



CLINICAL REASONING CORNER

Recurrent Episodes of Breathlessness in a Young Man

Bahae Eddine El Kheyyat, MD¹

¹ Zaporizhzhia State Medical University, Zaporizhzhia, Ukraine

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ABSTRACT

A 34-year-old man presented with seven months of paroxysmal, rapid-onset throat tightness and a feeling of breathlessness. Despite three emergency department visits, he was discharged without a confirmed diagnosis. Empiric treatment for panic attacks and asthma did not prevent recurrence. Respiratory rate, oxygen saturation, chest radiography, arterial blood gas, and an electrocardiogram were normal during evaluated episodes. During a subsequent medical encounter, reframing prompted scrutiny of the initial hypotheses and ultimately led to diagnostic resolution. This case illustrates how anchoring bias can persist across encounters when an unconfirmed diagnostic label is carried forward and emphasizes the importance of iterative diagnostic testing, particularly when the treatment of the presumed diagnosis is ineffective.

CASE PRESENTATION

A 34-year-old man was referred to the outpatient clinic by his general practitioner for seven months of recurrent, sudden-onset breathlessness and throat tightness. Episodes occurred at rest, including while eating, watching television, or sitting quietly. It was unclear if it was also triggered by exertion as the patient lived a predominantly sedentary lifestyle. All

episodes were similar, lasting approximately two minutes and resolving spontaneously without cough or wheezing. He was completely well between episodes. He had no significant past medical or surgical history, including childhood asthma, other chronic respiratory disease, symptoms of gastroesophageal reflux disease, occupational exposure to dust or chemical irritants, odor sensitivity, or dysphonia. There was no family history of asthma, atopy, cardiopulmonary disease, or similar symptoms. He did not smoke or drink alcohol.

He had presented to the emergency department (ED) three times with these symptoms. At the first visit, respiratory rate and other vital signs were normal, including an oxygen saturation of 99% on room air. The physical examination, chest radiograph (CXR), and electrocardiogram (ECG) were normal; he was given lorazepam and discharged with a diagnosis of panic attack. The second episode was clinically similar, characterized by increased work of breathing but unremarkable chest examination. His breathing normalized spontaneously before nebulized albuterol was administered. He was subsequently discharged with an inhaler and a working diagnosis of asthma. No spirometry, bronchodilator reversibility testing, or fractional exhaled nitric oxide (FeNO) measurement had been performed. On follow-up, he reported no benefit from albuterol. During the third ED evaluation,

he again reported sudden breathlessness and throat tightness. Vital signs and examination of the chest remained normal, with no wheezing. An arterial blood gas (ABG) obtained while symptomatic was as follows: pH 7.41, PaO₂ 97 mmHg, and PaCO₂ 38 mmHg (all values within normal limits). He was discharged without a confirmed diagnosis and referred to the outpatient clinic.

Upon presentation to the clinic, he appeared well and comfortable at rest. Work of breathing was normal and chest auscultation was clear. Forced inspiration elicited a brief high-pitched sound that was loudest over the neck and diminished toward the chest. There was no expiratory component. This sound persisted after bronchodilator administration. Although the patient noticed it during each prior episode, this important finding had not been documented during his preceding ED visits.

DISCUSSION

The key to this case is better qualifying and refining the dyspnea. Three questions, readily answerable at the bedside, were used as a clinical localization heuristic: 1.) *Was the respiratory difficulty predominantly inspiratory or expiratory?* 2.) *Were the abnormal respiratory sounds louder over the neck or chest?* And, 3.) *Was the patient normal between episodes?* In this patient, inspiratory symptoms, a neck-localized sound, and complete resolution between episodes favored an intermittent upper-airway process. This framework guided us to consider and evaluate for vocal cord dysfunction (VCD)/inducible laryngeal obstruction (ILO).^{1,2} The Pittsburgh Vocal Cord Dysfunction Index, a clinical scoring tool to help differentiate asthma from VCD, was obtained.³ Throat tightness contributed 4 points and the absence of wheezing 2 points, giving a total score of 6, above the published cutoff of 4. Dysphonia and odor sensitivity were absent and contributed no additional points. The results of the index increased suspicion for VCD/ILO, but alone were not diagnostic.

