Original Research

THE SILENT BONE THIEF: A 60-YEAR-OLD MAN'S JOURNEY FROM CHRONIC BACKACHE TO A DIAGNOSIS OF MULTIPLE MYELOMA

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ABSTRACT
Multiple myeloma (MM) is a malignant plasma cell neoplasm that remains notoriously difficult to diagnose at its earliest stages, often masquerading as benign musculoskeletal or haematological conditions. We report the case of a 60-year-old male shopkeeper who presented with a six-month history of insidious back pain and progressive generalised weakness. A systematic workup revealed markedly elevated total protein with grossly inverted albumin-to-globulin ratio, normocytic normochromic anaemia, renal impairment, elevated inflammatory markers, and on serum protein electrophoresis — an M-band (3.9 g/dL) in the gamma globulin region with elevated kappa light chains. Peripheral blood smear showed normocytic normochromic red cells. Bone marrow aspirate imprint smear demonstrated numerous plasma cells, flame cells, binucleated forms and plasmablasts. Bone marrow trephine biopsy confirmed plasma cell neoplasm — multiple myeloma. Radiological imaging demonstrated extensive lytic bone lesions and vertebral osteoporosis. The case illustrates the diagnostic odyssey inherent to MM and underscores the critical importance of a thorough workup in an elderly male presenting with back pain and anaemia.

Keywords: Multiple Myeloma, Plasma Cell Neoplasm, Peripheral Blood Smear, Bone Marrow Biopsy, Lytic Lesions, M-Band, Kappa Light Chain, Bortezomib.

1. INTRODUCTION
Multiple myeloma (MM) is a clonal malignancy of terminally differentiated plasma cells, accounting for approximately 10% of all haematological malignancies and around 1–2% of all cancer deaths worldwide. It predominantly afflicts adults over the age of 60, with a slight male preponderance. The disease is characterised by the uncontrolled proliferation of neoplastic plasma cells within the bone marrow, leading to the production of a monoclonal immunoglobulin protein (M-protein) and the consequent devastation of bone, renal function, and immune homeostasis.
The classical clinical hallmarks of MM are encapsulated by the mnemonic **CRAB**: hyperCalcaemia, Renal insufficiency, Anaemia, and Bone lesions. However, the insidious onset of symptoms particularly chronic backache frequently leads to delayed diagnosis, with patients often attributed a diagnosis of degenerative disc disease, osteoporosis, or musculoskeletal strain for months to years before the underlying malignancy is unmasked.
We present the case of a 60-year-old male who navigated this diagnostic labyrinth for six months before a meticulous clinical and laboratory evaluation including peripheral blood smear, bone marrow aspiration and biopsy, serum protein electrophoresis with kappa light chain quantification, and MRI spine led to the definitive diagnosis of multiple myeloma.

2. CASE PRESENTATION
2.1 Patient Demographics and Chief Complaints
Mr. X, a 60-year-old male shopkeeper of moderate build and nutrition (height 161 cm, weight 50 kg, BMI 19.3 kg/m²), presented to the Department of General Medicine with worsening back pain for 15 days, generalised weakness for 10 days, and poor appetite for 10 days.
2.2 History of Presenting Illness
The patient's history revealed that his back pain had been a quiet, persistent companion for the preceding six months. Initially mild, managed with analgesics

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prescribed at a local facility with reportedly unremarkable investigations, the pain had intensified dramatically over the preceding fortnight. It was dull and aching in character, non-radiating, aggravated by activity, and worsened nocturnally — a pattern that, in retrospect, should have raised suspicion for an underlying neoplastic aetiology. Concomitant generalised weakness had also progressed over six months. He denied weight loss, trauma, fever, respiratory symptoms, urinary complaints, or neurological symptoms.

2.3 Physical Examination

On examination, the patient was conscious, coherent, cooperative, and oriented. Pallor was present. Examination of the oral cavity revealed a bald (atrophic) tongue with pale pink discolouration. Pulse was 105 bpm (tachycardia), BP 150/80 mmHg, RR 16/min, temperature 98.4°F, SpO_2 98% on room air. Musculoskeletal examination showed thoracic kyphosis and scoliosis, with bony tenderness over the lumbar spine. A systolic murmur was noted in the pulmonary area, consistent with a high-output flow murmur due to profound anaemia.

