

## ORIGINAL RESEARCH

# Clinical and histopathological profile of patients with multiple myeloma and renal involvement

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## Abstract

**Background:** Multiple Myeloma (MM) is a malignant disorder of plasma cell characterised by the clonal expansion of aberrant plasma cells within the bone marrow. This leads to the excessive production of monoclonal immunoglobulins (M-protein) and associated with organ dysfunction. Renal disease is a frequent and a potentially significant complication of multiple myeloma. The range of renal lesions observed in the patients with multiple myeloma is diverse. The aim of present study is to find the clinical and histopathological profile of patients with multiple myeloma and renal involvement and their clinico-pathological correlation. **Methods:** This was an observational hospital based study where 33 patients with multiple myeloma as well as renal involvement who agreed for renal biopsy were included. Demographic data, mode of presentation and need for dialysis were noted. Appropriate laboratory tests and radiological survey were done. **Results:** The median age of the patients in our study was 56 years. Fatigue was the most common clinical feature. The indications for renal biopsy were acute kidney injury (AKI), chronic/progressive reduction in glomerular filtration rate (GFR) with normal sized kidneys and nephrotic syndrome. Histopathological findings in the patients with acute kidney injury included myeloma cast nephropathy, acute tubulointerstitial nephropathy, acute tubular necrosis and IgA (Immunoglobulin A) nephropathy. In cases with chronic decline in GFR, observed lesions were myeloma cast nephropathy, monoclonal immunoglobulin deposition disease, membranoproliferative glomerulonephritis, chronic interstitial nephropathy and diabetic nephropathy. For patients presenting with nephrotic syndrome, amyloidosis and monoclonal immunoglobulin deposition disease were predominant. **Conclusions:** Renal involvement is considered as one of the main feature of multiple myeloma and is often the initial manifestation of the disease. Renal biopsy is crucial for establishing a specific diagnosis and providing valuable prognostic and therapeutic insights.

**Keywords:** Acute kidney injury; Amyloidosis; Chronic kidney disease; Multiple myeloma; Myeloma cast nephropathy

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## Introduction

Multiple Myeloma (MM) is a malignant plasma cell disorder characterised by the clonal proliferation of aberrant plasma cells in the bone marrow that leads to excessive production of monoclonal immunoglobulins (M-protein) and associated with organ dysfunction. Kidney disease is a common and potentially serious complication of multiple myeloma, affecting approximately 20% to 25% of patients [1] and in up to 50% patients [2] during the course of their disease. The range of kidney impairment in multiple myeloma extends from mild asymptomatic dysfunction to severe acute kidney injury requiring dialysis, or chronic kidney disease progressing to ESRD (End Stage Renal Disease) [2, 3].

Renal insufficiency in patients with MM is due to a significant degree to the pathologic effect of monoclonal light chains on renal tissue, with predominant renal tubular and, in smaller

degree, glomerular involvement. Hypercalcemia plays a lesser role as a cause of renal insufficiency. Contributing causes include dehydration, nephrotoxic substances (e.g., antibiotics and non-steroidal anti-inflammatory drugs) and contrast media, which can compound renal injury but are seldom the sole causative agent. Monoclonal light chains inflict injury on all parts of the nephron, such as glomeruli, tubules, interstitium and vasculature, by various pathogenic mechanisms and result in a wide range of histological and clinical presentations. The most frequent kidney injury associated with multiple myeloma (MM) is myeloma cast nephropathy (MCN). Amyloidosis, monoclonal immunoglobulin deposition disease (MIDD), and acquired adult Fanconi syndrome may occur sporadically in the same individual patient [4]. Interestingly, no consistent correlation is observed between clinical presentation and the underlying renal pathology, and renal outcome can't be forecasted on the basis of clinical presentation [5].

Kaustubh Bora's study revealed that the northeastern zone accounted for only 9.6% of India's total MM burden [6]. However, there is a paucity of data from the North Eastern Part of India regarding the presentation of MM patients with renal involvement. So this study was undertaken to see the clinical presentation, histopathological profile and clinico-pathological correlation of this subset of MM patients with renal involvement.

