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ACUTE LARYNGEAL DYSPNOEA REVEALING ANTERIOR CERVICAL HYPEROSTOSIS (FORESTIER'S DISEASE)

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ABSTRACT

Forestier's disease, or diffuse idiopathic spinal hyperostosis, can exceptionally lead to acute obstruction of the upper airways when the hyperostosis is located in the cervical region. We report the case of an 87-year-old man admitted for acute inspiratory dyspnoea and progressive dysphonia. Endoscopic examination confirmed left laryngeal paralysis and pharyngolaryngeal oedema. Imaging revealed a large anterior hyperostosis compressing the airway. This case illustrates the interaction between mechanical compression, local inflammation, and chronic laryngeal paralysis, which can lead to severe dyspnoea. Early recognition and appropriate imaging allow for rapid and effective management.

KEYWORDS

Diffuse idiopathic skeletal hyperostosis, airway obstruction, laryngeal nerve paralysis, dyspnoea, cervical vertebrae

LEARNING POINTS

- Cervical hyperostosis can cause acute laryngeal dyspnoea through mechanical compression and associated oedema.
- The combination of an acute inflammatory process and pre-existing recurrent paralysis exacerbates the severity of the clinical picture.
- Cervicothoracic imaging and nasofibroscopy are essential for establishing the diagnosis and guiding management.

INTRODUCTION

Forestier's disease, also known as diffuse idiopathic spinal hyperostosis (DISH), is a degenerative condition characterized by progressive ossification of the ligaments, particularly the anterior longitudinal ligament. It is a rare and little-known condition, with few descriptions in the scientific literature. Although it is usually asymptomatic, its cervical involvement can cause various compressive symptoms such as dysphagia, dysphonia, or snoring. In rare cases, pharyngolaryngeal compression can become severe enough to cause acute, life-threatening dyspnoea. Here we describe a rare case of upper airway obstruction associated with significant anterior cervical hyperostosis in an elderly patient.

CASE DESCRIPTION

An 87-year-old Caucasian man of European origin was treated for progressive inspiratory dyspnoea accompanied

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by recent-onset dysphonia. He had initially been hospitalized following a fall without major trauma. His medical history included high blood pressure, anxiety and depression, gout, lower limb arteriopathy with medial calcification, and severe snoring. He did not have a history of alcohol or tobacco abuse. He had undergone total hip replacement and total knee replacement surgery. On admission, the patient presented with inspiratory dyspnoea with stridor, marked dysphonia, and orthopnoea. There were no signs of vascular overload, and the biological level of the NTPROBNP marker was normal at 233 pg/ml (laboratory standard: <300 pg/ml). The oral cavity was normal on examination. Nasofibroscopy revealed significant oedema of the pharyngolaryngeal structures, bulging of the posterior pharyngeal wall, and paralysis of the left vocal cord. The glottis was narrowed and difficult to visualize, suggesting significant partial obstruction. A cervical spine computed tomography (CT) scan with contrast showed extensive anterior hyperostosis mainly at the C2–C3, C4–C5, and C5–C6 levels, resulting in a marked reduction in the airway *(Fig. 1 and 2)*. Extensive uncodisc-arthrosis, disc bulging, and thickening of the prevertebral soft tissues were also observed. A CT scan of the cervical and thoracic spine confirmed thickening of the left ventricular wall and narrowing of the glottic airway. The cervical magnetic resonance imaging (MRI) ruled out spondylodiscitis and confirmed the extent of degenerative changes and anterior hyperostosis. Lumbar MRI ruled out any sacroiliac involvement characteristic of ankylosing spondylitis.

Emergency treatment for this respiratory distress combined intravenous corticosteroid therapy with methylprednisolone, adrenaline aerosols, and inhaled budesonide. The patient's condition improved rapidly, with a noticeable reduction in dyspnoea and dysphonia. Follow-up nasofibroscopy showed significant regression of the oedema and improved visualization of the glottis. The left laryngeal paralysis was interpreted as a chronic phenomenon.

