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AN UNUSUAL CAUSE OF ACUTE INTERSTITIAL NEPHRITIS

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ABSTRACT

Multiple myeloma is a monoclonal proliferation of B-cell lineage, often referred to as plasma cell dyscrasias. Early diagnosis, is of immense importance because initiation of treatment at early stage has a vital role in the prognosis. This case was presented to enlighten an uncommon cause of acute interstitial nephritis. A 55 year old Indian women presented to medical outpatient department with epigastric discomfort and non oliguric renal failure. Investigation reports revealed non nephrotic range proteinuria, elevated urine eosinophils, urine bencejonce protein was positive, serum electrophoresis revealed M band and immunofixation confirmed multiple myeloma. Kidney biopsy was done and patient was diagnosed to have myeloma kidney. Patient found to have elevated serum beta-2-microglobulin and a reduced serum albumin which suggests poor prognosis. The patient is on chemotherapy and on regular follow up. Recognition of the disease before the onset of complications and early initiation of therapy offers good prognosis as it can prolong lifespan and prevent fatal complications.

KEY WORDS: multiple myeloma, beta 2 microglobulin, acute interstitial nephritis

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CASE PRESENTATION

A 68 year old female came to outpatient department with complaints of nausea and vomiting for 3 weeks contained mainly food particles , abdominal pain on and off for 2 weeks .Patient was a known case of diabetic and hypertensive since 5 years under regular treatment .Patient gave a recent past history of fever before 1 month ,for which she took over the counter medications without proper prescriptions. The medications, mainly contained BETA LACTAM antibiotics, NSAID’S and Acetaminophen. She had a normal bladder and bowel habit. On examination, her haemodynamic status was normal and systemic examination was also normal. Urine output was normal. Lab investigations such as complete blood count were normal except for Eosinophilia, serum urea -150 mg/dl and creatinine-6 mg/dl. Urine routine showed eosinophils. Sugar, puscells, other cells were negative. Serum electrolytes were normal. USG abdomen showed normal and near equal sized kidneys, corticomedullary differentiation well maintained. Since the patient presented with non oliguric acute renal failure with uremic gastritis following illicit analgesic abuse we initially thought it was drug induced interstitial nephritis. The offending agents were withdrawn and she was well hydrated but the serum urea and creatinine was increasing day by day .serum urea -165 ,creatinine 8.2.Since it is atypical picture of acute interstitial nephritis we proceeded with renal biopsy which revealed protein casts and light microscopy showed light chain deposits suggestive of myeloma of the kidney.

![Renal biopsy showing lambda light chain deposits in immunofluorescent microscopy](https://www.jpahs.net)

FIGURE 1

Then we proceeded with other investigations for myeloma,which revealed positive bence jones proteins and serum electrophoresis showed classical M-band. Beta 2 microglobulin was 6,serum albumin was 3.1 gm/dl. Patient was treated with chemotherapy, hemo dialysis for renal failure following which renal functions improved. The patient is now under regular follow up.

DISCUSSION

Acute interstitial nephritis is an acute inflammation of the kidney characterized by
cellular and fluid exudation in the interstitial tissue, accompanied by, but not purulent in character, and the lesions may be both diffuse and focal. It accounts for 10-15% cases of intrinsic renal failure. Although drugs accounts for the majority of cases, AIN also occurs in infectious diseases (bacterial, viral, parasitic), immunological diseases and acute obstructive disorders such as light chain cast nephropathy (myeloma kidney), acute phosphate and urate nephropathy. There are about more than 100 drugs that can cause AINs. Some common drugs causing this disorder are methicillins and other penicillins, sulfonamides, diuretics, rifampicin, allopurinol, cephalosporins and cimetidine. NSAIDs can interstitial nephritis with nephrotic syndrome. Though drugs account for more than 70% of cases, some cases have other causes also hence our case was brought to the lime light as it is caused by myeloma. The patient had myeloma, which had sudden flaring up after NSAID abuse.

