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Case Report

Cutaneous Cytomegalovirus-Associated Ulceration Mimicking Pyoderma Gangrenosum in an Iatrogenically Immunosuppressed Patient: A Case Report

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Abstract: Cytomegalovirus (CMV) cutaneous ulceration is a rare but important mimic of inflammatory ulcerative dermatoses in immunocompromised patients. We report a case of a 43-year-old man with chronic inflammatory demyelinating polyneuropathy receiving rituximab, mycophenolate mofetil, and intravenous immunoglobulin who presented with progressive, painful anogenital ulcers initially diagnosed as pyoderma gangrenosum (PG). Despite escalating immunosuppression, including methotrexate and high-dose corticosteroids, the ulcers worsened. Multiple biopsies were performed over the disease course showing only nonspecific ulceration and were repeatedly negative for HSV; CMV was identified only when directed immunohistochemistry was applied to a repeat inpatient biopsy, revealing positive nuclear staining with nucleomegaly. CMV DNA was subsequently detected by PCR in both lesional tissue and blood (viral load 1099 IU/mL). HSV-1 was repeatedly detected from surface groin and oral specimens, although tissue-based HSV testing was negative; *Pseudomonas aeruginosa* was felt to represent colonization, contributing to diagnostic complexity. Ulceration was therefore best characterized as CMV-associated, with possible clinically significant CMV/HSV coinfection. Immunosuppression was reduced, and valganciclovir therapy was initiated, resulting in clinical improvement and resolution of viremia. This case highlights the importance of reconsidering the diagnosis in presumed PG that worsens with immunosuppression and emphasizes the need for early biopsy with directed viral testing. Recognition of CMV as an infectious mimic can prevent inappropriate escalation of immunosuppression and reduce morbidity in immunocompromised hosts.

Keywords: cytomegalovirus; herpes simplex virus; pyoderma gangrenosum; immunosuppression; genital ulcer; valganciclovir

1. Introduction

Pyoderma gangrenosum (PG) is a neutrophilic dermatosis and is a clinical diagnosis of exclusion; histopathology is supportive but not diagnostic. Misdiagnosis of PG can lead to inappropriate immunosuppression and worsening of occult infection, particularly in immunocompromised patients, where infectious mimics may be overlooked. Cytomegalovirus (CMV) cutaneous ulceration is rare, heterogeneous in morphology, and most often seen in iatrogenically immunocompromised hosts. The morphology can be nonspecific, and reported lesions may resemble vasculitic ulcers, inflammatory ulcers, or other ulcerative dermatoses. We report a case of CMV-associated cutaneous ulceration initially treated as PG with concurrent HSV-1 viral infection, bacterial colonization, and diagnostic confusion from a mycobacterial isolate. A review of the published literature on

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cutaneous CMV ulceration is presented to contextualize the clinical features, diagnostic challenges, and management of this entity.

2. Case

A 43-year-old man with chronic inflammatory demyelinating polyneuropathy (CIDP) managed with rituximab, intravenous immunoglobulin, and mycophenolate mofetil, presented to inpatient dermatology with progressive, exquisitely painful ulcers involving the groin, genitals, perineum, and oral mucosa. Initially, these painful, progressively enlarging inflammatory ulcers without an identified infectious cause led to an outside working diagnosis of superficial PG, prompting treatment with clobetasol and tacrolimus ointment. When the lesions failed to improve after 6 weeks, methotrexate was added; however, the ulcers progressed (Figure 1A). Over the next 2 months, superficial cultures grew *Pseudomonas aeruginosa* and *Mycobacterium fortuitum*, though the latter was considered a contaminant rather than the primary pathogen, given it grew from a single superficial wound culture, had a negative acid-fast staining, with absence of granulomatous inflammation. Methotrexate was discontinued, and the patient was started on escalating doses of prednisone (up to 60 mg daily).

