Ann Hematol (2004) 83:487–488
DOI 10.1007/s00277-003-0839-2

CASE REPORT

Peter Vuylsteke · Celine Bertrand · Gregor E. G. Verhoef · Peter Vandenberghe

Case of megaloblastic anemia caused by intestinal taeniasis

Received: 23 April 2003 / Accepted: 10 December 2003 / Published online: 17 January 2004

Abstract A 61-year-old eutrophic male was diagnosed with vitamin B12 deficiency and megaloblastic anemia. A modified Schilling test suggested intestinal malabsorption unrelated to intrinsic factor deficiency. Subsequent colonoscopy revealed the presence of a *Taenia* tapeworm. The anemia resolved within days under therapy with niclosamide and temporary vitamin B12 supplements. The present case suggests that, in addition to other well-known parasitic agents, e.g., *Diphyllobothrium latum* and *Giardia lamblia*, *Taenia* infestation can also be a cause of intestinal vitamin B12 malabsorption.

Keywords Megaloblastic anemia · Taeniasis

Introduction

Parasites can give rise to malabsorption patterns in the host, and they can do so through different mechanisms. In the present report, a case of selective vitamin B12 malabsorption is presented.

Case report

A 61-year-old man was admitted with progressive fatigue and impaired performance status over the last 3 months. He had a history of congenital mental retardation and was known to have consumed alcohol to excess over the preceding 10 years. He was unmarried and lived with his mother. His eating habits were irregular and he frequently ate raw ground meat dishes. On examination, he was pale, nonicteric, and eutrophic. Neurological examination was unremarkable. Laboratory tests revealed macrocytic anemia [hemoglobin (Hb) 8.0 g/dl, mean corpuscular volume (MCV) 124 fl], neutrophil hypersegmentation, low haptoglobin levels, a slightly increased indirect bilirubin of 1.41 mg/dl (normal: 0.20–1.00), and a high lactate dehydrogenase (LDH) level of 4344 U/l (normal: 240–480). Bone marrow aspirate was indicative of megaloblastic anemia with hyperactive erythropoiesis and megaloblastic appearance of all precursor cells. On admission, the patient had a strongly decreased serum vitamin B12 level of less than 75 ng/l (normal: 170–800), while serum folic acid and erythrocyte folic acid were within the normal range. Based on these findings, the diagnosis of megaloblastic anemia due to vitamin B12 deficiency was made.

There were no complaints of abdominal pain, diarrhea, or malabsorption problems and no history of abdominal surgery. However, the patient had lost 5 kg in weight over the last 6 months. A Schilling test was performed and revealed intestinal malabsorption of vitamin B12 irrespective of the availability of intrinsic factor. Subsequent ileocolonoscopy revealed the presence of a tapeworm in the terminal ileum as well as adjacent localized erosions (Fig. 1). Microscopic examination of ileal biopsy samples showed an ulcer and signs of acute inflammation. Neutrophils were clearly present in the lamina propria and the epithelium covering the villi. Eosinophils were not prominent. Architectural anomalies or granulomas, suggestive of Crohn's disease, were not observed (Fig. 2). Examination of the tapeworm revealed a *Taenia* species. Although no further subtyping of the species was performed, *Taenia saginatum* is the most likely species given the history of raw

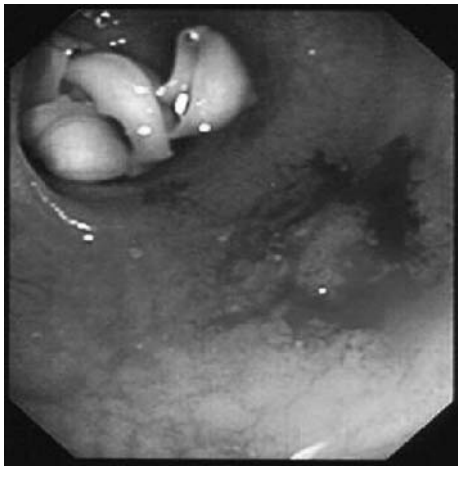

Fig. 1 Endoscopic view of the terminal ileum, showing *Taenia* tapeworm, and a local erosion in the lower right quadrant

P. Vuylsteke · C. Bertrand · G. E. G. Verhoef · P. Vandenberghe (✉)
Hematology Unit,
Department of Internal Medicine & Center of Human Genetics,
University Hospital Leuven,
49 Herestraat, 3000 Leuven, Belgium
e-mail: peter.vandenberghe@uz.kuleuven.ac.be
Tel.: +32-16-345903
Fax: +32-16-34 6051

