Osteoporotic Vertebral Compression Fractures

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Disclosures:

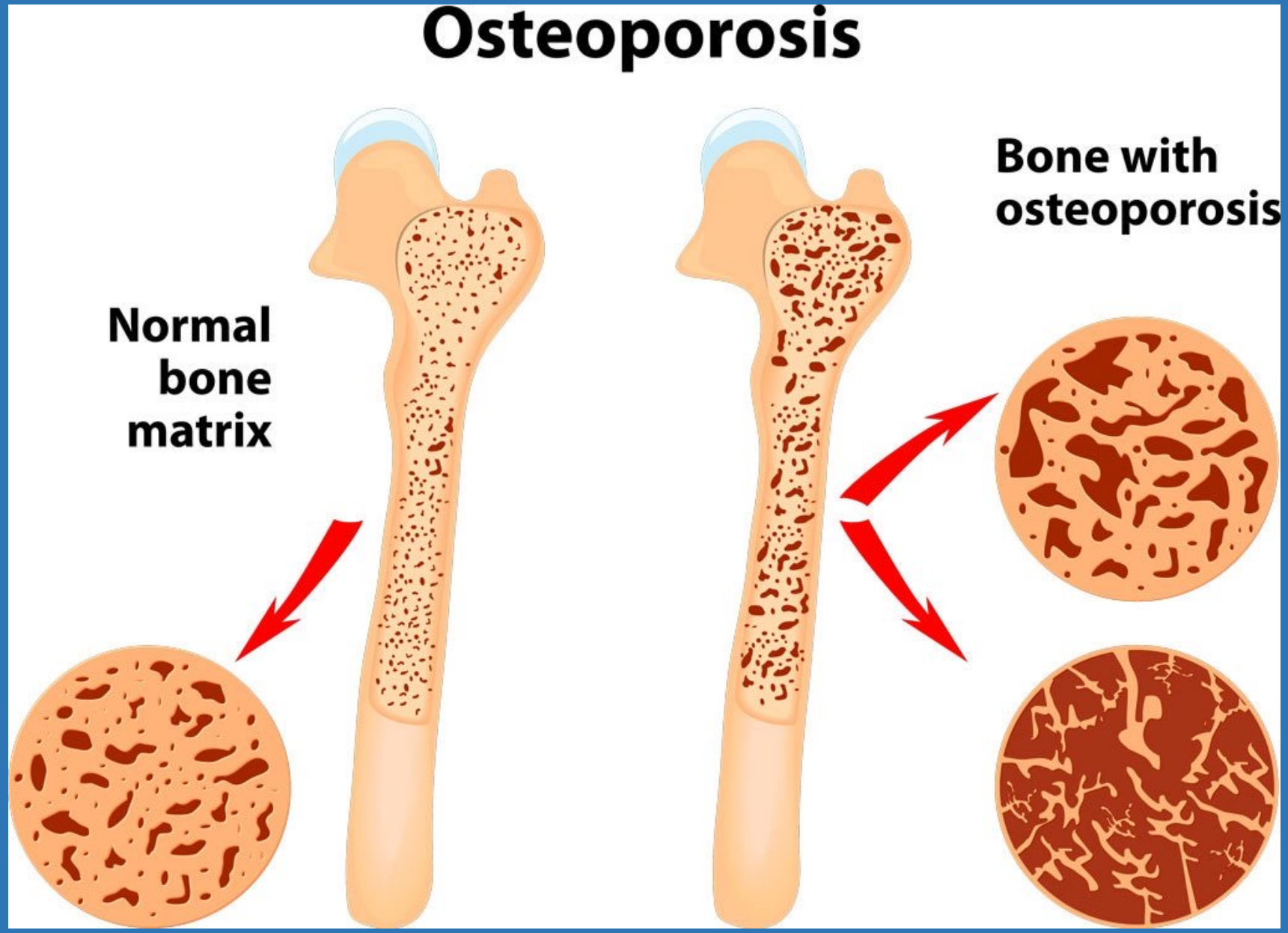
- None

About me:

- From Ada, OK
- Graduated from East Central University
- Medical School at OSU-COM
- Residency in orthopedic surgery at St. Anthony Hospital - OKC, OK
- Fellowship in spine surgery at Texas Back Institute

Osteoporosis:

- Refers to excessive bone loss as reflected by the deterioration of bone mass and microarchitecture which results in compromised bone strength.
- Generally, peak bone mass is reached in the third decade of life. Subsequently, there is a decline in bone mass, which is accelerated during menopause in women.
- The development of osteoporosis is asymptomatic, but it’s ultimate consequences (fragility fractures) pose tremendous medical and economical challenges to patients and society.

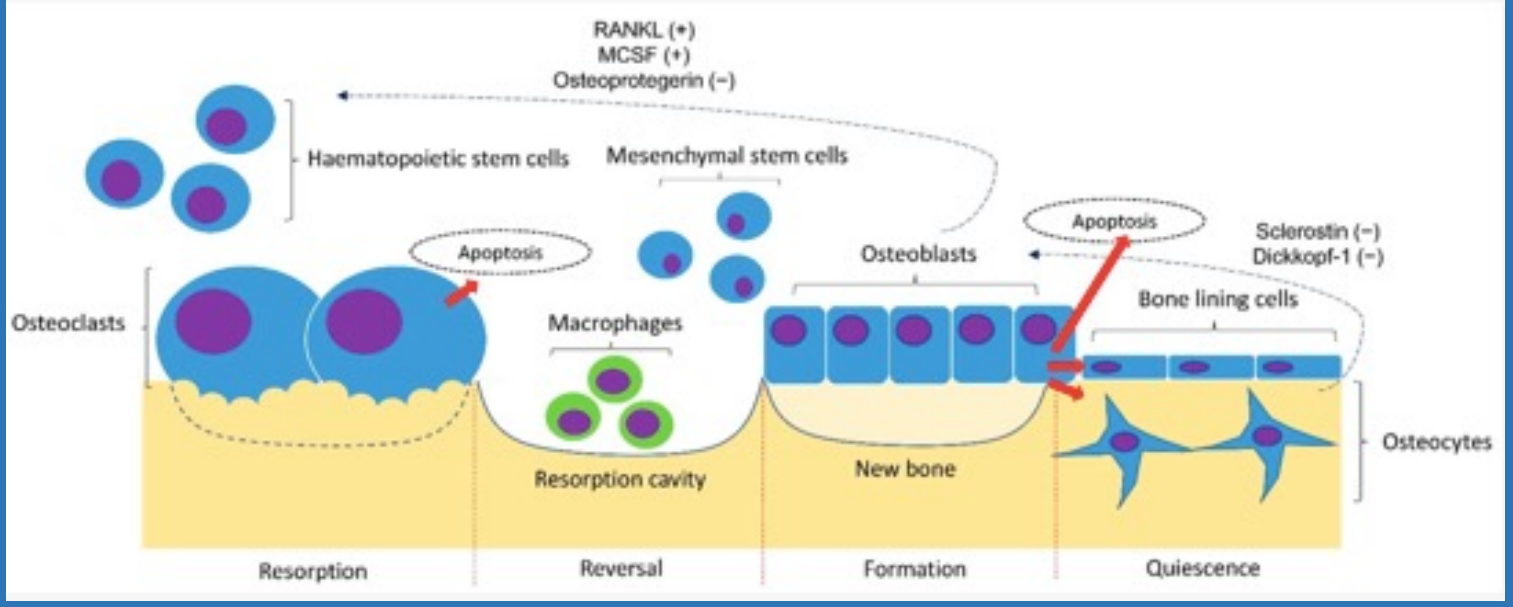

Osteoporosis
Normal bone matrix
Bone with osteoporosis

Pathophysiology:

- Traditionally osteoporosis was thought to be caused by estrogen deficiency in postmenopausal women and secondary hyperparathyroidism due to menopause or vitamin D deficiency. In reality, osteoporosis is a complex multifactorial disease caused by a complex interplay of genetic, intrinsic, exogenous, and lifestyle factors.

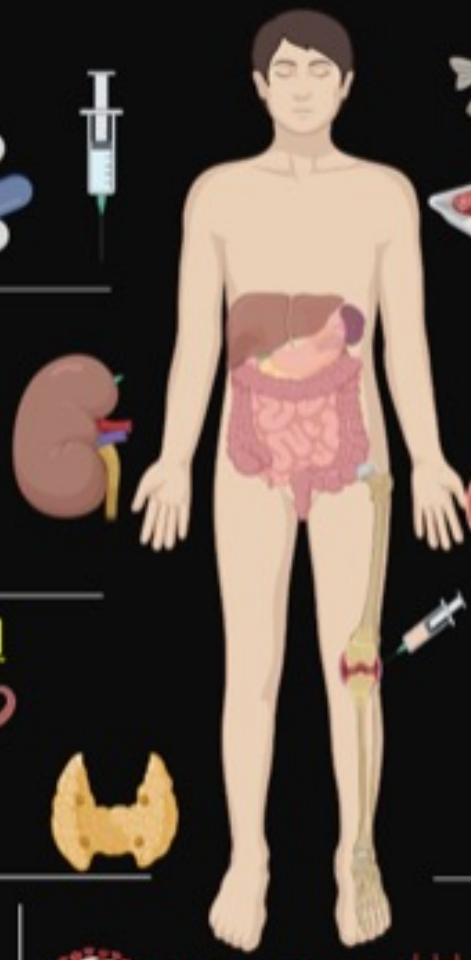
- Osteoblasts, osteoclasts, and osteocytes are the 3 main cells involves in bone formation and remodeling.
- In short, osteoclasts migrate to a damaged site and perform resorption before undergoing apoptosis then serving in osteogenesis. Macrophages further help with resorption in the reversal phase. Next osteoblasts are stimulated and migrate to perform bone formation. Some osteoblasts will be embedded in the bone and become osteocytes. Osteocytes act as mechanoreceptors and play regulatory roles in bone remodeling through signaling proteins.

