Diuretics and Idiopathic Oedema

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Dear Sir,

There has been considerable controversy concerning the role of diuretics in the pathogenesis of idiopathic oedema (IO) of women. The syndrome is characterized by excessive postural fall of plasma volume and progressive accumulation of fluid during the day. It has been proposed that the syndrome is entirely due to diuretics which cause the hypovolaemia and stimulate renal sodium and water-retaining mechanisms [1]. Diuretic withdrawal leads to acute sodium and water retention due to prolonged action of the renin/angiotensin system, and this 'rebound oedema' perpetuates the syndrome and the apparent need for the diuretic. This proposal has not met with universal agreement [2-6]. Our recent experience also indicates that diuretics are not an important factor in the pathogenesis of the disorder.

Twenty-four women who met the diagnostic considerations of Thorn [7] for IO have been seen in our department during the last 3 years. Their age range was 17-54 years (mean 34 years), and the duration of symptoms varied from a few months to several years. At presentation, 11 women were taking a diuretic; 6 had received diuretics in the past, but had discontinued them as ineffective, and 7 women had never taken a diuretic. It was possible to withdraw diuretics without difficulty in 8 patients, and 2 women who refused to discontinue treatment were changed to spironolactone. These 10 patients have continued to have episodes of generalized swelling. In only 1 woman did diuretic abuse seem an important factor.

This patient, a 35-year-old woman with 2 children, had a history of intermittent swelling for 10 years and for 3 years had been treated with furosemide, the dose of

Fig. 1. Changes in weight, plasma aldosterone, and plasma renin activity in a woman with idiopathic oedema following withdrawal of fruse-mide.

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which she would vary between 80 and 120 mg daily depending on the severity of her symptoms. She was persuaded to discontinue the furosemide which was supervised in hospital with bed rest and a low-sodium diet to minimize 'rebound oedema'. A combined thyrotro-phin-releasing hormone and luteinizing hormone releasing hormone (TRH/LH-RH) test, which has previously been reported as abnormal in IO [8], was performed and showed exaggerated prolactin and luteinizing hormone responses. Diuretic withdrawal was followed by weight gain and uncomfortable swelling (fig. 1). Plasma renin activity (reference range 0.6–5.6 ng ml⁻¹ h⁻¹) and plasma aldosterone concentration (reference range 100–500 pmol l⁻¹) were measured at intervals and were initially high, but rapidly returned to normal followed by natriu-resis and a fall in body weight. In these respects this patient behaved in the typical manner of a diuretic abuser [1]. However, diuretic withdrawal has not resulted in resolution of her symptoms which have persisted during a 3-year follow-up period. It was unlikely that she had occultly reverted to diuretic abuse, as the plasma renin activity measured at 6 and 9 months after stopping the diuretic was normal. The TRH/LH-RH test repeated at these times remained abnormal, suggesting that these abnormalities may be related to the primary fault in the disorder.

Diuretic abuse as the cause for IO is an attractive proposal, as it would explain the postural hypovolaemia and increased renal sodium-retaining mechanisms. However, we have not found it to be an important factor in our patients. Several conditions including early hypothyroid-ism, mild cardiac failure, and changes in dietary sodium and carbohydrate may produce syndromes similar to IO and need to be excluded before the diagnosis is established. Diuretic abuse would also appear to be in this category.

References

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Erratum

In the paper by Rydzewski et al., entitled ‘Concentration of Three Thrombin Inhibitors in the Nephrotic Syndrome in Adults’, published in *Nephron* 42: 200–203 (1986), the term α macroglobulin (α M) should be corrected and read α_2 -macroglobulin (α_2 -M).

Obituary

Prof. C. J. Hodson†

We regret to announce the death of Professor C.J. Hodson who passed away on December 1, 1985. He was a