DOI: 10.20514/2226-6704-2022-12-4-254-266
EDN: TLTRAB
УДК 616.711-007.234-001.5-079.4

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ОСТРЫЙ ОСТЕОПОРЕТИЧЕСКИЙ ПЕРЕЛОМ ПОЗВОНОЧНИКА. ЧАСТЬ 1. ОПРЕДЕЛЕНИЯ, КЛИНИЧЕСКАЯ КАРТИНА, ОЦЕНКА БОЛЕВОГО СИНДРОМА, ДИАГНОСТИЧЕСКАЯ ВИЗУАЛИЗАЦИЯ, ВВЕДЕНИЕ В ДИФФЕРЕНЦИАЛЬНЫЙ ДИАГНОЗ

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Acute Osteoporotic Vertebral Fracture. Part 1. Definitions, Clinical Presentation, Pain Assessment, Diagnostic Imaging, Introduction to Differential Diagnosis

Резюме

Остеопороз — широко распространенное метаболическое заболевание скелета среди лиц 50 лет и старше. Значимым проявлением заболевания являются остеопоретические переломы, которые могут оказывать существенное влияние на качество жизни. Целью данной публикации является рассмотрение подходов к ведению пациентов с острым остеопоретическим переломом. Данная работа разделена на две части. В первой части рассматриваются общие сведения об остеопорозе, варианты течения остеопоретического перелома, дифференциальный

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диагноз болевого синдрома, методы визуализации переломов, дифференциальная диагностика остеопороза. Во второй части работы рассматриваются особенности дифференциальной диагностики остеопоретического перелома по данным визуализирующих методов, немедикаментозные, медикаментозные и хирургические методы лечения.

Ключевые слова: *остеопоретический перелом, остеопороз, перелом позвоночника*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

Источники финансирования

Авторы заявляют об отсутствии финансирования при проведении исследования

Статья получена 06.11.2021 г.

Принята к публикации 04.02.2022 г.

Для цитирования: Лялина В.В., Борщенко И.А., Борисовская С.В. и др. ОСТРЫЙ ОСТЕОПОРЕТИЧЕСКИЙ ПЕРЕЛОМ ПОЗВОНОЧНИКА. ЧАСТЬ 1. ОПРЕДЕЛЕНИЯ, КЛИНИЧЕСКАЯ КАРТИНА, ОЦЕНКА БОЛЕВОГО СИНДРОМА, ДИАГНОСТИЧЕСКАЯ ВИЗУАЛИЗАЦИЯ, ВВЕДЕНИЕ В ДИФФЕРЕНЦИАЛЬНЫЙ ДИАГНОЗ. Архивъ внутренней медицины. 2022; 12(4): 254-266. DOI: 10.20514/2226-6704-2022-12-4-254-266. EDN: TLTRAB

Abstract

Osteoporosis is a widespread metabolic disease of the skeleton among the elderly. Osteoporotic fractures are significant manifestation of the disease, which can substantially affect the quality of life. The purpose of this article is to review approaches to the management of patients with acute osteoporotic fracture. This article consists of two parts. The first part reviews general information about osteoporosis, clinical course of osteoporotic fracture, differential diagnosis of pain syndrome, methods of visualization of fractures, differential diagnosis of osteoporosis. In the second part, we discuss differential diagnosis of osteoporotic fracture according to the data of imaging methods, non-pharmacologic, pharmacologic and surgical methods of treatment.

Key words: *osteoporotic fracture, osteoporosis, vertebral fracture*

Conflict of interests

The authors declare no conflict of interests

Sources of funding

The authors declare no funding for this study

Article received on 06.11.2021

Accepted for publication on 04.02.2022

For citation: Lyalina V.V., Borshenko I.A., Borisovskaya S.V. et al Acute Osteoporotic Vertebral Fracture. Part 1. Definitions, Clinical Presentation, Pain Assessment, Diagnostic Imaging, Introduction to Differential Diagnosis. The Russian Archives of Internal Medicine. 2022; 12(4): 254-266. DOI: 10.20514/2226-6704-2022-12-4-254-266. EDN: TLTRAB

25(OH)D — 25-hydroxycalciferol, CT — computed tomography, DXA — dual energy X-ray absorptiometry, ESR — erythrocyte sedimentation rate, GIT — gastrointestinal tract, MPS — myofascial pain syndrome, MRI — magnetic resonance imaging, OP fracture — osteoporotic fracture, STIR — Short Tau Inversion Recovery (inversion recovery spin echo sequence, fat suppression mode)

Introduction

Osteoporosis is a metabolic disease of the skeleton characterized by decreasing bone mass, impaired micro-architectonics of bone tissue and, as a result, minimal trauma fractures [1].

Two opposite processes constantly take place in bone tissue: bone formation by osteoblasts, and bone resorption determined by osteoclasts. Osteoblasts are derived from immature progenitor cells in periosteum and bone marrow; they produce and mineralize bone matrix composed primarily of type I collagen. Insulin-like growth factor II and transforming growth factor-beta stimulate the formation of bone tissue by mature osteoblasts. Osteoblasts surrounded by matrix transform into osteocytes that stop participating in the processes of mineralization and matrix synthesis, however, participate in the paracrine regulation of active osteoblasts, and also, according to some data, inhibit the formation of osteoclasts. Osteoclasts are derived from cells of monocyte-macrophage series. Osteoclast activity is regulated by: parathyroid hormone, calcitonin and interleukin-6; soluble factors such as macrophage colony stimulating factor (deficiency of this factor causes osteopetrosis); transcription factors. Maximum bone mass in humans is observed at the age of about 30 years; then there is a gradual decrease in bone mass [1, 2].

Dysregulated bone formation processes can result in severe skeletal disorders characterized by decreased (e.g., osteoporosis) or increased (e.g., osteopetrosis) bone mass. Bone tissue remodeling depends on the level of estrogens, the state of phosphorus and calcium metabolism, the level of parathyroid hormone, vitamin D, growth hormone, calcitonin, thyroid hormones, glucocorticoids, senescence and senescence-associated secretory phenotype, etc. [1, 3].