3. INVESTIGATIONS

3.1 Peripheral Blood Smear

Complete blood count on admission revealed severe normocytic normochromic anaemia: haemoglobin 6.6 g/dL (nadir 5.0 g/dL), RBC 2.17 × 10^6/µL, MCV 90 fL. Peripheral blood smear was reported on 27/02/2026. Red blood cells showed normocytic normochromic morphology with no haemoparasites or RBC inclusions. WBC was normal in total count, distribution, and morphology. Platelets were adequate in number, seen in singles and occasional clumps. The impression confirmed normocytic normochromic anaemia.

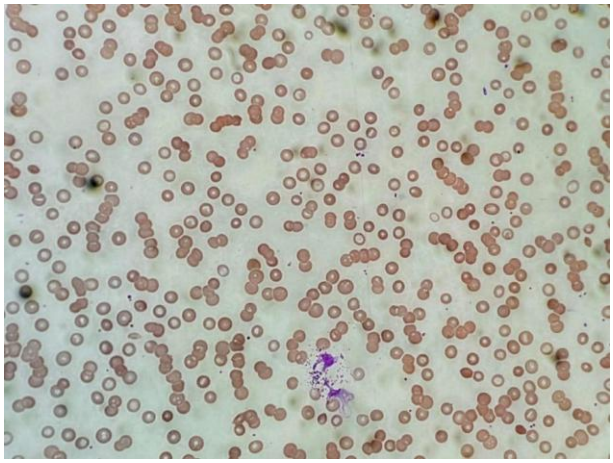

Figure 1A: PBS Microscopy — Normocytic normochromic RBCs with normal WBC morphology (Leishman stain, ×400)

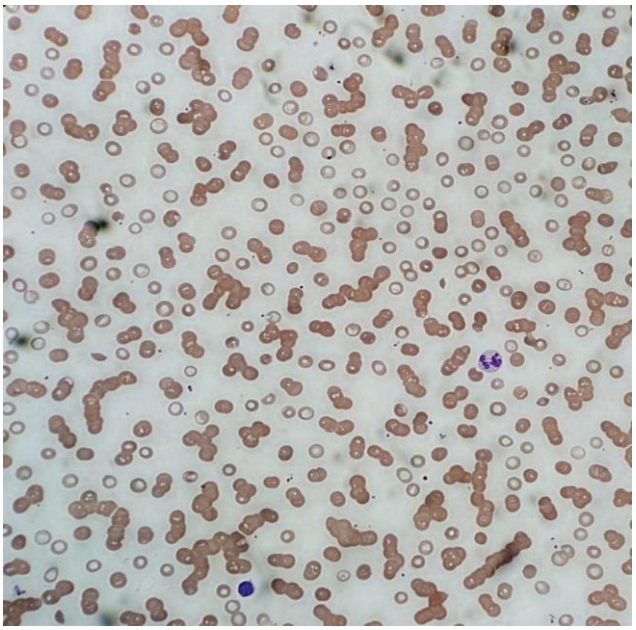

Figure 1B: PBS Microscopy — RBC rouleaux formation and relative pallor indicating significant anaemia (Leishman stain, ×400)

3.2 Biochemical Profile

The biochemical profile revealed a pattern highly evocative of a plasma cell dyscrasia. Total serum protein was markedly elevated at 11.60 g/dL (reference: 6.1–8.1 g/dL), with a disproportionately elevated globulin fraction of 8.01 g/dL and a severely inverted albumin-to-globulin (A/G) ratio of 0.44. Renal function tests demonstrated azotaemia (serum urea 64 mg/dL) and elevated creatinine (1.46 mg/dL). ALP was elevated at 354 U/L; CRP at 49.10 mg/L; serum ferritin at 524.26 ng/mL; ESR 22 mm/1st hr. Serum calcium was within normal limits (9.8 mg/dL). Urine Bence Jones protein was negative.

