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**Original Research Article**

# Renal Function Tests in Multiple Myeloma Patients before and After Chemotherapy

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**Conflict of interest: Nil**

**Abstract:**

**Objective:** This study was done to assess renal involvement in multiple myeloma patients with the aid of renal function tests before and after chemotherapy.

**Material and Methods:** The present study was conducted in the Department of Biochemistry in collaboration with Department of Medicine, Pt B D Sharma PGIMS, and Rohtak. Twenty newly diagnosed patients of Multiple Myeloma (MM) were enrolled for the study. Five mL of venous blood was collected aseptically from antecubital vein in a plain red capped vacutainer after taking written consent. Samples were processed by centrifugation and analysed on the same day. We had been done biomarkers of renal profiles including S.Urea, S.Creatinine, Creatinine Clearance (GFR), S.uric acid, B.Sugar were analyzed on auto-analyzer (Randox Suzuka, United Kingdom, model no. 6L7WD5J) by using kits provided by Randox laboratories.

**Results**: It was observed that mean levels of S. Urea (46.00±17.26), S. Creatinine (1.77±1.05), GFR (50.10±26.90), S. Uric acid (4.69±1.39), B. Sugar (117±14.41) in MM patients before chemotherapy. The mean levels of S. Urea (28.20±9.99), S. Creatinine (0.97±0.18), GFR (78.03±25.14), S. Uric acid (4.29±0.57), B. Sugar (132±9.88) in MM patients after chemotherapy.

**Conclusion:** In conclusion, abnormal renal profile of MM patients is due to toxic effects of monoclonal light chain and plasmocytic infiltration of renal tissues. After treatment renal function improves which leads to improvement in renal profile.

**Keywords:** Multiple myeloma (MM), Renal Profile, Chemotherapy.



## Introduction

Multiple myeloma (MM) is a neoplastic disorder characterized by a single clone abundance of plasma cells in the bone marrow and generating a monoclonal immunoglobulin, which cause end organ damage and related complications such as anemia, renal insufficiency, hypercalcemia, skeletal events and infection. [1] Myeloma arises from an asymptomatic premalignant proliferation of monoclonal plasma cells that are derived from post germinal-center B cells. Multistep genetic and micro-environmental changes lead to the transformation of these cells into a malignant neoplasm. [2] Its incidence increases with age and is more common in males than females and blacks have twice the incidence of whites. It accounts 1.3% of all malignancies in whites and 2% in blacks and 13% of all hematologic cancer in whites and 33% in blacks. It is more common at 60-70 yrs of age. [3] In India, the incidence varies 0.3-1.9/100,000 for males and 0.4-1.3 / 100,000 for females. [4]

**Diagnostic criteria for multiple myeloma [5]**

1. Clonal bone marrow plasma cells more than or equal to 10% or biopsy proven plasmacytoma.
2. Presence of serum or urinary monoclonal protein (except in patients with true non secretory multiple myeloma).
3. Evidence of myeloma related organ dysfunction commmonly summerized under the CRAB acronym.
   a. C: Hypercalcemia: serum calcium more than or equal to 11.5mg/dl.
   b. R: Renal insufficiency: Serum creatinine > 2mg/dL or estimated creatinine clearance< 40ml/ min.
   c. A: Anemia: normochromic, normocytic with a hemoglobin value of 2g/dL below the lower limit of normal or a hemoglobin < 10g/dL.
   d. B: Bone lesions: Radiologically detected lytic lesion, severe oesteopenia or pathologic fracture.

