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Lepromatous leprosy with laryngeal involvement

Sir,

Leprosy,caused by *Mycobacterium leprae,* classically affects the skin and peripheral nerves, while involvement of the testes, eyes, lymph node, liver, spleen, bone, bone marrow, muscle and larynx is uncommon. Laryngeal involvement presents with cough, hoarseness of voice, dyspnea and rarely, life-threatening airway obstruction.[1] Implementation of multidrug therapy has further reduced the occurrence of airway obstruction due to laryngeal leprosy, except a few sporadic cases.[2] In the Philippines, newly diagnosed leprosy cases reached 1908 in 2017, almost 90% being multibacillary. Among them 1.9% had grade 2 disabilities at presentation, thus suggesting a relatively high rate of delayed detection.[3]

A 48-year-old Filipino male with longstanding lepromatous skin lesions presented with progressive hoarseness and dysphonia, subsequently followed by difficulty in breathing. The patient had been delaying treatment due to economic constraints and finally approached us with worsening symptoms. Cutaneous examination demonstrated leonine facies, madarosis, saddle nose deformity, nodules and ulcerations on bilateral ears and deformities affecting his feet [Figure 1]. Monofilament test revealed impaired sensation over all digits, although no peripheral nerve was enlarged. Motor test, laboratory tests and chest X-ray were unremarkable. Skin biopsy and Fite-Faraco stain confirmed lepromatous leprosy [Figure 2].

Indirect laryngoscopy demonstrated a fungating epiglottic mass, enlarged arytenoids and markedly narrowed glottic opening due to thickened vocal cords [Figures 3a and 3b]. We observed a friable fungating mass extending from the arytenoids to the true vocal folds on direct laryngoscopy. [Figure 3c]. A computerized tomography scan of the neck demonstrated narrowed glottic opening due to thickened epiglottis, aryepiglottic folds and false vocal cords [Figure 3d]. Histopathology of the arytenoid mass with Fite-Faraco stain showed numerous acid fast bacilli [Figure 3e], confirming the diagnosis of lepromatous leprosy associated laryngeal involvement. Emergency tracheostomy was performed by the otorhinolaryngology surgeons. Post-procedure, we started multidrug therapy for multibacillary leprosy. While on his 2nd blister pack, the patient underwent fiberoptic endoscopic evaluation to detect lack of vocal cord mobility and impaired swallowing.

After his completion of 11th blister pack, we noticed the resolution of ulcers on both ears, without any new skin lesions or sensory or motor deficits. Re-evaluation by the otorhinolaryngology department demonstrated persistent thickening of his epiglottis and vocal folds, thus mandating maintenance tracheostomy. After six months, the otorhinolaryngology department shall reassess the patient to determine feasibility of decannulation.

In all reported cases of leprosy with laryngeal involvement, lepromatous leprosy with widespread skin and cutaneous nerve involvement preceded their laryngeal symptoms. Laryngeal lesions develop insidiously, with an asymptomatic phase for years. Symptoms include hoarseness, dry cough, sore throat, difficulty in breathing, dysphagia, dysphonia, aphonia, odynophagia, raspy breathing, stridor and pain when speaking.[1,4] Laryngeal involvement is confirmed by histopathologic demonstration of acid-fast bacilli.[1]

Usually, laryngeal leprosy starts from the epiglottis.[4,5] This occurs as inspired air flows across the posterior choanae

How to cite this article: de Guia CE, Tablizo AV. Lepromatous leprosy with laryngeal involvement. Indian J Dermatol Venereol Leprol 2022;88:106-9.

