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PULMONARY LOBECTOMY AND THE POTENTIAL OCCURRENCE OF POSTOPERATIVE DYSAUTONOMIA

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ABSTRACT

Despite advances, pulmonary lobectomy can lead to postoperative dysautonomia, a dysfunction of the autonomic nervous system. This occurs due to possible vagus nerve injury during surgery, causing chronic cough or gastrointestinal issues. Additionally, surgical stress alters autonomic activity, manifesting as hemodynamic instability and orthostatic intolerance, which may include Postural Orthostatic Tachycardia Syndrome (POTS). Further symptoms include fatigue and cognitive problems. Although not common, early recognition of these signs is vital for proper patient management and to improve their quality of life.

Keywords: Lobectomy, Dysautonomia, Vagus nerve, Postoperative period, Postural orthostatic tachycardia syndrome.

INTRODUCTION

Pulmonary lobectomy is a surgical procedure widely used in the treatment of various pulmonary conditions, notably lung cancer. Despite advances in surgical techniques, such as video-assisted thoracoscopic surgery, the procedure is not without complications.[1] The autonomic nervous system (ANS), which controls involuntary bodily functions such as heart rate, blood pressure, digestion, and respiration, may be susceptible to dysfunction due to surgical events.[2] Dysautonomia, characterized by an imbalance in ANS regulation, emerges as a potential—though less explored—complication in the postoperative context of pulmonary lobectomy.

A narrative literature review was conducted using the PubMed database to identify articles published from 2015 onward. The search was performed using a combination of the following descriptors: Lobectomy, Dysautonomia, Vagus Nerve, Postoperative Period, and Postural Orthostatic Tachycardia Syndrome. The objective was to compile and synthesize relevant information regarding the relationship between lobectomy, autonomic dysfunctions involving the vagus nerve in the postoperative period, and the occurrence of Postural Orthostatic Tachycardia Syndrome.

Potential Mechanisms of Dysautonomia Following Pulmonary Lobectomy

The occurrence of dysautonomia after pulmonary lobectomy can be explained by multiple interconnected mechanisms:

Vagus Nerve Injury

The vagus nerve (cranial nerve X) is the main component of the parasympathetic nervous system, with extensive thoracic branches that innervate vital organs, including the lungs and heart.[3] Figure 1 illustrates the course of the vagus nerve in the thorax.[4] During lobectomy, the dissection and manipulation of anatomical structures adjacent to the vagus nerve and its branches—such as the bronchi, pulmonary vessels, and mediastinal lymph nodes—may result in direct injury, stretching, or neural disruption.[5]

The vagus nerve (cranial nerve X) emerges as the principal component of the parasympathetic nervous system, exhibiting extensive thoracic branches that innervate vital organs, including the lungs and heart.[3] This widespread innervation plays a crucial role in regulating essential autonomic functions such as heart rate, bronchoconstriction, and gastrointestinal motility. As illustrated in Figure 1, the course of the vagus nerve through the thorax is complex and intricate, with its detailed anatomy and multiple branching points well established in the medical literature.[4] A precise understanding of its topography is essential for thoracic surgeons, given its vulnerability during surgical interventions.

In thoracic surgical procedures such as lobectomy, the anatomical proximity of the vagus nerve and its branches to essential structures—notably the bronchi, pulmonary vessels, and mediastinal lymph nodes—makes it particularly susceptible to iatrogenic injury. The meticulous dissection and manipulation required in these regions may inadvertently result in various types of vagal trauma, ranging from excessive stretching to complete neural disruption.[5] Such injuries can lead to a range of postoperative complications, including hoarseness, dysphagia, bradycardia, or gastrointestinal motility disorders, all of which directly affect patient recovery and quality of life. Therefore, identification and preservation of the vagus nerve are considered critical priorities to reduce the risk of morbidity and optimize surgical outcomes in thoracic procedures.