Senescence and decreased gonadal function are the most important factors in the development of osteoporosis. Estrogen deficiency leads to bone loss not only in postmenopausal women, but also in men. Results of studies conducted revealed that the rate of bone loss increases significantly in the first few years after menopause onset. Estrogen deficiency leads to increased number of osteoclasts and decreased number of osteoblasts what, in general, results in bone mass loss. The risk of fractures in post-menopausal period is inversely related to estrogen levels. Osteoblasts, osteocytes and osteoclasts express estrogen receptors. In addition, estrogen has indirect effect on bones through cytokines and paracrine factors [3].

Senile osteoporosis is associated with both excessive activity of osteoclasts and progressively decreasing number of osteoblasts. At the age of 30+, bone resorption exceeds bone formation; it results in osteopenia and, in severe cases, in osteoporosis. Cortical bone loss in women amounts to 30–40 %, and cancellous bone loss — to 50 %; these values for men are 15–20 % and 25–30 %, respectively. Senescence leads to thinning of cortical layer, increased porosity of cortical tissue, and thinning of trabeculae. [3]

Calcium, vitamin D and parathyroid hormone are involved in the regulation of bone formation. Calcium deficiency in the diet or its malabsorption in the intestine can lead to secondary hyperparathyroidism. Parathyroid hormone is secreted in response to low serum calcium level. It increases bone resorption (what, in its turn, increases plasma calcium levels), reduces calcium excretion by kidneys, and increases renal production of 1.25-dihydroxyvitamin D (active hormonal form of vitamin D) that increases calcium and phosphorus absorption, and inhibits synthesis of parathyroid hormone. Vitamin D deficiency is common among the elderly and can result in secondary hyperparathyroidism due to reduced intestinal absorption of calcium [3].

Generally, all effects on bone tissue metabolism are realized via main regulation systems of osteoblastogenesis (canonical Wnt signaling pathway) and osteoclastogenesis (RANKL/RANK/OPG pathway). Changes in the expression of molecules that regulate osteoblastogenesis and osteoclastogenesis due to aging and the negative influence of other factors lead to decreased bone strength that can have presentation as impaired internal microarchitectonics, decreased bone mass and, as a result, minimal trauma fractures [1].

In Russia, 34 % of women and 27 % of men 50+ are diagnosed with osteoporosis, and the incidence of osteopenia is 43 and 44 %, respectively. The incidence of osteoporosis increases with age [4].

Osteoporosis may be primary or secondary. Primary osteoporosis develops as a separate disease that is not associated with other causes of reduced skeletal bone strength. 95 % of osteoporosis in postmenopausal women (postmenopausal osteoporosis) and 80 % of osteoporosis in men 50+ are cases of primary osteoporosis [5]. Primary osteoporosis also includes idiopathic osteoporosis that develops in women before menopause, in men under the age of 50, and juvenile osteoporosis (in children under the age of 18). Idiopathic and juvenile types of primary osteoporosis are extremely rare.

Secondary osteoporosis is caused by various diseases or conditions, as well as medications. The list of possible causes of secondary osteoporosis includes more than 70 diseases and pathological conditions and at least 20 drug categories and separate medications. 5 % of osteoporosis in women and 20 % in men correspond to secondary osteoporosis [5].

Osteoporosis of mixed genesis is also possible. For example, women with primary postmenopausal osteoporosis may develop secondary glucocorticoid-induced osteoporosis associated with administration of glucocorticoids.

The most significant clinical sign of osteoporosis is an osteoporotic fracture (OP fracture). Fractures with underlying osteoporosis occur due to a minimal trauma (for example, falls from standing height, weight lifting, or even coughing, sneezing, awkward turn/flexion of trunk, bumpy ride in a car, etc.), therefore, such fractures are also called low energy, or low trauma, or pathological. The term “pathological fracture” refers to the fractures that result from a disease, not from a traumatic effect, for example, a fracture in patients with metastatic skeletal disease, Paget’s disease, etc., thus, a fracture in osteoporosis is also a pathological one [1].

OP fractures occur most often in certain areas of the skeleton, therefore, they are called “marker fractures” [6]. The typical fractures in osteoporosis are those of the proximal femur (“femoral neck”), distal radial metaphysis, proximal humerus, and vertebral bodies. Fractures of ribs, pelvic bones, and tibia are also possible. The vertebral compression fractures are the most common type of OP fracture. [7]. They tend to happen in the mid-thoracic and thoracolumbar spine (Th7 — L2) [8]. Vertebral fractures due to osteoporosis are diagnosed in 7–12 % of men and 7–16 % of women 50+. According to several reports, the incidence of such fractures reaches 30 % in women 75+ [9]. A history of OP fracture is a risk factor for subsequent fractures. Approximately 19 % of patients with vertebral compression fractures will have another fracture next year [10].

Clinical presentation of OP vertebral fracture

There are two types of vertebral damage in osteoporosis: acute compression fracture of vertebral body, and chronic compression deformity.

Chronic compression deformity

Slow gradual compression of vertebrae ("delayed fracture") is asymptomatic or low symptomatic for a long time. Patients complain of aching pain or a sensation of heaviness in the lumbar and/or lower thoracic regions of moderate or slight intensity, rapid back fatigue in a standing position [11]. As a rule, two or three vertebrae are involved in deformation, and in this case, there is no significant deformation of a whole spinal column. Such fractures often become incidental findings during imaging studies (radiography, computed tomography (CT), magnetic resonance imaging (MRI)).

Multiple compression or complete compression of single vertebrae results in a gradual decrease in patient's height, development of thoracic kyphosis and other deformities of trunk. Most patients develop more or less significant pain syndrome and have restrictions in daily motor performance.

