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Case Report

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Laryngeal zoster: two esoteric cases

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ABSTRACT

Herpes zoster is a neurocutaneous disease resulting from the reactivation of varicella-zoster virus in dorsal sensory or cranial nerve ganglia. A thorough knowledge of laryngeal zoster is important to diagnose Zoster sine herpete. Case I shows polyneuritic involvement. Case II shows mononeuritic presentation. The clinical picture of laryngeal zoster varies from mucocutaneous lesions and paralysis to that mimicking malignancy leading to missed diagnoses.

Keywords: Laryngeal zoster, Zoster sine herpete, Herpes zoster

INTRODUCTION

Primary infection by varicella zoster virus causes chickenpox. The virus remains latent in nerve cell bodies and less frequently in the non neuronal cells of dorsal root, cranial nerve or autonomic ganglia. Herpes Zoster (HZ/shingles) results from reactivation of latent infection, occurring in 15-30% of the individuals who had chickenpox.[1]

Cephalic zoster presents as lesions involving dermatomes of the pinna, face, neck, nasal mucosa, oral cavity, pharynx and larynx. Motor palsies of face, ocular muscles, tongue, palate, pharynx and vocal cords, loss of taste and deafness are also seen. Distribution of lesions and weakness correspond to the ganglia involved. In HZ ophthalmicus and oticus presentation is clear but in glossopharyngeal and vagal involvement, the clinical picture is varied. It can be isolated or associated with other neurological features. Early identification and intervention are key to reducing duration of the disease and preventing complications.

CASE REPORT

Case 1

A 67 year old gentleman with no comorbidities presented with fever and progressive painful swallowing for 7 days. He was being treated by his physician and was on day 3 of Acyclovir. He was referred to us for sudden hoarseness and worsening dysphagia. Examination showed mucosal blisters on the right side of hard and soft palate with right palatal paresis. Tongue movement was normal.

Flexible laryngoscopy showed similar lesions with slough on the right side of nasopharynx, base of tongue, lingual and laryngeal surfaces of epiglottis, right aryepiglottic fold and right pyriform fossa with right vocal cord paralysis, cord being fixed in paramedian position (Figure 1). There was no cervical lymphadenopathy. Differential diagnosis included carcinoma hypopharynx, and HZ with polyneuritic involvement, confirmed with Tzanck smear. Biopsy was deferred by 1 week. Antiviral medication with supportive measures were continued. In 7 days he improved showing healing mucosal lesions and vocal compensation. After 4 months, follow up showed complete recovery of vocal cord movement.

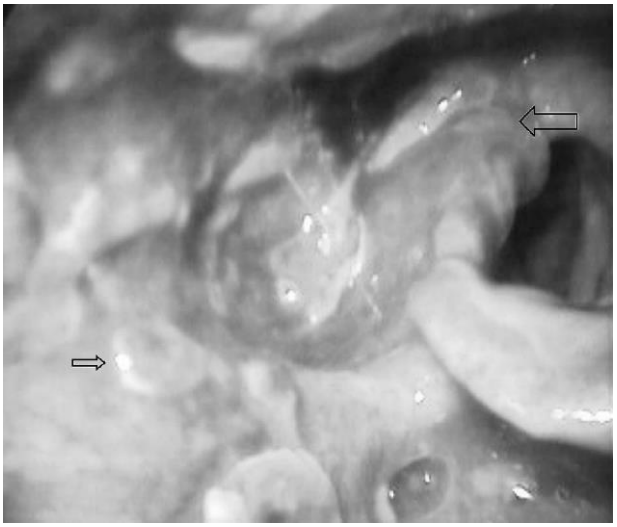

Figure 1: Vesicles over right aryepiglottic fold (large arrow), pyriform fossa (small arrow) and right vocal cord palsy seen on laryngoscopic examination.

Case 2

A 71 year old lady with no comorbidities was referred from a peripheral centre with dysphagia of 2 weeks and severe left otalgia. Dysphagia progressed from solids to liquids, becoming painful. She was a betel nut and leaf chewer. Assessment showed left vocal cord fixed in paramedian position with a sloughy mass in left pyriform fossa. Flexible laryngoscopy confirmed fixity of cord and an exophytic lesion involving medial wall of left pyriform fossa with left aryepiglottic fold bulge. Rest of the pharynx was normal. There was no cervical lymphadenopathy. Left ear examination revealed scabs of multiple healed skin lesions on lateral aspect of pinna and normal tympanic membrane.

