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شبكة المعلومـــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـسـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون آية تغيرات

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To be kept away from dust in dry cool place of
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بالرسالة صفحات

لم ترد بالأصـــل

Effects of Gastroesophageal Reflux Disease on the Larynx

Thesis
Submitted for partial fulfillment
Of MD degree in Phoniatrics

By

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الدراسات العليا

محضر

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الطبيب / علاء الدين محمود حسن مصطفي

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في امراض التخاطب

تحت عنوان : باللغة الانجليزية: Effects of gastroesphageal reflux disease on the larynx.

اللغة العربية: تأثير الارتجـاع المعدي المريئي علي الحنجرة

بناء على موافقة الجامعة بتاريخ٧/ ٥/ ٢٠٠٥ تم تشكيل لجنة الفحص والمناقشة للرسالة المذكورة أعلاه على النحو التالى :

١-١-٠١د/ طارق محمد كامل عن المشرفين

٢-١-٠١د/ صادق وفيق زاهر ممتحن داخلى

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بعد فحص الرسالة بواسطة كل عضو منفردا وكتابة تقارير منفردة لكل منهم انعقدت اللجنة مجتمعة في يوم الخميس بتاريخ ١٢/٥/٢٠٠٥ الساعة ٩ صباحا مدرج العيادة الخارجية بكلية الطب – جامعة القاهرة وذلك لمناقشة الطالب في جلسة علنية في موضوع الرسالة والنتائج التى توصل اليها وكذلك الاسس العلمية التى قام عليها البحث .

قرار اللجنة : قبول الرسالة

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توقيعات أعضاء الجنة :-

المشرف الممتحن الممتحن الداخلى الممتحن الخارجى

١٠د/ طارق محمد كامل ١٠د/ صادق وفيق زاهر ١٠د/ عليه محمود الشوبري

facilitate proper evaluation and treatment and to arrest progression to complications *(Toohill, et al, 1989)*.

In patients with Gastro Esophageal Reflux Disease (GERD) gastric distention induced experimentally increases the number of transit L.E.S. relaxations, which are the dominant cause of reflux episodes. As the number of transient relaxation's increase the frequency of reflux episodes increase *(Fennerty, and Sampliner, 1993)*. Physiologic GERD, indeed, the occurrence of as many as 50 GER episodes a day, usually after meal, accepted as being within normal range *(Koufman, 1991)*. Patients with pathological reflux often experience many episodes of short duration reflux and /or several prolonged episodes where the acid may stay in the esophagus for up to several hours *(Orlando, 1986)* GERD is a clinical term that refers to GER that is excessive and that causes symptoms & tissue damage (usually heartburn, and oesophagitis) (Koufman, 1991). GERD is associated with laryngopharyngeal conditions, which range from mild symptoms to life threatening ones. It is a one of the most common problems encountered by physicians in many specialties (Koufman, 1988). The clinical manifestations of GERD include conditions that are occult chronic –intermittent, and life threatening. Until recently, it was mistakenly believed that patients who denied heartburn could not have GERD. It is now recognized that GERD may produce at least two different consultations of symptoms and squeals

- in gastroentrology patients with esophagitis, and
- in otolaryngology patients with throat complaints, many of who do not have heartburn or esophagitis *(koufman, 1998)*.

The most common symptom reported by patients is a "lump in the throat" (globus sensation). Tauber, et al, 2002 founded that throat clearing is the most frequent symptom, but *Koufman, 1991, and Hill, et al, 1997* have shown that in 23 to 60

percent of patients presenting with globus sensation, GERD is the etiologic factor.

Other symptoms include constant throat clearing (caused by increased secretions and irritation of the laryngeal mucosa); dysphonia (caused by edema or inflammatory lesions of the true vocal folds); chronic sore throat (often misdiagnosed as recurrent or chronic tonsillitis); coughing; cervical dysphagia (caused by dysfunction of the upper esophageal sphincter); halitosis; buccal burning; otalgia (explained by the common sensory innervations of the esophagus and external auditory canal by the 10th cranial nerve); food sticking in the throat; pharyngeal tightness; a choking sensation; aerophagia; and water brash (hyper salivation). Laryngopharyngeal reflux should be suspected in patients who present with any of these symptoms.

(Ahuja , et al, 1997).

