



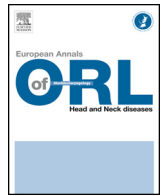
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What is your diagnosis?

Laryngeal dyspnoea and COVID-19

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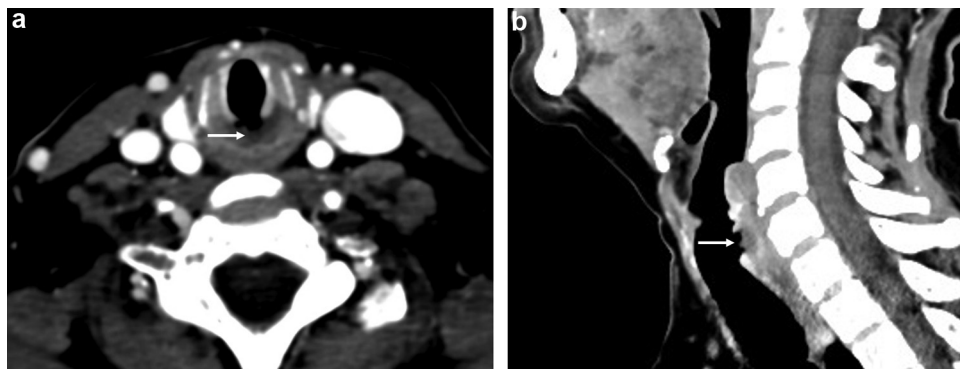


Fig. 1. Axial (a) and sagittal (b) contrast-enhanced head and neck CT scan demonstrated a posterior subglottic mucosal ulceration measuring $17 \times 9 \times 15$ mm, associated with chondrolysis of the inferior part of the cricoid cartilage (white arrow).

1. Case report

A 69-year-old woman was referred to the emergency room with inspiratory dyspnoea that had progressively worsened over 6 days. One month previously, she had experienced an episode of severe SARS-CoV2 pneumonia (PCR-positive) complicated by acute respiratory distress syndrome requiring 7 days of intubation in the intensive care unit with 48 hours in the prone position, followed by 7 days of conventional hospitalization. This woman's history included coronary artery stenosis treated by angioplasty with drug-eluting stent, hypertension and type 2 diabetes. On arrival in the emergency room, she was afebrile but presented dyspnoea during speech with a two-toned voice and a respiratory rate of 12. Clinical examination of the neck and oral cavity did not reveal any abnormality. Nasolaryngoscopy revealed glottic and subglottic oedema with a necrotic appearance of the subglottic mucosa below the posterior commissure. Bilateral cricoarytenoid hypomobility was

also observed. C-reactive protein and complete blood count were normal and SARS-CoV2 PCR was negative. In this context of respiratory emergency, systemic corticosteroid therapy at a dosage of 1 mg/kg daily was initiated, together with corticosteroid inhaler. Clinical improvement was rapidly observed. Follow-up nasolaryngoscopy showed improvement of glottic and subglottic oedema allowing transfer to the supine position. Contrast-enhanced head and neck CT scan was then performed (Fig. 1), followed by upper airway endoscopy to confirm the diagnosis and to obtain histological and microbiological samples. Following this assessment, empirical antibiotic therapy with amoxicillin/clavulanic acid (intravenous Augmentin® at the dose of 1 g t.i.d.) was initiated immediately after taking samples, followed by oral antibiotics 48 hours later.

What is your diagnosis?

