Sonographic Features of Necrosed Renal Papillae Causing Hydronephrosis

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Objective. To describe the sonographic appearance of ureteric obstruction due to necrosed papillae.

Methods. In this study, carried out over 3 years 6 months, patients with diabetes mellitus who had renal colic were studied by sonography. Results. In 15 patients with hydronephrosis, there was a soft tissue mass of necrosed papillae filling the ureteric lumen at the site of obstruction. Necrosed papillae were seen in medullary cavities of the ipsilateral kidney in 5 patients. Ureteroscopic removal of necrosed papillae was done in 13 patients. One patient was not fit for an invasive procedure.

Conclusions. When patients predisposed to renal papillary necrosis have renal colic, and sonography fails to show a ureteric calculus, it is best to look for necrosed papillae in the ureter, which may be causing obstruction. Key words: diabetes mellitus; hydronephrosis; renal colic; renal papillary necrosis; sonography.

Sonography is a well-recognized imaging modality for evaluating hydronephrosis. Sonographic features of papillary necrosis are reported. One of the clinical manifestations of renal papillary necrosis (RPN) is ureteric colic due to obstruction of the ureter by sloughed off papillae. We present the sonographic appearance of this condition.

Materials and Methods

The study group comprised 15 consecutive patients studied over 3 years 6 months. All of them had pain in the loins and were referred for sonography to evaluate the urinary system. All had diabetes mellitus for various periods, ranging from 5 to 20 years. They were taking oral antidiabetic agents or insulin but had poor control. Nine patients were female, and 6 were male. Three patients also had fever with chills, and 2 had hematuria.
Sonography was done with HDI 3500 and HDI 5000 systems (Philips Medical Systems, Bothell, WA) and broadband convex probes of 2 to 5 and 4 to 7 MHz and a linear probe of 5 to 12 MHz. There was mild to moderate dilatation of the pelvicalyceal system of the kidney on the side of pain. On tracing the ureter, there was no evidence of a calculus in the ureter at the site of obstruction, which would have explained the obstruction. Instead, a mass of soft tissue was seen filling the ureter at the site of obstruction (Figs. 1–3), indicating that a necrosed papilla was producing the obstruction. All the patients had routine urinanalysis. Urine cultures were done for the 3 patients who had fever.

Results

All 15 patients in this series had hydronephrosis due to obstruction by a nonshadowing soft tissue mass in the lumen of the ureter. The site of obstruction was in the lower ureter in 6 patients, mid ureter in 4 patients, and upper ureter in 5 patients. In 5 patients, necrosed papillae were seen in cavities in the medullary region of the ipsilateral kidney communicating with the calyces (Fig. 4); this feature is identical to the ring sign seen on excretory urography. Classic ring calcifications of papillary necrosis were seen in 2 patients. Urinanalysis was not contributory in any patient. Urine culture results were negative for the 3 patients who had cultures done. Fourteen patients underwent cystoscopy and ureteroscopy, which in 13 revealed necrosed papillae in the ureter as the cause of obstruction (Fig. 5A), and the same were removed. In 1 patient, necrosed papillae were not seen, but the ureteric orifice was patulous, which may have suggested recent passage. In 5 patients, necrosed papillae were seen in calyces as well (Fig. 5B), and raw areas of papillary necrosis were also seen, after the removal of the necrosed papillae (Fig. 5C). One patient was not fit for any invasive procedure. Two patients returned with recurrence of symptoms during the study period and had similar findings on sonography.

Discussion

Renal papillary necrosis is a complication that develops in the course of a variety of systemic diseases, such as diabetes mellitus, analgesic nephropathy, and sickle cell hemoglobinopathy. The basic manifestation is tubulointerstitial nephritis accompanied by compromised medullary blood flow that ultimately results in focal or diffuse necrosis of various segments of the renal medulla. Renal papillary necrosis has been classified into 2 forms, depending on the location of the necrotic process: a medullary form, in which the focal areas of the innermost renal medullary regions are necrotic but the fornices and papillary tip remain viable, resulting finally in a cavity in the medulla communicating with the calyx by a tract through the tip of the papilla; and a papillary form, in which the calyceal fornices and the

Figure 1. Oblique sonogram of the left flank showing a necrosed papilla (arrow) filling the lumen of the left upper ureter and a dilated proximal ureter (UR).

Figure 2. Oblique sonogram of the right lower quadrant showing a necrosed papilla (arrow) in the mid ureter. UR indicates dilated proximal ureter.
entire papillary tip are destroyed, finally resulting in necrosed papillae (Fig. 6). The exact prevalence of RPN is unknown. The frequency with which the lesion is encountered at autopsy varies from one geographic region to another. The disease is principally one of older individuals, the average reported age of patients being 55 years, with nearly half of the cases occurring in those older than 40 years. The lesion is uncommon in individuals younger than 40 years, except for those with sickle cell hemoglobinopathy, in whom the disease occurs at younger ages.

