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Asymptomatic Tracheobronchopathia Osteochondroplastica: A Case Report at the Akanda Army Training Hospital of Gabon

Hilda Birinda[1*], Rana Farelle Atome Minang[1], Yannick Armand Ivala Mendome[1], Aude Jessica Atoume N'Noume[1], Raphael Okoue Ondo[2], Romain Tchoua[2], Jean Marcel Mandji Lawson[1]

[1]Department of Emergency Anesthesia Resuscitation, Akanda Army Training Hospital, Libreville, Gabon
[2]Department of Emergency Anesthesia Resuscitation, Omar Bongo Ondimba Army Training Hospital, Libreville, Gabon
Email: *hildabirinda@gmail.com, ranaatome@gmail.com, yazface@yahoo.fr, atoume89@yahoo.fr, raphaelokoue@gmail.com, mandji@live.fr, rtrtchoua@yahoo.fr

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Abstract

Tracheobronchopathia osteochondroplastica is an idiopathic condition affecting the tracheobronchial tree. It may be asymptomatic or manifest as respiratory symptoms. We describe here a case of an incidental finding within hours of admission in a patient with multiple comorbidities who was initially managed for vascular leukoencephalopathy. Although Tracheobronchopathia osteochondroplastica is a rare condition, it should be suspected based on medical imaging results.

Keywords

Tracheobronchopathia Osteochondroplastica, Thoracic Computed Tomography, Bronchial Fibroscopy, Rare Disease

1. Introduction

Tracheobronchopathia osteochondroplastica is a non-malignant condition characterized by the presence of nodules of varying sizes and numbers protruding into the lumen of the trachea and bronchi [1] [2]. These nodules are generally located on the anterolateral wall of the tracheobronchial tree, which is composed of cartilaginous rings. The posterior wall, made of connective and muscular tissue, is spared [3]. Often diagnosed in adulthood, tracheobronchopathia osteochondroplastica affects both men and women. Its incidence is thought to be underestimated due to its generally rare diagnosis and nonspecific respiratory symptoms. The condition can progress to stenosis or obstruction of the airways [3]-[5].

2. Case Report

This case report concerns a 68-year-old woman, Mrs. MM, a retired nurse, with modifiable cardiovascular risk factors: type 2 diabetes treated with insulin therapy, hypertension treated with dual therapy (amlodipine-valsartan), complicated by end-stage renal disease, and hemodialysis three times a week for the past seven years. This patient was initially admitted for a non-febrile confusional state that had been progressively developing for one month. The family reported back pain associated with lower limb muscle weakness that had been progressing for several weeks. The family history revealed no episodes of hemoptysis, chronic cough, recurrent respiratory infections, or wheezing.

The neurological examination revealed a Glasgow Coma Scale score of 12/15, with impaired verbal response, flaccid tetraparesis, a positive Hoffman sign, and negative deep tendon reflexes in all four limbs, suggestive of peripheral neuropathy. No sphincter dysfunction was noted. The pleuropulmonary and other system examinations were normal. A brain CT scan is consistent with chronic vascular leukoencephalopathy. Blood sugar and sodium levels are normal. The calcium level was 2.62 mmol/L. In addition, a mild biological inflammatory syndrome is noted, with C-reactive protein at 20.36 mg/L. A chest X-ray, performed as part of the standard workup, shows a calcified appearance of the tracheobronchial wall (Figure 1).

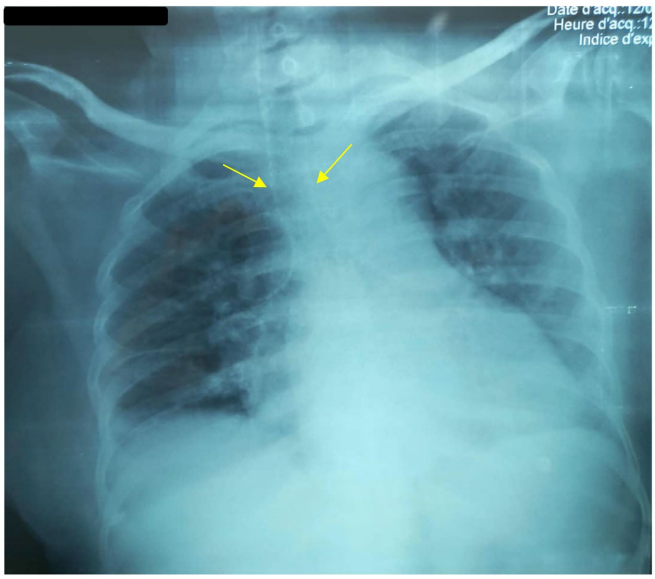

Figure 1. Chest X-ray (frontal view) showing calcification of the tracheobronchial wall (yellow arrows).

