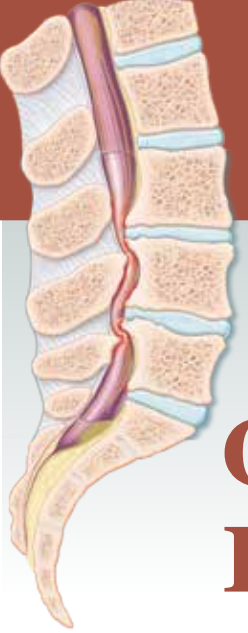

Osteoporotic Vertebral Compression Fractures: Diagnosis and Management

ABSTRACT

Osteoporotic vertebral compression fractures (VCFs) are the most common fragility fracture and have significant impact on numerous indices of health quality. High risk patients should be identified and appropriate preventative therapy initiated. The majority of VCFs can be managed in a non-operative fashion, with analgesia as required to support progressive mobilization. Patients who fail non-operative measures may be considered for percutaneous vertebral augmentation. However, the efficacy of these procedures in altering the natural history of recovery is controversial. Surgery has a limited role in the initial management of VCFs and is typically restricted to the rare circumstance of VCF associated with acute neurological dysfunction.

KEYWORDS: osteoporosis, vertebral compression fracture, vertebroplasty, kyphoplasty

CME Pre-test Quiz

Introduction

Osteoporosis is a disorder of low bone density and disrupted bone microarchitecture that results in an increased risk of fractures. Osteoporosis is defined as having a bone mineral density of 2.5 or more standard deviations below the peak bone mass for young adults (i.e. T-score <= -2.5). Osteoporotic, or fragility, fractures are responsible for excess mortality, morbidity, admission to health care institutions and significant economic burden at both an individual and population level.[25,26] Osteoporosis can affect any bone in the body, but most fragility fractures occur at the hip, spine, wrist or ribs.[8,25] Approximately 40% of Caucasian women and 13% of men 50 years and older will experience one clinically recognized fragility fracture in their lifetime.[18,23,25] The risk of developing a VCF is strongly associated with decreasing

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bone mineral density (BMD), with the risk increasing two fold with each standard deviation below the mean BMD, as determined by dual-energy x-ray absorptiometry (DEXA).[7,33] Osteoporosis continues to be undertreated in Canada, as current estimates suggest that fewer than 20% of females and 10% of males who have sustained a fragility fracture, receive therapies to prevent future fractures.[26]

Vertebral compression fractures (VCFs) are the most common osteoporotic fracture, accounting for approximately 700,000 of the total 1.5 million annual osteoporotic fractures in the USA.[33] VCFs typically lead to severe back pain, decreased mobility, sleep loss and, most significantly, are associated with a 15% increased risk of age-adjusted mortality.[30] According to the Canadian Multicenter Osteoporosis Study, an estimated 23.5% of Canadian women and 21.5% of men 50 years and older will develop a VCF in their lifetime10. The rate of vertebral fractures increases from an annual incidence of 0.9% and prevalence of 5-10% among middle-aged women in their 50-60s, to an incidence of 1.7% and prevalence of greater than 30% among those 80 years and older.[33] The economic burden of VCFs is substantial, and in the first year after a VCF, patients have been found to require primary care services at a rate 14 times greater than the general population.[7,33] The annual US medical burden for VCF management was estimated at $13.8 billion in 2001,[14,33] with this cost projected to increase by a further 50% in 2025.[14]

Risk Factors for Osteoporotic Fractures and Diagnostic Evaluation

Given the significant health and socioeconomic burden osteoporotic VCFs have on both patients and society in general, it is important to identify low-BMD patients early, refer them for BMD testing and offer appropriate prophylactic treatment, as indicated. Risk factors for low BMD include female sex, prior fragility fractures, parental hip fractures, menopause, previous glucocorticoid use (>=3 month use of equivalent steroid dose of prednisone 7.5mg daily), current smoker, high alcoholic intake (>=3 drinks per day), rheumatoid arthritis, insufficient dietary calcium, low vitamin D production/consumption, low body mass index and lack of weight bearing exercise.[26]

According to the 2010 Clinical Practice Guidelines for diagnosis and management of osteoporosis in Canada, all men and women above age 50 should be assessed for risk factors for osteoporosis and fragility fractures.[26] The assessment should include a combination of history and physical examination (Table 1).[26] BMD testing should be performed on all patients >= 65 years old, and all

Table 1: Recommendations for Clinical Assessment[26]

Assessment	Recommended Elements
History	Identify risk factors for low BMD, future fractures and falls • Prior fragility fractures • Parental hip fractures • Menopause • Glucocorticoid use • Current smoking • High alcohol intake (>= 3 units per day) • Rheumatoid arthritis • Inquire about falls in the previous 12 months • Inquire about gait and balance
Physical exam	Measure weight (weight loss of >10% since age 25 is significant) Measure height annually (prospective loss >2cm or historic loss of >6cm) Measure rib to pelvis distance (>= 2 fingers' breadth) Measure occiput-to-wall distance (for kyphosis) (>5cm) Assess fall risk by using Get-up-and-Go-Test

menopausal females or males >=50 who meet any clinical risk factors listed in Table 2.[26]

While BMD is strongly correlated with the risk of fragility fracture, it is not the only determinant of fracture risk. Multiple clinical factors (e.g. prior fracture, glucocorticoid use) contribute to fracture risk independent of BMD. As such, it is important to determine the 10-year fracture risk of the patient in order to guide medical management and not simply determine BMD. The two most commonly used tools in Canada for estimating this risk of a major osteoporotic fracture (hip, vertebra, forearm or proximal humerus) are the Canadian Association of Radiologist and Osteoporosis Canada (CAROC) and the Fracture Risk Assessment Tool (FRAX). Both of these tools use the DEXA determined T-score of the femoral neck in addition to numerous clinical risk factors to determine the overall fracture risk. Both tools have been vali-

