Vertebral compression fractures

ABSTRACT

This review gives a practical guide to the investigation and management of osteoporotic vertebral compression fractures. With an ageing population, the burden of disease and health system costs attributable to this common injury continue to rise. This article outlines the epidemiology, clinical and radiological assessment of vertebral compression fractures, and key decisions that must be made in their management. It reviews the indications and evidence for conservative *vs* operative treatment, discusses the rationale for vertebroplasty, kyphoplasty and spinal stabilization, and looks at outcomes in this vulnerable patient population. It also reviews key evidence underlying decision making including National Institute for Health and Care Excellence guidelines.

Vertebral compression fractures are the most common type of fragility fracture, with an estimated annual incidence in the UK of approximately 120 000 (Iqbal and Sobhan, 2002). They are associated with significant morbidity, with patients often developing chronic pain, and are an important marker of frailty. Indeed, in the year after a vertebral compression fracture, the risk of sustaining a fractured neck of femur is increased fivefold and there is a 20% risk of sustaining a further vertebral compression fracture (Lindsay et al, 2001). With an ageing population, the incidence continues to rise and is nearly eightfold higher in women aged between 85 and 89 years of age compared to those aged 60–64 years.

The cost of managing fragility fractures in the UK was estimated at £2.3 billion in 2011 and is projected to increase to more than £6 billion by 2036 (National Institute for Health and Care Excellence, 2012b). Understanding the pathophysiology and management of vertebral compression fractures is therefore of key clinical and health economic importance.

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Risk factors

Vertebral compression fractures in the elderly are usually caused by low energy trauma in osteoporotic patients. The main risk factors, in addition to any factor that increases susceptibility to falls, are therefore those related to an increased risk of osteoporosis as outlined in *Table 1* (National Institute for Health and Care Excellence, 2012a).

Clinical assessment

Vertebral compression fractures usually present with acute, axial back pain, although many occur on a background of chronic lower back pain (often as a result of previous vertebral compression fractures) and have a more insidious onset. There may be a history of trauma although patients with severe osteoporosis can fracture during trivial events such as coughing or sneezing and there is evidence to suggest that up to 30% of compression fractures in patients with severe osteoporosis occur while the patient is in bed (Kim and Vaccaro, 2006). A history of malignancy, red flag symptoms including recent weight loss or night sweats, and risk factors for osteoporosis as outlined above should be sought. Patients must also be asked about neurological deficit, including bladder and bowel function, and when these symptoms began.

Initial assessment for suspected vertebral compression fracture centres on three key questions:

1. Is the fracture new or old?
2. Is there any neurological deficit?
3. Is the fracture stable or unstable?

On clinical examination, patients with an acute vertebral compression fracture usually report local tenderness at the level of the fracture although this may not be present in cases of anterior column fracture. A kyphotic deformity can also be indicative of previous fractures. A full

Table 1. Risk factors for osteoporosis
Age >65 years men, >50 years women
Previous osteoporotic vertebral compression fracture
Smoking
Asian or Caucasian race
Long-term corticosteroid use
Family history
Rheumatoid arthritis and connective tissue disease
From National Institute for Health and Care Excellence (2012a)

neurological assessment is required including evaluation for radiculopathic features and markers of cauda equina compression including perineal sensory loss. If there is any suspicion of new onset neurological deficit urgent discussion with the local spinal surgical team is required.

Imaging

Initial imaging involves anteroposterior and lateral radiographs of the affected spinal region. Vertebral compression fractures most commonly occur at the thoracolumbar junction (T12–L1) and the mid-thoracic regions (T6–T8) (Patel et al, 1991). Lateral radiographs may show a wedge-type fracture pattern with loss of anterior vertebral height or a burst-type pattern with loss of height of both the anterior and posterior aspects of the vertebral body. Distinguishing old fractures from acute fractures on plain radiographs is challenging. Classically, acute fractures will show well-demarcated fracture lines, while sclerotic margins and osteophyte formation are more suggestive of older fractures. However, the possibility of an acute-on-chronic injury should always be considered. Further imaging in the form of computed tomography or magnetic resonance imaging is recommended to help delineate the fracture pattern, assess stability and, in the case of magnetic resonance imaging, to allow assessment of fracture chronicity (*Figure 1*).

