An undiagnosed cause of chronic cough

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Abstract

Tracheomalacia (TM) refers to loss of tracheal rigidity and resulting susceptibility to collapse. It is usually an incidental finding during investigations of other illness. The main symptoms are dyspnoea, cough, sputum production and hemoptysis. Most cases are considered as respiratory infection and are treated symptomatically. Acquired TM results from damage to trachea due to various conditions such as inflammation, chronic pressure, or medical/surgical procedures. The diagnosis is done by end-expiratory dynamic tracheal imaging, which demonstrates typical crescentic narrowing of trachea and reduced antero-posterior diameter <50% of normal. Management include conservative measures like cough suppressants or surgical measures like tracheoplasty, stenting or surgical repair. We are reporting a case of chronic cough, which was subsequently diagnosed as TM.

Keywords: Chronic cough, tracheomalacia, undiagnosed cause

Introduction

Tracheomalacia (TM) is one of the rare causes of chronic cough. Many times, it is an incidental finding during investigations of other illness. This is a unique case of acquired TM, which was diagnosed after 8 years of insult due to lack of awareness of this clinical entity. Only few cases of acquired TM have been reported.

Case Report

A 50-year-old female presented with a history of fever, abdominal pain and loose stools for the duration of 1-week. She had dry cough since 2 weeks and was on symptomatic treatment. She did not have history of vomiting, dyspnoea, sputum production or haemoptysis. She did not have previous history of obstructive airway disease or antitubercular treatment. She was known to have type 2 diabetes mellitus, hypertension and hypothyroidism since many years, on regular medicines. She underwent thyroid surgery for multinodular goitre 18 years back. She had surgical site infection with involvement of trachea 1-week after surgery. On detailed enquiry, it was found that she had on and off dry cough since last 8 years, which was treated symptomatically with cough suppressants and antibiotics many times. But, symptoms used to recur each time.

Physical examination revealed a moderately built woman, weight of 62 kg, afebrile, blood pressure of 80/50 mmHg, pulse rate of 110/min, SpO₂ of 94 % on room air. She had a surgical scar of thyroid surgery over anterior side of neck. She did not have pallor, icterus, cyanosis, clubbing, pedal oedema or lymphadenopathy. She was dehydrated. Her peripheries were cool and peripheral pulses were not well palpable. Gastrointestinal system examination was unremarkable. Upper respiratory tract examination was normal. She had scattered coarse crackles at bilateral lung bases.

The investigations showed:
- Total count: 15,500 (4000–10,000 cells/cm³)
- Haemoglobin: 11.2 (13–16 mg/dL)
- Platelets: 231,000 (150,000–400,000 cells/µL)

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- C-reactive protein: 31.3 (0–5 mg/L)
- Serum creatinine: 1.9 (0.6–1.3 mg/dL)
- Sodium: 133 (136–145 mmol/L), potassium: 3.0 (3.5–5.1 mmol/L)
- Aspartate aminotransferase: 663 (0–38 U/L), alanine transaminase: 317 (0–41 U/L)
- Sensitive thyroid-stimulating hormone: 0.69 (0.27–4.2 µU/mL)
- Chest radiograph: Normal
- High-resolution computed tomography (HRCT) of the thorax with dynamic tracheal imaging: Normal tracheal appearance seen during inspiration [Figure 1]. A typical crescentric tracheal appearance with >50% narrowing of lumen s/o TM seen during expiration [Figure 2]. Other findings include centrilobular emphysematous cyst in anterior segment of right lower lobe.

On initial clinical evaluation, the diagnosis of acute gastroenteritis with sepsis, prerenal acute kidney injury and upper respiratory infection was considered. The diagnosis of sepsis was confirmed by elevated inflammatory markers and growth of *Escherichia coli* in blood culture. She was treated in Intensive Care Unit (ICU) with appropriate antibiotics. Tracheal pathology was suspected in view of history of chronic cough and previous surgery. The diagnosis of TM was made after HRCT thorax with dynamic tracheal imaging and in consultation with the respiratory physician. She ideally needed bronchoscopy, followed by tracheoplasty or stenting, but due to her morbid condition, conservative treatment with cough suppressant was chosen. She received influenza vaccination also.

