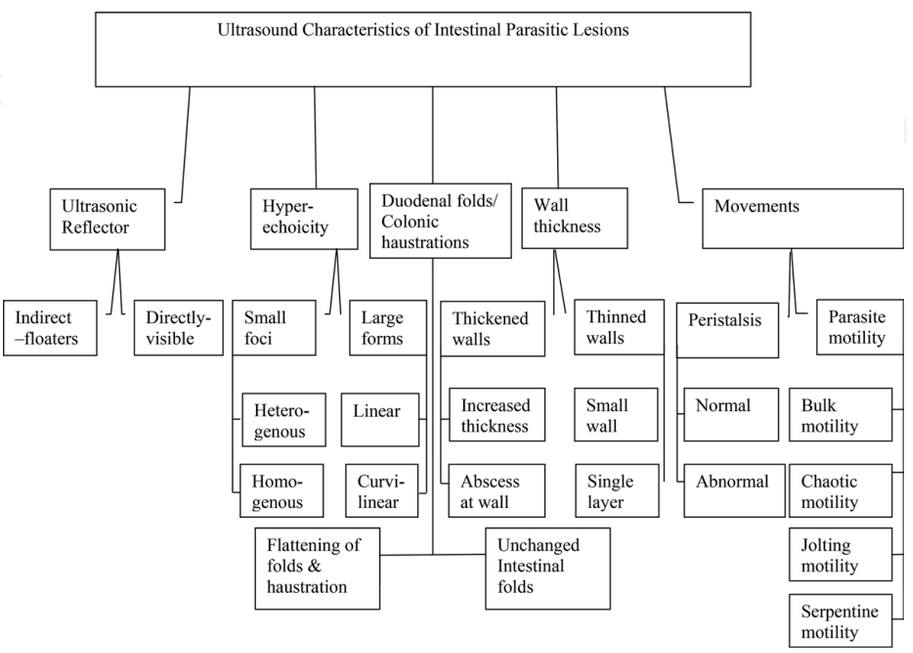

Figure 1.
Schematic diagram of the classification based on ultrasound imaging features of intestinal parasites.

2.5 Changes in duodenal folds and colonic haustrations

1. Flattening of duodenal folds and colonic haustrations, e.g., *Giardia*.

2. Thinning of duodenal folds and colonic haustrations, e.g., *Giardia* in immune compromised patients.

3. Thickening of duodenal folds and colonic haustrations, e.g., advanced disease in giardiasis and amebiasis.

Figure 1 shows the schematic diagram for ultrasonic characterization of the lesions caused by the different types of common intestinal parasites (protozoan and helminthes).

3. Technique of high-frequency ultrasound duodenography and colonography

3.1 Patient examination

To evaluate the intestine, patient preparation is needed. All ultrasound examinations are performed with and without water contrast after overnight fasting (for at least 16 h) using standard scanning procedure [4]. Water contrast imaging is performed by having adult subjects take at least 1 L of water prior to examination. Subject examination is performed in the supine horizontal, left-posterior oblique, and left-lateral decubitus positions using the intercostal and subcostal approaches. The internal organs, including liver, gall bladder, spleen, pancreas, duodenum, colon, and kidneys, are routinely evaluated in all subjects. The abdominal ultrasound examination are performed using B-mode and color flow Doppler ultrasonography with 2.5 and 7.5 MHz annular array transducers of a duplex color flow Doppler ultrasound system (Agilent HP/Philips SONOS 5500, Philips Medical Systems, Cambridge, MA, USA). The examination begins with deeply located abdominal structures using 2.5 MHz probe. The detailed examination of duodenal walls and folds is performed using 7.5 MHz probe beginning in the right hypochondriac (1) and epigastric (2) regions (see **Figure 2**). This is followed by the examination of the colonic walls and haustra [3, 4] of the ascending colon starting from the McBurney's point that lies one-third of the distance laterally on a line drawn from the umbilicus to the right anterior superior iliac spine. The examination proceeds upward to the right lumbar (4), right hypochondriac (1), and turning clockwise to the epigastric (2), left-hypochondriac (3), left-lumbar (6), and left-iliac (9) regions (see **Figure 2**).

Color flow Doppler sonography is performed to examine the localization of lesions in relation to vessels and body abdominal cavities. All ultrasound studies including measurements and grading of echogenicity are performed by a trained sonographer using built-in software [4]. Measurements are taken between peristaltic waves on a water contrast image.

3.2 Sonographic findings in normal duodenum

The duodenal wall is visualized as alternate bands of moderately echogenic mucosa with hyperechoic core submucosa which are thrown into folds of Kerckring, arranged circularly, a middle hypoechoic muscularis layer and an outer hyperechoic serosa layer [3, 4]. **Figure 3** shows the measurement end-points including wall thickness in the duodenum (with water contrast) (**Figure 3**; within double blue

arrow ends), measured between two mucosal folds of Kerckring [4] (**Figure 3**; white arrow heads), from the surface of the moderately echogenic mucosa, through the hyperechoic submucosa (**Figure 3**; red arrow) and hypoechoic muscularis (**Figure 3**; within double brown arrow ends) to the hyperechoic serosa layer (**Figure 2**; bottom red arrow head). The wall thickness of the duodenum is 3.5 ± 2.2 mm [4].

