

Case Report

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Infection or Malignancy, or Both? Kaposi Sarcoma Complicated by MRSA Infection: A Case Report

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ABSTRACT

Background: Kaposi sarcoma is an angioproliferative malignancy that presents as violaceous, pink, or red macules, plaques, or nodules. It is caused by human herpesvirus-8 infection of endothelial cells and primarily occurs in immunocompromised individuals, mainly those with human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS). These lesions may sometimes mimic or be misdiagnosed as cellulitis. Human herpesvirus -8 can also infect B cells, leading to cytokine-mediated syndromes such as multicentric Castleman disease, inflammatory cytokine syndromes, and lymphoproliferative disorders including primary effusion lymphoma and large B-cell lymphoma.

Case Presentation: A 46-year-old male with hypertension and prior methicillin-resistant *Staphylococcus aureus* (MRSA) cellulitis presented with facial swelling and presumed cellulitis. Further assessment revealed multiple violaceous papules across the body. HIV/AIDS was diagnosed with a markedly reduced CD4 count during workup for disseminated rash and recurrent infections. CT maxillofacial imaging demonstrated a soft tissue mass of the left palate with bony erosion and inflammatory changes. Chest CT angiography showed innumerable bilateral mass-like opacities concerning for metastatic disease or septic emboli. Left facial wound, blood, and bronchoalveolar lavage cultures grew MRSA. Skin biopsy from the chest wall lesions and palatal mass confirmed Kaposi sarcoma. The patient was treated with intravenous daptomycin per infectious disease recommendations and paclitaxel per oncology guidance, with subsequent clinical and radiographic improvement.

Conclusion: We conclude that history-taking, physical examination, and multidisciplinary collaboration are critical for timely diagnosis and management. In this case, recurrent cellulitis and disseminated rash prompted evaluation for underlying immunosuppression, leading to HIV diagnosis and appropriate treatment with favorable outcomes.

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Background

Kaposi Sarcoma (KS) is an angioproliferative tumor that mostly presents as cutaneous lesions, which appear as purple-blue macules, papules, and nodules, and was named after Viennese dermatologist Moritz Kaposi, who first described it as “idiopathic multiple-pigmented sarcoma of the skin” in 1872[1]. In 1994, human herpesvirus-8 (HHV-8) was first identified in these lesions and later confirmed as the etiology of KS. Moreover, there are four types of Kaposi sarcoma was identified, 1) classic KS: identified among mediterranean elder men, 2) Endemic KS identified among adults and children in sub-Saharan Africa, 3) Iatrogenic KS identifies on organ transplant patients, and 4) Epidemic KS which is associated with acquired immunodeficiency syndrome (AIDS) secondary to human immunodeficiency virus (HIV) and

subtype identified among HIV -negative men who have sex with men [2].

A patient who has untreated HIV would develop AIDS, causing a drop in CD4 count to less than 200 cells/mm³, which activates the dormant HHV-8, causing cutaneous lesions or Kaposi sarcoma[3]. Furthermore, a low CD4 count will have a broken skin barrier, which can allow the bacteria into the dermis, causing cellulitis and methicillin-resistant *Staphylococcus aureus* (MRSA) cellulitis is the most common among all, eventually causing bacteremia in patients with HIV/AIDS[4]. Moreover, KS may occasionally resemble cellulitis or be misdiagnosed as cellulitis, as observed in our patients [5,6].

We aim to present this case to demonstrate that KS can sometimes present in a manner that closely mimics cellulitis. Therefore, it is important to consider multiple skin and lesion biopsies before

settling on a diagnosis of cellulitis. In addition, involving a multidisciplinary team can help differentiate KS from cellulitis, enabling timely diagnosis and appropriate management before symptoms worsen.

Case Presentation

A 46-year-old male with a history of hypertension presented with an acute onset of left facial swelling beginning on the day of admission. Symptoms initially began as a small pimple on the left temple three days prior, which progressively enlarged, became erythematous, and led to significant facial swelling. He reported a prior episode of MRSA cellulitis one year earlier that resolved with antibiotics. He denied intravenous drug use. Patient's vitals on presentation: temperature: 99.5 °C, blood pressure: 144/88 mm Hg, heart rate: 114 beats/minute, respiratory rate: 20 breaths/minute. On physical examination, the patient had a pink erythematous, edematous plaque with crusting and pustules on the left lateral cheek extending to the upper and lower eyelids, a red-purple plaque on the left hard palate, and scattered soft, purple papules involving the abdomen, trunk, arms, and legs. On further questioning, the patient reported a one-year history of a purple rash on the body. Given recurrent cellulitis and disseminated rash or papules, testing for Human Immunodeficiency Virus was performed.

