Osteoporotic Compression Fracture with Anterolisthesis in a Geriatric Diabetic Patient: A Conservative Management Approach

Nikhitha Shetty[1,2], Nirmala Rajesh[1], Aishwarya[2]

[1] Srinivas University, Mangaluru, - 575 001. Karnataka State, INDlA.
[2] Department of Medical Imaging Sciences, College of Health Sciences, Gulf Medical University, Ajman, UAE

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***Abstract*- Background:** Osteoporotic fractures in the elderly population represent a significant clinical challenge, particularly when complicated by multiple comorbidities. This case report presents the diagnostic workup and conservative management of a complex spinal fracture in a geriatric patient with diabetes mellitus.
Case Presentation: A 72-year-old female with type II diabetes mellitus, hypertension, and hypercholesterolemia presented with severe back pain radiating to the left lower limb and shoulder following a fall at home. Comprehensive imaging including X-ray, DEXA scan, and MRI revealed a T12 compression fracture with vertebral collapse, anterolisthesis of L4-L5, diffuse osteopenia, and secondary spinal canal stenosis. Laboratory findings demonstrated vitamin D and calcium deficiency with elevated alkaline phosphatase. Given the patient's advanced age and medical comorbidities, a conservative treatment approach was implemented consisting of electrotherapy, heat therapy, kinesiotherapy, and geriatric rehabilitation, along with pharmacological management and nutritional supplementation.
Conclusion: This case emphasizes the importance of multimodal diagnostic imaging and multidisciplinary collaboration in managing complex musculoskeletal conditions in elderly patients. Conservative management can provide effective pain relief and functional improvement when surgical intervention carries significant risk due to age and comorbidities.

***Index Terms*-** Osteoporotic fracture, T12 compression fracture, anterolisthesis, geriatric patient, conservative management, DEXA scan, type II diabetes mellitus

I. INTRODUCTION

Osteoporotic fractures, particularly those affecting the spine, represent a substantial challenge in geriatric healthcare due to their complex nature and high prevalence in aging populations(1–3). These fractures typically result from osteoporosis, a systemic skeletal disease characterized by reduced bone mass and microarchitectural deterioration, which predisposes individuals to fractures even with minimal trauma(3,4). The global burden of osteoporotic fractures continues to increase with population aging, affecting approximately 200 million women worldwide and resulting in significant morbidity, mortality, and healthcare costs(1–3).

The thoracolumbar junction (T11-L2) is particularly vulnerable to compression fractures due to the transition from the relatively rigid thoracic spine to the more mobile lumbar spine(2,5,6). Vertebral compression fractures can lead to chronic pain, kyphotic deformity, height loss, and reduced quality of life, with studies demonstrating increased mortality rates in patients with these fractures(2,4,5,6). When occurring in patients with multiple comorbidities such as diabetes mellitus, these fractures present additional management challenges due to compromised bone quality and increased surgical risks(8–10).

Type II diabetes mellitus has been increasingly recognized as a significant risk factor for osteoporotic fractures, with epidemiological studies demonstrating a 40-70% increased risk of hip and vertebral fractures in diabetic patients compared to non-diabetic individuals(8,9,11,12). Despite normal or even elevated bone mineral density in some diabetic patients, bone quality is compromised through multiple mechanisms including accumulation of

advanced glycation end products, oxidative stress, chronic inflammation, microvascular complications, and alterations in bone cell function and bone matrix composition(8,11,13). This creates a paradoxical situation where fracture risk is increased despite apparently adequate bone density, highlighting the limitations of DEXA scanning alone in assessing fracture risk in diabetic populations(13,14,15).

We present the case of a 72-year-old female patient with multiple comorbidities including osteoporosis, type II diabetes mellitus of 25 years duration, and hypertension, who sustained a T12 compression fracture following a fall at home. Her condition was further complicated by anterolisthesis of L4-L5, multilevel spondylotic changes, and severe radiating pain with neurological compromise. This case highlights the complexity of managing osteoporotic fractures in elderly diabetic patients and emphasizes the role of comprehensive diagnostic imaging, metabolic assessment, and conservative therapeutic approaches in optimizing outcomes while minimizing surgical risks in this vulnerable population(7,8,10,12,14).

II. CASE PRESENTATION

A 72-year-old female patient presented to the Department of Medical Imaging on April 22, 2024, with chief complaints of severe back pain radiating to the left lower limb and shoulder. The patient reported a fall at home between January and February 2024, after which she developed persistent and worsening back pain despite undergoing physiotherapy at a local specialty hospital. The delay in seeking comprehensive evaluation highlights a common pattern in elderly patients who may initially attribute symptoms to normal aging or who experience gradual progression of symptoms following osteoporotic fractures(2,3).

The patient had a significant medical history including type II diabetes mellitus of 25 years duration, hypertension of 25 years duration, hypercholesterolemia, gastroesophageal reflux disease, and known osteoporosis(8,11). Her current medication regimen included Glucophage XR 1g (metformin) for glycemic control, Tulip 20mg (atorvastatin) for hypercholesterolemia, Aspirin 100mg for cardiovascular protection, Gasec 20mg (omeprazole) for gastroesophageal reflux, Januvia 100mg (sitagliptin) for blood glucose control, Coversyl 5mg (perindopril) for hypertension, and Diamicron MR 60mg (gliclazide) for diabetes management. This polypharmacy regimen is typical of elderly patients with multiple chronic conditions and requires careful consideration when planning additional therapeutic interventions(15).

Physical examination revealed severe tenderness at the thoracolumbar junction (T12-L1 region) with a positive straight leg raise (SLR) test indicating nerve root irritation, consistent with radiculopathy secondary to spinal pathology(16). The patient demonstrated difficulty ambulating with visible kyphotic posture, a common finding in patients with vertebral compression fractures that reflects both pain-related postural changes and structural deformity from vertebral collapse(2,5,7). Pain radiated from the lower back to the left lower extremity and shoulder, suggesting multilevel spinal involvement and possible referred pain patterns. There was marked reduction in range of motion in the lumbar spine, accompanied by muscle spasm in the paraspinal region, which is a typical protective mechanism in response to spinal instability and pain(16).

