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Case Report

Pregnancy and lactation associated osteoporosis: unrecognized cause of musculoskeletal pain syndrome during the peri-pregnancy period

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ABSTRACT

Pregnancy- and lactation-associated osteoporosis (PLO) is a rare disorder, usually occurring in late pregnancy and the early post-partum period. The prevalence, etiology, pathogenesis and therapy remains unclear. Three clinical cases of PLO present patients with multiple severe osteoporotic fractures during the peri-pregnancy period and different treatment strategies.

Keywords: Fractures, Lactation, Osteoporosis, Pregnancy

INTRODUCTION

Pregnancy- and lactation-associated osteoporosis (PLO) is a rare syndrome characterized by significant changes in calcium and bone homeostasis during pregnancy and the early post-partum period.[1] At this time, calcium demands are increased to meet the needs of the growing fetus and the losses that occur during lactation.[1,2]

PLO leads to musculoskeletal pain syndrome, fragile bones and increased risk of fractures during late pregnancy and early post-partum period.[1,3] The three case reports present the clinical findings of young women who developed severe PLO with vertebral fractures during a peri-pregnancy period.

CASE REPORT

We came across a 34-year-old primiparous woman with severe lower back pain that worsened two months after a Cesarean delivery. Her height was 168.5 cm, body weight was 62 kg, and BMI was 21.7 kg/m^2. A CT scan exhibited multiple vertebral osteoporotic fractures of the thoracic (Th11, Th12) and lumbar (L1, L2) spine (Figure 1).

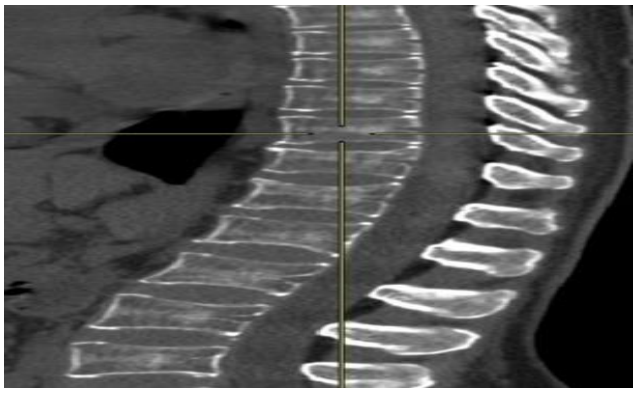

Figure 1: A CT scan of the thoracic and lumbar spine in case 1.

The lumbar spine bone mineral density (BMD) was measured using dual x-ray absorptiometry (DXA) and was below the expected range of age (–2.7 SD). The patient had serum 25-hydroxyvitamin D (25(OH)D)

deficiency (11.5 ng/mL). Biochemical markers of bone turnover were increased: osteocalcin level was 52.8 ng/ml, cross-linked C-terminal telopeptide of type I collagen (β CTX) level was 0.766 ng/mL. The laboratory assessments (including complete blood count, calcium, total blood cholesterol, low-density lipoprotein cholesterol, liver enzymes, alkaline phosphatase, creatinine, parathyroid hormone, protein electrophoresis) revealed no abnormality.

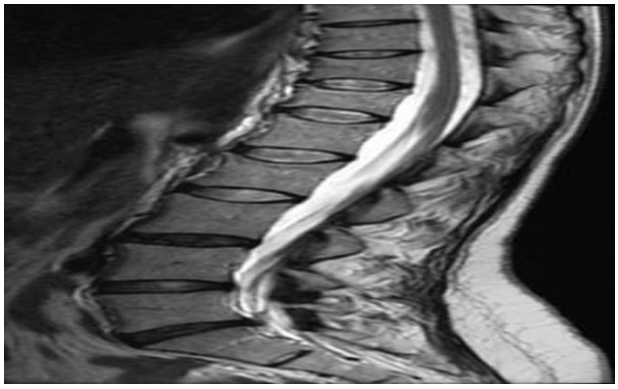

Figure 2: A MRI scan of the thoracic and lumbar spine in case 2.

