DOI: 10.20514/2226-6704-2022-12-6-438-449
EDN: LZINTG
УДК 616.711-007.234-06:616.711-001.5-079.4

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ОСТРЫЙ ОСТЕОПОРЕТИЧЕСКИЙ ПЕРЕЛОМ ПОЗВОНОЧНИКА. ЧАСТЬ 2. ДИФФЕРЕНЦИАЛЬНАЯ ДИАГНОСТИКА ПО ДАННЫМ ВИЗУАЛИЗИРУЮЩИХ МЕТОДОВ. КОНСЕРВАТИВНОЕ И ХИРУРГИЧЕСКОЕ ЛЕЧЕНИЕ

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Acute Osteoporotic Vertebral Fracture. Part 2. Differential Diagnostics According to the Data of Imaging Methods. Conservative and Surgical Treatment

Резюме

Остеопороз — широко распространенное метаболическое заболевание скелета среди лиц старше 50 лет. 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 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, дифференциальная диагностика*

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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 14.12.2021 г.

Принята к публикации 04.07.2022 г.

Для цитирования: Лялина В.В., Борщенко И.А., Борисовская С.В. и др. ОСТРЫЙ ОСТЕОПОРЕТИЧЕСКИЙ ПЕРЕЛОМ ПОЗВОНОЧНИКА. ЧАСТЬ 2. ДИФФЕРЕНЦИАЛЬНАЯ ДИАГНОСТИКА ПО ДАННЫМ ВИЗУАЛИЗИРУЮЩИХ МЕТОДОВ. КОНСЕРВАТИВНОЕ И ХИРУРГИЧЕСКОЕ ЛЕЧЕНИЕ. Архивъ внутренней медицины. 2022; 12(6): 438-449. DOI: 10.20514/2226-6704-2022-12-6-438-449. EDN: LZINTG

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, differential diagnosis*

Conflict of interests

The authors declare no conflict of interests

Sources of funding

The authors declare no funding for this study

Article received on 14.12.2021

Accepted for publication on 04.07.2022

For citation: Lyalina V.V., Borshenko I.A., Borisovskaya S.V. et al. Acute Osteoporotic Vertebral Fracture. Part 2. Differential Diagnostics According to the Data of Imaging Methods. Conservative and Surgical Treatments. The Russian Archives of Internal Medicine. 2022; 12(6): 438-449. DOI: 10.20514/2226-6704-2022-12-6-438-449. EDN: LZINTG

CT — computed tomography, DXA — Dual energy X-ray absorptiometry, KP — kyphoplasty, MM — multiple myeloma, MPS — myofascial pain syndrome, MRI — magnetic resonance imaging, OP fracture — osteoporotic fracture, T1-WI — T1-weighted image, T2-WI — T2 — weighted image, VP — vertebroplasty

Acute osteoporotic vertebral fracture (OP fracture) is one of the most common structural injuries of spine in elderly individuals. In most cases, such a fracture is accompanied by pronounced pain syndrome and a significant decrease in patient's motor activity. Such nonspecific clinical manifestations require careful verification of fracture, as well as differential diagnosis with other diseases that can lead to vertebral fracture. Acute OP fracture can be managed using conservative and surgical treatment methods.

Differential diagnosis of acute OP fracture based on the results of imaging studies

A low-energy vertebral fracture can develop due to osteoporosis or have other causes, including vertebral hemangioma, metastatic lesion, or primary malignant tumor in vertebral body (including multiple myeloma).

Fractures that are a result of these diseases are clinically indistinguishable, since they are present as nonspecific signs: acute pain in the area of injured vertebra and secondary limited range of motion. Diagnostic imaging can confirm the presence of fracture and are the first step in differential diagnosis determining the need for further examination and its direction.

Vertebral hemangioma

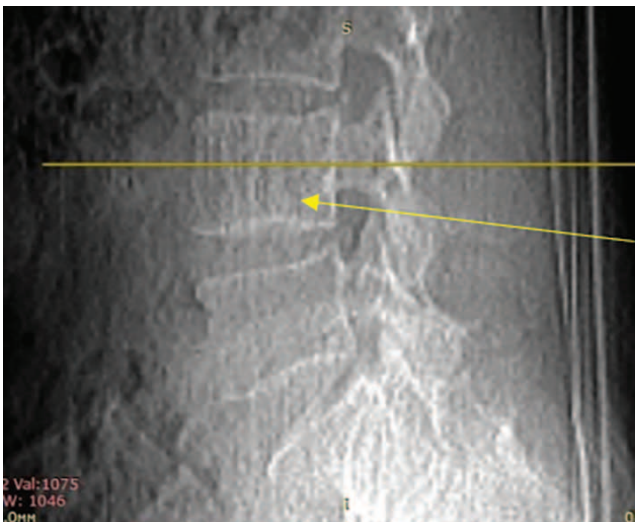
Vertebral hemangioma is a common benign vascular tumor and the most common neoplasm of spinal column; it occurs in 10–20 % of adults [1]. In most cases, hemangiomas are asymptomatic and are found incidentally. Hemangiomas can be single and multiple. Typically, they are rounded lesions with sharp contours, several millimeters in diameter, however, they may be large and cover the entire vertebral body. It is these hemangiomas that can cause a pathological fracture of vertebral body.

Histologically, hemangiomas include thin-walled vessels and sinuses lined by a layer of endothelial cells interspersed with sparse bone trabeculae oriented along the spinal axis. Adipose stroma is located between the vessels [2].

