Nodular Kaposi's sarcoma associated with colon cancer

S. Majewski, A. Nawrocka, A. Wydrzynska, W. Owczarek, R. A. Schwartz

KEY WORDS

Kaposi's sarcoma, nodular Kaposi's sarcoma, colon cancer

SUMMARY

Kaposi's sarcoma (KS) is a multicentric, malignant neoplasm of a complex and still unclear etiology. We present the case of 87-year-old man referred to our dermatology department with the coexistence of two separate malignances: nodular KS and colon cancer. The patient observed the cutaneous nodules 3 months after colon cancer surgery. The question arises of whether the intestinal malignancy–associated weakened immune system exacerbated what was potentially preexisting but unapparent KS, or actually triggered development of KS.

Introduction

Kaposi's sarcoma (KS), first described in 1872 by Moritz Kaposi (1837–1902), is a multicentric, malignant neoplasm of unknown cause. It arises from endothelial cells, probably in a subgroup of lymphatic origin (1). The four main clinical subtypes of KS are: *classic*, most frequently observed in elderly men in Italy and Greece; *endemic* as seen in African (predominantly sub-Saharan) men; *iatrogenic* in transplant recipients or immunosuppressed patients (2), and *epidemic* HIV-related KS (3).The etiology of KS remains unclear. Because one of the key factors is infection with human herpes virus-8 (HHV8), present in all clinical subtypes of the disease (4), KS seems to be an interesting model of virus-associated transformation. However, only a small number of people infected with HHV8 develop KS. Therefore it seems to be a necessary component but still insufficient to entirely explain development of Kaposi's sarcoma (5).

Case report

A 87-year-old Polish man was referred to the dermatology department with a 6-month history of disseminated cutaneous nodules originally located on the right knee, then spreading to both elbows, forearms, and dorsal surfaces of hands. They were violaceous, discrete, and confluent, evolving into large tumors with an ulcerative surface (Fig. 1). There were also purple macules of varying size and shape on the extremities and pronounced edema of the upper left extremity. The patient denied being of Italian, Greek, or Jewish lineage. Three months prior to onset, the patient underwent a left hemicolectomy for a perforat-

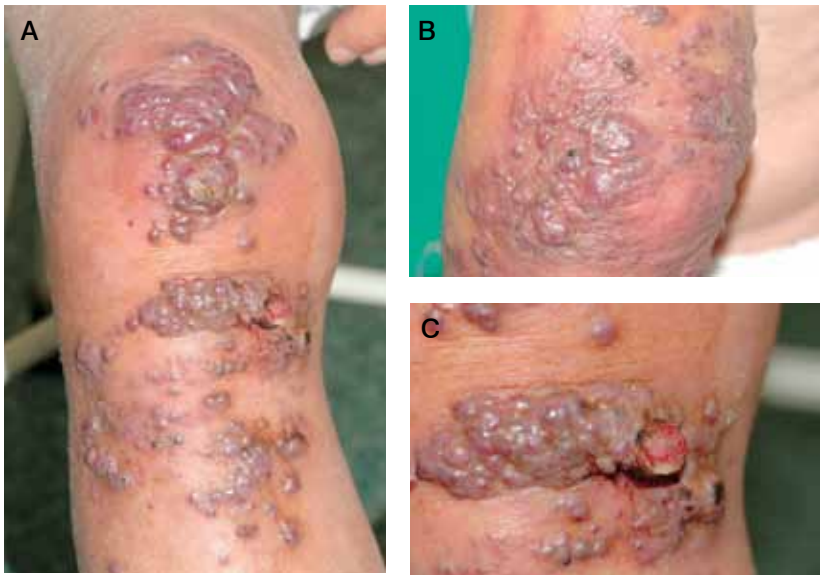

Figure 1. Nodular KS on the right knee (A), left elbow (B), with a close-up of the knee (C).

ing tumor of the descending colon, staged as tubular carcinoma G2, pT3, N0, and M1. Due to his advanced age and general clinical condition, the patient received no further gastroenterological oncological treatment. No violaceous nodules suggestive of KS were identified in the examined gut.

The laboratory findings revealed an elevated white blood cell count of 13.9 × 10^3/ml), mild anemia with hemoglobin 10.8 g/dl and hematocrit 32.6%, and a CEA marker level of 3.2 ng/ml (normal range < 3.0 ng/ml). A chest X-ray did not show any significant abnormalities. An ultrasound scan of the abdominal cavity documented a 10 mm gall stone in the gall bladder and a 23 × 21 mm structure in the second segment consistent with a colon cancer metastasis. A computed tomography scan of the abdomen also revealed an enlarged prostate and a cyst in the left kidney. An ultrasound scan of the edematous left upper limb did not show venous thrombosis.

A biopsy specimen from a violaceous nodule of the forearm showed eosinophilic spindle cells forming irregular slit-like vascular spaces, noticeable hemorrhage, and a chronic inflammatory infiltrate composed mainly of lymphocytes (Fig. 2). Most of the spindle cells expressed CD34, visualized with immunochemistry staining (Fig. 3).

