

DIAGNOSIS

Anti-gliadin IgG (39.3 U/ml), anti-gliadin IgA (>300 U/ml), anti-tissue transglutaminase IgG (>300/ml), and anti-tissue transglutaminase IgA (>300/ml) were strongly positive. Her ileus improved within two days of stopping oral nutrition and switching to parenteral fluids. She was diagnosed with coeliac disease and started on a gluten-free diet, osteoporosis treatment, and iron replacement therapy. Her diarrhoea improved within five days.

Two weeks later, she was doing well and a repeated coeliac serology showed a decrease of the following antibodies: anti-gliadin IgG 20.5 U/ml, and anti-tissue transglutaminase IgG 59.3 U/l. After 12 months, she was symptomless under the gluten-free diet.

Chronic diarrhoea, characterised as three or more loose stools per day lasting for at least four months, is caused by inflammatory bowel disease, irritable bowel disease, malabsorption syndromes, chronic infections, food, and drugs. Coeliac disease, another cause of chronic diarrhoea, is a state of heightened immunological responsiveness to ingested gluten in genetically susceptible individuals. The inflammation and intestinal mucosal damage may cause a wide spectrum of gastrointestinal symptoms, nutritional abnormalities, and systemic complications including anaemia and osteoporosis, secondary autoimmunity and malignancy.

This disease was once considered a disease of children and was thought to rarely occur in the elderly. In fact, the peak age of diagnosis is in the fourth and fifth decades of life¹ and some evidence shows an increased rate of diagnosis among adults and the elderly.² Overall, 19 to 34% of newly diagnosed coeliac disease patients are over 60 years of age.³ The incidence rates are increasing among all age groups, including the elderly.³

Coeliac disease presents a spectrum of clinical features that range from severe malabsorption with nutritional deficiencies to presentation with a single symptom, such as anaemia, osteoporosis or osteomalacia. Intestinal symptoms are less prominent in elderly coeliac patients than in younger ones. Some elderly patients may present with acute complications such as intestinal obstruction or perforation. Although the exact mechanism of obstruction is not known, it is suggested to originate from electrolyte deficiency due to primary malabsorption or increased faecal loss or both or from disturbed motility of the gastrointestinal system due to gluten-driven mucosal inflammation. Motor abnormalities may present radiologically with a decreased number of jejunal folds, an increased number of ileal folds, small bowel dilatation,

and intussusception. Our patient's obstruction and ileal dilatation improved after withholding gluten. Our patient presented twice with intestinal obstruction and her clinical picture was improved inadvertently by stopping oral nutrition.

Intestinal lymphoma is rarely associated with coeliac disease. The diagnosis may be difficult since it may be multifocal. Our endoscopic biopsy did not show any evidence of lymphoma. Imaging studies were negative for any involvement for the extranodal sites. The clinical course may exclude lymphoma: during gluten-free follow-up of 12 months, she was doing well; in lymphoma, however, overall survival is 7-10 months despite chemotherapy.⁴

REFERENCES

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