Initial thoracic spine X-ray in anteroposterior and lateral views, performed on April 22, 2024, demonstrated straightening of thoracic curvature, likely secondary to muscle spasm, with diffuse osteopenia involving all visualized vertebrae(4,5)(**Fig 1&2)**. There was anterior wedge compression of the D12 (T12) vertebral body with reduced anterior and central height, a characteristic pattern of osteoporotic compression fractures in which the anterior column fails under axial loading while the posterior elements remain relatively intact(2,5,6). Adjacent vertebral bodies and intervertebral disc heights were preserved, which helped exclude acute traumatic injury at multiple levels and suggested a chronic osteoporotic process. Lumbosacral spine X-ray in anteroposterior and lateral views revealed exaggeration of lumbar lordotic curvature with diffuse osteopenia throughout visualized

bones(4,5). Spondylotic changes with marginal osteophytes were noted at L3-L5 levels, reflecting degenerative changes common in this age group, and atherosclerotic calcification was visible within the abdominal aorta(17). Intervertebral disc heights remained preserved throughout the lumbar spine, suggesting that degenerative disc disease was not a major contributing factor to the patient's symptoms.

DEXA scanning was performed on May 6, 2024, to quantify bone mineral density and assess fracture risk according to WHO criteria(4,17,18). The lumbar spine (L1-L4) demonstrated a T-score of -1.18, placing the patient in the osteopenia range (T-score between -1.0 and -2.5), with a Z-score of -0.06, which was within normal limits for her age(4,17,18). The left hip assessment (femoral neck, trochanter, and Ward's area) showed a T-score of 0.05, indicating normal bone density according to WHO criteria, but the Z-score was 1.65, showing subtle signs of osteoporosis when compared to an age-matched population(4,17,18). These DEXA results revealed a paradoxical finding with osteopenia in the lumbar spine despite relatively preserved hip bone density. The discordant T-scores between spine and hip are characteristic of diabetic bone disease, where bone quality is compromised despite apparently adequate bone mineral density measurements(8,9,11,13). This phenomenon underscores the limitations of DEXA scanning as the sole tool for fracture risk assessment in diabetic patients and highlights the need for comprehensive clinical evaluation that considers disease duration, glycemic control, and presence of diabetic complications(9,13,18).

MRI examination of the thoracolumbar spine without contrast, performed on May 9, 2024, provided detailed soft tissue characterization and confirmed the extent of spinal pathology(7,19)**(Fig 3-8)**. The study revealed complete collapse of the T12 vertebra with loss of vertebral body height and posterior tilt of the superior endplate. Importantly, there was no marrow edema or soft tissue mass, effectively excluding malignant etiology such as metastatic disease or multiple myeloma, as well as acute traumatic fracture which would typically demonstrate bone marrow edema signal on fluid-sensitive sequences(19,20). The absence of marrow edema several months after the initial fall suggested that this represented a chronic osteoporotic compression fracture with no acute component. Grade I anterolisthesis of L4 over L5 vertebra was identified, representing forward slippage of the upper vertebra on the lower vertebra, typically resulting from degenerative changes in the facet joints and intervertebral disc(20). Multilevel disc desiccation and spondylotic changes were present, consistent with age-related degenerative changes of the spine.

The L4-L5 level showed posterior broad-based disc protrusion causing significant bilateral neural foraminal narrowing, with impingement of bilateral traversing and exiting nerve roots resulting in secondary spinal canal stenosis(20,21). This finding explained the patient's radicular symptoms and positive straight leg raise test, as nerve root compression can produce radiating pain, paresthesias, and motor weakness in the distribution of the affected nerve roots(16). The combination of central canal stenosis from disc protrusion and foraminal stenosis from degenerative changes created a "double crush" phenomenon affecting both traversing nerve roots (which continue down the spinal canal) and exiting nerve roots (which exit through the neural foramen at that level)(16). The thoracic spinal cord demonstrated normal bulk and signal intensity throughout its visualized length, an important finding that excluded spinal cord compression or intrinsic cord pathology such as myelomalacia. There were no paraspinal soft tissue masses or abnormal fluid collections to suggest infection or hematoma(16).

Follow-up thoracic and lumbosacral spine X-rays performed on July 30, 2024, approximately three months after initial presentation, demonstrated progression of thoracic kyphosis at the lower thoracic level with persistent D12 vertebral body collapse showing no interval healing(2,7)**(Fig 9-11).** The lack of radiographic healing at three months is not uncommon in elderly osteoporotic patients, particularly those with metabolic derangements, as bone healing capacity decreases with age and is further compromised by diabetes mellitus and vitamin D deficiency(13,22). Continued diffuse osteopenia was present, and atherosclerotic calcification in the abdominal aorta remained unchanged, reflecting the chronic nature of vascular disease in this patient. Left shoulder X-ray in anteroposterior and axial views revealed sclerosis involving the greater tuberosity of the humerus, suggestive of chronic rotator cuff pathology or post-traumatic changes from the fall(23). The glenohumeral and

acromioclavicular joints showed normal alignment, and periarticular soft tissues were unremarkable, suggesting that the shoulder pathology was chronic rather than acute and likely related to degenerative changes or remote injury.

Comprehensive laboratory evaluation was performed to assess nutritional status and bone metabolism, which is essential in the evaluation of any patient with osteoporotic fractures(12,22). Laboratory results demonstrated vitamin D (25-hydroxyvitamin D) deficiency, with levels below the normal range of 30-100 ng/mL, as well as low serum calcium levels below the reference range of 8.5-10.5 mg/dL. Alkaline phosphatase was elevated above the normal range of 30-120 U/L. The elevated alkaline phosphatase in conjunction with low vitamin D and calcium levels is consistent with increased bone turnover associated with fracture healing and underlying metabolic bone disease.[8,9,27] In the context of osteoporotic fractures, elevated alkaline phosphatase reflects increased osteoblastic activity as the body attempts to repair the fracture and remodel bone. Vitamin D deficiency impairs intestinal calcium absorption, contributing to both calcium deficiency and compensatory increases in bone resorption through secondary hyperparathyroidism(22).This creates a vicious cycle where inadequate calcium and vitamin D lead to increased bone turnover, further weakening the skeleton and increasing fracture risk(22).

Given the patient's advanced age (72 years), multiple comorbidities including diabetes mellitus and cardiovascular disease, and the risks associated with surgical intervention in this population, a comprehensive conservative management approach was implemented(7,10,12,14). The decision to pursue non-operative management was based on several factors including the absence of progressive neurological deficit, the patient's medical comorbidities that increase surgical risk, the chronic nature of the fracture without acute instability, and the patient's preference to avoid surgery if possible(7,10). Evidence from randomized controlled trials has demonstrated that conservative management can achieve outcomes comparable to surgical interventions such as vertebroplasty and kyphoplasty in selected patients with osteoporotic vertebral compression fractures, while avoiding surgical complications(10,24).

