

Early Versus Delayed Endoscopic Clearance of Necrosed Papillae in Non-Calculous Acute Pyelonephritis: A Prospective Comparative Study from a Tertiary Care Centre

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Abstract

Introduction: Renal papillary necrosis complicating acute pyelonephritis is seen with increasing frequency in poorly controlled type 2 diabetes. Sloughed papillae obstruct the collecting system and sustain infection. Emergency decompression with a double-J stent is standard practice, but there is no agreement on when the necrotic tissue should be cleared endoscopically. We compared clearance at 2 to 3 months with clearance at 9 to 12 months.

Methods: Prospective, non-randomised comparative study conducted at Department of Urology, Sri Siddhartha Medical College, Sri Siddhartha Academy of Higher Education, Tumkur between April 2022 and April 2025. Sixty adults with radiologically confirmed non-calculous acute pyelonephritis with papillary necrosis and hydronephrosis underwent emergency 6 Fr/24 cm silicone double-J stenting, followed by two weeks of intravenous and two weeks of oral culture-directed antibiotics. Allocation to early clearance at 2 to 3 months (Group A) or delayed clearance at 9 to 12 months (Group B) followed the protocol of the admitting consultant unit, determined by the day of admission roster. The primary outcome was complete clearance of necrosed papillae in a single session. Secondary outcomes were operative time, re-stenting, peri-operative sepsis, stent burden, and chronic pyelonephritis on dimercaptosuccinic acid scintigraphy performed at least six months after clearance. Chronic pyelonephritis was modelled by multivariable logistic regression adjusting for baseline chronic kidney disease, glycated haemoglobin and age.

Results: Fifty-three of 60 patients completed follow-up (28 in Group A, 25 in Group B). Mean age was 54.0 (SD 10.6) years, 47 patients (88.7%) were female, 45 (84.9%) had type 2 diabetes and mean glycated haemoglobin was 9.1% (SD 1.9). Complete single-session clearance was achieved in 20 of 28 (71.4%) in Group A and 23 of 25 (92.0%) in Group B ($p = 0.081$). Mean operative time was 127.7 (SD 28.5) minutes against 75.8 (SD 20.9) minutes ($p < 0.001$). Peri-operative sepsis occurred in 5 patients (17.9%) and 1 patient (4.0%) respectively ($p = 0.196$). Median stent dwell was 80 days against 322 days ($p < 0.001$). Chronic pyelonephritis was identified in 6 of 28 (21.4%) in Group A and 17 of 25 (68.0%) in Group B ($p < 0.001$), and delayed clearance remained independent associated with it after adjustment (adjusted odds ratio 8.13, 95% CI 1.97 to 33.56, $p = 0.004$). Mean estimated glomerular filtration rate fell by 1.0 (SD 6.1) mL/min/1.73 m² in Group A and by 7.5 (SD 7.0) in Group B ($p < 0.001$).

Conclusions: Delaying clearance to 9 to 12 months makes the endoscopic procedure easier, faster and safer in the short term, but is independently associated with renal cortical scarring and accelerated loss of filtration. The two strategies compared here are the extremes of a spectrum, and the balance of harms suggests the optimal window lies between them. An adequately powered randomised trial comparing intermediate timing against these two anchors is the logical next step.

Keywords: Renal papillary necrosis; acute pyelonephritis; type 2 diabetes mellitus; ureteroscopy; ureteral stent; chronic pyelonephritis; DMSA scintigraphy.

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Introduction

Renal papillary necrosis is an old diagnosis whose epidemiology has quietly shifted. Analgesic nephropathy and sickle cell disease once dominated the case mix described in Western series. [1-3] In Indian tertiary practice today, most of what reaches the urology ward arises on a background of poorly controlled type 2 diabetes. [4,5] The reason is anatomical as much as metabolic. The medullary pyramid sits at the watershed of the renal circulation and tolerates ischaemia badly. [1] Add small vessel disease and then an infection to that, and a blood supply that was already marginal tips into frank infarction. The papilla sloughs.

