DOI: 10.53469/jcmp.2026.08(04).27

Endplate Resorption Accompanied by Bone Cement Fragmentation and Displacement in a 3-Year Follow-up after Osteoporotic Vertebral Compression Fracture Surgery

Kemao Luo[2], Guangqiang Meng[1], Jiawei Wei[3],Pan Zhang[3], Zheng Han[3], Song Wu[3], Xiaobing Song[3], Yunlong Pan[3], Hua Huang[3]

[1]Correspondent, Man, Guizhou Zunyi, 563500 Chief Physician
[2]First author, male Guizhou Zunyi, Han Chinese master's student; Spinal direction
[3]Members of the Orthopedics Department of Daozhen Gelao and Miao Autonomous County Hospital of Traditional Chinese Medicine

Abstract: ***Objective:*** *To report a case of vertebral endplate cracks, gradual absorption, and eventual stress-induced bone cement fracture and displacement following minimally invasive vertebroplasty in a patient with osteoporotic vertebral compression fractures confirmed through imaging follow-up.* ***Methods:*** *A retrospective study was conducted by reviewing the same patient from 2022 to 2025 at Daozhen County Traditional Chinese Medicine Hospital, Guizhou Province. Clinical and imaging data were analyzed for an elderly patient with osteoporotic fractures after surgery, which included X-ray, CT, and MRI showing disappearance of the affected vertebral endplates and pseudoarthrosis formation. The 63-year-old female patient suffered an injury leading to outpatient MRI revealing T12 vertebral damage, compression in T6, T8, L1-4 vertebrae, and local inflammatory changes in L4 and L5. Two-year postoperative follow-up showed disappearance of the upper endplate of the affected vertebra and the lower endplate of the adjacent vertebra along with pseudoarthrosis formation.* ***Results:*** *Imaging showed endplate cracks and fractures in vertebral bodies after minimally invasive osteoporotic compression fracture surgery, with stress leading to endplate absorption and bone cement fracture and displacement.* ***Conclusion:*** *Postoperative endplate cracks, fractures, absorption, and eventual bone cement fracture and displacement following vertebroplasty in osteoporotic vertebral compression fractures are strongly associated with osteoporosis, vertebral stress, and physical activity.*

Keywords: OVCF, Minimally invasive vertebral surgery, Endplate resorption, Bone cement-fracture and displacement**.**

1. Introduction

Osteoporosis is a systemic bone disease characterized by a decrease in overall bone mass, thinning and increased porosity of the endplates, as well as reduced, thickened, and fractured trabeculae. These changes lead to an increased risk of fragility fractures throughout the skeleton. It primarily affects elderly postmenopausal women with reduced bone mass who have not received regular anti-osteoporosis treatment. This results in decreased bone calcium, bone resorption exceeding bone formation, reduced organic bone matrix, and increased mineral content, collectively contributing to heightened bone fragility. The incidence of osteoporotic fractures is positively correlated with age and is significantly higher in elderly female patients compared to males of the same age. With the aging population in China, combined with insufficient dietary calcium intake, inadequate nutrient absorption, and lack of sufficient sunlight exposure for vitamin D synthesis, senile osteoporotic fractures have become increasingly common and have garnered growing attention. The severity of osteoporosis is typically classified based on bone density test results, which categorize the condition into the following four stages [1]: Stage 1: Normal Bone Mass (T-score ≥ -1). Clinical Manifestations: Typically, there are no obvious symptoms such as lower back pain or generalized body pain. Posture is normal, height is unaffected, and daily activities are not impaired. Pathological Features: Bone mineral density (BMD) falls within the normal range with sufficient bone mass. Bone resorption and bone formation are in relative balance, maintaining normal bone strength and a low risk of fracture. Stage 2: Osteopenia / Low Bone Mass (T-score between -1 and -2.5), Clinical Manifestations: Most patients are asymptomatic. Some may experience occasional, mild lower back soreness or pain, which is often overlooked and does not interfere with normal life and activities. Pathological Features: Bone mass begins to decline slowly. Bone resorption slightly outpaces bone formation. Trabeculae become thinner with widened spaces, leading to a decrease in bone strength and a beginning increase in fracture risk. Stage 3: Osteoporosis (T-score ≤ -2.5), Clinical Manifestations: Generalized bone pain may occur, primarily in the lower back, often radiating to the paraspinal muscles. Pain shows little relief when resting in a non-weight-bearing position. It worsens with prolonged standing, severe coughing, or sudden increases in abdominal pressure. Postural changes such as loss of height and kyphosis (dowager's hump) may appear. Minor trauma, like bending over or a fall, can easily lead to fractures.

Pathological Features: BMD is significantly reduced. Trabeculae are fractured and perforated, and the cortical bone thins. The bone microstructure is severely compromised, leading to markedly decreased bone strength and a significantly increased risk of fracture. Stage 4: Severe Osteoporosis (T-score ≤ -2.5 with one or more fragility fractures), Clinical Manifestations: Chronic pain is prominent, such as persistent lower back pain unrelieved by rest. Pain at fracture sites is severe and limits mobility (e.g., inability to walk after a hip fracture, requiring prolonged bed rest). Severe kyphosis and thoracic deformity may occur, potentially impairing respiratory function and causing symptoms like chest tightness and shortness of breath. Quality of life is severely diminished, and the risk of mortality increases.

Pathological Features: BMD is extremely low. Bones are exceedingly fragile with severely compromised microstructure, resulting in a very high risk of recurrent fractures. Prolonged bed rest can also lead to complications such as pneumonia, thrombosis, and pressure ulcers.

2. Osteoporosis is Classified into Type I and Type II

2.1 Type I Osteoporosis (Postmenopausal Osteoporosis)

This condition leads to rapid bone loss throughout the skeleton over a relatively short period in postmenopausal women. The primary characteristic is a reduction in trabecular bone mass, where the trabeculae become thickened, forming a "palisading sign" on imaging. The main cause of this bone loss is the sharp decline in estrogen secretion following menopause, resulting in systemic bone loss and decreased bone mineral density. Within the first five years after menopause, bone mineral density decreases by 2%–3% annually, and the cumulative incidence of osteoporosis reaches 10%–15% [2]. The incidence of postmenopausal osteoporosis increases over time. Approximately 30% to 50% of postmenopausal women are affected by osteoporosis, with the spine, hip, and radius being the most commonly involved sites [3]. It is common in Type I osteoporosis.

Pathogenesis: It is mainly associated with a sharp drop in estrogen levels in postmenopausal women. Estrogen exerts a protective effect on the skeleton and can inhibit osteoclast activity. Postmenopausal estrogen deficiency leads to increased osteoclast activity, with bone resorption being much faster than bone formation, resulting in a gradual decrease in bone mass. The high-risk group for this osteoporosis is typically women within 5 to 10 years after menopause, namely those aged approximately 50 to 70 years, as well as women who experience artificial menopause due to factors such as bilateral oophorectomy, chemotherapy and radiotherapy. Osteoporosis caused by estrogen deficiency is mainly characterized by trabecular bone loss, with a relatively rapid rate of bone loss in the early stage. Common fracture sites: vertebral bodies of the spine, distal radius, and femoral neck.

