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FIRST DIAGNOSIS OF MULTIPLE MYELOMA IN A KIDNEY TRANSPLANT RECIPIENT (A CASE REPORT)

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Background. Kidney graft failure due to recurrence of previously undiagnosed multiple myeloma (MM) is a rare event. This report presents a clinical case of kidney transplantation complicated by graft dysfunction one year after surgery caused by recurrence of undiagnosed MM. **Clinical observation.** A 75-year-old man with a history of arterial hypertension and diabetes mellitus was admitted to the dialysis unit with symptoms of uremia. He had not previously been followed by a nephrologist. Renal ultrasonography revealed diffuse parenchymal changes. Considering the two diseases, chronic kidney disease (CKD C5) secondary to hypertensive and diabetic nephropathy was diagnosed, and maintenance hemodialysis was initiated. After 11 months, the patient underwent deceased-donor kidney transplantation. One year post-transplantation, graft dysfunction developed, prompting a transplant kidney biopsy. Histopathological examination revealed changes characteristic of MM-associated kidney injury, including light-chain cast nephropathy (LCCN, κ type) combined with light-chain proximal tubulopathy (LCPT, κ type), focal segmental glomerulosclerosis, tubulointerstitial nephritis, and acute tubular necrosis. Further evaluation confirmed MM with Bence–Jones proteinuria, stage III B, and myeloma nephropathy of the renal allograft. The patient was transferred to the hematology department for chemotherapy, resulting in partial hematologic remission. However, renal graft function was not restored, and the patient remained on maintenance hemodialysis. **Conclusion.** MM-associated kidney injury is a rare clinical event. In routine clinical practice, thorough pre-transplant evaluation and accurate determination of the etiology of CKD are essential.

Keywords: kidney transplantation, multiple myeloma, cast nephropathy, light-chain proximal tubulopathy.

INTRODUCTION

Patients who have undergone kidney transplantation (KT) are at an increased risk of developing malignant tumors, which impair quality of life and reduce life expectancy. Multiple myeloma (MM) is a B-cell malignancy characterized by the clonal proliferation of plasma cells producing monoclonal immunoglobulin. Although MM is relatively rare in the general population, it is quite common among all hematopoietic and lymphoid tissues, with a frequency of up to 10–15% [1–3].

The course of MM is frequently complicated by renal involvement, leading to the development of acute kidney injury (AKI) as well as acute and chronic kidney disease (CKD), which exacerbate disease severity and prognosis [3, 4]. Kidney damage in MM is heterogeneous and may predominantly affect either the tubulointerstitial compartment or the glomerular apparatus. The most common renal lesion associated with MM is light-chain cast nephropathy (LCCN), which results from excessive production and urinary excretion of monoclonal free immunoglobulin light chains. These light chains exert a direct toxic effect on proximal tubular epithelial cells and contribute to the formation of casts in the distal tubules. In addition to LCCN, other forms of MM-related kidney injury include amyloid light-chain (AL) amyloidosis, characterized by the deposition of monoclonal immunoglobulins with light and/or heavy chains in the glomerular and tubular basement membranes, and, less commonly, light-chain proximal tubulopathy (LCPT). The latter is defined by the accumulation of monoclonal light chains, most commonly kappa, within proximal tubular cells, often accompanied by crystal formation [5–8]. The coexistence of LCCN and LCPT is rare and has been reported only in a limited number of clinical case descriptions [9–11].

There have been isolated clinical reports on cases of primary MM diagnosed in kidney transplant (KT) recipients, resulting in renal allograft dysfunction. Post-KT MM may occur through several mechanisms: (1) recurrence of pre-existing MM, (2) *de novo* MM, or (3) MM associated with the donor kidney [12–16]. In clinical practice, the most frequently encountered scenario is relapse of previously undiagnosed MM in transplant candidates, whereas *de novo* and donor-associated MM are considerably less common.

