REVIEW ARTICLE

Imaging in Laryngeal Tumors

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Abstract

Imaging plays an important role in the diagnostic evaluation of laryngeal cancers. This article discusses important technical issues related to cross-sectional imaging modalities, imaging anatomy, patterns of tumor spread and the contribution of imaging in pretreatment staging and post-treatment surveillance.

Keywords: Laryngeal tumors, cross-sectional imaging.

INTRODUCTION

Clinical examination (including endoscopy) and imaging are complementary to each other. Endoscopy gives an accurate assessment of mucosal involvement, but it cannot evaluate the extent of submucosal disease. CT and MRI are excellent modalities to evaluate the submucosal spaces, cartilage and extralaryngeal soft tissues. Thus, the combination of information from clinical examination and imaging provides the most accurate staging of laryngeal malignancy.

TECHNICAL CONSIDERATIONS

CT versus MRI

Modern day multidetector CT scanners can perform the complete study of larynx within few seconds, giving high resolution images unhampered by artifacts caused by breathing or swallowing. Using the overlapping axial images, it is possible to obtain excellent sagittal and coronal reformations.

The staging accuracy of MRI is slightly higher because of more accurate assessment of cartilage involvement and pre-epiglottic, paraglottic extension of tumor.

However, compared to CT, a standard MRI study of larynx takes considerably longer and requires more cooperation from the patient to prevent image degradation from artifacts produced by breathing, swallowing or coughing. MRI is also costlier and less available compared to CT. Hence, CT scan is more widely used for initial staging and MRI is more often used as a problem solving tool.

Examination Technique

Imaging is usually done during quiet breathing rather than breath holding because the abducted position of the true vocal cords facilitates evaluation of the anterior and posterior commissures. In selected cases, additional scans may be obtained with phonation or modified Valsalva maneuvers (e.g. to evaluate the laryngeal ventricle). Axial slices measuring 2 to 3 mm are obtained from the base of tongue to the trachea parallel to the plane of vocal cord (parallel to the plane of hyoid bone on CT). Focused 1 mm thin slices are required for high detail work (e.g. for evaluation of relationship of tumor to laryngeal ventricle). Image acquisition with helical CT makes it possible to obtain reformation images in any desired plane. Administration of intravenous contrast is necessary for lymph node evaluation on CT.

MRI study typically consists of T2-w sequences with fat saturation, T1-w sequences and T1-w fat saturated sequences with gadolinium in the axial and coronal planes. The sagittal plane is useful to assess potential tongue base involvement.

LARYNGEAL ANATOMY ON CROSS-SECTIONAL IMAGING

On axial scans, the upper sections show the epiglottis and connecting folds (Fig. 1). The lateral edges of the epiglottis merge with the aryepiglottic folds which converge posteriorly. Fat containing pre-epiglottic and paraglottic spaces appear dark (hypodense to muscle) on CT, hyperintense on T1-w MR sequence with intermediate signal on

T2-w sequences. Fat saturation techniques render fat hypointense. The supraglottis ends at the superior surface of true vocal cords.

On axial imaging, the false cords can be differentiated from the true vocal folds by the presence of paraglottic fat in the false cords (Fig. 2). At the glottic level, the paraglottic space contains soft tissue which represents the thyro-arytenoid muscle (Fig. 3). The laryngeal ventricle is best seen on coronal images.

Between the attachment sites of the vocal cords to the thyroid cartilage is a "bare" area–the anterior commissure, where the laryngeal mucosa abuts the thyroid cartilage. The mucosa covering the posterior wall of the glottis between the arytenoid cartilages is known as the posterior commissure. There is no submucosal tissue present in the anterior and posterior commissures (Fig. 3).

At the level of the cord, the airway narrows, more inferiorly it widens to a rounder appearance at the level of the subglottis. The paraglottic space stops at the upper margin of the cricoid. No soft tissue should be visible within the ring of cricoid cartilage, and the air column appears to abut the inner surface of the cartilage (Fig. 4).