Parameter	Result	Reference Range
Haemoglobin	6.6 g/dL ↓	13–17 g/dL
RBC Count	2.17 × 10⁶/µL ↓	4.5–5.5 × 10⁶/µL
MCV	90 fL (Normal)	83–101 fL
Total Protein	11.60 g/dL ↑	6.1–8.1 g/dL
Albumin	3.59 g/dL	3.2–4.6 g/dL
Globulin	8.01 g/dL ↑	2.5–3.5 g/dL
A/G Ratio	0.44 ↓ (Inverted)	1.1–2.0
Serum Creatinine	1.46 mg/dL ↑	0.66–1.25 mg/dL
Serum Urea	64.0 mg/dL ↑	19.0–42.8 mg/dL
ALP	354 U/L ↑	38–126 U/L
CRP	49.10 mg/L ↑	0–10 mg/L
Serum Ferritin	524.26 ng/mL ↑	70–435 ng/mL
Serum Calcium	9.8 mg/dL (Normal)	8.4–10.2 mg/dL
Serum PTH (1-84)	<4.0 pg/mL ↓	9.2–44.6 pg/mL
ESR	22 mm/1st hr	0–20 mm/1st hr
Serum LDH	178 U/L (Normal)	120–246 U/L
Urine Bence Jones Protein	Negative	—

Table 1: Key laboratory investigations on admission.

3.3 Serum Protein Electrophoresis & Kappa Light Chain

Serum protein electrophoresis (Metropolis Laboratory, 04/03/2026) was a landmark investigation. It revealed total protein 9.44 g/dL, serum albumin 2.80 g/dL, gamma globulin 4.30 g/dL (reference 0.8–1.4), and an A/G ratio of 0.41. Most crucially, an M-band of 3.9 g/dL was identified in the gamma globulin region on capillary electrophoresis, with a comment that immunofixation was mandated for further evaluation. Serum kappa light chain levels were markedly elevated at 234.27, corroborating monoclonal plasma cell secretory activity and solidifying the diagnosis.

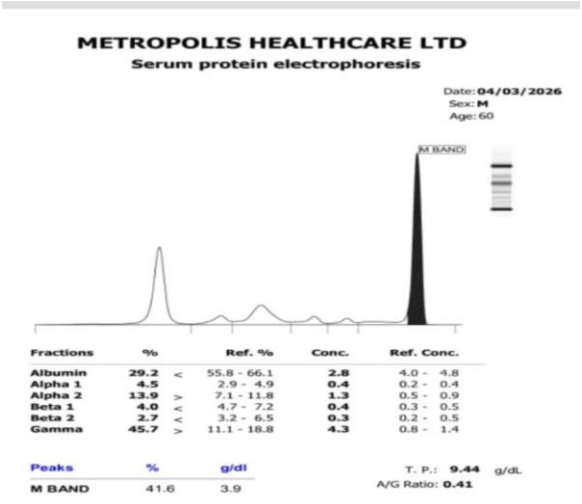

Figure 2: Serum Protein Electrophoresis (Metropolis Healthcare, 04/03/2026) — Densitometric scan demonstrating a tall, sharp restricted band (M-band, 3.9 g/dL, 41.6%) in the gamma globulin region, with

corresponding dense band on the gel strip (right). Albumin markedly reduced (29.2%) with gross elevation of gamma globulin fraction (45.7%) — the classical electrophoretic signature of monoclonal gammopathy.

3.4 Bone Marrow Aspiration and Imprint Smear

Bone marrow aspiration was performed on 03/03/2026 (BM-25/26). The aspirate smears were bloody, depicting mainly fatty marrow with dispersed stromal fragments and a good number of plasma cells with eccentrically placed nuclei and abundant cytoplasm. The imprint smear was the definitive diagnostic advance: it was cellular and demonstrated numerous plasma cells scattered amidst haematopoietic elements. A good number of the plasma cells displayed binucleation with a few multinucleated forms. A few flame cells and occasional plasmablasts were noted. Normal haematopoietic elements appeared reduced in number with severe suppression of myeloid precursors. No granulomas were seen.

Impression: Imprint Smear: Features are suspicious for plasma cell disorder — Neoplastic.

