

Case Report

Percutaneous Vertebroplasty for Pregnancy-Associated Osteoporotic Vertebral Compression Fractures

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Osteoporosis is a worldwide problem and it mainly affects postmenopausal women. Osteoporosis associated with pregnancy or lactation is a rare condition. The incidence and mechanism of this phenomenon has not been clarified, but it can cause one or more vertebral compression fractures with severe, prolonged back pain in the affected women. We experienced this uncommon case, treated it with percutaneous vertebroplasty. A 35-year-old woman visited our hospital with complaints of severe back pain and flank pain 2 months after normal vaginal delivery. She was diagnosed with osteoporotic vertebral compression fractures on the T5, 8, 9 and 11 vertebral bodies and we performed percutaneous vertebroplasty on the T8, 9 and 11 vertebrae with a good result. We present here an unusual case of pregnancy-associated compression fractures treated by percutaneous vertebroplasty.

KEY WORDS : Osteoporotic vertebral compression fracture · Percutaneous vertebroplasty · Pregnancy-associated osteoporosis.

INTRODUCTION

Osteoporosis is a common disease entity of the elderly and this especially occurs in postmenopausal women. Vertebral compression fracture (VCF) is one of the severe complications of osteoporosis. Pregnancy-associated osteoporosis is a very rare condition in which even minimal trauma can cause fracture. However, making the diagnosis is difficult due to the special conditions surrounding pregnancy, birth and breast feeding.

The possible causes have been reported to be the increased need for calcium and hereditary factors. There has recently been an emphasis on the role of parathyroid hormone related protein (PTHrP), but this has not yet been clarified. Most of the reported cases of VCF were treated with conservative maneuvers such as bed rest, medication or appliance of back braces. In one case, the patient suffered from osteoporosis VCF that occurred during light daily activity after delivery, and the pain was sustained after con-

servative treatments. Percutaneous vertebroplasty (PVP) was performed with good results.

CASE REPORT

A 35-year-old woman visited our hospital due to severe back pain and both flanks pain after a normal vaginal delivery. She was previously healthy without any other diseases, and she had given birth to her first baby two months prior to the visit. One month previously while holding the baby, she suffered from severe back pain. She then went to a local clinic for treatment, but the pain did not lessen and she was transferred to our hospital. The patient suffered from severe pain that she could not stand or sit, but she did not show any other neurologic deficits. On past medical history, she underwent a surgery due to ectopic pregnancy, and experienced an spontaneous abortion two years ago. When the woman was hospitalized, lactation was prohibited. She did not have any disease that could cause osteoporosis and she was not taking any medicine such as corticosteroid or thyroid hormone. She was a non-smoker and there was no positive family history for osteoporosis or any related risk factors. With 150 cm tall and 42 kg body weight, she was relatively thin, and her vital signs were stable.

Tenderness was noted on her back on the physical exami-

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nation, and there was no history of trauma. On the X-ray image, there was mild height loss of the T8, 9 and 11 vertebral bodies. On MRI, there were multiple VCFs seen on the T5, 8, 9 and 11 vertebrae. There were no signs of metabolic or infectious bone disease on the laboratory and radiologic findings. We performed PVP on the T8, 9 and 11 vertebrae to reduce her back pain. Tenderness was not noted at T5, so it was skipped. The cytology of bone marrow was taken during the PVP, and it was negative for malignancy. Shortly after the procedure, her symptoms were improved and she was comfortable in her daily living activities. Her VAS score was 8 pre-operatively, and it improved to 1 (Fig. 1, 2).

Complete blood count was in the normal ranges. Erythrocyte sedimentation rate was mildly increased to 43 mm/hr (normal range : 0-20 mm/hr). The renal function, thyroid function test were in the normal ranges. FSH, LH, estrogen and progesterone levels were also in the normal ranges, as were the serum total and ionized calcium level and the phosphorus levels. The renal secretion of calcium and phosphorus in the 24-hour urine were within the normal limits at 158.0 mg/day (30.0-200.0 mg/day) and 0.41 g/day (0.4-1.3 g/day), respectively. The parathyroid hormone and 25-OH vitamin D were in the normal ranges at 24.95 pg/mL (15.00-65.00) and 10.60 ng/mL (4.80-52.80) respectively. The biochemical bone markers are slightly increased. Alkaline phosphatase and deoxypyridinoline were slightly increased to 152 IU/L (25-129 IU/L), 17.77 nM/mMcr (3.00-7.40 nM/mMcr). Yet, osteocalcin was in normal range with

14.46 ng/mL (5.00-18.00 ng/mL). The bone mineral density was measured with a T-score of -5.45. We prescribed a daily intake of 1,500 mg of calcium carbonate and cholecalciferol at 400 IU and a weekly 35 mg dose of risedronate.

