Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202522268

Integrated Pharmacy, Nursing, and Physical Therapy Approaches to the Management of Vertebral Compression Fractures

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Abstract

Background: Vertebral Compression Fractures (VCFs) are the most common osteoporotic fracture, posing a significant clinical and public health burden, especially in aging populations. They result from compromised vertebral bodies failing under load, leading to pain, kyphotic deformity, functional decline, and an increased risk of subsequent fractures.

Aim: This article aims to provide a comprehensive overview of the integrated management of VCFs. It synthesizes information on the etiology, pathophysiology, clinical evaluation, and evidence-based treatment strategies, emphasizing a multidisciplinary approach involving pharmacy, nursing, and physical therapy.

Methods: The study is a detailed narrative review. It consolidates current evidence and clinical guidelines on VCF management, covering classification systems (e.g., AO Spine, Osteoporotic Fracture classification), diagnostic imaging (X-ray, CT, MRI), and a spectrum of management options from conservative care to surgical intervention.

Results: The review finds that effective VCF management is multifaceted. Conservative management, including analgesia, bracing, and physical therapy, is first-line for stable fractures. For unstable or painful fractures refractory to conservative care, vertebral augmentation procedures (vertebroplasty, kyphoplasty) or surgical stabilization are effective. The prognosis is highly variable and depends on fracture morphology, underlying etiology (osteoporosis, trauma, malignancy), and patient comorbidities. A successful outcome hinges on an interprofessional team to optimize bone health, manage pain, restore function, and prevent complications.

Conclusion: A patient-centered, interprofessional approach is fundamental to managing VCFs, integrating medical, rehabilitative, and surgical strategies to improve outcomes and quality of life.

Keywords: Vertebral Compression Fracture, Osteoporosis, Kyphoplasty, Interprofessional Care, Pain Management, Spinal Rehabilitation

Introduction

Vertebral compression fractures (VCFs) represent the most common form of osteoporotic fracture and constitute a major clinical and public health challenge worldwide.[1] They typically occur when compromised vertebral bodies fail under axial or compressive loading, most often involving the anterior column and resulting in a wedge-shaped deformity of the vertebral body. This structural collapse alters normal spinal alignment, leading to segmental kyphosis, loss of height, and progressive sagittal imbalance. From a pathophysiological standpoint, reduced bone mineral density and impaired bone microarchitecture weaken vertebral trabeculae, decreasing the threshold for fracture even under low-energy or routine daily activities such as bending, lifting light objects, or minor falls.[1] Consequently, VCFs are increasingly recognized not only as isolated skeletal injuries but also as markers of systemic skeletal fragility and underlying

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Receive Date: 31 October 2025, Revise Date: 22 November 2025, Accept Date: 24 November 2025

metabolic bone disease. The burden of VCFs on individuals and healthcare systems is substantial, particularly in aging populations with a high prevalence of osteoporosis.[2] Epidemiological data demonstrate a steep age-related increase in vertebral fracture incidence, affecting both sexes, though women remain disproportionately affected due to accelerated postmenopausal bone loss and generally lower baseline bone mass.[2] Population-based studies have reported that vertebral fracture prevalence increases from approximately 3% in individuals younger than 60 years to nearly 20% in those aged 70 years and older, emphasizing age as a critical determinant of risk.[2] This trend is exacerbated by global demographic transitions toward older age structures, with a corresponding rise in the absolute number of individuals at risk of osteoporotic fractures. Recent large-scale epidemiological analyses have shown an approximate 14% increase in fracture incidence between 2009 and 2019, underscoring VCFs as an expanding clinical and economic concern in aging societies and highlighting the urgency of optimized prevention, early detection, and management strategies.[3]

Clinically, VCFs are associated with acute and often severe back pain, impaired mobility, and significant functional limitations that may lead to long-term disability.[1] The resulting kyphotic deformity can compromise pulmonary function, increase intra-abdominal pressure, and alter load distribution across adjacent vertebrae, thereby predisposing patients to additional fractures and further postural deterioration.[1] These structural and functional consequences frequently translate into loss of independence, reduced participation in activities of daily living, increased risk of falls, and a marked decline in health-related quality of life. Importantly, a single vertebral fracture substantially elevates the risk of subsequent vertebral and non-vertebral osteoporotic fractures, creating a "fracture cascade" that amplifies morbidity, mortality, and healthcare utilization over time.[1],[2] This cascade effect positions VCFs as sentinel events that should trigger comprehensive evaluation for osteoporosis and other modifiable risk factors to prevent future fractures. From a health system perspective, VCFs contribute significantly to direct and indirect healthcare costs through emergency visits, hospital admissions, pharmacological treatment, spinal imaging, interventional procedures, and prolonged rehabilitation requirements.[3] Conservative management—anchored in multimodal analgesia, spinal bracing, and structured rehabilitation—remains the first-line approach for most stable fractures without neurological compromise.[1] Nevertheless, a subset of patients experiences persistent pain, progressive deformity, or instability, necessitating minimally invasive procedures such as vertebroplasty or kyphoplasty, or, less commonly, more extensive surgical stabilization.[1],[3] Given the complex interplay of pain control, functional restoration, fall prevention, and secondary fracture prevention, optimal care for patients with VCFs increasingly relies on an interprofessional model that integrates medical, pharmacological, nursing, and rehabilitative expertise. Within this framework, evidence-based guidelines emphasize early recognition of VCFs, rapid initiation of appropriate therapy, and coordinated long-term follow-up to address bone health, functional recovery, and quality of life.[1],[3] Understanding the current evidence surrounding VCF epidemiology, pathophysiology, clinical consequences, and management is therefore essential for guiding comprehensive, patient-centered, and resource-conscious care in contemporary clinical practice.

Etiology

Vertebral compression fractures (VCFs) most commonly arise from osteoporosis, which remains the predominant cause of fragility fractures in older adults due to progressive declines in bone mineral density and deterioration of trabecular architecture.[3] Osteoporotic bone demonstrates reduced mechanical strength and impaired ability to withstand axial loads, resulting in structural failure of the vertebral body under forces that would be considered physiologically normal in healthy individuals. Consequently, routine daily activities such as bending, lifting light objects, or even minimal-impact events like coughing or minor falls may precipitate a fracture. The clear association between advancing age and increasing fracture incidence reflects the cumulative effects of bone remodeling imbalance, hormonal changes, decreased physical activity, and nutritional deficiencies that contribute to skeletal fragility in aging populations.[3] As demographic trends shift toward a larger proportion of elderly individuals, the prevalence of osteoporosis-related VCFs continues to rise globally, underscoring the importance of early intervention and preventive strategies. While osteoporosis accounts for the majority of VCFs, younger individuals experience these fractures through a distinctly different mechanism, typically involving high-energy trauma. Motor vehicle collisions, sports-related injuries, or falls from significant heights generate substantial axial compression and flexion forces capable of overwhelming the structural capacity of otherwise healthy vertebrae.[4][5] In such cases, the magnitude of force required to produce a VCF is considerably higher, reflecting the relative robustness of the vertebral bodies in younger populations. This creates a bimodal distribution pattern of VCF etiology: low-energy osteoporotic fractures in the elderly and high-energy traumatic fractures in younger adults, each with differing clinical implications, management priorities, and prognostic considerations.[6] Additionally, pathological processes involving neoplastic infiltration of the vertebral bodies

represent another important etiological category. Conditions such as multiple myeloma, metastatic carcinomas, or primary bone malignancies compromise vertebral integrity by disrupting normal bone turnover, promoting osteolysis, and weakening cortical and trabecular structures, thereby increasing susceptibility to compression failure even under physiological loading conditions.[7] Recognition of these varied etiologies is essential for appropriate diagnostic evaluation, as therapeutic strategies differ substantially between osteoporotic, traumatic, and neoplastic fractures.

The Thoracolumbar Injury Classification and Severity (TLICS) Score

Injury Category	Point Value
Injury morphology	
Compression	1
Burst	2
Translation or rotation	3
Distraction	4
PLC status	
Intact	0
Injury suspected or indeterminate	2
Injured	3
Neurologic status	
Intact	0
Nerve root involvement	2
Spinal cord or conus medullaris injury	
Incomplete	3
Complete	2
Cauda equina syndrome	3

Scores: <4 non operative, 4 non operative or operative, >4 operative; PLC- posterior ligamentous complex

Fig. 1: Thoracolumbar Injury Classification and Severity Score.