He was admitted 10 days later with a pain crisis and worsening genital, perineal, and oral ulcers (Figure 1B). On admission the differential included PG, Behçet disease, autoimmune blistering disease, Crohn-related ulceration, and infection. Quantitative immunoglobulins and peripheral lymphocyte counts obtained during admission were within normal limits. Early wound and mucosal swabs demonstrated HSV-1 and mixed bacterial flora, including *P. aeruginosa* and *Citrobacter freundii* complex. The patient had severe pain, inability to sit or ambulate fully, and substantial functional decline; he failed to improve with wound care and piperacillin-tazobactam.

Inpatient dermatology review questioned the diagnosis of PG because the lesions had worsened despite substantial immunosuppression; additionally, there was no peripheral undermining, pathergy or associated inflammatory arthritis or enteropathy. Several biopsies were obtained over the disease course; specimens showed only nonspecific cutaneous ulceration with a mixed inflammatory infiltrate and were repeatedly negative for HSV (i.e., no herpetic cytopathic changes). As the ulcers progressed, a repeat punch biopsy of the left proximal anteromedial thigh was obtained for histopathology as well as tissue bacterial culture, fungal culture, mycobacterial culture, Leishmania culture/PCR, and CMV PCR. Histopathology demonstrated an ulcer edge with neutrophils and fibrin with a mixed inflammatory infiltrate extending into the papillary and upper reticular dermis. Special stains including PAS-F, GMS, Ziehl-Neelsen, HSV I/II, spirochete, and CD1a were negative, with no herpetic viral cytopathic changes identified. Notably, on the same tissue block, CMV immunostaining showed positive nuclear staining with nucleomegaly, supporting CMV-associated ulceration.

HSV-1 was repeatedly recovered from surface groin and oral swabs from admission onward, whereas CMV emerged only later (when specifically tested), on the final thigh biopsy. No oral mucosal biopsy was obtained, so oral CMV involvement could be neither confirmed nor excluded histologically. CMV DNA was then detected by NAT/PCR in blood (viral load 1099 IU/mL) as well as a lesional swab. Importantly, tissue-based HSV testing was negative across all biopsies, and HSV-1 was recovered only from surface groin and oral specimens. Superficial bacterial cultures repeatedly yielded *P. aeruginosa* (suggesting colonization rather than a primary driver of disease), while Leishmania PCR was negative and fungal studies were unrevealing.

Management shifted from immunosuppression toward antimicrobial therapy and wound support. Prednisone was rapidly tapered; mycophenolate mofetil was discontinued and further doses of rituximab and IVIG were held in coordination with neurology. Valganciclovir 900 mg twice daily was initiated and continued for approximately 6 weeks, with infectious diseases follow-up planned after discharge. As there are no guidelines for isolated cutaneous CMV disease, the duration was not fixed, and therapy was continued under the direction of infectious disease physicians, until the CMV viremia cleared and the ulcers showed sustained re-epithelialization. Wound care consisted of gentle cleansing with sterile water, topical silver sulfadiazine 1% cream, a catalytic advanced wound-care matrix, and a petrolatum-based barrier beneath a non-adherent dressing or directly on the wound. Acetic acid soaks were attempted for *Pseudomonas* control but discontinued because of pain and poor tolerance. Pain management required multimodal therapy including hydromorphone initially and later topical lidocaine, gabapentin, amitriptyline, and scheduled acetaminophen.

Following valganciclovir initiation and reduction of immunosuppression, the lesions gradually dried and crusted, with visible re-epithelialization and slow but clear clinical improvement (Figure 1C,D). Valganciclovir was completed as planned without treatment-related adverse events. At 6-week outpatient follow-up, the patient had completed valganciclovir with resolution of CMV viremia; repeat ulcer testing was negative for CMV and HSV PCR. The patient's pain and wound burden improved slowly, though a substantial residual wound required ongoing outpatient dermatology and wound care follow-up.