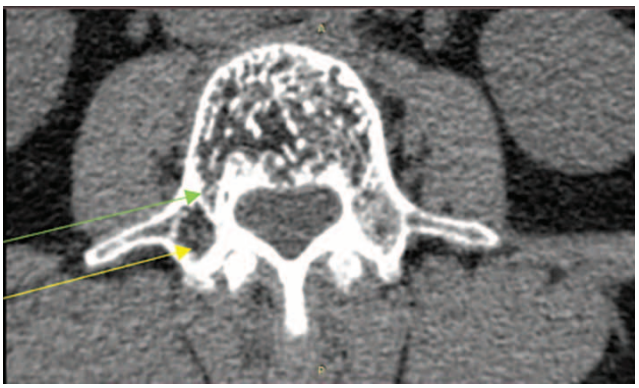
An X-ray image reveals typical longitudinal striation of the vertebral body in the presence of rare and thickened bone trabeculae ("honeycomb" appearance) [3]. Computed tomography (CT) demonstrates hemangioma

as low-density focus with the inclusion of rare bone trabeculae; the tissue of vertebral body on axial sections resembles honeycomb. According to magnetic resonance imaging (MRI), in the T1-weighted image (T1-WI), there is increased signal intensity due to the adipose tissue in lesion. In T2-weighted mode (T2-WI), the signal intensity is also increased due to the high water content, and this signal is usually more intense than the signal from adipose tissue, which distinguishes hemangioma from local fat deposits [4].

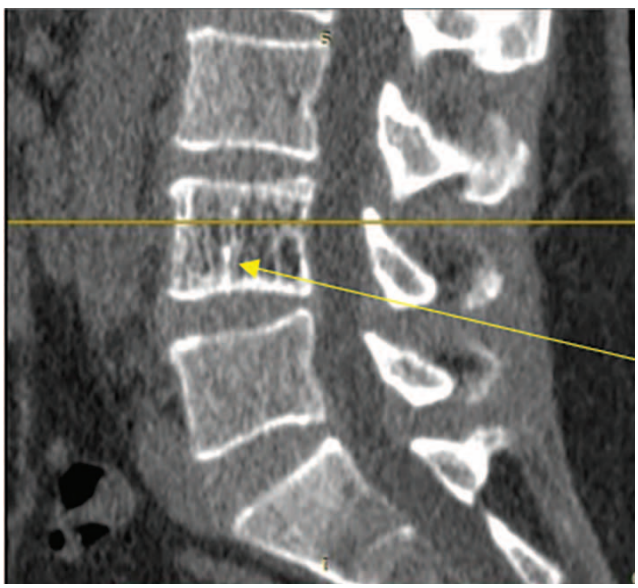
A vertebral body fracture due to a large hemangioma has no clinical differences from an OP fracture. In several cases differential diagnosis can be performed using MRI and CT imaging, however, most often, this diagnosis can be established only if the patient has a known history of hemangioma or results of previous studies. As a rule, the final diagnosis can be established after a bone biopsy performed during reconstructive surgery. The examples of hemangiomas demonstrated with the help of imaging techniques are shown in Figure 1.



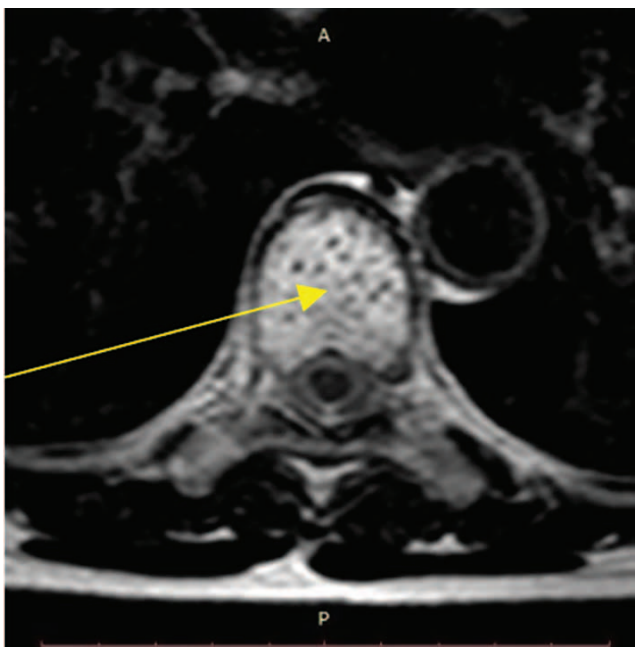
Picture 1a. *Radiography in lateral projection. Thickened longitudinally oriented trabeculas ("corduroy sign") are visible on the background of increased transparency of the bone tissue of the vertebral body*



Picture 1в. *CT, axial slice through the L4 vertebral pedicle. The hemangioma occupies over a half of the vertebral body and expands to the right pedicle and the articular process of the vertebra. Cross section through the hemangioma appears as a polka-dot pattern*



Picture 1б. *CT, sagittal reconstruction. Thickened longitudinally oriented trabeculas ("corduroy sign") are visible on the background of increased transparency of the bone tissue of the vertebral body. "Corduroy sign" is much better visualized than by radiography*



Picture 1г. *MRI T2 WI, axial slice through the middle of the Th8 body. Aggressive hemangioma, honeycomb pattern*

Figure 1. *Hemangioma of the L4 vertebral body (Observation by I.A. Borshenko)*
Abbreviation: CT — computed tomography, MRI — magnetic resonance imaging, T2 WI — T2 weighted imaging

Multiple myeloma

Multiple myeloma (MM) is a B-cell malignant tumor with the morphological substrate of plasma cells that produce monoclonal immunoglobulin [5]. Thus, MM refers to peripheral B-cell lymphoid tumors and is characterized by the bone marrow infiltration with plasma cells, the presence of monoclonal immunoglobulin in serum and/or urine, and osteolytic bone lesions. MM accounts for approximately 1 % of all malignant tumors and up to 10–15 % of all tumors of the hematopoietic and lymphoid tissues. This disease develops predominantly in elderly individuals. The average age of new patients is about 70 years. In 2020, the incidence of MM in Russia was 2.64 per 100,000 of population [6].