DISCUSSION

Forestier's disease, or senile ankylosing hyperostosis of the spine, is a rheumatological disease of unknown aetiology first described in 1950 by Forestier and Rotes-Querol[1]. It is characterized by ossification of the paravertebral ligaments and peripheral enthesis. Most often asymptomatic, DISH is discovered incidentally on standard X-rays. More rarely, it manifests as neck pain, spinal stiffness, or dysphagia[2]. DISH is the second most common cause of ventral cervical osteophytes after degenerative conditions, with a prevalence of 2 to 4% in patients over 40 years of age, reaching up to 28% in those over 80 years of age[3]. Although DISH has been reported more frequently in certain ethnic groups—particularly in Ashkenazi Jewish and Japanese populations—its occurrence in European patients is well documented, especially in elderly men[4]. In this case, the patient's ethnicity did not represent a specific risk factor beyond the known associations with age and male sex. The pathogenesis of this disease remains poorly understood. Several studies have explored the associations between DISH and trauma, as well as endocrine and metabolic diseases, but the strongest correlations observed are related to gender and age[5]. The epidemiological profile is marked by a strong male predominance and a frequent association with metabolic disorders (obesity, type 2 diabetes, dyslipidaemia), described



Figure 1. Sagittal computed tomography scan of the cervical spine (bone window) showing massive anterior osteophytes of the cervical vertebrae, forming a continuous bony ridge consistent with diffuse idiopathic spinal hyperostosis.

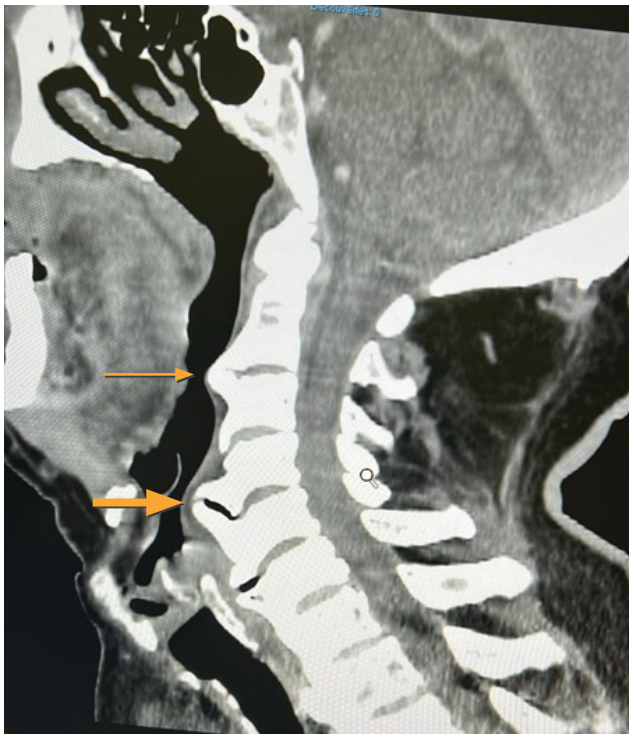


Figure 2. Sagittal computed tomography scan of the cervical spine (bone window). Slice showing voluminous anterior hyperostoses from C3 to C6, characteristic of diffuse idiopathic spinal hyperostosis, with marked reduction of the retropharyngeal space.

in particular by Mader et al.[6]. The exact pathophysiology of DISH remains poorly understood. Several authors suggest a contributory role for hormonal and metabolic factors, particularly hyperinsulinism and IGF-1, consistent with the strong association reported between DISH and metabolic syndrome[7]. He and Chen also highlight a consistent correlation with male sex, advanced age, and cardiovascular comorbidities[8].

When the cervical spine is affected, the symptoms can become severe. The literature shows that C3–C5 levels are most often responsible for dysphagia or compression of the hypopharynx, while lower osteophytes (C5–C7) can cause paresis or paralysis of the vocal cords by compressing the recurrent laryngeal nerve[8,9]. Swallowing disorders affect up to 28% of patients with cervical DISH, of whom 8–10% may require surgical management[10].

