

CASE REPORT I

Multiple myeloma presenting with digital gangrene and renal impairment in a frail older adult: A case report

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Abstract

Multiple myeloma (MM) is a plasma cell malignancy that primarily affects the elderly, with high mortality rates. MM can present with a wide range of clinical manifestations. While common features include anaemia, renal dysfunction, bone pain, and hypercalcaemia, atypical presentations may delay diagnosis and affect outcomes. We report the case of a 74-year-old frail man with multiple comorbidities who presented with progressive digital gangrene and worsening renal function. Initial investigations ruled out peripheral arterial disease and raised the possibility of vasculitis. However, serum and urine electrophoresis revealed monoclonal kappa light-chain gammopathy. The bone marrow biopsy confirmed the diagnosis of MM, while imaging revealed spinal lytic lesions. Renal biopsy revealed both cast nephropathy and membranoproliferative glomerulonephritis. Patient refused chemotherapy, and while on supportive care, he died of sepsis and renal failure. This case highlights an unusual presentation of MM, emphasising the fact that apart from vasculitis, monoclonal gammopathies should also be considered as a differential diagnosis for digital ischaemia and renal impairment in older patients. Furthermore, it emphasises the significance of integrating the patient's functional status, comorbidities, and treatment preferences into clinical decision-making. In frail older adults, shared decision-making ensures that care remains patientcentred, appropriate, and ethically grounded.

Keywords: frail older adult, digital gangrene, renal impairment, multiple myeloma

Introduction

Multiple Myeloma (MM) is a B-cell malignancy characterised by the abnormal proliferation of plasma cells within the bone marrow, leading to the production of a monoclonal immunoglobulin. The disease primarily affects older adults, with a median age at diagnosis of 69 years. Thirty-five per cent of patients are diagnosed at age 75 or older, including 10% at age 85 or older [2].

According to the International Agency for Research on Cancer (IARC), in 2018, MM affected approximately 160,000 individuals globally, resulting in around 106,000 deaths [1]. Mortality rates are highest in patients above 75

years, with early deaths particularly frequent among those over 70. Several factors—including treatment tolerance, comorbidities, and functional status—contribute to poor outcomes in this age group [3] [4].

MM can range from an asymptomatic condition like monoclonal gammopathy of undetermined significance (MGUS) to severe presentations involving multiple organs. Classic features include the CRAB acronym—Calcium elevation, Renal dysfunction, Anaemia, and Bone lesions—driven by abnormal immunoglobulin production and bone marrow infiltration. Anaemia is the most common manifestation of MM, found in about 73% of MM patients at presentation, while hypercalcemia, bone

pain, and renal dysfunction occur in 28%, 58%, and 48% of patients, respectively [5].

Beyond the typical findings, MM can occasionally present with less common signs, involving the vascular, neurological, and dermatological systems. Peripheral ischemia and digital gangrene are rare and may be mistaken for autoimmune or vascular pathologies. When present, these are most often linked to cryoglobulinemia or hyperviscosity states [6].

We present a case of a frail elderly man with multiple comorbidities who presented with progressive digital gangrene and renal dysfunction, which led to the diagnosis of MM.

Case description

Mr J, a 74-year-old unmarried, retired teacher from the suburban outskirts of Colombo, presented to the National Hospital in Colombo in May 2022 with a three-day history of blackish discolouration of the fingertips. The symptoms began in the right second and third fingers as a dusky hue and rapidly progressed to involve all ten digits. He experienced numbness and tingling sensations in the fingers but reported no pain. Over the preceding three months, he had also experienced poor appetite and notable unintentional weight loss.

He had multiple comorbidities, including a 25-year history of type 2 diabetes mellitus with microvascular complications: diabetic neuropathy, nephropathy, and retinopathy. He also had hypertension for 15 years and a previous non-ST elevation myocardial infarction three years ago. His medications included Mixtard insulin (35 units in the morning, 15 units in the evening), metformin SR 500 mg twice daily, empagliflozin 10 mg daily, Aspirin 75 mg at night, enalapril 2.5 mg twice daily, bisoprolol 2.5 mg twice daily, atorvastatin 40 mg at night, and isosorbide mononitrate 30 mg daily.

Three months before this presentation, he fell in the bathroom and sustained a right-sided neck of femur fracture. Though he underwent Austin Moore hemiarthroplasty, he was unable to bear weight on the affected leg and remained bed-bound. He became fully dependent for activities of daily living, including transfer, mobility, toileting, bathing, grooming and feeding. According to the Clinical Frailty Scale, he was at Level 7—severely frail on admission. He lived with his sister's family and had the support of a full-time caregiver. He had no known drug allergies and no family history of autoimmune or malignant diseases.

