Case Report

Acute graft dysfunction due to Kaposi sarcoma involving the bladder in a renal transplant recipient

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Introduction

Kaposi sarcoma in renal transplant patients is no longer rare. Its incidence is 400- to 500-fold higher than that seen in a control population of the same ethnic origin [1]. These findings are confirmed by the Cincinnati Transplant Tumor Registry. When non-melanoma skin cancers and in situ carcinomas of the uterine cervix are excluded, Kaposi sarcoma comprises 5.7% of neoplasms [2].

Sixty per cent of patients with Kaposi sarcoma have non-visceral Kaposi sarcoma confined to the skin, conjunctiva, or oropharyngeal mucosa. Forty per cent have visceral disease that involves mainly the gastrointestinal tract, lungs, and lymph nodes, but other organs are also involved [3].

Kaposi sarcoma in the bladder in renal transplant recipients is rarely reported [4]. We record here an unusual case of Kaposi sarcoma that manifested as graft dysfunction due to obstruction by the sarcoma itself. Our case suggests that Kaposi sarcoma involving the bladder may be a cause of graft dysfunction in renal transplant recipients.

On admission, abdominal ultrasonography and computed tomography (CT) scan were performed for evaluation of graft dysfunction. Hydronephrosis of the grafted kidney (Figure 1A) and a nodular mass on the bladder were detected (Figures 1B, C). Under the presumptive diagnosis of obstructive nephropathy by bladder cancer, the mass was removed via a trans-urethral approach. The histological findings of the tumour mass were consistent with Kaposi sarcoma, and further systemic evaluation of the case revealed Kaposi sarcoma involving the skin, lung, and stomach.

After resection of the tumour mass on the bladder, the serum creatinine decreased dramatically from 6.7 to 2.5 mg/dl. Cyclosporin was reduced (from 225 mg/day to 125 mg/day) and then withdrawn during chemotherapy. Combination chemotherapy consisting of doxorubicin 20–30 mg/m², bleomycin 10 mg/m², vincristine 2 mg (ABV) was administered intravenously every 3 weeks. Follow-up serum creatinine concentration was approximately 2.0 mg/dl (Figure 2).

The subject was treated as an outpatient with low-dose cyclosporin (trough level 50–100 ng/ml) and low-dose prednisolone (5–7.5 mg/day) for 18 months, with a relatively well-functioning graft (serum creatinine level 1.5–2.0 mg/dl). During the follow-up period, no further visceral involvement of Kaposi sarcoma was observed, but cutaneous lesions appeared sporadically in relation to the level of immunosuppression.

Discussion

We report an unusual case of Kaposi sarcoma that manifested as graft dysfunction due to sarcoma involving the bladder. Initially, acute rejection was suspected, but radiological evaluation revealed obstructive nephropathy by a tumour mass that was confirmed as Kaposi sarcoma. Further systemic evaluation of this case revealed a disseminated Kaposi sarcoma involving the skin, lung, bladder, and stomach. A retrospective review of this case shows that cutaneous Kaposi sarcoma may
have been diagnosed if careful physical examination had been carried out at the outpatient clinic.

In general, visceral Kaposi sarcoma involves the gastrointestinal tract, lungs, and lymph nodes; however, it may develop in any organ (testis, heart, liver, pancreas, and bladder) [5]. When a systemic work-up for disseminated Kaposi sarcoma is required, rarer sites must be evaluated. This is likely to result in an increased incidence of Kaposi sarcoma of the bladder being reported.

The first line of approach to treating post-transplant Kaposi sarcoma is to reduce and eventually stop anti-rejection medications [6–8]. With this strategy, a 30% complete remission can be expected. In addition, surgical excision and local irradiation are needed for localized lesions, and chemotherapy is needed in cases with widespread lesions. In our case, three treatment strategies were designed. First, the cyclosporin dosage was reduced; second, transurethral resection of the tumour mass on the bladder was carried out; and third, chemotherapy for disseminated Kaposi sarcoma was started. After resection of the tumour mass on the bladder, serum creatinine decreased dramatically and further improvement of renal function was observed with reduction of the cyclosporin dose and subsequent chemotherapy. We chose a less nephrotoxic chemotherapy regimen (ABV) [9] and immunosuppressants were discontinued during chemotherapy to reduce the risk of fatal infection due to over-immunosuppression [10].

The therapeutic goals for renal transplant recipients with Kaposi sarcoma are to maintain a well-functioning graft and to cure the Kaposi sarcoma without terminating immunosuppressants. It is practically difficult to achieve two goals at the same time and it is also difficult to discontinue immunosuppression if the Kaposi sarcoma is not life threatening. With reduction or cessation of immunosuppressive therapy, the remission rate is 27% in visceral Kaposi sarcoma. However, such therapy does have a price, as 59% of renal recipients in whom it was successful lost their grafts [3]. On the other hand, impressive results were reported in a study in which eight of 13 patients (61%) had complete remission, following reduction of immunosuppressive therapy, and 69% of the patients had functioning grafts many months after reduction or withdrawal of immunosuppression [11].

In this case, complete remission of Kaposi sarcoma was induced with chemotherapy, and low-dose cyclosporin and steroids were administered to save graft function. During the follow-up period, the cutaneous Kaposi sarcoma fluctuated according to the level of immunosuppression, but graft function was well maintained. At present the clinical outcome of our case is unpredictable, but our therapeutic approach

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### Table 1: Treatment Schedule

<table>
<thead>
<tr>
<th>Day</th>
<th>Prednisolone (mg/day)</th>
<th>Cyclosporin (mg/day)</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>225</td>
<td>125</td>
<td>TUR</td>
</tr>
<tr>
<td>20</td>
<td>125</td>
<td>125</td>
<td>Chemotherapy</td>
</tr>
</tbody>
</table>

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### Fig. 1. Kaposi sarcoma involving the bladder. (A) Abdominal CT scan shows marked enlargement of grafted kidney, hydronephrosis, and hydrourerter. (B) Abdominal CT scan shows round mass on bladder wall (arrow). (C) Abdominal ultrasonography shows mass on the bladder wall (arrow).

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### Fig. 2. Clinical course. Note dramatic decrease of serum creatinine after removal of tumour mass on the bladder and further decrease of serum creatinine with reduction of cyclosporin and subsequent chemotherapy. CsA, cyclosporin; Pd, prednisolone; TUR, transurethral resection.
Graft dysfunction from Kaposi sarcoma in RT recipient

seems to be reasonable for prevention of graft loss in renal transplant recipients with Kaposi sarcoma.

In conclusion, Kaposi sarcoma involving the bladder in the renal transplant recipient is rare but it should be considered as one of the causes of graft dysfunction.

References


Received for publication: 18.10.99
Accepted in revised form: 5.7.00