Anatomical and Biomechanical Characteristics

The thoracolumbar junction, typically spanning T11 to L2, is especially prone to VCFs owing to its unique anatomical and biomechanical properties. As the transition zone between the relatively rigid, rib-stabilized thoracic spine and the more mobile lumbar spine, this region experiences concentrated mechanical stresses during flexion, extension, and axial loading. The abrupt shift in rigidity results in heightened shear and compressive forces that predispose the vertebral bodies in this region to structural failure under both traumatic and osteoporotic conditions. Understanding these biomechanical principles is fundamental to interpreting fracture mechanisms and predicting patterns of instability. Anatomically, the spinal column is traditionally conceptualized as comprising three key structural columns. The anterior column includes the anterior longitudinal ligament, anterior annulus fibrosus, and anterior portion of the vertebral body. The middle column consists of the posterior vertebral body, posterior annulus, and posterior longitudinal ligament, while the posterior column comprises the ligamentum flavum, neural arch, facet joints, and posterior ligamentous complex. Historically, VCFs were characterized by injury confined solely to the anterior column, implying inherent mechanical stability. Disruption of the middle column, by contrast, was associated with burst fractures, which signify a higher degree of instability and pose greater risks for retropulsion of bony fragments into the spinal canal.

Contemporary classification systems have revised this traditional distinction. The AO Spine Thoracolumbar Injury Classification System designates compression injuries as type A fractures, with subtypes A3 and A4 involving posterior vertebral body wall compromise, thereby extending the definition beyond isolated anterior column involvement.[8] Similarly, the more recent Osteoporotic Fracture (OF) classification—developed by the Spine Section of the German Society for Orthopaedics and Trauma (DGOU) and adopted by AO Spine—integrates osteoporotic fracture patterns that include posterior vertebral wall disruption. Within this framework, OF types 3 through 5 specifically identify osteoporotic fractures with posterior wall involvement, highlighting their mechanical significance and shifting the conceptualization of VCFs from strictly stable, anterior column injuries to a broader spectrum of potentially unstable fracture morphologies.[9][10] This expanded understanding plays a critical role in guiding clinical decision-making, as posterior wall involvement often necessitates more vigilant monitoring, advanced imaging, and consideration of interventional or surgical management strategies.

Epidemiology

Vertebral compression fractures (VCFs) represent the most common fragility fractures encountered in clinical practice and remain a significant global health concern due to their rising incidence and substantial burden on aging populations.[11] In the United States alone, approximately 1 to 1.5 million VCFs occur annually, illustrating the pervasive nature of these injuries within both community and hospital settings.[11] The prevalence increases markedly with age, particularly among postmenopausal women, in whom osteoporosis and accelerated bone loss substantially heighten fracture vulnerability. Epidemiological data indicate that nearly 25% of women aged 50 years and older have sustained at least one VCF, either symptomatic or incidentally detected during imaging performed for unrelated conditions.[11] Among individuals aged 80 years and older, the prevalence rises dramatically to between 40% and 50%, underscoring the close relationship between aging, reduced bone mineral density, and vertebral fragility.[12] These statistics highlight VCFs as a

hallmark of skeletal aging and a sentinel event in the natural history of osteoporosis. Anatomical distribution also exhibits consistent epidemiological patterns. Recent reports indicate that approximately 60% to 75% of VCFs occur at the thoracolumbar junction—the region extending from T12 to L2—where the abrupt transition between the rigid thoracic spine and more mobile lumbar segments creates biomechanical vulnerability.[12] An additional 30% of fractures occur within the L2 to L5 region, reinforcing the susceptibility of the lower spine to compressive and flexion forces. These regional patterns reflect both mechanical strain distribution and age-related degeneration, providing valuable insight for diagnostic vigilance and preventive strategies.

In contrast to older adults, where low-energy mechanisms predominate, VCFs in younger patients typically arise from high-energy trauma. Approximately 50% of spinal fractures in this population result from motor vehicle collisions, while another 25% stem from falls from significant heights, workplace injuries, or athletic trauma.[11] This etiological contrast underscores the bimodal distribution of VCFs, where age and trauma severity play defining roles. Notably, in elderly patients, up to 30% of VCFs are reported to occur while the individual is in bed or performing minimal activities, reflecting the profound skeletal fragility associated with advanced osteoporosis.[12] The epidemiologic burden of VCFs is expected to intensify as global demographics shift toward older age structures. Currently, an estimated 10 million Americans carry a diagnosis of osteoporosis, with an additional 34 million classified as having osteopenia, placing them at increased risk of future fractures.[11] With continued increases in life expectancy, the number of individuals at risk is projected to grow substantially in the coming decades. Population-based studies report annual VCF incidence rates of approximately 10.7 per 1000 women and 5.7 per 1000 men, reflecting both the sex disparity in bone density decline and the broader public health implications of osteoporosis.[11] Collectively, these epidemiological trends emphasize the urgent need for early detection, targeted preventive interventions, and comprehensive management strategies to mitigate the rising clinical and socioeconomic impact of vertebral compression fractures [11][12].

Pathophysiology

The pathophysiology of vertebral compression fractures (VCFs) involves the failure of the vertebral body when applied axial forces exceed its structural capacity, leading to deformation, collapse, or fragmentation of the vertebral body. Under typical circumstances, the vertebral column is designed to withstand significant compressive loads through the combined strength of cortical bone, trabecular architecture, and intervertebral disc support. However, when these forces become excessive—such as during acute trauma—or when the bone is significantly weakened, as in osteoporosis, the vertebra becomes unable to resist compression, resulting in structural failure. Axial loading is the most common mechanism and may be accompanied by flexion or rotational forces around an instantaneous axis of rotation, producing complex strain patterns that preferentially affect the anterior portion of the vertebral body. In most VCFs, the initial failure point occurs in the anterior column, where the trabecular bone is more metabolically active and structurally less dense than the posterior vertebral body. When compressive forces exceed the load-bearing threshold of this region, the anterior height of the vertebral body decreases, creating the characteristic wedge-shaped deformity associated with compression fractures. With greater force intensity, collapse may extend into the middle column, resulting in burst fractures, which involve retropulsion of bone fragments into the spinal canal and carry a higher risk of neurological compromise. These fracture patterns reflect the interplay between the magnitude of mechanical loading and the intrinsic integrity of the vertebral body [11][12].

The resulting kyphotic deformity alters normal spinal biomechanics, shifting the center of gravity anteriorly and increasing the bending moment applied to adjacent vertebrae. This altered alignment magnifies stress on contiguous motion segments, predisposing them to subsequent fracture and contributing to a progressive cycle of structural deterioration. The compensatory muscular and ligamentous demands placed on the spine to maintain posture further exacerbate pain and functional impairment. In osteoporotic individuals, this pathophysiological cascade is amplified, as significantly reduced bone mineral density weakens trabecular connectivity and cortical thickness, lowering the threshold for fracture even under normal physiological loading. Repeated microfractures and progressive deformity ultimately contribute to a downward spiral of compromised biomechanics, diminished mobility, heightened fall risk, and increased susceptibility to additional VCFs. Thus, the pathophysiology of VCFs reflects an intricate interaction between mechanical forces, bone quality, and spinal alignment, emphasizing the clinical importance of early identification and intervention to prevent fracture progression and cumulative disability [11][12].

History and Physical

Clinical Features

Evaluation of vertebral compression fractures (VCFs) begins with a structured and comprehensive history and physical examination, tailored to the mechanism of injury and the overall clinical context. In high-energy trauma, assessment should commence only after the patient has been stabilized using Advanced Trauma Life Support (ATLS) principles, giving priority to securing the

airway, ensuring adequate breathing, and maintaining circulation. Once life-threatening conditions have been addressed, focused attention can be directed toward the spine. At this stage, clinicians must perform a detailed neurological assessment, including evaluation of motor strength, sensory function, and deep tendon reflexes in all extremities, while also assessing sphincter tone, bladder function, and bowel control to detect early signs of spinal cord or cauda equina compromise. Particular care is required in polytrauma patients, where concomitant injuries to the head, thorax, abdomen, pelvis, or long bones may coexist and distract from or mask spinal pathology. The physical examination of the spine should be systematic and gentle. Inspection may reveal bruising, abrasions, or swelling over the affected region, as well as postural changes such as focal kyphosis or loss of normal lumbar lordosis. Palpation along the spinous processes often elicits localized tenderness at the level of fracture, and in some cases, paraspinal muscle spasm may be appreciated. Pain may be exacerbated by axial loading, such as when the patient attempts to sit, stand, or change position. Range of motion is frequently limited due to pain, and patients may adopt a guarded posture to minimize discomfort. In the context of high-energy trauma, log-roll techniques should be employed to avoid exacerbating spinal injury during examination and transfer [11][12].

