Clinical Reports

Successful reversal of sleep apnoea syndrome following treatment for acromegaly, confirmed by polygraphic studies

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Summary: A patient with acromegaly was shown to have obstructive sleep apnoea by polygraphic recordings. Following successful hypophysectomy, further recordings demonstrated resolution of obstructive sleep apnoea.

Introduction

Obstructive sleep apnoea has been associated with acromegaly.1 We report a case in which objective evidence, from polygraphic sleep studies, demonstrated reversal of this condition in an acromegalic patient following hypophysectomy. This reversal, which has been suggested from other cases, has not previously been fully documented by sleep studies.

Case report

A 67 year old man complained of ‘gaps in memory’, daytime somnolence, dating back at least 13 years, and his wife complained that his breathing was irregular and noisy at night. He had typical clinical features of acromegaly and this diagnosis was confirmed biochemically from a glucose tolerance test with human growth hormone measured at a basal level of 29 mIU/l rising to 50 mIU/l at 120 minutes, following a 50 g glucose load.

Polygraphic sleep studies were performed using simultaneous electroencephalogram, electrocardiogram, ear lobe oximetry, air flow and chest movement recordings. On average, the patient had 10.1 periods of 10 second apnoea per hour, mainly of the obstructive type. Ear lobe oximetry showed many dips in arterial saturation, falling as low as 67% (mean 91%). These results confirmed a diagnosis of the sleep apnoea syndrome. The patient was initially unwilling to undergo surgical treatment and was treated with bromocriptine in doses up to 30 mg daily. However, not surprisingly, he had persisting biochemical evidence of acromegaly.

Trans-sphenoidal hypophysectomy removed an eosinophilic adenoma and the patient was started on hormonal replacement therapy with thyroxine and hydrocortisone. Three months after treatment, his growth hormone levels were not elevated, with a basal level of 3.0 falling to 2.0 mIU/litre 120 minutes after a glucose load. During the year following treatment, his growth hormone levels were not elevated, with a basal level of 3.0 falling to 2.0 mIU/litre 120 minutes after a glucose load. During the year following treatment, the patient reported resolution of his episodes of somnolence and improvement in his nocturnal breathing. The sleep studies were repeated 13 months after treatment (Figure 1). Less than one 10 second period of apnoea per hour of sleep occurred, with arterial saturation much less variable, dipping only to 85% (mean 95%).

Discussion

The sleep apnoea syndrome, mainly due to obstructive sleep apnoea, is well recognized in acromegaly.1,2 The mechanisms appear to be obstruction of the naso-pharyngeal airway by collapse of the walls and hyperplasia of mucosa.3

Resolution of symptoms associated with sleep apnoea in acromegaly after yttrium-90 implantation4 has been reported and improvement in sleep studies has also been observed in one patient as a result of treatment with a somatostatin analogue.5 However, we report here reversal of objective polygraphic sleep study evidence of the sleep apnoea syndrome following successful treatment for acromegaly.

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Figure 1 Polygraphic sleep recordings of apnoea, ear lobe oximetry and electroencephalographic stages, before and after hypophysectomy.

References