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CASE REPORT

Cessation of teriparatide in order to heal a humeral fracture: a case report

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Teriparatide (Forteo; Eli Lilly, Indianapolis, IN, USA) is a recombinant protein consisting of a biologically active portion of the parathyroid hormone (PTH) molecule. Acting on bone and renal G-protein receptors, the net effect of the medication is to increase cortical and cancellous bone mass.[8-10] Given in a continuous fashion, Teriparatide can favor bone reabsorption; however, if pulsed by daily dosing, it can favor bone formation.[7,17] PTH has been proven to increase cortical thickness without increasing porosity in osteoporotic patients.[5] Teriparatide has also been shown to decrease vertebral fracture risk in postmenopausal women with osteoporosis.[11,12] The drug is currently U.S. Food and Drug Administration-approved to treat patients with severe osteoporosis at high risk for future fracture. Teriparatide's ability to increase bone mass and decrease fracture risk has led to the investigation of PTH peptide 1-34 as an adjunct to assist in fracture healing.[2]

The current literature has focused on teriparatide use as an adjunct to heal acute fractures and fracture nonunions.[14] All of these studies have demonstrated that teriparatide has a positive outcome on fracture healing, but no studies have proven an outright negative outcome with its use. We present a case report of a proximal humeral nonunion with successful healing after termination of teriparatide therapy initially begun for fracture prevention.

Case report

A 43-year-old woman with a medical history significant for chronic regional pain syndrome and osteopenia sustained a right proximal humeral fracture after a fall from standing (Fig. 1). At the time of injury, the patient had been taking 20 μg of teriparatide daily for approximately 2 months as treatment for osteopenia. One week after her injury, the patient underwent open reduction internal fixation via locking plate construct (Fig. 2). During her hospitalization, the patient was found to have hypocalcemia and hypoalbuminemia, which were corrected before discharge via calcium, vitamin D, and protein supplementation. The patient was monitored closely as an outpatient with radiographs and clinical examination. Radiographs at the 4-month visit revealed a complete lack of any callous formation and smooth sclerotic fracture borders concerning for the beginning of a nonunion (Fig. 3, *A*). Due to the lack of any radiographic evidence of healing as well as continued pain at the 4 month postoperative visit, the patient began bone-stimulator treatment for proximal humeral nonunion; however, she was unable to tolerate the treatment secondary to chronic regional pain syndrome. A nonunion laboratory workup was also done at that time and was negative for metabolic, endocrine, or infectious abnormalities. Her albumin and prealbumin levels had corrected to within normal reference range values. At this time, conservative and surgical treatment options were given, and the patient decided on continued observation. The patient was monitored with monthly radiographs at 5, 7, and 9 months. She persisted to have no radiographic evidence of fracture healing (Fig. 3, *B* and *C*) and continued pain on physical examination. At 1 year postoperatively, the patient