Further investigations were pursued. Laboratory testing was unremarkable (Table 1); echocardiography showed normal left ventricular function without outflow tract obstruction. Spirometry demonstrated

Table 1. Selected Laboratory Data

Test	Result	Reference Range
Sodium (mmol/L)	140	135-145
Potassium (mmol/L)	3.9	3.4-5.0
Chloride (mmol/L)	99	98-108
Carbon Dioxide (mmol/L)	24	23-32
Urea Nitrogen (mg/dL)	12	8-25
Creatinine (mg/dL)	1.3	0.6-1.5
Glucose (mg/dL)	92	70-110
Calcium (mg/dL)	10.0	8.5-10.5
Total IgE (IU/mL)	36	<100
Thyroid Stimulating Hormone (mIU/L)	2.0	0.4-4.0
White Cell Count (per μ L)	5,000	4,500-11,000
Absolute Eosinophil Count (per μ L)	90	<500
Hemoglobin (g/dL)	14.0	13.5-17.4
Platelets (per μ L)	300,000	150,000-400,000

normal pre- and post-bronchodilator values without significant reversibility; FeNO testing was normal (Table 2). The flow-volume loop showed inspiratory truncation with preservation of the expiratory contour, consistent with variable extrathoracic obstruction.

These findings prompted a laryngoscopy with provocation. Flexible nasolaryngoscopy at rest was unremarkable, with normal vocal-fold appearance and symmetric abduction. Exercise provocation using ten minutes of stationary cycling reproduced his usual symptoms. Repeat laryngoscopy was performed, revealing paradoxical adduction of the vocal folds during inspiration, exceeding 50% of the glottic aperture, along with a small residual posterior glottic gap between the arytenoid cartilages. No structural lesion, vocal-fold palsy, or mass was identified.

Table 2. Pulmonary Testing			
Test	Pre-bronchodilator	Post-bronchodilator	Reference
FEV ₁	3.5 L (93% predicted)	3.5 L (93% predicted)	Normal
FVC	4.3 L (96% predicted)	4.3 L (96% predicted)	Normal
FEV ₁ /FVC	0.81	0.81	>0.8
Bronchodilator change in FEV ₁	N/A	Increase of 1%	<12%
FeNO	8 ppb	N/A	<25 ppb

Abbreviations: FEV₁ (forced expiratory volume in 1 second); FVC (forced vital capacity); FEV₁/FVC (ratio of forced expiratory volume in 1 second to forced vital capacity); FeNO (fractional exhaled nitric oxide).

Performing a cognitive autopsy, the diagnostic delay was not the absence of data, but how the data was framed. Panic attacks and asthma are indeed common causes of dyspnea in a young person and remain reasonable diagnostic considerations. Anxiety may accompany VCD/ILO or develop in response to frightening airway symptoms. However, attributing these symptoms to a psychiatric cause should be a diagnosis of exclusion and should not substitute for airway localization and objective assessment.^{1,2,6} Asthma is characterized by variable expiratory airflow limitation, and objective testing should be interpreted in the context of clinical probability rather than as a single exclusionary test.⁴ Normal spirometry, FeNO, or blood eosinophils therefore do not independently exclude asthma; however, when these findings are considered alongside a clinical pattern that is atypical for asthma, they should prompt reconsideration of the initial diagnostic framework.⁴ However, the frequent coexistence of asthma and VCD/ILO may further entrench an initial diagnosis of asthma, making diagnostic reconsideration more difficult. In this patient, repeatedly normal examinations, including the absence of expiratory wheezing, along with normal pre- and post-bronchodilator spirometry without significant reversibility, argued against lower-airway obstruction. A low FeNO and normal eosinophil count provided additional evidence against type 2 airway inflammation. Instead, the throat tightness and neck-localized, inspiratory sounds pointed toward an upper-

airway process. As the episodes were sudden, brief, and self-resolving, with no residual signature between attacks, a dynamic condition, like VCD/ILO, was ultimately considered.¹⁻⁵

The second visit illustrates how treatment timing can distort diagnostic reasoning. His symptoms had resolved before nebulized albuterol was administered, so this did not establish a bronchodilator response. The asthma label had nevertheless been applied before objective confirmation and was carried forward between encounters. In a retrospective analysis of 292 patients (with diagnoses of VCD, asthma, both, and controls), 42.4% of those with VCD had previously been diagnosed with asthma, with an average reported delay of 9 years before VCD diagnosis; asthma coexisted in nearly one third of patients.⁵ An unconfirmed impression can therefore become a working diagnosis that persists despite discordant findings. Such anchoring bias can also cause new information to be interpreted through this established, but incorrect, diagnostic frame.⁶

