Giardiasis: a Cause of Intestinal Malabsorption

POVL RIIS
Professor, Medical Department (Gastroenterology), Gentofte University Hospital, Hellerup, Denmark

Despite increasing absolute numbers of patients with tropical and subtropical diseases in Western Europe, caused by the expanding tourist industry, such cases are still relatively rare when seen among unselected internal medical patients. The psychological factors involved in the physician’s diagnostic alertness are shown in Fig. 1; confronted with a diagnosed case, diagnostic vigilance increases sharply. During the following months the disease in question is sought for out of proportion to its true incidence. This can lead to an abuse of specific diagnostic tests but is otherwise harmless. Partly as a rebound phenomenon and partly because of the presence of more prevalent diagnostic alternatives, the vigilance subsides and may sometimes reach zero, i.e. the diagnostic possibility is temporarily forgotten. Only with increasing diagnostic experience will a satisfying congruity between diagnostic imagination and true incidence be achieved.

THE PARASITE
Giardiasis (lambliasis) is caused by the flagellated protozoan Giardia lamblia (Giardia intestinalis). The first description was made by Antoni van Leewenhoek in his 34th letter, dated 11th November 1681, to Robert Hook (Lund, 1950), based on an examination of his own diarrhoeic stools. The present name of the protozoan stems from the nineteenth century Prague physician W. D. Lambl and the French zoologist A. Giard.
Giardia intestinalis is related to Trichomonas vaginalis. It exists in two stages, the cyst and the trophozoite. The latter resides in the proximal small bowel. It has the shape of a stratospheric balloon or a fancy Chinese kite, its size is 10-20 x 2-4 μm, and it has two nuclei and four pairs of flagellae (Spencer and Monroe, 1961) (Fig. 2). The cysts are formed in the distal small bowel, and measure 10 μm. Within the double contoured cyst wall four nuclei and retracted flagellae are seen. The trophozoite cannot exist outside the body but the cysts can survive for months in a watery milieu.

The natural host is man. Transmission takes place through cysts via a faecal-oral route, either from person to person or through infected drinking water (Rendtorff, 1954; Lancet, 1974).

**INCIDENCE**

Giardiasis is known all over the world (Haas and Bück, 1967; Wählin, 1971). The reported higher incidence in tropical and subtropical areas might be related, not to the climate, but to poorer sanitation (Thompson et al., 1974). The reason for frequent infections while travelling in Eastern Europe, especially the U.S.S.R., (Walzer et al., 1971; Shultz et al., 1974) is probably the same.

Children are more readily infected than adults, with increasing incidence in lower socio-economic strata.

The overall incidence of giardiasis in Western European patient groups from gastroenterological departments (when patients are examined consecutively) is approximately 0.5 to 7 per cent (Petersen, 1972). Recent Nordic studies of groups of patients with gastroenterological symptoms have given these figures: 3.2 per cent in Norway (Petersen, 1972), and 0.5 per cent in Denmark (Danø et al., 1973). In other words, all Western European interns will meet patients with Giardia intestinalis, but at long intervals, so that the diagnostic possibility may be forgotten (Pettersson and Selroos, 1971).
PATHOGENICITY
That *Giardia* trophozoites or cysts are found in patients does not *per se* imply that the protozoan is the cause of the symptoms. Coincidental non-pathogenic presence of the parasite could be an alternative interpretation. However, much clinical evidence supports the concept that the organism is facultatively pathogenic. The number of infected persons who exhibit symptoms with a possible causal relationship varies from series to series. The highest degree of correlation is found between giardiasis and diarrhoea, with or without malabsorption. It has been suggested, but not yet proven, that giardiasis is especially liable to infect, and to provoke symptoms in, patients with hypogammaglobulinaemia, lactose malabsorption, etc.

SYMPTOMS
Based on epidemic cases (especially those reacting dramatically to treatment) the typical clinical picture can be described. The incubation period averages 14.5 days (range 1 to 43 days) (Schultz *et al.*, 1974). Diarrhoea, in the form of frequent, non-bloody, loose, bulky stools, lasting for more than a week is a cardinal symptom. Accompanying symptoms are anorexia, nausea, vomiting, flatulence and fever (Brady and Wolfe, 1974; *British Medical Journal*, 1974a, b). In severe cases the patients lose weight and signs of malabsorption are present (Hoskins *et al.*, 1967; Ament and Rubin, 1972; Danø and Soltøft, 1973; Geddes, 1973; Losowsky *et al.*, 1974). The malabsorption seems to be enterogenic, with impaired xylose absorption, lactase insufficiency (primary or secondary?), rapid transit time and sometimes villous atrophy as seen in sprue, together with inflammation of the jejunal lamina propria. Whether the parasite causes malabsorption by damage to the intestinal mucosa or by absorptive competition is not known.

If hypogammaglobulinaemia is present in a patient with giardiasis and malabsorption, the causal relationships are difficult to disentangle. Hypogammaglobulinaemia itself can cause diarrhoea and malabsorption, and probably disposes to infection with *Giardia intestinalis*. On the other hand, treatment of the giardiasis might definitely improve the malabsorption, which assigns a more central role to the giardiasis. The number of cases are too few for controlled clinical investigations, and one has to rely on the more biased interpretation of the effect of therapy on disease activity in isolated cases. Specific malabsorption (vitamin B₁₂, folic acid) is sometimes seen.

DIAGNOSIS
Diagnostic tests are based on the demonstration of cysts in stools, trophozoites in duodenal aspirates, and trophozoites in jejunal biopsies.

