Nontraumatic Fracture Risk With Diabetes Mellitus and Impaired Fasting Glucose in Older White and Black Adults

The Health, Aging, and Body Composition Study

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Background: Diabetes mellitus (DM) and related complications may increase clinical fracture risk in older adults.

Methods: Our objectives were to determine if type 2 diabetes mellitus or impaired fasting glucose was associated with higher fracture rates in older adults and to evaluate how diabetic individuals with fractures differed from those without fractures. The Health, Aging, and Body Composition Study participants were well-functioning, community-dwelling men and women aged 70 to 79 years (N = 2979; 42% black), of whom 19% had DM and 6% had impaired fasting glucose at baseline. Incident nontraumatic clinical fractures were verified by radiology reports for a mean ± SD of 4.5 ± 1.1 years. Cox proportional hazards regression models determined how DM and impaired fasting glucose affected subsequent risk of fracture.

Results: Diabetes mellitus was associated with elevated fracture risk (relative risk, 1.64; 95% confidence interval, 1.07-2.51) after adjustment for a hip bone mineral density (BMD) and fracture risk factors. Impaired fasting glucose was not significantly associated with fractures (relative risk, 1.34; 95% confidence interval, 0.67-2.67). Diabetic participants with fractures had lower hip BMD (0.818 g/cm² vs 0.967 g/cm²; P < .001) and lean mass (44.3 kg vs 51.7 kg) and were more likely to have reduced peripheral sensation (35% vs 14%), transient ischemic attack/stroke (20% vs 8%), a lower physical performance battery score (5.0 vs 7.0), and falls (37% vs 21%) compared with diabetic participants without fractures (P < .05).

Conclusions: These results indicate that older white and black adults with DM are at higher fracture risk compared with nondiabetic adults with a similar BMD since a higher risk of nontraumatic fractures was found after adjustment for hip BMD. Fracture prevention needs to target specific risk factors found in older adults with DM.

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OSTEOPOROSIS IN PEOPLE with diabetes is poorly understood. Recent data from prospective cohorts in the United States, Norway, and Australia indicate that adults with type 2 diabetes mellitus (DM) may have a higher risk of fracture compared with nondiabetic adults, in spite of the higher bone mineral density (BMD) and body weight that is associated with diabetes. Most evidence exists for older white women compared with older white men and black adults. Although type 2 DM is more prevalent among older black than white adults in the United States, it is not known whether the effect of DM on fracture risk is similar in this age group. The relationship of impaired fasting glucose (IFG), or prediabetes, to fracture risk in older adults is uncertain because previous studies did not measure fasting plasma glucose (FPG) levels or perform separate analyses in nondiabetic adults.

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Underlying factors that may contribute to the higher fracture risk in older diabetic adults, given their higher body weight and higher BMD compared with nondiabetic adults, are not clear. Falling and impaired motor abilities are more common in diabetic adults and might explain some of the increased risk for fractures. Based on findings from previous cohorts, possible explanations for higher fracture risk in older adults with
diabetes may be diabetes-related medication use,\textsuperscript{2-5} longer disease duration,\textsuperscript{1,4} and/or complications such as impaired vision\textsuperscript{1,4} or neurologic impairments (eg, neuropathy and stroke).\textsuperscript{1,2}

The objectives of the present study were to determine (1) if type 2 diabetes mellitus or IFG is independently associated with a higher risk for clinical fractures for white and black men and women and (2) if individuals with both type 2 DM and fractures have characteristics similar to those with type 2 DM without fractures.

**METHODS**

**STUDY PARTICIPANTS**

Participants were 3075 well-functioning older white and black adults (48% male and 42% black), aged 70 to 79 years, in the Health, Aging, and Body Composition Study (Health ABC), a prospective cohort study investigating changes in body composition as a common pathway by which multiple diseases contribute to disability. Recruited participants were a volunteer group from a large mass mailing to a random sample of white Medicare beneficiaries and all age-eligible black community residents in Pittsburgh, Pa, and Memphis, Tenn, with a baseline examination in 1997-1998. Eligible participants reported no difficulty walking a quarter of a mile (400 m), climbing 10 steps, or performing activities of daily living; were free of life-threatening cancers with no active treatment within the past 3 years; and planned to remain within the study area for at least 3 years. Participants provided informed consent prior to examinations, approved by the institutional review boards at the University of Pittsburgh and the University of Tennessee Health Science Center. We excluded participants with missing diabetes or IFG status (n=22), diabetes onset in childhood (≤20 years old) (n=9), or oral steroid use (n=69), and 2979 participants remained (914 white men, 542 black men, 816 white women, and 707 black women).

