to have caused the 'vanishing duct syndrome'³. But reports implicating glibenclamide as a cause of DICC or what has been aptly termed 'pseudo-primary biliary cirrhosis'³, appear to be rare. Our literature search found one earlier report in the English literature concerning a 66-year-old woman who developed severe cholestatic hepatitis, eight months after having been on glibenclamide 7.5 mg/day. Unlike our patient, she recovered very rapidly within eight weeks of drug withdrawal⁴.

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References

- 1. Kaplan MM. Primary biliary cirrhosis. N Engl J Med 1987;316 : 521-8.
- Sherlock S. The syndrome of disappearing intrahepatic bile ducts. Lancet. 1987;ii : 933-6.
- 3. Zimmerman HJ. Hepatotoxicity. Dis Mon 1993;39 : 715-21.
- Lambert M, Geubel A, Rahier J, Branquinho F. Cholestatic hepatitis associated with glibenclamide therapy. Eur J Gastroenterol Hepatol 1990;2 : 389-91.

Road Traffic Accidents in Patients with Obstructive Sleep Apnoea

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Summary

Three patients involved in road traffic accidents were suspected to have obstructive sleep apnoea (OSA). Two of them fell asleep while riding motorcycles and one patient fell asleep behind the wheel of a truck causing it to overturn. The diagnosis of OSA in each case was suspected based on a history of loud snoring, restless sleep, and excessive daytime somnolence and was confirmed by sleep studies.

Key Words: Road traffic accidents, Obstructive sleep apnoea

Introduction

Excessive daytime sleepiness, one of the main symptoms of obstructive sleep apnoea (OSA) syndrome,¹ can lead to real dozing off especially in monotonous situations, such as driving. Studies have shown that patients with OSA have a high rate of automobile accident^{2,3}. We report three cases to illustrate this fact. Recognising and effectively treating these impaired drivers may prevent potentially serious injury to themselves and other road users.

CASE REPORTS

Case Reports

Case 1

A 40-year-old obese male van driver (weight 94 kg, height 1.62 m, body mass index 35.8 kg m⁻²) was admitted for hypercapnic respiratory failure and right heart failure. He gave a history of loud snoring, excessive daytime sleepiness and a tendency to fall asleep while riding motorcycle or driving. The van he was driving once knocked into a car but there was no human injury. A few months before the present admission, he had sought treatment at the emergency unit of our hospital for minor injuries he sustained when the motorcycle he was riding knocked into a road divider. However, the casualty doctor did not suspect the diagnosis of OSA then.

Overnight polysomnography (PSG) performed this admission documented 50 obstructive apnoeas and hypopnoeas per hour of sleep. His sleep architecture was greatly deranged, with 78% stage 1, 19% stage 2, 2% stage 3, 0% stage 4, and 1% rapid eye movement (REM) sleep. His apnoeas and hypopnoeas and associated oxygen desaturation were markedly reduced while he was using nasal continuous positive airway pressure (CPAP) of 14 cm H_2O . Increased proportions of stage 3, stage 4 and REM sleep were recorded by a repeat PSG study. Domiciliary nasal CPAP therapy at night resulted in improvement in his daytime somnolence and the other symptoms.

Case 2

A 43-year-old man with Pierre Robin sequence, a congenital deformity giving rise to micrognathia, was admitted for assessment of his complains of loud snoring, nocturnal awakenings with a choking sensation, excessive daytime somnolence and chronic fatigue. One month prior to admission, the truck he was driving overturned when he dozed off during a long distance journey. He admitted to have fallen asleep behind the wheel on several occasions when he was caught in traffic jams, at traffic light junctions and during long distance travel. He denied taking alcohol before driving.

Overnight PSG revealed an apnoea-hypopnoea index (number of apnoeas plus hypopnoeas per hour of sleep) of 101. His apnoeas and hypopnoeas, and sleep architecture were markedly improved when he received nasal CPAP treatment. His chronic fatigue, daytime sleepiness and driving ability also improved.

Case 3

A 34-year-old obese man (weight 132 kg, height 1.66 m, body mass index 47.9 kg m⁻²) who worked as a crane driver fell from his motorcycle on his way to work. He was admitted through the emergency room with chest injuries and a left haemothorax. He needed endotracheal intubation and mechanical ventilation for 4 days because of hypercapnic respiratory failure. He denied being under the influence of alcohol or other drugs.

The patient had loud snoring and daytime sleepiness for at least 3 years and he was witnessed by his wife to have apnoeic episodes during sleep. Three weeks after the accident, he underwent an overnight PSG study which revealed 113 obstructive apnoeas and hypopnoeas per hour of sleep associated with marked oxygen desaturation. His sleep architecture was grossly abnormal with predominance of stage 1 and stage 2 sleep. He was prescribed nasal CPAP of 16 cm H_2O to be used nightly after a second PSG study.

Discussion

These three cases illustrate that drivers with untreated sleep apnoea can be involved in road traffic accidents which may result in potentially serious injury to themselves and other road users. Controlled studies in the United States² and Canada³ have shown that patients with OSA are involved in automobile accidents approximately two and one half times more frequently than other licensed drivers. About a quarter of patients with sleep apnoea reported falling asleep at least once per week while driving².

Apnoeas and hypopnoeas result in brief awakenings from sleep caused by the increased inspiratory effort¹. This arousal restores upper airway dilating muscle tone required to reestablish airway patency but it disrupts sleep. Episodes of upper airway narrowing, terminated by arousal, may recur many hundred times in a night. The fragmentation of sleep caused by apnoeas and hypopnoeas leads to deprivation of REM sleep and slow wave (stage 3 and stage 4) sleep and accounts for the excessive daytime sleepiness.

It is important to identify patients with sleep apnoea because effective treatment of the condition is available in the form of nasal CPAP ventilation¹. Drivers who are involved in road traffic accidents should be asked whether they suffer from sleep apnoea. A seriously impaired driver with sleep apnoea is putting himself and others at risk. Once identified, such a patient must be warned about the risks of driving and should be advised to stop driving until they are successfully treated.

References

- 1. Douglas NJ, Polo O. Pathogenesis of obstructive sleep apnoea/ hypopnoea syndrome. Lancet 1994;344 : 653-5.
- Findley L, Unverzadt M, Suratt P. Automobile accidents in patients with obstructive sleep apnoea. Am Rev Respir Dis 1988;138 : 337-40.
- George C, Nickerson P, Hanly P, Millar T, Kryger M. Sleep apnoea patients have more automobile accidents. Lancet 1987; 8556 : 447.

Concordant Childhood Acute Lymphoblastic Leukemia in Monozygotic Twins

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Summary

Two 4-year-old monozygotic Chinese, female twins developed concordant childhood acute lymphoblastic leukemia (ALL) within an interval of about 2 weeks. Based on morphology and cytochemistry findings of the bone marrow blast cells, a diagnosis of ALL, L1 was made. Immunophenotyping showed the blast cells of both twins expressed similar antigens, i.e. HLA-DR, CD10, CD13, CD19, CD22 and CD34.

Identical blood group, same HLA (human leucocyte antigen) genotype, sex and similar appearance suggest that the twins are monozygotic. Since the bone marrow leukemic cells of both twins were identical in morphology and expressed the same antigens with almost similar percentages of positivity, it is likely that the blast cells were derived from the same single clone. Based on the single clone hypothesis, the leukemogenic event must have arisen in utero in one twin and the cells from the abnormal clone then spread to the other twin via shared placental anastomoses.

Key Words: Concordant, Acute lymphoblastic leukemia, Twins, Monozygotic