

Squamous Cell Carcinoma of True Vocal Cords (Ti) Lesion Metastasis to Lung - a Case Report

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Introduction

Squamous cell carcinoma of true vocal cords is the most common tumor in all laryngeal cancers¹. Its distant metastasis is uncommon due to anatomical structure, lymphatic drainage and poor blood supply². Incidence of distant metastasis is also low due to diagnosis of tumor in early course of disease; even slight alteration on the surface of the vocal cords produce voice changes^{1,3}.

Case Report

In December 1999, a 38 year old male was referred to us as a diagnosed case of Squamous cell carcinoma of True vocal cords (Glottic) region (Figure 1).

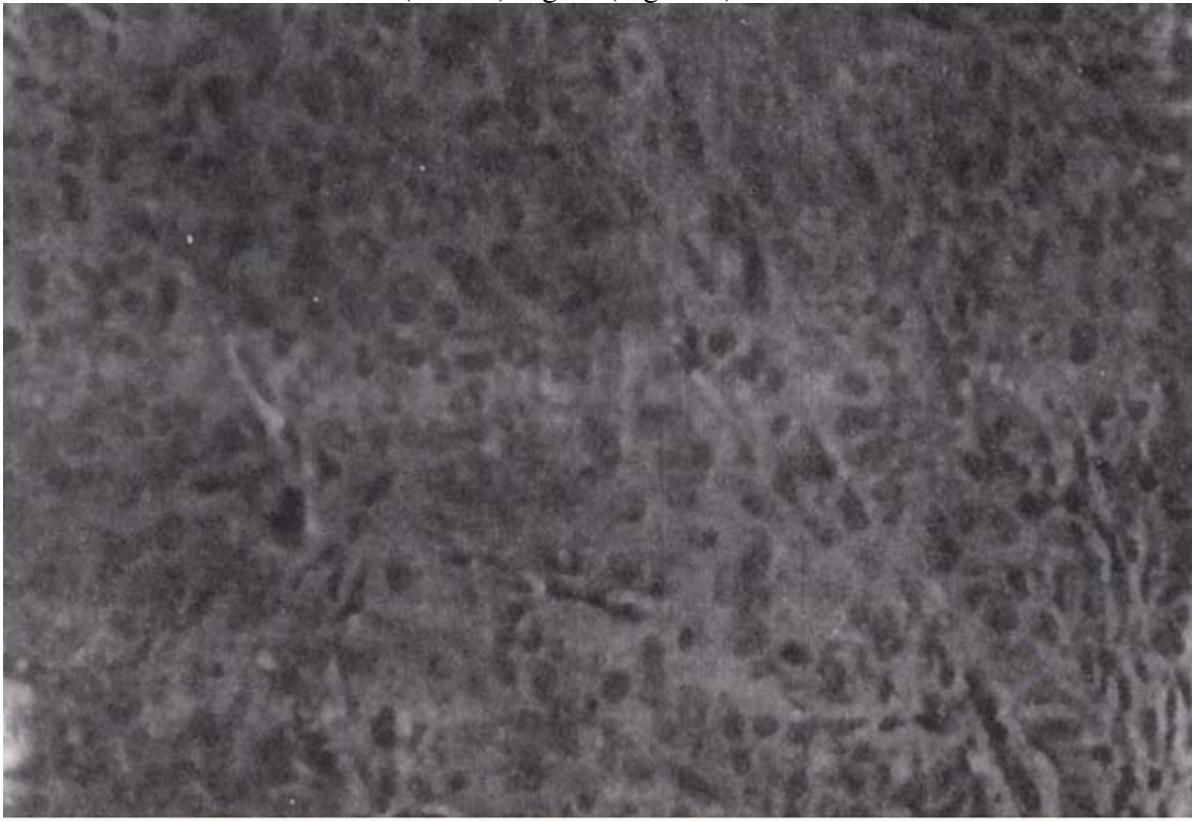


Figure 1. Laryngeal biopsy showing cell carcinoma before treatment.

After initial work-up including CT Scan, X-ray chest, U/S whole abdomen; the stage of disease was labeled as T1 N0 M0 / Stage I. The plan of treatment was decided by radical radiotherapy (cobalt). Total became productive. For this complaint he went to the chest physician who advised him an X-ray chest; which showed multiple well-defined opacities with cavitation in upper lobe

of right lung (Figure 2).