In low-energy or atraumatic presentations, the history focuses on identifying risk factors for fragility fractures and secondary causes of osteoporosis. Clinicians should inquire about advanced age, low body mass index, previous low-impact fractures, and long-term glucocorticoid therapy, all of which significantly increase fracture risk. Lifestyle factors such as current smoking and excessive alcohol intake, as well as dietary calcium and vitamin D insufficiency, are equally important contributors. Detailed medical history should explore conditions associated with secondary osteoporosis, including rheumatoid arthritis, chronic immobility, endocrine disorders such as hypogonadism and thyroid disease, inflammatory bowel disease with malabsorption, diabetes mellitus, chronic obstructive pulmonary disease, and a history of solid organ transplantation or prolonged immunosuppressive therapy. Medication review is essential, with attention to drugs that impair bone density or increase fall risk, such as sedatives, anticonvulsants, and certain psychotropic agents. Acute onset back pain is the hallmark symptom of symptomatic VCFs. Patients often describe a sudden, sharp pain localized to the thoracic or lumbar region, frequently triggered by an innocuous activity such as bending, lifting, or even coughing. The pain may radiate anteriorly around the chest or abdomen but typically follows a band-like distribution corresponding to the affected vertebral level. Despite this, a substantial proportion of VCFs are clinically silent and discovered incidentally on imaging performed for other reasons. When symptomatic fractures remain unrecognized or undertreated, they can lead to progressive kyphotic deformity, chronic axial pain, reduced mobility, impaired pulmonary function, and substantial limitations in activities of daily living. Over time, these factors contribute to deconditioning, increased fall risk, loss of independence, and diminished quality of life [11][12].

Red Flag Clinical Symptoms

In addition to mechanical and osteoporotic causes, clinicians must maintain a high index of suspicion for neoplastic etiologies, particularly in patients with low-energy fractures or atypical clinical features. Hematological malignancies such as multiple myeloma, as well as metastatic disease from primary cancers of the breast, lung, prostate, kidney, or thyroid, may infiltrate the vertebral bodies and predispose them to pathological compression fractures. Careful history taking should therefore include targeted questions about systemic “red flag” symptoms. Nocturnal pain that disrupts sleep, pain that is progressive and unrelenting or not clearly related to activity, and pain occurring at multiple skeletal sites are all concerning malignancy. Additional red flags include unexplained weight loss, anorexia, fatigue, night sweats, or a known history of primary cancer. Red flag neurological symptoms also warrant urgent attention. New-onset weakness, gait disturbance, saddle anesthesia, urinary retention, fecal incontinence, or severe radicular pain may indicate spinal cord compression or cauda equina syndrome, requiring immediate imaging and neurosurgical or orthopedic consultation. In all cases, meticulous documentation of the history, physical examination findings, and any red flag features is essential. These records establish a baseline for subsequent comparison, guide the choice of diagnostic imaging and laboratory investigations, inform therapeutic decision-making, and support coordinated long-term management among multidisciplinary teams [11][12].

Evaluation

The evaluation of vertebral compression fractures (VCFs) requires a systematic, multimodal approach that integrates clinical assessment with radiographic and, when appropriate, laboratory investigations. In patients with suspected VCFs, initial imaging typically begins with anteroposterior and lateral plain radiographs of the relevant spinal segments. In the acute trauma setting, these radiographs are first obtained with the patient in a supine position while maintaining full spinal precautions, in accordance with standard trauma protocols, to avoid exacerbating any unstable injuries.[13] Supine films allow rapid screening for gross vertebral height loss, malalignment, or obvious fracture lines, while preserving spinal immobilization

until stability is confirmed. However, supine radiographs can underestimate the true extent of vertebral body collapse because the absence of axial loading tends to mask dynamic deformity. For this reason, once the patient is hemodynamically stable and spinal precautions have been appropriately managed, upright or standing radiographs should be obtained to more accurately characterize vertebral height loss, kyphotic angulation, and overall fracture morphology under physiological load-bearing conditions.[13][14][15] These upright images are particularly valuable in osteoporotic populations, where subtle height loss or progressive deformity may only become evident with gravity-dependent loading. Beyond plain radiography, a careful clinical evaluation is essential. A detailed history should document the mechanism of injury, onset and character of pain, presence of neurological symptoms, prior fractures or known osteoporosis, and any history suggestive of malignancy or infection. The physical examination focuses on localized spinal tenderness, deformity, and neurological status, including motor, sensory, and reflex evaluation in all extremities, as well as bowel and bladder function. This clinical context guides the choice and urgency of advanced imaging modalities and helps differentiate between benign osteoporotic fractures, high-energy traumatic injuries, and fractures secondary to neoplastic disease.[7][13]

Imaging Studies

Computed tomography (CT) plays a central role in the acute evaluation of VCFs, especially in high-energy trauma. CT provides high-resolution, multiplanar reconstructions that allow precise delineation of fracture morphology, including involvement of the posterior vertebral wall, canal compromise, and subtle comminution that may be missed on plain radiographs.[13][14] CT is therefore indispensable for operative planning, assessment of spinal stability, and detection of associated injuries such as rib, pelvic, or posterior element fractures. In polytrauma settings, CT of the entire spine is often performed as part of whole-body trauma protocols, facilitating early and comprehensive detection of clinically significant injuries.[15] Magnetic resonance imaging (MRI) further enhances diagnostic accuracy by offering superior soft tissue contrast and the ability to assess neural structures, intervertebral discs, ligaments, and bone marrow. MRI is particularly valuable when neurological deficits are present, when ligamentous injuries are suspected, or when the distinction between acute and chronic fractures is clinically important.[16] Acute fractures typically demonstrate bone marrow edema, characterized by increased signal intensity on short tau inversion recovery (STIR) or fat-suppressed T2-weighted sequences and decreased signal on T1-weighted images. In contrast, chronic or healed fractures lack this edema pattern, appearing more uniformly hypointense on STIR and T2-weighted sequences. MRI therefore helps determine whether a vertebral deformity is responsible for new-onset pain or represents an old, asymptomatic lesion. MRI is also crucial for differentiating benign osteoporotic fractures from malignant pathological fractures. Features such as abnormal marrow signal extending into the pedicles or posterior elements, convex posterior vertebral border, and soft tissue mass-like extension into the epidural space or paravertebral region strongly suggest a malignant process.[16] Conversely, benign osteoporotic fractures often show band-like horizontal hypointense lines near the endplates on T1- and T2-weighted imaging, corresponding to fracture clefts, as well as the so-called “fluid sign,” a focal fluid collection within the vertebral body cleft, both of which are considered more typical of benignity.[16] These imaging distinctions have important implications for subsequent workup, including biopsy, systemic staging, and oncologic referral when malignancy is suspected.[7][16]

Vertebral Compression Fracture Classification Systems

To standardize communication, guide treatment decisions, and predict outcomes, several classification systems have been developed for VCFs. Their use depends on the underlying mechanism of injury—high-energy trauma, low-energy osteoporotic mechanisms, or neoplastic involvement—and they assist clinicians in assessing the severity and stability of fractures.

High energy trauma

In the context of high-energy trauma, the AO Spine Thoracolumbar Spine Injury Classification System is widely adopted. This system categorizes injuries primarily by morphology and integrity of the posterior tension band.[8] Type A injuries are defined as compression injuries involving failure of the anterior column under compression forces without disruption of the posterior tension band. Within this group, type A0 represents minor or insignificant fractures such as isolated spinous or transverse process injuries, typically without structural compromise of the vertebral body.[8] Type A1 injuries correspond to wedge compression fractures that affect a single endplate and spare the posterior vertebral wall, generally considered stable. Type A2 fractures are characterized as split or “pincer” fractures involving both endplates but still without posterior wall involvement, often reflecting a more complex compressive mechanism. Type A3 and A4 fractures extend the concept of compression injuries to include partial failure of the posterior vertebral wall. In type A3 injuries, one endplate and the posterior vertebral wall are involved while the posterior tension band remains intact, indicating increased risk of canal compromise but preservation of overall ligamentous stability.[8] Type A4 injuries involve both endplates and the posterior wall while still maintaining an intact posterior tension band,

representing more severe structural compromise and greater potential for progressive deformity under load. In contrast, type B injuries describe disruption of the posterior or anterior tension band due to flexion-distraction or extension forces, conferring significant mechanical instability and higher risk of progressive deformity. Type C injuries are characterized by translational or rotational displacement with complete disruption of all spinal elements, reflecting profound instability and typically mandating surgical stabilization.[8] Among these, only the type A category is strictly considered within the spectrum of compression injuries, while types B and C denote more complex instability patterns beyond pure compression [8].