**DIABETES MELLITUS**

Diabetes mellitus was defined as self-report of physician diagnosis, hypoglycemic medication use, or FPG level of 126 mg/dL or higher (≥7.0 mmol/L), and IFG was defined as a fasting glucose level of 110 mg/dL or higher (≥6.1 mmol/L) but lower than 126 mg/dL (<7.0 mmol/L), based on American Diabetes Association 2003 criteria.\textsuperscript{20} Nondiabetic participants had FPG levels lower than 110 mg/dL (<6.1 mmol/L). Baseline clinic measures also included a 75-g oral glucose tolerance test only for those not using diabetes medications), fasting insulin (only for those not using exogenous insulin), and glycosylated hemoglobin as previously reported.\textsuperscript{21} Poor glycemic control was defined as a glycosylated hemoglobin level of 7% or higher.\textsuperscript{20} Preexisting diabetes, diabetes duration, and hypoglycemic medication use were determined by an interviewer-administered questionnaire.

**FRACTURE OUTCOME**

Incident nontraumatic clinical fractures were assessed every 6 months by self-report and verified by radiology reports for a mean ± SD of 4.5 ± 1.1 years of follow-up. Nontraumatic fractures were defined as those occurring spontaneously or from modest trauma (eg, fall from a standing height). Based on medical record review, fractures were excluded if they were due to excessive trauma (n=18; eg, motor vehicle crash), a pathologic condition (n=4; eg, cancer), or stress (n=7), or were of other/unknown cause (n=19).

**BMD AND BODY COMPOSITION AT BASELINE**

Height was measured using a stadiometer and weight was measured with a calibrated balance beam scale. Body mass index (BMI) was calculated as weight divided by the square of height in meters. Total hip BMD (in grams per centimeter squared), whole body bone mineral-free lean mass and fat mass were assessed at both field centers by dual-energy x-ray absorptiometry (Hologic 4500A, software version 9.03; Hologic Inc, Bedford, Mass). Quality assurance measurements of dual-energy x-ray absorptiometry performed at both study sites ensured scanner reliability and identical scan protocols. Computed tomography (CT) in Pittsburgh (General Electric 9800 Advantage; General Electric, Milwaukee, Wis) and Memphis (Siemens Somatom Plus; Siemens Medical Systems, Iselin, NJ; or Picker PQ2000S; Marconi Medical Systems, Cleveland, Ohio) of the abdomen was used to measure the abdominal visceral fat area (in centimeters squared) at the L4-L5 disk space as previously described.\textsuperscript{22} Computed tomography of the spine L3 region was conducted only in Pittsburgh to obtain bone volume and volumetric trabecular BMD (in milligrams per milliliter). Computed tomography data were analyzed with a standardized protocol at the University of Colorado Health Sciences Center, Denver, for soft tissue and at the University of California, San Francisco, for BMD.\textsuperscript{23,24}

