Asymmetrical bone loss in a patient with poliomyelitis: an indication for anti-osteoporotic therapy

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Summary

Background. Poliomyelitis survivors suffer from post-myelitic complications including osteoporosis that are often overlooked.

Methods. We report a case of a 49-year-old lady with history of poliomyelitis with resultant flaccid paralysis of the involved limb.

Results. The bone mineral density revealed asymmetrical severe osteoporosis in the poliomyelitic limb. Given the risk of falls and fractures, she was commenced on oral bisphosphonate therapy.

Conclusion. Poliomyelitis is an important acquired risk factor for regional osteoporosis. This condition should be detected and treated in this cohort of patients who are clearly at higher risk of fractures.

KEY WORDS: poliomyelitis; osteoporosis; bone mineral density.

Case report

A 49-year-old female was referred to us after a dual energy X-ray absorptiometry (DXA) scan. She had a history of childhood poliomyelitis affecting the left lower-limb, resulting in flaccid paralysis and reduced muscle power of the left lower-limb. Although fully ambulant with use of a walking aid, she was unable to weight-bear on the affected limb. She was perimenopausal and denied prolonged glucocorticoid intake, smoking or alcohol consumption. She had no history of fractures prior to this and there was no family history of osteoporosis.

On examination, she was a medium built lady, with a body mass index of 22.4 kg/m². Measured limb-length was 86 cm in the affected limb (left) and 88 cm in the contralateral limb. There was profound muscle wasting and flaccid paralysis over the left lower limb with a muscle power of 4 out of a score of 5.

DXA scan (LUNAR Prodigy®) revealed significant discrepancy in bone mineral density (BMD) between both femoral necks. The BMD of the left neck-of-femur (NOF) was significantly reduced at 0.504 g/cm² (Z score: -3.2) compared to the right NOF which was 0.798 g/cm² (Z score: -1.1), seen in Figure 1. Lumbar spine BMD was 1.001 g/cm² (Z score: -1.4), Figure 2. Laboratory investigations reported serum corrected calcium: 2.4 mmol/L (N: 2.2-2.6), phosphate: 1.0 mmol/L (N: 0.7-1.1), alkaline phosphatase: 78 IU/L (N: 50-136), 25(OH) vitamin D3: 29 ng/ml, intact PTH: 2.1 pmol/L (N: 1.5-7.8), normal liver function, renal function, thyroid function and complete-blood-count that ruled out other causes of secondary osteoporosis.

She was diagnosed with regional osteoporosis of the left hip secondary to poliomyelitis. The FRAX® score revealed a 10 year probability of fractures as high as 4.5% (hip) and 6.3% (major) in the affected limb. Considering her fall and fracture risk in the pathologically underdeveloped poliomyelitic limb, she was commenced on anti-resorptive therapy with oral bisphosphonate—weekly alendronate of 70 mg and supplemental calcium carbonate of 1 gm/day and vitamin D (cholecalciferol) of 1000 IU/day. She was also given advice on fall prevention and referred to the physiotherapist for lower limb muscle strengthening exercises. A repeat DXA scan of both hips and lumbar spine is scheduled at 2 years from commencing anti-osteoporotic therapy.

Discussion

Poliomyelitis is estimated to have affected more than one million people worldwide and was the commonest cause of disability amongst children in the 1950s-60s. Those survivors who are now in the older adult age group (50-70 years) suffer from morbidities collectively termed post-myelitic-syndrome (1, 2). The post-myelitic complications which include increased risk of falls and osteoporosis are often overlooked.

To date, there is sparse data that looked into the problem of osteoporosis amongst poliomyelitis survivors. The study by Mohammad et al. highlighted that up to 96% of polio survivors had osteoporosis or osteopenia in the affected limb and the major osteoporotic fracture incidence in this cohort was as high as 38% over 5 years (3). Almost all fractures involved the femoral neck of the affected or atrophic limb (3, 4). Despite the alarming rates of fractures only less than a quarter were treated with anti-osteoporotic agents.

The increased risk of fractures are directly related to increased risk of falls. Falls is a major concern amongst poliomyelitis sur-
Survivors with up to 60-80% of survivors having reported to have fallen at least once in the past year and the incidence increases with advancing age (2, 3, 5). The fall risk is believed to be four times higher than the age matched normal population and one third who fell had sustained fragility fractures in the affected limb (5). The other general factors that contribute to increased risk of osteoporosis and fragility fractures in this cohort of patients are probably the advancing age, lack of physical activity from muscle weakness and lack of sunlight exposure that lead to vitamin D deficiency.

Although the susceptibility of falling and the other mentioned factors are a clear reason for increased fracture risk in poliomyelitic survivors, this fails to explain the disproportionately greater prevalence of fractures in the atrophic limb. Generally poliomyelitic patients had lower BMD than their age and gender matched healthy counterparts and the atrophic leg is...
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shown to have a significantly lower femoral neck BMD compared to the contralateral leg (6). The regional osteoporosis in the atrophic leg occurs as a result of flaccid paralysis, muscle disuse, underdeveloped growth of the limb and non-weight bearing bone (3-6). The regional osteoporosis and the interplay between other local factors such as reduced stability and poorly developed muscle in the atrophic limb leads to increased risk of fractures in that limb.

Hence, our case report underscores the importance of screening aging poliomyelitis survivors early using a simple tool such as a DXA scan on both the afflicted atrophic limb and the contralateral limb as well as the lumbar spine for comparison. As shown by Marshall et al., the prediction of osteoporotic-fracture risk using BMD is site-specific (7). Therefore the finding of regional osteoporosis in the atrophic limb should warrant commencement of anti-osteoporotic therapy with calcium and vitamin D supplementation, more so in the context poliomyelitis survivors who are clearly at increased fracture risk for the reasons highlighted above. Fall prevention would play an essential role in preventing fractures in this cohort of individuals besides advocating lower limb muscle strengthening exercise and adequate calcium and vitamin D supplementation that would improve stability and musculoskeletal function.

Further systematic research is warranted to determine the appropriate type of anti-osteoporotic therapy, the exact timing in which treatment should be commenced and the duration treatment should be continued, weighing the benefits and risks of long-term anti-osteoporotic therapy in this special group of aging poliomyelitis survivors.

References