Furthermore, the size of the osteophytes plays a decisive role: a thickness greater than 10–12 mm is strongly correlated with swallowing disorders and the risk of aspiration[11]. Finally, compressive symptoms can range from simple pharyngeal discomfort to acute respiratory distress or cord paralysis, as highlighted by Pruijn et al.[9] and as evidenced by our reported clinical case.

The diagnosis is essentially radiological, based on the criteria established by Resnick & Niwayama[12], namely:

1. "Flowing" ossification of the anterior longitudinal ligament on at least four contiguous vertebrae;
2. Preservation of disc spaces;
3. Absence of involvement of the sacroiliac joints or facet joints.

CT scan allows for accurate assessment of the degree of laryngopharyngeal compression and remains the gold standard preoperative examination.

All authors emphasize the need to rule out severe ear, nose, and throat (ENT) diagnoses such as hypopharyngeal tumour, Zenker's diverticulum, retropharyngeal infection, or spondylodiscitis, conditions that can mimic the symptoms of cervical DISH[7].

In the absence of respiratory distress, first- line of treatment is conservative:

- Dietary adjustments;
- Speech therapy (swallowing, cervical posture);
- Nonsteroidal anti-inflammatory drugs or short-acting corticosteroids[5,8,10].

Mesolella et al.[12] show that with intensive speech therapy, combined with anti-inflammatory treatment (indomethacin), surgery can sometimes be avoided even in the presence of severe dysphagia.

In the long term, patients with symptomatic cervical DISH may require periodic ENT and rheumatological follow-up to monitor potential recurrence of compressive symptoms. Conservative management—including dietary adaptation, speech therapy, and intermittent anti-inflammatory therapy—may be sufficient in stable cases. However, if symptoms recur or progressive airway or oesophageal compression develops, anterior cervical osteophytectomy may be considered, as it remains the definitive treatment in cases of persistent dysphagia, recurrent dyspnoea, or vocal cord dysfunction[3,12].

When symptoms persist or worsen, or in cases of dyspnoea/stridor, anterior cervical osteotomy is indicated. Postoperative results are generally excellent: the literature reports an improvement in dysphagia in more than 90% of cases[9]. However, radiological recurrence is possible, as shown by Miyamoto et al., with almost constant osteophytic regrowth in the long term, even if symptomatic recurrence remains rarer[13].

CONCLUSION

DISH should be recognized as a possible—albeit rare and often underestimated—cause of dysphagia, dysphonia, and laryngeal dyspnoea in older individuals. The presence of massive anterior cervical osteophytes in direct contact with the aerodigestive junction in our patient allowed us to link the laryngeal symptoms to mechanical compression exerted by vertebral hyperostosis, rather than to a tumour or infectious lesion. This case illustrates the importance of a structured diagnostic pathway, combining a complete ENT examination, endoscopy of the upper aerodigestive tract, and cervical imaging (X-rays, CT scan, possibly MRI). This approach remains essential in order to rule out neoplasia of the larynx, hypopharynx, or oesophagus, whose clinical presentation may be nonspecific and confused with that of cervical DISH. Management must be multidisciplinary, involving an otolaryngologist, rheumatologist, radiologist, possibly an orthopaedic surgeon or neurosurgeon, and a speech therapist. When symptoms are moderate and do not pose a respiratory threat, conservative treatment including dietary adjustments, speech therapy, and anti-inflammatory treatment may be sufficient. However, in cases of severe dysphagia, weight loss, stridor, or laryngeal dyspnoea, anterior cervical osteotomy is an effective treatment option, with rapid and lasting improvement in swallowing and respiratory symptoms in the majority of published series.

Cervical DISH should therefore be included in the systematic differential diagnosis of swallowing disorders and upper dyspnoea in elderly patients, in order to provide early and appropriate care that can prevent potentially life-threatening acute complications.

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