

Multiple Myeloma Associated with HIV Infection in a Transgender Woman: Case Report and Review of an Integrated Management Approach

Tomás Andrés Lasso Arequipa ^{1,*}, Erika Daniela Criollo Pullupaxi ², Camila Sinai Yánez Montenegro ², Elian Andrés Urvina Maila ², Kimberly Camila Lastra Samaniego ², Joselyn Carolina Piarpuezan Contento ², Carlos Andrés Vallejo Betancourt ¹

¹ School of Medicine, Pontificia Universidad Católica del Ecuador, Quito, Pichincha, Ecuador.

² School of Medicine, Universidad de Las Américas (UDLA), Quito, Pichincha, Ecuador.

* Correspondence: tomasandreslasso@gmail.com.

Abstract: Multiple myeloma (MM) is a malignant hematologic neoplasm characterized by the clonal proliferation of plasma cells within the bone marrow, associated with the production of monoclonal immunoglobulins that lead to progressive bone destruction. This condition typically manifests with hypercalcemia, renal insufficiency, anemia, and lytic bone lesions, which constitute the CRAB criteria used for diagnosis. MM accounts for approximately 10% of hematopoietic neoplasms and about 1% of all cancers worldwide. The median age at diagnosis ranges from 65 to 70 years, and the disease is rare in individuals younger than 40 years. Established risk factors for MM include advanced age (predominantly affecting individuals over 65 years), male sex, African descent, obesity, the presence of monoclonal gammopathy of undetermined significance (MGUS), and certain precursor plasma cell disorders. MGUS is the most common precursor condition, with an estimated annual progression rate of approximately 1% to active multiple myeloma. Beyond classical risk factors, chronic states of immunosuppression, such as human immunodeficiency virus (HIV) infection, have been associated with humoral immune dysfunction and the development of plasma cell disorders. Epidemiological studies and case series suggest that patients living with HIV have an increased risk of developing monoclonal plasma cell disorders, including MGUS and MM, with some reports documenting a 2- to 5-fold higher risk compared with the general population. Clinical presentation in these patients also appears to occur at younger ages and with atypical features, posing an additional diagnostic challenge. This case highlights the importance of early diagnosis and comprehensive management in patients with HIV/AIDS, particularly those with poor adherence to antiretroviral therapy, who may be more susceptible to developing malignant hematologic conditions such as MM.

Keywords: Multiple Myeloma; HIV; Transgender; Hypocalcemia; Hyperphosphatemia; Parathyroid Hormone; Calcitonin; Electrophoresis; Mineral Bone Metabolism.

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1. Introduction

Multiple myeloma (MM) is a malignant hematologic neoplasm characterized by the clonal proliferation of malignant plasma cells within the bone marrow, leading to excessive production of monoclonal immunoglobulins, also known as M proteins [1]. These proteins disrupt normal renal function and may cause lytic bone lesions, hypercalcemia,

and anemia. The pathophysiology of MM is closely linked to a complex tumor microenvironment within the bone marrow, where malignant plasma cells interact with other cellular components, including osteoblasts and osteoclasts, thereby promoting bone resorption and angiogenesis [2]. The presence of a monoclonal protein spike, together with urinary immunoglobulin light chains, constitutes a hallmark finding that enables definitive diagnosis [3].

Although MM predominantly affects older adults, its association with conditions that compromise immune function, such as human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), has garnered increasing attention in recent years. Patients with advanced HIV/AIDS exhibit a higher susceptibility to the development of hematologic malignancies, including MM, due to chronic immunosuppression that facilitates uncontrolled proliferation of malignant plasma cells. Furthermore, poor adherence to antiretroviral therapy (ART) further exacerbates the risk of malignancy by impairing immune recovery and allowing unregulated tumor growth [3].

HIV infection leads to a decline in cellular immune responses, particularly affecting T-cell function, which in turn compromises the immune system's ability to regulate malignant plasma cell proliferation. Patients with HIV who do not adhere adequately to ART experience prolonged immunodeficiency, significantly increasing their risk of developing hematologic malignancies such as MM. Several studies have demonstrated that individuals with advanced HIV/AIDS have a substantially higher risk of MM compared with the general population, largely due to chronic immune system dysregulation and altered B-cell responses [4].

