Chasing incessant urinary tract infections results in an intriguing case of myeloma kidney.

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Background:

Light chain cast nephropathy, formerly known as myeloma kidney, is the primary cause of renal failure in multiple myeloma (MM). At the time of presentation, about 50% of patients had kidney involvement, strongly associated with higher mortality rates.

Case presentation:
A 68-year-old white female with a history of osteoarthritis on chronic NSAIDs and recurrent UTIs, proteinuria treated with long-term antibiotics, presented to the Emergency Department with the chief complaint of lightheadedness, described as episodic, accompanied by gait instability, polyuria, polydipsia, back pain, and constipation. Negative for urinary symptoms or weight loss. Medical records obtained from Primary Care Physician proven history of proteinuria, pyuria, recurrent UTIs with no significant UFC, and normal kidney function. Upon presentation unremarkable physical examination. Laboratory showed leukocytosis, anemia, and severely impaired kidney function. Further work-up such as ionized calcium, parathyroid hormone, and vitamin D compatible with malignant hypercalcemia. Urinalysis showed bacteriuria and proteinuria. Therapy was started with isotonic fluids, calcitonin, and bisphosphonates avoided in the setting of severe kidney impairment, and nephrotoxin agents were removed. Due to high suspicion of underlying MM Bence-jones protein urine showed M spike 78.5%. To confirm the diagnosis a bone marrow biopsy was done and showed plasma cell myeloma (95% lambda-restricted plasma cells). Due to new onset kidney disease, aimed to high suspicion for nephropathy, a kidney biopsy was done showing light chain cast nephropathy, and chronic active interstitial nephritis. Renal replacement therapy was contemplated if there was no improvement. The oncologist and Nephrologist were consulted, and the patient successfully started on pulse steroids and received the first dose of bortezomib. There was a remarkable improvement in kidney function, and hypercalcemia was resolved. The patient was discharged safely to continue to be followed by the Oncologist.

Conclusions:
Light chain cast nephropathy should be suspected in patients who do not carry a diagnosis of MM but have evidence of a monoclonal protein (M-protein) in the urine. More testing is warranted to evaluate MM. Clinical features include acute kidney injury, proteinuria, electrolyte abnormalities, and evidence of tubular dysfunction. This can occur as the first manifestation or develop later during myeloma. The key is found if a monoclonal protein is involved in the pathogenesis of kidney disease; in most cases, kidney biopsy is needed. The treatment consists of anti-myeloma therapy, fluid management, and, in patients with severe acute kidney injury, dialysis. In terms of prognosis, patients with significant kidney dysfunction at presentation tend to have worse outcomes than those without. Reported rates of kidney function improvement in patients with newly diagnosed myeloma treated with bortezomib-based chemotherapy range between 50-80%. This case has

clinical relevance in patients with persistent proteinuria, UTI´s, risk factors, new onset of kidney impairment, these features could be masking an underlying serious and deadly pathology. When facing these cases, a thorough workup is needed.