Diabetes mellitus remains the most frequent condition associated with RPN. The high frequency with which RPN occurs in diabetic persons can be appreciated from its finding in 2.7% to 7.2% of autopsies of patients with diabetes mellitus, whereas in nondiabetic patients, its frequency at autopsy is less than 0.3%. Radiologic evidence of RPN has been reported in as many as 23.7% of patients with long-standing insulin-dependent diabetes mellitus. Diabetic persons with recurrent episodes of urinary tract

Figure 3. Oblique sonogram through the urinary bladder showing the soft tissue of a necrosed papilla filling the distal-most ureter (arrow). BL indicates urinary bladder, and UR, dilated proximal ureter.

Figure 4. Coronal (A), longitudinal (B), and transverse (C) sonograms of the kidney showing necrosed papillae (arrows) in the necrotic medullary cavities.
infection or acute infections of the kidney are particularly prone to development of RPN. In diabetic persons, the lesion characteristically affects elderly women, with a female-male preponderance as high as 5:1. The relatively poor nutrient blood supply to the papillary tip, compared with the remainder of the kidney and pyramids, renders this anatomic segment particularly susceptible. In its early phases, the necrosis appears to involve a few papillae to which RPN may remain localized, or it might become progressive and gradually involve a greater number of papillae in 1 or both kidneys. The lesions usually, but not invariably, involve both kidneys. Renal papillary necrosis is bilateral in three fourths of the cases reported in the literature. In patients in whom 1 kidney was involved at the time of first examination, RPN developed in the other kidney over 1 to 4 years.1

On gross examination, the foci of necrosis are friable with a well-circumscribed area of yellowish gray or grayish red sharply demarcating the involved areas in the distal regions of the affected pyramids. Where the papilla has sloughed, there

Figure 5. A, Ureteroscopy showing a necrosed papilla in the ureter at the site of obstruction. B, Necrosed papilla removed. C, Ureteroscopy showing a sloughed off necrosed papilla lying free in the calyx. D, Ragged margins (arrows) of papillary necrosis after removal of the sloughed-off papilla.
will be a ragged surface at the point of separation. In chronic forms of the disease, varying amounts of calcification are present. The clinical manifestations of papillary necrosis are variable, depending on several factors: (1) the localization of the necrotic process; (2) the number of papillae involved, which will ultimately determine the level of renal insufficiency that develops; (3) the presence or absence of superimposed infection; and (4) the presence of an obstruction. In papillary form, the necrotic lesions are well demarcated and form a sequestrum that may be sloughed and excreted into the urine, be reabsorbed, leaving sinuses or cavities at their site, or remain in situ to become calcified. The passage of the sloughed papillae may be associated with lumbar pain, ureteral colic, and hematuria. All the patients in this series had colic due to passage of sloughed off papillae.

All the patients in this series had diabetes that was controlled poorly. They had symptoms of renal colic for which sonography was done. On sonography, the necrosed papilla was seen as a nonshadowing soft tissue mass filling the lumen of the ureter at the site of obstruction in the different parts of the ureter. This feature was seen in all patients. The appearance of a nonshadowing soft tissue mass in the ureter also can be seen with a clot, sludge, or fungal ball, and these cannot be differentiated sonographically. The ipsilateral kidney showed necrosed papillae in necrotic medullary cavities, a feature identical to the ring sign on excretory urography, in 5 patients. The ring sign on excretory urography is caused by the detached papilla lying in the contrast agent–filled cavity. When the necrosed papilla is sloughed off, a cavity communicating with the collecting system is shown sonographically (Fig. 7). The cavity of papillary necrosis can be differentiated from hydronephrosis by the irregular contour of the cavity and by extension of the cavity up to the arcuate arteries. In hydronephrosis, all the calyces are clubbed, whereas in papillary necrosis, 1 or a few cavities are seen that extend beyond the level of other calyces. Alternatively, the necrosed papilla can remain in situ and become calcified, which is shown sonographically as the classic ring or garlandlike calcification of the medulla. Calcifications in a single papilla are shown as a ring, and in cases of multiple calcified papillae, they are shown as a garland of calcifications around the renal sinus echoes. In this series, 2 patients had the classic ring calcifications of papillary necrosis. While tracing the dilated ureter, the mid and distal portions of the ureter may be obscured by bowel gas, causing difficulty. In women, endovaginal sonography will be useful for imaging the distal ureter.

In the particular clinical situation in which patients who are predisposed to RPN have renal colic, and sonography fails to reveal a calculus in the ureter, it is best to look for a soft tissue mass in the lumen at the site of obstruction. This finding is indicative of obstruction of the ureter by a necrosed papilla. This finding is further corroborated if necrosed papillae are seen in the necrotic medullary cavities.

Figure 7. Transverse sonogram of the kidney showing irregular and empty medullary cavities (C) remaining after a passing of a sloughed-off necrosed papilla.
References