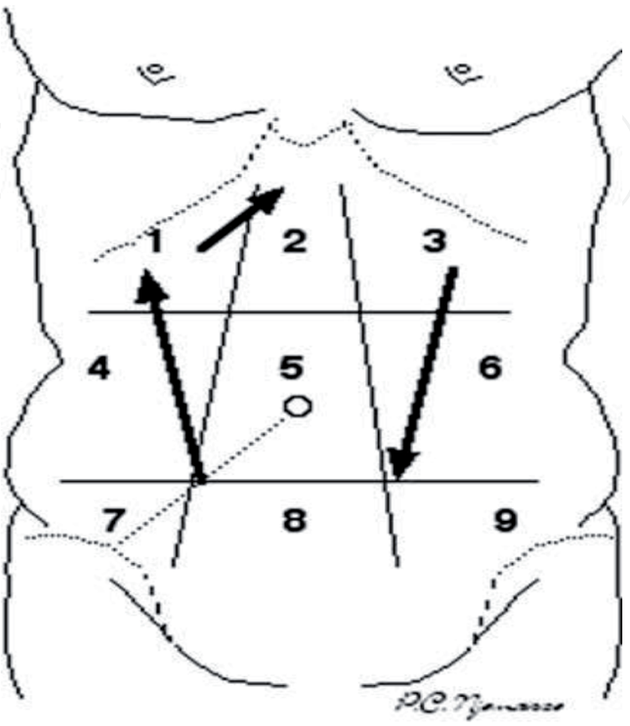

Figure 2.
Locations for placement of transducer for examination of the intestine.

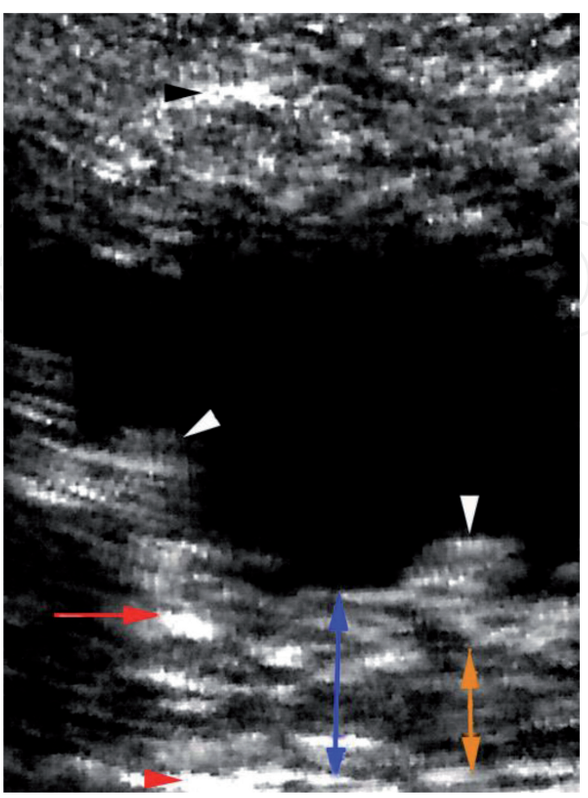

Figure 3.
Measurement end-points of the duodenum with water contrast.

3.3 Sonographic findings in normal colon

Figure 4A shows the colonic wall comprising alternate hypoechoic and hyperechoic bands corresponding to the histological layers. The latter comprises a moderately echogenic mucosa, a hyperechoic core submucosa, a hypoechoic muscularis layer, and an outer hyperechoic serosa [3, 4]. The outer layer of the longitudinal muscle in the colon demonstrated a relatively hyperechoic *tenia coli librae*. The haustra are isoechoic with the mucosa. The wall thickness of the colon (**Figure 4A**; with water contrast), (**Figure 4A**; within double blue arrow ends) is measured between two haustra (**Figure 4A**; white arrow heads), from the surface of the moderately echogenic mucosa (**Figure 4A**; top arrow of the double blue arrows), through the hyperechoic submucosa (**Figure 4A**; brown arrow), and hypoechoic muscularis (**Figure 4A**, within double brown arrow ends) layers, to the hyperechoic serosa layer (**Figure 4A**; bottom brown arrow head); and diameter measurement is taken from near wall serosa (**Figure 4A**; top brown arrow head) to far wall serosa (**Figure 4A**; bottom brown arrow head). The measurement cursor line is aligned perpendicular to the echogenic *tenia coli librae* (**Figure 4A**; cursor line between brown arrow heads) which runs midway between the near and far wall serosa in long axis view of the ascending and descending colon. The ascending colon diameter is 32.0 ± 13.3 mm and wall thickness is 3.9 ± 1.4 mm [4]. The descending colon diameter is 30.7 ± 8.5 mm and wall thickness is 3.8 ± 0.8 mm [4]. **Figure 4B** (white arrows) shows the haustra with pyramidal shape, regular contour, homogenous and spaced at regular intervals [3, 4].

3.4 Normal colonic peristalsis

Colonic peristalsis was observed sonographically in control subjects using real-time images taken with water contrast. Local movements of the colon aid the absorption of water and help to form feces by providing a kneading action. The peristaltic movements are brought about by contractions of segments of circular muscles and