DISCUSSION

Pregnancy-associated osteoporosis is not a common phenomenon, but it can be accompanied by substantial bone loss, resulting in fracture by minimal trauma^{7,11}. We experienced one unusual case of osteoporotic VCFs that developed after normal delivery, and we performed PVP and obtained good result.

The causes of pregnancy-associated osteoporosis are not well known, but several hypotheses after evaluating the prevalence of fractures in 35 women who were diagnosed with pregnancy-associated osteoporosis. However, the cause of the prevalence of this malady remains unclear. A higher prevalence of fractures at an early age in the mothers of patients with pregnancy-associated osteoporosis has been observed, and this suggests a previous condition of a lower bone mass of a genetic origin.

A change in calcium metabolism may contribute to the cause of pregnancy-associated osteoporosis^{4,6,12}. Under the normal environment of pregnancy and lactation, the calcium demand increases and a relative deficiency of calcium can occur and so bone absorption increases. Eventually, bone demineralization may increase. However this explanation is not so attractive because over-activated bone absorption is

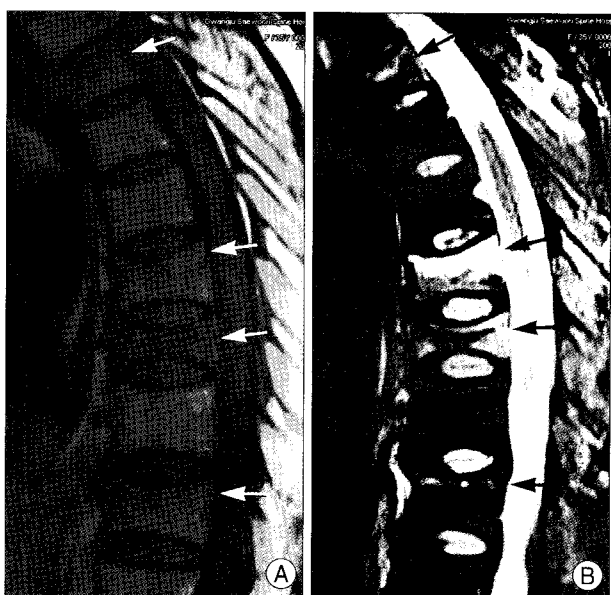


Fig. 1. Preoperative MRI findings showing the compression fractures on T4, 8, 9, 11. A : Sagittal T1-weighted MRI shows low signal intensity on T4, 8, 9, 11. B : Fat suppression T2-weighted MRI shows high signal intensity on T4, 8, 9, 11.

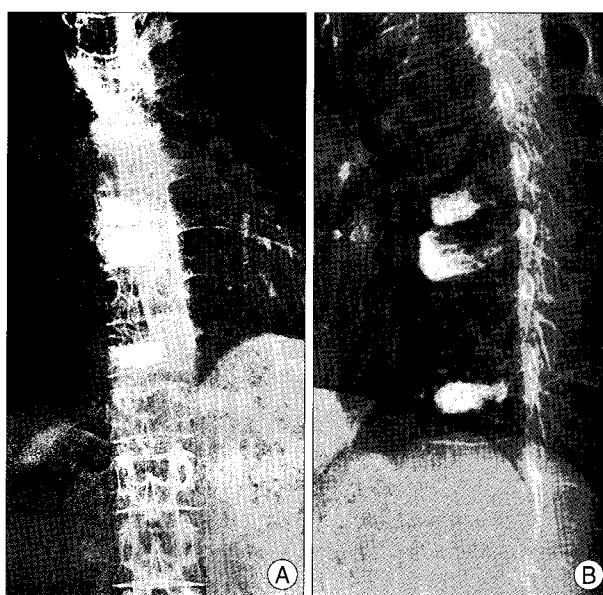


Fig. 2. Postoperative X-ray images showing the cement augmentation on T8, 9, 11. A : Anteroposterior radiograph demonstrate the central filling of cement. B : Lateral radiograph demonstrate the increased anterior body height.

regulated by a normal buffering system, so theoretically it is not feasible to be osteoporotic only through this explanation.