The complications of MM are multisystemic and may involve multiple organs. Hematologically, patients frequently develop anemia as a consequence of bone marrow infiltration by malignant plasma cells, resulting in impaired erythropoiesis. Leukopenia and thrombocytosis may also occur, increasing the risk of infections and coagulation disorders. Renal involvement is common, as myeloma-related nephropathy arises from the precipitation of immunoglobulin light chains within the renal tubules, leading to acute or chronic renal failure [4].

Regarding the skeletal system, lytic bone lesions are a defining feature of MM and result from osteoclast-mediated bone resorption induced by interactions with malignant plasma cells. This process leads to bone destruction, pathological fractures, and osteoporosis, which is commonly observed due to excessive osteoclast activation. These lytic lesions are readily detectable on radiographic imaging. MM may also be associated with hypercalcemia, secondary to calcium release from affected bone tissue [5].

The patient has a history of cosmetic procedures involving biopolymers, which may have induced chronic inflammation in the treated areas. However, current scientific literature does not provide sufficient evidence to establish a direct causal relationship between biopolymer exposure and the development of MM. In this case, the most relevant and well-established risk factor remains advanced HIV infection, which constitutes the primary underlying contributor to disease development.

The patient presented with a complex clinical picture, including a history of advanced HIV/AIDS with poor treatment adherence and multiple associated complications. On admission, the patient exhibited constitutional symptoms, asthenia, and lower limb muscle weakness. Physical examination revealed progressive edema, deviation of the fingers, impaired hand grip, and joint pain. Initial laboratory studies demonstrated significant anemia, thrombocytosis, and an active inflammatory response, evidenced by elevated C-reactive protein levels. Osteoporosis and metabolic profile abnormalities were also identified, while negative rheumatoid markers helped rule out rheumatologic disease. Based on these findings, a malignant hematologic disorder such as MM was suspected, prompting further diagnostic evaluation, including molecular studies, serum protein electrophoresis, and Bence Jones protein analysis, to confirm the presence of monoclonal gammopathy and assess the likelihood of MM.

2. Case Report

A 32-year-old patient, identified as a transgender woman for the past 22 years, born and residing in Quito, Ecuador, presented with a complex medical history. Her past medical history was significant for anemia diagnosed three months prior and long-standing human immunodeficiency virus (HIV) infection, currently on antiretroviral therapy (ART) with poor adherence. Surgical history included rhinoplasty performed one year earlier and multiple cosmetic procedures involving biopolymer injections in the gluteal region, breasts, face, and malar areas approximately ten years ago. Family history revealed a maternal grandmother with hypertension and diabetes mellitus and a maternal grandfather with gastric cancer. Sexual history was notable for multiple sexual partners, with sexual activity exclusively with men. Regarding toxic habits, the patient reported alcohol consumption every weekend, reaching intoxication, from the age of 24 to the present.

The patient had been hospitalized in June 2025 for evaluation of constitutional syndrome and diarrhea, characterized by unquantified weight loss, asthenia, and lower limb muscle weakness. In November, she presented to the emergency department of Hospital Enrique Garcés with progressive edema of both upper and lower limbs of 20 days' duration, accompanied by severe pain rated 9/10 on the visual analog scale (VAS). She also reported deviation of the fingers with difficulty in hand grip, as well as dysphagia and odynophagia.

Upon admission, vital signs were as follows: blood pressure 100/54 mmHg, heart rate 110 beats per minute, respiratory rate 19 breaths per minute, and temperature 36.5 °C. On general physical examination, the patient was conscious and oriented, with a Glasgow Coma Scale score of 15/15, afebrile, with pale sclerae and semi-moist oral mucosa. Dentition was in fair condition. Oropharyngeal examination revealed erythema with whitish plaques; tonsils were not hypertrophic. Neck examination showed a non-enlarged thyroid (grade 0A) and no palpable lymphadenopathy. The chest was symmetrical with preserved expansion. Pulmonary auscultation revealed normal vesicular breath sounds with crackles at the right lung base. Abdominal examination showed a soft, non-tender, depressible abdomen with splenomegaly and preserved bowel sounds. The inguino-genital region showed no apparent abnormalities.