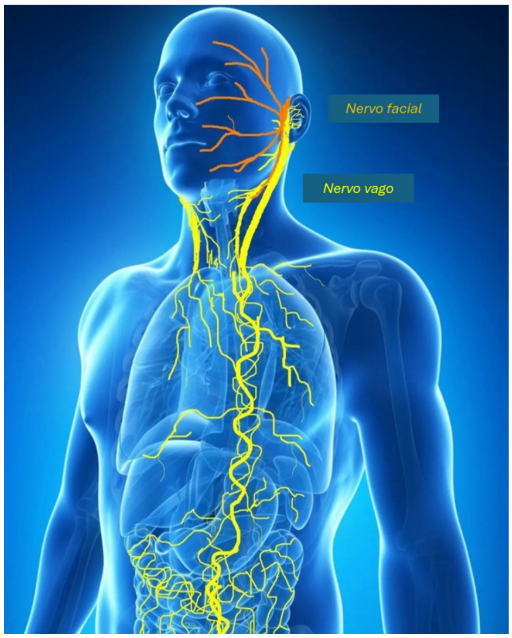

Figure 1. Path of the Vagus Nerve. The image shows the path of the vagus nerve (and a section of the facial nerve)[4]

Clinical Implications: Vagal injury can disrupt autonomic homeostasis, clinically manifesting as chronic cough due to its role in the cough reflex arc.[5] Additionally, gastrointestinal dysfunctions—such as gastroparesis (delayed gastric emptying), constipation, or diarrhea—may occur, given the vagus nerve's influence on digestive tract motility.[6]

Alterations in Sympathetic and Parasympathetic Activity

Surgical stress, the systemic inflammatory response, and postoperative pain are factors that can induce significant modulations in autonomic nervous system (ANS) activity. Major surgeries, including thoracic procedures, have been shown to precipitate postoperative autonomic dysfunction.[7] This dysfunction may present with the following characteristics:

- Hemodynamic Instability: Fluctuations in blood pressure (hypotension or hypertension) and cardiac arrhythmias (tachycardia or bradycardia) are manifestations of autonomic dysregulation.[7]
- Orthostatic Intolerance: The inability to maintain blood pressure homeostasis upon transitioning to an upright position can lead to dizziness, vertigo, or syncope. Postural Orthostatic Tachycardia Syndrome (POTS), a form of dysautonomia frequently triggered by surgical events, exemplifies this mechanism.[8]
- Fatigue and Cognitive Dysfunction: Persistent fatigue, disproportionate to physical effort, and cognitive deficits such as difficulty concentrating and "brain fog" may reflect both autonomic and cerebral dysfunction.[1]
- Sudomotor and Thermoregulatory Dysfunctions: Alterations in sweating (hypo- or hyperhidrosis) and in the regulation of body temperature are indicative of autonomic dysregulation.[2]
- Sleep Disturbances: Postoperative disruption of sleep patterns may also be associated with autonomic dysfunction.[1]

Inflammatory Response and Oxidative Stress

Surgical trauma and the subsequent inflammatory response can release mediators that directly affect the integrity or function of autonomic nerves. Additionally, postoperative oxidative stress may contribute to neural injury.[7]

Clinical Manifestations of Dysautonomia After Pulmonary Lobectomy

The symptoms of dysautonomia are diverse and may be nonspecific, making diagnosis challenging. In the postoperative context of pulmonary lobectomy, manifestations may include:

- Dizziness, vertigo, presyncope, or syncope, particularly with postural changes.[8]
- Palpitations or tachycardia at rest or with minimal exertion.[8]
- Severe fatigue and exercise intolerance.[1]
- Gastrointestinal symptoms such as nausea, vomiting, constipation, or diarrhea.[6]
- Unexplained dyspnea at rest.[1]
- Difficulty concentrating, memory problems, or "brain fog".[1]
- Abnormalities in thermoregulation and sweating patterns.[2]
- Persistent chronic cough.[5]

Diagnosis of Dysautonomia / POTS

The diagnosis of dysautonomia, and specifically of POTS, is challenging due to the wide

variety of symptoms and the absence of a single definitive diagnostic test. It requires a comprehensive approach that includes a detailed clinical history, physical examination, and specific tests to assess autonomic function.[9] The diagnostic criteria for POTS include chronic symptoms of orthostatic intolerance—such as dizziness, presyncope, palpitations, weakness, fatigue, nausea, and "brain fog"—that worsen upon standing and improve when lying down.[9] Additional criteria include increases in heart rate: a sustained increase of at least 30 beats per minute (bpm) or a heart rate of 120 bpm or more within the first 10 minutes of transitioning from a supine to a standing position (in adults). In adolescents, the increase must be at least 40 bpm[9]. There must be an absence of classical orthostatic hypotension, defined as a drop in systolic blood pressure not exceeding 20 mmHg and in diastolic pressure not exceeding 10 mmHg within the first three minutes of standing. The presence of significant orthostatic hypotension suggests other forms of dysautonomia.[9]

Care should also focus on ruling out other conditions that may cause similar symptoms, such as dehydration, anemia, thyroid disorders, pheochromocytoma, structural heart disease, or primary arrhythmias.[9]

