Effects of cinacalcet on vascular calcification in haemodialysis patients

Dear Sir,

We read with great interest the recent report by Floege et al. [1] describing the study design and subject baseline characteristics in the ADVANCE Study, a prospective randomized study with the objective to assess the effects of cinacalcet on progression of coronary artery calcification (CAC) in haemodialysis patients. Waiting for these important results, we want to refer our experience on this topic. Although limited because of the small sample size, as far as we know, no similar description has been previously published. Eleven patients on haemodialysis with secondary hyperparathyroidism (PTHi >400 pg/mL) resistant to conventional treatment began cinacalcet treatment. We measured at that time the CAC score with a multi-detector 16-row helical CT scanner and quantified it in Agatston units with the ‘Syngo Calcium Scoring’ software. Measurements were obtained at baseline, 12 and 36 months after starting cinacalcet. Three patients have been lost to follow-up (two received a kidney transplant and one died) after 1 year. Basal and last measurements for Ca, P, Ca × P and PTHi were 9.73 vs. 9 mg/dL, 5.5 vs. 4.8 mg/dL, 53 vs. 43 and 608 vs. 230 pg/mL, corresponding to an 8%, 13%, 19% and 62% decrease, respectively (P<0.05 for all values). Mean cinacalcet doses stabilized at 50 ± 22 mg/day. The CAC score results were (mean±SE) 1027±346 vs. 1268±418 (Figure 1). This corresponds to an annualized percentage CAC progression of 8%, clearly lower as compared with the 18% reported in the literature [2]. Importantly, these results do not change even if we consider only the five patients with a basal CAC >400 Agatston units (1628±690 vs. 1965±912 at 3 years) as these patients are known to be at particular risk of progression.

These preliminary results are in agreement with those obtained in experiments performed in vitro and in animal models of kidney failure that demonstrate inhibition of vascular calcification progression with calcimimetics. Certainly, CAC is a surrogate marker of cardiovascular events, and the long-term clinical implications of treatment, including patients’ survival, are less clear. To answer this question definitively, we will have to wait for the results of further prospective, long-term randomized controlled trials, such as the EVOLVE study, that focus on cardiovascular events and mortality [3].

Conflict of interest statement. None declared.

Editorial note: Floege et al. had no further comments on this letter.

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Fig. 1. CAC score results.