

**DISSEMINATED KAPOSI'S SARCOMA ONE YEAR AFTER KIDNEY  
TRANSPLANTATION: CASE REPORT**

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**ABSTRACT**

The chronic use of immunosuppressive agents is associated with the long-term risk of a wide variety of malignancies, including Kaposi's sarcoma (KS), in renal transplant recipients compared with those of the general population. An increased risk of post-transplant KS may be related to HHV-8 infection, and pre-transplantation HHV-8 seropositivity is a risk factor. Management of Kaposi's sarcoma generally includes radiotherapy for focal disease and chemotherapy for systemic disease. In renal transplant patients, the dose reduction or cessation of immunosuppressive drugs is the main approach for the treatment. Here we describe a case of 59-year-old woman who developed disseminated Kaposi's sarcoma one year after kidney transplantation, her immunosuppressive therapy was prednisolone 20 mg/day, Tacrolimus (Prograf®) 4 mg/day, and mycophenolate mofetil (MMF) 500 gr/day. The decision was made for monotherapy immunosuppression with 5 mg prednisolone, then systemic chemotherapy with paclitaxel with good progress under treatment.

In renal transplant patients, the dose reduction or cessation of immunosuppressive drugs is the main approach for the treatment.

**KEYWORDS:** immunosuppressive drugs, renal transplantation, Kaposi sarcoma.

**INTRODUCTION**

Long-term immunosuppression in kidney transplant recipient increases the risk of malignancy approximately 100 times as high as in the general population.<sup>[1]</sup>

Kaposi's sarcoma (KS) is one of these tumors with 500 fold more frequently in renal transplant recipients than the general population.<sup>[2,3]</sup>

Patients on immunosuppressive therapy post renal transplant have weakened innate and adaptive immunity leading to infection of human herpes virus 8 (HHV8), a recognized oncogenic virus responsible for causing Kaposi's sarcoma.<sup>[4]</sup>

Traditionally, it is recognized as an AIDS-defining presentation. In the transplant setting, depletion of T cell immunity by immunosuppression can permit opportunistic infections like HHV8, especially if the patients immunosuppression drug levels are targeted to high.<sup>[5]</sup>

KS is an angioproliferative neoplasm characterized by reddish-brown or purple-blue plaques or nodules on

cutaneous or mucosal surfaces, including the skin, lungs, gastrointestinal tract and lymphoid tissue.<sup>[6]</sup>

The mean interval that reported in international series was about 13 months.<sup>[2]</sup> The ratio of male to female in post-transplant KS is 3.3:1 to 1:1 and the mean age at the time of diagnosis are 43 years, which is younger than among patients with classic KS.<sup>[3]</sup>

**Case report**

Here we describe a case of 59-year-old woman who developed KS a fourteen months after kidney transplantation. The cause of end-stage renal disease (ESRD) in the patient was primary crescentic glomerulonephritis and was managed with hemodialysis for 5 years before transplantation. He gets kidney transplant from her sister.

His immunosuppressive therapy was prednisolone 20 mg/day, Tacrolimus (Prograf®) 4 mg/day, and mycophenolate mofetil (MMF) 500 gr/day.

Six months later, the patient presented a cytomegalovirus infection associated with neutropenia treated with intravenous Ganciclovir and Neupogen.

One year after transplantation, the patient presented a red-purplish macular rash on her left leg associated with edema of the lower left limb with progressive worsening. [Figure 1] and recent occurrence of left inguinal lymphadenopathy.

Physical examination revealed large cutaneous purplish confluent infiltrative plaques on the left leg with necrosis lesions highly evocative of Kaposi sarcoma [figure 2]



**Figure 1: One year after transplantation.**



**Figure 2: Significant spread of skin lesions before treatment.**

Para-aortic, parailiac, pelvic and left inguinal lymphadenopathies were detected by computer tomography imagination showing multiple pathologically enlarged lymph nodes.

Biopsy, excision of the left inguinal lymphadenopathy was performed and confirmed the diagnosis of KS: histology revealed the presence of fusiform cells of endothelial origin and neovessels while Immunohistochemical staining revealed positive labelling for CD34 and HHV8.

**Nephrologically:** The decision was made for monotherapy immunosuppression with 5 mg prednisolone.

On the oncological level: the patient started chemotherapy with paclitaxel 135 mg/m<sup>2</sup> every 3 weeks with good clinical tolerance.

After 3 cycles of chemotherapy, disappearance of the left inguinal lymphadenopathy with reduction of the diffuse skin lesion of the left lower limb and a good radiological response, therefore chemotherapy was continued. [Figure 3]



**Figure 3: After 3 cycles of chemotherapy.**

## DISCUSSION

KS is an angioproliferative cutaneous cancer caused by human herpesvirus 8.1 Skin lesions are typical and make the diagnosis. These are purplish-blue lesions presenting as non-painful, non-itchy, macules, papules, plaques or nodules.<sup>[7]</sup>

Due to their vascular etiology, they can ulcerate and bleed. The incidence of KS is higher in transplant patients than in non-immunosuppressed populations.<sup>[8]</sup>

Majority of cases of post-transplant KS has been reported in patients from Mediterranean, Jewish, Arabic, Caribbean, or African descent.<sup>[6]</sup>

In kidney transplant recipients, the intensity and duration of immunosuppression, and the presence of HHV8 serology pre transplantation increase the risk of developing KS, which occurs 13 months after transplantation (range few weeks to 18 years).<sup>[8]</sup> In our case, KS has occurred in the year of the kidney transplantation.

Lesions usually begin on the lower limbs, with multifocal and asymptomatic development. KS may remain localised to the skin, but dissemination to visceral mucosa of the trachea, lungs and gastrointestinal tract is common in immunosuppressed patients.<sup>[9]</sup>

Management of Kaposi's sarcoma generally includes radiotherapy for focal disease and chemotherapy for systemic disease.

The chemotherapy drugs Taxol® (paclitaxel) and Taxotere® (docetaxel) appear to be active treatments for disseminated Kaposi's sarcoma.

Taxol has been found to cause tumors to shrink significantly in the majority of patients with advanced Kaposi's sarcoma who have not responded to previous systemic chemotherapy. Overall, more than half (56%) of patients treated with Taxol experience an anticancer response, and the responses last an average of almost nine months. The majority of patients who responded (70%) did so after six weeks of treatment and four cycles of therapy. However, complete anticancer responses were observed in only four of the 54 patients who responded. Patients who responded to treatment also experienced a significant improvement in quality of life. It has not been determined whether Taxol may extend survival.<sup>[10]</sup>

Taxotere has also been shown to produce partial anticancer responses in nearly half of patients with recurrent disease and produced stable disease in another one-third of patients. On average, patients survived more than two years without disease progression.<sup>[11]</sup>

In our case, chemotherapy with paclitaxel was preferred and the result was good.

In renal transplant patients, The usual management is to reduce the doses of immunosuppressive drugs once Kaposi's sarcoma is diagnosed. This requires carefully balancing the risk of death from generalized Kaposi's sarcoma and the risk of organ rejection and complications of renal failure that may occur if the immunosuppressive therapy is discontinued.<sup>[9]</sup>

In our case the patient used monotherapy immunosuppression with 5 mg prednisolone.

## CONCLUSION

This case serves as a reminder to be vigilant of the complications from immunosuppression such as opportunistic infection and malignancy in the transplant cohort.

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