Histological examination of renal allograft biopsy specimens most often reveals LCCN, while other pat-

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terns of graft injury are reported less frequently; in rare cases, LCPT has been reported [13, 14, 17, 18]. A review of the available literature identified only a single reported clinical case documenting the coexistence of LCCN and LCPT in a transplanted kidney in a recipient with subsequently confirmed MM [14].

Here, we present our own rare clinical case of a KT recipient in whom graft dysfunction, developing one year after transplantation, was the first manifestation of previously undiagnosed MM, leading to combined allograft injury in the form of LCCN and LCPT.

CLINICAL CASE

Patient S., a 75-year-old man, was admitted to the emergency department of a medical facility due to a worsening condition. His complaints included increasing weakness, nausea, loss of appetite, and weight loss of 8–10 kg. A few days prior to admission, the patient had undergone self-initiated laboratory testing at a medical center, which revealed markedly elevated serum creatinine (778 µmol/L), hyperkalemia (potassium 5.5 mmol/L), and increased blood urea (37.2 mmol/L). Ultrasound examination of the abdominal organs, kidneys, and prostate demonstrated hepatomegaly and diffuse parenchymal changes in the liver, pancreas, kidneys, and prostate. No additional medical records were available at the time of admission.

According to the patient and his relative, he had a long-standing history of arterial hypertension, with maximal blood pressure values reaching 170/100 mmHg, which was well controlled with valsartan (160 mg/day) and indapamide (2.5 mg/day). Four years earlier, he had been diagnosed with type 2 diabetes mellitus and treated with metformin (1000 mg/day), achieving satisfactory blood glucose control.

A week prior to the current deterioration in his condition, the patient experienced an episode of syncope. Subsequent evaluation revealed anemia, with a hemoglobin level of 111 g/L. Upper gastrointestinal endoscopy and colonoscopy were performed; however, no source of bleeding was identified. At that time, serum creatinine was already significantly elevated (590 µmol/L), and the patient was advised to see a nephrologist, which was not done.

Upon admission, the patient's condition was moderate. Body temperature was 36.6 °C. Height was 174 cm, body weight 78 kg, and body mass index 25.8 kg/m^2. He had a normal body habitus with a normosthenic constitution. Skin was jaundiced; no peripheral edema. No abnormalities were detected on neurological or mental status examination, nor were there pathological findings involving the musculoskeletal or endocrine systems. Respiratory rate was 17 breaths per minute. Lung auscultation revealed vesicular breath sounds evenly distributed over all lung fields, without wheezes. Cardiac auscultation demonstrated muffled heart sounds with a regular rhythm. Heart rate was 93 beats per minute. Blood pressure was 160/80 mm Hg in both arms. The abdomen was of normal configuration, soft, non-tender on palpation, and painless in all quadrants, with no signs of peritoneal irritation. The lower edge of the right hepatic lobe protruded 1 cm below the costal margin. The kidneys were not palpable, and percussion over the lumbar region was painless. Urine output was 500 mL/day over several days. The patient reported frequent urination at night in small volumes.

There was no family history of disease and no known allergies. Epidemiological history was unremarkable: the patient denied contact with infectious sources and reported vaccination against SARS-CoV-2 and hepatitis B virus. Serological testing for HBsAg, anti-HCV antibodies, HIV, and syphilis (RW) was negative.

Laboratory test results. A complete blood count revealed mild anemia with hemoglobin of 113 g/L. White blood cell count (10.8 × 10^9/L), platelet count (382 × 10^9/L), and leukocyte differential were within normal limits. Urinalysis was unremarkable; proteinuria was 0.5 g/L. Biochemical blood analysis showed total protein 69 g/L, albumin 46 g/L, urea 62 mmol/L, creatinine 1342 µmol/L, glucose 6.5 mmol/L, potassium 6.1 mmol/L, bicarbonate 12.9 mmol/L, and arterial pH 7.27. Bilirubin, liver enzymes, and cholesterol levels were within reference ranges. Lactate dehydrogenase (LDH) was mildly elevated at 259 U/L (reference range 110–210 U/L), and C-reactive protein was 3 mg/L. Glycated hemoglobin (HbA1c) was 7.6%. Serological tests for hepatitis B, hepatitis C, and HIV were negative; SARS-CoV-2 RNA was not detected.