2.2 Type II Osteoporosis is Also Known as Senile Osteoporosis

Pathogenesis with aging, a decline in sex hormone levels (e.g., postmenopausal estrogen deficiency or androgen hypofunction) leads to a significant reduction in the osteoblasts' capacity for differentiation, proliferation and mineralization, directly impairing bone formation. Additionally, since the lifespan of osteoblasts is shorter than that of osteoclasts, the number of osteoclasts in the elderly gradually exceeds that of osteoblasts. Ultimately, this results in bone formation being insufficient to compensate for bone resorption, causing a progressive loss of bone mass. In addition, elderly patients have poor mobility, which leads to reduced outdoor activities and inadequate sunlight exposure, causing a decrease in vitamin D synthesis. Hypothyroidism and other conditions can also exacerbate osteoporosis. Affected population: It generally refers to osteoporosis that develops after the age of 70, in which both trabecular and cortical bone experience bone mass loss, with bone mineral density decreasing by 2%–3% annually. The lesions are most prominent at the junctions of trabecular and cortical bone such as the vertebral bodies, distal ulna and radius, and hip joints. Among these, fractures of the femoral neck and intertrochanteric region have relatively severe consequences: patients are unable to stand and have to stay in bed for a long time passively, which increases the risk of disuse osteoporosis and other complications [4].

3. Etiology of Osteoporosis

Osteoporosis is a systemic, multifactorial metabolic bone disease, and the research on its etiology has gone through a long process of understanding. Scholars have adopted various approaches to explore the etiology of osteoporosis and attempted to elucidate its pathophysiological mechanisms [5]. 1. Endocrine Factors: (1) Sex Hormones: Estrogen, androgen and progesterone in sex hormones inhibit bone metabolism, promote bone formation and directly suppress osteoclast activity, playing a key role in maintaining bone mass. (2) Calcitonin: Calcitonin is a polypeptide produced by thyroid cells, consisting of 32 amino acid residues. It relieves pain caused by osteoporosis, reduces osteoclast survival and inhibits bone resorption. In addition, calcitonin exerts an analgesic effect through the neural center.2. Genetic Factors: Multiple genetic factors regulate bone metabolism and determine bone mineral density, Vitamin D is an important hormone that regulates bone metabolism, and it promotes the body's rapid absorption of calcium by binding to vitamin D receptors. 3. Nutritional factors: The chewing and digestive functions of the elderly decline, leading to long-term nutritional deficiencies from dietary intake, mainly involving insufficient consumption of protein, vitamins, inorganic minerals (calcium, phosphorus), and trace elements.4. Disuse factors: Regular outdoor exercise over the long term for the elderly can increase skeletal weight-bearing of the body and prolong sunlight exposure time. Increased skeletal weight-bearing can raise the turnover rate of osteogenesis and boost the activity of osteoblasts; ultimately, it can significantly enhance bone remodeling and bone mass, improve bone strength and bone mineral density, and reduce the risk of fractures.

4. Treatment Options for Osteoporotic Fractures

Fracture reduction, internal fixation with a nail-rod system, regular long-term weight-bearing exercise and scientific anti-osteoporosis therapy constitute the basic therapeutic principles for osteoporotic vertebral compression fractures. (OVCF）. For osteoporotic fractures, an individualized and targeted treatment plan should be formulated before surgery based on the fractured vertebral body, the degree of osteoporosis, fracture classification and the patient's underlying medical conditions. Non-surgical or surgical treatment can be adopted, with the goal of restoring the pre-injury quality of life as early as possible. The optimal treatment should prioritize minimal invasiveness, rapid functional recovery, pain relief and enhanced recovery after surgery (ERAS) [6]. The surgical approach should be selected comprehensively with reference to imaging data before operation. Percutaneous Vertebroplasty (PVP) and

Percutaneous Kyphoplasty (PKP) yield comparable therapeutic effects; however, PKP is recommended for patients with severe vertebral compression fractures (vertebral body compression exceeding 1/3) who require restoration of the fractured vertebral height and are complicated with kyphosis [7]. Percutaneous Kyphoplasty (PKP) can significantly reduce the risk of bone cement leakage during surgery. In addition, intraoperative biopsy can be performed to differentiate it from spinal pathological fractures. The commonly used minimally invasive treatments are osteoporotic vertebral augmentation surgeries, which mainly include Percutaneous Vertebroplasty (PVP) and Percutaneous Kyphoplasty (PKP). At present, there is no conclusive evidence on whether there are differences in the therapeutic effects between these minimally invasive surgeries and non-surgical treatments for OVCF [8]. The nonunion rate of OVCF fractures is relatively low, usually below 5% to 10%, but patients with severe osteoporosis or other comorbidities may have a significantly higher nonunion rate [9]. Patients with a significant decrease in bone mineral density are more prone to fracture nonunion, which is particularly common in elderly patients [9]. The incidence of vertebral clefts in patients with ordinary osteoporosis is approximately 10%-30%, while in those with severe osteoporosis or multiple-level vertebral compression fractures, the incidence can exceed 30%. The presence of clefts usually indicates a significant decrease in bone mineral density and impaired local fracture healing, a condition that is more prominent in the thoracolumbar segment with high dynamic load [10]. Without scientific and regular anti-osteoporotic treatment, the incidence of osteoporotic vertebral compression fractures (OVCF) is positively correlated with age. Indications for minimally invasive vertebral augmentation surgery include failure of conservative treatment for 6 months, and the acute phase (6 weeks post-injury) where persistent low back pain remains and the patient is still unable to ambulate [10]. Unstable and nonunion fractures in OVCF; intravertebral cystic degeneration with extensive necrosis of vertebral body cells (osteoporotic Kummell's disease without neurological injury); prolonged bed rest is not advisable. For elderly patients opting for surgery, it is recommended that the procedure be performed as early as possible after injury (within 6 weeks for patients aged over 65 years and within 3 weeks for those aged over 70 years) to shorten bed rest duration and reduce complications. Conservative treatment: is indicated for mild to moderate vertebral compression fractures confirmed by imaging examinations, with mild symptoms and signs, and patients who can resume ambulation after 24 hours of analgesic treatment. Conservative treatment modalities include prolonged bed rest, trunk bracing, administration of analgesics and anti-osteoporotic drugs, early ambulation, and rehabilitation exercises. Regular reexaminations and evaluations of the vertebral body are required to obtain an accurate understanding of changes in vertebral compression [11].

5. Prevention and Treatment of Osteoporotic Fractures

5.1 Lifestyle Therapy

Lifestyle interventions include smoking cessation and alcohol reduction, as smoking and excessive alcohol consumption can lower bone mass and significantly increase the risk of fractures. Regular weight-bearing exercises such as brisk walking and jogging help improve bone mineral density. Resistance training: Light-weight training can stimulate muscle contraction, thereby indirectly enhancing bone strength. Balance training: Practices like Tai Chi and yoga reduce the risk of falls and aid in fall prevention. Pharmacological. Therapy: Pharmacological therapy is the core of osteoporosis management, with the primary goals of inhibiting bone resorption, promoting bone formation, directly increasing bone mass and reducing fracture risk.