**OTHER COVARIATES**

Health and medical histories (smoking, alcohol consumption, and osteoporosis or fractures); weight changes over prior 12 months (gain or loss ≥2.25 kg) and adult life (constant within 4.5 kg, gradual gain or loss >4.5 kg, marked gain or loss >4.5 kg, and fluctuation); diabetes-related complications (cerebrovascular disease [transient ischemic attack or stroke], cardiovascular disease [bypass or coronary artery bypass graft, carotid endarterectomy, myocardial infarction, angina, or congestive heart failure], kidney disease, retinopathy or retinal disease, and cataracts); weekly physical activity in kilocalories from walking and exercise; and falls in prior 12 months were determined by an interviewer-administered questionnaire at baseline. Mediations from the prior week were brought to the clinic. Thiazide diuretic, statin, estrogen, osteoporosis medications (bisphosphonates, calcitonin, raloxifene, and fluoride), and calcium and vitamin D supplement use was coded using the Iowa Drug Information System ingredient codes.\textsuperscript{25} Serum creatinine levels of 1.5 mg/dL or higher (≥132.6 µmol/L) for men and 1.3 mg/dL or higher (≥114.9 µmol/L) for women defined renal insufficiency.\textsuperscript{26} An ankle-brachial index less than 0.9 indicated subclinical cardiovascular disease as previously described.\textsuperscript{27} The Health ABC performance battery was a supplemented version of the lower-extremity performance test used in the Established Populations for the Epidemiologic Studies of the Elderly (chair stands, standing balance, and 6-m walk for gait speed)\textsuperscript{28} with increased test duration, a single foot stand, and a narrow walk test of balance as previously described (score range, 0-12).\textsuperscript{29} Year 4 nerve function available for most participants included self-reported pain or numbness in the legs or feet (n=2314; monofilament testing (n=2291; reduced sensation defined as inability to feel 3 of 4 touches at the great toe for both 1.4-g and 10-g monofilament), vibration threshold in micrometers (n=2252; VSA-3000 Vibratory Sensory Analyzer; Medoc, Chapel Hill, NC); and peroneal motor nerve conduction amplitude in millivolts and velocity in meters per second from the popliteal fossa to the ankle (n=2168 and 2034, respectively; NeuroMax 8; XLTEK, Oakville, Ontario).
Women mellitus; HBA1c, glycosylated hemoglobin; IFG, impaired fasting glucose; NA, not applicable.

Table 1. Baseline Descriptive Characteristics by DM and IFG Status for Men and Women

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>DM</th>
<th>IFG</th>
<th>No DM</th>
<th>DM</th>
<th>IFG</th>
<th>No DM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 323)</td>
<td>(n = 106)</td>
<td>(n = 1027)</td>
<td>(n = 243)</td>
<td>(n = 71)</td>
<td>(n = 1209)</td>
</tr>
<tr>
<td>Black race, %</td>
<td>46.7*</td>
<td>35.8</td>
<td>34.4</td>
<td>71.2*</td>
<td>52.1</td>
<td>41.1</td>
</tr>
<tr>
<td>Age, median, y</td>
<td>74.0</td>
<td>74.0</td>
<td>73.0</td>
<td>73.0</td>
<td>73.0</td>
<td>73.0</td>
</tr>
<tr>
<td>Fractured after age 45 y, %</td>
<td>16.8</td>
<td>14.4</td>
<td>16.2</td>
<td>18.5†</td>
<td>27.5</td>
<td>29.3</td>
</tr>
<tr>
<td>Current smoker, %</td>
<td>9.6</td>
<td>9.4</td>
<td>11.4</td>
<td>9.9</td>
<td>9.9</td>
<td>10.2</td>
</tr>
<tr>
<td>Current drinker, %</td>
<td>45.7*</td>
<td>64.2</td>
<td>60.5</td>
<td>21.8*</td>
<td>53.5</td>
<td>45.9</td>
</tr>
<tr>
<td>Oral estrogen use, %</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>15.6†</td>
<td>12.7†</td>
<td>23.3</td>
</tr>
<tr>
<td>Osteoporosis medication use, %</td>
<td>1.2</td>
<td>1.9</td>
<td>0.6</td>
<td>4.1†</td>
<td>4.2</td>
<td>8.0</td>
</tr>
<tr>
<td>Calcium supplement use, %</td>
<td>6.2</td>
<td>7.5</td>
<td>7.3</td>
<td>15.2*</td>
<td>12.7*</td>
<td>31.3</td>
</tr>
<tr>
<td>Vitamin D supplement use, %</td>
<td>2.5</td>
<td>4.7</td>
<td>4.1</td>
<td>7.0*</td>
<td>1.4†</td>
<td>14.8</td>
</tr>
<tr>
<td>Fallen in past year, %</td>
<td>19.3</td>
<td>16.0</td>
<td>18.1</td>
<td>26.0</td>
<td>21.1</td>
<td>24.1</td>
</tr>
<tr>
<td>Performance battery (0-12), median score</td>
<td>7.0*</td>
<td>8.0</td>
<td>8.0</td>
<td>6.0*</td>
<td>7.0</td>
<td>7.0</td>
</tr>
<tr>
<td>Height, median, cm</td>
<td>173.2</td>
<td>172.3</td>
<td>173.4</td>
<td>159.5†</td>
<td>160.2</td>
<td>159.4</td>
</tr>
<tr>
<td>Weight, median, kg</td>
<td>84.0*</td>
<td>83.4†</td>
<td>79.0</td>
<td>77.2*</td>
<td>77.1*</td>
<td>67.3</td>
</tr>
<tr>
<td>BMI, median</td>
<td>28.0*</td>
<td>27.7*</td>
<td>26.3</td>
<td>30.1*</td>
<td>30.0*</td>
<td>26.2</td>
</tr>
<tr>
<td>Lost ≥2.25 kg, past year, %</td>
<td>47.9†</td>
<td>40.4†</td>
<td>27.3</td>
<td>52.3*</td>
<td>33.3</td>
<td>32.4</td>
</tr>
<tr>
<td>Fasting insulin, median, µIU/mL</td>
<td>8.4†</td>
<td>9.3†</td>
<td>6.3</td>
<td>10.1*</td>
<td>10.8*</td>
<td>6.7</td>
</tr>
<tr>
<td>HbA1c, mean ± SD, %</td>
<td>7.9 ± 1.5*</td>
<td>6.4 ± 0.8*</td>
<td>6.0 ± 0.5</td>
<td>7.9 ± 1.6*</td>
<td>6.5 ± 0.6*</td>
<td>6.0 ± 0.5</td>
</tr>
<tr>
<td>Total hip BMD, median, g/cm²</td>
<td>1.013*</td>
<td>0.989†</td>
<td>0.945</td>
<td>0.868*</td>
<td>0.866*</td>
<td>0.782</td>
</tr>
<tr>
<td>Bone mineral-free lean mass, median, kg</td>
<td>57.1*</td>
<td>55.7</td>
<td>54.2</td>
<td>43.4*</td>
<td>42.3*</td>
<td>38.7</td>
</tr>
<tr>
<td>Total fat mass, median, kg</td>
<td>24.3*</td>
<td>24.3†</td>
<td>21.9</td>
<td>30.8*</td>
<td>31.3*</td>
<td>26.7</td>
</tr>
<tr>
<td>Visceral fat, median, cm²</td>
<td>156.2*</td>
<td>163.3†</td>
<td>138.0</td>
<td>157.4*</td>
<td>154.2*</td>
<td>113.9</td>
</tr>
</tbody>
</table>