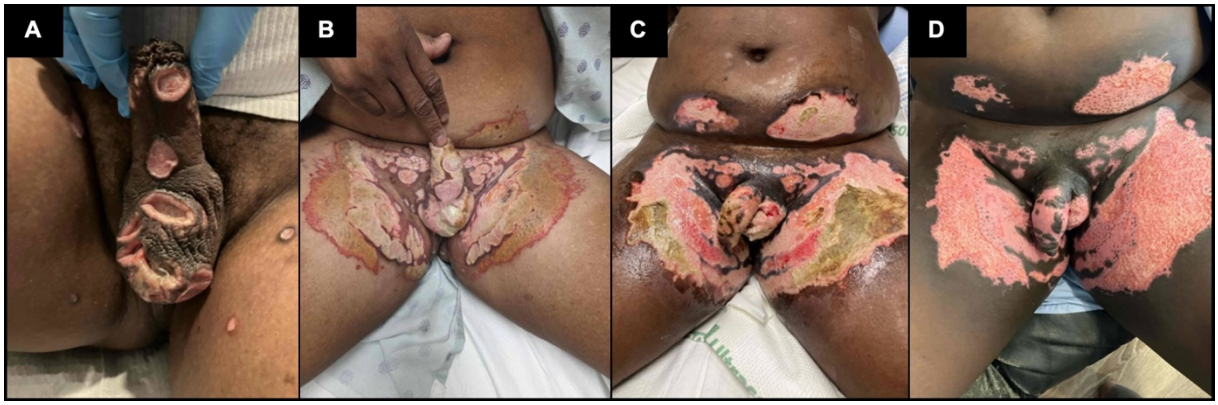


Figure 1. Clinical images demonstrating the progression of the cutaneous CMV infection (**A**,**B**), followed by re-epithelization after discontinuing immunosuppressant therapies and initiating valganciclovir (**C**,**D**).

3. Discussion

This case illustrates several important diagnostic and management principles relevant to the evaluation of refractory anogenital ulcers in immunocompromised patients. First, progressive ulceration despite escalating corticosteroids, methotrexate, and background immunosuppression should raise concern that a presumed inflammatory diagnosis may be wrong or incomplete. PG remains a diagnosis of exclusion, and misdiagnosis is well documented in the literature. Weenig et al. found that 10% of patients treated for PG at a single institution ultimately had an alternative diagnosis, most commonly vascular disease, vasculitis, malignancy, or primary infection, with a median of 10 months of inappropriate therapy received; in 12% of cases, immunosuppressive treatment actively worsened the underlying condition [1]. Misdiagnosis remains frequent despite the availability of clinical diagnostic frameworks and contributes directly to increased morbidity and mortality [2]. Since PG is a diagnosis of exclusion, a thorough workup, including biopsy in most cases, is important to evaluate for mimics, especially if the presentation is atypical or fails to respond to treatment. In this case, the patient's immunosuppressive regimen including rituximab, mycophenolate, methotrexate, and high-dose corticosteroid may have contributed to the risk of CMV reactivation. Rather than controlling ulceration, escalating immunosuppression may have contributed to ulcer progression by allowing CMV viral proliferation [3]. The simultaneous withdrawal of multiple immunosuppressive agents and initiation of antiviral therapy makes it impossible to determine the independent contribution of each intervention.

Second, CMV-associated cutaneous disease may present with nonspecific ulceration and may only be recognized when biopsy material is subjected to directed CMV testing including immunohistochemistry or PCR. In this case, histopathology showed an ulcer edge with neutrophilic inflammation, a pattern seen in both infection and PG, with special stains returning negative. Only CMV immunohistochemistry revealed positive nuclear staining with nucleomegaly, prompting investigation of serum CMV found to be elevated at 1099 IU/mL. Hartsough et al. reported a 12% false-negative rate for CMV detection by light microscopy alone and highlighted that immunohistochemistry improves detection and may be less dependent on the patient's stage of viral infection [4].

