A 41-year-old white male with a prior medical history of insulin dependent diabetes and hypertension was admitted from outpatient clinic with nausea, emesis, back pain and an elevated creatinine level above his baseline. He works as a correctional officer and noted back injury about six months prior while transferring a patient off a stretcher. Over the past month, his back pain flared intermittently with moderate exercise. He described the pain as sharp and radiating down his back into his bilateral buttocks. He denied any weakness in his lower extremities and had no bowel or bladder incontinence. His pain improves with ibuprofen, up to 600 milligrams every six hours and methocarbamol. He had obtained the most relief from 30 mg ketorolac injections administered at two separate visits to his local emergency department over the last two weeks. He controls his diabetes with daily injections of insulin glargine. He has no known complications of diabetes including neuropathy, nephropathy or retinopathy. He is compliant with his diabetic diet but has not exercised regularly for several months due to back pain. His weight increased over the past year with diabetic diet but has not exercised regularly for several months due to back pain. His weight increased over the past year with diabetic diet but has not exercised regularly for several months due to back pain. His weight increased over the past year with diabetic diet but has not exercised regularly for several months due to back pain. His weight increased over the past year with diabetic diet but has not exercised regularly for several months due to back pain. His weight increased over the past year with diabetic diet but has not exercised regularly for several months due to back pain. His weight increased over the past year with diabetic diet but has not exercised regularly for several months due to back pain. 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13,000 deaths/year are attributed to MM in the United States. This correlates with an annual incidence of approximately seven per 100,000 men and women per year. Mean age at presentation is seventy, but 36 percent are under sixty-five years. The risk of MM increases with body mass index. There is an association between Agent Orange exposure and MM. The diagnosis of MM is suspected when one (or more) of the following clinical findings occur at presentation: Increased serum creatinine level, renal insufficiency, anemia and bone lesions. (CRAB) All four were present on admission in our patient. Given the non-specific nature of this quartet, these factors must be related to an underlying plasma cell proliferative disorder to make the diagnosis. More specific clinical presentations include increased total serum protein concentration and/or the presence of a monoclonal protein in the urine or serum; hypercalcemia, can be either symptomatic or discovered incidentally. Systemic signs or symptoms suggestive of malignancy (unexplained anemia), and bone pain with lytic lesions discovered on routine skeletal films or other imaging modalities. Interestingly as in our case, MM-related bone pain as a presenting complaint occurs in approximately sixty percent of patients. Pain typically involves the central skeleton and is induced by movement. Positron emission tomography (PET/CT), and magnetic resonance (MRI) have a higher sensitivity than skeletal surveys for detection of bone lesions and enables earlier detection which improves prognosis. MRI is the most sensitive modality for bone involvement, while PET/CT may be more sensitive for non-osseous tissue involvement. Despite the lower sensitivity, CT is used most frequently for its convenience and lower cost.

Acute renal failure is a rare presentation and can manifest with a bland urinalysis or rarely nephrotic syndrome due to concurrent immunoglobulin light chain (AL) amyloidosis. The two major causes of renal insufficiency in patients with MM are light chain cast nephropathy (also called myeloma kidney) and hypercalcemia. Patients who do not secrete light chains are not at risk for myeloma kidney. In the absence of other causes of renal failure, a presumptive diagnosis of light chain cast nephropathy is made in the setting of high free light chain (FLC) levels. Renal biopsy should be performed to characterize the histologic changes in patients who may have other potential risk factors for renal impairment, in whom the mechanism of kidney injury is not clear such as seen in our case. Renal insufficiency, can be acute or insidious in onset, occurs in approximately fifty percent of patients with MM. A variety of etiologic mechanisms may be involved. These include excess production of monoclonal light chains causing cast nephropathy, deposition of intact light chains causing nephrotic syndrome (light chain deposition disease), amyloidosis, hypercalcemia; radiocontrast media-induced acute renal failure (usually due to dehydration in the presence of Bence Jones proteinuria) and, infrequently, very high levels of uric acid. Ionized calcium should be measured if the patient has a high serum calcium level but no symptoms of hypercalcemia, as elevation of serum calcium concentration may not occur due to binding of the M protein with calcium. Severe hypercalcemia can act as an unmeasured cation and result in a low anion gap as seen in our case. Decreased anion gap may also be due to the high IgG molecule manifesting as additional cations. All patients with MM should take precautions to minimize risk of renal damage. These include avoidance of nephrotoxins such as aminoglycosides and NSAID’s and maintenance of hydration by drinking enough fluids to produce 3 liters of urine per day. Certain medications frequently used for the treatment of MM or its complications such as zoledronic acid may require dose adjustment for renal insufficiency or may contribute to renal failure. The treatment of renal insufficiency is directed at the underlying cause and may require the use of plasmapheresis and/or hemodialysis in the setting of acute renal failure. The presence of acute renal failure due to light-chain cast nephropathy may also have an impact on the choice of initial chemotherapy. Our patient was treated with bortezomib, cyclophosphamide, plus dexamethasone, which do not need dose reductions for renal failure. Weekly doses of the drugs used in these regimens can be adjusted based on counts and other adverse effects. Usually, renal function improves with therapy, as in our patient.

The initial evaluation of our patient found him eligible for high-dose therapy (HDT) based on his age, performance status, and limited comorbidities. With the introduction of novel agents, thalidomide, bortezomib and lenalidomide, as part of induction treatment have markedly improved the rate of complete response (CR). These have not increased toxicity and improved time to progression (TTP). Progression-free survival (PFS) and overall survival (OS) have significantly improved. Long term follow-up and disease progression will determine further treatment.

REFERENCES


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