## Methods

Present study was a single center observational study conducted in the Department of Nephrology and Department of Hematology, Gauhati Medical College and Hospital, Assam from 01 July 2016 to 30 June 2017. 33 patients of MM with renal impairment who were willing for renal biopsy were enrolled in the study.

Demographic data, mode of presentation and need for dialysis were noted. Routine blood and urine tests, urine for Bence Jones protein, serum B2 microglobulin levels and radiological survey for lytic bone lesions were done. Serum protein electrophoresis (SPEP) was done by the agarose gel method (agarose gel electrophoresis). The monoclonal protein type was determined by immunoelectrophoresis. Bone marrow aspiration and its biopsy was done in all patients. Patients were classified based on the International Staging System (ISS) for MM [7].

Multiple myeloma was diagnosed by the Revised International Myeloma Working Group diagnostic criteria for multiple myeloma [8].

Renal biopsies were performed as a routine clinical practice after obtaining informed consent from the patients. Common indications included acute kidney injury, chronic progressive decline in glomerular filtration rate with normal sized kidneys and nephrotic syndrome (NS). Renal biopsy specimens were processed using standard techniques, including immunofluorescence and light microscopy. For light microscopy, all samples were counter-stained with hematoxylin and eosin, Masson trichrome, periodic acid—Schiff and Jones methenamine silver. Immunofluorescence was performed on 3  $\mu$ m cryostat sections, which were further stained with specific polyclonal antibodies against IgG, IgM, IgA, C3, C1q, kappa (K) and lambda ( $\lambda$ ) light chains.

The present study proposal was approved by the Institutional Ethics Committee of Gauhati Medical College & Hospital, Guwahati, Assam.

## Statistical analysis

Statistical analysis was performed using IBM SPSS (V29, Guwahati, Assam, India) and Microsoft Excel (Version 16.97.2, Microsoft, Guwahati, Assam, India). Numerical data are presented as mean  $\pm$  standard deviation (S.D.) or median based on normality of distribution. *p* value was determined by Pearson Chi-square to analyse categorical data and Student's *t*-test and ANOVA (Analysis of Variance) was used for the continuous variables followed by Tukey *Post Hoc* tests for multiple comparison was performed. *p*-value less than 0.05 was judged as significant.

## Results and observations

33 patients of MM with renal impairment were enrolled in the study out of a total of 71 cases of newly diagnosed MM in the study period. The indications for performed renal biopsies were AKI in 57.57% patients, CKD (Chronic Kidney Disease) in 30.30% and NS in 12.12% patients.

### Clinical features

Of total 33 MM patients with renal involvement, fatigue was found to be the most common clinical feature in 87.87% patients followed by anaemia in 84.84%, weight loss in 42.42%, bone pain in 36.36%, oliguria in 36.36%, pedal oedema in 30.3% and lytic bone lesions in 24.24% of patients.

### Precipitating factors for renal failure in AKI

The most common factor responsible for AKI was dehydration in 73.7% patients. Nephrotoxic drugs and hypercalcemia were identified in 47.4% patients each. Infections precipitated AKI in 36.8% patients. 33% had more than 1 precipitating factor. No precipitating factor could be identified in 15.7% patients.

### Frequency of paraprotein associated lesions on kidney biopsy

MCN was the most frequent paraprotein associated lesion seen in 48.48% patients. MIDD (which included only Light chain deposition disease) and amyloidosis were observed in 9.09% patients each.

### Frequency of non-paraprotein associated lesions on kidney biopsy

Non-paraprotein-associated renal lesions were diverse in nature. Acute tubular necrosis (ATN) and acute interstitial nephritis (AIN) were the most common lesions found in 12.12% and 9.09% patients respectively. IgA nephropathy (IgAN), chronic interstitial nephropathy (CIN), C3 glomerulonephritis (C3G) and diabetic nephropathy (DN) were found in 3% each.

### Clinical stage (ISS) of the patients with MM and renal involvement

75.75% patients were in stage III and 24.24% patients were in stage II.

