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Case Report
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A Case Report: Paroxysmal Vocal Cord Dysfunction

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Introduction

PVCD has been described as paroxysmal periods of vocal cord closure during inspiration or expiration leading to varying airway obstruction causing symptoms such as episodic dyspnea, wheezing, stridor and subjective respiratory distress. It is very commonly mistaken for asthma or anaphylaxis and often leads to unnecessary drug use, hospitalization and in the acute setting possible tracheal intubation. We present a young female with multiple psychiatric comorbidities who presented with stridor and was found to have PVCD.

Case Presentation

A 19 year old female with a history of depression, PTSD, polysubstance abuse and previous suicidal attempts presented for the third time in two days to the emergency department with stridor and dyspnea. On previous visits she has received intramuscular epinephrine and steroids with temporary symptomatic relief. On admission patient hemodynamically stable, SPO2 98% room air and on exam hoarsening of her voice, stridor and inspiratory wheezing. Chest radiography unremarkable, CBC and BMP within the normal limits. She received methylprednisolone. Her home medications were lamictal, quetiapine, gabapentin, duloxetine, these were held on admission suspecting the patient was having an anaphylactic reaction secondary to medications. One day after her medications were discontinued her respiratory symptoms were resolved. Suddenly, the patient developed again stridor and dyspnea, when examining the patient her SPO2 was 98% room air, inspiratory wheezing bilaterally but more prominent in the cervical area. Otorhinolaryngology was consulted; they performed a laryngoscopy that showed aberrant adduction of the true vocal folds with inspiration, returning to normal position during expiration. The patient was educated about exercises to perform when these episodes, she was set up with speech therapy as an outpatient [1,2].

Discussion

PVCD pathogenesis remains uncertain but both organic (GERD, irritants) and non-organic (psychological, emotional, exercise) causes have been identified as precipitating factors. There is a reported female predominance with most of the patients having an underlying psychiatric disorder. Clinical presentation includes intermittent asthma-like or anaphylactic symptoms that do not respond to standard treatment. In asymptomatic periods, physical exam is normal. Gold standard for diagnosis is direct visualization of the upper airway showing complete vocal cord adduction of the anterior two-thirds during inspiration with formation of a small posterior diamond-shaped cleft.

Treatment includes identification and management of contributing factors. Short term termination of acute attacks involves relaxation techniques like pursing lips and panting. Benzodiazepines, helium-oxygen gas mixtures, continuous positive airway pressure, topical lidocaine, and botulinum toxin have been reported to be effective. The long-term management includes speech therapy as a mainstay. Literature suggests that PVCD is generally a self-limiting disorder with most patients having no long-term sequela once the diagnosis has been established.

Conclusion

PVCD can be an underlying cause of stridor and dyspnea especially in the setting of recurrent hospitalizations and failure of response to conventional asthma/anaphylaxis therapy. This diagnosis should be kept in mind to avoid high medical utilization, unnecessary high-dose steroid use. Initial management includes control of underlying triggers and vocal cord exercises.

References

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