Repeat ultrasound examination demonstrated kidneys of normal size: right kidney 105×62 mm and left kidney 105×64 mm. Renal contours were smooth and well defined. Parenchymal thickness measured 18 mm on the right and 21 mm on the left. The parenchyma exhibited diffusely heterogeneous echogenicity bilaterally. On color doppler flow imaging (CDFI), renal vascularization was preserved and traceable to the capsule. Corticomedullary differentiation was clearly maintained on both sides. The renal pelvicalyceal system was not dilated. Renal sinuses were hyperechoic and contained multiple punctate hyperechoic inclusions measuring 2–3 mm with indistinct acoustic shadows, consistent with microlithiasis. No focal masses were identified.

Conclusion: Ultrasound findings are consistent with diffuse changes in the renal parenchyma.

Echocardiography. Mild dilation of the left cardiac chambers, basal and mid portions of the right ventricle, and ascending aorta was noted. Calcification of the aorta, mitral valve, and aortic valve was present. There were mild regurgitation of the aortic, mitral, tricuspid, and pulmonary valves. Concentric left ventricular hypertrophy was identified. Global left ventricular systolic

function was preserved, with grade II diastolic dysfunction. Minimal pericardial effusion was detected.

Based on the patient's medical history, the presence of two comorbid conditions associated with a high risk of CKD, and the results of laboratory and instrumental investigations, the following diagnosis was established. Primary diagnosis. CKD stage C5 (estimated glomerular filtration rate 8 mL/min), secondary to hypertensive and diabetic nephropathy. Concomitant conditions. Arterial hypertension, stage 3, grade 2, with a very high cardiovascular risk (risk category 4); chronic heart failure, functional class III according to the NYHA classification; and type 2 diabetes mellitus.

Given the high level of azotemia in the setting of reduced diuresis, a tunneled central venous dialysis catheter was inserted, and renal replacement therapy with hemodialysis was initiated. Subsequently, an arteriovenous fistula was created. The patient was discharged for continuation of maintenance hemodialysis on an outpatient basis at the place of residence. The hemodialysis regimen was adequate, with a single-session Kt/V of 1.5–1.7 and a urea reduction ratio of 70–72%. The patient's clinical condition remained stable: blood pressure ranged from 130–140/90 mmHg on antihypertensive therapy; fasting plasma glucose was 5.8–7.0 mmol/L; hemoglobin levels ranged from 88 to 98 g/L; and serum iron, ferritin, calcium, and albumin were within reference ranges. Hyperphosphatemia was noted (1.7–2.2 mmol/L). Following additional evaluation, the patient was placed on the kidney transplant waiting list.

Deceased-donor KT was performed 11 months after initiation of maintenance hemodialysis. Induction and maintenance immunosuppressive therapy were administered according to standard protocols. No intraoperative or early postoperative complications were observed, and the graft demonstrated immediate function. Urine output was 1.8–2.3 L/day. Ultrasound examination revealed the renal allograft located in the right iliac fossa, measuring 121×55 mm (measured medially from the suture line). The parenchyma exhibited increased echogenicity with prominent hypoechoic pyramids, while corticomedullary differentiation was preserved. The renal pelvicalyceal system was not dilated. On CDFI, the vascular pattern was intact, and the renal artery and vein were visualized up to their anastomoses with the iliac vessels. The patient was discharged in stable condition for outpatient follow-up (Table).

Two months after kidney transplant, the patient's condition deteriorated, manifested by a reduction in urine output to 500 mL/day, the development of mild peripheral edema of the shins and feet, and hyperazotemia (Table). Glycated hemoglobin was 6.7%. Polymerase chain reaction testing for cytomegalovirus and Epstein–Barr virus DNA in the blood was negative. A renal allograft biopsy was not performed. During inpatient management, infusion therapy was administered and immunosuppressive therapy was adjusted. This resulted in clinical and laboratory improvement, with restoration of satisfactory graft function (serum creatinine 150 µmol/L, urea 15.7 mmol/L, daily proteinuria 0.7 g). Outpatient follow-up continued.

