Case Report

The vegetarian who could not sleep: a case report

Sanjeewa Sumathipala*

Department of Family Medicine, Primary Health Care Corporation, Doha, Qatar

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*Correspondence:
Dr. Sanjeewa Sumathipala,
E-mail: sansumathipala@gmail.com

ABSTRACT

Sleep disorders are not unusual presentations to family medicine clinicians. Similarly, patients may have diets that can render them susceptible to vitamin deficiencies. Although not a commonly considered risk factor for sleep disorders, vitamin B12 is involved in the production of melatonin, a hormone regulating the sleep-wake cycle, and supplementation of vitamin B12 has been used to treat sleep disorders. This case study described a patient who presented with a sleep disorder and who was a vegetarian. Although he lacked obvious manifestations of vitamin B12 deficiency, his serum level was almost half the lower limit of normal, and with vitamin B12 supplementation, his sleep improved.

Keywords: Insomnia, Vitamin B12 deficiency, Sleep-cycle

INTRODUCTION

Insomnia is a subjective sleep problem that occurs despite sufficient opportunities and situations for sleep.¹ The prevalence of insomnia is considered to lie between 10-30%.² Sleep disorders can be related to variety of factors, including the use of caffeine, alcohol and comorbid diseases. The true prevalence of vitamin B12 deficiency is not known, however, one study found that 15% of adults aged more than 65 years had laboratory evidence of vitamin B12 deficiency.³ Vitamin B12 is a water-soluble vitamin and humans are incapable of making its own vitamin B12, relying entirely on dietary sources. Vitamin B12 is derived from meat, fish, and dairy, and a diet lacking those sources can lead to vitamin B12 deficiency.⁴ Hence, vegetarians are at risk of developing a vitamin B12 deficiency. The deficiency can present in a protean manner and include neurological features, hematological changes, and even skin discoloration.⁵ Associations between sleep disorders and levels of micronutrients as well as vitamin B12 have been suggested.⁶,⁷

CASE REPORT

An adult male recently undertook a telephone consultation with the family medicine physician regarding a complaint of insomnia. The patient described an inability to sleep that had been present for two years. The patient had been prescribed clonazepam by a physician and he has been taking this for the two years. He had not been taking another other medication consistently.

He had a past medical history of hypertension and dyslipidemia. There was no significant surgical history. The patient was teetotal and a vegetarian. He has no significant family history, and he works as a driver for a household.

The patient did not describe any symptoms to suggest depression or anxiety as primary causes for his insomnia. He said that he had tried at times to miss one day of his clonazepam, but his insomnia worsened on those corresponding nights.
The clinician requested hematological investigations to include thyroid function tests and a vitamin B12 level. The thyroid function test was normal. However, the vitamin B12 returned at almost half of lower limit of normal of 145 pmol/L. His serum folate was within normal limits and his intrinsic factor was negative.

The patient was invited to come for a face to face consultation and when examined, did not have any features of demyelination. He went on to receive parenteral vitamin B12 which has corrected his vitamin B12 level to normal. He was referred to the psychiatry team for management of his insomnia given the reliance on clonazepam medication. He has also been referred to a dietician to ensure his dietary intake of vitamin B12 is adequate.

The patient is being followed up by the family medicine physician to manage his overall health. The patient has reported an improvement in his sleep pattern; however, he is yet to be fully weaned from his clonazepam. It is hoped that once he is weaned off his clonazepam, the improvement in his sleep will be sustained.

DISCUSSION

In this case, the patient’s main concern was to ensure that he could continue to have medication that had been prescribed to help him sleep. A careful history ascertained that depression or anxiety were neither the precipitating cause or a consequence of his inability to sleep well. Given his dietary history, and the association that can occur with insomnia, his serum vitamin B12 blood test was requested to check that there was no deficiency that required treatment. A serum level of less than 148 pmol/L is considered sensitive enough to diagnose 97% of people with vitamin B12 deficiency. Vitamin B12 levels of less than 75 pmol/L are considered to have clinical or metabolic evidence of vitamin B12 deficiency.

Sleep has been divided into non-rapid eye movement (NREM), consisting of light sleep (stages N1 and N2) and slow-wave sleep (stage N3), and rapid eye movement (REM) sleep occurs periodically in cycles of approximately 90-120 minutes of sleep. Melatonin is a hormone produced by mammalian pineal glands, helping to regulate the sleep-wake cycle. It has been postulated that melatonin acts in the brain to attenuate the wake-promoting signal of the circadian clock to enable sleep.

Studies have made associations between vitamin B12 and sleep disorder, for instance postulating that since vitamin B12 is involved in the synthesis of melatonin, its deficiency could result in sleep disturbance.

Traditionally, there are several risk factors for sleep disorders, although vitamin B12 is not frequently considered amongst the common risk factors for insomnia, in research on animals, intravenously administered vitamin B12 promoted effects on the sleep of rat. In human studies, vitamin B12 administration has resulted in improvement of sleep and a relapse of sleep disturbance when the vitamin B12 was withdrawn. Oral vitamin B12 dose of 1.5 mg once a day or three times a day or was used, however, in these relatively small studies, the baseline vitamin B12 level was not recorded. Although the effects of vitamin B12 on sleep are yet to be fully clarified, vitamin B12 deficiency should be considered as a possible contributory factor for insomnia, especially when other risk factors for this deficiency are present in the history obtained from the patient.

CONCLUSION

The relationship of low vitamin B12 and insomnia is mentioned in various research literature, it is not a commonly cited possible cause for sleep disorders. In this case, the patient’s vegetarian diet was a clue that a low vitamin B12 was a possibility. However, there were no other apparent symptoms or signs to suggest that vitamin B12 was a possibility. As such, if the serum vitamin B12 was not requested in this patient presenting with insomnia, his nutritional deficiency might have deteriorated leading to other serious manifestations of this hypovitaminosis. A holistic approach to insomnia is required, and this is one single case study, there does also need to be focused attention to underlying pathology and the association with certain diets should lead the clinician to consider whether vitamin B12 deficiency is present.

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REFERENCES