Table 2: Indication for Bone Mineral Density Testing[26]

Older Adults (age>= 50 years)	Younger Adults (age < 50 years)
Age >= 65 years	Fragility fractures
Clinical Risk Factors for fractures (menopausal	Prolonged use of glucocorticoids
women, men age 50-64 years old)	Use of high risk medications[2]
Fragility fracture after age 40 years	Hypogonadism or premature menopause (age <45 years)
Prolonged glucocorticoid use[1]	Malabsorption syndrome
Use of other high-risk medications[2]	Primary hyperparathyroidism
Parental hip fracture	
Vertebral fracture ore osteopenia identified on radiography	Other disorders strongly associated with rapid bone loss and/or fracture
Current smoker	
High alcoholic intake	
Low body weight (<60 kg) or major weight loss	
(>10% of body weight at age 25 years)	
Rheumatoid arthritis	
Disorders strongly associated with osteoporosis	

[1]At least three months cumulative therapy in the previous year at a prednisone equivalent dose >=7.5mg daily
[2]For example, aromatase inhibitors or androgen deprivation therapy

dated for the Canadian population. The CAROC and FRAX tool can be found at http://www.osteoporosis.ca/multimedia/FractureRiskTool/index.html#/Home and https://www.shef.ac.uk/FRAX/tool.aspx?country=19, respectively. Both predictive tools have a high concordance of 90% in risk categorization and correlate well with observed fracture rates for women and men.[17] Either can be used for risk stratification.

Prevention and Treatment of Osteoporosis Fractures

Both non-pharmacological and pharmacological therapies are important in the prevention of osteoporotic fractures. It is important to encourage basic bone health for all individuals over the age of 50 years old. This includes regular weight bearing exercise, daily calcium intake of 1200mg, daily Vitamin D intake of 800-2000 IU and education around fall prevention

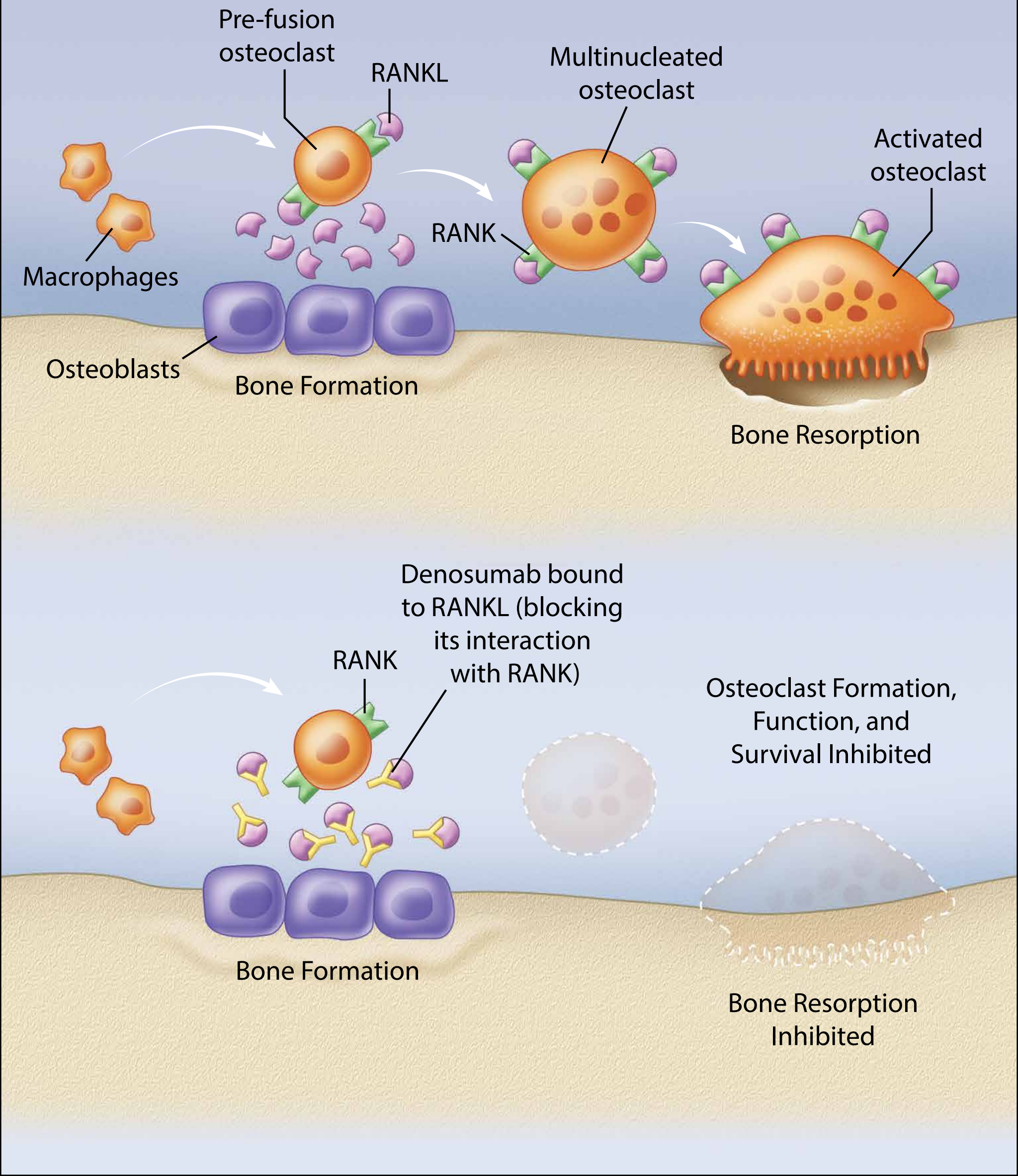
strategies. The decision to start a patient on pharmacological therapy should be predicated on an assessment of fracture risk by means of a validated fracture prediction tool (e.g. FRAX or CAROC).[26] Any patient in the high-risk category (> 20% 10 year probability of osteoporotic fracture), a history of more than one fragility fracture or a fragility fracture involving the hip or vertebra should be offered pharmacological therapy. Patients in the moderate risk group should also be considered for pharmacological therapy, using an individualized approach to treatment decisions that involves a careful clinical evaluation to identify additional risk factors (Table 3).[26] For patients in the low risk category, no therapy is required beyond education on lifestyle measures, including exercise, fall prevention, smoking cessation and optimization of calcium and vitamin D intake.[26] Given BMD decrease with advancing age, it is advisable to consider rescreening patients who are low risk on initial evaluation. However, there are no consensus guidelines regarding the most appropriate time interval for rescreening.[8]