Bone marrow damage in the presence of MM can be both diffuse and focal.

The main clinical signs of MM is the bone pain. One of the typical localizations of myeloma is vertebrae; in most cases thoracic and lumbar spine regions are affected. Therefore, the decreased growth due to vertebral compression deformation and acute compression fractures can develop. Laboratory tests reveal normochromic normocytic anemia, pronounced acceleration of erythrocyte sedimentation rate (ESR), increased total protein level, dysproteinemia with M-gradient (paraprotein, monoclonal immunoglobulin), hypercalcemia, proteinuria [5].

The results obtained by imaging techniques help to provisionally identify four main MM patterns; the first three are the most relevant in terms of differential diagnosis of acute OP fracture [7]:

- disseminated form with multiple, well-defined demarcated lytic lesions,
- disseminated form of diffuse osteopenia type,
- solitary plasmacytoma (single lesion in vertebral body or in pelvic bones),
- osteosclerosing myeloma.

Diagnosis of MM is based on the results of laboratory tests, as well as on the data of morphological, immunohistochemical and cytogenetic tests of bone marrow biopsy material. However, in the case of an acute vertebral compression fracture in a patient without the known history of MM, the first step in diagnosis is likely to be the assessment of imaging results.

Figure 2a. *MRI, T1 WI, sagittal slice. Low signal intensity from the vertebral body, significant decrease in height over the entire area of the vertebral body, flat shape of the vertebra (vertebra plana), bulging posterior wall of the vertebral body*

Figure 2б. *MRI, T2 WI, sagittal slice. Mixed signal from the vertebral body, bulging of the posterior wall of the vertebral body*

Figure 2в. *CT, axial slice. Destruction in the vertebral body and pedicles, as well as the posterior wall of the vertebral body*

Fugure 2. *Compression pathological fracture of the Th 10 vertebral body in a patient with focal myeloma (Observation by I.A. Borshenko, V.V. Lyalina)*

Abbreviation: CT — computed tomography, MRI — magnetic resonance imaging, T1 WI — T1 weighted imaging, T2 WI — T2 weighted imaging

X-ray generally has low sensitivity detecting no more than 60 % of myeloma lesions [7]. X-ray results suggest MM only if there are multiple "stamped" lytic bone lesions. However, this signs also cannot be specific. In all other respects, X-ray imaging will confirm, but not differentiate, the presence of osteopenia, fracture, and/or multiple vertebral compression deformities.

On MRI, myeloma focus is visualized as a rounded area of low intensity T1-WI signal and high intensity signal on T2-WI with fat suppression. In diffuse MM form, a uniformly low signal from the affected bones on T1-WI and a uniform, slightly inhomogeneous increased signal on T2-WI is observed [8]. Moreover, MR-imaging provides detailed examination of the condition of arches, transverse and spinous processes that can also be involved in the myeloma process, as well as of the epidural space of spine that may include epidural soft tissue component leading to the compression of spinal cord and its roots.

One of the MM types is solitary vertebral plasmacytoma that is defined on MRI as a typical "mini brain" appearance. [9].

However, one should understand that the differential diagnosis of an OP fracture and a MM-related fracture can be difficult in the case of acute vertebral fracture. First of all, this is due to the fact that the appearance of the damaged vertebra is non-specific and is mainly represented by deformity and pronounced bone edema. In some cases, MM may be suspected based on such typical changes as diffuse focal lesions of other vertebrae or the presence of epidural component. However, in the case of a diffuse osteopenic type, the MRI presentation will be low-informative, and in the case of an acute fracture due to a solitary plasmacytoma, differential diagnosis based on MRI results will be impossible. The final diagnosis of MM is based on the results of biopsy and laboratory tests. An example of a pathological fracture in the presence of focal myeloma obtained with the help of imaging techniques is shown in Figure 2.

Vertebral metastatic lesion

The most osteotropic types are breast, prostate and lung cancers, as well as kidney, adrenal, thyroid and ovarian cancer [10]. The presence of metastases is often complicated by a compression vertebral fracture. The most typical localizations of metastases in spine are the lower thoracic and upper lumbar regions; fractures most often occur in these regions, as it happens in osteoporosis [11]. Metastases can be divided into osteolytic, osteoblastic and mixed [10].

X-ray cannot reveal small lytic lesions, as well as does not provide adequately detailed visualization of the structures of spinal canal. Damage to the posterior parts of vertebra, including pedicles, is typical ("missing pedicle", or "winking owl sign" that is assessed on the frontal image); this fact can be useful in several cases for the differential diagnosis of fractures. However, it should be kept in mind that this symptom is non-specific [9].

CT presentation depends on the degree of metastasis mineralization. Lytic metastases (the most common form) appear as a lesion of hypointense signal with uneven contours. Destruction of the posterior cortical plate and asymmetric insertion of plus-tissue into the spinal canal are typical signs. Sclerotic metastases look like an area of hyperintense signal and, as a rule, do not spread beyond the vertebra. Typical features also include impaired trabecular structure of the vertebral body, the presence of destruction foci in the spongy substance, as well as in anterior and posterior cortical plates where asymmetric fractures are developed, partial destruction of endplates, insignificant changes in the anteroposterior size of vertebral body [8].

