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Polyostotic Paget's Disease of Bone Involving the Pelvis and Lumbar Spine in A 78-Year-Old Female: A Case Report

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ABSTRACT

Background: Paget's disease of bone (PDB) is a chronic, focal disorder of bone remodelling characterised by disorganised osteoclastic resorption followed by exuberant, structurally abnormal bone formation. It typically affects individuals over 55 years of age and is comparatively uncommon in the Indian subcontinent, where it may be under-recognised.

Case Report: A 78-year-old female presented with insidious-onset, progressive low back pain of 18 months' duration with difficulty in ambulation. Radiographs of the pelvis and lumbosacral spine showed a coarsened, disorganised trabecular pattern with mixed lytic and sclerotic areas involving both iliac bones and the pubic rami, thickening of the iliopectineal line, cortical thickening, and a "picture-frame" appearance of the lumbar vertebrae. Serum alkaline phosphatase (ALP) was markedly elevated at 684 IU/L (reference 40-129 IU/L) with normal calcium, phosphate and parathyroid hormone, and secondary causes of osteosclerosis were excluded. Technetium-99m MDP bone scintigraphy confirmed a polyostotic distribution. Following correction of vitamin D status, she received a single intravenous infusion of zoledronic acid 5 mg with analgesia and supervised physiotherapy. At 6 months, pain had improved from a visual analogue scale score of 8/10 to 3/10 and serum ALP had fallen to 198 IU/L.

Conclusion: PDB should be considered in elderly patients with chronic skeletal pain and mixed lytic-sclerotic radiographic changes. Characteristic radiological signs with an elevated ALP and normal calcium and phosphate permit accurate non-invasive diagnosis in most cases, and timely bisphosphonate therapy relieves symptoms and substantially reduces bone turnover.

Keywords: *Paget's disease of bone, osteitis deformans, bone remodelling, zoledronic acid, alkaline phosphatase, case report*

INTRODUCTION

Paget's disease of bone (PDB), also termed osteitis deformans, is a chronic, focal disorder of skeletal remodelling first

described by Sir James Paget in 1877.[1] The condition is characterised by an initial phase of intense, disorganised osteoclastic bone resorption, followed by a compensatory phase of rapid but architecturally abnormal osteoblastic bone formation, culminating in sclerotic, structurally weak, and hypervascular bone that is prone to deformity and fracture.[2]

PDB predominantly affects individuals in the sixth decade of life and beyond, with a slight male preponderance. The disease demonstrates striking geographical clustering, with the highest prevalence reported in the United Kingdom, Western Europe, Australia, and New Zealand, and comparatively low prevalence in Scandinavia, Asia, and Africa.[3] Contemporary epidemiological data indicate a continuing secular decline in both incidence and severity in historically high-prevalence populations, a trend documented in United Kingdom primary care data and in a 20-year Canadian population study, and attributed to a combination of earlier diagnosis, milder phenotypes, and as-yet-unidentified environmental change.[4,5] Reports from the Indian subcontinent remain sparse, and the true prevalence is likely to be underestimated because a substantial proportion of patients are asymptomatic and never come to medical attention.[6]

The aetiology of PDB is incompletely understood and is considered multifactorial, involving an interaction between genetic susceptibility and environmental triggers. Mutations in the SQSTM1 gene, which encodes the sequestosome-1/p62 protein involved in RANK-NF-κB signalling, are identified in a substantial proportion of familial cases.[7] A long-postulated but still unproven role for chronic paramyxoviral infection of osteoclasts has also been described.[8]

The axial skeleton is most commonly affected, with the pelvis, lumbar spine, femur, tibia, and skull involved in descending order of frequency.[3] Clinical presentation ranges from an entirely asymptomatic incidental radiological finding through to bone pain, deformity, secondary osteoarthritis, pathological fracture, neurological compression syndromes, high-output cardiac failure, and, rarely, sarcomatous degeneration.[9]

We report a case of polyostotic Paget's disease involving the pelvis and lumbar spine in an elderly female, diagnosed non-invasively and treated with a single infusion of zoledronic acid, and discuss the pathophysiology, diagnostic approach, differential diagnosis, and current management principles relevant to the practising orthopaedic surgeon. This report has been prepared in accordance with the CARE (CAse REport) guidelines.