Abbreviations: BMD, bone mineral density; BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); DM, diabetes mellitus; HbA1c, glycosylated hemoglobin; IFG, impaired fasting glucose; NA, not applicable.

*P<.001 for patients with DM or IFG vs nondiabetic participants within sex groups.
†P<.05 for patients with DM or IFG vs nondiabetic participants within sex groups.

STATISTICAL ANALYSES

Participants were followed prospectively for fracture outcomes, and other data were from the baseline study visit, with the exception of year 4 nerve function measures. Incidence rates were calculated per 1000 person-years. Differences in prevalence and univariate associations between participants with DM or IFG and nondiabetic participants were tested separately by sex using Pearson χ² methods. Fisher exact methods were used when the expected value of any cell was less than 5. For continuous variables, partial correlation coefficients were computed and nonparametric 1-way Mann-Whitney tests were performed for comparisons between patients with DM or IFG and nondiabetic participants.

Additional known and hypothesized risk factors for fracture and diabetes were included in models as possible confounders or mediators for the association of diabetes and fractures. Cox proportional hazards regression models determined the independent effect of DM or IFG on risk of incident fracture, while adjusting for age, race, sex, site (Memphis or Pittsburgh), total hip BMD, bone volume, falls, lean and fat mass, visceral fat, fasting insulin, serum creatinine, smoking, drinking, Health ABC performance battery categorical score (0-12), weekly physical activity from walking and exercise, gain or loss of 2.25 kg or more in past 12 months, adult weight changes, current drinker, current smoker, race, clinic site, and bone-active medication use. Diabetes-related complications (history of cardiovascular disease, transient ischemic attack or stroke, kidney disease, retinopathy or retinal disease, cataracts, and nerve function [pain or numbness in legs or feet, monofilament detection, vibration threshold, and nerve conduction amplitude and velocity]) were entered in the model to assess their role as potential mediators. Date of first fracture or last follow-up if no fracture occurred was used to censor data. Models were built progressively by entering variables in the following order: demographics, hip BMD, body composition factors, other potential confounders, and diabetes-related complications. Sex, race, clinic site, and diabetes variables were forced into the model, and other variables were removed at P>.10. Given the low number of fractures, multivariate analyses for fracture risk including only diabetic participants were not performed. Data were analyzed using SPSS statistical software (SPSS Inc, Chicago, Ill).