Figure 1. Bone remodelling cycle. The bone remodelling cycle is governed by osteoclasts, osteoblasts and osteocytes derived from the respective stem cell lineage. The differentiation of osteoclasts is stimulated by the receptor activator of nuclear factor kappa-B ligand (RANKL) and macrophage colony-stimulating factor (MSCF) and inhibited by osteoprotegerin (OPG) synthesised by osteoblasts and osteocytes. The osteogenesis of osteoblasts is inhibited by sclerostin and Dickkopf-1 synthesised by osteoblasts. Notes: +, promoting factor; −, inhibiting factor.



- The remodeling process is coordinated delicately to maintain bone mineral homeostasis and strength.
  - Osteoclasts are stimulated by RANKL and MSCF. The are inhibited by OPG.
  - Osteocytes synthesize sclerostin and Dickkopf-1 that inhibits Wnt signaling pathway and osteoblasts (formation).
  - Bone loss occurs when the rate of formation is lower than the rate of resorption.

Secondary Osteoporosis



Drug-induced

- Steroids
- PPIs
- Anti-epileptics
- Anti-coagulants

Nutritional

- Bad dietary habits
- Starvation
- Anorexia/Bulemia
- Exessive Alcohol

Renal

- Hyperparath. bone dis.
- Adynamic bone dis.
- Osteomalacia
- Mixed ROD

Gastro-Intestinal

- Malabsorption
- Liver Diseases
- IBD
- IBS

Endocrinological

- DM
- Hypogonadism
- Thyroid/PTH disorders

Immunological

- Inflamatory arthritis
- SLE
- Multiple Sclerosis

Others

- Smoking
- Disuse
- Genetic causes

Infections

HIV HCV HBV HZV
COVID-19 TB Osteomyelitis

Hemato-oncological

- Hemolytic anemias
- Malignancies

Diagnosis:

- The diagnosis of osteoporosis is most commonly established by measuring bone bone mineral density (BMD).
- The current standard is for measuring BMD is dual energy x-ray absorptiometry (DEXA). It is the actual expression of the bone in absolute terms of grams of mineral (primarily Ca++) per sq cm of the scanned bone.
- BMD measurements of the hip and spine are used to establish or confirm the diagnosis of osteoporosis or to predict future fracture risk and monitor patients.
- T-scores represent the standard deviation from the peak value in young adults (20-29 year olds).

Diagnosis:

- A T score of -2.5 or less is considered osteoporosis, -1.5 to -2.5 denotes osteopenia, -1 or greater is normal.
- For premenopausal women, men less than 50 years of age, and children, the BMD diagnostic classification as defined by the WHO should not be applied. The International Society for Clinical Densitometry (ISCD) recommends using ethnic- or race-adjusted Z-scores: Z-scores of −2.0 or lower are defined as “low bone mineral density for chronological age”
- The United States Preventive Services Task Force (USPSTF) recommends testing of all women >65 and younger women whose fracture risk is equal to or greater than that of a 65 year old white female.

Table 5

Indications for measuring BMD (13)

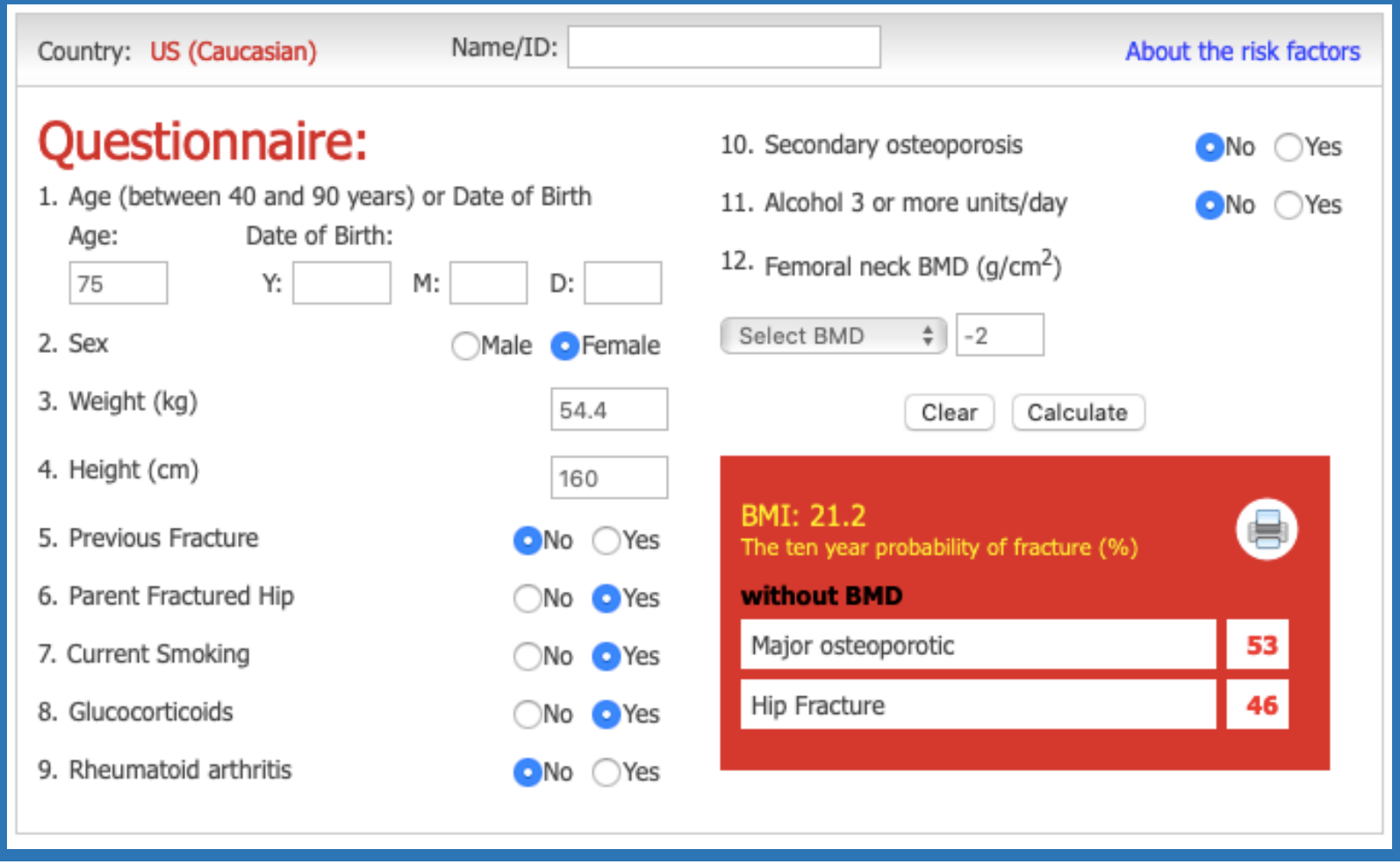
Older adults (age >50 yr)	**Younger adults (age <50 yr)**
Age ≥65 yr (both women and men)	Fragility fracture
Clinical risk factors for fracture (menopausal women, men aged 50–64 yr)	Hypogonadism or premature menopause (age<45 yr)
	Malabsorption syndrome
- Fragility fracture after the age of 40 yr	Prolonged use of glucocorticoids*
- Prolonged use of glucocorticoids	Use of other high-risk medications**
- Use of other high-risk medications	Primary hyperparathyroidism
- Vertebral fracture or osteopenia identified on radiography	Other disorders strongly associated with rapid bone loss and/or Fracture
- Other disorders strongly associated with osteoporosis	
- Current smoking	
- High alcohol intake	
- Vertebral fracture or osteopenia identified on radiography	
- Low body weight (<60 kg) or major weight loss (>10% of body weight at the age of 25 yr)	
- Rheumatoid arthritis	
- Parental hip fracture	

Problems with Diagnosing Osteoporosis

Of the 2259 postmenopausal women who reported fractures at 1 year*

Fracture Risk Assessment Tool (FRAX):

- The most important health consequence of osteoporosis is fractures. Recently, algorithms have been developed to predict the risk of fracture in individuals that incorporate significant predictors of fracture risk in addition to BMD.
- Estimating the 10-year risk of a major osteoporotic fracture (i.e., fracture of the hip, vertebra (clinical), forearm, or proximal humerus) is possible with algorithms that integrate the weight of clinical risk fractures for fracture risk with or without information on the BMD have been developed.
- They can be used to compute the 10-year probability of hip fracture or a major osteoporotic fracture (clinical spine, hip, forearm, or humerus).



