SLEEP APNOEA PRESENTING AS SEVERE HYPERTENSION AND SILENT OCCIPITAL HAEMORRHAGE

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ABSTRACT

A 39-year old Chinese man presented with an acute onset of severe headache, accelerated hypertension and subsequently an unexpected extensive right occipital haemorrhage. These were found to be related to a sleep apnoea syndrome which had been unrecognized for many years despite its typical symptoms of loud snoring and excessive daytime sleepiness. Weight reduction led to significant clinical but not polysomnographic improvement of the sleep apnoea syndrome.

Keywords : Sleep Apnoea, Hypertension, Occipital Haemorrhage

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INTRODUCTION

Sleep apnoea is a common disorder [1] that has been under-recognized in Western countries until several years ago [2] and is still so in most Asian countries. Although snoring and excessive daytime sleepiness are now much better known to be the typical presenting symptoms [3], we report a patient who had the unusual presentation of severe hypertension, headache and silent occipital haemorrhage.

CASE REPORT

A 39-year old Chinese jewellery maker in Hong Kong was urgently admitted for severe frontal headache for 2 days. He had a 5-year history of progressive weight gain and daytime sleepiness. He frequently dozed off at work and snored loudly at night. With past good health he had been repeatedly reassured by doctors that he was a "healthy deep sleeper". In the medical ward, he sustained high blood pressure of 190/120 and was treated with complete bed rest, metoprolol (100 mg o.m., 50 mg p.m.) and oral analgesic which gave only partial relief to the headache. Marked hypersomnolence associated with loud "resuscitative" snoring and excessive motor activity were noted. Examination showed a short obese man (78 kg; height 162 cm) who easily dozed off during conversation and could not fully co-operate with detailed neurological testing. Other systems were normal. A diagnosis of hypertension and possible sleep apnoea syndrome was made.

His blood pressure was gradually stabilized at 120/80 while routine laboratory investigations including 24 hour urine for VMA were normal. An inverted circadian rhythm of blood cortisol was noted but subsequent standard dose dexamethasone suppression test to exclude Cushing's syndrome was normal. He was referred for sleep assessment but the persistent severe headache led to a neurological reassessment which showed pronounced neck rigidity and marked left hemianopia confirmed with perimetric charting. There were no motor or sensory deficits in the face or limbs. Urgent CAT scan showed extensive right occipital lobe hematoma with intraventricular haemorrhage. This was treated conservatively. Overnight polysomnography a week later confirmed significant sleep apnoea and sleep fragmentation (Table I). ENT examination revealed a narrow oropharynx related to bulky tissues rather than any specific physical obstruction. The patient was put on a 2000 Cal diet, physical exercise for weight reduction and was discharged on metoprolol 50 mg b.d.

Seven weeks later, he reduced his body weight to 72.5 kg with significant diminution of daytime sleepiness and to a lesser extent, snoring. He felt like a different person. There was no more headache and blood pressure was well controlled. At 6 months' follow-up, a repeat

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CAT scan showed resolved intraventricular and intracerebral hematoma leaving a mildly dilated right lateral ventricle and slight midline shift, while clinical as well as perimetric reassessment indicated improved vision. Polysomnography showed reduction of the apnoea index and improved vision. Polysomnography showed reduction of the apnoea index and improved sleep architecture (Table I). The weight loss was maintained at 2-year follow-up, and the patient declined surgical treatment.

Table I

Polysomnographic findings before & after weight loss

	Pre-weight loss (78 kg)	Post-weight loss (72.5 kg)
Sleep stage 1	27.0%	18.4%
2	63.3%	63.2%
3	2.3%	5.3%
4	0.0%	0.0%
REM	7.4%	13.1%
Actual Sleep time	7h 46m 40s	7h 7m 20s
No. of apnoea episodes	287	181
No. of hypopnea episodes	120	85
Longest episode	54 secs	53 secs
Shortest episode	10 secs	10 secs
Lowest SaO (REM)	71%	77%
Lowest SaO (NREM)	83%	77%
Latency of apnoea attack	3 mins	37 mins
Apnoea index	36.9	25.4
Hypopnea index	15.3	11.9
Apnoea + Hypopnea index	52.2	37.3
Nature of apnoea : obstructive	203 episodes	139 episodes
mixed	84	42

DISCUSSION

Arterial hypertension is a well recognized complication of the obstructive sleep apnoea syndrome [4], whose treatment can reduce antihypertensive dosage. A 34-year old man with obstructive sleep apnoea and an established history of hypertension and diabetes mellitus presenting with stroke causing right-sided hemiplegia was reported before [5]. Our patient is different in his sudden onset of severe hypertension and intracerebral haemorrhage not associated with obvious neurological deficits; we do not know of similar case reports. There was also no evidence of trauma, coagulopathy or a vascular anomaly to account for the haemorrhage. The frontal headache was implicitly assumed to be due to sleep apnoea. However, it occurred suddenly and throughout the day rather than in the morning as in most sleep apneic patients [3]. He probably had the haemorrhage on first presentation, but it was not until it progressed to intraventricular haemorrhage and severe neck rigidity that neurological re-examination and the right diagnosis were made. The stress of severe headache, bedrest during hospitalization and his hypersomnolent state were inter-related and would explain the disturbed cortisol rhythm and the initial difficulty in an accurate neurological diagnosis. The presence of high blood pressure and persistent severe headache in a sleep apneic patient should therefore warrant a detailed neurological assessment.

The patient was falsely reassured to be a "deep sleeper" when actually his sleep, like sleep apneics in general, is fragmented and deficient in SWS (slow wave sleep) which is believed to be the recuperative part of the human sleep [6]. Although obesity, a well known association with the sleep apnoea syndrome [7], is not common in Chinese in Hong Kong, we have seen an increasing number of referrals of sleep apnoea over the past one year. Weight reduction is frequently difficult in these patients, but the absence of a readily correctible obstruction such as large tonsils [8] would make it worth a try. The weight loss of 10 kg led to significant improvement in clinical symptoms though less so polygraphically. Snorers with no excessive daytime sleepiness have been shown to have hundreds of apneic episodes on polysomnographic assessment [9]. It appears that there could be a threshold beyond which a slight increase in the severity of apnoea could lead to dramatic clinical deterioration. Through mechnical and metabolic mechanisms [7], weight reduction could make that clinical difference, although other methods of treatment may still be indicated in view of the long-term complications of the sleep apnoea syndrome [3].

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