Gastro esophageal reflux (GER) has been implicated in the Pathogenesis of several otolaryngologic disorders, such as chronic/posterior laryngitis, laryngeal contact ulcer *(Cherry, et al, 1970) (Koufman , 1998),* or granuloma *(Goldberg, et al, 1978, Miko, 1989, Oslon, 1991, and Toohill, et al, 1997),* paroxysmal laryngospasm, *(Loughlin, et al, 1996, and Toohill, et al, 1989)* vocal fold nodules *(Kuhn, et al, 1998, Toohill, et al, 1997,and Ulualp, et al, 1998)* Reinke's edema, *(Toohill, et al, 1997)* subglottic stenosis, *(Jindal, et al, 1994, and Little, et al, 1985),* laryngotracheal stenosis, *(Bain, et al, 1983, Koufman, 1991,and Toohill, et al, 1998),* globus pharyngeus *(Hallewell and Cole ,1970, Hill ,et al, 1997 , Koufman , 1991 , and Toohill, et al , 1989)* laryngeal *(Koufman , 1991 , Morrison ,1988, and Ward and Hanson, 1988)* and hypopharyngeal carcinoma *(Price , et al ,1990)* , chronic sinusitis, (*Barbero, 1996, DiBasic et al ,1998, and Ulualp,et al, 1998-1999*) and sudden infant death syndrome *(Leape ,et al,1977).* Despite discoveries that have yielded a better understanding of Laryngopharyngeal Reflux (LPR) and how it

differs from GERD, much is still not known. LPR remains controversial, partly because the gastroenterology model of reflux disease (i.e. GERD) does not seem to apply to patients with LPR. The term laryngopharyngeal reflux itself was coined because otolaryngologists wanted a new diagnostic term to designate reflux in otolaryngology patients. The clinical dichotomy of reflux patients who are seen by gastroenterologists and those who are seen by otolaryngologists warrants the use of two different diagnostic designations. Several other terms have been used for LPR in the medical literature (e.g. Atypical reflux, Extra esophageal reflux, Gastro pharyngeal reflux, Laryngeal reflux, Pharyngoesophageal reflux, Reflux laryngitis, and Supraesophageal reflux).

(Koufman, 2000).

Symptoms, findings, and mechanisms of reflux on (ORL patients and GI patients) are different. ORL patients do not have esophagitis and its principal symptoms, heartburn .Traditional reflux tests are often falsely negative in patients with LPR. LPR patients' generally are upright, daytime refluxers, whereas esophagitis patients are usually supine nocturnal refluxers, *(Koufman, 1991, Wiener, et al, 1989-1986).*LPR patients have relatively good esophageal motor function, while esophagitis patients do not, *(Koufman, 1991, 1988, Wiener, et al, 1989).* function of the upper esophageal sphincter tends to be poorer in LPR patients, *(Koufman, 1991, 1988, Wiener, et al, 1989).* Thus, LPR appears to have a different pathophysiology, and hence different symptoms, findings, and manifestations. *(Koufman, 1996).*Because of its varied manifestations, patient referrals occur to the cardiologist, gastroenterologist, pulmonary specialist, general surgeon, and to the otolaryngologist. So confusion because each specialist may relate the symptoms only to his or her area of expertise. *(Toohill, et al, 1997).*

Aim of the Work

The aim of this study is to find the relationship between the Gastro esophageal reflux disease GERD and different laryngeal disorders in order to reach better understanding and diagnosis of such disorder as a preliminary step for proper management.

Historical perspective

GASTROESOPHAGEAL REFLUX DISEASE (GERD) originally identified in 1968 by Cherry and Margulies as an etiologic factor in laryngeal disease. Yet, long before this landmark report, unrecognized Virchow who in 1880 *(cited from Koufman , 1998)* coined the term "pachydermia verrucosa larynges" detected laryngeal manifestations of reflux disease to describe the annular epithelial overgrowth centered on the posterior glottis. Although Virchow believed it to be related to gross vocal abuse, it would later be recognized as pathognomonic for laryngeal reflux *(Shaw and Seari, 1997).*

In 1980, Jackson introduced the esophagoscope, and the modern era of esophagology began. In 1989, using a crude manometric system. *Meltzer, 1899 (cited from Koufman, 1998)* described esophageal peristalsis. Although *Tilesion, 1906, Mosher, 1921, and Jackson, (cited from Koufman , 1998)* described the anatomic, endoscopic, and pathologic findings of stricture and peptic ulcer of the esophagus, they did not consider the cause to be related to reflux of the gastric contents. *Mosher, 1921(cited from Koufman , 1998)* attributed esophageal stricture formation to gall bladder disease, stomach causes, or foreign body *(Shaw, et al, 1997).*

