Hyperbaric oxygen treatment (HBOT) in a case of traumatic chondronecrosis of the cricoid cartilage

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Abstract

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Cricoid chondronecrosis is a rare entity and is scarcely reported in the literature. Its prevalence is increasing in the form of chondroradionecrosis among the survivorship of head and neck carcinoma patients treated with radiotherapy. We have reported a case of cricoid chondronecroisis caused by trauma from repeated tracheostomy. The patient presented with hoarseness and dyspnoea. Radiological findings in multidetector computed tomography showed disintegration of the cricoid and confirmed the diagnosis. Conservative treatment was given in the form of antibiotics, steroids and nebulised anticholinergics and bronchodilators. However, the patient did not improve and his condition worsened throughout two months of hospitalisation. He was referred for hyperbaric oxygen treatment, which was given over 30 sessions. This was associated with improvement in his condition and he was able to be decannulated from tracheostomy. Six monthly follow up of the patient showed a well-healed tracheostomy scar.

Introduction

Traumatic chondronecrosis is a rare consequence of cricoid trauma.[1] Trauma to the cartilage either from direct assault or during airway management can lead to chondronecrosis, which is difficult to manage.[2] Treatment typically consists of the use of antibiotics, steroids and surgery in severe life-threatening conditions, but prognosis remains poor.[1,2] Hyperbaric oxygen treatment (HBOT) has occasionally been reported as effective in cases failing to heal with other interventions. We report a case of traumatic chondronecrosis of the cricoid in which HBOT was used after the condition failed to improve with antibiotics and steroids.

Case report

The patient gave written consent for publication of his case details and medical images.

A 31-year-old male presented to our centre with difficulty breathing and hoarseness of voice. He had been injured in a hand grenade blast while participating in a military training activity eight months previously. He suffered from a severe head injury, his left thumb was blown away, and he also sustained a comminuted fracture base of the first metacarpal of the same hand. After initial stabilisation at the nearby hospital, he was transferred to a tertiary care hospital. His definitive management included right decompressive hemicraniectomy and surgical debridement of the left hand under general anaesthesia. On the sixth day of surgery, he developed difficulty in breathing due to poor cough reflex and retention of secretions. His oxygen saturation was decreasing and he was managed with a planned percutaneous tracheostomy, which remained *in situ* for five days. After stabilisation, decannulation of his tracheostomy was performed. The patient remained in the hospital for another two weeks for observation. Once, he appeared stable without any active intervention, he was discharged from the hospital for recovery. During the convalescent period at home, he developed acute breathlessness and again reported to a hospital near his home. He was diagnosed with acute laryngotracheobronchitis and managed conservatively with medication. He responded well and fully recovered.

After two months, the patient again developed severe breathlessness and hoarseness. An emergency tracheostomy procedure was performed with 8.0 mm inner diameter, single lumen, cuffed, non-fenestrated tracheostomy tube. After 10 days, the tracheostomy tube was changed to a 7.0 mm inner diameter, double lumen, fenestrated tube. Pre- (without contrast) and post- (with contrast) multidetector computed tomography (CT) views of the neck were obtained from the base of the skull to the carina. This revealed fragmentation of the cricoid cartilage lamina predominantly along the

posterior aspect with extensive soft tissue component causing partial airway narrowing (Figure 1). Contrast media was used for better visualisation of the soft tissue. Based on the above findings he was diagnosed with chondronecrosis of the cricoid cartilage. He was managed with antibiotics, corticosteroids and nebulisation, for around three months.