MRI is the most high-sensitive method for detecting metastases (more than 90 %), including the early stage of metastatic process; it also allows detailed analyzing of the state of spinal canal. *Lytic* metastasis is characterized by a hypointense signal on T1-WI and hyper- or isointense signal on T2-WI; *osteoblastic* metastasis is hyperintense in T1- and T2-WI; *mixed* metastasis is hypointense on T1-WI and hypo- and/or hyperintense on T2-WI. Process spreading to vertebral posterior structures is also well visualized, as well as its paraspinal spreading [9].

The presentation of a metastasis-related vertebral fracture is non-specific. The metastatic origin of the fracture can be clearly defined by such typical signs as damage to the posterior parts of vertebra, spreading of plus-tissue, destruction of posterior cortical plate (that sometimes looks like a "bulge" into the spinal canal), damage to other vertebrae [10]. However, the differential diagnosis of an OP fracture and a metastatic fracture in the absence of these signs is difficult. A distinctive feature of a "benign" from a "malignant" fracture is a change in signal characteristics during dynamic MRI: in cases of a "benign" fracture, the signal returns to normal range in 1–3 months. However, this sign is not reliable, since bone marrow edema and the associated signal change in an OP fracture can persist for more than three months. Final diagnosis is established based on the results of morphological study. [12, 13].

An example of L1 metastatic lesion according to the results of imaging techniques is shown in Figure 3.

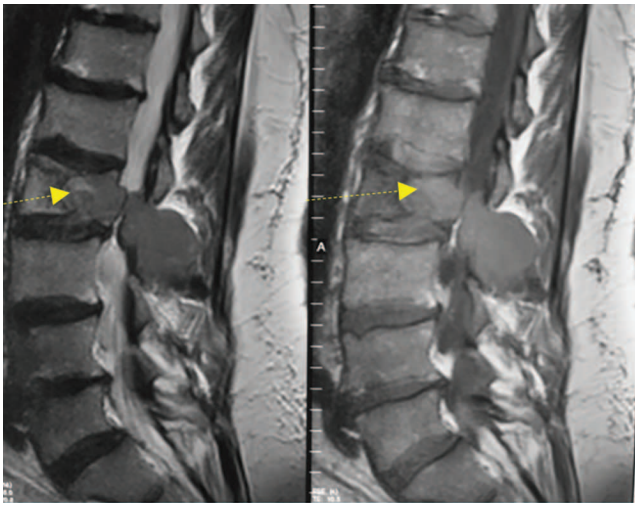
Treatment

Treatment of a patient with an acute OP fracture can last up to three months or more and involves drug and non-drug conservative treatment, and, in certain cases, surgical treatment as well.

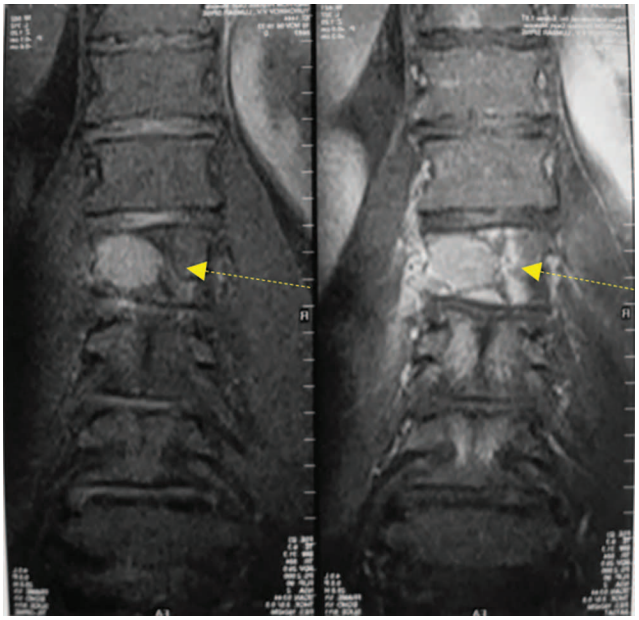
Non-drug treatment methods

Physical activity and wearing corsets

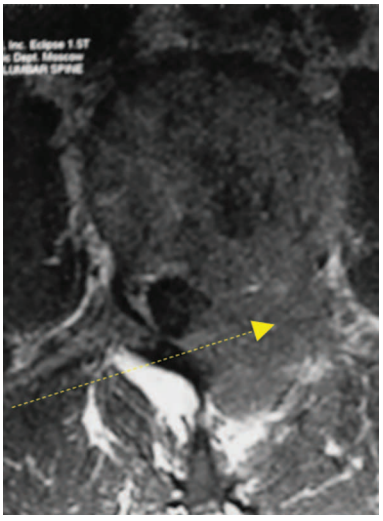
Patients are recommended to restart physical activity as soon as possible. Long-term bed rest is not



Picture 3а. *MRI, T2 WI (left), T1 WI (right) sagittal slice. Metastatic growth in the L1 vertebral body involves the left pedicle, expands into the spinal canal, compresses and displaces the dural sac, affects the posterior structures of the vertebra, arch and intervertebral joint and forms of a paravertebral mass in the spinal muscles*



Picture 3б. *MRI, T2 WI (left), T1 WI (right) frontal slice. There is a focal lesion of the left half of the L1 vertebral body with a expansion to the left side of the vertebral arch, into the lumen of the spinal canal and beyond the vertebra into the paravertebral tissues on the left*



Picture 3в. *MRI, T1 WI axial slice. There is a focal lesion of the left half of the L1 vertebral body with an expansion to the left side of the vertebral arch, into the lumen of the spinal canal and beyond the vertebra into the paravertebral tissues on the left*

