ORIGINAL ARTICLE

CONTENTS

Clinical and laboratory correlates of kidney function in multiple myeloma patients

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ABSTRACT

Aim: To investigate the relationships of kidney function with clinical and laboratory parameters in multiple myeloma (MM) patients**.**

Materials and Methods: A cross-sectional study involved 105 MM patients. Data included clinical manifestations and standard laboratory parameters. Kidney function was assessed via estimated glomerular filtration rate (eGFR), serum creatinine, urea, uric acid (UA), calcium (Ca), and albumin-to-creatinine ratio (ACR). The markers of MM activity and burden included M-protein, beta-2 microglobulin (β2m), albumin, hemoglobin (Hb), lactate dehydrogenase (LDH) and platelets (PLT). Rank biserial correlation assessed associations between symptoms and laboratory parameters. Rank-based canonical correlation analysis (RCCA) explored the multivariate relationship between six kidney function indicators and six MM-related markers.

Results: Common laboratory abnormalities included elevated β2m (90,5 %) and anemia (indicated by low Hb in 52,4 % of patients). Frequent symptoms included bone pain (71,4 %) and weakness (68,6 %). Symptoms like weakness/breathlessness correlated significantly with (β2m, M-protein) and renal impairment (creatinine, ACR, eGFR). RCCA identified one significant canonical correlation (R1=0,497; p=0,013), linking impaired renal function (characterized by low eGFR, high ACR, creatinine and urea) with a myeloma profile indicative of disease activity and burden (high β2m, low Hb, low albumin, and high M-protein).

Conclusions: The study confirms a significant multivariate association between a profile of impaired renal function and markers reflecting MM activity, hematopoietic suppression and systemic burden. These findings underscore the multifactorial nature of MM-related kidney injury and highlight the clinical utility of monitoring key laboratory markers (including eGFR, ACR, creatinine, β2m, Hb and albumin) alongside clinical evaluation for comprehensive assessment and management of MM patients.

KEY WORDS: multiple myeloma, chronic kidney disease, anemia

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INTRODUCTION

Multiple myeloma (MM) is a malignant hematological disease characterized by the proliferation of plasma cells in the bone marrow and excessive production of monoclonal immunoglobulin – the M-protein [1]. MM is a multi-stage process that begins with the formation of a plasma cell clone, which subsequently undergoes malignant transformation, leading to the development of plasma cell myeloma – the final stage of the disease. The disease manifests as anemia, recurrent infections, osteolytic bone lesions, and hypercalcemia. One of the most frequent complications of this disease is kidney damage, which occurs in 20-50% of patients at initial presentation, and 10-13% of patients require dialysis at the time of diagnosis.

This significantly increases morbidity and mortality and limits treatment strategies [2, 3]. MM is associated with the highest rate of kidney damage among all cancer [4]. The mechanism of kidney damage in MM is multifactorial; however, the most common basis of "myeloma kidney" is the excessive excretion of free light chains that deposit in the distal and proximal tubules of nephrons. Another cause that leads to impaired renal function is hypercalcemia and hypercalciuria with subsequent hypovolemia, which leads to prerenal renal failure. Hyperuricemia and tumor lysis syndrome, amyloidosis, and microthrombotic lesions are also significant. In addition, nephrotoxic cytostatic drugs used in modern chemotherapy regimens, radiocontrast agents, and nonsteroidal anti-inflammatory drugs can lead to kidney dysfunction [5]. Additional risk factors for chronic kidney disease (CKD) in MM are the combined occurrences of hypertension and type 2 diabetes mellitus.

Despite the fact that this topic is well researched, it has not lost its relevance due to advances in understanding the pathogenesis of MM and CKD, as well as the development of new treatments for both nosologies. And the fact that CKD is one of the main causes of death in patients with MM further justifies the importance of timely intervention.