For USA use only

Consider FDA-approved medical therapies in postmenopausal women and men aged 50 years and older, based on the following:

- A hip or vertebral (clinical or morphometric) fracture
- T-score ≤ -2.5 at the femoral neck or spine after appropriate evaluation to exclude secondary causes
- Low bone mass (T-score between -1.0 and -2.5 at the femoral neck or spine) and a 10-year probability of a hip fracture ≥ 3% or a 10-year probability of a major osteoporosis-related fracture ≥ 20% based on the US-adapted WHO algorithm
- Clinicians judgment and/or patient preferences may indicate treatment for people with 10-year fracture probabilities above or below these levels

Table 6
2020 AACE Diagnosis of Osteoporosis in Postmenopausal Women

1. T-score –2.5 or below in the lumbar spine, femoral neck, total proximal femur, or 1/3 radius
2. Low-trauma spine or hip fracture (*regardless of bone mineral density*)
3. T-score between –1.0 and –2.5 **and** a fragility fracture of proximal humerus, pelvis, or distal forearm
4. T-score between –1.0 and –2.5 **and** high FRAX® (or if available, TBS-adjusted FRAX®) fracture probability based on country-specific thresholds

Abbreviations: AACE = American Association of Clinical Endocrinologists; FRAX® = fracture risk assessment tool; TBS = trabecular bone score.

Osteoporosis Treatment:

- Refer to Bone Health Specialist if available.
- Check appropriate labs to rule out secondary causes:
  - Serum Ca++, phosphorous, magnesium, CBC, CMP, alk phos, urine calcium, serum albumin, vitamin D 25(OH), TSH, liver function test, testosterone, PTH.
- Address any modifiable factors:
  - Alcohol, exercise, medications…etc

Pharmaceutical Treatment:

- Antiresorptive:
  - Bisphosphonates
  - Denosumab
  - SERMS
  - Calcitonin
  - Increase bone mass by 2-5%, reduce fractures by 50%
- Anabolic:
  - Teriparatide
  - Abaloparatide
  - Romosozumab
  - Increse bone mass by 14-20%, reduce fractures by 75%

Fragility fractures:

- 2 million people/year sustain a osteoporosis related fracture with an estimated cost of $19B to medicare alone.
- Fragility fractures typically occur after a fall from standing, walking, or sitting.
- In the U.S. <20% of patients with a fragility fracture receive care for osteoporosis.
- Without treatment patients commonly sustain secondary fractures resulting in morbidity, disability, institutionalization, and increased mortality.

Vertebral Compression Fractures:

- An estimated 1.5 million vertebral compression fractures occur each year in the U.S.
- 25% of post menopausal females will experience a VCF in their lifetime. This incidence increases to 40% at age 80.
- These fractures rarely require hospitalization, but they have the potential to cause significant disability, morbidity, and often incapacitating pain.
- Multiple adjacent VCFs can lead to progressive kyphosis which can results in decreased appetite and pulmonary function.

Mortality following the diagnosis of a vertebral compression fracture in the Medicare population

Edmund Lau [1], Kevin Ong, Steven Kurtz, Jordana Schmier, Av Edidin

- Reviewed >97k patients with new dx of VCF. Patient's with a fracture were compared to a matched control group.
- Survival rates for patient's with a VCF were 53.9%, 30.9%, and 10.5% at 3, 5, and 7 years respectively, which were significantly lower than the matched groups.
- Mortality risks was greater for men than women.
- The difference in mortality rates between the two groups were greatest when the patients were younger at the time of fracture, this difference declined as the age at time of fracture increased.

Etiology of VCFs:

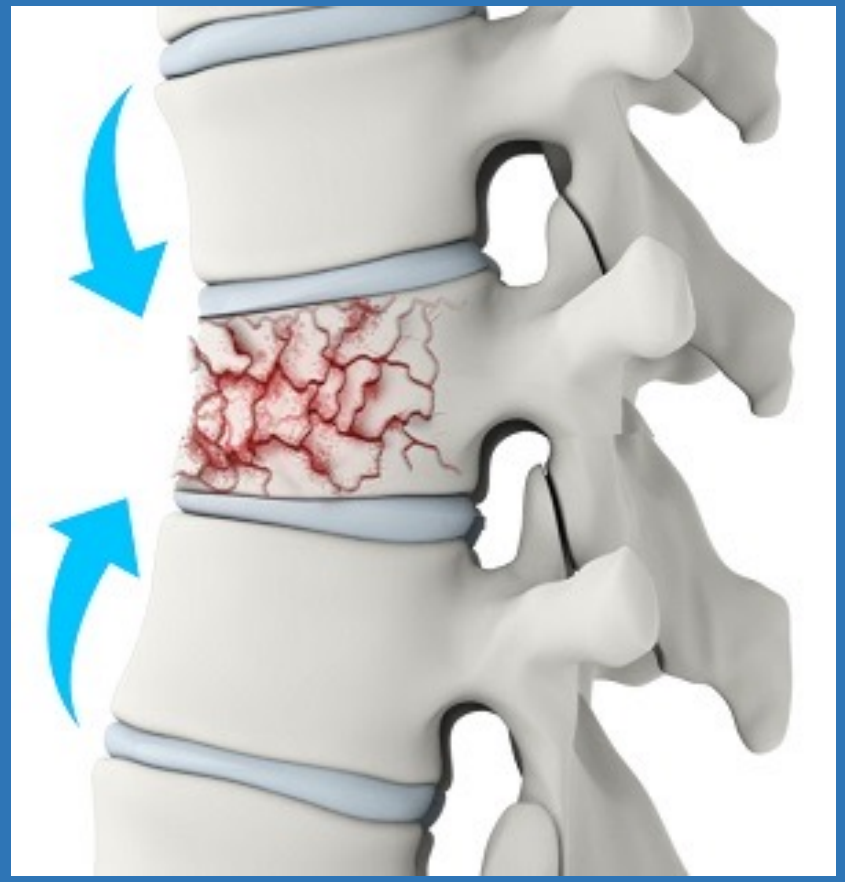
- The MC etiology is osteoporosis although they can also be caused by trauma, infection, and neoplasms.
- Postmenopausal females are at greatest risk due to hormonal changes resulting in decreased bone mineral density (BMD).
- It's estimated that 44M Americans have osteoporosis and an additional 34M have low bone mass.
- Studies have suggested that having 1 VCF increases the risk of future fractures by 5-fold and having 2 or more VCFs increases the risk of future fractures by 12-fold.

VCF Presentation:

- Compression fracture of the thoracolumbar spine involve a flexion compression mechanism of the anterior column of the spine.
- Pain is the most common symptoms.
- Neurologic deficits are very rare since the fracture doesn't require retropulsion of bone fragments into the spinal canal (burst fx).
- When patient's have very weak bone the injury mechanism can be quite low energy. Some examples include lifting a heavy object, vigorous cough or sneeze, turning in bed, or a fall.
- Many times the patient's do not remember a specific incidence.

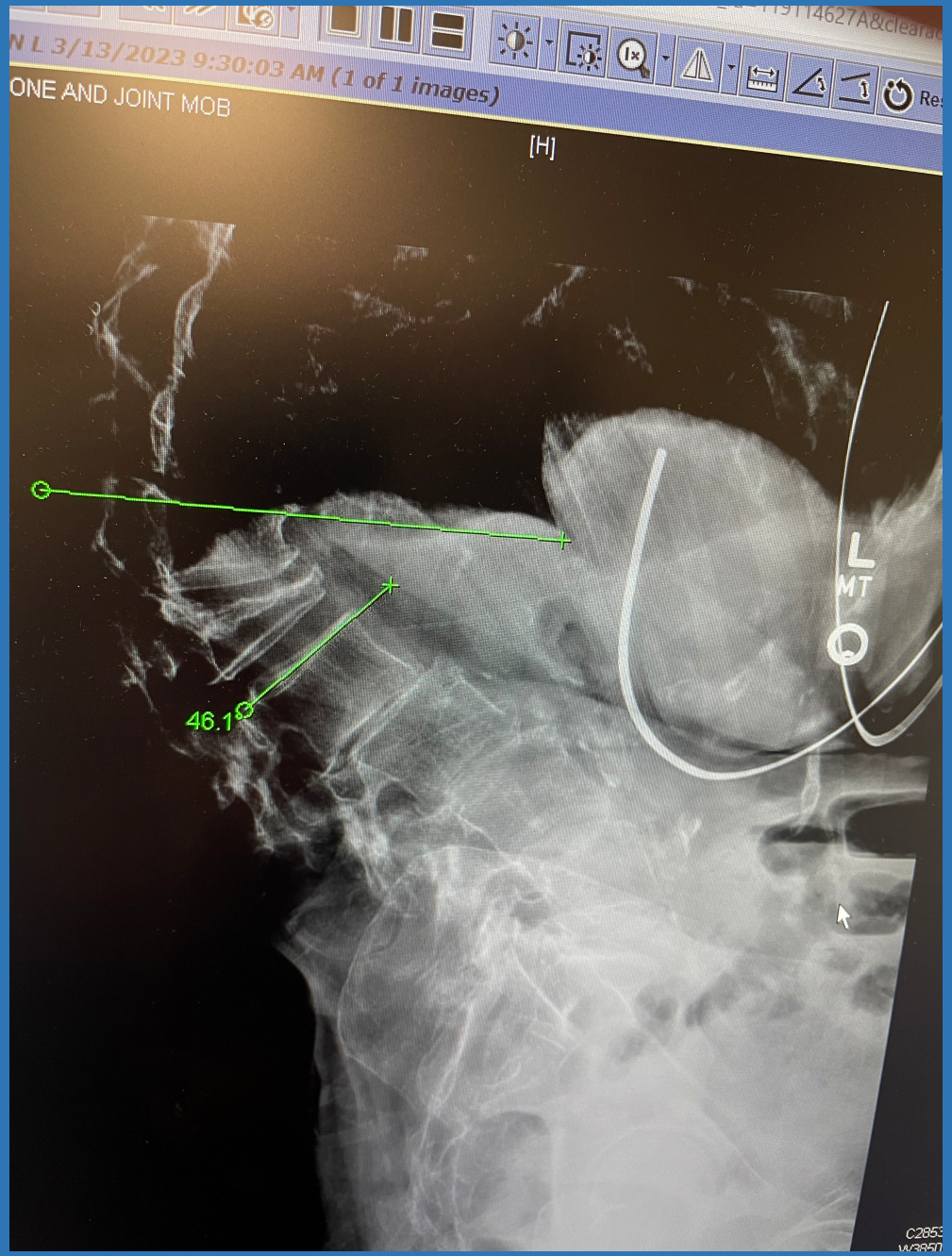
VCF Presentation:

