



Pregnancy and Lactation-Associated Osteoporosis

Gebelik ve Emzirmeye İlişkili Osteoporoz

© Mehmet ALBAYRAK

Özel Tekirdağ Yaşam Hospital, Clinic of Orthopedics and Traumatology, Tekirdağ, Turkey

ABSTRACT

Osteoporosis in pregnancy and during lactation period is a rare clinical problem with unknown etiology and pathophysiology. Pregnancy and lactation may have significant impact on bone loss and this may result in osteoporosis and related fractures. The symptoms often occur during the first pregnancy and usually do not recur. Secondary causes of osteoporosis that may cause back pain and fractures during pregnancy should be taught in differential diagnosis. Still no guidelines exist about the accurate treatment of this rarely seen osteoporosis type, this means each case must be evaluated on an individual basis in order to decide for a treatment plan. In this case report, a 25-year-old woman, complaining of severe back pain after twenty days of giving birth, is reported.

Keywords: Pregnancy, lactation, osteoporosis

ÖZ

Gebeliğe ve emzirmeye bağlı gelişen osteoporoz etiyolojisi ve patofizyolojisi tam olarak bilinmeyen ve nadir görülen bir klinik problemdir. Gebelik ve emzirme kemik kaybı üzerinde belirgin bir rol oynayarak gebeliğe bağlı osteoporoz ve buna bağlı gelişebilecek olan kırıklara sebep olur. Semptomlar genellikle ilk gebelikten sonra olur ve tekrar etmez. Gebelik sırasında ağrı ve kırık sebebi olabilen kemik erimesinin sekonder sebepleri ayırıcı tanıda düşünülmelidir. Bu nadir görülen osteoporozun belirlenmiş bir tedavi algoritması yoktur, bu yüzden her olgu bireysel olarak ele alınmalıdır ve tedavi planı belirlenmelidir. Bu olgu sunumu ile 25 yaşında doğum yaptıktan yirmi gün sonra şiddetli bel ağrısı başlayan bir olgu rapor edilmiştir.

Anahtar Kelimeler: Gebelik, emzirme, osteoporoz

INTRODUCTION

Pregnancy and lactation associated osteoporosis (PLAO) is a very rare condition seen during the last three months of the pregnancy or at early postpartum period^{1,2}. It was first defined by Nordin and Roper³ in 1955. It is characterized by pain due to vertebral fractures. There are cases in which rarely pelvis, sacral and wrist fractures have been reported⁴. Its etiology has not been clearly defined. Risk factors associated with pregnancy-related osteoporosis include genetic factors, physical inactivity, malnutrition, low body weight, osteoporotic fracture history in family members, and secondary osteoporosis^{3,4}. The high prevalence of fracture seen in female relatives of patients

diagnosed with PLAO suggests an underlying genetic component^{2,5,6}. Anorexia and oligomenoria history can be seen in PLAO patients⁷.

CASE REPORT

Mild low back pain started in 25-year-old primigravid woman without a previously known disease or trauma in her last month of pregnancy. Twenty days after giving birth by cesarean section under spinal epidural anesthesia, a very severe low back pain suddenly occurred without trauma. There was continuous low back pain at the degree limiting daily activities. She did not benefit from the analgesic treatment which was prescribed by a physician who she visited in another center with a diagnosis

Address for Correspondence: Mehmet ALBAYRAK MD, Özel Tekirdağ Yaşam Hospital, Clinic of Orthopedics and Traumatology, Tekirdağ, Turkey

Phone: +90 533 660 50 13 **E-mail:** doktorm.albayrak@gmail.com **ORCID ID:** orcid.org/0000-0002-4074-7024

Received: 12.03.2021 **Accepted:** 13.05.2021

of myalgia. Two months later after birthing, the patient applied to our hospital suffering from ongoing pains.

In her medical history, there was no disease or drug use that would suggest the secondary osteoporosis. The causes of secondary osteoporosis like smoking and alcohol use, menstrual irregularity, estrogen deficiency, history of fracture as a result of mild trauma, rheumatoid arthritis, infertility treatment with clomiphene, type 1 osteogenesis imperfecta, gluten enteropathy, corticosteroid or heparin usage, anorexia nervosa, chemotherapy, chronic obstructive pulmonary disease, diabetes mellitus, hypothyroidism, hyperthyroidism, chronic liver disease, and long term immobilization were all asked and none of them were positive.

Eating habits were questioned and it was detected that due to nausea and sometimes vomiting during pregnancy period, the intake of calcium with diet or supplementation was not sufficient. The patient claimed that calcium intake with diet was also low at long term before pregnancy.

Family history was normal as well. Her body mass index was 21.6 kg/m² (165 cm, 59 kg).

