EDITORIAL

MALNUTRITION RELATED DIABETES

Expert Committee of W.H.O. has recently elaborated on the classification of Diabetes Mellitus (W.H.O. 1985; TRS 727)[1].

Malnutrition Related Diabetes (MRD) emerges as a distinct entity in this presentation. MRD is subdivided into Protein Deficient Pancreatic Diabetes (PDPD) and Fibrocalculus Pancreatic Diabetes (FCPD).

There is considerable overlap in the characterization of these two types, and except for abdominal pain pancreatic calculi on radiology and loss of some exocrine pancreatic function in the latter group (FCPD), there seems to be hardly any difference between the two types.

It is likely that such sub-categorization will defeat the very purpose for which the entity malnutrition related diabetes has been given recognition.

Pointers for Malnutrition Related Diabetes are :

1. Epidemiological observation relating to certain geographic locations and economic conditions (23° North and South of the Equator), GNP < $ 230.

2. Low body weight (BMI < 19). While malnutrition may not be overt in all cases, daily protein intake is usually 30 gm. or less. In some instances, evidence of ingestion of cyanogens and in others consumption of coarse cereals which may contain some pancreatotoxins is forthcoming.

3. Clinical presentation in adolescence (mid-twenties) while conventional IDDM or NIDDM usually do not have onset at this age.

4. Moderate to severe hyperglycaemia, but no acetonuria or acetonemia leading to ketoacidosis. Insulin is required in higher than normal dosage to achieve euglycaemia, and not to prevent ketoacidosis.

5. C-peptide level is of intermediate range but may be related to residual beta cell function, quality of metabolic control and the duration of the illness.

The presentation can be thus categorized on the basis of Clinical and Investigative data with objectivity under headings of absolute, relative or absent characteristics

Absolute	Relative	Absent
Geographical location : Equator N 23° S 23°	History of recurrent abdominal pain (suggesting pancreatitis)	No history of biliary disease or alcohol consumption.
History of consumption of low protein diet < 30 g or cereals that contain cyanogens	Radiologically pancreatic calcification.	Absent genetic Marker (no specific or distinctive HLA type).
Subclinical or overt undernutrition, BMI < 19	U/S : reduced size of pancreas, dilated ducts or ductal stones.	ICA negative (cytoplasmic islet cell antibodies).
Onset at adolescence < 30 yrs	Exocrine pancreatic malfunction	Absent lymphocytic infiltrate in pancreas (insulinitis or systemic autoimmune disorder).
Moderate to severe hyperglycaemia.	C-peptide of intermediate value	Non-responsive to sulphonylureas.
Serum acetone value < 5.0 mg/dl.		
Insulin dependent > 1.5U/Kg/ 24 hrs.		

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1. Diabetics from South India, who have better body build (normal or overweight), later age of onset (30-40 yrs.), absence of ketosis, and having pancreatic fibrosis with calcification, and responding to oral sulphonylureas should be recognized as having secondary diabetes (pancreatic diabetes) and not undernutrition related diabetes. Some have designated this entity as NIDDY (Non-insulin dependent diabetes in the young).[3]

2. Another type of diabetes observed in South India[4] but not among undernourished, (BMI > 18) is being designated as Insulin Requiring Diabetes Mellitus (IRDM). The patients are below 30 yrs. of age, ketosis is not seen despite persistent

hyperglycaemia and being without insulin for prolonged periods. Insulin in higher than usual dose is required to attain euglycaemia. According to its authors, this group is heterogenous and includes secondary diabetes as well as diabetes associated with certain genetic syndromes. Some authors have even commented on the need for intermittent insulin administration in such instances and used another designation for it : Phasic Insulin Dependent diabetes mellitus[5]. Such additional categorizations seem to be unnecessary, as undernutrition related diabetes has all the above features except that it does not include genetic syndromes related diabetes or the conventional secondary diabetes (hormone or drug induced).

As regards experience of working in this area, the most usual characteristics of undernutrition related diabetes seem to be age of onset < 30 yrs. low body mass index (< 19), absence of ketosis (serum acetone < 5.0 mg/dl.)

Evidence of pancreatic fibrosis or calculi may be present variably.

In such instances, (history suggesting pancreatitis;) recurrent abdominal pain, (radiological evidence for pancreatic calculi), reduced size of pancreas (on ultrasonography) and disturbance of exocrine pancreatic function (faecal fat, xylose test) may also be degrees. C-peptide levels are in the intermediate range, indicating that residual beta cell function exists.

This type of diabetes is distinctive from IDDM, as ketoacidosis is absent, C-peptide is present in intermediate values and ICA are absent. It is also distinct from NIDDM type as body mass index is low, and glycaemia is unstable and not responsive to sulphonylureas.

There has been no distinctive HLA type association with undernutrition diabetes. Again, in such diabetics islet cell antibodies or a subtle autoimmune profile is lacking. Histologically in the pancreas there is no lymphocytic aggregation, but atrophic changes predominate. Calculi are found intra ductally, with abundant viscous mucus around, incriminating compositional changes of pancreatic secretion in stone formation.[5] Though not recognized in initial screening, calculi may become evident over a period of time.

Natural history of malnutrition related diabetes is also distinctive; infections may supervene or the microangiopathic renal involvement may lead to end-stage renal failure.

It is important to recognise the etiogenesis of the entity as this may provide preventive public health measures. Malnutrition merely predisposes the islet cell to some diabetogenic agent (s) that damages pancreatic endogenous and exogenic function to varying degree. If this agent/s can be identified, preventive measures can be evolved.

Protein deficient and fibrocalculus pancreatic diabetes may indeed be different phases in the evolution of the same disease, or may be related to the severity of the noxious agent damaging the islet cells or exocrine cells.

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