There is a wide range of pharmacological agents available in Canada for the treatment of osteoporosis. The options include anti-resorptive agents such as bisphosphonates, the RANK ligand inhibitor denosumab, selective estrogen receptor modulators, hormone therapy, calcitonin and the bone-forming agent teriparatide. There is consistent and high level

Table 3: Factors that Warrant Consideration for Pharmacological Therapy in Moderate Risk Patients[26]

• Additional vertebral fracture(s) (>25% height loss with end-plate disruption) identified on vertebral fracture assessment or lateral spine x-ray
• Previous wrist fracture in individuals older than age 65 or those with T-score<=-2.5
• Lumbar spine T-score much lower than femoral neck T-score
• Rapid bone loss
• Men on androgen deprivation therapy for prostate cancer
• Women on aromatase inhibitor therapy for breast cancer
• Long-term or repeated glucocorticoid use (oral or parenteral) that does not meet conventional criteria for recent prolonged systemic glucocorticoid use
• Recurrent falls defined as falling 2 or more times in the past 12 months
• Other disorders strongly associated with osteoporosis, rapid bone loss or fractures

Denosumab and Its Role in the Inhibition of Osteoclast Formation, Function, and Survival



evidence from randomized clinical trials that all therapies currently available in Canada reduce the risk of VCFs for menopausal women with a high fracture risk.[2,6,20]

In contrast, while there is limited randomized data assessing the reduction of fracture rate as a primary outcome for men, systemic reviews and meta-analyses have found reduction in vertebral fractures with bisphosphonates.[21,31] The evidence of other agents in men is limited. In general, pharmacotherapy reduces the risk of fracture by 30-70% depending on the agent and the level of adherence. Table 4 outlines the mechanism of action, efficacy and side effects of various pharmacological options.[8] For patients with significant back pain secondary to VCFs, both calcitonin and teriparatide has been shown to significant reduce fracture-associated pain.[13,15] Table 5 outlines the recommended first-line agents for fracture prevention in osteoporosis.

VCFs: Clinical Presentation

VCFs commonly occur in the mid-thoracic or thoracolumbar transition zone (T11 – L2) of the spine.[33] When symptomatic, patients often complain of sudden-onset severe, focal, back pain with little or no history of trauma. Back pain is typically mechanical in nature, such that it is exacerbated with sitting or ambulation and improves when lying supine.[33] While acute pain is the norm, VCFs may develop insidiously with chronic compression fractures only incidentally noted on radiographs performed for other reasons.

For the majority of VCFs only the anterior portion of the vertebral body collapses, with the posterior vertebral body wall remaining intact. As such, VCFs typically develop a wedge-shaped morphology that results in a kyphotic deformity of the spine, particularly in the presence of multiple VCFs with significant height loss. Such a kyphotic deformity, colloquially termed “Dowager’s hump”, may lead to chronic progressive back pain even after the fracture has healed.[33] In severe cases, this kyphotic deformity can result in restrictive pulmonary disease.[16]

Neurological injury is very uncommon (~0.05% of cases) in the setting of osteoporotic VCFs, largely because the low energy mechanism of injury does not routinely promote disruption of the posterior vertebral body wall. Even in those fractures where the posterior vertebral wall is disrupted (correctly termed burst fractures, vis-à-vis compression fractures), the low energy traumatic forces do not typically result in significant retropulsion of fracture fragments into the spinal canal. In the rare cases where boney retropulsion results in thecal sac compression, patients can present with symptoms consistent with thoracic myelopathy or cauda equina syndrome, requiring urgent surgical consultation.

Table 4: Pharmacologic Treatments for Osteoporosis[8]

Drug Category	Drug Name/Dose	Mechanism of Action	Efficacy
Bisphosphonates	• Aledronate 10mg po daily or 70mg po weekly • Risedronate 5mg po daily, 35mg po weekly, 150mg po monthly • Zoledronic acid 5mg IV yearly	• Decreases bone resorption by attenuating osteoclast activity	• All listed bisphosphonates have been shown to increase bone density and decreases both vertebral and non-vertebral fractures by 25-75%
Estrogen	• Conjugated estrogens (+ progesterone if intact uterus); dosing varies resorption	• Suppressive effects on osteoclast; decreases bone	• Increase bone mass • Decrease risk of vertebral and non-vertebral fractures by 23-34%
Selective Estrogen Receptor Modulators	• Raloxifen 60mg po daily agonist on bone tissue thus suppressive effect on osteoclasts • Acts as estrogen antagonist in uterine and breast tissue	• Act as estrogen • Reduces risk of invasive breast cancer	• Increases bone mass • Decreases vertebral compression fractures by 30-55%
Parathyroid hormone	• Teriparatide 20mcg daily sc injection	• Stimulates bone formation	• Increases bone mass • Decreases vertebral and non-vertebral fractures by 65% and 53% respectively
RANK ligand inhibitor	• Denosumab 60mg sc every 6 months osteoclast formation, activity and survival	• Decreases bone resorption by attenuating	• Increases bone mass • Decreases risk of vertebral fractures by 68%, hip fractures by 40% and other non-vertebral fractures by 20%

