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Multiple Myeloma Presenting with Autoimmune Disease in Low-Resource Settings: A Diagnostic Challenge

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ABSTRACT

This paper presents a clinically significant case of multiple myeloma (MM) coexisting with autoimmune disease, highlighting the diagnostic and therapeutic challenges encountered in low-resource settings. Multiple myeloma is a malignant plasma cell disorder characterized by clonal proliferation within the bone marrow and associated end-organ damage, while autoimmune diseases such as rheumatoid arthritis (RA) involve chronic immune dysregulation and inflammation. The overlap in clinical features between these conditions particularly anemia, bone pain, and elevated inflammatory markers, can obscure diagnosis and delay appropriate management. The report centers on a 58-year-old patient initially managed for autoimmune arthritis based on clinical and laboratory findings, including elevated inflammatory markers and positive autoimmune serology. Despite initial improvement with immunosuppressive therapy, the patient later developed additional symptoms such as fatigue, renal abnormalities, and bone pain. Further investigations revealed hallmark features of MM, including lytic bone lesions, Bence Jones proteinuria, and monoclonal gammopathy confirmed by serum protein electrophoresis and bone marrow biopsy. The patient was subsequently treated with chemotherapy, resulting in clinical improvement. This case underscores the complex interplay between chronic immune activation and malignant transformation, supporting the hypothesis that prolonged antigenic stimulation in autoimmune diseases may predispose to plasma cell dyscrasias. It also highlights the importance of maintaining a high index of suspicion when patients with autoimmune conditions present with atypical or progressive symptoms. Early recognition and multidisciplinary collaboration are critical for improving outcomes. The study emphasizes the need for better diagnostic access, clinician awareness, and healthcare support systems, particularly in resource-limited environments.

Keywords

Anemia, Bone pain, Multiple Myeloma, Autoimmune disease, Low-resource settings.

Introduction

Multiple myeloma, also known as Kahler's disease is a clonal plasma cell malignancy characterized by the neoplastic proliferation and accumulation of terminally differentiated B cells within the bone marrow, resulting in the mass production of structurally and functionally homogenous monoclonal immunoglobulin [1]. Multiple myeloma expresses by altering the body's normal design of hematopoiesis, bone marrow microenvironment, and drives

end organ damage typically manifesting as hypercalcemia, renal impairment, anemia and Osteolytic bone disease [1]. It is the second most common hematologic malignancy following non-Hodgkin's lymphoma (NHL) [2].

Multiple myeloma accounts for 0.9% of all cancer diagnosis worldwide and about 588,161 cases are reported each year [3-5]. In the United States, it accounts for 1.8% of all new cases and 18% of all hematologic malignancies [6]. It is slightly more common in men than in women with incidence rate of 1.5:1 [7]. It is often diagnosed among the geriatric population, aged 65-74 years and about twice as common among Blacks/African Americans than in Whites [8]. However, there is paucity of data on multiple myeloma in Africa. A retrospective study conducted by Clement et al in Uganda demonstrated that the median age of clinical presentation in the black patients was 52 years which is about 10 years younger than the median age for the Whites [9]. In Nigeria, it is estimated to account for 8.2% of all hematological malignancies, also with a slight male predilection than female [10].

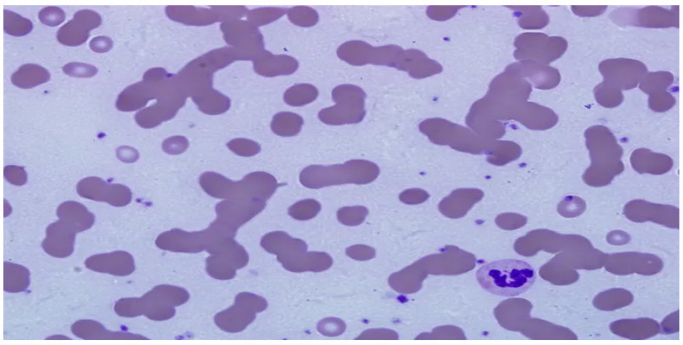
Multiple myeloma typically develops from monoclonal gammopathy of undetermined significance (MGUS), an asymptomatic condition in which monoclonal immunoglobulins are present without organ damage. MGUS, found in 3% of people older than 50 years usually arises from post-germinal center plasma cells and carries about 1% yearly risk of progressing to MM. Although, the exact causes are unclear, genetic changes that enhance plasma cell survival and proliferation along with additional "second hit" cytogenetic abnormalities are thought to drive the transition from MGUS to myeloma [11].