- Approximately 30% of fractures occur while the patient is in bed.
- Patient's who sustain VCF's who don't have low bone density typically sustain a severe trauma such as fall from height or MVC.
- When patient's are younger than 55 years old malignancy should be considered as a possible cause of fracture.
- Vertebral compression fractures often may produce only low grade back pain. Over time if fractures continue to worsen the patient may experience progressive loss of stature leading to the paraspinal muscles having to work to maintain posture. This can result in fatigued muscles and chronic back pain that can persist even after the fracture has healed.



VCFs

- Untreated patient's with progressive collapse may develop increased thoracic kyphosis or loss of lumbar lordosis.
- In severe cases increased pressure on the thoracic and abdominal cavity can lead to early satiety and weight loss as well as decreased pulmonary function.
- Other known complications are: constipation, bowel obstruction, DVT, progressive muscle weakness, respiratory disturbances (pneumonia, atelectasis), and increased morbidity and mortality.
- When there is more than 50% collapse the adjacent levels have to support the additional load. This can lead to advanced degeneration of the spine and also make these levels more susceptible to VCFs.
- Almost 75% of VCFs occur around the thoracolumbar junction (T10-L2). This is a transition zone from the rigid thoracic spine to the mobile lumbar spine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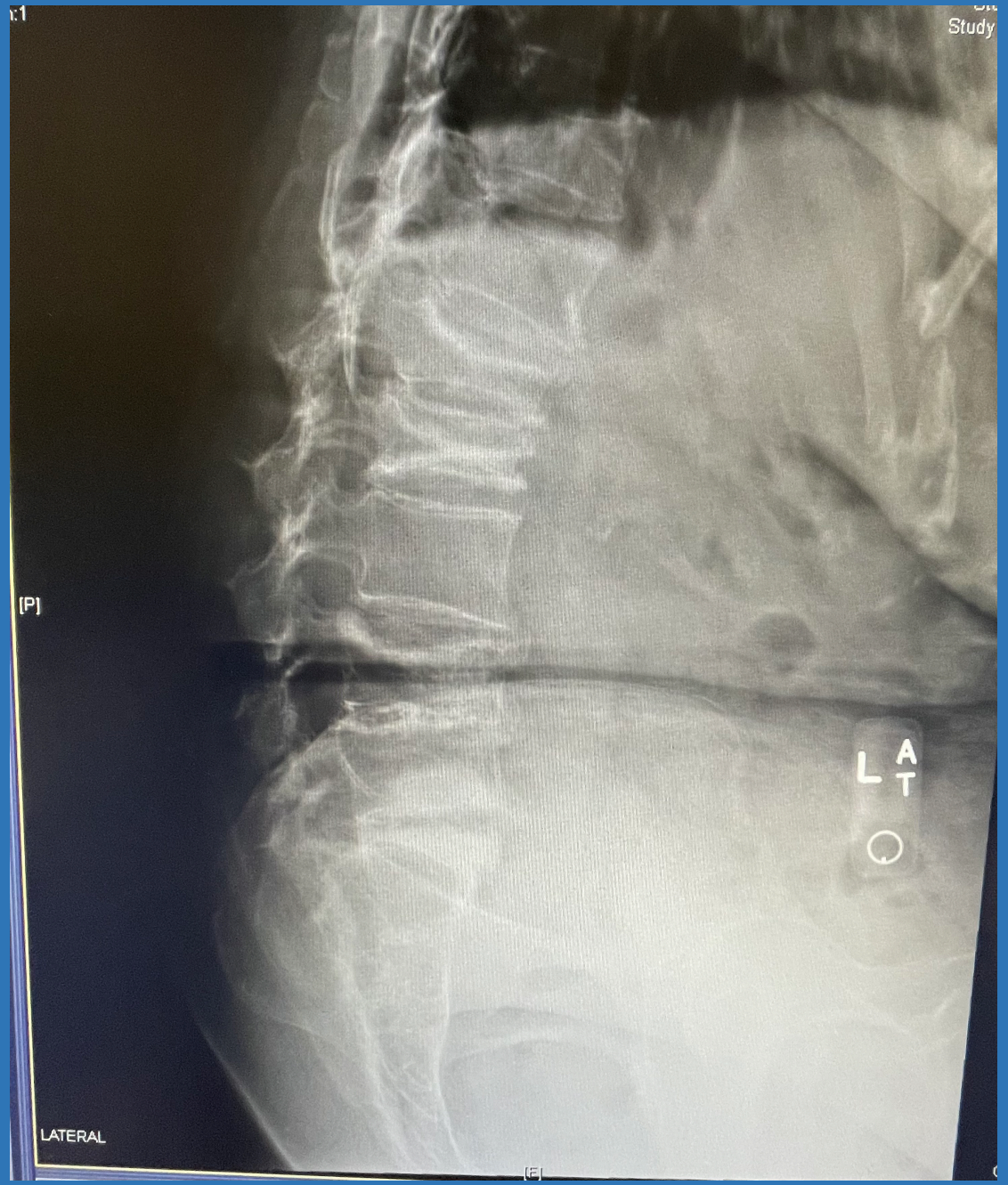
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Symptoms	Complications
Sudden onset of back pain	Continuous low-grade back pain
Intensity of pain increases during standing or walking	Thoracic kyphosis and lumbar lordosis
Intensity of pain decreases when lying on the back	Impaired pulmonary function
Pain increases during palpation over the affected level	Protuberant abdomen, and early satiety and weight loss
Decreased spinal mobility because of pain	Increased osteoporosis because of inactivity
	Deep vein thrombosis because of inactivity
	Decreased respiratory capacity because of kyphosis, which in turn leads to atelectasis pneumonia
	Low self-esteem and emotional and social problems

Risk Factors for VCFs

Modifiable	Nonmodifiable
Alcohol consumption	Advanced age
Tobacco use	Female sex
Osteoporosis	Caucasian race
Estrogen deficiency	Dementia
Early menopause	Susceptibility to falling
Bilateral salpingo-oophorectomy	History of fractures in adulthood
Premenopausal amenorrhea for more than one year	History of fractures in a first-degree relative
Frailty	
Impaired eyesight	
Insufficient physical activity	
Low body weight	
Dietary calcium deficiency	
Vitamin D deficiency	

Risk Factors:

- Many medications are known to decrease bone mineral density.
  - Steroids, heparin, warfarin, cyclosporine, loop diuretics, retinoids, chemotherapeutic drugs (methotrexate), antiseizure medications, Proton Pump Inhibitors, SSRIs.
- Interestingly, obesity is protective against fractures as it decreased the risk of bone loss. High stress on bone induces bone remodeling. Additionally, obesity leads to increased quantities of estrogen which promotes osteoblast activity.

Diagnosis of VCF:

- X-rays should be ordered if you are suspicious of VCF.
- Subtle fractures can be very difficult to identify on XR, especially in the upper thoracic spine. Many studies have reported the relatively high frequency that these are missed by radiologist.
- CT: Can be ordered initially if you are suspicious or if not identified on initial x-ray.
- MRI: Good for helping differentiate acute versus chronic fracture (many pts have multiple fxs). Some insurance companies require prior to intervention.