Table 4 continued: Pharmacologic Treatments for Osteoporosis[8]

Drug Category	Side Effects	Notes
Bisphosphonates	• Esophageal irritation • Osteonecrosis of the jaw (rare when used for long term osteoporosis treatment) • Low trauma atypical femur fractures (very rare, associated with >5 year use)	• Oral bisphosphonates should be taken on an empty stomach with lots of water • Patients should remain upright for 3-60 min after ingestion • Do not use if creatinine clearance is ≤ 35ml/min
Estrogen	• DVT and PE • Cardiovascular disease (in women >10 year post-menopause) • Stroke • Invasive breast cancer (only seen in combined estrogen/progesterone group)	• Only considered first line agent in women with menopausal symptoms
Selective Estrogen Receptor Modulators	• DVT and PE • Hot flashes • Leg cramps	• Does not reduce risk for non-vertebral fractures • Consider in women who have history of breast cancer and osteoporosis
Parathyroid hormone	• Leg cramps • Nausea • Dizziness • Possible increased risk of osteosarcoma	• Avoid in patients with an increased risk of osteosarcoma (history of Paget's disease, bony radiation, skeletal metastasis). • Lifetime duration of use should not exceed 18-24 months • When stopped, should be replaced by other anti-resorptive osteoporosis treatment, such as bisphosphonates
RANK ligand inhibitor	• Hypocalcemia • Increased risk of cellulitis • Osteonecrosis of jaw (very rare) • Atypical femur fractures (very rare)	• No restrictions in dosing according to renal function

Table 5: First line Therapies with Evidence for Fracture Prevention in Postmenopausal Women*[26]

Type of Fracture	Anti-resorptive Therapy						Bone Formation Therapy
	Bisphosphonates			Denosumab	Raloxifen	Hormone therapy (Estrogen)**	Teriparatide
	Alendronate	Risedronate	Zoledronic Acid				
Vertebral	✓	✓	✓	✓	✓	✓	✓
Hip	✓	✓	✓	✓		✓	
Non-vertebral	✓	✓	✓	✓		✓	✓

Non-vertebral fractures are a composite endpoint including hip, femur, tibia, humerus, radius and clavicle.
*For post-menopausal women, P indicates first line therapies and Grade A recommendation. For men requiring treatment, alendronate, risedronate and zoledronic acid can be used as first line therapies for prevention of fractures (Grade D)
** Hormone therapy (estrogen) can be used a first line agents in women with menopausal symptoms.

VCFs: Diagnostic Evaluation

Patients who are osteoporotic or have significant risk factors for VCFs should be screened annually, as described above. A few simple screening measurements, which can be performed in an office setting can help to significantly improve the likelihood of detecting a VCF on radiographic studies. These include a prospective height loss of greater than 2cm or a height loss, based on history, of more than 6cm, a rib-to-pelvis distance of less than 2 fingerbreadths, or a occipital-to-wall distance greater than 5 cm.[26]

For a patient presenting with signs and symptoms of a VCF, radiological studies should be ordered. The most cost-effective initial imaging is a plain radiograph of the thoracic and lumbar spine in both lateral and anteroposterior (AP) projections. A plain radiograph will allow quick assessment of degree of height loss, spinal alignment and progression of deformity on sequential images. VCFs are defined as having a minimum of 20% height loss relative to the unaffected portion of the vertebral body. Following plain radiograph identification of a VCF, an upright X-ray should be performed. Significant height loss or increased kyphotic angulation on this upright

radiograph as compared to the supine image may suggest fracture instability[9] and warrants further investigation with cross-sectional imaging such as computed tomography (CT). A plain radiograph may also have certain characteristics of osteopenia include increased skeletal lucency, loss of horizontal trabeculae and decreased cortical thickness, but increased relative opacity of end plates and vertical trabeculae.[1] Prior comparison films can help determine if an event is acute, but the age of the fracture can be difficult to determine accurately with radiographs alone.

While imaging studies such as magnetic resonance imaging (MRI) and/or CT can provide substantially increased anatomic detail, they are only indicated in a) patients with red flag signs or symptoms of pathological fractures of a neoplastic or infectious etiology, b) in patients with neurological dysfunction, c) substantial morphological changes between supine and upright radiographs suggesting mechanical instability or, d) if interventional management is being considered for refractory pain (discussed below).

CT of the spine is ideal for assessment of boney anatomy and allows for an accurate assessment of loss of height, fragment retropulsion and canal compromise. However, in patients with osteoporotic VCFs and no red flags (neurological symptoms, or symptoms suggestive of pathological fractures of a neoplastic or infectious etiology) such information has no clinical relevance. A CT scan may be ordered to help surgical planning in percutaneous vertebral cement augmentation (PVCA) cases (described below) as it can characterize the pedicle anatomy which may be useful to determine the appropriate needle path. A CT scan can also reveal chronic fractures through the presence of cortication.[33] MRI, however, is the best study to assess fracture age, as it will show boney edema associated with acute and sub-acute fractures and suggests that the VCF has not yet healed. Acute fractures exhibit low signal intensity on T1-weighted sequences and high signal intensity on T2 or short tau inversion recovery (STIR) sequences.[14] Only patients with refractory pain combined with “active” VCFs, as defined by the presence of bone edema on MRI should be considered for interventional management such as PVCA. Moreover, MRI can help detect a pathological fracture secondary to a malignancy or infection, if this is considered possible based on history or physical examination characteristics. MRI is the best test for the evaluation of neural compression in the rare setting of neurological symptoms.

