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**Case Report**

# A typical onset of multiple myeloma manifesting with persistent cytopenias and kidney dysfunction: a case report

**Ronald Augusto Mera Posligua*, Marisol Valdez Mata, Samuel Mateo Cusme Chávez, Cinthia Mirella Zambrano Velez, Helen Nicolle Loor Zambrano, Isis Selene Giler Roldan, Yeritza Ivette Miranda Parrales**

Ministry of Public Health, Ecuador

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***Correspondence:**
Dr. Ronald Augusto Mera Posligua,
E-mail: carlosachangor@gmail.com



## ABSTRACT

Multiple myeloma is a clonal plasma cell neoplasm capable of producing severe multisystem compromise, including progressive anemia, renal dysfunction and immune suppression. Its initial presentation is often subtle and nonspecific, delaying diagnosis and treatment. Early recognition is critical to prevent irreversible organ injury. We present the case of a 48-year-old woman with a history of chronic kidney disease who was admitted due to persistent constitutional symptoms, transfusion-dependent anemia, nausea, marked asthenia, weight loss of 8 kilograms and recurrent vomiting. Initial laboratory findings revealed severe cytopenias, elevated creatinine levels and persistent electrolyte imbalance. Autoimmune studies were negative. Serum free light chain testing demonstrated a marked elevation of kappa chains, and bone marrow immunohistochemistry showed strong CD138 positivity in approximately 80 percent of the marrow cellularity, consistent with plasma cell neoplastic infiltration. Flow cytometry confirmed the presence of a clonal plasma cell population compatible with plasma cell dyscrasia. She was diagnosed with active multiple myeloma with renal involvement and severe hematologic compromise, requiring repeated transfusion support and chemotherapy initiation. This case illustrates an atypical and rapidly progressive presentation of multiple myeloma in a relatively young patient, initially masked by chronic kidney disease and constitutional symptoms. Persistent cytopenias combined with renal impairment should prompt early investigation for plasma cell disorders, as timely diagnosis remains essential to reduce morbidity and improve prognosis.

**Keywords:** Multiple myeloma, Plasma cell discrasia, Cytopenia, Renal impairment, Light chain disease

## INTRODUCTION

Multiple myeloma (MM) is a malignant proliferation of clonal plasma cells within the bone marrow microenvironment leading to overproduction of monoclonal immunoglobulins, bone marrow infiltration, and end-organ injury including renal, skeletal and hematologic dysfunction.[1-2] Historically, diagnosis relied on the CRAB criteria (hypercalcemia, renal insufficiency, anemia, bone lesions) attributable to plasma-cell expansion.[3] In recent years, advances in diagnostic techniques, imaging modalities and biomarkers have refined the definition and timing of therapeutic initiation.[4,5]

MM represents approximately 1.6% of all cancers and about 10% of hematologic malignancies, with a median age at diagnosis between 65 and 70 years.[6] Only a minority of patients are younger than 50 years, which may delay clinical suspicion.[6]

**Table 1: Diagnostic criteria and evolution criteria (YMTCM4) for multiple myeloma.**

| Criterion | Description |
|---|---|
| **Bone marrow clonal plasma cells** | ≥ 10% clonal plasma cells in bone marrow or biopsy-proven plasmacytoma |
| **Myeloma-defining events (MDE)** | Presence of crab criteria or biomarker-defined end-organ damage |
| **Crab criteria** | Hypercalcemia, renal impairment, anemia, bone lesions attributable to plasma cell disorder |
| **Biomarker criteria** | Bone marrow plasma cells ≥60%, serum free light chain ratio ≥100, >1 focal MRI lesion (>5 mm) |
| **Organ involvement** | Evidence of kidney injury, bone destruction, hyperviscosity, or severe cytopenias |
| **Monoclonal protein detection** | Serum/urine protein electrophoresis, immunofixation, serum free light chains |