On physical examination, he was afebrile and pale. All ten fingertips showed blackish discolouration

(Figures 1 & 2). There were no gangrenous changes in the toes. Livedo reticularis was visible on both thighs and

lower legs (Figure 3). Peripheral pulses were palpable in all limbs. No peripheral stigmata of infective endocarditis and cardiovascular examination revealed a regular pulse at 98 bpm and blood pressure of 90/60 mmHg. Jugular venous pressure was not elevated. Respiratory and abdominal exams were unremarkable.

Examination of the back revealed pressure ulcers: grade II at the left greater trochanter and grade III at the sacrum, both covered in slough. Neurologically, he exhibited distal muscle wasting, diminished ankle reflexes, and a stocking-type sensory loss in the lower limbs up to the knees.

Peripheral arterial disease was initially suspected; however, arterial and venous Doppler ultrasonography demonstrated patent vessels. A broader differential diagnosis was considered, including cardioembolic events, hypercoagulable states, and systemic vasculitis. Investigation findings are summarised below (Table 1). Urinalysis revealed proteinuria and red cell casts, and serum creatinine was elevated, raising suspicion for glomerular disease. Empirical intravenous methylprednisolone pulses were initiated while awaiting the renal biopsy report.

Despite immunosuppressive therapy, his digital necrosis and renal function continued to worsen. Further investigations revealed a monoclonal band in the gamma region on serum protein electrophoresis. Immunofixation confirmed a monoclonal gammopathy of Kappa light chains. The serum Kappa/Lambda ratio was elevated to 51.9 (reference range: 0.26–1.65). Urinary immunofixation showed Kappa-type Bence Jones proteinuria.

Bone marrow aspiration and trephine biopsy demonstrated 20% plasma cell infiltration, confirming MM. Imaging studies showed a wedge fracture at T12 and multiple lytic lesions in the lumbar spine, with no additional lesions on skull or long bone X-rays. In view of the presence of monoclonal Kappa light chains, increased plasma cell count on bone marrow examination, and multiple osteolytic lesions, a diagnosis of multiple myeloma was made.

The diagnosis of MM was discussed in detail with the patient and his family along with the disease prognosis and available treatment options. Mr. J was deemed to have full decision-making capacity and, after discussions with his close relatives, he opted not to pursue chemotherapy.

Over the course of his hospital stay, his condition deteriorated further, marked by a progressive rise in serum creatinine levels. He subsequently developed aspiration pneumonia and sepsis, which contributed to further clinical decline and he passed away on the 18th day of hospitalisation. The renal biopsy report, received posthumously, revealed a membranoproliferative glomerulonephritis (MPGN) pattern with crescent formation in one of four glomeruli, and coexisting cast nephropathy

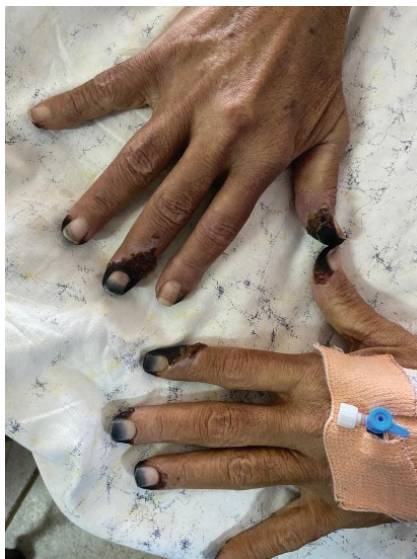


Figure 1. Digital necrosis involving all 10 finger tips.



Figure 2. Livedo reticularis involving lower limbs.

