

## The vacuum cleft sign: an uncommon radiological sign

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**Abstract** The intravertebral vacuum cleft sign (VCS) is an uncommon radiological sign, characterized by a radiolucent zone in the vertebral body. It is composed of 95% nitrogen and small amounts of oxygen and carbon dioxide. Post-traumatic ischemic necrosis could be its physiopathological mechanism, along with other pathologies like osteoporosis, corticosteroid therapy, diabetes, arteriosclerosis, alcoholism, multiple myeloma, bone metastasis and osteomyelitis. The broad diagnosis is made by antero-posterior X-ray, but computed tomography scan (CT scan) and magnetic resonance imaging (MRI) may help with the differential diagnosis. The aims of this paper are, on one hand, to communicate the clinical case of a 73-year-old osteoporotic woman with traumatic vertebral fractures who developed this sign in her radiological survey. On the other hand, its secondary aims are to review the medical literature about this sign and to show the clinical and radiological evolution after a percutaneous vertebroplasty.

**Keywords** Intravertebral vacuum cleft sign · Osteoporotic fractures · Percutaneous vertebroplasty · Vertebral fractures

### Introduction

The vacuum cleft sign (VCS) is an uncommon radiological sign, characterized by an intravertebral trans-

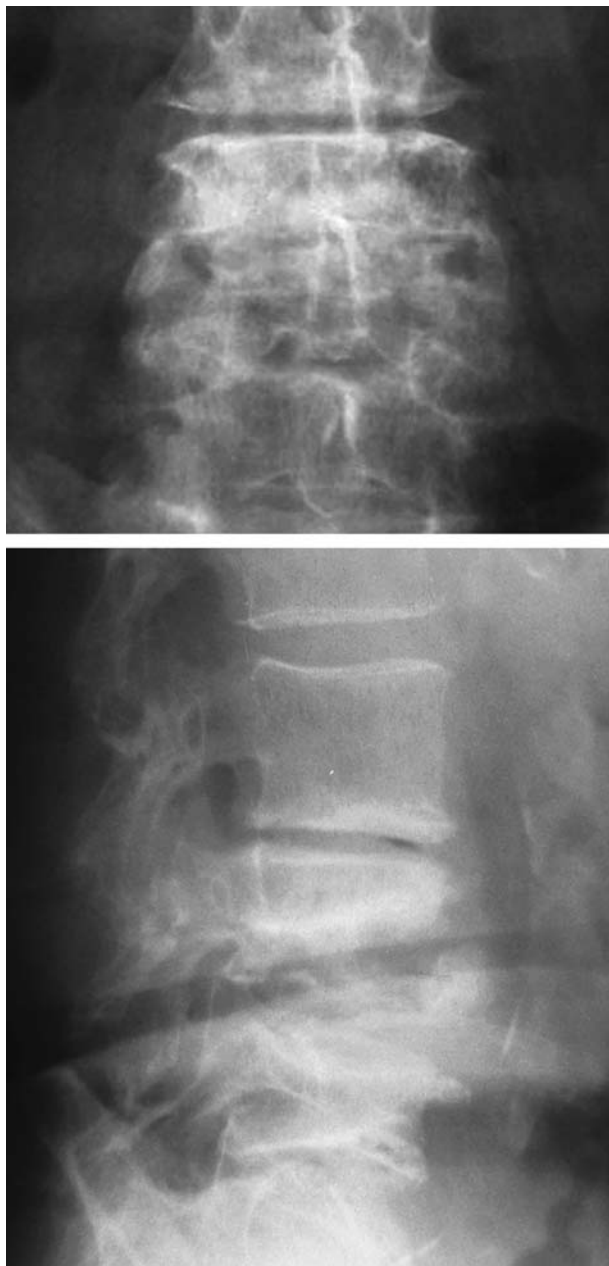
verse, linear or semilunar radiolucent shadow [1]. In 1891, Kümmel reported a delayed vertebral collapse that appeared from weeks to months after a traumatic, nutritional, vasomotor or neurological injury [2]. In 1971, Schmorl and Junghanns gave the anatomopathological support for this concept, but they could not determine its physiopathology. It was not until 1978 that Maldague [3] reported the association between the presence of gas within the vertebral body and ischemia. Since then, ischemia secondary to trauma is thought to be the physiopathological mechanism of this sign, leading to osteonecrosis and delayed vertebral collapse. The aim of this paper is to present a case report of a woman with osteoporotic traumatic vertebral fracture that developed the VCS in the radiological follow-up. In addition to this, we have done a review of the VCS medical literature to date, and we show the clinical and radiological progression after a percutaneous vertebroplasty.

