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Cutaneous cytomegalovirus in mixed serostatus kidney transplant patient

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Abstract

Cutaneous cytomegalovirus infection is a rare but serious complication in solid organ transplant recipients. We present a 47-year-old male kidney transplant recipient with a chronic, nonhealing right lower extremity ulcer. Initial biopsies revealed septic vasculopathy, leading to treatment with sodium thiosulfate and antibiotics for suspected calciphylaxis. Despite regular wound care, the ulcer continued to worsen. After completing 6 months of cytomegalovirus prophylaxis, surveillance viral levels remained undetectable, but the ulcer progressed considerably. Worsening severity prompted hospitalization, during which cytomegalovirus viremia was detected, and an ulcer biopsy confirmed cytomegalovirus inclusion bodies. Antiviral therapy was reinitiated, resulting in rapid and sustained wound improvement. Therefore, this case underscores cytomegalovirus' potential for cutaneous invasion in transplant recipients, even without preceding viremia, and highlights the importance of considering cutaneous cytomegalovirus in nonhealing ulcers posttransplant, especially in serodiscordant recipients.

Keywords

infectious disease, pathology, wound, dermatology

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A 47-year-old male with a history of deceased donor kidney transplant complicated by graft dysfunction and donor cytomegalovirus (CMV) seropositivity, diabetes mellitus, and atrial fibrillation, presented for evaluation of a nonhealing ulceration of the right lower extremity (RLE) for 1 year. Four months posttransplant, the patient developed RLE swelling associated with multiple painful, necrotic, punched-out ulcers (Figure 1(a)). Biopsy at that time revealed numerous gram-negative bacteria within the deep dermis and medium-sized vessels, consistent with septic vasculopathy. The patient was treated with sodium thiosulfate and antibiotics for possible calciphylaxis with superimposed cellulitis. Over the next several weeks, the ulcers continued to progress despite regular wound care, prompting a second hospitalization. A repeat punch biopsy demonstrated a nonspecific ulcer consistent with ecthyma. Antibiotics were discontinued after tissue culture showed polymicrobial growth that was attributed to colonization. Of note, valganciclovir was discontinued at discharge as the patient had completed 6 months of posttransplant CMV prophylaxis per standard American Transplant Society Guidelines.[1] The patient was eventually readmitted for a third hospitalization due to new-onset CMV viremia of 1,200,000 IU/mL noted on outpatient surveillance labs. His wound had now progressed to a large, ulcerated plaque with islands of friable, exophytic granulation tissue, and chronic scarring (Figure 1(b)). A final incisional biopsy revealed extensive viral inclusion bodies with positive staining for CMV on immunohistochemistry (Figure 2). Bacterial wound cultures once again showed polymicrobial growth with various organisms. Dramatic sustained improvement was noted within 1 month of restarting antiviral therapy (Figure 1(c)). In addition, the patient's immunosuppressive regimen was changed given the CMV viremia. His initial regimen included cyclosporine and mycophenolic acid. Per the transplant team's recommendations, mycophenolic acid was discontinued, prednisone 5 mg daily was initiated, and cyclosporine was continued.

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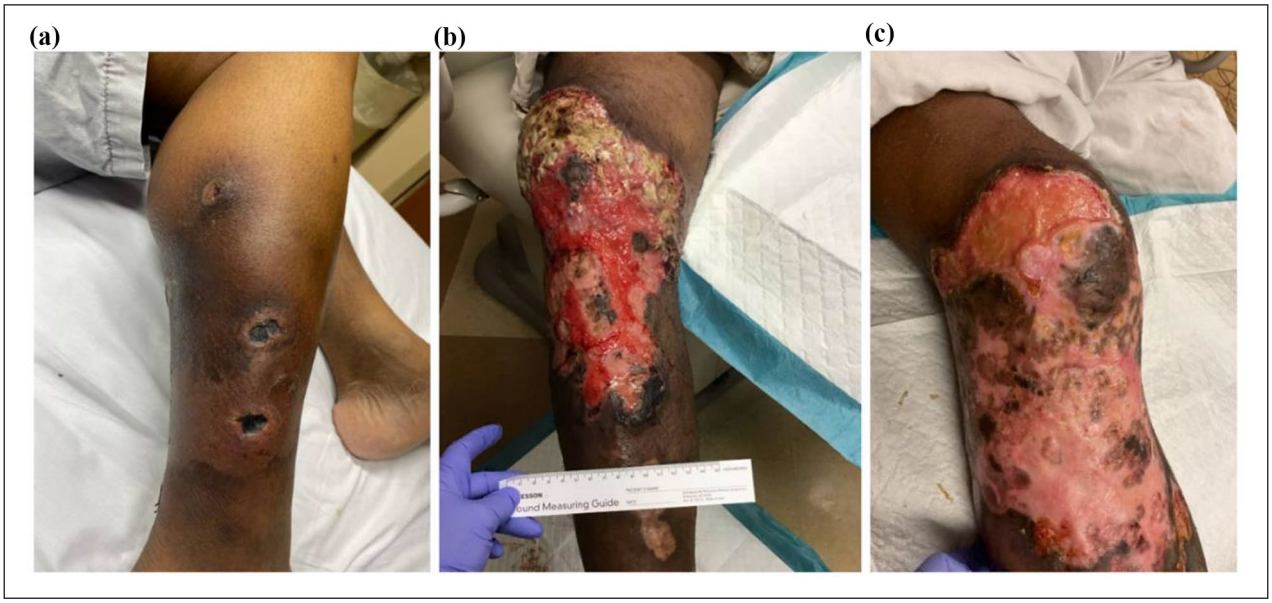

