A Review of the Clinical Presentation of Dientamoebiasis

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Abstract. Among 750 symptomatic and asymptomatic patients, Dientamoeba fragilis was detected at a prevalence of
5.2% and more common than Giardia intestinalis. Most infected patients presented with diarrhea and abdominal pain
with symptoms greater than 2 weeks duration being common. Bacterial and viral causes of infection were excluded by
routine microbiological techniques. Treatment of D. fragilis infection with either iodoquinol, paromomycin, or combina-
tion therapy resulted in the eradication of the parasite and complete resolution of symptoms. Treatment failure/relapses
were associated only with the use of metronidazole. Nineteen patients were examined for pin worm, no Enterobius ver-
icularis, a proposed vector of transmission, were detected. Intermittent shedding of D. fragilis was found to be highly
variable. These studies confirm the pathogenic nature of D. fragilis and we recommend laboratories routinely test for the
organism.

INTRODUCTION

Dientamoeba fragilis is a protozoan parasite found in the
gastrointestinal tract of humans. It was first described in the
scientific literature in 1918 by Jepps and Dobell who initially
considered it as a non-pathogenic commensal.1 At that time the
organism was classified as an amoeba but subsequent antigenic
analysis, electron microscopy, and molecular studies of the
small subunit ribosomal RNA (SSU rRNA) gene have shown
that the organism is closely related to the trichomonads.2–5
Recent studies have documented the pathogenic potential of
this organism with the majority of D. fragilis-infected patients
presenting with gastrointestinal symptoms including diarrhea,
loose stools, and abdominal pain.6–9 Stark and others9 reported
that chronic symptoms are common with dientamoebiasis
with 32% of patients suffering from persistent diarrhea. Other
researchers have also showed the propensity of the organism
to cause prolonged diarrhea.10 Dientamoeba has also been
recently implicated as a possible etiological agent in irritable
bowel syndrome (IBS).11,12

The organism has a worldwide distribution and the prev-
ance rates of D. fragilis vary widely from 0.4% to 42%.13 In contrast to many pathogenic protozoa, which have a high
prevalence in developing regions of the world, high prev-
ance rates of D. fragilis have been reported from countries
where high levels of health standards are to be expected: 4.5%
prevalence from Italy,14 6.3% from Belgium, 9.4% from the
United States,15 11.7% from Sweden,16 and 16.9% from the
British Isles.17 Several reports have also identified D. fragilis
as the most common pathogenic protozoan found in stool when
appropriate diagnostic methods are used.18,19

Diagnosis of D. fragilis has traditionally relied upon micros-
copy of fixed fecal smears. Because of the “fragile” nature of the
organism, prompt fixation of clinical specimens is essential as
the trophozoites degenerate rapidly once passed in stool
samples.20 More recently, xenic culture methods have been
used for diagnosis of D. fragilis18,21,22 along with both conven-
tional and real-time polymerase chain reaction (RT-PCR).23–26
A recent study evaluated microscopy, culture, conventional
PCR, and RT-PCR for the diagnosis of D. fragilis and reported
that RT-PCR was the most sensitive of all diagnostic meth-
ods for the detection of D. fragilis (Stark and others, submitted
for publication). Daily shedding of D. fragilis trophozoites has
been shown to be highly variable, with intermittent shedding
occurring regularly. This intermittent shedding can confound
diagnosis if only single samples are examined. Because of
the high sensitivity of RT-PCR, this diagnostic method is less
influenced by intermittent shedding than other methods such as
microscopy, culture, and conventional PCR,20 and as such is
now the diagnostic method of choice.

Many studies have shown that the elimination of D. fragilis
with antimicrobial agents usually relieves clinical symptoms.20,27
As such, the treatment of symptomatic patients with D. fragilis
infections is warranted. The most common antimicrobials used
to treat D. fragilis include iodoquinol (diidohydroxyquin),28,29
metronidazole,8,30 tetracycline,31 paramomycin,32 newer nitro-
midazoles derivatives such as secnidazole and ornidazole,33,34
or combination therapy.35 However, there is currently no con-
sensus as to the best practice for the treatment of dientamoebi-
asis, and no large-scale randomized control trials have been
undertaken to evaluate treatment options.

The aim of this study was to document primarily the preva-
ance and clinical features of D. fragilis infection in patients
with gastrointestinal disease presenting to this Hospital. The
results show that correct diagnosis and treatment of dientamoebi-
asis results in the improvement of clinical signs associated
with infection.