### Case report

We present the case of a 73-year-old woman who was referred to us in November 1999 because of low backache. She had had it for 18 months after a post-traumatic vertebral collapse of the fourth lumbar vertebra because of trauma (fall of her own height). Her medical records showed diabetes type 2, hypertension, hypercholesterolemia and coronary artery disease. She had undergone percutaneous transluminal coronary angioplasty on two occasions. At the time of her visit, she was on atenolol 50 mg per day, simvastatin 10 mg per day, chlorpropamida 250 mg per day and isosorbide mononitrate 90 mg per day. She had risk factors for osteoporosis as follows: low calcium intake, low sunlight exposure and no physical activity. A complete radiological evaluation of the spine, bone mineral density of the lumbar spine and left hip, and bone metabolism evaluation were performed. Radiological evaluation showed vertebral collapse of the seventh dorsal, first

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**Fig. 1** Anteroposterior (top) and lateral (bottom) lumbar spine X-ray. In the lateral view, specially focused on the fourth lumbar vertebra, vertebral body collapse is seen

lumbar and fourth lumbar vertebra (Fig. 1), with reduced secondary trabeculae and osteopenia in the dorsal and lumbar spine. Bone mineral density (October 1998) showed a T-score of  $-2.6$  in the fourth lumbar vertebra and a T-score of  $-2.6$  in the left hip.

The laboratory tests are shown in Table 1. In laboratory tests, the electrophoretic proteinogram, blood count and serum creatinine value were normal, excluding therefore a diagnosis of multiple myeloma and renal insufficiency as secondary causes of osteoporosis. A diagnosis of postmenopausal osteoporosis was made, and treatment with alendronate (first 10 mg daily, and

later 70 mg weekly) together with 1,000 mg per day of calcium and 400 UI per day of vitamin D2 was initiated. Generally, treatment was well-tolerated, but it was discontinued several times because of gastric acidity.

The densitometric follow-up under treatment is shown in Table 2. In spite of the treatment, the densitometric benefit was low, and in August 2001 a new vertebral collapse (the fifth lumbar vertebra) was detected. In 2001, the VCS in the fifth lumbar vertebra was suspected in an X-ray evaluation. Because of this, complementary diagnostic studies were performed. An intravertebral (the fifth lumbar vertebra) heterogeneous gas collection was detected on CT scan, confirming the VCS. Vertebral body collapse (the fourth lumbar vertebra) resulted in an overall decrease in the volume of bone with associated intraosseous cleft formation (Figs. 2 and 3).

On magnetic resonance imaging (MRI) and on T1-weighted imaging, a low intensity area was observed inside the collapsed vertebral body of the fourth lumbar vertebra (Fig. 4), while on a T2-weighted imaging and on a fat-suppressed sequence, the signal intensity was markedly high. Because of the back pain and the radiological follow-up of the patient, a CT-guided biopsy of the fourth lumbar vertebra was done to rule out infection and/or a neoplastic process (Fig. 5). The material was sent to be cultured and to anatomopathological study. The results ruled out infection (osteomyelitis) and malignancy (metastasis), respectively. With the aim to restrict back pain and improve low-back mobility, a percutaneous vertebroplasty of the fourth lumbar vertebra was done, with significant clinical improvement (Fig. 6).

## Discussion

This radiological phenomenon reveals bone gas formation [4]. It is composed of 95% nitrogen and small amounts of oxygen and carbon dioxide. Radiolucent linear shadows are the radiological expression of the anatomopathological subchondral necrotic bone fracture. This could be perceived at various sites, for example, the humeral and femoral epiphyses [1]. The VCS was studied by some authors who demonstrated the presence of empty osteocytic lagoons within the bone trabeculae in the histological assay [3, 5].

