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Benign Erosion of Laryngeal Cartilage: Report of Two Cases

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Erosion and destruction of laryngeal cartilage are usually seen in the setting of malignant or aggressive processes. Cricoid and thyroid cartilage involvement has been observed in carcinomas of the larynx, hypopharynx, and thyroid gland. Chondrosarcoma may cause a defect in the cartilage, and necrosis can occur after radiation therapy, whether or not there is superimposed bacterial superinfection [1, 2]. The pannus of rheumatoid arthritis may extend beyond the synovial cricoarytenoid and cricothyroid joints to destroy the cartilaginous skeleton itself [3, 4], and prolonged intubation may lead to aseptic necrosis [5]. One case of a benign thyroglossal duct cyst that eroded the adjacent thyroid cartilage has been described [6].

We have not found other reports of remodeling of laryngeal cartilage by adjacent benign structures. Two examples are presented here.

Case Reports

Case 1

An 80-year-old man had a CT examination (GE 9800, General Electric Medical Systems, Milwaukee, WI) to evaluate a left parotid gland mass. Five-millimeter contiguous axial scans were obtained after IV administration of iodinated contrast medium. The lateral digital scout view revealed marked productive and degenerative changes of the cervical spine (Fig. 1A). On CT images obtained at the level of the cricoid cartilage, a large anterior vertebral osteophyte deviated the larynx to the right. The posteromedial aspect of the calcified cricoid ring adjacent to the osteophyte was thinned, with preservation of a scalloped but intact cortical margin (Fig. 1B). No soft-tissue mass was identified.

Case 2

A 72-year-old woman was evaluated with CT for painful neck swelling and dysphagia 5 years after partial thyroidectomy for multinodular goiter. The same scanning technique was used as in case 1. The thyroid gland was enlarged with inhomogeneous parenchyma and foci of calcification interspersed with areas of low attenuation. The gland extended around the right thyroid ala, behind the larynx, and into the tracheoesophageal groove (Fig. 2A). The ossified right thyroid ala was remodeled and shortened as compared with the left ala. The cortex appeared intact (Fig. 2B). The patient underwent total thyroidectomy with removal of a multinodular goiter.

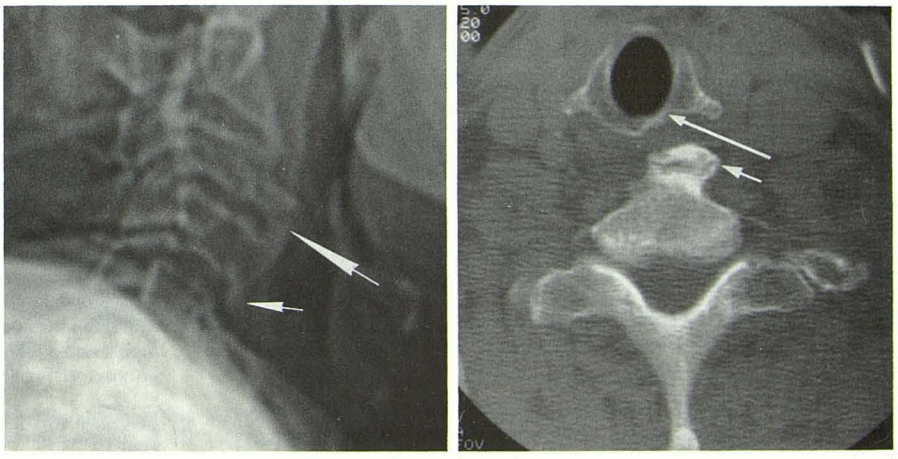

Fig. 1.—Case 1.
***A*, Lateral digital scout view from CT shows a large bridging anterior osteophyte at C3–C4 (*large arrow*) and a smaller anterior inferior osteophyte at C4 (*small arrow*). There is loss of height of C3 and C4 vertebral bodies.**
***B*, Bone algorithm axial scan at level of cricoid cartilage shows prominent anterior osteophyte (*short arrow*) separated from cricoid cartilage by esophagus and cricopharyngeal muscle. The posterior cricoid lamina has smooth, scalloped erosion (*long arrow*).**