The radiologic appearance of laryngeal cartilage may vary considerably depending on the degree of ossification.[1] On CT, ossified cartilage shows a high attenuating outer and inner cortex and a central low attenuating medullary space. Nonossified cartilage has soft tissue attenuation. On MR imaging, nonossified cartilage has an intermediate to low signal intensity on T1- and T2-w images. Cortical bone has very low signal intensity on T1- and T2-w images, whereas the medullary cavity of ossified cartilage has high signal intensity on T1-w images and intermediate signal

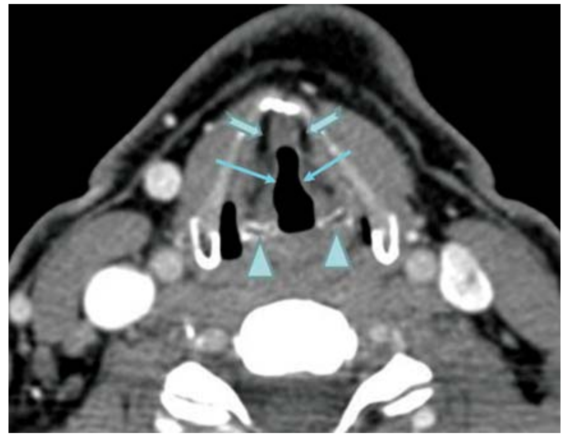


Fig. 2: Axial CT scan through the supraglottis at the level of false cords (arrows). The arytenoid cartilages (arrowheads) are seen posteriorly. The paraglottic space at this level contains mainly fat (notched arrows)

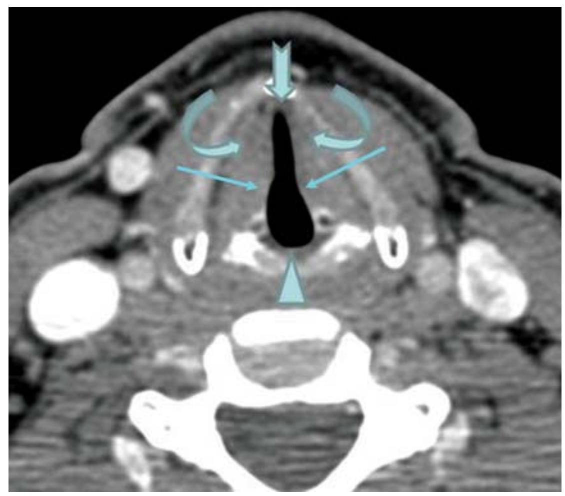


Fig. 3: Axial CT scan through the glottis showing the true vocal cords (arrows), anterior (notched arrow) and posterior commissures (arrowhead). The paraglottic space (curved arrows), at this level, has soft tissue density due to the presence of thyroarytenoid muscle. There is no submucosal tissue at the commissures

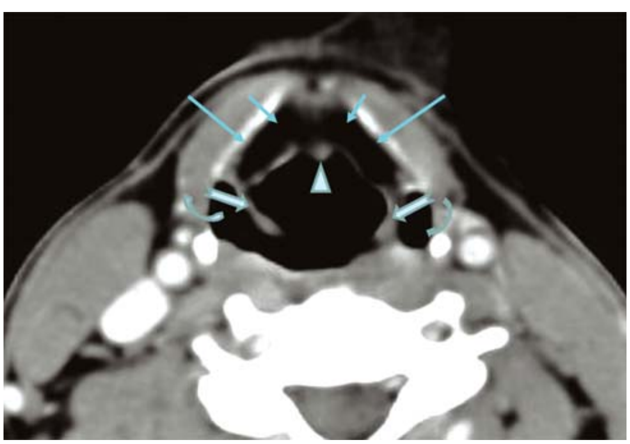


Fig. 1: Axial CT scan through the supraglottis showing the epiglottis (arrowhead), aryepiglottic folds (notched arrows), thyroid lamina (lines) and pyriform sinuses (curved arrows). The pre-epiglottic space is filled with fat (arrows)

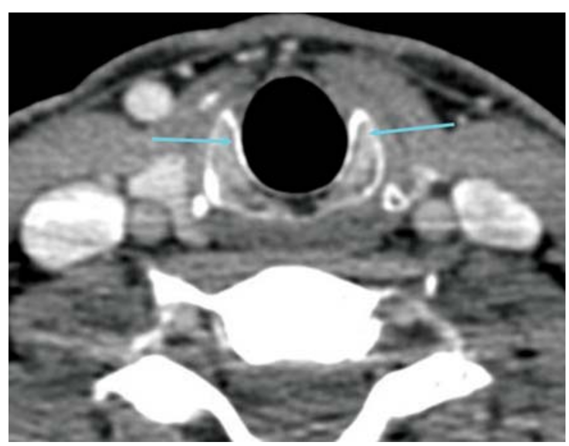


Fig. 4: Axial CT section at the subglottic level. Luminal air abuts the inner surface of cricoid cartilage (arrows) as no submucosal tissue is present at this level

intensity on T2-w images due to the high content of fatty tissue.