Reported cases of cutaneous CMV ulceration, limited to case reports and small series are summarized in Table 1, and offer useful context. Anogenital ulceration appears to be the most commonly reported presentation, followed by oral involvement, while concurrent oral and anogenital disease, as in our patient, is uncommon (Table 1). The anogenital predilection may be attributed to autoinoculation from urinary or fecal viral shedding [5]. Most reported cases have occurred in solid organ transplant recipients, with fewer among patients with hematologic malignancies, autoimmune disease, and HIV. Reported management most often consisted of intravenous ganciclovir or oral valganciclovir, with reduction or discontinuation of immunosuppression as adjunctive treatment (Table 1). Outcomes have generally been favorable for patients with timely recognition and therapy, although deaths have been described, typically in the setting of delayed diagnosis, systemic dissemination, or complications of the underlying disease. No guidelines exist for antiviral treatment specific to cutaneous CMV; management is based on recommendations for other end-organ disease [3,6].

Table 1. Narrative summary of reported cases of cutaneous cytomegalovirus (CMV) infections.

Ref No.	Reference	Year	Country	Age/Sex/Initial Suspected Diagnosis	Comorbidities & Immunosuppressant Therapy (IS)	Anatomic Site	Co-Infections	Management/Outcome
1	[7]	2003	USA	48-F	Rheumatoid arthritis IS: Pred, AZA, MTX	Peripheral ulcers	None	Ceftazidime, doxycycline, Bactrim for pneumonia IV ganciclovir & foscarnet Died 24 days following admission
2	[5]	2006	South Korea	Case 1: 15-M	5 hematologic malignancies, 2 liver transplants, 2 kidney transplants IS: not reported	Case 1: Diffuse rash	HSV-2 infection in 1/9 patients	All treated with IV ganciclovir All skin lesions resolved within 1 or 2 months 3 patients with hematological malignancies died due to worsening of their disease
				Case 2: 54-M		Case 2: Multiple ulcers to buttocks		
				Case 3: 73-M		Case 3: Multiple ulcers to buttocks		
				Case 4: 24-F		Case 4: Multiple ulcers to buttocks		
				Case 5: 6 Mo-M		Case 5: Solitary nodule to back		
				Case 6: 54-M		Case 6: Multiple ulcers to buttocks		
				Case 7: 57-M		Case 7: Solitary nodule anus		
				Case 8: 31-M		Case 8: Multiple ulcers to penile shaft		
				Case 9: 62-F		Case 9: Multiple ulcers to perineum All cases: Anogenital ulcers (66.7%); other cutaneous sites		
3	[8]	2008	Australia	Case 1: 39-F HSV infection	Renal transplant IS: CsA, MMF, Pred	Perianal ulceration	HSV2	IV valacyclovir, then oral valganciclovir x2 weeks, then PO ganciclovir 1 g BID x3 months Survived; resolved after 3 months
				Case 2: 62-F HSV infection	Renal transplant IS: CsA, MMF, Pred, basiliximab	Lateral tongue ulcer followed by diffuse petechial rash to face	Not reported	Valacyclovir 500 mg TID, MMF dose reduced, then stopped. Pred reduced. Valganciclovir BID x3 weeks Survived; full resolution
				Case 3: 34-M CMV reactivation	Renal transplant IS: CsA, MMF, methylprednisolone, daclizumab. 3-month prophylactic oral ganciclovir	Deep tongue ulcer, nodular erythematous lesion right pinna	Not reported	Oral ciprofloxacin & doxycycline; IV ticarcillin-clavulanic acid, gentamicin; MMF stopped 1 week prior to admission PO/IV ganciclovir for total of 3 weeks; discharged on reduced MMF dose and valacyclovir x2 months as prophylaxis Full resolution of ulcers. Patient died shortly after from myocardial infarction
4	[9]	2008	USA	67-M HSV, EBV, CMV, drug eruption	Renal transplant IS: Thymoglobulin, corticosteroids, MMF, Tacro CMV prophylaxis: valganciclovir 900 mg daily x6 months	3 oral ulcers to left lower gingiva, right buccal mucosa, right lateral tongue	None (HSV, CVC, EBV ruled out)	Tacro lowered, MMF switched to AZA; PCN, IV clindamycin x5 days; prophylactic valganciclovir discontinued Survived; ulcers resolved after lowering immunosuppressant dosing
5	[10]	2008	Brazil	30-F Not specified	Renal transplant; IS: Tacro, Pred, AZA, MMF	Oral ulcers to buccal, palate, premolar region, superior fore teeth, inferior lip mucosa	HSV, Candida albicans	Acyclovir (1 g daily x10 days); topical nystatin gargles Survived; full resolution