### Demographics and hematologic characteristics of 33 MM patients at the time of renal biopsy

The clinical and hematologic characteristics of all MM patients with renal involvement and those with the histopathological finding of MCN, MIDD and Amyloid is shown in Table 1. The finding of the three paraprotein associated lesions did not differ by age, sex, presence of M peak on serum electrophoresis, type of paraprotein involved, free light chain type or percentage of plasma cells in the bone marrow of patients with MM and renal involvement. Hypercalcemia tended to be more frequent in MCN and MIDD than amyloid. The median haemoglobin level was reduced in patients with MCN and MIDD compared to those with amyloidosis. B2 microglobulin levels were lower

**Table 1. Demographics and hematologic characteristics of 33 multiple myeloma patients at the time of renal biopsy.**

Characteristic	All study patients	MCN	MIDD	Amyloid	<i>p</i> -value			
					3 way comparison	MCN vs. MIDD	MCN vs. Amyloid	Amyloid vs. MIDD
No. of patients	33	16	3	3				
Male sex	22	12	2	3	0.571	0.764	0.330	0.273
Age (yr)	56	53.5	56	55	0.632	0.997	0.966	0.996
“M” peak in serum electrophoresis	15	8	1	0	0.260	0.596	0.107	0.273
Myeloma type								
IgG	21	10	1	2				
IgA	4	4	0	0	0.254	0.067	0.439	0.414
IgD	1	1	0	0				
LC	7	1	2	1				
FLC type								
λ	18 (54.5)	7 (43.7)	1 (33.3)	3 (100.0)	0.167	0.737	0.073	0.083
K	15 (45.4)	9 (56.2)	2 (66.6)	0				
Plasma cells in bone marrow (%)	35.0	44.5	34.0	27.0	0.839	0.587	0.897	0.604
Hypercalcemia	11 (33.3)	6 (37.5)	3 (100.0)	0	0.039	0.047	0.200	0.014
Median hemoglobin (mg/dL)	7.1	7.2	6.7	11.2	0.023	0.349	0.014	0.011
B2 microglobulin	8900.0	14,145.5	10,149.0	6700.0	0.131	0.223	0.152	0.047
Uric acid (mg/dL)	8.80	9.45	9.20	6.70	0.091	0.386	0.035	0.298

MCN: myeloma cast nephropathy; MIDD: monoclonal immunoglobulin deposition disease; λ: lambda; K: kappa; Ig: Immunoglobulin; LC: Light Chain; FLC: Free Light Chain. Continuous variables given in the table are as median; categorical variables, as counts with percentages or as fractions (number affected/total) with corresponding percentages. *p*-value < 0.05 are considered statistically significant.

in patients with amyloidosis compared to those with MIDD, although there was no significant difference between MCN and amyloidosis. The median uric acid level was increased in patients with MCN compared to those with amyloidosis.

#### Renal characteristics at renal biopsy in 33 MM patients

The renal characteristics of all the patients with MM and renal involvement and histopathological findings of MCN, MIDD and amyloid is shown in Table 2. Median 24 hr urinary protein was higher in amyloidosis than in MCN or MIDD. Median serum albumin levels were lower in amyloidosis than MCN. The median serum creatinine level was lower and eGFR (Estimated Glomerular Filtration Rate) level was increased in amyloidosis than MCN and MIDD. Interstitial fibrosis and tubular atrophy was more severe in MIDD and MCN than amyloidosis. The proportion of globally sclerotic glomeruli were not significantly different in MCN, amyloidosis and MIDD.

#### Treatment and outcome

Among the patients with AKI or CKD, the median follow-up was 4 months. Patients were treated with Bortezomib, Cyclophosphamide and Dexamethasone (VCd). All patients received hydration, blood transfusion and allopurinol as needed. None received therapeutic plasma exchange (TPE). Dialysis

support was needed in 15 patients, corresponding to a histological diagnosis of MCN (9), dual finding of MCN and ATN (2), DN (1), MIDD (1) and AIN (1). After the end of 4 months of follow up, 34% (10) remained dialysis dependent, 38% (11) progressed to various stages of CKD, 24% (7) achieved normal renal functions and 3% (1) were lost to follow-up.

Among the patients presenting as NS corresponding to a histological diagnosis of amyloidosis (3) and MIDD (1), all patients received VCd. There was no response to treatment during the median follow-up of 2 months.