At the molecular level, early oncogenic events frequently involve immunoglobulin heavy chain (IgH) translocations at chromosome 14q32, juxtaposing oncogenes such as CCND1, FGFR3, or MAF to the potent IgH enhancer. These early lesions equip with a survival and proliferative advantage but are insufficient alone for complete malignant transformation [12]. Multiple myeloma progression is enhanced by mutations in signaling pathways such as the RAS/RAF/MEK/ERK pathways, dysregulation of MYC, alteration in DNA repair mechanisms, and epigentic reprogramming. High-risk cytogenetic abnormalities such as del(17p) involving TP53 or t(4;14) further promote genomic instability and clinical behavior [12].

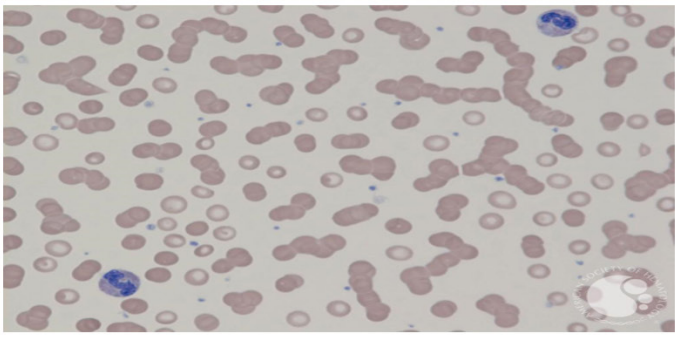
Monoclonal and Polyclonal gammopathy are characterized by elevated immunoglobulins but each has different biological processes. In monoclonal gammopathy, a single B cell undergoes transformation and begins to proliferate abnormally, producing large quantities of a single, identical immunoglobulin often referred to as M protein or paraprotein. Because the antibodies produced are structurally identical, they appear as a sharp well-defined "M-spike" on serum protein electrophoresis usually in the gamma region often signaling an underlying neoplastic or pre-neoplastic process. Contrastingly, Polyclonal gammopathy reflects a physiologic process instead of a malignant one. Here, multiple clones of B-cells are stimulated by chronic infection, inflammation or autoimmune activity resulting in the production of diverse kinds of immunoglobulins. On electrophoresis, it appears broad based with diffuse elevation in the gamma region rather than a sharp spike, indicating diversity in antibody production [13]. Unlike monoclonal gammopathy, the antibodies produced are varied and often functional, representing an ongoing immune response. Thus, instead of pinpointing a neoplasm, polyclonal gammopathy serves as a marker of chronic immune stimulation [13].

The symptoms of MM are oftentimes nonspecific and could mimic a variety of other conditions such as Waldenstrom macroglobulinemia, Amyloidosis, metastatic bone disease, and autoimmune diseases. Patients could present with bone pain and fatigue, anemia, elevated creatinine level, hypercalcemia and bone abnormalities (primary lytic lesions, osteoporosis, and fractures), repeated infections, neuropathy, bleeding and unexplainable weight loss [14]. There is the typical CRAB manifestation of multiple myeloma which stands for hypercalcemia, renal impairment, anemia and lytic bone lesions [15]. Studies have shown that early manifestations of multiple myeloma can be attributed to pre-existing conditions such as renal impairments, diabetes mellitus and mild hepatic disease which can further delay the diagnosis [15].