Table 1. Baseline characteristics of the study cohort (N=105)

Indicators	Median [Q1; Q3]	95 % CI
Body mass index (kg/m^2)	27,3 [24,5; 30,2]	[26,1; 28,3]
Systolic blood pressure (SBP, mm Hg)	135,0 [120,0; 145,0]	[130,0; 140,0]
Diastolic blood pressure (DBP, mm Hg)	90,0 [80,0; 95,0]	[85,0; 90,0]
Leukocytes (WBC, ×10^9/L)	4,9 [3,6; 6,4]	[4,4; 5,4]
Erythrocytes (RBC, ×10^{12}/L)	3,9 [3,6; 4,2]	[3,8; 4,0]
Hemoglobin (Hb, g/L)	126,0 [114,0; 135,0]	[121,0; 129,0]
Platelets (PLT, ×10^9/L)	196,0 [146,0; 241,0]	[177,0; 216,0]
Erythrocyte sedimentation rate (ESR, mm/h)	24,0 [12,0; 37,0]	[19,0; 28,0]
ALP (U/L)	74,0 [49,8; 92,8]	[61,6; 80,3]
ALT (U/L)	20,5 [15,5; 30,0]	[17,8; 24,3]
AST (U/L)	19,9 [16,0; 23,6]	[18,0; 20,9]
GGT (U/L)	35,0 [25,0; 44,0]	[32,0; 37,0]
Glucose (mmol/L)	5,5 [5,0; 6,0]	[5,3; 5,7]
Total protein (TP, g/L)	69,5 [63,8; 73,0]	[67,8; 70,7]
UA (μmol/L)	278,7 [216,7; 345,2]	[246,0; 308,3]
Ca (mmol/L)	2,36 [2,27; 2,48]	[2,29; 2,37]
Urea (mmol/L)	5,4 [4,3; 7,1]	[4,9; 5,9]
Creatinine (μmol/L)	80,1 [61,0; 95,4]	[73,1; 83,9]
eGFR (CKD-EPI, mL/min/1,73m^2)	85,0 [61,0; 101,0]	[77,0; 92,0]
β2m (mg/L)	3,7 [2,2; 6,1]	[3,0; 4,9]
Fibrinogen (g/L)	3,0 [2,1; 3,8]	[2,4; 3,2]
D-dimer (ng/mL)	320,0 [220,0; 580,0]	[250,0; 440,0]
Albumin (g/L)	40,8 [36,4; 43,8]	[38,6; 42,1]
M-protein (g/L)*	8,82 [4,9; 23,7]	[7,35; 18,7]
ACR (mg/g)	30,1 [5,6; 45,4]	[11,4; 34,1]
LDH	285,1 [246,0; 345,5]	[269; 310]
CRP (C-reactive protein)	6 [6; 6]	[6; 6]

Note: * — M-protein was detected in 35 out of 105 patients (33,3 % [95 % CI 24,6-43,3 %])

AIM

The aim of the study was to investigate the relationships of kidney function with clinical and laboratory parameters in MM patients.

MATERIALS AND METHODS

The study included 105 patients with MM who were undergoing treatment or observation at the State Institution "Institute of Pathology and Cell Therapy of the National Academy of Medical Sciences of Ukraine" (Lviv, Ukraine). The diagnosis of MM was made according to NCCN Clinical Practice Guidelines in Oncology, Version 2.2024 [6].

Inclusion criteria were: documented MM, age between 18 and 85 years, patient consent to participate in the study, and ability to cooperate adequately during the study process. Exclusion criteria were: patient refusal to participate, age <18 years, evidence of acute infectious processes of any etiology, pregnancy and lactation, decompensated heart failure and mental disorders.

The study was conducted in accordance with the Helsinki Declaration, the Convention for the Protection of Human Rights and Biomedicine, and the legislation of Ukraine, and was approved by the Ethics Committee of Scientific Research of the Danylo Halytskyi Lviv National Medical University: Protocol No. 1, dated January 23, 2023. All patients signed an informed consent form prior to the study.

The mean age of the subjects was 57,2±8,80 years (mean ± standard deviation [SD]). The cohort included 50 (47,6% [95% CI 38,3–57,1%]) women and 55 (52,4% [95% CI 42,9–61,7%]) men.

