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Worsening Back Pain: To Inject or Not to Inject That Is the Question

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Stem Case and Key Questions Content

A 91 year old female is admitted to the hospital after experiencing 2 weeks of worsening back pain. She has a long standing history of back pain managed with Tylenol. While walking independently at home, she fell in her bathroom. Now pain limits her mobility. She has fallen many times in the past few months but never experienced the present degree of pain. The pain is localized to her back, and non-radiating. There is no obvious neuro-deficit. Her medical history is significant for hypertension, coronary artery disease and dementia. The pain service is consulted for possible interventional therapy because opioid therapy has been unsuccessful.

1. How should we proceed? Do we have enough information?

The history is revisited. The patient's son says her mother lives alone with a 24/7 helper at home. He visits her every day. She could walk easily with a walker to the bathroom before the fall. She does not take any narcotics and did not complain of severe back pain previously. She is hard of hearing. He realizes that his mother is old; he wants her to go back to baseline status but requests no heroic measures.

2. What is the next step?

A physical examination is conducted. She cannot sit, stand or even turn by herself in the bed. With the help of a nurse she is turned to her side. Examination reveals no bruises and mild to moderate kyphosis. Her back is not tender to palpation but it's tender to deep percussion. She moves both her legs but her strength cannot be tested because she is uncooperative. The patient moans and groans during the physical examination in the presence of her son who says she was not like this 2 -3 weeks ago.

3. What should we do now?

An MRI of the spine shows a multiple compression fracture at T12, L2 and L4, spinal stenosis, grade I anterolisthesis of L4 over L5, severe degenerative disc disease, and facet arthropathy with severe ligamentaum hypertrophy contributing to spinal stenosis at L2-3, L3-4 and L4-5 levels.

4. What are your questions for the radiologist?

You ask the radiologist to comment on the age of the fractures. He says T12 is definitely old but L2 and L4 are of undetermined age.

5. Should laboratory tests be ordered in anticipation for injection therapy?
A CBC and coagulation profile done on admission reveals that her Hb is 9.5, Platelets are 110, and INR is 1.3. She takes aspirin daily.

6. After a week in the hospital, is there any urgency to treat this back pain?
The patient is visited by the physical therapy team and her primary team orders a hard brace. The physical therapy team is not sure how active the patient can be with a fracture in her back as they were not given any guidelines by the primary team. The patient does not want to wear the hard brace in bed.

7. What would you tell the Physical Therapy team and the nurse?
You call the nurse and tell her the brace can stay off while the patient is in bed. You tell the physical therapy team that the goal is to make the patient ambulate as much as possible to prevent bedsores and deconditioning.

8. How do you correlate MRI finding with her back pain? What is the value of dating compression fracture? How do you date a compression fracture? What if MRI is contraindicated?
MRI does not explain her pain completely. Clinically tenderness over mid back with L2 compression fracture suggests that her pain might be from the compression fracture. MRI with STIR images is best imaging technique to date a compression fracture. Acute fracture is differentiated from chronic fracture by the presence of inflammation and increase blood flow which can be seen on MRI. If MRI is contraindicated, then CT with PET scan can reveal the same information.

9. Should you consider kyphoplasty or vertebroplasty?
You suggest to the patient's son about the possibility of kyphoplasty or vertebroplasty. The patient's son is reluctant and is agreeable to proceed if this is the best option. You arrange a physical therapy session in the presence of the patient's son. Two people are needed to help the patient sit at the bedside. When asked if she has pain she says her back hurts.

10. Is it a clear signal to proceed? What's the role of facet joints in causing pain in compression fracture?
Her son is not convinced of the seriousness of his mother's pain. He wants to know risks of the procedure. You explain that the risks of procedure are infection, bleeding, and life-threatening embolism. He wants to know which of the two procedures is best for his mother. You explain to the son that both vertebroplasty and kyphoplasty are equally safe and effective procedures. There is less risk of cement embolism with kyphoplasty which you recommend. The patient is scheduled for L2 kyphoplasty.