Back pain in chronic compression deformity is primarily represented by myotonic and vertebral pain syndromes. Vertebral deformity is also accompanied by structural changes in intervertebral discs, facet joints, ligaments; involvement of spinal cord roots, narrowing of spinal canal, and other disorders are also possible. In this regard, discogenic, radicular, facet and other pain syndromes may develop.

Acute compression vertebral fracture

Acute compression vertebral fracture is diagnosed mainly in women 15–20 years after menopause [11]. An acute fracture of vertebral body, like other OP fractures, is a result of a low energy impact. Unlike OP fractures of other localizations, most vertebral fractures are caused not by a fall, but by a compression that occurs during lifting weights, or changing body position, or during routine daily activities; there is often no indication of a traumatic moment [11].

Clinical presentation of an acute fracture

This fracture is accompanied by sharp pain in the area of damaged vertebra [6]. Vertebrae with maximum axial load (T10-12 and L1-2) are typically involved [11]. If thoracic vertebrae are damaged, girdle pain is possible; if lumbar vertebrae are involved, pain may irradiate to the anterior part of abdomen or to the posterior superior iliac spine; it is especially typical for L1 fracture [6, 11]. Pain irradiation to the limb caused by an OP fracture is rare, unlike pain caused by intervertebral hernias, however, it is possible if a nerve root is compressed by bone fragments or a simultaneous protrusion of an intervertebral disc.

Pain in acute fracture, as in the case of chronic compression deformity, usually includes vertebral and myotonic components. This pain is caused by periosteal hemorrhage, a large number of simultaneously occurring microfractures of trabeculae, and spasm of paravertebral muscles [12]. Other types of pain are also possible depending on the degree of damage and the nature of the impact of damaged vertebra on the surrounding structures.

Pain severity can be different: from moderate and tolerable that resolves spontaneously to pronounced that requires hospitalization and potent pain medications. Acute pain lasts, as a rule, for 1–2 weeks, then it gradually decreases during 2–3 months [11]. Longer duration of pain may indicate a non-healing fracture and/or progressive compression.

Pain after a fracture occurred can be either acute and paroxysmal with certain movements, or monotonous and dull. Spinal extension, sitting position, attempts to lie on one side from a sitting position, turning in bed, and the Valsalva maneuver often aggravate pain and may be accompanied by muscle spasms [8].

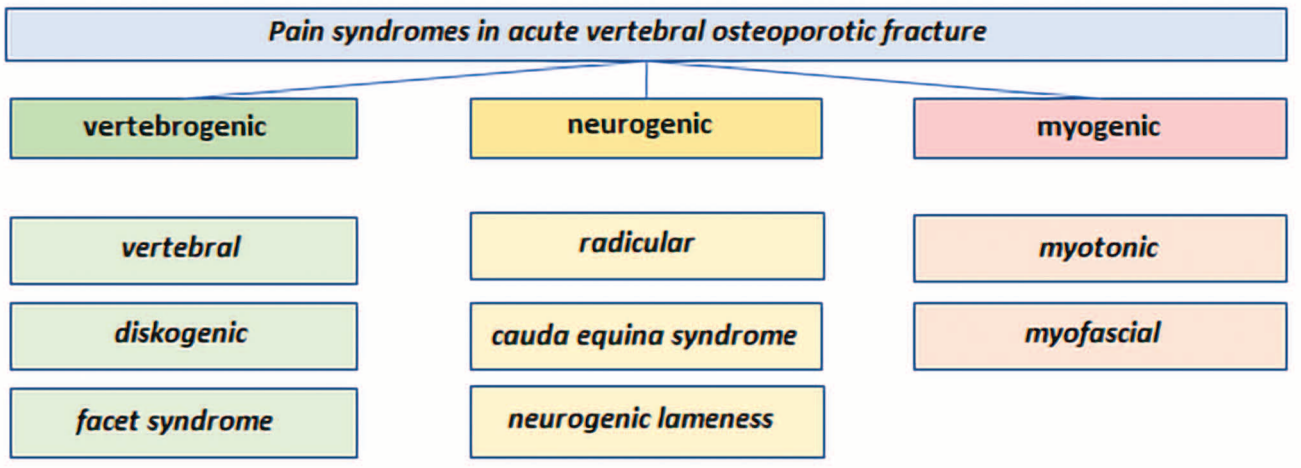
Palpation and/or percussion of spinous processes and paravertebral structures may be painful [8]. Palpation is carried out with a patient standing, with moderate pressure along the midvertebral line. Percussion is also performed with a patient standing. For percussion, a physician positions the palm of one hand over the patient's spine, and taps on it with the closed fist of the other hand. Tenderness on palpation/percussion indicates possible vertebral injury and is a highly specific clinical sign.

A patient also requires neurological examination to exclude possible compression of roots or spinal cord. Sensory deficits and weakness in limbs may indicate root compression or the presence of bone fragments in spinal canal; in such cases urgent surgical treatment may be required.

Differential diagnosis of pain syndrome in acute OP vertebral fracture

Vertebral and myotonic syndromes usually predominate in the clinical presentation of an acute OP fracture [13], however, there can be other pain syndromes that may require different treatment approaches. Pain status of each patient should be detailed as much as possible in order to select proper disease management.

Three groups of pain syndromes are routinely distinguished when it comes to spine diseases: vertebrogenic, neurological and myologic (scheme 1) [14].



Scheme 1. *Pain syndromes in acute vertebral osteoporotic fracture*

The term "vertebrogenic pain" describes pain associated with any pathology of the spine itself. Pronounced structural changes in spine, in turn, can lead to neurological disorders (radicular syndrome, cauda syndrome, neurogenic lameness, myelopathy) that are characterized by neurological pain syndromes. It is also reasonable to identify myogenic pain syndromes associated with the reaction of soft skeleton to structural changes in spine.

Vertebral pain develops with the direct damage to vertebrae. In addition to an OP-fracture, such pain can be caused by an infectious lesion of vertebra (osteomyelitis, tuberculosis) or metastasis. By nature, it is pain with a mechanical rhythm that is accompanied by tenderness of one or two spinous processes during palpation/percussion [8].