METHODS AND MATERIALS

Faecal specimens. Data from single fecal specimens
(N = 750) submitted to the Department of Microbiology at
St. Vincent’s Hospital, Sydney, from January 2008 until March
2009, were included in the study. Samples were submitted from
both symptomatic patients (N = 650) with gastrointestinal
symptoms or asymptomatic stools submitted for routine fecal
occult blood screen testing (N = 150). Clinical information
was collected on any patient who was diagnosed with D. fragilis
infection. Follow-up stool samples were collected 2–4 weeks
after treatment and underwent microscopy and RT-PCR
(see below). Specimens underwent routine bacteriological
and virological screening as previously described to rule
out bacterial and viral (adenovirus and rotavirus) causes of
infection.9

Microscopy. Wet smears for microscopic detection of white
and red blood cells were made from fresh feces and portions

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doi:10.4269/ajtmh.2010.09-0478
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of the stool samples fixed in sodium acetate-acetic acid-formalin (SAF) for further staining. Preparations were stained with a modified iron-haematoxylin stain (Fronine, Australia), incorporating a carbol fuschin staining step for the detection of coccidian protozoa, according to the manufacturer’s recommendations and examined by oil immersion microscopy (1,000x magnification). Approximately 250 fields of view were examined on each slide. Definitive diagnosis was based on the characteristic morphology of the parasite found in the permanently stained smears and/or wet preparations. Microscopy was performed on sticky-tape preparations to detect Enterobius vermicularis ova as previously described, a minimum of two (N = 2–4) consecutive tapes were examined to “rule out” infection.

**DNA extraction and RT-PCR for D. fragilis.** All stool samples underwent direct DNA extraction using the QIAamp DNA stool minikit (Qiagen, Hilden, Germany) using a portion of the fresh stool sample as previously described. The RT-PCR was performed as previously described with the following changes to the reaction conditions; 10 min at 95°C followed by 40 cycles of 95°C for 15 sec and 60°C for 60 sec. To exclude inhibition as a contributor to negative results, all samples were spiked with an equal volume of genomic DNA from D. fragilis and run in parallel with an unspiked specimen.

**Trophozoite shedding experiment.** A family, comprising of two adults (54 and 48 years of age) and two children (14 and 9 years of age), all diagnosed with D. fragilis infections underwent examination of stool specimens on a daily basis for 10 days to determine the shedding of D. fragilis. Samples were collected and underwent microscopy as described previously.

## RESULTS

Microscopy detected parasites in 112/750 samples (Table 1). The prevalence of enteric parasites in this patient population was 14.9%. The majority of parasitic infections were with only one species of parasite (N = 90); however, 14 patients had two parasite species present, whereas eight had three or more. The RT-PCR detected D. fragilis in 39 patient samples and D. fragilis was found to be the most common pathogenic protozoa at 5.2% prevalence and the second most common protozoan parasite detected after Blastocystis hominis. Other protozoa were detected in conjunction with D. fragilis in 9/39 (23%) samples.

The clinical features of D. fragilis infection are reported in Table 2. The age range of infected patients was 3–75 years of age with the majority of infection in children under 10 (N = 14) and then adults in the 41–60 age range (N = 18) (Figure 1), with the median age of infection 34.5 years. Males and females were infected at a 1/1.16 ratio. None of the patients were immunosuppressed. The majority of infected patients did not present with a leukocytosis or red blood cells in their stools, and only 3/22 (13.6%) patients had a peripheral eosinophilia. Out of the 39 D. fragilis-infected patients 9 had other organism present: 3 Blastocystis spp., 2 Cryptosporidium spp., 1 Entamoeba coli and Iodamoeba butschlii, 1 Endolimax nana, and 1 Clostridium difficile. To rule out symptoms being caused by these organisms, those considered pathogenic and capable of causing gastrointestinal symptoms (Cryptosporidium and Clostridium difficile) were excluded from the data dealing with