Figure 3. *Metastatic lesion of the L1, MRI visualization, T1 WI, T2 WI (observation by Solomin V.D.)*
Abbreviation: CT — computed tomography, MRI — magnetic resonance imaging, T1 WI — T1 weighted imaging, T2 WI — T2 weighted imaging

recommended. Vertebral OP fracture significantly limits the overall physical activity of the patient; this fact results in the risk of pulmonary and thrombotic complications, contributes to the further loss of bone mass and muscle strength, and general detraining. In this regard, it is recommended, if possible, to start gymnastics literally from the first day of fracture using adequate anesthesia. The patients who have to temporarily stay in bed due to a fracture, on day 4–8, are recommended to turn from side to side with adequate anesthesia; then, if their condition allows, it is recommended to get out of bed with back support with a corset (corset should be put on in supine position) for short time (for 10 minutes up to 10 times a day). 3 weeks after fracture and for the next 10 weeks, patients should comply with the regimen of "intermittent rest in horizontal position": 2 hours in vertical position followed by 20 minutes in lying position [13, 14]. Physical exercises to improve balance and adequate strength training are recommended as prescribed by an exercise therapy physician.

Rigid/semi-rigid lumbar or thoracolumbar corset facilitates patient's verticalization, reduces pain severity by limiting the motion of the affected spine, and contributes to the early restart of physical activity [15]. However, many patients with previous pronounced deformity (kyphoscoliosis with torso shortening and decreased

costo-iliac distance) experience significant difficulties and discomfort from wearing a corset that diminish its therapeutic effect. An important negative aspect of the use of corsets is the development of muscle atrophy, so their use is recommended during the first three months after fracture, but not longer.

Drug treatment

Drug treatment includes pain relief and specific treatment for osteoporosis [5].

Treatment for osteoporosis

Primary osteoporosis is managed with bone resorption modulators (bisphosphonates, RANKL inhibitors receptor activator of nuclear factor-kappa B ligand), teriparatide), as well as vitamin D agents (cholecalciferol and alfacalcidol). Management of secondary osteoporosis also requires compensation for the underlying disease. If the fracture appeared during treatment with one or another bone resorption modulator, then a question should be raised whether it is reasonable to continue taking or replacing this agent.

Anesthesia

The choice of drug products depends primarily on the intensity and type of pain syndrome.

In most cases, pain syndrome is represented by vertebral and myotonic components. In this regard, nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen (paracetamol), muscle relaxants, and lidocaine patches are recommended as first-line agents for mild to moderate pain (Table 1) [16]. Drug pain relief is often not effective enough or is poorly tolerated. Given the wide variability in individual efficacy and tolerability, it is recommended to select NSAIDs and muscle relaxants using agents belonging to different chemical groups. If the pain is still not reduced within one to two weeks, then tramadol and/or calcitonin may be used, as well as a decision on surgical treatment should be made.

If myofascial trigger points were found, therapeutic blockades are recommended.

In the case of the prevalence of radicular pain, the development of radiculopathy signs and other neurological symptoms, an individual decision on further treatment strategy is recommended. [17]. Conservative pathogenetic treatment of radicular syndrome can

Table 1. *Drugs for pain relief in acute vertebral osteoporotic fracture*

Medications	Dosing	Side effects
Paracetamol	500-100 mg q4-8h (daily doses up to 3 g/day may be used)	Nephropathy, anemia, thrombocytopenia, hepatotoxicity, hypersensitivity, acute renal tubular necrosis
Non-steroidal anti-inflammatory drugs	Ibuprofen 200-800 mg q8h Naproxen, 200-500 mg q12h	Atrial fibrillation, bleeding, cardiovascular disease, edema, gastritis, gastrointestinal bleeding, heart failure, hypertension, kidney disease, gastric ulcer
Calcitonin	200 UI q24h intranasal 2-4 weeks. Alternate nostrils from one day to the next	Decreased appetite, dizziness, flashes, gastrointestinal disorders, headache, hypertension, hypocalcemia, rash, rhinitis, weight gain
Lidocaine patch 5 %	Stick on the affected area for 12 hours	Dermatitis, edema, exacerbation of pain, skin depigmentation, urticaria
Myorelaxants	Tolperisone — 50 mg q8-12h, then gradually increase the dose to 150 mg q8-12h	Dizziness, drowsiness, sedation, vomiting, dry mouth, constipation, headache
	The initial dose of tizanidine is 2 mg 3 times a day, then a gradual increase in the daily dose by 2-4 mg at intervals of 3-7 days to 12-24 mg/day, divided into 3-4 doses at regular intervals. Do not exceed 36 mg/day. Do not abruptly withdrawal	
Opioid analgesics	Tramadol — 50-200 mg/day Maximum daily dose — 400 mg	Dependence, confusion, drowsiness, dizziness, tachycardia, orthostatic hypotension, dry mouth, nausea, vomiting, increased sweating, miosis
Central analgesics	Nefopam Per os — 30-90 mg q8h; IM — 20 mg q6-8h; IV in NaCl 0,9 %– 20 mg q6-8h Maximum daily dose — 120 mg P.S. During the administration of the drug and within 15-20 minutes after the injection, the patient must be in the supine position	Dizziness, drowsiness, sleep disturbances, nervousness, thinking disturbances, a feeling of a veil over vision, nausea, vomiting, dry mouth, increased sweating, tachycardia, urinary retention. In rare cases — euphoria, hallucinations, convulsions
	Flupirtine* 200-600 mg/day. Maximum daily dose — 600 mg.	Dizziness, heartburn, nausea, vomiting, constipation or diarrhea, flatulence, abdominal pain, dryness of the oral mucosa, anorexia, depression, sleep disturbances, sweating, anxiety, nervousness, tremors, headache

Note: * currently not available in Russia

be used that includes neurotropic agents (lipoic acid, B vitamins, medications that improve microcirculation), or agents aimed at reducing neuropathic pain can be administered (gabapentin, pregabalin, etc.). In cases with the development of compressive radiculopathy — it often develops when a compression fracture is combined with degenerative spinal stenosis — injection of epidural steroid to the compressed root under X-ray monitoring can be used to relieve pain.