All patients underwent a thorough collection of complaints, medical history, and life history, as well as general clinical and laboratory examinations. These examinations included: a complete blood count, biochemical

Table 2. Association of clinical manifestations with laboratory and demographic parameters in MM patients (rank biserial correlation coefficient [r]; N=105)

Parameter	Weakness	Bone pain	Loss of body weight	Breath-lessness	Neuropa-thy	Infections	Diarrhea	Constipa-tion	Dyspep-sia
SBP	0,25*	0,15	0,40*	0,10	−0,04	−0,23	−0,09	−0,08	−0,29**
DBP	0,23*	0,16	0,06	0,09	0,04	−0,20	0,02	−0,18	−0,19
WBC	0,18	−0,02	0,37*	−0,04	−0,05	−0,13	0,18	−0,19	0,32**
RBC	−0,50***	−0,37**	−0,50**	−0,75***	0,13	0,05	−0,20	0,03	−0,02
Hb	−0,62***	−0,38**	−0,47**	−0,90***	0,16	0,05	−0,25*	−0,03	−0,13
PLT	−0,06	−0,22	−0,05	−0,43***	0,19	0,20	−0,00	−0,24	0,11
ESR	0,43***	0,36**	0,36*	0,44***	−0,09	−0,05	0,14	0,05	0,14
ALP	0,12	0,25*	0,61***	0,17	−0,21	−0,30*	0,12	−0,08	−0,13
ALT	−0,10	−0,19	0,03	−0,03	−0,23*	−0,24*	−0,10	−0,18	−0,07
AST	0,05	0,14	0,13	0,23	−0,24*	−0,27*	0,11	−0,21	−0,08
GGT	−0,09	−0,14	0,20	0,11	−0,05	−0,01	0,03	−0,11	−0,11
Glucose	−0,05	−0,06	0,04	−0,08	−0,03	−0,05	0,13	0,37*	−0,09
TP	0,03	0,11	0,32	0,04	−0,02	−0,21	−0,11	0,28	0,02
UA	0,16	0,05	0,31	0,27*	−0,03	−0,24*	0,16	−0,02	0,16
Urea	0,10	0,09	0,33	0,21	−0,26*	−0,15	0,07	−0,05	0,19
Creatinine	0,26*	0,06	0,31	0,39**	−0,13	−0,33**	0,08	0,15	0,17
eGFR	−0,30*	−0,15	−0,40*	−0,46***	0,19	0,31**	−0,12	−0,01	−0,22
β2m	0,39***	0,37**	0,43*	0,42***	−0,11	−0,25*	0,20	−0,07	0,03
Fibrinogen	0,16	0,09	0,10	−0,02	0,13	0,14	−0,09	0,04	−0,18
D−dimer	0,04	0,17	0,12	0,20	−0,05	−0,10	−0,09	0,03	−0,08
Albumin	0,00	−0,08	−0,10	−0,17	0,11	−0,14	−0,01	−0,14	−0,02
M−protein	0,44***	0,31**	0,47**	0,31**	−0,07	−0,31**	−0,11	−0,05	−0,01
ACR	0,29**	0,18	0,26	0,29*	−0,17	−0,29*	0,16	−0,05	0,33**

Note: * − $p < 0,05$; ** − $p < 0,01$; *** − $p < 0,001$

blood tests (alanine aminotransferase [ALT], aspartate aminotransferase [AST], alkaline phosphatase [ALP], gamma-glutamyl transferase [GGT], lactate dehydrogenase [LDH], creatinine, urea, uric acid [UA], glucose, calcium [Ca], beta-2 microglobulin [β2m]), coagulation profile, blood protein electrophoresis, determination of immunoglobulin levels, and the determination of the albumin/creatinine ratio (ACR) in urine.

Hematological and biochemical blood tests, and coagulation profiles were performed using standard methods. Serum protein electrophoresis was performed on a Hellabio-Vitatron electrophoretic system (Greece). The assessment of albumin and creatinine excretion in urine was conducted using test strips from MICROLABBU-PLAN (Czech Republic). To assess renal function, the estimated glomerular filtration rate (eGFR) was calculated using an online calculator, employing the CKD-EPI formula (2021 modification) according to the KDIGO 2024 recommendations [7]. Baseline characteristics are presented in Table 1.

All statistical analyses were performed using R software, version 4.4.2.

Descriptive statistics were calculated for baseline patient characteristics. Continuous variables were assessed for normality using the Shapiro-Wilk test. Due to deviations from normality for most variables, continuous data were summarized and presented as median (1st Quartile [Q1]; 3rd Quartile [Q3]). The 95% confidence interval (CI) for the median was also calculated. Categorical variables, including the presence or absence of clinical manifestations, were described using absolute (n) and relative (%) frequencies. The 95 % CI for these proportions was computed using the Wilson score interval method.