Axial slices are ideal for evaluating both thyroid and cricoid cartilages. Identification of the arytenoid cartilage facilitates localization of true and false cords. Beginning superiorly, the upper arytenoid is first seen at the level of the false cord as a triangular structure. The base of the arytenoids is indicated by the anteriorly oriented vocal process at more caudal level.[2]

LARYNGEAL CANCER

Squamous cell carcinoma accounts for more than 90% of laryngeal tumors. Though it originates as a mucosal lesion, infiltration of deep spaces and laryngeal cartilage is common. Depending on the site of origin (i.e. supraglottic, glottic or subglottic) typical patterns of spread is seen.[1]

Supraglottic Carcinoma

Tumors of the epiglottis (ventral supraglottic carcinomas) primarily invade the pre-epiglottic space.

Tumors that originate in the region of the petiole often invade the low pre-epiglottic space, and via the anterior commissure, the glottis or subglottis, thus becoming trans-glottic tumors.

Tumors originating from the false cord, laryngeal ventricle or aryepiglottic fold (lateral supraglottic carcinomas) primarily infiltrate the paraglottic space.

Tumors arising in the arytenoids and interarytenoid region (posterior supraglottic carcinomas) tend to infiltrate the postcricoid portion of the hypopharynx.

Lymphatic spread is common; level 2/3 nodal metastases are seen.

Glottic Carcinoma

Glottic carcinoma typically arises from the anterior half of the vocal cord and primarily spreads into the anterior commissure. The anterior attachment of the true vocal cords consists of dense, avascular fibroelastic tissue that acts as a relative barrier to early glottic cancer. Once the tumor has reached the anterior commissure, it may easily spread into the supraglottis or subglottis. There is also high incidence of thyroid cartilage invasion and extralaryngeal spread through the cricothyroid membrane.

When glottic tumor spreads laterally, it eventually invades the thyroarytenoid muscle, thus leading to vocal cord fixation. Tumor spread within the paraglottic space is limited by the conus elasticus medially and the perichondrium of the thyroid ala laterally; so further spread occurs mainly in a cephalad or caudad direction or, via the cricothyroid membrane, into the perilaryngeal tissue.

Subglottic spread is relatively common and may either occur superficial or deep to the elastic cone. Posterior extension of a glottic cancer into the anterior process of the arytenoid is relatively uncommon, and initial involvement of the posterior commissure is rare.

Lymphatic metastases from glottic carcinoma are uncommon as long as the tumor is confined to the endolarynx.

Subglottic Carcinoma

Involvement of the subglottis by laryngeal cancer usually represents inferior spread of a glottic or supraglottic tumor rather than a primary tumor originating in the subglottis. True subglottic tumors are relatively uncommon and tend to spread to the trachea or invade the thyroid gland and the cervical esophagus. Lymph node metastases from subglottic carcinomas are much more common than from glottic carcinoma. Primary drainage is directed toward the paratracheal and pretracheal nodes. These nodes drain to the lower jugular or upper mediastinal nodes.

T Staging of Laryngeal Tumors[3]

Supraglottic Carcinoma

- T1 Tumor limited to one subsite with normal vocal cord mobility.
- T2 Tumor involves mucosa of more than one supraglottic subsite or glottis or extralaryngeal mucosa (e.g. pyriform sinus) with normal vocal cord mobility.
- T3 Tumor limited to larynx with vocal cord fixation and/or invasion of postcricoid, pre-epiglottic region, paraglottic space and/or minor thyroid cartilage erosion (e.g. inner cortex).
- T4a Tumor involves tissues beyond the larynx and/or laryngeal cartilage (e.g. trachea, deep extrinsic tongue muscles, strap muscles, thyroid or esophagus).
- T4b Tumor invades prevertebral space, encases the carotid artery or invades mediastinal structures.

