Reciprocal Causal Relationship between Laryngopharyngeal Reflux and Eustachian Tube Obstruction

Abstract

My own experience in the medical treatment of a lot of patients for 20 years has proved that many cases have both Laryngopharyngeal reflux (LPR) and Eustachian tube obstruction (ETO) at the same time. In these cases, ETO can be a cause of LPR, or, conversely, LPR can be a cause of ETO, and hence it is natural that a concept of a ‘reciprocal causal relationship between LPR and ETO’ emerges from it. A combination like ‘hearing loss’ or/and ‘ear fullness’ or/and ‘dizziness (vertigo)’ or/and ‘tinnitus’ or/and ‘headache (migraine)’ due to ETO, is regarded as consisting of major symptoms originating from ETO. In addition to nausea, vomiting and perspiration as the common symptoms accompanied by vertigo, any other multiple complaints from LPR or Gastro esophageal reflux disease (GERD) also may be clinical manifestations originating from ETO. Reversely, the fact that LPR can be a cause of ETO also has been proved by recent researches. In conclusion, treating patients regarding in an emergency room or outpatient department, a wide and diverse variety of symptoms and diseases originating from both LPR or GERD, and ETO, has to be considered with the mutual connection observantly. And they should be subjected to the therapeutic test of inflation of the tubes as a first step in a thorough clinical investigation. Ideally normal middle ear cavity pressure with perfectly equal balance between both ears is the core prerequisite before diagnosis and treatment for any symptoms and diseases.

Keywords: Eustachian tube catheterization; Eustachian tube obstruction; Laryngopharyngeal reflux; Gastro esophageal reflux; Vertigo; Nausea; Vomiting; Hearing loss; Ear fullness; Tinnitus; Headache

Abbreviations: ETO: Eustachian Tube Obstruction; LPR: Laryngopharyngeal Reflux; GERD: Gastro Esophageal Reflux Disease; VOR: Vestibulo Ocular Reflex; MEEs: Middle Ear Effusions

Introduction

My own experience in the medical treatment of a lot of patients for 20 years has proved that many cases have both Laryngopharyngeal reflux (LPR) and Eustachian tube obstruction (ETO) at the same time. I have realized keenly the necessity of definitizing the cause-and-effect relationship between both of them, and it should be universally applicable to management of many cases of this type. Needless to say, it can be a different matter without any connection between both at all in some patients; hence they should be studied on a case by case.

Blocked Eustachian tubes can cause several symptoms, including ears that hurt and feel full, ringing or popping noises, hearing problems, feeling a little dizzy [1]. These 5 symptoms can be regarded as major in multiple symptoms originated from ETO. Mechanical obstruction of the Eustachian tube may be either intrinsic or extrinsic. Intrinsic mechanical obstruction is usually caused by inflammation of the mucous membrane lining of the Eustachian tube or an allergic diathesis causing edema of the tubal mucosa [2]. Extrinsic mechanical obstruction is caused by obstructing masses such as hypertrophic adenoid tissue or nasopharyngeal tumors [2].

Though ETO as one of the principal causes of vertigo was already recognized by many respected senior doctors a long time ago, it still has received but scant attention both in the literature and in practice for a long time. Many patients of this type suffer unnecessarily the distressing symptoms of vertigo, nausea, vomiting, and perspiration sometimes for long periods, when their physicians fail to recognize the cause in the emergency room or outpatient department, and have no skill to institute the simple procedure of mechanical inflation of the Eustachian tubes which would bring them relief. What’s even worse, many special clinical tests of vestibular function are performed even in that case such as vertigo due to ETO. It is just an infinite error.