### Table 1

Number and prevalence of parasites detected

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Number detected (prevalence)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blastocystis hominis</td>
<td>72 (9.6%)</td>
</tr>
<tr>
<td>Dientamoeba fragilis</td>
<td>39 (5.2%)</td>
</tr>
<tr>
<td>Giardia intestinalis</td>
<td>15 (2.0%)</td>
</tr>
<tr>
<td>Endolimax nana</td>
<td>10 (1.3%)</td>
</tr>
<tr>
<td>Cryptosporidium spp.</td>
<td>6 (0.8%)</td>
</tr>
<tr>
<td>Entamoeba dispar/histolytica/moshkovskii</td>
<td>5 (0.7%)</td>
</tr>
<tr>
<td>E. hartmann</td>
<td>4 (0.5%)</td>
</tr>
<tr>
<td>Iodamoeba butschlii</td>
<td>3 (0.4%)</td>
</tr>
<tr>
<td>Enteromonas hominis</td>
<td>2 (0.3%)</td>
</tr>
<tr>
<td>Chilomastix mesniti</td>
<td>2 (0.3%)</td>
</tr>
<tr>
<td>Strongyloides stercoralis larvae</td>
<td>1 (0.1%)</td>
</tr>
</tbody>
</table>

### Table 2

Clinical features of dientamoebias

<table>
<thead>
<tr>
<th>Clinical features of dientamoebias</th>
<th>No. of Dientamoeba fragilis-infected patients (N = 39)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age range</td>
<td>3–75</td>
</tr>
<tr>
<td>Median age</td>
<td>34.5</td>
</tr>
<tr>
<td>Sex-male</td>
<td>18</td>
</tr>
<tr>
<td>Female</td>
<td>21</td>
</tr>
<tr>
<td>Male/female ratio</td>
<td>1/1.16</td>
</tr>
<tr>
<td>Microbiological features</td>
<td></td>
</tr>
<tr>
<td>Leukocytes in stool</td>
<td>1/39</td>
</tr>
<tr>
<td>Red blood cells</td>
<td>1/39</td>
</tr>
<tr>
<td>Eosinophilia</td>
<td>3/22</td>
</tr>
<tr>
<td>Other enteric protozoa present</td>
<td>9/39 (23%)</td>
</tr>
<tr>
<td>Pathogenic or potentially pathogenic protozoa present</td>
<td>5/9</td>
</tr>
<tr>
<td>Blastocystis hominis</td>
<td>3/9</td>
</tr>
<tr>
<td>Cryptosporidium spp.</td>
<td>2/9</td>
</tr>
<tr>
<td>Non-pathogenic protozoa present</td>
<td>4/9</td>
</tr>
<tr>
<td>Enterobius vermicularis ova present</td>
<td>0/19</td>
</tr>
<tr>
<td>Clinical features of D. fragilis infected patients*</td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td>30/36 (83.3%)</td>
</tr>
<tr>
<td>Chronic diarrhea (&gt; 2 weeks)</td>
<td>9/36 (25%)</td>
</tr>
<tr>
<td>Loose stools</td>
<td>26/36 (72.2%)</td>
</tr>
<tr>
<td>Abdominal pain/discomfort</td>
<td>28/36 (77.7%)</td>
</tr>
<tr>
<td>Faecal urgency</td>
<td>17/36 (47.2%)</td>
</tr>
<tr>
<td>Vomiting and/or nausea</td>
<td>3/36 (8.3%)</td>
</tr>
<tr>
<td>Fever</td>
<td>2/36 (5.5%)</td>
</tr>
<tr>
<td>Overseas travel</td>
<td>5/36 (13.8%)</td>
</tr>
<tr>
<td>Clinical features of D. fragilis negative patient cohort</td>
<td></td>
</tr>
<tr>
<td>Diarrhea</td>
<td>92/539 (17%)</td>
</tr>
<tr>
<td>Chronic diarrhea (&gt; 2 weeks)</td>
<td>11/539 (2%)</td>
</tr>
<tr>
<td>Loose stools</td>
<td>120/539 (22%)</td>
</tr>
<tr>
<td>Abdominal pain/discomfort</td>
<td>82/539 (15.2%)</td>
</tr>
<tr>
<td>Faecal urgency</td>
<td>136/539 (25%)</td>
</tr>
<tr>
<td>Vomiting and/or nausea</td>
<td>43/539 (8%)</td>
</tr>
<tr>
<td>Fever</td>
<td>17/539 (3.1%)</td>
</tr>
<tr>
<td>Overseas travel</td>
<td>25/539 (4.6%)</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
</tr>
<tr>
<td>Metronidazole</td>
<td>28/35 (treatment failures/relapses 6/28)</td>
</tr>
<tr>
<td>Iodoquinol</td>
<td>3/35 (treatment failures/relapses 0/3)</td>
</tr>
<tr>
<td>Paramomycin</td>
<td>5/35 (treatment failures/relapses 0/5)</td>
</tr>
<tr>
<td>Combination therapy†</td>
<td>4/35 (treatment failures/relapses 0/4)</td>
</tr>
<tr>
<td>Total treatment failures/reinfection</td>
<td>9/30 (30%)</td>
</tr>
<tr>
<td>Resolution of symptoms after successful treatment</td>
<td>33/35 (94.3%)</td>
</tr>
</tbody>
</table>

*Two patients with D. fragilis were co-infected with Cryptosporidium, and one patient was co-infected with Campylobacter jejuni and was excluded from the clinical features description.