Inspection and palpation of the upper extremities revealed ulnar deviation of both hands, more pronounced at the metacarpophalangeal joints, consistent with chronic joint involvement. There was bilateral superficial tenderness of the hands with clinical signs of active inflammation. Non-pitting inflammatory edema with bilateral distribution and associated trophic skin changes was also observed. No active ulcerative skin lesions were documented at the time of evaluation.

Examination of the lower extremities demonstrated superficial tenderness in both limbs, particularly at the ankles and toes, indicating joint involvement (Figure 1A). Findings were symmetrical, with palpable distal pulses, capillary refill time of less than three seconds, and non-pitting edema of the right ankle and bilateral toes. Given the clinical course and physical examination findings, a rheumatology consultation was requested for joint evaluation and multidisciplinary management.

2.1 Laboratory and Diagnostic Findings

Laboratory studies obtained at admission revealed the following findings. Complete blood count showed leukopenia with a white blood cell count of $4.00 \times 10^3/\mu\text{L}$ (reference range: 4.09–9.75), neutrophilia at 77.5% (reference range: 41.2–73.5), lymphopenia of $0.52 \times 10^3/\mu\text{L}$ (reference range: 1.92–6.44), and monocytopenia of $0.17 \times 10^3/\mu\text{L}$ (reference range: 0.19–0.68). Hemoglobin was 9.7 g/dL (reference range: 11.3–17.6) and hematocrit 30.7% (reference range: 34.7–53.6), consistent with significant anemia and an active systemic inflammatory response. Platelet count was markedly elevated at $588 \times 10^3/\mu\text{L}$ (reference

range: 131–387), indicating thrombocytosis. Inflammatory markers showed a significantly elevated C-reactive protein (CRP) level of 8.89 mg/dL (reference range: 0–0.4).

Figure 1. A. Clinical photograph demonstrating marked edema of the fingers and distal portion of the left hand, with discoloration of the fingernails of both hands showing a yellowish hue. Articular deformities involving several fingers are evident, suggestive of inflammatory joint disease, along with marked asymmetry. Anteroposterior radiographs of the left and right hands show abnormalities at the metacarpophalangeal joints, including bone erosions at the proximal interphalangeal joints. Ulnar deviation, joint deformity, and narrowing of the joint space are also observed.



The biochemical profile revealed glucose 77 mg/dL (reference range: 70–99), urea 31.4 mg/dL (reference range: 16.86–43.37), decreased creatinine at 0.74 mg/dL (reference range: 0.8–1.3), and hypoalbuminemia with albumin levels of 2.7 g/dL (reference range: 3.5–5.5). Thyroid function tests showed normal TSH at 3.28 μ IU/mL (reference range: 0.35–5.1) and normal free T4 at 0.95 ng/dL (reference range: 0.87–1.85). The electrolyte panel was within normal limits, including sodium 138 mmol/L (reference range: 137–147), potassium 4.25 mmol/L (reference range: 3.5–5.3), and chloride 107 mmol/L (reference range: 99–110). Procalcitonin level was 0.2 ng/mL (reference range: 0–0.2).

Based on these findings, the patient was admitted to the internal medicine service for multidisciplinary management involving internal medicine, endocrinology, rheumatology, and hematology. Treatment included antibiotic therapy, optimization of antiretroviral therapy, calcifediol, calcium carbonate prescribed by endocrinology, and magnesium citrate, with close monitoring of clinical evolution, daily serum calcium levels, renal function, and follow-up by the involved specialties.

Further complementary studies were obtained. Molecular testing revealed a CD4+ T-cell count of 79 cells/ μ L (reference range: 500–1,500), indicating severe immunosuppression consistent with advanced HIV infection (AIDS) and a high risk for opportunistic infections. HIV viral load testing showed detectable virus with <40 copies/mL (log 1.60), indicating minimal viral replication. Autoimmune studies demonstrated negative anti-cyclic citrullinated peptide (anti-CCP) antibodies and a normal quantitative rheumatoid factor level of 13 IU/mL (reference range: 0–18), suggesting that the musculoskeletal findings were not attributable to rheumatoid arthritis. Bone mineral density assessment revealed the following results: lumbar spine (L1–L4) T-score -2.4, proximal femur T-score -2.4, and distal radius T-score -2.5 (reference values: normal > -1.0; osteopenia -1.0 to -2.5; osteoporosis < -2.5), consistent with osteoporosis and significant bone mineral loss.