Rank biserial correlation coefficient (r) was calculated to assess the pairwise association between clinical manifestations and baseline laboratory, demographic and disease markers.

To investigate the multivariate linear relationships between groups of variables, rank-based canonical correlation analysis (RCCA) was performed. One set comprised six kidney function indicators (eGFR, creatinine, ACR, urea, UA, and Ca). The second set comprised six indicators related to myeloma and its systemic effects

(M-protein, β2m, albumin, Hb, LDH, and PLT). The RCCA was conducted on patients with complete data for all variables included in the analysis. The overall significance of the model and the sequential significance of the canonical correlations were evaluated using Wilk's Lambda statistic with Rao's F-approximation. Canonical correlations and structure correlations (loadings) between the original variables and the derived canonical variates were examined for interpretation. For all statistical tests, a p-value < 0,05 was considered statistically significant.

RESULTS

Analysis of laboratory parameters in the studied cohort (N=105) revealed frequent abnormalities characteristic of MM. Among elevated parameters, increased β2m was observed in 95 (90,5% [95% CI 83,4–94,7 %]) patients, followed by a high ESR in 76 (72,4% [95% CI 63,2–80,0%]) patients, and elevated total protein levels in 18 (17,1% [95% CI 11,1–25,5%]) patients. Renal impairment (indicated by elevated creatinine) was detected in 21 (20,0% [95% CI 13,5–28,6%]) patients, and increased CRP was detected in 22 (21,0% [95% CI 14,3–29,7%]) patients. Hypercalcemia was noted in 15 (14,2% [95% CI 7,6–26,2%]) patients, while elevated UA in 11 (10,5% [95% CI 6,1–18,9%]) patients and elevated LDH in 9 (8,6%[95% CI 4,6–16,2%]) patients were less frequent.

Concurrently, the analysis showed significant rates of decreased parameters. Anemia was highly prevalent, with decreased Hb found in 55 (52,4% [95% CI 42,8–61,7%]) patients and reduced RBC observed in 52 (49,5% [95% CI 40,1–59,0%]) patients. Other cytopenias included thrombocytopenia, occurring in 44 (41,9% [95% CI 32,9–51,5%]) patients, and leukopenia in 36 (34,3% [95% CI 25,9–43,8%]) patients. Furthermore, prognostically significant hypoalbuminemia was detected in 35 (33,3% [95% CI 25,0–42,8%]) patients. Collectively, these findings highlight the typical patterns of paraproteinemia, hematopoietic suppression, renal involvement, and altered protein metabolism associated with MM in this patient group.

The frequency of major clinical manifestations of MM was evaluated in the cohort (N=105). The most common findings were bone pain, which was present in 75 (71,4% [95% CI 62,2–79,2%]) patients, and weakness, observed in 72 (68,6% [95% CI 59,2–76,7%] patients. Neuropathy occurred in 55 (52,4% [95% CI 42,9–61,7%]) patients. Dyspepsia was reported by 47 (44,8% [95% CI 35,6–54,3%]) patients. Frequent infections affected 42 (40,0% [95% CI 31,1–49,6%]) patients, followed by loss of body weight in 39 (37,1% [95% CI 28,5–46,7%]) patients, breathlessness in 32 (30,5% [95% CI 22,5–39,8%]) patients, diarrhea in 28 (26,7% [95% CI 19,1–35,8%]) patients, and constipation in 13 (12,4% [95% CI 7,4–20,0%]) patients.

Analysis using rank biserial correlation revealed significant associations between clinical manifestations and laboratory parameters in the studied cohort (Table 2). Notably, symptoms indicative of significant disease burden and systemic impact, such as weakness and particularly breathlessness, demonstrated strong positive correlations with markers of inflammation and tumor load (ESR, β2m, M-protein) and strong negative correlations with markers of anemia (Hb, RBC). The association between breathlessness and low Hb was exceptionally strong (r= –0,90; p<0,001). Furthermore, weakness, breathlessness, and loss of body weight were significantly associated with laboratory indicators of impaired renal function, including elevated creatinine and ACR, and reduced eGFR.