CASE REPORT

A 78-year-old female presented to the orthopaedic outpatient department with insidious-onset, progressive low back pain of 18 months' duration, associated with difficulty in walking and a progressively reduced walking distance. The pain was described as a deep, persistent ache, present both at rest and on activity, with nocturnal exacerbation, and was only partially relieved by simple analgesics. Pain intensity at presentation was recorded as 8/10 on a visual analogue scale (VAS). There was no history of preceding trauma. The patient denied fever, weight loss, loss of appetite, or other constitutional symptoms. There was no history of malignancy, chronic renal disease, or prolonged drug intake, and no history of hearing loss, headache, or progressive increase in head size. Bladder and bowel function were normal, and there were no radicular or claudicant symptoms in the lower limbs. Family history was non-contributory.

Clinical Examination

On general examination the patient was conscious and oriented, with no pallor, icterus, or lymphadenopathy, and vital parameters were within normal limits. Local examination of the lumbosacral spine revealed no visible deformity, scar, or swelling. There was diffuse paraspinal

tenderness in the lower lumbar region with restriction of terminal lumbar flexion and extension. Bilateral hip movements were terminally restricted and mildly painful. There was no palpable local rise of temperature or bony swelling. Neurological examination of both lower limbs demonstrated normal tone, power, sensation, and deep tendon reflexes, with a negative straight leg raise bilaterally. Peripheral pulses were well felt. Gait was antalgic with a shortened stride length.

Radiological Findings

Radiographs of the pelvis in the anteroposterior view (Figure 1) demonstrated a diffusely coarsened, disorganised trabecular pattern with mixed lytic and sclerotic ("cotton-wool") areas involving both iliac bones, most marked in the iliac wings and extending into the supra-acetabular regions. There was thickening and accentuation of the iliopectineal and ilioischial lines, generalised cortical thickening, and loss of the normal corticomedullary differentiation. The pubic rami showed similar trabecular coarsening with cortical expansion. Both hip joints showed mild superomedial joint space narrowing consistent with secondary degenerative change. No cortical breach, periosteal reaction, or associated soft tissue mass was identified.

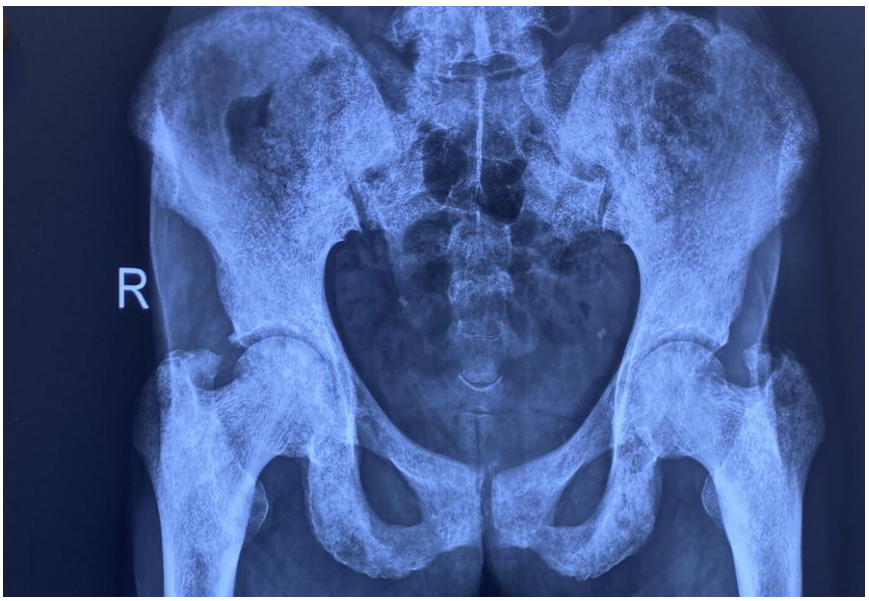

Figure 1: Anteroposterior radiograph of the pelvis demonstrating a diffusely coarsened and disorganised trabecular pattern with mixed lytic and sclerotic areas involving both iliac bones and the pubic rami, thickening of the iliopectineal line, generalised cortical thickening, and loss of normal corticomedullary differentiation. Mild superomedial narrowing of both hip joints is noted, consistent with secondary degenerative change. Patient identifiers have been removed.

Anteroposterior and lateral radiographs of the lumbosacral spine (Figure 2) demonstrated increased vertebral density with a coarsened trabecular pattern and accentuation of the peripheral cortical margins, producing the characteristic "picture-frame" appearance of the vertebral body. Vertebral body height was largely maintained, with no vertebral collapse, and the intervertebral disc spaces were preserved. Facetal and endplate degenerative changes were noted at the lower lumbar levels. There was no paravertebral soft tissue shadow, no destructive lesion, and no evidence of pedicle erosion.