Low-energy trauma and osteoporotic vertebral compression fractures

For low-energy trauma and osteoporotic VCFs, the AO Spine DGOU Osteoporotic Fracture (OF) classification offers a tailored framework that incorporates both fracture morphology and the degree of posterior wall involvement.[9] OF 1 fractures are defined by the absence of overt vertebral deformation on radiographs or CT, with vertebral edema apparent only on MRI-STIR sequences, indicating a purely edematous lesion or microfracture. OF 2 fractures display deformation of a single endplate with no or only minor involvement of the posterior wall, typically less than one-fifth of the vertebral body depth, and are usually considered relatively stable.[9] OF 3 fractures involve a single endplate with distinct posterior wall compromise exceeding one-fifth of the vertebral body depth, signifying a higher likelihood of instability and progressive kyphotic deformity. OF 4 fractures reflect substantial structural failure involving both endplates and the posterior wall, often manifesting as vertebral body collapse, pincer-type configurations, or marked loss of vertebral height; these injuries carry a considerable risk of progression and frequently require more aggressive management.[9] OF 5 fractures represent the most severe category, characterized by distraction, rotation, or complex instability affecting both anterior and posterior columns, and are typically associated with gross mechanical instability. The OF score has been developed as a validated decision-making tool that complements the OF morphological classification by integrating clinical parameters relevant to treatment choice.[10] The score assigns points for fracture morphology by multiplying the OF grade (from 1 to 5) by two, yielding between 2 and 10 points. Additional points are added or subtracted based on osteoporosis severity, deformity progression, pain intensity, neurological status, mobility, and overall health. A T-score less than −3 adds one point for severe osteoporosis. Evidence of radiographic deformity progression contributes one point, whereas stable morphology over time subtracts one point. Pain severity is incorporated using the visual analogue scale (VAS), adding one point when VAS is 4 or higher and subtracting one point when pain is less than 4.[10] The presence of fracture-related neurological deficit adds two points, reflecting its critical impact on treatment urgency. Patient mobility and general health status also influence the score: the ability to mobilize without assistance subtracts one point, while severe comorbidities—such as an American Society of Anesthesiologists (ASA) score greater than 3, body mass index below 20 kg/m², nursing dependency, or anticoagulation—each contribute negative points up to a maximum deduction of two.[10] Cumulative scores of 5 or less generally favor conservative management, a score of 6 is considered equivocal or "indifferent," and scores greater than 6 support consideration of surgical or interventional procedures.[10] This structured approach helps individualize treatment, balancing fracture morphology with patient-specific factors.

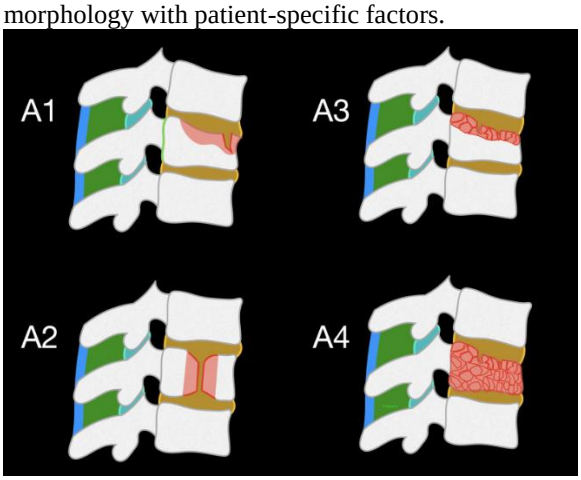

Fig. 2: AO Spine Thoracolumbar Spine Injury Classification.

Fractures secondary to neoplastic conditions

When VCFs arise from neoplastic processes, such as multiple myeloma or metastatic disease, additional classification and scoring systems are utilized to capture both fracture severity and spinal stability. The Genant semi-quantitative grading system is frequently employed to describe vertebral deformity on imaging.[7] In this system, grade 1 denotes mild height loss of approximately 20% to 25%, grade 2 corresponds to moderate height loss between 25% and 40%, and grade 3 reflects severe height loss exceeding 40%. These grades can be applied across multiple vertebral levels, providing an overview of fracture burden in systemic conditions like osteoporosis and myeloma. The Spinal Instability Neoplastic Score (SINS) offers a complementary, clinically oriented framework for assessing instability and the need for surgical consultation in neoplastic spinal disease.[17][18] SINS evaluates six parameters: spinal location (e.g., junctional versus mobile segments), presence and character of pain (particularly mechanical pain that worsens with movement and improves with recumbency), lesion

type (lytic, blastic, or mixed), spinal alignment, degree of vertebral body collapse, and involvement of posterolateral elements, such as pedicles or facets. Each parameter is assigned to a numerical value, and the sum yields a total score. Scores from 0 to 6 indicate a stable lesion that typically can be managed conservatively. Scores between 7 and 12 suggest potential or impending instability and warrant prompt surgical consultation. Scores of 13 to 18 signify overt instability, often necessitating surgical stabilization or other invasive interventions.[17][18] In addition to imaging-based evaluations, laboratory assessment in suspected myeloma-related fractures should include β2-microglobulin, monoclonal protein quantification, serum and urine light chains, and creatinine levels, as these biomarkers correlate with disease burden, renal involvement, and fracture severity.[7] Altogether, a meticulous evaluation of VCFs integrates targeted clinical assessment, appropriate radiographic and advanced imaging, and, when indicated, laboratory evaluation and specialized classification systems. This comprehensive approach enables accurate diagnosis, distinction between benign and pathological fractures, assessment of mechanical stability, and informed selection of conservative, interventional, or surgical treatment pathways tailored to the individual patient.

Treatment / Management

The management of vertebral compression fractures (VCFs) is multifaceted and must be individualized, integrating both patient-specific and fracture-specific factors. Key considerations include the mechanism of injury, fracture morphology, neurological status, bone quality, overall health, and patient preferences. Broadly, treatment strategies fall into conservative measures, surgical or interventional procedures, and structured interprofessional care for complex conditions such as osteoporosis and multiple myeloma. The overarching goals are to relieve pain, restore and maintain mobility, prevent or limit progressive deformity, protect neurological function, and optimize long-term quality of life. To support standardized and evidence-informed decision-making, several classification and scoring systems have been developed, including the Thoracolumbar Injury Classification and Severity (TLICS) scale and the AO Spine Thoracolumbar Injury Classification and Severity Score, as well as the Osteoporotic Fracture (OF) classification and OF score for osteoporotic fractures.[19][20][10] The TLICS system, introduced in 2005, was designed to provide more uniform management recommendations for thoracolumbar injuries, particularly in trauma patients.[19] It incorporates three critical parameters: the integrity of the posterior ligamentous complex (PLC), the morphology of the injury, and the patient's neurological status. Each of these components is assigned a numerical value, and the sum yields a total score ranging from 1 to 10. Scores less than 4 generally favor nonoperative management, whereas scores greater than 4 support surgical intervention; a score of 4 falls into an intermediate zone in which either conservative or surgical treatment may be appropriate based on clinical judgment and patient-specific factors.[19][20] The strength of TLICS lies in its focus on neurological compromise (which may necessitate decompression), fracture morphology (which reflects biomechanical stability), and PLC integrity (which is closely associated with the risk of delayed instability). However, the system is sometimes criticized as being overly broad, and as a result, many clinicians increasingly rely on the more detailed AO Spine Thoracolumbar Injury Classification system to refine clinical decision-making, especially in complex or borderline cases.[20]

Conservative Management

Conservative management remains the cornerstone of treatment for many stable VCFs, especially those without significant neurological deficit or major mechanical instability. Core components of nonoperative care include adequate analgesia, early and appropriately supervised mobilization, structured physical therapy, and spinal bracing when indicated. Analgesic regimens typically involve a stepwise approach, starting with acetaminophen and nonsteroidal anti-inflammatory drugs, and escalating to short-term use of opioids in more severe cases. Adjunctive agents such as calcitonin or neuropathic pain medications may be used in selected patients. The aim is to control pain sufficiently to allow early mobilization, which is essential to reduce the risks associated with prolonged bed rest, such as deconditioning, venous thromboembolism, and pressure injuries. Spinal orthoses are frequently employed as part of conservative therapy. Thoracolumbosacral orthoses (TLSOs) are generally used for fractures involving the thoracic spine and upper lumbar segments, whereas lumbosacral orthoses are more appropriate for injuries in the lower lumbar region. Bracing is usually prescribed for a period of 4 to 12 weeks, with the exact duration tailored to the patient's symptoms, radiographic progression, and overall clinical course.[24] The primary goals of bracing are to limit painful spinal motion, support the injured segment, and facilitate earlier ambulation. However, brace use must be balanced against potential downsides, including discomfort, skin breakdown, reduced pulmonary function, and further loss of muscle strength, particularly in older or frail patients. For this reason, patient education, careful fitting, and close follow-up are crucial. Radiographic and clinical criteria guide the discontinuation of bracing. Radiographic healing can be demonstrated by the absence of progressive vertebral collapse or segmental kyphosis on standing radiographs, evidence of bony consolidation on CT, or resolution of bone marrow edema and fracture-related

hyperintense signals on STIR sequences in MRI. Clinically, substantial pain reduction and improved function are essential indicators that bracing may be safely tapered.[13][14][15] Despite its effectiveness in controlling acute pain in stable fractures, conservative management does not reverse established kyphotic deformity and may be associated with persistent pain or pseudoarthrosis in some patients, particularly in severe osteoporotic fractures.[24]

