



Figure 1: Multi-stage deep-learning application for the opportunistic screening of bone's fragility risk from X-ray

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VITAMIN D METABOLITE RATIO (VMR) OFFERS THE BEST APPROACH TO EVALUATE FUNCTIONAL VITAMIN D DEFICIENCY: RESULTS OF THE SARCOPHAGE STUDY

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Backgrounds

The vitamin D metabolite ratio (VMR) has recently shown to be a best indicator of vitamin D deficiency than 25-hydroxyvitamin D (25(OH)VTD) alone. This study aims at validating these results in a large independent cohort of older individuals. Methods

The SarcoPhAge cohort is a Belgian cohort of community-dwelling older adults. 25(OH)VTD and 24.25(OH)₂VTD were measured on 204 sera collected at the second follow-up using liquid chromatography-tandem mass spectrometry CDC certified method. VMR was calculated according to the formula: VMR= (24.25(OH)₂VTD / 25(OH)VTD) *100. Results

According to cut-offs for vitamin D deficiency established in the literature, 35 individuals (17.2%), 40 individuals (19,6%) and 19 individuals (9.3%) had 25(OH)VTD < 20ng/mL, 24.25(OH)₂VTD < 1.2 ng/mL and VMR < 4%, respectively. 25(OH)VTD, 24.25(OH)₂VTD and VMR were all independently associated with PTH but the best association was observed with VMR (rho: -0.292; p-value < 0.0001). When categorizing 25(OH)VTD, 24.25(OH)₂VTD, and VMR into quartiles, it was observed that only 24.25(OH)₂VTD and VMR exhibited a noteworthy elevation in PTH levels across quartiles (p= 0.002 and p<0.0001, respectively). Additionally, VMR

was independently associated with appendicular lean mass (rho: -0.220; p-value =0.0018) and BMI (rho: -0.173; p-value =0.0156) but not with fat mass. Conclusions In this study, we confirmed that VMR is the best biomarker to study functional deficiency of vitamin D.

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CALCITRIOL SUPPLEMENTATION AFTER KIDNEY TRANSPLANTATION: RESULTS OF A DOUBLE-BLINDED, RANDOMIZED, PLACEBO-CONTROLLED TRIAL

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Objective: Vitamin D deficiency is highly prevalent following kidney transplantation (KT) and results in bone loss. We performed a randomized placebo-controlled trial of calcitriol administration in the 1st 12 months post-KT hypothesizing that calcitriol preserves bone quality and strength.

Methods: Participants >18 yo undergoing a first KT were recruited from at Columbia University between 2013 and 2015. Participants were randomized 1:1 to either daily placebo or daily calcitriol 0.5 mcg. All participants received cholecalciferol 1,000 IU daily. Participants had labs and imaging at baseline (pre-KT) and 12 months post-KT. Areal bone mineral density (BMD) was measured by DXA at the spine, hip and forearm. Cortical and trabecular volumetric BMD, microarchitecture, geometry, total bone strength, and vascular calcifications were measured by high resolution peripeperal QCT (HRpQCT) at the radius and tibia. Primary analyses were intent to treat. **Results:** The study included 32 and 29 participants in the calcitriol and placebo group respectively. In the calcitriol and placebo groups: mean±sd age was 51±14 and 51±13 yrs; 67% and 70% were male; and 100% and 97% were Caucasian respectively. PTH declined -63±11% in calcitriol and -62±6% in placebo (p<0.001 for both); there was no between-group difference (p=0.9). BSAP increased 63±36% in placebo and 9.6±13.2 in calcitriol; CTX decreased by -69±7% in placebo and by -77±5% in calcitriol; there were no between-group difference for either (p=0.8, p=0.3, respectively). There were no within or between group differences in changes to areal BMD by DXA or to geometry, microarchitecture or mechanical estimates of bone strength by HRpQCT (Table 1). Hypercalcemia occurred in 39% and 3% in the calcitriol and placebo groups respectively (p<0.001) but vascular calcification did not progress (p>0.05).

Conclusion: Compared to placebo, calcitriol administration during the first 12 months of KT did not affect skeletal measures. These findings suggest that the addition of calcitriol to cholecalciferol in KT recipients does not improve skeletal health but causes hypercalcemia.