THE pain SOURCE
L3
L4
L5
MRI
T1-weighted
image
Vertebral Compression Fracture
- L3 Superior Endplate -


THE pain SOURCE
L3
L4
L5
MRI
T2-weighted
image
Vertebral Compression Fracture
- L3 Superior Endplate -

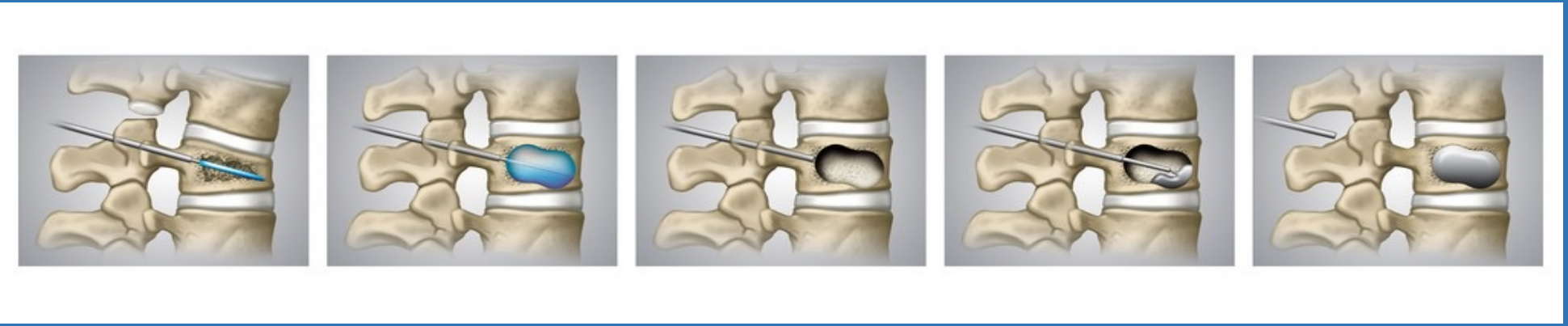

THE pain SOURCE
L3
L4
L5
MRI
STIR
image
Vertebral Compression Fracture
- L3 Superior Endplate -

Treatment:

- Osteoporosis treatment is arguably the most important action you can take to help prevent future fragility fractures.
- Non-surgical treatment consists of immobilization, bracing, pain relief, physical therapy (typically exacerbates symptoms acutely), activity modification, possible injections.
- Bracing: some studies show improved outcomes with bracing while other studies show no difference in outcome in braced versus non-braced cohorts.
- Many insurances require 6 weeks of documented conservative care before they will cover any type of intervention.

Vertebral augmentation

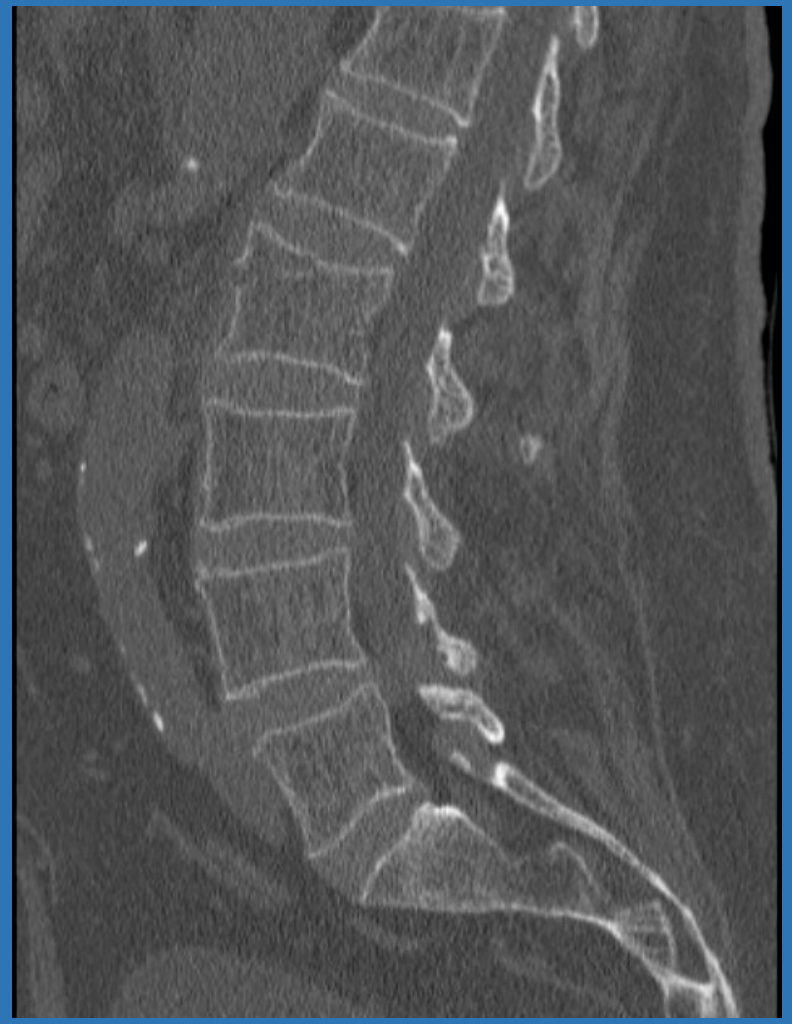
- Vertebroplasty – Injection of bone cement, no void creation
- Kyphoplasty – Void creation with a mechanical device, such as a balloon, followed by the injection of bone cement.



Balloon Kyphoplasty

- Over 1500 Studies confirm efficacy
- Superior Pain relief over conservative therapy
- Higher QOL scores
- Decrease mortality rate compared to non-treatment
- Decrease in Opiate intake
- Decrease in length of hospital stays
- Decrease in overall treatment cost

-79/M, fall from standing height.



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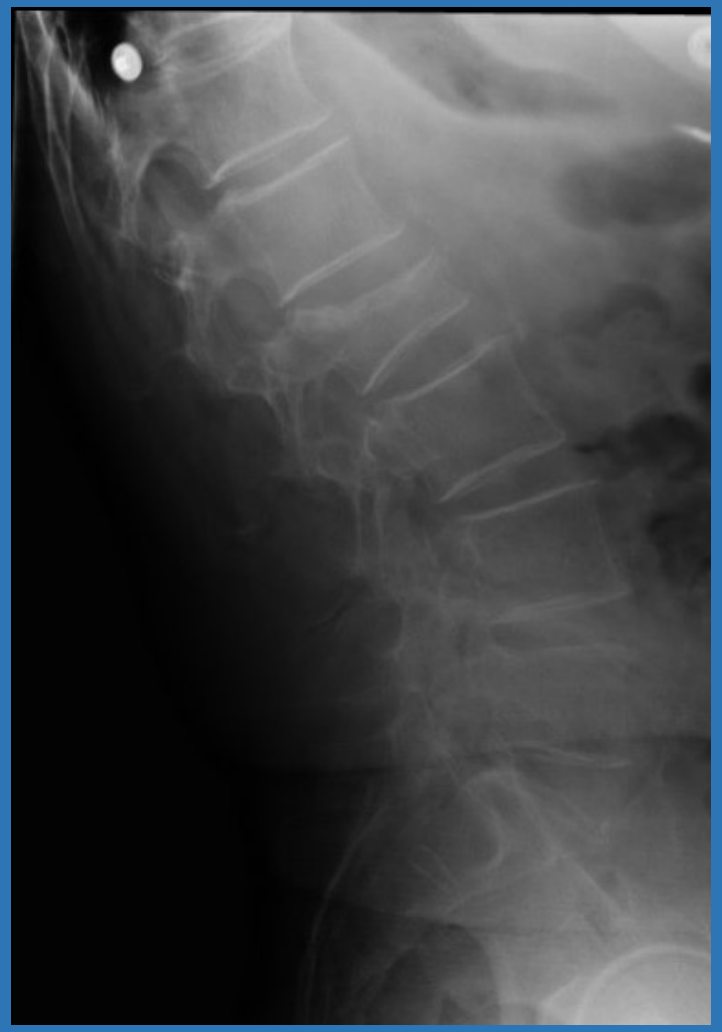