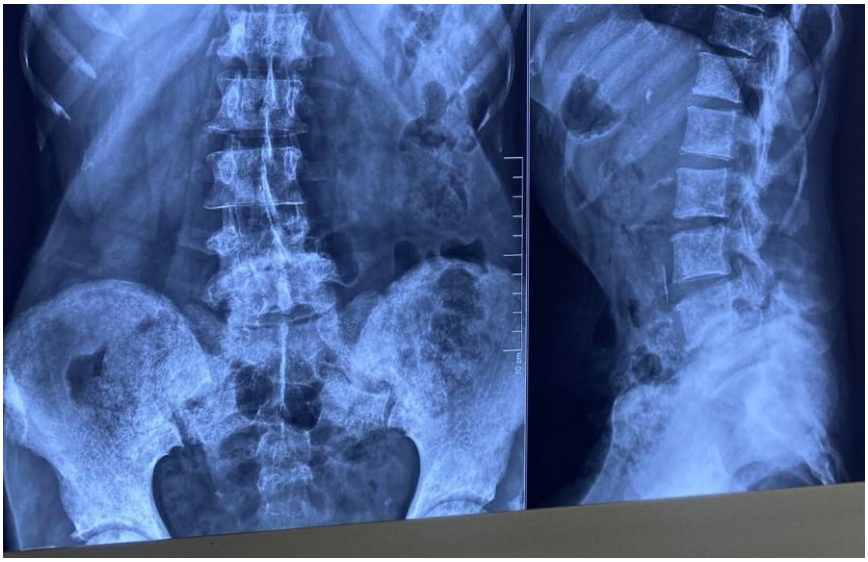

Figure 2: Anteroposterior (left) and lateral (right) radiographs of the lumbosacral spine showing increased vertebral body density with a coarsened internal trabecular pattern and accentuation of the peripheral cortical margins, producing the characteristic "picture-frame" appearance. Vertebral body heights and intervertebral disc spaces are largely preserved. Patient identifiers have been removed.

Laboratory Investigations

Serum alkaline phosphatase (ALP) was markedly elevated at 684 IU/L (reference range 40-129 IU/L), representing approximately a fivefold elevation above the upper limit of normal. Serum calcium, serum phosphate, and serum parathyroid hormone were within normal limits. Renal and hepatic function tests were normal, and gamma-glutamyl transferase was normal, confirming a skeletal rather than hepatobiliary source of the raised ALP. Serum 25-hydroxyvitamin D was 24 ng/mL, indicating vitamin D insufficiency requiring correction prior to antiresorptive therapy. Complete blood count, erythrocyte sedimentation rate, and C-reactive protein were within normal limits. Serum protein electrophoresis and urinary Bence Jones protein were negative. These findings excluded multiple myeloma, metastatic disease, renal osteodystrophy, and osteomalacia as alternative explanations for the radiological appearances.

A whole-body technetium-99m methylene diphosphonate (Tc-99m MDP) bone scan demonstrated intense, homogeneous, well-demarcated tracer uptake corresponding to the involved pelvic and lumbar segments, confirming the polyostotic distribution and excluding additional clinically silent lesions. Bone biopsy was not performed, as the combination of characteristic radiological features, markedly elevated ALP with normal calcium and phosphate, and a typical scintigraphic pattern was considered diagnostic. The episode of care is summarised in Table 1.

Table 1: Timeline of the episode of care.

Time point	Event
18 months before presentation	Onset of insidious low back pain
Initial visit (Day 0)	Clinical examination; radiographs of pelvis and lumbosacral spine
Week 1	Biochemical evaluation — serum ALP 684 IU/L; calcium, phosphate and PTH normal; myeloma screen negative
Week 2	Tc-99m MDP whole-body bone scan confirming polyostotic distribution
Week 3	Intravenous zoledronic acid 5 mg infusion; calcium and cholecalciferol supplementation commenced
3 months	Symptomatic improvement in back pain; increased walking distance
6 months	VAS 8/10 → 3/10; serum ALP 684 → 198 IU/L; functional improvement sustained

Management

The patient was counselled regarding the nature, natural history, and prognosis of the condition. Vitamin D and calcium status were optimised prior to antiresorptive therapy in order to prevent post-infusion hypocalcaemia. In the third week following presentation she received a single intravenous infusion of zoledronic acid 5 mg over 15 minutes with adequate pre-infusion hydration, followed by continued oral calcium and cholecalciferol supplementation. Analgesia was provided with paracetamol supplemented by short-course non-steroidal anti-inflammatory therapy, and a supervised physiotherapy programme comprising core strengthening, hip abductor strengthening, and gait training was instituted. A walking aid was prescribed to reduce loading and improve ambulatory confidence. The infusion was well tolerated; no acute-phase reaction, symptomatic hypocalcaemia, or renal impairment was observed.