A CT scan is performed to complement the chest X-ray. It reveals, firstly, an increase in bone density of the tracheobronchial cartilage, which is irregular and beaded, without reduction of the lumen (Figure 2), sparing the posterior wall (Figure 3).

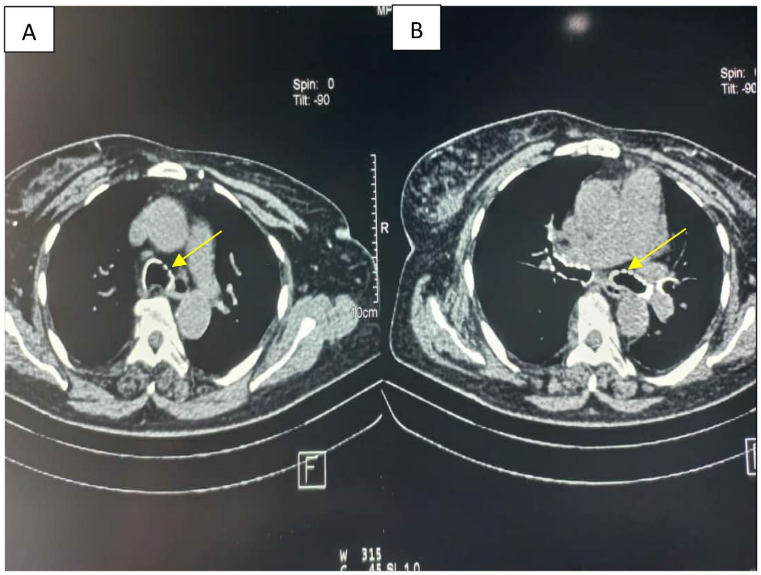

Figure 2. Thoracic CT scan showing calcified nodules of the thickened tracheobronchial tree bordering the anterolateral wall on images (A) and (B) (yellow arrows).

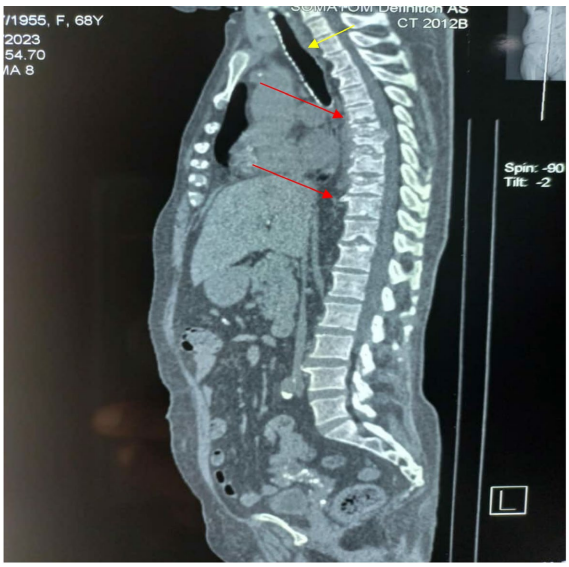

Figure 3. Sagittal CT scan of the spine showing the posterior wall of the trachea spared by calcified nodules (yellow arrow) and degenerative lesions of the spine (red arrows).

Furthermore, it demonstrates, in the thoracic region, multilevel disc and vertebral destruction including vertebral compression fractures from T7 to T9, disc narrowing and subchondral erosions, and lysis of the posterior wall of T6, T7, and T9, without ruling out a potentially life-threatening spinal cord injury. In the lumbar region, subchondral erosions of the vertebral endplates are noted, more pronounced in L3. At the level of L4 - L5, a non-conflicting disc protrusion is found (**Figure 3**). Hormonal testing revealed an elevated parathyroid hormone level (279.8 pg/ml). The secondary diagnosis was post-hemodialysis erosive spondylitis with suspected spinal cord injury. The patient was referred to a neurosurgical unit

for initial management. However, bronchoscopy with biopsy for histopathological analysis could not be performed to definitively diagnose.