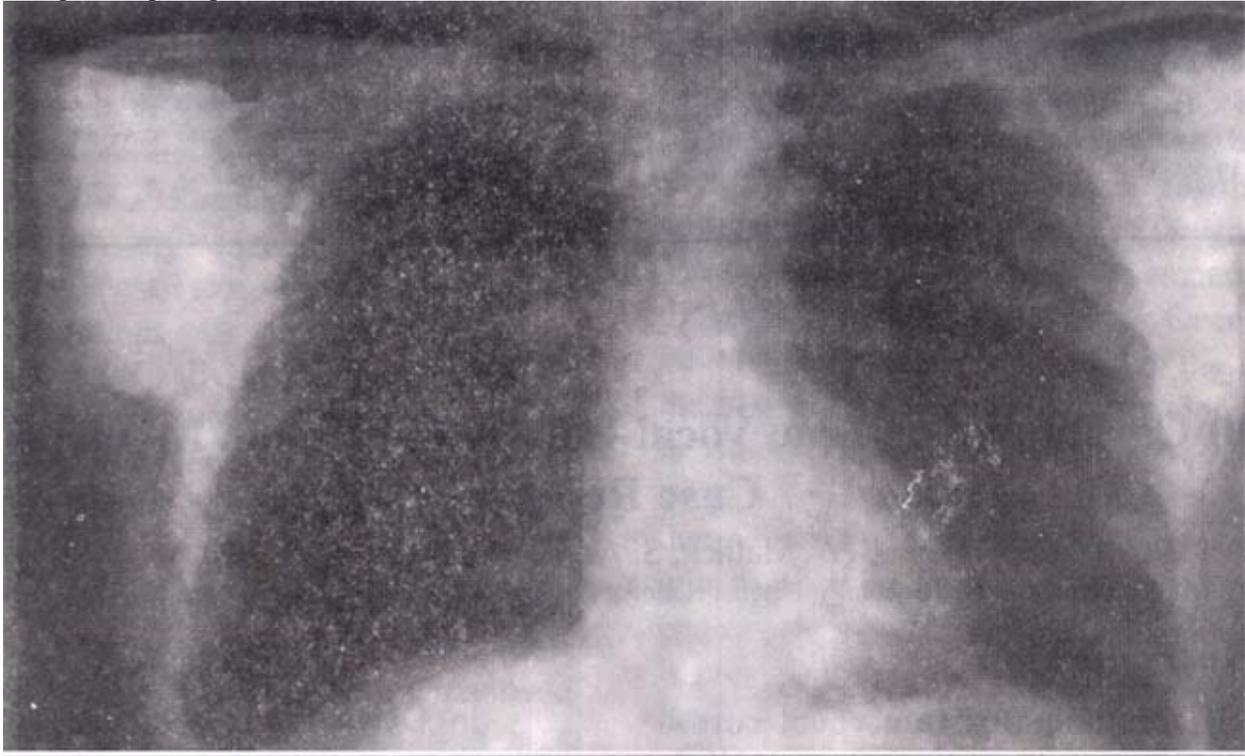


Figure 2. X-ray chest showing multiple well defined opacities with cavitation in right upper lobe.

On the basis of the X-ray findings alone a diagnosis of Pulmonary Tuberculosis was established and anti Tuberculosis treatment started. Despite 2 months of treatment he did not show a satisfactory response.

In January 2001, he came to oncology clinic for the above mentioned complaints. On the basis of history, clinical examination, X-ray findings and failure to treatment, an urgent CT Scan chest was performed which showed multiple well-marginated soft tissue densities in the right lung measuring 3.8 x 3.8cms in anterior segment of right upper lobe. The other two densities were lying in the posterior basal segment of right lung. No evidence of lymphadenopathy was noted, (Figures 3, 4).

