fresh osteoporotic fractures. The patient was admitted in April 2000 for percutaneous vertebroplasties. After local anaesthesia, the needle was placed transpedicularly into the vertebral bodies. Under fluoroscopic control the cement was injected without any complication in the vertebral bodies T10 and T11 (fig 1A). As the needle was pulled out of the vertebral body L1, the patient felt an intense pain in the left thigh. A computed tomographic (CT) scan performed within a few hours depicted a haematoma in the left psoas muscle. The patient did not feel any respiratory or thoracic discomfort and was discharged a few days later.

Because of persisting pain in the lower back the patient was referred to our clinic six months later. His history disclosed heavy smoking and a weight loss of 7 kg during the past five months. He denied any respiratory symptoms. Clinical examination showed a body weight of 46 kg and a height of 1.64 m. Cardiac and respiratory findings were normal. The gait was ataxic, with absent Achilles tendon reflexes and marked muscle wasting. The chest radiograph did not show any signs of a neoplastic disease, but a large number of fine radiodense lines with a branching pattern spreading throughout both lungs was seen (figs 1B and D).

DISCUSSION

Percutaneous vertebroplasty is a minimally invasive technique mainly for the treatment of vertebral fractures in osteoporosis and fractures due to spinal metastasis.[3–5] In up to 90% of cases, immediate pain relief is reported. The risk of cement extravasation into the venous system and the spinal canal represents the major hazard of this technique. The leakage of acrylic cement outside the vertebral body occurs in up to 65% but remains silent in most cases. The extravasation of cement into the inferior vena cava and subsequently into the lungs is rare. To date, only three cases of patients with pulmonary embolism caused by percutaneous vertebroplasty have been reported.[3 6] All the cases showed paravertebral venous opacity, the embolisation was documented by CT scan and two of the patients remained asymptomatic.[3] In addition, one case of lethal pulmonary embolism was reported after percutaneous vertebroplasty in a series of patients with spinal metastasis. It is important to note, however, that no cement was found in the pulmonary arteries.[7]

The risk of cement leakage depends on the vascular anatomy and fracture pattern, on the one hand, and technical aspects, on the other. The case presented here shows a typical fracture pattern without involvement of the posterior wall and offers no additional risks for this treatment. The viscosity of PMMA cement is a crucial aspect during the procedure. The occurrence of this diffuse, extensive lung embolisation is only possible when a considerable amount of cement is injected in a very low viscous state. The cases mentioned above showed only globular cement in major pulmonary vessels. Furthermore, sufficient radio-opacity of the cement is mandatory. In addition, the placement of the tip of the needle needs to be controlled by CT scan or fluoroscopy. As the performing radiologist did not detect dislocation of cement, we suspect a lesion of the basivertebral vein or a horizontal subarticular collecting vein draining into the vena cava inferior at the L1 level in our case. The value of prior vertebrography is controversial. Some authors recommend a venography to exclude needle placement directly within the basivertebral venous plexus. Others argue that the contrast media has different chemical and physical properties and nearly always escapes through the venous plexus. Our experience suggests that extravasation cannot be avoided by previous venography and, therefore, meticulous monitoring of the cement flow during the procedure is crucial.

This case illustrates for the first time diffuse pulmonary cement emboli as a complication of percutaneous vertebroplasty. It supports the notion that plugging a small percentage of arterial pulmonary vessels does not result in respiratory symptoms. However, whether the stiffness or the chemical properties of the cement, or both, may lead to secondary pulmonary lesions is unknown.

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No association between human parvovirus B19 infection and Sjögren's syndrome

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The association of human parvovirus B19 (HPVB19) infection with autoimmune disease, including systemic lupus erythematosus, rheumatoid arthritis, polymyositis, and vasculitis, has been suggested, although the exact relationship between the infection and these disorders has not been fully elucidated.[1 2] A recent report showed serological evidence of past B19 infection associated with the presence of cytopenia in patients with primary Sjögren's syndrome (SS).[3] To gain more information about the aetiopathogenetic role of HPVB19 for this disease, we evaluated the presence of the viral genome in minor salivary glands from patients with primary SS.

