Vertebral Osteonecrosis and Percutaneous Vertebroplasty

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Vertebral osteonecrosis is characterized by the presence of the intravertebral vacuum phenomenon. It is a relatively uncommon disease and although it may be caused by different pathologies, the most frequent cause is posttraumatic. The explanation for the presence of intravertebral gas is not known completely. We present the case of a 74-year-old patient who after suffering a vertebral traumatism, to complain of intense vertebral pain. A simple radiological study, CT scan, and magnetic resonance confirmed the presence of intravertebral vacuum phenomenon. We studied this radiological sign and then commented on its evolution after percutaneous vertebroplasty.

Key words: Osteoporotic fractures. Vertebral osteonecrosis. Percutaneous vertebroplasty.

Palabras clave: Fracturas osteoporóticas. Osteonecrosis vertebral. Vertebroplastia percutánea.

Introduction

The empty intravertebral space phenomenon (EIVS) is a very rare radiologic sign and, although it is not pathognomonic, it is the most relevant characteristic of vertebral osteonecrosis.1 It wasn’t until 1979 when Maldague et al2 associated this presence of gas with ischemia of the vertebral body. In spite of the publication of some cases of osteonecrosis without vertebral body collapse,3 in the majority of cases this phenomenon is localized to a fractured and collapsed vertebra, especially in older patients and who had previous osteoporosis, although the exact mechanism of intravertebral gas formation is unknown. The purpose of this article is to present the case of a woman with a traumatic vertebral fracture who developed EIVS and to review the literature related to this, with a final comment on the case progression after percutaneous vertebroplasty.

Clinical Case

A 74-year-old woman with a history of hypertension treated with atenolol 100 mg/day and depression treated with venlafaxin 75 mg/day, was diagnosed with osteoporosis 10 years prior had suffering a bilateral Colles fracture 4 years prior and had several posttraumatic vertebral fractures (D9, L2, and L3), receiving treatment with 2 g a day of strontium ranelate, 1000 mg a day of calcium, and 400 U a day of vitamin D. Her general conditions were acceptable but in November 2006 she suffered a fall on her back that led to a loss of movement due to intense pain in the dorsolumbar spine. She was admitted to the emergency room of her hospital of reference where laboratory analysis and vertebral x-rays were performed, being diagnosed with recent and past vertebral fracture and was discharged with the recommendation of rest and
analgesics (paracetamol 1g/8 h, tramadol 150 mg/12 h, and ibuprophen 600 mg/12 h). Treatment was switched from strontium ranelate to subcutaneous teriparatide and after 1 month was hospitalized for a complete laboratory and radiologic study because the intense dorsolumbar pain, severe limitation for mobility, and gait. Blood analysis showed: urea, 61 (normal, 10-50) mg/dL; uric acid, 7.5 (2.4-6) mg/dL; cholesterol, 260 (150-220) mg/dL; ALT, 37 (5-31) U/L; ferritin, 311.5 (30-300) ng/mL; CRP, 8.6 (0.01-6) mg/L; and intact PTH, 2.2 (1.6-6.9) pmol/L. The rest of the analysis, hemogram, ESR, proteins, thyroid hormone tests, phosphorus, and calcium 24-hour determinations, CEA, vitamin D3, and telopeptides were normal. During 1 week and in this order, several imaging tests were performed: simple x-ray of the dorsolumbar spine: vertebral fracture-collapse on D9, D11, L1, L2, and L3. Computed tomography (CT) of the dorsal column: fracture-collapse of D12 with EIVS indicative of osteonecrosis (Figure 1), vertebral collapse of D9, D11, L2, and L3. ⁹⁹m⁸⁹Tc bone scan: intense osteogenic reaction of D12 vertebral body. Magnetic resonance (MR) of the dorsolumbar spine: past osteoporotic fractures in a moderate degree of D9, L1, and severe on D11 and L3. D12 osteoporosis fracture in a moderate degree, acute-subacute progression, with diffuse reduction in signal on T1 sequences and a diffuse increase of the signal on a potentiated T2 sequence with fat-suppression (STIR). Centrally, there was an image of EIVS in both sequences, secondary to intravertebral air and a liquid focus of intensity on STIR, very indicative of osteonecrosis (Figure 2). Due to the persistence of invalidating dorsolumbar pain, the case was referred to the department of radiology and rheumatology of IMAS Hospital del Mar in Barcelona and the patient was hospitalized, 2 months after the trauma, for a vertebroplasty of D12 that was performed through a right yuxtapedicular approach with 2-3 mL of methylmetacrilate and also on D11 vertebral body that presented an older collapse, but with a certain degree of posterior edema which also suggested vertebral necrosis, also through a right yuxtapedicular approach and with 2 mL of methylmetacrilate. The tolerance to the procedure was adequate and the response to pain (previous VAS, 8/10; VAS after vertebroplasty, 3/10). One month after the intervention the patient continued with a certain limitation of dorsolumbar mobility and the only treatment she was receiving was subcutaneous teriparatide, 2000 mg a day of calcium, 800 U of vitamin D, no analgesics, and had a VAS of 2/10. Figure 3 shows a simple x-ray of the dorsal spine, 6 weeks after the vertebroplasty.