For patients with pronounced radiculopathy, inadequate effectiveness of conservative treatment, decompensated spinal stenosis, or developed neurogenic intermittent claudication syndrome, a decision should be made in regard to the microsurgical expansion of spinal canal (microdecompression) [18].

Signs of cauda equina syndrome are always an indication for urgent decompression of the nerve structures of cauda equina, since prolonged compression of these nerve structures leads to irreversible neurological deficit, especially — to the dysfunction of pelvic organs [19].

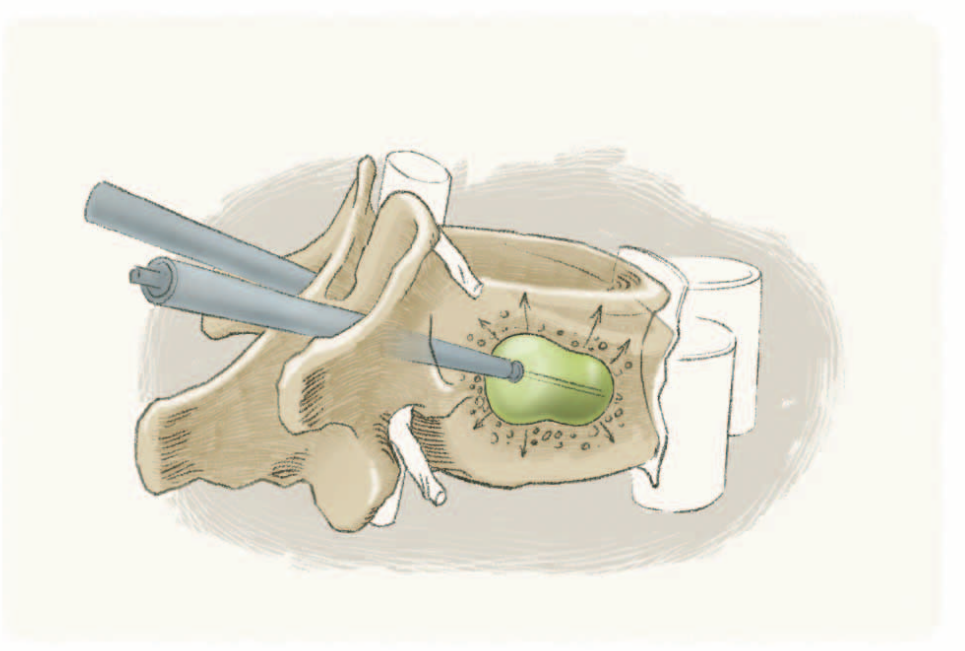
Pain facet syndrome is observed quite often in elderly patients, in particular, in the presence of osteoporosis [20]. However, the clinical presentation of acute vertebral fracture is usually stronger, and treatment for facet arthropathy is usually started several months after the compression fracture was healed. In such cases, blockades of the posterior medial branch of spinal nerve that innervates these joints are widely used, often combining local anesthesia with topical steroids [20]. If such blockades are effective, the technique of radiofrequency or endoscopic denervation of facet joints can be applied that allows achieving long-term pain relief.

Surgical management of acute vertebral fracture

In cases of choosing a surgical technique, vertebroplasty or kyphoplasty is commonly used. The purpose of these surgeries is to reduce pain and to correct or stabilize the shape of vertebrae.

In 1987, P. Geliber et al. described the management of a vertebral body tumor by injecting cement (polymethyl methacrylate) into the affected vertebra [21]. This procedure was called "vertebroplasty" and is actively used to manage various vertebral fractures (osteoporotic, traumatic, tumor). Vertebroplasty does not imply correction of vertebral shape; it only helps to "fix" the existing shape. During vertebroplasty (VP), methacrylate-based bone cement is injected into the spongy substance of vertebral body; it hardens within 10–15 minutes and prevents further vertebral deformation [22].

Afterwards, balloon kyphoplasty (KP) was developed: one or two balloons are inserted into a broken vertebral body [23]. When these balloons are inflated, endplates are moved apart, thereby reducing the kyphotic deformation of vertebra (that is, a kind of "straightening" and restoration of vertebral shape is achieved); a cavity in vertebral body is also made for the following injection of cement. KP mechanism is presented in Figure 4. Both surgeries (both VP and CP) are performed under the monitoring using image intensifier. As a rule, 5–7 ml of cement is injected into the ventral and central part of vertebral body. It should not be filled "tightly", as this does not always correlate with the analgesic effect, however, increases the risk of cement leakage.

