Asymptomatic diffuse pulmonary embolism caused by acrylic cement: an unusual complication of percutaneous vertebroplasty

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Percutaneous vertebroplasty was first performed in 1984. Galibert *et al* treated a cervical vertebral angioma by percutaneous puncture and injection of polymethylmethacrylate (PMMA) cement into the vertebral body.[1] Shortly thereafter vertebroplasty was done also in lytic metastatic bone lesions.[2] Vertebroplasty for the treatment of vertebral fractures in osteoporosis has gained fast acceptance in the past two years. Its efficacy is documented in several clinical studies.[3 4] However, open questions about indication, technical aspects, and complications remain. We report the first case of extensive but clinically silent cement embolisation into the lungs.

CASE REPORT

A 67 year old man presented with upper abdominal pain. A chest radiograph suggested multiple vertebral fractures as a possible explanation. Magnetic resonance imaging confirmed

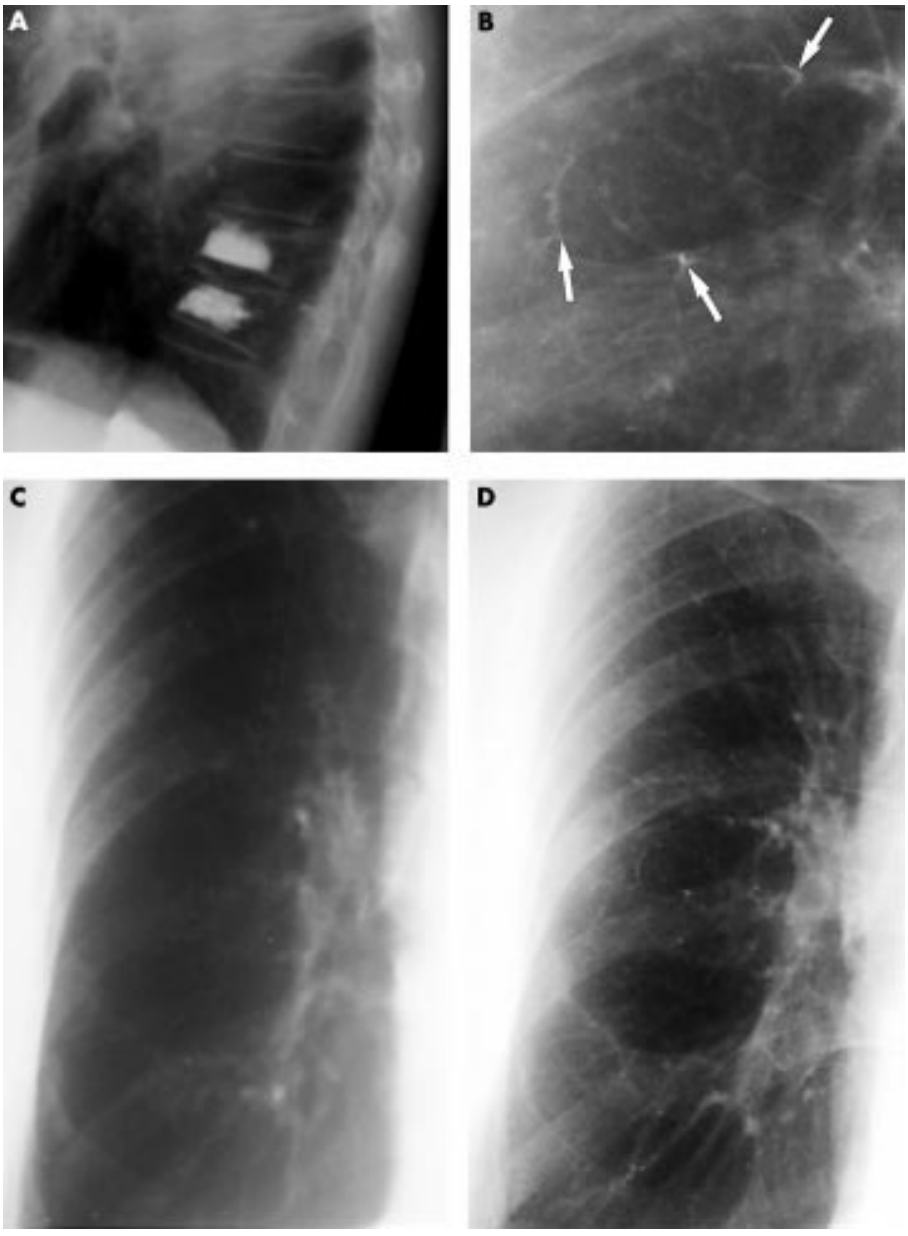

Figure 1 (A) Six months after vertebroplasties the chest radiographs show radiodense cement in the fractured vertebral bodies T10, T11, and L1. (C and D) Posteroanterior views of the right lung. The pulmonary arterial vessels look normal before the vertebroplasties (C). In contrast, multiple radiodense lines along the pulmonary vessels are detectable thereafter. (B) Shows a more detailed view of (D). Three of the cement emboli are marked with white arrows.

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.