Received: April, 2021 **Accepted:** July, 2021 **EPub Ahead of Print:** October, 2021 **Published:** December 2021

DOI: 10.25259/IJDVL_381_2021 **PMID:** 34672475

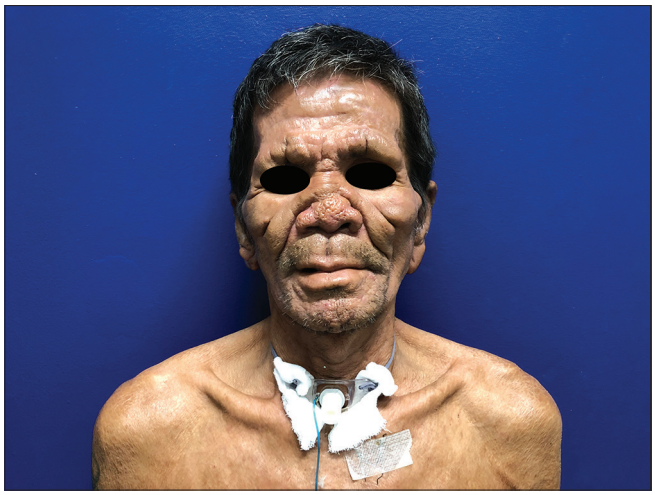

Figure 1a: Leonine facies, madarosis and saddle nose deformity

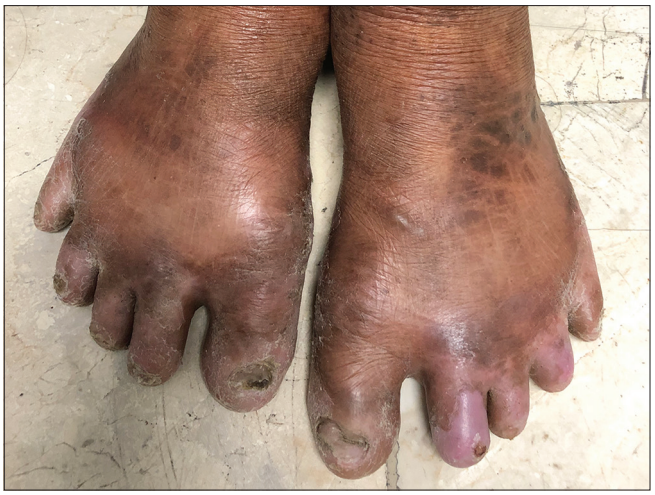

Figure 1b: Grade 2 deformities affecting bilateral feet.

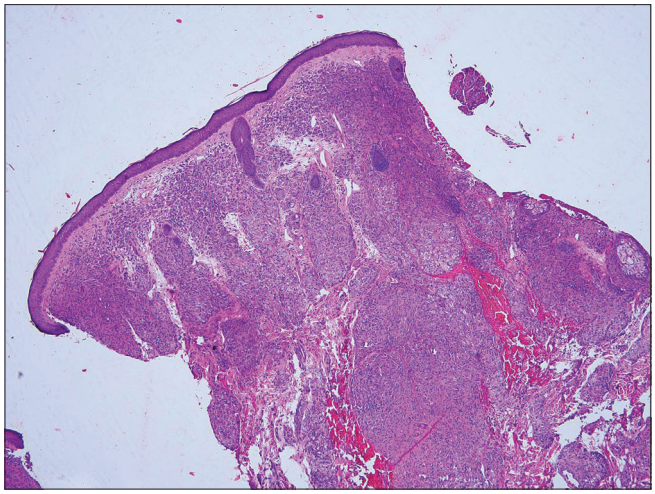

Figure 2a: Atrophic epidermis and nodular perivascular, periadnexal and perineural infiltrates on histology (H&E, ×40)

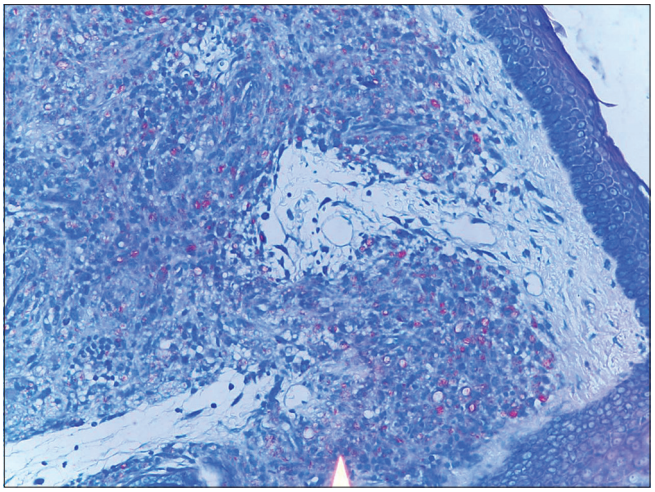

Figure 2b: Fite-Faraco stain demonstrating a bacillary index of 6+ (Fite-Faraco, ×400)