5.2 Basic Therapy

Calcium and vitamin D supplementation is the cornerstone of basic therapy, especially for patients with osteoporotic fractures in the early stage. Calcium serves as the fundamental raw material for bone formation. The recommended daily calcium intake varies according to an individual's age, gender and health status. Adequate calcium supplementation is particularly necessary for the elderly, postmenopausal women and patients with chronic diseases [12]. Data shows that daily supplementation with 500-1000 mg of calcium and 800-1000 IU of vitamin D3 can significantly improve bone mineral density and reduce the incidence of fractures in people aged 65 and above. Chinese guidelines recommend a daily calcium intake of 600-800 mg for adults, and the intake should be increased to 1000-1200 mg for those over 50 years old. The average dietary calcium intake of residents in China is about 400 mg per day, meaning an additional daily supplementation of 500-600 mg is required.

Calcium carbonate has high solubility in an acidic environment and high bioavailability in the human body. Calcium citrate causes mild gastrointestinal side effects, has poor water solubility, and reduces the risk of kidney stones; however, its calcium content is low, making it suitable for patients with hypochlorhydria and a history of kidney stones. Calcium phosphate dissolves slowly in gastric acid, can release Ca^{2+} stably to minimize the impact on blood calcium levels, and can be directly converted into hydroxyapatite. Calcium chloride has extremely high solubility, dissolves rapidly to release a large amount of calcium ions, and features high absorption efficiency, which can quickly raise blood calcium concentration, but it is highly likely to cause gastrointestinal irritation. Calcium supplementation should be avoided in patients with hypercalcemia and hypercalciuria [13].

5.3 Pharmacological Prevention and Treatment of Osteoporotic Fractures

It includes antiresorptive agents, bone-forming agents, drugs with other mechanisms, and traditional Chinese medicines (TCMs) [14]. Antiresorptive Agents: 1) Bisphosphonates: Common agents include alendronate sodium, ibandronate sodium, risedronate sodium, and zoledronic acid. These drugs can increase bone mass in the vertebrae and hip joints and significantly reduce the risk of fractures. Patients must remain upright for more than 30 minutes after taking bisphosphonates. The use of these drugs is contraindicated in patients with esophageal stricture and those unable to stand or sit upright.

Zoledronic acid is available as 5 mg/vial for the treatment of osteoporosis, while the 4 mg formulation is indicated for metastatic tumors (a non-osteoporotic indication). Its use is contraindicated in patients with a creatinine clearance rate of less than 35 ml/min. 2) Calcitonins [15]: These agents inhibit osteoclast activity, effectively slowing down acute bone loss and alleviating associated pain. Clinically commonly used preparations include salmon calcitonin and eel calcitonin. 3) Receptor Activator of Nuclear Factor-κB Ligand (RANKL) [16]: It exerts its effects by binding to its receptor RANK, primarily activating the metabolism, activation and survival of osteoclasts. In the process of bone turnover, the RANKL/RANK signaling pathway serves as a key regulatory mechanism that modulates bone mass by enhancing osteoclastic resorption. The representative drug is denosumab: it inhibits the formation, functional activity and survival time of osteoclasts, thereby reducing bone resorption and attenuating bone mass loss, indirectly enhancing bone strength with no impact on renal function. 4) Selective Estrogen Receptor Modulators (SERMs) [17]: SERMs [16] can exert protective effects similar to endogenous estrogen, such as slowing bone loss, thereby increasing bone mineral density. A commonly used medication is raloxifene. 5) Estrogen Therapy [17]: By reducing osteoclast activity and stimulating osteoblast activity, estrogen can effectively improve bone mineral density, demonstrating significant efficacy particularly in postmenopausal women. It may be an option for patients undergoing menopause.

5.4 Bone-Forming Agents: Parathyroid Hormone Analogues, such as Teriparatide and Abaloparatide [18]

This class consists of human recombinant N-terminal fragments (amino acids 1–34), which retain the active region of parathyroid hormone (PTH). PTH analogues are among the few available bone-forming agents and, unlike anti-resorptive drugs (e.g., bisphosphonates), can reverse bone loss. Treatment duration is typically limited to two years. They work through intermittent administration to promote osteoblast activation and bone formation, thereby reducing excessive bone resorption and mitigating potential side effects. Teriparatide promotes bone matrix formation and mineralization [19], enhancing the growth rate, quality, and function of bone tissue, thereby restoring normal bone structure and mechanical properties. It significantly reduces the risk of vertebral and hip fractures in postmenopausal women. For women aged 65 and older with a bone mineral density T-score below -2.5, postmenopausal individuals who have experienced vertebral or hip fractures, or those who have sustained osteoporotic fractures while on bisphosphonate therapy, the use of bone-forming agents is recommended.

5.5 Other Mech Anistic Drugs [20]

1 Active vitamin D and its analogues: The main condition of active vitamin D is calcium phosphorus absorption in the small intestine, enhancing renal calcium reabsorption, and bone calcium release. This maintains calcium phosphorus metabolic balance in the body. This is particularly important for maintaining bone density, preventing osteoporosis, and promoting bone strength; such as osteotriol, suitable for elderly and osteoporosis patients with renal dysfunction; significantly reduces bone fracture risk. Vitamin K group: [21] Primarily refers to Vitamin K2 (menaquinones, **MKs**), which can be synthesized by intestinal bacteria in the human body. Among these, **MK-7** (a long-chain form of Vitamin K2) enhances bone mineralization and reduces the risk of osteoporosis by activating the carboxylation of bone proteins (such as osteocalcin), thereby promoting the effective utilization of calcium.

5.6 Traditional Chinese Medicine (TCM) [22]

In the early stage of osteoporotic fracture, TCM with effects of promoting blood circulation, removing stasis, reducing swelling, and alleviating pain may be considered in addition to conventional anti-osteoporosis drugs. In the later fracture stage, herbs that strengthen tendons and bones, supplement qi and nourish blood, and benefit the liver, spleen, and kidneys are selected. Prepared rehmannia root and epimedium help regulate bone metabolic balance. Eucommia bark and astragalus root exert anti-inflammatory effects and modulate endocrine function. Drynaria rhizome and morinda root are commonly used. Signaling pathways and molecular mechanisms: Substances such as deer antler glue can influence related pathways. Components like ginseng, solomon’s seal, and drynaria rhizome are able to regulate bone metabolism through multiple targets. Compounds such as icariin and tetramethylpyrazine have become potential targets for new drug development due to their ability to modulate multiple bone metabolism signaling pathways. Most TCM herbs show positive effects in increasing bone density, inhibiting bone loss, and improving bone microstructure. Meanwhile, compared with conventional drugs, they generally have fewer side effects and are more suitable for long-term use.

