A floppy child with failure to thrive

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A 13-month old girl presented in February, 2000, with flaccid paraparesis after an upper respiratory tract infection. She had been born at term (birthweight 4·2 kg) and her neurological development had been previously normal for her age. When we examined her, she was pale, listless, and had a bulging abdomen, diffuse weakness, hypotonia, and reduced deep tendon reflexes. Her weight was below the 5th percentile, but her height and head circumference were at the 50th percentile. We admitted her with a diagnosis of Guillain-Barré syndrome. Full blood count, serum electrolytes, creatine kinase, Lyme serology, serum lead concentrations, thyroid function, vitamin B12 and vitamin E—were all within normal limits. We did a lumbar puncture, but the cerebrospinal fluid was unremarkable. Chest radiographs, and brain and spinal MRI showed no abnormalities. We reviewed her growth chart, and saw that her height had shifted from the 25th percentile to the 5th percentile in the past 5 months, so we considered the possibility of malabsorption. She had IgA antibodies against gliadin, reticulin, and the endomysium. Duodenal biopsy showed villous atrophy and intraepithelial lymphocytosis (figure), findings consistent with coeliac disease.

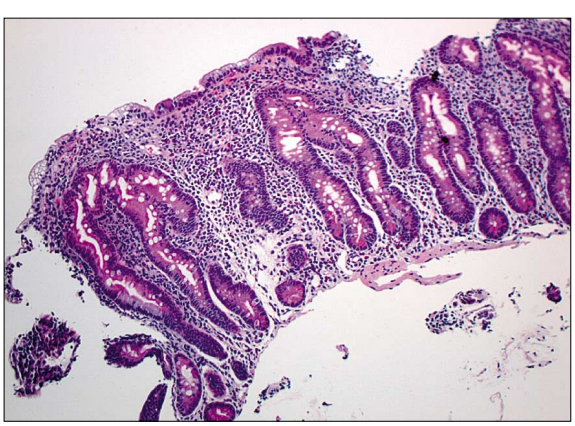

Figure: **Biopsy of duodenal mucosa showing villous atrophy and lymphocytic infiltration**

We placed our patient on a gluten-free diet and within 2 weeks she was sitting independently and pulling herself up to standing. Her strength, tone, and reflexes returned to normal. After 2 months her weight was at the 25th percentile and after 7 months it was at the 50th percentile. We did not do nerve conduction studies because the patient showed such rapid clinical improvement. We screened the rest of her family and diagnosed her older sister with coeliac disease. When last seen in January, 2005, our patient was fit and healthy.

Two cases of peripheral neuropathy associated with biopsy-proven coeliac disease have been reported.[1,2] In these individuals, gastrointestinal symptoms preceded neurological symptoms. Vitamin deficiencies used to be considered the cause of the neurological complications associated with coeliac disease. However, autoimmune mechanisms might also be responsible for the neuronal damage. Patients with neurological complications and anti-neuronal antibodies, improved clinically on a gluten-free diet.[3] The incidence of coeliac disease is rising in the USA,[4] but patients are seldom screened in the absence of gastrointestinal symptoms. Atypical presentations increase the delay in diagnosis of coeliac disease in children, with a mean duration of 13 months before diagnosis in the USA, compared with 3 months in Europe.[5] Rapid diagnosis of coeliac disease and initiation of a gluten-free diet are essential to achieving catch-up growth in affected children.

References

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