A56 The Effects of Diabetes Mellitus (DM) on Bone Fracture Susceptibility and Repair With Applications to Non-Accidental Forensic Fracture Interpretation

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Learning Overview: The focus of this research is the examination of the effect of DM upon bone fracture risk and subsequent bone healing. The goals of the study are to identify the bony signatures of DM, determine whether they can be differentiated from other comorbidities affecting the skeleton, and evaluate whether the presence of DM increases fracture risk and alters fracture healing in diabetic individuals.

Impact on the Forensic Science Community: This research will impact the forensic science community by providing greater understanding of the influence of DM on bone fracture and healing and lead to the potential for identification of this condition in forensic remains. It will ultimately aid in improving accuracy of forensic trauma interpretations of accidental vs. non-accidental fracture and Time Since Injury (TSI).

Previous research has suggested that diabetes, a chronic metabolic disease characterized by increased blood glucose levels, can result in an array of deleterious effects upon the human skeleton, including irregular distribution of bone mass, decreased bone strength, increased fracture susceptibility, extended healing times, and poor bone regeneration. DM complications and treatments, including hyperglycemia, increased Advanced Glycation End product (AGE) formation, reactive oxygen species generation, inflammation, and the ingestion of insulin and oral antibiotic medicines, can compromise bone health, including its formation, mass, density, distribution, healing process, velocity, and overall healing outcome. DM has been associated with an increase in osteoclastic and thus resorptive bone processes at the expense of osteoblastic (bone forming) ones, although this interpretation has been questioned.[1,2]

However, the specific effects of DM on bone quality and strength, fracture risk, and fracture healing have rarely been studied from an anthropological perspective using known diabetic decedent collections. In the current research, 44 known diabetic decedents, ranging in age from 21 to 84 years, comprise the combined study sample, derived from the University of Tennessee William Bass collection (self-identified as diabetic), the Terry collection (diabetes informed by medical records), and the Radford University Forensic Science Institute (medical records). The majority are affected by Type 2 DM; however, at least two decedents are noted as Type 1. Variables recorded from this sample include presence, type, and location of fracture, character and status of bone healing, and microscopic evidence for bone distribution, density, and overall bone quality (using a 3D digital microscope at 5x–200x magnification).

It is hypothesized that this diabetic sample will manifest a higher fracture frequency and greater number of fractures in non-union or a non-advanced stage of bone repair and remodeling compared to that observed for the non-diabetic decedents in each collection.

Results generally support the stated hypothesis. Fracture frequency is significantly higher (44%) in the diabetic sample, accompanied by earlier (rather than later) fracture healing status for the majority of fractures. At least three decedents manifest amputations in their lower limbs. Microscopic analysis of exposed trabeculae reveals evidence for diminished bone volume in healing fractures, reflected by the presence of smaller bone calluses. Also evident is irregular bone distribution, macroporosity, and overall poor bone quality and strength in the diabetic sample.

However, a confounding limitation of the study was the presence of significant comorbidities associated with diabetic decedents. A majority of the diabetic individuals manifest evidence for osteoporosis, cancers, and other diseases that influence bone quality and complicate attempts to definitively identify bony signatures of DM.

Bone repair for the diabetic sample is interpreted using a model of anabolic compromise in conjunction with catabolic excess and informs the interpretations regarding TSI for fractures observed in diabetic decedents in a forensic context. In conclusion, given the increased fracture risk and fracture healing time variability, as well as decreased bone regenerative capacity for diabetic individuals, it is recommended that caution be exercised in interpretations of non-accidental bone fracture and its TSI in diabetic decedents, particularly for those who are elderly and manifesting associated comorbidities such as osteoporosis.

Reference(s):

1. Marin, M., Luyten, F.P., Van der Schueren, B., Kerckhofs, G., Vandamme, K. The Impact of Type 2 Diabetes on Bone Fracture Healing. *Frontiers in Endocrinology* (Lausanne) (2018):9:6. doi: 10.3389/fendo.2018.00006.
2. Kayal, R.A., Tsatsas, D., Bauer, M.A., Allen, B., Al-Sebaei, M.O., Kakar, S., Leone C. W., et al. Diminished Bone Formation During Diabetic Fracture Healing is Related to the Premature Resorption of Cartilage Associated with Increased Osteoclast Activity. *Journal of Bone and Mineral Research* (2007):22(4):560-568.

Diabetes Mellitus, Bone Fracture, Bone Healing