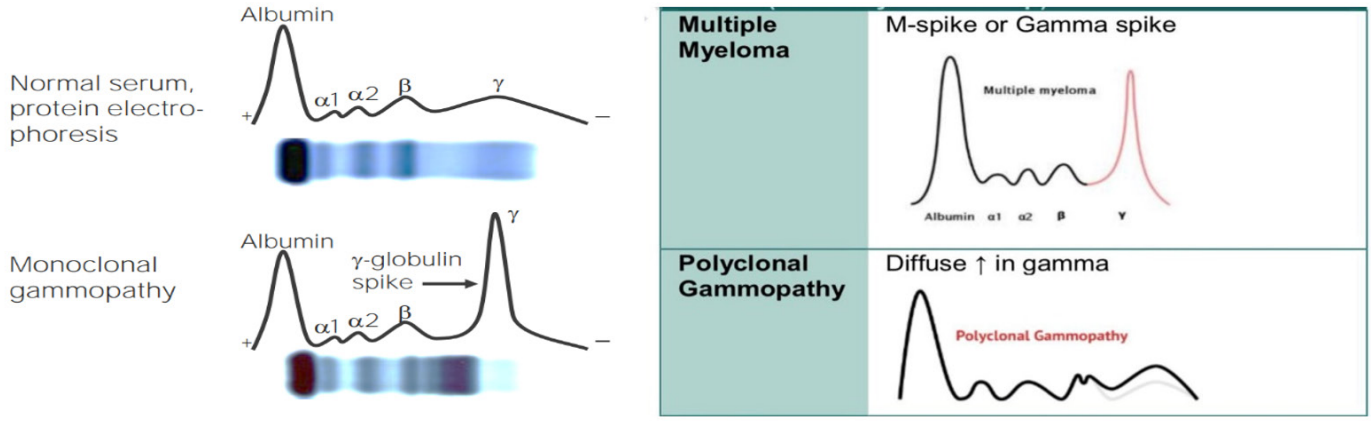
Multiple myeloma is part of a spectrum of plasma cell proliferative disorders and thus, the correct identification and diagnosis of MM is imperative. According to the World Health Organization guidelines in line with International Myeloma Working Group criteria, the diagnosis of Multiple Myeloma requires a combination of pathological and clinical findings rather than a single definitive test. Essentially, there must be clear evidence of a clonal plasma



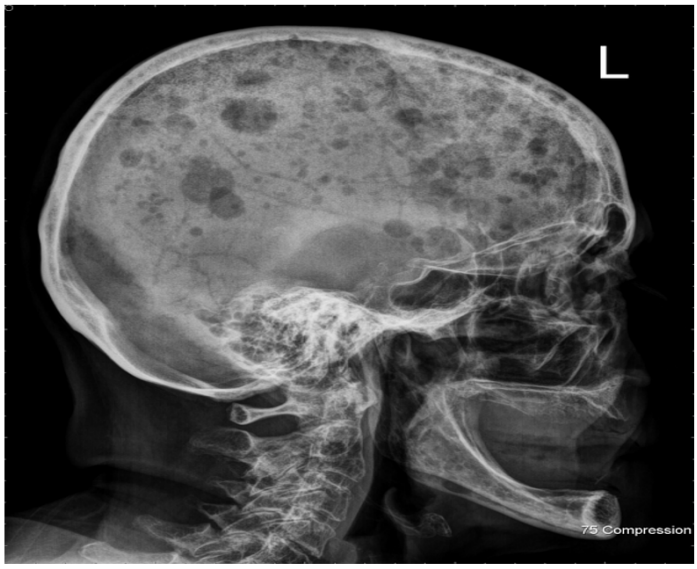
Rouleaux formation in MM (webpathology.com)