Table

Laboratory test results for Patient S.

Parameter	Before kidney transplant	After kidney transplant					
		3 days	1 month Discharge from hospital	2 months Deterioration	6 months	9 months	12 months
Hemoglobin (g/L)	140	108	111	111	113	120	89
White blood cells ($\times 10^9$/L)	9.1	5.9	6.4	5.9	6.5	7.2	2.3
– Neutrophils (%)	63	72	65	66	71	69	28
– Lymphocytes (%)	23	14	20	26	23	26	46
– Monocytes (%)	12	13	14	7	7	6	20
Platelets ($\times 10^9$/L)	238	233	252	293	221	234	221
Creatinine (µmol/L)	576	237	157	337	132	100	469
Urea (mmol/L)	18	19.1	12.9	29.6	16.7	12.6	29.3
Uric acid (µmol/L)		440	386	403	388	289	626
Total protein (g/L)	67	61	62	62	67	69	59
Albumin (g/L)	44	43	41	44	48	44	44
Total cholesterol (mmol/L)	5	4.4	4.8	5.1	4.8	5.1	2.9
Glucose (mmol/L)	6	8.8	5.5	8.6	8.5	7.8	6.8
Total calcium (mmol/L)		2.5	2.3	2.4	2.4	2. 3	2.5
Phosphorus (mmol/L)		1.31	1.22	1.33	1.23	1.71	1.93
Potassium (mmol/L)	4.7	3.7	4.7	4.9	5.2	4.8	4.2
Proteinuria (g/day)	–	1.2	0.9	0.9	1.77	1. 6	2.3
Tacrolimus (ng/mL)	–	8.3	7.8	4.3	7.6	6.4	6.8

One year after KT, the patient's condition deteriorated again, characterized by progressive anemia, leukopenia, and worsening renal allograft function (Table). Polymerase chain reaction testing for cytomegalovirus DNA in the blood was positive, while Epstein–Barr virus DNA was not detected. Ultrasound examination of the renal allograft revealed no significant interval changes. The graft measured 110×44×45 mm (measured medially from the suture line). The parenchyma demonstrated moderately increased echogenicity with preserved corticomedullary differentiation. The pelvicalyceal system was not dilated. On CDFI, intrarenal perfusion was preserved, and the renal artery and vein were visualized to their anastomoses with the iliac vessels. Mycophenolate mofetil was discontinued, antiviral therapy with valganciclovir was initiated, and red blood cell transfusion was performed. Renal allograft dysfunction persisted, prompting a diagnostic biopsy to determine the etiology.

Conclusion. The morphological features were most consistent with renal involvement by multiple myeloma, including LCCN (κ type) in combination with proximal light chain tubulopathy (LCPT, κ type). Additional findings included focal segmental glomerulosclerosis, tubulointerstitial nephritis, and acute tubular necrosis (Figs. 1–3). Electron microscopy was not performed due to technical limitations.

Based on the morphological findings from the renal allograft biopsy, a hematology consultation and further investigations were undertaken. Bone marrow examination: bone marrow aspirate was hypercellular, with plasma cell (myeloma cell) metaplasia (66.4%); the plasma cells were predominantly large, morphologically polymorphic, and anaplastic, with the presence of multinucleated forms. Erythropoiesis was normoblastic. Urinalysis for Bence–Jones protein was positive. Serum immunoglobulin levels were markedly reduced: IgG 1.7 g/L (reference range 7–16 g/L), IgA 0.13 g/L (reference range 0.7–4 g/L), and IgM 0.04 g/L (reference range 0.4–2.3 g/L). Skull X-ray revealed a focal area of

