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Spontaneous Insufficiency Fracture of the Lateral Malleolus in a patient with Primary Hyperparathyroidism.

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Introduction

Most ankle fractures in practice are of traumatic origin, however some may occur without a history of proceeding injury. Insufficiency fractures around the ankle are rare. We present the presentation, treatment and literature review of a patient with primary hyperparathyroidism presenting with insufficiency fracture of lateral malleolus.

Case report

A 57 year old patient, initially presented to the fracture clinic some days following the onset of symptoms of pain over the outer aspect of her right ankle. The pain was said to have started suddenly when she was asleep in bed without a history of preceding trauma. GP consultation was sought 3 days later and as the pain was unrelenting an A&E referral was ordered 10 days hence. A fracture clinic appointment was made via A&E department as a Weber B fracture of the lateral malleolus was identified clinically and radiologically (Fig.1).

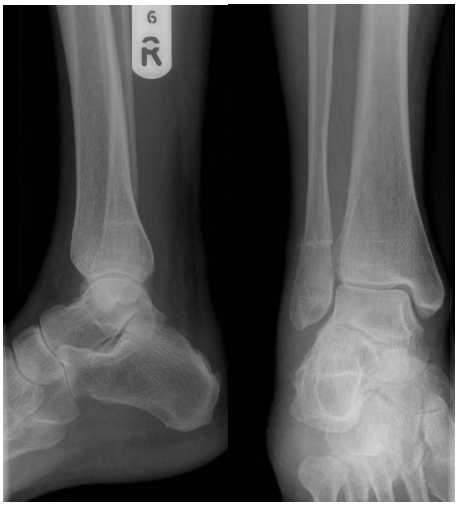

Figure 1 X-ray (R) ankle showing a Weber B fracture**.**

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Patient gave a past medical history of recurrent nephrolithiasis and a primary adenoma of the parathyroid gland for which she had undergone a parathyroidectomy approximately a year prior to presentation. Other significant medical conditions in the past were intestinal polyps, Achilles tendonitis, De Quervains's stenosing tenovaginitis. Her biochemical profile had shown increased levels of parathormone at 188pg/ml [normal range: 10-55pg/ml], along with raised levels of serum Calcium at 2.7mmol/l [normal range: 2.02-2.60mmol/l] and phosphorous level at 0.65mmol/l [normal range: 0.87-1.45mmol/l].

Management

The patient was treated non-surgically in a below knee plaster of Paris cast for a period of a total of 8 weeks during which time she remained non weight-bearing. Upon the removal of her plaster cast, the fractured lateral malleolus was noted to be healed with periosteal callus (Fig.2).

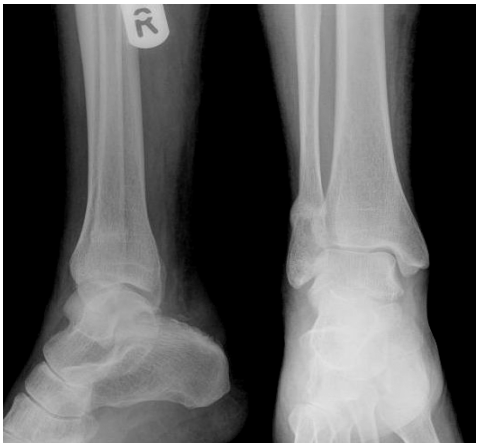

Figure 2 X-ray 8 weeks after presentation showing formation of periosteal callus.

On her latest follow-up the ankle has regained a full range of movement withnormal function. During follow-up in the fracture clinic the patient was advised a bone densitometry which showed osteopenia (T score = -2.14) of the left hip(Fig.3) and osteopenia (T score = -1.68) of the spine (Fig.4) where **T-score** is the number

of standard deviations above or below the young adult mean bone mineral density. The World Health Organization defines osteoporosis as a T-score of less than -2.5.

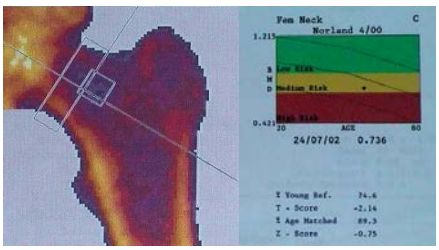

Figure 3 Osteopenia of left hip

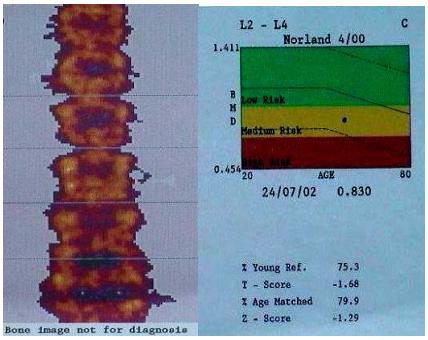

Figure 4 Osteopenia of spine

Discussion

Spontaneous onset of pain in a patient with primary hyperparathyroidism should raise the suspicion of an insufficiency fracture. By definition insufficiency fractures occur when normal repetitive forces are applied to bone that has been weakened by an underlying disease process. A stress fracture is an overuse injury. It occurs when muscles become fatigued and are unable to absorb shock and repeated impacts. Over time the fatigued muscle transfers this stress to the bone, resulting in a small crack (a stress fracture), and finally pathological fractures occur when a normal force is exerted on a bone that has been weakened by a focal underlying disease process. In this case the fracture was associated with a primary hyper parathyroidism. Under physiological conditions, the parathyroid glands function to maintain appropriate serum calcium concentrations and to regulate bone metabolism by means of the production of parathyroid hormone (PTH). In the non-pathological state, PTH secretion increases in response to low serum calcium concentrations and enhances the synthesis of 1,25-dihydroxyvitamin D. PTH and 1,25-dihydroxyvitamin D act together to increase calcium reabsorption in the gut and kidney and to promote osteoclastic resorption and the demineralization of bone.[1, 2]

Primary hyperparathyroidism is caused by an overproduction of PTH, in excess of the amount required by the body.[3,4] The effects of hyperparathyroidism on bone are numerous. Excess PTH results in an increase in bone breakdown by means of osteoclastic resorption with subsequent fibrous replacement and reactive osteoblastic activity. The bone may have microfractures, with subsequent hemorrhage and growth of fibrous tissue and an influx of macrophages. The process of bone resorption and fibrous replacement results in the characteristic radiological features of generalized bone demineralization, resorption, cysts, brown tumors, and pathological fractures.

In this case, the onset of pain in the ankle area without a history of trauma was misleading, more so due to the ongoing presence of Achilles tendinitis. Bone pains in hyperparathyroidism are not uncommon, however the sudden onset and the persistence of symptoms should alert the treating clinician about the presence of a concealed fracture to request early plain radiographs.

Conclusion

One should suspect a pathological fracture or an insufficiency fracture if a patient has a history of a metabolic disorder and presents with insidious onset of pain. Fracture healing may not be delayed as evidenced by satisfactory periosteal callus formation within the appropriate period of time for such fractures. However one must exercise caution and care while treating fractures with underlying metabolic disorders.

References

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