VCFs: Management

Non-operative Management

Non-operative management is the mainstay of treatment of VCFs.

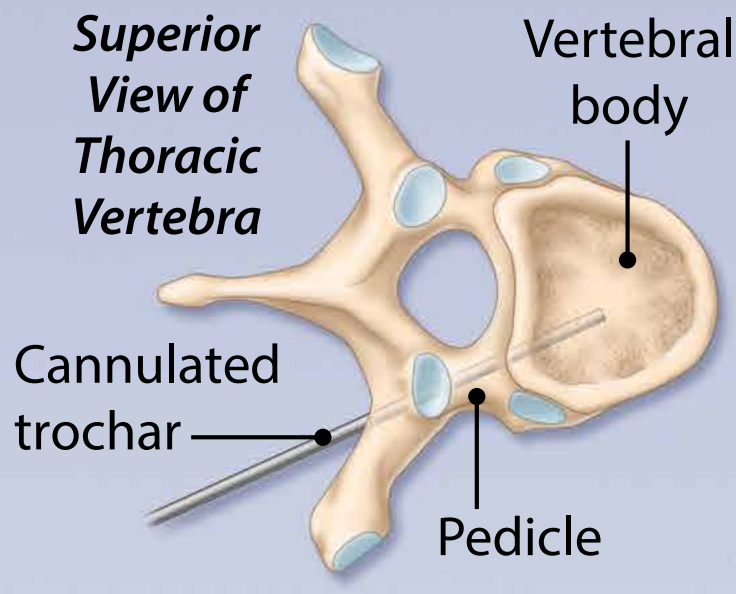
There are two main goals; the first goal is to provide pain relief and facilitate functional rehabilitation, as accomplished with analgesia and initial bed rest followed by gradually increasing activity as tolerated based on pain levels. For the subset of patients intolerant of ambulation after institution of an analgesic regiment and a short period (i.e. a few days) of bed rest, orthotic bracing can be considered. The second goal of treatment is to properly complete an assessment of ongoing fracture risk and provide appropriate prophylactic treatment against further frailty fractures (as described previously).

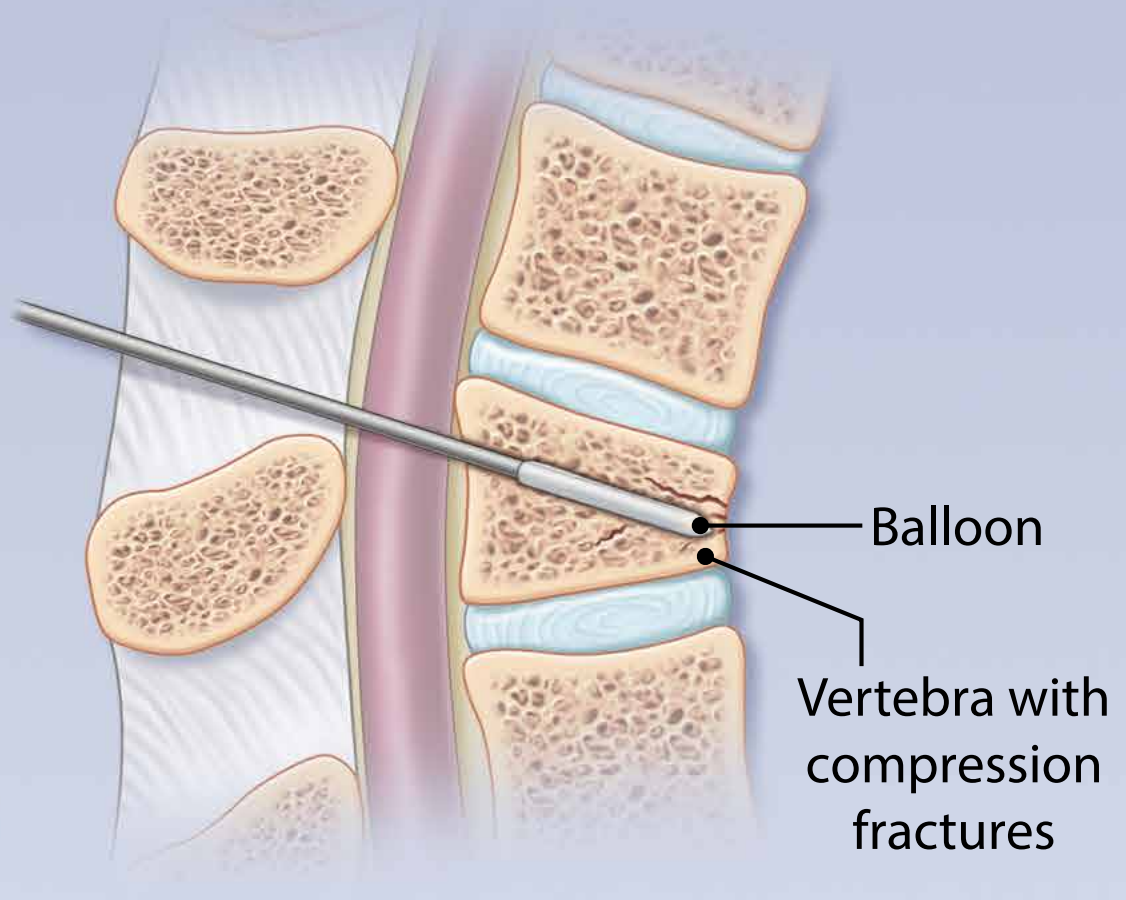
Acute pain from a new VCF usually improves over a period of 6 weeks.[19] The first line analgesic medications should be acetaminophen and non-steroidal anti-inflammatory drugs (NSAIDs). The risk of gastrointestinal bleeding and renal insufficiency should be taken into account when prescribing NSAIDs. Opioids should be prescribed for patients failing first-line therapy. Opioids such as oxycodone or codeine may be combined with acetaminophen for additive effects. Opioids, however also can have significant side effects in the elderly, including reduced gastrointestinal motility, urinary retention, reduced respiratory drive, somnolence, cognitive deficits, and an increased fall risk.[4]

