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REVIEW ARTICLE

To do one and to get more: Part I. Diabetes and bone

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Abstract: Type 2 diabetes mellitus (T2DM), is a chronic metabolic disease, characterized by the presence of hyperglycemia and insulin resistance. The key treatment strategies for T2DM include modification of lifestyle, medications, and continuous glucose monitoring. DM patients often have DM-associated morbidities and comorbidities; however, disorders of musculoskeletal system are often neglected, compared to other major systems in DM patients. Based on sharing similar pathophysiology of DM and osteoporosis, it is supposed that the use of antidiabetic agents (ADAs) may not only provide the lowering glucose level effect and the maintenance of the sugar homeostasis to directly delay the tissue damage secondary to hyperglycemia but also offer the benefits, such as the prevention of developing osteoporosis and fractures. Based on the current review, evidence shows the positive correlation between DM and osteoporosis or fracture, but the effectiveness of using ADA in the prevention of osteoporosis and subsequent reduction of fracture seems to be inconclusive. Although the benefits of ADA on bone health are uncertain, the potential value of "To do one and to get more" therapeutic strategy should be always persuaded. At least, one of the key treatment strategies as an establishment of healthy lifestyle may work, because it improves the status of insulin resistance and subsequently helps DM control, prevents the DM-related micro- and macrovascular injury, and possibly strengthens the general performance of musculoskeletal system. With stronger musculoskeletal system support, the risk of "fall" may be decreased, because it is associated with fracture. Although the ADA available in the market does not satisfy the policy of "To do one and to get more" yet, we are looking forward to seeing the continuously advanced technology of drug development on diabetic control, and hope to see their extra-sugar–lowering effects.

Keywords: Antidiabetic agents; Bone health; Diabetes mellitus (DM); Extra-glycemic effects

1. INTRODUCTION

Diabetes mellitus (DM), including type 1 DM (T1DM) and type 2 DM (T2DM), is a common but complex disease, associated with lots of comorbidities and a leading cause of mortality.[1–8] Risk factors associated with developing DM, especially T2DM accounting for >90% of all cases of DM worldwide, include elderly population,[9–11] family history (multiple genes),[11–18] overweight,[3,4,17–21] obesity,[3,4,17–21] pregnancy, such as the development of gestational DM,[21–24] dietary (calories intake, fibers, essential micronutrition, and mineral elements),[25–29] lifestyle factors (exercise, rest, and others),[3,4,30–32] environmental factors, interactions with other microorganisms (microbiotas), and many others.[33–35] The key pathophysiologic mechanism of DM is based on the inability of faulty pancreatic β-cells to secret a normal amount of insulin to maintain normal body consumption, and/or peripheral tissue has a decreased susceptibility to insulin, resulting in hyperglycemia and insulin resistance.[3,4,36] The main goal of DM management is normalized blood sugar and maintenance of sugar homeostasis to avoid and/or delay DM-related tissue or organ damage. The management strategies include lifestyle modification, pharmacological therapy, and routine and continuous blood glucose monitoring.[9,10] Lifestyle modification, such as caloric restriction, body weight reduction (in part, through reducing extrinsic insulin resistance), regular and appropriate exercise, and others, is recommended as first-line therapy to manage prediabetes and DM.[21–24,27,28,30–32,36] The major strength of lifestyle modification is based on its safety and potential effectiveness, which are considered as overt benefits to harms.[37–39] However, DM patients have a tendency to lower total adherence to healthy eating patterns or poorer consumption of major food groups (grains, fruits, vegetables, proteins, seeds, nuts, and dairy)[17,18,30,31]; therefore, many DM patients still need further pharmacologic agent therapy (antidiabetic agents [ADAs], glucose-lowering drugs) to overcome the underlying pathophysiological mechanisms of DM, such as inadequate and

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inappropriate secretion of insulin, defect and deficiency of insulin receptor, and malfunction about interaction of insulin and insulin receptor.[9,10,40–42] Since pharmacological therapy is not avoidable, physicians should be familiar with different pharmacologic options to target DM, since as shown above and below, DM is frequently associated with major morbidities or concomitant comorbidities.[9,10,43–47] Finally, routine and continuous blood sugar monitoring to make sure that DM patients state in normalized and stabilized sugar homeostasis is a confirmatory step for successful DM control.