The previously mentioned Pittsburgh Vocal Cord Dysfunction Index is another supported clinical assessment tool to distinguish VCD/ILO from asthma, but again is not a diagnostic endpoint. The index incorporates throat tightness, dysphonia, absence of wheezing, and odor triggers, with a score ≥ 4 used to distinguish VCD/ILO from asthma with a sensitivity and specificity of 0.83 and 0.95, respectively.³ This

patient's score was 6 and further increased clinical suspicion for the diagnosis, but did not obviate the need for laryngoscopic evaluation. Recent consensus recommendations emphasize direct laryngoscopic visualization as the reference standard for confirming VCD/ILO, with symptom provocation when feasible. Clinical features such as abrupt symptom onset and throat tightness may help identify patients in whom laryngoscopic evaluation should be prioritized. When symptoms are intermittent, provocation may increase the likelihood of capturing the transient laryngeal abnormalities characteristic of this condition.⁸⁻¹⁰

Unfortunately, a normal ABG does not distinguish VCD/ILO from asthma or, by itself, localize dyspnea outside the lungs. Asthma can occur with normal gas exchange, particularly when airflow limitation is not severe, and other pulmonary disorders can also present with a normal ABG. In this case, the ABG was informative only in the context of the broader pattern:

respiratory distress localized to the neck during inspiration, without hypoxemia or hypercapnia. Dynamic laryngeal narrowing, as in VCD/ILO, increases inspiratory resistance and work of breathing while gas exchange may remain preserved.^{1,2}

The differential diagnosis for dyspnea driven by an acute upper-airway disease includes anaphylaxis, angioedema, structural laryngeal obstruction, and other causes of stridor; VCD/ILO can mimic many of these.⁷ Here, stereotyped episodes, spontaneous resolution, absence of an allergic trigger or systemic features, as well as normality between attacks, argued against anaphylaxis or persistent structural obstruction. These alternatives should nevertheless be considered when episodic dyspnea with upper-airway localization develops, especially when accompanied by symptoms like stridor, swelling, urticaria, hypotension, or relevant allergen exposure.⁷