While vertigo originating from ETO is a distinctive clinical entity, the reason these cases are so consistently overlooked probably is that they are examined usually by the physician, audiologist, otolaryngologist or otologist, who is likely to think in terms of disturbances in the digestive, circulatory, nervous systems or only vestibular organ function and hence to ignore the possibility that violent symptoms of dizziness, nausea, vomiting and perspiration may be attributable to ETO. In addition to these above-mentioned major symptoms, the collateral symptoms accompanied by them, e.g., symptoms of LPR or GERD such as nausea, vomiting and perspiration as the common symptoms accompanied by vertigo should be observed closely. I believe that more variable symptoms related to both of LPR or GERD, e.g., back pain and numbness of fingers or toes, than many symptoms of ETO as one of the principal causes of vertigo that a concept of a ‘reciprocal causal relationship between LPR and ETO’ emerges from it.
In these cases, some process should be performed to rule out possible mechanical causes for such as middle ear effusion or/and ETO before all. Moreover, ideally normal middle ear cavity pressure with balance between both ears is the essential prerequisite to be checked before any other tests for vestibular function, tinnitus, ear fullness, sensorineural hearing loss, headache, earaches, itching sensation of ear, migrainous vertigo, etc. What is first, necessary and sufficient for it? Eustachian tube catheterization (so-called ‘Rosenmuller method’) [3]. It is true, of course, that there are many other conditions which may cause vertigo, but since obstruction of the Eustachian tube is one of the most obvious, and also the most easily corrected, every patient with symptoms of dizziness and nausea should be subjected to the therapeutic test of inflation of the tubes as a first step in a thorough clinical investigation [4].

Dizziness or vertigo from ETO can be explained by increase of inner pressure of vestibular organ. It is because negative middle ear pressure gives rise to the retraction of tympanic membrane, and it makes the stapes push against the oval window [3]. It seems obvious that obstruction of the Eustachian tube somehow disturbs the air pressure and causes stimulation of the perilymph, which interferes with normal balance as maintained by the labyrinthine mechanism [4]. In this light, we cannot help accepting the clear proposition that ETO is the obvious cause for ‘loss of vestibular function’, and gives rise to ‘peripheral vestibular loss’ at the end. Recognition of the head’s movement relative to the body is provided by the linear (otolithic macula) and angular (semicircular canals) acceleration receptors of the inner ear. Electrical activity generated within the inner ear travels along the vestibular nerve (primary afferent neuronal pathway) to the central vestibular nuclei of the brainstem, forming second-order neuronal pathways that become the vestibulo-ocular reflex (VOR), the vestibule spinal tracts, and the vestibule cerebellar tracts. Pathways derived from vestibular information also travel to the brainstem emetic centers, which serves to explain vegetative symptoms such as nausea, vomiting, and perspiration that a patient typically experiences following an acute unilateral vestibular loss (Figure 1) [3]. So to speak, ETO which somehow disturbs the air pressure and causes stimulation of the perilymph, interferes with normal balance as maintained by the labyrinthine mechanism. And such electrical activity generated within the inner ear, travels to the brainstem emetic centers, which serves to explain vegetative symptoms such as nausea, vomiting, and perspiration that a patient typically experiences following an acute unilateral vestibular loss. Specifically, in considering the principal causes of this vestibular loss, ETO should never be excluded from them.

Figure 1: Schematic representation of the vestibular system and its pathways.
Unilateral Peripheral Vestibular Loss With a loss of unilateral vestibular function the patient acutely experiences the sensation of true vertigo from interruptions of VOR pathways and tends to lie perfectly still, as any movement aggravates vegetative symptoms such as nausea and vomiting that arise from the emetic centers. Nystagmus beating away from the side of the lesion is the cardinal physical sign that obeys Alexander's law (the quick phase of the nystagmus induced by the imbalance in activity at the level of the vestibular nuclei is greatest in amplitude and frequency when the eyes are turned away from the side of the lesion) [5]. Interruption in vestibospinal tract pathways causes the patient to fall or list toward the affected side. Findings of ipsilateral hemispheric cerebellar dysfunction presenting with behaviors such as past-pointing, an inability to perform rapid alternating movements (dysdiadochokinesis), and gait ataxia reflect acute vestibulo-cerebellar tract involvement [6]. The vertigo is caused in most instances and perhaps in all, by unilateral Eustachian obstruction or by more complete obstruction on one side than the other. The direction of the gait furnishes a clue to the side which may be affected, for in most of the cases the staggering is in the direction of the obstructed side [4]. If more easily explained, unilateral ETO cause a loss of unilateral vestibular function, and the loss of unilateral vestibular function cause Unilateral Peripheral Vestibular Loss Vertigo with nausea, vomiting, and nystagmus.

