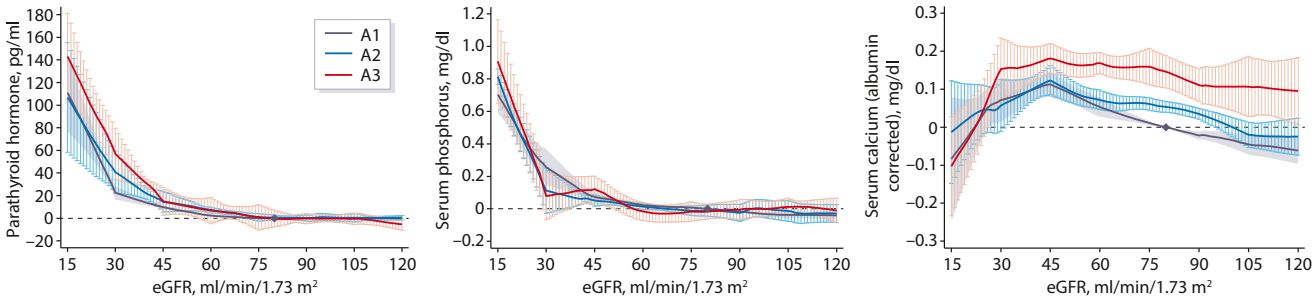

Figure 35 | Association between estimated glomerular filtration rate (eGFR) with serum concentrations of parathyroid hormone, phosphate, and serum calcium in general population and high-risk cohorts from the Chronic Kidney Disease Prognosis Consortium, by level of albuminuria (A1–A3). The *y* axis represents the meta-analyzed absolute difference from the mean adjusted value at an eGFR of 80 ml/min per 1.73 m^2 and albumin excretion <30 mg/g (<3 mg/mmol). A1, albuminuria <30 mg/g (<3 mg/mmol); A2, albuminuria 30–300 mg/g (3–30 mg/mmol); A3, >300 mg/g (>30 mg/mmol). Reproduced from *American Journal of Kidney Diseases*, volume 73, issue 2, Inker LA, Grams ME, Levey AS, et al. Relationship of estimated GFR and albuminuria to concurrent laboratory abnormalities: an individual participant data meta-analysis in a Global Consortium, pages 206–217, Copyright © 2018, with permission from the National Kidney Foundation, Inc.[541]

extraskeletal (i.e., vascular) calcification related to these abnormalities of metabolism. It has been recommended that in people with CKD G3a–G5, treatments of CKD-MBD should be based on serial assessments of phosphate, calcium, and PTH levels considered together.[20]

Higher serum phosphate concentrations are associated with mortality,[602] and experimental data suggest that serum phosphate concentration is directly related to bone disease, vascular calcification,[603,604] and CVD. Low-phosphorus diets and binders are used to help lower serum phosphate to reduce the long-term complications of CKD-MBD, although more research is needed to fully understand the disease-modifying impact of these interventions.[605] Similarly, despite evidence suggesting no benefit on clinical outcomes,[606] vitamin D replacement and calcimimetics to control PTH levels and to maintain calcium within the normal range are also common strategies. For recommendations regarding selection and dosing with specific therapeutic agents and research, please see the KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD).[20]

3.14 Hyperuricemia

Definition and prevalence. Uric acid is the end product of the metabolism of purine compounds, and both increased urate production and decreased kidney excretion of uric acid can lead to hyperuricemia. The American College of Rheumatology defines hyperuricemia as a serum uric acid concentration of ≥6.8 mg/dl (approximately ≥400 μmol/l).[607]

Data from the US National Health and Nutrition Examination Survey (NHANES) 2015–2016 found that the crude adult prevalence of gout (defined as self-reported, doctor diagnosis, or uric acid–lowering therapy use) was 3.9% with a higher prevalence in men than women (5.2% vs. 2.7%). After adjustment for age and sex, an eGFR consistent with CKD G3 was associated with about twice the prevalence of gout (odds ratio: 1.96; 95% CI: 1.05–3.66).[608]

Recommendation 3.14.1: We recommend people with CKD and symptomatic hyperuricemia should be offered uric acid–lowering intervention *(1C)*.

The Work Group placed high value on avoiding the unpleasant symptoms of acute gout and preventing long-term complications of recurrent gout among people with CKD. There are well-tolerated and low-cost oral medications that can effectively lower blood uric acid concentration in people with CKD.

Key information

Balance of benefits and harms. Systematic review of the management of gout by the American College of Rheumatology found strong evidence for uric acid lowering in people with tophaceous gout, radiographic damage due to gout, or frequent gout flares; some of whom also had CKD.[607]

The ERT assessed the safety of uric acid–lowering therapy and found that uric acid lowering did not increase adverse events among people with CKD and particularly focused on risk of cutaneous reactions and hypersensitivity (pooled RR: 1.00; 95% CI: 0.60–1.65) and hepatotoxicity (pooled RR: 0.92; 95% CI: 0.37–2.30). Uric acid–lowering therapy was also found not to modify the risk of cardiovascular events or all-cause mortality in people with CKD.[150,609,610] This reassuring cardiovascular safety profile is consistent with general population data. In the open-label Allopurinol and Cardiovascular Outcomes in Patients With Ischemic Heart Disease (ALL-HEART) randomized trial, 5721 people aged ≥60 years with ischemic heart disease but no history of gout were included. Allopurinol did not modify cardiovascular risk compared with standard care (HR for the composite primary outcome of nonfatal myocardial infarction, nonfatal stroke, or cardiovascular death: 1.04; 95% CI: 0.89–1.21). Findings were similar when 540 people