Association between osteoporosis and statins therapy

Statins therapy is found to be associated with a decreased risk of osteoporosis.1 2 Moreover, a number of observational studies reported that statins therapy was associated with a decreased risk of osteoporotic fractures,3 4 but other studies did not.5 6 Some points are discussed here.

First, a study conducted by Leutner et al7 published in Annals of the Rheumatic Diseases reported that low dose of statins therapy was associated with a decreased risk of osteoporosis, but high dose of statins therapy was associated with an increased risk of osteoporosis and maybe subsequent fractures. However, a cohort study reported that people taking high-potency statins such as atorvastatin or rosuvastatin were at lower risk of developing osteoporotic fractures when compared with those taking simvastatin.3 A case–control study by Cheng et al8 reported that current use of statins seemed to have a protective effect against hip fracture in older people (adjusted OR 0.73, 95% CI 0.65 to 0.82).4 There was also a dose-dependent effect of statins use on the protective effect of hip fracture in Cheng et al’s study.4 That is, the higher the dose of statins use, the lower the risk of hip fracture. This finding was not compatible with Leutner et al’s study showing that high dose of statins use was associated with an increased risk of osteoporosis.7 Third, blood lipid and bone mineral density are dynamic change. In clinical practice, the initial levels of blood lipid would determine which potency of statins should be used. Once the treatment goal of blood lipid is achieved, the dose of statins can be reduced. If the treatment goal is not achieved, the dose of statins can be increased. In addition, bone mineral density cannot be measured every day. It is difficult to assess that bone mineral density is affected by low dose or high dose of statins in retrospective studies. Only randomised controlled trials have a chance to investigate whether the dosage or the potency of statins would affect bone mineral density. Then the causation between statins therapy and osteoporosis can be clarified. Fourth, currently there is no research to definitely prove the causal relationship between statins therapy and osteoporosis. The US Preventive Services Task Force does not recommend that persons on statins therapy should screen for osteoporosis to prevent osteoporotic fractures.7 It indicates that at present persons taking statins do not need to worry about the risk of osteoporosis. Finally, I agree with Leutner et al’s comments that future studies should focus on the causal relationship between the dosage-potency of statins and osteoporosis rather than the association.

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