6. Rehabilitation Strategies for Osteoporotic Fractures

6.1 Functional Exercise

Acute Phase (0-6 weeks): Protection and Stabilization Phase. Primary Goals: Reduce pain, control inflammation, promote fracture healing, and prevent secondary injury. Exercise Interventions: Appropriately restrict movement of the fracture site using braces, splints, or surgical internal fixation devices. Perform non-weight-bearing or light weight-bearing activities, such as active range-of-motion exercises for joints away from the fracture site (e.g., ankle pumps). Gentle isometric strength exercises can be performed if pain allows, to prevent muscle atrophy. Recovery Phase (6-12 weeks): Range of Motion Expansion and Strength. Enhancement. Phase. Primary Goals: Gradually restore soft tissue and joint range of motion, and initiate localized muscle strength training. Exercise Interventions: Gradually introduce low-intensity, non-resistance active exercises, such as joint flexion-extension functional training. Guide patients in balance training and improving proprioception (e.g., single-leg standing for hip fracture patients). Encourage limited weight-bearing activities, adjusting rehabilitation progress based on medical advice. Functional Restoration Phase (12 weeks and beyond)

6.2 Comprehensive Rehabilitation and Functional Training Phase

Main Objectives: Restore normal daily activity capacity and prevent long-term complications such as muscle weakness, joint stiffness, and secondary joint injury. Training Interventions: Gradually incorporate moderate- to high-intensity resistance training based on the patient's specific condition (e.g., exercising with light loads). Implement functional activity training that simulates daily movements (such as stair climbing and sit-to-stand transitions). For patients with reduced bone density, recommend weight-bearing exercises aimed at improving bone density, such as brisk walking or standing jumps. Hip Fracture Rehabilitation Acute Phase: Bed rest is advised to minimize weight-bearing. Ankle pump exercises can be initiated early postoperatively to prevent thrombosis. Recovery Phase: Gradually introduce weight-bearing standing exercises, combined with active hip flexion and extension movements, along with seated and standing balance training. Functional Restoration Phase: Focus on lower limb muscle strength training (e.g., leg extensions using elastic resistance bands) to enhance gait stability and stride control. Research Support: Multiple studies indicate that combined balance and resistance training significantly improves recovery after hip fracture surgery, enhancing quality of life and preventing secondary falls. Vertebral Fracture Rehabilitation. Acute Phase: Avoid vigorous bending and twisting motions; a back brace can be used to stabilize the injured area. Recovery Phase: Incorporate lightweight, weight-bearing back extensor muscle training (using exercise balls or rehabilitation equipment) to prevent muscle weakening due to prolonged inactivity. Functional Restoration Phase: Combine whole-body flexibility and balance exercises (such as yoga or Tai Chi) to mitigate long-term risks associated with osteoporosis. Research Support: Evidence-based research recommends multi-component exercise programs, including balance training and spinal stabilization exercises, which can significantly alleviate chronic pain and functional impairment. Summary Osteoporotic fractures commonly affect sites such as the vertebrae, hip, and forearm, leading to a significant decline in bone density and strength. This increases the risk of subsequent fractures and reduces patients' quality of life. As a non-pharmacological intervention, functional exercise helps alleviate post-fracture dysfunction and prevent further fractures by enhancing muscle strength, balance, flexibility, and bone density.

7. Case Information

Patient Case Summary. Patient: Ms. He, female, 63 years old. Chief omplaint: Presented with "low back pain and limited mobility for 4 days following a fall." Diagnosis and Treatment Timeline with Key Findings: Initial Visit (November 2, 2022): Examination: Thoracolumbar Digital Radiography (DR). Findings: Suggested degenerative changes in the thoracolumbar spine; flattening of the T6 and T12 vertebral bodies; flattening of the L1-L3 vertebral bodies; osteoporosis. Further Investigation (November 6, 2022): Examination: Lumbar spine contrast-enhanced Magnetic Resonance Imaging (MRI). Findings: Revealed injury to the T12 vertebral body; flattening of the T6, T8, and L1-L4 vertebral bodies; localized inflammatory changes in the L4 and L5 vertebral bodies. Diagnosis and Admission: Admitted with a diagnosis of "T12 vertebral compression fracture." Treatment (Post-admission): Pre-operative: Completed necessary examinations, and surgical contraindications were ruled out. Surgery (November 2022): Underwent "Percutaneous Vertebroplasty (PVP) for T12 vertebral fracture." Post-operative Review (Initial): Lumbar DR showed "postoperative changes in the T12 vertebra." Post-operative Follow-up Examinations: Review in January 2023: Imaging still indicated "postoperative changes in the T12 vertebra." Review in June and September 2024: Imaging revealed new findings: disappearance of the T12 superior endplate and the T11 inferior endplate, with significant enhancement indicating vertebral calcification.

