REVIEW ARTICLE

Renal involvement in multiple myeloma

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KEY WORDS

acute kidney injury, chronic kidney disease, multiple myeloma, multiple myeloma nephropathy

ABSTRACT

In this paper, the spectrum of renal involvement in the course of multiple myeloma (MM) is discussed. We describe the most important pathophysiological mechanisms underlying the development of renal complications observed in MM. In particular, we focused on the correlations between morphological changes in the kidneys and clinical signs and symptoms. Physicochemical characteristics of light chains that are synthesized in excess are critically important in the development of different types of renal involvement. It seems that patients with MM should be actively treated regardless of the type of lesions because the current methods allow to reverse renal lesions and reduce the negative effect of renal damage on prognosis in these patients.

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Introduction Multiple myeloma (MM) is a hematological disorder, which due to its multifaceted clinical manifestations attracts the attention of various medical specialists, including neurologists, nephrologists, cardiologists, orthopedists, and others. Current trends in the diagnosis and treatment of MM were reviewed in the *Polish Archives of Internal Medicine* a few years ago.[1] In the present review, we would like to focus more specifically on renal manifestations in the course of the disease. Clinical and laboratory signs of renal involvement are present in up to 75% of the patients with various stages of MM. No strict correlation exists between the clinical manifestation and underlying renal pathology, which makes it unreliable to predict renal outcome based solely on clinical symptoms. In this paper, we discuss the most important pathophysiological pathways of renal damage in the course of MM, their morphological consequences, and the clinical context. The list of renal manifestations of MM (probably not complete) is shown in TABLE 1.

Clinical manifestations of multiple myeloma Renal failure occurs in 25% to 75% of the patients with MM. The discrepancies in reporting the prevalence of kidney involvement is caused by many factors, including use of different definitions of chronic kidney disease (CKD), use of different methods to measure renal function (creatinine clearance, serum creatinine concentration, anthropometric formulas), difficulties in distinguishing between CKD, rapidly progressing renal failure, acute kidney injury (AKI), acute-on-chronic renal failure, etc. These different forms of renal involvement are all described simply as "renal insufficiency" in hematological literature.

It is well known that any impairment of renal function significantly and adversely affects the prognosis in MM. Traditionally, i.e., before the era of unlimited access to dialysis, CKD was considered the second most important cause of death in this disease, after infection.[2] According to the recent American and European registry reports, between 0.9% and 1.5% of the patients initiating long-term dialysis suffer from MM; paradoxically, this may reflect therapeutic success – patients in whom renal function has not recovered still survive long enough to be chronically dialyzed.[3,4] Successfully treated MM can result in complete recovery of renal function. In these cases, an unfavorable effect of CKD on prognosis can be fully reversed. It is estimated that in up to 60% of the patients with MM and CKD or AKI, hematological treatment may reverse kidney damage or at least significantly improve renal function; recent data indicate that fast and early reduction in plasma light chains (LCs) is critical for successful long-term renal outcome.[5,6]

Proteinuria is another common manifestation of renal involvement in MM; it is observed in up to 80% of the cases with renal manifestations, and

TABLE 1 Spectrum of renal lesions observed in the course of multiple myeloma[11-14,19]

frequent (at least in 10% of all cases with renal involvement)
myeloma cast nephropathy (myeloma kidney)
amyloidosis
LCDD
acute and chronic interstitial nephritis; ATN
mixed forms (myeloma cast nephropathy + ATN, myeloma cast nephropathy + LCDD)
rare
proximal tubule disorders resembling Fanconi syndrome
hypercalcemic nephropathy
acute uric acid nephropathy/tumor lysis syndrome
obstructive nephropathy
rare glomerular forms: immunotactoid nephropathy, fibrillar nephropathy, cryoglobulinemic nephropathy, membranous nephropathy, thrombotic microangiopathy
iatrogenic lesions: contrast media, bisphosphonates, lenalidomide, furosemide

Abbreviations: ATN – acute tubular necrosis, LCDD – light chain deposition disease

TABLE 2 Associations between clinical manifestations and types of renal injury in multiple myeloma[5-9,25]