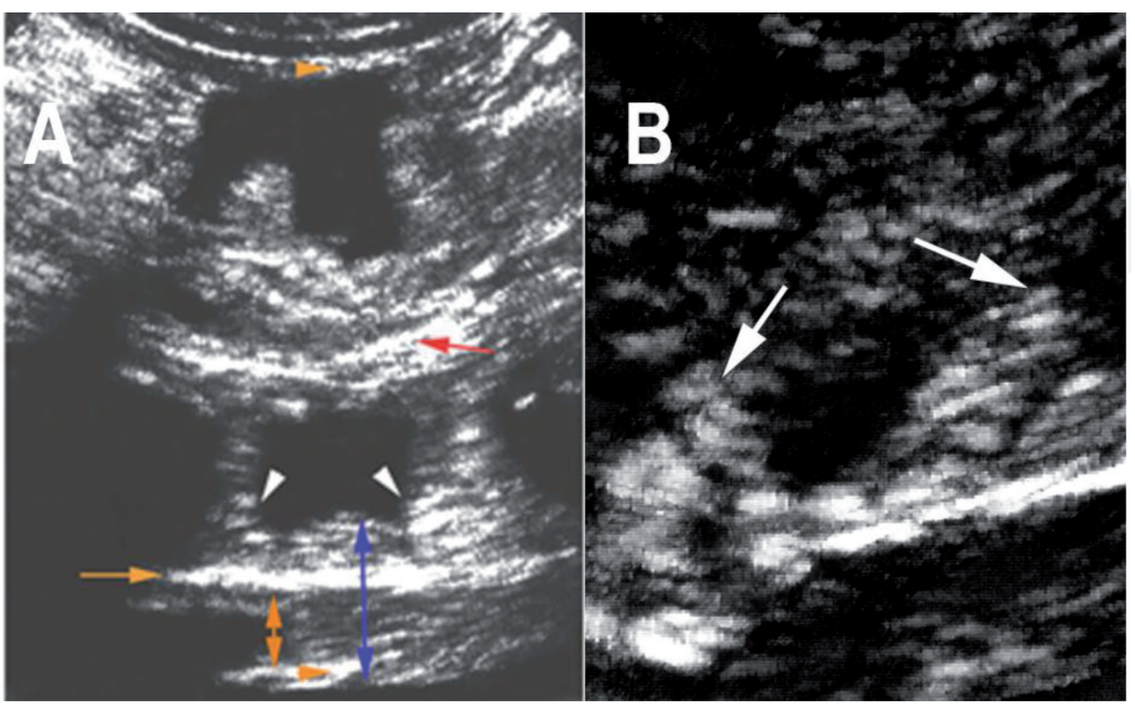

Figure 4.
Measurement end-points in the ascending and descending colon with water contrast (A), and pyramidal shaped haustra in the ascending colon (B, white arrows).

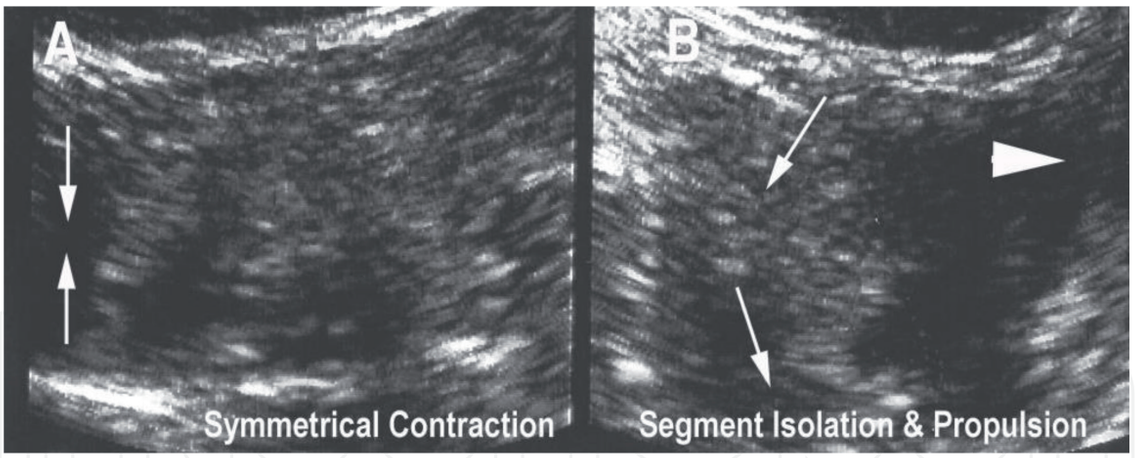

Figure 5.
Normal colonic peristalsis showing symmetrical contraction rings (A, white arrows) that isolate the segments followed by antegrade propulsion of colonic inclusions (B, white arrow head).

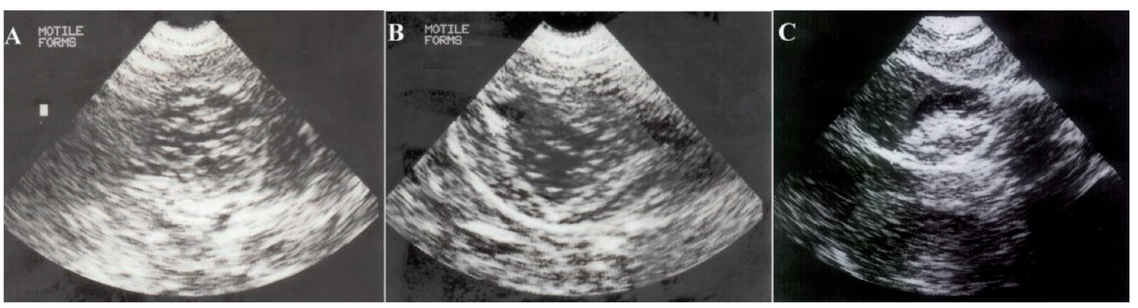

Figure 6.
Shows hyperechoic floating foci within the duodenum without water contrast (A), and with water contrast (B); while there is a condensed hyperechoic foci with bulk motility of Amoeba with water contrast (C).

the adjacent portions of the *tenia coli* [4]. The latter movements are termed segmentations; they produce folds of the wall known as haustra (**Figure 5A**; white arrow heads). During peristalsis, there are circumferential symmetrical contraction rings (**Figure 5A**; white arrows) formed between adjacent haustra, and sequential segment isolation from the rest of the gastrointestinal tract (GIT) followed by antegrade propulsion of content (**Figure 6B**; white arrows) [4]. Contractions of the smooth muscle in the colonic walls produced a rise in intraluminal pressure within the isolated chamber [5, 6]. This is followed by relaxation of one of the two rings enclosing a segment, and results in peristaltic propulsion of the colonic inclusions (**Figure 5B**; white big arrow head). The walls maintain symmetric contours during contraction (**Figure 5A**; white arrows) and relaxation (**Figure 5B**; white arrows) [4].