A calcium homeostasis is maintained by regulating intestinal calcium absorption, renal calcium excretion and skeletal calcium release^{5,9}. It is known that intestinal calcium absorption and renal excretion rates increase during pregnancy. Enhanced maternal bone resorption, however, is reported to play a major role in compensation for calcium loss during lactation⁴. Calcium requirements increased during pregnancy and lactation. A total calcium storage in term fetuses is average 21 g (range 13-33). About eighty percent of calcium requirement is used at the third trimester of pregnancy, when a rapid mineralization of the fetal bone occurred. During lactation, maternal calcium loss is 280-400 mg/day (up to 1,000 mg/day), four times higher than during pregnancy¹³.

It is known that a decrease in serum calcium of as little as 0.5 mg/100 mL is sufficient to double serum PTH. So, a decrease in the level of ionized serum calcium would be the simplest explanation of maternal hyperparathyroidism since this represents an established stimulus of PTH secretion. However, there is no correlation between the serum levels of calcium and PTH during pregnancy, and the depression of serum calcium is associated with the explanation of plasma calcium and the lowering of serum proteins. For these reasons, relative hypocalcemia is not an adequate explanation for maternal hyperparathyroidism. The specific signal for the hypersecretion of PTH has not been identified during pregnancy.

Estrogen and PTH have generally opposite effects on bone metabolism and levels of estrogenic hormones are, of course, greatly increased during pregnancy. Thus, estrogen limits the osteolytic action of PTH while permitting its action on the gut and kidney to be continued, providing the extra calcium needed in gestation. Even though the effects of progesterone are still controversial, it might inhibit bone resorption and prevent postmenopausal bone loss.

Other studies have focused on PTHrP. Reid et al.¹⁰ suggest PTHrP is deeply associated with the development of pregnancy-associated osteoporosis. During lactation, the regulation of bone loss probably involves several factors: calcium lost in breast milk, the hypoestrogenic state and the increased levels of PTHrP that are in circulation^{1,10}. The bone absorption increases with the decreased bone density. After the end of lactation, the PTHrP levels are normalized, and the bone density is restored. The PTHrP is suggested to be calcium mobilizing factors that are related to pregnancy-associated osteoporosis. Unfortunately, we did not check the PTHrP in our case.

For the treatment of pregnancy-associated VCFs, most

reports have administered conservative treatments like bed rest, analgesics, corset application and medical treatment. Bayram et al.² performed kyphoplasty on pregnancy-associated VCF, and they got good results. Surgical treatment has the advantages of early ambulation and dissolution of back pain over conservative treatment. We think that PVP is a minimally invasive and effective procedure for the VCF, and this can be applied to pregnancy-associated VCFs⁸.

Despite a conventional use of polymethylmethacrylate as bone cement, calcium phosphate may be a good alternative of bone cement, which is known as a biocompatible material³. When a pregnant or lactating woman complain of severe back pain, the clinician should take into account of osteoporotic compression fracture. The X-ray, MRI and bone mineral density should also be examined.

Pregnancy-associated osteoporosis is not very common, but it can cause VCFs or hip joint fractures. The clinician can easily misdiagnose these cases, so attention must be paid to the possibility of pregnancy-associated osteoporotic VCFs. We think that PVP can be an effective and minimally invasive procedure for the treatment of the pregnancy-associated osteoporotic VCFs.

CONCLUSION

When a pregnant or lactating woman complain of back pain, we should take into account of pregnancy-associated osteoporosis not to make misdiagnosis. Pregnancy-associated osteoporosis is rare, but it may be accompanied by a substantial bone mineral loss, resulting in VCFs. We report an unusual case of pregnancy-associated compression fractures treated well by percutaneous vertebroplasty.

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