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Squamous Cell Carcinoma Arising From Lupus Vulgaris On The Neck

Boyunda Lupus Vulgaristen Gelişen Skuamoz Hücreli Karsinom

Abstract
Lupus vulgaris (LV) is the most commonly encountered form of cutaneous tuberculosis, and the most common site of involvement is the head and neck. We report the case of a 45-year-old man with a squamous cell carcinoma arising from a plaque of LV with a 20-year history on the malar region and the neck. The patient presented with a 3-month history of a squamous cell carcinoma enlarging on the left neck. He has been successfully treated with tumor excision and antituberculous therapy.

Key words: Lupus vulgaris, squamous cell carcinoma, cutaneous tuberculosis

INTRODUCTION
Lupus vulgaris (LV) is usually the result of dissemination from an endogenous focus during a period of lowered resistance and Mycobacterium tuberculosis bacillemia in a previously sensitized host with a strongly positive delayed hypersensitivity to tuberculin. The spread is mostly hematogenous or lymphatic. The contagious spread is also seen particularly in cervical adenitis or pulmonary tuberculosis, or sometimes in exogenous infection at the site of primary inoculation or after BCG vaccination (1). Development of malignancies is an important complication of LV. Although the most commonly encountered form is squamous cell carcinoma (SCC), various type of malignancy was reported. In many source, it was briefly named as “lupus carcinoma”. SCC development rate from LV lesions is in a range of 0.5% to 10.5%. A Medline search revealed 22 such cases, the majority being published before 1990 (2). Lupus carcinoma development on the neck lesions were reported in 4 patients (3,4). To our knowledge, hereby, we present the fifth case in the literature.

CASE REPORT
A 45-year-old man was admitted to our clinic with the complaint of longstanding erythematous lesion over the left malar area and the left neck of 20-year duration. Verrucous-exophytic tumour of the left neck had developed over 3-month. His previous and family histories were not contributory. He had remained undiagnosed and no treatment had been given throughout 20 years.
Dermatological examination revealed an erythematos, nontender, well-defined plaque of 30x40 cm in size over the left malar area and the left neck. An exophytic, infiltrated and ulcerated tumoral lesion of brownish-red, 3x4 cm in size was observed on the center of the plaque (Fig. 1). Diascopic examination gave an apple-jelly appearance. The systemic examination was normal. He was afebrile, and there was no submandibular or cervical lymphadenopathy. No BCG scar was visible.

Incisional biopsies from plaque and ulcerated lesions were performed. The entire dermis was composed of non-caseous granulomatous inflammation which contains epitheloid histiocytes, lymphocytes, and large numbers of Langhans type giant cells (Fig 2A). The ulcerated lesion of histopathological examination showed proliferation of atypical keratinocytes with eosinophilic, occasionally dyskeratotic cytoplasm; their nuclei were hyperchromatic and occasionally mitotic. These cells invaded the epidermis and formed tumoral nodules that contained horny pearl and invaded the upper and mid-dermis (Fig 2B).

Sputum, stool and urine cultures were negative. Laboratory tests showed a normal blood count. VDRL and HIV tests were negative. Fungal and standard bacterial cultures from the skin biopsy were negative. Ziehl-Neelsen and periodic acid-Schiff stains did not demonstrate any acid-fast bacilli. Tuberculin skin test was positive with erythema and induration of 12 mm after 48 hours. Mycobacterium tuberculosis was cultured from the biopsy specimen after five weeks. PCR for M. tuberculosis complex gave a negative result. Cranial, chest, and abdominal computed tomographic scans showed no evidence of any active or metastatic diseases. Underlying bone and joint disease was excluded by radionuclide scanning.

The exophytic-ulcerated lesion of the neck excised under local anesthesia. The patient was treated with four-drug therapy consisting of isoniazid (5 mg/kg), rifampin (10 mg/kg), pyrazinamide (25 mg/kg), and ethambutol (15 mg/kg) daily for two months, followed by dual therapy with isoniazid and rifampin for ten months. His cutaneous lesions significantly regressed by leaving atrophic scars after treatment.

**Figure 1.** Erythematous, well-defined plaque over the left malar area and the left neck, and exophytic, ulcerated tumoral lesion on the middle plaque

**Figure 2.** A. Non-caseous granulomatous inflammation in the dermis (H&E x4). B. Well-differentiated squamous cell carcinoma with verrucous surface invading the dermis (H&E x4)
DISCUSSION
LV is often located on the face. Other sites of predilection are the nose, ears, chin, neck, and, rarely, extremities, buttock and trunk (5). In about one-third of cases, lupus vulgaris may develop at the site of scrofuloderma or in its vicinity (6). Sehgal et al. (7) suggested that LV is probably occurred by direct inoculation of the tuberculous bacilli into the abraded skin in a sensitized host, and suggested a diagnosis of secondary inoculation cutaneous tuberculosis for this entity. In our case, dissemination from an unknown primary focus to the skin and lymph nodes or primary inoculation is the most likely pathogenic mechanism. Although it is quite rare, especially in long-lasting LV lesions and scars, malignant transformation is an expected consequence. Most commonly reported malignancies, such as basal cell carcinomas can be seen (8,9), Bowen’s disease, syringoid eccrine carcinomas, melanomas and lymphomas were also reported (2). SCC may originate directly from LV lesions or from scar tissue. Duration between beginnings of LV lesions to development of carcinoma ranges from 2 to 79 years (8). In our case, SCC was detected on active LV plaque. Causes of lupus carcinoma remain obscure. In older cases, X-ray therapy has been an important factor in carcinogenesis. Possible factors triggering the malign transformation are chronic inflammation, cicatricial changes, physical and chemical trauma. Sunlight is an important factor especially on the head and neck tumors (8,10). In our patient, chronic inflammation on a sun-exposed area may contribute SCC development.

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