

Diabetes and malnutrition

by Jasbir S. Bajaj

Contrary to popular belief, diabetes mellitus is not a disease that affects only the affluent. It strikes the poor and under-nourished as well. A greater awareness of this fact, as a result of a large mass of epidemiological data, has resulted in the new international classification of diabetes. Malnutrition-related diabetes mellitus (MRDM) was recognised by a recent WHO study group as a clinical class distinct from non-insulin (NIDDM) and insulin dependent diabetes mellitus (IDDM). In several developing countries it may constitute 30 to 70 per cent of all cases of youth-onset diabetes. Indeed, epidemiological data do indicate a low incidence of IDDM and a high prevalence of MRDM in most of the developing world. Clinical features include characteristic leanness with sub-normal body mass, moderate to severe increase in blood glucose, the requirement of large doses of insulin to achieve normalcy in blood glucose, and a frequent history of malnutrition in early childhood.

The clinical spectrum of MRDM in most parts of the world lends itself to a further classification into two sub-types, namely fibro-calculeous pancreatic diabetes (FCPD) and protein deficient diabetes mellitus (PDDM). This classification is based on simple clinical observations together with basic laboratory data, readily obtainable in the setting of primary health care in developing countries.

The previous terminology of tropical diabetes served its purpose in the past, when it was essential to accord recognition to the fact that most young people with diabetes mellitus in tropical developing countries exhibited features distinct from those observed in youth-onset diabetes in the industrialised societies. With the increase in

knowledge of causes and mechanisms, as well as of metabolic changes that occur in this type of diabetes, it was considered essential to propose a clinical, rather than geographical, descriptive classification.

A worldwide disorder, diabetes mellitus is recognised as a growing problem in developing countries. In the island of Nauru, in the Western Pacific Region, one-quarter of the adult population are known to suffer from the non-insulin-dependent form of diabetes. In migrant communities of Asian Indians now living in Fiji, Mauritius, Singapore and South Africa, some ten per cent of adults suffer from the disease. Whilst prevalence may be low in populations which maintain a traditional lifestyle, no country is free of the disease.

Whereas non-insulin-dependent diabetes is generally associated with over-nutrition, diabetes is also associated with under-nutrition. Official recognition of the malnutrition-related form of diabetes emerged from the report of a WHO study group on diabetes in 1985 (published in WHO's Technical Report Series No. 727).

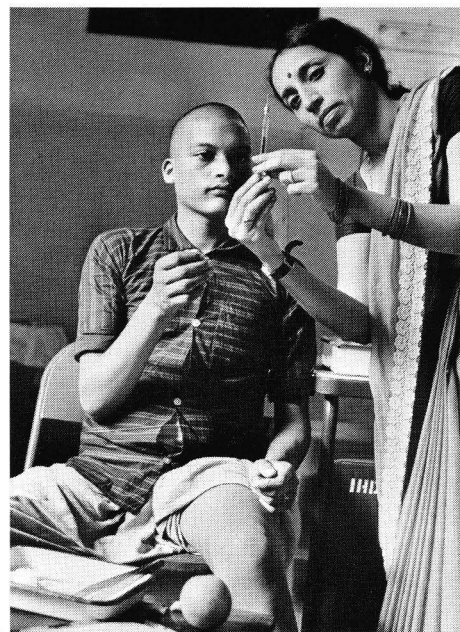
In July this year, WHO co-sponsored a meeting organised by the Wellcome Tropical Institute in London which, for the first time, brought together experts from around the world to consider the problem of malnutrition-related diabetes and its potential prevention.

The FCPD syndrome has been described from several countries, including Bangladesh, Ghana, India, Indonesia, Jamaica, Madagascar, Nigeria, Sri Lanka, Thailand, Uganda, Zaire and Zambia. The prevalence is especially high in South India (Kerala), Indonesia, Nigeria and Zaire. In these countries, 30 to 35 per cent of young diabetics with age of onset of disease below 30 years have this type of diabetes. The key feature is widespread formation of calculi (stones) in the main pancreatic duct and its

branches, *without* any calcification in the pancreas itself. The calcification is easily shown by radiological examination, or by newer techniques such as ultrasonography and computerised tomography. The natural history of the disease is characterised by attacks of abdominal pain at a young age—usually below ten years, diagnosis of diabetes during the following five to ten years, leading on to diabetes-related complications by the age of 30 years.

Although the precise cause and mechanism of FCPD is not known, there is evidence to suggest association with protein malnutrition in early childhood and with excessive intake of cassava, also known as tapioca. Cassava is one of the major tuber crops, grown in more than 80 countries located on both sides of the equator. While it has achieved considerable agricultural importance as a major source of cattle feed in the countries of the European Economic Community, it also provides an important source of calories for around 500 million people living in tropical developing countries.