The patient was given two courses of chemotherapy: dexamethasone, ondansetron, dacarbazine, doxorubicin, and vinblastine. The patient tolerated the administrated treatment very well and there was no further progression of the Kaposi's sarcoma lesions.

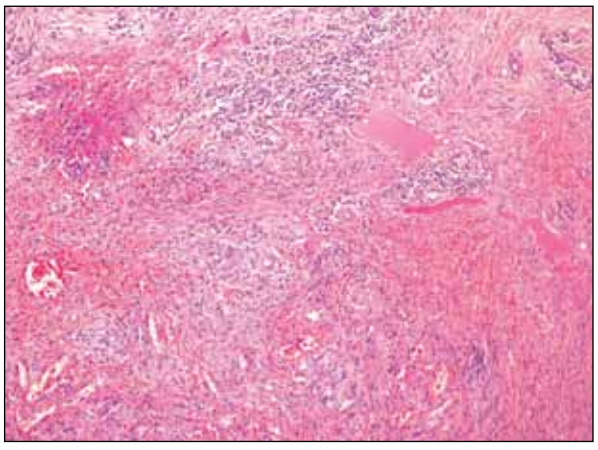

Figure 2. Kaposi's sarcoma: hematoxylin-eosin staining, original magnification ×100.

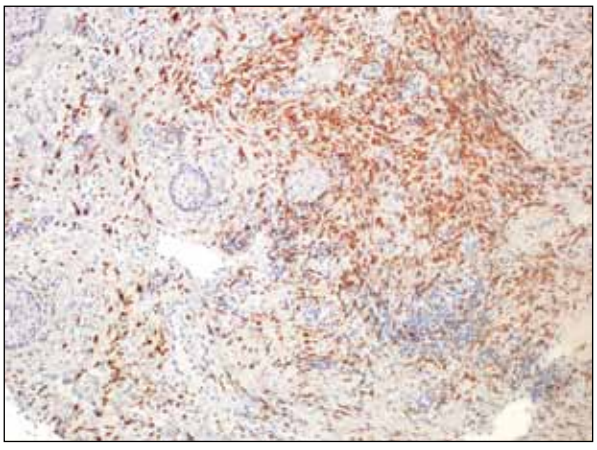

Figure 3. Immunochemistry staining of spindle cells with the use of CD34 marker, original magnification ×100.

Discussion

For many years, an increased incidence of secondary neoplasms in patients with KS has been observed. Several studies have demonstrated KS in association with non-Hodgkin's lymphoma, Hodgkin's lymphoma, leukemia, and breast cancer. Other primary neoplasms, involving the colon, lung, stomach, larynx, liver, pancreas, or kidney, were also reported, although less frequently (6, 7). Development of secondary KS seems to be mediated by mechanisms similar to those for hematopoietic neoplasms and certain non-hematopoietic neoplasms such as breast cancer (6). This report documents the coexistence of two separate malignances: nodular KS and colon cancer. The patient observed the cutaneous nodules 3 months after colon cancer surgery. Therefore, the question arises of whether the intestinal malignancy–associated weakened immune system exacerbated possibly preexisting but unapparent KS, or actually triggered development of KS.

REFERENCES

1. Schwartz RA, Micali G, Nasca MR, et al. Kaposi sarcoma: a continuing conundrum. J Am Acad Dermatol. 2008;59:179–206.
2. Hoshaw RA, Schwartz RA. Kaposi's sarcoma after immunosuppressive therapy with prednisone. Arch Dermatol. 1980;116:1280–2.
3. Haverkos HW. Multifactorial etiology of Kaposi's sarcoma: a hypothesis. J Biosci. 2008; 33: 643-51.
4. Mancuso R, Biffi R, Valli M, et al. HHV8 a subtype is associated with rapidly evolving classic Kaposi's sarcoma. J Med Virol. 2008;80:2153–60.
5. Dourmishev LA, Dourmishev AL, Palmeri D, et al. Molecular genetics of Kaposi's sarcoma-associated herpesvirus (human herpesvirus-8) epidemiology and pathogenesis. Microbiol Mol Biol Rev. 2003;67:175–212.
6. Iscovich J, Boffetta P, Winkelmann R, et al. Classic Kaposi's sarcoma as a second primary neoplasm. Int J Cancer. 1999;80:178–82.
7. Büyükpamukçu M, Varan A, Yazici N, et al. Second malignant neoplasms following the treatment of brain tumors in children. J Child Neurol. 2006;21:433–6.

AUTHORS' ADDRESSES

Sebastian Majewski, MD, Department of Dermatology, Military Institute of Medicine, Szaserów 128, 04-141 Warsaw, Poland, corresponding author, Tel. +48 604 770 317, Fax +48 22 8105520, E-mail: smajewski@gmail.com

Agnieszka Nawrocka, MD, same address

Anna Wydrzynska, MD, same address

Witold Owczarek, MD, same address

Robert A. Schwartz, MD, PhD, Department of Dermatology and Pathology, New Jersey Medical School, Newark, NJ, USA