There are a number of criteria for a fracture to be considered ‘unstable’. Burst-type fractures with >50% loss of vertebral height, significant fragment retropulsion, >25–35° of kyphosis and fractures with involvement of the posterior column bony or ligamentous structures should be considered unstable. Computed tomography will enable column integrity to be assessed and can be used to characterize the extent of any retropulsion. Magnetic resonance imaging allows assessment of fracture chronicity by demonstrating vertebral body oedema within an acute fracture, although this may not always be seen. Magnetic resonance imaging is also important when planning subsequent management (as patients with evidence of vertebral body oedema may be amenable to balloon kyphoplasty or vertebroplasty), as well as allowing for evaluation of neural compromise and ligamentous integrity.

Treatment

Treatment of vertebral compression fractures aims to reduce pain, restore mobility and reduce the risk of further vertebral compression fracture. Treatment modalities include conservative management, balloon kyphoplasty and vertebroplasty, and surgical stabilization. In all cases patients must also be considered for treatment of underlying osteoporosis and falls prevention.

Conservative treatment

Analgesia should follow the World Health Organization pain ladder and, for stable fractures, early mobilization should be encouraged as pain allows. The use of orthoses such as a thoracolumbosacral orthosis may be helpful for temporary support but there is little evidence to support their efficacy and they are not a form of definitive stabilization (Urquhart et al, 2017). Advice can also be sought from a multidisciplinary pain team as pain following a vertebral compression fracture may be highly disabling and often becomes chronic.

Figure 1. **a.** Lateral radiograph showing T11 and T12 burst fractures and superior endplate fracture of L2. **b.** Magnetic resonance imaging (sagittal STIR sequence) of the same patient showing oedema within the T12 vertebral body suggesting this is an acute fracture.

Balloon kyphoplasty

Patients with an acute fracture, with oedema present within the vertebral body on magnetic resonance imaging, may be candidates for balloon kyphoplasty which primarily aims to relieve pain but may also prevent further collapse and partially restore vertebral height to reduce kyphotic deformity. Kyphoplasty involves inserting a balloon-like device into the fractured vertebra(e) under radiographic guidance, usually using sedation and local anaesthetic. Inflating the balloon then helps to restore vertebral height. Following this, the balloon is deflated and the air-filled space injected with bone cement (*Figure 2*). There is also the option to stent during kyphoplasty. This involves inserting a small balloon catheter surrounded by a metal stent into the vertebra with the stent remaining in the cavity when the balloon is deflated and withdrawn, functioning as a scaffold to prevent the vertebra from losing height as the balloon is deflated.

