Bibliografía

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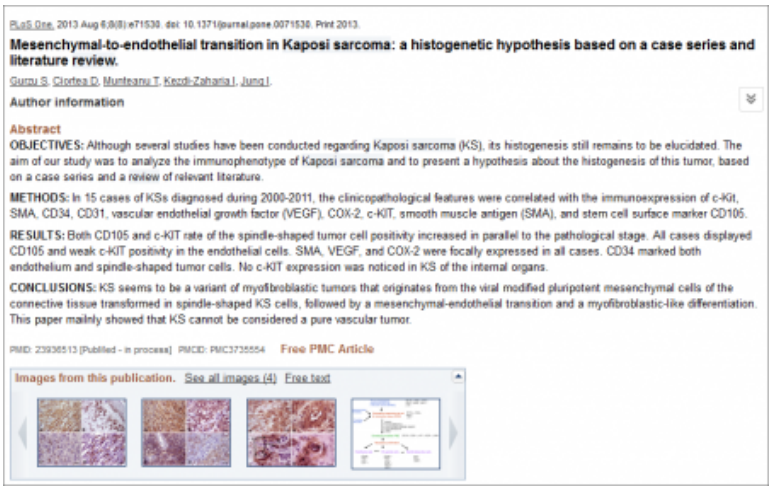

PLoS One. 2013 Aug 6;8(8):e71530. doi: 10.1371/journal.pone.0071530. Print 2013.

Mesenchymal-to-endothelial transition in Kaposi sarcoma: a histogenetic hypothesis based on a case series and literature review.

Gurzu S, Ciortea D, Munteanu T, Kezdi-Zaharia I, Jung I.

Author information

Abstract

OBJECTIVES: Although several studies have been conducted regarding Kaposi sarcoma (KS), its histogenesis still remains to be elucidated. The aim of our study was to analyze the immunophenotype of Kaposi sarcoma and to present a hypothesis about the histogenesis of this tumor, based on a case series and a review of relevant literature.

METHODS: In 15 cases of KSs diagnosed during 2000-2011, the clinicopathological features were correlated with the immunoexpression of c-Kit, SMA, CD34, CD31, vascular endothelial growth factor (VEGF), COX-2, c-KIT, smooth muscle antigen (SMA), and stem cell surface marker CD105.

RESULTS: Both CD105 and c-KIT rate of the spindle-shaped tumor cell positivity increased in parallel to the pathological stage. All cases displayed CD105 and weak c-KIT positivity in the endothelial cells. SMA, VEGF, and COX-2 were focally expressed in all cases. CD34 marked both endothelium and spindle-shaped tumor cells. No c-KIT expression was noticed in KS of the internal organs.

CONCLUSIONS: KS seems to be a variant of myofibroblastic tumors that originates from the viral modified pluripotent mesenchymal cells of the connective tissue transformed in spindle-shaped KS cells, followed by a mesenchymal-endothelial transition and a myofibroblastic-like differentiation. This paper mainly showed that KS cannot be considered a pure vascular tumor.

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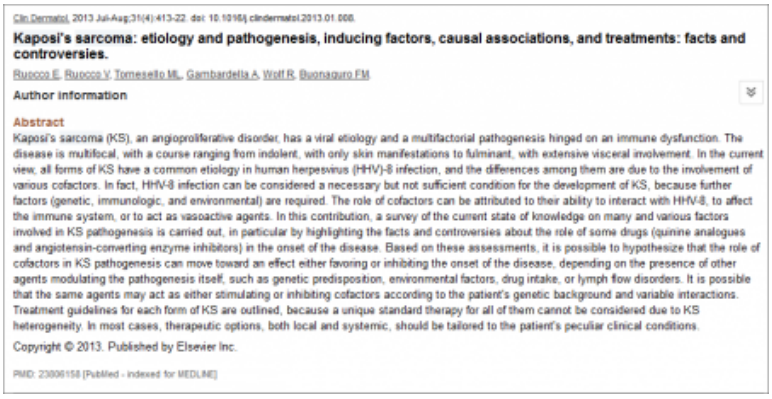

Clin Dermatol. 2013 Jul-Aug;31(4):413-22. doi: 10.1016/j.clindermatol.2013.01.008.

Kaposi's sarcoma: etiology and pathogenesis, inducing factors, causal associations, and treatments: facts and controversies.

Rupoco E, Rupoco V, Tomesello ML, Gambardella A, Wolf R, Buonaguro FM

Author information

Abstract

Kaposi's sarcoma (KS), an angioproliferative disorder, has a viral etiology and a multifactorial pathogenesis hinged on an immune dysfunction. The disease is multifocal, with a course ranging from indolent, with only skin manifestations to fulminant, with extensive visceral involvement. In the current view, all forms of KS have a common etiology in human herpesvirus (HHV)-8 infection, and the differences among them are due to the involvement of various cofactors. In fact, HHV-8 infection can be considered a necessary but not sufficient condition for the development of KS, because further factors (genetic, immunologic, and environmental) are required. The role of cofactors can be attributed to their ability to interact with HHV-8, to affect the immune system, or to act as vasoactive agents. In this contribution, a survey of the current state of knowledge on many and various factors involved in KS pathogenesis is carried out, in particular by highlighting the facts and controversies about the role of some drugs (quinine analogues and angiotensin-converting enzyme inhibitors) in the onset of the disease. Based on these assessments, it is possible to hypothesize that the role of cofactors in KS pathogenesis can move toward an effect either favoring or inhibiting the onset of the disease, depending on the presence of other agents modulating the pathogenesis itself, such as genetic predisposition, environmental factors, drug intake, or lymph flow disorders. It is possible that the same agents may act as either stimulating or inhibiting cofactors according to the patient's genetic background and variable interactions. Treatment guidelines for each form of KS are outlined, because a unique standard therapy for all of them cannot be considered due to KS heterogeneity. In most cases, therapeutic options, both local and systemic, should be tailored to the patient's peculiar clinical conditions.

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