Surgical Management

Surgical or interventional management is considered when fractures are unstable, associated with significant deformity or neurological compromise, or refractory to adequate conservative therapy. Key determinants of surgical necessity include the degree of vertebral body collapse, involvement of the posterior wall, progression of kyphosis, canal compromise with neurological symptoms, and failure to achieve pain relief or mobilization with nonoperative care. Modern fracture classification systems, including AO Spine and OF classifications, provide structured frameworks for these decisions.[8][10] Among minimally invasive options, cement augmentation techniques—vertebroplasty and balloon kyphoplasty—have become widely used for both osteoporotic and certain pathological VCFs. Vertebroplasty involves percutaneous injection of polymethylmethacrylate (PMMA) cement into the fractured vertebral body, primarily to stabilize microfractures and reduce pain rather than restore height. Balloon kyphoplasty, by contrast, uses an inflatable balloon tamp to create a cavity and partially restore vertebral height before cement injection, which improves segmental alignment and reduces kyphosis. Kyphoplasty is associated with a lower risk of cement leakage compared with vertebroplasty because of the controlled, low-pressure injection and the formation of a confined cement cavity.[21] Recent studies suggest that kyphoplasty can provide more rapid and substantial improvements in pain, function, and mobility compared with conservative therapy alone, particularly in patients with acute, painful osteoporotic fractures.[21] In selected patients with unstable compression fractures—such as those with marked posterior wall involvement, progressive deformity, or significant canal compromise—instrumentation with internal fixation, with or without fusion, may be required. This can include short-segment or long-segment posterior pedicle screw fixation, often combined with vertebral cement augmentation in osteoporotic bone to enhance screw purchase. Fusion may be added when long-term stability is desired or when extensive ligamentous disruption is present. The choice between minimally invasive versus open techniques depends on patient factors, fracture complexity, and surgeon expertise. The goals of surgical management are to restore spinal alignment, decompress neural elements when required, stabilize the injured segment, and facilitate early mobilization.

High Energy Trauma Management

In high-energy trauma, management decisions are heavily informed by the AO Spine Thoracolumbar Injury Classification and the associated Thoracolumbar Injury Severity Score, which provide a structured, quantitative approach to evaluating fracture severity and guiding treatment.[22][23] This system assigns points based on three major domains: fracture morphology, neurological status, and clinical modifiers. Fracture morphology is stratified into three main types with increasing severity: type A (compression), type B (tension band disruption), and type C (translation or dislocation). Within type A injuries, A0 lesions, such as minor spinous or transverse process fractures, score zero points; A1 wedge compression fractures involving a single endplate score one point; A2 split or pincer fractures involving both endplates score two points; A3 fractures that involve a single endplate and the posterior wall score three points; and A4 fractures with both endplates and posterior wall involvement score five points, reflecting higher instability risk.[22] Type B injuries, which indicate disruption of the posterior or anterior tension band without significant translational displacement, are more destabilizing and therefore carry higher scores. B1 injuries, such as bony chance fractures, are assigned five points, while B2 and B3 lesions—representing more extensive or complex tension band failures—receive six and seven points, respectively. Type C injuries, characterized by translation or dislocation with complete disruption of all structural elements, are considered the most severe, scoring eight points and almost invariably necessitating surgical stabilization.[22]

Neurological status contributes additional points. A neurologically intact patient (N0) receives zero points. Transient neurological deficits that have resolved by the time of evaluation (N1) score one point, whereas persistent radiculopathy (N2) earns two points. More severe deficits, such as incomplete spinal cord injury or cauda equina syndrome (N3), and complete spinal cord injury (N4), are each assigned four points, reflecting their critical importance. When the neurological exam cannot be reliably assessed—such as in sedated or intubated patients—an Nx designation is applied, scoring three points.[22] Clinical modifiers further refine the assessment. An indeterminate status of the posterior ligamentous complex (PLC) is designated as M1 and adds one point, capturing uncertainty related to potential instability. Other comorbid conditions, including osteoporosis and ankylosing spondylitis, are grouped under M2. Although these conditions substantially influence clinical management, they do not change the numeric score in the AO system;

instead, they are recorded as important contextual factors.[22] The cumulative AO Spine Thoracolumbar Injury Severity Score then guides treatment. Total scores of three or less typically indicate that conservative management is appropriate, focusing on analgesia, bracing, and rehabilitation. Scores of four or five fall into a gray zone, in which either conservative or surgical treatment may be reasonable, depending on patient comorbidities, preferences, and surgeon judgment. Scores of six or more usually support surgical intervention owing to significant instability or neurological compromise.[23] Nonetheless, management must remain individualized: highly fragile or medically complex patients with high scores may still be treated nonoperatively, whereas select patients with relatively low scores may undergo surgery to achieve faster mobilization or address specific concerns.

Low Energy Trauma (Osteoporotic Fractures)

Low-energy osteoporotic vertebral compression fractures (OVCFs) are managed primarily using the AO Spine DGOU Osteoporotic Fracture (OF) classification and the corresponding OF score, which together offer a comprehensive framework that incorporates fracture morphology, bone quality, clinical symptoms, and patient status.[10] Most osteoporotic fractures initially receive conservative therapy, particularly those classified as stable and associated with manageable pain. Standard nonoperative care includes multimodal analgesia, early but protected mobilization, physical therapy focused on core strengthening and balance, and external bracing with TLSO or lumbosacral orthoses depending on fracture level.[24] Conservative treatment typically extends over 4 to 12 weeks, with serial radiographs or advanced imaging used to monitor for progressive deformity or collapse. The OF score translates the OF morphological grade and clinical parameters into a numeric guide for management. Morphology contributes the largest component, with grades OF 1 through OF 5 multiplied by two, contributing between 2 and 10 points.[10] Additional clinical factors are layered onto this base: severe osteoporosis, defined by a T-score below −3, adds one point; documented deformity progression on follow-up imaging adds another point, whereas absence of progression subtracts one point. Symptomatically, a visual analogue scale (VAS) pain score of 4 or higher adds one point, while milder pain subtracts one point. The presence of fracture-associated neurological deficits contributes two points, underscoring their importance. Functional status also modifies the score: the ability to ambulate without assistance and good overall health (e.g., lower ASA classification, BMI ≥20 kg/m², independence from nursing care, and absence of high-risk anticoagulation) each reduce the score, up to a maximum of two points.[10] OF scores of 0 to 5 generally support conservative therapy, a score of 6 is considered equivocal and requires individualized decisions, and scores above 6 favor surgical or interventional treatment. Adherence to these score-based recommendations has been associated with better short-term outcomes and lower complication rates, whereas deviations correlate with suboptimal recovery.[10]

When surgery is indicated, specific strategies are tailored to OF fracture types. For OF 1 fracture, which show edema without overt deformation, vertebral augmentation with kyphoplasty or vertebroplasty is often sufficient, especially when pain is severe and persistent. OF 2 fractures, with limited deformation and minimal posterior wall involvement, can be managed with vertebral augmentation alone or, in rare circumstances, short-segment posterior fixation if deformity progresses. OF 3 fractures—with clear posterior wall compromise—are often treated with kyphoplasty or vertebroplasty, sometimes supplemented by short-segment posterior fixation in younger or more active patients, while long-segment or combined anterior–posterior constructs are reserved for more complex configurations.[25] OF 4 fractures, which exhibit substantial vertebral collapse and posterior wall involvement, are typically managed with short-segment posterior instrumentation, frequently combined with vertebral cement augmentation to improve stability. In highly comorbid or low-demand elderly patients, stand-alone vertebral augmentation may be considered, especially when surgical risk is high or deformity progression appears limited, though clinicians must remain aware that up to 14% may experience neurological deterioration over follow-up.[25] Long-segment posterior fixation is sometimes indicated for mid-thoracic fractures with significant kyphotic angulation or multilevel involvement. OF 5 fractures are deemed highly unstable and generally require operative stabilization. Options include long-segment posterior instrumentation extending several levels above and below the fracture or short-segment constructs reinforced with cement-augmented pedicle screws. Although these procedures carry higher complication rates, they consistently achieve superior pain relief, functional recovery, and quality of life compared with conservative management in appropriately selected patients.[26][27]

Preoperative Risk Assessment and Optimization

Preoperative risk stratification and optimization are particularly important in geriatric patients with osteoporotic VCFs, who often present with multiple comorbidities. Nonmodifiable risk factors—such as advanced age (especially older than 90 years), frailty, low BMI, male sex, and chronic neurodegenerative diseases like Parkinson disease—are associated with increased perioperative complications and higher mortality.[28] Modifiable factors, including hypoalbuminemia, chronic kidney disease, uncontrolled hypertension, cardiac

arrhythmias, and chronic pulmonary disease, should be carefully addressed before surgery. An interdisciplinary team involving internists, anesthesiologists, nutritionists, and physiotherapists can optimize medical management, nutritional support, and respiratory function, thereby reducing perioperative risks and improving postoperative outcomes.[28] This structured approach is crucial in determining whether patients are suitable candidates for more extensive surgical procedures or should instead receive less invasive or conservative therapies.