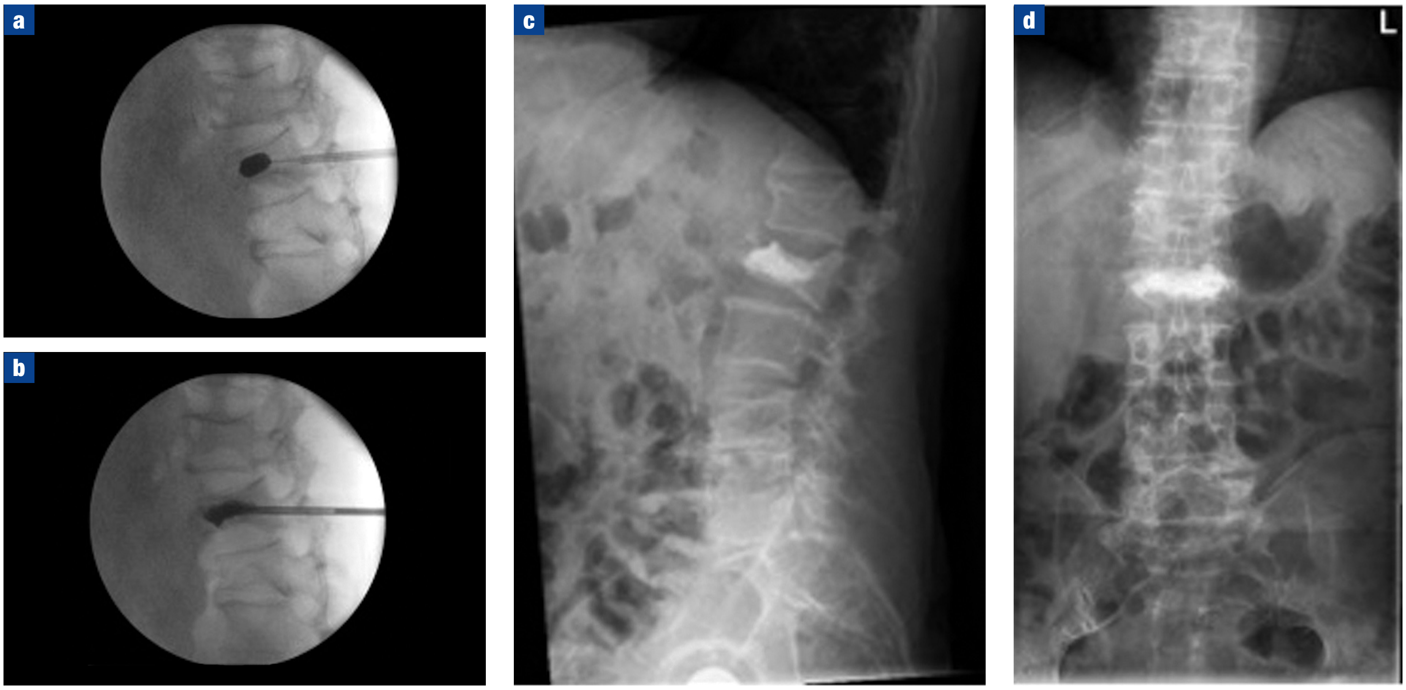

Figure 2. Balloon kyphoplasty procedure. **a.** Under image intensifier guidance an entry needle is inserted into the fractured vertebral body via the pedicle and a balloon inflated. **b.** The balloon is deflated, removed, and radio-opaque cement injected into the cavity created by the balloon. **c.** Postoperative lateral and **(d)** anteroposterior radiographs confirm appropriate positioning of cement within the vertebral body.

The National Institute for Health and Care Excellence (2006) supports the use of percutaneous balloon kyphoplasty as an option for treating vertebral fractures but there are a number of important considerations. National Institute for Health and Care Excellence guidance states that kyphoplasty is recommended for patients with 'severe on-going pain after a recent unhealed fracture despite optimal pain management', and only in whom the 'pain has been confirmed to be at the level of the fracture by physical examination and imaging' (National Institute for Health and Care Excellence, 2006).

Vertebroplasty

Vertebroplasty involves injecting bone cement directly into the vertebral body without prior balloon inflation. It is also approved by the National Institute for Health and Care Excellence (2003), although since a balloon is not used to expand the cavity the cement used is less viscous and must be injected at higher pressure and thus the risk of cement leakage is greater (National Institute for Health and Care Excellence, 2003).

An open-label prospective randomized trial, VERTOS II, showed improved pain control and physical function with vertebroplasty at 1-year follow up when compared to conservative management (Klazen et al, 2010a). However, two randomized controlled trials comparing vertebroplasty and sham procedures reported no difference in functional outcomes or pain at 6 months follow-up (Buchbinder et al, 2009; Kallmes et al, 2009). These latter trials included patients with both acute and chronic injuries. The VERTOS IV trial, which is yet to be reported, seeks to shed light on this topic by means of strict inclusion criteria including evidence of oedema on magnetic resonance imaging and symptom duration less than 6 weeks (Firanescu et al, 2011). Both vertebroplasty and kyphoplasty are widely used depending upon local availability and expertise. Further research is needed to elucidate the cost-benefit profile of each. The authors advise discussing all cases of vertebral compression fracture with the local spinal surgical service to confirm and clarify local policy.

The optimal timing of kyphoplasty and vertebroplasty is also controversial. In the authors' view, balloon kyphoplasty may be considered if a patient has an acute fracture with evidence of vertebral body oedema on magnetic resonance imaging and persistent symptoms. As symptoms may improve over time, a period of conservative management is often trialled first. However, intervening early may prevent further vertebral collapse, especially in the case of anterior wedge fractures, and allow more rapid mobilization (Klazen et al, 2010a). Again, evidence from open-label randomized trials supports early intervention, yet masked trials have not shown the same benefits. The VAPOUR trial showed vertebroplasty was more effective at managing pain than placebo in vertebral compression fracture within 6 weeks of onset in elderly patients (Clark et al, 2016). Subgroup analysis showed that the maximal benefits were in patients with thoracolumbar fractures. A meta-analysis comparing vertebroplasty and kyphoplasty has reported equivalent outcomes with both techniques, with no difference in either short- or long-term pain and disability scores (Gu et al, 2016).

Adverse reactions to both kyphoplasty and vertebroplasty include infection, bleeding, neurological damage as a result of needle insertion, systemic reactions and cement leakage, including into the adjacent disc, with a rate of 9% in kyphoplasty and up to 41% in vertebroplasty (Hulme et al, 2006). New vertebral fracture caused by kyphoplasty can be as high as 11.1% and often occur in the vertebrae adjacent to that containing cement as a result of increased stiffness of the spine at this level (Taylor et al, 2007). There is also a very small risk that the balloon can rupture in kyphoplasty resulting in retention of balloon fragments within the vertebral body.