11. How do you plan for kyphoplasty? What do you think of her lab? If patient had more than one compression fracture, how many would you do in one setting?
You evaluate the patient's vertebral anatomy. The walls of her vertebral body seem intact. There is mild retropulsion of posterior wall but no cord compression. In the OR, the vertebral body is accessed via the transpediculuar approach. A balloon is used to create a cavity, but it blows out the side wall of the vertebra. You perform the egg -shell technique to plug the hole. It works well. You inject 0.5 ml cement at a time under live fluoroscopy to a total of 2 ml of cement in the body of the vertebra. You notice a cement leak in front of the vertebral body. You stop

and inject a little more. As the cement hardens so you try to inject cement a little faster. You notice that cement is backtracking towards the spinal canal.

12. What is an optimal volume of cement injection per vertebra? What are the dangers of cement leak outside the vertebral body including intravascular leak?
The procedure is terminated. The patient is hemodynamically stable. She recovers from sedation and is now wide awake in the PACU complaining of leg pain. She moves her leg but her strength is hard to assess because she is agitated by new pain.

13. What has happened? Do we need a CT or MRI?
The patient moves her legs slowly, and her knee reflexes are intact. The ankle reflexes are hard to obtain and she has negative Babinski sign. A CT of the spine is done which shows cement leak in the spinal canal, more than you suspected. There is some leakage towards the neural foramen. The radiologist does not suspect cord compression but suggests an MRI to be sure. You ask for a surgical consult. The surgeon considers patient too high a risk for surgery and recommends against getting an MRI as she does not show signs of cord or severe nerve compression.
The next day the patient is still having radicular leg pain that limits her activity. You suggest a lumbar epidural steroid injection. The son agrees. After lumbar epidural steroid injection, she experiences significant pain relief but still is not walking yet. The son wants to know why. You explain that she needs extensive rehabilitation to improve. She is discharged to inpatient rehabilitation.

14. Did we cure the patient's pain? Why /why not? What can still cause back pain after kyphoplasty? Should we expect to see her again?
The patient's problem is osteoporosis, not compression fracture. There is no cure for osteoporosis. Drugs only slow down the progression of osteoporosis but do not reverse it. Kyphoplasty is known to slightly increase the risk of compression fracture at the adjacent level. In hindsight would you have performed kyphoplasty in this patient?

Model Discussion Content

Epidemiology of the Problem:
Osteoporosis is a systemic skeletal disorder characterized by low bone mass and micro-architectural deterioration of bone tissue. In the United States, up to 10 million individuals have the disease, and almost 34 million more have low bone mass, increasing their risk for fracture later in life.[1] In the US, osteoporosis is responsible for more than 1.5 million fractures annually, half of which happens in the spine.

Vertebral fractures can be symptomatic or found incidentally on x-rays called asymptomatic fractures. The incidence of asymptomatic vertebral fractures approximately doubles in women every ten years, from less than 5/1,000 person years at age 50 to about 25/1,000 person years by the late 70s. In men, the incidence also increases with age, but at a slower rate. The lifetime risk of a clinical vertebral fracture at the age of 50 years is 3.1% for women and 1.2% for men.[2] Clinical vertebral fractures are associated with substantial morbidity, causing back pain, kyphosis, loss of height and poor quality of life. Data from the Study of Osteoporotic Fractures in

the US shows that mortality increases with the number of vertebral fractures. In 1998 it was estimated that osteoporotic fractures cost £942 million annually in the UK, of which only £12 million was due to the acute cost of vertebral fractures. Previous vertebral fracture increases the risk of further vertebral fractures and hip fracture.[3] Early intervention with treatment for osteoporosis may prevent further vertebral fractures and the associated back pain.
Patients develop thoracic kyphosis and lumbar lordosis, and may have reduced exercise tolerance. They have been found to have reduced spinal extensor muscle strength. Since self-care and dressing often become difficult, patients become dependent on family members for help. The quality of life is most affected by lumbar compression fracture.[4]

Pathogenesis of back pain associated with vertebral fractures:
The nature of pain associated with vertebral fracture is multifactorial. Back pain may arise either directly from vertebral fracture or indirectly from the consequences of spinal deformity. There is also mechanical strain on associated muscles, posterior ligaments and facet joints. Nerve root compression may result in radicular pain, and rarely fracture compress the spinal cord. Compression of cord was found in 2% of a series of 497 older patients admitted with acute vertebral fractures.