The pharmacological approach included nutritional supplementation with Milk Calcium 1200 mg and Cholecalciferol (Vitamin D3) 400 IU, both administered orally once daily for 60 days to address the documented deficiencies(12,22). Current guidelines recommend vitamin D supplementation to achieve serum 25-hydroxyvitamin D levels above 30 ng/mL, with higher doses often required in deficient patients to achieve repletion(22). Calcium supplementation of 1000-1200 mg daily is recommended for postmenopausal women with osteoporosis, particularly those with dietary calcium intake below recommended levels(12). Pain management consisted of Paracetamol 500 mg three times daily for 10 days, Celecoxib 200 mg orally twice daily for 15 days as a selective COX-2 inhibitor, and Loxoprofen 100 mg topical application to the affected area once daily for 15 days(14,25). The use of selective COX-2 inhibitors reduces gastrointestinal toxicity compared to non-selective NSAIDs while providing effective analgesia and anti-inflammatory effects(25). Muscle relaxation was achieved with Tolperisone HCl 150 mg orally once daily for 15 days, which acts centrally to reduce muscle spasm without causing significant sedation. To prevent NSAID-induced gastric complications, particularly important given the patient's pre-existing gastroesophageal reflux disease, Esomeprazole 20 mg was administered orally once daily for 15 days as a proton pump inhibitor(26).

The rehabilitation program incorporated multiple therapeutic modalities based on evidence for their efficacy in promoting recovery and preventing disability in elderly patients with spinal fractures(27). Infrared electrotherapy using low-wavelength red light was applied to penetrate deep into bone tissue, with theoretical mechanisms including enhanced ATP production through cytochrome c oxidase activation, increased collagen synthesis, improved microcirculation, and reduced inflammation through modulation of inflammatory mediators(28). Heat therapy was utilized to improve circulation, reduce muscle spasm, and ease stiffness, thereby enhancing mobility and interrupting pain signals through activation of thermal receptors and modulation of pain pathways(27). Kinesiotherapy, comprising passive and active movement exercises including stretching and therapeutic massage, was implemented to rehabilitate injuries and enhance mobility and strength while preventing the deconditioning

that commonly occurs in elderly patients with painful conditions who limit their physical activity(27,29). A specialized geriatric rehabilitation program focusing on fall prevention, balance training, and safe mobility techniques tailored to elderly patients was incorporated to address the multifaceted needs of this age group and reduce the risk of subsequent falls and fractures(29,30). Fall prevention is particularly critical as the risk of subsequent fractures is substantially elevated following an initial osteoporotic fracture, with studies demonstrating a two- to five-fold increased risk of future fractures(30).

A Dynaelnoxa Lumbosacral Corset was provided to the patient, worn for 15 days to provide external spinal support and limit painful motion during the acute recovery phase(5). Spinal orthoses in the management of vertebral compression fractures serve multiple functions including pain reduction through mechanical support, limiting spinal motion to allow healing, improving posture, and providing proprioceptive feedback that may enhance balance and prevent falls. However, prolonged use of rigid bracing is generally discouraged as it may lead to muscle atrophy and deconditioning, hence the limited 15-day duration in this patient's treatment plan(5).

The patient was followed at regular intervals to assess treatment response and functional improvement, which is essential for monitoring progress and adjusting the treatment plan as needed(7,12). Follow-up imaging at 3 months (July 30, 2024) demonstrated a stable T12 compression fracture with no further collapse, which represented a favorable outcome indicating that the conservative management approach successfully prevented progressive vertebral deformity(2,7). Progression of compensatory thoracic kyphosis was noted, which is a common finding as the spine attempts to maintain sagittal balance following vertebral compression fractures(2). No new fractures were identified on follow-up imaging and maintained alignment of other spinal segments suggested adequate spinal stability.

Clinical improvement was substantial, with significant reduction in pain intensity as measured by Visual Analog Scale, improving from 8/10 at presentation to 4/10 at three-month follow-up, representing a clinically meaningful improvement that exceeded the minimal clinically important difference of 1.5-2.0 points on the Visual Analog Scale(31). The patient demonstrated improved mobility and ability to perform activities of daily living, with enhanced overall quality of life as reported subjectively by the patient. Laboratory follow-up confirmed normalization of calcium and vitamin D levels with supplementation, which was essential for ongoing bone health and fracture prevention(12,22). The patient was counseled on the importance of continuing calcium and vitamin D supplementation long-term, maintaining adequate dietary calcium intake, participating in regular weight-bearing exercise appropriate for her age and functional status, and considering anti-osteoporotic pharmacotherapy with bisphosphonates or other bone-active agents to reduce future fracture risk(12,32).

III. DISCUSSION

This case illustrates the complex interplay between osteoporosis, type II diabetes mellitus, and traumatic injury in an elderly patient, highlighting several important clinical considerations in the management of geriatric spinal fractures that have broad applicability to the care of similar patients in clinical practice(2,3,7,8). Type II diabetes mellitus significantly compromises bone microarchitecture through multiple mechanisms that extend beyond simple reduction in bone mass(8,11,13). Despite normal or even elevated bone mineral density on DEXA scanning, as observed in this patient's hip measurements, diabetic patients demonstrate substantially increased fracture risk in epidemiological studies.(9,11,13) This paradox results from accumulation of advanced glycation end products in bone collagen, which occurs when chronic hyperglycemia leads to non-enzymatic glycation of proteins, resulting in cross-linking that reduces bone toughness, flexibility, and ability to dissipate energy(8,13). Additionally, chronic hyperglycemia and insulin resistance promote osteoblast apoptosis through oxidative stress and inflammatory cytokines, while simultaneously enhancing osteoclast-mediated bone resorption through upregulation of RANKL expression, creating a state of high bone turnover with net bone loss despite normal bone density measurements(8,11,13). Diabetic complications including peripheral neuropathy increase fall risk through

impaired proprioception and balance, while microvascular disease may impair bone perfusion and healing capacity(13,30).

The documented deficiencies in vitamin D and calcium created a substrate for accelerated bone loss that compounded the patient's underlying osteoporosis and diabetic bone disease(22). Vitamin D plays crucial roles in calcium homeostasis and bone metabolism beyond its classical function of regulating intestinal calcium absorption(22). Vitamin D receptors are present on osteoblasts where they mediate direct effects on bone formation, and vitamin D also modulates immune function and inflammatory responses that affect bone remodeling(22). Severe vitamin D deficiency leads to decreased intestinal calcium absorption, resulting in mild hypocalcemia that triggers compensatory secondary hyperparathyroidism with increased parathyroid hormone secretion, which in turn increases bone resorption to maintain serum calcium levels at the expense of skeletal integrity(22). The elevated alkaline phosphatase observed in this patient reflects increased osteoblastic activity in response to both the healing fracture and the underlying metabolic bone disease, as alkaline phosphatase is released by osteoblasts during active bone formation(12). While elevated alkaline phosphatase can indicate pathological bone turnover, it also represents the body's attempt to heal the fracture and remodel damaged bone(12).

