Diabetes and osteoporotic fracture. Do clinicians take care of the risk?

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The number of patients with lifestyle-related diseases is rapidly increasing in worldwide. In recent studies both type 1 diabetes and type 2 diabetes mellitus (DM) was found firmly associated with high fracture risk. Although type 1 DM is associated with lower bone density, those with type 2 DM usually have normal/elevated bone density. Yet both types of DM, bone appears to be more fragile in microarchitecture. Not only hyperglycemia, but also oxidative stress induced deposition of advanced glycosylation endproducts (AGEs) in collagen, reduced serum level of insulin like growth factor 1 (IGF-1), hypercalciuria, renal failure, microangiopathy and inflammation are various mechanisms were hold responsible for lower bone quality in diabetic population. Additionally falls arising from diabetes-related comorbidities are another possible contributing factor for pathologic fracture in DM.

Here we present 13 years-old boy history of type 1 DM with right humerus posttraumatic fracture 1 months ago. Better knowledge on how diabetes and its treatments influence bone tissue will achieve the effective prevention of high fracture risk in both type 1 and type 2 DM patients.

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Dear Editor,

In recent years studies have found evidence related to the increased risk of fractures of the hip and other areas in Type 1 and Type 2 diabetes mellitus (DM). While frequent falls clearly have a place in this increased risk, reduced bone strength also plays a role. Type 1 DM is related to reduced bone density and Type 2 generally with increased bone density. However, bones are seen to be more fragile in both types of DM. Bone tissue is affected by multiple pathways in DM including the accumulation of advanced glycosylation products in obesity, hyperinsulinaemia and collagen. It has even been thought that the administration of some oral anti-diabetic agents (Thiazolidinediones) in DM treatment could increase the risk of fracture in elderly females.

Clinicians must be aware of the increased fracture risk associated with DM. The best approach to preventing future fracture risk is an understanding of the underlying mechanism of the increased risk of fracture [1]. The case is here presented of a 13-year old male patient with a history of Type 1 DM who developed a post-traumatic right humerus fracture. The aim of this paper is to emphasise the effect of DM on bone metabolism.

A 13-year old male presented with complaints of restricted movement in the right shoulder and pain in the anterior of both thighs. One month previously a fracture had been diagnosed as a result of a fall on the arm. The shoulder had been bandaged (Velpeau) in the Orthopaedic Clinic. It was learned from the history that at the age of 3 years, the patient had gone into a coma and was subsequently diagnosed with Type 1 DM. Insulin treatment was started, but the family stated that blood sugar levels were not regularly monitored and attention was not paid to the diet.

In the physical examination after 1 month evaluating the range of movement of the right shoulder joint, slight restriction was determined in internal rotation. In all other directions, the shoulder movements were full and painless. In the laboratory examinations, fasting blood sugar was determined as 327 mg/dl (70-105), blood urea nitrogen: 45 mg/dl (15-40), AST: 22 U/L (5-34), ALT: 13 U/L (0-55), ALP: 313 IU/ml (40-150), calcium: 9.2 mg/dl (8.4-10.2), phosphorus: 4.2 mg/dl (2.3-4.7), vitamin D: 12.1 ng/ml (10-44), parathormone: 48 pg/ml (15-65), thyroid stimulating antibody (TSH): 4.72 uIU/ml (0.27-4.2), sTf: 3.33 pg/ml, sT4: 1.36 ng/dl (0.93-1.7) and hemoglobin A1c%: 13% [4-6]. Full blood count, acute phase reactants and the lipid profile were within normal limits.

The fracture which developed following the fall was thought to be due to osteoporosis which had developed secondary to poorly controlled blood sugar levels. An osteoporotic appearance was determined on the conventional radiographs. The insulin dosage used by the patient to control blood sugar levels was assessed and ibuprofen 2 x 200mg/day oral tablets were started. As osteoporosis treatment, 1200 mg/day calcium carbonate and colecalciferol 800 IU 1 x 1 oral tablet/day were started. The patient was informed about preventing osteoporosis.

Proximal humerus fractures are rare in paediatric traumatology [1-2]. While metaphyseal fractures comprise 70% of cases, the remaining 30% are epiphyseal separations. It is useful to have good knowledge of the development and anatomy of the proximal humerus, various fractures, separation types, potential complications and especially interpretation of radiological findings seen in children [2].

In non-complicated shoulder injuries, the treatment approach generally includes conservative methods. The vast majority of bandages used are known as Velpeau bandages. This bandage was first used by Velpeau in Paris in 1795. In a Velpeau bandage, the injured arm is placed crosswise on the chest and the bandage is wrapped round the axillary, the injured shoulder and passing over the forearm below the elbow joins again to the axilla [3]. In the current case, a Velpeau bandage was seen to be appropriate as the first approach and at the 1-month follow-up, successful union was determined in the fracture line.

Post-traumatic fractures are the most common complaint of orthopaedic emergencies. In addition, which fractures are related to osteoporosis is an important subject for clinicians and researchers. Warriner et al [4] made a systematic review of literature of which fracture types and anatomic regions were related to osteoporosis and it was determined that femoral neck, lumbar or thoracic vertebra pathological fractures were related to osteoporosis at a high rate and proximal humerus fractures, skull and facial bone fractures at a low rate.

Throughout the world, lifestyle-related diseases are increasing. DM, dyslipidemia and metabolic syndrome including hypertension lead to several complications, primarily cardiovascular diseases. Current studies have shown that lifestyle-related factors increase the risk of osteoporotic fracture. In addition, how bone tissue is affected by metabolic diseases is the subject of ongoing research. Low grade metabolic acidosis and increased oxidative stress as a complication of DM contribute to each other in the bones and double the risk of bone fracture in diabetic individuals. The negative effect of the oxidative and/or glycolisation stress of DM on bone tissue is thought to increase the risk of fracture [6].

DM reduces bone mineral density. Previous studies have shown that it is not just through hyperglycaemia of DM, but at the same time low mineral density is caused through mechanisms such as reducing the beneficial enzymatic cruciate ligaments in oxidative stress and the accumulation of harmful advanced glycated end products (AGEs) of non-enzymatic cruciate ligaments (such as Pentosidine in bone tissue) [7].

There is an anabolic effect of insulin on bone mass in Type 1 and Type 2 DM patients, consistent with the hypo/hyperinsulinemia stage. In addition, despite the presence of hyperinsulinemia, DM supports the effect of other potential pathogenic variables of increased fracture risk (hyperglycaemia, diabetic complications, lifestyle factors) [10].

If there is insufficient potential for remodelling because of the severity of the blow, angulation or the patient’s age, retrograde elastic stable intramedullary nailing (ESIN) provides successful stabilisation. Although a thoracobrachial abstraction corset is still an effective choice, it has started to be used less often. When closed reduction is not successful, there may occasionally be a need for open reduction in severe fractures [2].

Successful precautions in the prevention of bone fractures in the DM population are directed at understanding the effects on bone tissue of the disease itself and the treatments applied. Therefore, tight glycemic control in both Type 1 and Type 2 DM, sufficient calcium and vitamin D intake, and scanning for low mineral density are important approaches in the prevention and treatment of DM complications and control of osteoporosis. Care must be paid to TZD treatment in elderly female patients with Type 2 DM in particular. While DM and osteoporotic patients can be treated with the same medications as non-diabetic patients, it must be noted that there have not been sufficient studies related to the effect of anti-osteoporotic medications on the diabetic population (including the paediatric age group).
References