History of vertebral augmentation technique
In 1984 Galibert and Deramond performed the first vertebral augmentation on a woman who had had severe neck pain for several years. The procedure was repeated in six more people before a first report was published in 1987. The technique was later used to treat compression fractures in France and was introduced in the US at the University of Virginia in the 1990s. Kyphoplasty was later introduced by Kyphon.®.

Clinical Features of Compression Fracture:
Compression fractures manifest as localized back pain but 20% of the patients are generally asymptomatic; the compression fracture is found incidentally on radiological study. Patients describe pain as deep achy, worse with activity, especially sitting, standing or bending. The spine is tender to palpation and deep percussion, which is a hallmark of acute fracture.
In a series of 210 patients with acute back pain associated with vertebral fracture, two patterns were identified. In the first group, there is severe collapse of the vertebra and the pain lasts for few weeks. In the second group, the collapse is slow and progressive and happens in multiple episodes over many months. The pain in second group is not crippling and is stretched over months.[5] The clinical effects of compression fracture vary from minimal loss of height and pseudoarthrosis producing persistent instability, to multiple fractures causing loss of height and kyphosis. Neurological compression is rare. Cases of complete paralysis have been described.
The pain of acute compression fracture is severe for the first 7 to 10 days after the fracture. In a study assessing pain in 56 hospitalized women with acute fracture, pain had decreased by only 22% at day 7, and by only 33% at day 14. Analgesic consumption fell by 16% at day 5 and by 33% at day 14. Gennari et al found that spontaneous pain, measured with a visual analogue scale, was only significantly decreased by day 15, but at day 30 it had decreased by approximately 40%. In the same study pressure-provoked pain only began to decrease by day

20 and had decreased by 50% at day 30.[6] There are few long-term studies of the pain of acute vertebral compression fracture. Generally speaking, pain duration is variable, but pain remains for about 2-3 weeks. Pain relief happens after 4-6 weeks.

Management of Compression Fracture

The immediate aims of treating fracture are pain relief and ambulation. Pain relief can usually be accomplished with oral analgesics, such as non-steroidal anti-inflammatory drugs. Short term use of oral narcotics may be needed in some patients. Bed rest is not encouraged. All efforts are geared to keep the patient mobile including use of a cane, walker, braces, or trigger point injection. If a patient is not ambulatory, aggressive intervention is needed. The long term goal is prevention of a compression fracture by slowing down or arresting osteoporosis with medications.

Rationale for Vertebral Augmentation

Conservatively treated fractures show pain improvement over 12 months for 60% of patients; 40% still have pain 1 year after fracture. Conservative treatment of painful vertebral compression fractures usually consists of bed rest, bracing, and narcotic analgesia. This conservative management is not risk free and cause many well documented complications like development of pressure sore, cognitive impairment, side effects from the use of different medications all of which complicate recovery of the patients. Bone density decreases approximately 2% per week, a serious concern in patients with osteoporosis, who are unlikely to regain the lost bone mass. The most dramatic bone loss occurs in the first 12 weeks of immobilization. Muscle strength decreases 1%-3% per day or 10%-15% per week. Almost half of normal strength is lost within 3-5 weeks of immobilization, and the rate of recovery from disuse weakness is slower than the rate of loss. Patients who undergo an augmentation therapy have immediate and considerable improvement in pain and mobility after treatment. This prompt return to function is the basis for all augmentation therapy as waiting for the pain to ease up for patient to be functional again would take anywhere 2-12 weeks[7] which elderly people can ill afford.