The hoarseness and dyspnoea did not improve with the medication and his clinical condition deteriorated over the three months while the tracheostomy tube was in situ. He was referred to our centre for HBOT, given the poor wound healing even after adequate treatment. Problem wounds are an approved indication for HBOT per the Undersea and Hyperbaric Medical Society (UHMS). After ascertaining fitness for HBOT, a trial session was given at 243 kPa (2.4 atmospheres absolute) for 60 minutes without an air break. For the purposes of treatment, the tracheostomy tube was temporarily closed and HBOT was given via a face mask. A hyperbaric physician remained in the chamber with the patient in case of any complications arising from closure of the tracheostomy. The patient tolerated the trial session without any complaints or complications. During HBOT, antibiotics were stopped and steroids were tapered off slowly. Tracheostomy care was given simultaneously with the ongoing HBOT to avoid late complications of the tracheostomy. Although HBOT can cause acute complications in terms of barotraumas, oxygen toxicity and chronic complications like pulmonary changes and cataracts none of these problems were observed.

Breathing started becoming comfortable at the 8th to 10th session of HBOT and dyspnoea improved at around the 22nd session. Decannulation was successfully performed after the 26th session but HBOT was continued for six days a week for a total of 30 sessions. The hoarseness improved significantly during and after HBOT. Indirect laryngoscopy after the completion of 30 sessions of HBOT showed oedema subsidence in the glottis and subglottic area, a decrease in the size of granulation tissue, and a reversal of narrowing in the subglottic area. The patient underwent repeat neck CT at one month follow-up which confirmed the remodelling of the cricoid cartilage architecture with opening up of the airway (Figure 2). At the 3rd month follow up, complete resolution of dyspnoea was noted, and the tracheostomy scar had healed well without any complication.

Discussion

While cricoid chondronecrosis is a rare entity and is scarcely reported in the literature, the prevalence of chondroradionecrosis is increasing among survivors of head and neck carcinoma treated with radiotherapy.[3] We present a case of traumatic chondronecrosis of cricoid cartilage. The presentation of cricoid chondronecrosis may include hoarseness, dyspnoea, dysphagia and open discharging sinuses over the neck. All of these symptoms have highly debilitating consequences for the patients.

Figure 1
Computed tomography scan of neck region showing fragmented cricoid cartilage (yellow arrow)

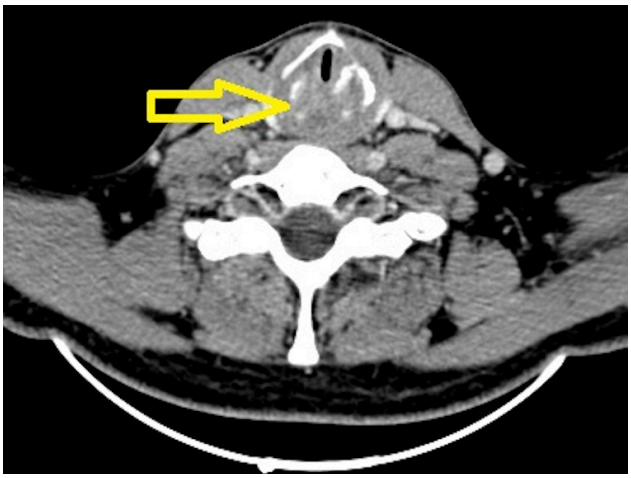
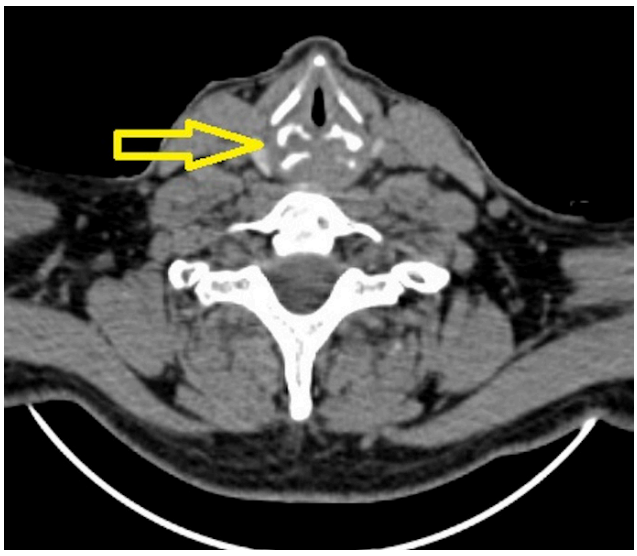


