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Thickening of the tracheal wall

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A 63-year-old female patient presented with a 1-year history of recurrent polyarthritis and a 3-month history of dry cough. Her laboratory test results were normal. Physical examination revealed deformity in both ears, with inflammatory signs. A chest CT showed diffuse thickening of the walls of the trachea and main bronchi (Figure 1). Her lung parenchyma was normal.

Diffuse thickening of the tracheal wall has a large number of possible etiologies—amyloidosis; relapsing polychondritis (RP); tracheopathia osteochondroplastica (TPO); infections, such as tuberculosis, paracoccidioidomycosis, and rhinoscleroma; granulomatosis with polyangiitis; sarcoidosis; lymphomas; etc. Some imaging features can be useful in narrowing the differential diagnosis, such as the presence of calcifications, and define whether the entire tracheal circumference is affected or whether the lesion spares the posterior membranous wall, affecting only the cartilaginous portion. In the case presented here, two CT features are of note: the wall thickening has calcifications in its entire longitudinal extension; and the posterior membranous portion of the tracheal wall is spared.

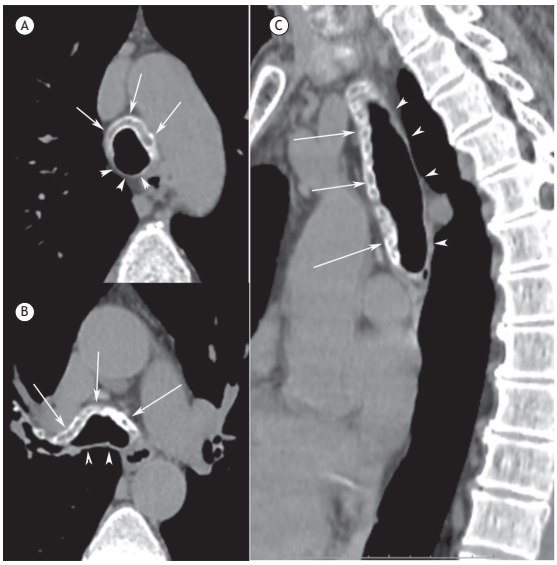

Figure 1. Axial CT slices (in A and B) and coronal CT reconstruction (in C) showing diffuse thickening of the anterior wall of the trachea and main bronchi, with calcifications (arrows). Note that the posterior wall (arrowheads) is spared.

Tracheal wall calcifications can be observed in healthy patients, being related to senility. However, calcifications associated with wall thickening can be seen in amyloidosis, TPO, and RP. In amyloidosis, involvement is circumferential, also affecting the posterior membranous wall. Therefore, in the case presented here, the differential diagnosis is restricted to two diseases: TPO and RP.

TPO is a disease of unknown etiology, characterized by the formation of small, usually calcified submucosal nodules protruding into the tracheal lumen. The disease restricts itself to the tracheobronchial tree. It may be asymptomatic, or it may result in cough, dyspnea, wheezing, or, occasionally, hemoptysis.

RP is characterized by recurrent, potentially severe episodes of inflammation of cartilaginous tissues, including the cartilage of the ear, nose, peripheral joints, and tracheobronchial tree. Airway symptoms include progressive dyspnea, cough, stridor, and hoarseness, which are due to destruction and fibrosis of the cartilaginous rings of the larynx and trachea, leading to luminal collapse, and also to airway narrowing caused by inflammation and cicatricial fibrosis.

Our patient had, in addition to characteristic tracheobronchial changes on chest CT, a seronegative arthropathy and auricular chondritis. On the basis of these elements, a diagnosis of RP was made. In most cases, biopsy is not necessary for diagnostic confirmation.

RECOMMENDED READING

1. Marchiori E, Pozes AS, Souza Junior AS, Escuissato DL, Irion KL, Araújo Neto Cd, et al. Diffuse abnormalities of the trachea: computed tomography findings. J Bras Pneumol. 2008;34(1):47-54. https://doi.org/10.1590/S1806-37132008000100009

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