Follow-up and Outcome

At 3 months the patient reported appreciable symptomatic improvement in back pain with an increase in pain-free walking distance. At 6 months, pain intensity had improved from a baseline VAS score of 8/10 to 3/10, and serum ALP had fallen from 684 IU/L to 198 IU/L — a reduction of approximately 71% from baseline. This represents a substantial biochemical response, although ALP remained modestly above the upper reference limit, indicating persisting low-grade disease activity rather than complete biochemical remission. Follow-up radiographs showed no interval progression, deformity, or fracture. The patient was enrolled in long-term surveillance with periodic clinical review and serial ALP estimation, with the plan to consider retreatment with zoledronic acid in the event of biochemical relapse or symptom recurrence.

Patient Perspective

The patient reported that the persistent back pain had significantly restricted her ability to perform household activities and to walk to the local place of worship, and that she had previously attributed her symptoms to "old age" and had not sought formal medical evaluation for a prolonged period. Following treatment, she reported being able to resume most household activities and to walk considerably further with reduced discomfort, and expressed satisfaction with the single-infusion treatment regimen and with the explanation provided regarding her diagnosis.

DISCUSSION

This case illustrates a classical presentation of polyostotic Paget's disease of bone involving the pelvis and lumbar spine in an elderly patient, diagnosed non-invasively on the basis of characteristic radiological and biochemical findings and treated with a single infusion of zoledronic acid. The case is of particular interest given the relatively low reported prevalence of PDB in the Indian population, where the condition may be under-recognised and misattributed to degenerative disease, metastatic bone disease, or metabolic bone disorders, with a predominance of polyostotic and symptomatic presentations in reported Indian series.[6] The 18-month interval between symptom onset and diagnosis in this patient is consistent with the diagnostic delay commonly reported in low-prevalence settings, and reflects the tendency for chronic axial pain in the elderly to be attributed to degenerative change without further biochemical evaluation.

Pathophysiology

The pathological hallmark of PDB is a focal, markedly accelerated but disorganised bone remodelling cycle. The initiating abnormality resides within the osteoclast: pagetic osteoclasts are increased in number, abnormally large, and contain up to 100 nuclei, with a substantially increased resorptive capacity.[2] This osteolytic phase

is followed by an intense compensatory osteoblastic response, in which new bone is deposited rapidly and haphazardly, producing a disorganised mosaic pattern of irregular cement lines that is pathognomonic on histology. The resultant bone is expanded and sclerotic on imaging but mechanically inferior, being both less stiff and more prone to microdamage accumulation, deformity, and fracture.[9] The marrow space is replaced by loose, richly vascular fibrous connective tissue, accounting for the increased local temperature over affected superficial bones, the risk of substantial intraoperative haemorrhage, and, in extensive polyostotic disease, the rare complication of high-output cardiac failure.[10]

At the molecular level, mutations in SQSTM1 (p62), most commonly the p.P392L variant, are the most consistently identified genetic abnormality and are found in approximately 5-10% of sporadic and up to 40-50% of familial cases. The p62 protein functions as a scaffold within the RANKL-RANK-NF-κB signalling axis, and loss-of-function mutations lead to sustained NF-κB activation, enhanced osteoclastogenesis, and increased osteoclast sensitivity to RANKL and 1,25-dihydroxyvitamin D.[7,11] Genome-wide association studies have subsequently identified additional susceptibility loci including CSF1, OPTN, TM7SF4/DC-STAMP, and RIN3.[11] The historically proposed paramyxovirus hypothesis, based on the identification of nuclear inclusion bodies resembling viral nucleocapsids within pagetic osteoclasts, remains unconfirmed and is not universally reproducible.[8]