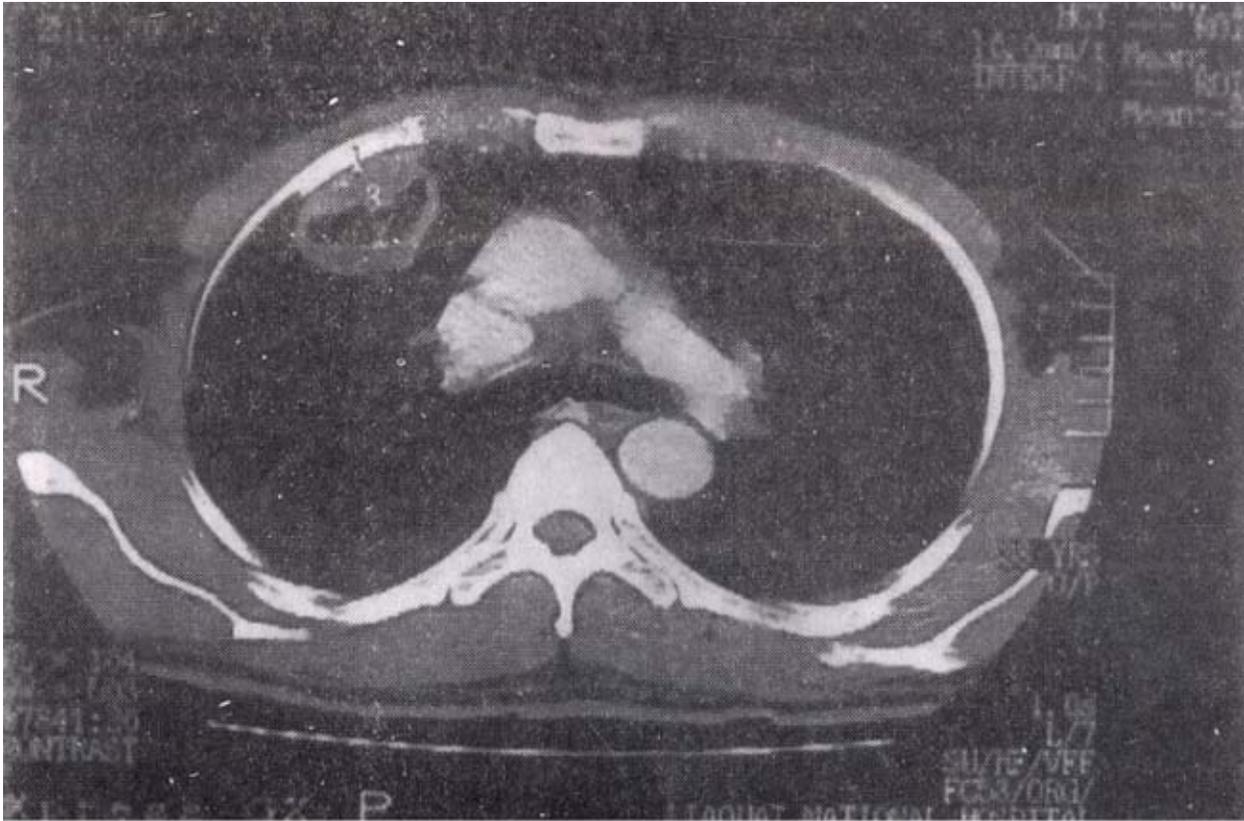


Figure 3. CT scan showing upper lobe cavitary lesion in the anterior segment of right upper lobe.