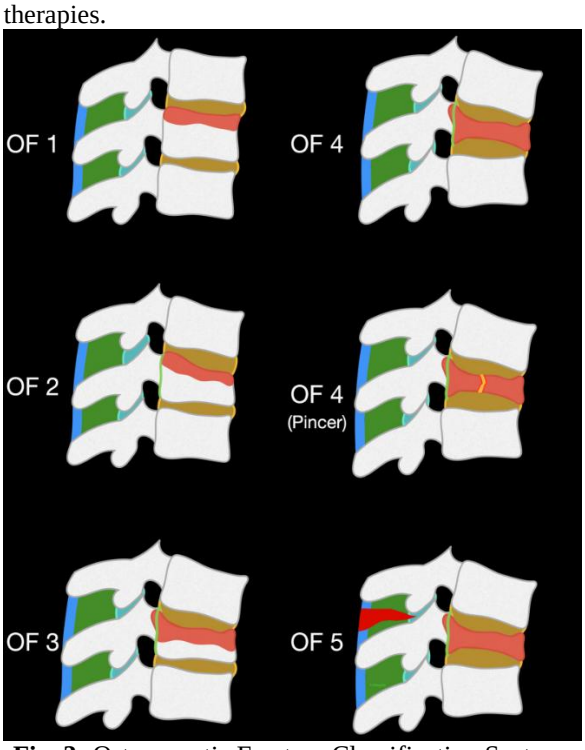

Fig. 3: Osteoporotic Fracture Classification System.

Multiple Myeloma Fractures

VCFs associated with multiple myeloma (MM) and other neoplastic conditions require a distinctly interprofessional and oncologically informed management strategy. Pathological fractures in MM occur in the context of widespread osteolytic disease, immunosuppression, and systemic therapy, which collectively alter healing potential and increase complication risks.[7][18] Initial treatment emphasizes pain control, early mobilization, and systemic bone support with bisphosphonates or other antiresorptive agents, alongside standard myeloma therapies. External bracing with TLSO can provide symptomatic relief and mechanical support, especially in patients not immediately eligible for surgery. Vertebral augmentation, particularly balloon kyphoplasty, is considered the mainstay surgical intervention for MM-related VCFs. Balloon kyphoplasty offers several advantages over vertebroplasty in this population, including improved restoration of vertebral height, better correction of kyphotic deformity, and a reduced rate of cement leakage owing to the controlled cavity creation.[29] Importantly, kyphoplasty has been shown to be effective even when the posterior wall is compromised, allowing pain relief and partial realignment without excessive risk of cement extravasation. Evidence suggests that performing kyphoplasty relatively early, typically within 4 to 8 weeks of fracture onset, maximizes functional recovery and reduces the duration of severe pain.[29] In cases where fractures are accompanied by substantial mechanical instability, as quantified by a Spinal Instability Neoplastic Score (SINS) greater than 12, posterior instrumentation becomes necessary.[17][18] Pedicle screw–rod constructs, often supplemented with vertebral cement augmentation, can restore stability, reduce pain, and improve or preserve neurological function. Because myeloma significantly compromises bone quality, cement augmentation of pedicle screws is frequently employed to enhance fixation. Novel materials such as carbon fiber–reinforced pedicle screws may be chosen to reduce imaging artifacts, thereby facilitating more accurate postoperative MRI and radiation therapy planning.[18] Whenever feasible, minimally invasive or mini-open approaches are preferred in MM patients to minimize blood loss, infection risk, and recovery time, although more extensive open decompression may still be required when there is marked epidural tumor burden causing neural compression.

Anterior column reconstruction is reserved for selected cases of severe anterior vertebral body destruction or pronounced segmental instability that cannot be adequately managed with posterior-only constructs. These procedures may involve corpectomy with placement of an expandable cage and anterior plating, often combined with posterior instrumentation to achieve circumferential stability. Given their higher morbidity, such operations are typically limited to patients with reasonable functional status and sufficient life expectancy to benefit from extensive reconstruction.[18] Postoperatively, early resumption or initiation of systemic myeloma therapy is essential to control disease progression. Radiotherapy plays an important complementary role, providing local tumor control and pain relief in symptomatic vertebral lesions or residual disease after surgery.[18] Concomitant use of bisphosphonates, along with calcium and vitamin D supplementation, is recommended to improve bone mineral density, reduce skeletal-related events, and support the structural benefits achieved by surgical intervention. Through this integrated, multidisciplinary approach—combining targeted surgical stabilization, vertebral augmentation, systemic anti-myeloma therapy, and bone-directed treatments—patients with MM-related VCFs can achieve meaningful reductions in pain, improved mobility, and enhanced health-related quality of life.[18][29]

Differential Diagnosis

The differential diagnosis of back pain in a patient with a suspected vertebral compression fracture (VCF) is broad and requires a systematic and holistic approach. Before any imaging is obtained, clinicians should carefully consider nonspinal etiologies, as back pain may be referred from thoracic, abdominal, retroperitoneal, or pelvic structures. Musculoskeletal causes, such as paraspinal muscle strain, ligamentous sprain, degenerative spondylosis, or facet joint arthropathy, are frequent and often coexist with osteoporotic changes, potentially obscuring the clinical picture. Pulmonary processes, including pneumonia, pulmonary embolism, or pleuritis, may manifest as thoracic back pain, particularly when the lower lobes or pleura are involved. Similarly, abdominal and retroperitoneal pathologies—such as pancreatitis, peptic ulcer disease, cholecystitis, abdominal aortic aneurysm, renal colic, or pyelonephritis—can present with midline or flank pain that mimics vertebral pathology. In the lower thoracic and upper lumbar regions, careful evaluation for cardiovascular or vascular causes, particularly aortic dissection or aneurysms, is essential given their life-threatening nature .[18][29] Once imaging reveals a vertebral body fracture, the diagnostic focus shifts to characterizing the fracture pattern and distinguishing simple compression fractures from more unstable or complex injuries. Close inspection of the posterior vertebral cortex, posterior wall, pedicles, and posterior elements is mandatory to identify features of burst fractures, flexion-distraction injuries, or translational injuries that carry a higher risk of neurological compromise and may necessitate surgical stabilization. Cross-sectional imaging with CT refines this assessment by delineating retropulsed fragments, canal compromise, and involvement of the posterior elements, while MRI allows evaluation of the posterior ligamentous complex, spinal cord, and nerve roots .[18][29]

At the same time, pathological fractures must be differentiated from benign osteoporotic fractures. Red flags such as disproportionate pain, involvement of noncontiguous levels, lytic or blastic changes on imaging, or a known history of malignancy raise suspicion for metastatic disease or hematologic malignancies such as multiple myeloma. Infectious etiologies, including vertebral osteomyelitis and discitis, should also be considered, particularly in patients with fever, elevated inflammatory markers, recent bacteremia, intravenous drug use, or immunosuppression. These conditions may mimic VCFs radiographically in early stages but evolve to show endplate destruction, paravertebral soft tissue masses, or epidural abscesses on MRI. In younger patients with trauma, high-energy mechanisms require consideration of concomitant spinal cord injury, ligamentous disruption, or associated fractures of the pelvis and ribs, necessitating a full trauma workup. Ultimately, accurate differentiation among these entities relies on integrating history, physical examination, laboratory tests, and advanced imaging to ensure that vertebral fractures are correctly classified and that dangerous mimics or coexisting conditions are not overlooked .[18][29]

Prognosis

The prognosis of vertebral compression fractures (VCFs) is heterogeneous and influenced by the underlying etiology, patient age, baseline functional status, comorbidities, and fracture morphology. In older adults with osteoporotic VCFs, numerous studies demonstrate an increase in mortality compared with age-matched controls, reflecting not only the fracture itself but also the systemic vulnerability associated with frailty and chronic disease. Survival rates have been reported as approximately 53.9% at three years, 30.9% at five years, and 10.5% at seven years following an osteoporotic VCF, underscoring the long-term prognostic implications of these injuries.[30]

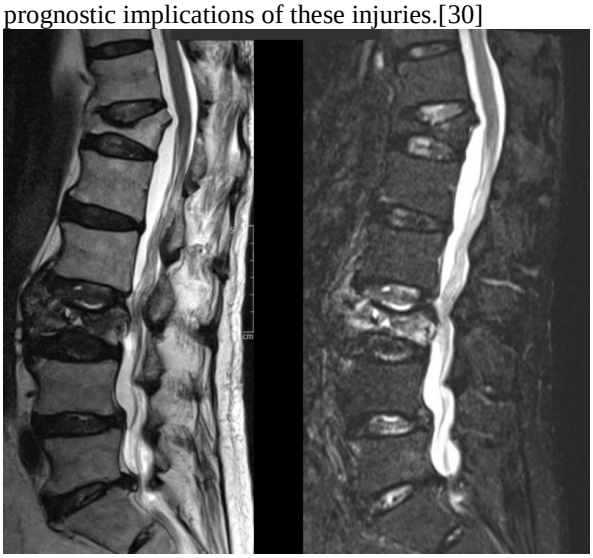

Fig. 4: STIR Imaging. Sagittal T2-weighted (left) and STIR (right) MRI sequences demonstrate two vertebral compression fractures.

