Kaposi’s sarcoma complicating long-term corticotherapy for rheumatoid arthritis

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INTRODUCTION

Kaposi’s sarcoma (KS) is a lymphoangioproliferative neoplasm induced by human herpes virus 8 (HHV-8). KS caused by iatrogenic immunosuppression is rare. Iatrogenic KS can present a therapeutic dilemma, as decreasing the level of immunosuppression may not be possible or may come at the high cost of organ rejection, such as in the case of organ transplant recipients or organ damage in the case of patients with autoimmune disorders. We report a new case of Kaposi’s sarcoma induced by corticosteroids.

CASE REPORT

A 44-year-old woman suffering from rheumatoid arthritis for 30 years treated with low doses of prednisone who developed Kaposi’s sarcoma (KS). Skin biopsy confirmed plaque-stage KS and imaging ruled out systemic involvement. Diagnosis of cutaneous iatrogenic KS was made, corticosteroid was gradually stopped and replaced by methotrexate to treat rheumatoid arthritis. The lesions regressed then relapsed. we opted for surgical excision of nodules with radiotherapy for lesions of the feet and legs with improvement.

DISCUSSION

Kaposi’s sarcoma has been reported in patients receiving immunosuppressive therapy, most of whom are organ transplant recipients. The development of Kaposi’s sarcoma after treatment with corticosteroids has been reported in only 38 patients who have not had acquired immunodeficiency syndrome or undergone organ transplantation [1]. Data from the literature clearly show that corticosteroid therapy may trigger the development of Kaposi’s sarcoma in patients who usually possess several other pathogenetic factors of that disease, such as pre-existing immune deficiency, environmental (viruses) or genetic factors [2]. In the literature only 8 rheumatoid arthritis patients who developed KS during steroid therapy have been reported [3]. In some cases, withdrawing corticosteroids...
may result in complete remission of the cutaneous lesions [4]. In-vitro studies suggest corticosteroids induce KS by indirectly inhibiting transforming growth factor beta, a protein that inhibits growth of endothelial cells [5]. In our patient, the lesions regressed after discontinuation of corticosteroid therapy but reappeared with methotrexate, prompting alternative treatment.

CONCLUSION

Maintaining immunosuppression leads to further progression of iatrogenic KS. However, restoring immune defenses by reducing or withdrawing immunosuppression may induce spontaneous KS remission and represents the first-line therapy for iatrogenic KS. Iatrogenic KS can present a therapeutic dilemma, as decreasing the level of immunosuppression may not be possible or may come at the high cost of organ rejection, such as in the case of organ transplant recipients or organ damage in the case of patients with autoimmune disorders. This case highlights the therapeutic challenge of iatrogenic KS, as reduction or removal of immunosuppression may not always lead to resolution of KS. In these cases or in cases in which reduction of immunosuppression may not be possible, other therapy such as surgery or radiotherapy should be considered.

REFERENCES