Stools should be examined repeatedly (at least three times a week), because the excretion of cysts takes place intermittently. Smears are examined, either in saline
or in solutions of iron haematoxylin, trichrome or iodine (Lugol's solution) (Spencer and Monroe, 1961; Petersen, 1972).

The necessity of concentrating the faecal samples is much disputed (Petersen, 1972). Repeated examinations of simple smears seem satisfactory.

Duodenal juice obtained by aspiration (around the region of Treitz's ligament) contains the trophozoites, if infected. The number of swarming mobile parasites can be very large in fresh unstained specimens, and this direct technique seems to be as good as the use of fixed and stained preparations. It has to be remembered that trophozoites are very vulnerable outside the body, so that examination must take place within 2 to 3 hours of the aspiration. The patient must be fasting, the intubation should be controlled by X-rays, the aspiration time should amount to half an hour and, when stained preparations are produced, the aspirate should be centrifuged under cold conditions at 3,000 rev/min, and the sediment smeared, air dried, fixed for 5 minutes in methanol and stained as blood smears by the May-Grünwald-Giemsa technique.

Examination of jejunal biopsies should probably be reserved for severe cases, where the clinical evidence of giardiasis (recent travelling, etc.) is good. Biopsy should be by standard technique. The true invasion of trophozoites as demonstrated in some intestinal biopsies is still disputed (Petersen, 1972).

Positive findings in faeces, duodenal/jejunal aspirate and intestinal biopsy do not show complete concordance. Whether one should start with stool examination or duodenal/jejunal aspiration is not clear. Probably most clinicians base their decisions on purely practical premises. If the clinical evidence is very suggestive of giardiasis, at least two different ways of demonstration should be used.

THE CLINICAL DIAGNOSIS
The traditional nosographic description is necessary background knowledge for the clinician, but it is not a sufficient basis for making the the diagnosis of giardiasis when the cases are encountered in the daily routine, mixed up with diseases of a different nature presenting similar symptoms. To increase the number of accurate diagnoses one must be aware of the diagnostic pathways related to the disease in question. In giardiasis, as seen in Western Europe, they are the following—

1. The patient has just returned from an area where the disease is endemic. The major symptom is subacute diarrhoea, and some of the travelling companions are ill, too. In such a case giardiasis should have a high diagnostic priority.

2. The patient has not been travelling recently. The case is an isolated one, and the cardinal symptom is diarrhoea.

(a) If the duration has been less than two weeks, bacterial and viral enterocolitis should be given a higher diagnostic priority.

(b) If the duration has been more than two weeks, the following questions should be asked:
Have there been similar attacks earlier?

Are there signs of functional bowel disease (irritable colon)?

Are signs of inflammation present (imprints, biopsies)?

Is the diarrhoea accompanied by pain?

Is steatorrhoea present?

The possibility of giardiasis should be seriously considered when subacute diarrhoea is combined with pain, and especially steatorrhoea.

Important differential diagnoses in Western Europe are: traveller's diarrhoea, the irritable colon syndrome, infections with *Yersinia enterocolitica*, lactose malabsorption, Crohn's disease, pancreatitis, carcinoid syndrome and ischaemic bowel disease. Non-specific laboratory tests are of little or no diagnostic importance (Leegaard, 1971). Important specific tests (besides direct demonstration of trophozoites or cysts) are: faecal fat excretion, D-xylose test, small bowel biopsy, serum B₁₂, vitamin B₁₂ absorption tests and intestinal lactase activity.

**TREATMENT**

Even if giardiasis is known to be a self-limiting disease in many cases, proven cases ought to be treated.

As a rule, treatment is successful because the parasite is very sensitive. Most authors nowadays recommend metronidazole 400 mg given three times daily for 10 days. The treatment can be repeated if not effective after the first course.

An alternative drug is mepacrine chloride 100 mg given three times daily for 7 days—or Tinidazole (Andersson et al., 1972).

Given the criteria for a clinical diagnosis of giardiasis, the presence of trophozoites or cysts, symptoms compatible with giardiasis, and the exclusion of other diagnoses, the disappearance of both parasites and symptoms is necessary to show that treatment has been effective.

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FLOREAT EPSOM

Henry Wick’s cows, grazing on Epsom Common in 1618, would not drink from the water hole he had found. So he did, was duly purged and realised that he was on to a good thing. Soon Epsom was swarming with Londoners drinking what the cows would not touch. So fashionable and so full of people was Epsom that Pepys could not find lodging there. At least Charles II added to its fame and provided a stable for Nell Gwynn’s horses. The whole operation was made respectable by Dr Nehemiah Grew, a Fellow of the College and of the Royal Society. He evaporated a gallon of water from Epsom Common and got two drachms of impure magnesium sulphate. Epsom salts had come to stay. So did the drinkers who used the Well House that provided a ballroom, gaming room and two bowling greens to fill in the time between draughts of the waters. In 1706 Livingstone, a cunning apothecary, bored a well in the town, built the necessary rooms for entertainment and bought up the lease of the Common well, promptly closing it. He prospered with his monopoly for some years but the new craze for health from sea bathing took the crowds away. But Epsom salts continued to gain popularity with the medical profession until they ousted ‘sal mirabile Glauberi’, the sodium sulphate made first in 1658 by Johann Rudolph Glauber.

With the ethos of Arnold and the properties of Epsom salts it was inevitable that the public school, founded in 1855 for the sons of doctors, would be built at Epsom.