**Discussion**

EIVS reveals the presence of gas within the bone. It is usually composed of nitrogen in 95% and, in a lesser quantity, oxygen and nitrogen dioxide. Since the description by Maldague et al.,² it is accepted that the physiopathology of EIVS is due to vertebral osteonecrosis, relatively frequent in older patients with osteoporosis, in which there is inadequate vertebral vascularization, errors in the repair process and alterations in healing, although

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**Figure 1.** Computerized tomography image that shows an empty intervertebral space on D12.

**Figure 2.** Magnetic resonance image in a fat-suppression stage that shows an image of emptiness in the intervertebral space and occupying bony edema of D12.
the mechanism for the formation of this gas remains hypothetical. The frequent observation that disk gas adjacent to EIVS has led to the hypothesis that migrating gas from a contiguous disk through a fractured vertebral platform might be involved. It has also been proposed that it could be the result of vaporization and accumulation of the gas in a rift which is dissolved into the tissues that envelop the vertebrae, or, rarer still and not related to the case we presented, the production of intravertebral gas could be related to osteomyelitis produced by gas-forming bacteria.

EIVS can be observed on an anteroposterior and lateral x-ray of the spine in the form of a linear image or a half-moon, transparent and which curiously, can intensify upon extension of the spine or disappear upon flexion. CT imaging of the gaseous collection shows a more homogeneous and irregular form than x-rays. In the case presented, the initial dorsal column x-ray did not lead to the suspicion of osteonecrosis because EIVS was not seen, but it was detected with CT 1 month after trauma, maybe due to the fact that the intrasosseous hematoma, which results from acute vertebral collapse, occupied the spaces between the bone fragments and impeded the collection of gas in these areas. Both the bone scan, in which a non-specific increase in uptake of the affected vertebra, as well as MR helped confirm its localization. With RM in T1 sequence, there was an image of low intensity with the density of air, but T2 revealed that this intensity was time dependent and the position of the patient: during the first 10 minutes from a supine position, images tend to be of low intensity, between 20 and 40 min the intensity increases markedly because the fluid slowly enters the rift. These changes in signal indicate that we are faced with an acute process that is susceptible to treatment with a vertebroplasty.

Treatment of vertebral osteonecrosis is the same as the one for osteoporotic vertebral fracture, rest, analgesia and immobilization, and if after 4 to 6 weeks there is persisting pain, and after ruling out neurologic, muscular, or orthopedic complications, more aggressive treatments must be considered such as vertebroplasty or xiphoplasty. The patient we presented did not improve after a month of conservative treatment and, faced with the risk of a severe vertebral collapse, we opted for a percutaneous vertebroplasty. This technique consists of the percutaneous injection of polymethylmetacrilate inside the vertebral body of the osteonecrotic vertebrae, through a yuxtapedicular or posterolateral catheter approach, guided fluoroscopically or with CT. Once the treatment is finalized, the needles are removed while the patient remains recumbent for 1 or 2 hours and is then discharged. Pain relief can generally be appreciated between 2 and 24 hours after the intervention, as was the case with our patient. The majority of the series published to date are retrospective studies and coincide with a therapeutic functional response in the order of 90%, the number of treated vertebrae varies between 1 and 3 per patient and the rate if post-therapeutic complications, such as escaping cement, hemorrhage, or others is estimated in less than 10%. Prospective studies have demonstrated an improvement in spinal alignment of patients treated for xiphosis correction or for restoring the height of the vertebral spine. Some works have proven, however, a high degree of new fractures, that present between 40% and 50% on vertebrae adjacent to the vertebroplasty and occur earlier than other fractures, signaling to a possible mechanical conflict between the osteoporotic vertebrae and the cemented one.

One month after the intervention the patient still had some limitation but was not under analgesic treatment and showed a high degree of satisfaction.

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References