#### Discussion

Kidney involvement in MM is observed in approximately 50% of cases at presentation [2], while our study identified it in 46.47% patients. The majority of patients were in the age group of 41–60 years and median age of the patients was 56 years in contrast to a median age of 60–70 years in western studies [2, 9]. Prakash *et al.* [10] reported that the mean age of patients with MM presenting as AKI was  $59.3 \pm 7.4$  years. Sakhuja *et al.* [11] has found a mean age of  $57.1 \pm 10.8$  years in MM patients and renal involvement was documented in 26.9%. A male predominance was seen in this study which was reflected in most of the studies on MM and renal involvement

**Table 2. Renal characteristics at renal biopsy in 33 multiple myeloma patients.**

Characteristic	All study patients	MCN	MIDD	Amyloid	p-value			
					3 way comparison	MCN vs. MIDD	MCN vs. Amyloid	Amyloid vs. MIDD
All patients	33	16	3	3				
24 hour urinary protein	6.100	4.950	5.200	19.000	0.012	0.956	0.004	0.020
Serum albumin (g/dL)	3.200	3.400	3.100	2.400	0.119	0.979	0.044	0.109
Microscopic hematuria	10 (30.3)	7 (43.7)	1 (33.3)	0	0.349	0.737	0.149	0.273
Bence Jones proteinuria	14	7	3	1	0.167	0.073	0.737	0.083
Serum creatinine (mg/dL)	5.40	7.40	8.89	0.90	0.028	0.472	0.014	0.016
eGFR (mL/min/1.73 m <sup>2</sup> )	11.28	7.13	6.40	93.12	<0.0001	0.4150	<0.0001	<0.0001
Requiring Hemodialysis	10	6	1	0	0.440	0.891	0.200	0.270
Percentage of glomeruli that are globally sclerotic	11	18	24	0	0.190	0.648	0.096	0.104
Tubular atrophy/interstitial fibrosis (%)	25.0	33.5	42.0	12.0	0.066	0.194	0.076	0.022

MCN: myeloma cast nephropathy; MIDD: monoclonal immunoglobulin deposition disease; eGFR: Estimated Glomerular Filtration Rate. Continuous variables are given as median; categorical variables, number (percentage). p-value < 0.05 are considered statistically significant.

[9–11]. In the previous study by Julie Lin *et al.* [12] on patients with MIDD, there was almost equal sex distribution with a ratio of 1.09:1.

Fatigue and anaemia were found to be the most common clinical presentation with bone pain in only 36% of patients while a North Indian study have found anaemia (85%) and bone pains (68%) to be more frequent [10]. The most common factor responsible for AKI was dehydration in 73.7% of patients followed by nephrotoxic drugs, hypercalcemia and infections. Other studies found hypercalcemia to be a more common factor for AKI than nephrotoxic drugs [10, 11].

The main indication for renal biopsy was AKI, similar to as reported by Nasr *et al.* [13]. Overall 66.7% of patients had paraprotein associated lesions while 33.3% of patients had non-paraprotein associated lesions. ATN and AIN were considered as the most common non-paraprotein associated lesions in AKI while CIN, DN and C3G were found in CKD. Dominant C3 deposits on immunofluorescence led to the diagnosis of C3G. Zand *et al.* [14] hypothesized that C3 glomerulopathy can manifest in MM due to monoclonal immunoglobulins causing dysregulation of the complement pathway. In the study by Nasr *et al.* [13], in 23% of the cases, the pathological diagnosis could not be clearly attributed to paraprotein-related injury, with nephroangiosclerosis (6%) and DN (5%) being

the most common. A kidney biopsy in MM is not always required as renal involvement is associated on the basis of clinical and laboratory findings (*e.g.*, Bence Jones proteinuria, elevated serum free light chains, *etc.*). However, some atypical features that does not fit the typical presentation of MCN may warrant a kidney biopsy for accurate diagnosis. Nephrotic syndrome may suggest Amyloid Light chain (AL) Amyloidosis or MIDD rather than MCN which is treated primarily with light chain reduction (*e.g.*, TPE, chemotherapy), while the previous two may require additional therapies. Absence of significant proteinuria may suggest alternative causes as hypertensive nephrosclerosis, DN or tubulointerstitial disease. Minimal light chain excretion raises the suspicion of non-myeloma related CKD. Other atypical features include slowly progressive CKD without AKI episodes, glomerular hematuria and presence of thrombotic microangiopathy (TMA) features which might be due to MM, Bortezomib or paraprotein related endothelial injury.

