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The Possible Role of Insulin Release in Cardiovascular Morbidity and Mortality in NIDDM Patients

In his commentary, Lebovitz (1) underlines the roles of hyperglycemia and insulin resistance as risk factors for macrovascular disease in non-insulin-dependent diabetes mellitus (NIDDM), but he does not emphasize the roles of obesity and insulin release. NIDDM is a different disease in obese (mainly insulin resistant) and nonobese (mainly insulinopenic) patients. Body weight also seems important in the clinical picture of secondary failure (SF) of oral hypoglycemic agents, as SF is usually irreversible in nonobese patients, but reversible after weight loss in the obese (2).

In SF patients, we have evaluated risk factors for major complications and mortality. In 1989, 76 SF patients, 51 obese (body mass index [BMI] 32.8 ± 0.8 kg/m^2), and 25 nonobese (BMI 23.6 ± 0.3, $P < 0.01$), were hospitalized to ameliorate metabolic control, including body-weight reduction and adjustment of insulin treatment (patients with serum creatinine levels >1.5 mg/dl were excluded), and to evaluate C-peptide release and diabetic complications. Obese patients differed from nonobese in hypertension (systolic blood pressure [BP] 157 ± 2.9 vs. 147 ± 4.5 mmHg, $P < 0.05$; diastolic BP 91 ± 1.8 vs. 85 ± 1.9 mmHg, $P < 0.05$); use of hypotensive drugs 31/51 vs. 7/25, $P < 0.05$) and C-peptide levels (2.2 ± 0.3 vs. 1.2 ± 0.2 ng/ml, $P < 0.05$). On univariate analysis in 76 patients, ischemic heart disease (IHD) was associated with C-peptide (2.2 ± 0.3 vs. 1.0 ± 0.2 ng/ml, $P < 0.01$ at 0 min; 3.7 ± 0.5 vs. 1.7 ± 0.4 ng/ml 6 min after intravenous glucagon, $P < 0.05$), serum creatinine levels (CREA, 1.0 ± 0.0 vs. 0.9 ± 0.0 mg/dl, $P < 0.01$), and diastolic BP (92 ± 1.7 vs. 86 ± 2.1 mmHg, $P < 0.05$). CREA correlated with age ($r = 0.29$, $P < 0.05$). At stepwise regression analysis (Statview II, Macintosh), C-peptide and CREA were significant risk factors for IHD, age and sex for CREA.

Status was ascertained 48 months later in 39 patients. Six patients (five obese) had died. At baseline they differed from live patients for age (65 ± 1.4 vs. 58 ± 1.5 y, $P < 0.05$), BMI (31.6 ± 2.1 vs. 27.1 ± 0.9, $P < 0.05$), insulin dose (51 ± 12.5 vs. 29.4 ± 3.6 U/day, $P < 0.05$), C-peptide (1.1 ± 0.2 vs. 0.6 ± 0.1 ng/ml at 0 min, $P < 0.01$; 1.6 ± 0.3 vs. 1.0 ± 0.1 ng/ml at 6 min, $P < 0.05$), serum triglycerides (298 ± 75 vs. 156 ± 13 mg/dl, $P < 0.01$), CREA (1.2 ± 0.1 vs. 0.9 ± 0.0 mg/dl, $P < 0.01$), and IHD (5/6 vs. 10/33, $P < 0.05$). At stepwise regression analysis, C-peptide was the only significant risk factor for mortality.

In our view, these data underline the primary role of insulin resistance, linked inter alia to BMI, in cardiovascular morbidity and mortality and suggest that insulin or drugs stimulating insulin release should be avoided in the presence of well preserved insulin release (1–3), as is in obese NIDDM patients.

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Intrauterine Devices Are Safe and Effective Contraceptives for Type I Diabetic Women

Further evidence

Two prospective controlled trials (1,2) with state-of-the-art copper intrauterine devices (IUDs) in type I diabetic women have shown that IUDs are as safe and effective as in nondiabetic women. However, there is still a widespread belief among physicians that diabetic women have a higher risk of IUD-associated pelvic inflammatory disease (PID) (3). The skepticism is probably due to the fact that in one of the above studies, 103 women were followed for only 1 year (1) and in the other study, only 58 women were evaluated, albeit for 3 years