Kaposi’s Varicelliform Eruption Associated With Meningitis by Herpes Simplex Type I

Erupção Variceliforme de Kaposi Associada à meningite por Herpes Simplex Tipo I

**ABSTRACT**

Kaposi’s varicelliform eruption (eczema herpeticum) is a serious infection caused principally by the herpes simplex virus 1. The typical clinical presentation is marked by the appearance of vesiculopapular lesions that spreads to involve the entire cutaneous surface. Here, we are reporting a case of Kaposi’s varicelliform eruption with systemic dissemination caused by HSV1 in a patient with no prior history of dermatoses or immunodeficiency. This report aims to promote the recognition of this potentially fatal condition, which can be treated effectively with early diagnosis.

**Keywords:** Kaposi Varicelliform Eruption, Exanthema, Rash, Herpes simplex virus

**RESUMO**

Erupção Variceliforme de Kaposi (eczema herpeticum) é uma infecção grave causada principalmente pelo herpes simplex virus 1. Sua apresentação clínica típica é marcada pelo aparecimento de lesões vesiculo papular que se espalham para envolver a totalidade da superfície cutânea. Neste estudo, relatamos um caso de erupção variceliforme de Kaposi com disseminação sistêmica causada pelo HSV1 em um paciente sem história prévia de dermatoses ou imunodeficiências. Este relato tem como objetivo promover o reconhecimento desta condição potencialmente fatal, que pode ser tratada de forma eficaz com o diagnóstico precoce.

**Palavras-chave:** Erupção Variceliforme de Kaposi, Exantema, Herpes simplex virus
INTRODUCTION
Kaposi's varicelliform eruption (eczema herpeticum), first described by Dr. Moritz Kaposi in 1887, refers to a skin rash that is caused by a variety of viruses (1), with 80% of infections attributed to herpes simplex virus type 1. Other viral causes are generally seen in patients with pre-existing dermatoses or a cellular or humoral immunodeficiency (2). Close human contacts usually provide the source of infection, and the viral particles generally enter through eczematous skin. Typically, widely disseminated vesicular lesions suddenly appear on the infected person's skin, and the patient develops a fever which persists for the first few days of the rash. The Lesions become umbilicated, then pustular and varicella-like (aspect varicelliform) with mass dissemination (3).

The infection is characterized by a vesicular eruption, which progresses to elementary umbilicated lesions. The lesions then crust over and disseminate after a few days, leaving widespread areas of denuded skin, hemorrhage, and scabbing. The cutaneous presentation is accompanied by fever, regional adenopathy and malaise. The lesions may be limited to the skin, or in more severe cases may involve internal organs, which jeopardizes the patient's life (4).

The diagnosis of Kaposi's varicelliform eruption is mainly derived clinically, but can be confirmed quickly with Tzanck smear (Giemsa coloration of herpetic vesicle fluid which allows the observation of intranuclear inclusions and multinucleated giant cells) (5). This report aims to promote the recognition of this potentially serious condition which can be treated effectively with early diagnosis.

CASE REPORT
A 52-year-old woman, native and current resident of the mountainous region of Junín, Perú, and with no prior history of any medical complaints, presented to the emergency room with a nine day history of subjective fevers, malaise, and a vesicular rash that began on the face and later spread over her entire body sparing the mucosal surfaces. Three days after the initial onset of symptoms, she developed abdominal pain, nausea, vomiting, and hemoptysis with associated dyspnea on moderate exertion. For these reasons, the patient was admitted to the local hospital. Upon initial exam, she was noted to have altered mental status, worsening respiratory distress, oliguria, and a poor general status which prompted transfer to our hospital. The patient's history revealed a family contact with a recent varicella diagnosis; the patient's 21-year-old daughter was diagnosed three weeks prior to the onset of her fevers and rash.

Soon after being admitted to our hospital, the patient experienced a generalized tonic-clonic seizure which lasted approximately 40 seconds. Physical exam showed deviations in her vital signs; she was febrile (38.6°C), tachypnic, and tachycardic. An innumerable, amount of umbilicated blood-filled vesicular lesions present on an erythematous base covered the patient's body, with some converging to leave areas of denuded skin and hemorrhage on the chest, upper, and lower extremities. Hemorrhagic bullous lesions and blood-crusted scabs were found on the palmar surfaces of her hands (Figures 1, 2, and 3).

Further examination of the mucosal surfaces revealed violaceous macular lesions on the oral hard palate. Additionally, there were decreased breath sounds in the lower third of the right hemithorax with diffuse bilateral rhonchi audible. Neurological exam revealed a GCS score of 13. The patient was easily distracted and failed to look at the examiner, but her pupils were reactive to light and isochoric. While the patient was able to move all four extremities spontaneously, she showed diffusely diminished deep tendon reflexes, equivocal plantar reflexes, nuchal rigidity, and a positive Kernig sign.

