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CASE STUDY

Goitre: a cause of obstructive sleep apnoea in euthyroid patients

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Goitre: a cause of obstructive sleep apnoea in euthyroid patients. P.C. Deegan, V.M. McNamara, W.E. Morgan. ©ERS Journals 1997.

ABSTRACT: While hypothyroidism is considered to predispose to obstructive sleep apnoea (OSA), the presence of a goitre itself is not a recognized cause of OSA.

We present the cases of two euthyroid patients with large goitres and clinical evidence of OSA, whose OSA symptoms significantly improved following partial thyroidectomy.

This finding suggests that the goitre contributed to their symptoms.

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Hypothyroidism is viewed as a risk factor both for obstructive [1–5] and central [6, 7] sleep apnoea. Replacement therapy with thyroxine usually leads to amelioration or even cessation of the associated apnoea. Proposed mechanisms linking the two conditions include: narrowing of the upper airway (UA) by deposition of mucopolysaccharides and protein extravasation into the tissues of the face, tongue and pharyngeal structures [1], dysfunction of UA dilator muscles due to hypothyroid myopathy [2, 4], or abnormalities of ventilatory control [3, 6]. However, little is known about whether an enlarged thyroid gland contributes to sleep-disordered breathing, with only one previous report of an acromegalic patient with obstructive sleep apnoea (OSA) and respiratory failure, who improved after thyroidectomy [7]. We present the cases of two euthyroid patients with large goitres and OSA requiring treatment with nasal continuous positive airway pressure (nCPAP).

Case No. 1

A 59 year old man, who presented after a gardening accident, was incidentally found to have a deviated trachea on examination and this was confirmed on chest radiography. Clinically, he was euthyroid and had no symptoms of UA obstruction. However, he had biochemical evidence of hyperthyroidism, with an elevated serum thyroxine (T_4) of 196 nmol·L^{-1} (reference range 53–135 nmol·L^{-1}) and free T_4 index of 190 (reference range 59–127). A computerized tomographic (CT) scan of the neck and thoracic inlet revealed diffuse enlargement of the thyroid gland, especially of the left lobe, extending upwards to the hyoid arch and retrosternally down to the level of the aortic arch. Prior to surgery, the patient was made euthyroid with carbimazole. However, during follow-up, he reported excessive daytime sleepiness, with heavy snoring, grunting and gasping during sleep. Examination of his UA revealed a deviated nasal septum and a bulky uvula.

On overnight oximetry, the patient had a total of 58 dips in arterial oxygen saturation (S_{a,O_2}) of ≥4% (7.4 episodes·h^{-1}), the lowest being to 75%, and an obstructed pattern of breathing and heavy snoring on simultaneous video-recording. A diagnosis of clinically significant OSA was made. He had a further sleep study whilst on 7.5 cmH_2O of nCPAP, with a subjective improvement in sleep quality, abolition of snoring and a reduction in the number of ≥4% dips in S_{a,O_2} to a total of 11. After commencing nCPAP at home, he reported that he no longer snored, with more restful sleep and a reduction in daytime sleepiness. After 1 month of continuous therapy with nCPAP, he underwent hemithyroidectomy; the left lobe of his thyroid, measuring 13 × 7 × 6 cm in size and weighing 150 g, was removed through a left collar incision. Histology revealed a multinodular goitre. At this point, his height was 171.5 cm and weight 77.5 kg, giving a body mass index (BMI) of 26.2 kg·m^{-2}, with normal spirometry and arterial blood values.

On returning home, the patient discontinued nCPAP and continued to sleep well, without any daytime somnolence. A repeat sleep study off nCPAP 4 months later revealed 28 dips of ≥4% in O_2 saturation (3.6 episodes·h^{-1}), the lowest saturation being 89%, and he had similar findings on a further sleep study 8 months later. During this period his weight remained unchanged, being 77.2 kg at the time of the last sleep study. On review 3.5 yrs later, he had maintained his improvement, with only occasional snoring and no daytime sleepiness.

Case No. 2

A 70 year old man was referred by his general practitioner, for investigation and treatment of suspected

