Sequential Measurements of Intestinal Permeability to [⁵¹Cr]EDTA in Children with Henoch-Schönlein Purpura Nephritis

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Dear Sir,

In 1988, we demonstrated an increased intestinal permeability to [⁵¹Cr]EDTA in some children presenting with idiopathic IgA nephropathy or with Henoch-Schönlein purpura nephritis [1]. Three further studies of intestinal permeability were reported in adult idiopathic IgA nephropathy [2-4]. Two [2,3] -but not the 3rd one [4] - displayed occasionally the same abnormality. Layward et al. [3] pointed out a marked increase of gut permeability in a patient displaying macroscopic hematuria 3 weeks later. The authors [3] hypothesized that this finding might be observed transiently only during relapse and emphasized the need of sequential studies.

In order to clarify this point, we have tested a second time 4 patients included in our previous study [1] who all presented initially with Henoch-Schönlein purpura nephritis, microscopic hematuria, high levels of circulating IgA-containing immune complexes (IgA-CIC) and markedly increased intestinal permeability. This 2nd evaluation was performed after apparent complete recovery. The intestinal permeability was measured as described [1] using [⁵¹Cr]EDTA as a probe and the results of 24-hour urine excretion of [⁵¹Cr]EDTA were expressed in percentage of the oral dose administered. IgA-CIC plasma levels were determined by the measurement of the IgA content of 2.5% polyethylene glycol precipitates of sera [1]. In all cases, an intestinal permeability which had returned to nor-

Hematuria No hematuria

Fig. 1. Twenty-four-hour urine excretions of [⁵¹Cr]EDTA in 4 patients suffering from Henoch-Schönlein purpura nephritis. The tests were performed in the same patients during microscopic hematuria episodes and after complete recovery. - = Highest normal control value [1].

mal was found (fig. 1), and IgA-CIC plasma levels remained in the normal range (data not shown).

We have therefore shown that the increased intestinal permeability reported in IgA-associated nephropathies in children [1]

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is a transient phenomenon associated with illness activity (at least in Henoch-Schönlein purpura nephritis). It could be speculated that this transitory increase of intestinal permeability might result from intestinal lesions taking place only under some definite circumstances such as for example the ingestion of lactose in patients with intestinal lactase deficiency or of specific foods in patients with alimentary allergy or intolerance. However, it can also be postulated that the abnormality is only the consequence of IgA-CIC deposition in the intestinal wall. Indeed, it is very probable that IgA-CIC may also accumulate in the mucosal intestinal wall (as in the skin and the kidney) since we have observed abundant IgA deposits in a biopsy of the rectal mucosa of 1 patient presenting initially with isolated rectorrhagiae and developing thereafter typical Henoch-Schönlein purpura [pers. unpubl. observation]. Correlations between sequential measurements of intestinal permeability and mucosal histology might be contributive for the understanding of the cause and/of the eventual etiopathogenic role of the transient increase of intestinal permeability observed in some patients with IgA-associated nephropathies. However, it is unlikely that such studies will be performed for obvious ethical reasons.

References

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