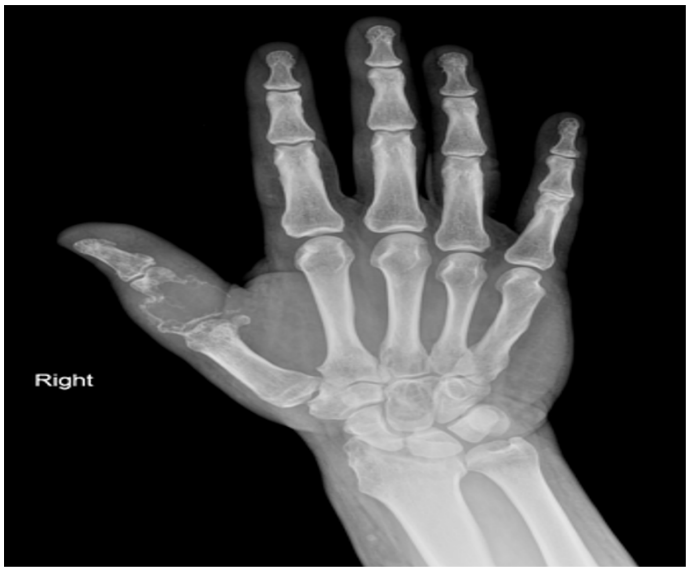
Clumping cells in MM (webpathology.com)



Serum Protein Electrophoresis (Sciencedirect.com)



Lytic lesion on a skull Radiograph (Med.estrategia.com)



Lytic lesion on a limb radiograph

cell population of > or =10% plasma cells in the bone marrow or a confirmed plasmacytoma in conjunction with at least one of the hallmarks of active disease. These features include the well-known CRAB manifestations (hypercalcemia, renal dysfunction, anemia and bone lesions) or specific high-risk biomarkers such as markedly elevated plasma burden or characteristic focal lesion on MRI. Deploying this diagnostic approach helps in early recognition of the disease before irreversible organ damage develops. To establish a multiple myeloma diagnosis the following investigations should be carried out; complete blood count with differentials and platelet count, ESR, BUN, creatinine, electrolytes, albumin calcium levels, serum LDH and beta 2 microglobulin, serum immunoglobulins, serum protein electrophoresis (SPEP), serum immunofixation electrophoresis (SIFE), 24-hour proteinuria, urine protein electrophoresis (UPEP), urine immunofixation electrophoresis (UIFE), serum-free light chain (FLC) assay [11].

Multiple myeloma has previously been diagnosed with greater than or equal to 10% of clonal bone marrow plasma cells on bone marrow biopsy or if a biopsy proven plasmacytoma was proven in addition to at least one of the following CRAB criteria; serum calcium level greater than 0.25mmol/L higher than upper limit of normal or above 2.75mmol/L, renal insufficiency with creatinine above 2mg/dL or creatinine clearance less than 40mL/min, anemia with hemoglobin less than 10g/dL and one or more osteolytic bone lesions on skeletal radiography.

The goals of treatment are to reduce symptoms, slow disease progression, provide long periods of remission and lengthen survival while preserving quality of life. Treatment options include chemotherapy, autologous stem cell transplantation (ASCT) and radiation therapy. The treatment plan depends on whether a patient is a high risk or standard risk patient. The induction regimen for high-risk patients include four cycles of daratumumab, bortezomib, lenalidomide and dexamethasone followed by an early ASCT for transplant eligible patients and a proteasome inhibitor-based maintenance therapy should be initiated after. The induction for standard risk is typically same with the exception of daratumumab.

The maintenance following the ASCT is with lenalidomide [11].

Rheumatoid arthritis is a systemic, chronic, symmetrical autoimmune disease that affects the joints [15]. It typically starts by affecting the small joints, progresses to the larger joints and ultimately, there is destruction of the bones and cartilages of joins with weakness of the tendons and ligaments [16]. It could affect any joint with a synovial membrane lining and there is the chance of serious affectation of organs such as the heart, skin, lungs and eyes [17].

Rheumatoid arthritis has an estimated global prevalence of 1% and is one of the leading causes of chronic morbidity in industrialized nations [18,19]. While there is limited study to represent Africa, the crude prevalence was estimated to be 0.36% [20]. RA is underreported in Nigeria. In a retrospective study, covering a period of about 8 years in a rheumatologic clinic, it was discovered that of a total of 1,623 patients who presented with complaints, 12.3% had RA with a higher female prevalence, 2.4:1 and mean age of 46.9 years [21].

