Clinical Reports

A patient with coexisting narcolepsy and morbid jealousy showing favourable response to fluoxetine

Yun-Kwok Wing, Sing Lee, Helen F.K. Chiu, Crover K.W. Ho and Char-Nie Chen

Department of Psychiatry, Prince of Wales Hospital, Chinese University of Hong Kong, Shatin, Hong Kong

Summary: A 37 year old Chinese man suffered from coexisting narcolepsy and morbid jealousy which were precipitated by head injury 5 years previously. Fluoxetine 20 mg/day reduced his narcoleptic symptoms and morbid jealousy but not his sleepiness. On defaulting treatment, the patient's symptoms and marital problem recurred. A common central serotonin disturbance might be involved in mediating the sleep disorder and associated psychopathology.

Introduction

Narcolepsy is a common sleep disorder leading to daytime hypersomnolence. Associated psychopathologies such as personality change, depression and low self-esteem^{1,2} are not uncommon, but may be easily under-recognized. Although marital problems may occur, morbid jealousy has been rarely described.^{3,4}

Case report

A 37 year old Chinese married cleansing worker was referred to our sleep disorders clinic for excessive daytime somnolence and behavioural change for 5 years. He had enjoyed his marriage until a traffic accident 5 years ago when his car was crushed by a truck. After a transient loss of consciousness, he was admitted to a general hospital for observation for a few days. Several months later, his wife complained of his episodes of daytime sleepiness. He also reported attacks of sleep paralysis on a few occasions, but not hypnagogic and hypnopompic hallucinations. His wife witnessed several cataplectic attacks which were usually provoked by extreme anger. Moreover, he would fall asleep irresistibly 4–5 times a day while sitting, reading, driving and even eating. As a result, he changed from driving to a less demanding cleansing work. He did not use any stimulants, psychoactive drugs or alcohol.

He was described premorbidly as a considerate

person, but became irritable, impulsive and suspicious after the accident. He accused his wife of infidelity and frequently tried to extract a confession from her. He also searched for evidence of her extramarital affair by inspecting the linen bed and her underwear for semen stains. Often, he expressed doubt towards his own belief in view of the lack of confirmatory evidence. His wife pleaded innocence and felt annoyed by his mistrust. They had regular sex, but she disliked his increasing rudeness and they had slept separately in the subsequent 3 years. The patient did not suffer from any sexual dysfunction.

Physical assessment including nasal and oropharyngeal examination was unremarkable. His body weight was 64 kg with a height of 1.64 m. He scored only 2 on the 17-item Hamilton rating scale of depression (range 0–53). Neuro-psychological assessment revealed no cognitive deficit and an average intelligence quotient of 93 with no significant verbal-performance discrepancy on the Weschler Adult Intelligence Test. HLA typing was A2, Aw33, B17, B13 and DR2. A plain computerized tomogram of the brain was normal. He had a slightly raised haemoglobin level of 17.1 g/dl, but the renal, liver and thyroid functions were normal.

After a night's adaptation, nocturnal polysomnography was performed using the ambulatory Medilog 9000 system. The actual sleep time was 6 hours with a fragmented sleep architecture, consisting of very little slow-wave sleep: stage 1, 120 minutes (32.8% of actual sleep time); stage 2, 126.5 minutes (34.6%); slow wave sleep, 12 minutes (3.3%); total REM time, 107 minutes (29.3%). The REM latency (the time from onset of sleep to onset

Correspondence: Yun-Kwok Wing, M.R.C.P., M.R.C. Psych.

Accepted: 27 May 1993

of first REM period) was much shortened to 6 minutes. There were eight episodes of REM sleep, six of which occurred just after transient awakenings. The patient also underwent the multiple sleep latency test which is an objective standardized measurement of daytime sleepiness during which he slept in the laboratory at daytime for five scheduled periods of 20 minutes each.⁵ A shortened mean sleep latency (normal >10 minutes) of 2.3 minutes and the presence of sleep onset REM in all five scheduled sleep periods were documented.

Informed consent was obtained for a trial of fluoxetine (20 mg/day) because of its reported usefulness in both narcolepsy⁶ and morbid jealousy.⁷ After 4 days' treatment, the sleep efficiency was unchanged but there was marked reduction of REM sleep and mild increase of stage 1, 2 and slow wave sleep: total sleep time, 419 minutes; stage 1, 165 minutes (39.4%); stage 2, 194 minutes (46.3%); slow wave sleep, 30 minutes (7.2%); REM sleep, 30 minutes (7.2%). The REM latency was significantly lengthened to 275 minutes. When the sleep assessment was repeated 3 months later, both the reduction of REM sleep and lengthening of REM latency persisted but had become less prominent: total sleep time, 463 minutes; stage 1, 189.5 minutes (40.9%); stage 2, 137.5 minutes (29.7%); slow wave sleep, 31.5 minutes (6.8%); REM sleep, 104.5 minutes (22%); REM latency, 92 minutes. The multiple sleep latency test showed a mean sleep latency of 1.8 minutes but there was only one episode of sleep-onset REM occurring at 3.5 minutes. His body weight remained unchanged. The Hamilton depression rating score was 4.

The patient reported improvement in irritability and impulsivity. There was a diminution of his accusatory behaviour towards his wife, who confirmed this. There was no attack of sleep paralysis and cataplexy. The drug was well tolerated and his wife confirmed his medication compliance. Unfortunately, he defaulted follow-up after 6 months of treatment, apparently because of the fear of psychiatric stigma. When he was contacted a year later, his narcoleptic symptoms and morbid jealousy had exacerbated. However, he declined to accept any medical help and the couple was planning a divorce.