MM is common in those individuals who works in agriculture field or with allergies. Hypersensitivity and defective immune response play a role in the occurence of this tumor. [6]

Renal failure is most common feature in MM. It is due to the toxic effects of monoclonal light chains which effects, mainly to renal tubules, less to glomeruli. Circulating monoclonal light chains are freely filtered through the glomerulus and go to the proximal tubule where they are catabolized. The endocytosis of these free light chains occur by proximal tubular cells, through a receptor-mediated process by binding to the cubilin/megalin. [7,8]

In MM light chain are produced in excess amount so they overcomes the capacity of the tubular cells to catabolize them so they appear in the tubular fluid and form tubular casts with Tamm-Horsfall Protein, it is a glycoprotein synthesized by the cells present in the medullary thick ascending limb of the loop of Henle. These casts leads to the obstruction of the distal tubule and the thick ascending loop of Henle. [9,10] When a light chain is nephrotoxic, it cause renal impairment in the early course of the disease, even before other clinical features of MM appear.

Kidney injury is a common complication of multiple myeloma and other plasma cell disorders and it is associated with increased mortality. It occur due to multiple pathogenic mechanism which are due to nephrotoxic monoclonal Ig and some are independent of paraprotein deposition. [11] Some of light chains are resistant to chemical degradation and they collect as precipitates that interfere with proximal tubule function. Some light chains are degraded and they induce pro inflammatory cytokines, such as monocyte chemo attractant protein-1 and IL-6 and 8. These light chains cause the production of reactive oxygen species, which cause inflammatory cell infiltration, matrix deposition, fibrosis or epithelial-mesenchymal transformation of renal tubular epithelial cells. [12]

Myeloma-related renal failure which occur from Ig-independent mechanisms. These are hypercalcemia, uric acid nephropathy, volume depletion, sepsis, rhabdomyolysis, nonsteroidal anti-inflammatory drugs, and renin-angiotensin system inhibitors. [13]

Diagnosis of MM is done by identification of abnormal monoclonal plasma cells in the bone marrow, M protein in the serum or urine. A complete blood count, peripheral blood smear, bio-chemistry includes calcium, creatinine, β2-M, lactate dehydrogenase and routine urinalysis. Serum protein electrophoresis, immunofixation, nephelometric quantitation of immunoglobulins and measurement of free light chains. [5]

Treatment options for MM are chemotherapy and stem cell transplantation. The chemotherapy for MM includes an initial induction regimen followed by maintenance therapy and on subsequent progression, management of relapsed disease.

Three-drug regimens that contains Bortezomib, Cyclophosphamide and Dexamethasone (VCD) Bortezomib, Thalidomide and Dexamethasone (VTD) and Bortezomib, Lenalidomide and Dexamethasone (VRD) are highly effective in newly diagnosed myeloma patients. VTD show better response rates and progression-free survival compared with Thalidomide and Dexamethasone (TD) and Bortezomib and Dexamethasone (VD). **[14]**

VTD regimen consisted of four 3-week cycles of Bortezomib 1.3 mg/m$^2$ administered subcutaneously (SC) on days 1, 4, 8, and 11.

Dexamethasone 40 mg on days 1-4 and 9-12.

Thalidomide 100 mg/day administered orally.

VCD regimen composed of four 3-week cycles of Bortezomib 1.3 mg/m$^2$ administered subcutaneously (SC) on days 1, 4, 8, and 11.

Dexamethasone 40 mg on days 1-4 and 9-12.

Cyclophosphamide 500 mg/m$^2$ administered orally on days 1, 8, and 15.

Including bisphosphonates, antibiotics, and antiviral for prophylaxis. [15]

The main goal of myeloma treatment is to reduce the level of light-chain production with chemotherapeutic regimens alone or followed by autologous stem cell transplant (ASCT). [16] In myeloma patients who are new to dialysis, ASCT cause recovery of renal function and discontinuation of dialysis in upto 24% of cases. So ASCT is the preferred therapy in patients with myeloma with advanced renal failure. [17,18]

Renal failure in MM is reverted by high fluid intake alone, or hydration combined with anti-myeloma treatment, the agents that are not excreted by the kidneys. Alkalinization of urine and treatment of hypercalcemia is also done. Bisphosphonates are very useful for the management of malignancy-related hypercalcemia, but they can cause renal failure and hypocalcemia in MM patients. [19] Calcitonin also reduce calcium levels without causing hypocalcemia and risk of renal toxicity. Free light chains which are removed with plasma exchange or in combination with anti-myeloma therapy, can prevent irreversible renal failure. The free-light chains are also removed with the help of dialysis. Hemodialysis membrane removes the circulating light chains more efficiently. [20]

**Aim & Objectives:** This study was done to assess renal involvement in multiple myeloma patients with the aid of renal function tests before and after chemotherapy.