3. Discussion

Tracheobronchopathia osteochondroplastica was first identified and described in the mid-19th century. The first microscopic description reported ossified deposits in the larynx, trachea, and bronchi of a 38-year-old man who died of pulmonary tuberculosis [6] [7]. Over time, it has emerged as a rare condition affecting men and women equally. It is often diagnosed in adulthood, and only exceptionally in children [8] [9]. Although some cases have been described with severe respiratory symptoms such as dyspnea or hemoptysis [4] [5], diagnosis is predominantly made in asymptomatic patients. With an estimated incidence of 0.11%, the prevalence of tracheobronchopathia osteochondroplastica is relatively low [4] [10]. The exact etiology is unknown. Several theories exist regarding the pathogenesis, including ecchondrosis and exostosis of cartilaginous rings, and metaplasia of elastic tissue [7] [10]-[13]. There is no formal evidence concerning genetic susceptibility or the role of smoking [10]. Associations with chronic inflammatory conditions, chemical or mechanical irritation, and degenerative or metabolic abnormalities have been described [10] [12]. This observation illustrates the case of asymptomatic tracheobronchopathia osteochondroplastica in a woman in her sixties. It occurred in a context of multiple pathologies involving parathyroid hormone hypersecretion secondary to chronic renal failure. This hypersecretion sustains the phenomenon of vertebral bone demineralization and causes the degenerative lesions found on the spinal CT scan (**Figure 3**). Even though situations of increased parathyroid hormone levels can lead to tracheal calcifications, no formal link has been established between tracheobronchopathia osteochondroplastica and phosphocalcic metabolism disorders [14].

There are numerous cases where tracheobronchopathia osteochondroplastica has also been diagnosed incidentally [15]. The discovery is generally made either during bronchoscopy or during a computed tomography examination, as was the case for this patient. Bronchoscopic examination with biopsy confirms the diagnosis through the histopathological examination of the collected tissues. The biopsies reveal the abnormal presence of cartilage and bone tissue in the tracheal and bronchial submucosa. In this clinical case, bronchoscopy was not possible. However, imaging exams suggested a diagnosis of tracheobronchopathia osteochondroplastica. On chest X-ray, the diagnosis is suspected in the presence of an irregular, beaded appearance of the tracheal or bronchial wall related to calcifications. This feature is found in 11% of cases [16]. Chest CT scan reveals the presence of multiple calcified nodules, typically sparing the posterior wall of the trachea and projecting into the proximal tracheal or bronchial lumen [17]. These characteristics are unique and consistent with those described (**Figure 2** and **Figure 3**). This CT scan description is considered pathognomonic according to some authors [18]. The nodules, 1 to 3 mm in size, cross the airway lumen and cause

thickening of the tracheobronchial wall [1]. Patients with a life-threatening progression have been reported. Osteocartilaginous nodules originating from the tracheobronchial cartilage rings can cause stenosis or obstruction of the airways [4] [10] [11] [19] [20]. Cases of tracheal stenosis have been treated with laser therapy [21]. There is no clearly established consensus on management. Treatment depends on the presentation of the disease. However, this disease is benign, and in most of the cases described, it progresses slowly [22].

The absence of specific signs explains why this pathology was not detected earlier in this patient. On the other hand, its association with an erosive spondyloarthropathy, potentially linked to a spinal cord lesion, was a limiting factor in achieving a definitive diagnosis of tracheobronchopathia osteochondroplastica. Priority was given to the treatment of degenerative bone lesions that could compromise the neurological prognosis. This clinical case reveals that imaging signs, even subtle ones, can significantly strengthen the diagnostic suspicion of tracheobronchopathia osteochondroplastica [1] [4]. To achieve this, careful examination of the tracheobronchial tree on imaging scans deserves attention in routine practice.

4. Conclusion

This case report aims to help clinicians recognize imaging features as quickly as possible, especially in asymptomatic patients. Given that tracheobronchopathia osteochondroplastica is a rare, slowly progressing disease, its bronchoscopic diagnosis is at risk of being delayed due to an underlying urgent condition.

Informed Consent

Informed verbal consent was obtained from the legal guardian for the publication of this clinical case and the associated images.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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