Figure 1. Initial presentation of punched-out ulcers with biopsy findings consistent with septic vasculopathy (a), ulcer progression over time without antiviral therapy (b), and rapid clinical improvement 1 month after restarting antiviral therapy (c).

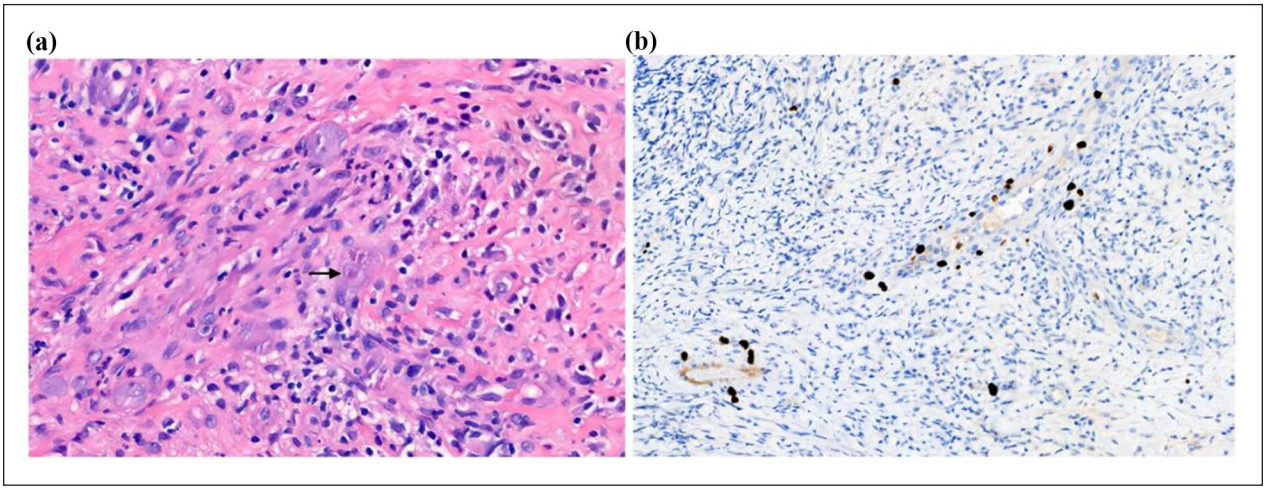

Figure 2. Histologic section showing enlarged, round, and polygonal infected cells with characteristic intranuclear Cowdry bodies (a) and CMV immunohistochemical stain highlighting multiple infected cells (b).
CMV: cytomegalovirus.

CMV disease in transplant recipients can involve various organ systems, most commonly the gastrointestinal tract, liver, and lungs.[2] Cutaneous manifestations, though rare and poorly understood, may present as morbilliform exanthems, pustules, petechiae, and ulcers.[3] Among these, perianal ulcerations are the most prevalent and occur secondary to viral shedding from the gastrointestinal tract.[3] Seronegative recipients of seropositive CMV donors, like our patient, are at higher risk of developing CMV infection and disease and are routinely placed on antiviral prophylaxis for the first 6 months after transplant.[1] Our case demonstrates a novel presentation of cutaneous CMV wherein viral infection likely resulted from the seeding of already damaged skin and soft tissue from the patient's donor graft following discontinuation of valganciclovir prophylaxis. Our argument for CMV superinfection is supported by the absence of CMV on initial biopsies, wound progression after discontinuation of antiviral prophylaxis, CMV detection in later biopsies, and significant clinical improvement with antiviral therapy. It is notable that the patient's surveillance CMV serum viral levels remained undetectable for several months during wound progression after the discontinuation of antiviral prophylaxis. Invasive end-organ CMV without preceding

viremia has been well documented in solid organ transplants.[4] Furthermore, CMV has demonstrated a predilection for vulnerable cutaneous tissue, particularly in immunocompromised individuals, where it exhibits accelerated tissue invasion.[5] This invasive process begins with the infection of compromised endothelial and perivascular stromal cells, leading to the development of an exanthem.[5,6] Without a robust immune response, this process may progress to vasculitis, characterized by infarcted, purpuric nodules that eventually ulcerate.[6] Therefore, CMV should remain on the differential diagnosis for nonhealing ulcers even in the absence of viremia, especially in serodiscordant transplant recipients like our patients. Collectively, our case demonstrates a rare cutaneous manifestation of tissue-invasive CMV with subsequent viremia and underscores the importance of considering cutaneous CMV in transplant recipients who discontinue antiviral prophylaxis.

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Consent to participate

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