The presence of abdominal aortic atherosclerotic calcification in our patient reflects systemic vascular disease, which is common in diabetic patients with long-standing hypercholesterolemia and represents a manifestation of chronic cardiovascular disease(17,33). This finding has implications for bone health beyond its cardiovascular significance, as vascular calcification and osteoporosis share common pathophysiological pathways and may represent different manifestations of calcium dysregulation, with calcium being inappropriately deposited in vascular walls while simultaneously being lost from bone(33). The "calcification paradox" describes how patients with osteoporosis often have increased vascular calcification, mediated by shared regulatory factors including osteoprotegerin, receptor activator of nuclear factor-kappa B ligand, and bone morphogenetic proteins(33). Furthermore, vascular disease may impair bone perfusion and compromise bone healing capacity, potentially contributing to the lack of radiographic healing observed at three-month follow-up in this patient(33).

This case demonstrates the value of comprehensive multimodal imaging in characterizing spinal pathology and guiding treatment decisions in complex patients(4,5,7,19). Plain radiography initially identified the vertebral compression and diffuse osteopenia, providing rapid and cost-effective assessment of bony architecture that remains the first-line imaging modality for suspected vertebral fractures(5). DEXA scanning quantified bone mineral density and confirmed osteopenia according to WHO criteria, though it revealed the characteristic discordance between spine and hip seen in diabetic bone disease, highlighting the limitations of DEXA as the sole assessment tool in diabetic patients(4,9,13,18). MRI proved essential for soft tissue characterization, definitively excluding acute marrow edema which would suggest recent trauma or ongoing fracture healing, identifying the extent of neural compromise from the L4-L5 disc protrusion and spinal canal stenosis, and documenting the absence of malignancy or infection which can present with vertebral collapse(19,20). The lack of marrow edema signal on MRI several months after the injury indicated a chronic osteoporotic fracture without acute component, which influenced the decision toward conservative rather than interventional management such as vertebroplasty, which is most effective when performed in the acute or subacute phase when marrow edema is present(10,19,24).

The Grade I anterolisthesis of L4 on L5, combined with broad-based disc protrusion and degenerative changes, created a complex pattern of neural compromise affecting multiple anatomical structures(21). Foraminal narrowing affected exiting nerve roots that pass through the neural foramen at that level, central canal stenosis affected traversing nerve roots that continue down the spinal canal to exit at lower levels, and mechanical instability from the anterolisthesis contributed to dynamic compression during movement(21). This anatomical complexity explained the patient's severe radicular symptoms including radiating pain to the lower extremity and positive straight leg raise test and guided the conservative treatment approach that targeted both mechanical

support through bracing and pain control through multimodal analgesia and physical therapy(16,27). The presence of both central stenosis and foraminal stenosis creates a more challenging clinical scenario than either pathology alone, as decompression at multiple levels may be required if surgical intervention becomes necessary.

The decision to pursue conservative management rather than surgical intervention including vertebroplasty, kyphoplasty, or spinal fusion was multifactorial and based on careful consideration of risks and benefits in this individual patient(7,10,24). At 72 years of age with a 25-year history of diabetes mellitus, hypertension, and hypercholesterolemia, this patient faced significant perioperative risks that increase with age and medical comorbidity burden.(10,15,34) Surgical complications in elderly diabetic patients include poor wound healing due to impaired immune function and microvascular disease, increased infection risk from hyperglycemia and immune dysfunction, instrumentation failure related to poor bone quality, cardiopulmonary complications from anesthesia and surgery, and prolonged recovery times(34). Furthermore, randomized controlled trials have demonstrated that vertebroplasty provides no significant benefit over sham procedure in pain reduction or functional improvement in many patients with osteoporotic vertebral compression fractures, though there may be benefits in selected patients with acute fractures and severe pain(10,24). The absence of progressive neurological deficit and the patient's ability to tolerate conservative therapies supported a non-operative approach, as urgent surgical intervention is typically reserved for patients with spinal cord compression, cauda equina syndrome, or rapidly progressive neurological deficits(7,10).

The pain management strategy employed a combination of non-opioid analgesics to minimize adverse effects while maximizing efficacy, an approach supported by current pain management guidelines that emphasize multimodal analgesia(14,25,35). Celecoxib, a selective COX-2 inhibitor, provided potent anti-inflammatory effects through inhibition of cyclooxygenase-2, the inducible form of the enzyme responsible for prostaglandin synthesis at sites of inflammation, while exhibiting reduced gastrointestinal toxicity compared to non-selective NSAIDs that also inhibit COX-1, which protects the gastric mucosa(25). Prophylactic proton pump inhibitor therapy with esomeprazole further minimized gastric complications, particularly important given the patient's pre-existing gastroesophageal reflux disease and the well-documented increased risk of upper gastrointestinal bleeding with NSAID use in elderly patients(26). Tolperisone, a centrally acting muscle relaxant, addressed the significant muscle spasm component contributing to the patient's pain through modulation of spinal reflexes and reduction of abnormal muscle tone without causing the sedation and cognitive impairment associated with benzodiazepines, which are particularly problematic in elderly patients(36). Topical loxoprofen provided local anti-inflammatory effects through transdermal delivery without significant systemic exposure, offering an additional analgesic modality with minimal systemic side effects(37). The deliberate avoidance of opioid analgesics in this elderly patient reflected current best practices recognizing the substantial risks of opioid therapy in older adults including falls, fractures, cognitive impairment, constipation, and respiratory depression(35).

Correcting vitamin D and calcium deficiencies represented a fundamental intervention addressing the underlying metabolic bone disease that contributed to fracture occurrence and impaired healing capacity(12,22). While calcium supplementation alone cannot reverse established osteoporosis, it provides essential substrate for bone formation and reduces compensatory parathyroid hormone elevation that drives bone resorption(12). Vitamin D supplementation improves calcium absorption from the intestine, directly enhances osteoblast function through genomic and non-genomic mechanisms mediated by the vitamin D receptor, and may have additional benefits for muscle strength and fall prevention through effects on skeletal muscle(22,30). Studies have demonstrated that vitamin D supplementation reduces fall risk by approximately 20% in elderly individuals, likely through improvements in muscle strength and neuromuscular function, providing an additional benefit beyond skeletal health(30). Given the patient's documented deficiencies, the supplementation regimen provided was appropriate for repletion, though higher doses are often required in severely deficient patients to achieve optimal serum 25-hydroxyvitamin D levels above 30 ng/mL, with some experts recommending target levels of 40-60 ng/mL for optimal skeletal health(22).