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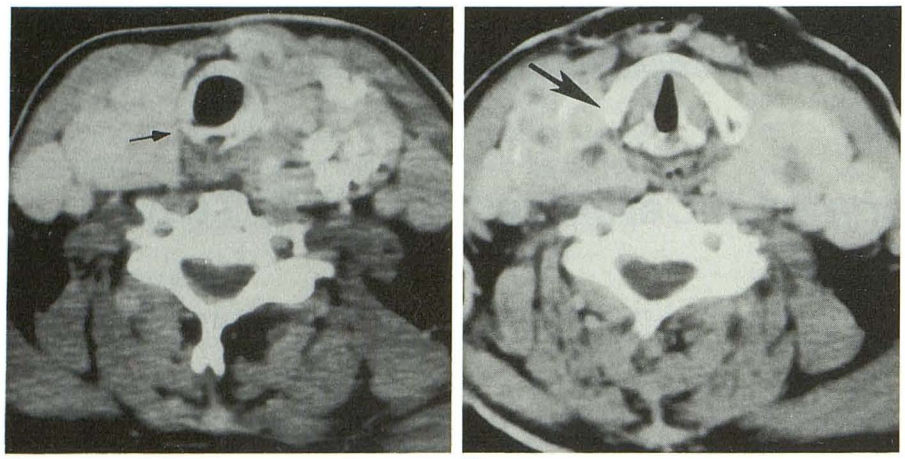

Fig. 2.—Case 2.
***A*, Axial postcontrast CT scan through level of cricoid cartilage shows both left and right lobes of thyroid gland to be enlarged, with gross calcifications in left lobe. There is thinning of the right cricoid arch (*arrow*).**
***B*, Axial scan 15 mm higher than *A* shows extension of the posterior aspects of both lobes into the tracheoesophageal grooves. There is also truncation of the right thyroid cartilage ala (*arrow*).**

Discussion

The cricoid and thyroid are hyaline cartilages covered by a thin perichondrium. Nonossified laryngeal cartilage is avascular. Two sets of nutrient vessels, located within external and internal perichondrium, nourish cartilage by diffusion [5]. By adulthood, laryngeal cartilage frequently has calcified or ossified. It then appears on CT as a marrow-filled corticated structure, interspersed with residual islands of nonmineralized cartilage [4].

The pathophysiology of destruction of cricoid and thyroid cartilage depends on the insult. Therapeutic radiation causes a small-vessel arteritis and thrombosis in surrounding soft tissues. The resultant ischemia initiates a perichondritis, which leads to necrosis and sequestration of cartilage. If the integrity of the cartilage or its perichondrium is disrupted (e.g., by biopsy), this process may be accelerated by bacterial superinfection [7].

Cricoid injury has been reported as a complication of endotracheal and nasogastric intubation. The latter is usually an acute injury secondary to ulceration of the retrocricoid mucosa, leading to perichondritis and chondritis, and ultimately to cricoid necrosis. Charlin et al. [5] reported endotracheal intubation as a cause of cricoid injury, with an initial CT appearance of a low-attenuation mass replacing cartilage. Subsequent evaluation revealed resolution of the mass, but there was persistent cricoid demineralization in the same location. Because the injury occurred weeks after intubation, the authors hypothesized that ischemia was secondary to pressure damage to perichondrial nutrient vessels.

The synovium that lines the cricoarytenoid and cricothyroid joints is susceptible to rheumatoid arthritis. Lymphocyte infiltration and synovial proliferation are followed by formation of granulation tissue (pannus), which can destroy cartilage. Ultimately, a fibrous or, rarely, bony ankylosis of the affected joint develops [3].

Normal remodeling of bone occurs in an orderly sequence. Osteoclasts are activated, perhaps by abnormal pressure on bone, resorb both organic and inorganic components, and then become quiescent [8]. The gradual destruction of bone stimulates proliferation of new vessels. Where these invade the remaining bone, osteoblasts form new bone [9], maintaining the remodeled cortical margin. (By this mechanism, too, marginal osteophytes form at joints subjected to abnormal weight bearing.) This combination of events is the likeliest explanation for the benign erosions and remodeling we observed in two patients with ossified laryngeal cartilages. Although histologic proof is not available, the intact cortex and scalloped contour of the affected cartilages suggest gradual erosive change caused by pressure.

In conclusion, the remodeling of laryngeal cartilage resulting from benign processes should not be confused with the destructive changes of inflammatory or malignant lesions.

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