Discogenic pain originates from damaged intervertebral disc. This pain is described as extradermatomal (i.e., with no definite localization in a dermatome). Discogenic pain is most often observed in lumbar region; its typical sign is the bilateral pain in lumbar region that extends to buttocks [14, 15]. The pain is aggravated during spine flexion (forward lean), rotation, prolonged sitting or standing, as well as coughing/sneezing/straining, and is relieved in lying position. Typical signs are pain provocation during vibration load (tuning fork test) and the so-called "centralization" (onset/intensification of midline back pain that is provoked by flexion) [14].

Arthrogenic (facet) pain indicates arthrosis and/or overload of facet (zygapophyseal) joints. Its sign is a dull monotonous diffuse pain that aggravates after long standing, with extension and rotation of spine (during these movements, there occurs a strong tension of joint capsules and decrease in the volume of joint with close contact of articular surfaces), and relieves at sitting, walking, slight bending. Facet pain that originates from lumbar region often irradiates to the proximal thigh mimicking radicular pain syndrome, however, unlike it, facet pain never extends below the popliteal fossa. This pain may also irradiate to buttocks, groin, lower abdomen, and sometimes even to perineum [16]. Diagnostic block of facet joints is often used for the differential diagnosis of arthrogenic pain.

Radicular (neuropathic) pain is unilateral, with irradiation to leg often below the knee. This pain spreads along the dermatome (Figure 1), is asymmetrical (unilateral), is accompanied by sensory (numbness, paresthesia) and motor (paresis) disorders in the area of innervation by the corresponding root. Pain in limb is often the single sign of radiculopathy [15]. Table 1 presents the clinical features of radicular pain.

Cauda syndrome is a cauda equina syndrome. It is characterized by severe back pain spreading to both legs (symmetrically or asymmetrically), with the development of weakness and impaired sensitivity in legs and S-dermatomes (intergluteal fold), as well as impaired pelvic functions [19].

Neurogenic lameness develops with spinal stenosis (narrowing of spinal canal) that leads to the compression of nerve structures before their exit the intervertebral foramina. This causes lumbar pain; heaviness and weakness in legs; numbness, paresthesia and weakness in lower part of legs. Painful sensations usually appear when walking or standing for a long time and disappear after a short rest and when leaning forward [14].

Myofascial and myotonic pain syndromes. Changes in muscles can both be a separate cause of back pain, and accompany pain syndromes of other types what is a very common situation. Myofascial pain syndrome (MPS) is characterized by the formation of painful tight areas in muscles that are a result of acute or chronic overload of separate muscles. These areas are called

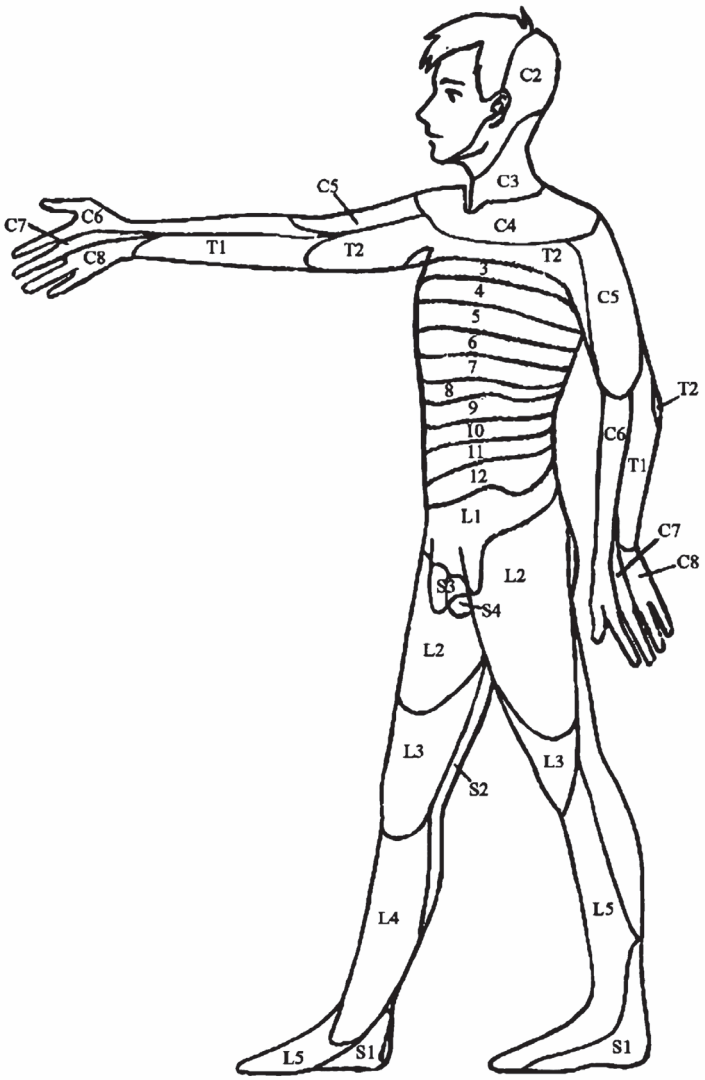


Figure 1. *Human dermatomes. According to Hawkes H.C., et al. (2019) [17]. Illustrator A.K. Rudykh*

Table 1. *Characteristics of radicular pain (adapted from Wolf J.K. (1981) [18], with additions).*

