Bloody Pleural Effusion and Ascites Associated With Kaposi Sarcoma in a Kidney Transplant Patient

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Isolated pleural involvement is rare in Kaposi sarcoma (KS). We report an unusual case of bloody pleural effusion and ascites associated with KS in a kidney transplant recipient. A 50-year-old man who had received kidney transplantation from a living unrelated donor presented with a massive left-side pleural effusion, ascites, and a skin lesion. The pleural effusion and ascites were bloody. The skin biopsy specimens showed KS infiltration (proliferation of spindle-shaped cells). Immunosuppressive therapy was discontinued. Although chemotherapy with paclitaxel was started, the patient died. To our knowledge, this is the first report of bloody pleural effusion and ascites associated with KS. Kaposi sarcoma can cause concomitant serositis in kidney transplant patients and should be considered as a differential diagnosis.

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Case Report

INTRODUCTION

Kaposi sarcoma (KS) in allograft recipients is a type of the disease that is associated with immunosuppressive agent therapy. The most common clinical presentation of KS arising after organ transplantation is the development of cutaneous or mucosal lesions in approximately 90% of patients, while in 10%, the disease is limited to the viscera.[1] Visceral involvement in KS occurs in 40% of transplant patients. The most commonly involved organs are the gastrointestinal tract, lungs, and lymph nodes. Pulmonary KS presents with pneumonia and pleural effusion.[2] Isolated pleural KS is very rare in kidney transplant patients.[3] Here, we report a case of bloody pleural effusion and ascites associated with KS in a kidney transplant patient.

CASE REPORT

A 50-year-old man with chronic obstructive uropathy caused by a urinary calculi underwent kidney transplantation from a living unrelated donor. One year later, he presented with dyspnea and cough. On admission, immunosuppressive drugs consisting of cyclosporine,100 mg/d, mycophenolate mofetil, 2 g/d, and prednisolone, 5 mg/d, were administered. Chest examination revealed diminished breath sounds on the left side. A reddish-blue macule was observed on the abdominal skin and the right leg (Figure 1). The

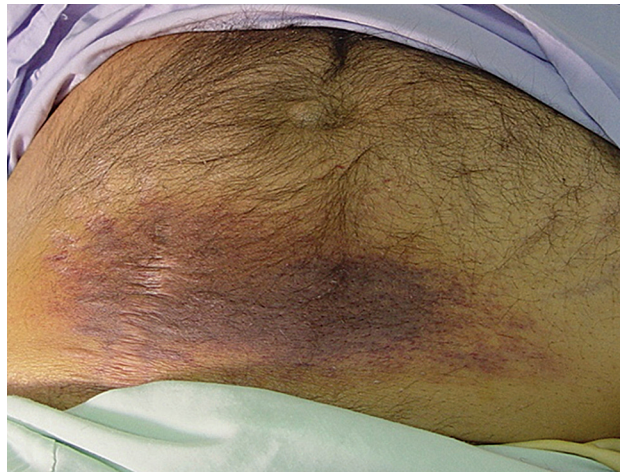

Figure 1. Reddish-blue macule on the abdominal skin.

thoracic computed tomography scan revealed a massive left-side plural effusion without mediastinal lymphadenopathy, mass lesion, or opacity of the lung parenchyma (Figure 2). Abdominal ultrasonography revealed massive ascites without organomegaly.

The pleural effusion was drained, and a biochemical analysis revealed the following: lactate dehydrogenase (LDH) level, 607 IU/L; protein level, 3.6 mg/dL, leukocyte count, 90 cell/μL; erythrocyte count, 100 000 cell/μL, glucose level, 102 mg/dL; and adenosine deaminase level, 14 μg/L. The results of the smear test and culture were negative. The smear and culture were also negative for acid-fast bacilli. Pleural biopsy showed fibrinoid pleuritis. Skin biopsy showed proliferation of irregular vascular channels with partly surround preexisting blood vessels in some area in the dermis (Figure 3).

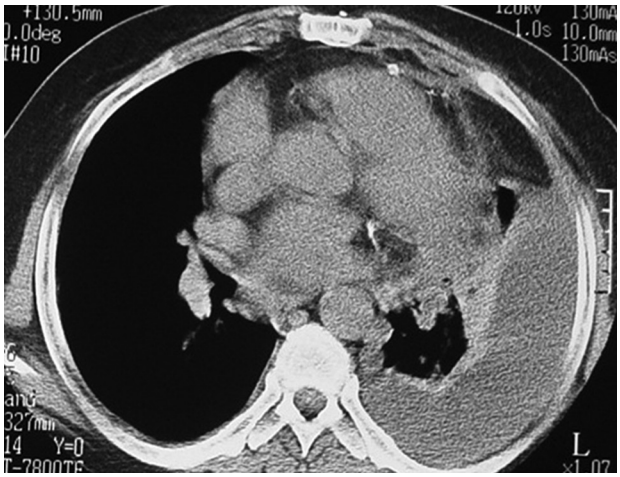

Figure 2. A massive left side plural effusion without mediastinal lymphadenopathy, mass lesion, or opacity of the lung parenchyma.

