

Untangling asthma, inducible laryngeal obstruction, and dysfunctional breathing in a competitive sportsperson

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Associate Editor: Bei He

Abstract

A young woman with historically mild asthma experienced worsening breathlessness and cough with competitive ice skating. Despite optimizing and escalating treatment for her eosinophilic asthma, and addressing known exacerbating factors, her symptoms remained uncontrolled and refractory to bronchodilators and oral corticosteroids. Objective testing suggested her presentation was out of keeping with asthma alone, and she was suspected to have comorbid dysfunctional breathing and/or inducible laryngeal obstruction. Evidence was required to confirm the diagnoses, assess each condition's contribution to her symptom burden, and guide therapy. As exercise was a predominant trigger, she proceeded to cardiopulmonary exercise test with continuous laryngoscopy during exercise (CPET-CLE). Testing confirmed the presence of two forms of inducible laryngeal obstruction and evidence of hyperventilation predominant dysfunctional breathing. This case highlights the importance of identifying coexisting conditions in difficult-to-treat asthma, and the value of structured multidisciplinary assessment in referral centres for such individuals.

KEYWORDS

asthma, breathlessness, dysfunctional breathing, inducible laryngeal obstruction, vocal cord dysfunction

INTRODUCTION

Uncontrolled asthma is common in Australia, partly due to undertreatment, severe disease, and unrecognized contributory comorbidities.^{1,2} Inducible laryngeal obstruction (ILO) and dysfunctional breathing (DB) are recognized drivers of poor symptom control.^{3–5} Guidelines recommend structured multidisciplinary assessment to identify and address issues systematically.^{2,6–8} However this often does not occur due to limited expertise or resources.

CASE REPORT

A competitive ice skater was referred to a tertiary hospital respiratory outpatient service for assessment of worsening breathlessness and cough during exercise. She was diagnosed with asthma at the age of 14, and her symptoms had been well controlled on ICS/LABA. Over the past 2 years

she had become more breathless with exercise, especially when ice skating. Symptoms were also triggered by smoke pollution, perfumes, aerosols, and potent smells. The onset and escalation of breathlessness, throat and upper chest tightness was rapid, within seconds of exposure to irritants, and often triggered non-productive coughing fits. Symptom duration varied between 5 and 30 min and resolved spontaneously. Salbutamol and oral corticosteroids were ineffective during attacks, and she was hospitalized three times over the past 2 years for such attacks—one such is detailed below.

During an ice-skating competition, the patient was exposed to cold dry air, and high intensity exercise, but also aerosols and perfumes in the changing rooms. Her sudden onset shortness of breath, chest tightness and wheeze-like sounds on both inspiration and expiration were unresponsive to high-dose short-acting bronchodilators delivered via spacer, prompting hospital attendance. On admission her oxygen saturations were 97%, respiratory rate 28, heart rate

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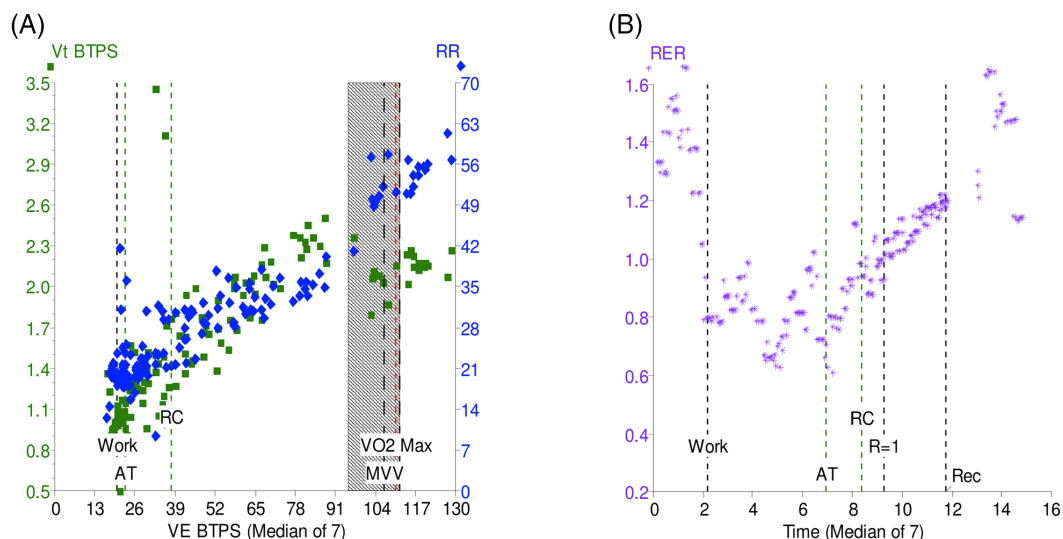


