Summary

Laryngopharyngeal reflux (LPR) refers to the backflow of stomach contents into the throat that is into the hypo-pharynx. There are numerous synonymous for LPR in the medical literature; reflux laryngitis, laryngeal reflux, gastropharyngeal reflux, pharyngoesophageal reflux, supraesophageal reflux, extraesophageal reflux, atypical reflux. The most accepted of these terms is extraesophageal reflux **(Koufman et al., 2002)**.

LPR is an underlying etiology in 40–60% of patients with various voice disorders. LPR has been reported in up to 10% of patients presenting to an otolaryngologist office and 1% of patients to primary care physicians. Although there are no published figures as to the prevalence of LPR in general population, it is estimated that 50% of all patients presenting with voice or swallowing disorder exhibit LPR. LPR is one of the foremost recognized etiological factors behind the development of various inflammatory disorders of the upper aero-digestive tract **(Printza et al., 2011, Kamani et al., 2012)**.

Laryngopharyngeal reflux and Gastroesophageal reflux disease (GERD) are different disorders:

1. LPR causes irritation and changes in the larynx. GERD is caused by the backflow of gastric contents into the esophagus, which leads to tissue damage or esophagitis and heartburn.
2. LPR is not commonly postprandial.
3. Patients with LPR are predominantly upright (daytime) refluxers
4. There are no prolonged periods of acid exposure, no dysmotility and

no prolonged esophageal acid clearance in LPR.

5. Although this level of esophageal reflux does not cause heart burn and esophagitis, the more fragile laryngeal epithelium may still be injured. The larynx and pharynx are also devoid of the acid clearance mechanism found in the esophagus and thus is far more liable to peptic injury.
6. It is believed that the primary defect in GERD is lower esophageal dysfunction whereas the primary defect in LPR is upper esophageal sphincter dysfunction, **(Vardar et al., 2012)**.

Patient with LPR present with nonspecific symptoms like globus sensation, vocal fatigue, hoarseness, chronic throat clearing, dysphagia, chronic cough. They do not usually have symptoms of gastroesophageal reflux **(Ptok & Ptok, 2012)**.

Laryngoscopic findings are also nonspecific. The most common laryngoscopic finding is reflux laryngitis. The most frequently observed LPR related findings are interarytenoid erythema or hyperemia, infraglottic edema (pseudo sulcus), ventricular obliteration, posterior commissure hypertrophy and/or pachyderma, granuloma or granulation tissue formation, and thick excessive endolaryngeal mucus. A normal laryngeal examination cannot rule out the presence of LPR, however, observation of these findings along with the presence of suggestive laryngeal symptoms gives important information for the diagnosis **(Ozmen et al., 2012; Ptok & Ptok, 2012)**..

Reflux finding score (RFS) is an 8 item clinical severity rating scale based on fibroptic findings. It ranges from 0 to 26 (worst score). The scale includes most common laryngeal findings related to LPR and any individual with RFS greater than 7 has more than 95% probability of

having LPR. RFS accurately document treatment efficacy in patients with LPR **(Belafsky et al., 2002)**.

Reflux symptom index (RSI) is a 9 item self administered outcome instrument. It accurately documents symptoms of patients with LPR. This index appears to be valid and is highly reproducible. An RSI of more than 13 is considered to indicate LPR. It ranges from 0 to 45 (worst possible score) **(Mesallam & Stemple, 2007)**.

Response to empiric treatment with PPI (the 'Omeprazole test') is a more common and acceptable initial diagnostic strategy for uncomplicated LPR. Dual probe pH monitoring remains the most specific and sensitive test available to diagnose LPR. The establishment of diagnosis by pH monitoring for LPR is reserved by American Gastroenterological Association for patients who do not respond to initial acid suppression and the use of pH monitoring as initial diagnostic study is also recommended in patients with more severe conditions possibly related to LPR such as sub-glottic stenosis and severe laryngospasm **(Friedman et al., 2012)**.

Standard therapeutic intervention for LPR includes life style modifications, medical and surgical treatment. Proton pump inhibitors have become the treatment of choice even though conflicting results exists in their response **(Sen & Georgalas, 2005)**.

Because of the lack of laryngopharyngeal protective mechanism, treatment requires acid suppression to be complete, aggressive and prolonged therapy than GERD and treatment failure is not uncommon **(Coca-Pelaz et al., 2012)**.

Nissen fundoplication is an effective treatment for most LPR symptoms, although patients with adult-onset asthma and repetitive throat clearing appear to benefit least from surgical intervention **(van der Westhuizen et al., 2011)**.

Transoral incisionless fundoplication (TIF) improved surgical outcome of treatment of LPR. Heartburn was eliminated in 65% and improved by >50% in 86% of patients. Regurgitation was eliminated in 80% of patients. Atypical LPR symptoms such as hoarseness, coughing, and throat clearing were eliminated in 63% of patients as measured by Reflux Symptom Index scores **(Trad et al., 2012)**.