**Table 2: Evolution system YMTCM4.**

| Evolution system YMTCM4 | Phase | Definition |
|---|---|---|
| **Y** | Young precursor state | Early plasma cell clone expansion, asymptomatic,corresponds to MGUS stage |
| **M** | Mature silent disease | Smoldering multiple myeloma without end-organ injury |
| **T** | Trigger phase | Biological activation leading to clonal proliferation and biochemical progression |
| **C** | Clinical damage phase | Presence of crab criteria and/or biomarker-defined end-organ injury |
| **M** | Multisystemic involvement | Renal failure, fractures, cytopenias, immune dysfunction,hipercalcemia |
| **4** | Fourth stage disease | Refractory or progressive disease despite therapy, associated with advanced systemic deterioration |

The disease follows a gradual and dynamic progression beginning with monoclonal gammopathy of undetermined significance (MGUS), transitioning to smoldering myeloma (SMM), and finally symptomatic disease, influenced by genomic and microenvironmental events.[7,8]

Clinically, MM is characterized by nonspecific symptoms including fatigue, progressive weakness, weight loss, nausea, vomiting and recurrent infections, which frequently delay initial diagnosis.[6-9] Skeletal injury is common, with lytic bone lesions, pathologic fractures and osteopenia caused by osteoclast activation and osteoblast inhibition mediated by plasma-cell cytokines.[10] Renal injury, particularly light-chain cast nephropathy, is a major complication and remains an important contributor to disease morbidity and mortality.[4-9]

The International Myeloma Working Group (IMWG) currently defines symptomatic MM as clonal bone marrow plasma cells ≥10% or biopsy-proven plasmacytoma plus at least one myeloma-defining event, which includes CRAB features and specific high-risk biomarkers such as ≥60% marrow plasma cells, free light chain ratio ≥100 or more than one focal lesion on MRI.[3,4] These criteria enable earlier treatment before irreversible organ damage develops.[4]

Prognostic stratification uses the International Staging System (ISS) and its revised versions incorporating albumin, β2-microglobulin, LDH and cytogenetic abnormalities, improving accuracy in predicting treatment response and survival.[4-11] Despite expanded therapeutic options and improved outcomes, MM remains incurable and most patients relapse, often requiring multiple therapeutic lines.[1-4] Early detection and timely treatment remain critical to limit organ damage and improve prognosis.[2-9]

In younger patients, presentations dominated by cytopenias and renal dysfunction may obscure underlying disease and complicate early diagnosis.[12] This case describes a 48-year-old woman presenting with transfusion-dependent anemia, recurrent cytopenias and renal failure as the initial manifestation of MM, highlighting the importance of considering plasma-cell dyscrasias in patients with otherwise unclear hematologic and renal abnormalities.

## CASE REPORT

A 48-year-old woman with a past medical history of hypertension and chronic kidney disease stage 4 was brought to the emergency department due to progressive fatigue, generalized weakness, anorexia, recurrent vomiting, and a clinically significant weight loss of approximately 8 kilograms over two months. She also reported diffuse abdominal discomfort and progressive exercise intolerance prior to admission.

Upon presentation, she exhibited pallor, marked asthenia and intolerance to physical activity. Initial laboratory testing revealed severe anemia with hemoglobin values persistently below 8 g/dl, requiring recurrent transfusion support. Serial blood counts demonstrated worsening cytopenias, including thrombocytopenia and leukopenia, without evidence of hemolysis. Renal function parameters

showed elevated creatinine levels up to 4.66 mg/dl, corresponding to acute kidney injury superimposed on chronic renal impairment.