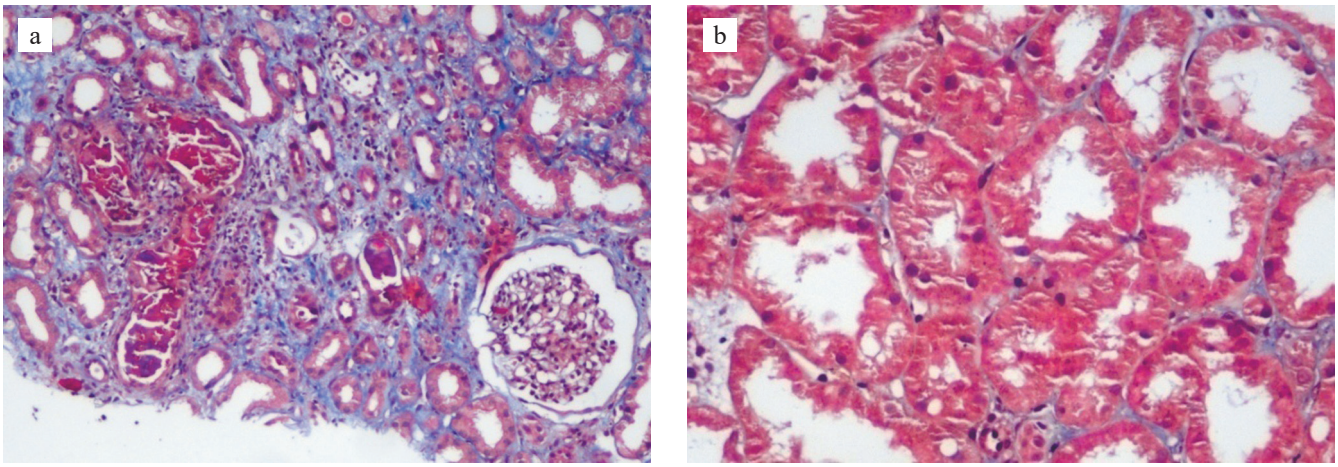

Fig. 1. Biopsy of the kidney transplant: (a) Unchanged glomerulus without mesangial or endocapillary hypercellularity. Interstitial fibrosis with accompanying tubular atrophy. Fragmented xanthophilic casts in the tubular lumen. Masson's trichrome stain, ×100. (b) Xanthophilic granules in tubular epithelial cells. Masson's trichrome stain, ×200

