

Dysphonia Revealing Early Syphilis

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Sir,

Syphilis has reappeared in the form of epidemics in France and other western countries since the end of the 1990s, affecting mostly homosexual and bisexual male patients half of whom are HIV-positive (1–4). During their haematogenous spread the spirochetes can infect various organs including the head and neck. Although rare, the larynx may be involved at any stage of the disease, but mostly at the later ones. We report here the case of a bisexual HIV-positive patient who presented acute dysphonia and dysphagia one week before a typical syphilitic eruption occurred.

CASE REPORT

A 48-year-old bisexual man was referred for a diffuse papular eruption that had been evolving for one week. His medical history was notable for: (i) HIV infection for 18 years without any follow-up or therapy; (ii) secondary syphilis diagnosed at the same period 18 years ago and treated with three injections of benzathine penicillin; and (iii) thoracic zoster eruption one year previously.

The patient acknowledged having sex with multiple sexual partners. He practiced safe sex for anogenital intercourse, but not always for oral sex. He denied any other sexually transmitted disease, drug abuse or medication. His last Venereal Disease Research Laboratory (VDRL) control, performed one year prior to consultation, was negative. His CD4 level fluctuated between 200 and 400 cells/mm³, whereas his HIV viral load was unknown.

Physical examination revealed generalized, small, polymorphous, infiltrated papules predominating on the upper and lower limbs, the trunk and the back. The palms and soles were also involved. Several lesions were observed over the genitalia, with a collarette of scale in the periphery. The oral mucosa was free of any lesion. The clinical aspects were compatible with secondary syphilis. Cervical, axillar and inguinal lymphadenopathies were palpated. Constitutional symptoms, such as fever, malaise, headaches or arthralgias, were absent. There was no clinical manifestation of symptomatic neurosyphilis. The patient complained only of a severe dysphonia and mild dysphagia, which appeared one week before the skin rash. A diagnosis of acute laryngitis was made at the onset of dysphonia.

Laboratory tests revealed that VDRL and *Treponema pallidum* haemagglutination assay (TPHA) were above

512 IU and 10,240 IU, respectively. Lymphocytes CD4 count was 230/mm³, with an elevated viral load of over 500,000 copies/ml. White blood count, platelets, kidney and liver functions were within the normal range. An elevated titre of VDRL prompted us to perform a lumbar puncture. The cerebrospinal fluid was clear, disclosing 8 cells/mm³, normal glucose and protein levels, and a negative Gram-stain. Laryngoscopy examination displayed mucous patches compatible with specific location of syphilis. No histological or direct examination was performed.

The patient was treated with three intramuscular injections of 2.4×10⁶ units of benzathine penicillin, once per week, with an excellent tolerance. Dysphonia began to improve after two injections, whereas the cutaneous lesions had almost completely vanished. The patient was then lost to follow-up.

DISCUSSION

Albeit extremely rare nowadays, the larynx may be involved at any stage of syphilis (5). Most such cases occur during the secondary and tertiary stages of the disease (6, 7). In the case of oral mucous patches, laryngeal involvement is constant (5). Diffuse laryngeal hyperemia is observed during the secondary phase in association with erythematous maculopapular papules that progress to mucous patches throughout the larynx, especially in the supraglottic region (6). These lesions are highly contagious and ulcerations expose the patient to the risk of bacterial infection leading to perichondritis. Secondary laryngeal lesions can either be asymptomatic or, as in our case, lead to disabling symptoms, such as dysphonia and/or dysphagia.

Tertiary syphilis appears 15–20 years after the first stage in the case of under-recognition of the disease. It is characterized by gumma formation (8, 9), ulcers, chronic granulomatous infiltration and fibrosis (6). In the absence of treatment, perichondritis, chondritis, fibrosis and scarring are the consequences of deep ulcerative invasion of the thyroid and cricoid cartilages (6). Laryngoscopic examination reveals single or multiple necrotic ulcers, mimicking carcinoma. Histology can display pseudo-epitheliomatous hyperplasia with submucosal perivascular infiltration of plasmacytes and lymphocytes and some degrees of vasculitis (7).

HIV can alter the clinical manifestations of syphilis (3). To the best of our knowledge, there is no report

mentioning specific laryngeal manifestations of syphilis during HIV co-infection. Moreover, the clinical response of early syphilis to therapy is not altered by HIV infection, even though serological responses may be delayed (4). High titres of VDRL (over 52 IU) in this case prompted us to perform a lumbar puncture, which was completely normal in the absence of neurological anomaly.

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