With compensation (implying the existence of a normal functioning CNS and contra lateral peripheral vestibular system) there may be minimal symptomatology that is only brought out by very rapid head movements. The spontaneous nystagmus disappears, vegetative symptoms resolve, gait improves, and in the case of a chronic condition the patient may experience only a slight imbalance when turning quickly [6]. This point is the reason why any vestibular function test should never be performed before correction of ETO and without normalization of middle ear pressure. Every clinical tests being used in the assessment of vestibular function should be performed in the state of 'ideally normal middle ear cavity pressure with perfectly equal balance between both ears'.

Bilateral Peripheral Vestibular Loss Vertigo is not a feature of a bilateral vestibular loss even when it occurs in an acute fashion. Injury to the end organs as might occur in systemic amino glycoside vestibulo toxicity causes a bilateral loss of function that tends to be electrically symmetric at the level of the vestibular nuclei in the brainstem. Instead the patient tends to complain of oscillopsia (visual blurring with head movement) and imbalance. The gait is typically broad-based and ataxic, especially with eyes closed. Falls are not infrequent and in many instances the patient requires assistive devices for ambulation or is relegated to a wheelchair. Compensation is generally unlikely to occur despite the best efforts of vestibular rehabilitation therapy and a greater reliance on information from visual and proprioceptive receptors [6]. That is to say, bilateral ETO cause a bilateral loss of vestibular function, but does not cause Bilateral Peripheral Vestibular Loss Vertigo. We can realize that bilateral ETO cause oscillopsia and imbalance, but no vertigo.

ETO can already cause a loss of a vestibular function. Unilateral ETO cause unilateral peripheral vestibular loss vertigo including vegetative symptoms such as nausea, vomiting, perspiration, and nystagmus, but bilateral ETO cause such complaints of oscillopsia and imbalance. At right now, it is reasonable to get a question, what is the normal range of middle ear cavity pressure that never makes any impact on the vestibular function? Therefore, as I mentioned above, it is established that every clinical tests being used in the assessment of vestibular function should be performed in the state of 'ideally normal middle ear cavity pressure with perfectly equal balance between both ears'. Kim HY mentions; Ideally normal middle ear cavity pressure with balance between both ears is the essential prerequisite to be checked before any other tests for vestibular function, tinnitus, ear fullness, sensorineural hearing loss, headache, earaches, itching sensation of ear, migrainous vertigo, etc. [3].

Discussion

Laryngopharyngeal Reflux due to Eustachian Tube Obstruction

The cases of insidious onset are the ones most likely to be overlooked. In these instances, because the gastrointestinal symptoms are predominant, the patients are likely to be subjected to various types of treatment over long periods for diseases of the digestive system. When the onset of the symptoms of nausea and vertigo is sudden and severe, the diagnosis is more easily established, and, if the proper treatment is instituted promptly, the relief is striking and dramatic [4]. The gastrointestinal symptoms like nausea and vomiting related to vertigo can be also regarded as symptoms of LPR or GERD. And we can accept an intuition that every variable symptom and disease associated LPR or GERD should be included in the multiple symptoms and diseases originated from ETO.

Eustachian Tube Obstruction due to Laryngopharyngeal Reflux

In the patients having LPR and ETO, ETO is not just a cause of LPR as seen above. On the contrary to this, LPR can be a cause of ETO. Because reflux material from stomach can reach the nasopharynx and the Eustachian tubes, and block the tubes directly or cause inflammation and adhesion and collapse of them (Table 1) [7].

Al-Saab et al. [8] present the study group of patients with middle ear effusions (MEEs) and adenoidal tissue biopsies were obtained from patients undergoing simultaneous tympanostomy tube placement and adenoidecomy. In the control group, adenoid specimens were taken during adenoidecomy (+/- tonsillectomy) from children with no history of OME. The adenoid tissues were analyzed immunohistochemically to confirm the presence of pepsinogen. Normal gastric tissue was used as a positive control and lymphatic tissue as a negative control. Total pepsinogen levels of MEE were measured with enzyme-linked immunosorbent assay. Adenoid tissue of the OME group (n = 25) demonstrated significantly higher pepsinogen immuno reactivity when compared with the adenoid tissue of the control group (n = 29), specifically in staining of both the epithelia (p < .0001) and the subepithelia, (p < .001). The presence of pepsinogen was detected in 84% of MEEs from the OME group, at concentrations 1.86 to 12.5 times higher than that of serum. Pepsinogen messenger...
ribonucleic acid was not demonstrated in any of the adenoid tissues of the OME group. Conclusively, LPR plays an important role in the pathogenesis of OME as gastric reflux reaches the middle ear through the nasopharynx and Eustachian tube to cause OME [8].