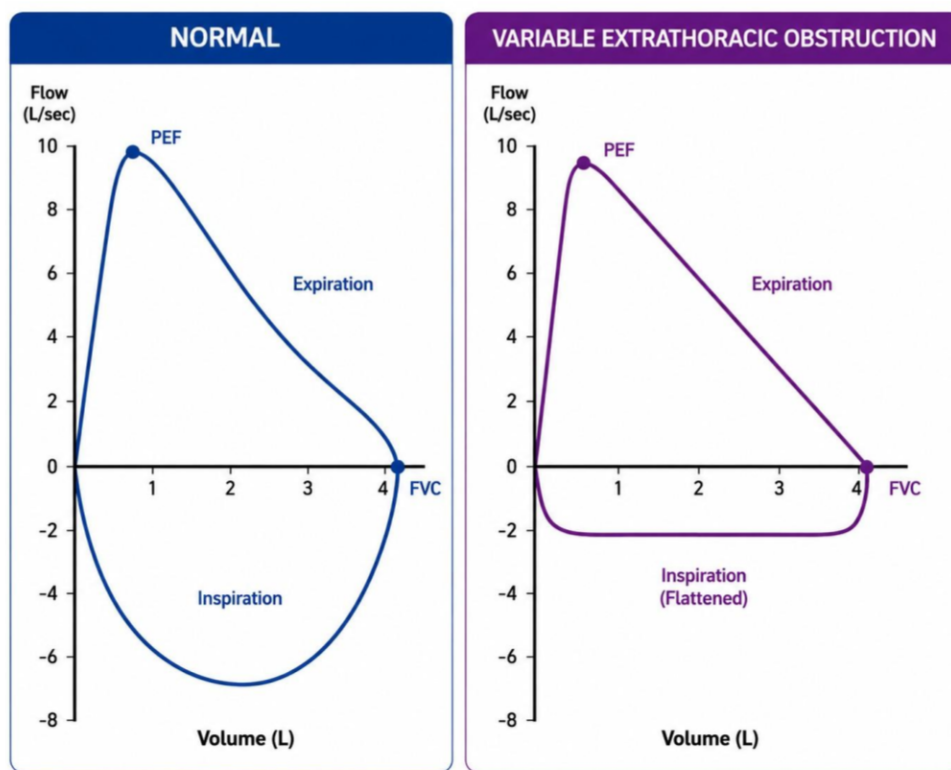


Figure 1. Author-created schematic representation of normal and variable extrathoracic obstructive flow-volume loop patterns. **Left (blue):** normal pattern showing a smooth expiratory arch above the axis and a full rounded inspiratory curve below. **Right (purple):** variable extrathoracic obstruction showing a preserved expiratory arch with a flattened inspiratory limb—the pattern seen in vocal cord dysfunction (VCD)/inducible laryngeal obstruction (ILO).

The flow-volume loop provided an additional localization clue. Inspiratory truncation with preserved expiratory contour suggests variable extrathoracic obstruction (Figure 1) but is not diagnostic; one review found truncation in only 28% of reported VCD patients.¹ Because VCD/ILO is a dynamic disorder, diagnostic testing is most informative when symptoms are present or reproduced through appropriate provocation.^{2,8} In this patient, exercise reproduced his characteristic symptoms, allowing direct visualization of paradoxical inspiratory vocal-fold adduction. The adduction exceeded 50% of the glottic aperture and was accompanied by a small posterior glottic gap, with no structural lesion. This evaluation provided definitive confirmation despite a normal resting laryngoscopy. Thus, when the history strongly suggests an intermittent laryngeal disorder, a normal resting examination should not preclude further evaluation with provocation in appropriately selected patients.^{2,8-10}

Management commonly involves speech and language therapy, including laryngeal control exercises and breathing retraining. Associated conditions such as reflux, rhinitis, irritant exposure, and psychological or behavioral factors should be assessed and treated when clinically relevant.^{1,2,8} This patient reported no reflux symptoms, occupational irritant exposure, odor sensitivity, or dysphonia; formal psychiatric screening was not performed, though the patient did not report any symptoms. The absence of objective evidence supporting asthma at this evaluation should be interpreted just as that—one evaluation. Although the clinical picture and accumulated evidence support VCD/ILO as the primary diagnosis, coexisting asthma cannot be excluded given the imperfect sensitivity of spirometry. Repeat testing should be considered when clinically indicated.^{4,5} The etiology of intermittent symptoms can be difficult to diagnose, particularly when evaluation is performed while the patient is asymptomatic. Thus, whenever feasible, symptom provocation, combined with careful attention to the time course—dynamic and intermittent in this case—can provide complementary diagnostic footholds for solving the recurrent clinical problem.

FINAL DIAGNOSIS

Vocal Cord Dysfunction / Inducible Laryngeal Obstruction

The patient has undergone speech and language therapy with notable improvement in symptoms.

KEY TEACHING POINTS

- The respiratory phase, location of abnormal respiratory sounds, and the presence (or absence) of symptoms between episodes can help localize episodic dyspnea before diagnostic testing.
- Normal oxygenation does not exclude important pulmonary disease or explain away substantial respiratory distress; arterial blood gas results must be interpreted in the clinical context.
- Normal spirometry, low FeNO, or a normal blood eosinophil count at a single assessment does not independently exclude asthma; asthma may coexist with VCD/ILO.
- Clinical scoring systems and inspiratory flow-volume-loop abnormalities can increase suspicion for VCD/ILO, but laryngoscopy during symptoms, with provocation when needed, is required for diagnostic confirmation.
- When an unconfirmed diagnosis is carried across clinical encounters, anchoring bias can delay reconsideration; convergence of the clinical pattern and objective findings should prompt reassessment of the working diagnosis.

CONFLICT OF INTEREST STATEMENT

The author declares no conflict of interest.

CORRESPONDENCE

Bahae Eddine El Kheyyat, MD

Email: bahaeeddineelkheyyat@gmail.com

ORCID

Bahae Eddine El Kheyyat 

<https://orcid.org/0009-0008-8673-9880>

ETHICS STATEMENT

The author(s) obtained verbal informed consent, witnessed and documented in accordance with institutional guidelines, for publication of medical information and images.

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