The exact etiology of RA is unknown but it has been largely linked to an interplay between genetic susceptibility and environmental factors [22]. Female gender, family history of RA and HLA genotype are all linked to genetic factors [23].

Rheumatoid arthritis (RA) develops through a breakdown of immune tolerance, leading to the activation of autoreactive T cells. Regulatory T cells (Tregs), which normally suppress autoimmune responses, become dysfunctional in RA. Pro-inflammatory T cell subsets such as Th17 and Th1 cells expand and produce key cytokines (IL-17, IFNγ, GM-CSF) that drive synovial inflammation [24]. IL-17 amplifies synovial inflammation by inducing additional inflammatory mediators and promoting osteoclast differentiation, thereby accelerating bone erosion. A central role in RA pathology is played by fibroblast-like synoviocytes (FLS) which proliferates excessively when activated [25].

Overall, RA arises from the interaction of genetic predisposition and environmental triggers, leading to loss of self-tolerance, chronic joint infiltration by autoreactive immune cells, hyperplastic and invasive synovium, and persistent cytokine-driven inflammation and destruction [26].

RA typically affects more than one joint and small joints are usually the first to be affected. The affectation is symmetrical. It presents as joint pain, tenderness, swelling or stiffness that lasts for 6 weeks or more. Patients with RA also presents with malaise, fatigue, low grade fever, weight loss and flare. The extra-articular manifestations include dryness, pain, redness and increased sensitivity of the eyes to light, dryness of the mouth, gum inflammation or infection, rheumatoid nodules, scarring of the lungs, anemia, vasculitis and visceral involvement [27].

There is no single diagnostic test for RA. The diagnosis is made through the combination of clinical symptoms, physical examinations, laboratory investigations and imaging. The blood tests to be done include Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP), Rheumatoid Factor (RF), Anti Citrullinated Protein Antibody (ACPA) also called Anti Cyclic Citrullinated Protein (CCP) and Full Blood Count (FBC) to exclude other conditions. Imaging such as X-rays are also important [28].

The goals of treatment of RA are to reduce joint inflammation and pain, maximize joint function and prevent joint destruction and deformity. The treatment is multidisciplinary which involves use of pharmacological agents, weight bearing exercises and counselling [29]. The first line management of RA involves use of Non-Steroidal Anti-Inflammatory Drugs (NSAIDS) such as acetylsalicylate, naproxen, ibuprofen, among others and corticosteroids. Second line management involve the use of Disease Modifying Antirheumatic Drugs (DMARDs), methotrexate and hydroxychloroquine. Newer medications include leflunomide, infliximab, golimumab, anakinra. Tocilizumab is a biologic agent which inhibits IL-6 and can be used for people who have not been effectively treated with DMARDs [30]. Joint surgery such as arthroplasty is the last resort for the treatment of RA to relieve pain and restore the joint function [30].

Case Presentation

A 58-year old Clergy who resides in Ondo state presented to the hematology clinic, UNIMEDTHC, on referral from the orthopedic clinic on account of bilateral shoulder pain of 10 months duration, patient acknowledged having a genotype of AS and had an X-ray report which showed rotator cuff syndrome. Patient's claim of being an AS raised the suspicion of the Orthopedic management team that patient might be SS since there was no record of previous investigations to substantiate his claims and thus he was referred to the hematology clinic. He had first of all presented to the General Outpatient Department (GOPD) with the same complaint of bilateral shoulder pain and was referred to the Orthopedic clinic after being managed by the out-patient department team for Osteoarthritis with no visible improvements. He was asked to carry out some investigations (FBC, Anti-CCP, ESR, FBS, PBF Hb1Ac, C-reactive protein, HPLC {High performance liquid chromatography}) and to report back to the hematology clinic in 2 weeks. Patient was also instructed to continue the use of previously administered drugs which are prednisolone, arthrotec, cocodamol, vitamin C, ferrous II and amilodipine because patient is a known hypertensive.