Table 1. *Cont.*

Ref No.	Reference	Year	Country	Age/Sex/Initial Suspected Diagnosis	Comorbidities & Immunosuppressant Therapy (IS)	Anatomic Site	Co-Infections	Management/Outcome
6	[11]	2009	Spain	Case 1: 65-F	Pred, Tacro, MMF	Oral ulcers to hard palate & buccal mucosa	Not reported	IV ganciclovir All survived; early diagnosis emphasized as key
				Case 2: 49-F	Pred, CsA, MMF	Oral ulcers to buccal mucosa, soft palate, floor of mouth		
				Case 3: 64-F	Pred, Tacro, MMF	Buccal mucosa ulcer		
				Case 4: 48-F	Pred, CsA, MMF	Tongue and soft palate ulcers		
				Case 5: 48-M	Pred, CsA, Aza	Buccal mucosa and hard palate ulcers		
				Case 6: 30-M	Pred, Tacro, MMF	Ulcers to floor of mouth		
7	[12]	2014	Portugal	41-F HSV genital ulcer	HIV; HARRT therapy: tenofovir/emtricitabine, raltegravir, ritonavir, darunavir	Vulvar ulceration	HSV	Oral acyclovir, several oral/topical antifungals, oral antibiotics IV acyclovir x4 weeks; valganciclovir 900 mg BID x6 months Survived; complete resolution after valganciclovir
8	[13]	2015	USA	64-F Septicemia, cholestatic jaundice, hepatic encephalopathy	Mixed connective tissue disease, interstitial lung disease IS: Pred, AZA 6 weeks prior to presentation, Pred increased from 2 mg to 40 mg daily	Bilateral lower extremity bullae and ulcerations	Not reported	Ceftazidime, ciprofloxacin; broadened to meropenem; AZA was stopped; prednisone dose was tapered off IV ganciclovir Survived, full resolution
9	[14]	2016	Iran	M (age not specified) HSV infection	Renal transplant; IS: Alemtuzumab, methylprednisolone induction, then MMF, Tacro, Pred	2 ulcerative lesions on the face (one on lower lip, one on left nasolabial fold)	HSV	IV ganciclovir x4 weeks Survived; resolved
10	[15]	2016	USA	76-F Disseminated shingles, herpes infection	Sjogren's syndrome, interstitial lung disease Chronic IS: MMF, Pred, hydroxychloroquine, and rituximab	Ulcers to lower back, buttocks, perineum, and oral mucosa	None	TCS, topical nystatin, acyclovir x7 days Valganciclovir 450 mg BID x14 days, then continued indefinitely. Prednisone reduced, MMF, rituximab, hydroxychloroquine all discontinued
11	[16]	2016	Japan	48-F Not specified	TEN; IS: Pred, IVIG, PLEX	Ulcers to the lower lip after TEN-related lesions were healing	Not reported	Ganciclovir (route/duration not specified) Complete resolution
12	[16]	2016	Japan	72-F CMV-related ulcers vs rheumatoid arthritis related ulcers	Rheumatoid arthritis; IS: high dose Pred, MTX	Multiple ulcers to bilateral lower extremities	Not reported	Died of respiratory failure from known cause prior to receiving CMV related treatment
13	[17]	2019	Japan	57-F Recurrent aphthous stomatitis, HSV stomatitis	Kidney transplant; IS: basiliximab, Pred, MMF, Tacro	Oral ulcers to the tongue and upper palate; bullous ulcers to left cheek and index finger	HSV, Candida species	Oral valacyclovir x1 month, then oral valganciclovir. Skin lesions resolved, oral ulcers persisted Switched to IV ganciclovir x3 weeks, oral valganciclovir x1 month maintenance, oral ulcers resolved Survived, full resolution
14	[18]	2021	Iran	20-F SLE flare	SLE; IS: Pred, hydroxychloroquine. Pred increased, AZA initiated	Ulceration to the right knee, left elbow, bilateral legs, perianal region	Oral candidiasis	Oral acyclovir 800 mg TID, oral fluconazole daily, IV amikacin TID, IV clindamycin TID, oral levofloxacin BID. Prednisolone does tapered IV ganciclovir x12 days, discharged with oral valganciclovir (450 mg daily) as maintenance therapy