In addition, CT maxillofacial with contrast demonstrated a soft tissue mass of the left palate with bony erosion involving the inferior medial wall of the left maxillary sinus and extensive inflammatory changes consistent with cellulitis, without a drainable abscess (Figures 1 and 2). CT angiography of the chest showed no pulmonary embolism but revealed innumerable bilateral mass-like opacities concerning for metastatic disease or septic emboli (Figures 3 and 4). Imaging also demonstrated bilateral adrenal enlargement (right greater than left) with ill-defined retroperitoneal soft tissue stranding. Laboratory evaluation confirmed HIV-1 infection with an absolute CD4 count of 11 cells/mm³. Other lab workup: hepatitis panel non-reactive; rapid plasma regain test non-reactive; blood cultures were positive for MRSA. The patient was initially treated with intravenous (IV) vancomycin 1 gram every eight hours and IV ampicillin-sulbactam 3 grams every eight hours, later narrowed to IV vancomycin.

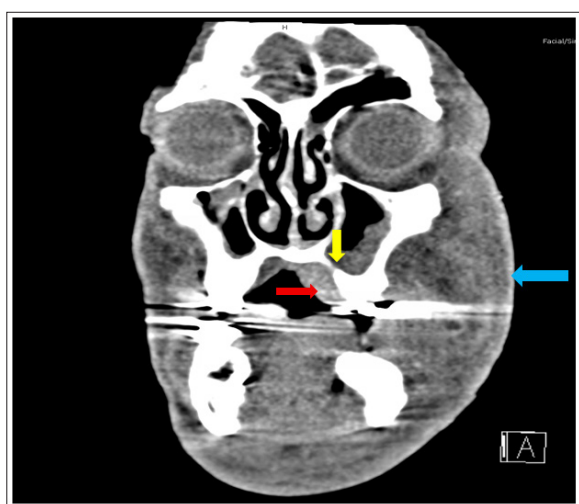


Figure 1: Computer Tomography of the Maxillofacial Region with Contrast (Coronal Section). Red Arrow: Left Palatine Mass, Yellow Arrow: Bony Erosion, Blue Arrows: Facial Swelling

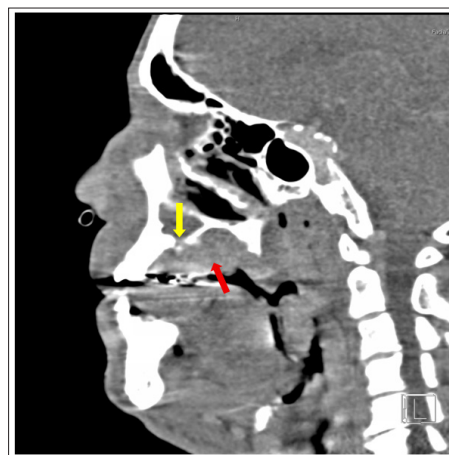


Figure 2: Computer Tomography of the Maxillofacial Region with Contrast (Sagittal Section). Red Arrow: Left Palatine Mass, Yellow Arrow: Bony Erosion



Figure 3: Computer Tomography of the Chest with Contrast (Axial Section, Lung Window). Red Arrows: Lung Infiltrates

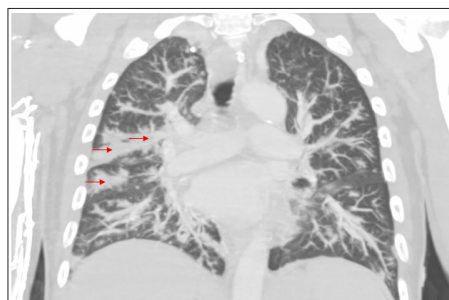


Figure 4: Computer Tomography of the Chest with Contrast (Coronal Section, Lung Window). Red Arrows: Lung Infiltrates

Dermatology performed a right chest skin punch biopsy, confirming KS. ENT biopsy of the left palatine mass also confirmed KS. Left facial wound and bronchoalveolar lavage cultures were positive for MRSA following pulmonology consultation. Infectious disease started sulfamethoxazole-trimethoprim double-strength (800 mg/160 mg) once daily for *Pneumocystis jirovecii* prophylaxis; IV vancomycin was transitioned to IV daptomycin for six weeks; and antiretroviral therapy (ART) with bictegravir/emtricitabine/tenofovir (50-200-25 mg) was started. Oncology initiated paclitaxel 100 mg/m² every two weeks for Kaposi sarcoma, and was also concerned about cytokine release syndrome associated with KS.