over the epiglottis reducing local temperature by ~2°C, thus favouring the growth of *Mycobacterium leprae* due to its predilection for cooler temperatures. Once the epiglottis is affected, infiltration of other laryngeal structures may occur. Vocal cord infiltration is responsible for hoarseness, due to restricted mobility; vocal cords are usually involved later in the disease process, hence the lag time between cutaneous and laryngeal symptoms.[4]

Long-standing laryngeal lesions may become ulcerative or fibrotic. The former results in early development of granulation tissue with subsequent inflammation and rapid edema, which may lead to acute respiratory distress.[4] Conversely, in the fibrotic form, gradual fibrous tissue formation occurs, possibly leading to laryngeal stenosis and manifesting as aphonia or rarely an upper airway obstruction requiring tracheostomy.[4,5]

Multidrug therapy effectively reduces bacterial count in laryngeal leprosy,[4] although the prognosis remains unaffected, as evidenced by variable treatment outcomes. While few authors reported normalizations of voice post-treatment, most developed persistent dysphonia. Rarely, emergency tracheostomy is necessary, similar to our patient, due to irreversible destruction and scarring of the larynx.[2,5] Although multidrug therapy is effective, the authors hypothesize that fibrotic form of laryngeal leprosy is recalcitrant to treatment, owing to underlying scarring and fibrous tissue deposition.

The present rare case of laryngeal leprosy highlights the importance of laryngeal examination in patients with lepromatous leprosy to prevent complications such as laryngeal stenosis and airway obstruction. Moreover, this case emphasizes the need for health education and

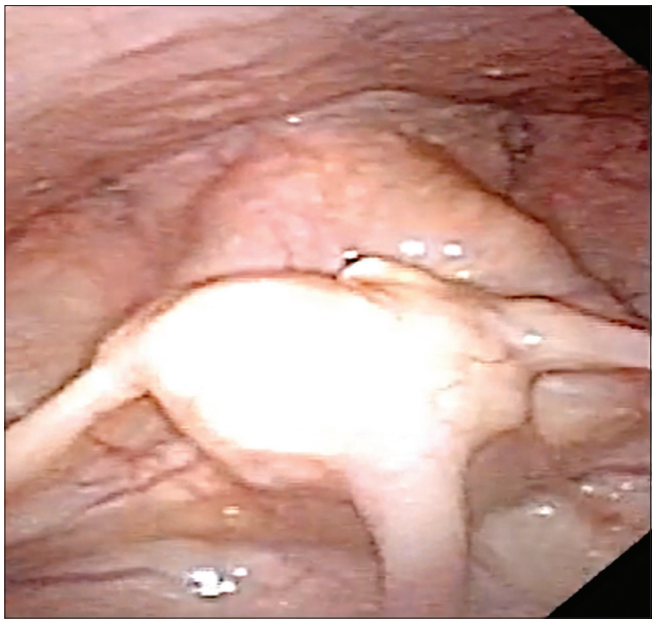

Figure 3a: Indirect laryngoscopy demonstrated a solitary fungating mass on the epiglottis