Predominant renal syndrome	Major type of renal lesion
acute kidney injury	myeloma cast nephropathy
	acute tubular necrosis
	iatrogenic effects
	direct infiltration of renal parenchyma
	acute tubulo-interstitial nephropathy
proteinuria/nephrotic syndrome	LCDD
	amyloidosis
	rare types of glomerular involvement
chronic renal failure	amyloidosis
	myeloma cast nephropathy
	LCDD
Fanconi syndrome	proximal tubulopathy

Abbreviations: see TABLE 1

in 15% to 20% of these cases it reaches the nephrotic range, i.e., exceeds 3.5 g/24 h.[7] Proximal tubular cell dysfunction resulting in glycosuria, aminoaciduria, hyperphosphaturia, and renal tubular acidosis secondary to bicarbonate loss (acquired Fanconi syndrome) is probably the most subtle manifestation of renal MM that is particularly difficult to detect.[8] TABLE 2 summarizes associations between clinical manifestations and type of renal injury in MM.

Pathophysiology of kidney damage in multiple myeloma The type of renal involvement in MM depends on numerous factors, including stage and activity of the disease, type of LCs synthesized in excess, the renal load of LCs (amount of filtered LCs), target cells that interact with certain type of LCs, urine pH, calciuria, amount of urinary Tamm-Horsfall (TH) protein in distal nephron, and others. The individual physicochemical characteristics of LCs appear to be critical, hence one could expect, in the particular case of MM, the presence of LCs that are predominantly glomerulopathic (leading to LC deposition disease [LCDD]), amyloidogenic, tubulopathic, or cast-forming.

Light chain deposition disease Up to 70% of all LCDD cases develop in the course of MM; the remaining 30% may be caused by other diseases. This type of renal manifestation is quite rarely diagnosed on autopsy, whereas it is present in 20% to 25% of the cases of renal biopsies performed in patients with MM (which is obvious given the fact that proteinuria and nephrotic range proteinuria are the best-recognized indications for biopsy). Patients with LCDD are younger than those with other types of renal involvement (median age, 58 years), almost universally manifest with heavy proteinuria, and, in most cases, with elevated serum creatinine. In about 35% of the cases, LC deposits can also be found in other organs, most frequently in the heart and liver.[5,6,9-12] Recent studies have demonstrated that in a very early phase of LCDD, glomerulopathic κLCs freely pass through the filtration membrane and accumulate subendothelially in the glomerular basement membrane (GBM). This results in submicroscopic GBM damage and selective proteinuria (predominantly albuminuria) resembling minimal change disease. Of note, LCs may be deposited within glomerulus or be completely reabsorbed in the proximal tubules (until complete saturation of their resorptive capacity is reached). This explains why in MM, even with advanced renal lesions, LCs may not be detectable in urine.[13] In the next stage of glomerular involvement, LCs reach mesangial compartment and stimulate the proliferation of mesangial cells. At this stage, the disease appears as proliferative glomerulonephritis (mesangial proliferative or membranoproliferative pattern of injury). Granular deposits containing κLCs can be identified within mesangium. With time, mesangial cells synthesize in excess extracellular matrix (ECM) proteins, including type IV collagen, laminin, fibronectin, and tenascin C. Simultaneously, the activity of enzymes that catabolize ECM (including metalloproteinase 7) decreases, which additionally enhances the accumulation of ECM. At this stage, glomeruli resemble nodular sclerosis, similar to that observed in advanced diabetic nephropathy.[13] Proteinuria may be present in all mentioned stages, accompanied by a gradually decreasing glomerular filtration rate (GFR).

Amyloidosis Amyloid (AL type) is found in less than 5% of the patients with MM on autopsy and in 15% to 35% patients with MM and renal symptoms who undergo renal biopsy. Since only from 15% to 35% of the patients with AL amyloidosis suffer from MM, most cases of AL amyloidosis seem to be primary disease.

As in the case of LCDD, proteinuria is present in up to 80% of the patients, and in more than 50%,

it exceeds the nephrotic threshold. More than half of the patients also have different stages of renal failure. The disease is diagnosed based on kidney biopsy with amyloid identification using Congo red staining and specific anti-LC antibodies that identify the type of amyloid. As in other forms of systemic amyloidosis, also in MM--related AL amyloidosis the biopsies of other tissues (i.e., the rectum, gum, or subcatenous fat) may be helpful. In contrast to LCDD, AL amyloidosis is formed mostly by λLCs. Interestingly, in some cases of AL amyloidosis (up to 35%), λLCs cannot be identified because their immunogenic fragments are damaged or hidden (the same applies to κLC in LCDD).[14] The assessment of circulating free LCs, with identification of high absolute amount of clonal LCs as well as an abnormal κ-to-λ ratio has shown to be a sensitive method for the detection of AL amyloidosis; changes of free LC concentration in plasma are predictive of the outcome and response to treatment in this disease.[15-18]