2. TYPE 2 DIABETES MELLITUS AND MORBIDITIES OR CONCOMITANT COMORBIDITIES

Since DM is a chronic systemic inflammatory disease and shares the similar pathophysiological disorders of many other chronic diseases or aging process, such as cancers, hereditary diseases, and many others,[4,7,8,10,12–15,48–51] DM is associated with many morbidities. Morbidity from DM is mainly secondary to microvascular damage and dysfunction (hypertension, retinopathy, nephropathy, neuropathy, diabetic foot, and nonalcoholic fatty liver), macrovascular injury and occlusion (hypertension, atherosclerotic cardiovascular disease), and acute complications of life-threatening diseases, including hyperglycemia (hyperglycemia hyperosmolar nonketotic coma and diabetic ketoacidosis) or hypoglycemia.[52–62] DM is also a main cause of end-stage of renal disease (ESRD or kidney failure), lower-extremity amputation, and blindness. All DM-related loss of vital and nonvital organ functions contribute to significant impairment of normal daily activity and dramatic deterioration of quality of life (QoL) in DM. Additionally, the world with its increasingly aging processes stands to suffer from the heavy socioeconomic burden resulting from the severe morbidity and mortality imposed by DM and DM-associated disorders or DM-accompanied comorbidities. Therefore, appropriate treatment for DM patients is critical and of paramount importance. Among these, pharmacological therapy by ADA (glucose-lowering agents) plays a decisive role to achieve this purpose.[9,10,40,41,43,44] For the following discussion, we would like to focus on one silent and often neglected issue-osteoporosis and its related fracture.[63–67] Compared with cardiovascular accidents, ESRD, lower limb amputations, or blindness, osteoporosis does not represent the common causes of morbidity and mortality in T2DM, but there is no doubt that DM and osteoporosis frequently attack elderly population and both are rapidly assuming epidemic proportions in developed countries.[56,57] Additionally, osteoporosis and fracture may make these DM people debilitating and bedridden directly and indirectly; and all further worsen their QoL and also cut the lifespan in this population.

3. MEASUREMENT OF BONE HEALTH

Bone quantity can be evaluated by dual-energy X-ray absorptiometry (DEXA) to assess bone mineral content or bone mineral density (BMD) and the definition of osteoporosis is relatively standard. Based on the definition from the International Osteoporosis Foundation, BMD, usually measured at the lumbar spine and hip is used to diagnose osteoporosis when the measurement is 2.5 or more SDs below peak bone mass (minimum T score, ≤–2.5).[68] However, it is hard to reproducible of DEXA report, partly because differences between DEXA machines (exceeding 10% between some manufacturers but still substantial within the same manufacturer) are dramatically large as well as partly because the patient and technologist who are involved with DEXA examination also influence the final BMD presentation by altering patient positioning during examination. Additionally, hip may be a better target site for examination, because hip may not be significantly biased by degenerative changes, which may produce overestimation in BMD, resulting in measurement errors.[68]

To evaluate the bone quality, it is more difficult compared to use DEXA to assess bone quantity, since there are many strategies to test the bone quality, which include (1) imaging modalities, such as radiography (with limitations as low sensitivity, unable to further visualize the microstructure of bone specimens as well as two-dimensional [2D] image to evaluate bone shape, cortical thickness, cortical-medullar index, and trabecular homogeneity index), DEXA, computed tomography (CT)-based techniques (the three-dimensional [3D] microstructure level as quantitative CT [QCT], high-resolution peripheral QCT with advantages as measurement of density-independent of overlying tissue, avoidance of bone size interference effect, acceptable safety as well as higher accuracy and 3D visualization, and micro-CT, called as the gold standard to assess bone quality by allowing objective and quantitative evaluation of trabecular bone structure (TBS), including bone volume fraction, bone surface density, specific bone surface, trabecular thickness, trabecular number, trabecular separation, structure model index, and connectivity density), and magnetic resonance image (MRI)-based modalities (high-resolution-MRI, micro-MRI, and nuclear MR); (2) mechanical testing approaches, such as traditional testing methods (often combination with micro-CT) and microindentation testing; and study of compositional characterization to evaluate bone quality and bone compositions, such as degree of bone mineralization (separating bone into an increased status resulting in bone stiffer and more resistant to mechanical loading, and increased brittleness-high tendency to crack propagation, and decreased toughness-the ability to deform without fracturing and too low status contributing to bone softening, reduced stiffness and strength); and finally (3) organic composition, such as collagen glycation and collagen cross-links to evaluate advanced glycation end products (AGEs), which are correlated with bone fragility by altering bone matrix properties and to measure the extent of nonenzymatic glycation that is linked to alterations in the microarchitecture and microdamage of cancellous bone.[69] Recently, another term described as lacunar-canalicular bone remodeling system (LCBRS) is also reported to be reflective of bone quality, because suppression of LCBRS may decrease bone fracture toughness.[70] Additionally, the LCBRS can distinguish aging process and other osteolytic osteolysis phenotypes, and the former is typically characterized by decreased lacunar size, increased lacunar sphericity, decreased lacunar number, and decreased viable osteocytes; but the latter is presented as increased lacunar size, decreased lacunar sphericity, decreased perilacunar mineralization and no change in viable osteocytes.[70] Unfortunately, tools that require synchrotron radiation are only limited to very few researchers, suggesting that evaluation of bone quality still faces a long way to get popularity. The focus of this section is on techniques available in routine clinical practice to introduce the tools to evaluate bone quality and quantity. The following is the correlation between bone health and DM.

