The patient developed urinary retention, persistent motor deficit, and worsening CSF leukocyte count on repeat lumbar puncture, raising suspicion of CMV polyradiculopathy. Ganciclovir was initiated, and given the possibility of refractory or resistant infection, foscarnet was added, totaling 28 days of combined therapy. CSF PCR for CMV returned positive. By the end of treatment, there was marked clinical response, with reduced CSF cellularity and progressive functional improvement. Electroneuromyography showed acute demyelinating sensory-motor polyradiculoneuritis consistent with Guillain–Barré syndrome. However, the context of severe immunosuppression and inflammatory CSF supported the diagnosis of a Guillain-Barré-like syndrome secondary to CMV meningo-radiculitis. This case illustrates the diagnostic complexity of neurologic manifestations in AIDS, in which opportunistic infections can mimic autoimmune diseases. Considering CMV, even without retinal signs or classic manifestations, was decisive given the subacute, refractory course, abnormal CSF, and lack of response to initial empiric therapies.

Keywords: Radiculopathy, CMV, Guillain-Barré syndrome, HIV.

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SARCOMA DE KAPOSI IN AN IMMUNOCOMPETENT OLDER ADULT WITH FATAL OUTCOME

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Human herpesvirus 8 (HHV-8), or Kaposi sarcoma-associated herpesvirus (KSHV), is a relatively recent scientific discovery and is found in patients with some degree of immune compromise, and it may progress to organ dysfunction and death. A 72-year-old male patient with sequelae of cerebrovascular accident. No other prior comorbidities or history of recurrent infections. One year earlier, he developed hardened edema of the lower limbs, associated with violaceous skin lesions. The lesions progressed to the trunk, genitalia, and upper limbs, as well as the appearance of similar lesions in the oral cavity. He presented clinical worsening due to secondary bacterial infection, requiring hospitalization. A skin lesion biopsy was performed, and the pathology report was compatible with superficial nodular chronic dermatitis, with red blood cell extravasation. Given the diagnostic uncertainty, a new cutaneous infection within one month, together with the onset of diarrhea, fever, and asthenia, the patient was referred to a tertiary center and clinical investigation was continued. Upon admission to a teaching hospital in the Center-West region of Brazil, he was febrile, anasarcous, and confused. On the plantar surfaces he had violaceous papules, some with honey-colored crusts, associated with local inflammation and purulent secretion. He underwent investigation for autoimmune diseases by the rheumatology team, with negative results for antinuclear factor (ANA), rheumatoid factor, and anti-cytoplasmic antibodies. Serologies were negative for human immunodeficiency virus (HIV) and syphilis. Fungal, bacterial, and mycobacterial cultures from a foot tissue sample were negative. The biopsy showed an intradermal spindle-cell lesion. Immunohistochemistry was compatible with a neoplasm of vascular histogenesis with positive human herpesvirus 8 (HHV-8), and Cluster of Differentiation CD31 and CD34 also positive, consistent with the hypothesis of Kaposi sarcoma. The patient died despite broad-spectrum antimicrobial therapy, before obtaining the results described above. Despite no known prior immunosuppression, we present a severe case of Kaposi sarcoma, with late diagnosis that made timely treatment impossible, culminating in death.

Keywords: Kaposi sarcoma, Human herpesvirus 8, Immunocompetent, Older adult, HHV-8.

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NEXT-GENERATION SEQUENCING APPLIED TO THE IDENTIFICATION OF CMV ANTIVIRAL RESISTANCE MUTATIONS: DEVELOPMENT AND VALIDATION OF AN ASSAY FOR CLINICAL USE

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Introduction/Objective: Cytomegalovirus (CMV), a member of the Herpesviridae family, is widely distributed globally and usually causes asymptomatic infection or self-limited symptoms, followed by latency for an indeterminate time in immunocompetent individuals. However, in immunosuppressed patients, it can trigger severe clinical manifestations due to reactivation. The emergence of strains resistant to the antivirals ganciclovir, foscarnet, cidofovir, maribavir, and letermovir represents a growing challenge in treatment. In this context, early detection of mutations associated with viral