Survival of patients with AL amyloidosis is significantly worse compared with that of patients with LCDD, and the prognosis is worsened mainly by extrarenal deposits.[3]

Proximal tubulopathy and nonspecific interstitial fibrosis LCs accessing the proximal tubule are reabsorbed in excess by proximal tubular epithelial cells (PTC). If these are of the Vκ1 LC subtype, they may lead to another type of renal damage, namely, crystalline-inclusion LC nephropathy (proximal tubule disorders resembling Fanconi syndrome). The condition is particularly rare (1%) but it may also be overlooked due to very discrete clinical symptoms (glucosuria, aminoaciduria, hyperphosphaturia, hypercalciuria, mild metabolic acidosis with bicarbonate loss). Most of the above parameters (except glucose and urine pH) are not routinely assessed; therefore, it is important to consider MM in elderly patients with glucosuria without hyperglycemia and/or with metabolic acidosis accompanied by high urine pH.[19]

Interstitial kidney fibrosis is one of the most important mechanisms of chronic renal damage, almost universal in all nephropathies, particularly in those with proteinuria. Recently, the role of injured tubular cells has been highlighted in this process. Namely, PTC may undergo epithelial-to-mesenchymal transition (EMT) when challenged with an excess urinary protein. The cells loose their epithelial phenotype, interrupt (digest) the underlying tubular basement membrane, and migrate into the interstitium where they acquire myofibroblastic pheonotype and start to synthesize ECM proteins – de-differentiated PTC contribute to interstitial fibrosis. These mechanisms were also described in MM: LCs reabsorbed by PTC activate the nuclear factor κB, which in turn stimulates the transcription of several genes encoding proteins responsible for EMT (in particular interleukin 6, interleukin 8, and macrophage/monocyte chemotactic protein). LCs also activate the synthesis of transforming growth factor-β (TGF-β), one of the most potent profibrotic growth factors. LCs appeared to be more potent activators of EMT than albumin in nephrotic syndrome and more potent inductors of TGF-β synthesis than one of its strongest stimulator – cyclosporine.[20-22] Of note, LCs enter the PTC via the megalin- and cubilin-mediated endocytosis (megalin and cubilin are nonspecific receptors responsible for reabsorption of many proteins filtered in glomerulus, including albumin and vitamin D-binding protein). It has been demonstrated that silencing cubilin and megalin may prevent PTC from the damage induced by LCs – under these circumstances LCs do not enter PTC.[23]

Myeloma cast nephropathy Myeloma cast nephropathy (or myeloma kidney) is probably the most frequent and most important renal manifestation of MM. This entity is observed in more than 50% of the patients who died from MM and renal involvement, and in 40% to 60% of renal biopsies performed in subjects with MM.[7,8,10,24] The most important factor promoting cast formation in LC-saturated urine is the TH protein. The rate of cast formation depends largely on the type and characteristics of LCs (predominantly κLCs) as well as the specific features of TH protein, for example the degree of its glycation. The interaction between κLC and TH protein resulting in cast precipitation is enhanced particularly in the presence of high urinary calcium and sodium; thus, hypercalciuria and natriuresis promote cast formation. Hence some drugs, for example loop diuretics, used both for diuresis stimulation and as an calciuric agent, may worsen the course of myeloma cast nephropathy. The disorder almost always manifests with renal failure (in up to 50% of the cases acute or rapidly developing); proteinuria, although present in about two-thirds of the cases, usually does not reach the levels observed in LCDD or amyloidosis. Myeloma cast nephropathy is associated with exceptionally poor prognosis for both renal and overall survival. The disorder is observed in more advanced stages of myeloma and is associated with extremely high plasma LC levels (usually higher than 1000 mg/l, i.e., 50- to 100-fold higher than normal). Myeloma cast nephropathy as an underlying cause of end-stage renal disease (ESRD) is also associated with significantly worsened survival after the start of dialysis compared with ESRD secondary to other forms of renal involvement due to MM.[7,25] Identification of specific domains on LCs responsible for TH-protein binding has recently allowed to develop competitor peptides that bind with LCs and prevent from cast precipitation and loss of renal function in the rodent model of MM.[26,27] This may indicate new perspectives to prevent kidney complications in MM.