This case highlights several important clinical implications for managing osteoporotic fractures in elderly diabetic patients that extend beyond the specific details of this case to inform broader clinical practice(3,7,8,12). First, clinicians must recognize that normal DEXA scans do not exclude fracture risk in diabetic patients, and bone quality assessment tools that incorporate diabetes as an independent risk factor along with measures of bone microarchitecture may improve risk stratification beyond traditional bone mineral density measurements(8,9,11,13,18). The development of fracture risk assessment tools such as FRAX has improved fracture prediction by incorporating clinical risk factors beyond bone mineral density, though these tools may still underestimate fracture risk in diabetic patients due to the unique aspects of diabetic bone disease(18,38). Emerging technologies including high-resolution peripheral quantitative computed tomography and trabecular bone score may provide additional information about bone microarchitecture and quality that better predicts fracture risk in diabetic populations, though their clinical utility and cost-effectiveness require further validation(38).

Second, all patients with osteoporotic fractures should undergo comprehensive metabolic evaluation including screening for vitamin D, calcium, parathyroid hormone, thyroid function, and other potential secondary causes of osteoporosis, as identifying and treating reversible factors can improve bone health and reduce future fracture risk(12,22). The high prevalence of vitamin D deficiency in elderly populations, particularly those with limited sun exposure or dark skin pigmentation, makes routine screening cost-effective in the context of fragility fractures(22). Third, the decision between conservative and surgical management must be individualized, considering patient age, comorbidities, functional status, fracture characteristics, and patient preferences, as successful conservative management can achieve satisfactory outcomes while avoiding surgical risks in appropriately selected patients(7,10,24). Shared decision-making that incorporates patient values and preferences is essential in this population where treatment decisions often involve tradeoffs between potential benefits and risks(7,14).

Fourth, optimal management requires multidisciplinary collaboration between radiologists who provide accurate diagnosis and characterization of pathology, endocrinologists or metabolic bone specialists who address underlying bone disease and metabolic derangements, pain specialists or physiatrists who coordinate multimodal pain management and rehabilitation, physical therapists who implement exercise and mobility programs, and primary care providers who coordinate overall care and ensure treatment adherence(7,12,27). This team-based approach recognizes that osteoporotic fractures in elderly patients with multiple comorbidities cannot be optimally managed by any single specialty acting in isolation (7,12). Finally, beyond acute fracture treatment, patients require ongoing management with pharmacological osteoporosis therapy including bisphosphonates, denosumab, or anabolic agents such as teriparatide or romosozumab to increase bone mass and reduce future fracture risk, combined with fall prevention strategies addressing home safety, medication optimization, vision correction, and balance training, as well as continued nutritional optimization and regular physical activity to prevent future fractures(12,30,32,35). The concept of osteoporosis as a chronic disease requiring long-term management rather than a single acute event must be emphasized to patients and caregivers to optimize adherence to preventive strategies(12).

This case report has several limitations that should be acknowledged. The delay in seeking definitive care following the fall (January-February to April) may have allowed progressive vertebral collapse and compensatory changes that might have been prevented with earlier intervention, though it also represents a realistic scenario of how patients often present in clinical practice(2,3,7). Long-term follow-up beyond 3 months would provide additional information about fracture healing, functional outcomes, development of new fractures, and durability of treatment effects, as osteoporotic fractures may take many months to fully heal and patients remain at high risk for subsequent fractures(3,7,30). Quantitative assessment of pain and functional status using validated instruments such as the Oswestry Disability Index or Roland-Morris Disability Questionnaire would have strengthened outcome reporting and allowed comparison with published literature, though the Visual Analog Scale for pain

provides a standardized measure that showed clinically meaningful improvement.(31) Additionally, bone turnover markers including C-telopeptide of type I collagen as a marker of bone resorption and procollagen type I N-terminal propeptide as a marker of bone formation, along with parathyroid hormone levels, would have provided more comprehensive assessment of bone metabolism and helped guide treatment decisions regarding anti-resorptive versus anabolic osteoporosis therapy(12). Measurement of hemoglobin A1c to assess long-term glycemic control in this diabetic patient would have provided additional information relevant to bone health and fracture risk(8,13).

IV. CONCLUSION

This case demonstrates the successful conservative management of a complex osteoporotic T12 compression fracture with concurrent L4-L5 anterolisthesis in a 72-year-old female with multiple comorbidities including type II diabetes mellitus of 25 years duration, hypertension, hypercholesterolemia, and documented vitamin D and calcium deficiency(7–10,22). The case illustrates several key principles that have broad applicability to the management of osteoporotic fractures in elderly patients: comprehensive multimodal imaging including radiography, DEXA, and MRI is essential for complete characterization of spinal pathology, exclusion of alternative diagnoses, and appropriate treatment planning; type II diabetes mellitus significantly affects bone quality through multiple mechanisms beyond what is reflected in bone mineral density measurements, creating increased fracture risk despite apparently adequate DEXA results and necessitating heightened clinical suspicion and comprehensive risk assessment; metabolic assessment revealing vitamin D and calcium deficiency provides therapeutic targets that address underlying pathophysiology and improve bone health; and conservative management combining pharmacological therapy, physical rehabilitation, nutritional optimization, and orthotic support can achieve satisfactory clinical outcomes including meaningful pain reduction and functional improvement while avoiding surgical risks in appropriately selected elderly patients with multiple comorbidities(4,5,7–14,22,27).

The success of conservative management in this case emphasizes the importance of individualized treatment planning that considers patient-specific factors including age, comorbidities, functional status, and preferences; multidisciplinary collaboration between radiologists, endocrinologists, pain specialists, physical therapists, and primary care providers to coordinate comprehensive care; and comprehensive attention to both mechanical aspects of spinal pathology and metabolic aspects of bone health(7,12,14,27). Moving forward, continued advancement in bone quality assessment beyond traditional bone mineral density measurement, fracture risk stratification incorporating diabetic bone disease and other clinical risk factors, and development of targeted therapies for metabolic bone disorders will further improve outcomes for this vulnerable patient population(4,8,12,13,18,38).The integration of emerging technologies such as high-resolution peripheral quantitative computed tomography and novel biomarkers of bone turnover and quality may enable earlier identification of high-risk patients and more precise targeting of interventions(38).