Table 1. *Cont.*

Ref No.	Reference	Year	Country	Age/Sex/Initial Suspected Diagnosis	Comorbidities & Immunosuppressant Therapy (IS)	Anatomic Site	Co-Infections	Management/Outcome
15	[19]	2021	Ireland	66-F Crohn's flare, impetigo, HSV	Renal transplant, Crohn's disease; IS: infliximab, mesalamine, Pred	Multiple ulcers to the back & lower extremities	HSV-1 (positive on skin swab; IHC negative on autopsy); Pseudomonas aeruginosa	Penicillin G & piperacillin-tazobactam, broadened to meropenem & vancomycin Valganciclovir started, died on hospital day 6
16	[20]	2021	India	46-M Not specified	Renal transplant; IS: Tacro, MMF, steroids	Erythematous to violaceous plaque on the ankle (protuberant growth)	Klebsiella pneumonia	IV antibiotics & antifungals. Tacro reduced, MMF stopped IV ganciclovir x7 days, oral antivirals x14 days Died (secondary sepsis)
17	[21]	2022	Taiwan	74-M Cellulitis vs herpes zoster	Large B-cell lymphoma; IS: Rituximab, cyclophosphamide, doxorubicin, vincristine, prednisolone	A band of erythematous lesions on the abdomen (no ulcers)	Not reported	Ganciclovir + CMV IVIG x2 days, then foscarnet, valacyclovir and antibiotics (type not specified) Died (multi-organ failure, 1 month later)
18	[4]	2024	USA	Case 1: 79-M HSV, squamous dysplasia, early LS/LP	Early lichen sclerosis vs lichen planus IS: TCS, ILS, topical, TCI (tacrolimus), acitretin, injected fluorouracil	Peripheral ulcers	Not reported	Oral acyclovir x5 days. 3-weeks of valganciclovir, then continued on lower maintenance dose
				Case 2: 85-M Not specified	Metastatic Merkel cell carcinoma IS: MMF, Tacro	Right thumb purpuric plaque	Not reported	Valganciclovir Died from multiorgan failure 1 month after initiation
				Case 3: 47-M Not specified	Untreated HIV/AIDS, CMV retinitis IS: None reported	Left perianal ulcer	Not reported	IV ganciclovir x2 weeks then transitioned to oral valganciclovir Resolution not specified
19	[3]	2025	USA	Case 1: 71-M Herpetic infection	Hodgkin's lymphoma, primary biliary cirrhosis; IS not reported	Perianal polycyclic ulcerations	None	IV ganciclovir, with improvement over 17 days Died from septic shock & multiple organ dysfunction
				Case 2: 55-M HSV infection	Liver transplant for primary sclerosing cholangitis & cholangiocarcinoma; IS: MMF, Tacro, Pred	Suprapubic & penile ulcers	HSV	IV ganciclovir, resulting in improvement of the rash, treated x1 month Ulcers resolved but later died due to pneumonia
				Case 3: 69-F HSV infection	Non-small cell lung cancer; IS: carboplatin pemetrexed	Multiple ulcers to buttocks & gluteal cleft	None	Over 1 month of IV valganciclovir, then prophylactic valacyclovir dosing
				Case 4: 75-F HSV infection vs allergic contact dermatitis	Colon adenocarcinoma & breast cancer IS: Not reported	Multiple ulcers to buttocks & gluteal cleft	HSV	IV ganciclovir x14 days with improvement in ulcerations Patient died after pursuing hospice treatment
20	This paper	2026	Canada	Case 5: 43-M Pyoderma grangrenosum	Chronic inflammatory demyelinating polyneuropathy (CIDP) IS: Rituximab, IVIG, MMF, MTX, TCS, TCI	Oral & perianal mucosa	HSV-1, pseudomonas aeruginosa	Valacyclovir, piperacillin-tazobactam, reduction to MMF, stopped Rituximab Valganciclovir Survived/resolution of ulcers