4. BONE HEALTH AND DIABETES MELLITUS

Evidence shows both quality and quantity of bone have a strong causal relationship with DM, contributing to the increased risk of osteoporosis and it is associated with fracture and even though a risk of fracture is also increased dramatically in those DM patients without osteoporosis.

As early as 2016, one review based on 21 studies enrolling 6 995 272 subjects showed that DM, regardless T1DM or T2DM was associated with an increased risk of hip fractures (risk ratio or relative risk [RR], 2.07; 95% CI, 1.83-2.33). Furthermore, T1DM patients had much higher risk of the development of

hip fracture with RR 5.76 (95% CI, 3.66-9.07) compared with T2DM (RR, 1.34; 95% CI, 1.19-1.51).[71] In 2017, Dytfeld and Michalak[72] focused on postmenopausal women and found that T2DM postmenopausal women had a higher risk of hip fracture (odds ratio [OR], 1.30; 95% CI, 1.10-1.57).

In 2019, Wang et al[73] found that DM subjects have an increased risk of total (RR, 1.32; 95% CI, 1.17-1.48), hip (RR, 1.77; 95% CI, 1.56-2.02), upper arm (RR, 1.47; 95% CI, 1.02-2.10), and ankle fracture (RR, 1.24; 95% CI, 1.10-1.40). Their study also confirmed that an increased risk of fracture is more apparent in T1DM, since compared to T2DM, T1DM subjects had a greater risk of total (1.24; 95% CI, 1.08-1.41), hip (RR, 3.43; 95% CI, 2.27-5.17), and ankle fractures (RR, 1.71; 95% CI, 1.06-2.78).[73] Another meta-analysis found that T2DM had a lower risk of prevalent (RR, 0.84; 95% CI, 0.74-0.95) but increased risk of incident vertebral fractures (OR, 1.35; 95% CI, 1.27-1.44).[74] The current meta-analysis also showed subjects with T2DM and vertebral fractures faced the high risk of mortality, because these patients had at least a 2-fold increase in mortality compared with those without T2DM and vertebral fractures (hazard ratio [HR], 2.11; 95% CI, 1.72-2.59) or with vertebral fractures alone (HR, 1.84; 95% CI, 1.49-2.28) and marginally increased compared with T2DM patients alone (HR, 1.23; 95% CI, 0.99-1.52).[74]

Epidemiological studies have supported a close correlation between DM and osteoporosis or fractures, suggesting that underlying mechanisms of DM or DM-related complications may cause malfunction of bone structure. It reported that DM can compromise bone metabolism, impair osteocytes or osteoblasts function (a reduction of alkaline phosphatases activities, as an example) or damage the extracellular matrix (ECM) due to over inflammation and oxidative stress as well as dysregulation of adipokine and incretin or other cytokines; and additionally, obesity, insulin resistance, abnormal blood sugar, increased bone marrow adiposity, overproduction of AGEs with resultant the formation of collagen-AGEs to alter the ECM, dysfunction of muscle and connective tissue, and microvascular and macrovascular disorders further result in malfunction of normal bone turnover, change of bone microarchitecture, decreased mineralization and decreased bone toughness, contributing to osteoporosis, and bone fragility in DM.[75–78] Blood sugar-related to the bone health has been reported before. Study attempting to determine the negative impact of blood sugar on BMD and TBS in T2DM showed that good glycemic compensation with glycated hemoglobin (HbA1c) value <7.0% did not lead to BMD changes but had significantly better TBS.[57] However, there was a negative correlation between TBS and HbA1C with glycemic fasting.[57] Moreover, several large-scale population-based cohort studies have also demonstrated the close link between poor glycemic control and fracture risks.[79,80]

Although fracture and fracture-related mortality are significantly increased in DM compared to in non-DM, there are still many confounding factors modifying the risk, which included sex, age, rate, fracture sites, study design, and geographical regions. Additionally, as shown above, T1DM has a much higher risk of fracture compared to T2DM. All hint the antidiabetic treatment may also influence the development of fracture in T2DM patients. One meta-analysis showed insulin was associated with a significantly increased risk of fracture in T2DM (RR, 1.24; 95% CI, 1.07-1.44), contributing to the conclusion that treatment with insulin increased the risk of fractures among T2DM patients compared with oral ADA,[81] suggesting that antidiabetic drugs may also play a certain degree of positive or negative impact on the prevention of bone fractures in these T2DM patients.