Glottic Carcinoma

- T1 Tumor limited to vocal cord(s) with normal mobility.
- T1a Tumor limited to one vocal cord.
- T1b Tumor involves both vocal cords.
- T2 Tumor extends to supra- and/or subglottic larynx or region outside the supraglottis (with or without impaired vocal cord mobility).
- T3 Tumor limited to larynx with vocal cord fixation and/or invasion of paraglottic space and/or minor thyroid cartilage erosion (e.g. inner cortex).
- T4a Tumor involves tissues beyond the larynx and/or laryngeal cartilage (e.g. trachea, deep extrinsic tongue muscles, strap muscles, thyroid or esophagus).
- T4b Tumor invades prevertebral space, encases the carotid artery or invades mediastinal structures.

Subglottic Carcinoma

- T1 Tumor limited to the subglottis.
- T2 Tumor extends to the vocal cords with normal or impaired cord mobility.
- T3 Tumor limited to larynx with vocal cord fixation.
- T4a Tumor involves tissues beyond the larynx and/or laryngeal cartilage (e.g. trachea, deep extrinsic tongue muscles, strap muscles, thyroid or esophagus).
- T4b Tumor invades prevertebral space, encases the carotid artery or invades mediastinal structures.

WHAT INFORMATION THE CLINICIAN WANTS FROM IMAGING?

1. Relationship of Tumor to the Ventricular Complex

This is a very important consideration in deciding the type of surgery. In most cases, this can be accurately determined by endoscopy. Imaging is helpful in situations where endoscopic evaluation is difficult, such as bulky supraglottic tumors or uncooperative patients. On axial images, the location of ventricle is determined by identifying the transition from fat in the paraglottic space (supraglottis) to soft tissue in the paraglottic space (which represents the thyroarytenoid muscle). Understanding tumor relationship to ventricle is easier in coronal scans (Fig. 5).

2. Subglottic Extension

This region is difficult to evaluate endoscopically, but both CT and MRI have high sensitivity and specificity. The normal subglottis is lined only by a thin layer of mucosa, hence any additional soft tissue is pathological. The reported sensitivity of CT and MR in detecting subglottic involvement is 93% and 96%, respectively compared with 82 to 86% sensitivity of endoscopic biopsy.[1]

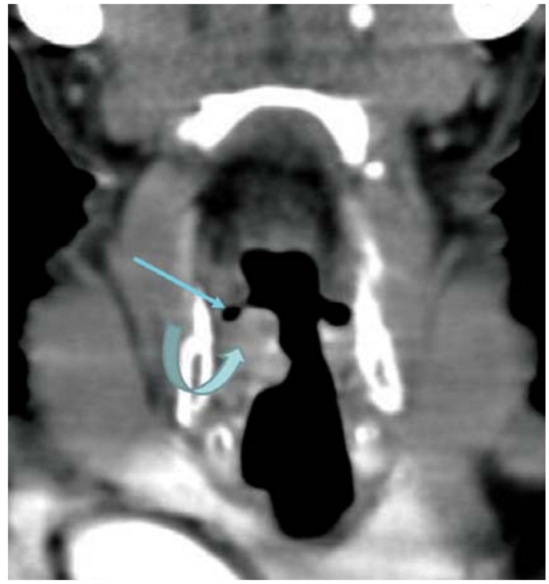


Fig. 5: Coronal reformation from helical CT displaying relationship of right glottic tumor (curved arrow) to the ventricle (arrow)

3. Submucosal Infiltration

Fat in the pre-epiglottic space and the paraglottic space has unique appearance on CT and MRI. Hence, replacement of fat by tumor tissue is easily appreciable on imaging (Fig. 6). The reported sensitivity of CT, MR in detecting pre-epiglottic space invasion is 100% and the corresponding specificities are 93% and 84 to 90% respectively.[1]

For paraglottic space invasion, CT and MR have sensitivity of 93% and 97% respectively. However, the specificity varies between 50 to 76%. The reduced specificity is because of peritumoral inflammatory changes leading to overestimation of tumor spread.[1]

4. Anterior and Posterior Extension of Glottic Tumors

In region of the commissures, the mucosa is directly abutting the cartilage with no submucosal tissue in between. Tumor in the anterior commissure is frequently associated with thyroid cartilage invasion or extralaryngeal extension through cricothyroid membrane[4] (Fig. 7). Posterior glottic tumors may invade the cricoarytenoid joint and further extend into submucosa of postcricoid pharynx. Both findings have tremendous surgical significance but are often undetectable on endoscopy.