We studied 10 women with SS (mean (SD) age 45 (9) years) and 10 healthy controls matched for age (43 (6) years) and sex. SS was diagnosed according to European criteria.[4] Each subject taking part in the study underwent minor salivary gland 6 mm punch biopsy under local anaesthesia. Histological evaluation of biopsy samples was carried out according to Chisholm and Mason's classification.[5] They were also analysed for the presence of DNA sequence coding for the HPVB19 non-structural protein (NS1) amplified by nested polymerase chain reaction (PCR) as a marker of infection. The outer primer pairs were P1 and P6, corresponding to nucleotides 1399–1422 and 1682–1659. In the second amplification, the P2 and P5 inner nested primer pairs, corresponding to nucleotides 1498–1525 and 1660–1576, were used. The 103 base pair (bp) diagnostic fragment was subsequently detected by ethidium bromide staining after agarose gel electrophoresis. Each sample was tested in duplicate. A 10^{-9} dilution of a reference serum containing about 10–100 HPVB19 genome copies was used as positive control. Negative water controls were extracted concomitantly with the diagnostic specimens in order to monitor possible contamination during the extraction step. Additional negative controls were included in each PCR run to eliminate the possibility of carryover contamination. A 268 bp fragment of the β-globin gene was amplified using primers PC04 and GH20 as a test for the absence of Taq DNA polymerase inhibitors and to estimate the quantity of DNA extracted from each minor salivary gland. A serial 10-fold dilution of DNA extracted from a known number of Hep-2 cells was used as positive control. Negative controls were simultaneously extracted water samples. PCR products were then analysed by agarose gel electrophoresis.

Blood samples from each patient were tested for the presence of anti-B19 IgM and IgG using a commercially available enzyme linked immunosorbent assay (ELISA) (Pantec, Torino-Italy).

All minor salivary gland samples of patients with SS were rated as grade III or IV according to Chisholm-Mason's classification. In the control group, only three subjects were rated as grade I or II (subjects 13, 14, and 17, table 1).

The DNA sequence coding for NS1 of HPVB19 was found in a salivary gland specimen from one case of SS (patient 2) and from one control subject (subject 19). Both cases showed a high titre of anti-B19 IgG antibodies and the absence of specific IgM antibodies. In the patient with SS (patient 2) the presence of IgG B19 antibodies was associated with more than one focus score, whereas in the control subject (subject 19) the presence of DNAPVB19 was not associated with lymphocytic infiltrate. Anti-B19 IgG antibodies, but no anti-B19 IgM antibodies, were detected in three other subjects, including one with SS (patient 6) and two controls (subjects 12 and 17). The results of the study showed that the prevalence of past B19 infection in patients with primary SS was similar to that of the control group. Furthermore, none of the patients with SS showed serological markers of recent infection from HPVB19.

Table 1 Histological and serological features in patients with primary SS and controls. 1–10 are patients, 11–20 controls

Subjects	Lymphocytic infiltrate (grade)	IgG	IgM	DNAPVB19
Patients				
1	IV	–	–	–
2	III	+	–	+
3	IV	–	–	–
4	III	–	–	–
5	III	–	–	–
6	IV	+	–	–
7	III	–	–	–
8	IV	–	–	–
9	IV	–	–	–
10	IV	–	–	–
Controls				
11	Absent	–	–	–
12	Absent	+	–	–
13	II	–	–	–
14	I	–	–	–
15	Absent	–	–	–
16	Absent	–	–	–
17	I	+	–	–
18	Absent	–	–	–
19	Absent	+	–	+
20	Absent	–	–	–

B19DNA can also be found, and can persist, in the salivary glands without inevitably inducing a lymphocytic infiltrate in this tissue. Our results suggest that B19 constituents may also be found in salivary gland tissue. However, the presence of viral DNA in the salivary glands of patients with SS appears to be incidental, and it does not support an association between SS and HPVB19 infection.

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