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Case Report

Non-Hodgkin lymphoma of vocal cord causing "pseudo-palsy"

Shrikant V. Solav[1]*, Rajlaxmi R. Jagtap[1], Shailendra V. Savale[1], Reena Bharadwaj[2], Chethana R.[3]

[1]Spectlab, Nuclear Medicine Services, Bavdhan, Pune, Maharashtra, India
[2]Department of Pathology, Bharati Vidyapeeth Medical College, Pune, Maharashtra, India
[3]Department of ENT, Bharati Vidyapeeth Medical College, Pune, Maharashtra, India

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***Correspondence:**
Dr. Shrikant V. Solav,
E-mail: drsolav@gmail.com

ABSTRACT

Non epithelial lesion of vocal cord is identified as vocal cord palsy/ impaired movement without obvious mucosal ulceration. There are several nonepithelial lesions of vocal cord. Lymphoma, plasmacytoma, liposarcoma, lipoma, myxofibrolipoma, hemangioma, rhabdomyosarcoma are amongst the important differential diagnosis. These tumors occur in the submucosal layer of larynx and hence the mucosa appears intact at endoscopy. The lesions are typical non circumscribed, and invasion of adjoining structures helps in differentiating benign from malignant lesions. Presented here is a case of vocal cord (pseudo) palsy who underwent an 18F-fluorodeoxyglucose positron emission tomography computed tomography (18F-FDG PET CT) scan which revealed a hypermetabolic trans glottic soft tissue lesion. Histology confirmed the diagnosis of Extra-nodal High Grade Diffuse Large B Cell Non-Hodgkin lymphoma (DLBCL NHL).

Keywords: Vocal cord (pseudo) palsy, FDG PET CT, Non-Hodgkin lymphoma

INTRODUCTION

Vocal cord lesions usually present with hoarseness of voice, dysphonia. At endoscopy a mucosal lesion usually shows ulcerations or leukoplakia. Vocal folds include the vocal ligament and its superficial mucosal covering. Benign vocal fold lesions such as nodules, polyps, cysts all present with change of voice.

Videolaryngostroboscopy helps in distinguishing various entities to a certain extent. Hypomobility of vocal cord could be due to various causes. Neurogenic vocal cord palsy results from involvement of vagus nerve anywhere in its path from its nucleus of origin in medulla up to its innervation. However, non-neurologic impaired vocal cord movement could be related to crico-arytenoid joint dysfunction secondary to joint subluxation/dislocation, tumor infiltration.[1] Neoplastic lesion may cause vocal fold hypomobility or immobility. The cause could be mechanical bulk of the tumor responsible for impaired mobility or infiltration of innervation nerves.[2] We would like to introduce the term "pseudo vocal palsy" to address impaired vocal cord movement in absence of demonstrable lesion along the path of vagus nerve to distinguish these entities.

Majority of vocal cord neoplasms are epithelial in origin that is squamous cell carcinoma. Non epithelial neoplasms comprise about 2-5% of laryngeal neoplasms and majority are malignant.[3]

We present a 39-year-old with change of voice and evidence of impaired movement of vocal cord who was found to have hypermetabolic trans glottic lesion in 18F-FDG PET CT scan. Histology confirmed the diagnosis of extra-nodal high grade diffuse large B cell non-Hodgkin lymphoma (DLBCL NHL).

CASE REPORT

A 39-year-old woman presented with change of voice of one-month duration. Laryngoscopy revealed vocal cord palsy with intact mucosa (Figure 1).

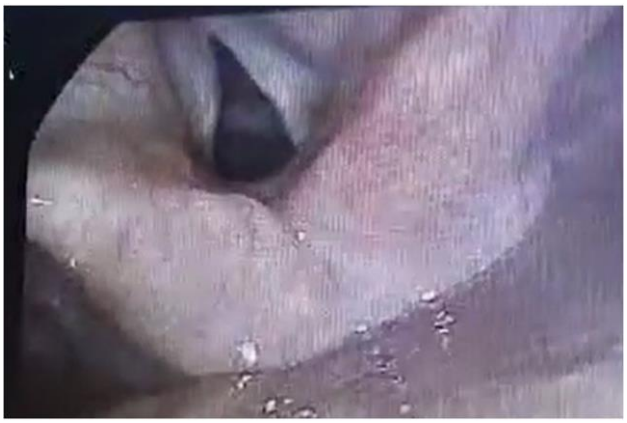

Figure 1. Photograph of vocal cords with impaired mobility of left vocal cord and intact mucosa.

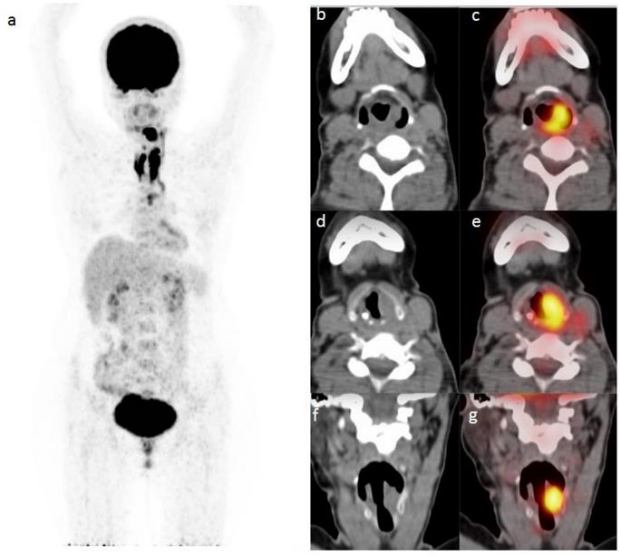

Figure 2: (a) Maximum intensity projection (MIP) image of 18F-fluorodeoxyglucose positron emission tomography computed tomography (18F-FDG PET CT); (b-e) increased FDG uptake in soft tissue nodule in left vocal cord in transaxial view and (f & g) coronal view.

