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Beyond the Inability to Burp: A Comprehensive Pathophysiological Theory of Retrograde Cricopharyngeal Dysfunction

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The first documented cases of retrograde cricopharyngeal dysfunction (R-CPD), a condition characterized by the inability to burp, were reported independently by Kahrilas et al.[1] and Waterman and Castell.[2] While these initial case reports provided preliminary insights, it was not until 2019 that Bastian and Smithson published the first case series, establishing R-CPD as a distinct clinical condition characterized by absent or incomplete upper esophageal sphincter (UES) relaxation in response to esophageal gas distention, resulting in a constellation of characteristic symptoms.[3] Despite an increasing number of cohort studies published in the past 6 years,[4] fundamental questions regarding R-CPD etiology remain unresolved. To date, no comprehensive hypothesis has been proposed to explain the pathogenesis of R-CPD and its associated symptomatology. This perspective paper presents a novel etiological hypothesis considering etiological findings and heterogeneous clinical patterns observed in R-CPD.

The potential etiological role of gastroesophageal reflux disease (GERD) and laryngopharyngeal reflux disease (LPRD) has been suggested,[4] alongside genetic factors,[4] in R-CPD pathogenesis. However, the marked heterogeneity in symptom severity among patients—ranging from mild manifestations to severe cases requiring botulinum toxin injection into the UES—remains incompletely understood.[4] For example, in clinical practice, some patients report the story of family members or friends with the inability to burp but no troublesome symptoms. The heterogeneity in clinical presentations of R-CPD could be explained by both the diverse esophageal motility patterns observed in LPRD patients[5] and the similarities in high-resolution manometry (HRM) findings between R-CPD and LPRD.[5]

Previous research has established that proximal reflux events can trigger UES spasms.[6] Recent investigations using 24-hour hypopharyngeal-esophageal multichannel intraluminal impedance-pH monitoring have identified 2 distinct phenotypes among patients with confirmed LPRD: those presenting with isolated LPRD and normal esophageal motility, and those exhibiting concurrent LPRD and esophageal dysmotility, with the latter potentially serving as a contributing factor to LPRD pathogenesis.[5,6] The integrity of anterograde esophageal motility represents a fundamental protective mechanism against esophagopharyngeal reflux events through its capacity to clear refluxate from the proximal to the distal esophagus. Although LPRD patients with and without esophageal dysmotility exhibit only modest clinical distinctions, the presence of impaired esophageal body motility may explain the heterogeneous clinical presentations observed in R-CPD.

Although GERD or LPRD may occur during childhood in most R-CPD patients,[4,7] those with preserved esophageal motility typically experience less/no severe esophageal

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symptoms, including gurgling noises, chest pain, or esophageal distension, due to efficient propulsion of air through the lower gastrointestinal tract. Given the vagus nerve's regulatory role in upper and lower gastrointestinal motility and its positive correlation with esophageal function, R-CPD patients with preserved esophageal motility and adequate vagal tone generally exhibit milder or no digestive symptoms, including reduced flatulence and bloating. Conversely, patients with childhood GERD or LPRD and impaired esophageal motility typically experience more severe symptomatology due to esophageal air accumulation and ineffective clearance through the lower gastrointestinal tract. The similarity in HRM findings between LPRD and R-CPD patient populations can support this theory. Thus, in 2024, Anderson et al.[8] reported that 67% (95% CI: 54%-78%) of R-CPD patients had abnormalities in HRM with ineffective motility found in 41%, and complete absence of peristalsis in 23% of cases. In the same vein, Yousef et al.[9] observed that 70% of patients with R-CPD demonstrated higher rates of ineffective swallows and 81% had incomplete bolus clearance. This hypothesis model considers that acute stress and anxiety are both conditions leading to esophageal dysmotility and reduced protective mechanisms against reflux. While LPRD patients demonstrate higher stress and anxiety levels compared to asymptomatic controls,[10] R-CPD patients exhibit an anxious/stressed personality profile that should be related to both baseline disposition and troublesome symptoms limiting their daily life.[4] Anxiety and stress demonstrate a significant association with autonomic nervous system dysfunction, particularly manifesting as diminished vagal tonicity, which subsequently impairs esophageal motility.[10] The emerging recognition of autonomic dysfunction as a fundamental etiological mechanism in LPRD[10] provides substantial support for the present theoretical framework. Given that autonomic nervous system function exhibits considerable diurnal variation in response to daily stressors, the brief duration of HRM examination (approximately 45 minutes) may be insufficient to capture the full spectrum of esophageal dysmotility events in R-CPD patients with troublesome symptoms.

The implementation of quantitative autonomic function assessment methodologies—specifically heart rate variability analysis and Sudoscan-based sympathetic function testing—combined with experimental 24-hour HRM (smallest probe), warrants investigation in prospective controlled studies to elucidate the interrelationships between autonomic function, personality characteristics, and esophageal motility patterns in R-CPD cohorts versus controls. More digestive examinations are required in children with R-CPD. The injection of botulinum toxin into the cricopharyngeal sphincter could be indicated as soon as possible to limit the impact of R-CPD on the patient's quality of life. Because proton pump inhibitors just reduce the H+ production in the stomach without changing the number/duration of reflux events, the LPRD treatment requires post-meal alginate rather than proton pump inhibitors to reduce the number and duration of reflux events, and the irritation of the proximal esophagus and upper respiratory tract.[10]

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Ethical Considerations

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Consent to Participate

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Author Contributions

Jerome R. Lechien: Design, acquisition of data, drafting, final approval, and accountability for the work; final approval of the version to be published; agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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