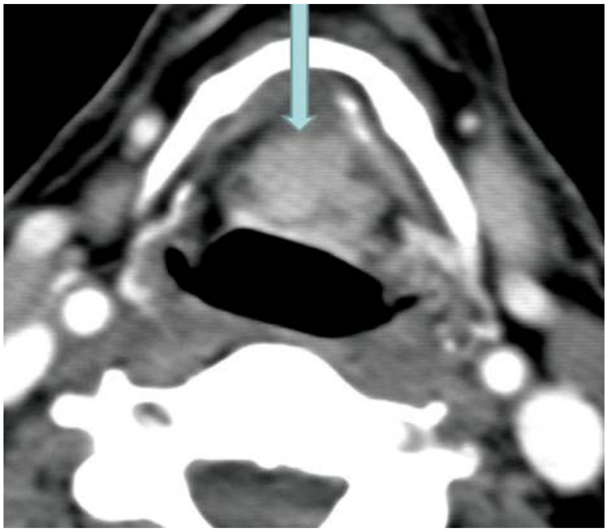


Fig. 6: Enhancing soft tissue in the normally fat-filled pre-epiglottic space indicates tumor infiltration (arrow)

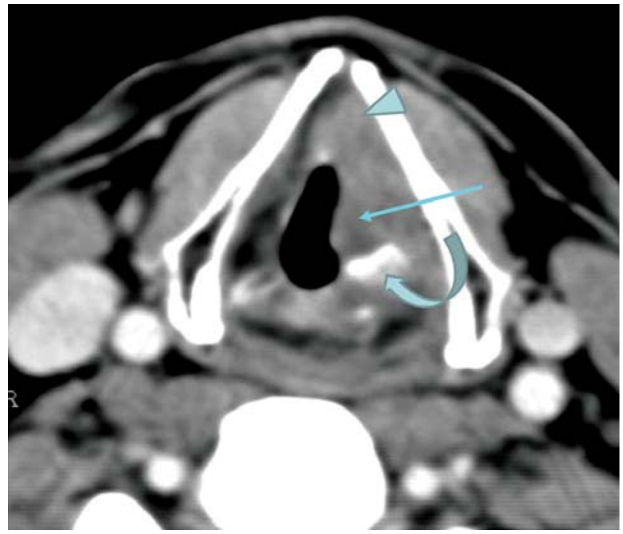


Fig. 8: Left supraglottic carcinoma (arrow) with paraglottic space involvement (arrowhead) indicated by replacement of the paraglottic fat with soft tissue. Sclerosis of the left arytenoid cartilage (curved arrow) is also observed

Fig. 7: Axial CT scan through the subglottis in a case of anterior glottic malignancy with anterior commissure involvement shows soft tissue within the cricoid ring (arrowhead) indicating subglottic extension. Extralaryngeal spread (arrows) through cricothyroid membrane is also seen in the midline anteriorly

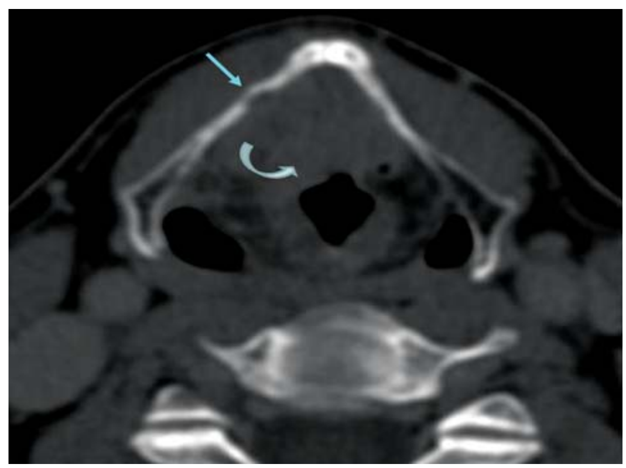


Fig. 9: Cartilage erosion-Axial CT scan viewed at bone window settings reveals erosion of inner cortex of right thyroid lamina (arrow) in a case of supraglottic carcinoma (curved arrow)

5. Laryngeal Cartilage Invasion

Cartilaginous invasion is an important consideration in treatment decisions. As clinical examination is highly insensitive in detecting cartilage invasion, the assessment is totally dependent on imaging.