**Material and Methods**

The present study was conducted in the Department of Biochemistry in collaboration with the Department of Medicine (Clinical Hematology Unit), Pt. B.D. Sharma PGIMS, and Rohtak. Twenty newly diagnosed patients of MM were enrolled for the study. The diagnosis was made by history, clinical examination, bone marrow examination and electrophoretic studies and other tests. Patients were included and excluded as per following criteria.

**Inclusion criteria:** Multiple myeloma patients after confirmed diagnosis.

**Exclusion criteria:** Patients on steroids, suffering from any renal, liver pathology or any other malignancy.

**Cases were categorized into two groups:**

**Group I:** At the time of presentation (pre-treatment) i.e. before the start of chemotherapy.

**Group II:** After 6 months of chemotherapy.

An informed written consent was obtained from the patients who participated in the study. Five mL of venous blood sample was collected under aseptic conditions from the patients before and after the treatment, in a plain red capped vacutainer.

Samples were processed within one hour of collection. Serum was separated by centrifugation (2000rpm for 10 minutes) after clotting and analysed on the same day.

We had been done biomarkers of renal profiles including S.Urea, S.Creatinine, Creatinine Clearance (GFR), S.Uric acid, B.Sugar were analyzed on auto-analyzer (Randox Suzuka, United Kingdom, model no. 6L7WD5J) by using kits provided by Randox laboratories.

All the analyses were performed by using the statistical package (IBM SPSS 20). Data were considered to be significant if p value < 0.05. Results were expressed as Mean± SD.

**Results and Observations**

**Table 1: Mean serum urea (mg/dL), creatinine (mg/dL), creatinine clearance (GFR) (mL/min/1.73m$^2$), uric acid (mg/dL), blood sugar (mg/dL) levels in group I and group II expressed in Mean±SD (Range).**

| Parameters with normal range | Group I | Group II | p-value |
|---|---|---|---|
| **S. urea**<br>(6 to 20 mg/dL) | 46.00±17.26<br>(20 to 72) | 28.20±9.99<br>(12 to 48) | **0.000** |
| **S. creatinine**<br>(For males 0.72 to 1.18 mg/dL)<br>(For females 0.55 to 1.02 mg/dL) | 1.77±1.05<br>(0.8 to 4.4) | 0.97±0.18<br>(0.7 to 1.4) | **0.002** |
| **Creatinine Clearance (GFR)** (Glomerular filtration rate) >60 mL/min/1.73m$^2$) | 50.10±26.90<br>(10.7 to 108.8) | 78.03±25.14<br>(42.0 to 126.9) | **0.000** |
| **S. uric acid**<br>(For males 3.5 to 7.2 mg/dL)<br>(For females 2.6 to 6.6 mg/dL) | 4.69±1.39<br>(2.8 to 8.0) | 4.29±0.57<br>( 3.2 to 5.8) | 0.200 |
| **Blood sugar**<br>(Fasting-75-115 mg/dL)<br>(Post-prandial-75-140 mg/dL) | 117±14.41<br>(100 to 150) | 132±9.88<br>( 110 to 148) | **0.000** |

