Lipoatrophy and monocomponent porcine insulin

Lipoatrophy occurs in about 10% of patients receiving conventional insulin.1 Highly purified porcine insulin is not generally thought to cause this complication and is normally advocated for its treatment.1 We describe here, however, a case of lipoatrophy in a patient treated with monocomponent insulin.

Case report
A woman, now aged 60, was first seen in June 1958 after glycosuria had been found on routine urine analysis. An oral glucose tolerance test confirmed the diagnosis of diabetes mellitus and she was treated with diet and chlorpropamide (250 mg/day). During November 1977 she was admitted with ketocidosis precipitated by an upper respiratory tract infection and was treated with continuous intravenous infusion of monocomponent insulin (Actrapid MC). While in hospital she received one injection of Insulin Novo Rapitard (16 units subcutaneously). She was discharged taking insulin zinc suspension (Monotard MC) 36 units a day subcutaneously.

In November 1979 she developed lipoatrophy at injection sites on her thighs (figure) and upper arms. The hospital case notes and treatment sheets and her general practitioner’s records yielded no evidence that she had received any insulin other than those mentioned. Biopsy specimens taken from the edge of lipoatrophic areas were snap-frozen in liquid nitrogen, and immunofluorescence analysis was performed.3 Mild speckling of C3 was seen at the dermal-epidermal junction and a mild diffuse increase in fibrin and fibrinogen in the upper dermis, but no IgM, IgG, C3, or Clq was deposited in the blood vessel walls. Serum insulin binding capacity, assayed by a second antibody coprecipitation assay specific for IgG antibody,7 was 15.9 μg/l. Serum insulin-specific IgE, measured by radioimmunoassay, was normal.

She was instructed to inject Actrapid MC (12 units) and Monotard MC (36 units) into the middle of the lipoatrophic areas. By July 1980 the affected areas had filled out (figure).

Comment
A similar case of insulin-induced lipoatrophy was reported by Hanai et al in Japan.4 Their patient had also been treated initially by diet and with a sulphonylurea and was later transferred to Monotard MC insulin. Unfortunately, the response to treatment was not mentioned. Our patient, however, responded well to monocomponent porcine insulin injected into the affected areas.

The cause of insulin-induced lipoatrophy is unknown. An immune pathogenesis in patients treated with conventional insulins was suggested by Reeves et al.4 They showed abnormal deposition of immunological components in the dermal blood vessels of all biopsy specimens taken from the edge of lipoatrophic sites. These were not present in our patient. The findings of a mild speckling of C3 at the dermal-epidermal junction and a mild diffuse increase in fibrin and fibrinogen in the upper dermis are non-specific and alone are insufficient to confirm the presence of an immunological reaction. The insulin binding capacity in this patient was 15.9 μg/l; Reeves et al reported a range of 12.7-58.8 μg/l, with a mean of 33.1 μg/l in patients with lipoatrophy who had received conventional insulins and of less than 22.3 μg/l in patients who had received conventional insulins but did not have lipoatrophy.4 There was no evidence of a local immunological reaction in our patient.

We suggest that the pathogenesis of lipoatrophy in our patient, treated exclusively with monocomponent insulin, may differ from that in conventionally treated patients.

We thank Dr W G Reeves, Department of Immunology, University Hospital, Nottingham, for his reports on the immunofluorescence studies and assay of insulin binding capacity.