In the physical examination, there was an increase in the thoracic kyphosis, decrease in the lumbar lordosis and mildly left facing scoliosis in the thoracic region by inspection. Palpation revealed severe muscle spasms both in thoracic and lumbar paravertebral muscles. While there was pain and limited movement in all directions in the trunk, range of motion was severely compromised. No abnormality was detected in the bilateral upper and lower extremity motor and sensory examination. Deep tendon reflexes were normoactive and no pathological reflexes were detected.

The tests of complete blood count, routine biochemistry, sedimentation, C-reactive protein, *Brucella* agglutination test, parathormone, calcium excretion in urine, alkaline phosphatase, lactate dehydrogenase, and thyroid function were requested from the patient. No abnormality was detected in laboratory examinations except for phosphorus 5 mg/dL (N: 2.3-4.7 mg/dL) and 25-OH vitamin D 23.2 ng/mL (N: >30 ng/mL). Tumor markers were in normal range and no pathology was detected with mammary ultrasonography and mammography, parathyroid scintigraphy.

Bone mineral density (BMD) of the patient was measured by using the dual-energy X-ray absorptiometry method and the value were found to be low according to corresponding age group. The scores are listed in Table 1.

On plain radiographs, there was an arching at frontal T5-T12 and L1-L3 vertebrae. In the magnetic resonance imaging (MRI) of thoracic and lumbar vertebrae, it was detected that there was a widespread arch in T5-7-9-11-12, and L1-L3 vertebrae,

height loss at varying grades in all of mentioned vertebrae and edematous signal changes and compression fractures in the corpuses of them (Figures 1, 2). Osteoporosis was thought to be the cause of compression fractures.

By excluding all of the reasons for the secondary osteoporosis, the patient was diagnosed as PLA0.



Figure 1. T1 sagittal magnetic resonance imaging at the beginning



Figure 2. T2 sagittal magnetic resonance imaging at the beginning

The treatment was started by the termination of lactation since during lactation, the body would have needed twice as calcium than normal. Also, the patient was prescribed 1200 mg calcium/day, 2000 IU vitamin D3/day. As analgesic medication, non-steroidal anti-inflammatory drugs were prescribed. Alendronate 70 mg/week was added to the treatment. A dorsal splint corset was applied. Calcium rich diet was recommended to the patient. Moreover, home based exercise program, including stretching and strengthening the low back, waist extensors, strengthening isometric abdominal muscles, and 45 minutes of walking exercises were advised after consulting with the physical therapy and rehabilitation department. The patient was discharged by controls once a month for three months and then once every three months up to end of one year. The treatment regimen was continued as started for one year.

At the end of one-year follow-up, no additional fractures were observed in control X-rays and MRI. It was observed that there were improvements in BMD values in parallel with the decrease of the patient's complaints. Corset treatment was stopped. After one year, the patient came for controls once in

three months up to end of two years, thereby continuing the same treatment.

At the end of two-year follow-up, it was observed that fractures were completely healed in the control X-rays and MRI (Figures 3, 4). BMD values were closer to normal at the end of two years (Table 1). It was observed that the patient's complaints completely ended at the end of two years. The treatment was ended.

DISCUSSION

Although PLA0 is a rare condition, when low back pain occurs in the last trimester of pregnancy or in early postpartum period, it should be considered in differential diagnosis and not to be misdiagnosed⁵⁻⁹. PLA0 may be more common than the current literature suggests¹⁰. Detailed examination and exclusion of the causes of secondary osteoporosis are important and should be carried out in every patient⁵⁻⁹. The risk factors for osteoporosis should be considered and if there is any reason underlying, it should be treated when detected. Evaluating the patient independently is very important for the treatment to be more effective¹¹. The most important



Figure 3. T1 sagittal magnetic resonance imaging at the end of two years

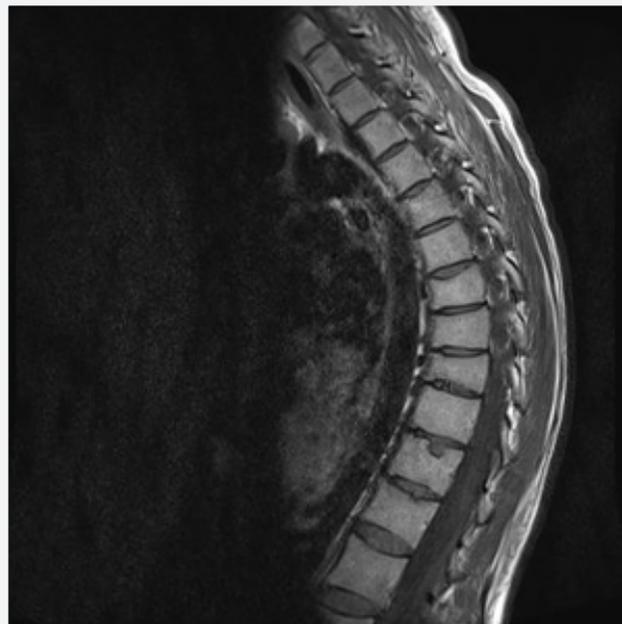


Figure 4. T2 sagittal magnetic resonance imaging at the end of two years

Table 1. BMD scores at the beginning, at one-year follow-up and at two-year follow-up