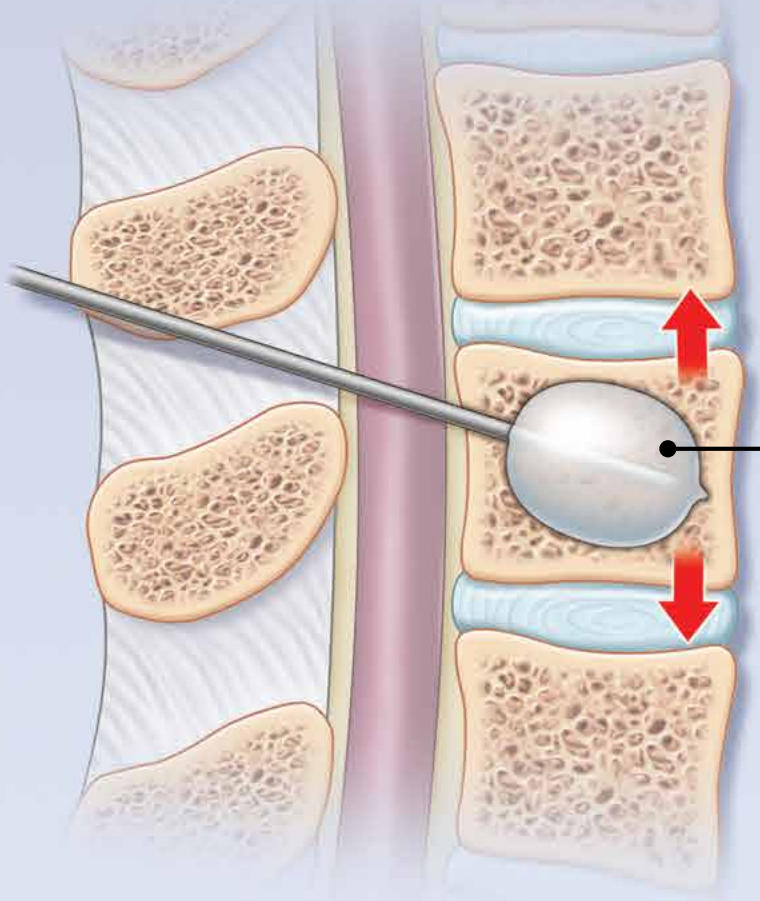
Medications used to treat osteoporosis may also provide pain relief in patients with acute VCFs. A recent systematic review of 13 randomized controlled trials by Knopp-Shiota et al (2012)[13] noted that calcitonin significantly reduced the severity of acute pain related to osteoporotic VCFs. Pain was reduced by 1 week with continued improvement through 4 weeks. Similarly, Nevitt et al. (2006)[24] demonstrated that teriparatide (Forteo), provided pain relief in osteoporotic VCFs; patients randomized to teriparatide had a reduced incidence of new or increasing back pain compared with a placebo, hormone replacement therapy or alendronate.

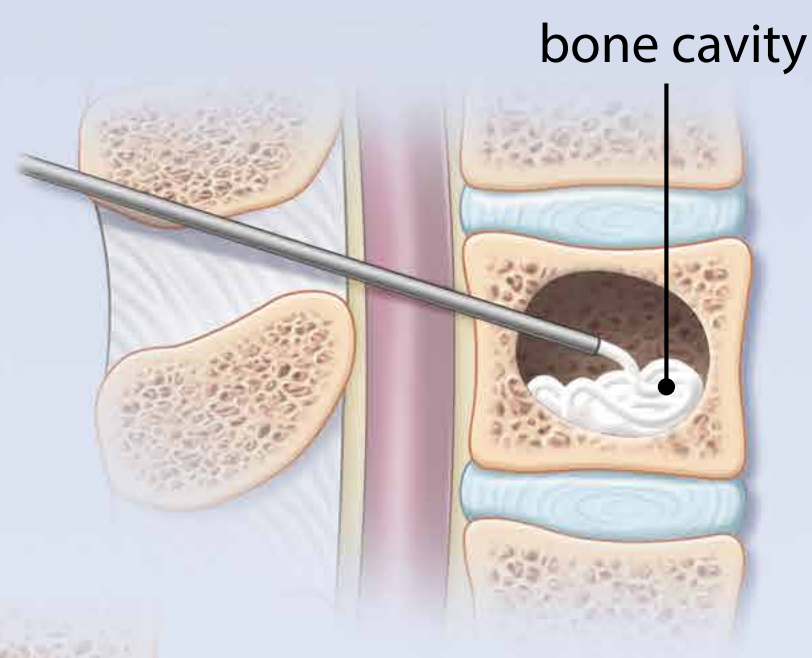
Bracing is often used in the non-operative management of osteoporotic VCFs, but the evidence to support this practice is limited. The primary goal of bracing is to reduce mechanical pain by stabilizing the spine, with a secondary goal of limiting progression of spinal deformity.[29] The majority of evidence supporting bracing of spinal fractures is in the setting of acute non-osteoporotic fractures such as thoracolumbar burst fractures, which, from both a fracture mechanism (high vs. low energy) and bone biology perspective, is not readily translatable to the clinical scenario of osteoporotic VCFs. To date, there has only be one randomized control trial (Pfeifer et al., 2004)[28] that showed bracing for 6 months in patients with osteoporotic VCF was associated with