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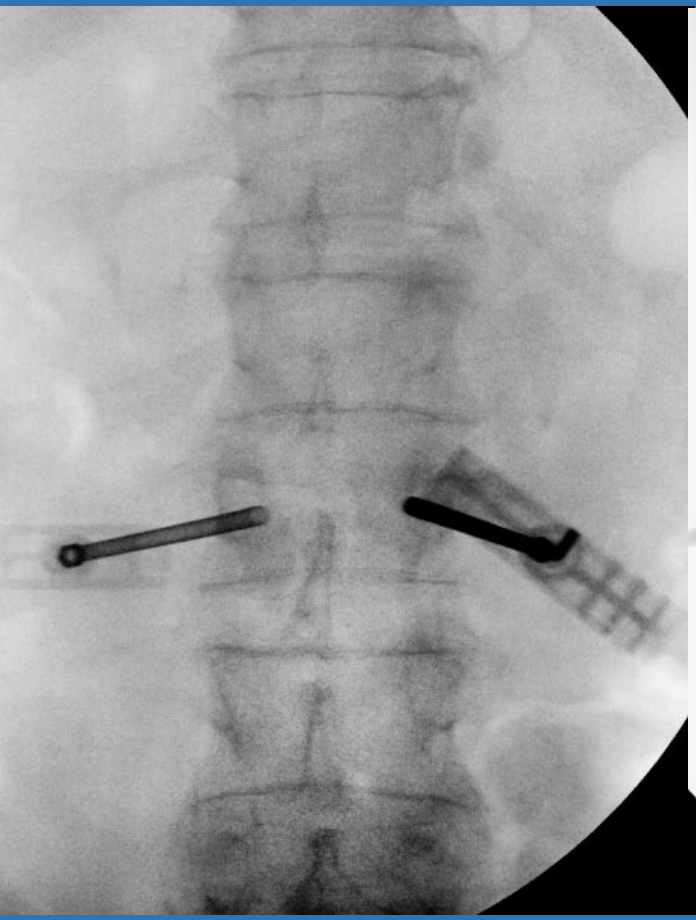
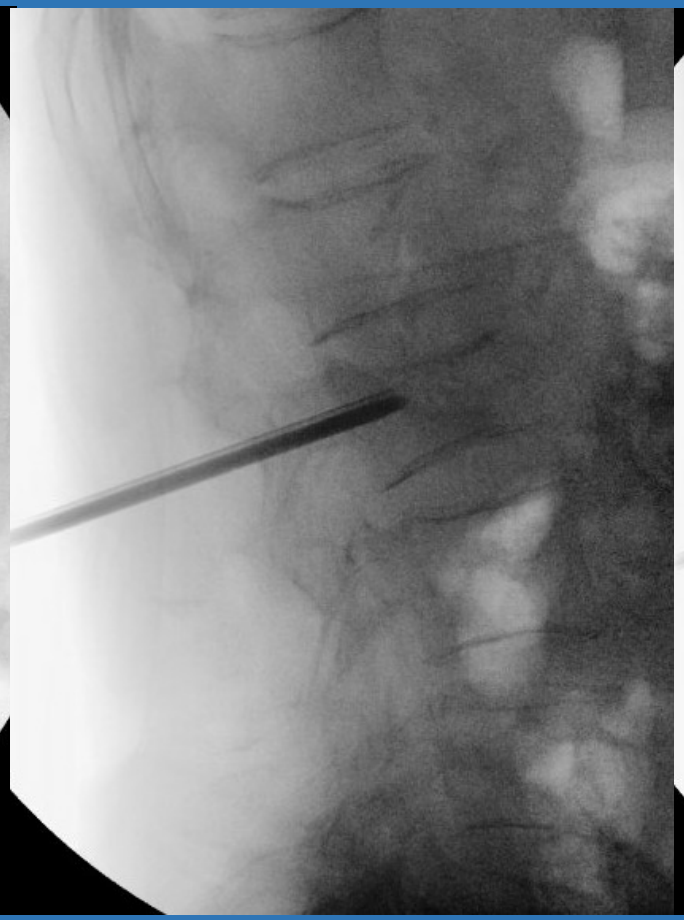
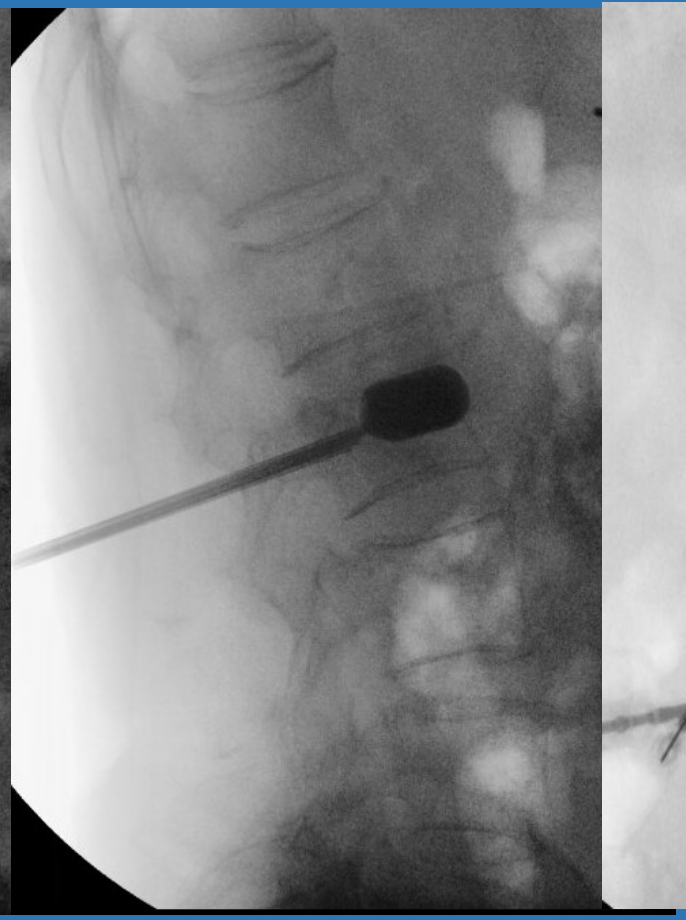
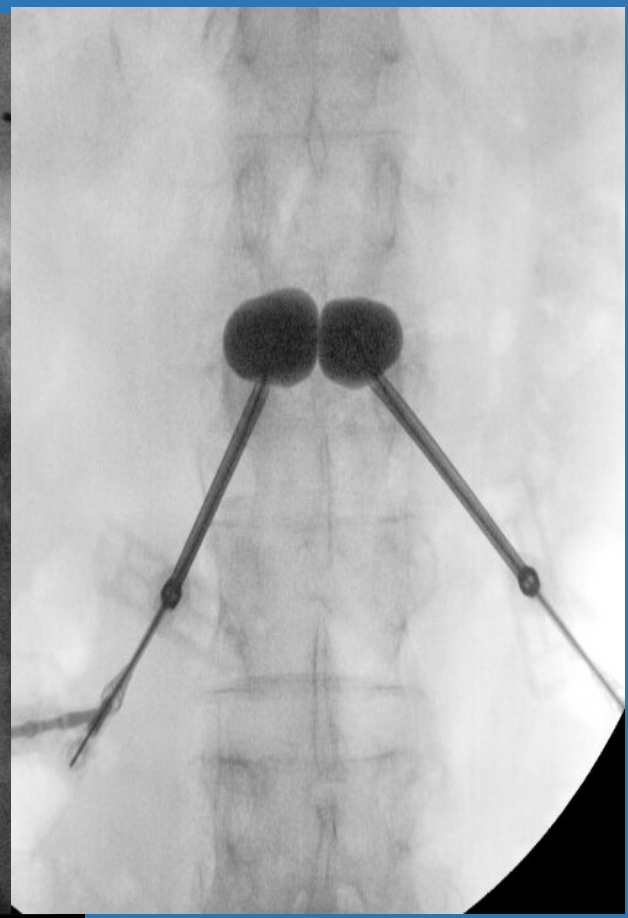
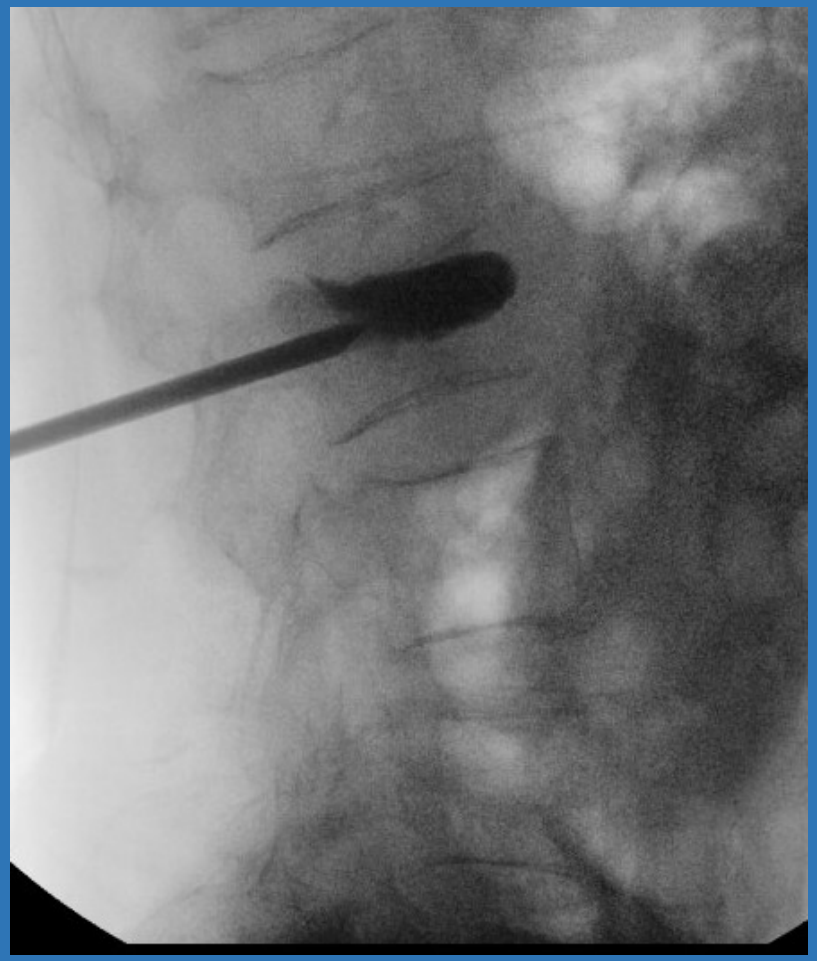
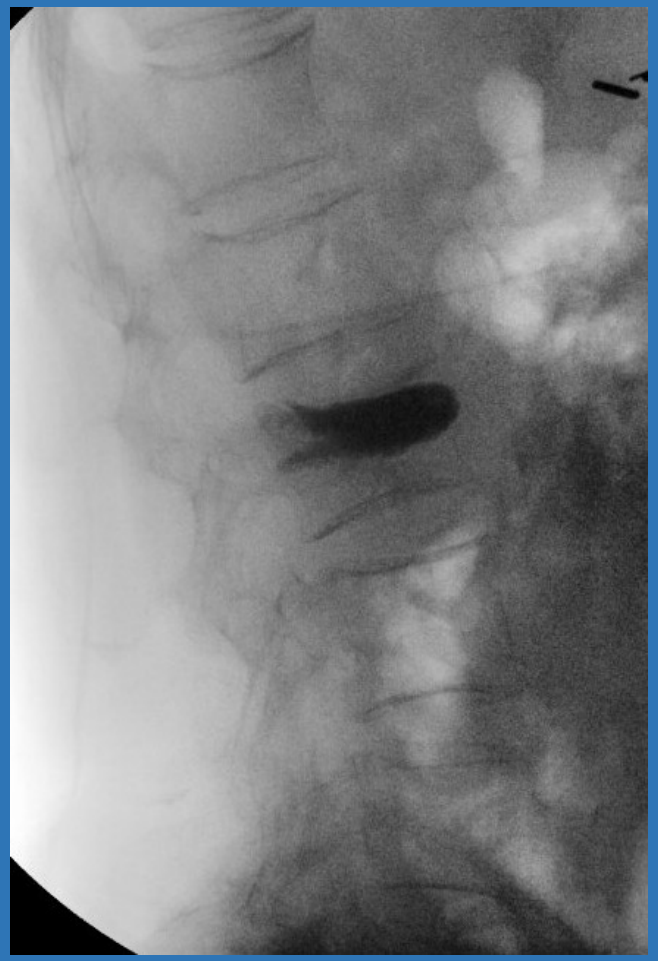
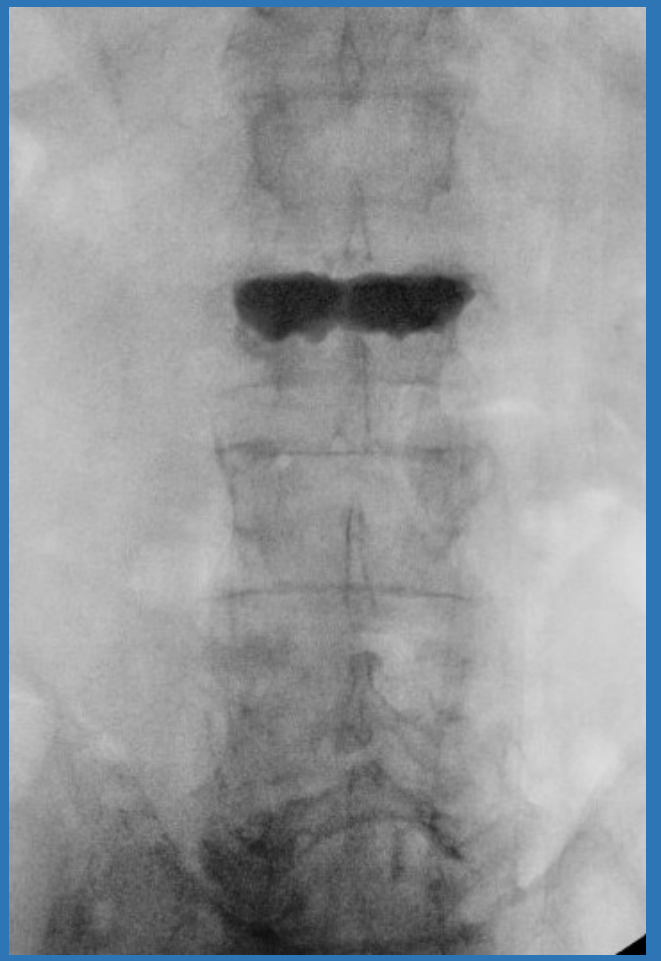