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2. Answer

The clinical history is suggestive of cricoid chondritis due to the combination of recent intubation and persistent glottic and subglottic oedema in an afebrile patient. CT scan showed a posterior subglottic mucosal ulceration measuring $17 \times 9 \times 15$ mm, associated with chondrolysis of the inferior part of the cricoid cartilage, confirming the diagnosis of cricoid chondronecrosis. Histological examination of endoscopic samples demonstrated simple oedematous and inflammatory changes with no signs of carcinoma. Mycological and mycobacteriological samples were sterile. Bacteriological culture showed a few rare colonies of commensal micro-organisms (*Parvimonas micra*, *Atopobium rima*, *Streptococcus constellatus*, *Streptococcus anginosus*) and *Staphylococcus aureus*. In view of the patient's clinical improvement, it was decided not to modify antibiotic therapy despite the presence of a methicillin-resistant *Staphylococcus aureus*, after consultation with the infectious disease specialists, who considered that the presence of *Staphylococcus aureus* in endoscopic samples corresponded to chronic non-pathogenic bacterial carriage related to her intensive care unit stay, for which they did not recommend any specific antibiotic therapy. The diagnosis of aseptic chondronecrosis of the cricoid cartilage was therefore adopted. Subglottic trauma after endotracheal intubation is relatively common, but cricoid chondronecrosis is rare and associated with an uncertain prognosis. The duration of intubation is a recognized risk factor, but the diagnosis should be considered even after brief intubation (less than 7 days), in the presence of progressive onset of post-extubation laryngeal dyspnoea following a dyspnoea-free interval [1]. Chondronecrosis is generally considered to be due to ulceration of the mucous membrane over the cricoid cartilage due to compression by the endotracheal tube cuff, resulting in impaired blood supply of the posteroinferior laryngeal pedicle [2]. This mechanism leads to perichondritis followed by chondritis and even necrosis of the cricoid cartilage [3]. Clinically, this lesion is responsible for laryngeal dyspnoea associated with glottic and subglottic oedema with contiguous bilateral impaired vocal cord mobility due to extension of oedema to the cricoarytenoid joint, accompanied by an infiltrated or even necrotic appearance of the subglottic mucous membrane with the presence of inflammatory granulomas. Contrast-enhanced CT scan of the neck and chest should be performed whenever this diagnosis is suspected [4]. CT features vary according to the extent of the lesion and usually show demineralization of the cricoid cartilage, which has a hypodense appearance [4]. Other characteristic radiological signs have also been described in the literature: (i) chondrolysis with fragmentation of the cartilage and disruption of the cartilage ring, (ii) cartilage sclerosis, and (iii) oedema of adjacent soft tissues [4].

Management is medical and surgical. No standard protocol has been defined and treatment varies according to the severity of chondronecrosis. Endoscopy is performed routinely to confirm the diagnosis based on detailed examination of the upper airways and collection of deep microbiological and histological samples. Endoscopy may be accompanied by surgery to improve

the laryngeal airway when medical treatment has not allowed any respiratory improvement. Tracheostomy should be preferred to posterior cordotomy or arytenoidectomy to avoid any further laryngeal mutilation. Placement of a laryngotracheal stent, such as Montgomery's tube, may sometimes be necessary [3]. However, this type of stent is associated with a risk of acute obstruction due to tube torsion. When the stent protrudes beyond the arytenoid cartilages, it can also cause pain or aspiration. The use of laryngotracheal stents is therefore reserved for highly specialized centres. In terms of medical treatment, there is a general consensus in the literature concerning the major role of systemic corticosteroid therapy at a dose of 1 mg/kg daily for 7 days together with antibiotic therapy adapted to culture results [2]. However, the real impact of these therapies on the course of the disease remains unknown. In particular, antibiotic therapy may have been of limited value in our case due to the presence of non-pathogenic micro-organisms or simple colonization on microbiological specimens. However, exposed cartilage with signs of chondritis is at high risk of becoming infected despite the initial absence of infection. Some teams may therefore prefer a more interventionist approach to antibiotic therapy. Empirical antibiotic therapy is often administered in the presence of early signs of cricoarytenoid arthritis to prevent progression to full-blown chondritis. Two types of chondronecrosis of the cricoid cartilage can therefore be distinguished: septic and aseptic [5]. Corticosteroid therapy alone may also predispose to extension of septic chondronecrosis and should therefore only be proposed after contrast-enhanced CT scan of the neck and chest and upper airway endoscopy [2]. Since the beginning of the COVID-19 pandemic, corticosteroid therapy must be reserved for severe clinical forms of the various conditions in which it has been clearly established to allow rapid improvement of symptoms [6]. Due to the sudden and massive increase in the number of patients urgently intubated in intensive care units, sometimes repeatedly, as a result of rapid clinical deterioration, an increased incidence of cricoid chondronecrosis could be observed in the context of the COVID-19 pandemic.

Disclosure of interest

The authors declare that they have no competing interest.

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