3.5 Hyperechoic floating foci with chaotic and bulk motility

The parasitic lesions are located in the duodenum and colon and verified by morphology in stool analysis [6–9]. The microscopic protozoan parasites could not be imaged directly with ultrasound, but could be seen indirectly as they float on intestinal inclusions called floaters which reflect the ultrasound waves [10]. The flagellated protozoan *Giardia* appear as hyperechoic small foci on floaters which are either heterogeneous or homogeneous depending on the floater substance without water contrast (**Figure 6A**), and with water contrast (**Figure 6B**) in the duodenum. Giardial lesion imaged with water contrast present as lesser echogenic HFF with chaotic motility, defined as sonographically observed rapid floatation movements in all directions by hyperechoic floating foci, between peristaltic waves (**Figure 6A, B**) [4].

The *Amoeba* with pseudopods on fatty dense floaters are echogenic and move by slow bulk motion en-mass in a given direction in helical fashion (**Figure 6C**) [4, 8]. The fatty substances as floaters are more echogenic without water contrast (**Figure 7A**) than non-fatty substances or after water contrast (**Figure 7B**).

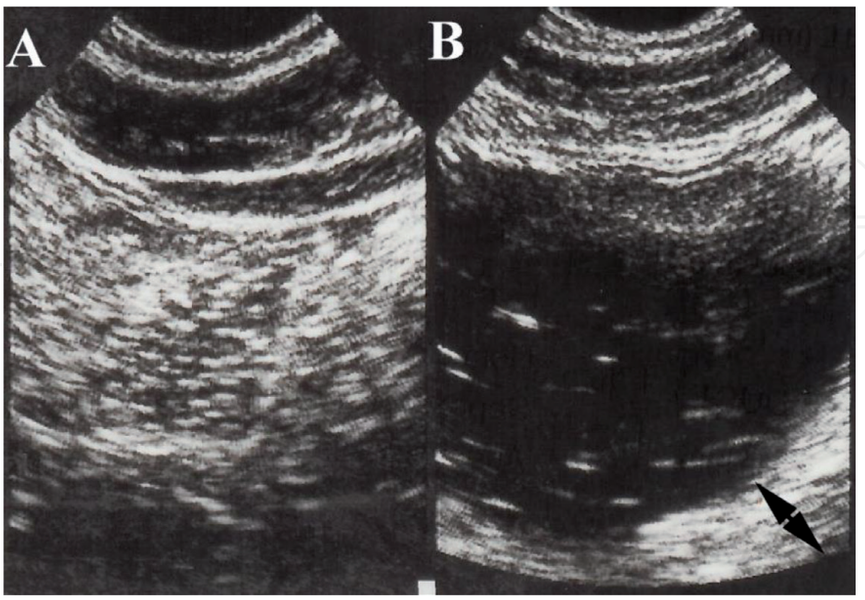

Figure 7.
The ascending colon with hyperechoic floating foci (HFF) without water contrast (A) and with water contrast the image appears hypoechoic (B). The HFF display fast chaotic motility by the flagellated Giardia on floaters.