This case contributes to the growing literature supporting conservative management as a viable option for osteoporotic vertebral compression fractures in elderly patients with significant comorbidities, while highlighting the critical importance of addressing underlying metabolic derangements including vitamin D and calcium deficiency, implementing comprehensive rehabilitation strategies that address both pain management and functional restoration, optimizing pharmacological management of comorbid conditions, and providing long-term follow-up with preventive strategies to reduce future fracture risk.(7,10,12,22,27,30,35). The case underscores that osteoporosis should be viewed as a chronic disease requiring sustained management rather than a single acute event, with the goal of optimizing bone health and preventing subsequent fractures that carry substantial morbidity and mortality in the elderly population(3,12,30).

APPENDIX

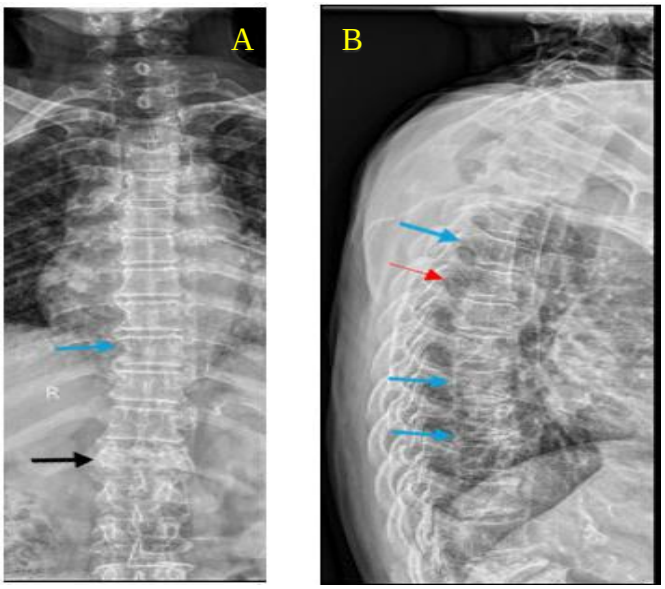

Figure 1:AP Image (A) Blue arrow indicates diffuse osteopenia and black arrow showing anterior wedge compression. Lateral Image (B) Blue arrow indicating diffuse osteopenia and red arrow shows Straightening of thoracic curvature

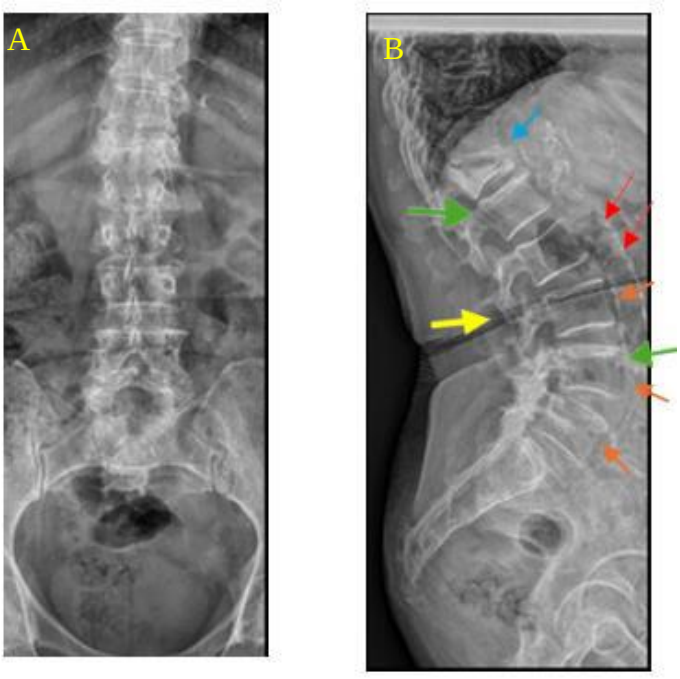

Figure 2: Lateral image () yellow arrow indicates lordotic curvature. Green arrow shows Diffuse osteopenia along with orange arrow marking spondylotic changes from L3 to L5 levels. Red Arrow indicates Atherosclerotic Calcification in Abdominal aorta.

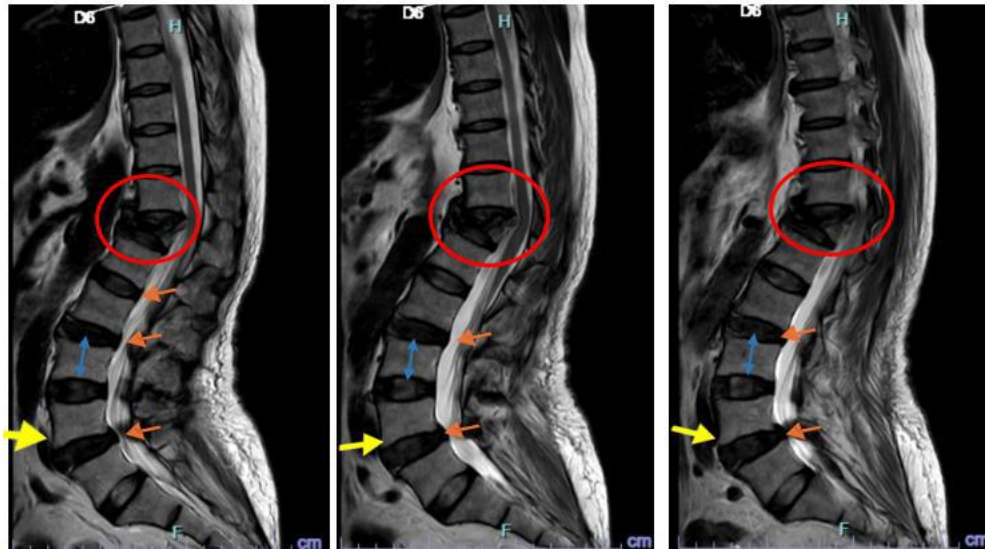

Figure 3: MRI Sequences (eSag_T2-TSE) red marking indicates D12 vertebral collapse with yellow marking indicating anterolisthesis. Orange marking signifies Disc desiccation and blue indicating spondylotic changes.