Mortality Outcomes of Vertebral Augmentation (Vertebroplasty and/or Balloon Kyphoplasty) for Osteoporotic Vertebral Compression Fractures: A Systematic Review and Meta-Analysis

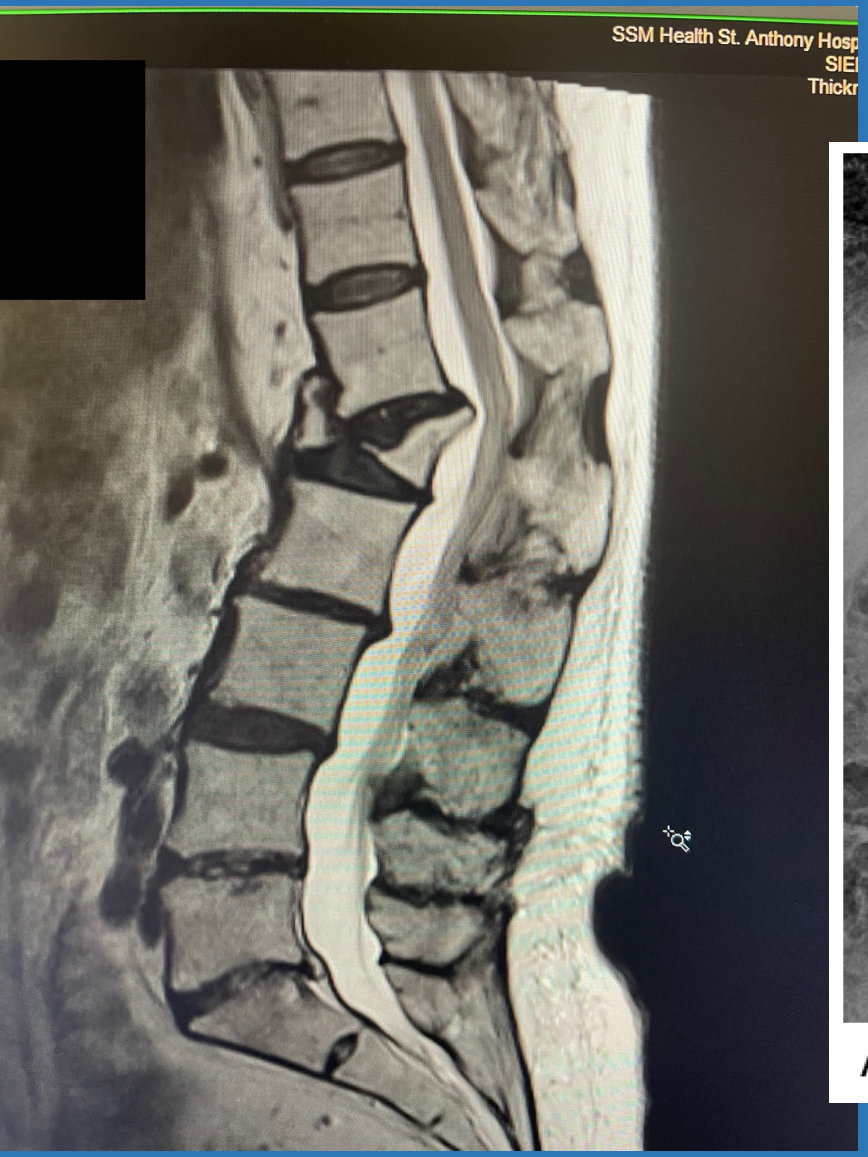
Kenji Hinde, Julian Maingard, Joshua A. Hirsch, Kevin Phan, Hamed Asadi, Ronil V. Chandra

- Reviewed 7 meta-analysis studies that consisted of over 2 million patients with osteoporotic vertebral compression fractures.
- Compared mortality outcomes in patients who underwent nonsurgical management versus those who underwent vertebral augmentation.
- Found that patients who underwent vertebral augmentation had a 22% reduction in mortality up to 10 years compared to those in the nonsurgical group.

Secondary Fracture Rate After Vertebral Osteoporotic Compression Fracture Is Decreased by Anti-Osteoporotic Medication but Not Increased by Cement Augmentation

Emily S Mills [1], Raymond J Hah [1], Zoe Fresquez [1], Kevin Mertz [1], Zorica Buser [1], Ram K Alluri [1], Paul A Anderson [2]