In a landmark study, *Winkelstein, 1935, (cited from Koufman , 1998)* reviewed the prevailing beliefs that the causes of esophagitis were irritative (mechanical, thermal, and chemical irritants, including alcohol and tobacco); specific (syphilis, tuberculosis, and actinomycosis): and secondary to cardio spasm, diverticula's or neoplasm. He described five patients with heartburn, dysphagia, esophagitis, stricture, and esophageal spasm and stated, "the type of substernal pain, heartburn, sourer regurgitations and the hyper chlorhydria in all recall the clinical features of peptic ulcer of the esophagus." Three patients

had a history of a preexisting peptic ulcer. All patients responded to anti reflux management, although four had periodic relapses, and one required a gastrectomy. In addition to reviewing the symptom complex in these patients, Winkelsiein reported the endoscopic and radiographic findings *(Delahunty and Cherry , 1968)*

Throughout the 1940s and 1950s, hiatal hernia equated with GERD and surgical correction of symptomatic sliding hiatal hernia became the focus of management *(Urschel and Paulson, 1967).* The belief that the sphincteric function of the lower esophageal sphincter (LES) was created by mechanical factors was supported by the failure of anatomists and operating surgeons to find a separate muscle at the Gastroesphageal junction *(Urschel and Paulson et al, 1967)* .

In *1950, Berenberg and Newhauser* provided the most accurate and comprehensive description of GERD. LES (Lower Esophageal Sphincter) dysfunction (low resting pressure) was believed to be the sole factor responsible for reflux. However, knowledge about the physiology of esophagogastric function and the mechanisms of GERD was still limited *(Jamieson and Duranceau, 1988).*

With the introduction of the flexible fiberoptic esophagoscope in 1962 some aspects of diagnostic esophagology moved out of the operating room and into the gastroenterologists's laboratory. However, despite advances in diagnosis, GERD research remained almost exclusively focused on esophagitis *(Koufman, 2000).*Bernstein, et al, 1958 introduced the acid perfusion test, which proved to be more sensitive than contrast radiography for diagnosing reflux esophagitis. These diagnostic tests and others were for the consequences and complications of esophagitis *(Jamieson, et al, 1988).*

In 1967, prolonged pH monitoring was introduced which is highly sensitive and specific for the reflux event itself .In 1986, double-probe (simultaneous esophageal and pharyngeal) PH monitoring was introduced by Wiener, et al, 1986. This test has resulted in new insights in pathophysiology of GERD. Perhaps because of esophageal manometry and fiberoptic esophagoscopy, the literature on GERD since the 1960s has been primarily within the specialty of Gastroentrology *(Wiener, et al, 1986).*

Today, GERD is the subject of considerable interest in otolaryngology *(Koufman, 2000)* . Laryngopharyngeal reflux (LPR) went unrecognized as a clinical entity until 1968, when the first reports linking LPR with the development of vocal process granulomas appeared in the otolaryngology literature. *(Cherry, et al, 1968, and Delahunty, et al, 1968)* Since that time, LPR has been implicated in the etiology of a host of laryngeal conditions, including muscle tension ("functional") dysphonia *(Morrison, et al, 1988, and koufman, 2000),* subglottic stenosis, *(Bain, et al, 1983 and Jindal, et al, 1994,* laryngospasm, *(Chodosh, 1977 , and Bortolotti, 1989),* pachydermia, *(Koufman, 1991, and Delahunty, 1972),* leukoplakia, (Koufman, 1991), and vocal fold carcinoma, *(Koufman, 1991, Glanz and Kleinsasser, 1976, and Oson, 1983).*

In the 1980s, the first reports of otolaryngologic patients with hoarseness and other throat complaints studied using ambulatory 24-hour pH monitoring began to appear, *(Wiener, et al, 1986, and Koufman, et al, 1988),* and in*1987, Wiener et al,* reported the use of simultaneous monitoring of the pH in the pharynx and in the distal esophagus, so-called "double-probe" pH monitoring. By placing a pH probe in the hypopharynx behind the laryngeal inlet. This diagnostic technique used to document the presence of extraesophageal reflux, i.e., true laryngopharyngeal reflux.