FIGURE 1 Cardiopulmonary exercise test (CPET) demonstrating atypical respiratory response to exercise. (A) The relationship between tidal volumes (Vt) and respiratory rate (RR) is disrupted, with periods of high RR and low Vt both in early and in late exercise. Ventilation was excessive relative to metabolic demand, with peak RR ~60 rpm. (B) The respiratory exchange ratio (RER) is high prior to work onset, erratic during work phase, and high during the recovery phase. This is in keeping with periods of low CO₂ from hyperventilation-predominant dysfunctional breathing pattern.

120 bpm sinus rhythm, blood pressure 116/70 mmHg and afebrile. She was speaking in short sentences and “widespread inspiratory and expiratory sounds” was noted on auscultation. Her presentation to a local hospital resulted in adrenaline administration, high flow nasal prongs, and high-dependency unit admission for suspected severe asthma exacerbation. She was not intubated. Her relevant results included: potassium 2.7 mmol/L, lactate 4.4 mmol/L, eosinophil count of 90 cells/ μ L. Chest x-ray was unremarkable. She was treated for a non-infective asthma exacerbation and was admitted under respiratory medicine for 4 days. Her peak flows in hospital varied from 200 to 280 L/min with diurnal variation noted, with a baseline of 420 L/min. Subsequent outpatient investigations demonstrated normal spirometry with no airflow limitation or bronchial hyperreactivity; FEV1 2.86 L (93%), FVC 3.36 L (97%) FEV1/FVC 85, bronchodilator response in FEV1 + 0.16 (+5%). FeNO 31 ppb.

Her persistent, severe, symptoms which were poorly responsive to guideline-based asthma management prompted referral to a specialist multidisciplinary asthma service. The reassuring objective investigations suggested a likely confounding comorbidity. Inducible laryngeal obstruction (ILO) and dysfunctional breathing were suspected to be complicating her eosinophilic asthma. She underwent the following investigations (Figure 1).

Flexible nasoendoscopy with provocation (perfume challenge)

Normal anatomy and mucosa of the nasal passages, upper airway, larynx, and vocal cords with symmetrical cord movement on phonation. There was obvious vocal cord

adduction throughout inspiration following a perfume challenge, confirming ILO at the glottic level. Symptoms were not fully representative of her presentation (Figure 2).

Cardiopulmonary exercise test with continuous laryngoscopy during exercise (CPET-CLE)

Throughout CPET-CLE the patient intermittently developed vocal cord adduction during inspiration, which was accompanied by her typical symptoms. Exercise-induced ILO (EILO) was confirmed, with total Maat score 10 indicating grade III (severe) EILO. EILODI score 34/48. Her CPET was also significant for abnormal hyperventilation and loss of breathing reserve with ETCO₂ 22 mmHg, which indicated dysfunctional breathing. Exercise capacity, aerobic capacity, circulatory response and post-exercise spirometry were all within normal limits (Figure 2).

She was diagnosed with “classic” ILO, exercise induced laryngeal obstruction (EILO), and dysfunctional breathing pattern (DB). She underwent individualized therapy with both speech pathology and physiotherapy with good response.