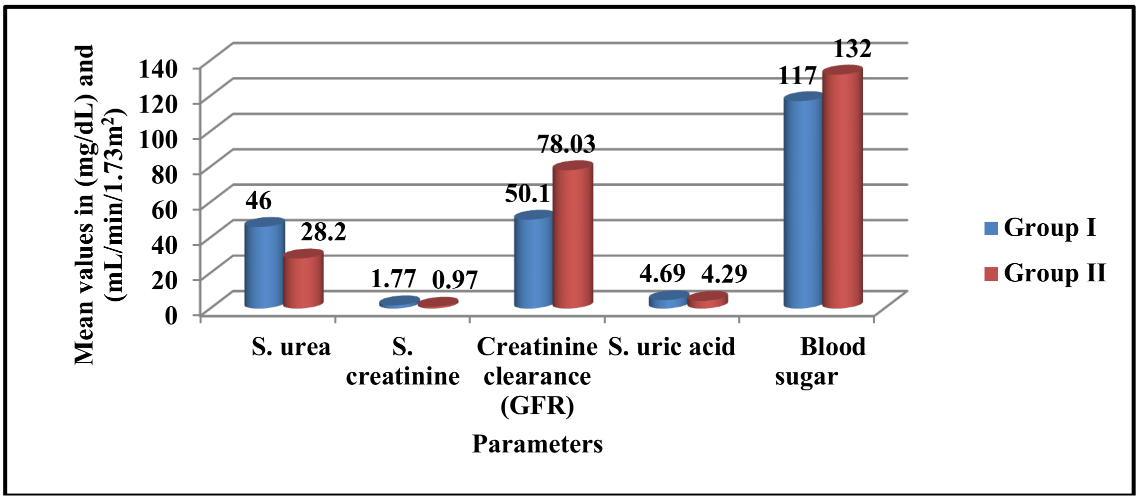


**Figure 1: Mean serum urea, creatinine, creatinine clearance, uric acid and blood sugar levels in group I and group II**

## Discussion

In the present study, mean serum urea levels in group I was 46.00±17.26 mg/dL (range 20 to 72 mg/dL) and in group II was 28.20±9.99 mg/dL (range 12 to 48 mg/dL). It was decreased in group II and this decrease in mean urea levels was statistically significant (p-value 0.000). This increase in the mean urea levels in (group I) is due to renal insufficiency in MM patients.[1]

The mean serum creatinine levels in group I was 1.77±1.05 mg/dL (range 0.8 to 4.4 mg/dL) and in group II was 0.97±0.18 mg/dL (range 0.7 to 1.4 mg/dL). It was decreased in group II and this decrease in mean creatinine levels was statistically significant (p-value 0.002). Kyle et al, also reported increase in the creatinine levels in MM patients out of which some patients with an initial creatinine value lower than 5 mg/dL responded to therapy, but dialysis was required in the other patients. [21] Kaur et al, also reported increase in the creatinine levels in 22 patients (out of 28) of MM.[4] The mean creatinine clearance (GFR) in group I was 50.10±26.90 mL/min/1.73m$^2$ (range 10.7 to 108.8 mL/min/1.73m$^2$) and in group II was 78.03±25.14 mL/min/1.73m$^2$ (range 42.0 to 126.9 mL/ min/1.73m$^2$). This difference in mean creatinine clearance (GFR) was statistically significant with p-value 0.000. It was observed when serum creatinine level decreased after chemotherapy (group II), Creatinine clearance (GFR) also improved in these patients due to improvement of renal functions.

The mean serum uric acid levels in group I was 4.69±1.39 mg/dL (range 2.8 to 8.0 mg/dL) and in group II was 4.29±0.57 mg/dL (range 3.2 to 5.8 mg/dL). It was decreased in group II but this was not statistically significant (p-value 0.200).

In this study mean uric acid levels were within normal range. Kyle et al, reported the serum uric acid levels were more than 8.0 mg/dL in 27% of patients and more than 12 mg/dL in 2% of patients out of 1027 patients.[21]

The mean blood sugar levels in group I was 117±14.41 mg/dL (range 100 to 150 mg/dL) and in group II was 132±9.88 mg/dL (range 110 to 148 mg/dL). This difference in mean serum sugar level was statistically significant (p-value 0.000). In group II mean of blood sugar was higher than group I which may be due to dexamethasone which causes increase blood sugar levels.