RESULTS

Overall, 19% (n = 566) of 2979 participants had DM and 6% (n = 177) had IFG. Black men had the highest prevalence of DM (28%), followed by black women (25%), white men (19%), and white women (9%). Men and women with DM or IFG had significantly higher unadjusted total hip BMD compared with their nondiabetic counterparts of the same sex (Table 1). Participants with DM or IFG had significantly higher weight, BMI, lean mass, fat mass, visceral fat, and fasting insulin level compared with participants without DM for each sex (Table 1). Associations for BMD and body composition were consistent for each race-sex group (data not shown). Participants with DM and men with IFG were more likely to have lost 2.25 kg or more in the year prior to baseline examination compared with nondiabetic participants. Diabetic women were less likely to report a fracture after age 45 years or use bone-active medication compared with nondiabetic women. Bone-active medication use was also lower among women with IFG. Nontraumatic fracture incidence per 1000 person-years was 11.7 for diabetic participants, 11.4 for those with IFG, and 12.4 for those without DM. The most common
fracture sites were the radius/ulna (21%), spine (18%), hip (18%), tibia/fibula/ankle (10%), and foot (9%) and did not significantly differ by diabetes or IFG status.

MULTIVARIATE COX PROPORTIONAL HAZARDS REGRESSION ANALYSES

Diabetes mellitus, although not IFG, was significantly associated with higher risk of fractures compared with not having DM (Table 2), with diabetic participants having a 64% increase in fractures (relative risk, 1.64; 95% confidence interval, 1.07-2.51). The risk of fracture with IFG was approximately half of that for DM but not significant (relative risk, 1.34; 0.67-2.67). The higher fracture risk among diabetic participants was observed after adjusting for either the higher BMD or higher lean mass and fat mass. Analyses with addition of other covariates listed in the methods were not significant, did not affect the association between diabetes and fractures, and were therefore excluded from the final model. Diabetes-related complications were not related to fracture risk in the models. Removal of clinical vertebral fractures (n=30) or participants with DM taking insulin (n=122) or inclusion of additional individuals with 200-mg/dL or greater (≥11.1-mmol/L) 2-hour plasma glucose levels from the 75-g oral glucose tolerance test (n=136) did not change results. For Pittsburgh participants with available data (n=1446), bone volume was not related to fracture. There was no interaction between either sex or race and diabetes or IFG, so modeling was not performed separately by sex or race.

PARTICIPANTS WITH DM

Oral hypoglycemic agents and insulin were used by 46% and 22%, respectively, of participants with DM. Blacks used insulin more frequently compared with whites (27% vs 15%; P<.001), but there were no race or sex differences for use of oral hypoglycemic medications. Many participants had a short duration of DM (47% ≤ 5 years; median, 6.0 years), and 27% of white men, 21% of black men, 17% of white women, and 14% of black women did not report a prior diagnosis. Blacks had slightly longer median DM duration compared with whites (8.0 years vs 5.0 years; P=.07), but women and men had similar lengths of duration.

Compared with those without fractures, diabetic participants with fractures were more likely to be women (70% vs 41%; P<.01) but were similar in race. Diabetic participants with incident fractures had lower total hip BMD (0.818 g/cm² vs 0.967 g/cm²; P<.001) and lean mass (44.3 kg vs 51.7 kg; P=.001) but no difference in fat mass (28.9 kg vs 26.8 kg; P=.55) compared with those without fractures. Reduced peripheral sensation, falls and recurrent falls (year prior to baseline examination), lower physical performance battery scores, and history of transient ischemic attack or stroke were significantly more prevalent in the diabetic participants with incident fractures; however, poor glycemic control, longer DM duration, and insulin use were not significantly different (Table 3).