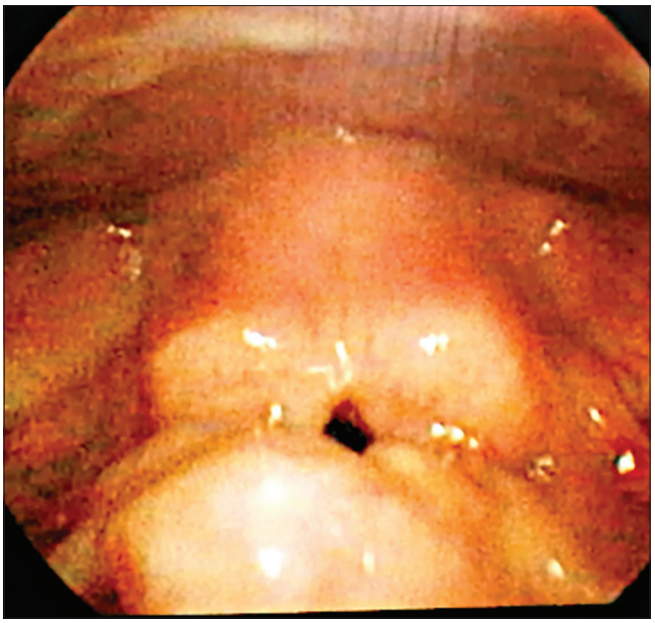

Figure 3b: Bilaterally enlarged arytenoids and markedly narrowed glottic opening

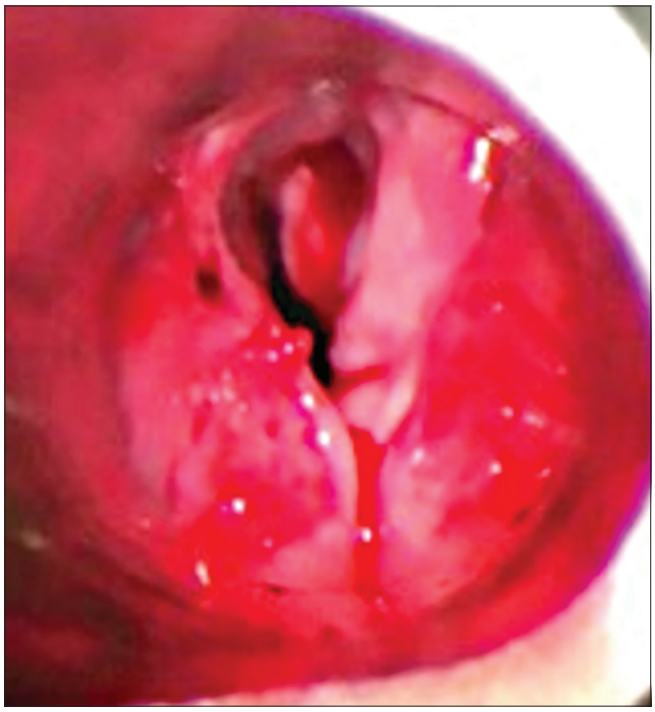

Figure 3c: Direct laryngoscopy demonstrating a friable fungating mass extending from the arytenoids up to the true vocal folds

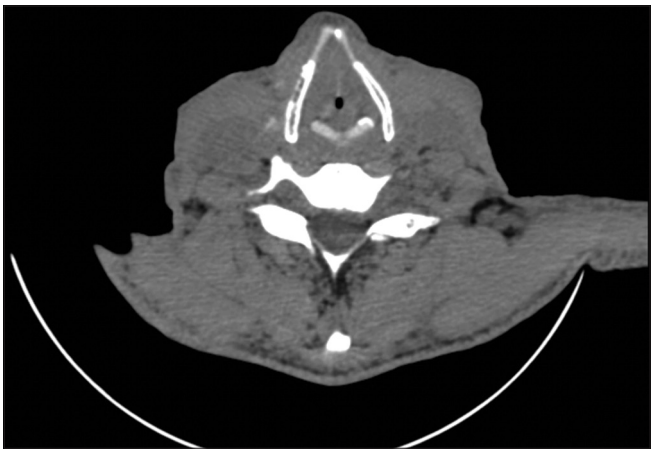

Figure 3d: Plain CT scan of the neck demonstrating narrowed glottic opening due to thickened epiglottis, aryepiglottic folds and false vocal cords

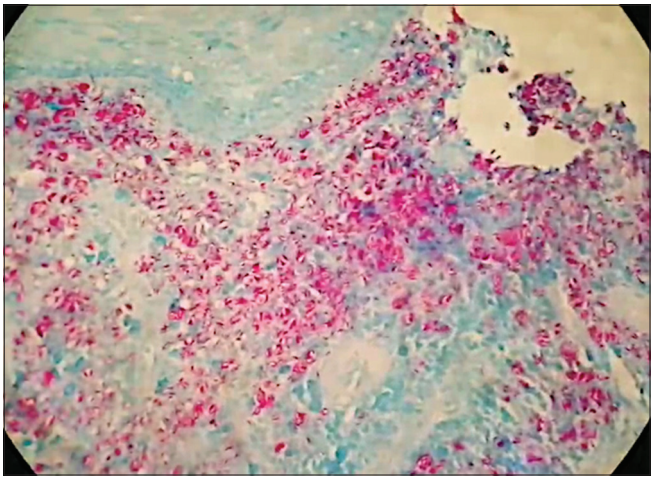

Figure 3e: Numerous AFB noted on the arytenoid mass Fite-Faraco stain (Fite-Faraco, ×400)