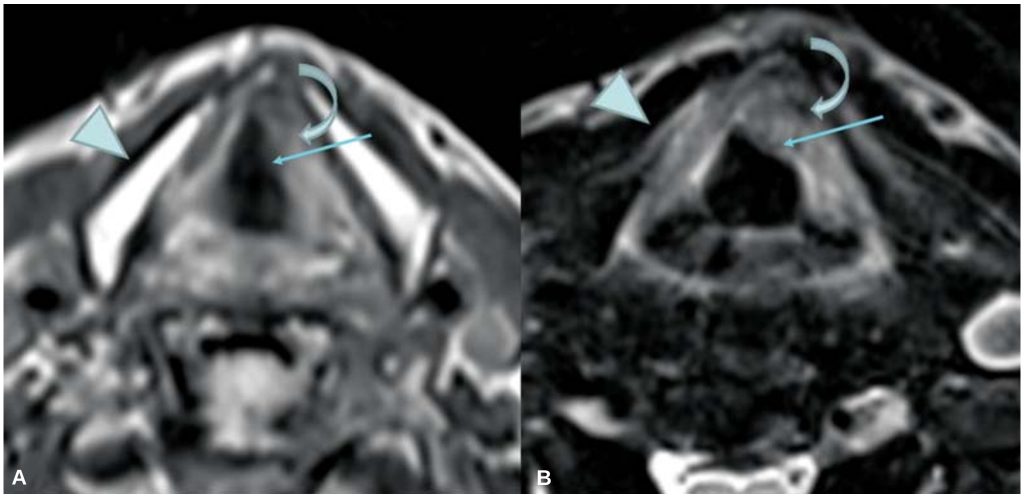
The signs of cartilage invasion on CT are:

1. Extralaryngeal tumor spread
2. Sclerosis
3. Erosion/lysis. Each of these CT signs corresponds to distinct histologic findings.[5]

Sclerosis (Fig. 8) corresponds to histological findings of early perichondrial invasion or microscopic intra-cartilaginous tumor spread inducing new bone formation. It is a sensitive sign, but specificity varies from one cartilage to another, being lowest in the thyroid cartilage (40%) and higher in the cricoid and arytenoid cartilages (76% and 79% respectively).[5]

With progressive invasion, minor and major osteolysis is seen within the areas of new bone formation. Minor areas of osteolysis correspond to the CT criterion of erosion (Fig. 9), whereas major areas of osteolysis correspond to the CT criteria of lysis. Histologically erosion and lysis corresponds to destruction of bone by osteoclasts. These

Figs 10A and B: T1- and T2-w coronal images showing left supraglottic carcinoma (arrows) destroying the thyroid cartilage (curved arrows) with extralaryngeal spread (arrowheads). Tumor tissue is seen replacing the normal hyperintense marrow signal on T1-w sequence and gives abnormal hyperintense signal in the marrow on T2-w sequence



Figs 11A and B: T1-w and T2-w axial MR images shows anterior glottic tumor (arrow) with paraglottic space involvement (curved arrow). Normal MR signal of the ossified thyroid cartilage (hyperintense marrow on T1, intermediate signal on T2) (arrow head) rules out cartilage invasion with high degree of confidence

signs have high specificity (93%), but they are not very sensitive.[5]

Extralaryngeal spread (Figs 10A and B) is highly specific (95%), but because it is seen only in very advanced cases, sensitivity is low.[5]

MRI criteria for cartilage involvement by tumor are high signal on T2-w images and/or enhancement in the cartilage adjacent to the tumor on postcontrast T1-w sequence. If these signs are absent, cartilage invasion can be ruled out with a high level of confidence (negative predictive value 92 to 96%) (Figs 11A and B). However, it should be noted that because peritumoral inflammatory changes also display similar signal changes, the positive predictive value is only 68 to 71%.[6]

6. Lymph Node Metastasis

Lymphadenopathy is best evaluated on contrast scans. The criteria most useful for evaluating lymph node involvement are size and central necrosis. Regardless of size, if central necrosis is seen, the node is considered to be pathologic.