These figures highlight that a VCF in an older adult is often a marker event, signaling advanced skeletal fragility, increased fall risk, and frequently the presence of multiple comorbidities rather than an isolated orthopedic problem. Within osteoporotic fractures, prognosis varies by OF classification. OF 4 fractures, which involve pronounced compromise of both vertebral endplates and the posterior wall, are associated with substantial structural instability and a higher risk of progressive deformity. If inadequately treated or managed solely with conservative measures in patients with significant symptoms or deformity progression, these fractures may lead to chronic pain, impaired mobility, and diminished health-related quality of life.

Neurological complications, including radiculopathy and, less commonly, spinal cord or cauda equina compromise, are more likely in this group when posterior wall involvement is significant. However, patients who undergo timely surgical management—most commonly short-segment hybrid stabilization with pedicle screw instrumentation combined with cement augmentation—typically experience favorable short-term functional outcomes, improved pain control, and earlier mobilization, even though the overall complication rate is higher due to age and comorbidities.[25]

OF 5 fractures, representing the most unstable osteoporotic patterns with failure of anterior or posterior tension bands, carry an even more guarded prognosis if left untreated. Surgical stabilization in these cases is generally recommended, as it improves segmental alignment, reduces pain, and restores sufficient stability to permit rehabilitation. Functional outcomes and quality of life usually improve significantly following surgery, with many patients regaining pre-injury levels of basic mobility or better.[26] Nonetheless, these benefits are offset by a high rate of general complications in this frail population, including urinary tract infections, pneumonia, delirium, and thromboembolic events. Consequently, meticulous perioperative care, early mobilization, and close medical management are crucial to translating structural improvements into durable clinical gains.[26] For patients with vertebral fractures secondary to multiple myeloma (MM), prognosis has markedly improved in recent decades due to advances in systemic therapy, including proteasome inhibitors, immunomodulatory agents, monoclonal antibodies, and autologous stem cell transplantation. Local management of VCFs in MM using vertebral augmentation, particularly balloon kyphoplasty, provides rapid and substantial pain relief, improved vertebral height restoration, and reduction of segmental kyphosis, which together enhance functional status and allow earlier initiation or continuation of systemic therapy.[29] Early intervention within four to eight weeks of fracture onset appears to correlate with superior outcomes, including improved ambulation and reduced opioid requirements.[29] Despite these benefits, MM patients remain susceptible to high rates of adjacent-level fractures due to ongoing skeletal involvement, as well as perioperative complications related to immunosuppression, anemia, renal dysfunction, and infection risk. Prognosis in this group is therefore determined by the interplay between effective oncologic control of the underlying malignancy and successful mechanical management of the spinal pathology. Overall, a proactive approach that addresses bone health, systemic disease, and fractured stability collectively offers the best opportunity to improve survival, preserve independence, and maintain quality of life in patients with VCFs across etiologies.[25][26][29][30]

Complications

Complications associated with vertebral compression fractures (VCFs) arise from both the natural history of the disease and the interventions used to treat it. Nonoperative management, although appealing for its lower immediate procedural risk, can lead to a cascade of adverse consequences, particularly when fractures are unstable or poorly controlled from a pain and biomechanical standpoint. Chronic back pain is one of the most common complications, often resulting from persistent micro-motion at the fracture site, progressive kyphotic deformity, and secondary degenerative changes in adjacent spinal segments. As kyphosis increases, global sagittal imbalance may develop, shifting the center of gravity anteriorly and imposing greater demands on paraspinal musculature. This can lead to fatigue, reduced walking tolerance, and a significant decline in activities of daily living. In the thoracic spine, severe kyphotic deformity may compromise pulmonary function by reducing chest wall compliance and vital capacity, predisposing patients to recurrent respiratory infections and decreased exercise tolerance. Over time, these biomechanical and physiologic alterations can contribute to deconditioning, increased fall risk, and further fracture events, particularly in frail or osteoporotic individuals [29][30]. Persistent vertebral instability in untreated or inadequately treated fractures may culminate in pseudoarthrosis, characterized by nonunion and ongoing pain. Such cases often become refractory to conservative measures and may eventually require delayed surgical intervention, which is typically more complex due to established deformities and soft tissue changes. Moreover, prolonged immobility in patients managed conservatively without adequate pain relief can lead to secondary complications such as pressure ulcers, deep vein thrombosis, urinary tract infections, and muscle wasting, especially among older adults and those with multiple comorbidities.

Surgical and interventional treatments introduce their own spectrum of complications. Cement augmentation techniques, including vertebroplasty and balloon kyphoplasty, are generally safe but carry the risk of cement leakage beyond the confines of the vertebral body. Most leakages are clinically silent; however, in rare instances, cement can extravasate into the spinal canal or neural foramina, compressing nerve roots or the spinal cord and resulting in new or worsened neurological deficits. Even more rarely, cement can migrate into venous channels, leading to pulmonary cement embolism or, in exceptional cases, cerebral embolic events, causing serious cardiopulmonary or neurologic compromise. Although these events are infrequent, their potential severity mandates

meticulous technique, careful patient selection, and real-time imaging guidance [29][30]. Augmentation of a fractured vertebra alters local biomechanics by increasing the stiffness of the treated segment relative to adjacent levels. This stiffness mismatch may redistribute mechanical loads to neighboring vertebrae, theoretically contributing to the risk of adjacent-level fractures. While it can be difficult to distinguish the relative contribution of altered biomechanics from the underlying osteoporotic process, clinicians should remain aware of this possibility and monitor patients closely after augmentation. Posterior stabilization procedures, particularly multilevel instrumentation and combined anterior–posterior approaches, carry additional risks, including surgical site infection, hardware failure, screw loosening, and iatrogenic neurologic injury during decompression. Despite the advent of minimally invasive techniques, which reduce soft tissue trauma, blood loss, and postoperative pain, these approaches may offer limited capacity for extensive deformity correction or complex decompression and still require careful execution. Cement augmentation of pedicle screws and involved vertebrae (hybrid stabilization) can mitigate implant-related complications in osteoporotic bone by improving fixation stability but also introduces the same cement-related risks as vertebral augmentation [29][30]. Finally, systemic complications are common in patients undergoing surgery for VCFs, particularly among older adults and those with severe comorbidities. Pneumonia, urinary tract infections, delirium, cardiac events, and thromboembolism are frequently encountered and may overshadow the orthopedic success of the procedure. Comprehensive preoperative optimization, evidence-based perioperative care, aggressive pulmonary hygiene, early mobilization, and vigilant postoperative monitoring are essential to minimize these risks. Careful risk–benefit analysis and shared decision-making with patients and families help ensure that the chosen treatment strategy aligns with the patient's health status, goals of care, and tolerance for potential complications [29][30].

Patient Education

Deterrence of vertebral compression fractures (VCFs) relies heavily on education and empowerment of patients, particularly those at elevated risk due to osteoporosis, prior fragility fractures, or chronic glucocorticoid use. A central element of preventive care is promoting bone health through lifestyle and pharmacologic strategies. Patients should receive clear, accessible information about the importance of adequate calcium and vitamin D intake, either through diet or supplementation, as well as the value of regular weight-bearing and resistance exercises in maintaining bone mineral density and improving balance and muscle strength. Such exercises not only enhance skeletal integrity but also reduce fall risk, a critical determinant of fracture occurrence in older adults. Clinicians must also emphasize modifiable risk factors, including smoking cessation and moderation of alcohol consumption, both of which adversely influence bone turnover and fracture risk [29][30]. Equally important is patient adherence to prescribed osteoporosis therapies, such as bisphosphonates, denosumab, selective estrogen receptor modulators, or parathyroid hormone analogues. Patients should understand that these medications are preventive and long-term in nature, with benefits accruing over months to years. Counseling should address common concerns about side effects, clarify realistic expectations, and underscore the consequences of untreated osteoporosis, including VCFs, hip fractures, and loss of independence. Educational materials, group classes, and reinforcement by nurses, pharmacists, and physical therapists can all improve medication adherence and lifestyle modification [29][30].