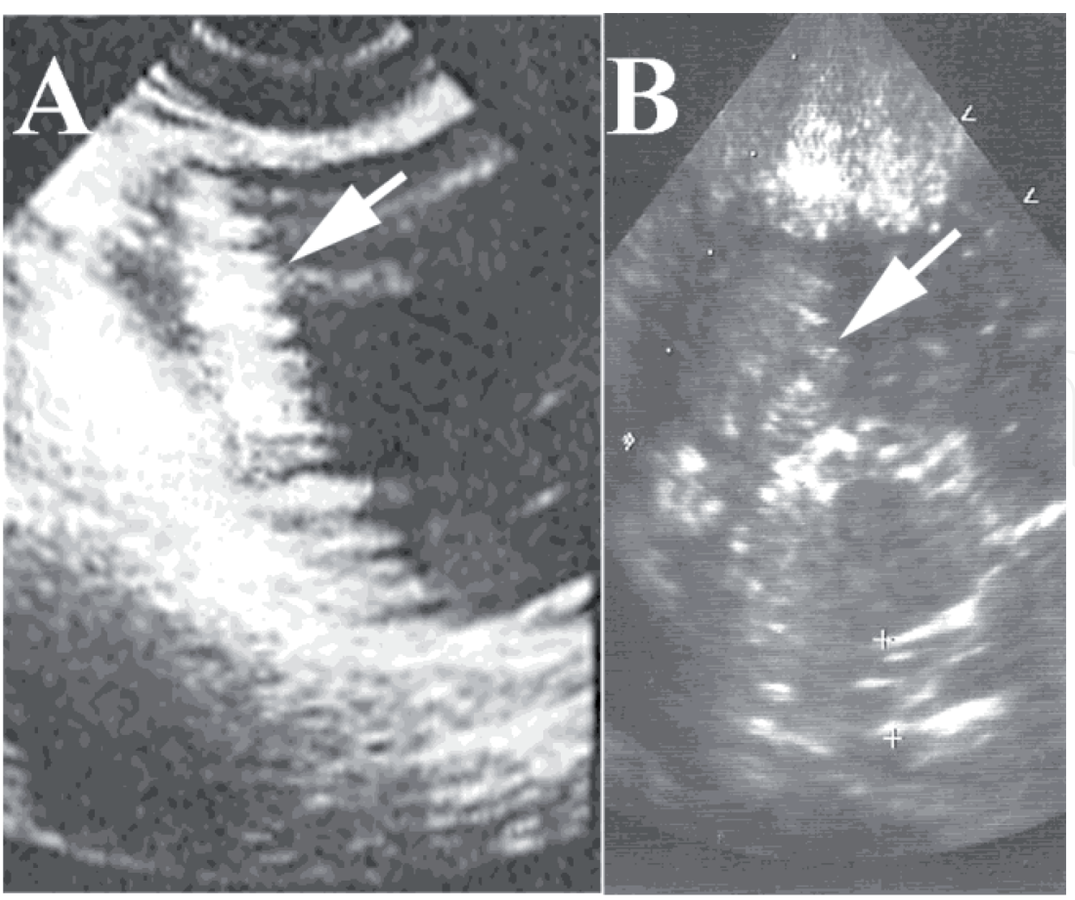

Figure 8.
Helminths parasites as large hyperechoic linear form as seen with Ascaris (A, white arrow) compared to hypercurvilinear form as seen with Taenia (B, white arrow).

3.6 Hyperechoic linear and curvilinear forms with serpentine and jolting motility

The macroscopic helminthic parasites could be visualized directly, and the body parts seen in the reflected ultrasound waves, sometimes with internal details of the gut of the worm. Some parasites may appear as large hyperechoic linear forms (HLF) with rapid spasmodic movement in what could be described as voluntary jolting motility (**Figure 8A**), seen in ascariasis [11, 12]. Other forms of helminthes are hyperechoic curvilinear forms (HCF), which display slow "serpentine" motility (**Figure 8B**), seen in teniasis [13]. The parasites were all identified by morphology in stool analysis.

4. Differential diagnosis

Table 1 shows the differential characteristics of protozoan and helminthic lesions. The protozoan parasites increase wall thickness of the layers and cross-sectional diameter of the duodenum, which occasionally caused flattening of duodenal folds of Kerckring. There is increased wall thickness of the ascending and descending colon of all three layers or at some specific sites due to the presence of amebic abscess [4]. The colonic haustrations are normal in simple infections, but could be flattened in advanced disease. The protozoan HFF could be differentiated by parasite motility. The major differential diagnosis for giardiasis is the ciliated protozoal infection—balantidiasis, that could potentially give rise to HFF. Balantidiasis [14] is caused by a ciliated protozoa—*Balantidium coli*; and is excluded based on clinical, epidemiologic, and laboratory findings. In patients with giardiasis there is absence of history of balantidial dysentery. Amebic lesion demonstrates bulk slow motility as a large mass of the lesion actively moves in one direction at a time. In contrast, giardial lesion demonstrates chaotic motility observed as rapid floatation movements in all directions between peristaltic waves. The helminthic lesion could be differentiated based on size/form and motility. *Ascaris lumbricoides* shows a large hyperechoic linear form (HLF) with jolting motility, different from *Ancylostoma duodenale* which is a relatively small HLF with jolting motility. *Taenia saginata* presents as large hyperechoic curvilinear form with serpentine motility.

4.1 Peculiarities of giardial lesions in the duodenum and colon

Giardial lesions could be distinguished by location, and changes in wall thickness, increased echogenicity of wall tissue, increased cross-sectional diameter, flattening or loss of duodenal folds and/or colonic haustration, presence of HFF with chaotic motility, presence of perilesional tissue echogenicity, and abnormal colonic peristalsis [4]. In patients with giardiasis, the duodenal wall thickness (6.3 ± 1.3 mm) is greater than that in healthy controls (**Figure 9A**, white arrow head), with loss of folds of Kerckring (**Figure 9A**, two white big arrows) and HFF (**Figure 9A**, small white arrow) [4]. In severe disease, the thickness of the duodenal wall could be several times of that seen in normal controls (**Figure 9B**, two white arrow heads), causing compression of adjacent tissues with a thin area of perilesional edema (**Figure 9B**, white arrow) [4].

In symptomatic giardiasis, the wall thickness (8.8 ± 1.4 mm) of the ascending colon is greater than that in healthy controls [3, 4]. Similarly, the wall thickness (9.2 ± 1.2 mm) of the descending colon is greater than that in healthy controls [4]. The increased wall thickness of the ascending colon is best seen with water contrast imaging (**Figure 7A**, **B**). In immune-compromised patients, giardial lesions could cause thinning of intestinal walls to only a single layer wall (**Figure 9C**) with loss of intestinal haustrations and folds (**Figure 9D**) [4].

Differential diagnosis based on ultrasound characteristics of intestinal parasites								
Group of parasite	**Genus**	**Locomotion**	**Ultrasonic reflector**	**Duodenal folds/ haustrations**	**Wall thickness**	**Hyperechoicity**	**Lesion motility**	**Intestinal peristalsis**
Protozoan	*Giardia*	Flagellates	Floaters	Flattening in advanced diseases	Thickened walls	Small foci	Chaotic motility	Abnormal retropulsion
	Amoebida	Pseudopods	Floaters	Loss at amebic ulcer sites	Amebic granuloma	Small foci	Bulk motility	Abnormal
Helminths	*Ascaris*	Serpentine	Parasite body part large	Normal	Normal	Large linear form	Jolting motility	Normal
	Taenia	Serpentine	Parasite body part is large	Normal	Normal	Large curvilinear form	Serpentine motility	Normal
	Ancylostoma	Serpentine	Parasite body part is small	Normal	Normal	Small linear form	Jolting motility	Normal

Table 1.
Differential diagnosis of protozoa and helminthes intestinal parasites.