Radix	Site of pain	Irradiation	Sensory Disorders	Muscle weakness	Reflex alterations
Th	Girdle pain and dysesthesia in the area of the corresponding dermatomes				
L1	Below the groin fold	Groin area	Groin area	Hip flexion	Cremasteric
L2	Middle third of the anterior thigh	Groin area, Anterior thigh	Anterior thigh	Hip flexion, hip adduction	Adductor
L3	Anterior thigh and knee	Anterior thigh, knee	Distal anteromedial thigh, knee area	Lower leg extension, thigh flexion and adduction	Knee, Adductor
L4	Middle part of the lower leg and ankle	Anterior thigh, knee	Medial thigh	Lower leg extension, thigh flexion and adduction	Adductor
L5	Buttocks, posterolateral thigh, lower leg and foot	Posterolateral surface of the thigh, lateral surface of the lower leg, medial edge of the foot up to I — II fingers	Lateral surface of the lower leg, dorsum of the foot, I — II toes	Dorsiflexion of the foot (flap foot) and big toe, hip extension	No
S1	Posterior surface of the leg and buttocks	The back of the thigh and lower leg, lateral edge of the foot	Posterolateral surface of the leg, the lateral edge of the foot	Plantar flexion of the foot and toes, flexion of the lower leg and thigh	Achilles

trigger points, or myofascial nodules; the outdated name of "myogelosis" is also often observed. MPS is characterized by local "spot" and/or regional pain while its area often does not coincide with the topographic boundaries of the trigger muscle and can extend far beyond its limits. MPS results in asymmetric restriction of movements. When the affected muscle is stretched, the pain decreases. Main diagnostic method is palpation when sharply painful trigger points can be found in certain areas of muscle. When trigger points are stimulated, patient's habitual pain restarts or increases [20]. Myofascial pain can be debilitating, persisting for many years, and has a significant impact on motor activity and, in general, on patient's quality of life. MPS associated with the large square muscle of lower back and with piriformis muscle is more often detected with underlying structural damage of lower thoracic and lumbar regions [21, 22].

Myotonic pain, on the contrary, is more extensive, dull, aching, and dragging. It is triggered by movements and increases significantly in positions when the muscles surrounding the spinal column are stretched. Pain can also increase with prolonged staying in one posture (during driving a car, a long flight, etc.). Paravertebral muscles are tight, tense, and painful on palpation [21]. Secondary muscle pain can become chronic and persist independently, even after the initial cause disappears.

Diagnosis

Diagnostic search in a patient with an acute OP fracture involves the verification and classification of a fracture itself, as well as the differential diagnosis of its causes.

Fracture verification

Visualization methods.

To verify an acute OP fracture, radiography, computed tomography (CT) and magnetic resonance imaging (MRI) are used. Use of these methods is presented in Figures 2 and 3.

A patient with a suspected acute vertebral body compression fracture should first have an X-ray of the thoracic and/or lumbar spine.

X-ray is a fast, affordable, and low-cost method [23]. It allows identifying the deformity of vertebral body, however, not the age of the fracture what is especially important in cases when healing should be evaluated over time, as well as in situations when the fracture occurred with already existing multiple deformities of other vertebrae. In addition, X-ray demonstrates only bone structures of the spine; it does not allow assessing the state of other structures (discs, ligaments, spinal canal), roots and spinal cord.

CT is also a fast and fairly affordable method [23]. Unlike X-ray, CT provides more detailed information about the state of the bone structures of spine, allowing not only to assess the anatomical integrity, but also to find compression deformities of a separate part of vertebra. In addition, CT evaluates the condition of spinal canal and its contents. Therefore, CT may be the method of choice if a fracture is suspected. Disadvantages of CT include high cost and predominant visualization of bone structures.

MRI demonstrates in detail all the structures of spine, spinal cord and roots, and also allows assessing the stage and changes in fracture healing over time based on the parameters of bone edema (Figures 2c and 2d) [23]. From this point of view, MRI is preferable to radiography and CT, however, the use of MRI is limited by cost, inequal availability, and contraindications. Moreover, one should keep in mind that spinal MRI is a long examination that requires about 30 minutes when a patient should be motionless in the tomograph. For a patient in acute fracture stage and with severe pain, this may be

Thus, X-ray and/or CT help to quickly diagnose a vertebral fracture and to obtain approximate information about the state of surrounding structures. If the results of these examinations and/or clinical presentation give the reason to suspect significant damage to intervertebral discs, nerve roots, spinal cord, etc., associated with a fracture, then MRI is mandatory. In addition, indications for MRI include the ineffectiveness of conservative treatment, progression of symptoms, and the need to assess the fracture over time.

Classification of OP fractures

Both acute and chronic OP fractures are classified according to their shape and grade.

According to the shape, biconcave ("medium deformation"), wedge-shaped ("anterior deformation"), and compression ("posterior deformation", "compression deformation") fractures are distinguished (Figure 4). [18] Anterior wedge-shaped deformity is the most common [8].

Depending on the decrease of vertebral height, 3 grades of fractures are distinguished: Grade 1 — decrease in vertebral height by 20–25 %, Grade 2 — by 25–40 %, Grade 3 — >40 % [18]. This classification is convenient and illustrative, however, it gives no idea of the changes in the spatial geometry of vertebra after fracture, thus, creating a misleading impression of "damage in one plane". Besides, keep in mind the possibility of combined compression and comminuted injuries in acute OP fracture that can cause neurological complications.

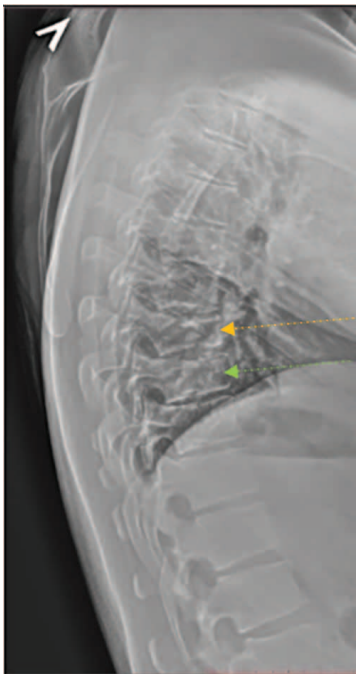


Figure 2а. *Digital radiography of the thoracic spine: acute compression fracture of the T8 (yellow arrow), T9 (green arrow) vertebrae: decrease in the height of the ventral part of the body, wedge-shaped vertebral body*