**Multiple myeloma**

It is a clonal expansion of abnormal, proliferating plasma cells producing a monoclonal paraprotein, mainly IgG (55%) or IgA (20%) and rarely IgM and IgD. There are variety of clinical presentations in myeloma. 20% of patients of presents asymptomatic and detected by lab investigations. Skeletal presentations occur due to areas of osteolysis with expanding plasma cell in the marrow. Again, this can be either symptomatic (bone pain, vertebral fractures) or asymptomatic (radiological evidence). Metabolic presentations often due to hypercalcemia where calcium is mobilized from bone during osteolytic activity. Haematological presentation may involve all the three lineages due to expanding plasma cell in bone marrow. Renal presentation can happen like our case usually presents with acute renal failure, which may be due to myeloma itself or by hypocalcemia. Infectious manifestations occur as there is deficient, normal immunoglobulin secretion. Some can manifest with neurological complications like radiculopathy, cord compression due to plasma cell proliferation and amyloid infiltration. Hyperviscosity can cause complications like bruising, epistaxis, headache can be present.

**Prognosis**

The disease has a prolonged course, but carries a poor prognosis. Most patients succumb within 4 years time. Certain translocations have also been associated with a poor prognosis: t(4;14), t(14;16), and del17p13. The median survival with these translocations is about 25 months.

**International staging system (2005) of multiple myeloma is**

- Stage I: $\beta_2$ microglobulin ($\beta_2$M) < 3.5 mg/L, albumin $\geq$ 3.5 g/dL
- Stage II: $\beta_2$M < 3.5 mg/L and albumin < 3.5 g/dL; or $\beta_2$M 3.5–5.5 mg/L irrespective of the serum albumin
- Stage III: $\beta_2$M $\geq$ 5.5 mg/L

**Durie-Salmon staging system (1975)**

- stage I: all of
  - Hb > 10g/dL
  - normal calcium
  - Skeletal survey: normal or single plasmacytoma or osteoporosis
  - Serum paraprotein level < 5 g/dL if IgG, < 3 g/dL if IgA
  - Urinary light chain excretion < 4 g/24h
- stage II: fulfilling the criteria of neither I nor III
  - stage III: one or more of
    - Hb < 8.5g/dL
    - high calcium > 12 mg/dL
• Skeletal survey: Three or more lytic bone lesions
• Serum paraprotein> 7g/dL if IgG, > 5 g/dL if IgA
• Urinary light chain excretion > 12g/24h

**Differential diagnosis**
1. Smouldering myeloma
2. Plasma cell leukemia
3. Non secretary myeloma
4. POEMsyndrome
5. Waldenstrom’s macroglobulinemia
6. Heavy chain disease.
7. Monoclonal gammapathy of undetermined significance.

**Chemotherapy options for myeloma**
1. Pulse dexamethasone
2. Dexamethasone and thalidomide
3. Lenalidomide and dexamethasone
4. Melphalan ,prednisolone and thalidomide
5. Bortezombib
6. Pomalidomide is recently approved may be used in the case of refractory or relapsing plasma cell neoplasm.

Patients who are not eligible for HCT are usually treated with combinations of melphalan and prednisone with or without bortezombib or thalidomide and its analogues 3,4.

Stem cell transplant along with chemotherapy may increase the period of survival. With high-dose therapy followed by autologous stem cell transplantation, the median survival has been estimated in 2003 to be approximately 4.5 years, compared to a median of approximately 3.5 years with "standard" therapy.5

Plasmapheresis can also be instituted to remove free light chains if needed6,7

**CONCLUSION**

Multiple myeloma is a monoclonal proliferation of B-cell lineage, often referred to as plasma cell dyscrasias. There are varieties of clinical presentations in myeloma. 20 % of patients of presents asymptomatic and detected by lab investigations. Early diagnosis is of immense importance because initiation of treatment at an early stage has a vital role on the prognosis. This case was presented to highlight an uncommon cause of acute interstitial nephritis.

**REFERENCES**