Clinical Presentation and Natural History

It is estimated that up to 70-80% of patients with PDB are asymptomatic at the time of diagnosis, the condition being detected incidentally on radiographs obtained for unrelated reasons or on the finding of an isolated elevation of serum ALP during routine biochemistry.[3,9] When symptomatic, bone pain is the commonest complaint, characteristically deep, persistent, present at rest, and often worse at night — a pattern that helps distinguish it from mechanical degenerative pain, and one that was clearly present in our patient. Pain in PDB may arise from increased metabolic activity within the pagetic bone itself, from secondary osteoarthritis of an adjacent joint, from microfracture, or from nerve compression.[9]

Pelvic involvement, as in the present case, frequently produces secondary hip osteoarthritis through alteration of acetabular architecture and, in advanced disease, acetabular protrusio. Spinal involvement may cause back pain, spinal stenosis, and rarely neurological compromise through vertebral expansion, ligamentous ossification, pathological fracture, or a vascular steal phenomenon.[12] Long bone involvement classically produces bowing deformity of the femur and tibia (the "sabre tibia") with the associated risk of insufficiency and complete pathological fracture, which characteristically occurs on the convex, tension side of the deformity.[13] Skull involvement may result in progressive sensorineural or conductive hearing loss, cranial nerve palsies, and basilar invagination.

Sarcomatous transformation, most commonly to osteosarcoma, is the most feared complication of PDB but is rare, occurring in fewer than 1% of cases. It should be suspected when a patient with established PDB develops new, rapidly progressive, or disproportionate pain, a new soft tissue mass, cortical destruction, or an abrupt further rise in ALP, and carries a poor prognosis.[14]

Diagnosis and Differential Diagnosis

The diagnosis of PDB is, in the majority of cases, established non-invasively through the combination of characteristic plain radiographic appearances and an elevated serum ALP with otherwise normal calcium and phosphate metabolism, an approach endorsed by both the UK clinical guideline and the recent Italian Society of Osteoporosis, Mineral Metabolism and Skeletal Diseases (SIOMMMS) position

paper.[15,16] Plain radiography remains the single most useful investigation and typically demonstrates trabecular coarsening, cortical thickening, bone expansion, and mixed lytic and sclerotic areas. In the skull, the "osteoporosis circumscripta" of the lytic phase or the "cotton-wool" appearance of the mixed phase may be seen. In the vertebral body, the combination of a coarsened internal trabecular pattern with accentuated peripheral cortical margins produces the classical "picture-frame" vertebra, while diffuse homogeneous sclerosis of a single vertebra yields the "ivory vertebra" appearance.[12] In the pelvis, thickening of the iliopectineal line is an early and characteristic sign.

Total serum ALP is the recommended first-line biochemical marker for both diagnosis and monitoring of disease activity and response to treatment; where hepatobiliary disease coexists, bone-specific ALP or procollagen type I N-terminal propeptide (P1NP) may be substituted. [15,16] Radionuclide bone scintigraphy is the most sensitive modality for defining the full extent of skeletal involvement and for identifying clinically silent lesions, and is recommended at the time of initial diagnosis.[15]

The principal differential diagnoses of the radiological picture described include osteoblastic skeletal metastases (particularly from prostate and breast primaries), which characteristically lack bone expansion and cortical thickening; multiple myeloma; renal osteodystrophy; fibrous dysplasia, which typically presents at a younger age with a ground-glass matrix; chronic osteomyelitis; and sclerosing bone dysplasias such as melorheostosis.[17] In the present case, the presence of bone expansion with cortical thickening, preservation of vertebral body height and disc spaces, normal calcium and phosphate, a negative myeloma screen, and a typical scintigraphic distribution together made the diagnosis of PDB secure without recourse to biopsy.

Management

Nitrogen-containing bisphosphonates are the mainstay of pharmacological treatment. A single intravenous infusion of zoledronic acid 5 mg is currently the treatment of choice, having demonstrated superior and substantially more durable biochemical remission than oral risedronate in randomised comparison, with the majority of responders maintaining remission for several years after a single dose.[18,19] A long-term observational cohort has further characterised the durability of response and identified higher baseline ALP and more extensive skeletal involvement as predictors of the need for eventual retreatment.[20] Alternative agents include oral risedronate, alendronate, and pamidronate. Adequate correction of vitamin D deficiency and calcium supplementation before and after infusion is essential to prevent symptomatic hypocalcaemia, as was undertaken in our patient, whose baseline 25-hydroxyvitamin D of 24 ng/mL indicated insufficiency. Calcitonin is now reserved for patients in whom bisphosphonates are contraindicated.