Oral ADA includes sulfonylurea (glyburide, glipizide, and glimepiride), biguanide (metformin), α-glucosidase inhibitors (acarbose and miglitol), meglitinides (repaglinide and nateglinide), thiazolidinediones (pioglitazone and rosiglitazone), dipeptidyl peptidase-4 (DPP-4) inhibitors (DPP-4i, sitagliptin, saxagliptin, linagliptin, and alogliptin), glucagon-like peptide-1 receptor agonists (GLP-1 receptor agonists), and sodium-glucose cotransporter type 2 (SGLT2) inhibitors (canagliflozin, empagliflozin, dapagliflozin, and ertugliflozin).[9,10,44,82]

5. ANTIDIABETIC AGENTS AND BONE

A recent meta-analysis[82] addressing the effects of ADA on fracture reduction may provide very interesting findings about the current topic – could we prescribe one medication (ADAs) to treat existing diseases (DM) and reduce the diseases-related morbidities or concomitant comorbidities (osteoporosis or fracture)? The enrolled ADAs include DPP-4i, GLP-1 receptor agonists, sulfonylurea, thiazolidinediones, SGLT2 inhibitors, and other nonspecified.[82] However, although the results of every study seemed to be varied greatly, after a summation of data to offer the results from a meta-analysis, the final outcomes seemed to be relatively consistent. That is to say, there is absent of any statistically significant difference of nearly all ADAs in reduction of bone fractures. Similarly, nearly all ADAs are also not associated with statistically significantly increased risks of fractures. However, the interpreted data are interesting, although they do not reach the statistical significance. The use of some ADA favored the trend of decreased risk of fractures, but by contrast, some of them tend to increase the risk of fractures, regardless of which categories or which agent at the same category were prescribed.

In term of DPP-4i,[82–89] almost all are balanced for fracture reduction, including vildagliptin (RR, 1.17; 95% CI, 0.23-6.16), sitagliptin (RR, 1.29; 95% CI, 0.27-6.47), omarigliptin (RR, 1.33; 95% CI, 0.21-8.24), saxagliptin (RR, 2.04; 95% CI, 0.38-12.09), linagliptin (RR, 0.9; 95% CI, 0.18-4.66), and alogliptin (RR, 0.76; 95% CI, 0.12-4.87); however, trelagliptin seemed to significantly increase the risk of fracture with RR of 3.51 (95% CI, 1.58-13.70).[82]

In terms of sulfonylureas, the data also failed to support the benefits of fracture reduction, although the trend seemed to favor the use of this type of medication for fracture reduction during DM control, including glimepiride (RR, 0.45; 95% CI, 0.31-4.25), glipizide (RR, 0.67; 95% CI, 0.12-3.74), gliclazide (RR, 0.75; 95% CI, 0.05-9.46), and glibenclamide (RR, 0.98; 95% CI, 0.22-4.25).[82–84]

In terms of thiazolidinediones, risk of fracture seemed to be increased, although no statistically significant difference was achieved, including pioglitazone (RR, 1.14; 95% CI, 0.31-4.25) and rosiglitazone (RR, 1.20; 95% CI, 0.21-6.83).[82,83]

In terms of SGLT2 inhibitors, similar to DPP-4i, the results seemed to be varied, although most still failed to show any statistically significant difference between the use of SGLT2 inhibitors and non-SGLT2 inhibitors users. The canagliflozin (RR, 0.62; 95% CI, 0.13-3.08) and dapagliflozin, (RR, 0.9; 95% CI, 0.16-5.14) seemed to be favorable for fracture reduction, but the empagliflozin (RR, 1.19; 95% CI, 0.24-5.89) and ertugliflozin (RR, 2.47; 95% CI, 0.16-9.95) seemed to increase the fracture rate in the current SGLT2 inhibitor users.[82,86]

Although in Taiwan, DPP-4 inhibitors (DPP-4i) effect on the bone health in T2DM patients seemed to be supported by large-scale population-based study as shown by Dr. Chang's group,[56] and additionally, DPP-4 has been extensively reviewed to provide the possible benefits on bone and subcutaneous tissue (extracelluar matrix), which are key factors of bone health.[89,90] Similar to many large-scale population-based studies,[31,56,63,83–86,91–98] many biases, which cannot be totally excluded, may decrease the reliability and reproducibility of the data analysis. Additionally,