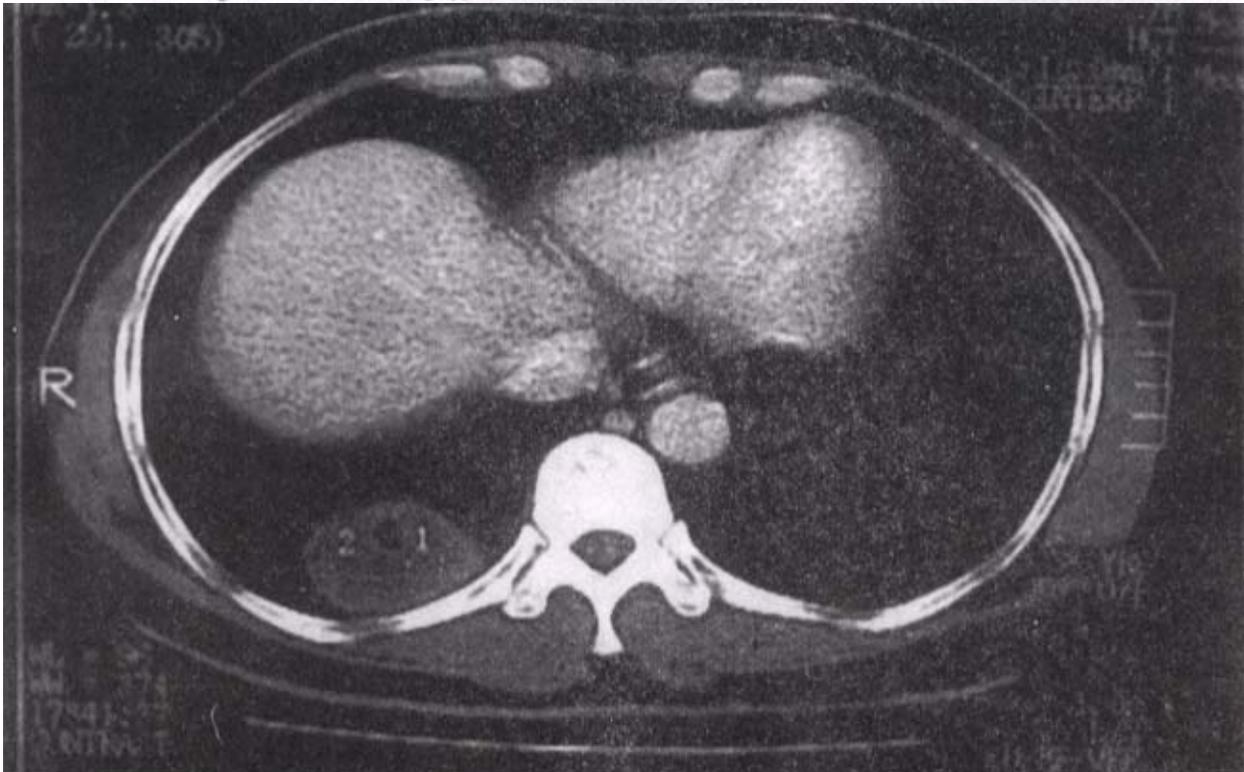


Figure 4. CT scan showing opacity in the basal segment of right lung.

In view of CT findings; the U/S Abdomen was performed to evaluate the distant metastasis.

Biopsy of primary site and lung mass was performed. U/S abdomen and biopsy of primary site revealed no evidence of disease but biopsy from lung mass showed atypical cell highly suggestive of Squamous cell carcinoma (Figures 5 and 6).

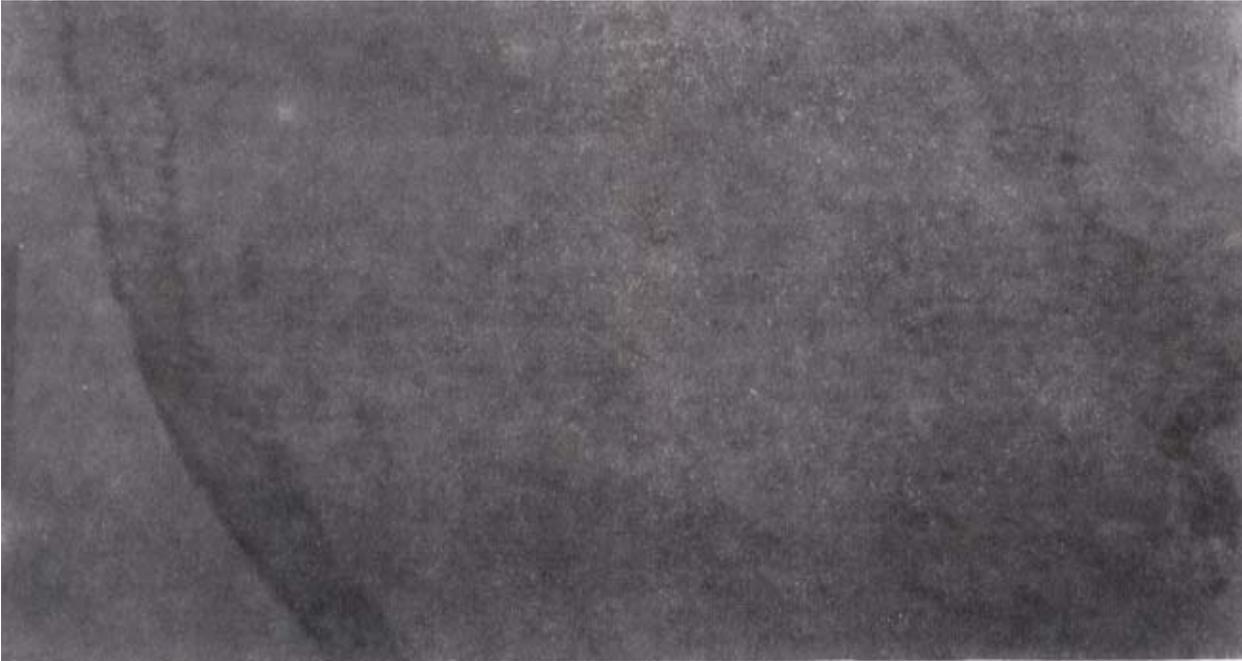


Figure 5 Post radiation laryngeal biopsy showing no evidence of disease.