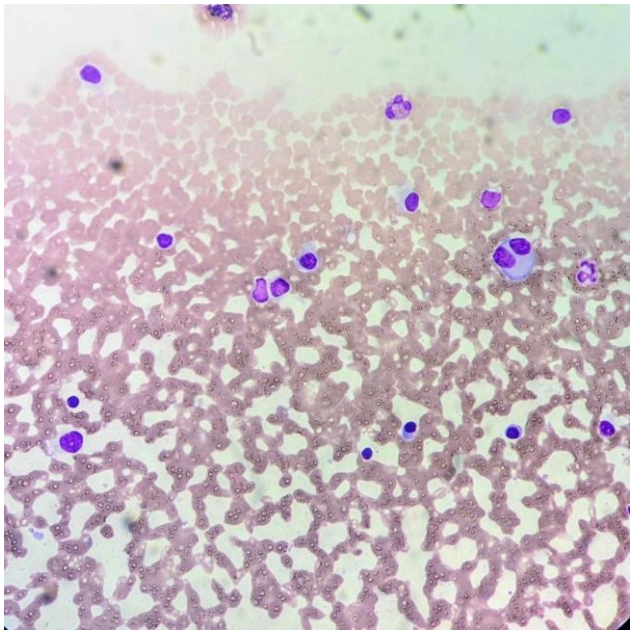

Figure 3: Bone Marrow Aspirate Imprint Smear (Leishman stain, ×400) — Cellular smear showing scattered plasma cells with eccentrically placed nuclei and abundant cytoplasm among haematopoietic elements. Note suppression of normal myeloid precursors consistent with marrow infiltration by neoplastic plasma cells.

3.5 Bone Marrow Trephine Biopsy

Bone marrow trephine biopsy (BX-971/26) provided definitive histological confirmation. A 2 cm linear grey-white to grey-brown tissue core was received. Microscopy depicted sheets of mononuclear plasma cells with eccentrically placed nuclei, granular-to-condensed chromatin, and inconspicuous nucleoli. Larger, immature cells (plasmablasts) with prominent nucleoli were interspersed. Moderate suppression of other haematopoietic elements was noted with only occasional megakaryocytes. No granulomas were identified.

Impression: Biopsy (Bone Marrow): Features are suggestive of Plasma Cell Neoplasm — Myeloma. Advised: clinico-radiological correlation, serobiochemical profile, electrophoresis for M-band, immunofixation, and tumour markers (Beta-2 microglobulin) evaluation.

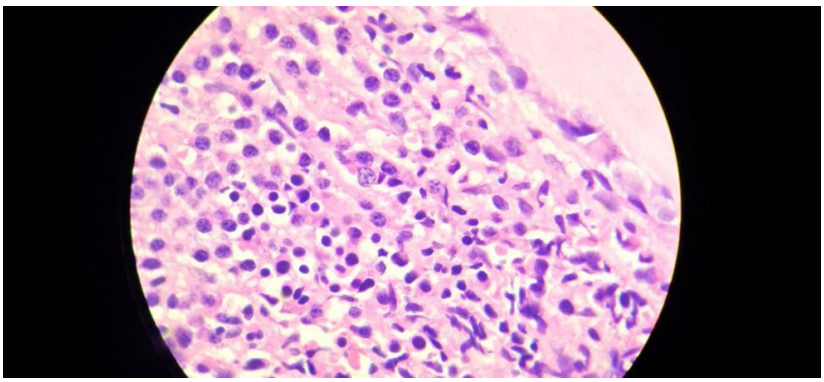

Figure 4A: Bone Marrow Trephine Biopsy (H&E, ×40) — Low-power view showing sheets of mononuclear plasma cells replacing normal haematopoietic marrow with bony trabeculae at periphery.

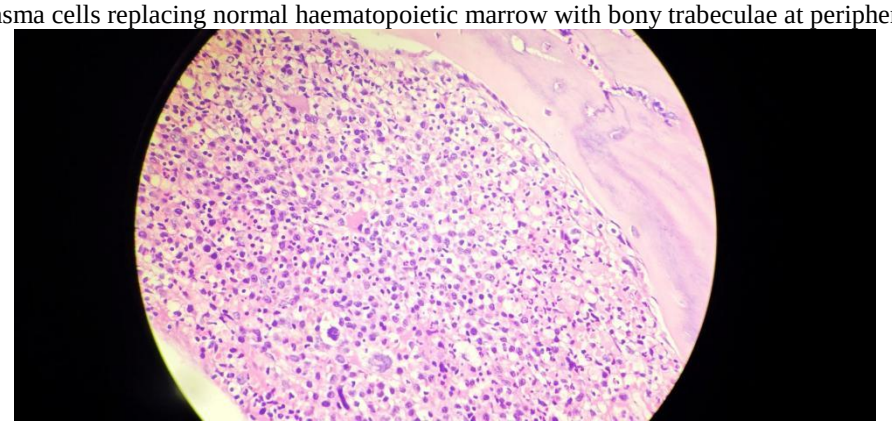

Figure 4B: Bone Marrow Trephine Biopsy (H&E, ×40) — Dense plasma cell infiltration with moderate suppression of other haematopoietic elements and occasional residual megakaryocytes.