CT scan showed ill-defined enhancing lesion in left vocal cord 15×13×18 millimeter in size. An 18F-FDG PET CT was performed using 8 millicurie dose administered intravenously (in overnight fasting state) and images were acquired on Siemens Horizon 16 PET CT scanner at one hour. It showed an FDG avid soft tissue lesion in the left vocal cord measuring 17×11 mm with maximum standardized uptake value (SUVmax) 17.6 (Figure 2). There was contiguous extension along the left ary-epiglottic fold and left para glottic extension. The left laryngeal ventricle was effaced giving an impression of trans glottic neoplasm. Left level III neck node measured 14 mm with SUVmax of 5.0. Thyroid gland showed diffuse FDG uptake attributed to chronic thyroiditis. Histology confirmed extra-nodal high grade diffuse large B cell non-hodgkin lymphoma, non-germinal cell type. Immunohistochemistry marker was positive for CD20 (Figure 3), Ki 67 proliferative index was 70%. CD 10 and Bcl 6 negative with MUM 1, confirming non-germinal centre B cell type NHL.

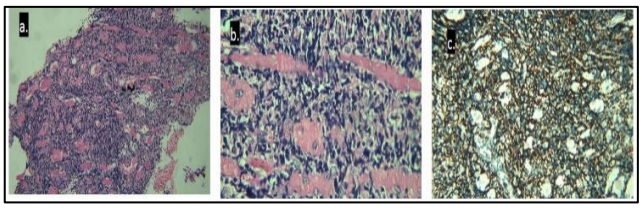

Figure 3: (a) Microphotographs of hematoxylin eosin-stained slides at 20x magnification showing large cells in sheets, (b) hematoxylin eosin stained slides at 40x magnification showing tumour cells with high nucleo-cytoplasmic ratio with scanty cytoplasm and prominent nucleoli; (c) immunohistochemistry slides at 40x magnification showing cd 20 positivity.

DISCUSSION

Our patient presented with change of voice and was found to be having vocal cord palsy with intact mucosa. As there was no lesion in the pathway of vagus nerve or its branches in the neck or mediastinum, there was no reason to suspect neurogenic involvement of vocal cord. Hence, we like to introduce the term “pseudo-palsy of vocal cord” to represent impaired vocal cord movement in absence of obvious involvement of vagus nerve or its branches. One might counter argue that the terminal fibers of recurrent laryngeal nerve could be involved at crico-arytenoid or inter-arytenoid muscles. The lesion had trans glottic extension of aryepiglottic fold. Histology showed NHL.

Laryngeal lymphoma accounts for less than 1% of laryngeal tumors.[4] It is difficult to differentiate between mucosal and submucosal lesions on imaging alone.[5] Laryngoscopy helps in distinguishing these lesions. A tiny fraction of mucosal lesions arising from the laryngeal ventricle may be missed at laryngoscopy.[6] Thus, if there is a tumor seen on imaging with intact mucosa at laryngoscopy, a non-epithelial neoplasm is likely. Extra-nodal NHL most frequently involves gastrointestinal tract. Head and neck NHL are second in order of frequency.[7]

Within the larynx NHL can arise from the submucosal lymphoid tissue from B cell lineage. MALT (mucosa associated lymphoid tumor) usually arise from ary-epiglottic fold and epiglottis.[8] Only about 100 cases of NHL involving the larynx have been reported.[9]

Macroscopically, these tumors appear as polypoidal submucous lesions without ulceration. Immuno histochemistry helps in identification of subtypes of NHL (B cell versus T cell lineage). NHL of larynx has predilection for ary-epiglottic fold- epiglottis and vestibule. 10 Our patient had trans glottic pattern of involvement. Laryngeal lymphomas are usually supraglottic and do not extend in intragalactic larynx.[11] The differential diagnosis of hypermetabolic laryngeal lesions on 18F-FDG PET CT scan include squamous cell carcinoma, lymphoma, various types of sarcomas though the later may show variable degree of metabolic activity. In addition to these enhancing laryngeal lesions on magnetic resonance imaging (MRI) include adenoid cystic carcinoma, carcinoma of minor salivary gland, rhabdomyosarcoma, chondrosarcoma, leiomyoma, spindle cell neoplasms, angiosarcoma, fibrosarcoma, enchondroma, tuberculoma, trachea-bronchial papilloma. Necrosis and calcification are usually not seen and favor more aggressive pathology such as chondrosarcoma.

CONCLUSION

The present case highlights the importance of 18F-FDG PET CT in identifying non epithelial laryngeal pathology in view of intact mucosa on endoscopy giving a false impression of vocal cord palsy that was unlikely to be neurogenic in origin.

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