We also encountered a 29-year-old patient with musculoskeletal pain syndrome began in the third trimester of pregnancy and deteriorated immediately post-partum. Her height was 162 cm, body weight was 57 kg, and BMI was 21.8 kg/m^2. MRI scan showed vertebral compressions of the thoracic (Th11, Th12) and lumbar (L1) spine, osteoporotic fracture of the L2 vertebra (Figure 2). The lumbar spine BMD was measured using DXA and was below the expected range of age (–4.5 SD). The white blood cells count was decreased (3.370/ μL) and 25-OH vitamin D level was slightly decreased (21.6 ng/mL). Osteocalcin level and β CTX level was increased 56.6 ng/ml and 0.759 ng/ml, respectively. The red blood cells, platelets, calcium, liver enzymes, reactive protein, thyroid function tests, prolactin, alkaline phosphatase, protein electrophoresis revealed normal findings.

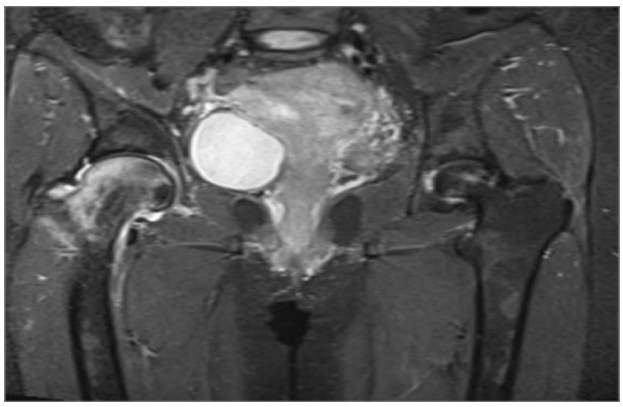

Figure 3: A MRI of the pelvic bones in case 3.

Finally, we report a case of a 35-years-old patient with complaints of progressive pain in the back, hip and right lower extremity, accompanied by reduced general mobility. Her height was 166 cm, body weight was 58 kg, and BMI was 20.0 kg/m^2. MRI revealed multiple sacral and femoral osteoporotic fractures. The lumbar spine and femoral BMD was below the expected range of age (–3.9 SD, –3.6 SD, –2.8 SD, respectively). The laboratory assessments (including the 25(OH)D vitamin, osteocalcin, β CTX) showed no abnormality. Neither of patients had any disease or other osteoporosis risk factors.

DISCUSSION

All the patients were diagnosed with PLO, and an appropriate treatment was initiated. As the etiology and pathogenesis of PLO remain incomprehensible, there is no mutually agreed opinion in the management of this condition.[4] The aim of therapy was to increase BMD and prevent new fractures and the development of chronic pain. Patients were primarily recommended to discontinue breastfeeding, maintain a balanced diet and rest.

Patients were administered with vitamin D supplementation (5000 IU/day) and calcium (500 mg/day). For pain management, nonsteroidal anti-inflammatory drugs and muscle strengthening exercises, and the thoracolumbar corset was recommended to all the patients. In case 1 daily injection of teriparatide for 10 months was used. In case 2 once-yearly infusion of zoledronic acid and L2 vertebroplasty was performed. In case 3 ibandronic acid injection every three months for two times was conducted.

PLO involves bone loss, but the process does not increase susceptibility to osteoporosis in the long term.[5] However, the transitory reduction in bone mass may pose some women at risk, possibly as a result of the previous osteopenia.[1,5]

CONCLUSION

PLO, although a rare disorder and often confused with other causes of lower back pain associated with pregnancy and lactation, should be kept in mind when a new and expecting mother develops persistent back pain. PLO can result in devastating physical, psychosocial and economic consequences, thus monitoring the patients with risk factors or secondary causes of osteoporosis, early diagnosis and management are essential for increasing the quality of life. Different treatment strategies equally well help to reach targets of the therapy in cases of PLO: increase BMD, prevent chronic pain and new fractures and improve a quality of life.

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