There is evidence that osteonecrosis could be the mechanism of the production of gas, leading to a delayed post-traumatic vertebral collapse. Primary vertebral osteonecrosis is uncommon, and its incidence is unknown. Ischemic bone necrosis, on the other hand, is shared by all pathologies that develop VCS: osteoporosis, long-term corticosteroid therapy, diabetes, arteriosclerosis, alcoholism, multiple myeloma, metastatic bone disease, acute trauma and osteomyelitis [2, 3, 4].

Ischemia takes place in the anterior segment of the vertebral body, which is supplied by the anterior peripheral and metaphyseal arteries. Vascular insult

**Table 1** Initial and follow-up biochemical values, with alendronate, calcium and vitamin D treatment. 25(OH)-D3 = 25(OH)-vitamin D

Measured value	References values	25 November 1999	24 July 2001	09 November 2001	25 May 2002
Serum calcium	2.2–2.625 mmol/l	2.45	2.3	2.325	2.4
Serum phosphate	1.45–1.68 mmol/l	1.23	1.32	1.13	1.23
25(OH)-D3	0.048–0.084 pmol/l	0.042	0.064	0.074	0.047
Urinary calcium 24 h	4–5.5 mmol/d	4.35	7.15	–	–
Fasting calcium/creatinine	>0.10 mg Ca/mg creat	0.29	0.24	0.25	0.23
Fasting dipyrindamol/creatinine	3.0–8 µmol dipyr/µmol creat	7.8	5.2	5.1	4.8

**Table 2** Densitometric values of lumbar spine and left femoral hip of the patient. *BMD* bone mineral density

Date	BMD L1-L3 (g/cm <sup>2</sup> )	T score L2-L3	BMD femoral hip (g/cm <sup>2</sup> )	T-score femoral hip
10/98	0.840	–1.6	0.680	–2.6
4/00	0.789	–2.4	0.617	–3.0
7/01	0.783	–2.3	0.602	–3.1
7/02	0.837	–1.7	0.618	–3.0
7/03	0.858	–1.5	0.671	–2.6

**Fig. 2** Sagittal and coronal lumbar spine CT scan. Intravertebral vacuum cleft sign at the fifth lumbar spine level (*point*) and intervertebral vacuum cleft sign between the third and fourth lumbar spine vertebrae (*asterisk*) are shown

leads to vertebral collapse, and because of this, to an insufficient revascularization and bone fracture healing process. This produces a vicious circle with further ischemia and vertebral collapse progression.

Diminished bone volume—with gas accumulation in a low-pressure area—is produced in the vertebral collapse [1]. A prolonged supine position could lead to a fluid replacement of the cleft. In the case we are presenting, gas accumulation was observed inside the fifth lumbar vertebra, but not in the fourth, which contained a fluid collection. Independently, VCS could be detected in young adults or in the elderly; the trauma and VCS formation could take days to years to disappear. The VCS persisted as time passed.

It is mainly seen in the low thoracic and superior lumbar levels; nevertheless, cases also have been

described from the fifth thoracic to the third lumbar vertebra, and a rare case in the fifth lumbar vertebra [5]. It could affect one or, rarely, more than one vertebra. In acute vertebral collapse, VCS could not be seen since an intraosseous hematoma covered the cleft between the bone fragments.

Rarely, intravertebral gas production is associated with osteomyelitis caused by gas-forming bacteria. This is an important VCS differential diagnostic factor. An infection-related gas formation is produced under high-pressure areas, presenting a small and non-linear bubble-like appearance, and in the context of a soft tissue inflammatory process surrounding it [2]. Besides, the typical VCS is seen as a radiolucent, linear and transverse shadow. Image support led us to exclude infection in our patient, which was confirmed by vertebral biopsy and posterior bacteriological cultures.

VCS is observed in frontal plain X-ray films of the spine. Images in the lateral view allow the identification of both an intra- or intervertebral lesion and the putting out of intestinal gas interference over the radiolucent zone. A maneuver such as stress extension in the lateral view of the spine provides the best position to detect VCS. The intravertebral vacuum phenomenon is seen with extension stress views or the supine position, and disappears when the cleft is closed, as with flexion or the standing position [2]. When repeated stress views were obtained sequentially after supine positioning of the patient, radiolucency of the cleft decreased and disappeared. Because of this, in suspected cases, it is recommended to keep the patient in a standing or sitting position at least 1 h before the examination [2].

