Endocrine and Metabolic Aspects of Various Sleep Disorders

MUDr. Zuzana Vimmerová Lattová

Abstract:

Recent epidemiological and experimental data suggest a negative influence of shortened or disturbed night sleep on glucose tolerance. However, no comparative studies of glucose metabolism have been conducted in clinical sleep disorders. Dysfunction of the HPA axis may play a causative role in some sleep disorders and in other sleep disorders it may be secondary to the sleep disorder. Moreover, dysfunction of the HPA axis is regarded as a possible causative factor for the impaired glucose sensitivity associated with disturbed sleep. However, data on HPA system activity in sleep disorders are sparse and conflicting.

We studied 25 obstructive sleep apnea (OSA) patients, 18 restless legs syndrome (RLS) patients, 21 patients with primary insomnia and compared them to 33 healthy controls. We performed oral glucose tolerance test and assessed additional parameters of glucose metabolism. The dynamic response of the HPA system was assessed by the DEX-CRH-test which combines suppression (dexamethasone) and stimulation (CRH) of the stress hormone system.

Compared to controls, increased rates of impaired glucose tolerance were found in OSA (OR: 4.9) and RLS (OR: 4.7), but not in primary insomnia. In addition, HbA1c values were significantly increased in the same two patient groups. Significant positive correlations were found between 2-h plasma glucose values and the apnea-arousal-index in OSA ($r = 0.56$; $p,0.05$) and the periodic leg movement-arousal-index in RLS ($r = 0.56$, $p,0.05$). Sleep duration and other quantitative aspects of sleep were similar among patient groups.

After HPA axis suppression the number of non-suppressors did not differ among groups. Following CRH stimulation we did not detect differences in ACTH or cortisol levels and adrenocortical responsivity to ACTH was comparable among groups. These results for the first time document normal HPA system feedback sensitivity in various sleep disorders.