Keeping phosphorus under control to improve the quality of patients with renal failure

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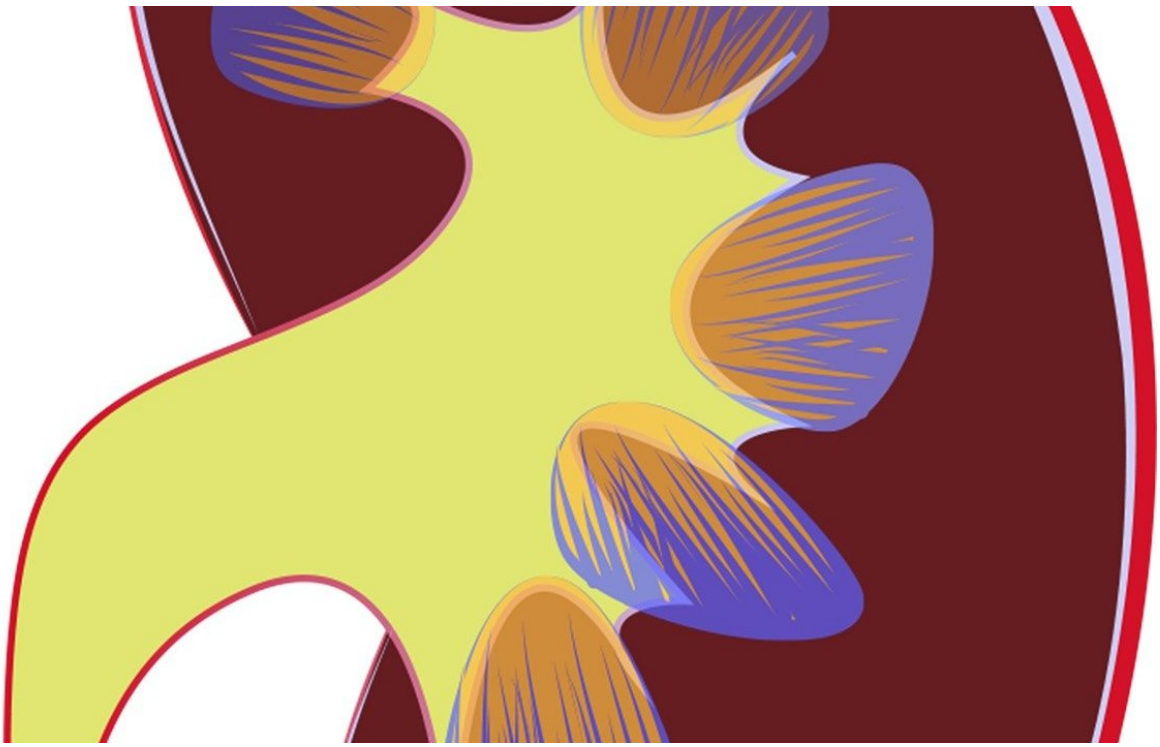

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The FGF23 (Fibroblast growth factor 23) hormone originates in bone to communicate with the kidney, whose duty it is to excrete excess phosphorus that the bone detects. Its main function is bone-kidney

communication to eliminate phosphorus, but what happens when the person has renal failure and kidneys that do not work in addition to an impaired excretion ability? The failing kidney is unable to abide by FGF23. Nevertheless, the bone keeps producing this hormone because it does not detect that the kidney has stopped working, resulting in a build-up of FGF23 in the blood.

The build-up of this hormone causes troublesome effects on other organs. For instance, the cardiovascular system is affected by high levels of this hormone; the heart enlarges (this is known as hypertrophy), which increases risk of death. This led a research team made up of researchers from the University of Cordoba's Medical School, the GC13 and GC07 groups from the Maimonides Institute of Biomedical Research (IMIBIC) and the Nephrology Unit at the Queen Sofia University Hospital to focus on lowering levels of FGF23.

After a clinical trial on 21 patients in dialysis treatment for 40 weeks, they were able to prove that a diet with low phosphorus intake along with medication based on phosphate binders, which prevent the body from absorbing phosphate, corrects the high levels of phosphorus and produces a considerable decrease of the FGF23 hormone. Therefore, the effort of lowering phosphorus levels would also work to decrease the concentration of FGF23 and reduce the risks of circulation and heart problems.

Another advance coming out of this research project is differentiating two parts of the hormone: intact FGF23 (iFGF23) and FGF23 c-terminal (c-FGF23). In this research project involving 150 patients, the complete intact hormone was considered, and conversely the resulting parts of fragmentation (c-terminal), since the final quantity of these depends on specific factors.

While phosphorus is responsible for more than 60 percent of the

molecule values in both cases, phosphorus and calcium in the blood are defining for the intact hormone; for the c-terminal, inflammation and the time that the patient has undergone dialysis are factors that increase hormone concentration.

Future research aims to study how phosphorus reduction increases life expectancy in the long term.

More information: Cristian Rodelo-Haad et al, Phosphate control in reducing FGF23 levels in hemodialysis patients, *PLOS ONE* (2018). DOI: 10.1371/journal.pone.0201537

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