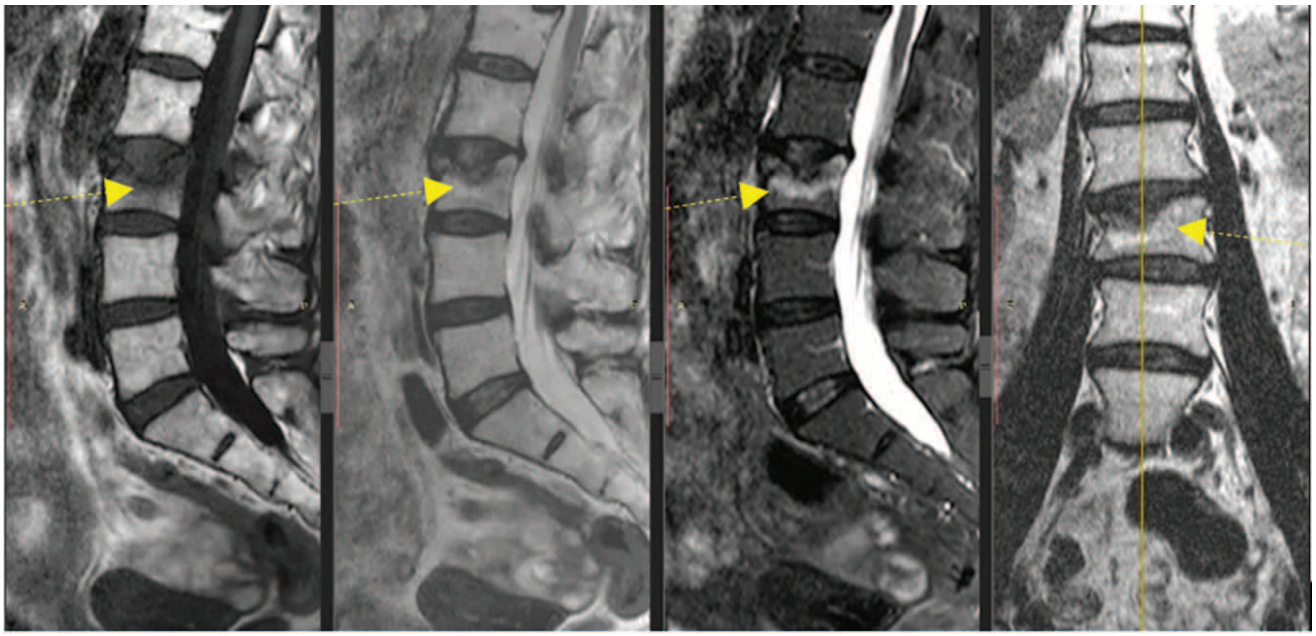


Picture 4. *Balloon kyphoplasty. Illustrator A.K. Rudykh*
Comments: the process of inflating a balloon in a fractured vertebral body to detect a cemented formation in the form of a cavity

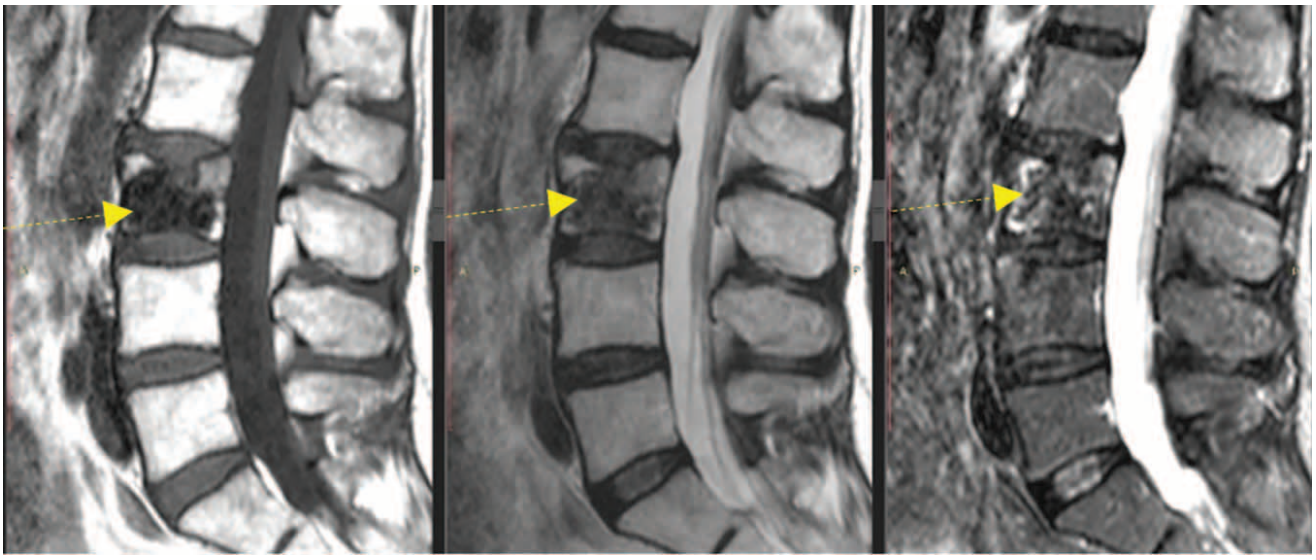
The advantage of KP is the lower risk of cement leakage due to the preliminary making a cavity in vertebral body. However, KP requires a fairly wide tunnel in vertebral pedicle (5–6 mm) for balloon placement that can cause technical difficulties for the surgeon, especially in cases of vertebral compression-comminuted fracture, causing iatrogenic displacement. Thus, a comminuted fracture of vertebral body and thin pedicles make this surgery difficult, so, in such cases VP is preferable [2] {Борщенко, 2014, Практика спинальной хирургии в условиях частной клиники}. The results of surgical treatment are shown in Figure 5.

VP/KP surgery with the strengthening of one vertebra lasts about 30–40 minutes. If cement is injected into two or more vertebrae, the duration of intervention increases. Most often, one or two vertebrae are treated.