Discussion

Both the clinical and polysomnographic features of our patient fulfilled the diagnostic criteria of narcolepsy as proposed by the International Classification of Sleep Disorders.⁸ The clinical triad of recurrent irresistible sleep attacks, cataplexy and sleep paralysis, as well as the polysomnographic features of shortened sleep and REM sleep latency, presence of more than two sleep-onset REM

periods during multiple sleep latency test were characteristic.

The relationship between the sleep disorder and head injury seems complex. Although there is a possibility that the accident could be caused by pre-existing undiagnosed narcolepsy, the absence of any conspicuous symptoms prior to the accident tended to support the contrary. The narcoleptic syndrome is a recognized complication of head injury,⁹ and it is now recommended that post-traumatic hypersomnia should only be diagnosed if the relevant features failed to meet the criteria of specific sleep disorders such as narcolepsy.⁸ Apart from characteristic clinical and polysomnographic features, the presence of HLA DR2 in our patient is in keeping with reports of an almost 100% association with HLA DR2 in narcoleptics of other ethnic backgrounds.¹⁰ This strong association with class II HLA antigen in narcolepsy serves as a useful genetic marker, and it is now believed that genetic factor is a prerequisite even in the setting of head injury in narcoleptic patients.¹⁰

Psychopathology occurs commonly in narcoleptic patients. This is usually considered as a consequence of chronic sleepiness and disturbed nocturnal sleep but a common altered central monoamine disturbance is also postulated.¹⁰ Personality change, depression and obsessive symptoms were said to occur in nearly 50% of narcoleptic patients.^{1,2} These patients frequently encountered marital difficulties and approximately one-fifth of them might end up with separation or divorce.¹ Nevertheless, morbid jealousy has been reported uncommonly and was usually seen as a psychotic condition in previous reports.^{3,4} However, these reports might be biased by the use of stimulant treatments which could themselves induce psychosis. Our patient's preoccupation with his wife's infidelity did not reach delusional degree and might best be described as an obsessive phenomenon,⁷ in which the jealous thoughts resembled ruminations while the checking for evidence of infidelity paralleled compulsion.

The treatment of psychopathology associated with narcolepsy is usually limited to the management of the sleep problem and general support.¹ However, the improvement of temperament and morbid jealousy of our patient with fluoxetine may suggest an additional remedy. The exact mechanism of fluoxetine is unclear but it is possible that it exerts its effect by correcting a central serotonin dysfunction which may underlie obsessionality, impulsivity and affective instability¹¹ and/or through an indirect symptomatic alleviation of the sleep disorder. The effect of fluoxetine on our patient's sleep was complex. The REM sleep suppression occurred immediately after 4 days of treatment, and accounted for the disappearance of sleep paralysis and cataplexy. Fluoxetine also

affected non-REM sleep but to a lesser degree,^{12,13} as indicated by the increase in stage 1 and slow wave sleep. However, the interesting finding was at 3 months when the amount of REM sleep began to increase again. This might suggest that maximal REM suppression occurred at the beginning of treatment. Similar results obtained in animal

studies supported the notion that serotonin modulates in a time-limited manner rather than controls REM sleep permanently.¹³ As narcolepsy and the accompanying psychopathology may be notoriously difficult to treat, the possible positive effect of fluoxetine deserves further study.

References

1. Broughton, R., Ghanem, Q., Hishikawa, Y., Sugita, Y., Nevismalova, S. & Roth, B. Life effects of narcolepsy: relationships to geographical origin (North American, Asian or European) and to other patient and illness variables. *Can J Neurol Sci* 1983, **10**: 100–104.
2. Kales, A., Soldatos, C.R., Bixler, E.O. *et al.* Narcolepsy-cataplexy II. Psychosocial consequences and associated psychopathology. *Arch Neurol* 1982, **39**: 169–171.
3. Pfefferbaum, A. & Berger, P.A. Narcolepsy, paranoid psychosis, and tardive dyskinesia: a pharmacological dilemma. *J Nerv Ment Dis* 1977, **164**: 293–297.
4. Coren, H.Z. & Strain, J.J. A case of narcolepsy with psychosis. *Comp Psychiatry* 1965, **6**: 191–199.
5. Richardson, G.S., Carskadon, M.A., Flagg, W., Hoed, J.V.A., Dement, W.C. & Mitler, M.M. Excessive daytime sleepiness in man: multiple sleep latency measurement in narcoleptic and control subjects. *Electroencephalography Clin Neurophysiol* 1978, **45**: 621–627.
6. Langdon, N., Shindler, J., Parkes, J.D. & Bandak, S. Fluoxetine in the treatment of cataplexy. *Sleep* 1986, **9**: 371–373.
7. Lane, R.D. Successful fluoxetine treatment of pathologic jealousy. *J Clin Psychiatry* 1990, **51**: 345–346.
8. *The International Classification of Sleep Disorders: Diagnostic and Coding Manual*. American Sleep Disorders Association, 1990.
9. Guilleminault, C., Faull, K.F., Miles, L. & Hoed, J.V.D. Posttraumatic excessive daytime sleepiness: a review of 20 patients. *Neurology* 1983, **33**: 1584–1589.
10. Aldrich, M.S., Narcolepsy. *N Engl J Med* 1990, **323**: 389–394.
11. Lopez-Ibor, J.J., Jr. The involvement of serotonin in psychiatric disorders and behaviour. *Br J Psychiatry* 1988, **153** (Suppl 3): 26–39.
12. Nicholson, A.N. & Pascoe, P.A. Studies on the modulation of the sleep–wakefulness continuum in man by fluoxetine, a 5-HT uptake inhibitor. *Neuropharmacology* 1988, **27**: 597–602.
13. Slater, I.H., Jones, G.T. & Moore, R.A. Inhibition of REM sleep by fluoxetine, a specific inhibitor of serotonin uptake. *Neuropharmacology* 1978, **17**: 383–389.