On the next appointed clinic visit, patient came back with a PCV of 26.5%, Hb – 8.60, WBC – 6,400, Lymphocytes – 44.50, Neutrophils – 49.50, Eosinophils – 1.00, Basophils – 1.00, Platelets – 179,000, RBC – 3.63, MCV – 73.10, MCH – 73.60 MCHC – 31.30, PBF – Anisopoikilocytosis, Hypochromic, microcytic, polychromasia, clumping RBC, Pencil cells, Target cells, lymphocytosis, all these revealed Iron deficiency anaemia ? autoimmune, Anti-CCP – 21 IU/ml, ESR – 89mm in 1hr, FBS – 6.0, HPLC – AC, C-reactive protein - >200 mg/l. These results are consistent with an auto-

immune disorder associated with arthritis and patient was managed accordingly (Steroids were continued, analgesics, metformin, hydroxychloroquine and low dose cyclophosphamide were also used) and patient improved significantly. However, to confirm the working diagnosis, patient was requested to carry out Anti-nuclear antibody test which eventually came out positive (1:80).

After about 3 months on the 4th clinic visit, patient reported again to the hematology clinic with complaints of sudden recurrent fatigability, palpitations, episodes of vomiting, nausea, recurrent yellowing of eyes, back pain, nocturia, excessive frothiness of urine and frequent urination. Following physical examination, the patient was found to be pale, had hepatomegaly, renal angle tenderness and bilateral leg swelling and his blood pressure reading was 120/80mmHg. Patient was admitted and the following investigations were carried out; FBC, ESR, Hb1Ac, FBS + 2 HPP, Lumbosacral X-ray, Serology (HBV, HCV, Retroviral), Abdominopelvic scan, LFT + GGT, EuCr, Coomb's test. FBC results showed anaemia, ESR - >130mm/hr, FBS – 13.20, 2 HPP – 18.5, Serology – HBV positive, HCV and retroviral negative, LFT + GGT – Elevated transaminase enzymes, Total protein – 6.6g/dl, Albumin – 3.0g/dl, Globulin – 3.6g/dl, Other electrolytes are normal except calcium, Calcium – 2.02mg/dl, Abdominopelvic scan – BPH with obstructive uropathy, Coomb's test – Positive, Lumbosacral + chest x-ray – Shows punched out lytic lesions, Bence jones protein – Positive. All these are consistent with a Multiple myeloma diagnosis and so led to confirmatory investigations of bone marrow which shows elevated plasma cells, protein electrophoresis which shows 'M' spike in the gamma region.

Patient was transfused with 3 units of red-packed cells, administered broad spectrum antibiotics (IV clindamycin and levofloxacin), Duodat for BPH, Metformin for the sugar level, Micronac XR and patient was stabilized. Afterwards, chemotherapy was commenced with the CDTZ regimen (Cyclophosphamide, Dexamethasone, Thalidomide and Zoledronic acid).

Patient was on the ward for 2 weeks and was discharged with a PCV of 30.9%. The PCV consistently remained at this level despite having received about 3 sets of chemotherapy session after discharge, a testament of favorable response to treatment and drastic improvements. However, patient complained of mild pains around the ribs, wrist joint and knee joint some days after the chemotherapy session and the attending physician requested for a repeat of the Anti-nuclear antibody test which showed 1:80, which confirmed autoimmune disorder. Patient was then placed on previously recommended drugs for autoimmune disorder from day 6 to day 28.

Patient as at the last clinic has received 15 cycles of chemotherapy and is currently doing well and carrying out his daily activities without any setbacks. Patient current investigations show; PCV - 33%, FBS – 5.9, Hb1Ac – 7.1, PSA – 1.2, EuCr – within normal range, LFT – within normal range.

Discussion

The association between multiple myeloma (MM) and autoimmune diseases has been a subject of scientific interest for several decades, with early observations dating back to the 1960s [29]. The mechanism of action is primarily anchored in the "Chronic antigenic stimulation" hypothesis. Several autoimmune disorders, including systemic lupus erythematosus, Sjögren's syndrome, ankylosing spondylitis, Rheumatoid arthritis and various autoimmune hematological conditions, have been investigated for potential associations with MM [29,30]. These conditions share common features such as chronic inflammation, persistent immune activation, and altered B-cell function, all of which may contribute to plasma cell transformation.