- Reviewed 36,145 patients.
- Secondary fractures occurred in:
  - Non surgical management – 21.8%
  - Kyphoplasty – 18.5%
  - Vertebroplasty – 14.5%
- Osteoporosis medications given to 7.8% (2,833), not given to 92.2% (33,312).
- Secondary fracture rate with osteoporotic medications was 10.1%, without medications it was 21.9%.
- Conclusions:
  - Osteoporotic medicaitons reduced the rate of secondary fracture by over half.
  - Only 7.8% of patients were placed on medications.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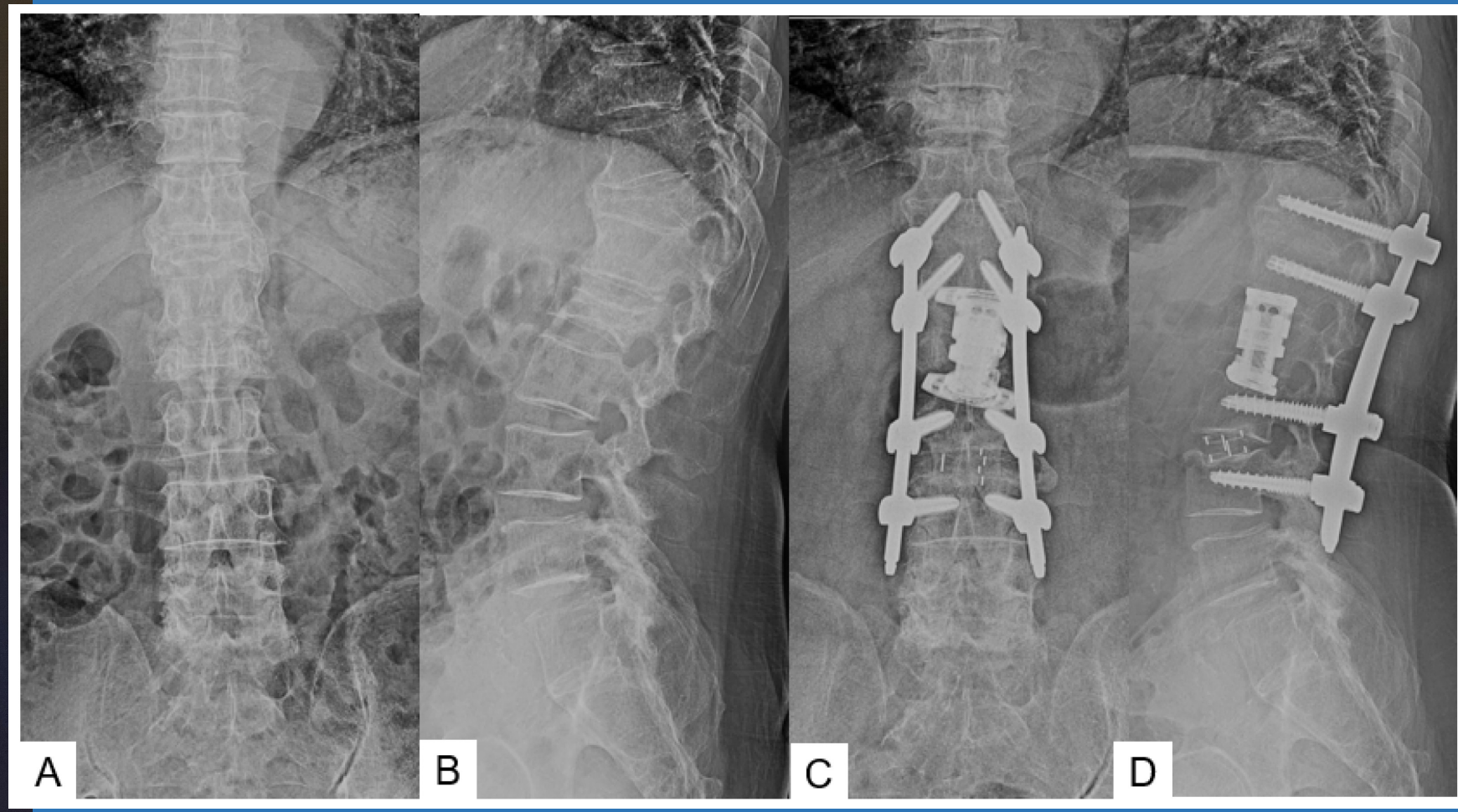
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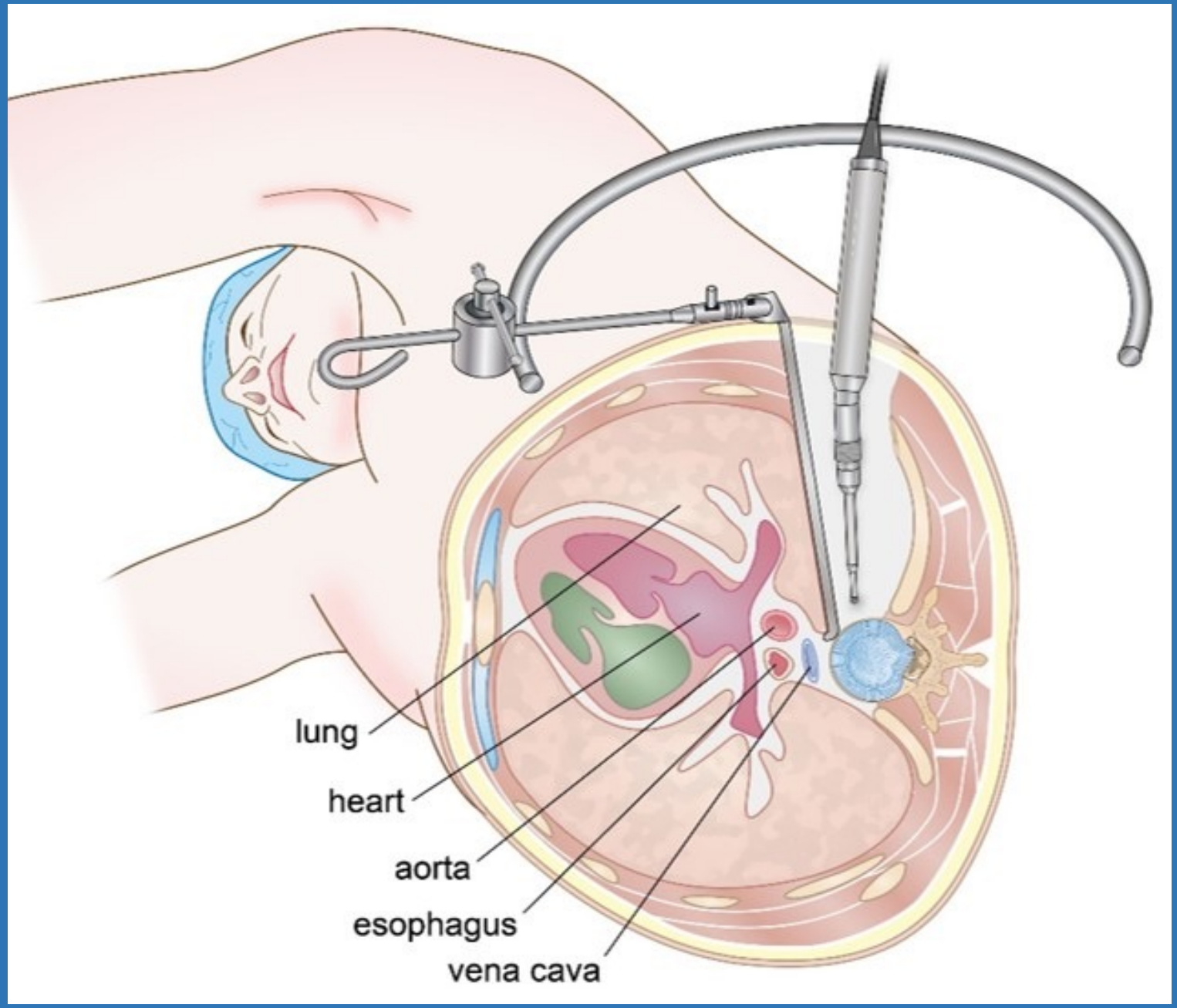
lung
heart
aorta
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vena cava

AACE/ACE 2020 POSTMENOPAUSAL OSTEOPOROSIS TREATMENT ALGORITHM

Lumbar spine or femoral neck or total hip T-score of ≤ -2.5, a history of fragility fracture, or high FRAX® fracture probability*

Evaluate for causes of secondary osteoporosis

Correct calcium/vitamin D deficiency and address causes of secondary osteoporosis

- Recommend pharmacologic therapy
- Education on lifestyle measures, fall prevention, benefits and risks of medications

High risk/no prior fractures**

- Alendronate, denosumab, risedronate, zoledronate***
- **Alternate therapy:** Ibandronate, raloxifene

Reassess yearly for response to therapy and fracture risk

Increasing or stable BMD and no fractures

Consider a drug holiday after 5 years of oral and 3 years of IV bisphosphonate therapy

Resume therapy when a fracture occurs, BMD declines beyond LSC, BTM's rise to pretreatment values or patient meets initial treatment criteria

Progression of bone loss or recurrent fractures

- Assess compliance
- Re-evaluate for causes of secondary osteoporosis and factors leading to suboptimal response to therapy

- Switch to injectable antiresorptive if on oral agent
- Switch to abaloparatide, romosozumab, or teriparatide if on injectable antiresorptive or at very high risk of fracture
- Factors leading to suboptimal response

ABBREVIATIONS GUIDE

BMD – bone mineral density
LSC – least significant change
BTM – bone turnover marker

Very high risk/prior fractures**

- Abaloparatide, denosumab, romosozumab, teriparatide, zoledronate***
- Alternate therapy: Alendronate, risedronate

Reassess yearly for response to therapy and fracture risk

Denosumab	Romosozumab for 1 year	Abaloparatide or teriparatide for up to 2 years	Zoledronate
Continue therapy until the patient is no longer high risk and ensure transition with another antiresorptive agent.	Sequential therapy with oral or injectable antiresorptive agent	Sequential therapy with oral or injectable antiresorptive agent	• If stable, continue therapy for 6 years**** • If progression of bone loss or recurrent fractures, consider switching to abaloparatide, teriparatide or romosozumab

* 10 year major osteoporotic fracture risk ≥ 20% or hip fracture risk ≥ 3%. Non-US countries/regions may have different thresholds.

** Indicators of very high fracture risk in patients with low bone density would include advanced age, frailty, glucocorticoids, very low T scores, or increased fall risk.

*** Medications are listed alphabetically.

**** Consider a drug holiday after 6 years of IV zoledronate. During the holiday, an anabolic agent or a weaker antiresorptive such as raloxifene could be used.

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