Sleep in Type 2 Diabetes

A Vigg*, Avanti Vigg**, A Vigg**

Abstract

Objectives: To examine the relationship between glycemic control in Type 2 diabetics and sleep duration and quality of sleep.

Methods: There were 220 adult non-insulin dependent diabetics (Type 2) with 69 males and 151 females. The mean age was 59.4 ± 9.2 yrs (Mean ± SD), mean BMI 35.6 ± 9.9 kg/m², and mean HbA₁c 8.2 ± 2.1%. Detailed questions about history and management of diabetes, height, weight and the Pittsburgh Sleep Quality Index (PSQI) were obtained to measure subjective sleep quality. HbA₁c data were obtained to measure glycemic control in the past three months.

Results: The mean amount of self-reported sleep was 6.10 ± 1.66 hours. The mean difference between preferred and actual weekday sleep was 1.83 ± 2.01 hours indicating significant perceived sleep debt. The mean Pittsburgh Sleep Quality Index (PSQI) score was 8.3 and 71% of patients had a score of > 5 which is clinically diagnostic for poor sleep.

Conclusion: A significant proportion of Type 2 diabetics have reduced sleep and there is a definite association between glycemic control and both quality and quantity of sleep.

INTRODUCTION

Sleep disordered breathing (SDB) is an increasingly recognized common chronic condition characterized by sleep fragmentation due to repetitive episodes of upper airway obstruction. Recently two separate well documented studies¹,² both involving large sample sizes and careful assessments of SDB by full polysomnography add to a growing body of evidence supporting the existence of a link between SDB and insulin resistance independent of degree of obesity. Another study by Vgontzas et al³ recently reported that fasting glucose and insulin levels are significantly higher in patients with SDB when compared with weight-matched controls. Furthermore, studies have identified impairment in metabolism as one of the consequences of sleep loss, suggesting that decrements in sleep duration and/or quality may increase the severity of metabolic disorders, particularly diabetes.⁴

Aim of the Study

This study tests the hypothesis that there is a relationship between glycemic control in Type 2 diabetic subjects and sleep duration and quality of sleep.

MATERIAL AND METHODS

Total of 220 adult subjects were studied and there were 69 males and 151 females with Type 2 diabetes treated at the outpatient diabetic clinic. The mean age was 59.4 ± 9.2 years. All these subjects were not recently diagnosed and had blood tests within 90 days of the interview. The survey included questions about history and management of diabetes, height, weight and the Pittsburgh Sleep Quality Index (PSQI) that specifically measures the subjective sleep quality. The PSQI yields a global score that is the sum of seven component scores, each of which addresses a specific aspect of subjective sleep quality. These seven components comprised of sleep quality, latency, duration, sleep efficiency, use of medications, sleep disturbance and daytime dysfunction. It is a self-rating questionnaire for measuring subjects sleep quality and specifically assesses the preceding one-month period. It has good interval consistency and test retest reliability and its validity is supported by polysomnographic results.⁶ The PSQI has been recently validated and subjective sleep quality in healthy elderly men and women has been quantified by Buysse et al. Hemoglobin A₁c (HbA₁c) data were obtained from the patients medical records to reflect glycemic control over the past three months. HbA₁c values less than 7.0% were considered to be in the normal range. It consists of several domains related to sleep quality, sleep efficiency, sleep latency, total duration of sleep, sleep maintenance, use of medications and daytime dysfunction.
dysfunction as shown by previous studies.

**RESULTS**

The mean (± SD) age was 57.4 (± 12.9) years, mean BMI was 35.6 (± 9.9) kg/m² and mean HbA₁c was 8.2 (± 2.1)%. The mean amount of self-reported sleep was 6.10 (± 1.66) hours on the weekdays (Table 1). On weekdays, sleep duration was significantly lower than the conventional average of 7.0 hours (p < 0.001). The mean difference between preferred and actual weekday sleep was 1.83 (± 2.01) hours, which was an indication of perceived sleep debt. The mean PSQI score is 8.3 (± 4.6) and 71% of these patients had a score greater than 5 and this is indicative of poor quality of sleep. There was a significant positive association between HbA₁c and PSQI (r = 0.201; p = 0.003) (Fig. 1) and between HbA₁c and perceived sleep debt (r = 0.202; p = 0.003). Thus, there was an association between worse sleep and perceived sleep debt with poorer glycemic control. There was a trend towards a negative association between HbA₁c and amount of weekday sleep (r = 0.131; p = 0.053). The less reported sleep was also associated with worse glycemic control. Because of the association of pain with diabetes, those who responded that their sleep was disrupted by pain (n = 47) more than twice a week were factored out in a separate analysis. In this subgroup the association between HbA₁c and PSQI remained significant (r = 0.163; p = 0.034) as does the relationship between HbA₁c and perceived sleep debt (r = 0.220; p = 0.004). There was still a trend relating HbA₁c to weekday sleep duration (r = 0.141; p = 0.065). However, this was not statistically significant. In this group, 67% of those patients with PSQI scores greater than 5 had the mean weekday sleep duration of 6.35 (± 1.61) hours, which was also significantly lower than the conventional average of 7.0 hours (p < 0.001).

**DISCUSSION**

Ideally we should have had a control group of non-diabetic subjects in our study to facilitate a direct comparison. However, it was not feasible at the time of doing the study. Our study has clearly shown that type 2 diabetic patients have reduced sleep when compared to general population and this has been further substantiated by recent study by Vgoutzas *et al.* and Mander *et al.* This has a definite bearing on glyemic control. Earlier studies have shown that sleep loss achieved by bedtime curtailment in normal healthy young adults results in marked alterations of glucose metabolism and function. This suggests that sleep loss per se in the absence of breathing abnormalities may promote insulin resistance. The state of ‘sleep debt’ is in itself associated with increased sympathetic nervous activity. Normal sleep duration has decreased from 9 hours in 1910 to an average of 7 hours today and many individuals are in bed for 5-6 hours per night on a chronic basis. Consistent with such short habitual bedtimes, time in bed in the patients who participated in the study by Punjabi and coworkers was between 6.5 and 7 hours. It is likely that both the prevalence and severity of SDB are increased by chronic sleep curtailment. The possibility that the increased incidence of obesity, diabetes and SDB in the community may be partly related to insufficient sleep has been recently recognized and this needs to be further validated.

**CONCLUSIONS**

In this study, a high proportion of Type 2 diabetic subjects were found to have disturbed and reduced sleep relative to the general population. The data support an association between glycemic control and sleep quality and quantity. These findings suggest that sleep hygiene should be a part of routine diabetes management.

**Acknowledgement**

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**REFERENCES**

1. Punjabi NM, Sorkin JD, Katzel L, Goldberg A, Schwartz A, Smith PL. Sleep-disordered breathing and insulin resistance

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**Table 1 : Patient's characteristics**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Age</td>
<td>59.4 ± 9.2 years</td>
</tr>
<tr>
<td>Mean BMI</td>
<td>35.6 ± 9.9 kg/m²</td>
</tr>
<tr>
<td>Mean HbA₁c</td>
<td>8.2 ± 2.1%</td>
</tr>
<tr>
<td>Mean self reported sleep</td>
<td>6.10 ± 1.66 hours</td>
</tr>
<tr>
<td>Mean PSQI</td>
<td>8.3 ± 4.6</td>
</tr>
<tr>
<td>Mean difference between preferred and actual weekday sleep</td>
<td>1.83 ± 2.01 hours</td>
</tr>
</tbody>
</table>

**Fig. 1 : Relationship between PSQI and HbA₁c in total sample.**


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