Table 1 Evolution of blood results over time. Treatment was started on day 3

	Day 0	Day 4	Day 7	Day 9	2 months	9 months	19 months	Normal
Hb (g/dl)	9.1	7.9	9.1	9.3	13.8	15.5	15.3	12–16
Hct (%)	0.26	0.23	0.28	0.28	0.43	0.45	0.44	0.37–0.47
RBC count (10^{12}/l)	2.07	1.86	2.19	2.36	4.50	4.77	4.53	3.9–5.6
MCV (fl)	123.7	122.6	125.6	120.8	94.7	96.2	98	76–96
WBC count (10^9/l)	5.2	6.8	3.8	4.4	4.9	6.0	6.3	4–10
Platelets (10^9/l)	144	111	129	141	127	-	-	150–450
LDH (U/l)	4344	3821	2461	1702	-	-	286	240–480
Vitamin B12 (ng/l)	75	-	-	-	-	-	200	170–800

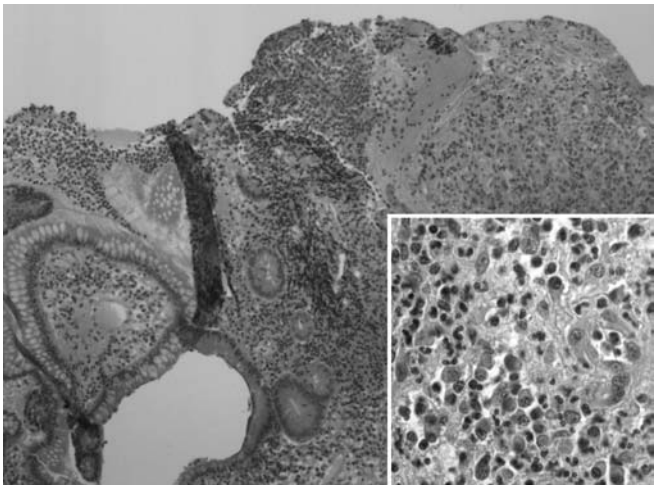

Fig. 2 Ileal biopsy sample showing an ulcer and signs of acute granulocytic inflammation

beef eating. No other parasites, cysts, or eggs were identified on three consecutive stool samples. Therapy with niclosamide was started and temporary vitamin B12 supplements were given (six daily injections of 1000 μg of cyanocobalamin, followed by monthly injections). The anemia resolved within days and the patient remained well. Two months later, a follow-up examination showed complete recovery of Hb and an improved performance status. Five months later, the monthly administration of vitamin B12 supplements was stopped, and 19 months after the event, there were still no signs of anemia (Table 1).

Discussion

To the best of our knowledge, intestinal taeniasis has not been reported as a potential cause of selective malabsorption of vitamin B12. Although a jejunal biopsy was not done to rule out giardiasis, infestation with *Giardia lamblia* seems unlikely in the present case for several reasons. The patient did not have any typical symptoms such as abdominal pain, foul smelling, loose stools, or profound malabsorption [1, 2]. Moreover, *Giardia* trophocytes or cysts, which can usually be detected in 50–70% of cases with a single stool specimen and in 90% after three specimens, were not found in three repetitive stool samples from our patient [3]. Of note, no *Taenia* eggs or proglottids were detected either, but stool examination is not a very sensitive diagnostic test for *Taenia* before the tapeworm has reached full maturation, which may take several months [4]. Finally, *Giardia lamblia* infestation is localised to the duodenum and jejunum and is not known to cause inflammation with ulceration in the terminal ileum.

Mucosal injury as seen here in the terminal ileum might be caused either directly by the movement and feeding of the parasite or, indirectly, by the host's immune response to the presence of the parasite. Once the immune response has been activated beyond a certain threshold, symptoms may start to occur and the impact on the health and nutritional status of the host can be severe [5, 6]. Intestinal disorders such as Crohn's disease are unlikely in the present case on clinical and histologic grounds.

Eradication therapy with niclosamide was given together with a course of parenteral vitamin B12. After correction of the anemia, vitamin B12 supplementation was stopped and the patient is now under further clinical follow-up. We conclude that the present case suggests that intestinal taeniasis can be a cause of selective vitamin B12 malabsorption, the exact mechanism of which is uncertain.

Acknowledgements We thank Dr. Edmond Charlier for clinical and laboratory data and Dr. Christopher Butler for reading the manuscript.

References

1. Gheorghescu B, Gherman I, Jovin GH, Suseanu I, Merculiev E, Nedea M, Aposteanu G (1976) Absorption studies in patients with parasitic infestation of the small intestine, before and after treatment. Med Interna 14:31–38
2. Brasitus TA (1983) Parasites and malabsorption. Clin Gastroenterol 12:495–510
3. Hiatt RA, Markell EK, Ng E (1995) How many stool examinations are necessary to detect pathogenic intestinal protozoa? Am J Trop Med Hyg 53:36–39
4. Beljaev AE, Madarang ME, Matuchansky C, Lenormand Y (2000) Taenia saginatum (correspondence). N Engl J Med 342:1139–1140
5. Hoste H (2001) Adaptive physiological processes in host during gastrointestinal parasitism. Int J Parasitol 31:231–244
6. Lunn PG, Northrop-Clewes CA (1993) The impact of gastrointestinal parasites on protein-energy malnutrition in man. Proc Nutr Soc 52:101–111