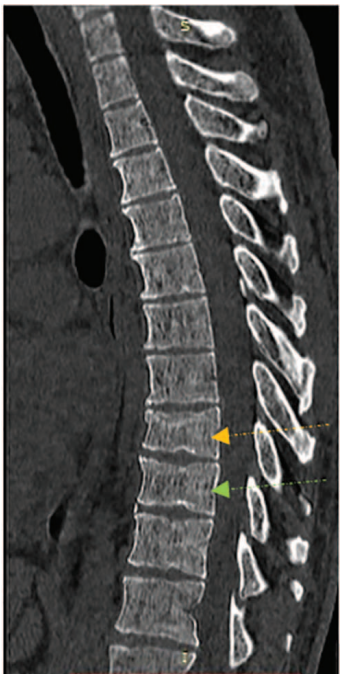


Figure 2б. *CT of the thoracic spine, sagittal reconstruction. Acute compression fracture of the Th8 (yellow arrow) and Th9 (green arrow) vertebrae: decrease in the height of the ventral part of the bodies, wedge-shaped shape of the vertebral bodies, fracture line can be traced in the compression zone as well as compaction of the spongy part of the bodies, a bony «notch» along the ventral surface as a sign of acute compression in the Th8 vertebra*

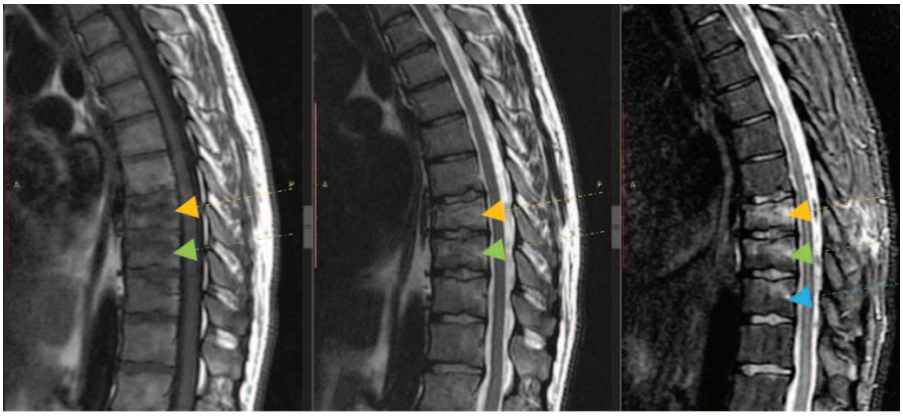


Figure 2в. *MRI of the thoracic spine: acute compression fracture of the T8 (yellow arrow), T9 (green arrow) vertebrae: decrease in the height of the ventral part of the bodies, wedge-shaped shape of the vertebral bodies, decreased signal intensity in T1 WI, an increased intensity in T2 WI, significantly increased signal intensity in STIR mode from the body as a manifestation of an acute bone edema on the background of a "fresh" fracture. Similar changes in the body of the Th10 vertebra (blue arrow), as a reflection of bone contusion or incipient compression fracture.*

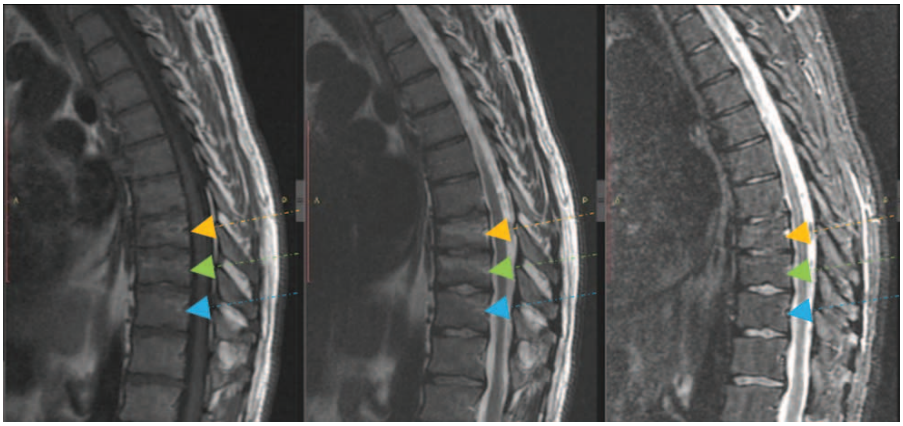
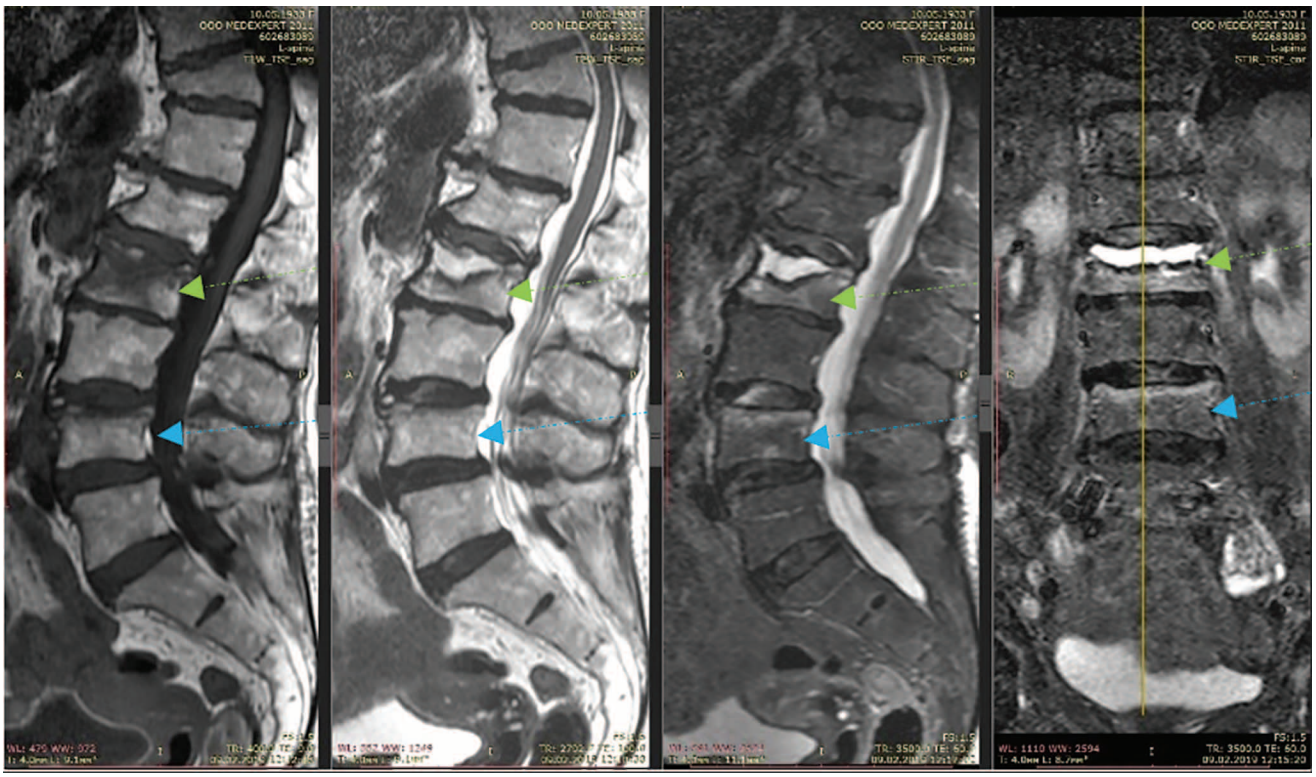