Therapeutic considerations It should be emphasized that none of the renal complications of MM should lead to the decision of withholding

pharmacotherapy or hematopoietic stem cell transplantation, although the rate of adverse effects of these therapies is expected to be higher compared with patients with normal renal function.[28] Results of numerous trials published recently clearly demonstrate that novel therapies used in MM, such as bortezomib or lenalidomide, are almost equally effective in patients with and without renal insufficiency in terms of hemato-oncological criteria (time to progression of a disease, progression-free survival, overall survival, etc.). Use of these therapies resulted also in an increased rate of renal function recovery. The safety profile of bortezomib in unadjusted dosage appeared to be similar in patients with renal failure.[29-31] The dose of lenalidomide needs to be adjusted to the estimated GFR rate in order to limit potential toxicity, since this drug is eliminated with urine in an unchanged form in up to 84%.[32-34] It should be kept in mind, however, that the above new agents – although very promising – are not registered as the first-line treatment and are available with certain restrictions; hence high-dose steroids still remain the first-line therapy for renal involvement in everyday practice.

The use of bisphosphonates in patients with advanced bone lesions, hypercalcemia, and concomitant renal failure remains a controversial issue. On the one hand, treating hypercalcemia may limit kidney damage. However, according to the Food and Drug Administration (FDA) labeling and other regulatory documents, most of bisphosphonates should not be used in patients with GFR below 30 ml/min. Although many reports have shown safety of these drugs in patients with MM and kidney failure, their use in patients with significantly compromised kidney function should be considered as an off-label therapy.[35,36] It seems that limiting the use of bisphosphonates in patients with kidney disease needs to be revised because the FDA regulations are based on toxicology studies performed in animals and reports of AKI following rapid intravenous injection of zoledronic acid; retrospective analyses of large databases of postmenopausal women treated with bisphosphonates for osteoporosis indicate that these drugs are equally effective and safe also in patients with the GFR below 30 ml/min.[37,38]

Dialysis using high cut-off dialysis membranes (i.e., with large pores permeable to free LCs) has emerged recently as a promising treatment strategy in patients with kidney failure due to myeloma cast nephropathy.[6,39-41]

Summary and conclusions In our brief review, we have discussed the main kidney disorders in MM and some new aspects of their pathophysiology. One should keep in mind, however, that in most cases these renal manifestations usually overlap and usually 2 or 3 manifestations may be observed simultaneously (not infrequently with superimposing iatrogenic damage). In addition, background chronic nephropathy may be present in affected patients (for example diabetic or hypertensive). We believe that this dangerous, but also intriguing, disease provides new insight into the pathophysiology, pathomorphology, and clinical appearance of renal diseases. It seems that nephrologists are important partners and collaborators of hematologists in the diagnosis and treatment of MM, since renal manifestations are commonly detected first. It could also be postulated that kidney biopsy should be considered more frequently (possibly in most, if not all cases of MM) to establish a more precise renal and overall prognosis.

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ARTYKUŁ POGLĄDOWY

Zmiany nerkowe w przebiegu szpiczaka plazmocytowego

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nefropatia szpiczakowa, przewlekła choroba nerek, ostre uszkodzenie nerek, szpiczak plazmocytowy

STRESZCZENIE

W tym artykule omówiono spektrum zmian obserwowanych w nerkach u chorych na szpiczaka plazmocytowego (*multiple myeloma* – MM). Skupino się na najważniejszych mechanizmach patofizjologicznych prowadzących do rozwoju MM. Szczególnie dużo uwagi poświęcono zależnościom między zmianami morfologicznymi w nerkach oraz objawami klinicznymi. Czynnikiem decydującym o rozwoju poszczególnych postaci zajęcia nerek jest przede wszystkim fizykochemiczna charakterystyka syntetyzowanych w nadmiarze łańcuchów lekkich. Wydaje się, że pacjenci z MM powinni być aktywnie leczeni niezależnie od rodzaju spotykanych zmian ponieważ współczesne metody pozwalają na regresję zmian nerkowych i zniwelowanie niekorzystnego wpływu uszkodzenia nerek na rokowanie tych chorych.

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