	L1-L4 T-score	L1-L4 Z-score	Femur neck T-score	Femur neck Z-score
BMD 0 day	-1.8	-2.8	-1.5	-1.8
BMD 1 year	-1.5	-2.8	-1.5	-1.8
BMD 2 years	-1.4	-1.1	1.1	-1.6

BMD: Bone mineral density

part of the treatment is not to miss other spinal fractures, so evaluation of the whole vertebral column is mandatory¹². It is known that subsequent pregnancies are not contraindicated⁵⁻⁹. Pregnancy and lactation are not major risk factors for BMD. However, breastfeeding should be terminated because of both drug use and possible calcium deficiency^{6,10}. Findings of BMD usually improve within a year. Regular calcium, vitamin D supplementation should be applied to these patients and exercises should be recommended. Bisphosphonate therapy administered soon after presentation substantially increases spinal bone density in patients with pregnancy-related osteoporosis¹³.

CONCLUSION

Although PLAO is a rare entity, it must be kept in mind in pregnant or in new mothers who come with back pain and must not be misdiagnosed. The question which has not been answered yet is whether the recovery in these patients is spontaneous or a result of the treatment. Further investigations or case series are needed for to solve this unanswered question¹⁴. The most important point of the treatment is that the treatment should be specific to the patient.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Externally peer-reviewed.

Financial Disclosure: The author declared that this study received no financial support.

References

1. Cosman F, de Beur SJ, LeBoff MS, Lewiecki EM, Tanner B, Randall S, et al. R; National Osteoporosis Foundation. Clinician's Guide to Prevention and Treatment of Osteoporosis. *Osteoporos Int*. 2014;25:2359-81.
2. Polat SB, Evranos B, Aydin C, Cuhaci N, Ersoy R, Cakir B. Effective treatment of severe pregnancy and lactation-related osteoporosis with teriparatide: case report and review of the literature. *Gynecol Endocrinol*. 2015;31:522-5.
3. Nordin BE, Roper A. Post-pregnancy osteoporosis; a syndrome? *Lancet*. 1955;268:431-4.
4. Lebel E, Mishukov Y, Babchenko L, Samueloff A, Zimran A, Elstein D. Bone mineral density in gravida: effect of pregnancies and breast-feeding in women of differing ages and parity. *J Osteoporos*. 2014;2014:897182.
5. Ofluoglu O, Ofluoglu D. A case report: pregnancy-induced severe osteoporosis with eight vertebral fractures. *Rheumatol Int*. 2008;29:197-201.
6. Holmberg-Marttila D, Sievänen H, Tuimala R. Changes in bone mineral density during pregnancy and postpartum: prospective data on five women. *Osteoporos Int*. 1999;10:41-6.
7. Tsvetov G, Levy S, Benbassat C, Shraga-Slutsky I, Hirsch D. Influence of number of deliveries and total breast-feeding time on bone mineral density in premenopausal and young postmenopausal women. *Maturitas*. 2014;77:249-54.
8. More C, Bhattoa HP, Bettembuk P, Balogh A. The effects of pregnancy and lactation on hormonal status and biochemical markers of bone turnover. *Eur J Obstet Gynecol Reprod Biol*. 2003;106:209-13.
9. Ghannam NN, Hammami MM, Bakheet SM, Khan BA. Bone mineral density of the spine and femur in healthy Saudi females: relation to vitamin D status, pregnancy, and lactation. *Calcif Tissue Int*. 1999;65:23-8.
10. Dunne F, Walters B, Marshall T, Heath DA. Pregnancy associated osteoporosis. *Clin Endocrinol (Oxf)*. 1993;39:487-90.
11. Leere JS, Vestergaard P. Calcium Metabolic Disorders in Pregnancy: Primary Hyperparathyroidism, Pregnancy-Induced Osteoporosis, and Vitamin D Deficiency in Pregnancy. *Endocrinol Metab Clin North Am*. 2019;48:643-55.
12. Yun KY, Han SE, Kim SC, Joo JK, Lee KS. Pregnancy-related osteoporosis and spinal fractures. *Obstet Gynecol Sci*. 2017;60:133-7.
13. O'Sullivan SM, Grey AB, Singh R, Reid IR. Bisphosphonates in pregnancy and lactation-associated osteoporosis. *Osteoporos Int*. 2006;17:1008-12.
14. Raffaetà G, Mazzantini M, Menconi A, Bottai V, Falossi F, Celauro I, et al. Osteoporosis with vertebral fractures associated with pregnancy: two case reports. *Clin Cases Miner Bone Metab*. 2014;11:136-8.