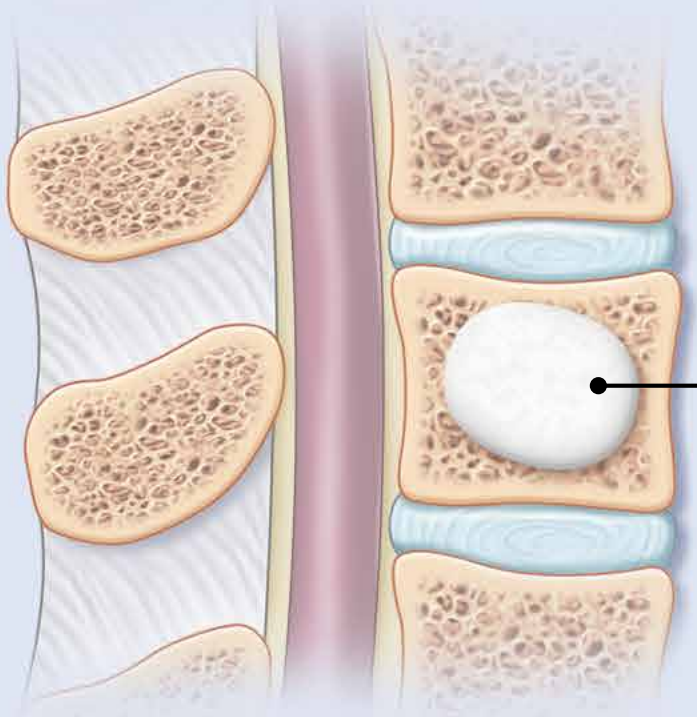
Percutaneous Vertebral Cement Augmentation (PVCA) Technique











a marginal clinical benefit (38% decrease in average back pain, a 15% improvement in overall well-being and 27% decrease in limitations of daily living), as compared to patients that did not complete a bracing trial. Bracing is not wholly benign and there are numerous potential complications, including patient discomfort, leading to decreased compliance, pressure ulceration if braces are ill-fitted, and deconditioning and muscle atrophy of the trunk and paraspinal muscles in the setting of prolonged bracing. Given the paucity of evidence to support bracing in the setting of osteoporotic VCFs, combined with the potential pitfalls, it is our view that bracing should only be considered in individuals that are unable to mobilize secondary to axial back pain complaints after optimization of analgesia and bed rest of a few days duration.

Operative Management

While there is no universally accepted time period that constitutes a reasonable trial of non-operative management, the vast majority of patients should realize significant pain relief by 6 weeks post-VCF.[33] In the small subset of patients that continue to have unremitting pain, consideration of operative management is appropriate. Patients that have significant back pain around the fracture area that increases with axial loading and have MRI evidence of bone edema consistent with an acute or sub-acute fracture can be reasonably considered as candidates for intervention to decrease VCF related back pain. Patients with nonspecific back pain and those with cross-sectional imaging (CT and/or MRI) that demonstrates healed fractures without active marrow edema are highly unlikely to benefit from operative intervention. Obviously, patients that present with objective acute neurological dysfunction (e.g. cauda equina syndrome or myelopathy) secondary to neural element compression require urgent decompression surgery. Other neurological symptoms such as radiculopathy, neurogenic claudication, and evidence of instability on upright radiographs do not require urgent surgical management but reasonably warrant a spine surgical referral on an elective basis.

The most commonly performed interventional procedure for osteoporotic VCFs are percutaneous vertebral cement augmentation (PVCA) methods such as vertebroplasty or kyphoplasty. PVCA involves fluoroscopically-guided transpedicular insertion of a cannulated trochar into the central aspect of the target vertebral body, with injection of radio-opaque cement, most commonly polymethylmethacrylate (PMMA), into the vertebra.[11] These procedures have recently gained widespread acceptance as an effective method

of pain relief and have become routine therapy for osteoporotic VCFs unresponsive to initial non-operative care. The mechanism of pain relief following PVCA is not well understood, but is postulated to be secondary to a combination of improved mechanical stability, as afforded by the injected cement and destruction of the somatic nerves innervating the adjacent soft tissue due to the PMMA hardening exothermic reaction.[27]

Vertebroplasty and kyphoplasty differ only in the addition of an inflatable balloon that is threaded into the VCF after transpedicular trochar placement and then expanded to compact the adjacent trabecular bone, thus creating a cavity for PMMA injection. Theoretically, the balloon expansion of kyphoplasty can provide improved kyphosis correction in comparison to vertebroplasty but the degree to which this correction occurs is typically marginal, inconsistent and of unclear clinical significance.[27] PVCA procedures are usually well tolerated and can typically be performed as outpatient procedures.

Despite the widespread adoption of PVCA in the setting of osteoporotic VCFs, there is significant controversy surrounding their true efficacy. McGirt et al. (2009)[22] published a systematic review outlining all vertebral augmentation procedures over a 20 year period. The article outline 74 vertebroplasty studies (1 level I, 3 level II and 70 level IV) and 34 kyphoplasty studies (all level IV) for osteoporotic VCFs. There was good evidence (level I) that vertebroplasty results in superior pain control within the first 2 weeks of intervention compared to optimal medical management.[22] There was fair evidence (level II-III) that vertebroplasty results in less analgesia use, less disability and greater improvement in general health when compared to medical management. Additionally, there was some evidence that by 2 years after intervention, vertebroplasty provides similar degree of pain control and physical function as optimal medical management.[22] Consistent with these results, the Vertos II trial[12] randomized 202 patients into vertebroplasty versus medical management and demonstrated that vertebroplasty resulted in greater pain relief than medical management at both 1 month and 1 year.