In this biracial cohort of elderly men and women, type 2 diabetes mellitus was associated with a 64% increase in...
incident clinical fractures compared with nondiabetic participants, after adjustment for the higher BMD, lean mass and fat mass, and greater weight changes among participants with DM. Our results suggest that diabetic participants with similar BMD as nondiabetic participants were more likely to have a fracture. Prior longitudinal studies of diabetes and fracture risk did not include older black adults, although similar age-adjusted increased risks of hip fracture were found for older white and Mexican American adults with type 2 DM. Hip fracture rate was not higher among older Norwegian white men with a duration of DM longer than 5 years, but adjustment for BMD was not done. Our previous work indicated that diabetic adults in this cohort had significantly lower spine bone volume compared with nondiabetic adults, which could affect bone strength, but did not account for increased fractures in diabetic participants.

We are aware of 1 previous report on the association of IFG and prospective fracture from an Australian cohort that found no increased risk of fractures for participants with IFG compared with those with FPG levels lower than 90 mg/dL (≤5 mmol/L), although self-reported or treated diabetic adults were included in each group. We hypothesized that the IFG group may be associated with an intermediate risk for fractures; however, this finding was not statistically significant likely owing to the small number of fractures among participants with IFG.

We did not observe a significant mediating effect of diabetes-related complications on fracture risk in multivariate analyses including both diabetic and nondiabetic participants, possibly because of low power. An Australian study reported that diabetic retinopathy and cataracts were associated with an increased risk of fracture in diabetic adults, though they did not control for other diabetes-related complications in their analyses. Other studies did not find a relationship with impaired vision. Loss of protective sensation, more frequent falls, lower physical performance battery scores, and greater history of transient ischemic attack or stroke were found for the diabetic adults with vs without fractures, suggesting a role for neurologic or motor impairments. One previous study noted that increased risk of fracture associated with diabetes was not significant after adjustment for stroke, motor ability, and impaired vision. Stroke, even years after the event occurs, is a well-recognized risk for fracture because it increases the risk of falling and motor impairments. Falls are also more common among older adults with poor nerve function and foot problems, which occur more often with neuropathy, were linked to fractures in a large multiracial case-control study. The Study of Osteoporotic Fractures found that loss of protective sensation was associated with increased risk of nonvertebral fracture in older white women in addition to diabetes. Further prospective studies are needed to confirm that diabetes complications place older diabetic adults at higher risk for fracture.

Insulin use did not account for the higher fracture rate in the diabetic participants, and the risk remained the same when insulin users were excluded, which is similar to findings from the Norwegian prospective study. This result is in contrast to several cohorts in which diabetic adults using insulin had increased fracture risk, particularly for foot and hip fractures. Even when we compared insulin use within participants with DM, those with fractures were not more likely to use insulin. Our exclusion of diabetic participants with a childhood onset of disease ensures that most of this older cohort had type 2 DM.

Diabetic participants with and without fractures were similar in their glycemic control (as measured by glycated hemoglobin) and disease duration. Poor glycemic control was not reported in most assessments of fracture risk for older adults with DM, although a glycated hemoglobin level higher than 9.5% was not found to be related to hip fracture risk in a Norwegian cohort. Diabetes duration longer than 10 to 15 years increased fracture risk in most previous reports. We do not know why duration was not related to fracture risk in our study, although our study participants were from a narrow age cohort and may not have the range in duration compared with others studies. As follow-up continues, we may accrue sufficient numbers of fractures to look further at a duration effect.

Our study is unique in its inclusion of white and black adults, stratification of fracture risk by FPG categories, radiologic confirmation of fracture outcomes, and wide range of diabetes-related complications. Nevertheless, our study has several limitations. Our data are for clinical fractures only and individuals with subclinical vertebral fractures would not be included. Some individuals with type 2 DM may not have participated in our study because of greater physical disability, though adjustments were made for performance battery scores and physical activity. It is likely that diabetes severity could increase fracture risk even though we assessed diabetes severity through disease duration, glycemic control, insulin use, and complications.

In conclusion, diabetic adults with a similar BMD to nondiabetic adults had a 64% higher risk for fracture. Diabetes-related complications may mediate this increased fracture risk. Given the large secular increases in the prevalence of diabetes, perhaps secondary to the current epidemic of obesity, higher fracture risk among adults with DM may have important public health consequences as the population ages. Adults with DM, already burdened with a greater risk for physical disability, may further decrease their function if they sustain fractures. Older adults with DM may benefit from fracture prevention efforts if they have greater risk factors for fractures and specific diabetic complications.

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