community awareness to promote early detection and treatment of this disease to prevent its permanent and life-threatening sequelae.

Acknowledgments

The authors would like to acknowledge Dr. Ma. Luisa Abad Venida for her insights and guidance on this case. The authors

would also like to acknowledge Dr. Danielle Nicolle D. Mejia for her contribution as the senior resident physician handling this case. The authors would also like to acknowledge Dr. Gianfranco C. Velasco, the resident-physician from the otorhinolaryngology-head and neck surgery service who first admitted the patient, oversaw his laryngoscopy, tracheostomy and fiberoptic endoscopic evaluation of swallowing procedure and provided the photographs of the larynx and laryngeal biopsy. Dr. Gianfranco C. Velasco provided invaluable information and insights on the patient's laryngeal condition.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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The ping-pong infection in gonorrhoea: Lest we forget

Sir,

Contact tracing in sexually transmitted infections is often understated. We describe a related entity, "the ping-pong" infection in gonococcal urethritis.

A 26-year-old unmarried man, working as ground staff at the airport, presented with dysuria and discharge per urethra for five days with no systemic symptoms. A few days ago, he had sexual contact with a female sex worker. Besides, he was in a regular non-marital sexual relationship (unprotected) for the past six months with a married woman, whose husband was an army personnel. Both the partners, however, were asymptomatic.

There was profuse discharge per urethra which was foul-smelling, greenish, thick, purulent and associated with perimeatal erythema. There was no inguinal lymphadenopathy. Gram smear of the discharge revealed abundant polymorphonuclear cells along with both intra and extracellular Gram-negative diplococci. A diagnosis of gonococcal urethritis was made which was confirmed by nucleic acid amplification test. An in-house opa gene and the *porA* pseudogene-based polymerase chain reaction assay were used for confirmation. Culture on modified Thayer Martin and chocolate agar medium was also done. The suspected colonies in culture were confirmed by Gram stain as well as oxidase, superoxol and rapid carbohydrate utilization tests. Antimicrobial susceptibility test of *Neisseria gonorrhoeae* isolates was done by disc diffusion method, minimum inhibitory concentration (MIC) was determined by E test and the results were interpreted using the breakpoint criteria of calibrated dichotomous sensitivity technique.[1] Low concentration antibiotic discs (Oxoid Basingstoke, UK) which included penicillin (0.5 IU), ciprofloxacin (1 microgram), nalidixic acid (30 μg), ceftriaxone (0.5 μg), cefpodoxime (10 micrograms), spectinomycin (100 μg), tetracycline (10 micrograms) and azithromycin (15 μg) were used. In addition, cefixime (5 micrograms) was tested and interpreted as per the Clinical and Laboratory Standards Institute **(**CLSI) guidelines.[2] The strain was susceptible to ceftriaxone (annular radius – 14 mm; MIC – 0.003 μg/ml), cefpodoxime, azithromycin (annular radius – 16 mm; MIC – 0.094 μg/ml) and spectinomycin. Cefpodoxime was used as a surrogate marker for oral cephalosporins. It exhibited high-level resistance to ciprofloxacin and plasmid-mediated high-level resistance to tetracycline (tetracycline-resistant

How to cite this article: Dev T, Mahajan N, Sood S, Gupta S. The ping-pong infection in gonorrhoea: Lest we forget. Indian J Dermatol Venereol Leprol 2022;88:109-11.

Received: April, 2021 **Accepted:** June, 2021 **EPub Ahead of Print:** August, 2021 **Published:** December 2021

DOI: 10.25259/IJDVL_413_2021 **PMID:** 34491678