Figure 6. Lung biopsy showing typical cells.

On the basis all investigations the stage of disease had been changed from Stage I to Stage IV. Therefore plan of palliative systemic combination chemotherapy comprising of Cisplatinum $75\text{mg}/\text{m}^2$ on day I and 5-Fluorouracil $750\text{mg}/\text{m}^2$ from day 2-6, with each cycle to be repeated after every 3-4 weeks was decided. After at least 2 courses of chemotherapy the response of treatment would be assessed clinically and radiologically to decide the further plan of management.

Discussion

True vocal cord carcinoma is the most common of all laryngeal cancers. It is rare before the age of 40 years¹. Most of them occur on the anterior two thirds of the cord, a small percentage on anterior commissure and rarely on the posterior commissure. Their growth characteristics are determined by the unique anatomy of the vocal cords². The sparsity of lymphatic drainage of the cord in all the areas other than posterior commissure makes metastasis of early lesions extremely unlikely³. The elastic layer within the larynx often diverts cancer that begins on the free edge of the cords and continues into the underlying vocalis muscle and paraglottic space, which is an inferiolateral pathway that leads out of the larynx through the cricothyroid space. With penetration into the underlying tissues, all degrees of motion impairment, from subtle mucous membrane stiffness to the frank fixation of the vocal cords can occur. The anterior commissure ligament forms the bridge between the anterior ends of the true vocal cords⁴. This structure lies immediately against the inner lamina of the thyroid cartilage and its presence usually retards the penetration of cancer of that area, often causing their diversion upwards onto the epiglottis and down onto the cricothyroid membrane. From there, these lesions can escape from the larynx into the neck. If the cancer overcomes the ligamentous barrier at the anterior commissure, the cartilage is penetrated^{2,3}.

Glottic cancer is often detected early in the course of the disease because even a slight alternation of the vibratory surface of the true vocal cords produces a voice change. Most of them are visible with routine laryngeal inspection, but a small percentage are obscure. Flexible endoscopes have broadened the capabilities to examine the larynx⁴. Once the lesion is discovered, the evaluation of its depth, bulk and cartilage invasion and the status of the regional lymph nodes are enhanced by CT or MRI. The former effectively demonstrates the vertical extension of tumor, especially in the subglottic and anterior commissure areas while the latter offers the advantages of multiplane visualization of the larynx. Invasion of laryngeal cartilage is important in treatment planning^{5,6}. In early glottic cancer TI, radiation or partial laryngectomy achieves excellent local control. With radiation, the voice is unquestionably better. After this treatment the voice is normal or near normal most of the time. On the other hand, all the patients are hoarse to varying degree after hemilaryngectomy or cordectomy⁷. In most centers, majority TI glottic cancers are treated with radiotherapy and partial or total laryngectomy is used as salvage operation in those who fail to respond to radiotherapy. The actual survival results for TI lesions treated by radiotherapy show local control of 91%. The local control with radiation alone in patients suitable for cordectomy was 97%. In both, local control increased to 100% when surgical salvage is added^{8,9}.

Tumor carcinogenesis is postulated to require a cascade of genetic events. In head and neck cancer a definitive carcinogenic pathway has not yet been established. However, information is accumulating describing chromosome deletions, rearrangements, particularly on chromosomes 3p, 9p and 17p. Alternations of the retinoid receptors or the epidermal growth factor receptors have also been described. Although specific oncogene activation has been observed, yet to date a

prevalent sequence of events has not been elucidated. Clearly genetic alternations have been seen at all stages and sites of this disease¹⁰.

