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Diabetologia

Letters

Comments

-to: Charlton R, Smith G, Day A (2001) Munchausen's syndrome manifesting as factitious hypoglycaemia. Diabetologia 44: 784–785

To the Editor: I read with interest the *Observations* of Drs. Charlton, Smith and Day in your June issue [1] about factitious hypoglycaemia. The authors searched and reviewed literature between 1966 and 1999 using Medline. They further state that they had identified 46 papers, containing 69 case reports, with a range of one to four case reports in each paper. I have to assume, therefore, that they inadvertently omitted our paper [2] which reported the largest number (10) of patients with surreptitious hypoglycaemia along with the longest follow-up (15 years). In our 1988 report, we divided patients presenting with factitious hypoglycaemia into two groups (five in each category): those who were not known previously to take insulin and those in whom use of insulin had been sanctioned by the medical profession. We outlined the difficulty of making and confirming the diagnosis, the prevalence of women (90%) with knowledge of the medical profession and the poor long-term prognosis of the majority of those patients (only three made a successful transition to a productive life after the diagnosis of factitious hypoglycaemia was established). These are essentially the same points now made by Charlton et al.

G. Grunberger

References

1. Charlton R, Smith G, Day A (2001) Munchausen's syndrome manifesting as factitious hypoglycaemia. Diabetologia 44: 784–785
2. Grunberger G, Weiner JL, Silverman R, Taylor S, Gorden P (1988) Factitious hypoglycemia due to surreptitious administration of insulin: Diagnosis, treatment, and long-term follow-up. Ann Int Med 108: 252–257

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-to: T. Skoog et al. (2001) Tumour necrosis factor-α (TNF-α) polymorphisms *–857C/A* and *–863C/A* are associated with TNF-α secretion from human adipose tissue. Diabetologia 44: 654–655

To the Editor: Skoog et al. have recently described that the secretion of TNF-α from adipose tissue varied among non-obese subjects according to the *TNF-α –863C/A* polymorphism [1]. Adipose tissue from subjects with the rare allele *–863/A* secreted less TNF-α than adipose tissue from non-obese subjects carrying the *–863C* allele. This indicated that *C* to *A* substitution at position *–863* represents a functional polymorphism, which leads to decreased *TNF-α* gene expression and thereby less production and secretion of the cytokine [1]. We have described that the *TNF-α –308G/A* polymorphism was associated, in parallel to constitutively different transcription rates of the cytokine, with increased body fat and insulin resistance [2]. We investigated whether the pattern of TNF-α secretion attributed to *TNF-α 863C/A* was also linked to a different insulin action.

We studied 28 consecutive healthy subjects. Six of them carried the *–863A* allele and 22 were homozygotes for the *–863C* allele (allele frequency 0.21/0.79, distribution in Hardy-Weinberg equilibrium). The two groups were similar in age, BMI and gender distribution (Table 1). The insulin sensitivity index (frequently sampled intravenous glucose tolerance test with minimal model analysis) was higher in *–863A* subjects than in