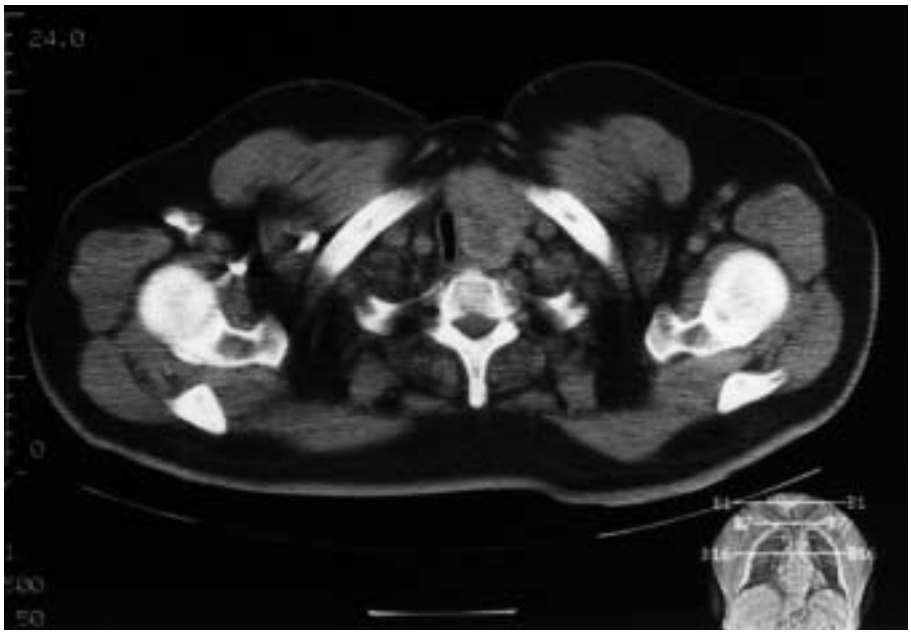

Fig. 1. – Computed tomographic scan at the level of the thoracic inlet in Case No. 2. The trachea is narrowed and deviated to the right by an enlarged thyroid gland.

hypothyroidism. He had a goitrous swelling in his neck, increasing in size over the preceding 3 yrs, with associated increasing shortness of breath, a weight gain of 20 kg, hoarseness, and a feeling of being tired all the time. His weight was 116.2 kg and height 180 cm, giving a BMI of 35.9 $kg \cdot m^{-2}$. However, he was biochemically euthyroid, with a T_4 of 17 $pmol \cdot L^{-1}$ (reference range 10–20 $pmol \cdot L^{-1}$) and thyroid stimulating hormone (TSH) of 0.1 $mU \cdot L^{-1}$ (0.5-5.5 $mU \cdot L^{-1}$). CT of his neck and thoracic inlet demonstrated generalized enlargement of the thyroid gland, particularly of the left lobe. Although there was no retrosternal extension, there was marked tracheal compression from the subglottic region to the thoracic inlet, with pronounced reduction in transverse tracheal diameter (fig. 1). Flow-volume loops showed no evidence of UA obstruction. The patient had a restrictive ventilatory defect, with a forced expiratory volume in one second (FEV_1) of 1.45 L (45% of predicted), and normal FEV_1/forced vital capacity (FVC) ratio; this finding was considered to be secondary to his obesity.

When assessed prior to surgery, the patient reported heavy snoring every night, with repeated arousals from sleep, apnoeas observed by his wife and increased daytime sleepiness. Examination of his UA revealed a very bulky uvula, with a small retroglossal space. On overnight oximetry, he had 102 dips in S_{a,O_2} of ≥4% over a period of 273 min (frequency of 22.4 $episodes \cdot h^{-1}$), and the lowest S_{a,O_2} was 60%. He snored heavily and video surveillance showed a breathing pattern consistent with UA obstruction. There was also a progressive decline in the baseline S_{a,O_2} during the night, compatible with an element of obesity hypoventilation. A repeat study 1 month later on 7.5 cmH_2O nCPAP demonstrated a significant reduction in desaturation frequency to 2.5 $episodes \cdot h^{-1}$. The patient felt less sleepy on the following morning, and this was maintained at home when he was commenced on domiciliary nCPAP.

After 2 months of therapy, the patient had the left lobe of his thyroid removed through a collar incision. It weighed 226 g and demonstrated a multinodular goitre on histological examination. On returning home, he discontinued his nCPAP therapy. At 1 month following his operation, he reported satisfactory sleep quality with no daytime sleepiness. His wife stated that he no longer snored and there were no observed apnoeas. His weight was unchanged at 116.4 kg. Overnight oximetry at home demonstrated few O_2 desaturations (3 $episodes \cdot h^{-1}$), with only a slight drift downwards in baseline to 90%. On review, 18 months postsurgery, his snoring had not returned and he had no daytime sleepiness.

Discussion

It was concluded in both of the cases presented that the observed improvement in symptoms was due to excision of the goitre, as neither patient experienced any postoperative weight reduction and there was no change in medication or level of alcohol consumption. It is unlikely that the goitre was the sole factor leading to UA obstruction, as both patients had abnormal UAs, and patient No. 2 was significantly obese. However, the enlarged thyroid gland probably converted a mild degree of obstructed breathing into a more severe form requiring treatment.

It is only possible to speculate about the underlying relationship between large goitres and OSA. A very large goitre can obstruct venous return from the head and neck, resulting in engorgement and oedema of UA structures and reduced UA patency [7]. However, there was no clinical or CT evidence of this in either patient. It has also been shown that thoracic inspiratory activity improves UA patency and opposes the collapsing action of negative UA pressure by providing inspiratory increases in caudal traction on the UA that are transmitted through structures in the neck [8]. The presence of a goitre, with displacement of the trachea and other cervical structures, could have interfered with this process. Alternatively, the enlarged thyroid gland may have impaired the function of muscles attached to the hyoid bone, the anterior movement of which increases the patency of the hypopharynx and reduces UA resistance [9]. Finally, there may be a simple mass loading effect of the enlarged thyroid gland on the UA when the subject is supine during sleep [10].

In conclusion, these cases illustrate two main points. Firstly, that patients presenting with large goitres and tracheal displacement should be asked about symptoms of the obstructive sleep apnoea syndrome, and examined for risk factors. Sleep studies could be performed in suspicious cases. Secondly, the link between goitre

and obstructive sleep apnoea in the above patients suggests that some degree of obstructive sleep apnoea could persist in hypothyroid patients who also have a goitre, even after they become euthyroid.

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