The patient was readmitted 8 weeks later with rectal pain and drainage, likely due to a pilonidal cyst that spontaneously drained in the emergency department, and also reported improvement in previous papules (Figure 5a - 5c). Laboratory work: ferritin 486 ng/ml, C-reactive protein 13.8 mg/dl, sedimentation rate

72 mm/hour, CD4 count 10 cells/mm³. CT abdomen showed new upper abdominal and retroperitoneal lymphadenopathy and mild splenomegaly. Repeat CT chest demonstrated interval improvement in pulmonary opacities, suggesting treatment response (Figures 6 and 7). Oncology consultant raised concerns about multicentric Castleman disease, KS inflammatory cytokine syndrome (KS-IRIS), and KS immune reconstitution inflammatory syndrome (KS-IRIS), and recommended interleukin-6 (IL-6, human and viral), HHV-8 viral load, and a lymph node biopsy with flow cytometry and latency-associated nuclear antigen (LANA) staining. Interventional radiology consultant noted no suitable biopsy target, but the patient declined further workup and left against medical advice prior to blood work and biopsy. With oncology follow-up, the patient reported improved appetite, 10-pound weight gain, and improvement in cutaneous lesions.



Figure 5a: Violaceous Papule Over Left Upper Chest



Figure 5b: Violaceous Papule Over Left Foot



Figure 5c: Violaceous Papule Over the Anterior Bilateral Lower Extremity



Figure 6: Computer Tomography of the Chest with Contrast (Axial Section, Lung Window), Yellow Arrows: Improved Lung Infiltrates

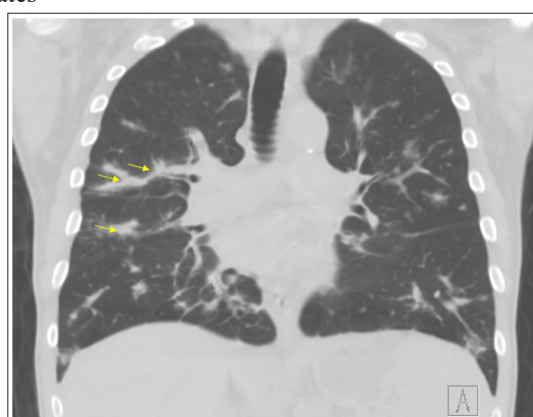


Figure 7: Computer Tomography of the Chest with Contrast (Coronal Section, Lung Window), Yellow Arrows: Improved Lung Infiltrates

One limitation of this case report is that we did not have photographs of the rash from the initial presentation, which makes it difficult to compare its progression and clearly demonstrate improvement over time.

Discussion

KS is an angio-proliferative malignancy caused by infection with human herpesvirus 8 (HHV-8), which primarily targets endothelial cells. Clinically, it presents as characteristic mucocutaneous violaceous papules or nodules. KS most commonly occurs in immunocompromised individuals, particularly those with HIV/AIDS, typically in the setting of CD4 counts <200 cells/mm³, as seen in our patient [1,3].

HHV-8 is also implicated in several other lymphoproliferative and inflammatory disorders, including multicentric Castleman disease (MCD), Kaposi sarcoma inflammatory cytokine syndrome (KICS), primary effusion lymphoma (PEL), and HHV-8-associated large B-cell lymphoma[7]. HHV-8-associated MCD represents one of the recognized subtypes of MCD and is characterized by lymphoproliferation and excessive cytokine release driven by the lytic phase of HHV-8 infection. Clinically, it presents with generalized lymphadenopathy, organomegaly, elevated inflammatory markers (including human and viral interleukin-6 [IL-6], interleukin-10 [IL-10]), elevated C-reactive peptide, abnormal liver function tests, anemia, thrombocytopenia, and elevated serum HHV-8 viral loads [8].

Table 1: Chronological Order of Diagnostics and Treatment. CT: Computer Tomography, PE: Pulmonary Embolism, Ab: Antibody, Ag: Antigen, HIV: Human Immunodeficiency Virus, MRSA: Methicillin-Resistant *Staphylococcus Aureus*, TMX-SMX: Trimethoprim-Sulfamethoxazole, PCP: *Pneumocystis Jirovecii* Pneumonia, CRP: C-Reactive Protein, ESR: Erythrocyte Sedimentation Rate, CD4: Cluster of Differentiation 4