Balwani et al, also observed renal complication in MM patients with acute kidney injury in 72% cases, chronic kidney disease in 24% and nephrotic syndrome in 0.03% cases. They also observed that 15 patients with acute kidney injury and 7 with chronic kidney disease were treated with hemodialysis. Four patients with acute kidney injury had complete recovery of renal function at 12 months of follow-up. 11 patients of acute kidney injury who required hemodialysis support initially later on recovered to non-dialyzable range of renal failure. Plasmapheresis was done along with high flux hemodialysis in 5 patients. 4 of 5 patients became hemodialysis independent while 1 patient progressed to chronic kidney disease requiring maintenance hemodialysis. 7 patients of chronic kidney disease remained on maintenance hemodialysis. In their study 4 patients had recovery of renal function after 6 weeks of dialysis, and their renal function was within normal range at follow-up of one year. Also MM had undergone remission in these 4 patients.

Therefore, dialysis treatment along with chemotherapy should be continued for several weeks if required. Rapid removal of light chains with chemotherapy and plasmapheresis may prevent irreversible renal dysfunction. [22]

De Martinis et al, studied that deficiency of Vit-D is associated with higher CRP, serum creatinine, ISS stage or lower serum albumin levels in patients of MM. Vit–D deficiency cause poorer outcomes in patients with MM. CRP and creatinine levels are related to the prognosis in MM. The higher levels of both CRP and creatinine cause poorer outcomes and survival in patients with MM. Vitamin D supplementation decrease the CRP levels supporting the anti-inflammatory role of vitamin D. [23]

Leung et al, studied that in patients with MM, myeloma kidney is the most common cause of severe irreversible renal failure. The tubulointerstitial injury occur due to high concentrations of circulating monoclonal free light chains which are produced by plasma cells. In severe renal failure, serum half-life of FLC is prolonged, so early reduction of serum FLCs concentrations is necessary, which gives an additional benefit to effective chemotherapy. For the direct removal of FLCs from the serum, there are two methods that is the plasma exchange and high cut-off haemodialysis. Plasma exchange removes only FLCs from the serum but its capacity is limited to reduce total body FLCs. High cut off hamodialysis is used safely for the extended period of time and has greater capacity for reducing total body FLCs.

The early reduction of FLCs is important for renal recovery, when FLCs are reduced by 60% on day 21, it is associated with renal recovery for 80% of the population. Patient survival is also strongly associated with renal recovery. The renal recovery is reduced when there is significant fibrosis. The patients who have both light chain deposition disease (LCDD) and Myeloma kidney on renal biopsy have non-significant reduction in the renal recovery. In the dialysis-dependent patients when there is reduction in the serum FLCs levels of 62% by day 12 then the median time to independence of dialysis is 27 days. When the serum FLCs are reduced by 75% then it is associated with the median time to independence of dialysis is 13 days, and in 50 to 74% reduction this time increased to 26 days and is 34 days when the reduction is 25 to 49%. So an early reduction of FLCs is related to early renal recovery in MM patients. [24]

## Conclusion

MM is a plasma cell disorder of elderly population between the age of 60-70 yrs. Renal and bone involvement is the most common feature. Reversal of renal function was achieved with chemotherapy and high flux hemodialysis in few cases. Rapid removal of light chains with chemotherapy and plasmapheresis prevents irreversible renal dysfunction. The prognosis was worse when renal failure develops during chemotherapy. [22] So early diagnosis and treatment of renal failure is important to improve the quality of life and prevent organ damage.