Given the currently available scientific data, the American Society of Interventional and Therapeutic Neuroradiology, the Society of Interventional Radiology, the American Association of Neurological Surgeons/Congress of Neurological Surgeons, and the American Society of Spine Radiology in a position paper in 2007 endorsed early intervention in favor of augmentation therapy for compression fracture. [8]

Patient Selection

Proper patient selection is a key for good outcome. Four characteristics are considered

1. Other potential sources of back pain in patients with a compression fracture
2. Anatomy of the fractured vertebral body
3. Age of the compression fracture
4. Early recognition of failure of medical therapy in patient with compression fracture

Information is sought to differentiate the pain of compression fracture from other etiologies such as disc herniation, spinal cord or nerve root compression, discogenic back pain, facet

arthropathy, or spinal stenosis. Best results are obtained when a recent compression fracture is treated. History helps to determine the site of pain and timing of fracture. Tenderness on physical examination should correlate with level of fracture on radiological studies. The timing of treatment has been liberalized as clinical experience with the technique has increased. In early published and unpublished treatment series, vertebroplasty was performed after conventional medical therapy (analgesics and bed rest) failed. Recent series have advocated treatment as early as a few weeks or even within days, after the occurrence of a painful compression fracture if pain is so severe as to require parenteral narcotics and hospitalization. Late treatment (after 6 months) is less likely to be successful in relieving pain; however, investigators have anecdotally reported symptomatic improvement with percutaneous vertebroplasty performed years after the initial injury.

History and physical examination is not always clear cut, so radiological workup is must when evaluating a suspected compression fracture. Pain is not well localized. The low thoracic and lumbar spine areas are imaged in a radiological study. Plain x ray can miss compression fracture in one third of cases. CT of lumbar spine or MRI of the spine is preferred when injection therapy is being considered.

The purpose of radiological studies is threefold. Primary purpose is to evaluate anatomy of the fractured vertebra but studies also help rule out infection and date the fracture. A vertebral body may be injected after ruling out a crushed body, cracked posterior body wall, or severe retroplusion of the posterior body wall. CT of the spine is good for evaluation of vertebral body anatomy. A thin section (3 mm or less) with sagittal reconstructions is the best modality for determining whether a fracture line extends through the posterior wall of the vertebral body. A CT scan cannot reveal the age of a fracture unless a recent study is available for comparison. If MRI is contraindicated, then CT scan with positive bone scan is a useful in evaluating the state of fracture. Persistent uptake by the spine is a reflection of a non-healed state. MRI with STIR images is best at evaluating age of the fracture. It's based on the principle of persistent edema in a non-healed fracture. Acute and subacute fractures which are less than 30 days old are hypointense in signal on T1-weighted images and hyperintense on T2- weighted and short T1 inversion recovery (STIR) sequences. Acute and subacute fractures may become isointense to normal vertebrae after administration of gadolinium contrast material. Approximately 1 month after fracture, the majority of osteoporotic VCFs become isointense to normal bone marrow on T1- and T2-weighted sequences. Fully healed compression fractures demonstrate a return of normal marrow signal or, alternatively, may appear hypointense on T1- and T2-weighted sequences when there is significant sclerosis. If the fracture is unstable, neurosurgical intervention is required. Any leg weakness or sign of cord compression must be vigorously and emergently evaluated with MRI and neurosurgical consultation.

<u>General Consideration for Augmentation Therapy</u>

1. Evaluate coagulation profile of the patient.
2. Vertebral augmentation pushes marrow into the blood vessels. Also cement can leak into blood vessels. All this can embarrass the pulmonary circulation and patient with pulmonary

hypertension should be monitored carefully during the procedure.
3. Active infection is contraindication for this procedure. Loss of more than 70% height makes the procedure technically challenging. Radiculopathy is not a contraindication to PV; however, the procedure may not improve these symptoms and may in some cases worsen them.
4. Spinal stenosis at the level is relative contraindication. Fracture line that open into spinal canal increases the risk of cement leaking into the canal.
Young patients with no underlying risk factor normally do not need the procedure.