CsA = Cyclosporin; MTX = Methotrexate; AZA = Azathioprine; Tacro = Tacrolimus; Pred = Prednisone; MMF = Mycophenolate mofetil; PCN = Penicillin; TCS = Topical corticosteroids; TCI = Topical calcineurin inhibitor; HSV = Herpes simplex virus; CMV = Cytomegalovirus.

Third, co-infection or colonization can coexist within CMV-associated ulcers, and distinguishing the two is often difficult [4]. In this case, HSV-1 and *P. aeruginosa* were repeatedly detected from groin and oral specimens; however, HSV testing on biopsy tissue was consistently negative and *P. aeruginosa* was felt to represent colonization rather than a primary driver of disease. The absence of granulomatous inflammation and negative acid-fast staining on multiple biopsies, including the biopsy that was positive for CMV, argued against true *M. fortuitum* infection, and the isolate was judged to represent contamination. Notably, detection of HSV-1 from surface specimens does not establish a tissue-level causal role, and a negative tissue HSV stain does not fully exclude an HSV contribution to the ulceration. Beyond its role as a diagnostic confounder, the repeated recovery of HSV-1 from mucocutaneous sites raises the possibility of a clinically significant CMV/HSV coinfection, as both viruses can co-occur and contribute to mucocutaneous ulceration in immunocompromised hosts. For this reason, and given the concurrent bacterial isolates, CMV-associated ulceration is the more accurate term than implying CMV was the sole cause. The potential for polymicrobial infection in immunocompromised hosts makes comprehensive microbiologic evaluation essential [5]. The presence of oral lesions alongside genital ulcers may initially suggest Behçet disease or severe HSV, but CMV should be considered in heavily immunosuppressed patients.

A strength of this case is its objective documentation of the disease course: serial photography (Figure 1A–D) captured both ulcer progression and re-epithelialization, and CMV was confirmed by immunohistochemistry, lesional and blood PCR. The main limitation is that valganciclovir was started and immunosuppression was reduced at the same time, with HSV-1 and bacteria also present, so improvement cannot be attributed to any single intervention, and CMV cannot be identified as the sole cause. From a practical standpoint, this case supports early biopsy in refractory anogenital ulcers and simultaneous evaluation for HSV, CMV, bacterial infection, fungal infection, and atypical pathogens. In patients already receiving immunomodulators, the threshold to diagnose PG should be high if the disease is worsening or non-responsive. CMV should be considered in heavily immunosuppressed hosts, especially if worsening despite appropriate immunosuppressive therapy [3].

4. Conclusions

Cutaneous CMV-associated ulceration should be considered in immunosuppressed patients with progressive anogenital or mucosal ulcers that do not behave as expected for PG. Tissue-based CMV viral testing is essential as biopsy morphology may be nonspecific. Early recognition of cutaneous CMV-associated ulceration can prevent further immunosuppressive escalation and allow timely antiviral therapy, reducing overall patient morbidity.

Author Contributions

R.A.D. and C.R.J.: Conceptualization, investigation, data curation, visualization, writing (original draft preparation), and writing (reviewing and editing). All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

Ethical review and approval were waived for this study due to the fact that this is a case report of a single patient's clinical course, which does not require research ethics board review under institutional policy. Written informed consent for publication of this case report and accompanying clinical images was obtained from the patient.

Informed Consent Statement

Written informed consent for publication of this case report and accompanying images was obtained from the patient.

Data Availability Statement

Data supporting the findings of this case report are contained within the article. Additional clinical data are not publicly available to protect patient privacy and confidentiality, consistent with the terms of the patient's consent for publication.

Conflicts of Interest

The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

A generative AI tool (Figure Labs AI) was used only to create the graphical abstract. No AI or AI-assisted technologies were used to write the manuscript or to produce the clinical images or tables.

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