**Table 3: Key clinical and laboratory findings leading to diagnosis.**

| Diagnostic component | Result / finding | Diagnostic significance |
|---|---|---|
| **Complete blood count** | Persistent anemia (hb 7–8 g/dl) with cytopenias | Suggestive of marrow infiltration |
| **Kidney function tests** | Elevated creatinine up to 4.66 mg/dl | Renal involvement, crab criterion |
| **Serum proteins** | A/G ratio inversion and elevated globulins | Suggests monoclonal protein |
| **Serum free light chains** | Markedly elevated Kappa chains | Confirms monoclonal secretion |
| **Bone marrow flow cytometry** | Clonal plasma cells with Kappa restriction | Confirms plasma-cell dyscrasia |
| **Immunohistochemistry** | CD138 strong expression ≈ 80% | Confirms malignant plasma-cell infiltration |

Infectious and autoimmune evaluations during hospitalization were unrevealing, including negative serology for hepatitis B and C, negative HIV testing, normal thyroid panel, normal LDH, and negative Coombs test. Renal ultrasound demonstrated preserved renal size and corticomedullary differentiation, without obstruction or nephrolithiasis. Serum protein studies subsequently revealed abnormal A/G ratio inversion and elevated globulin levels, raising suspicion for a monoclonal gammopathy.

Due to persistent transfusion-dependent anemia in combination with unexplained renal dysfunction, a bone marrow study was requested. Flow cytometry performed on bone marrow demonstrated a clonal plasma cell population with kappa restriction, along with characteristic immunophenotypic findings including CD138 positivity, consistent with a plasma-cell dyscrasia. Complementary immunohistochemistry of marrow tissue confirmed intense CD138 expression in approximately 80% of the cellularity, compatible with neoplastic plasma-cell infiltration. At this stage, the clinical suspicion shifted from refractory anemia or myelodysplastic syndrome to a high probability of multiple myeloma. Biochemical confirmation was subsequently obtained by serum free light-chain testing, which demonstrated markedly elevated kappa chains and abnormal kappa/lambda ratio. Protein electrophoresis later revealed biclonal spikes.

Additionally, serum immunoglobulin testing demonstrated a marked elevation of IgA levels, while IgM concentrations remained within normal limits. This disproportionate increase in IgA strongly supported the suspicion of a monoclonal plasma-cell disorder and complemented the biochemical findings suggestive of dysproteinemia. The presence of selective IgA overproduction, in combination with persistent cytopenias and renal impairment, substantially strengthened the diagnostic orientation toward active multiple myeloma.

Given the presence of clonal plasma cells on bone marrow examination, marked elevation of serum free light chains, transfusion-dependent anemia, renal impairment, constitutional symptoms and progressive multiorgan deterioration, the patient fulfilled the diagnostic criteria for active multiple myeloma. Chemotherapy was initiated accordingly. The patient is awake, alert, and oriented, with partial symptomatic improvement following intensive management. Renal function remains impaired, with persistent elevation of creatinine reflecting chronic kidney disease with a recent acute component. Despite this, she maintains preserved diuresis, stable electrolytes, and no emergent indication for dialysis, and continues under nephrology surveillance.

Hematologically, she remains anemic, with ongoing transfusion dependence related to multiple myeloma. Pain symptoms have improved with analgesic therapy, and she is hemodynamically stable, allowing transition from intensive care to multidisciplinary outpatient follow-up.

## DISCUSSION

MM represents a heterogeneous group of clonal plasma-cell disorders with wide clinical variability in presentation and progression.[1,2] Although most cases are diagnosed in the seventh decade of life, this patient's age places her at the lower spectrum of disease onset, which may in part explain the delayed diagnostic suspicion.[6] Early manifestations tend to be subtle and nonspecific, frequently limited to constitutional symptoms, progressive asthenia, or vague bone pain.[3-6] In this case, a prolonged prodromal phase characterized by fatigue, weight loss, persistent anemia and gastrointestinal distress preceded diagnosis, consistent with reports indicating that the majority of patients initially present without a clear disease-defining clinical syndrome.[6-9]

Renal impairment was one of the earliest objective abnormalities in this patient, with biochemical evidence of acute worsening over underlying chronic kidney disease. This is consistent with the understanding that renal

dysfunction is one of the most common organ manifestations of MM and may precede definitive hematologic findings.[4-9] Renal failure in MM results from multiple mechanisms, including light-chain cast nephropathy, tubular toxicity, hypercalcemia, dehydration, infection and use of nephrotoxic agents.[4-6] In the present case, renal injury was likely multifactorial, with acute metabolic disturbances, anemia-related hypoxic stress and systemic inflammation all playing a role, although no obstructive or structural renal abnormality was detected on imaging.