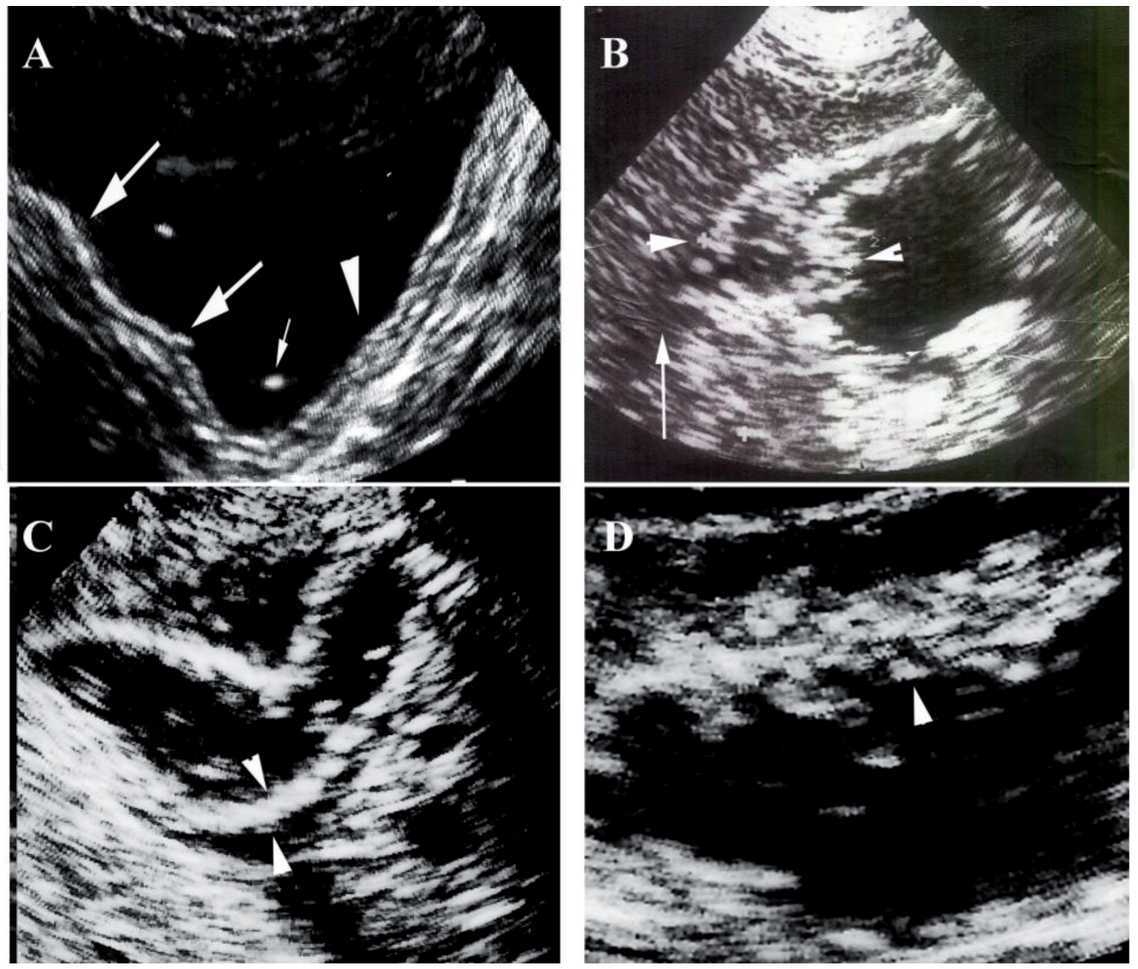

Figure 9.
Severe giardial lesions may cause duodenal wall thickening (A) with loss of folds, and in some cases there may be increased wall thickness (B). In immune-compromised patients there could be thinning of the intestinal wall to a single layer (C), and loss of colonic haustrations (D).

4.2 Abnormal colonic peristalsis in giardiasis

Colonic peristalsis could be observed sonographically with water contrast. The colon demonstrates circumferential asymmetric contraction rings that stretch the wall more on one end than the other (**Figure 10A**; white arrow heads). The giardial lesion alters the cytoskeleton of the colonic wall and the mode of intestinal peristalsis [3, 4]. The wall shows a concave contour and a "defect" during asymmetrical contraction (**Figure 10A**; top white arrow head). This is followed by relaxation and expansion into the preceding segment, in other words, by retropulsion (**Figure 10B**; white arrow) due to the defective intestinal motility [4].

In some patients, within the segment there is a dangling echogenic sheath, that falls short of the opposite wall, creating free-end septation with residual aperture (**Figure 10B**; double white small arrows). This sheath or septum is described as pseudo-haustration, since it lacked the oppositional arrangement of normal haustration [4]. Furthermore, the sheaths differ in shape, contour, and echogenicity from normal haustra. The residual aperture between the end of the sheath and colonic wall does not alter between contraction (**Figure 10A**; double white small arrows) and relaxation (**Figure 10B**; double white small arrows). This raises the possibility of partial obstruction of movement of intestinal contents by these pseudo-haustrations. The echogenicity of these pseudo-haustrations appeared similar to that of the echogenic submucosa, and showed anatomic continuity from that layer (**Figure 10B**; white small arrow head), until the sheath protrudes through

