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Bone metabolism and fracture risk in type 2 diabetic polyneuropathy

***Aims*:** To examine the risk of fractures and bone metabolism in individuals with type 2 diabetic polyneuropathy (DPN).
***Methods*:** In this cross-sectional study, males with type 2 diabetes (n=198), aged 54-70 were recruited in Shanghai Tongji Hospital between January 2017 and December 2020. There were 109 individuals with DPN and 89 individuals with type 2 diabetes without DPN. DPN was diagnosed based on vibration perception threshold and clinical evaluations using Toronto Clinical Neuropathy Score. Bone mineral density (BMD) was evaluated by dual-energy x-ray absorptiometry, and bone turnover markers, including cross-linked C-telopeptide (CTX) and alkaline phosphatase (BALP), were measured. The risk of hip fractures in 10 years was calculated with the WHO Fracture Risk Assessment Tool (FRAX).
***Results*:** Individuals with DPN had a lower level of CTX (0.32 ± 0.19 vs 0.38 ± 0.21 ng/mL, p = 0.038) and a higher level of BALP (15.28 ± 5.56 vs 12.58 ± 4.41 µg/mL, p = 0.003) compared to individuals without DPN. Individuals with DPN had a higher BMD at the lumbar spine, L1-L4, (1.05 ± 0.19 vs 0.95 ± 0.37, p = 0.027) and increased risk of hip fracture (0.98 ± 0.88 vs 0.68 ± 0.63, p = 0.009) compared to individuals without DPN. As for the risk of hip fracture, old age, use of insulin, and elevated levels of BALP and CTX were all positively related to the risk of hip fracture ($p<0.01$).
***Conclusions*:** Individuals with DPN had higher BALP and lower CTX levels, which may increase BMD at the lumbar spine and indicate an altered bone metabolism.

Comments. Individuals with DPN are susceptible to fractures in contrast to those without DPN (Kim JH et al *Clin Endocrinol (*Oxf). 201*2;77:51-5)*. The underlying mechanism of bone fragility could be partly attributed to the accumulation of advanced glycation end products in bone collagen and a decrease in bone turnover (Saito M et al *Curr Osteoporos Rep. 2014;12:181-8*.). Interestingly, BMD remains either normal or elevated in individuals with DPN.
The authors conclude that despite there being an increased BMD at the lumbar spine in patients with DPN, there are still signs of altered bone metabolism and an increased risk of hip fractures. This research study contributes to our understanding of the link between diabetic neuropathy and bone health, adding insights to the existing body of evidence. Future studies should include nerve conduction studies of peripheral nerves and utilize advanced imaging technologies to better understand the impact of DPN on bone metabolism. Additionally, there is a lack of prospective and longitudinal studies that are essential for evaluating bone metabolism and fracture risk over time in individuals with DPN. The recent addition of novel medications for the treatment of diabetes adds complexity, potentially affecting bone and muscle metabolism, and fracture risk in DPN patients.

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