IgG  $\lambda$  was the most frequent paraprotein involved as seen by Sakhuja *et al.* [11]. Previous studies have demonstrated that patients with light chain or IgD myeloma exhibit a significantly higher incidence of renal failure and amyloidosis compared to those with IgG or IgA myeloma [15]. Nasr *et al.* [13] reported that individuals with MCN had higher likelihood to



have detectable monoclonal immunoglobulins and a higher mean percentage of monoclonal plasma cells in the bone marrow than those with amyloidosis or MIDD [13] which was demonstrated in this study. Nasr *et al.* [13] observed that hypercalcemia was more frequently seen in patients with MCN in comparison to amyloidosis and lower median hemoglobin in MCN and MIDD compared to amyloidosis similar to the trend seen in our study.

MCN predominantly presented as AKI and in some cases CKD. In this study, 48% of the patients had MCN, with 33% (11) presenting with AKI and 15% (5) with CKD. NS with preserved creatinine levels was a presenting feature of AL amyloidosis while MIDD presented either as NS or CKD. The percentage of myeloma patients requiring dialysis has been reported to range from 2 to 66% [16, 17]. The severity of cast formation has been linked to the level of renal insufficiency and its reversibility [18]. Among the various histopathological lesions, the presence of myeloma cast nephropathy and elevated levels of free light chains were highly associated with the need for renal replacement therapy [18, 19]. In this study, out of 29 multiple myeloma patients with renal impairment, 52% (15) required dialysis, of whom 73% (11) had histological evidence of MCN. 40% (6) of patients requiring dialysis were successfully withdrawn following chemotherapy and supportive care. At the end of follow-up, 73% (8 patients) of those with myeloma cast nephropathy (MCN) remained dialysis-dependent. Although the role of therapeutic plasma exchange (TPE) in MCN is debatable, present treatment guidelines with expert opinion highlight TPE usage alongside plasma cell directed therapy and supportive therapy for the rapid reduction of circulating free light chains [20–22]. Owing to financial constraints, TPE was not utilized in any patient included in this study. Montseny *et al.* [23] found that maintenance hemodialysis was needed in 39%. In the study by Prakash *et al.* [10] which included MM patients with ARF (Acute Renal Failure), dialysis support was required in 77% of cases and renal function normalized in 38.5% with dialytic support and chemotherapy. In our study among the patients presenting as AKI, 24% (7) had complete normalization of renal function. Renal function was found to be improved in 62% (18) of patients who presented as AKI or CKD. In the study by Sakhuja *et al.* [11] renal functional improvement was noted in 33% of cases. Other previous studies [24, 25] have also demonstrated a significant higher rate of renal recovery with bortezomib-based regimens in MM, highlighting the value of treating patients with renal involvement.

## Limitations

Limitations include the small sample size, short follow up of the patients and non-usage of TPE in MCN due to financial constraints.

## Conclusions

Renal biopsy should be considered in cases of plasma cell dyscrasias when early therapeutic intervention is necessary to preserve renal function. This includes preventing end stage renal disease, potentially reversing the need for hemodialysis,

or distinguishing other conditions such as IgA nephropathy, hypertensive nephropathy or proliferative glomerulonephritis which may occur independently or as part of monoclonal gammopathy of renal significance (MGRS), AL amyloidosis or MCN where systemic therapy would be warranted.

## Availability of data and materials

The data supporting the findings of this study are not publicly available due to the privacy concerns, Institutional Policy and ethical restriction.

## Author contributions

MS and AM—designed the research study; analysed the data. AM—performed the research; wrote the manuscript. JB—provided help and advice on analysing the hematological aspect of the study. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

## Ethics approval and consent to participate

Our manuscripts reporting studies involving human participants, human data, approved by Institutional Ethics Committee of Gauhati Medical College & Hospital, Guwahati, Assam, India, No. MC/ 217/2016/214, dated 09 January 2016. Patient's written informed consent was waived by Gauhati Medical College & Hospital, Guwahati Assam, India.

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## Conflict of interest

The authors declare no conflict of interest.

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