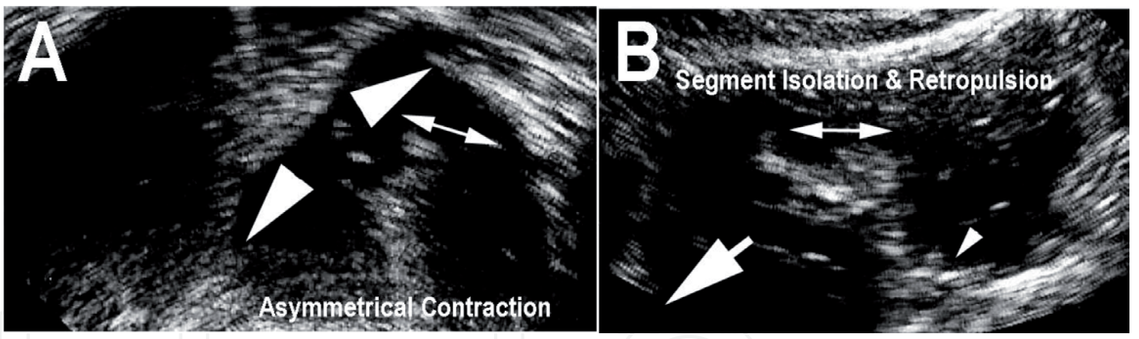

Figure 10.
Defective intestinal motility in giardiasis showing asymmetric contraction rings (A), followed by segment isolation and retropulsion (B).

the mucosal surface. This may suggest that these pseudo-haustrations derive from herniation of the submucosa through the mucosal surface (**Figure 10B**; white small arrow head) [4]. It has been suggested that a similar mechanism may result in invagination of the colonic wall after injury [6, 7].

4.3 Amebic lesions in the duodenum

In amebiasis, the duodenal wall thickness (5.4 ± 3 mm) is within normal limits [3, 4]. The wall echogenicity is usually not altered as seen in giardiasis. The folds of Kerkring have normal undulating contour. In contrast to giardiasis, in amebic lesion, HFF has increased echogenicity that moves slowly in bulk motion in helical fashion (**Figure 6C**).

4.4 Amebic lesions in the colon

Amebic lesions in the colon could be demonstrated using high-frequency B-mode ultrasound [8]. The wall thickness of the ascending colon (5.6 ± 3 mm) is marginally higher than in normal subjects but less than in patients with giardiasis [4]. There could be occasional collections demonstrated as well as delineated focal hyperechoic wall thickening lying on the mucosal surface (**Figure 11**, white arrow

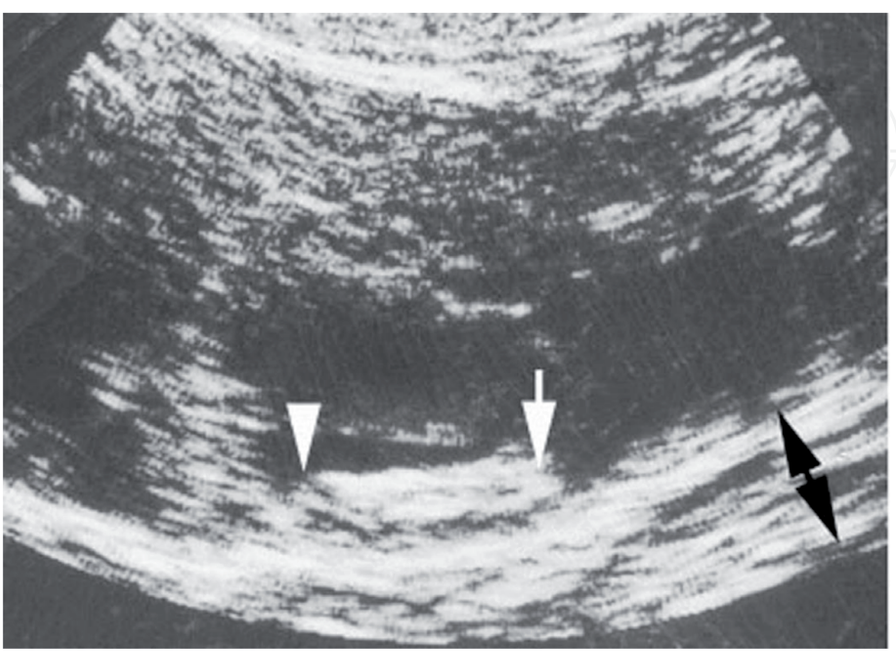

Figure 11.
Amebic abscess in the ascending colon with ameboma close to the haustra (between white arrow head and white arrow). The rest of the wall thickness remains within normal range (black double arrows).

heads). The focal thickening is associated with amebic granuloma (ameboma) and could be seen at the cecum, hepatic, and splenic flexures and sigmoid colon [4, 7]. It has been suggested that amebiasis may cause thickening of the submucosal layer due to hypervascularity of the bowel wall [8], but in contrast with generalized wall thickening observed in giardiasis, the changes in amebiasis are focal [4].