† Combination therapy comprised of doxycycline and iodoquinol, or secnidazole, nitazoxanide, and doxycycline.
symptoms and treatment. The majority of D. fragilis-infected patients \( N = 32 \) (88.8%) presented with at least one or more gastrointestinal symptoms; diarrhea \( N = 30/36 \) (83.3%) was the most common followed by abdominal pain \( N = 28/36 \) (77.7%), loose or abnormal stools \( N = 26/36 \) (72.2%), fecal urgency \( N = 17/36 \) (47.2%), vomiting and/or nausea \( N = 3/36 \) (8.3%), fever \( N = 2/36 \) (5.5%). Chronic infections, defined as presenting with prolonged diarrhea and symptoms for over 2 weeks’ duration were reported in 25% of patients. Nineteen patients agreed to undergo and submit sticky-tape tests for the detection of E. vermicularis, at least two and up to four consecutive tape tests were examined before they were considered negative, 15/19 of the patients were less than 15 years of age. No E. vermicularis eggs were detected in any of the samples submitted.

The treatment regimes including antimicrobial agents used, dosage, and length of treatment varied between patients. The following treatment regimes were used; metronidazole (400–750 mg PO, 8 hourly or once a day for 3 to 10 days duration), paramomycin (8–12 mg/kg PO daily for 7 to 10 days duration, iodoquinol (650 mg PO, daily for 10 to 12 days duration), and combination therapy comprising of doxycycline (100 mg, PO, twice a day for 10 days) and iodoquinol (650 mg PO, daily for 10 days), or secnidazole, nitazoxanid, and doxycycline (no dosage data available, 10 day course). The majority of patients (28/35) were treated with metronidazole for between 3 and 10 days duration. Metronidazole had a high rate of treatment failures/relapses with 21.4% of patients failing to clear the parasite that was detected on follow-up examination of stools, which ranged from 2 to 4 weeks after antimicrobial treatment. It is difficult to determine whether these were true treatment failures or reinfection from a common source. There was no correlation with dosage, length of treatment, and treatment failure with metronidazole. Only three patients were treated with iodoquinol because of its limited availability in Australia, however all patients responded to treatment not only clearing the infection but also reporting a resolution of symptoms. Paramomycin was used in the treatment of five patients all of which reported strong clinical improvement and clearance of the organism. Combination therapy was used in two patients both of whom failed to respond to the initial treatment of metronidazole. After receiving combination therapy both patients presented with no detectable parasites and clinical cure.

An experiment was conducted to determine the variability of parasite shedding within a family of four chronically infected patients before the resumption of treatment. The results are summarized in Table 3. One patient required 10 stool samples before D. fragilis was detected microscopically; in patient 2 D. fragilis was detected on 5 occasions among 10 stool samples, whereas in patient 3 D. fragilis was detected in 8 out of 10 samples. In the final patient, D. fragilis was detected in 7 out of 10 samples studied.

**DISCUSSION**

Dientamoeba fragilis continues to be a neglected cause of gastrointestinal disease in many countries throughout the world even though the balance of scientific evidence shows it is a relatively common enteropathogen.\(^6\) In this study D. fragilis was found to be the second most commonly detected protozoan parasite after Blastocystis hominis and more than two times as prevalent than Giardia intestinalis. This study, therefore, supports the facts that D. fragilis is a commonly encountered enteric protozoan parasite, that should be considered in any differential diagnosis of gastrointestinal disease.