Regarding specific symptoms, bone pain showed significant positive correlations with markers potentially reflecting bone turnover and disease activity (ALP, ESR, β2m, M-protein). Similarly, loss of body weight correlated positively with ALP and other markers of disease activity/inflammation (ESR, β2m, M-protein), as well as with higher SBP and WBC.

In contrast, correlations for neuropathy were less pronounced, exhibiting only weak negative associations with ALT, AST, Urea, and GFR. Findings concerning infections were somewhat counter-intuitive, showing negative correlations with several disease markers (M-protein, β2m, creatinine, ACR) while positively correlating with eGFR, suggesting a need for further investigation. Gastrointestinal symptoms generally exhibited fewer significant associations; constipation was weakly linked to higher glucose levels, whereas dyspepsia showed moderate correlations with higher WBC and ACR, and lower SBP. Gastrointestinal symptoms and manifestations of neuropathy are most often side effects of chemotherapeutic agents, rather than direct signs of MM or kidney damage.

Further analysis was conducted to comprehensively evaluate the relationships between MM markers and impaired renal function using a non-parametric approach. RCCA was performed on the ranks of the six kidney function indicators (eGFR, creatinine, ACR, urea, UA, Ca) and six indicators related to myeloma and its systemic effects (M-protein, β2m, albumin, Hb, LDH, PLT). The analysis yielded six pairs of canonical variates.

The overall relationship between the two sets of ranked variables was statistically significant according to the Wilk's Lambda criterion (Wilk's Lambda = 0,554; F (36; 411,2) = 1,642; p = 0,013). Further testing

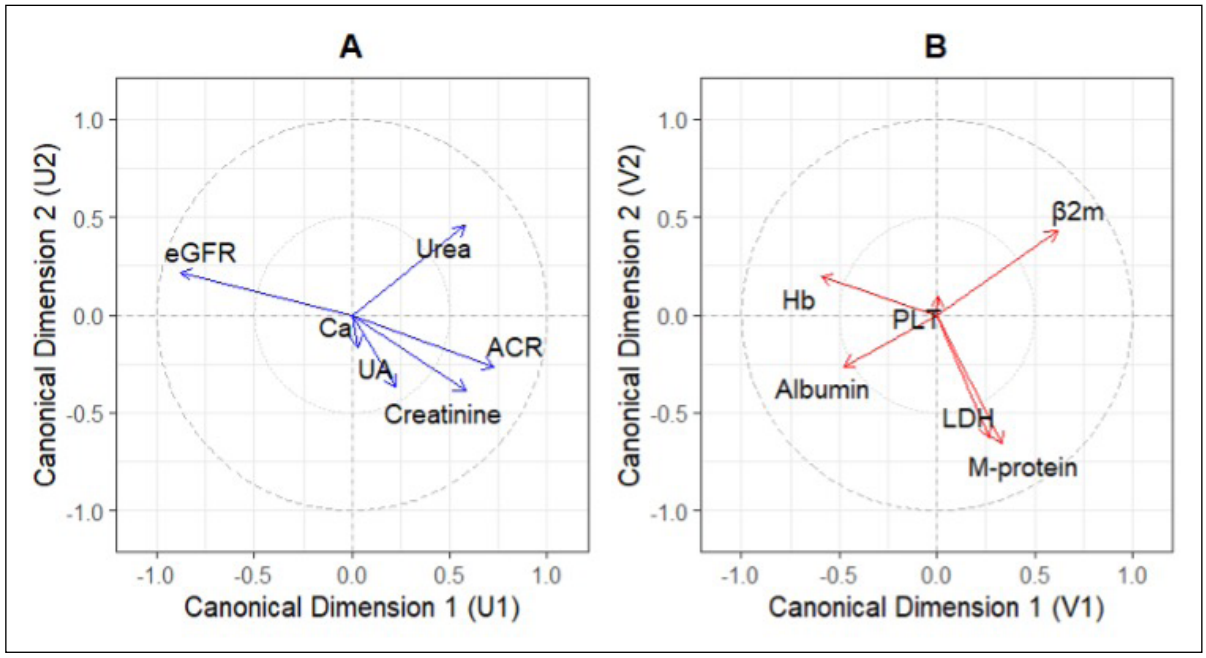

Fig. 1. Structure correlations from RCCA: kidney function indicators (A) and indicators related to myeloma and its systemic effects (B)

revealed that only the first canonical correlation (R1 = 0,497) was statistically significant. Subsequent canonical correlations were not statistically significant (R2 = 0,383, p = 0,228; R3 = 0,262, p = 0,566; R4-R6 were also non-significant).