In general, neck nodes measuring greater than 1 cm in short axis diameter are considered abnormal. However, for jugulodigastric nodes, short axis diameter greater than 1.5 cm is considered pathological.[2]

7. Carotid Involvement

Carotid involvement can be diagnosed if the encasement of the vessel is greater than 270º and lack of invasion can be diagnosed, if encasement is less than 180º. If the area of contact is between 180º and 270º, the radiologic distinction is not very reliable.[7]

8. Prevertebral Involvement

A small amount of fat tissue is present in the retropharyngeal space which can be identified on CT and MRI in most individuals. Preservation of this fat plane is a good indicator for lack of prevertebral involvement having a negative predictive value greater than 90%. However, obliteration of the fat plane is not a reliable indicator of prevertebral involvement unless there is obvious tumor in this region.[8]

POST-TREATMENT IMAGING

Postoperative and post irradiation necks are often difficult to examine clinically because of the bulky tissue of the flaps and postradiation thickening of skin and subcutaneous tissues. Hence, imaging plays a very important role in disease surveillance (Figs 12 to 14B). A baseline CT/MRI should be done 2 to 3 months after surgery, which will document the new anatomy of the individual. Comparing subsequent studies with the baseline scan prevents misinterpretation of postoperative fibrosis/granulation tissue as recurrence. In doubtful cases, PET-CT or CT guided FNAC/biopsy can be performed.

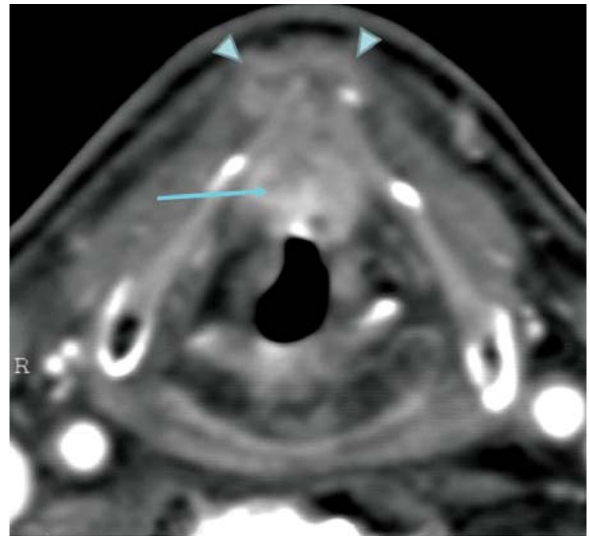


Fig. 12: Axial CT scan through the supraglottis in a patient treated previously with radiotherapy. Recurrent lesion (arrow) is seen in the midline anteriorly with extralaryngeal spread-strap muscle infiltration (arrowheads)

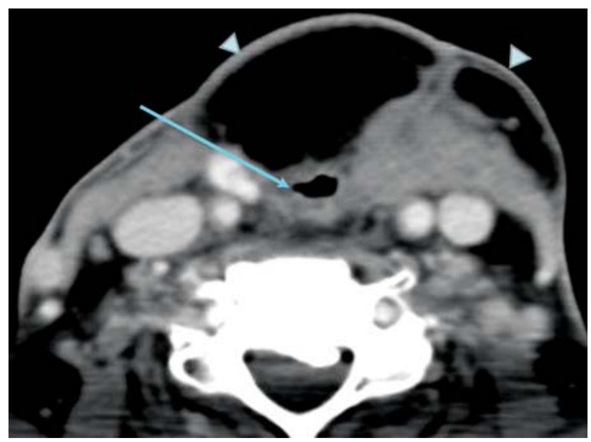
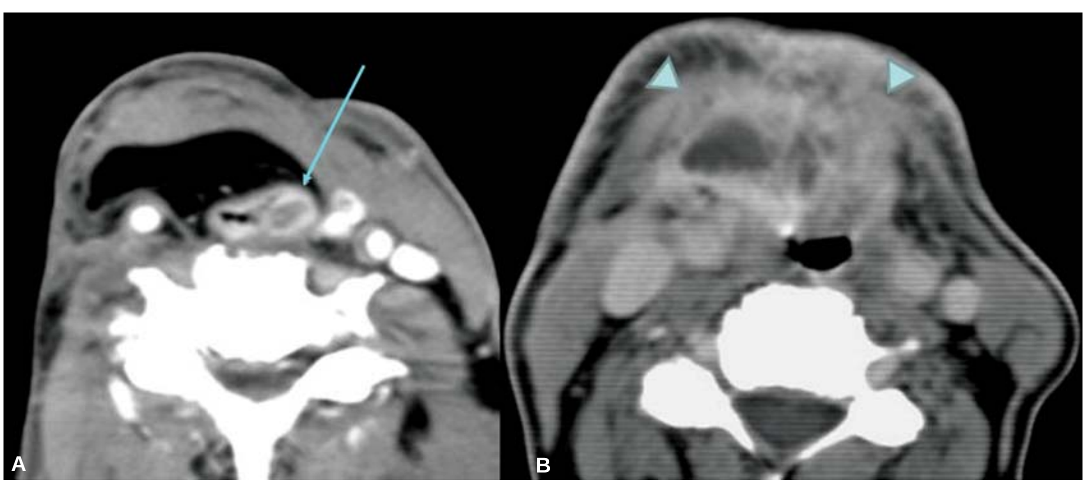