Open spinal stabilization

Open surgical stabilization may be required if the fracture is considered unstable, and is often coupled with local decompression if there is evidence of neurological deficit. Stabilization relieves pain by reducing movement at the fracture site and also prevents further displacement and potential nerve root or spinal cord compromise resulting from this. The surgery typically involves insertion of pedicle screws into the vertebrae above and below the level of the fracture, which are then secured together using connecting bars (*Figure 3*). By using the pedicle screws to distract the intact vertebral segments before tightening, ligamentotaxis can be achieved that enables reduction of retropulsed fragments and decompression of the spinal canal. In osteoporotic bone, cannulated pedicle screws allow insertion of cement into the vertebral body to allow greater purchase and reduce the risk of screw pull-out.

Important considerations

There should be a high index of suspicion for osteoporosis in patients with a vertebral compression fracture. A vertebral compression fracture should trigger a review and diagnosis and/or treatment optimization of underlying osteoporosis. Of note, bone density measurements are not essential before starting treatment for osteoporosis in patients with low-energy vertebral compression fractures but may be useful to confirm the diagnosis. Supplementary calcium and vitamin D, smoking cessation and reducing alcohol intake are crucial to preserve cortical thickness and trabecular microarchitecture, and consideration of secondary fracture prevention (e.g. through the use of bisphosphonates). Effective analgesia is also important as immobility caused by pain will result in further loss of muscle strength and function, increasing disability as well as the risk of falls. It should be noted that bone density measurements from the lumbar spine might be falsely normal, as concomitant osteoarthritis can cause sclerosis that increases local bone density and thus the average value, despite the vertebral body being osteopenic.

Long-term outcomes

In most patients with an acute vertebral compression fracture, pain decreases significantly with conservative management, predominantly in the first 6 months (Klazen et al, 2010b). However, in one prospective study, a third of patients still had severe pain necessitating oral analgesia and physical therapy 2 years after an acute vertebral compression fracture (Klazen et al, 2010b), and 20% of patients with a symptomatic vertebral compression fracture can be expected to develop chronic pain (Venmans et al, 2012). In the longer term, vertebral compression fractures have been shown to adversely affect quality of life, mental health and physical function (Silverman, 1992).

Patients whose pain is uncontrolled despite optimal therapy may benefit from a specialist multidisciplinary pain team review. Alternative therapies such as acupuncture, transcutaneous electrical nerve stimulation and cognitive behavioural therapy may help improve quality of life.

Vertebral body fractures can lead to kyphosis and a loss of height as they heal. This may affect the sagittal profile of the patient leading to poor posture. In turn, this may cause a compensatory increase in lumbar lordosis and secondary lower lumbar back pain as a result of facet joint overload arthropathy. This is a potentially avoidable consequence if vertebral body shape can be restored through the use of kyphoplasty.

Conclusions

Vertebral compression fractures are increasing in incidence and have a significant effect on quality of life and high socioeconomic costs. Clinically, the most important questions are whether there is any neurological deficit,

Figure 3. **a**. An acute burst fracture of T12 is shown on the sagittal computed tomography. **b.** This was treated with T11 to L1 posterior instrumented stabilization, with the pedicle screws inserted into T11 and L1 being connected via rods and cement introduced through these screws to increase construct stability. An incidental finding of a grade II L4/L5 spondylolisthesis is also noted.

KEY POINTS

- Vertebral compression fractures are increasingly common, with potentially devastating sequelae and significant costs to the health-care system.
- An understanding of the rationale for using either radiographs, computed tomography or magnetic resonance imaging to characterize these injuries is important.
- While conservative management is often used, surgical options in appropriate patients include kyphoplasty, vertebroplasty or spinal stabilization.
- There remains controversy around selection criteria for surgery and when it should be performed.
- All patients sustaining a vertebral compression fracture should undergo falls assessment and an osteoporosis screen.

whether spinal stability is compromised and if the fracture is new or old. Computed tomography and/or magnetic resonance imaging are therefore necessary for full evaluation. Balloon kyphoplasty and vertebroplasty are treatment options that may be useful for acute fractures, with unstable fractures requiring instrumented stabilization. The evidence surrounding the relative benefit and timing of each remains unclear but studies are currently in progress to answer these important questions. **BJHM**

Conflict of interest: none.

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