Given these findings, additional parameters were evaluated to assess bone-mineral metabolism. Laboratory results showed hypercalcemia at 12.9 mg/dL (reference range: 8.8–10.6), suggesting an underlying metabolic disturbance possibly related to a malignant process, and hyperphosphatemia at 5.8 mg/dL (reference range: 2.5–4.5), commonly observed in renal or neoplastic disorders. Serum magnesium was within normal limits at 2.2 mg/dL (reference range: 1.77–2.58). Parathyroid hormone (PTH) level was markedly de-

creased at 1.20 pg/mL (reference range: 12–88), indicating hypoparathyroidism or impaired PTH secretion. Vitamin D levels were deficient at 8.35 ng/mL (deficiency <20 ng/mL; insufficiency 21–29 ng/mL; sufficiency \geq 30 ng/mL), while calcitonin levels were normal at 8.9 pg/mL (reference range: 0–10).

In this clinical context, the combination of hypercalcemia, hyperphosphatemia, suppressed PTH levels, vitamin D deficiency, normal calcitonin, and radiological evidence of osteoporosis strongly suggested a bone–mineral metabolism disorder associated with an underlying malignancy. Severe immunosuppression, secondary to advanced HIV infection further increased the risk of skeletal complications in this patient. Consequently, an extensive diagnostic evaluation was initiated, including skeletal survey imaging and hematologic studies such as serum protein electrophoresis and urinary Bence Jones protein analysis, to further characterize the suspected neoplastic process.

Serum protein electrophoresis revealed the following fractions: albumin 56.2% (reference range: 54–65), alpha-1 globulins 3.3% (reference range: 2–4), alpha-2 globulins 7.4% (reference range: 6–9), beta globulins 11.2% (reference range: 8–14), and gamma globulins 22% (reference range: 10–20), indicating a significant elevation. A monoclonal spike of approximately 8.0 g/dL was identified in the gamma region, highly suggestive of a monoclonal gammopathy and consistent with multiple myeloma. Urinary analysis for Bence Jones proteins was positive, with kappa light chains measuring 100 mg/dL (reference range: 0–10), while lambda light chains were not detected. Protein immunofixation electrophoresis confirmed the presence of monoclonal kappa light chains, further supporting the diagnosis of multiple myeloma. Subsequently, a skeletal survey was carried out, the findings of which are described below (Figure 2 to 4).

Figure 2. Anteroposterior radiograph of the radius and ulna demonstrating multiple lytic lesions distributed along both bones (white arrows), characterized by decreased bone density. At the proximal epiphysis of the radius, an irregular bony protuberance is observed on the cortical surface (green arrows), compromising bone integrity and presenting as an area of reduced density consistent with a lytic lesion. The margins of this lesion are poorly defined, suggesting progressive bone destruction.



Figure 3. Anteroposterior radiograph of the tibia and fibula demonstrating multiple lytic lesions distributed along both bones with an overall reduction in bone density (white arrows). Loss of normal bone architecture is observed in the tibial diaphysis (red arrows). Additionally, prominent lytic lesions indicating bone destruction are evident in the proximal epiphysis (red circle).