On CT scan, gas collection may appear less homogeneous and irregular than in plain radiographs [2]. In our case, the intravertebral VCS was visible in the fifth lumbar vertebra image, but not in the fourth one, where there was fluid collection. Also, the intervertebral vacuum cleft sign may be exposed between the third and fourth intervertebral spaces.



**Fig. 3** Sagittal and coronal lumbar spine CT scan. Intravertebral vacuum cleft sign at the fifth lumbar spine level (*point*) and intervertebral vacuum cleft sign between the third and fourth lumbar spine vertebrae (*asterisk*) are shown

On T1-weighted MRI image, a low intensity area within the collapsed vertebral body is seen, particularly inside the cleft. On T2-weighted images, the intensity depends on the time and position of the patient. Within the first 10 min after supine positioning, low-intensity images are seen, and are markedly high between 20 and 40 min after positioning, suggesting fluid content. Sequentially T2-weighted images at 9, 18 and 27 min after supine positioning demonstrate low intensity inside the cleft, mixed low and high intensity, and high intensity, respectively. This suggests the low replacement of gas by fluid [2]. In the case reported, we observed that the fourth lumbar vertebra showed a low intensity signal on the T1-weighted image and a high intensity signal on

the T2-weighted image and fat-suppressed sequence, suggesting fluid occupation of the cleft.

VCS may disappear as result of fracture healing months or even years later [2]. This natural progression was not observed in our patient, since she was treated with a percutaneous vertebroplasty. Percutaneous vertebroplasty is a procedure in which bone cement is injected into a collapsed vertebral body. Acrylic cement is introduced into the vertebral body via a CT-guided cannula inserted through the pedicle. It is used for the treatment of painful osteoporotic compression fractures and osteolytic lesions of the spine [6, 7]. It results in good pain relief, is safe and has a rate of complications of less than 10% [8, 9, 10]. Prospective studies have



**Fig. 4** On T1-(left), T2-(middle) and fat-(right) suppressed-sequence-weighted MRI image, the vacuum cleft sign is seen to be occupied with liquid



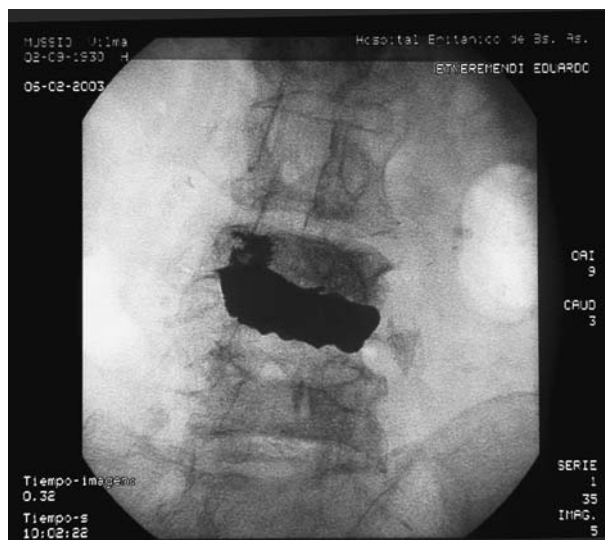
**Fig. 5** Axial view (fourth lumbar vertebra) CT scan guided-needle biopsy. Important vertebral body destruction and lagoon images full of liquid are observed

demonstrated significant improvement in mobility and function after treatment [8], as in our patient. Therefore, we introduced a clinical case in which VCS was evident late in the follow-up after trauma in a patient with osteoporosis. We reviewed the diagnostic criteria with various diagnostic imaging techniques, updated the physiopathologic knowledge and briefly discussed all advantages of percutaneous vertebroplasty in the treatment of vertebral fractures.

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**Fig. 6** Anteroposterior (top) and lateral (bottom) lumbar spine X-ray showing bone cement inside the fourth vertebral body

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