DISCUSSION

This case highlights three key points in managing difficult-to-control asthma. Firstly, the importance of identifying and acting upon a discrepancy between symptom severity and objective evidence. Both ILO and DB cause breathlessness, exercise intolerance, chest tightness and noisy breathing

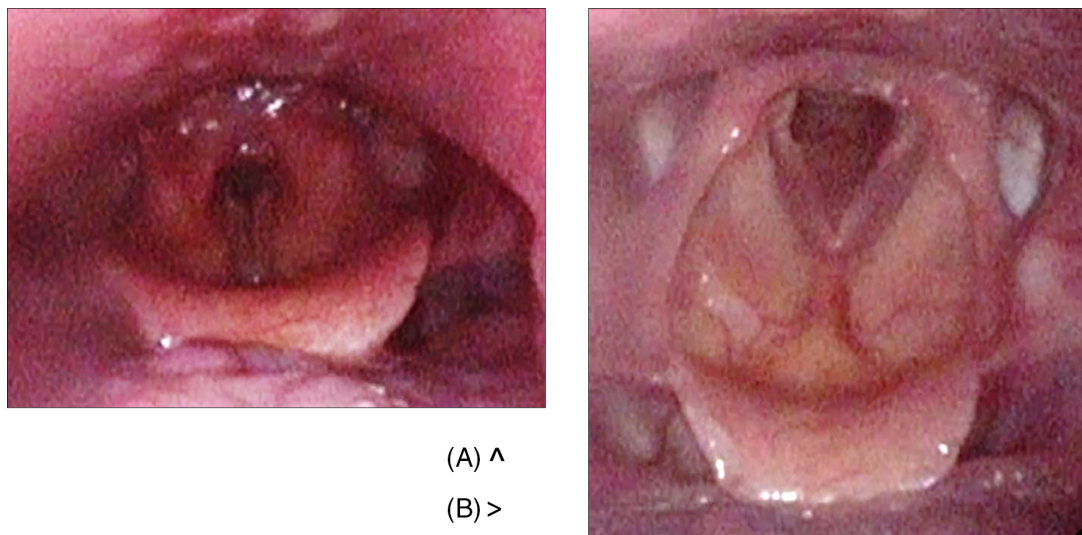


FIGURE 2 Flexible nasoendoscopy images taken during cardiopulmonary exercise testing with continuous laryngoscopy (CPET-CLE). Both images are during inspiratory phase near peak exercise demonstrating (A) glottic narrowing from vocal cord adduction, and supraglottic narrowing with medial rotation of the cuneiform tubercles. (B) Complete resolution of both glottic and supraglottic narrowing within seconds following patient-initiated nasal sniff and pursed lip exhale.

which can be difficult to differentiate from asthma. ILO and DB appear highly prevalent in asthmatics; up to 25% of asthma patients have comorbid ILO, and almost 30% have DB.^{3,5,9,10} These conditions frequently result in avoidable harm by way of symptom amplification, poor quality of life, escalation of toxic therapies, and unscheduled hospital attendances.¹¹ Secondly, a structured multidisciplinary approach improves outcomes, improves control of asthma if present, and reduces oral corticosteroid prescription.^{6,12} Since diagnosis and individualized allied health treatment, this patient has avoided hospitalization, and reduced oral corticosteroid and reliever use. Her symptom control has improved with the integration of ILO and DB therapies in her training. Finally, this case illustrates the pivotal role of complex testing in the hospital outpatient setting. Laryngoscopy with provocation—either with aerosol challenge or exercise—to reproduce presenting symptoms is gold standard in confirming an ILO diagnosis.¹³ This investigation requires a speech pathologist with experience in ILO diagnosis, severity scoring and management. Dysfunctional breathing currently has no gold standard investigation. However, CPET can definitively exclude pathologic causes of breathlessness whilst identifying abnormal respiratory patterns. Integration of CPET with continuous laryngoscopy permits the delineation of the relative varying contributions of co-existing pathologies over the exercise period.

In summary, identifying, diagnosing, and treating comorbidity is crucial in delivering optimal patient-centred asthma care and requires a multidisciplinary approach and objective testing in experienced hands.

AUTHOR CONTRIBUTIONS

Alice L. Crawford wrote the manuscript and figure legends, edited figures, completed literature review and references.

Niranjan Setty provided referencing and editing assistance. Brooke Kyle reviewed and edited the manuscript. Kate Baumwol provided flexible nasoendoscopy images, description of laryngeal findings and severity ratings. John Blakey provided the case, reviewed and edited the final manuscript.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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How to cite this article: Crawford AL, Setty N, Kyle B, Baumwol K, Blakey JD. Untangling asthma, inducible laryngeal obstruction, and dysfunctional breathing in a competitive sportsperson. *Respirology Case Reports*. 2024;12(1):e01282. <https://doi.org/10.1002/rcr2.1282>