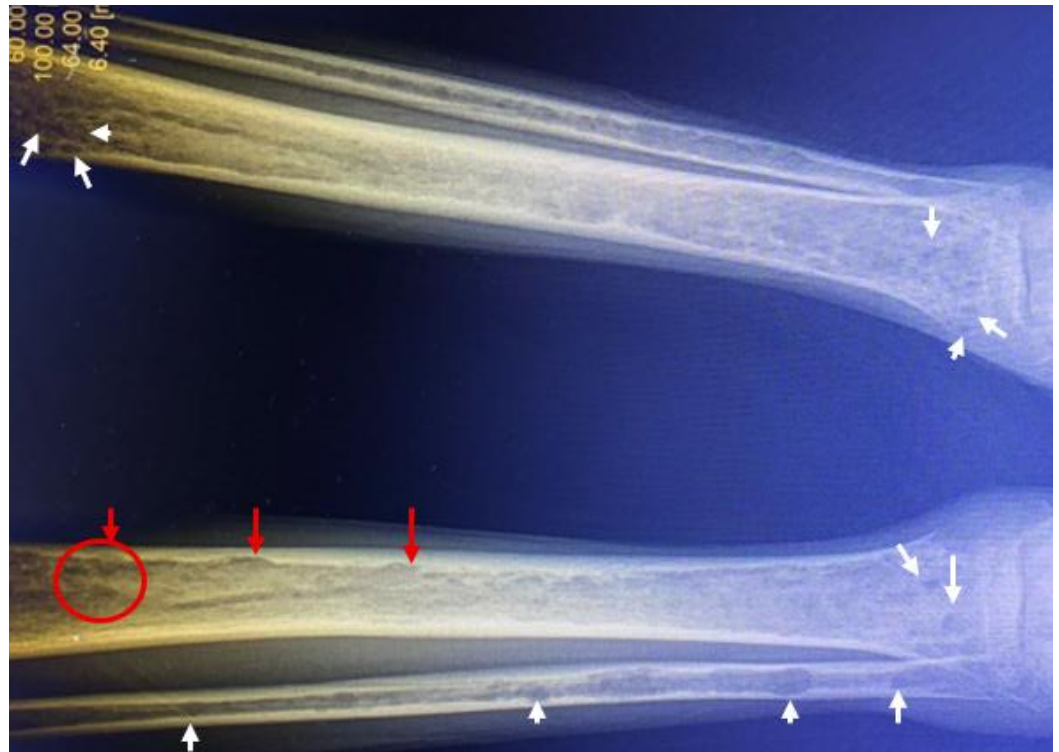


Figure 4. Posteroanterior skull radiograph demonstrating lytic lesions in the occipital region, characterized by areas of decreased bone density (white arrows). These lesions appear as radiolucent (dark) zones, suggesting underlying bone destruction.



It is important to note that during the course of hospitalization, follow-up laboratory tests were performed to monitor the patient's overall clinical status. These revealed a significant deterioration in renal function, with markedly elevated urea levels of 86.4 mg/dL

(reference range: 16.86–43.37) and creatinine of 2.35 mg/dL (reference range: 0.8–1.3), findings consistent with acute renal impairment, which may be related to multiple myeloma. Consequently, the therapeutic plan was adjusted according to the patient's clinical evolution. In addition, bisphosphonate therapy was initiated considering the documented disturbances in bone metabolism and the presence of hypercalcemia, particularly in the context of hypoparathyroidism and mineral imbalance.

This case illustrates a complex hematologic complication in a patient with advanced HIV/AIDS, in whom osteoarticular manifestations and metabolic study findings raise suspicion for multiple myeloma. It underscores the importance of early diagnosis, comprehensive evaluation, and appropriate management to prevent renal and skeletal complications and to optimize prognosis in the setting of severe immunosuppression.

4. Review

Human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS) is known to induce chronic immunosuppression, profoundly disrupting immune system function and increasing susceptibility to a wide range of malignancies, including multiple myeloma (MM). The association between these two conditions is supported by multiple studies suggesting that individuals living with HIV have a significantly increased risk of developing malignant hematologic disorders such as MM [8]. Chronic HIV-related immunosuppression, particularly in patients with poor adherence to antiretroviral therapy (ART), impairs immune surveillance mechanisms, thereby facilitating the proliferation of malignant plasma cells. Additionally, dysregulation of both B-cell and T-cell function in HIV-infected individuals contributes to uncontrolled plasma cell expansion and the subsequent development of MM [9].

In the present case, the patient was in the AIDS stage with a markedly reduced CD4+ T-cell count (79 cells/ μ L), indicating severe immunosuppression and an increased risk for hematologic malignancies. The CRAB criteria, hypercalcemia, renal insufficiency, anemia, and bone lesions, are essential for the diagnosis of MM. This patient fulfilled several of these criteria, including hypercalcemia (12.9 mg/dL), renal impairment with elevated creatinine levels, and multiple lytic bone lesions, all of which strongly support the diagnosis of multiple myeloma [9].