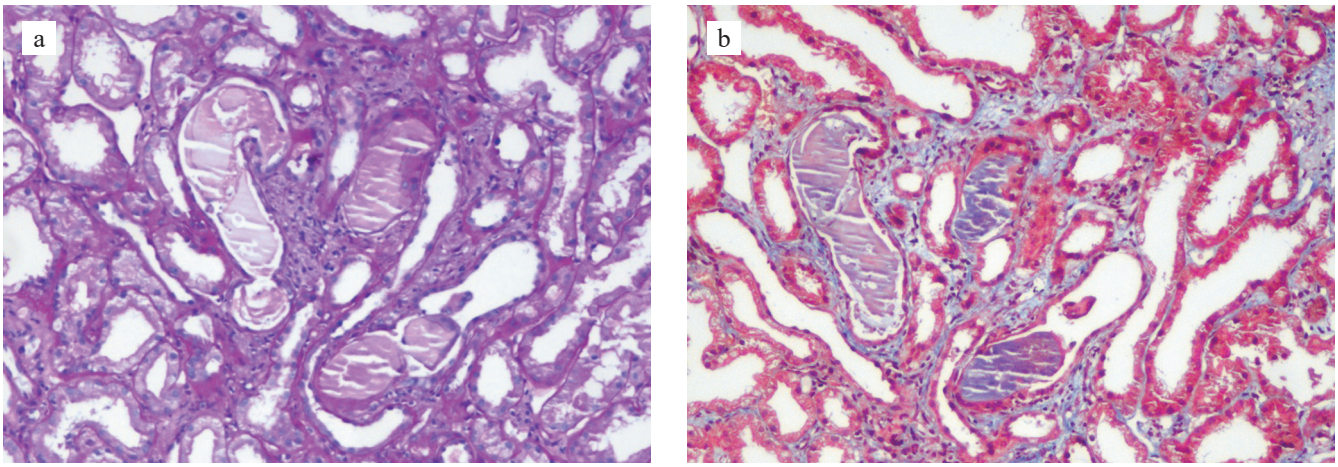

Fig. 2. Fragmented PAS-negative, fuchsinophilic casts, some surrounded by epithelial cells: (a) PAS (Periodic Acid-Schiff) stain, ×200; (b) Masson's trichrome stain, ×200

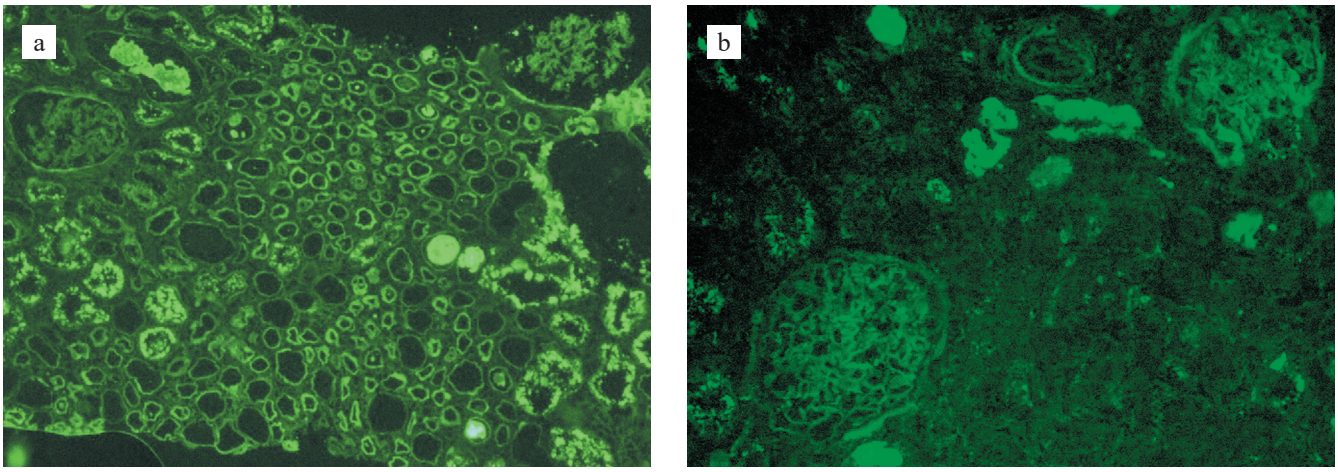

Fig. 3. Immunofluorescence study: (a) Kappa light-chain fluorescence in casts (++) and resorptive granules in the tubular epithelium (++), ×100; (b) Absence of lambda light-chain fluorescence, ×200

bone tissue brightening is visible at the junction of the right occipital and parietal bones.

Hematological diagnosis: MM with Bence Jones proteinuria, stage III B, complicated by myeloma nephropathy of the renal allograft, characterized by a combination of LCCN and LCPT. The diagnosis was established on the basis of bone marrow cytology (66.4% plasma cells), complete blood count findings (anemia), histological and immunohistochemical analysis of the renal allograft biopsy (LCCN and LCPT), detection of Bence Jones proteinuria, and skull X-ray results (areas of brightening).

The patient was transferred to the hematology ward for chemotherapy. Repeated courses of polychemotherapy resulted in partial hematological remission; however, recovery of renal allograft function was not achieved, and the patient continues maintenance treatment with scheduled hemodialysis.