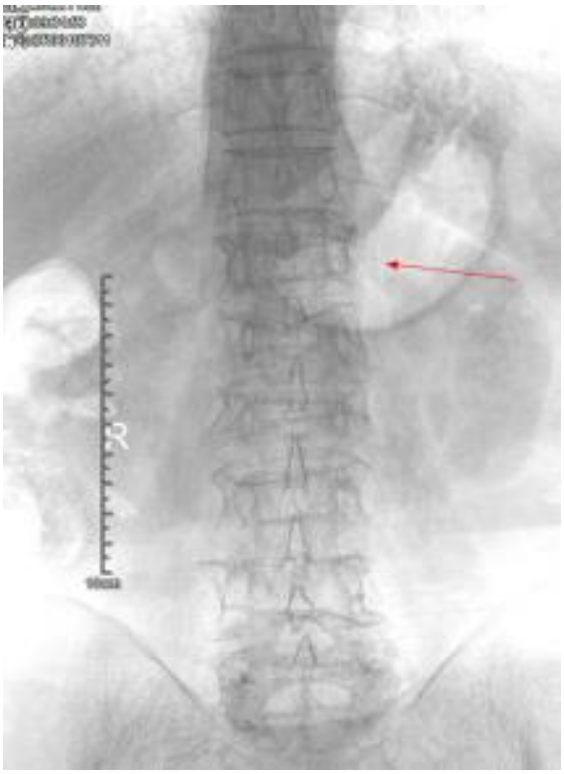

Figure 1

Figure 2

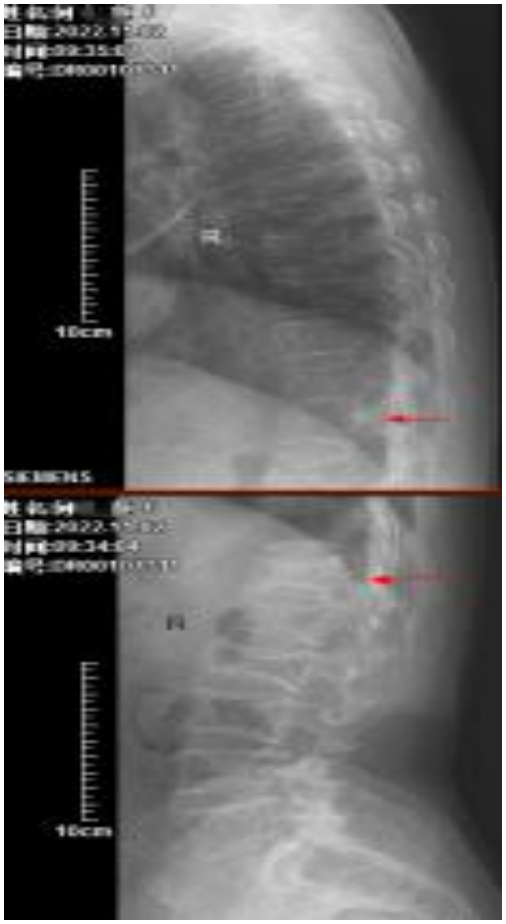

Figure 3

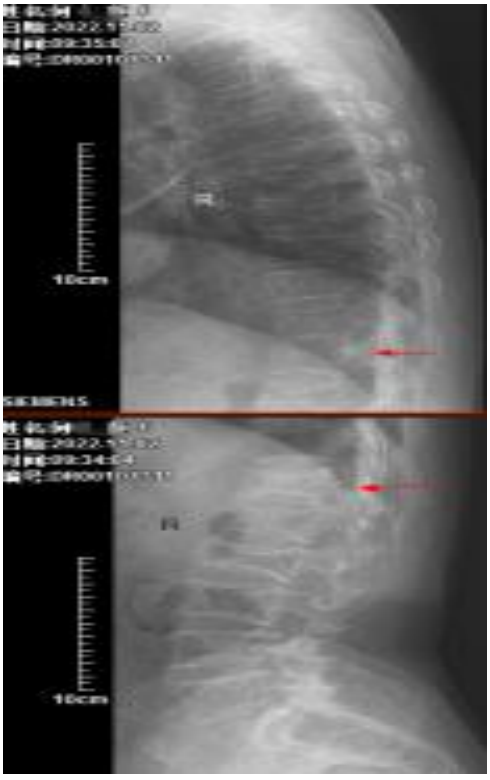

Figure 4

Figures 1-4: The patient's preoperative DR images from November 2022 revealed a vertebral compression fracture with an L1 endplate fracture. The fracture type was classified as a **Grade II biconcave deformity** according to the Genant visual semi-quantitative assessment. The superior endplate was still present but exhibited a fissure.

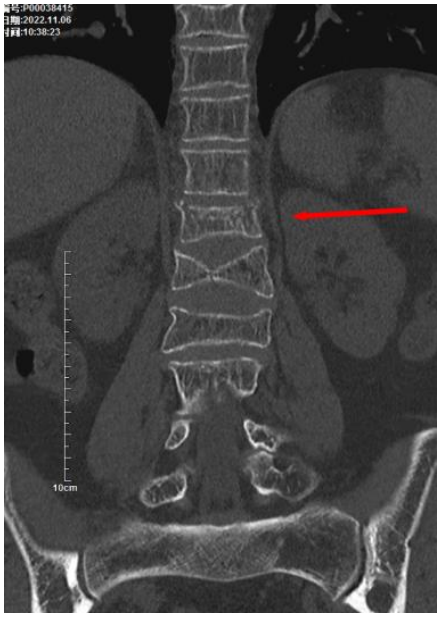

Figure 5

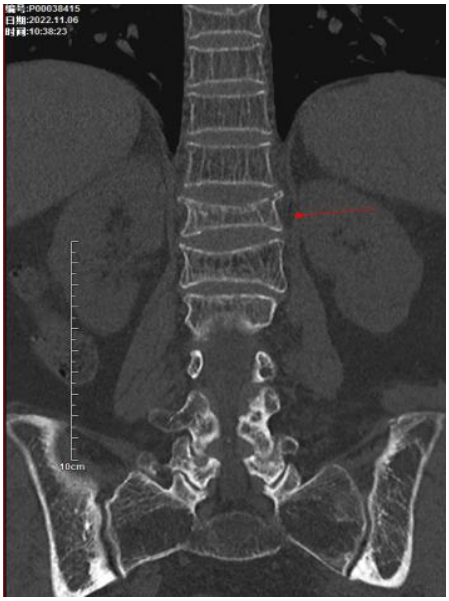

Figure 6

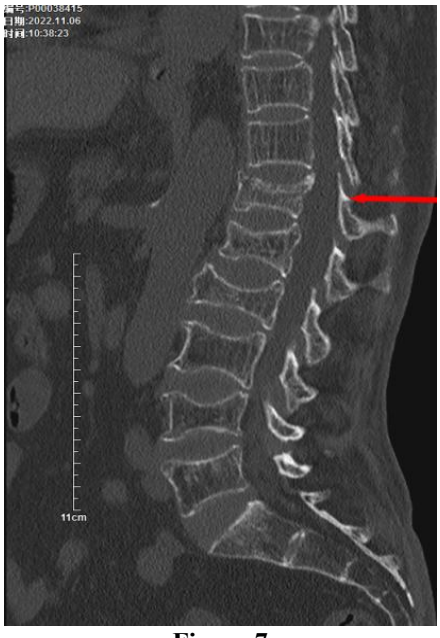

Figure 7

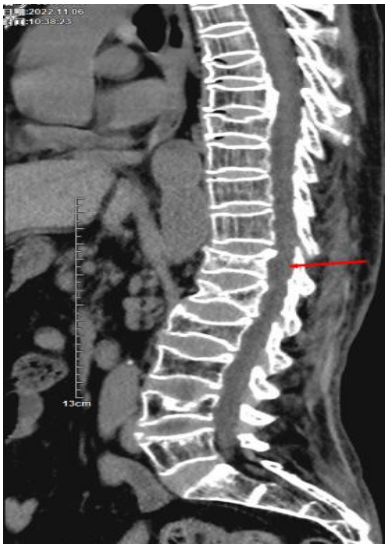

Figure 8

Figures 5-8: The patient's preoperative CT images from November 2022 showed a fresh vertebral fracture with an endplate fracture. A clear fissure sign in the superior endplate was particularly evident.

