Goiter and Uremia

L.

Laszlo

Hegedüs

Department of Internal Medicine and Endocrinology F, Herlev Hospital, Herlev, Denmark

Laszlo Hegedüs, MD, Department of Internal Medicine and Endocrinology F, Herlev Hospital, DK-2730 Herlev (Denmark)

Dear Sir,

We read with great interest the article by Sennesael et al. [1] addressing among others the question of goiter frequency in uremia. While we agree with their results and conclusions on free thyroid hormone concentrations, the data presented on thyroid size evaluation does not (for a number of reasons) allow any conclusion to be drawn as to the possible goitrogenic effect of uremia.

Attention has to be drawn to the inexactness and irreproducibility of the clinical evaluation (palpation) of thyroid size [2] and it has to be stressed that such an evaluation does not with any reliability allow a quantitative evaluation of thyroid size. This could have been achieved by using ultrasound [3] and is necessary if one wishes to demonstrate alterations in thyroid size in a range of thyroid volume where no goiter is evident. Therefore, an increase in thyroid volume could well have been demonstrated, in the study by Sennesael et al. [1], in spite of the failure to demonstrate a difference in goiter frequency between patients and controls. In fact this is exactly what we found in a recent investigation on this topic [4]. Furthermore, the necessity of matching the patients with the controls in respect to a number of variables has been shown to be of crucial importance if one wishes to compare goiter frequency with any reliability. Thus, sex, age and body weight [3], liver disease and alcoholism [5], smoking habits [6] and changes within the menstrual cycle (unpublished) all influence thyroid gland volume and thereby goiter frequency. The suggested geographical variations in goiter frequency in uremia has tentatively been attributed to differences in daily iodine intake and excretion. However, daily urinary iodine excretion is very similar in Brussels (60 µg/day [1] and Copenhagen (80 µg/day) [7] further supporting the view that enlargement of the thyroid gland is related primarily to uremia and not environmental factors. The explanation for the conflicting findings might, therefore, rather be the inexactness of the clinical evaluation of goiter, stressing the importance of an objective assessment of thyroid size.

Some authors have found that as many as 20–40% of patients with hyperparathyroidism had thyroid lesions of surgical significance [8] and we have shown that 9 of 23 consecutive patients with primary hyperparathyroidism had goiter and increased thyroid gland volume measured ultrasonically [9]. In view of the known association of uremia and hypercalcemia and hyperparathyroidism it is possible that the subjects with goiter (in our as well as other studies) were the ones with hypercalcemia and/or hyperparathyroidism. The goiter could possibly also be caused by mistaking grossly enlarged parathyroid glands for thyroid tissue. Such a palpatory distinction between the two tissues is certainly afflicted with much uncertainty.

Since the completion of our study [4] a number of these patients have been suspected of having hyperparathyroidism and many have been parathyroidectomized or are awaiting this operation. Ongoing studies will hopefully shed further light on the pathogenesis of thyroid enlargement in uremia.

Goiter and Uremia

151

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