The tubers may be peeled, boiled in water, and mashed. Slices of fresh tubers are fried in oil and form a common snack in Kerala. In Brazil, the tubers are often cooked in sugar syrup and eaten as a sweet meal. Gari, a food consumed in



A lesson in self-injection for a young diabetic in India.

Photo WHO/A. S. Kochhar



Patients waiting for a check-up at the diabetes clinic of a London hospital.

Photo WHO/P. Larsen

Nigeria and Ghana, is prepared by roasting the fermented pulp of cassava in hot pans. In the Philippines, the juice is squeezed out of the pulp which is then made into pellets and dried, and these are used as a substitute for rice and maize.

Kerala State, in the south of India, has the monopoly of its cultivation, with 83 per cent of the total area and 88 per cent of total production. And the largest prevalence of FCPD in India is reported from Kerala.

Cyanoglucosides are important chemical constituents of cassava. And it seems that excessive cassava intake *combined* with a low intake of proteins, deficient in certain amino acids, provides the essential milieu for the accumulation of cyanide in the body, with possible resultant toxicological effects.

Alternative sources of dietary cyanide include sorghum, yam, millet, maize, lima beans and linseed. Could there possibly be other toxic factors, especially food toxins, which may interact with malnutrition to produce similar clinical and morphological changes? We need much more information about other factors which, either singly or in concert with protein-energy malnutrition, may lead to FCPD syndrome.

As cyanoglucosides in cassava have also been implicated in the etiology of tropical ataxic neuropathy and of endemic goitre in several regions of Africa, the methods of reducing their content to within safe limits for human consumption constitute an important interven-

tion strategy. The suggested safe limit is 50 milligrams per kilogram of freshly grated cassava; any amount between 50 and 80 mg is probably toxic, 80 to 100 mg is definitely toxic, and above 100 mg may prove to be fatal. A large amount of cyanoglucosides can be eliminated by peeling and washing the tuber before eating. Sun drying the tapioca slices may destroy about 75 per cent, and cooking in water about 80 per cent.

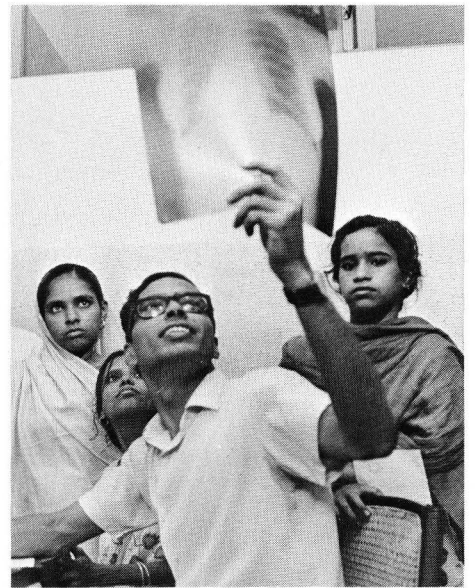
To improve the protein content of foods prepared from cassava, efforts have been made to supplement such foods with groundnut and soyabeans. It is also possible to mix cassava flour with wheat flour. Tapioca macaroni prepared by blending tapioca and groundnut flour with wheat semolina in the ratio of 60:15:25 has a final protein content of about 12 per cent.

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Research is being carried out to develop varieties of cassava that are low in cyanogenic glucosides but high in protein content. This has resulted in increases in the protein content as high as 42 per cent.

Protein-deficient diabetes mellitus (PDDM) has been described as J-type, K-type or M-type of diabetes. Essentially an absence of bouts of abdominal pain, a lack of demonstrable pancreatic calcification and an absence of pancreatic exocrine dysfunction differentiate FCPD from this sub-type. In contrast to FCPD, where an interaction

of malnutrition and environmental factors such as dietary toxins constitutes the key to the understanding of pathogenesis, there is now considerable evidence that protein malnutrition in early childhood initiated functional impairment of pancreatic beta-cells in the case of PDDM. Earlier observations which indicated alterations in carbohydrate metabolism in cases of kwashiorkor (a severe protein deficiency disease) provided a significant clue to the role of protein deficiency as a possible causative factor in PDDM. It seems that nutritional injury, in the form of protein malnutrition in early infancy and childhood, results in partial failure of beta-cell function. A continuing low level of protein intake may make this process progressive and irreversible, resulting in the clinical onset of diabetes mellitus at a young age.



Because of the high incidence of tuberculosis among diabetics, medical check-ups include a chest X-ray.

Photo WHO/A. S. Kochar

Whatever the causative mechanisms of these two types of the disease, there is clearly an urgent need to plan intervention strategies for diabetes care and control in developing countries, based on sound epidemiological and demographic premises. Only then can these countries move towards primary prevention of diabetes—a disease affecting a large segment of the young population. ■