Table 1: Symptoms and clinical manifestations reported to be related to laryngopharyngeal reflux

| Symptoms                  | Conditions                                      |
|---------------------------|-------------------------------------------------|
| Chronic dysphonia         | Reflux laryngitis                               |
| Intermittent dysphonia    | Subglottic stenosis                             |
| Vocal fatigue             | Carcinoma of the larynx                         |
| Voice breaks              | Endotracheal intubation injury                   |
| Chronic throat clearing   | Contact ulcers and granulomas                    |
| Excessive throat mucus “Postnasal drip” | Posterior glottic stenosis                      |
| Chronic cough             | Arytenoid fixation                              |
| Dysphagia                 | Paroxysmal laryngospasm                         |
| Globus                    | Paradoxical vocal foldmovement                   |
| Intermittent airway obstruction | Globus pharyngeus              |
| Chronic airway obstruction| Vocal nodules                                   |
| Wheezing                  | Polypoid degeneration                           |
|                           | Laryngomalacia                                  |
|                           | Pachydermalaryngis                              |
|                           | Recurrent leukoplakia                            |
|                           | Sudden infant death syndrome                     |
|                           | Sinusitis                                        |
|                           | Otitis media                                    |
|                           | Sleep apnea                                     |
|                           | Exacerbation of asthma                          |

Conclusion

My own experience in the medical treatment of a lot of patients for 20 years has proved that many cases have both LPR and ETO at the same time, and has let me make a concept about a reciprocal causal relationship between LPR and ETO. ETO can be a cause of Laryngopharyngeal reflux, or, conversely, LPR can be a cause of Eustachian tube obstruction.

I have mentioned a combination of 5 symptoms like ‘hearing loss’ or/and ‘ear fullness’ or/and ‘dizziness (vertigo)’ or/and ‘tinnitus’ or/and ‘headache (migraine)’ due to ETO. In addition to these above-mentioned major symptoms, the collateral symptoms accompanied by them, e.g., symptoms of LPR or GERD like nausea, vomiting and perspiration as the common symptoms accompanied by vertigo should be observed closely. I believe that more variable symptoms related to both of LPR or GERD, e.g., back pain and numbness of fingers or toes, than several symptoms gaining academic recognition at present, need to be proved definitely. On the grounds that LPR or GERD can be the direct result of the ETO, I strongly urge that ETO should be included as a direct cause of LPR or GERD academically.

Any vestibular function test should never be performed before correction of ETO and without normalization of middle ear pressure; because ETO can already cause a loss of a vestibular function. And the loss of vestibular function can cause a peripheral vestibular loss. Let me tell you, the results of vestibular function test without checking the middle ear pressure are just errors. If the abnormality of middle ear pressure was found, it should be corrected normally first. By extension, symptoms due to ETO can be subsided after it. Anyway, the objective finding of Eustachian tube obstruction should be corrected, regardless of whether there any symptoms? This point makes us make a mental note of the necessity of Eustachian tube catheterization for diagnosis and treatment of LPR or GERD and ETO.

Conclusively, treating patients regardless in emergency room or outpatient department, a wide and diverse variety of symptoms originating from both LPR or GERD and ETO, has to be considered with the mutual connection observantly. Because Eustachian tube catheterization may be of both diagnostic and therapeutic value, they should be subjected to the therapeutic test of inflation of the tubes as a first step in a thorough clinical investigation. Ideally normal middle ear cavity pressure with perfectly equal balance between both ears, is the core prerequisite before diagnosis and treatment for any symptom and disease. This is my thesis for my dear people including physicians and patients, even though it looks as though I might have to point out the obvious.

At the end, I introduce an idiom originated in an ancient event. ‘Review the old, learn the new.’

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