ISSN 2515-8260 Volume 09, Issue 07, 2022

AN UNUSUAL CA(U)SE OF VOCAL CORD PALSY

Manoj Sivasamy, Shreenidhi R, Kavitha MM, Jagadeesan M, Prasanna Karthik S

ABSTRACT:
Laryngeal involvement in lupus erythematosus is very rare and exceptionally revealing. The clinical manifestations could be quite varied. Vocal cord palsy (VCP) is a rare CNS involvement in the clinical course of SLE. We report a 51-year-old female who was admitted for acute onset inability to speak for 3 days and nasal regurgitation. Her ANA by IF showed a 3+ homogenous pattern. Anti – ds DNA, anti – SS A and SS B and anti-Ro 52 antibodies were also positive. Video Laryngoscopy (VDL) showed paralysis of the left vocal cord. The patient was treated with systemic corticosteroid with complete recovery with no residual vocal cord palsy.

KEYWORDS
Systemic lupus erythematosus (SLE), Vasculitis, Vocal cord, Immunosuppression.

INTRODUCTION:

Systemic Lupus Erythematosus (SLE) is an autoimmune condition affecting predominantly the skin, joints and kidney. Laryngeal involvement in SLE usually ranges from mild ulcerations and edema to necrotizing vasculitis with concomitant airway obstruction leading to death.

Laryngeal involvement in systemic lupus erythematosus (SLE), first described by Scarpelli DG[1] in 1959, is rare and often asymptomatic. It occurs mostly during SLE flares, with a usually favorable outcome, and is only exceptionally severe with life- threatening laryngeal obstruction. SLE may also indirectly involve the larynx and phonation by virtue of a vasculitic process involving cranial or peripheral nerves. Vocal cord palsy may be acquired due to a dysfunction of the recurrent laryngeal or vagus nerve[2,3]. A well recognized but rare association exists between recurrent laryngeal nerve palsy and SLE[4,5]. Failure or weakness of the vocal fold results in impaired vibration, with resulting dysphonia characterized by a hoarse voice. Well established associations include malignant invasion (e.g. mediastinal malignancy, iatrogenic injuries, viral and inflammatory conditions)[6]. From the limited literature on, Vocal cord palsy in SLE. Otorhinolaryngologic (ENT) manifestations during SLE are very rare, and include cervical polyadenopathies, laryngeal involvement, facial paralysis, trigeminal neuralgia and other less frequent and non-specific abnormalities[7].We herewith present this unusual manifestation of left side vocal cord palsy in a 51 year old female with SLE and a comprehensive review on this laryngeal involvement in cases of SLE.

CLINICAL PRESENTATION:

A 51 year old female, presented with acute onset inability to speak for 3 days and had nasal regurgitation. She also left sided ear discharge for the past 1 week. She was a known case of SLE for the past 6 years but not on regular treatment for the past 2 years. She was also a diabetic and hypertensive on regular medications and is under control. On examination, her vitals were stable. Central nervous system examination were unremarkable except for left sided vocal palsy. Video

Laryngoscopy (VDL) showed Left sided vocal palsy with no evidence of laryngeal edema or vasculitis lesions. Left ear examination revealed Chronic Suppurative Otitis Media (CSOM).

Her investigations revealed low hemoglobin levels (8.4 g/dL) along with a high ESR (110 mm/hr). Her serum C3 and C4 levels were reduced. Her renal and other parameters were within normal limits. ANA by immunofluorescence method showed a 3+ homogenous pattern. Anti – ds DNA, anti – SS A and SS B and anti-Ro 52 antibodies were also positive.

She was started on pulse steroid therapy (Inj. Methylprednisolone 1gm iv infusion for 3 days) followed by oral prednisolone therapy and hydroxychloroquine. In view of her active disease she was also given two doses of 1 gm of Rituximab (anti CD 20 monoclonal antibody) intravenously on day 0 and day 14.