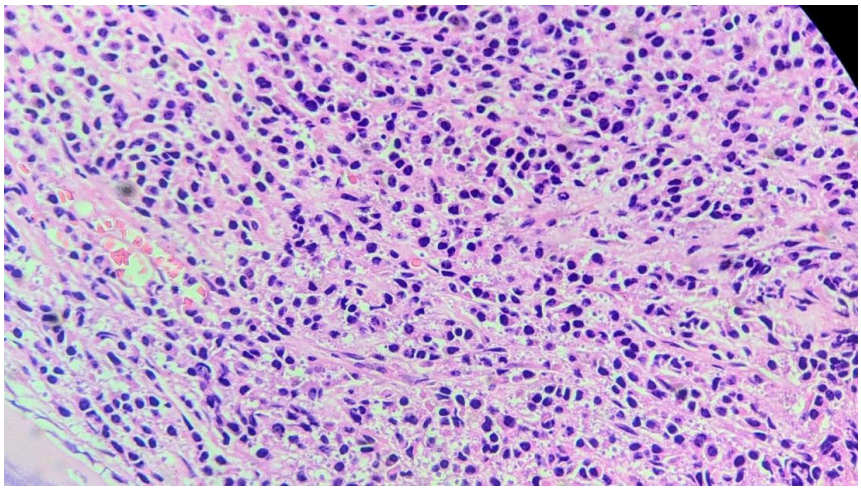

Figure 4C: Bone Marrow Trephine Biopsy (H&E, ×200) — High-power view demonstrating sheets of neoplastic plasma cells with eccentrically placed nuclei, abundant cytoplasm, condensed chromatin, and interspersed plasmablasts with prominent nucleoli — diagnostic of plasma cell myeloma.

3.6 Beta-2 Microglobulin (Prognostic Staging)

Beta-2 microglobulin (β_2M) was advised by the pathologist at the time of biopsy reporting and is a critical determinant of prognostic staging under the International Staging System (ISS). As per the ISS: Stage I is defined by β_2M <3.5 mg/L and albumin ≥3.5 g/dL (median survival 62 months); Stage II by β_2M <3.5 mg/L with albumin <3.5 g/dL, or β_2M 3.5–5.5 mg/L (median survival 44 months); and Stage III by β_2M >5.5 mg/L (median survival 29 months). In the present case, given the patient's serum albumin of 2.80 g/dL (below 3.5 g/dL) and the substantial renal burden, the clinical picture is suggestive of at minimum ISS Stage II, pending formal β_2M quantification. Formal β_2M assay result is recommended to be added prior to submission of this manuscript for formal ISS staging documentation.

ISS Stage	Criteria	Median Survival
Stage I	β_2M <3.5 mg/L AND Albumin ≥3.5 g/dL	62 months
Stage II	β_2M <3.5 with Albumin <3.5, OR β_2M 3.5–5.5 mg/L	44 months
Stage III	β_2M >5.5 mg/L	29 months

Table 2: International Staging System (ISS) for Multiple Myeloma. Stage II highlighted — most likely stage for this patient based on albumin 2.80 g/dL pending formal β_2M result.

3.7 Radiological Investigations

MRI of the lumbosacral spine demonstrated extensive diffuse osteoporosis, widespread lytic lesions in all visualised bony structures, and decreased vertebral body heights — features strongly suggestive of multiple myeloma.

Additional findings included sacralization of L5, lumbar scoliosis with leftward convexity, and diffuse disc bulge with central disc protrusion at L4–L5 causing moderate thecal sac compression. As part of the myeloma skeletal survey, a lateral skull radiograph was also obtained, which demonstrated the classical radiological hallmark of multiple myeloma.