Figure 2г. *MRI of the thoracic spine 3.5 months after the fracture. Signs of fracture consolidation and disappearance of bone edema: increased signal intensity from the body in T1 WI, iso-intensive or slightly hyperintense signal from the body in T2 WI, iso-intensive signal from the vertebral bodies in STIR mode as a reflection of the of edema resolution and replacement of this area with adipose tissue of the bone marrow. Regression of bone edema of the Th10 vertebra (blue arrow), the fracture did not develop, the vertebral body is not deformed*

Picture 2. *Acute compression fracture of the Th8, Th9, Th10 vertebrae*
Note: CT — computed tomography, MRI — magnetic resonance imaging, WI — weighted image, STIR — short tau inversion recovery)



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Picture 3 a, b, c, d. *MRI of the lumbar spine. Acute compression fracture of the L2 vertebral body (green arrow)*

Picture 3a. *Significantly decreased intensity of the signal from the vertebral body (green arrow) in the area of bone edema in T1 WI. Signal intensity in the line of bone compression and compaction of bone tissue is even lower*

Picture 3b. *zone of increased signal intensity from the preserved part of the vertebral body (green arrow) as a manifestation of bone edema, significantly increased signal intensity from the fracture zone, as a manifestation of hemorrhage in the fracture zone in T2-WI (sagittal)*

Pictures 3c and 3d. *Significantly increased signal intensity from the zone of fracture (green arrow), hemorrhage and bone edema (sagittal and frontal sections) in STIR mode*

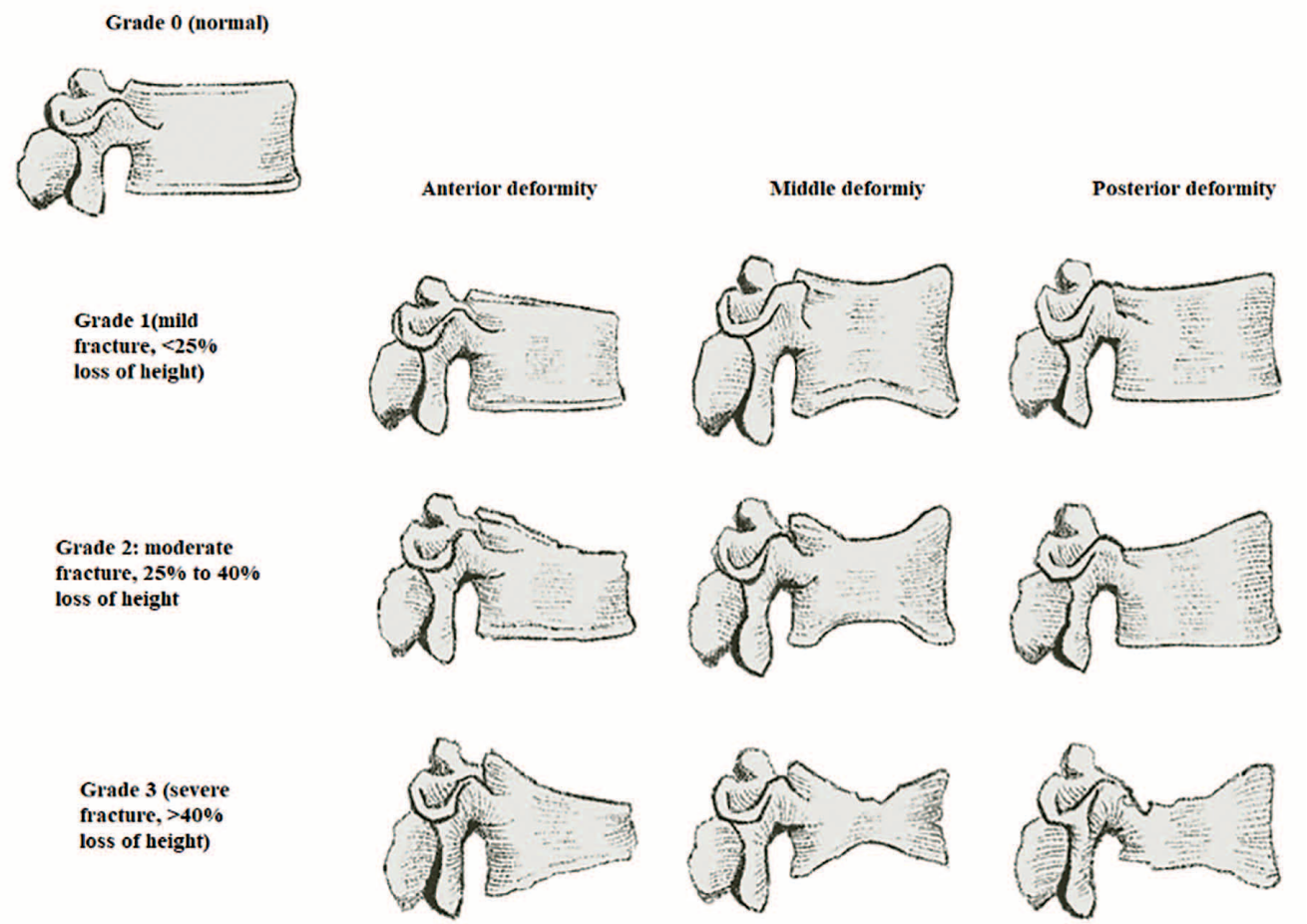
Minimal compression fracture of the superior part of the body of the L4 vertebra (blue arrow): decreased signal intensity from the subcortical parts of the body in T1 WI, T2 WI and an increased signal intensity in STIR mode from this area