DISCUSSION

MM in patients with CKD who have undergone KT is rare, with only isolated case reports and small case series reported [12–15]. Since the kidneys are the organs most commonly affected by MM, renal allograft dysfunction is often the first – and sometimes the only – clinical manifestation of the disease. In transplant recipients, differential diagnosis of graft dysfunction includes acute rejection, calcineurin inhibitor toxicity, viral infections, and other causes, including MM; therefore, renal allograft biopsy is a key diagnostic procedure.

Cast nephropathy, the most common renal lesion in MM, is characterized by distinctive morphological changes in renal tissue. Immunofluorescence studies demonstrating monoclonal light chain expression, together with additional hematological investigations, are essential for confirming the diagnosis. This diagnostic pathway was followed in the present clinical observation. One year after KT, the patient developed progressive allograft dysfunction, prompting a biopsy that revealed morphological features consistent with MM-related kidney injury, subsequently confirmed by further tests.

The biopsy results suggested combined renal involvement, including LCCN and LCPT; however, the latter was not confirmed by electron microscopy. Nevertheless, the presence of one or both patterns of renal injury did not influence the treatment strategy, as the patient was diagnosed with MM for the first time in the post-transplant period. Notably, the coexistence of LCCN and LCPT in a renal allograft represents an exceptionally rare manifestation of MM-related kidney injury and, to our knowledge, has been previously reported only by Singh et al. [14].

Post-transplant MM may develop either as a recurrence of previously diagnosed or undiagnosed disease present before KT, or as *de novo* post-transplant MM [12–15]. A single case of donor-derived MM has also been reported, occurring 7 years after KT; in that case, the kidney donor was the recipient's mother, who was diagnosed with MM 1 year after the transplant procedure [16].

Differentiating between recurrent MM present before transplantation and *de novo* post-transplant MM remains particularly challenging. This diagnostic difficulty is attributable to several factors that complicate the detection of MM in KT candidates. First, older patients, who are at higher risk for MM, often have comorbid conditions closely associated with CKD, which may mask underlying plasma cell disorders. Second, MM may exist in a subclinical or latent form, including non-secretory MM.

In both scenarios, patients typically do not undergo targeted diagnostic evaluations for MM prior to transplantation. Additionally, kidney biopsy is often avoided, as advanced nephrosclerosis is expected to yield limited diagnostic information, while the procedure itself carries a risk of complications [19, 20].

In the present case, we assume that MM was already present prior to KT, and that myeloma nephropathy was

the underlying cause of end-stage renal disease (ESRD) necessitating renal replacement therapy. The patient did not undergo targeted diagnostic testing for MM or kidney biopsy to determine the etiology of CKD, as he had a long-standing history of hypertension and diabetes mellitus (DM), both well-recognized causes of CKD.

In patients with DM, CKD is most commonly attributed to diabetic nephropathy, and kidney biopsy is therefore rarely performed in routine clinical practice. However, accumulating morphological evidence indicates that, even in the presence of DM, a substantial proportion of patients have non-diabetic kidney disease. Such cases are often characterized by atypical clinical features, including the absence of diabetic retinopathy, a short duration of DM (<5 years), hematuria, massive proteinuria, rapid decline in renal function, and extrarenal symptoms of systemic disease [21].

Retrospective analysis of the disease course in our patient before KT revealed several atypical features not characteristic of diabetic kidney disease, most notably severe anemia disproportionate to the degree of CKD and a relatively rapid progression to ESRD, both of which are characteristic of myeloma nephropathy. The diagnosis of ESRD was further supported by the lack of renal function recovery during maintenance hemodialysis, although it is recognized that certain extracorporeal techniques may facilitate the removal of free light chains and lead to restoration of AKI in patients with MM [22, 23].

Also noteworthy was the consistently high normal serum albumin level with low normal (before KT) and reduced (after KT) serum total protein content.

Establishing the correct etiology of CKD is a crucial component of pre-transplant evaluation, as it may significantly influence transplant outcomes due to the risk of disease recurrence in the patient's own kidneys. In this context, the feasibility of performing a native kidney biopsy should be carefully considered, particularly in older patients, including those with DM and hypertension, as well as in cases characterized by an atypical clinical course or rapid progression of renal failure.

