Histopathologic neuromuscular and soft tissue changes in the uvula and distal soft palate in heavy snoring and obstructive sleep apneic patients; a light, electron microscopic and quantitative image analysis guided studies.

Thesis submitted for partial fulfillment of M.D.degree in otorhinolaryngology.

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Summary

The present work was performed to study the histopathologic changes in the neuromuscular and soft tissues of the uvula and soft palate in patients with heavy snoring and OSA to check for the presence of **muscle**, gland and fat hypertrophy leading to severe mechanical airway obstruction (airway obstruction theory) and the occurrence of **muscle atrophy** secondary to local **efferent nerve degeneration** due to vibratory stretch trauma of heavy snoring (neurogenic theory).

Twenty five adult male patients were included in this study. 10 had heavy snoring without OSA, 10 had severe OSA and 5 nonsnoring nonapneic control subjects

Light microscopic examination with the aid of quantitative image analysis was used in addition to the transmission electron microscopy (TEM).

We have noticed significant increase in the muscle bulk in OSA patients associated with marked hypertrophy in the muscle fibers seen by light microscopy combined with atrophic changes noticed by electron microscopy.

We have found degeneration of the myelinated and unmyelinated nerve fibers, by TEM, in the entire 10 patients with OSA and 6 heavy snoring patients.

The amount of glandular tissue was significantly higher with hyperplasia and hypertrophy in the OSA and the heavy snoring patients. It was significantly related to BMI and RDI too.

OSA patients have significant higher amount of adipose tissue than the heavy snoring patients and the controls. But no significant relation was

observed between the amount of adipose tissue in the soft palate and the BMI and this means that increased BMI does not mean fatty infiltration of the soft palate.

Marked to moderate epithelial hyperplasia or acanthosis were seen in all of our OSA or heavy snoring patients.

The percentage of blood vessels between the patients and control was not significant. Moreover, there was no relation to any other constituents.

The age has no relation to any of the histopathological changes, so the neuromuscular changes were related to age.

All patients had higher BMI which was significantly related to RDI. Our results revealed that the higher the BMI, the worse the apnea index.

By through research in the international literature we could not find any published article on the normal electron microscopic picture of the soft palate and uvula.

From our results we believed that pathogenesis of heavy snoring and OSA passes into 2 stages; in the first stage, mechanical narrowing of the upper airway results from muscle fibers hypertrophy, palatine gland hyperplasia and excessive fatty tissue infiltration in the soft palate. While in the second stage neurogenic degeneration from the severe vibratory trauma of snoring result in muscle fibers atrophy which accelerate the progression of heavy snoring to OSA. Besides, the naturally narrow upper respiratory airway which accelerates the progression of heavy snoring to OSA