EDITORIAL

Non Insulin dependent diabetes in India

In a series of 5831 new diabetics registered in our clinic from 1982-89 only 132 (2.2%) were insulin-dependent (IDDs) and 5699 (97.8%) were non-insulin dependent (NIDDs). A large proportion of NIDDs are put on oral agents (52%), the main compounds used being sulfonylureas or sulfonylurea-biguanide combinations. The predominant sulfonylurea used is glibenclamide. Additionally, about 20% of these patients are treated with a combination of insulin and sulfonylurea. The proportion treated with diet alone (10%) and insulin alone (18%) is small. As these data originate from a specialised clinic, NIDDs controlled easily on diet alone are excluded by selection factors. The number treated with insulin-sulfonylurea combination is also large, as many insulin- resistant patients are likely to present themselves at a specialised centre.

Considering the fact that 98% of our diabetics are non-insulin dependent, there are fewer studies available on this group than deserved by its numerical strength. The Indian investigators have created and the WHO has accepted (1) the category of malnutrition-related diabetes (MRDM), divided into two major categories of fibrocalcific pancreatic diabetes (FCPD) and protein-deficient pancreatic diabetes (PDPD). The fibrocalcific pancreatic diabetes (FCPD), is an interesting group from the etiopathological standpoint yet it makes only 1% of all diabetics and 4% of young (< 30 yr age) diabetics seen in areas where it is supposedly rampant (2). While the clinical characteristics of FCPD are distinctive, it is difficult to say so for the PDPD. It is postulated that PDPD is caused by nutritional factors, but the precise nature of these factors is not known, and whether it is due to a protein deficiency, past or present, is highly conjectural. Although these patients are often undernourished, it is not clear whether under nutrition is the cause or the effect of the diabetic state. Some of these patients are well nourished, but it is surmised that they had nutritional problems earlier, a fact difficult to establish. It is possible that PDPD may be a forme fruste of IDDM presenting in a slightly older age group (2).

Besides the FCPD discussed above, the remainder of the patients from the vast group of NIDDs are not a homogenous population. There is urgent need for characterising the heterogenous subsets of NIDDM. A few outstanding subsets are: grossly obese; grossly undernourished; NIDDM bordering on insulin dependency; maturity-onset diabetes of young with autosomal dominant transmission (3); mild NIDDM (or IGTT) with hyperinsulinemia, obesity, hypertension and hyperlipidemia (Reaven syndrome) (4); insulin sensitive and insulin resistant variants (5); NIDDM which responds better to a combination of insulin and sulfonylurea therapy (6), islet-cell antibody positive NIDDM which often progresses into insulin dependency, NIDDM with structurally abnormal insulin secretion (7).

The incidence and natural history of complications is well worked out in IDDM but is not well worked out in NIDDM. Even in western countries, IDDM forms only 10-20 per cent of diabetics, yet it as always been more intensively studied than the NIDDM. The incidence and natural history of diabetic nephropathy is well worked out in IDDM. Nephropathy appears to be less frequent in NIDDs. End stage renal disease developed in 0.5% of NIDDs and 5.8% of IDDS over a 10-year period, yet NIDDM accounts for most patients of end stage renal failure seen at nephrology centres (8). Amongst NIDDs, there appears to be a few distinct groups, from the point of view of complications. There is a group which suffers from macrovascular disease. It is perhaps the largest group. There is also a group which predominantly develops microvascular complications. A small subset does not appear to develop vascular complications any faster than the non-diabetic group. We are not able to separate out these patients in advance with any degree of confidence, but there is no doubt that this separation will help immensely in planning our treatment strategies.

It is a pity that such a heterogeneous group is often studied by most investigators as a single, homogenous entity, which produces obvious difficulties in the interpretation of data.

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REFERENCES

1. Diabetes Mellitus: Report of WHO study Group (1985) Technical Report Series 727, WHO, Geneva.

2. Mohan V, Ramchandran A and Viswanathan M. Diabetes in the tropics. In the Diabetes Annual-4,

Alberti KGMM & Krall LP Editors, Elsevier, Amsterdam, 1988, p 46-55.

3. Tattersall RB & Fajan SS. A difference between inheritance of classical juvenile-onset and maturity-onset type diabetes in young people. Diabetes 1975: 24; 44-53.

4. Reaven GM, Role of Insulin resistance in human disease. Diabetes 1988: 37; 1595-1607.

5. Banerji MA, Lebovitz HE. Insulin-sensitive and insulin resistant variants in NIDDM. Diabetes 1987: 38; 784-92.

6. Lebovitz HE, Feinglos MN. Sulfonylurea drugs, mechanism of anti-diabetic action and thereapeutic usefulness. Diabetes Care 1978:1; 189-198.

7. Haneda M, Polonsky KS, Bergenstal RM, et al Familial hyperinsulinemia due to a structurally abnormal insulin: Definition of an emerging new clinical syndrome. N. Engl. J. Med. 1984:310; 1288-1294.

8. Cowie CC, Port FK, Wolfe RA, Savage PJ, Moll PP, Hawthorne VM. Disparities in incidence of diabetic end stage renal disease according to race and type of diabetes. New Eng. J. Med. 1989:321, 1074-9.