



Screening men for osteoporosis

I have many middle-aged and elderly men in my family practice, including some who are being treated for osteoporosis, and thus I read with interest the *CMAJ* article on managing osteoporosis in men by Aliya Khan and colleagues.¹ I was surprised by the authors' statement that "as in the 2002 guidelines [from the Osteoporosis Society of Canada], [bone mineral testing] for all men over 65 is advised."

The article did not provide any evidence to support this recommendation, so I turned to the cited guidelines.² The first sentence in the section on osteoporosis in men states that "there are insufficient data on the relation between [bone mineral density] and fracture risk in men." Neither the guidelines nor the article by Khan and colleagues provides information on the incidence of osteoporosis in men. It would seem to me that any further discussion should be postponed until such data are available. However, both documents go on to recommend screening for men over 65 years of age.

How can one propose screening for a disease when the incidence of the condition is unknown in the population in question and when use of the recommended screening tool cannot as yet be correlated with disease detection? Furthermore, no evidence was provided concerning the cost of screening, the number of cases of osteoporosis that would be diagnosed by screening all men (rather than only men at

high risk of developing the disease) and the number of subsequent fractures that would be prevented by screening.

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[The authors respond:]

We thank Dara Behroozi for raising an important point concerning our article.¹ Unfortunately, the data currently available on the cost-effectiveness of bone densitometry in men are very limited. Schousboe and colleagues recently evaluated the cost-effectiveness of bone densitometry followed by 5 years of oral bisphosphonate therapy to prevent fractures among older men with osteoporosis in the United States.² They concluded that bone densitometry for all men as young as 70 years of age may be cost-effective. This assumes a societal willingness to pay US\$100 000 per quality-adjusted life-year gained in addition to drug costs. The Osteoporosis Society of Canada's guidelines emphasize assessing key clinical risk factors for fracture (e.g., previous fracture history and glucocorticoid use) in conjunction with bone mineral density testing to determine treatment thresholds.³ In our article, we recommend targeting those at high risk of developing a fragility fracture for pharmacological intervention, which would improve the cost-effectiveness of treatment.

On the basis of current expert opinion, the Osteoporosis Society of Canada's recommendations for bone mineral density testing and clinical risk factor assessment are appropriate. It is true that in the absence of a defining fracture event, a low bone mineral density alone places a man at less risk for fracture than a woman of similar age with a similar bone mineral density. White men at age 60 have a 29% risk of experiencing a fracture.⁴ One-third of all hip fractures occur in men; they are associated with equivalent morbidity and higher mortality compared with those occurring in women.⁵ Vertebral fractures are also associated with similar morbidity in men and women.⁶ More detailed Canadian studies will help to assess the cost-effectiveness of bone mineral density screening in men. The Osteoporosis Society of Canada is in the process of completing an exhaustive evidence-based review of osteoporosis in men, the results of which will be reflected in future guidelines.

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Accuracy of point-of-care measurements

In a helpful case report in *CMAJ*, Peter Brindley and colleagues reported falsely elevated lactate levels obtained with a point-of-care analyzer and low plasma lactate levels obtained with laboratory testing in a patient who had ingested ethylene glycol.¹ Further investigation showed that the addition of even small amounts of glycolic acid or glyoxylic acid (the 2 predominant metabolites of ethylene glycol) to blood analyzed in the Radiometer ABL 700 point-of-care analyzer resulted in a

marked artifactual elevation in lactate levels. Such elevations were not seen with the other analyzers tested (including the Vitros laboratory analyzer).

We recently reported a comparable observation in a man who accidentally ingested a large amount of propylene glycol.² Arterial blood gas analysis with the Radiometer ABL 700 point-of-care analyzer showed a very high concentration of lactate (up to 39 mmol/L), but a normal reading was obtained with the Vitros laboratory analyzer. However, in our case the falsely elevated reading with the point-of-care analyzer was not caused by any interference with propylene glycol or its metabolites. Analysis with a sensitive and specific D-lactate kit revealed the presence of a very high amount (more than 100 mmol/L) of D-lactate but not L-lactate. Interference in this case was apparently due to the intestinal conversion of the orally ingested propylene glycol into D-lactate, which was erroneously measured as L-lactate by the point-of-care analyzer.

The key objective of point-of-care testing is to generate a result quickly so that appropriate treatment can be implemented, leading to an improved clinical or economic outcome. These 2 cases show that it is crucial, especially in patients intoxicated with glycols, to confirm extreme point-of-care results with laboratory testing.

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