Vertebroplasty and Kyphoplasty technique:
The body of the vertebra is injected with bone cement to strengthen the bone. It is important to have a good fluoroscopic view.
a. Kyphoplasty
The vertebral body is accessed using a 7-11 gauge cannula. Once the proper position of the cannula is confirmed by fluoroscopic imaging, a balloon is inserted via the cannula and is carefully inflated to restore the height of the collapsed vertebra. The balloon is then removed and cement is injected through the cannula to fill up the body of the vertebra.
b. Vertebroplasty
This procedure is similar to kyphoplasty except that the kit has smaller instruments (13 g cannula) and there is no attempt to restore vertebral height using a balloon.
The primary benefit of augmentation technique is immediate and lasting pain relief. Recovery time is short. When randomized controlled trials on vertebroplasty and kyphoplasty were reviewed, 8 studies of 241 were in patients age > 60 years. The reviewers concluded that vertebroplasty is no better than placebo, a conflict with the available level IIb evidence of a positive short-term effect of cement augmentation compared to standard medical therapy with regard to quality of life, function and pain relief. Kyphoplasty is not superior to vertebroplasty to relieve pain (evidence level IIb). Kyphoplasty is not cost-effective (evidence level IIb). Vertebroplasty has only short-term cost-effectiveness (evidence level IV). Vertebroplasty and kyphoplasty cannot be recommended as standard treatment for osteoporotic compression fracture.[9]
Currently a multicenter randomized sham control trial of vertebroplasty for painful acute osteoporotic vertebral fractures is underway. It will enroll 180 patients who have an acute fracture less than 6 weeks old.

Vertebroplasty versus kyphoplasty
No randomized controlled trials have been performed to compare vertebroplasty directly with kyphoplasty. When efficacy and safety of balloon kyphoplasty and vertebroplasty were compared for the management of patients with vertebral compression fractures, of 4,861 fractures treated by vertebroplasty and 1,070 fractures treated by kyphoplasty, reduction of pain was similar. Follow-up for vertebroplasty was for 5 yr; for kyphoplasty for 2 yr. The pooled case series also demonstrated improved vertebral height and kyphotic angle with vertebroplasty. The rates of pulmonary embolism, neurologic complications, and perioperative mortality were low with both procedures, although poorly reported across studies. [10]
Another evaluation of the efficacy of vertebroplasty and kyphoplasty with respect to pain relief,

restoration of mobility and vertebral body height, considered 4,456 vertebroplasty procedures and 1,624 kyphoplasty procedures. Pain relief was experienced by 87% of patients with vertebroplasty and 92% with kyphoplasty. Vertebral height was restored with kyphoplasty and, for a subset of patients with mobile fractures, with vertebroplasty. Cement leaks occurred for 41% and 9% of treated vertebrae for vertebroplasty and kyphoplasty, respectively. New fractures of adjacent vertebrae occurred after both procedures at rates that were higher than those in the general osteoporotic population but approximately equivalent to the general osteoporotic population that had had a previous vertebral fracture. In pooled data, the mean kyphotic angle restoration was 6.6 degrees for both procedures; however, in 34% of kyphoplasty patients and 39% of vertebroplasty patients' vertebral height or kyphotic deformity did not improve.[11]

Other investigators [12] found no difference in pain relief, function, incidence of cement leak, and risk of subsequent fracture after each procedure.
In summary, direct comparison between vertebroplasty and kyphoplasty is not possible because of a lack of good data comparing the two approaches. Indirect comparisons suggest that vertebroplasty and kyphoplasty reduce pain comparably. Both procedures appear to improve patient function in most series. Vertebral height restoration and reduction of kyphotic deformity are also similar for both procedures.