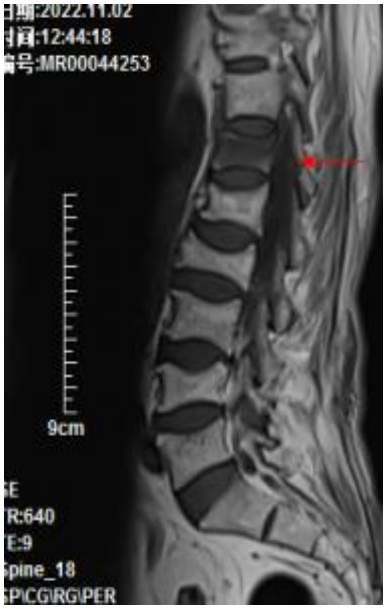

Figure 9

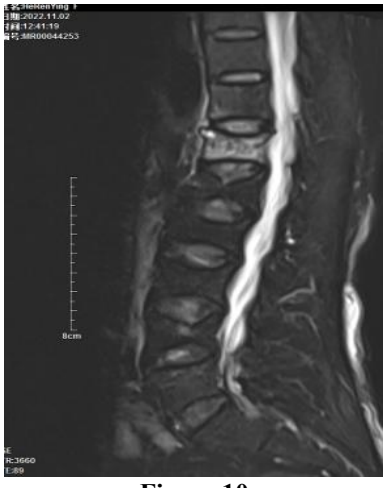

Figure 10

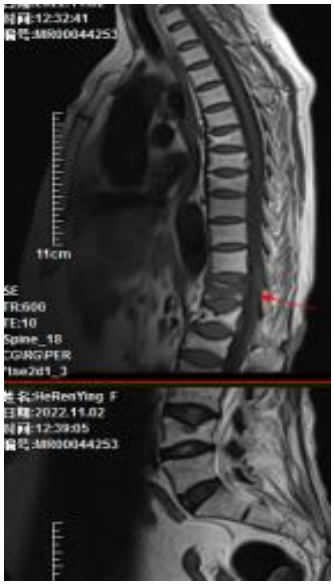

Figure 11

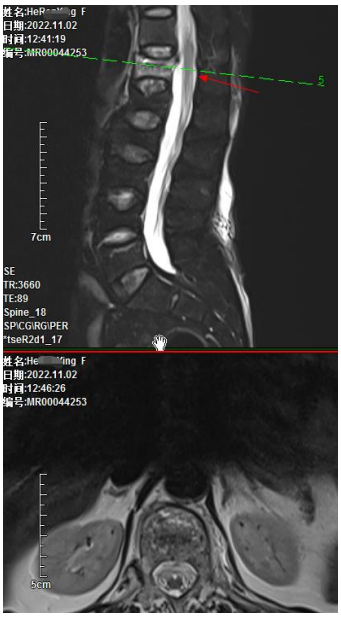

Figure 12

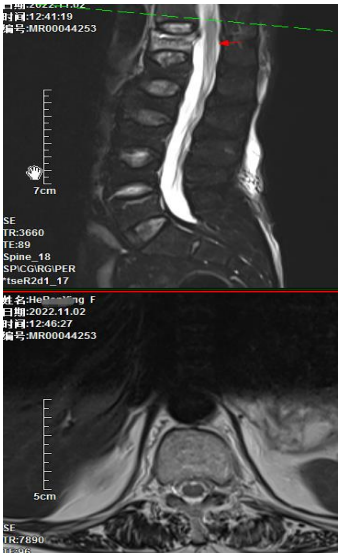

Figure 13

Figures 9-13: The patient's preoperative MRI (T1- and T2-weighted images) from November 2022 indicated a fresh vertebral fracture, and a fissure fracture of the superior

endplate was present.

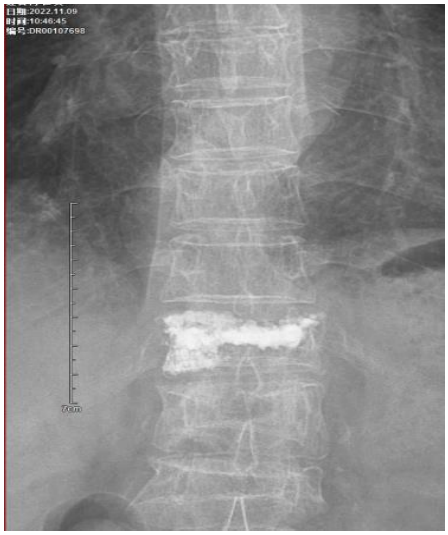

Figure 14

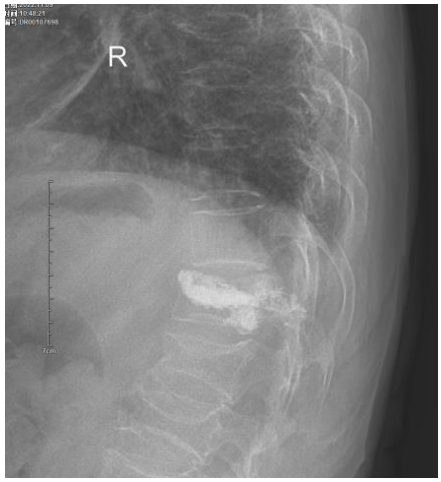

Figure 15

Figures 14-15: Postoperative review of the patient's thoracic spine DR shows that the bone cement filling in the T12 vertebral body appears adequate, occupying more than half of the vertebral body volume. There is suspected leakage of bone cement into the right superior endplate region. The T11 endplate remains intact.

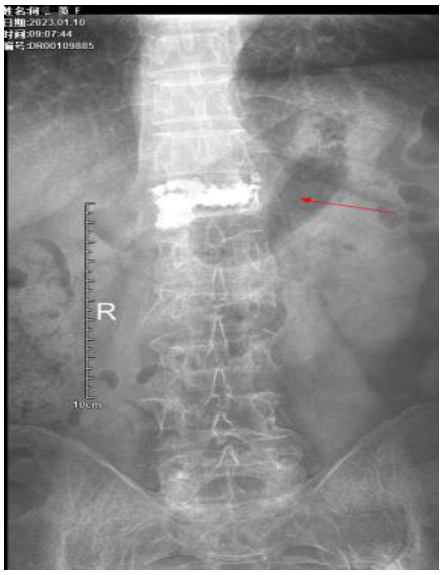

Figure 16

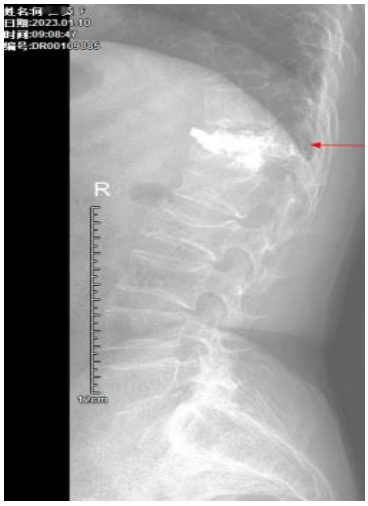

Figure 17

Figures 16-17: The patient's 2-month postoperative follow-up lumbar spine DR (January 2023) shows adequate vertebral body strengthening with well-dispersed bone cement and no resorption of the endplate observed. Compared to the immediate postoperative images, an increased thoracolumbar flexion angle is noted, and the bone cement in T12 appears elongated anteriorly.