Fall prevention is another foundational component of patient education. Clinicians should encourage patients and caregivers to review home environments for hazards such as loose rugs, poor lighting, cluttered walkways, and unstable furniture. Installation of grab bars in bathrooms, use of non-slip mats, and ensuring sturdy handrails on stairs are simple interventions that can markedly reduce fall risk. Vision and hearing assessments, appropriate footwear, and management of orthostatic hypotension or medication-induced dizziness are also essential aspects of fall prevention. Physical therapy–guided balance and gait training can provide individualized strategies for safe mobility and confidence-building, particularly in patients with prior falls or fear of falling. In addition to preventive education, patients should be informed about the early warning signs and symptoms of VCFs, including acute onset of midline back pain, pain exacerbated by standing or walking and relieved by lying down, and new or worsening kyphotic posture. Prompt medical evaluation in the presence of these symptoms can facilitate early diagnosis, timely initiation of treatment, and prevention of further vertebral collapse or deformity progression. Discussions about therapeutic options—ranging from conservative management with analgesia and bracing to interventional procedures like kyphoplasty—should be transparent and tailored to the patient's health status, values, and goals. Clearly outlining the risks and benefits of each modality, expected recovery trajectories, and potential impact on quality of life enables patients to participate actively in shared decision-making. Ultimately, a coordinated, patient-centered educational strategy that involves physicians, nurses, pharmacists, and physical therapists fosters adherence to preventive measures, supports informed choices, and helps reduce the incidence and consequences of VCFs over the long term [29][30].

Other Issues

Several key clinical pearls and special considerations can substantially influence the evaluation and management of vertebral compression fractures (VCFs). Foremost among these is the central importance of a detailed neurological examination. Because compression of neural elements—whether from retropulsed bone fragments, epidural hematoma, or tumor—can rapidly alter prognosis and treatment priorities, careful assessment of motor strength, sensory function, reflexes, and sphincter control should be performed in every patient with a suspected spinal fracture. Any new or progressive neurological deficit shifts management toward urgent imaging, typically MRI, and early surgical consultation. Another crucial practical consideration concerns the selection of patients for kyphoplasty or vertebroplasty. Although kyphoplasty is widely used and effective for many osteoporotic compression fractures, it is not universally appropriate. Contraindications include the presence of significant neurological compromise due to canal compromise, burst fractures with marked posterior vertebral body wall disruption, active spinal infection, systemic sepsis, or uncorrected coagulopathy or bleeding diathesis. In these situations, cement injection may exacerbate neural compression, disseminate infection, or increase hemorrhagic risk, making open or instrumented surgical approaches or staged management more appropriate. Furthermore, when there is uncertainty about the benign or malignant nature of a lesion, biopsy or advanced imaging should be considered before cement augmentation to avoid masking a neoplastic process [29][30]

Special attention must also be given to patients with underlying spinal disorders such as diffuse idiopathic skeletal hyperostosis (DISH) and ankylosing spondylitis (AS). In these conditions, the spine behaves biomechanically like a long, rigid lever arm due to extensive ossification of ligaments and joints. As a result, even low-energy trauma can produce highly unstable fractures that behave more like long-bone fractures than typical segmental spinal injuries. These fractures often traverse all three columns and carry a high risk of delayed displacement and neurological deterioration. Consequently, any suspected fracture in a patient with DISH or AS should be considered unstable until proven otherwise, and evaluation must include CT and, often, MRI to fully characterize the extent of injury. Surgical stabilization—usually with long-segment fixation—is frequently required, and nonoperative management is rarely appropriate given the high risk of catastrophic deterioration. Lastly, clinicians should recognize that VCFs often signal broader systemic issues. A first fragility fracture, especially in older adults, should trigger a comprehensive osteoporosis and fall-risk evaluation rather than being treated as an isolated event. Coordination with primary care physicians, endocrinologists, rheumatologists, and geriatric specialists can ensure appropriate investigation and long-term management of bone health, thereby reducing the likelihood of future fractures. Awareness of these pearls—prioritizing neurologic evaluation, recognizing contraindications to augmentation, identifying special high-risk spinal conditions, and viewing fractures as systemic red flags—enhances clinical decision-making and helps prevent avoidable complications [29][30]

Enhancing Healthcare Team Outcomes

Optimal care for patients with vertebral compression fractures (VCFs) is inherently interprofessional, requiring seamless collaboration among physicians, advanced practice practitioners, nurses, pharmacists, physical and occupational therapists, and, when appropriate, social workers and case managers. Physicians and advanced practice providers, including orthopedic surgeons, neurosurgeons, trauma surgeons, geriatricians, and internists, are responsible for initial diagnosis, classification of the fracture using systems such as the AO Spine or OF classifications, and formulation of a tailored treatment strategy. Their role includes not only technical decision-making—such as choosing between conservative management, vertebral augmentation, or instrumented stabilization—but also careful consideration of comorbidities, functional goals, and patient preferences. Ethical obligations include transparent communication with patients and families regarding the potential benefits and risks of proposed interventions, likely prognosis, and alternatives, particularly in frail or cognitively impaired individuals [29][30]. Nurses and trauma specialists contribute vitally to day-to-day patient care, monitoring for early signs of neurological deterioration, hemodynamic instability, and complications such as infection, thromboembolism, or delirium. In intensive care or high-dependency settings, they ensure timely implementation of physician orders, pain management protocols, and mobilization plans. Nurses also educate patients and families regarding brace application, skin care under orthoses, safe transfer techniques, and recognition of red-flag symptoms such as new weakness or bladder dysfunction. Their ongoing contact with patients positions them uniquely to reinforce fall prevention strategies and adherence to therapy, thereby promoting safe transitions from hospital to home or rehabilitation facilities. The overall prognosis is influenced not only by the fracture type and neurological status but also by the presence of associated injuries, the need for mechanical ventilation, and the quality of supportive nursing care.

Pharmacists play an essential role in optimizing pharmacologic management, particularly

in older adults with polypharmacy. They assist in selecting appropriate analgesics, adjusting doses for renal or hepatic impairment, and minimizing the use of medications that increase fall risk, such as sedative-hypnotics and certain psychotropics. Pharmacists also reinforce adherence to osteoporosis medications and supplements, identify potential drug–drug interactions, and provide guidance on perioperative management of anticoagulants and antiplatelet agents. Through this work, they reduce adverse drug events, enhance pain control, and support earlier mobilization [29][30]. Physical and occupational therapists are central to rehabilitation and functional recovery. Early physical therapy focuses on safe mobilization, gait training, posture correction, and strengthening of core and paraspinal musculature. Therapists also provide individualized home exercise programs and teach strategies to protect the spine during activities of daily living, such as lifting, bending, or transitioning from sitting to standing. Occupational therapists assess the patient's ability to perform self-care tasks and recommend adaptive equipment or environmental modifications to reduce strain and prevent falls at home. Their work directly reduces deconditioning, shortens hospital stays, and supports durable improvements in independence and quality of life. Social workers and case managers facilitate coordination of care across settings by arranging rehabilitation placements, organizing home health services, and assisting families with logistical and financial challenges. They help ensure continuity of care, adherence to follow-up appointments, and access to community resources such as osteoporosis education programs or fall-prevention workshops. From an ethical and quality-improvement standpoint, regular interdisciplinary case conferences, morbidity and mortality reviews, and protocol-based audits help teams identify gaps, refine treatment algorithms, and align practice with current evidence. Such collaborative reflection fosters a culture of continuous learning and patient safety. By integrating clinical expertise, vigilant nursing care, pharmacologic optimization, focused rehabilitation, and social support, the interprofessional team can significantly improve outcomes for patients with VCFs, reduce complication rates, and enhance both short- and long-term quality of life [29][30].

Conclusion:

In conclusion, the effective management of Vertebral Compression Fractures (VCFs) demands a comprehensive and integrated strategy that extends beyond treating the isolated fracture. A thorough evaluation, utilizing advanced imaging and structured classification systems, is crucial for accurate diagnosis and for distinguishing between osteoporotic, traumatic, and pathological fractures, which guides subsequent treatment. While stable fractures often respond well to conservative measures—including multimodal analgesia, spinal bracing, and targeted physical therapy—unstable or persistently painful fractures frequently require interventional procedures like kyphoplasty or surgical stabilization. Critically, VCFs should be recognized as sentinel events, particularly in older adults, signaling underlying skeletal fragility. Therefore, long-term management must prioritize secondary prevention through patient education, fall prevention strategies, and pharmacological treatment of osteoporosis to mitigate the risk of future fractures. Ultimately, optimal patient outcomes are achieved through a coordinated, interprofessional team approach. This model integrates the expertise of physicians, nurses, pharmacists, and physical therapists to address the multifaceted aspects of care, from acute pain control and functional rehabilitation to systemic bone health, thereby improving mobility, reducing disability, and enhancing the overall quality of life for affected individuals.

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