The clinical and laboratory findings observed in this patient are consistent with the CRAB criteria and represent classical manifestations of MM. The coexistence of hypercalcemia, renal dysfunction, anemia, and osteolytic lesions reinforces the diagnostic suspicion and aligns with previous reports emphasizing that such features should prompt consideration of MM in patients with advanced HIV infection and poor ART adherence. These findings further underscore the importance of including multiple myeloma in the differential diagnosis of immunocompromised patients presenting with systemic and osteoarticular symptoms.

Bone–mineral metabolism is primarily regulated by calcium, phosphorus, parathyroid hormone (PTH), and calcitonin. Under physiological conditions, calcium is essential for cellular function, muscle contraction, and coagulation, while phosphorus plays a critical role in bone mineralization. PTH promotes calcium release from bone into the bloodstream in response to hypocalcemia by stimulating bone resorption. However, in pathological states such as MM, excessive osteoclastic activity leads to increased bone resorption and subsequent hypercalcemia. In malignant hypercalcemia associated with MM, PTH secretion is typically suppressed despite elevated calcium levels. Calcitonin, in contrast, exerts opposing effects by inhibiting bone resorption and promoting calcium deposition in bone, thereby reducing serum calcium levels [10].

In this patient, the coexistence of hypercalcemia and hyperphosphatemia, along with suppressed PTH levels, suggests malignancy-related hypercalcemia, a condition in which parathyroid hormone secretion is physiologically inhibited in response to elevated calcium levels. This phenomenon is commonly observed in MM and reflects the inability of the parathyroid glands to appropriately regulate mineral metabolism in the presence of

excessive osteoclastic bone destruction. Furthermore, chronic HIV-related immunosuppression may exacerbate mineral metabolism abnormalities by impairing immune-mediated regulatory mechanisms involved in bone homeostasis [10].

The suppression of PTH provides a strong rationale for the use of bisphosphonates, which inhibit osteoclast activity and reduce bone resorption in patients with MM. Agents such as pamidronate and zoledronic acid are widely used to prevent skeletal-related events, reduce fracture risk, and improve bone stability in MM. In this case, bisphosphonate therapy was particularly important given the suppressed PTH levels, extensive osteolytic disease, and hypercalcemia, all of which increase the risk of progressive bone destruction. Chronic HIV-related immunosuppression further contributes to bone mineral imbalance and reduced bone density, highlighting the need for a comprehensive and multidisciplinary therapeutic approach [10]. Vitamin D deficiency, as observed in this patient, further contributes to disturbances in bone–mineral metabolism, as vitamin D is essential for intestinal calcium absorption and proper skeletal regulation. The presence of vitamin D deficiency, in combination with osteoporosis demonstrated on bone densitometry, underscores the deleterious impact of chronic immunosuppression on skeletal health in patients with HIV [11].

The diagnosis of MM in this case was confirmed through the integration of clinical, radiological, and laboratory findings. Serum protein electrophoresis demonstrated a monoclonal spike in the gamma fraction, consistent with monoclonal gammopathy and indicative of M-protein production by malignant plasma cells. Additionally, urinary Bence Jones protein analysis was positive for monoclonal kappa light chains, a hallmark finding in MM and a known contributor to myeloma-related nephropathy through tubular light chain deposition [12]. Following confirmation of the diagnosis, first-line systemic therapy was initiated using a standard triplet regimen consisting of a proteasome inhibitor (bortezomib), an immunomodulatory agent (lenalidomide), and a corticosteroid (dexamethasone). This combination is widely used in symptomatic MM to reduce tumor burden by targeting malignant plasma cells. Adjunctive therapy with bisphosphonates (zoledronic acid) was administered to mitigate bone destruction, alleviate bone pain associated with lytic lesions, and control hypercalcemia. Renal function was closely monitored throughout treatment to prevent and manage potential complications [12].

