

Diffuse Kidney and Retroperitoneal Infiltration by Plasmablastic Myeloma

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Abstract

Plasmablastic cell myeloma is a rare but aggressive form of multiple myeloma (MM) with a poor survival. Extramedullary localization of plasmablastic myeloma may be observed in more than two-thirds of patients mainly in the upper digestive tract. Herein, we report the first case of plasmablastic MM leading to acute kidney injury through renal and ureteral infiltration.

Keywords: Myeloma; Acute kidney disease; Retroperitoneal infiltration

Introduction

Multiple myeloma (MM) is a malignant plasma cell neoplasm accounting for 10% of all hematologic cancers [1]. Extramedullary manifestations such as acute kidney injury (AKI) may reveal MM in 20% of patients. MM-associated AKI is usually secondary to intrarenal lesions such as cast nephropathy, amyloidosis, hypercalcemia, hypovolemia or drug toxicity [2]. Herein, we report on an unusual case of AKI due to ureteral obstruction by an extramedullary localization of a plasmablastic myeloma, an atypical and aggressive form of MM.

Case Report

A 67-year-old male patient was referred to our department for AKI in the setting of an MM relapse. Four years earlier, the patient received a course of proteasome inhibitor (bortezomib) and dexamethasone for an IgA MM, and underwent a success-

ful autologous hematopoietic stem cell transplantation. He had been considered in complete hematological remission under lenalidomide as maintenance therapy.

On admission, the patient complained of abdominal pain, fluid overload, hypertension and oliguria. Serum creatinine was 635 $\mu\text{mol/L}$ and 24-h proteinuria was 3.92 g. Other laboratory tests showed calcemia 2.4 mmol/L, bicarbonates 22 mmol/L, albumin 23 g/dL and C-reactive protein 20 mg/L, a generative normocytic anemia (hemoglobin 9.2 g/dL), leukopenia (total white blood cells at 3,450/mm³), but normal platelets count (277,000/mm³). Immunofixation revealed a monoclonal IgA kappa with an excess of free light chains (kappa chain 772 mg/L, lambda chain 50 mg/L) and Bence-Jones proteinuria was detected. The lactate dehydrogenase (LDH) level was 7.79 $\mu\text{kat/L}$ (normal range, 2.25 - 3.75 $\mu\text{kat/L}$). Bone marrow aspiration was not performed as all clinical and biological findings were consistent with the diagnosis of MM relapse. Abdominal ultrasound showed enlarged kidneys without urinary tract obstruction.

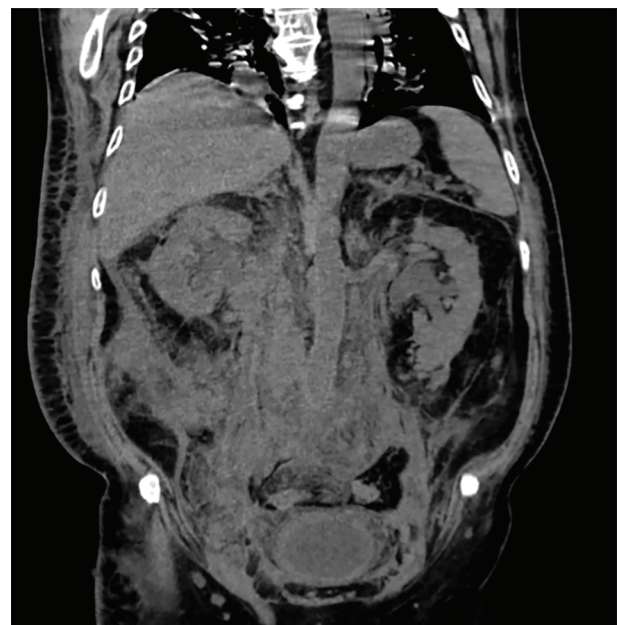


Figure 1. Abdominal CT scan. Large intra-abdominal mass arising from soft tissues and extending in the pelvic region. Dilatation of both ureters was noted.

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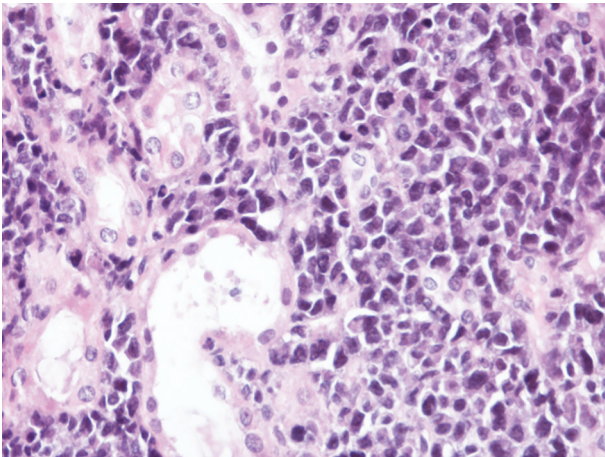


Figure 2. Kidney biopsy, light microscopy (HES, × 400). Diffuse an intense infiltration of the interstitium by plasmablasts.

The patient was started immediately on chemotherapy (dexamethasone and bortezomib). One week after initiation of therapy, he complained of dysuria. A bacterial prostatitis (*Escherichia coli*) was diagnosed and treated with parenteral antibiotics (piperacillin and tazobactam). He rapidly developed anuria requiring hemodialysis 7 days later. Despite hemodialysis, clinical symptoms worsened as he complained of abdominal pain associated with dyspnea. Abdominal CT scan identified a large retroperitoneal mass infiltrating both kidneys, compressing the ureters, and diffusing into the bladder (Fig. 1). The patient died 3 days later in the setting of uncontrolled sepsis.

A biopsy of the retroperitoneal mass and kidney disclosed a diffuse infiltration of conjunctive tissues and kidney by CD138⁺ CD20⁻ plasmacytoid cells with a high Ki67 expression (Fig. 2). The diagnosis of plasmablastic myeloma was made.

Discussion

MM is characterized by the clonal proliferation of plasma cells in the bone marrow and usually by the presence of a monoclonal Ig in the blood and/or the urine [1, 2]. In some patients, MM may take the form of plasmablastic myeloma, in which myeloma cells are detected in the circulation [3]. This plasmablastic transformation usually represents the terminal evolution of MM with a poor response to chemotherapy [3-6]. Plasmablastic myeloma cells are defined as having a fine reticular nuclear chromatin pattern, a large centrally placed nucleus (> 10 μm), a large nucleolus (> 2 μm) and a high nuclear/cytoplasmic ratio. The cytoplasm lacks a prominent perinuclear hof (area containing the Golgi apparatus) [6, 7].

Extramedullary localization of plasmablastic myeloma may be observed in more than two-thirds of patients [6] mainly in lymph nodes, the upper digestive tract, a common site of primary extramedullary plasmacytoma, and the central nervous system [6, 8]. Kidney involvement has been rarely reported [9].

Rare cases of AKI due to MM cells infiltrating the kid-

neys have been reported so far, within possible coexistent cast nephropathy [10-12]. To the best of our knowledge, this is the first case of plasmablastic MM leading to AKI through renal and peri-renal infiltration.

The rapid onset of ureteral obstruction, and the high proliferation of tumoral cells assessed by the Ki67 expression, reflect the aggressiveness of the plasmablastic form of MM.

Conclusion

Plasmablastic cell myeloma is a rare but aggressive form of MM with a poor survival. Plasmablastic transformation should be considered in MM presenting with AKI, high level of LDH and kidney enlargement and/or obstruction.

Conflicts of Interest

None.

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