

Highly aggressive squamous cell carcinoma in an HIV-infected patient

Carcinoma de células escamosas altamente agressivo em um paciente HIV-positivo

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ABSTRACT

Unusually aggressive forms of cutaneous squamous cell carcinoma are being increasingly recognized as a complication of HIV infection. We report the case of a 59-year-old male patient with advanced HIV infection who presented with a highly aggressive SCC lesion over the scalp area with destruction of the underlying parietal bone and fulminant clinical progression.

Key-words: HIV infection. Squamous cell carcinoma.

RESUMO

Formas altamente agressivas de carcinoma cutâneo de células escamosas vêm sendo reconhecidas como uma importante complicação da infecção pelo HIV. Descrevemos o caso de um paciente do sexo masculino, de 59 anos, que se apresentou com uma lesão altamente agressiva de SCC na região do couro cabeludo, com destruição do osso parietal subjacente e curso clínico fulminante.

Palavras-chaves: HIV/Aids. Carcinoma espino-celular. Carcinoma de células escamosas.

Cutaneous squamous cell carcinoma (SCC), also known as epidermoid carcinoma, is a malignant neoplasm of the keratinizing epidermal cells and accounts for around one-fifth of all cases of nonmelanoma skin cancer¹. Important etiologic factors to the development of SCC are host characteristics, such as age and skin pigmentation, and environmental elements, the most important of which being long-term sunlight exposure⁸. Actinic keratosis is known to be a major precursor lesion of SCC¹⁵. Other predisposing factors include ionizing radiation, such as therapy with ultraviolet A, exposure to chemical carcinogens, especially arsenic, and long-standing benign dermatoses and scars¹. We report on the case of a human immunodeficiency virus (HIV)-infected patient who developed a rapidly growing SCC of the scalp with a fatal outcome.

CASE REPORT

A 59-year-old white male retired driver presented with a one-year history of chronic weight loss, asthenia, anemia, disseminated scabies, herpes zoster and several recent episodes

of bacterial pneumonia. He gave a past history of multiple, unprotected, sexual intercourse with male and female partners and tested positive for HIV antibodies. He had a CD4 cell count of 130/mm³ and an HIV plasma viral load of 4.9 log/ml. A highly active antiretroviral regimen and *Pneumocystis carinii* prophylaxis were prescribed but the patient was soon lost to follow up. One year later the patient sought our service with an indurated, erythematous papule over the scalp area with a diameter of 3cm. The lesion was biopsied and histopathologic studies diagnosed actinic keratosis and a well differentiated SCC (Figure 1D). The patient was again lost to follow up until six months later when he presented with an extremely large infiltrating, vegetative mass over the parietal area (Figure 1A and B). Computed tomography scan study showed extensive soft tissue involvement with destruction of the underlying parietal bone (Figure 1C). Magnetic resonance imaging (not shown) found no evidence of involvement of the brain parenchyma and the superior sagittal sinus. The patient was not considered eligible for antineoplastic therapy due to the parietal bone invasion and his declining general state. He died of overwhelming sepsis two months later.