Common Diagnostic Tests

• Tilt Table Test: This is the primary test for diagnosing POTS. The patient is placed on a table that is then tilted to an upright position (typically 70 degrees), while heart rate and blood pressure are continuously monitored. A sustained increase in heart rate that meets diagnostic criteria, without significant hypotension, confirms the diagnosis of POTS.[9]

• Quantitative Autonomic Function Tests: These may include deep breathing tests (to assess parasympathetic function), the Valsalva maneuver (to assess both sympathetic and parasympathetic function), quantitative sudomotor axon reflex test (QSART), and pupillary function tests, all of which help identify specific patterns of autonomic dysfunction.[10, 11]

• Ambulatory Blood Pressure Monitoring (ABPM) and 24-Hour Holter Monitoring: These can be useful for detecting blood pressure patterns and arrhythmias not evident during routine office visits.

Potential Non-Pharmacological Treatments for Dysautonomia
POTS: Autonomic Rehabilitation

The treatment of dysautonomia and POTS is multifaceted and aims to relieve symptoms, improve quality of life, and, when possible, address the underlying cause. Increasing emphasis has been placed on autonomic rehabilitation, which integrates non-pharmacological strategies with progressive exercise programs.[12]

Non-Pharmacological Measures and Lifestyle Modifications

• Increased Fluid and Salt Intake: Helps increase blood volume, which can mitigate orthostatic hypotension and tachycardia. It is recommended to consume 2–3 liters of fluids per day and 10–12 grams of salt, if there are no contraindications.[9]

• Compression Stockings: May help reduce blood pooling in the legs and abdomen, improving venous return and decreasing tachycardia when standing.[9]

• Head-of-Bed Elevation: Sleeping with the head of the bed elevated by 10 cm can reduce nighttime diuresis and help adjust morning plasma volume.

• Smaller, More Frequent Meals: Avoid large meals rich in carbohydrates, which can divert blood flow to the gastrointestinal tract and exacerbate orthostatic symptoms.

• Avoiding Triggers: Identify and avoid factors that worsen symptoms, such as excessive heat, dehydration, alcohol, and prolonged standing.[12]

Autonomic Rehabilitation and Physical Exercise

Autonomic rehabilitation is a crucial component of treatment, especially for POTS, aiming to improve functional capacity and exercise tolerance. Adapted and progressive physical exercise can enhance cardiovascular conditioning and autonomic regulation.[12]

• Starting with Supine or Seated Exercises: For patients with severe orthostatic intolerance, exercises should begin in positions that minimize the effects of gravity, such as using a recumbent bike, swimming, or rowing.[12]

• Gradual Progression: The program should slowly increase in intensity and duration, with a gradual transition to upright exercises such as walking. Supervision by a physical therapist experienced in dysautonomia is essential to ensure safety and effectiveness.[13]

• Strength Training: Including muscle-strengthening exercises, especially for the legs and core, can support venous return and help stabilize blood pressure.[13]

• Heart Rate Monitoring: Patients should be advised to monitor their heart rate and symptoms to avoid overexertion, which can worsen symptoms and lead to demotivation.

Pharmacological Treatment

Medications may be used to control specific symptoms that do not respond to non-pharmacological measures.[9] Options include:

• Fludrocortisone: A mineralocorticoid that increases blood volume.

• Midodrine: An alpha-1 agonist that causes vasoconstriction and raises blood pressure.

• Low-dose beta-blockers (e.g., propranolol): May reduce tachycardia, especially in patients with sympathetic hyperactivity.

• Ivabradine: A medication that reduces heart rate without affecting blood pressure, useful for refractory tachycardia.

• Pyridostigmine: An acetylcholinesterase inhibitor that may enhance cholinergic transmission and alleviate some dysautonomic symptoms.

The choice of treatment should be individualized, taking into account the severity of symptoms, the presence of comorbidities, and the patient's response.

CONCLUSION

Although dysautonomia is not classified as a common complication in most patients undergoing pulmonary lobectomy, its occurrence is plausible and clinically relevant. The complexity of thoracic anatomy and the surgical manipulation near vital neural structures justify attention to this possibility. Early recognition of autonomic symptoms is crucial for proper management and for improving patients' postoperative quality of life. A thorough investigation is recommended in patients who

develop symptoms consistent with dysautonomia after pulmonary lobectomy, aiming for an accurate diagnosis and the implementation of effective therapeutic strategies.

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