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Letter to the Editor Open Access

# Roemheld syndrome: Apprehending arrhythmia in a different perspective

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An intriguing article by Bodur et al.[1] investigated the association between premature atrial contractions (PACs) and gastroesophageal reflux disease (GERD). The authors found that esophagitis and/or gastritis in patients with GERD symptoms were independently associated with the increased prevalence and number of PACs. The exact mechanism of esophagitis and/or gastritis-induced supraventricular arrhythmia is still unclear. However, several hypotheses have been proposed as the underlying pathomechanism discussed by the authors, including alteration in vagal and sympathetic balance and left atrium mechanical stimulation by esophageal distension.

Arrhythmias, particularly supraventricular arrhythmias, can be caused by several predisposing factors, including gastrointestinal problems. Roemheld syndrome, known as gastrocardiac syndrome, an overlap and link between the gastrointestinal symptoms and arrhythmias, was first observed by Ludwig Roemheld[2] later, several subsequent reports described this syndrome.

Finding the link and accurately diagnosing this syndrome is often difficult due to contributions from various possible mechanisms that may cause this syndrome, including vigorous exercise, inflammation within adjacent positioning of esophagus and atria, autoimmune disorders, common nerve innervations, impediments in coronary blood flow, and hiatal hernias.[2] Esophagus stimulation, whether mechanical or chemical by acid reflux, potentiates afferent vagal activity in GERD concomitant arrhythmia patients. Elevated vagal tone shortens effective atrial refractoriness, resulting in development of atrial arrhythmia.[3]

In addition, GERD may release various inflammatory cytokines, i.e., interleukin (IL)-1β and IL-6, which lead to systemic inflammation. The increase of circulating cytokines may prompt arrhythmia development, particularly atrial fibrillation (AF). In addition, acid reflux may cause lower esophagus inflammation and subsequently penetrate the esophagus wall, affecting the vagal nerves and leading to atrial myocarditis or local pericarditis.[2,3] This may happen due to adjacency of esophageal to atrium and alteration of local receptors. Consequently, cardiac rhythm afferent-efferent reflex mechanisms were induced, secondary to vagal nerve stimulation, thus inducing arrhythmia.[2]

The presence of hiatal hernia can also directly compress atrium, causing a decrease in blood supply to the heart, resulting in relative ischemia that can lead to arrhythmias. This hiatal hernia can also aggravate acid reflux in GERD.[2,3]

Esophageal acid exposure also has potential impact on coronary blood flow. It has been reported that coronary flow was reduced by acid instillation into esophagus. This phenomenon is mediated by autonomic reflex and postulated as one of leading causes of syndrome X, characterized by typical angina despite normal coronary arteries. This phenomenon is absent in denervated heart transplant recipients, supporting that reduced coronary blood flow is achieved through cardioesophageal reflex.[4]

As the first line GERD therapy, proton pump inhibitors (PPIs) may hold potential therapeutic effects in this situation. The PPIs have multimodal effects

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beyond acid suppression mediated by proton pump ($K^+$-$H^+$ ATPase) inhibition, including antioxidant and anti-inflammatory properties. They have potential antiarrhythmic and cardioprotective agent properties due to functional similarity proteins of the gastric $K^+$-$H^+$ ATPase within cardiac.[2,3]

Quinidine and disopyramide, antiarrhythmics with vagolytic properties,[5] can be used when arrhythmia is suspected due to vasovagal stimulation, while simultaneously treating the underlying gastrointestinal disease.

In conclusion, various pathophysiological mechanisms that may underlie the occurrence of arrhythmias in Roemheld syndrome should be investigated in patients presenting with gastrointestinal complaints and arrhythmias.

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