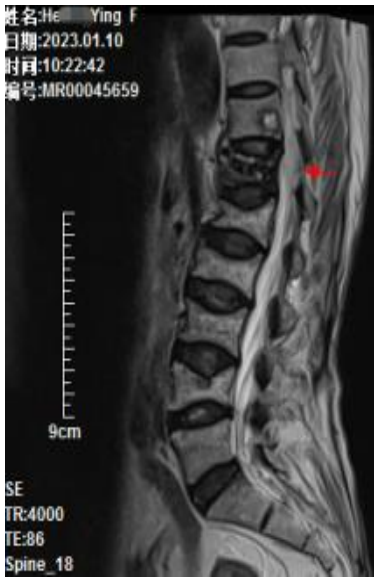

Figure 18

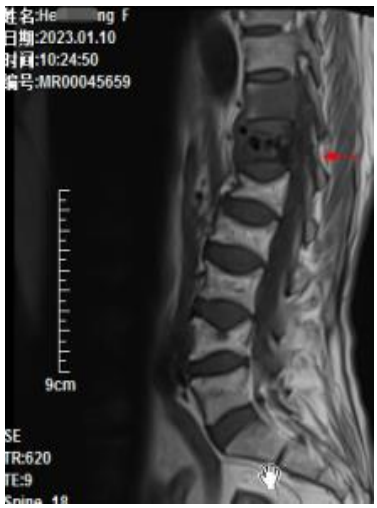

Figure 19

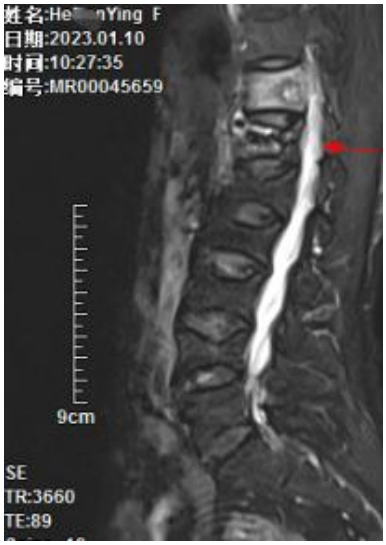

Figure 20

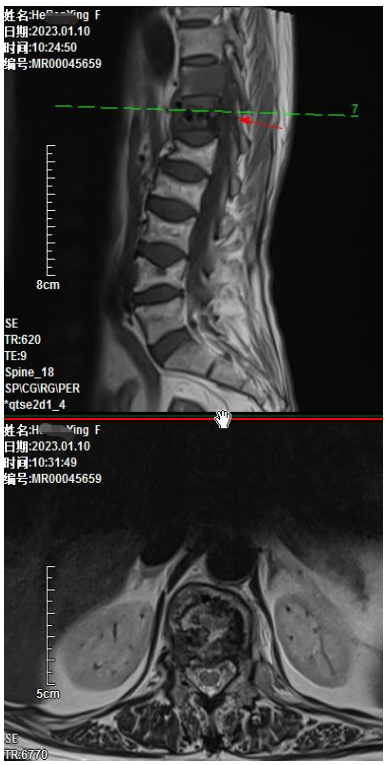

Figure 21

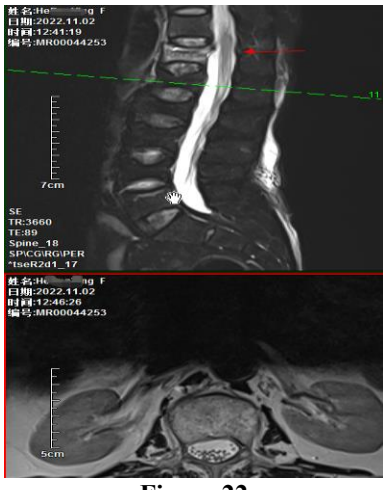

Figure 22

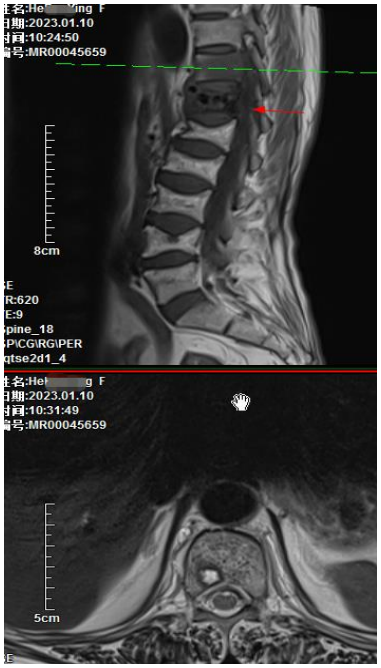

Figure 23

Figures 18-23: The patient's 2-month postoperative follow-up MRI (January 2023) shows adequate vertebral body strengthening with well-dispersed bone cement and no resorption of the endplate. No significant new fractures are observed. Posterior to the T11 vertebral body, new Schmorl's nodes and vertebral osteomyelitis are noted, but no significant endplate disappearance or curvature changes are evident.

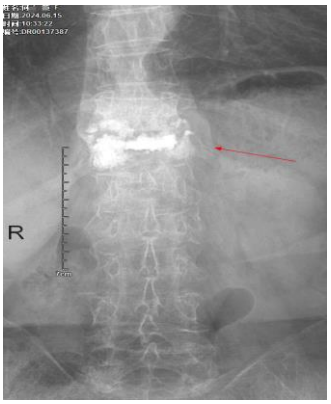

Figure 24

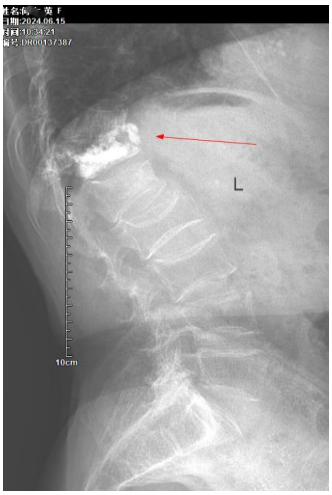

Figure 25

Figures 24-25: The patient's 19-month postoperative follow-up DR (June 2024) shows an increased vertebral

curvature. The anterior one-fourth of the T11 vertebral body is embedded into T12. The bone cement in T12 extends beyond the right endplate and shows a trend of fragmentation and displacement. The T11 vertebral endplate remains intact.

