Letters to the Editor

Heterogeneity in tropical pancreatic diabetes

Dear Sir,

With regard to the recently published article on heterogeneity in tropical pancreatic diabetes [1] one might want to regret that there often seems to exist a tendency to formulate premature conclusions concerning a clinical or biochemical finding so that the basic physiopathological or etiological features remain unravelled and unrecognized forever. In fact, the clinical and biochemical characteristics of juvenile-onset diabetes in the Indian sub-continent have been subject of our investigations since 1965 [2–7]. The patients develop clinically manifest diabetes in their mid-twenties, presenting with acute symptoms, a body mass index $< 19 \text{ kg} \cdot \text{m}^{-2}$ and they have no tendency to develop ketosis even when insulin therapy is interrupted. However, they require usually more than 2 units of insulin per kg of body wt/day for normalization of glycaemia and they do not respond to sulphonylurea treatment. The designation 'J' type diabetes for this syndrome has not been accepted by us as one should prefer not to employ eponyms, but to exactly describe the clinical characteristics until the etiological factors or physiopathological aspects of the syndrome become fully understood.

The similarity of clinical features of this type of juvenile diabetes frequently observed in India to the calcifying pancreatic diabetes or to the tropical pancreatic diabetes as observed in South India suggests the possibility that both disorders may represent stages of the same (mal-) nutrition related pathogenetic mechanism. The heterogeneity of C-peptide values measured in these patients may be an inconstant, temporary phenomenon as indicated in our recent studies [8, 9]. Again, in studies from the All India Institute of Medical Sciences on diabetes amongst young Indians, in this group, pancreatic calcification is demonstrable in 27%, steatorrhoea in 33% and overt clinical signs of malnutrition in up to 33%.

So far islet cell antibodies (ICA) have not been found in this group (as distinct from Type 1 (insulin-dependent) diabetes mellitus where in 31% of cases at the same centre ICA were detected). Also, no distinctive HLA haplotype is associated with this type of diabetes, thus rendering an HLA-related genetic pattern of transmission unlikely (unpublished data).

A group of investigators from Madras [10] have now designated such cases, i.e. young asthenic non-ketosis prone insulin resistant diabetic patients as insulin requiring diabetes mellitus (IRDM) or Type 3 diabetes. This is where the literature is likely to acquire yet another confusion through a new terminology. With regard to the report by Mohan et al. [1] it is probably misleading to include patients that can be managed on diet or sulphonylurea in the group under discussion. Furthermore, these authors have probably included a rather large number of lean Type 2 (non-insulin-dependent) diabetic patients in their series as 30% of their patients were well within or even above the normal weight range.

It is suggested that objective criteria as defined earlier [9] should be followed in identifying the atypical presentation of diabetes in the young from this country; such evaluations should include an assessment of pancreatic structure and function and analyses of the cause (s) for pancreatic injury. Diabetes of malnourished population and

calcifying pancreatic diabetes (tropical pancreatic diabetes) may well be representing the same disorders. New nomenclatures will not offer any clarification, neither does the assessment of C-peptide levels as this may just reflect the extent of preserved beta cell function as well as the degree of the present metabolic status. Concerted effort is needed for search of environmental or host factors as diabetes of this type may indeed be a preventable entity.

Yours Sincerely,
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