One must also remember that second malignancy is quite frequent in laryngeal cancer ranging from 15-21%. Some time it becomes difficult to differentiate between a metastatic lesion and second malignancy following criteria may be helpful in differentiating this:

1. Metastasis usually occurs early and most of the time within a year while second malignancy usually takes longer period of time to occur.
2. The histology must be different from the primary lesion in second malignancy as compared to metastatic disease, where it should be identical to primary lesion.
3. Metastatic lesions are usually multiple as compared to second malignancy which is usually solitary.

In our case the lung lesion appeared early within 8 months and the lesions were multiple. Histology from the lesions did not show any evidence of bronchial origin. Therefore in view of the above, we consider that in our case the lung lesion some more likely to metastatic, rather than second malignancy, and thus being reported.

Staging system

Ti: Confined to cords.

Tia: Involving single cord.

T2b: Involving both cords.

T2: Spread off the cord ± impaired vocal cords mobility.

T3: Tumor limited to larynx with fixed cord.

T4: Invasion into thyroid cartilage ± other tissue beyond the larynx.

Stage 0 Tis No MO Stages I-II have approximately a 70% (5 years) survival rate; Stages III and IV have less than 40% (5 years) survival rate and N3 nodal disease carries a <20% (3 years) survival rate¹¹.

Stage 0	Tis	No	MO
Stage I	T1	No	MO
Stage II	T2	No	MO
Stage III	T3	No	MO
	T1	N1	MO
	T2	N1	MO
	T3	N1	MO
Stage IV	T4	No, N1	MO
	Any T	N2, N3	MO
	Any T	Any N	M1

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Primary site	Distant metastasis
True vocal cords	3.1%
Supraglottic region	15%
True vocal cords (Lung)	0.03%

Conclusion

The lesion of the True Vocal cords usually does not metastasize due to sparcity of lymphatic drainage, anatomical barrier, presence of cartilage and poor blood supply in the region. Because of these the metastasis in Ti lesion is extremely rare (3.1%). Once the lesion extends to involve the surrounding structures (T2, T3), the lymph node metastasis increases (2.5%). Blood borne metastasis is extremely uncommon for the same reason but has been reported occasionally. This becomes more frequent when TI category of the tumor changes to T2 or T3 lesions. Our case which has been presented also had Ti lesion and remained so till the metastasis occurred. These characteristics of true vocal cords tumor should be kept in mind.

References

1. Laroson W, Biller H, Suen J, Cancer of larynx. In Myers O, Suen J, eds. Cancer of head and neck. ed2. New York: Churchill Livingstone 1989 533.
2. Olofsson J, Lords 1, Van Nostrand A. Vocal cord fixation in laryngeal carcinoma. Acta otolaryngol 1973; 75 86.
3. Sessions D, Orgura J, Fried M. Laryngeal carcinoma involving anterior commissure and subglottis. In : Alberti P, Bryce D, eds. Workshops from the central conference on laryngeal cancer. East Norwalk, CT Appleton - century - Goufs, 1976 : 674.
4. Harino M. structure of vocal fold in normal and disease states. In: Ludlow C, O Cornell H, eds. Proceedings of conference on the assessment of vocal pathology. Report. No II. Rockville, MD : American speech and hearing Association, 1981:11.
5. Castelijns J, Gerristen G, Kaiser M, et al. Invasion of laryngeal cartilage by cancer : comparison of CT to MRI. Radiology 1989; 167 : 199.
6. Castelijns J, Kaiser M, Valk J. MR imaging of laryngeal cancer. J comput Assttomogr 1987; 11: 34.
7. Harrison L, Solomon B, Miller S, Fass D, Armstrong J, Sessions R. Prospective computer assisted voice analysis for patient with early stage glottic cancer, a preliminary report of the functional result of laryngeal irradiation. Int J Radiat Oncol Biol Phys 1990; 19 : 123.
8. Constable W, White R, EL Makdi A, Fitz - Hugh O, Radiotherapeutic management. In Seventh National Cancer Conference proceedings Philadelphia: JB Lippincott, 1973 : 54. 11.
9. Pellititteri P, Kennedy T, Varbee D, et al. Radiotherapy, the main stay in the treatment of early glottic carcinoma. Archotolaryngeal Head Neck Surg 1991; 117 : 297.
10. Spitz MR Epidemiology and risks factors for head and neck cancer. Semin Oncol 1994; 21: 281 -8, Adapted from Merino et al, An analysis of distinct metastasis from SCC of upper respiratory and digestive tract, Cancer, 1977; 40:151.