Retinopathy in Tropical Pancreatic Diabetes

This letter is in reference to those of Davidson and Smith (1) and Couet and Drouin (2). Davidson and Smith refer to Geervarghese’s book published in 1968 (3), which mentions the low prevalence of retinopathy in tropical pancreatic diabetes (TPD) in southern India. We have recently shown that both sight-threatening forms of retinopathy, i.e., proliferative retinopathy and maculopatHy, are seen in patients with TPD when these patients are followed for long periods (4). Neuropathy (5) and nephropathy (6) are also seen in these patients. The frequency of microvascular complications was not significantly different from that of a matched group of non-insulin-dependent diabetic patients (6). Note that in Geervarghese’s more recent book (7), there is a separate chapter on diabetic retinopathy in TPD. The subject of microvascular complications in TPD has been reviewed by us recently (8,9). Macrocovascular complications are less common in this entity (6). This could be related to the relative youth, leanness, or low lipid levels of these patients (6). Prospective studies are needed to determine the natural history of vascular complications in TPD because of the special characteristics of this form of diabetes, which include association with protein-calorie malnutrition and relative infrequency of ketosis despite requiring insulin for stabilization of diabetes (10).

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LETTERS AND COMMENTS

extreme hyperkalemia associated with severe hyperglycemia and ketoacidosis.
A 62-yr-old diabetic male, insulin dependent since age 52 yr and complicated by proliferative retinopathy, peripheral neuropathy, and mild renal failure, had been admitted to our hospital for diabetic ketoacidosis and discharged a week before the episode reported herein. His only medications were 12 U regular insulin in the morning and 18 U before dinner and 30 U NPH insulin at night.
He was admitted to the emergency room in a stupor. Table 1 summarizes the main laboratory findings. The ECG showed lack of P-waves and marked widening of the QRS complexes. Endogenous CaCl₂, NaHCO₃, and regular insulin were administered, but he developed ventricular fibrillation and, finally, despite cardioversion, cardiac arrest. Intracardiac epinephrine was injected and resuscitative maneuvers continued over 45 min without success. In that critical situation an intracardiac injection of regular insulin (0.5 U/kg) was administered. Two minutes later he started a sinusual rhythm with subsequent hemodynamic normalization, and consciousness was restored. His hyperkalemia and ketoacidotic state was resolved in a few hours with conventional treatment. Later, the patient revealed that he abandoned insulin therapy 3 days before admission.
Hyperkalemia is a well-known initial finding in untreated diabetic ketoacidosis and is thought to be a major cause of death (3). Delirious effects on electrical activity of the heart are by far the most important consequences of hyperkalemia. The electrically active tissues of the heart are particularly sensitive to changes in the extracellular concentration of K⁺. Thus, hyperkalemia may result in dysrhythmia due to increased automaticity, reflecting repetitive depolarizations. At higher concentrations of K⁺ (8–9 meq/L), there is profound depression in impulse generation and conduction in all cardiac tissues, widening of the QRS complex, and...