Figure 1. Diffuse vesicular lesions on face
Figure 2. Vesicular lesions on chest and lower limbs
Auxiliary tests:
Sat O2 86%, finger stick 53.
Later, an arterial blood gas (ABG) with FiO2 50% shows: pH 7.3, PCO2 = 26, HCO3 = 12.4, anion gap = 16, PO2/FiO2 = 280.
CBC: WBC = 8560, 12% (1027) bands, platelets = 56000
Urea = 187, Creatinine = 4.9, K = 6.66
Albumin = 2.3, total bilirubin = 6.6, direct bilirubin = 6.5, indirect bilirubin = 0.4, ALT = 1679, AST = 924, Alkphos = 487.
PT = 13.6, INR = 0.9, PTT = 43.6
HIV ELISA negative, HTLV-1,2 negative, Coombs test: positive 2+, CRP = 77
CXR: Multiple, diffuse radiopaque lesions in bilateral lung fields with a macronodular pattern.
Lumbar puncture: OP = 17cm, leukocytes = 32, lymphocytes = 100%, RBC 12, glucose 54, protein 109, ADA 1.6
Following the lumbar puncture, antiviral treatment was initiated (intravenous acyclovir 500mg q8h) and the patient subsequently showed improvement, supporting our clinical diagnosis of Kaposi's varicelliform eruption with systemic dissemination. The lesions continued to improve and the patient was discharged 17 days after admission to our hospital. The diagnosis of HSV1 was later confirmed with PCR (GoTaq® DNA Polymerase) using the cerebrospinal fluid sample.

DISCUSSION
The severe and usually fatal form of Kaposi's varicelliform eruption occurs with systemic spread that affects the lung, brain, and adrenal glands. The cutaneous lesions may be complicated by co-infection with bacteria (S. aureus being the most frequently isolated bacterial agent(8-9)).

Various mechanisms have been proposed to explain the pathophysiology of Kaposi's varicelliform eruption, with the first being reduced activity of natural killer cells, the first line of defense against HSV infection. Secondly, certain co-morbidities that increase concentrations of IL-4, which in turn inhibits Th-1 cells and suppresses the secretion of IFN gamma, can heighten the patient's susceptibility to HSV infection(4,10).

To obtain the most valid viral culture sample, fluid from the vesicles should be collected and stained with fluorescent antibody to directly observe the virus in infected cells. Atypical lesions may be biopsied for subsequent analysis with PCR (detects small amounts of viral tissue DNA) (4, 11). Currently, the best diagnostic tool is PCR, which able to detect HSV DNA in CSF, and has a high sensitivity (98%) and specificity (100%) (13). However, the presence Tzanck positive vesicles (Giemsa) combined with seizures in the clinical picture points to a diagnosis of herpetic meningoencephalitis(14-15).

Various complications exist for uncontrolled systemic HSV1 infection. Herpes simplex associated thrombotic microangiopathy may precipitate acute
renal failure which resolves with adequate hydration and treatment of the underlying disease. Herpes simplex encephalitis, which presents primarily as focal encephalitis of the temporal lobe, is considered a medical emergency, and mortality reaches 70% without early intervention. Given its high lethal potential, antiviral treatment should be started immediately (acyclovir 10 mg/kg every 8 hrs (30 mg/kg/d) for 14-21 days) without waiting for diagnostic confirmation like in the case above. Early antiviral treatment reduces mortality by 10%.

Corticosteroids in treating herpes encephalitis are used to reduce inflammatory changes and cerebral edema secondary to the immune response, and adjunct steroid therapy is currently recommended as a therapeutic option. However, some reports concluded that the use of adjunct corticosteroid therapy did not appear to attenuate the neurological sequelae. Other options such as immunoglobulins or immunostimulants have not proven to be efficacious.

Although cerebrospinal fluid analysis may reveal a normal cell count or only erythrocyte presence, several studies have concluded that early administration of antiviral therapy is the only parameter capable of modifying the prognosis and should be continued until herpes simplex virus encephalitis is clearly ruled out.

Acknowledgement: We would like to thank Casey Krebs (Weill Cornell Medical College), Jason Kahn (University of Medicine and Dentistry of New Jersey-School of Osteopathic Medicine) and Kara Walter for the review of this article.

REFERENCES


Correspondence
Raúl Montalvo Otívo
Hospital Daniel Alcides Carrión –Infectious Diseases
Av Daniel Alcides Carrión cuadra 16
Huancayo-Perú
E-mail: otivo3@hotmail.com