Basic anesthetic technique for this intervention is local anesthesia. Potentiation in the form of ataralgesia



Picture 5a. *MRI of the lumbar spine. Hypointensive signal in T1 WI, areas of increased signal intensity in T2 WI. Significant increase in signal intensity in STIR mode as a manifestation of acute bone edema. Schmorl's hernia formation in the body of the L3 vertebra and decreased signal intensity along the line of bone compression*



Picture 5б. *MRI of the lumbar spine, 3 years after the fracture and puncture vertebroplasty of the L3 vertebra. An irregularly shaped area with a low signal in all modes — bone cement. The height of the vertebral body and normal spinal axis are preserved. No increase in the size of Schmorl's hernia in the body of L3 vertebra*

Figure 5. *Acute vertebral compression fracture of the L3, results of the vertebroplastic*
Abbreviation: MRI — magnetic resonance imaging, STIR — Short tau inversion recovery, T1 WI — T1 weighted imaging, T2 WI — T2 weighted imaging

is possible. Anesthesia with intradural administration of fentanyl in lumbar spine proved to be effective. In this case, fentanyl selectively binds to the segmental opioid receptors of spinal cord causing high-quality pain relief without affecting the motor functions of lower extremities.

Since the cement hardens already in the operating room 10–15 minutes after mixing and injection into vertebra, the patient can be activated almost immediately after returning to the ward. The verticalization of patient usually starts 30–60 minutes after the injection of cement. At this moment, the patient often notes a significant regression in vertebral pain syndrome.

Surgical indications in the acute period of vertebral OP fracture

Indications for performing VP/KP in case of a vertebral fracture are determined by the time elapsed after fracture, pain syndrome severity, and the presence of signs of fracture consolidation.

The severity of a vertebral fracture is primarily due not only to the time that has passed since the alleged vertebral injury, but also to the degree of bone tissue restructuring. Acute fracture process is associated with crushing of the bone trabeculae of the spongy substance of vertebral body. This can be observed on high-quality X-ray or CT images. On MRI, this situation is visible as the signs of the bone edema of vertebral body (decreased signal intensity on T1-WI, increased — on T2-WI, increased signal in the fat suppression mode (STIR mode)). An additional MRI sign of an acute fracture may be the line of the actual fracture with no bone trabeculae that can be observed as a line of reduced signal on T1-WI and T2-WI. On CT and X-ray images, a step-like deformation of a cortical plate is the sign of an acute fracture; it indicates a short period after the fracture when this "step" had no enough time to smooth out and transform.

If on X-ray or CT images there are established bone trabeculae in the area of vertebral deformation, and there are no MRI signs of bone edema, then such a fracture is considered chronic or consolidated.

Thus, the indication for VP/KP is a combination of two signs: the presence of an acute vertebral fracture in combination with a pronounced pain syndrome that is resistant to conservative treatment within 1–3 weeks [24, 25].

Actually, the time after fracture is not a criterion for surgery, since with low bone metabolism and delayed fracture consolidation, signs of an acute fracture may remain on MRI or CT for 1–6 months. Therefore, even 4-6 months after an OP fracture, if the signs of vertebral bone edema in combination with pain persist, an effective VP/KP can be performed. Such surgeries are performed on average 1–3 months after the fracture. At the same time, later than 6 months after the fracture, as a rule, vertebral remodeling is carried out and the reasonability of this type of surgical treatment becomes doubtful [2].

High pain tolerance, good physical activity of patient, effective analgesics and other conservative methods of treatment may be a relative contraindication for VP/KP surgery.

The degree of vertebral anatomical deformity is also not a criterion for determining indications for VP/KP, since even the first degree of vertebral compression can result in an extremely pronounced pain syndrome, and vice versa, complete compression crushing (vertebra plana) may not be accompanied by significant pain. However, a first-degree compression fracture in the transition zone where the inactive thoracic and highly mobile lumbar spine meet (Th11-L2 vertebrae) has a risk of deformity progression with the transition to higher degrees of compression. In this case, the risk of spinal kyphotic deformity increases that can lead to the development of a hump and chronic pain syndrome associated with the overload of spinal muscular and ligamentous apparatus. Therefore, in the case of an acute compression Th11-L2 fracture, it is recommended to expand the indications for VP/KP to prevent such biomechanical complications, even in the case of moderate primary pain syndrome [26–28].

Indications for surgery in delayed period

A compression fracture leads to the compaction of bone tissue (crushing of bone trabeculae). This in combination with natural reparative processes results in the spontaneous healing of fracture within 4–6 months while maintaining vertebral deformity. Therefore, VP/KP is most likely not indicated later than 6 months after fracture. However, osteoporosis can slow down reparative processes, so fracture healing may not occur, and bone loss and vertebral lysis may continue. In such cases, strengthening the vertebra with cement and performing VP/KP even later than 6 months after fracture can have a positive effect, i.e. significant decrease in pain and increased motor activity [29, 30].

The condition of vertebra is assessed by MRI and clinical signs. Signs of the process of crushing bone tissue, i.e. of the *ongoing fracture,* are decreased signal intensity from vertebral body on a T1-weighted image and increased signal intensity on a T2-weighted image. When drawing up a treatment plan, one should also keep in mind such signs of process severity as pain in the spinous process of damaged vertebra on palpation or percussion. The decision on the reasonability of VP/KP is made considering all the above factors [2].

Moreover, other surgical interventions are used in certain situations: neurological deficit (nerve roots compression), spinal instability according to the imaging, progression of kyphosis.

Conclusion

Differential diagnosis of an acute OP fracture based on clinical data is very difficult due to its non-specific signs and requires the use of medical imaging techniques, including CT and MRI. The treatment for acute OP fracture involves conservative and surgical methods that should be chosen in each individual case based on the results of thorough examination and ongoing monitoring of the patient.

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

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