4.5 *Ancylostoma* in the duodenum

Ancylostoma duodenale and *Necator americanus* are nematode parasites that cause hookworm infection, and have a complex life cycle [9]. The mature *A. duodenale* female worms measure up to 15 mm (longer during blood meal), and the male worms up to 10 mm. *A. duodenale* could be demonstrated using high-frequency B-mode ultrasound as has been seen with gastroscopy [15]. The deep buccal cavity of the female worm could be clearly demonstrated with mouth parts and head visible in motion (**Figure 12A**, **B**, arrows). The visibility of the head and mouth parts could allow differentiation from other helminths, for example, the expected image of the male worm should show two spicules and the characteristic bursa. The mouth image of the female worm differs from the shallow buccal cavity of *Strongyloides stercoralis* [9]. The female worm become attached to the wall of the small intestine by sucking part of the mucosa into their mouth parts (**Figure 12C**), and abandoned sites (**Figure 12D**) continue to bleed [9]. The worm ingests blood from their host

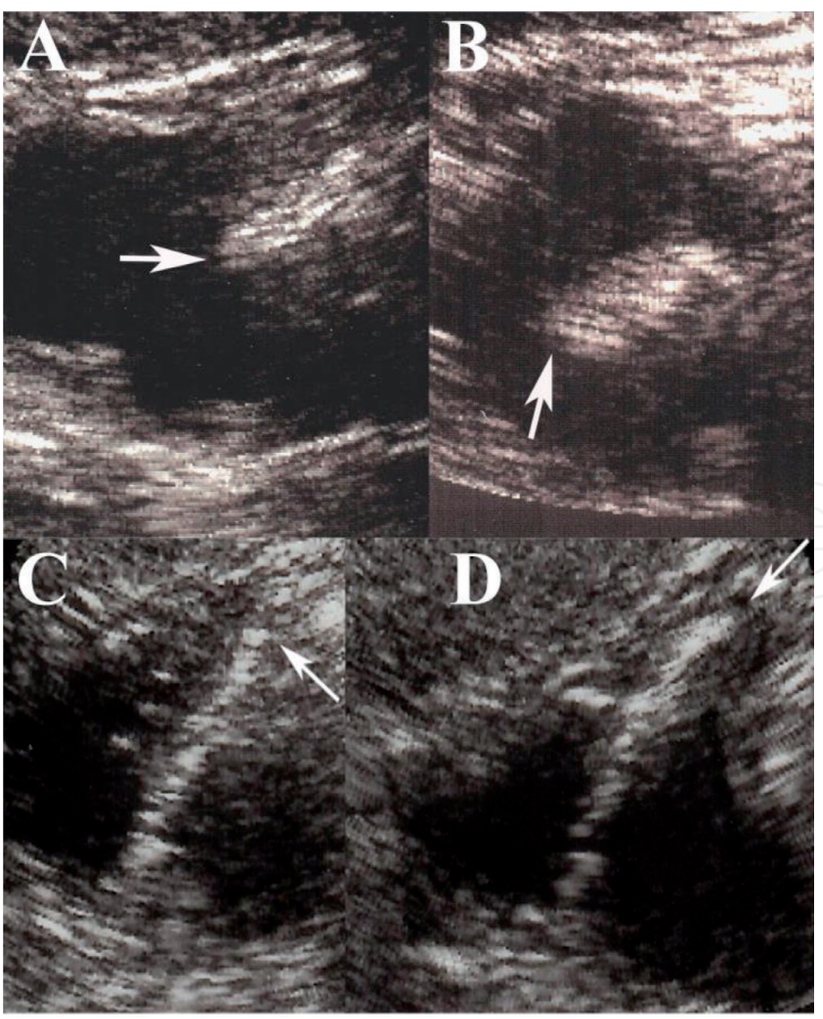

Figure 12.
Ancylostoma duodenale in the duodenum imaged in real-time with high-frequency B-mode ultrasound. The head and mouth parts move from one position (A) to another (B) to attach on the mucosal layer for blood meal (C, white arrow), and elongates and detaches thereafter (D, white arrow).

by the action of nematode anticoagulant protein c2 (NAPc2), a 85-amino acid protein with potent anticoagulant properties [16]. The process of ingestion of blood during a blood meal could be demonstrated using real-time high-frequency B-mode ultrasound. The worm is visualized in the proximal cephalid portion as a hyperechoic linear form (HLF) in active motion (**Figure 12A**, **B**, white arrow). The cephalid portion with two sharp pointed ends (buccal cavity) display jolting motility with jerking and leaping movements by spastic contractions and thickening (**Figure 12A**, **B**). The worm attaches to the mucosal surface at the duodenal near wall (**Figure 12C**). The worm could be observed attached to the mucosal layer (**Figure 12C**, white arrow) of the duodenum while ingesting blood. It could be seen elongating to about 20 mm in visible length, traversing the duodenal lumen. The caudal end lies in close proximity to the mucosal surface at the far wall. Under sonographic observation, the worm could be seen performing jolting motility, as it firmly attaches for a blood meal (**Figure 12C**, white arrow). It could be observed that as the worm elongates during a blood meal the entire visible length becomes hyperechoic. The proximal cephalid portion detaches from the mucosal surface (**Figure 12D**, white arrow) after the blood meal and the echogenicity of the worm changes with the upper one-third hyperechoic and the lower two-third hypoechoic. The latter is related to the movement of sucked blood through the gut of the worm. Stool analysis is used to reveal the eggs of *Ancylostoma duodenale* [9].

5. Common pitfalls of high-frequency ultrasound imaging of the intestine

1. Technical limitation of examination procedure due to abdominal tenderness, flatulence, or obesity.

2. Patient is unable to perform fasting due to health condition such as diabetes.

3. False negatives and false positives due to lack of proper technical access, condition of the patient, and similarity in motility patterns of the parasites.

4. Fluid filled bowel loops could alter the intestinal echoanatomy.

5. Constipation impairs bowel emptying and cause flatulence that technically impairs visualization due to fecal mass mimicking lesions in the intestine.

6. Large cystic lesions of the ovaries and fibromyoma may impair visualization of parts of the intestine.

7. Enlarged abdominal lymph nodes could also impair visualization of parts of the intestine.

8. Poor differentiations of colon cancer and lesions such as amebic abscess. However, the presence or absence of mesenteric lymph nodes and liver metastasis could aid differential diagnosis.

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