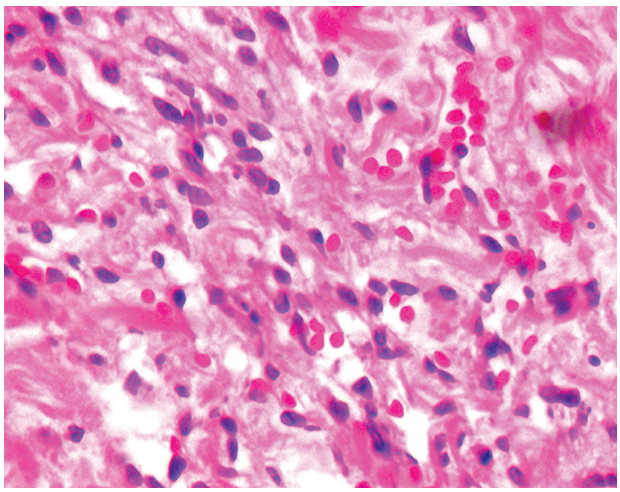

Figure 3. Proliferation of irregular vascular channels (spindle cells) with partly surround preexisting blood vessels in some area in the dermis (hematoxylin-eosin, × 40).

The gross appearance of the ascitic fluid was bloody. The results of the other laboratory tests were as follows: serum creatinine level, 2.1 mg/dL; serum LDH level, 1042 IU/L; serum protein level, 5.1 g/dL; leukocyte count, 11 500 cell/μL; hemoglobin level, 9.5 g/dL; platelet count, 151 000 cells/μL; prothrombin test time, 12 sec; international normalized ratio, 1.1; and partial thromboplastin time, 27 sec. Tests for human immunodeficiency virus, hepatitis B, hepatitis C, and cytomegalovirus infection were negative.

Mycophenolate mofetil was discontinued on the day of admission. Cyclosporine was tapered and discontinued 10 days after admission when clinical condition deteriorated. However, low-dose prednisolone was continued. Chemotherapy with paclitaxel was started 1 day before the patient died. However, his clinical condition deteriorated, and the patient needed ventilation support. He died because of cardiac arrest 3 weeks after admission.

DISCUSSION

Bloody pleural effusion associated with KS was diagnosed based on skin involvement simultaneously caused by KS. Differential diagnoses such as parapneumonic pleural effusion and tuberculosis were ruled out because the results of pleural pathology, thoracic computed tomography scan, and culture of pleural effusion were all negative.

The prevalence of pulmonary KS is different in various studies, and it ranges from zero to 19%.[4,5] Although KS simultaneously involves the lungs, pleura, and mediastinal lymph nodes,[2,6] our patient had isolated bloody pleural effusion associated with KS. Isolated pleural KS without mediastinal and lung parenchymal involvement is very rare. Gómez-Román and coworkers[3] reported isolated plural KS in a kidney transplant patient. The patient had exudative pleural effusion, which was not bloody, unlike that in our patient.

In addition to bloody pleural effusion, our patient also had bloody ascites. Unusual site of KS was reported after kidney transplantation.[7] However, to our knowledge, this is the first case of bloody ascites in a kidney transplant patient with KS. We did not perform a peritoneal biopsy. However, it

is likely that the KS lesion of the peritoneum was the only cause of ascites in our patient. Sabeel and colleagues[8] reported a case of a young woman with KS who developed both lymphoma and ascites. We did not perform bone marrow aspiration and biopsy to rule out lymphoma. However, the patient had no organomegaly and lymphadenopathy, and therefore, lymphoma was considered less likely. Previously, a patient with acquired immune deficiency syndrome was reported to develop bloody ascites, which was caused by peritoneal KS.[9] However, our patient tested negative for human immunodeficiency virus.

Kaposi sarcoma may respond to reduction or discontinuation of the immunosuppressive regimen, and this should be the first therapeutic maneuver.[10,11] However, the patients did not regress with this maneuver and we had to start chemotherapy with paclitaxel as a one option for treatment of KS.[12]

To our knowledge, this is the first report of bloody pleural effusion and ascites associated with KS. Kaposi sarcoma can cause concomitant serositis in kidney transplantat patients and should be considered as a differential diagnosis.

CONFLICT OF INTEREST

None declared.

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