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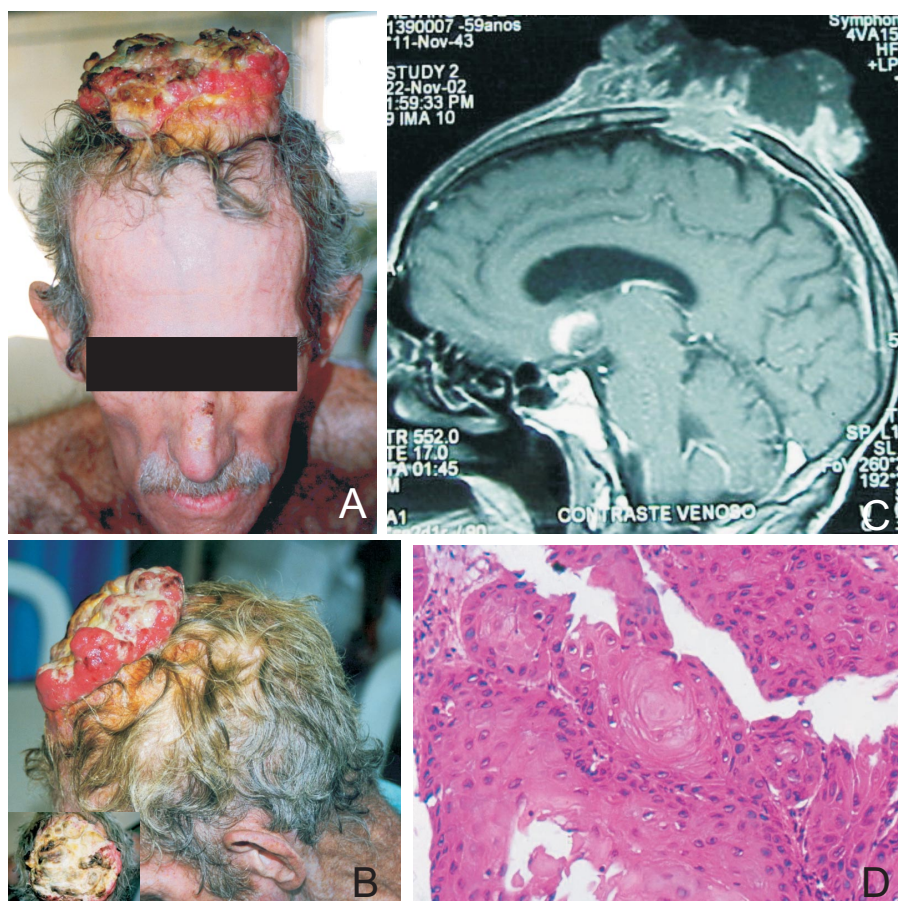


Figure 1 - Giant cutaneous squamous cell carcinoma of the scalp area. A) Frontal view of a large mass emerging from the parietal region; B) Lateral and upper (inset) view of the mass; necrotic areas can be seen; C) Sagittal view of the computed tomography scan study showing extensive soft tissue involvement and destruction of the parietal bone; D) histopathologic study demonstrates a well differentiated squamous cell carcinoma with irregular nests of epidermal cells, dermal invasion and keratin production (HE; 160x).

DISCUSSION

Cutaneous squamous cell carcinoma has long been associated with conditions leading to immunosuppression^{2, 13}. Epidemiologic investigations among subjects submitted to renal^{4, 5} and heart⁶ transplantation found the risk of SCC to be several times higher than in the general population. Patients prescribed immunosuppressant agents to treat diverse conditions such as inflammatory bowel disease and rheumatoid arthritis are also at an increased risk of developing SCC⁷. In face of these strikingly increased incidence rates of SCC among immunosuppressed patients, Kwa and cols⁸ stated in a 1992 review article that it was interesting that a similar increase had not yet been found in patients with AIDS.

Highly aggressive forms of SCC are increasingly being recognized among HIV-infected patients. Isolated case reports of aggressive SCC in the context of HIV infection date back to the early years of the epidemic^{11, 14}. In fact, it is known that SCC can present in unusually aggressive forms in association with immunodeficiency states such as renal transplantation⁴ and lymphoma^{3, 16}. Subsequent investigations found that the major predisposing factors to the development of SCC in association with HIV infection are a fair skin type, a positive family history, a past history of excessive sun exposure, and advanced stages of immunosuppression^{9, 10}.

Nguyen and cols¹² recently reported a case series of 10 patients diagnosed with aggressive SCC based on rapid growth rate, a diameter of over 1.5cm, a history of recurrence and/or evidence of metastasis. A total of 41 SCC lesions were recorded from these 10 patients. The head and neck were the most commonly involved sites (31 lesions), followed by the trunk (7 lesions) and extremities (1 lesion). Five patients had well differentiated tumors, 4 had intermediately differentiated lesions and 1 had a poorly differentiated SCC. The authors also found that patients initially undergoing combination surgery and radiation therapy or radical neck dissection had the best outcomes¹².

Our patient's fulminating clinical course, in conjunction with the reports cited above, highlight the importance of a rapid diagnosis and treatment of SCC lesions in HIV-infected patients if devastating growth of the primary lesion and metastatic spread are to be avoided. It is important to note that the development of SCC in HIV-infected patients seems to be determined by similar host and environmental predisposing factors as in the general population. Physicians caring for HIV-infected patients need to be aware of the possibility of an unusually aggressive behavior of SCC in such a setting. Primary prevention should be regularly instituted with sun avoidance and protection, as well as aggressive treatment of precancerous lesions such as actinic keratosis.

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