Coeliac disease in children of West Indian origin

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Abstract
Coeliac disease is uncommon in populations of non-European origin. Two English born West Indian children with coeliac disease are presented. The diagnosis should be considered in children of West Indian origin with chronic diarrhoea. (Arch Dis Child 1995; 73: 166–167)

Keywords: coeliac disease, West Indian children, HLA phenotypes.

The development of coeliac disease requires exposure to gluten and it is strongly influenced by genetic factors. Coeliac disease, that is, permanent gluten intolerance, predominantly affects populations of European origin. There are now reports of the disease in children from the Indian subcontinent, Lebanon, Iraq, Kuwait, Sudan, and Cuba but it appears to be very rare.1-6 Presentation in these patients is generally delayed to the second or third year of life. This may be due to prolonged breast feeding and late introduction of gluten in their diet.

Transient gluten intolerance has been described in children presenting at less than 2 years of age who eventually return to a normal diet.7 It is important to distinguish it from coeliac disease to avoid overdiagnosis, especially in the low risk population.

We report coeliac disease in two English born children of West Indian origin.

Case reports

CASE WM
This boy presented in 1974, aged 6 months, with failure to thrive and persistent diarrhoea. Small bowel histology showed a flat mucosa with severe villous atrophy and crypt hyperplasia. He was started on a gluten-free diet and he began to thrive. A pre-gluten challenge small bowel biopsy was normal at three years of age. He was then challenged with gluten for three months. His post-gluten challenge biopsy showed mild villous atrophy. He remained on a gluten containing diet and continued to thrive.

Another small bowel biopsy was carried out at 4-5 years of age and it appeared normal. At that stage, he was thought to have transient gluten intolerance.

He was monitored from the age of 6 years for gliadin and reticulin antibodies. Both antibodies became positive at 9 years of age and, although he remained well and thriving, a small intestinal biopsy was repeated. On this occasion, it showed a flat mucosa with severe villous atrophy and crypt hyperplasia. This confirmed the diagnosis of coeliac disease and he was advised to return to gluten-free diet. He remained well on subsequent review.

His mother was white and his father was a black Jamaican. HLA class II typing showed that he was positive for DQw3, DR4, DRw52, and DRw53.

Discussion
Although histology is crucial for the diagnosis, gliadin, endomysium, and reticulin antibodies have been shown to be very useful adjuncts for diagnosing and monitoring coeliac disease.10

As in patient WP, who was mentioned in a previous paper,7 histological relapse was heralded by the appearance of these antibodies and during that time he was in good health. This case illustrates the importance of long term follow up in children with the presumed
Coeliac disease in children of West Indian origin

Coeliac disease is a chronic autoimmune gastrointestinal disorder that occurs when the body reacts to gluten, a protein found in wheat, barley, and rye. In children of West Indian origin, the diagnosis of transient gluten intolerance can take many years to resolve. Indeed, it is difficult to make a final diagnosis of transient gluten intolerance, as children may take many years to relapse.7

Both children are a mixture of black West Indian and white ethnic backgrounds. The first case, with a distant Scottish relative, showed a typical presentation and response to gluten provocation, although the diagnosis was complicated by social circumstances. In contrast, patient WP, with mixed parentage, had a greatly delayed response to dietary challenge, only relapsing after six years of exposure to gluten. The HLA status may be relevant, being the commonly associated pattern in patient KM, but of less common association in patient WP. The association of coeliac disease with DR4 antigen has been reported in only a small number of patients, and different gene alleles are believed to be responsible.11 Furthermore, the degree of gluten sensitivity may be different in DR3/DQw2 negative patients as their antigliadin antibody titres are lower than in the DR3/DQw2 positive patients.13

The lack of reports of coeliac disease in children of West Indian origin may be partly due to low gluten consumption in the West Indies. However, the situation may change when they move to another country such as the United Kingdom where gluten consumption is considerably higher. The HLA-DR3 and HLA-DQw2 antigens12 14 15 have a stronger association with coeliac disease than HLA-B8 antigen which has a low prevalence in the West Indian population.16 As both of our patients possess class II HLA markers (DR3-DQw2 and DR4) for coeliac disease, it would be interesting to find out the prevalence of these antigens in the West Indies. It is also possible that the presence of coeliac disease in these cases is due to their mixed ancestry and it may not develop in West Indians of black African origin.

In conclusion, despite the uncommon occurrence of coeliac disease in the West Indies, the diagnosis should still be considered in children of West Indian origin with chronic diarrhoea.

2 Bitar JG, Salem AA, Nair AT. Celiac disease from the Middle East (report on ten cases seen at the American University of Beirut Hospital). J Med Leban 1970; 23: 423–44.