There is continuing debate regarding the appropriate threshold for treating asymptomatic disease. Current guidance recommends bisphosphonate therapy principally for symptomatic patients, while acknowledging that treatment may reasonably be considered in asymptomatic patients with active disease at skeletal sites where complications would be particularly consequential — the skull base, weight-bearing long bones, and the spine.[15,16] The PRISM and PRISM-EZ trials demonstrated that intensive, biochemically guided bisphosphonate therapy aimed at normalising ALP did not reduce the incidence of fracture, orthopaedic procedures, or quality-of-life decrement compared with symptom-directed treatment, and these findings underpin the current symptom-led approach.[21]

More recently, the Zoledronate in the Prevention of Paget's disease (ZiPP) randomised controlled trial examined whether genetic testing coupled with

prophylactic zoledronic acid could prevent disease development in asymptomatic SQSTM1 mutation carriers. Although the trial did not meet its primary endpoint, prophylactic zoledronic acid produced sustained suppression of bone turnover markers and a favourable signal with respect to the development and progression of bone lesions, raising the prospect of a genetically targeted preventive strategy in high-risk families.[22] In our patient, the observed 71% reduction in ALP at 6 months alongside marked symptomatic improvement is consistent with the expected therapeutic response, and the residual mild elevation supports continued biochemical surveillance with consideration of retreatment on relapse.

Surgical Considerations

Surgery in PDB is indicated for secondary osteoarthritis unresponsive to conservative measures, pathological or impending fracture, symptomatic deformity, spinal stenosis with neurological compromise, and suspected sarcomatous transformation.[13] Total hip arthroplasty in pagetic patients presents specific technical challenges including protrusio acetabuli, distorted proximal femoral geometry, sclerotic and hard bone that complicates canal preparation and increases the risk of iatrogenic fracture, and the potential for excessive intraoperative haemorrhage from hypervascular bone. Preoperative bisphosphonate therapy is advocated by many authors to reduce disease activity and vascularity, although high-quality evidence for this practice remains limited. Long-term arthroplasty outcomes in PDB are generally satisfactory, with an increased but acceptable rate of heterotopic ossification and revision compared with non-pagetic controls. [13,23]

Fracture fixation in pagetic bone requires careful preoperative templating, awareness of altered mechanical axes secondary to bowing deformity, and preparedness for difficult canal reaming; intramedullary fixation may necessitate corrective osteotomy to accommodate the implant within a deformed bone. In the present case, the mild secondary hip degenerative change was managed conservatively and did not warrant arthroplasty.

Limitations

This report is limited by the absence of histopathological confirmation, although tissue diagnosis is not routinely required where the clinical, radiological, and biochemical picture is characteristic. Genetic testing for SQSTM1 mutations was not performed, as it is not indicated in sporadic disease without a relevant family history. Advanced cross-sectional imaging was not undertaken, as it was not clinically indicated in the absence of features suggesting malignant transformation or neurological compromise. The follow-up period of 6 months, while adequate to demonstrate the expected early biochemical and symptomatic response, is insufficient to comment on the durability of remission or on the long-term prevention of skeletal complications; longer-term surveillance is ongoing.

CONCLUSION

We report a case of polyostotic Paget's disease of bone involving the pelvis and lumbar spine in a 78-year-old female, diagnosed non-invasively through characteristic radiographic features supported by a markedly elevated serum alkaline phosphatase with normal calcium and phosphate metabolism, and treated with a single intravenous infusion of zoledronic acid alongside analgesia and structured rehabilitation. At 6 months the patient demonstrated a 71% reduction in serum ALP with clinically meaningful pain relief and functional gain. Paget's disease of bone should be actively considered in any elderly patient presenting with chronic, poorly localised skeletal pain and mixed lytic-sclerotic radiographic changes with bone expansion and cortical thickening, particularly when an isolated elevation of serum alkaline phosphatase is present with normal calcium and phosphate. Attributing chronic axial pain in the elderly to degenerative change without biochemical

evaluation is a recognised cause of diagnostic delay, as illustrated by the 18-month interval in this case. Plain radiography and serum ALP will establish the diagnosis in the great majority of cases without the need for biopsy, and bone scintigraphy should be obtained at diagnosis to define the extent of skeletal involvement. Orthopaedic surgeons should remain alert to the technical difficulties of operating on pagetic bone and to the rare but grave possibility of sarcomatous transformation heralded by new or disproportionate pain.

Declaration by Authors

Informed Consent: Written informed consent was obtained from the patient for publication of this case report and the accompanying images. All patient identifiers have been removed.

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Conflict of Interest: The authors declare no conflict of interest.

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