

Growth Acceleration and Final Height After Treatment for Delayed Diagnosis of Celiac Disease

Short stature and growth failure may be the only clinical presentation of the so-called occult form of celiac disease (CD). This paper reports on 24 patients over 4 years of age in whom CD was diagnosed. Their initial presentation was short stature or retarded growth, with heights below the 5th percentile but without any other overt symptoms. The effect of treatment with a gluten-free diet (GFD) on catch-up growth and final height was determined.

Small-bowel biopsy demonstrated mucosal atrophy in all 24 patients. Antigluten antibody (AGA) titers were also found to be abnormal in the 13 of 24 patients whose levels were measured. Weight and height velocities, pubertal staging, bone age (BA), target height (TH, based on midparental height), and predicted height (PH, according to Tanner) were recorded at diagnosis and periodically after treatment was begun with GFD.

At diagnosis, 82% of patients were below the 3rd percentile for height and 58% were below

the 3rd percentile for weight. Nearly all of the patients (95%) had a delayed BA compared with chronologic age (range of delay, 1 to 6 years). All patients had catch-up growth following the institution of GFD, with increased height and weight velocities rapidly achieved during the first year of GFD. After 1 year of treatment, 87% of patients had a stable height velocity above the 50th percentile and their height standard deviation score (HSDS) improved significantly. By the third year, their HSDS showed less stature reduction than that observed at the time of diagnosis (-1.77 vs -2.52). The patients who reached an appropriate TH for midparental height were those in whom the diagnosis was made and treatment was started before puberty. In contrast, the patients who did not achieve a satisfactory final adult height were those who began dietary treatment after the onset of puberty.

Bosio L, et al. *J Pediatr Gastroenterol Nutr* 1990;11:324-329.

Editor's comment: Although the paper by Bosio et al does not address all pertinent issues of CD diagnosis and prognosis, it adds to the large volume of reports indicating that relatively asymptomatic short-statured children without weight deficits for height but with various degrees of retarded BA may have CD as the cause of their poor growth. This paper also confirms that the diagnosis of CD can be established only by a small-bowel biopsy, which shows the typical histologic findings, whereas other measurements of intestinal function (ie, xylose tolerance) may fail to detect any abnormality.

The paper by Bosio et al also suggests that when an appropriate diagnosis is made and when timely dietary treatment is given these patients exhibit catch-up growth and attain an appropriate height based on midparental height. In contrast, when there is a delay in the diagnosis and treatment with GFD is initiated after the onset of puberty, there may be an unsatisfactory final adult height.

Therefore, the clinician must be alert and must consider CD as a cause of short stature. Of course, there are other, perhaps more important reasons besides stature mandating that the accurate diagnosis of CD be made as early as possible. Since the only way to rule out CD is by small-bowel biopsy, the clinician must keep in mind the clinical indications for this procedure when a short child is evaluated. These vary in accordance with the geographic location and with the clinical history of the patient. In areas of the world where CD is frequent, it should be high on the list in the differential diagnosis of short stature. The clinical history usually reveals clues for consideration of CD. This entity

is not "occult" in the majority of these subjects. In the paper by Bosio et al summarized above as well as in other publications on the subject, it is clear that these patients have a frequent history of diarrhea, poor weight gain, and other gastrointestinal symptoms in infancy. These symptoms are often considered not important enough to be thoroughly evaluated by the physician, although patients are often treated by dietary manipulations. Other important clues to alert the clinician include a deteriorating height and weight pattern of growth and the presence of nutritional deficits, ie, iron deficiency associated with short stature. The paper by Bosio et al provided no data on the growth patterns that

preceded the diagnosis of CD in their patients, nor did it contain information regarding the presence or absence of nutritional abnormalities like iron deficiency. Patients with CD usually have a growth pattern typical of nutritional dwarfing with decelerating weight gain and height velocity, although they may not have weight loss or body weight deficits for height. Also, they often exhibit iron deficiency even though there may be no anemia. It is clear that following an appropriate diagnosis and treatment with GFD, the patient will exhibit catch-up growth and weight gain, which may occur rapidly during the initial stages of treatment.

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