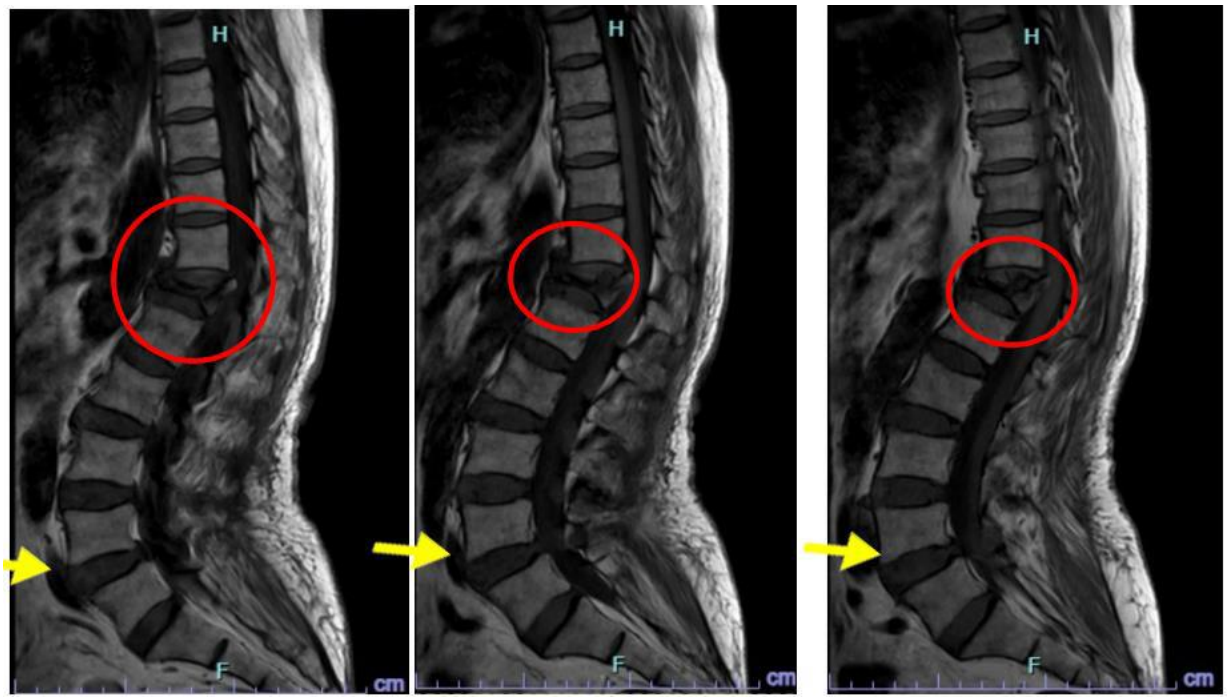

Figure 4: MRI Sequence (eSag_T1_TSE) red marking indicates D12 vertebral collapse with yellow indicating anterolisthesis

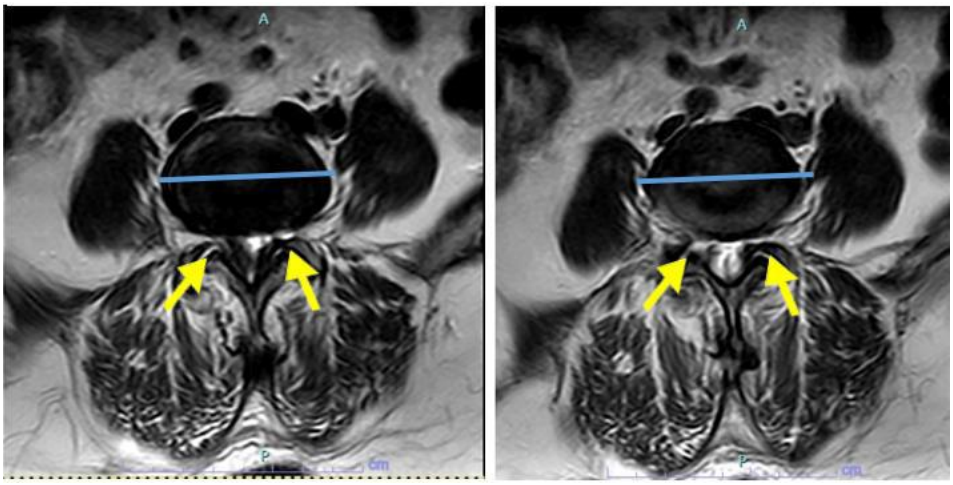

Figure 5:MRI Sequence (eAx_T2_TSE) blue marking signifies posterior broad-based protrusion along with yellow arrows indicating significant narrowing of bilateral neural foramina.

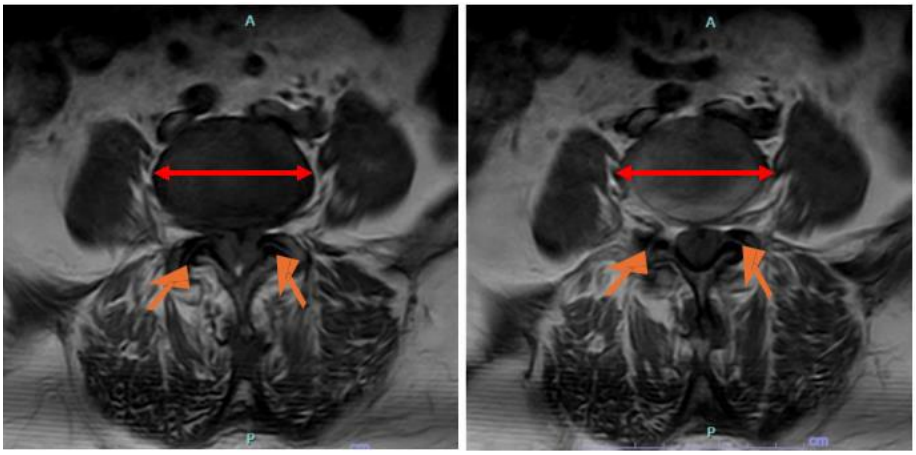

Figure 6:MRI Sequence (eAx_T1_TSE) Red marking indicates posterior broad-based protrusion along with orange arrows indicating significant narrowing of bilateral neural foramina

Figure 7:MRI Sequence (T2W_mdixon) red marking indicates presence of secondary spinal canal stenosis