She was managed conservatively for her left ear CSOM and was advised to undergo surgery at a later date. On the 5^{th} day after starting steroids, her voice retained back to normal and there was no nasal regurgitation. She was discharged after voice therapy. Follow up VDL scopy showed normal vocal cords.

DISCUSSION:

Complications of SLE are rare but could be very serious since laryngeal involvement is often closely associated with life-threatening airway obstructions.

Teitel et al.[3] reviewed 97 patients of SLE with laryngeal involvement, which ranged from mild ulcerations and edema to necrotizing vasculitis with airway obstructions, and reported that 11% (11 cases) had vocal cord paralysis. To the best of our knowledge, only few cases of vocal cord paralysis associated with SLE have been reported in the literature. Laryngeal involvement with SLE has been reported occasionally. It could be caused by direct infiltration of immune complexes on laryngeal mucosa or muscle or recurrent laryngeal neuropathy which is manifested either by vasculitis involving vasa nervorum, neuritis, or dilated pulmonary artery[3].

Pulmonary hypertension is another possible cause of left recurrent laryngeal neuropathy in this case. Three dimensional echocardiography indicated pulmonary hypertension. Furthermore, radiologic findings were consistent with interstitial lung disease. However, there was no significant dilation of the pulmonary artery visible on chest x-rays or CT. These findings suggest that nerve compression by the pulmonary artery was unlikely to be the cause of the recurrent laryngeal neuropathy. Thus, we suggest that recurrent laryngeal neuritis resulting from vasculitis of vasa nervorum was the causal factor explaining present case.

Kaleidoscope of systemic lupus symptoms and its dynamic course requires an appropriate interpretation of observed symptoms and an objective assessment of the disease activity. In those SLE patients who present with severe symptoms such as hoarseness of voice or dyspnea, an urgent ENT consultation and endoscopy may be necessary to manage potentially life threatening complications.

Systemic glucocorticoids have been the mainstay of treatment in SLE with laryngeal involvement, however some cases went on to develop obstructive respiratory distress warranting

emergency interventions such as tracheostomy and intubation[2]. Previous article describe that laryngeal involvement with SLE occurring mostly during acute phase of the disease[8].

CONCLUSION:

Vocal cord palsy in SLE is very rare. Patients with evidence of CNS vasculitis should be evaluated for systemic vasculitis and treated accordingly. CNS involvement in SLE is a challenge for the treating physician both diagnostic and therapeutic aspects.

REFERENCE:

1. Scarpelli DG, McCOY FW, Scott JK. Acute lupus erythematosus with laryngeal involvement. N Engl J Med. 1 Oct 1959;261:691-4.
2. Karim A, Ahmed S, Siddiqui R, Marder GS, Mattana J. Severe upper airway obstruction from cricoarytenoiditis as the sole presenting manifestation of a systemic lupus erythematosus flare. Chest. mars 2002;121(3):990-3.
3. Teitel AD, Mackenzie CR, Stern R, Paget SA. Laryngeal involvement in systemic lupus erythematosus. Semin Arthritis Rheum. 1992;22:203–14.
4. Lee JH, Sung IY, Park JH, Roh JL. Recurrent laryngeal neuropathy in a systemic lupus erythematosus (SLE) patient. Am J Phys Med Rehabil. 2008;87:69–70.
5. Gordon T, Dunn EC. Systemic lupus erythematosus and right recurrent laryngeal nerve palsy. Rheumatology. 1990;29:308–9.
6. Loughran C, Alves C, MacGregor FB. Current aetiology of unilateral vocal fold paralysis in a teaching hospital in the West of Scotland. J Laryngol Otol. 2002;116:907–10.
7. Loughran C, Alves C, MacGregor FB. Current aetiology of unilateral vocal fold paralysis in a teaching hospital in the West of Scotland. J Laryngol Otol. 2002;116:907–10.
8. Korbet SM, Block LJ, Lewis EJ. Laryngeal Complications in a Patient With Inactive Systemic Lupus Erythematosus. Arch Intern Med. 1984;144(9):1867–1868.