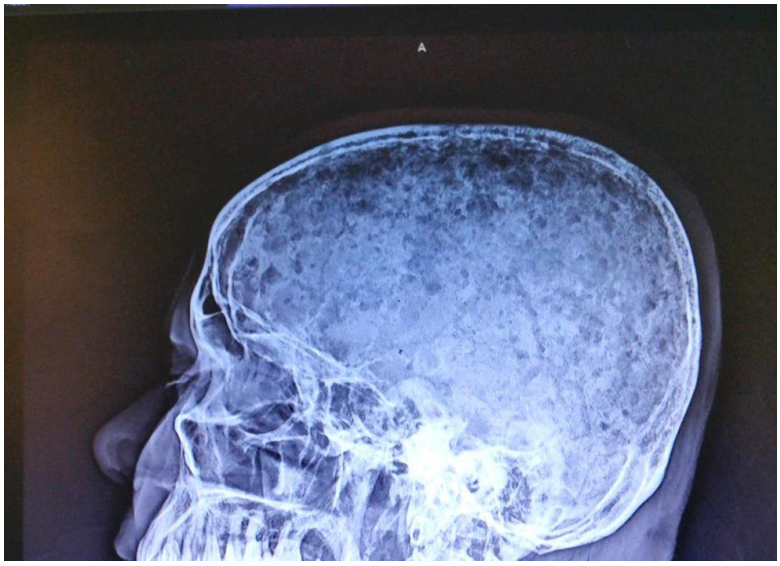

Figure 5: Lateral Skull Radiograph — Demonstrating multiple, discrete, well-defined "punched-out" lytic lesions scattered throughout the calvarium, producing the classical "pepper-pot skull" appearance pathognomonic of multiple myeloma. Lesions show no surrounding sclerotic margin or periosteal reaction, consistent with purely osteolytic, non-reactive bone destruction by infiltrating plasma cells.

4. FINAL DIAGNOSIS

Multiple Myeloma (Plasma Cell Neoplasm)

The diagnosis was established on convergence of: (1) symptomatic normocytic normochromic anaemia on peripheral blood smear with bone marrow suppression; (2) renal dysfunction; (3) extensive lytic bone lesions on MRI; (4) markedly elevated serum globulin with M-band (3.9 g/dL) on protein electrophoresis; (5) elevated kappa light chains (234.27); and (6) bone marrow trephine biopsy confirming plasma cell infiltration — all fulfilling IMWG diagnostic criteria for symptomatic multiple myeloma.

5. MANAGEMENT AND TREATMENT

Following multidisciplinary discussion, the patient was initiated on a bortezomib-thalidomide based induction regimen, the current standard of care for transplant-ineligible elderly patients:

Drug	Dose / Route	Duration
Inj. Bortezomib (Proteasome inhibitor)	1.3 mg/m² SC/IV weekly (28-day cycle)	6–8 cycles
Cap. Thalidomide (Immunomodulatory)	100 mg PO once daily (at night)	6 months
Tab. Acyclovir (Antiviral prophylaxis)	400 mg PO twice daily	6 months
Tab. Fluconazole (Antifungal prophylaxis)	100 mg PO once daily	Ongoing
Tab. Allopurinol (Tumour lysis prophylaxis)	100 mg PO thrice daily	2 weeks

Table 3: Chemotherapy and supportive medication regimen.

6. FOLLOW-UP PLAN

A structured follow-up protocol was instituted. Serial assessment for peripheral neuropathy (known toxicity of both bortezomib and thalidomide), haematological indices, and renal function was planned. The patient and caregivers were counselled regarding fever as a danger sign given the immunocompromised state, and active mobilisation was encouraged to mitigate venous thromboembolism and skeletal complications.

7. DISCUSSION

This case encapsulates the paradigmatic diagnostic challenge of multiple myeloma: a disease that whispers before it screams. The patient's six-month history of backache, attributed elsewhere to musculoskeletal causes, is tragically common in the MM narrative.

The peripheral blood smear findings of normocytic normochromic anaemia, while seemingly non-specific, are a cardinal early feature of MM and should prompt suspicion when seen alongside elevated total protein and an inverted A/G ratio. The markedly elevated globulin fraction of 8.01 g/dL

with inverted A/G ratio (0.44) was the single most important biochemical clue in this case. The serum protein electrophoresis revealing an M-band of 3.9 g/dL in the gamma region, combined with kappa light chain elevation at 234.27, provided biochemical confirmation of monoclonal paraprotein production.

The bone marrow imprint smear and trephine biopsy were pathognomonic. Flame cells — with their distinctive fiery cytoplasmic projections, characteristic of IgA myeloma — alongside plasmablasts and binucleated plasma cells in the context of suppressed normal haematopoiesis is the histological hallmark of MM. The negative Bence Jones proteinuria is not a reliable exclusionary finding, as non-secretory and IgG-secreting myelomas may not shed detectable urinary light chains.