Fig. 13: Normal appearance after total laryngectomy. The neopharynx (arrow) is covered anteriorly by a myocutaneous flap (arrowheads)



Figs 14A and B: Two examples of posttotal laryngectomy recurrence. (A) Enhancing nodule in the left wall of neopharynx (arrow) (B) Advanced recurrence infiltrating the surgical flap (arrowheads)

POSITRON EMISSION TOMOGRAPHY (PET)-CT

Because of its ability to distinguish postoperative fibrosis and granulation tissue from recurrent tumor, PET-CT has a well-established role in post-treatment disease surveillance.[2] Its current use in pretreatment evaluation is limited to detection of distant metastasis in selected patients. Studies with sufficiently large numbers investigating the routine use of PET-CT in staging of laryngeal cancer are lacking. Given the intrinsic limitation of spatial resolution of PET examination and its inability to assess lesions of small volume adequately, it appears unlikely that PET would improve T-staging of laryngeal cancer compared with clinical examination and CT/MRI. The accuracy of N-staging by PET is superior to other methods; however, currently, the negative predictive value of PET in nodal metastasis assessment is not sufficiently high.[9]

NONSQUAMOUS CELL TUMORS OF THE LARYNX

These constitute less than 10% of laryngeal neoplasms. Many of these are submucosally located and visible only as a bulge beneath intact mucosa on endoscopy. In certain cases, imaging can offer a histological diagnosis (Fig. 15), e.g. lipoma (fat density), chondrosarcoma (high T2 signal and stippled calcification), hemangioma (high T2 signal and phleboliths). However, in most cases imaging findings are nonspecific. Here the role of imaging is to map out the extent of tumor and to guide the endoscopist to appropriate site for biopsy.[4]

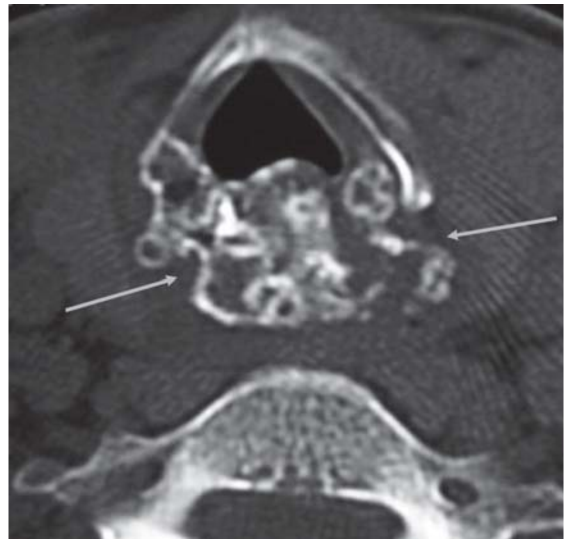


Fig. 15: Axial CT scan through the subglottis reveals a lobulated calcified lesion (arrows) arising from the endolaryngeal surface of posterior lamina of cricoid cartilage – chondroma

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