DPP-4i may increase the risk of cholecystitis in randomized controlled trials, especially with longer treatment duration,[99] although the population-based cohort study did not support the current use of DPP-4i was associated with an increased risk of bile duct and gallbladder disease compared with current use of at least 2 oral ADA (adjusted HR, 0.99; 95% CI, 0.75-1.32).[100] By contrast, the risk of bile duct and gallbladder disease seemed to be increased in DM treated with GLP-1 receptor analogues currently.[100] GLP-1 receptor analogues were associated with an increased risk of bile duct and gallbladder disease compared with current use of at least two oral ADA (adjusted HR, 1.79; 95% CI, 1.21-2.67).[100] Furthermore, GLP-1 receptor analogues were associated with an increased risk of cholecystectomy (adjusted HR, 2.08; 95% CI, 1.08-4.02),[100] suggesting that the benefits or risks of ADA may be more complicated than we expected before. Any claim about the extra-glycemic effects of ADA should be interpreted with caution, and all prescribed medications require more attention from physicians in routine clinical practice.

6. PREVENTION AND MANAGEMENT OF OSTEOPOROSIS IN T2DM: MODIFICATION OF LIFESTYLE

Based on uncertainty of ADA on the prevention of osteoporosis and reduction of fracture, the aim of the current review focusing on "To do one and to get more" therapeutic strategy may be supported by current evidence limiting the extra-sugar–lowering effects of ADA. Fortunately, modification of lifestyle to establish a healthy lifestyle may be working. For example, recent evidence favors that exercise can promote musculoskeletal development, based on various kinds of exercise models to study bone mass and epidemiological study from postmenopausal women.[101–103] Additionally, a recent meta-analysis suggested "sit-to-stand test" and "timed up and go" in favor of exercise intervention as a therapy in patients with DM and sarcopenia based on the results with a weighted mean difference (MD) of –1.57 (95% CI, –2.26 to –0.87) and MD of –0.61 (95% CI, –1.21 to –0.01), respectively, suggesting that exercise intervention as important part of a relevant therapy for DM patients with sarcopenia.[104] In fact, sarcopenia, implicated as both a cause (weaker glucose disposal and reduced metabolic rate and physical activity) and a consequence of DM (micro- and macrovascular complications to damage cellular function and cause cell death leading to loss of skeletal muscle mass and impairment of muscle function and strength), may be involved in accelerated loss of muscle component and impaired function associated with declining carrying out daily activities, as well as physical incapability, and tendency to falls and fracture, and subsequent contribution to mortality.[104] Additionally, physical training (exercise), based on the type, intensity, and duration of exercise, may improve mitochondrial function of muscle, attenuates oxidative stress, and increased the antioxidant capacity in muscle, and all may increase muscle mass and strength against the "fall" accident.[105]

Furthermore, it is reported that T2DM patients may be at higher risk of poor cognitive perspective of osteoporosis because recent study showed that these T2DM patients expressed a low self-efficacy for both exercise and calcium intake experience.[106] Moreover, other components of healthy lifestyle may be also important for all patients with chronic illnesses, including pre-DM and DM patients to prevent the occurrence of osteoporosis and reduce fracture, such as adequate nutritional support, vitamin D supplementation, calcium replacement, and essential micronutrition supports.[29,30,48,107–110] In fact, there are many agents also available in the prevention and reduction of risk of osteoporosis and fracture in DM patients[64,65,111]; however, many physicians may overlook the risk of disorders of musculoskeletal system compared to those called vital organs, such as cardiovascular, neurological and renal systems. Furthermore, too many prescriptions may result in low compliance of T2DM patients, and drug and drug interaction may result in unpredictive risk of drug-related adverse events. Therefore, to minimize the risk of the aforementioned dilemma, the current review emphasizes the urgent need of bi-, tri-, and multiextra–sugar-lowering effects of ADA.

In conclusion, with better understanding of underlying pathophysiology of DM, the field of DM therapy is entering a new era. Since the cure is nearly impossible for DM treatment in the current time, the DM control needs lifelong struggle and treatment.[112,113] The prescription of ADA should be according to the patient's age, patient's compliance, patient's general condition, disease course, drug efficacy, and drug-related side effects.[10,44] Therefore, the development of new T2DM drugs with better efficacy, fewer side effects and potential repositioning for any new indications or concomitant benefits for health promotion is a problem that researchers have been actively addressing.[44]

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