The biopsy pathologist's recommendation for Beta-2 microglobulin (β_2M) evaluation is clinically vital. β_2M is the single most powerful prognostic marker in MM and forms the backbone of the ISS. Given this patient's albumin of 2.80 g/dL (below 3.5 g/dL threshold), at minimum an ISS Stage II classification is expected, carrying a median survival of 44 months. Formal β_2M quantification should be completed and incorporated into the ongoing management plan.

Radiologically, the lateral skull radiograph in this patient reproduced the textbook "pepper-pot skull" sign — multiple discrete punched-out lytic lesions without sclerotic margins, reflecting unchecked osteoclastic bone resorption driven by RANKL upregulation from marrow-infiltrating plasma cells. This finding, together with the vertebral lytic lesions seen on MRI, fulfils the "B" (bone lesions) criterion of the CRAB framework and reinforces the systemic, multifocal nature of skeletal involvement in this patient.

The choice of bortezomib-thalidomide induction aligns with international guidelines demonstrating superior response rates compared to alkylator-based regimens in transplant-ineligible patients.

8. CONCLUSION

This case powerfully illustrates that chronic backache in an elderly patient is not always the benign companion it appears to be. A combination of normocytic normochromic anaemia on peripheral blood smear, markedly inverted A/G ratio, M-band on serum protein electrophoresis, elevated kappa light chains, and bone marrow biopsy confirming plasma cell infiltration — together with lytic bone lesions on imaging — established the diagnosis of multiple myeloma beyond doubt. Early identification and prompt institution of proteasome inhibitor-based therapy offers the best opportunity for disease control and improved quality of life.

WHAT THIS CASE ADDS — KEY LEARNING POINTS

1. In an elderly male with chronic back pain and anaemia, multiple myeloma must always be included in the differential — even when prior investigations were normal. A negative early workup does not exclude MM.
2. A markedly elevated total serum protein with severely inverted A/G ratio is a powerful biochemical red flag for plasma cell dyscrasia, often preceding more obvious clinical manifestations.
3. Peripheral blood smear showing normocytic normochromic anaemia, though non-specific, is an early and consistent haematological manifestation of MM and should prompt a full myeloma workup when seen alongside elevated globulins.
4. Negative Bence Jones proteinuria does NOT exclude multiple myeloma. Serum immunofixation and free light chain assay are far more sensitive and must always be requested.
5. Beta-2 microglobulin (β_2M) is the cornerstone of ISS prognostic staging in MM. It must be quantified at diagnosis and incorporated into the management plan.

6. PATIENT CONSENT

Written informed patient consent was obtained for publication of this case report. Patient identity has been fully anonymised in accordance with institutional and ethical guidelines.

CARE Checklist

CARE Guidelines for Case Reports — 13-Item Checklist

Reference: Gagnier JJ, et al. J Med Case Rep. 2013;7:223.

No.	Section	Description	Location	Present?
1	Title	Diagnosis + "case report" in title	Title	**Yes**
2	Keywords	2–5 keywords including "case report"	Abstract	**Yes**
3a	Abstract	What is unique; what does it add	Abstract	**Yes**
3b	Abstract	Main symptoms and clinical findings	Abstract	**Yes**
3c	Abstract	Diagnoses, interventions, outcomes	Abstract	**Yes**
3d	Abstract	Main take-away lessons	Abstract	**Yes**
4	Introduction	Why this case is unique (with refs)	Section 1	**Yes**
5	Patient Info	De-identified history and complaints	Sections 2.1–2.2	**Yes**

6	Clinical Findings	Physical exam findings	Section 2.3	**Yes**
7	Timeline	Key dates from symptoms to treatment	Sections 2, 3, 5	**Yes**
8	Diagnostic Assessment	Laboratory, imaging, histology; differential diagnosis	Section 3	**Yes**
9	Therapeutic Interventions	Drugs, doses, route, who performed	Section 5	**Yes**
10	Follow-up & Outcomes	Outcomes, test results, tolerability	Section 6	**Yes**
11a	Discussion	Strengths and limitations	Section 7	**Yes**
11b	Discussion	Relevant medical literature	Section 7	**Yes**
11c	Discussion	Rationale for conclusions	Section 7	**Yes**
11d	Discussion	Primary take-away lessons	Sections 8 + Learning Points	**Yes**
12	Patient Perspective	Patient view on treatment	Section 9 (consent only)	N/A
13	Informed Consent	Patient gave consent for publication	Section 9	**Yes**

Conflicts of Interest

The authors declare no conflicts of interest.

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