Complications

The most frequent complications after vertebral augmentation therapy have been described.[13] Combining direct posterior and intravascular migration, the published rate of cement leakage varies widely after percutaneous vertebroplasty from 0% to 76%. If PMMA leaks into the spinal canal or neural foramen, partial or complete paraplegia is possible, and decompression is required emergently. Leakage of cement into the paraspinal vasculature has been estimated in 24% to 39% of vertebroplasty cases. A striking 26% incidence of pulmonary cement embolism has been found with vertebroplasty. Small cement pieces, only revealed by CT scan were similar in size to the ones found after large joint replacement and did not generate lung reaction. Complication rates were compared in 42 vertebroplasty studies (4266 patients, 6506 fractures) and 10 kyphoplasty studies (957 patients, 1600 fractures).[14] The prevalence of cement leak was 19.7% after vertebroplasty and 7.0% after kyphoplasty. Cement leaks were symptomatic in 1.6% and 0.3% of patients respectively. New compression fractures were 17.9% and 14% respectively. Pulmonary embolism was 0.9% and 0.4% respectively, myocardial infarction 0.05% and 0.5% respectively. Less common complications included hematoma, rib fracture, infection, change in blood pressure or heart rate, pneumonia, and hypoxia.

After a one-year follow-up, a high degree of heterogeneity made it impossible to state that cement augmentation is as safe as conservative treatment with respect to occurrence of new fractures. The combined odds ratio of vertebroplasty and kyphoplasty versus conservative treatment was 0.96, hinting at the possibility that a little difference may exist.[15]

For every 1,000 patients treated with kyphoplasty each year, 1.7 patients could experience a

pulmonary embolism, 1.6 patients would experience spinal cord compression, 1.7 patients could experience radiculopathy, and 1.3 could die within the perioperative period (30 days).[10]

Is it possible that the infiltration of only the posterior elements could control a patient's pain?
Two recent studies raised the possibility that part of the pain in compression fracture could be due to facet subluxation and changes in loads over the facet joints/capsule as a consequence of vertebral collapse. In one report, 4 cases of compression fracture, pain was relieved by medial branch nerve blocks.[16] Posterior elements of the spine may be involved in pain generation and a diagnostic block before doing a vertebral augmentation injection may be helpful. The transpedicular approach for injecting cement often inadvertently causes neurotomy of medial branches and may even be the prime reason for pain relief.

New Methods
Stentoplasty. If a vertebral body shows a high degree of collapse and kyphotic deformity or some posterior wall involvement, stentoplasty, an evolution of kyphoplasty, restores height, and stabilizes the vertebral body with cement. Stentoplasty is a minimally invasive *during which two balloon mounted stents are placed through the pedicles of the fractured vertebral body.* The balloons are then deployed by an inflation system under fluoroscopic guidance until vertebral height is restored. The balloons are retrieved and the stent and the surrounding bone is filled with bone cement.

Skphoplasty. Another method for percutaneous cement augmentation of the spine is skyphoplasty with a device known as the Sky bone expander (Disc Orthopaedic Technology/Disc-O-Tech, Monroe Township, New Jersey). The diameter of the Skyphoplasty device is slightly larger than that of the kyphoplasty device. A stiff plastic tube is deployed through the cannula, placed inside the vertebral body and compressed in an accordion fashion into a popcorn like crenulated configuration, to create a cavity. The device is then removed and the cavity is filled with cement using a device similar to that employed in kyphoplasty. It is capable of creating higher pressures than those created by the kyphoplasty system. The plastic configuration used in skyphoplasty is less dependent on the character of bone and expands in a predictable fashion.
Experience with skyphoplasty is limited. When skyphoplasty was performed at 12 vertebral levels in 9 patients, pain from vertebral compression fractures was alleviated safely.[17] In another series of 40 single-level Sky bone expander skyphoplasties in 40 patients, the procedure was a viable alternative to balloon kyphoplasty. There were 3 cases of cement extravasation and 1 case in which the Sky bone expander could not be withdrawn from the vertebral body and had to be left in situ.

Lordoplasty. During lordoplasty adjacent vertebrae are reinforced and the fractured vertebra is reduced and reinforced.

Conclusion
There appears to be a case for vertebral augmentation therapy in selected patients with

vertebral compression fracture form cancer, multiple myeloma, and osteoporotic fractures with bone marrow edema. There is no consensus about the best candidates for the procedure or the best time to perform it. Currently medical management that produces pain relief within 3 weeks is the best treatment, with nearly universal good outcome at 1 year. Bone augmentation therapy should be delayed for at least a month after medical management has been shown to fail.[19]

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