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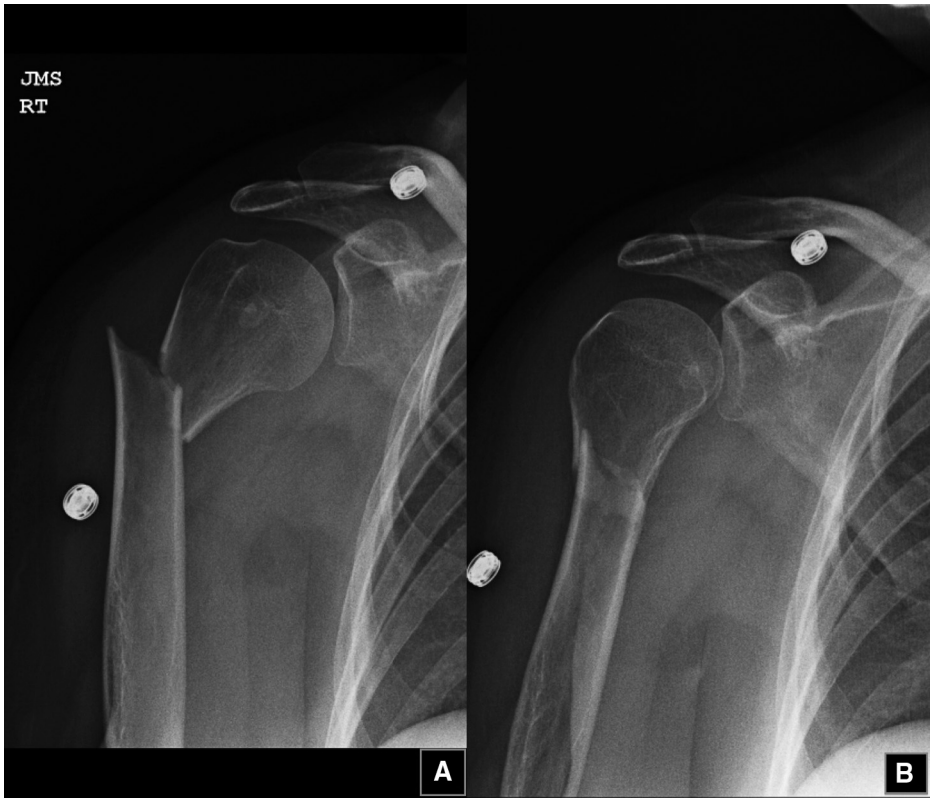

Figure 1 Anteroposterior radiographs in (**A**) external rotation and (**B**) internal rotation views on the date of injury (August 4, 2012).

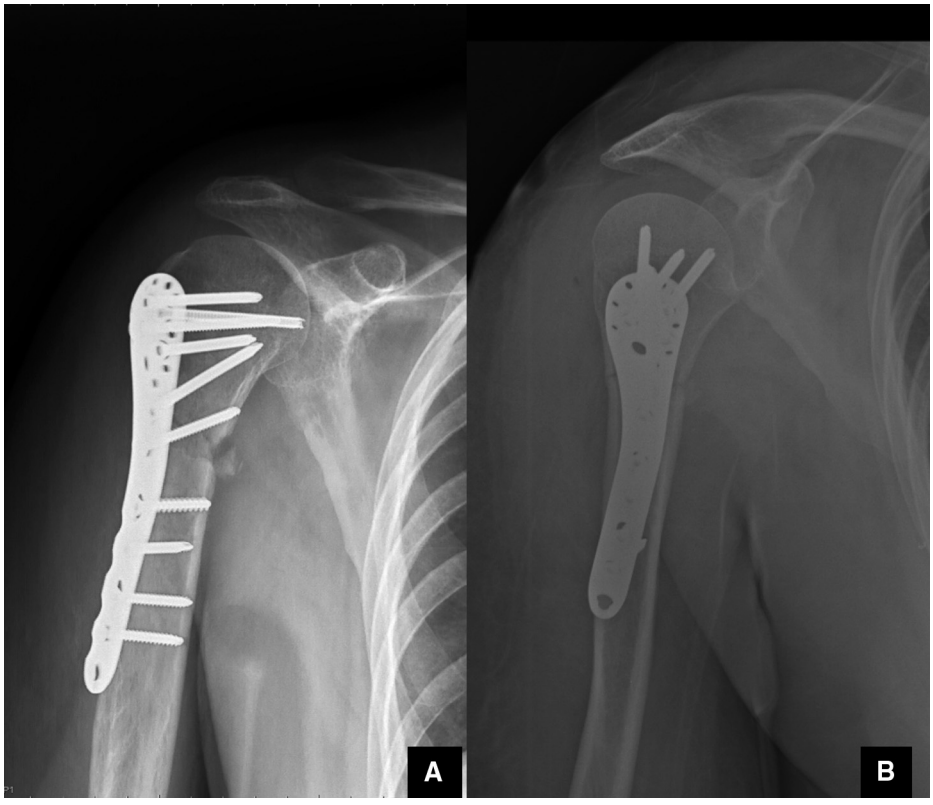

Figure 2 Anteroposterior radiographs in (**A**) external rotation and (**B**) internal rotation views in the immediate postoperative period (August 15, 2012).