Rheumatoid arthritis (RA), in particular, has been examined extensively in relation to MM and other plasma cell dyscrasias. In RA, the immune system exists in a state of persistent, high intensity activation, providing a fertile ground for plasma cell dyscrasias. RA is characterized by the continuous activation of B-cells and plasma cells to produce autoantibodies like Rheumatoid Factor and ACPA. This chronic proliferative pressure increases the statistical likelihood of stochastic genetic mutations such as t (11;14) or t (4;14) translocations leading to the emergence of a malignance clone [31].

At the molecular level, the interleukin-6 (IL-6) axis serves as a pivotal bridge between these two pathologies. In RA, IL-6 is a dominant driver of synovial inflammation and systemic bone resorption [32]. However, in the context of MM, the same cytokine acts as the primary growth and survival factor for malignant plasma cells. The systemic elevation of IL-6 in RA patients may effective acts as an extrinsic factor that promotes the transition from Monoclonal Gammopathy of Undetermined Significance (MGUS) to overt MM, suggesting that the microenvironment of a chronic autoimmune patient is uniquely primed for plasma cell survival [33].

Epidemiological data regarding this link has historically been complex and occasionally inconsistent. While many case reports and cohort studies describe the overall association as weak [33,34], epidemiological evidence indicates that individuals with RA may have a modestly increased risk of developing MM [35]. This observation supports the hypothesis that prolonged antigenic stimulation and sustained inflammatory activity in RA could promote clonal plasma cell expansion and malignant transformation [35]. Also, because RA patients undergo frequent blood monitoring and inflammatory marker checks, they are statistically more likely to have incidental paraproteins detected via serum protein electrophoresis (SPEP). This often results in earlier diagnosis of plasma cell disorders which can skew perceived correlation between the two diseases. Additionally, long-term immunosuppressive therapies used in RA management may further influence immune surveillance. Together, these findings suggest a biologically plausible link between chronic autoimmune activity and MM development, warranting further longitudinal and

mechanistic studies [31-36].

Conclusion

The coexistence of multiple myeloma and rheumatoid arthritis illustrates the complex interplay between chronic immune activation and malignant transformation. Rheumatoid arthritis provides a biologically permissive environment characterized by sustained antigenic stimulation, cytokine driven B-cell proliferation and increased cellular turnover. Within this milleu, the accumulation of stochastic genetic alterations may facilitate progression from premalignant state of monoclonal gammopathy to overt myeloma.

The association of this disease is clinically significant due to overlapping symptomatology particularly bone pain, anemia and elevated inflammatory markers which may obscure the diagnosis of Multiple myeloma. Also, the contribution of immunosuppressive therapies to malignancy risks remains inconclusive, emphasizing the need to distinguish disease-related mechanisms from treatment effects.

Finally, the coexistence of these conditions underscores the importance of maintaining a high index of suspicion in patients with longstanding or chronic rheumatoid arthritis who develop atypical clinical features. Early recognition is critical, as timely diagnosis of MM significantly influences prognosis and therapeutic outcomes.

Recommendations

- Clinicians managing rheumatoid arthritis should maintain a high index of suspicion for potential plasma cell dyscrasias. Persistent expression of atypical features such as non-articular bone pain, renal dysfunction, anemia and disproportionate elevation in ESR should prompt evaluation for multiple myeloma
- Given the overlap in musculoskeletal and systemic manifestations, clinicians should differentiate inflammatory joint diseases from bone lesions and utilize the appropriate imaging modalities (preferably MRI) when the suspicion arises
- Collaboration between rheumatologists, hematologists and pathologists is essential to ensure accurate diagnosis, optimize treatment strategy and balance immunosuppression with oncologic safety.
- The National Health Insurance Scheme should broaden its benefit package to include; comprehensive cancer care, long term management of autoimmune diseases and coverage for essential laboratory investigations such as Imaging and serum electrophoresis
- Government and stakeholders should establish special interventions funds for cancer care and introduce subsidies for high cost treatments.

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