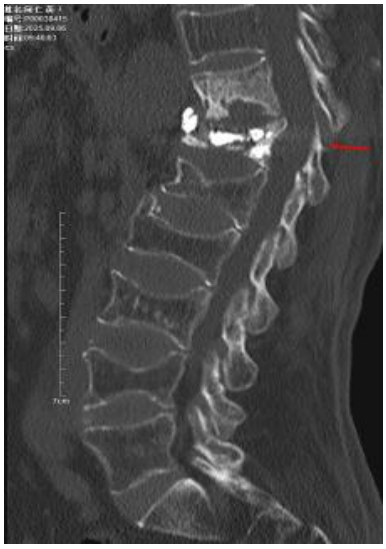

Figure 26

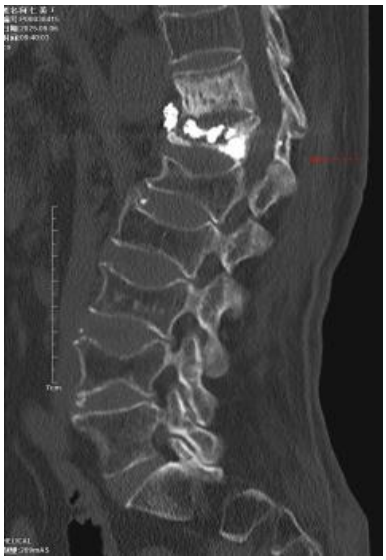

Figure 27

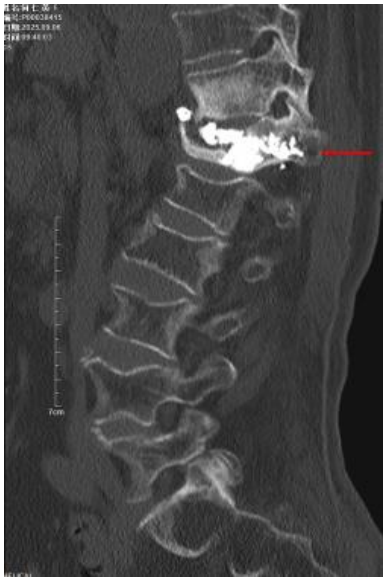

Figure 28

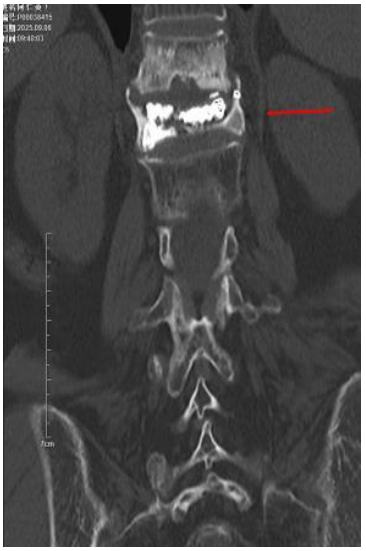

Figure 29

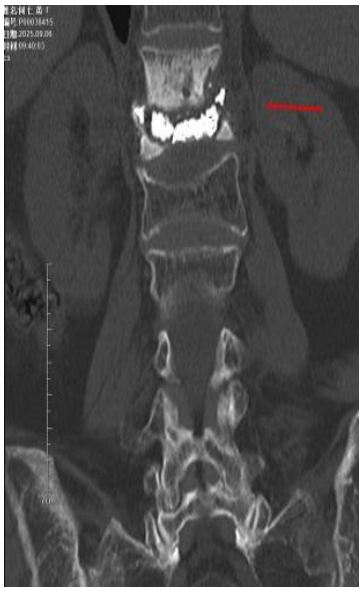

Figure 30

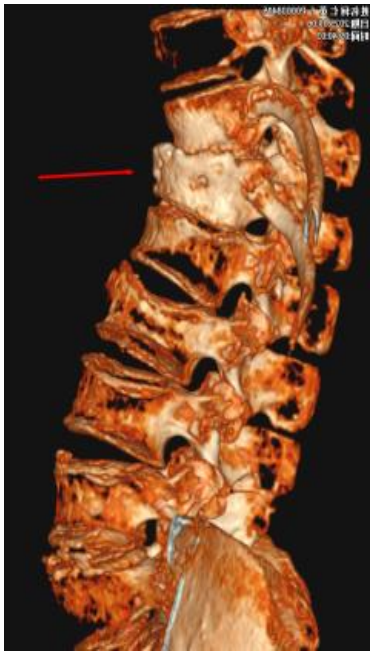

Figure 31

Figures 26-31: The patient's CT scan at 42 months

post-operation (September 2025) reveals: Possible resorption of the anterior one-fourth of the L1 superior endplate. Disappearance of the T12 superior endplate with significant collapse of the vertebral body. The bone cement shows evident fragmentation and displacement. A markedly increased body flexion angle. Initiation of resorption in the T11 inferior endplate. Clear evidence of chronic osteomyelitis in the T11 vertebral body.

Reasons for Endplate Disappearance: As a critical biomechanical structure for load transmission within the vertebra, the loss of endplate integrity leads to abnormal stress distribution. This is clearly associated with subsequent fractures in adjacent vertebrae. Studies indicate that endplate injury can increase stress concentration in adjacent vertebrae by up to 35%. Clinical research shows that patients with endplate fractures have a 2.8 times higher risk of subsequent vertebral fractures [23]. Pathological processes include: post-traumatic avascular necrosis, vertebral lamina fissures, nonunion of vertebral fractures, vertebral collapse, and pseudarthrosis formation in adjacent vertebrae [24].

7.1 Endplate Calcification and Reduced Blood Supply [25]

Intervertebral disc degeneration is closely associated with endplate calcification. The calcification of the endplate significantly impairs microcirculation and reduces the efficiency of nutrient transport, thereby depriving the surrounding vertebral bone of metabolic support. This vicious cycle further accelerates the combined degeneration of the endplate and the intervertebral disc [25]. The Endplate Serves as the Nutrient Pathway for Intervertebral Discs [26]: Intervertebral discs are avascular (lack blood vessels), and their nutrition primarily depends on the exchange of substances with adjacent vertebral bodies through the endplates. When tuberculous lesions involve the endplates, their normal structure and function are disrupted, leading to compromised nutrient supply to the intervertebral discs.

7.2 Intervertebral Disc Degeneration Exacerbates Endplate Destruction [27]

Nutritional disturbance leads to metabolic dysfunction of cells within the intervertebral disc, resulting in progressive degeneration and conditions such as annulus fibrosus rupture. Concurrently, disc degeneration further impacts the endplate, causing abnormal pressure distribution and stress loads on it, which accelerates the destruction of the endplate and ultimately leads to its disappearance.

7.3 Activation of Inflammatory Factors [28-30]:

During intervertebral disc degeneration, herniated discs can induce the release of local inflammatory factors, including mediators such as interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α). These factors not only directly damage endplate cells but also accelerate matrix degradation and induce cell apoptosis through oxidative stress mechanisms, further compromising the structural integrity of the endplate. They can also stimulate osteoclast activity, leading to the proliferation and heightened activation of osteoclasts. These osteoclasts then excessively resorb the bone tissue of the endplate, resulting in reduced bone mass, structural damage, and ultimately the disappearance of the endplate.

7.4 Increased Concentration of Mechanical Stress [31-32]

When intervertebral discs degenerate, their water content decreases and elasticity diminishes, leading to significant alterations in biomechanical properties. Degenerated discs are unable to effectively disperse applied loads, causing greater mechanical stress to be transferred onto the vertebral endplates. This localized stress concentration can induce micro-damage, collapse, or even fracture of the trabeculae within the endplate. Furthermore, in cases of severe osteoporosis, the thinning and increased fragility of vertebral trabeculae make the endplate more susceptible to deformation, collapse, or even complete disappearance under spinal pressure.

This section does not discuss abnormalities or disappearance of the endplate caused by congenital endplate dysplasia, or conditions such as endplate ischemia or infarction during growth (e.g., due to sickle cell anemia). Structural or functional abnormalities in the vertebral endplate may occur during embryonic development. Such conditions can lead to morphological changes in the vertebral body and issues related to spinal function. If the endplate structure fails to withstand pressure normally, it may cause localized pain or chronic discomfort, manifesting as restricted movement — particularly during bending or twisting. Endplate abnormalities may also result in reduced vertebral height or compression of nerve roots, leading to symptoms such as leg numbness or weakness. Magnetic resonance imaging (MRI) and computed tomography (CT) can reveal abnormalities such as irregular endplate thickness, abnormal cartilage tissue morphology, or reduced vertebral height.

Note: The disappearance of the vertebral endplate may be accompanied by symptoms such as pain and restricted movement. Diagnosis should be based on a comprehensive analysis of imaging findings (e.g., MRI, CT). If related symptoms occur, they should be addressed appropriately. The intravertebral vacuum cleft sign is primarily observed in the vertebral bodies at the thoracolumbar junction. This sign appears as a radiolucent area on imaging, typically larger in the coronal plane. The intravertebral vacuum cleft is considered indicative of local bone ischemia associated with unhealed vertebral collapse and is less commonly seen in vertebral collapse of neoplastic or inflammatory origin. Recognizing this sign in clinical practice can help suggest bone ischemia and avoid unnecessary investigations for more severe causes of vertebral collapse.

8. Summary

In summary, the postoperative occurrence of endplate rupture in the affected vertebra, resorption/disappearance of adjacent endplates, and pseudarthrosis formation at the involved vertebral segment in patients with osteoporotic vertebral compression fractures result from a combination of various etiological and pathological factors. However, osteoporosis serves as the fundamental pathological basis. Modern imaging techniques allow for better tracking and identification of the timing of endplate disappearance, the progression of pathological changes, and the process of vertebral alteration

in these patients.

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