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Background: Decreased bone mineral density (BMD) during pregnancy and lactation are rarely described in literature. We present this case of multiple compression fractures during and after pregnancy, highlighting the diagnostic and therapeutic dilemma of an overlooked diagnosis.
Clinical Case: A 23-year-old female without significant past medical history, suffers from an acute onset lower back pain in the third trimester of her first pregnancy. On initial evaluation, this back pain was thought to be musculoskeletal and was dismissed by her medical team. No imaging was ordered throughout the duration of pregnancy, despite the pain remaining unrelenting. On her second day post-partum, she heard a "pop" in her back and fell while holding her newborn. Imaging revealed multiple vertebral compression fractures, in different stages of acuity. Due to debilitating pain, the patient quit breastfeeding and ultimately would never hold her baby again. Her simple activities of daily living were stymied, both as a mother and secretary. It wasn't long before she couldn't even fax forms in her office and had to leave work with debility. Traumatized by these life events and continuing to be afflicted by this chronic pain, the patient decided against having any future children. Her compression fractures were managed with different types of analgesics in addition to vitamin D and calcium supplements.
At age 53 and 57, BMD scans showed a T-score >1 at both the lumbar spine and total hip. Forearm BMD was not evaluated at these times. At age 58, a CT spine demonstrated new compression fractures at T5-T12 & L1-L5. She subsequently underwent kyphoplasty of T5, T7 and T8. Fortunately, a bone core biopsy of these 3 vertebrae showed no malignant pathology. A follow up CT scan 6 months later showed stable compression fractures, along with multilevel degenerative changes and neural foraminal stenosis. At age 60, the patient would receive L4-L5 trans-foraminal epidural corticosteroid injection and referral to the bone clinic at a tertiary health center. Initial lab work on referral was significant for normal calcium, albumin, parathyroid hormone, vitamin D, kidney function, liver function, serum and urine protein electrophoresis and cross link N-telopeptide. Osteocalcin was low at 3.9 ng/mL (NL 8.8–37.6 ng/mL). Repeat BMD scan showed T-scores of 0.6, 1.1 and -2.6 at her lumbar spine, total hip and distal forearm respectively. Her osteoporosis is currently managed with teriparatide without active issues.
Conclusion: This case highlights the rare development of low BMD in pregnant and breastfeeding women, without prior risk factors, jeopardizing future quality of life. The evidence behind the incidence and pathophysiology underlying these changes remains deficient. There remains a dearth of guidelines for definition and treatment of osteoporosis and low BMD in young peripartum women.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORT
Lung Transplantation in a Patient With Osteogenesis Imperfecta and Osteoporosis

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Background: Osteogenesis imperfecta (OI) and transplantation (TP) are independently associated with fractures. Yet reports regarding the skeletal effects of organ TP in OI are limited. We report the early skeletal outcomes in an OI patient with osteoporosis who underwent lung TP.
Clinical Case: A 35-year-old man with moderate/severe OI and severe bronchiectasis was admitted for progressive respiratory failure and expedited lung TP evaluation. OI was diagnosed at age 10 after sustaining a hairline coccyx fracture when falling off a stool; scoliosis was diagnosed at age 14; additional fractures included ankle (18 y), toes (28 y) and rib (34 y). He had dentogenesis imperfecta, but no hearing loss, easy bruising or OI family history. Bronchiectasis also began at age 10 and progressed, with multiple drug resistant infections and glucocorticoid (GC) treatments. At admission, he was on 6L oxygen and bed-bound from dyspnea. Notably, he had been rejected twice for TP because of his bone disease. Admission medications included calcium, D3, famotidine, inhaled fluticasone, tobramycin, and tiotropium bromide. His exam was notable for height 5'5", BMI 16.5 kg/m2, kyphoscoliosis, blue sclera and joint laxity. Labs were notable for (mg/dl): serum calcium 9.4, magnesium 2.4, phosphate 4.4; albumin 4.2 g/dl, alkaline phosphatase 75 U/L, 25(OH) D 34 ng/ml, sCTX 535 pg/ml, urinary calcium 370 mg/24 hr. DXA showed T-scores of -4.7 (lumbar spine), -3.3 (femoral neck), -3.2 (total hip), -2.6 (1/3 radius). Endocrinology was consulted about the skeletal risk of lung transplantation.
Discussion and Follow-up: The patient's manifestations of OI increased the risk of adverse skeletal outcomes. His high CTX suggested increased bone resorption, often seen with OI; bone formation was not directly measured but in OI is frequently reduced. Notably, his bronchiectasis was likely related to the OI: in addition to restrictive lung disease in OI, the abnormal type 1 collagen likely alters alveolar structure and elasticity. His risk for post-TP fractures was high given that the expected post-TP bone loss would likely be exacerbated by high dose GCs further increasing bone resorption and reducing presumed low bone formation. Nevertheless, because he had never sustained a major fracture even without OI treatment, the decision was made to proceed. He received zoledronate (ZOL) 5 mg IV and underwent an uncomplicated double lung transplant; initial high dose GCs were tapered to prednisone 10 mg/d. Three months later, he has steadily rising lung function, excellent functional status, and is working full time. A current sCTX of 73 pg/ml suggests that bone loss is not increased. Admittedly, the patient remains within the early high-risk fracture window. Yet this case is the first report to our knowledge which suggests that lung TP in an OI patient treated with ZOL did not lead to fracture in the early post-TP period.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORT
Maxillary Brown Tumor as a Rare Complication of Hyperparathyroidism