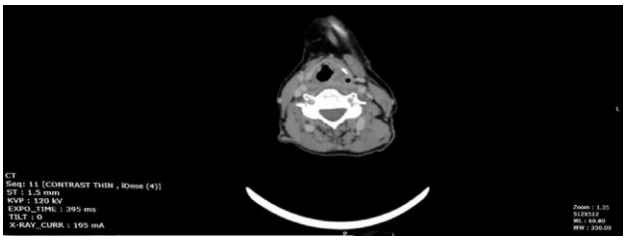

Figure 2: Contrast enhanced computed tomography neck showing left asymmetrical heterogeneously enhancing soft tissue thickening involving apex of left pyriform fossa and aryepiglottic fold partially obliterating left pyriform sinus.

Provisional diagnosis of malignancy of hypopharynx was made but the pinnal scabs and persistent otalgia indicated Zoster. A contrast enhanced computed tomography of neck (Figure 2), direct laryngoscopy and biopsy were done. After a dermatology opinion, we empirically started oral Valacyclovir 500 mg thrice a day for 10 days. IgM for varicella zoster was positive (Ig M >11.53 NTU). Histopathology confirmed viral pathology. Follow up after 2 weeks showed healed mucosal lesions with left vocal cord palsy. She recovered well, and was started on speech therapy. In 2 months, there was significant vocal recovery and in 4 months vocal cords were mobile.

DISCUSSION

Laryngeal Herpes was first described by Meyer in 1879.[2] Hunt et al published a landmark report of 60 laryngeal herpes cases in 1910.[3] The incidence rate of shingles ranges from 1.2 to 3.4 per 1000 persons/year in young healthy individuals, increasing to 3.9 to 11.8 per 1000 persons / year among those older than 65 years due to reduced cell mediated immunity. Some studies show an increased incidence in elderly females.[1] Other risk factors include immune deficiency seen in HIV infection, immunosuppressive drugs and immunotoxins. Shingles involvement of the trunk (T3 to L3 dermatomes) is most common.[4] In head and neck region, Herpes zoster ophthalmicus is most frequently seen.[5] Involvement of the larynx is rare. Isolated laryngeal herpes occurs 1 in 100 cases of laryngitis.[6]

Zosteric involvement of the larynx can be isolated or involving other areas as in herpes zoster oticus, and in trigeminal and glossopharyngeal nerve involvement. Clinical presentation varies from straightforward to obscure. Case I had classical unilateral mucosal lesions spreading over larynx and pharynx with neurological compromise. In zoster sine herpete, dermatomal lesions are absent but hyperesthesia, swelling, pain, neurological involvement and mucosal lesions are seen. Unilateral paralysis of soft palate, pharyngeal muscles or vocal cords especially when associated with otalgia or inflammatory changes in or at the hemi larynx indicate diagnosis.[7]

As in case II with a mass lesion in the larynx with/without neurological involvement, the differential diagnosis would include malignancy, granulomatous diseases or infections. In such cases biopsy is warranted. Occasionally presentation shows a stroke like picture with multiple cranial nerve involvement. Isolated Recurrent laryngeal nerve palsy is seen in zoster sine herpete. Prodromal symptoms include hyperesthesia of involved dermatome and systemic symptoms like fever, lassitude and anorexia preceding vesicular eruptions. Intractable hiccups has been reported in some cases as a prodrome, preceding HZ anywhere from 2 to 14 days.[8] The diagnosis of HZ is clinical in obvious cases. In obscure cases opt for Tzanck smear (84.7% sensitivity, 100% specificity).[9] Immunofluorescent staining for viral antigen is another test. Polymerase chain reaction for varicella zoster virus RNA in saliva & blisters is 90% sensitive. Complement fixation and enzyme immunoassay for IgM and IgG can be performed. IgM values show viral load and higher values possibly predict longer duration of lesions and paralysis.[10] The biochemical tests are significant in unclear cases and zoster sine herpete. Biopsy from lesions shows viral

pathology with giant cells containing intranuclear inclusion bodies.

Common complications of HZ are post herpetic neuralgia, superadded bacterial infection, persistent neurologic deficit, herpetic encephalitis, disseminated herpes and guillian barre like syndrome. Early use of antiviral and anti-inflammatory drugs prevent complications and reduce morbidity.

CONCLUSION

Thorough knowledge of HZ will arouse suspicion in obscure cases hastening diagnosis and upgrading treatment quality. With an increase of HZ vulnerable populations like the elderly and those immunocompromised by disease or therapy, laryngeal Zoster could be considered a significant cause of vocal cord palsy.

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