Is Measurement of Serum Antigliadin Antibodies in Patients with Suspected IgA Nephropathy Worthwhile?

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Dear Sir,

A-containing immune complexes are present in the sera of patients with IgA glomerulonephritis (IgA GN) [1] and may be formed by continuous exposure to exogenous antigens. Anecdotal reports of IgA GN associated with coeliac disease suggested that dietary gliadin might be one such antigen. Reported prevalences of raised serum IgA antigliadin antibody measured by ELISA in IgA GN include 3% by Fornasieri et al. [2] and 13% by Kumar et al. [3]. In contrast, French studies have obtained figures of 70% [4] and 53% [5] and suggest that the measurement of IgA antigliadin antibody is a useful diagnostic test. Rostoker et al. [6] suggested that the discrepancy was methodological: Fornasieri et al. [2] used neutral phosphate-buffered saline instead of alkaline buffer to prepare gluten, so that they in fact detected antibodies against water-soluble fractions of gluten (glutenin) and not gliadin. However, Rodriguez-Soriano et al. [7], using methods identical to those of Rostoker et al. [5], reported circulating levels of IgA antigliadin antibody in children with IgA GN similar to those of controls and patients with other types of GN.

We used an indirect immunofluorescence technique, which we have shown to have comparable sensitivity and specificity to an ELISA-based method for coeliac disease [8], to measure IgG and IgA antigliadin antibody in the sera of 27 patients (22 male, mean age 34 years, range 15-61) with biopsy-proved IgA GN. None was taking steroids or other immunosuppressive therapy at the time of serum sampling and none had symptoms suggesting malabsorption. IgG antigliadin antibody was detected in 3 patients: 2 at a titre of 1:10 and one at 1:40. Two of these (one with a titre of 1:40) had jejunal biopsies which were normal. No patient had detectable IgA antigliadin antibody and all were negative for antiendomysial and antijejunal antibody measured by indirect immunofluorescence.

As IgA GN is not associated with antiendomysial or antireticulin antibodies [3, 4, 6], and as there is no evidence that dietary gluten withdrawal alters the course of the disease [9], a significant association between coeliac disease and IgA GN seems unlikely. Our results confirm that testing for antigliadin antibody in patients with suspected IgA GN is not worthwhile.
References