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Diabetes and bone: an unexpected but intense relationship

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s a title of this editorial indicates, we have described the association between diabetes mellitus (DM) and osteoporosis as unexpected but intense. However, perhaps we should first say "still controversial for some." Today the proportion and intensity with which the disorders described in the quantity and quality of bone. In addition, alterations in metabolism are still under discussion. mineral, are associated with both type 1 diabetes (DMt1) and type 2 (DMt2), and influence an increase in the rate of fractures¹.

We know that diabetes juvenile onset may be associated with a reduction in the peak of bone mineral density (BMD), with the consequences that this entails for fractures at older ages. In addition, a greater than expected BMD has been described in DMt2, unlike DMt1, in which a reduction in BMD has been reported, especially associated with the appearance of chronic complications. In the current model, T2DM implies greater involvement of bone quality compared to reduced BMD, which would play a secondary role. In fact, there have been proposals to increase fracture risk estimates based on BMD (by multiplying them by up to 2)2, due to the low predictive value of the former. Likewise, functional hypoparathyroidism has been described in people with DM, in addition to disorders secondary to the appearance of nephropathy or other chronic complications.

Finally, we cannot ignore the increased risk of falls secondary to neuropathy, visual disturbances, cerebrovascular disease or hypoglycemia itself, which cause many of the most classic treatments for diabetes, such as sulfonylureas and other secretagogues or insulin¹.

In addition, people with diabetes suffer an unexpectedly high number of fractures in the appendicular skeleton (arms, ankles, legs...), so a role for neuropathy, and even microangiopathy localized in these areas, has been suggested.

In this issue, Martínez-Laguna et al.³ seek to determine if there are differences in the use of drugs bet-

ween people with T2DM and without diabetes using the powerful database of Primary Care in Catalonia (Information System for the Development of Research in Primary Care, SIDIAP). When selecting subjects with DMt2 older than 50 years and matching them with two similar non-diabetic groups, the analysis of their clinical characteristics and treatments yielded very interesting data.

First, a fracture rate was corroborated –prevalence actually– much higher than expected –in fact, almost excessively high: 1.3% vs 0.3% in subjects without DM–, but what is even more troubling, even with this enormous prevalence of fractures (which we must assume as osteoporotic in the main), is that the use of, for example, bisphosphonates was 30% lower in people with diabetes, which is unacceptable.

The multivariate analysis encouraged to clear the role of confusing variables, confirmed that the diabetic sufferer had a lower probability of being treated for osteoporosis.

Therefore, in addition to welcoming this new interesting research by the group led by Daniel Prieto Alhambra, we also remember that there are updated recommendations for assessing osteoporosis secondary to endocrine diseases², and even specific recommendations on anti-diabetic treatments and their impact on fracture risk⁴, promoted by the Working Group on Osteoporosis and Metabolic Bone Diseases of the Spanish Society of Endocrinology. We again recommend this work to all those concerned with treatment of osteoporosis and diabetes, a pair of conditions with a much more intense relationship than expected.

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