Poor adherence to ART represents a critical contributing factor in this case. Numerous studies have consistently demonstrated that suboptimal ART adherence is associated with inadequate viral suppression, progressive immune deterioration, and an increased burden of comorbidities, including hematologic and metabolic complications. Observational data in HIV-infected populations indicate that insufficient adherence may facilitate the development of severe comorbid conditions and malignancies by compromising long-term immune control. 11. In this patient, advanced HIV infection with an extremely low CD4+ count and a history of poor ART adherence significantly contributed to severe immunosuppression and increased susceptibility to MM. Close surveillance of such patients for early signs of malignancy is essential, as prolonged immunodeficiency may impair immune-mediated control of malignant cellular clones [9].

Finally, key factors in the diagnosis and management of multiple myeloma in the context of HIV/AIDS are closely interconnected. For example, chronic HIV-induced immunosuppression increases the risk of multiple myeloma by promoting immune dysregulation and the uncontrolled proliferation of malignant plasma cells. This pathophysiological interaction is often accompanied by disturbances in mineral metabolism; hypercalcemia and parathyroid hormone (PTH) abnormalities reinforce the association between multiple myeloma and bone–mineral disorders, contributing to reduced bone density and impaired renal function. Given these complexities, patient care should follow a comprehensive and multidisciplinary approach that prioritizes not only the treatment of multiple myeloma but also the management of renal and skeletal complications. Regulation of mineral metabolism, including the use of bisphosphonates to prevent fractures and limit bone destruction, is essential for improving clinical outcomes.

5. Discussion

Multiple studies have demonstrated that patients with advanced HIV/AIDS have an increased risk of developing hematologic malignancies, including multiple myeloma (MM). Chronic HIV-induced immunosuppression affects the immune system, particularly T lymphocytes, thereby facilitating the proliferation of malignant plasma cells. This process, combined with B-cell dysregulation, increases susceptibility to MM, especially in patients with poor adherence to antiretroviral therapy (ART) [11]. In the present case, the patient had a CD4+ T-cell count of 79 cells/ μ L, indicating severe immunosuppression and a high risk of opportunistic infections and hematologic malignancies such as MM [12]. Evidence suggests that advanced HIV infection creates a favorable environment for uncontrolled malignant plasma cell proliferation by disrupting immune homeostasis [9].

Regarding aesthetic history, the patient had previously undergone multiple procedures involving biopolymer injections. Although a causal relationship between biopolymers and multiple myeloma has not been definitively established, some studies suggest that chronic inflammation induced by these materials may alter immune function, potentially promoting mutations and proliferation of malignant plasma cells. However, there is currently no conclusive evidence to support this hypothesis, and further research is required. In this case, advanced HIV infection remains the primary contributing factor to the progression toward multiple myeloma [11].

Clinical and laboratory findings fulfilled the CRAB criteria, which are fundamental for the diagnosis of MM. The patient presented with hypercalcemia (12.9 mg/dL), renal insufficiency (elevated creatinine at 2.35 mg/dL and urea at 86.4 mg/dL), significant anemia (hemoglobin 9.7 g/dL), and multiple osteolytic lesions identified on radiographic imaging. The presence of these criteria strongly supports the diagnosis of multiple myeloma. Serum protein electrophoresis revealed a monoclonal spike in the gamma globulin fraction (22%), indicating the presence of monoclonal protein (M-protein), a hallmark of MM. This finding is consistent with a monoclonal gammopathy, a key diagnostic marker of multiple myeloma. Urinary Bence Jones protein analysis was positive, with kappa light chain levels of 100 mg/dL, a crucial finding for confirming MM, as these proteins are typically excreted by the kidneys in affected patients [12].

Significant alterations in mineral metabolism were observed, further supporting the diagnosis of MM. Elevated phosphorus levels (5.8 mg/dL) and hypercalcemia (12.9 mg/dL) indicated a mineral imbalance commonly seen in MM due to malignant plasma cell-mediated bone resorption. Hypercalcemia is a frequent complication of MM, as osteolytic activity leads to increased calcium release into the bloodstream. Suppressed parathyroid hormone (PTH) levels (1.20 pg/mL) were consistent with primary malignant hypercalcemia, representing a physiological response in which elevated calcium levels inhibit parathyroid hormone secretion. This disturbance in calcium homeostasis is a key component of MM pathophysiology and may be exacerbated by renal impairment, which is common in MM due to the deposition of monoclonal proteins in the kidneys. Management included calcium and vitamin D supplementation, which are essential for improving intestinal calcium absorption, restoring mineral balance, and normalizing serum levels. This approach reduces fracture risk and improves bone mineralization and is widely accepted in clinical practice [10-12].