**Discussion**

TM refers to loss of tracheal rigidity and resulting susceptibility to collapse. It may be diffuse or localized to a tracheal segment. The affected portion may be intrathoracic, in which airway obstruction is accentuated during expiration. Less common is extra thoracic obstruction, in which airway obstruction is most marked during inspiration. Tracheobronchomalacia is the term used to describe the condition when the mainstem bronchi are involved.

TM in adults may be classified as congenital or acquired. The congenital form, described more extensively in children, is related to a variety of congenital disorders e.g. mucopolysaccharidosis. The disorder may persist into adult life and is referred to as “idiopathic giant trachea,” “tracheomegaly,” or the “Mounier–Kuhn syndrome.” Bronchiectasis and recurrent respiratory infections are common. Although atrophy of the longitudinal elastic fibres and muscularis layer has been described, the aetiology of these changes is unclear.[1]

Acquired or secondary TM in adults is most common in men who are over 40 years of age. It may be related to a variety of conditions [Table 1].[2] Tracheostomy and endotracheal intubation are probably the most common aetiologies.[3] Risk factors include recurrent intubation, prolonged intubation and concurrent high-dose steroid therapy. It usually causes limited focal weakness of the trachea and dynamic airway obstruction. TM may be caused by conditions that are associated with chronic pressure on the

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**Table 1: Causes of acquired tracheomalacia**

| Condition                          |
|-----------------------------------|
| Posttraumatic                     |
| Postintubation                     |
| Posttracheotomy                    |
| External chest trauma             |
| Postlung transplantation           |
| Emphysema                         |
| Chronic infection/bronchitis      |
| Chronic inflammation              |
| Relapsing polychondritis          |
| Chronic external compression of the trachea |
| Malignancy                        |
| Benign tumours                    |
| Cyst                              |
| Abscesses                         |
| Aortic aneurysm                   |
| Vascular rings, previously undiagnosed in childhood |

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![Figure 1: Normal tracheal appearance during inspiration](image1)

![Figure 2: A typical crescentric narrowing of trachea during expiration](image2)

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tracheal wall (benign mediastinal goitre, malignancy, abscess or cyst), inflammation of the cartilaginous support or mucosa or interference with tracheal blood flow, exposure to mustard gas and gastroesophageal reflux disease. Traumatic injury to the central airways or surgical interventions may also lead to TM. In our case, the most likely aetiology is damage to cartilaginous wall of trachea by chronic inflammation.

TM should be suspected in patients with a history of chronic cough, sputum production and dyspnoea, not responding to symptomatic treatment. Wheezing and stridor may be present in patients with significant airway obstruction. The diagnosis is done by direct bronchoscopic visualization to confirm significant narrowing of the tracheal lumen during regular, forced expiration. Assessment of the central airways using end‑expiratory, dynamic, three‑dimensional computed tomography images is useful. The diagnosis is made when the diameters of the trachea or right or left mainstem bronchi exceed the upper limits of normal by three or more standard deviations.[3] It can also be defined as mild, if the lumen narrows to 50% of its initial size during expiration, moderate if it narrows to 25% of its initial size and severe if the anterior and posterior walls touch. [2] Other diagnostic methods may include pulmonary function test and chest radiography.

Management can be medical or surgical. Asymptomatic patients generally do not require therapy. Initial treatment of symptomatic patients includes optimization of underlying conditions like chronic obstructive pulmonary disease, tracheal stenosis or associated infections. A functional assessment is performed (e.g., pulmonary function tests, 6‑min walk test) in symptomatic patients to establish a baseline. Application of continuous positive airway pressure has been reported as beneficial. Other treatment options are tracheal silicone stenting or surgical repair. Silicone stents are inserted by rigid bronchoscopy and under general anaesthesia. It provides immediate improvement, but often migrates, which may manifest as a new cough.[4] The surgical methods include tracheobronchoplasty, conventional resection and reconstruction or tracheal replacement.

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Conflicts of interest
There are no conflicts of interest.

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