## References


1. Dimopoulos M, Bringhen S, Anttila P, Capra M, Cavo M, Cole C, Gasparetto C, Hungria V, Jenner M, Vorobyev V, Ruiz EY, Yin JY, Saleem R, Hellet M, Macé S, Paiva B, Vij R. Isatuximab as monotherapy and combined with dexamethasone in patients with relapsed/refractory multiple myeloma. Blood. 2021 Mar 4; 137(9):1154-1165. doi: 10.1182/ blood.2020008209.
2. Rajkumar SV. Multiple myeloma: 2022 update on diagnosis, risk stratification, and management. Am J Hematol. 2022 Aug; 97(8):1086-1107. doi: 10.1002/ajh.26590. Epub 2022 May 23.
3. Munshi NC, Longo DL, Anderson KC. Plasma cell disorders. In: Lango DL, Kasper DL, Jameson JL, Fauci AS, Hauser SL, Loscalzo J, editors. Harrison's Principles of Internal Medicine. 19th ed. New York: McGraw-Hill; 2015. p. 710-7.
4. Kaur P, Shah BS, Baja P. Multiple myeloma: a clinical and pathological profile. Gulf J Oncolog. 2014 Jul; 1(16):14-20.
5. Kyle RA, Rajkumar SV. Criteria for diagnosis, staging, risk stratification and response assessment of multiple myeloma. Leukemia. 2009 Jan; 23(1):3-9. doi: 10.1038/leu.20 08.291. Epub 2008 Oct 30. Erratum in: Leukemia. 2014 Apr; 28(4):980.
6. Georgakopoulou R, Fiste O, Sergentanis TN, Andrikopoulou A, Zagouri F, Gavriatopoulou M, Psaltopoulou T, Kastritis E, Terpos E, Dimopoulos MA. Occupational Exposure and Multiple Myeloma Risk: An Updated Review of Meta-Analyses. J Clin Med. 2021 Sep 16; 10(18):4179. doi: 10.3390/jcm10184179.
7. De S, Kuwahara S, Saito A. The endocytic receptor megalin and its associated proteins in proximal tubule epithelial cells. Membranes (Basel). 2014 Jul 11; 4(3):333-55. doi: 10.3390/membranes4030333.
8. Santostefano M, Zanchelli F, Zaccaria A, Poletti G, Fusaroli M. The ultrastructural basis of renal pathology in monoclonal gammopathies. J Nephrol. 2005 Nov-Dec; 18(6):659-75.
9. Hutchison CA, Batuman V, Behrens J, Bridoux F, Sirac C, Dispenzieri A, Herrera GA, Lachmann H, Sanders PW; International Kidney and Monoclonal Gammopathy Research Group. The pathogenesis and diagnosis of acute kidney injury in multiple myeloma. Nat Rev Nephrol. 2011 Nov 1; 8(1):43-51. doi: 10.1038/nrneph.2011.168.
10. Bridoux F, Leung N, Belmouaz M, Royal V, Ronco P, Nasr SH, Fermand JP; International Kidney and Monoclonal Gammopathy Research Group. Management of acute kidney injury in symptomatic multiple myeloma. Kidney Int. 2021 Mar; 99(3):570-580. doi: 10.1016/j.kint.2020.11.010. Epub 2021 Jan 10.
11. Heher EC, Rennke HG, Laubach JP, Richardson PG. Kidney disease and multiple myeloma. Clin J Am Soc Nephrol. 2013 Nov; 8(11):2007-17. doi: 10.2215/CJN.12231212. Epub 2013 Jul 18.
12. Kundu S, Jha SB, Rivera AP, Flores Monar GV, Islam H, Puttagunta SM, Islam R, Sange I. Multiple Myeloma and Renal Failure: Mechanisms, Diagnosis, and Management. Cureus. 2022 Feb 25; 14(2):e22585. doi: 10.7759/cur eus.22585.
13. Bridoux F, Cockwell P, Glezerman I, Gutgarts V, Hogan JJ, Jhaveri KD, Joly F, Nasr SH, Sawinski D, Leung N. Kidney injury and disease in patients with haematological malignancies. Nat Rev Nephrol. 2021 Jun; 17(6):386-401. doi: 10.1038/s41581-021-00405-7. Epub 2021 Mar 30.
14. Leiba M, Kedmi M, Duek A, Freidman T, Weiss M, Leiba R, Nagler A, Avigdor A. Bortezomib-cyclophosphamide-dexamethasone (VCD) versus bortezomib-thalidomide-dexamethasone (VTD) -based regimens as induction therapies in newly diagnosed transplant eligible patients with multiple myeloma: a meta-analysis. Br J Haematol. 2014 Sep; 166(5):702-10. doi: 10.1111/bjh.12946. Epub 2014 May 26.
15. Moreau P, Hulin C, Macro M, Caillot D, Chaleteix C, Roussel M, Garderet L, Royer B, Brechignac S, Tiab M, Puyade M, Escoffre M, Stoppa AM, Facon T, Pegourie B, Chaoui D, Jaccard A, Slama B, Marit G, Laribi K,