The prevalence of D. fragilis was found to be 5.2%, this is significantly higher than a previously reported prevalence from Australia of 0.9%.\(^7\) This increase in prevalence can be explained because an RT-PCR assay was used for the detection of D. fragilis as opposed to just relying on microscopy for the initial diagnosis.\(^8\) The RT-PCR assay has a reported sensitivity and specificity of 100% when compared with other diagnostic methods for the detection of D. fragilis.\(^2\) A recent evaluation of RT-PCR for the detection of D. fragilis found that low level shedding of the organism that occurs may be missed by microscopy alone, whereas RT-PCR will detect these low numbers of trophozoites.\(^8\) Furthermore, because of the high sensitivity of RT-PCR it is less influenced by intermittent shedding of D. fragilis than other diagnostic modalities, in particular microscopy.

Out of the 39 patients, in which D. fragilis was detected, nearly all (89%) presented with gastrointestinal complaints ranging from abdominal pain and discomfort to diarrhea. The average duration of diarrhea was 3–7 days. There are numerous reports from various parts of the world that also describe the association between infection by D. fragilis and various clinical symptoms, most commonly diarrhea and abdominal pain.\(^1,3,32,37\)

In this study, 25% of patients reported to have a chronic infection with symptoms that had persisted for over 2 week’s

**Table 3**

Detection of Dientamoeba fragilis in four chronically infected patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
<th>Day 8</th>
<th>Day 9</th>
<th>Day 10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
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<td>–</td>
<td>–</td>
<td>–</td>
<td>✓</td>
</tr>
<tr>
<td>2</td>
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<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>✓</td>
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</tr>
<tr>
<td>4</td>
<td>✓</td>
<td>–</td>
<td>–</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>–</td>
<td>–</td>
<td>✓</td>
</tr>
</tbody>
</table>

✓ D. fragilis trophozoites detected by permanent stain microscopy.
– No D. fragilis trophozoites detected by permanent stain microscopy.

**Figure 1.** Age distribution of Dientamoeba fragilis-infected patients.
duration. Previous reports have highlighted the propensity of the parasite to cause prolonged infection with chronic infections reported in the literature to last as long as 2 years.

Just over 11% of patients with D. fragilis infection were asymptomatic. It has been reported that not all infections with pathogenic protozoan parasites will develop enteric symptoms. Several studies have shown that G. intestinalis, Cryptosporidium spp., and Entamoeba histolytica-infected patients can shed the organism for weeks to months without developing any enteric symptoms. Another study has also reported asymptomatic infections in up to 15.4% of patients infected with D. fragilis.

In nine out of the 39 patients other protozoa were detected (B. hominis, E. coli, Endolimax nana, I. butschlii, Enteromonas hominis, and Cryptosporidium spp.). All of these protozoa are transmitted by the fecal oral route and these mixed infections would suggest a common source of infection, thus indicating that D. fragilis may also be spread this way. The mode of transmission of D. fragilis remains a mystery. Some researchers have postulated that because of the “fragile” nature of the organism, and the fact that no cyst stages have yet to be described, that transmission may occur by a helminth vector. Some studies have found coincidence rates of D. fragilis and E. vermicularis, higher than the prevalence of each parasite in similar populations, suggesting a common relation between the two parasites. However, a previous study from Australia found no correlation between E. vermicularis, a proposed vector of transmission, and D. fragilis infection. In this study, 19 patients also submitted “sticky-tape” tests for the diagnosis of E. vermicularis infection, no patients were found to be infected with pin worm using this test methodology. Another study found no correlation between pin worm and D. fragilis co-infection, whereas a study of a pediatric population found no D. fragilis infections were associated with E. vermicularis. These findings might suggest that E. vermicularis does not play a role in the transmission of D. fragilis.

Most patients diagnosed with D. fragilis were given treatment. Metronidazole was the most commonly administered antibiotic, and the duration of treatment varied from 3 to 10 days. It was found that 80% of patients treated with metronidazole (28/35) resulted in the clearance of D. fragilis from follow-up stools and complete resolution of gastrointestinal symptoms. However, 6/28 (21.4%) of patients who underwent metronidazole treatment failed to clear the infection parasitologically or clinically (even though parasites were not detected in stool samples the patient had ongoing symptoms). There was no correlation between the dose received, the duration of treatment, and treatment failure associated with metronidazole use. There are varying reports of the efficacy of metronidazole treatment of D. fragilis infections in the scientific literature. Preiss and others studied 125 pediatric patients with D. fragilis infections. They found metronidazole to be effective with 70% of patients eliminating the parasite and symptoms after one treatment. A second treatment was required for 21 patients with another drug. Ten patients had to be treated a third time to eliminate D. fragilis and accompanying abdominal complaints. They recommended a 10-day treatment with metronidazole for D. fragilis infections. A number of small case reports have also indicated that metronidazole is effective in treating D. fragilis infection; Cuffari and others showed that metronidazole was effective in treatment of five pediatric patients. While metronidazole was used in three patients with dientamoebiasis in New Zealand, the treatment eradicated the parasite in all patients, however one needed a further course of metronidazole in combination with oxytetracycline to finally eradicate the organism. A conflicting study from Sweden included 32 patients infected with D. fragilis who were treated with metronidazole. The drug was given at various doses for various lengths of time, and they found that only four patients responded to the metronidazole treatment. No details were given to the exact dosages or duration of treatment so it is difficult to comment on the clinical effect of metronidazole under these circumstances.