Day of hospitalization	Diagnostics	Results	Treatment
Day 1	CT maxillofacial	Left hard palate mass with bony erosion	Started on IV Unasyn and Vancomycin
	CT angiography chest	Ruled out PE, Innumerable bilateral mass-like opacities are highly suspicious for metastatic disease.	
	HIV Ab/Ag	Reactive	
	HIV 1 Ab	Reactive	
Day 2 to Day 5	Pulmonology did a bronchoscopy and BAL culture	MRSA	Discontinued IV Unasyn and Continued IV vancomycin
	ENT did the left palatine mass biopsy	Kaposi sarcoma	
	Dermatology did the biopsy of the right upper chest lesion	Kaposi sarcoma	
	Dermatology did the biopsy of the left cheek lesion	MRSA	
	Absolute CD4	11 /microL	
	Blood culture	MRSA	
Day 6 to 10 days	CT abdomen and pelvis	Nonspecific haziness in the periaortic retroperitoneum	1. Switched IV vancomycin to daptomycin. 2. Started on bictegrovir-emtricitabine-tenofovir alafenamide for HIV 3. TMX-SMX for PCP prophylaxis
Day 11 to 15			1. Started patient on paclitaxel 2. Discharged on IV daptomycin for 6 weeks
Second admission			
Day 1	CRP	13.8 mg/dl	1. Started on IV Cefepime and IV vancomycin 2. Infectious disease and hematology are consulted.
	ESR	72 mm/hour	
	Blood culture	No growth	
	CT abdomen and pelvis	new upper abdominal, retroperitoneal lymphadenopathy, and mild splenomegaly	
	CT chest	Improved ill-defined patchy airspace opacities	
Day 2 to 5	CD4 count	10 cells/mm ³	Left against medical advice and also declined a lymph node biopsy.
	ferritin	486 ng/ml	

KICS is a severe, complex inflammatory condition similar to MCD clinically and laboratory-wise but lacks characteristic lymph node biopsy findings. It is caused by excess cytokine release during HHV-8's lytic phase, when virions rapidly replicate, express viral genes, and produce viral IL-6 homologs [9]. In contrast to MCD, lymph node biopsy findings such as mantle zone "onion-skinning," LANA-positive cells, regressed germinal centers, and vascular proliferation with perivascular hyalinization are absent in KICS[10]. Moreover, KICS may be confused with KS-IRIS due to similar laboratory findings, except for the presence of an increased CD4 count or a decreased HIV viral load in KS-IRIS. KS-IRIS presents with worsening of pre-existing lesions or development of new lesions due to immune restoration targeting HHV-8, leading to an exaggerated inflammatory response occurring days to months after initiation of ART[11]. However, there are other hyperinflammatory syndromes like Hemophagocytic lymphohistiocytosis caused by HIV/AIDS and its opportunistic

infections. Diagnosis is confirmed when five out of eight criteria are met as per HLH-2004 guidelines [12].

Additionally, PEL is a rare, large-B-cell plasmablastic lymphoma that typically presents with malignant effusions in body cavities, including the pleural, pericardial, and peritoneal spaces. HHV-8-associated large B-cell lymphoma, in contrast, presents as a solid tumor [13]. At the molecular level, HHV-8 infects B cells, where the viral genome persists in a latent episomal form. This latency is maintained by the latency-associated nuclear antigen (LANA), which tethers the viral episome to host chromosomes during cell division, ensuring transmission to daughter cells. A subset of infected cells can transition to the lytic phase, resulting in cellular destruction and the release of proinflammatory cytokines that contribute to disease pathogenesis [14].

The current guidelines recommend liposomal doxorubicin as first-line therapy for Kaposi sarcoma, with paclitaxel as second-line therapy. HHV-8-associated MCD is typically treated with rituximab, often in combination with liposomal doxorubicin or etoposide. For PEL and HHV-8-associated large B-cell lymphoma, systemic chemotherapy regimens such as CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone) or EPOCH (etoposide, prednisone, vincristine, cyclophosphamide, and doxorubicin) are commonly used. Additionally, these therapies are typically started alongside ART [7].

Conclusion

We conclude that careful history-taking and physical examination helped us look beyond the initial presentation. In this case, recurrent cellulitis and a widespread violaceous rash raised suspicion for an underlying condition, urging us to evaluate for HIV/AIDS. Although the rash looked like cellulitis and a single biopsy might have been sufficient in the setting of bacteremia, but we performed biopsies from multiple sites due to the confirmed HIV infection and low CD4 count, planning to rule out Kaposi sarcoma. This approach led to early multidisciplinary involvement, enabling timely diagnosis and management of Kaposi sarcoma while also treating the bacteremia. Thus, providers should continue to focus on history and physical examinations. Not hesitating to perform multiple biopsies and to involve the multidisciplinary team early could be the key to patient-centric care in similar cases.

Declarations

Ethical and Consent to Participate: Not applicable. This manuscript is a case report and does not involve research on human participants requiring Institutional Review Board (IRB) approval.

Consent to Publish: Obtained.

Availability of Data and Materials: All raw data mentioned in the manuscript were obtained from the patients' electronic medical records.

Competing Interests: None of the co-authors has declared any conflict of interest.

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Authors' Contributions: SP conceptualized the manuscript and prepared the initial draft. MP and SP participated in the patient's clinical management during hospitalization. KB served as the infectious diseases consultant, providing clinical guidance for patient management and manuscript development. SP and MP critically reviewed the manuscript throughout its preparation. HCP conducted the literature review and contributed relevant evidence to support the manuscript. The manuscript was developed under the guidance of KB. All authors contributed to the review, revision, and editing of the manuscript and approved the final version for publication.

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