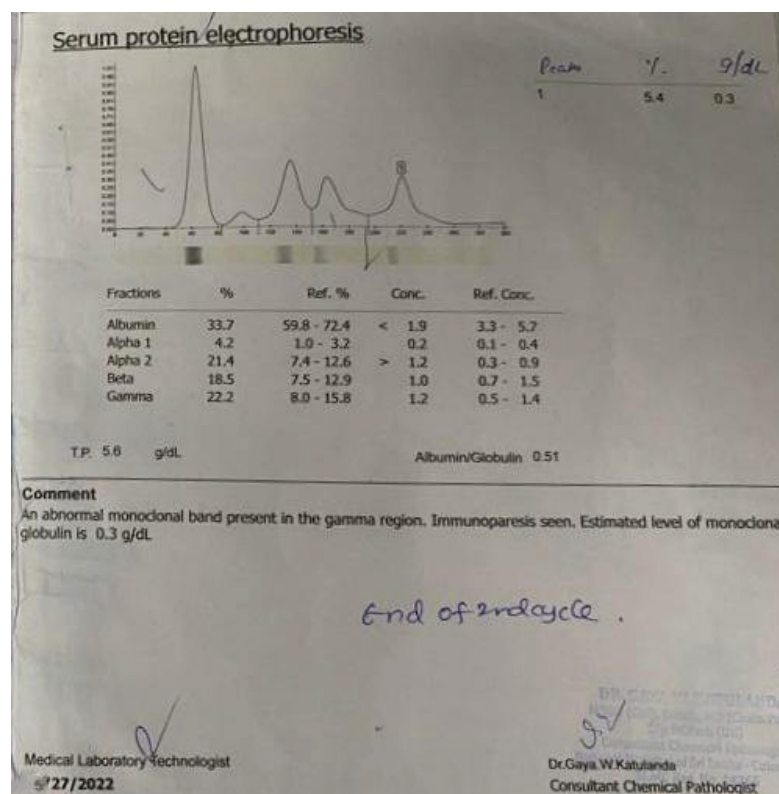


Figure 3. Serum protein electrophoresis.

Multiple myeloma presenting with digital gangrene and renal impairment

Table I. Investigations – Summary

Category	Investigation	Result
Hematology	Hemoglobin	8.6 g/dL
	Hematocrit	37%
	Total Leukocyte Count	9,200 /mm ³
	Neutrophils	86%
	Lymphocytes	9.2%
	Platelets	364,000 /mm ³
Renal Function	Blood Picture	Normochromic normocytic anemia with moderate rouleaux formation
	ESR	120 mm/h
	CRP	6.8 mg/dL
	Serum Creatinine	1.64 mg/dL
	Blood Urea Nitrogen (BUN)	65 mg/dL
	Sodium	136 mEq/L
Liver Function	Potassium	3.8 mEq/L
	Serum Albumin	2.2 g/dL
	Serum Globulin	7.1 g/dL
	Albumin/Globulin Ratio	Reversed
	ALT, AST	Within normal limits
	Alkaline Phosphatase (ALP)	240 U/L (High; reference: 45–150 U/L)
Calcium Metabolism	Serum Corrected Calcium	9.4 mg/dL (Within normal range)
Glucose	Random Blood Sugar	161 mg/dL
Coagulation Profile	PT	14.7 seconds
	INR	1.03
	APTT	29 seconds
Urinalysis	Urine Protein	+++
	RBC in Urine	6 /hpf
	WBC in Urine	4 /hpf
	Urine RBC Casts	Present
	Urine Dysmorphic RBCs	Positive
	Urine Protein-Creatinine Ratio	4.93 (Nephrotic range: > 2.5)
Protein Studies	Serum Protein Electrophoresis	Abnormal monoclonal band in gamma region with immunoparesis
	Serum Immunofixation	Monoclonal gammopathy of Kappa light chains
	Serum Free Light Chains	Elevated; Kappa/Lambda ratio: 51.9 (Ref: 0.26–1.65)
	Urine Immunofixation	Positive for Bence Jones proteinuria (Kappa type)
Bone Marrow	Bone Marrow Biopsy	20% plasma cells; consistent with multiple myeloma
Radiology	X-ray Spine	T12 wedge fracture; lytic lesions in lumbar vertebrae
	X-ray Skull & Long Bones	No additional lytic lesions
	Whole Body MRI	High signal in T11, T12, L1, and L3 vertebral bodies
Autoimmune Panel	Rheumatoid Factor	Negative
	ANA, C-ANCA, PANCA	Negative
	Serum Cryoglobulins	Negative
	Complement C3	127 mg/dL (Normal: 88–165)
Infectious Screening	Complement C4	34 mg/dL (Normal: 14–44)
	HIV, Hepatitis B & C	Negative
Cardiology	VDRL	Non-reactive
	ECG	Left ventricular hypertrophy
	Transthoracic Echocardiography	No vegetations, tumors, or embolic source; LVEF 65%
Imaging	Chest X-ray	Normal
	Renal Ultrasound	Altered corticomedullary differentiation; no hepatosplenomegaly
Biopsy	Renal Biopsy (posthumous)	Membranoproliferative GN with cellular crescent (1/4 glomeruli); co-existing cast nephropathy

Discussion

MM is a B-cell malignancy characterised by clonal proliferation of plasma cells that produce monoclonal immunoglobulins, and it predominantly affects older adults. According to the International Myeloma Working Group (IMWG) diagnostic criteria, there should be 10% or more clonal plasma cells in the bone marrow and/or a biopsy-proven plasmacytoma with any one or more myeloma-defining events (MDE) to diagnose MM. MDEs included in the criteria are end-organ damage attributable to the underlying plasmacell disorder (hypercalcemia, renal insufficiency, anaemia, or bone lesions), bone marrow clonal plasma cells $\geq 60\%$, serum involved to uninvolved free light chain (FLC) ratio ≥ 100 (provided involved FLC level is ≥ 100 mg/L), and more than one focal lesion (5 mm or more in size) on magnetic resonance imaging (MRI) [7].