Interpretation focused on the structure correlations (canonical loadings) for the first significant canonical dimension. The first canonical variate for the ranked kidney set (U1) was primarily characterized by a strong negative loading for eGFR (–0,88) and strong positive loadings for ACR (0,73), creatinine (0,58), and urea (0,58). UA exhibited a weak positive loading (0,22), while Ca showed a negligible loading (0,03) on U1. The corresponding canonical variate for the ranked myeloma set (V1) demonstrated strong positive loading for β2m (0,62) and strong negative loading for Hb (–0,59). Moderate negative loading was observed for albumin (–0,48) and moderate positive loading for M-protein (0,34). LDH displayed weak positive loading (0,27), whereas PLT had a negligible loading (0,01) on V1. (Fig. 1).

DISCUSSION

The most frequent symptoms in patients with MM are asthenia and bone pain, anemia, susceptibility to infections, hypercalcemia, and nephrotic syndrome, which is consistent with our results [8].

Most of the correlations we obtained in our study are weak or moderate, indicating the complex and multifactorial nature of MM. The presence of statistically significant correlations may indicate potential relationships between these parameters, which may be useful for understanding the pathophysiology of the disease and the clinical evaluation of patients.

It is traditionally believed that the presence of CKD at the time of MM diagnosis is a prognostically unfavourable factor that affects overall survival, treatment response, the risk of complications, and the quality of life of patients [9, 10].

The multifactorial nature of the relationship between MM and CKD, which includes numerous biomarkers of both nosologies, requires the use of complex analytical approaches. RCCA is one such multivariate method specifically designed to assess nonparametric relationships between two sets of variables.

The result of RCCA statistically compacts a set of known risk factors, such as low eGFR, low Hb, low albumin, high urea, high creatinine, and elevated ACR, into a single significant correlation dimension.

This reinforces the idea of the interconnectedness of CKD, anemia, increased β2m and hypoalbuminemia as a central characteristic of progressive MM, which is consistent with the results of regression models that identify these markers as prognostic factors.

RCCA does not identify independent predictors but captures a clinically significant and prognostically relevant pattern of target organ damage associated with MM. A similar study was conducted by W. Wei et al. [11] where they retrospectively investigated the relationship between clinicopathological characteristics and the frequency of kidney damage, response, and survival of patients with MM.

In particular, they found that secreted monoclonal immunoglobulin type IgG, free light chain κ/λ in serum,

elevated serum Ca, elevated urea, elevated UA, and ISS stage III were closely associated with kidney damage. Elevated LDH and CKD stage (G4-G5) were independent adverse factors influencing the overall survival of patients with MM and kidney damage. In addition, this study provided a model for predicting treatment response and kidney damage using 5 clinical signs, including Ca, MM stage, pre-treatment creatinine level, age, and sex [11].

The use of new biomarkers of acute kidney injury (NGAL, cystatin C, TIMP-2, IGFBP7) for earlier detection of kidney damage in patients with MM is being actively investigated. Kidney biopsy is becoming increasingly important for determining the type of kidney damage and prognosis, especially in cases of atypical clinical presentation or unexplained cause of kidney damage [12].

The shift in the diagnostic and research approach from a single marker to a cluster analysis of biomarkers, which will reflect the complex relationships in pathogenetic links, will allow for better prediction of the course and response to treatment, and thus improve therapeutic strategies.

STUDY LIMITATIONS

Despite its findings, this study has several limitations. Firstly, the cross-sectional design restricts the ability to establish causality between MM markers and renal function changes over time. Secondly, being a single-center study with 105 participants, the generalizability of results to broader MM populations may be limited. Thirdly, the analysis relied on standard laboratory markers, excluding newer kidney injury biomarkers, kidney biopsy data, detailed treatment histories, and molecular characteristics, which could provide deeper insights.

