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Reflux symptoms may develop in cases of throat mucosa injury, stress and related-autonomic nerve dysfunction

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Dear Editor,

We read the paper of Elton et al. entitled "Observations of increased gastroesophageal reflux symptomology in an anhydrous ammonia exposed population" [1]. The authors observed laryngopharyngeal reflux (LPR) symptoms in 15 patients exposed to anhydrous ammonia from a single chemical spill. Based on ABEA survey LPR symptom and sign outcomes, they reported that 93.3 % and 66 % of individuals experienced at least 3 cardinal symptoms or at least one sign of LPR, respectively. They concluded that there is an association between anhydrous ammonia exposure and the development of LPR. We congratulate authors for this interesting investigation of a poorly known topic. However, we would like to draw attention to several points.

Laryngopharyngeal reflux symptoms (e.g. throat clearing, cough, globus sensation, hoarseness) and findings (e.g. arytenoid erythema, vocal fold erythema or edema, posterior commissure hypertrophy) are nonspecific and may be commonly found in many irritative upper aerodigestive tract mucosa disorders; including tobacco or alcohol induced-pharyngolaryngitis, electronic cigarette exposure, air pollutants, or air conditioning [2–5]. For this reason, the LPR diagnosis requires the use of additional examinations, such as hypopharyngeal-esophageal multichannel intraluminal impedance-pH monitoring (HEMII-pH), which remains the most reliable tool for the identification of pharyngeal reflux event [6]. The use of HEMII-pH should establish the prevalence of LPR in this cohort of patients to better understand the nature of the relationship between anhydrous ammonia exposure and laryngopharyngeal symptoms and findings.

Indeed, the relationship between both conditions may be indirect. The exposure to toxic chemicals may increase the anxiety and the stress of patients over the post-exposure weeks because they are concerned for their health. The increase of anxiety and stress is known to be associated with autonomic nerve dysfunction and related gastroesophageal motility impairments [7]. Furthermore, previous studies supported the occurrence of an association between LPR and autonomic nerve dysfunction [8,9]. Thus, in the study of Elton et al., the post-exposure increases of gastroesophageal reflux and LPR symptoms and findings, especially heartburn (2/11 versus 10/11 patients), may be attributed to autonomic nerve dysfunction, which led to esophageal sphincter pressure impairments and related hypopharyngeal reflux events.

To sum, it is conceivable that the laryngopharyngeal symptoms and findings observed in the study of Elton et al. may be related to the sequalae of anhydrous ammonia exposure and the development of autonomic nerve dysfunction (i.e. stress); the latter increasing the risk of reflux. The association between both conditions could therefore be indirect, which is strengthened by the delay between the time of exposure and the development of symptoms. Note that this study strengthens the need to better understand the origin of symptoms and findings in patients exposed to irritative components, such as anhydrous ammonia; pepsin; or cigarette components. An interesting transversal research topic could be the study of the laryngopharyngeal and oral microbiota modifications in these conditions. Indeed, the microbiota is increasingly known for its important role in mucosa healing and homeostasis.

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Jerome R. Lechien[a,b,c,d,*], Sven Saussez[b,c], Miguel Mayo-Yanez[e], Giannicola Iannella[f], Luigi A. Vaira[g,h]

[a] *Department of Otolaryngology, Elsan Polyclinic of Poitiers, Poitiers, France*
[b] *Department of Human Anatomy and Experimental Oncology, Faculty of Medicine, UMONS Research Institute for Health Sciences and Technology, University of Mons (UMons), Mons, Belgium*
[c] *Division of Laryngology & Broncho-esophagology, EpiCURA Hospital, Baudour, Belgium*
[d] *Department of Otolaryngology-Head & Neck Surgery, Foch Hospital, School of Medicine, UFR Simone Veil, Université Versailles Saint-Quentin-en-Yvelines (Paris Saclay University), Paris, France*
[e] *Otorhinolaryngology-Head and Neck Surgery Department, Complexo Hospitalario Universitario A Coruña (CHUAC), A Coruña, Galicia, Spain*
[f] *Department of 'Organi di Senso', University "Sapienza", Viale dell'Università, 33, 00185 Rome, Italy*
[g] *Maxillofacial Surgery Operative Unit, Department of Medical, Surgical and Experimental Sciences, University of Sassari, Sassari, Italy*
[h] *Biomedical Science Department, PhD School of Biomedical Science, University of Sassari, Sassari, Italy*

[*] Corresponding author at: Division of Laryngology & Broncho-esophagology, Department of Otorhinolaryngology and Head and Neck Surgery, EpiCURA Hospital, Baudour, Belgium.
E-mail address: Jerome.Lechien@umons.ac.be (J.R. Lechien).