In our patient, the initial presentation with digital ischemia posed a diagnostic challenge. Cryoglobulinemia is the most frequently reported cause of such a presentation in MM [6]; however, qualitative testing for cryoglobulins was negative. Hyperviscosity syndrome due to high serum paraprotein levels may also result in sluggish blood flow and vascular occlusion, presenting with livedo reticularis, cyanosis, and gangrene [7].

Renal complications in MM are complex and may result from volume depletion, hypercalcemia, or direct tubular injury by light chains. Cast nephropathy is the most prevalent histological finding [8]. Our patient had both mesangio-proliferative glomerular nephritis (MPGN) and cast nephropathy. MPGN is typically immune-complex-mediated and is more commonly associated with infections or autoimmune conditions. When seen in MM, it may be secondary to monoclonal gammopathy.

At the initial presentation, systemic vasculitis was strongly suspected, and the presence of rapidly progressive glomerulonephritis (RPGN) with urinary red cell casts justified the use of high-dose steroid pulses and the decision to proceed with a renal biopsy. This was a shared decision made in consultation with the nephrology team, as the biopsy was expected to clarify the underlying pathology. At that stage, serum protein electrophoresis and bone marrow biopsy results confirming MM were still pending, and the diagnostic uncertainty influenced the course of action.

Ultimately, the biopsy revealed both MPGN and cast nephropathy, providing academic insight into the renal pathology. However, in retrospect, once MM was confirmed, the renal biopsy may not have been compulsory, especially given the patient's severe frailty and preference against chemotherapy. This case highlights the importance of carefully tailoring of investigations for

frail older adults, ensuring that a clear benefit to patient care justifies their risks.

The decision to initiate high-dose corticosteroids was made based on suspected vasculitis. Patient was already on low-dose aspirin for ischemic heart disease. There was no indication for anticoagulation, as the ischemic changes were more likely related to paraprotein-mediated vascular injury rather than thromboembolic disease.

Treatment decisions in MM are guided by the patient's age, comorbidities, and performance status [9]. Comprehensive geriatric assessment is a valuable tool to identify frailty and resolve occult health factors among older adults with MM [4]. Though autologous stem cell transplant (ASCT) is considered the standard of care, transplantation is less frequently performed for adults older than 65 years of age. There are numerous chemotherapeutic regimens used in the treatment of MM. The major classes include alkylating agents (melphalan, cyclophosphamide), proteasome inhibitors (bortezomib), immunomodulators (thalidomide, lenalidomide), and corticosteroids (dexamethasone, prednisone) [10].

Cutaneous manifestations such as digital gangrene in MM are associated with poor prognosis. A Korean case series of 1,228 MM patients demonstrated significantly reduced survival in those with skin involvement [11]. In this case, frailty, comorbidities, and the patient's personal preference against chemotherapy were central to the outcome. Additional supportive measures, such as meticulous wound and pressure ulcer care, infection prevention, nutritional optimisation, and early palliative care input, should have been prioritised over invasive diagnostics. This case emphasises the value of shared decision-making and a patient-centred approach in managing frail older adults.

Conclusion

Digital ischaemia and necrosis are rare and atypical presentations of MM, which may lead to delayed diagnosis and treatment. Although renal involvement and digital ischemia should always prompt investigations for vasculitis, monoclonal gammopathies should also be considered as a differential diagnosis, especially in elderly patients.

Equally important is the integration of the patient's frailty, comorbidities, decision-making capacity, and personal values into clinical planning. This case highlights that while diagnostic thoroughness is important, extensive invasive investigations may not always be appropriate in frail older adults, especially when treatment options are limited or declined. Greater emphasis should instead be placed on supportive measures, prevention of complications, and palliative input. Ultimately, individualised and ethically grounded decision-making ensures that care for older adults remains both clinically sound and patient centred.

Author Contributions: All authors listed contributed sufficiently to the project to be included as authors. DEMS Edirisinghe, D Dissanayake and M Senevitathne were involved in analysing and interpreting patients' clinical data and management of the patient. DEMS Edirisinghe contributed to conducting the literature review and writing the manuscript

Consent for publication: Informed consent for publication was obtained from patient's next of kin.

Declaration Conflicts of Interest: None declared

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