Only three patients were treated with iodoquinol, because of the limited availability of this antiparasitic drug in Australia. Iodoquinol proved to be an effective drug as it resulted in a parasitological and clinical cure in all patients. However, it is difficult to draw any conclusions on the efficacy as the group was small and only comprised of three patients. Iodoquinol is widely used to treat D. fragilis infections. Millet and others treated 12 patients suffering with D. fragilis infections with iodoquinol. Ten of the 12 treated patients eliminated the parasite, although three subjects required a second course of therapy. In a recent study, Bosman and others reported that 27/33 children had been successfully treated with clioquinol, a member of the same drug family as iodoquinol.

In this study, Paramomycin was used in the treatment of five patients. All these patients not only cleared D. fragilis infection, but also reported a strong clinical improvement with resolution of gastrointestinal symptoms. High rates of parasitological cure with paramomycin have been reported previously. However, given the small number of patients treated with this antiparasitic agent should be noted.

Combination therapy was used in four patients and either was comprised of doxycycline and iodoquinol (N = 2), or secnidazole, nitazoxanid, and doxycycline (N = 2) both for 10 days duration. Although all patients were cured after treatment, both patients treated with secnidazole, nitazoxanid, and doxycycline complained of various side effects. Secnidazole treatment of D. fragilis has been reported to be effective in achieving parasitological and clinical cure. A recent study in Turkey evaluated the use of secnidazole, a newer nitromidazole derivative, in 35 patients with D. fragilis infection. Dientamoeba fragilis was eradicated in all but one patient with a single dose of secnidazole, and a second dose was necessary in one patient.

Although there are different treatment regimes available for D. fragilis, there is still debate over what constitutes best clinical practice for the treatment of D. fragilis. To date most studies involving antimicrobial treatment have been case studies or small-scale studies. Large-scale randomized, double-blinded controlled studies are needed to determine the true efficacy of several of the antimicrobial agents mentioned previously in successfully treating D. fragilis infections. This study also highlights the variation of treatment regimes that are currently used to treat D. fragilis infection in Australia. There were variations in antimicrobial agent used, dose, and duration of treatment.

The shedding of D. fragilis was shown to be highly irregular and variable in this study, and required up to 10 stool examinations to detect D. fragilis trophozoites by microscopy. However, with such a small cohort of patients examined, results should be interpreted with caution. This intermittent variable shedding of D. fragilis has been documented previously.
Because of this variability, as with other enteric protozoan infections the collection of multiple stool specimens is essential to aid in diagnosis, in particular when using microscopy. Hiatt and others compared the sensitivity of examining one stool specimen to that of three specimens. Using conventional permanent staining it was found that the additional stool examinations increased the percentage of positive results by 31.1% for D. fragilis. There also can be problems in differentiating D. fragilis microscopically from other non-pathogenic protozoa, in particular E. nana as uninnucleated Dientamoeba prior to the karyosome becoming fragmented can be easily misdiagnosed as the non-pathogenic E. nana. It is therefore essential that multiple samples are collected when using microscopy as the only diagnostic modality.

In conclusion, this study highlights the pathogenic potential of D. fragilis and strongly implicates it as a common cause of gastrointestinal disease with the propensity to cause chronic infections. No D. fragilis was detected in the asymptomatic patient group and there was a marked increase in the number of gastroenterological symptoms of the D. fragilis positive cohort when compared with the D. fragilis negative cohort. We therefore recommend that all laboratories should routinely test for D. fragilis as the organism has been shown to respond favorably to a range of anti-microbial treatments. As such, it is essential that a correct clinical and laboratory diagnosis is made so treatment can be initiated.

Received August 19, 2009. Accepted for publication November 23, 2009.

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