Polysomnographic evaluation of sleep disordered breathing after lung transplantation

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Introduction:

A limited number of studies showed a high prevalence of sleep disordered breathing (SDB) in patients after lung transplantation (LTX) ranging from 24 to 100%. The large variation in prevalence is influenced by a difference in the definition of SDB and the time-frame after transplantation. This study aimed to examine the timing of onset, the pattern and the clinical impact of SDB after lung transplantation.

Methods:

In this prospective study, 15 patients (12 males/3 females, mean age: 52.3 years ± 11.3) underwent a polysomnography before LTX (278 ±174 days), 1 month after LTX (30 ± 12 days) and 1 year after LTX (366 ± 6 days). Presence of SDB was defined as an apnea/hypopnea index (AHI) > 15. If > 50% of the events were central, SDB was classified as central. Furthermore, standard follow-up with lung function and biochemistry was performed after 6 months and 1 year.

Results:

Mean AHI before transplantation was 5.8 with only 2 patients having SDB (both obstructive). One month after LTX, mean AHI was 12.4 (SDB in 2 patients – 1 obstructive, 1 central). One year later, mean AHI was 23.9 with 9/15 patients having SDB (3 obstructive – 6 central). Every patient had an AHI > 5 at one year post-LTX *(Figure 1)*. Patients with SDB had significantly less REM sleep, a significantly higher arousal/awake index, lower minimal saturation and lower diffusion capacity. Body mass index was not significantly different. Patients with central SDB were significantly smaller, had a lower diffusion capacity and showed a significantly higher prevalence of diastolic heart failure in comparison with patients without SDB. In comparison with patients with obstructive SDB, residual volume was also smaller in these patients.

Discussion:

This prospective study shows a high prevalence of SDB in patients one year after LTX. One month after LTX this high prevalence was not yet present. This suggests that SDB after lung transplantation develops rather late and is probably not a consequence of lung denervation or (short-term) use of immunosuppressives. Further research is needed to examine the underlying mechanisms.

Figure 1: AHI in patients before, 1 month and 1 year after LTX.