CASE REPORT

Twin airway abnormalities complicating the management of acute asthma: a case report

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Abstract

The term ‘refractory asthma’ includes patients with severe asthma, steroid-dependent and/or resistant asthma, difficult-to-treat asthma and irreversible asthma. In patients with difficult-to-treat asthma, exclusion of other causes of persistent wheeze like vocal cord dysfunction, upper airway obstruction and allergic bronchopulmonary aspergillosis is important. Besides, the presence of anatomical abnormalities that could affect effective medication delivery could also result in sub-optimal treatment response. These factors reiterate the need for a rigorous and systematic approach to rule out alternative co-existent diseases or abnormalities in a patient with difficult-to-treat asthma. We hereby report a case of an asthmatic patient with refractory bronchospasm despite optimal treatment, wherein work-up for an additional pathophysiological process aided in successful management of his symptoms.

INTRODUCTION

Asthma is a common respiratory illness and is managed with inhaled and oral agents like bronchodilators, corticosteroids and leukotriene-receptor antagonists. In patients who are unresponsive to treatment, consideration of an alternative diagnosis or associated illness is warranted.

We hereby report a 60-year-old asthmatic male patient who was unresponsive to the standard treatment of asthma and work-up for a second illness resulted in uncovering a rare congenital abnormality.

CASE REPORT

A 60-year-old, non-smoking, male asthmatic patient was referred to us with 3 weeks history of breathlessness, wheezing and dry cough. He had a history of acute severe asthma many years ago. However, he had not required ventilatory support during that period. Since then, he had self-medicated intermittently with oral salbutamol and theophylline only. There was no past history of major surgery or other medical illnesses.

On examination, the patient had dyspnoea at rest and tachycardia. Oxygen saturation was 90% on room air. Respiratory examination revealed bilateral polyphonic rhonchi; otherwise, he was normal. He was managed with nebulized bronchodilators, intravenous corticosteroids and oxygen inhalation, despite which he had persistent bronchospasm and breathlessness on minimal exertion.

Arterial blood gas analysis revealed hypoxemia (PaO$_2$: 54 mmHg) with respiratory alkalosis (pH: 7.43, PaCO$_2$: 32.3 mmHg and Bicarbonate: 20 mEq/l). Other laboratory tests, including work-up for allergic bronchopulmonary aspergillosis, were normal. Chest radiogram and computerized tomography (CT) thorax showed prominent bronchovascular markings and hyperinflated lung fields (Fig. 1a and b).
In view of persistent symptoms despite optimal management, bronchoscopy was performed to rule out vocal cord dysfunction and central airway tumour. It revealed three subglottic circumferential membranous structures causing partial tracheal obstruction. In addition, dynamic collapse of the posterior tracheal wall causing near total occlusion of the tracheal lumen was seen during expiration (Figs 2 and 3). Biopsies were taken from the subglottic membranous structures; histopathology was suggestive of a tracheal web (Fig. 4).

A diagnosis of asthma with excessive dynamic airway collapse (EDAC) and tracheal web was made. A trial of non-invasive ventilation (NIV) was given to overcome the EDAC; he reported symptomatic improvement and on auscultation wheeze disappeared completely at a continuous positive airway pressure (CPAP) of 8 cm of water. Thereafter, he was initiated on CPAP therapy.

An otorhinolaryngology opinion was taken for surgical excision/stenting of the tracheal web. A stent placement was recommended; however, the patient refused to undergo stent placement.

Hence the patient was discharged with advice to use a CPAP machine in addition to standard asthma treatment. On follow-up at 1 month, the patient was asymptomatic with the above measures. Spirometry was performed, which showed moderate airflow limitation with significant bronchodilator reversibility.
Pharmacological therapy was titrated accordingly. The patient was advised to discontinue CPAP, which did not result in worsening of symptoms. Hence he was advised to rigorously adhere to inhaled medications and report for regular follow-up.

**DISCUSSION**

The above case drives home the point that a high index of suspicion for complications of severe airflow limitation as well as for the presence of additional anatomical abnormalities is essential for the successful management of difficult-to-treat asthma. Some illnesses associated with asthma that result in poor therapeutic outcomes include vocal cord dysfunction and upper airway obstruction [1].

In literature, there has been a report of tracheal web misdiagnosed as asthma [2]. However, our patient is the first case of asthma and an associated congenital tracheal web. Tracheal webs may be congenital or occur as a complication of tracheal intubation or tracheostomy. Congenital webs are rare (1 in 10,000 births) and manifest in childhood with wheezing and dyspnoea [3]. However, in adults, tracheal webs have been described mostly post-intubation [4]. Our patient had never undergone tracheal intubation and thus probably had undiagnosed asymptomatic congenital tracheal web.

EDAC refers to abnormal and exaggerated bulging of the posterior tracheal membrane within the airway lumen during exhalation causing reduction in cross-sectional area of >50% [3]. It has been commonly reported in asthma and COPD patients, and is postulated to occur due to a negative transmural pressure gradient during expiration which causes invagination of the posterior wall and airway narrowing. This dynamic airflow obstruction can be demonstrated by bronchoscopy or dynamic CT and is treated with application of CPAP [6].

In our patient, the EDAC and tracheal web were confirmed bronchoscopically. While the EDAC occurred due to severe untreated asthma, the tracheal web was congenital and incidentally detected. The immediate disappearance of rhonchi and breathlessness after institution of non-invasive ventilation prompted us to postulate that the patient had an element of anatomical upper airway obstruction due to the tracheal web and EDAC, which led to persistent symptoms. Besides, the above two entities could have contributed to poor delivery of bronchodilators to the airway and thus a sub-optimal response to nebulized bronchodilators.

On follow-up, we were able to wean the patient from non-invasive ventilation after optimizing and ensuring compliance with asthma treatment. The timely performance of bronchoscopy in this patient aided in identification of the dual co-existent abnormalities, one of which was anatomical and hitherto asymptomatic and the other was a complication of chronic severe airflow limitation. Thus, this case reinforces the need to work-up for co-existent and associated pathologies in patients with difficult-to-treat asthma.

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