Picture 3. *Acute compression fracture of the L2 vertebral body*
Note: MRI — magnetic resonance imaging, WI — weighted image, STIR — short tau inversion recovery)

Differential diagnosis of osteoporosis

A routine method for diagnosing osteoporosis is X-ray densitometry (dual energy X-ray absorptiometry, DXA). The measurement of bone density in the region of lumbar vertebrae, as well as in proximal femur is considered to be the most informative method. Several parameters are calculated including the absolute bone density value (grams per square centimeter), as well as the T-score (difference between patient's bone density and the data in the reference base for the corresponding sex, race and age, expressed as standard deviations). The diagnosis of osteoporosis in individuals 50+ is based on the T-score that indicates how much patient's bone density differs from the normal value. Osteoporosis is diagnosed if this value in L1–L4 region and proximal femur is –2.5 standard deviations and lower [1].

In the case of compression deformities, especially multiple and accompanied by spinal curvature progressing over many years, the diagnosis of osteoporosis is not a challenge: results of spinal X-ray, examination findings and history are sufficient. However, one should understand that in patients with severe compression deformities, DXA in lumbar region is often false negative, i.e. bone density values are within normal or even elevated. First of all, this is due to the fact that the sagging vertebra becomes more compact



Picture 4. *Classification of vertebral deformities. According to H.K. Genant (1993) [24]. Illustrator A.K. Rudykh*

and is represented as an area of increased density. Moreover, aortic calcification, endplate sclerosis, ligament calcification, osteophyte proliferation, and other morphological changes that develop with age can contribute to the misrepresentation of results [23]. In such cases, one is recommended to focus on the parameters in the area of proximal femur or to make additional measurements in the distal third of forearm. [6].

If the fracture occurred for the first time in a patient with no known history of osteoporosis and normal shape of other vertebrae, then the cause should be established very thoroughly. It can be the following diseases, except osteoporosis: hyperparathyroidism, multiple myeloma, metastatic, infectious lesions and primary vertebral neoplasias [8]. Thus, DXA plays an important, however, not decisive role in the diagnostic search, since even positive results confirming osteoporosis do not allow us to assert the absence of other possible causes of fracture. On the other hand, negative results of densitometry (normal or slightly reduced bone density) does not mean the absence of osteoporosis, as it is a highly specific but low-sensitive test, and its result can be affected by many factors [6]. In some cases, the diagnosis of osteoporosis can be established even with a negative DXA result, if it is a minimal trauma fracture with all other causes that have been excluded [6].

The following approximate examination plan is recommended (Table 2):

The most difficult task from this list is the exclusion of a single metastatic and myeloma lesion of vertebra, as well as hemangioma; final diagnosis in some cases can only be established based on biopsy results. If there are strong suspicions of the secondary nature of vertebral damage and a single lesion of this vertebra is observed, then it is reasonable to first perform a needle biopsy [25]. If the patient has indications for surgical treatment of a fracture (vertebroplasty or kyphoplasty), then these interventions are recommended to be performed only after receiving the results of a histological test. This is required because the primary biopsy may not be informative enough; then a repeated sampling from vertebra will be required that is impossible with cement placed into vertebral body.

Table 2. Differential diagnosis of osteoporosis

Examinations	Assumed diseases
Parathyroid hormone, alkaline phosphatase, total calcium (serum)	Hyperparathyreosis
ESR, total protein, plasma protein fraction(serum)	Multiple myeloma
Phosphorus, 25(OH)D	Oncogenic osteomalacia
Skeletal scintigraphy, Comprehensive oncological examination	Metastatic bone lesion
DXA	Osteoporosis
Exclusion of secondary causes of osteoporosis	Endocrinological, rheumatological, gastrointestinal, renal diseases, blood disorders, drugs (steroids, aluminum in antacids, antiepileptic drugs, barbiturates, aromatase inhibitors), alcohol
Vertebral biopsy (If surgery (kyphoplasty or vertebroplasty) is planned, vertebral biopsy is mandatory)	Haemangioma, multiple myeloma, metastatic lesion, primary spine tumor

Note: ESR — erythrocyte sedimentation rate, 25(OH)D– 25-hydroxycalciferol, DXA — Dual-energy X-ray absorptiometry

Conclusion

Back pain is a complex clinical issue; it requires extensive differential diagnostic search. Osteoporotic fracture is one of the most common causes of back pain in elderly patients. Diagnosis of an osteoporotic fracture is based on a thorough analysis of clinical findings and laboratory test results, and also requires the targeted use of advanced imaging methods.

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Все авторы внесли существенный вклад в подготовку работы, прочли и одобрили финальную версию статьи перед публикацией

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