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Commentary

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The pathogenesis of calcium oxalate nephrolithiasis remains a mystery, but the suggestion that it is simply due to perturbations in urinary super saturations remains an inadequate explanation (1). It is likely due to much more complex and nuanced mechanisms that incorporate inorganic and organic components. How these components propagate into Randall plaques or calculi or even where these stone-forming events occur (vasa recta, collecting ducts, or the basement membrane of the loops of Henle) is debatable. Metabolic derangements leading to uncontrolled reactive oxygen species (ROS) generation or a reduced antioxidant capacity to alleviate oxidative stresses may play a role in Randall plaque formation through tissue damage and/or ROS-induced altered gene expression. Markers of oxidative stress/damage (e.g., N-acetyl-β-glucoseaminidase, malondialdehyde, or β-galactosidase) have been detected in animal models with calcium oxalate nephrolithiasis. Further supporting this hypothesis, medications (angiotensin converting enzyme inhibitors or statins) known to reduce oxidative stresses or diets high in antioxidants have been shown to decrease nephrolithiasis in experimental models.

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The mechanism by which increased oxidative stress/damage leads to Randall plaquess is unclear, but likely represents some combination of altered gene expression, tissue remodeling, biomineralization, and inflammation. Even the mechanistic timing is unknown as to whether the oxidative damage occurs first and then leads to Randall plaque formation or vice versa. This represents an area of promise and continued research is needed across several intersecting disease processes (hypertension, hyperlipidemia, diabetes mellitus, obesity, nephrolithiasis, etc) that likely have a shared mechanism/metabolic abnormality.

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