Hounsfield Density of Renal Papillae in Stone Formers: Analysis Based on Stone Composition

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Abbreviations and Acronyms

CT = computerized tomography

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Purpose: We examined renal papillary Hounsfield density in stone formers with all common stone subtypes to further understand the pathophysiology of stone formation.

Materials and Methods: Using computerized tomography we measured the Hounsfield density of a 0.2 cm^2 renal papillary area in patients with a single renal calyceal stone. Results were compared with those in patients without a nephrolithiasis history who served as controls. Stone composition was determined by stone passage or extraction during endoscopic procedures using infrared spectroscopy and polarized microscopy. We measured the Hounsfield density of the stone bearing calyx and of a single calyx from the upper, middle and lower poles of each kidney.

Results: Mean $\pm$ SD renal papillary Hounsfield density in controls was 36.2 ± 4.0 HU. In patients with stones Hounsfield density was significantly greater than in controls in stone bearing calyces, nonstone bearing calyces in the affected kidney and calyces in the contralateral nonstone bearing kidney for all stone composition subtypes (range 48.4 to 61.3 HU, each $p < 0.001$).

Conclusions: Patients with kidney stones regardless of composition showed the unique radiographic characteristic of increased renal papillary Hounsfield density. This was true for all calyces and for each kidney in all stone formers with a single renal calyceal stone. This radiographic evidence supports the role of renal papillary deposits or plaques in the pathophysiology of stone formation.

Key Words: kidney; kidney medulla; kidney calculi; tomography, x-ray computed; physiopathology

Computerized tomography is the gold standard for assessing urolithiasis at many institutions.[1,2] In addition to being the most accurate modality to determine stone size, number and location, CT attenuation values (HU) have been used to determine stone composition and differentiate calcium from noncalcareous stones.[3] It was previously reported that renal papillary Hounsfield density in patients with nephrolithiasis was significantly higher than in controls[4] and these data were reproduced in subsequent publications.[5,6] More recently a retrospective study demonstrated that increased renal papillary Hounsfield density is associated with an increased future risk of stones.[7] However, the phenomenon of increased renal papillary Hounsfield density in stone formers has not been studied with

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respect to stone composition. We determined renal papillary Hounsfield density in stone formers with varying stone compositions.

MATERIALS AND METHODS

Institutional review board approval was obtained before study initiation. We retrospectively searched urology and radiology department outpatient and hospital records to identify patients with nephrolithiasis. Those older than 18 years were included in study. Controls comprised patients evaluated for donor nephrectomy without a nephrolithiasis history and with no renal stones on renal donor CT. Stone formers were identified as patients who spontaneously passed a renal stone or received shock wave lithotripsy or ureteroscopic treatment of a renal stone. CT was done before stone passage or treatment. Study inclusion criteria for stone formers were a single renal calyceal stone of known composition in 1 kidney, no other stones in the stone bearing kidney, no stones in the contralateral kidney and a known stone composition. Stone composition was determined elsewhere by infrared spectrometry and polarizing microscopy. Stones were considered to be of a given composition if the composition was 80% or greater except for struvite stones, for which we used a 30% or greater benchmark.

CT images were retrospectively reviewed by 2 radiologists experienced with abdominal imaging. Reviewers were blinded to patient clinical details and images were reviewed independently. Analysis was done using version 4 of a picture archiving and communication system (Agfa, Richmond, Virginia). Renal papillary Hounsfield density was measured at 5× magnification by placing regions of interest with a mean size of 0.2 cm^2 in the region of the renal papillae. Mean attenuation values were recorded in HU. The Hounsfield density of a single calyx in the upper, middle and lower kidney poles was recorded. Figure 1 shows a stone bearing calyx, nonstone bearing calyces in a kidney with stone and calyces in the nonstone bearing kidney. Statistical analysis was done with JMP®, version 8.0. The Student t-test was used for statistical comparison.

RESULTS

A total of 15 controls and 72 patients with nephrolithiasis were included in study. There was no difference in mean age for any stone subtype vs controls. There was a significant difference in serum creatinine between patients with uric acid stones and controls (mean ± SD 1.28 ± 0.44 vs 0.94 ± 0.23 mg/dl, p = 0.03, see table). There was no significant difference in serum creatinine between any other stone subtype and controls.

Mean renal papillary Hounsfield density in controls (ie those without stones) was 36.2 ± 4.0 HU. In patients with stones Hounsfield density was significantly greater than in controls in stone bearing calyces, nonstone bearing calyces in the affected kidney and calyces in the contralateral nonstone bearing kidney for all stone composition subtypes (range 48.4 to 61.3 HU, each p <0.001, see table and fig. 2).

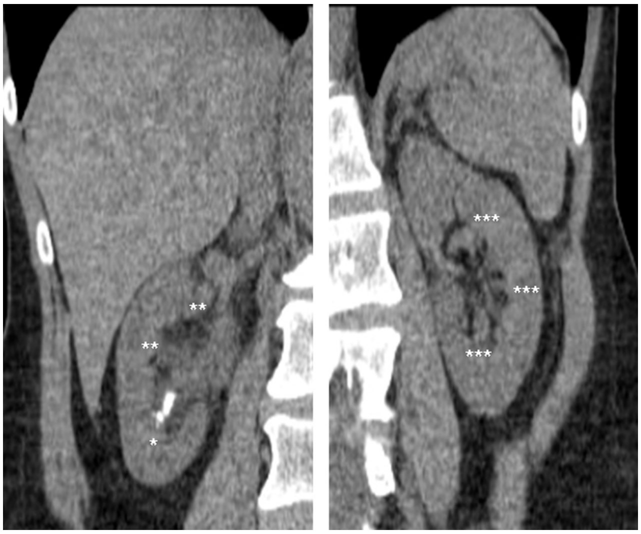

Figure 1. Hounsfield density recordings. Single asterisk indicates stone bearing calyx. Double asterisks indicate nonstone bearing calyces in stone bearing kidney. Triple asterisks indicate calyces in nonstone bearing kidney.

DISCUSSION

Previous studies demonstrated that renal papillary Hounsfield density is significantly greater in stone formers than in nonstone forming patients.[4–6,8] In a more recent retrospective study Ciudin et al found that increased renal papillary Hounsfield density was associated with a greater future risk of stone disease.[7] These findings are consistent with the observation that many patients with nephrolithiasis also have Randall plaques, which are calcium deposits in the renal papillae that are hypothesized to be a nidus for stone formation.[9,10]

In recent years many studies have been performed to better understand the pathogenesis of stone formation. Evidence suggests that different types of papillary plaques or concretions exist in addition to Randall plaques, and these crystal depositions likely have a central role in the pathogenesis of kidney stone disease.[11] Most study in this area has been done on calcium stones, presumably because they are the most common stone subtype. However, evidence was also reported of the role of intratubular deposits and plaque in the pathogenesis of uric acid and struvite stones, respectively.[12,13]

The current study builds on earlier investigations by our group. We previously reported on 17 patients with nephrolithiasis of an unknown stone composition.[4] In the current study we divided patients by stone composition and report the most common