The FREE trial, which was a prospective randomized controlled trial evaluating the efficacy and safety of kyphoplasty compared to medical management (non-surgical group) in vertebral VCFs also demonstrated similarly favorable results.[32] The authors randomized 300 patients into the kyphoplasty versus non-surgical care arm. Their primary endpoint was an improvement in the SF-36 physical component summary (PCS) at 1 month. The study showed a significant reduction in SF-36 PCS scores in

SUMMARY OF KEY POINTS

1. Osteoporosis is under diagnosed in Canada. Early diagnosis, fragility fracture risk stratification and initiation of preventative treatment is important, as osteoporotic vertebral compression fractures (VCFs) have a significant associated personal and societal health utility cost.

2. Patients suspected of having a VCF should have an AP and lateral X-ray of the suspected region. If VCF is confirmed, an upright X-ray should be performed to assess for stability. CT and/or MR imaging has limited utility in the absence of red flag signs or symptoms.

3. VCFs should be managed with initiation of an appropriate pain management regiment, early bed rest as required for pain control and gradual mobilization. Patients with refractory pain 4–6 weeks after onset can be considered for percutaneous vertebral cement augmentation (e.g. vertebroplasty), although the clinical efficacy of such procedures remains unclear.

the kyphoplasty compared to non-surgical arm at 1 month. However, this effect diminished by 12 months, as improved scores were noted in the non-kyphoplasty group, likely secondary to fracture healing over time.

In deference to the multiple studies suggesting a clinical benefit for PVCA techniques, two prospective, double-blinded randomized placebo controlled trails published in the New England Journal of Medicine questioned the efficacy of PVCA for osteoporotic VCFs. These studies, conducted by Buchbinder et al.[3] and Kallmes et al.[11] compared vertebroplasty with sham procedure groups, rather than comparing to a medical management group. Both of these studies reported no difference in pain control or function between the two arms; from 1 week to 6 months follow up in one study and 1 month follow-up in the other.[3,11] They concluded that the benefits of vertebroplasty in prior studies were secondary to a procedural placebo effect.[3,11,33] However, both of these studies had small sample numbers (78 and 131 patients, respectively), low volumes and infrequent rate of vertebroplasty performed at the centers over a long time interval, lack of clear inclusion criteria specifying patients with mechanical axial back pain and suboptimal volumes of cement injection.[3,11,33] In an attempt to address these shortcomings and provide some further clarity on the subject, there is an ongoing prospective randomized trial, VERTOS IV, that will compare vertebroplasty to a sham procedure similar to the studies conducted by Kallmes and Buchbinder, but using the stricter inclusion criteria of the VERTOS II trial.[12]

CME

Post-test Quiz

Members of the College of Family Physicians of Canada may claim MAINPRO-M2 Credits for this unaccredited educational program.

Operative Management: Complications

PVCA associated complication are uncommon and range from 0.3% to 3.9%.[22] Vertebral augmentation can promote fracture of adjacent vertebral bodies due to stiffness of the augmented body. Serious complications have been documented and include cement extravasation into the spinal canal or neurofo-ramen (0.4-4%),[22] with resulting myelopathy, paralysis or painful radiculopathy requiring open surgical decompression to remove the cement bolus. Rarely, cement boluses can enter the epidural venous system and result in pulmonary embolism.[22,33] Peri-procedure fatality risk is exceedingly low, with reports almost exclusively in relation to pulmonary cement embolism.[33]

PVCA is contraindication when there significant incompetence of the posterior vertebral wall with or without boney retropulsion, as the risk of cement extravasation into the canal is significant.[33] Further, PVCA is not technically feasible in cases with completely collapsed vertebral bodies. Other contraindications include active osteomyelitis of the fracture site, allergy to cement, and coagulopathy.[27]

Conclusions

Osteoporotic VCFs can have significant impact on a patient's quality of life and is associated with high socioeconomic cost and morbidity. It is essential to identify patients who are at risk of fragility fractures so appropriate therapy can be instituted to prevent VCFs. Patients who are suspected of suffering from an VCF should undergo plain radiographs of their thoracolumbar spine. Conservative management with analgesia and progressive mobilization should be attempted as a first line therapy. There is insufficient evidence to support the use of routine bracing of VCFs. If

CLINICAL PEARLS

A few screening measurements can be performed in the office setting to help significantly improve the likelihood of detecting a VCF on radiological studies. They include prospective height loss of greater than 2cm or a height loss, or a height loss based on history of more than 6cm, a rib-to-pelvis distance of less than 2 fingerbreadths, or an occipital-to-wall distance greater than 5cm.

Most patients with osteoporotic VCFs do not need a referral to a spine surgeon. Acute pain from a new VCF usually improves over a period of 6 weeks. Non-operative management should follow the WHO analgesic ladder starting with acetaminophen/NSAIDs followed by opioids, as necessary. The goal of treatment is to provide pain relief and facilitate early functional rehabilitation.

Patients with high or medium 10-year fracture risk should be considered for pharmacotherapy to prevent the progression of low bone mineral density and osteoporotic fractures.

patients fail non-operative therapy, and MRI imaging shows evidence of a non-healed acute/subacute fracture, consideration of PVCA procedures is reasonable, with the understanding that the true clinical benefit of PVCA is unclear at present. VCFs presenting with acute neurological dysfunction is very rare, but all such individuals should be referred urgently for decompressive and stabilizing spinal surgery.

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