ABSTRACT

CLINICAL AND BIOCHEMICAL CHARACTERIZATION OF MALNUTRITION RELATED DIABETES MELLITUS

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The aims of this study were to characterize clinically and biochemically Malnutrition Related Diabetes Mellitus (MRDM) as it presents in Jamaica (J-type or Phasic Insulin Dependent Diabetes Mellitus) and to examine the food toxin (linamarin)/malnutrition hypothesis as a possible aetiological factor in the onset of MRDM.

The study was broadly divided into two major areas:

(i) Clinical or patient study

(ii) Animal model study

The patient population consisted of thirty-four, (23 females, 17 males) who attend the diabetic outpatient clinic of the University Hospital of the West Indies. This group consisted of 14 phasic insulin dependent diabetes mellitus (PIDDM) (8 females, 6 males) 10 non-insulin dependent diabetes mellitus (NIDDM), (5 females, 5 males) and 10 non-insulin dependent diabetes mellitus (IDDM), (6 females, 4 males). Ten (10) normal (4 females, 6 males) subjects also participated.

The diabetic and normal subjects were matched for age, body mass index and duration of diabetes (among diabetics). the IDDM subjects had a mean BMI of

23.2±1.9 kg/m^2 and mean age of 54.0±5.4 years. The duration of diabetes in this population was 14.7±2.7 years. NIDDM patients had diabetes for 11.8±1.4 years mean age of 59.0±2.9 years and BMI was 24.1±0.9 kg/m^2. The PIDDM cohort had a mean BMI, age and duration of diabetes of 24.7±0.9 kg/m^2, 57.0±3.2 years and 16.0±1.3 years respectively. Normal controls had a mean age of 47.0±4.5 years and BMI of 22.7 kg/m^2.

Comparative studies were performed on insulin receptor binding, hormonal profile, glucose tolerance, blood status, renal, hepatic and pancreatic function. In addition, blood pressure measurements, flat abdominal x-ray and ultrasound were done, in types I, II and PIDDM along with the normal controls.

The results show a significantly decreased white and red blood cell binding to insulin ($P<0.05$), extensive kidney damage ($P<0.05$) and increased pancreatic echogenicity in PIDDM.

These findings support a separate identity of the latter syndrome from Types I and II diabetes mellitus.

In the animal model study, the dogs used were male and female mongrels obtained from the pre-clinical animal house where they were maintained on a diet of cornmeal cooked with chicken, fortified with Purina

laboratory chow and water ad libitum. Malnutrition was induced by restricting the diet to a very small quantity of cornmeal only for a period of 7-10 days. The recovered dogs were re-fed the normal diet with added milk and multi-vitamin supplement.

The total dog population was 35, (15 normals, 10 malnourished and 10 recovered). On each dog, a control glucose tolerance test, plasma insulin, counter-regulatory hormones, and receptor studies were done. After feeding linamarin, a residence time of half hour was allowed to elapse before investigations were repeated.

Linamarin dosage of 20 mg per kg body weight induced abnormally high blood glucose levels, and in two cases the hyperglycaemia was sustained for several days. There was an associated decrease in binding of insulin to erythrocytes and mononuclear leucocytes, as well as prevailing hypoinsulinaemia in the linamarin-induced hyperglycaemic conditions.

This animal study presents a possible aetiological model for Malnutrition Related Diabetes Mellitus.