Treatment with bisphosphonates resulted in significant improvement in bone resorption parameters four weeks after initiation, with a marked reduction in bone-related symptoms such as pain and limb edema. Serum calcium levels stabilized within the normal range (9.5 mg/dL), and follow-up bone densitometry demonstrated a mild increase in T-score at the lumbar spine and proximal hip, indicating improved bone density [10-12]. Following initiation of therapy with bortezomib, lenalidomide, and dexamethasone, the patient exhibited significant clinical improvement. Bone symptoms improved substantially, with reduced pain and inflammation in the extremities, leading to enhanced functional capacity. Hypercalcemia, which initially reached 12.9 mg/dL, was effectively controlled, with serum calcium levels stabilizing within normal limits after four weeks of

treatment. Renal function remained stable, with no further deterioration in creatinine or urea levels, reflecting appropriate management of metabolic complications. Follow-up serum protein electrophoresis showed a reduction in the monoclonal gamma fraction peak, indicating a favorable therapeutic response. Hematologic parameters also improved, with gradual normalization of hemoglobin levels and resolution of thrombocytosis. These findings suggest a positive response to initial therapy and underscore the importance of a multidisciplinary approach to the comprehensive management of this complex disease. Close follow-up is essential to monitor monoclonal protein levels, mineral metabolism, and renal function, as well as to optimize antiretroviral therapy adherence and control viral replication [12].

6. Conclusion

The present clinical case highlights the complexity of managing a young patient with advanced HIV infection and multiple myeloma (MM). Severe immunosuppression, reflected by a markedly low CD4+ T-cell count (79 cells/ μ L), represents a critical risk factor for the development of hematologic malignancies, particularly multiple myeloma. HIV-mediated immune dysfunction facilitates uncontrolled malignant plasma cell proliferation, thereby contributing to disease progression. The patient fulfilled the CRAB criteria (hypercalcemia, renal insufficiency, anemia, and bone lesions), which are essential for the diagnosis of multiple myeloma. Clinical findings, together with laboratory results demonstrating hypercalcemia (12.9 mg/dL), renal dysfunction (creatinine 2.35 mg/dL, urea 86.4 mg/dL), significant anemia (hemoglobin 9.7 g/dL), and multiple osteolytic bone lesions, strongly support the diagnostic suspicion of MM.

Regarding bone and mineral metabolism, the observed hypercalcemia is consistent with malignant plasma cell-mediated bone resorption in multiple myeloma. Suppressed parathyroid hormone (PTH) levels (1.20 pg/mL) represent an appropriate physiological response to primary malignant hypercalcemia, distinguishing this condition from secondary hypoparathyroidism typically observed in chronic kidney disease, thereby clarifying the initial pathophysiological interpretation. Vitamin D deficiency also appears to contribute to altered mineral metabolism and impaired bone health, which are common features in MM due to disrupted calcium homeostasis. Although a potential association between the use of biopolymers in aesthetic procedures and multiple myeloma has been hypothesized, this relationship should be interpreted with caution due to the lack of histopathological evidence and the presence of a far more significant triggering factor, namely advanced HIV infection. Further studies are required to explore this possible association.

Multidisciplinary management is essential in such cases, encompassing both treatment of the hematologic malignancy and management of associated metabolic and renal complications. Bisphosphonate therapy has proven effective in improving bone resorption parameters, stabilizing serum calcium levels, and enhancing bone mineral density. In addition, combination chemotherapy with bortezomib, lenalidomide, and dexamethasone resulted in a favorable clinical response, with significant improvement in bone-related symptoms, renal function, and hematologic parameters.

This case underscores the importance of early diagnosis and comprehensive management in patients with advanced HIV infection and multiple myeloma, emphasizing the need for a therapeutic approach that addresses associated comorbidities and optimizes clinical outcomes. Close monitoring of hematologic, renal, and metabolic parameters is crucial to guide treatment adjustments and achieve long-term disease control.

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