Serum and urine protein electrophoresis, commonly used as a screening test for MM, have limited diagnostic sensitivity, underscoring the value of kidney biopsy for definitive etiological diagnosis. When kidney biopsy cannot be performed and the cause of CKD remains unknown, it is recommended to assess monoclonal immunoglobulins in serum and daily urine using immunofixation, with quantification of the M-protein concentration, to rule out MM [24].

The development of *de novo* post-transplant MM in our recipient appears less likely. The patient lacked known risk factors associated with post-transplant MM, including Epstein–Barr virus infection or reactivation, concomitant cytomegalovirus infection, hepatitis C virus infection, or intensification of immunosuppressive therapy during the first episode of graft dysfunction. Moreover, renal allograft dysfunction was detected relatively soon after transplantation.

In this case, the diagnosis of MM was late – established only after KT, following the onset of graft dysfunction that prompted allograft biopsy, which revealed typical morphological features of severe myeloma nephropathy. Similar clinical scenarios have been reported by other authors [12–15]. It is plausible that performing an earlier transplant biopsy, during the first post-transplant hospitalization (approximately 2 months after surgery), might have enabled earlier diagnosis and initiation of therapy for MM, potentially preserving renal allograft function.

According to reports, several recipients with post-transplant MM experienced restoration of kidney transplant function during chemotherapy, but most patients ultimately required dialysis therapy [14, 25, 26].

The attitude of clinicians toward KT in patients with MM remains ambiguous. The limited number of such procedures can be attributed to the historically incurable nature of MM, the high risk of disease recurrence, and the increased susceptibility to infectious complications following transplantation. However, the introduction of novel chemotherapeutic agents and the widespread use of autologous hematopoietic stem cell transplantation (auto-HSCT) have markedly improved outcomes in patients with MM and ESRD, resulting in survival rates comparable to those of the general MM population.

As life expectancy in MM continues to increase, KT has emerged as a realistic therapeutic option for selected patients. Notably, patients with MM receiving dialysis therapy who have achieved a very good partial response or complete remission should not be excluded from the list of candidates for KT [27–29].

To date, only isolated cases and small series of successful KT in patients with MM have been reported, demonstrating satisfactory allograft function and sustained MM remission lasting several years [30–35]. In particular, Shah et al. [33] reported outcomes in five patients with MM who underwent auto-HSCT followed by KT after a median interval of 27 months. Disease recurrence occurred in two patients at 6 and 16 months post-transplant, while the remaining patients continue to be followed. Death-censored graft survival and overall patient survival were both 80% at four years.

Dykes et al. [34] compared graft and patient survival in KT recipients with pre-existing MM to those in the general KT population and demonstrated comparable graft and overall survival rates between MM patients with ESRD and age-matched controls. Additionally, isolated reports have described successful treatment of MM in KT recipients using modern chemotherapy regimens in combination with auto-HSCT [25, 26].

Importantly, patients who have undergone dual transplantation, autologous stem cell transplantation and KT, represent a particularly challenging group to manage.

These patients require combined immunosuppressive therapy, necessitating careful dose adjustment to minimize cytopenia, which increases the risk of opportunistic infections and other complications [35].

CONCLUSION

The clinical case presented represents a rare case of MM diagnosed in a KT recipient. Post-transplant MM, whether due to recurrence of previously undiagnosed disease or *de novo* MM, is associated with rapid decline and loss of kidney transplant function. In the present case, we have attempted to provide compelling arguments supporting recurrence of MM that remained undiagnosed during the pre-dialysis and dialysis stages of CKD. A thorough pre-transplant evaluation, including accurate determination of the etiology of CKD, is the key to timely diagnosis of MM in KT candidates. Furthermore, the development of renal allograft dysfunction mandates kidney transplant biopsy to establish the underlying cause.

Written informed consent was obtained from the patient for the publication of this clinical case and the results of the renal allograft biopsy.

The authors declare no conflict of interest.

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