Future directions. Prospective longitudinal studies are needed to track the temporal relationship between MM progression, treatment, and CKD development, clarifying causal links. Larger, multi-center studies would enhance statistical power and generalizability. Incorporating novel kidney biomarkers (e.g., NGAL, Cystatin C) and systematically correlating findings with kidney biopsy results (when available) could improve early detection and pathogenetic understanding. Investigating the specific impact of different MM therapies on renal outcomes is crucial. Finally, employing advanced statistical methods, including machine learning, may uncover complex interactions and improve predictive modeling for CKD in MM patients.

CONCLUSIONS

Multivariate analysis using rank-based canonical correlation identified a statistically significant relationship between a profile of impaired renal function (primarily characterized by low eGFR, high ACR, high creatinine, and high urea) and a profile indicative of myeloma activity and burden (including high β2m, low Hb, low albumin, and high M-protein). This finding statistically reinforces the strong interplay between key disease processes, particularly myeloma activity, hematopoietic suppression, and renal involvement.

These results emphasize the multifactorial pathophysiology of MM and underscore the importance of integrating both clinical symptoms and a range of laboratory markers for comprehensive patient assessment. Monitoring parameters related to renal function (eGFR, ACR, creatinine), anemia, and disease burden (β2m, M-protein), alongside clinical evaluation, is essential for understanding disease status and may help inform management strategies for patients with MM.

REFERENCES

1. Rajkumar SV. Multiple myeloma: 2020 update on diagnosis, risk-stratification and management. Am J Hematol. 2020;95(5):548–567. doi:10.1002/ajh.25791. DOI
2. Kundu S, Jha SB, Rivera AP et al. Multiple myeloma and renal failure: mechanisms, diagnosis, and management. Cureus. 2022;14(2):e22585. doi:10.7759/cureus.22585. DOI
3. Zhang Y, Pan J, Kang H et al. Prognosis of concurrent renal impairment at diagnosis of multiple myeloma: a systematic review. Ann Med. 2024;56(1):2380301. doi:10.1080/07853890.2024.2380301. DOI
4. Bridoux F, Leung N, Nasr SH et al. Kidney disease in multiple myeloma. Presse Med. 2025;54(1):104264. doi:10.1016/j.lpm.2024.104264. DOI
5. Menè P, Stoppacciaro A, Lai S, Festuccia F. Light chain cast nephropathy in multiple myeloma: prevalence, impact and management challenges. Int J Nephrol Renovasc Dis. 2022;15:173–183. doi:10.2147/IJNRD.S280179. DOI
6. Kumar SK, Callander NS, Adekola K et al. Multiple myeloma, version 2.2024, NCCN clinical practice guidelines in oncology. J Natl Compr Canc Netw. 2023;21(12):1281–1301. doi: 10.6004/jnccn.2023.0061. DOI
7. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int. 2024;105(4):S117–S314. doi: 10.1016/j.kint.2023.10.018. DOI
8. Ștefan G, Cinca S, Chiriac C et al. Multiple myeloma and kidney impairment at diagnosis: a nephrological perspective from an Eastern European country. Medicina (Kaunas). 2023;59(7):1326. doi:10.3390/medicina59071326. DOI

9. Khan R, Apewokin S, Grazziutti M et al. Renal insufficiency retains adverse prognostic implications despite renal function improvement following Total Therapy for newly diagnosed multiple myeloma. Leukemia. 2015;29(5):1195–1201. doi:10.1038/leu.2015.15. DOI
10. Sharma R, Jain A, Jandial A et al. Lack of renal recovery predicts poor survival in patients of multiple myeloma with renal impairment. Clin Lymphoma Myeloma Leuk. 2022;22(8):626–634. doi: 10.1016/j.clml.2022.04.013. DOI
11. Wei W, Shi H, Chen H et al. Clinicopathologic predictors of renal response and survival in newly diagnosed multiple myeloma with renal injury: a retrospective study. Clin Exp Med. 2025;25(1):48. doi:10.1007/s10238-025-01571-9. DOI
12. Woziwodzka K, Vesole DH, Małyszko J et al. New markers of renal failure in multiple myeloma and monoclonal gammopathies. J Clin Med. 2020;9(6):1652. doi: 10.3390/jcm9061652. DOI

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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