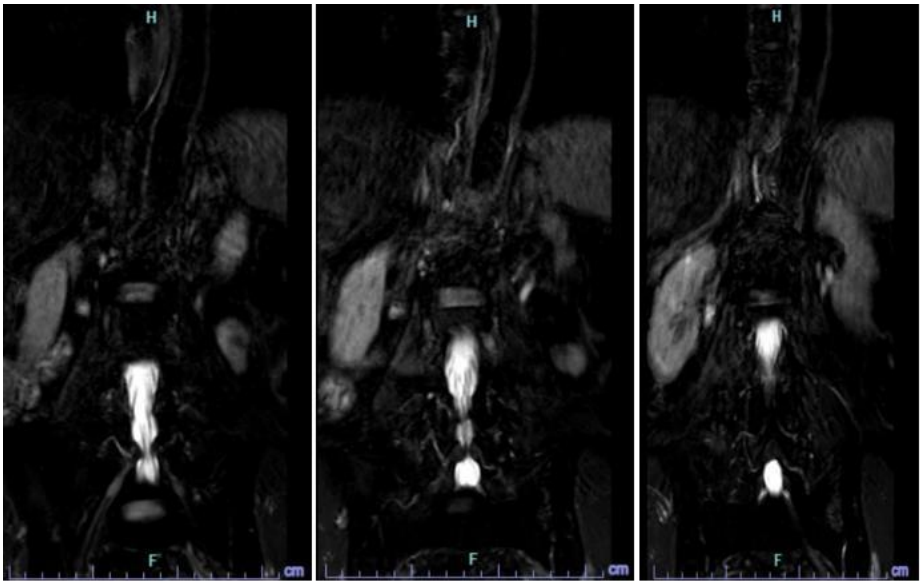

Figure 8:MRI Sequence (T2W_mdixon_cor) image shows the presence of secondary spinal canal stenosis in coronal view

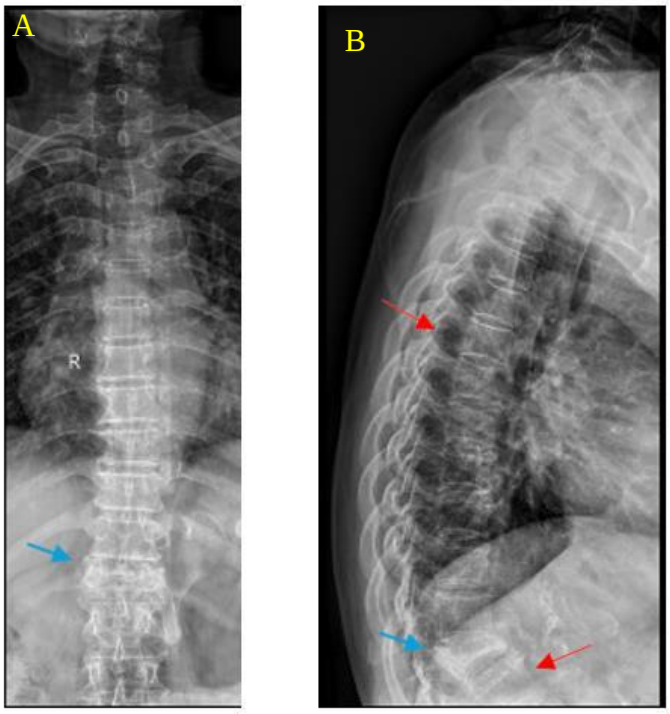

Figure 9:AP image (A) Blue arrow shows osteoporotic collapse, Lateral image (B) Red arrow indicates Straightening of thoracic curvature with increased kyphosis at the lower thoracic and blue arrow shows osteoporotic collapse.

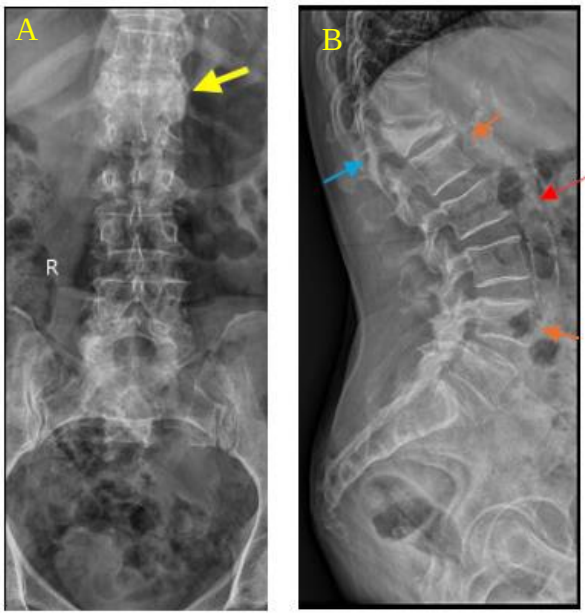

Figure 10:AP Image (A) yellow arrow indicates D12 vertebral body collapse. Lateral Image (B) Orange arrow signifies Diffuse osteopenia, and Blue Arrow indicates thoracic kyphosis. Red Arrow shows Atherosclerotic calcification.

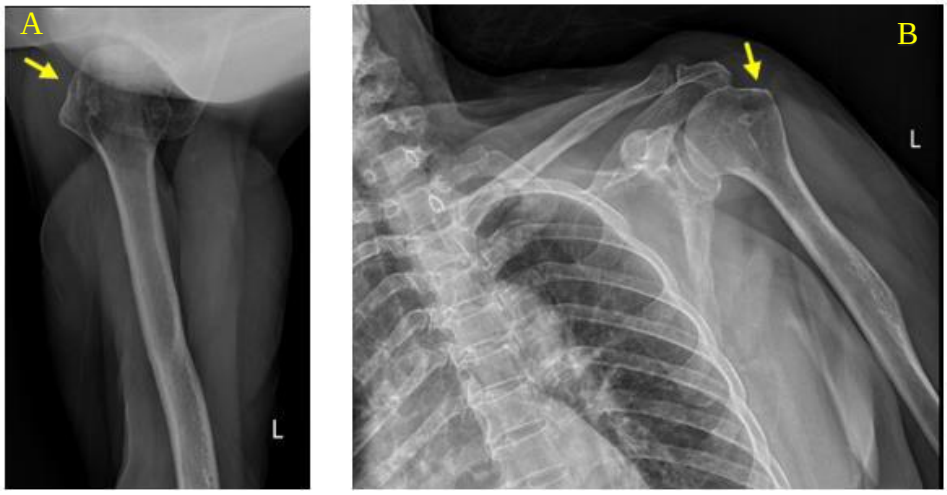

Figure 11:AP image (A) and Lateral Image (B) Yellow Arrow shows Sclerosis involving the greater tuberosity of the humerus

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AUTHORS

First Author –Nikhitha Shetty, MSc. Medical Imaging Technology,
1. Srinivas University, Mangaluru, - 575 001. Karnataka State, INDlA.
2. Department of Medical Imaging Sciences, College of Health Sciences, Gulf Medical University, Ajman, UAE
Nikhithashetty98@gmail.com

Second Author– Nirmala Rajesh, MSc. PhD
Srinivas University, Mangaluru, - 575001. Karnataka State, INDlA
Nimmiraj.naidu@gmail.com

Correspondence Author –Aishwarya SB, MSc. Medical Imaging Technology,
Department of Medical Imaging Sciences, College of Health Sciences, Gulf Medical University, Ajman, UAE
aishusb@gmail.com