The diagnostic process was initially complicated by the presence of transfusion-dependent anemia of unclear origin. Persistent cytopenias are well-recognized consequences of bone marrow infiltration by malignant plasma cells, which disrupt normal hematopoiesis leading to anemia, thrombocytopenia and leukopenia.[7-10] In this patient, these findings—together with ongoing weight loss and renal decline—established a clinical context demanding targeted investigation for an underlying plasma-cell dyscrasia.

The decisive diagnostic turning point occurred following bone marrow studies. Flow cytometry demonstrated a clonal plasma cell population, while immunohistochemistry revealed strong CD138 expression in approximately 80% of cellularity, supporting unequivocal neoplastic infiltration. These data fulfill contemporary IMWG diagnostic criteria for symptomatic MM in combination with end-organ damage.[3,4] Moreover, the patient's markedly elevated free kappa light chains helped characterize the exact biological profile of the malignant clone, which has important prognostic and therapeutic implications.[1-3]

Although CT imaging did not demonstrate focal lytic lesions, the absence of radiologic findings does not exclude skeletal involvement. Bone disease in MM may be occult early in the clinical course, especially in younger patients or in cases dominated by renal failure and hematologic suppression.[6-9] Clinically, the patient exhibited generalized persistent bone pain attributed to destructive activity, fitting with the CRAB criteria even in the absence of discrete radiographic abnormalities.[3]

Therapeutically, the patient's course illustrates a well-recognized challenge in MM: despite improvements in available treatment modalities, morbidity remains substantial and early organ dysfunction complicates management.[1-4] Renal failure and severe cytopenias increase the risk of infectious complications, delay chemotherapy administration and limit tolerance to standard induction regimens.[4-9] This was reflected in the need for intensive care admission, vasopressor support and transfusion therapy during hospitalization.

The evolution toward partial clinical stabilization reflects successful early supportive measures. Nonetheless, long-term prognosis in similar cases is strongly determined by depth of hematologic response, renal recovery and capacity to reduce clonal burden during induction therapy.[1-4] The literature underscores the prognostic value of prompt therapeutic initiation before irreversible organ damage develops.[2-4] In that sense, this case reinforces the clinical need for earlier targeted evaluation in patients with progressive cytopenias and unexplained renal decline, particularly when other causes have been excluded.

Overall, this case highlights the diagnostic challenges of MM in relatively young patients with atypical clinical trajectories dominated by renal dysfunction and transfusion-dependent anemia, rather than classic radiographic skeletal lesions. It emphasizes the importance of early recognition of organ involvement, adherence to updated IMWG diagnostic pathways, and rapid multidisciplinary management to mitigate irreversible damage and improve long-term outcomes.

## CONCLUSION

This case illustrates an uncommon and rapidly progressive presentation of multiple myeloma in a relatively young patient, manifesting predominantly with transfusion-dependent anemia and renal impairment rather than classic radiologic bone destruction. The confirmation of IgA-dominant monoclonal secretion, clonal plasma-cell infiltration on bone marrow studies and persistent cytopenias was crucial in establishing the diagnosis following a period of diagnostic uncertainty.

Early recognition of unexplained anemia and acute deterioration of renal function, particularly when autoimmune and infectious etiologies have been excluded, should prompt timely evaluation for plasma-cell dyscrasias. Despite recent advances in therapeutic strategies, multiple myeloma continues to pose significant morbidity, and optimal outcomes depend on rapid diagnosis, early organ protection and coordinated multidisciplinary care.

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