Godmer P, Luycx O, Eisenmann JC, Allangba O, Dib M, Araujo C, Fontan J, Belhadj K, Wetterwald M, Dorvaux V, Fermand JP, Rodon P, Kolb B, Glaisner S, Malfuson JV, Lenain P, Biron L, Planche L, Caillon H, Avet-Loiseau H, Dejoie T, Attal M. VTD is superior to VCD prior to intensive therapy in multiple myeloma: results of the prospective IFM2013-04 trial. Blood. 2016 May 26; 127(21):2569-74. doi: 10.1182/blood-2016-01-693580. Epub 2016 Mar 21.

16. Kyle RA, Rajkumar SV. Multiple myeloma. N Engl J Med. 2004 Oct 28; 351(18):1860-73. doi: 10.1056/NEJMra041875. Erratum in: N Engl J Med. 2005 Mar 17; 352(11):1163.
17. Al Hamed R, Bazarbachi AH, Malard F, Harousseau JL, Mohty M. Current status of autologous stem cell transplantation for multiple myeloma. Blood Cancer J. 2019 Apr 8; 9(4):44. doi: 10.1038/s41408-019-0205-9.
18. Bhatt P, Kloock C, Comenzo R. Relapsed/Refractory Multiple Myeloma: A Review of Available Therapies and Clinical Scenarios Encountered in Myeloma Relapse. Curr Oncol. 2023 Feb 15; 30(2):2322-2347. doi: 10.3390/curroncol30020179.
19. Perazella MA. Pharmacology behind Common Drug Nephrotoxicities. Clin J Am Soc Nephrol. 2018 Dec 7; 13(12):1897-1908. doi: 10.2215/CJN.00150118. Epub 2018 Apr 5.
20. Dimopoulos MA, Kastritis E, Rosinol L, Bladé J, Ludwig H. Pathogenesis and treatment of renal failure in multiple myeloma. Leukemia. 2008 Aug; 22(8):1485-93. doi: 10.1038/leu. 2008.131. Epub 2008 Jun 5.
21. Kyle RA, Gertz MA, Witzig TE, Lust JA, Lacy MQ, Dispenzieri A, Fonseca R, Rajkumar SV, Offord JR, Larson DR, Plevak ME, Therneau TM, Greipp PR. Review of 1027 patients with newly diagnosed multiple myeloma. Mayo Clin Proc. 2003 Jan; 78(1):21-33. doi: 10.4065/78.1.21.
22. Balwani MR, Gumber MR, Shah PR, Kute VB, Patel HV, Engineer DP, Gera DN, Godhani U, Gautam RS, Trivedi HL. Clinical profile of kidney involvement preceding diagnosis of multiple myeloma; a single center experience. J Nephropharmacol. 2015 Mar 29; 5(2):98-101.
23. De Martinis M, Allegra A, Sirufo MM, Tonacci A, Pioggia G, Raggiunti M, Ginaldi L, Gangemi S. Vitamin D Deficiency, Osteoporosis and Effect on Autoimmune Diseases and Hematopoiesis: A Review. Int J Mol Sci. 2021 Aug 17; 22(16):8855. doi: 10.3390/ijms2216 8855.
24. Leung N, Rajkumar SV. Multiple myeloma with acute light chain cast nephropathy. Blood Cancer J. 2023 Mar 29; 13(1):46. doi: 10.1038/ s41408-023-00806-w.