SUMMARY

‘The effect of laryngopharyngeal reflux on adenoid hypertrophia.’

Introduction: Adenoid hypertrophy is formed by the growth and expansion of the lymphoid follicules. Besides recurrent viral and bacterial infections, pathologic bacterial colonization, chronical bacterial infections, passive cigarette smoking and allergic episodes, LPR can be important causes of the adenoid hypertrophy. Adenoid tissue which is very small at birth reaches its biggest size between 3 – 7 years of ages. Hypertrophy of the adenoid tissue is one of the most common cause of snoring, oral respiration and hyponasal speech during childhood. The aim of this study is to analyze the effect of LPR on adenoid tissue hypertrophy.

Material and method: Sixty two patients between 2-35 years of ages who are undergone adenoidectomy with routine indications are taken into this study.Patients were questioned for the presence of the pirozis, regurgitation, excessive salivation of the mouth, dysphonia, familial GER, allergic rhinitis, apnea and frequent rhinosinusitis / rinit. The answers were noted as “present” or “absent”. During detailed Ear-Nose-Throat examination some findings such as; serous and purulent discharge in the nasal passage, serous otitis media, dome palate and orthodontic problems have been recorded. Flexible nasofarengolaryngoscopy have been applied to all patients by the doctor who has been carrying out the research. The size of the adenoidal tissue, the relative area covered in nasopharynx, whether there was hyperemia in the larynx and also aritenoid edema including some other laryngeal pathologies were recorded.

The presence of pepsin has been studied by Elisa in the adenoid tissue which is acquired during the operation and homogenized at certain concentration. The pathologic tissue variations due to reflux searched with routine immunohistochemical methods and routine staining techniques.

Results: One of the 62 samples is found positive for the presence of pepsin (%1,6 / 8,564 ng/ml). Significant relation and correlation have been found (aritenoid edema r=0,310;p=0,007, apne r=0,358;p=0,004) between the size of adenoid with the aritenoid edema (p=0,007) and apne (p=0,005). A statistically significant relationship between the size of adenoid with the lymphocyte (p=0,017) and subepithelial edema(p=0,017) have been discovered. Also correlation between the size of adenoid tissue with gland formation (r=0,271;p=0,033) and subepithelial

edema (r=0,291;p=0,022) have been relieved. A statistically significant relationship between aritenoid edema with laryngeal hyperemia (p<0,001), serous discharge and (p=0,036) purulent discharge (p=0,026) have been discovered. In none of the adenoid tissue samples of the 62 patients showed positive results for helicobacter pylori by using immunohistochemical staining. MUC5AC has been found 1+ in 28 (%45,1) patients, 2+ in 13 (%20,9) and 3+ in 2 (%3,3) of the 62 patients. But it has not been found in 19 (%30,7) patients.(43/62, %69,3)

Conclusion: With its mechanism and manifetasions LFR is a different clinical presentation from GER. LPR must be considered in cases of purulent rhinitis / rhinosinusitis and otitis media effusion with no response to the medical treatment. LPR can be a significant factor in the adenoidal tissue hypertrophy. İn children who have adenoidal hypertrophy, treatment of reflux should be considered as an important alternative option before surgical treatment.