Figure 2
Computed tomography scan of head and neck region showing restoration of cricoid cartilage (yellow arrow) with resolution airway narrowing



Understanding the anatomy of cricoid cartilage including its blood supply is necessary to interpret the pathology behind its necrosis. This cartilage is the only cartilage of the laryngeal box which completely encircles the airway. It is made up of hyaline cartilage and resembles a signet ring which is narrow in the anterior side and widens posteriorly. It is avascular and oxygen and nutrients through diffusion from the surrounding capillaries. Its capacity for regeneration is consequently poor and this is a slow process.[4] The external capillaries supply only the submucosa of the cartilage and the internal perichondrial plexus provides the majority of nutrients to the cartilage. The compromise of the internal perichondrial plexus often leads to chondronecrosis of the cricoid cartilage.[2] In our case, repeated episodes of tracheostomy were thought to be the causative factor.

In radiation-induced necrosis, radiation causes hypoxic, hypovascular, and hypocellular changes, which impairs cell replication and repair and causes cartilage breakdown.[5] Apart from the radiation exposure, any mechanical trauma in the form of intubation, direct mucosal damage by high cuff pressure of a tracheostomy tube, poorly fitted or excessive movement of the tube can cause breach in the mucosa. This exposure can trigger an inflammatory response which can lead to fibrosis or necrosis[6,7] A detailed history of the patient regarding exposure to the factors causing chondronecrosis of cricoid cartilage will generally indicate the pathology causing the disease. After a detailed history, investigation modalities like laryngoscopy and CT help in diagnosis. Laryngoscopy can find the narrowing of the airway, oedema and granulation tissue but it will be a non-specific sign. Computed tomography scanning can find and quantify the glottic and subglottic narrowing, the presence of air in the central lamina of the cricoid, a hypodensity or disruption or fragmentation of the cortex of the cricoid and helps in confirming the diagnosis.[8]

Conservative treatment of cricoid chondronecrosis includes antibiotics and corticosteroids. Although the use of HBOT has been supported by studies in radiation-induced soft tissue injury (including cartilage), it may also be useful in chondronecrosis of cricoid caused by mechanical trauma or other etiologies like aseptic necrosis. The use of HBOT in patients with tracheostomy tubes in situ needs special consideration, as airway narrowing may causes difficulty in using a face mask. Hoods, T-tubes or the use of a monoplace chamber may be preferred but it all depends upon the available resources. Hyperbaric oxygen has multiple potentially relevant mechanisms in this setting. Periodic reversal of hypoxia and supports oxygen dependent wound healing processes. The standing PO_2 gradient and hyperoxia-induced vasoconstriction may reduce local tissue oedema and thus reduce the airway narrowing causing symptomatic improvement.[9] The anti-inflammatory role of HBOT in decreasing the pro-inflammatory cytokines and increasing the anti-inflammatory cytokines is widely discussed and documented in the literature.[10] The intermittent increases in oxygen tension can induce many of the mediators and cellular mechanisms that are usually induced during hypoxia. This is called hyperoxic-hypoxic paradox.[11] The potential benefits to healing include elevation of hypoxia inducible factor 1 and 2, mobilisation of vasculogenic stem and progenitor cells, and local elaboration of vasculogenic cytokines. These cell-signaling effects seem induced by transient increases in reactive oxygen species.[12] All these factors work in tandem and help in healing.

Conclusions

In cases of chondronecrosis of the cricoid, a multidisciplinary approach to treatment must be considered. The treatment protocols for this rare entity are variable because of its scarce mention in the literature. Clinicians should be aware of HBOT as an option in management, particularly in serious or refractory cases. Although a randomised controlled trial of HBOT in this indication would be desirable, the rare and sporadic nature of cases make such a study unlikely. More reports or observational series are encouraged.

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