What follows is a mechanical problem layered on an infective one. Necrotic tissue migrates from the calyx into the renal pelvis and ureter, obstructs, and sustains the infection that produced it. Patients present with fever, flank pain, leucocytosis and a rising creatinine, and imaging shows hydroureteronephrosis with no stone to explain it. [6,7] The immediate management of this picture is not controversial. Decompression with a double-J stent, culture-directed antibiotics and correction of hyperglycaemia will settle the acute episode in the great majority. [8,9] What remains unsettled is the second half of the treatment. A sloughed papilla does not dissolve. Left in situ it behaves as a nidus, encrusts over months, and keeps the patient tethered to a stent. [10,11] Retrieving it requires cystoureteroscopy with check ureteroscopy or retrograde intrarenal surgery, and the timing of that second procedure is decided in most units by institutional habit rather than by evidence.

Two lines of reasoning pull against each other here. Intervening early frees the patient from the stent sooner and, in theory, shortens the run of low-grade obstruction and infection thought to drive parenchymal scarring. Waiting lets the necrotic papillae demarcate and separate from the underlying medulla, allows mucosal inflammation to settle, and ought to make retrieval cleaner. Neither position has been tested against the other. We compared early and delayed endoscopic clearance in patients with non-calculous acute pyelonephritis complicated by papillary necrosis and obstruction. We asked how completely and how quickly the papillae could be retrieved, what the procedure cost the patient in peri-operative morbidity and stent burden, and what became of the kidney over the following year.

Materials and Methods

Study design, setting and ethical approval: This was a prospective, non-randomised comparative study carried out in the Department of Urology, Sri Siddhartha Medical College, Sri Siddhartha

Academy of Higher Education, Tumkur between April 2022 and April 2025.

Participants: Adults older than 18 years with clinically and radiologically confirmed acute pyelonephritis were screened on admission. Patients were enrolled if noncontrast computed tomography demonstrated papillary necrosis together with hydroureteronephrosis or hydronephrosis. Patients were excluded if pyelonephritis was calculous, if the pyelonephritis was non-obstructive (that is, without hydroureteronephrosis or hydronephrosis), or if they had undergone urinary tract instrumentation within the preceding four weeks. Baseline chronic kidney disease was defined by KDIGO criteria as an estimated glomerular filtration rate below 60 mL/min/1.73 m² documented at the three-month review, once the acute episode had resolved. [12]

Indications for emergency drainage: Emergency drainage was undertaken where there was leucocytosis with a rising serum creatinine, hydroureteronephrosis on imaging, or a significant renal or perinephric collection.

Initial management: All patients underwent emergency retrograde double-J stenting with a 6 Fr, 24 cm silicon stent under appropriate anaesthesia as the clinical state allowed. Empirical intravenous antibiotics were commenced at admission and revised once urine culture and sensitivity results were available. Intravenous therapy was continued for two weeks and followed by two weeks of oral antibiotics. Glycaemic control was managed jointly with the department of endocrinology.

Group allocation: Allocation was not randomised. Two consultant units admit emergencies on alternate days at this centre, and the units follow different protocols for the timing of definitive clearance. Patients admitted under Unit I were scheduled for clearance between 2 and 3 months (Group A); those admitted under Unit II were scheduled between 9 and 12 months (Group B). Because the admitting unit is fixed by the day of presentation, allocation is quasi-random with respect to patient characteristics, but it is not concealed and the treating team was aware of group from the outset.

Definitive clearance procedure: The definitive procedure consisted of cystourethroscopy and stent removal, followed by check ureteroscopy with a 6/7.5 Fr semirigid ureteroscope, with flexible retrograde intrarenal surgery added where the pelvicalyceal system required inspection. The ureter, renal pelvis and every calyx were examined systematically and necrosed papillae were retrieved

with a nitinol basket or grasping forceps. Complete clearance was defined as the absence of any visible necrotic tissue in the ureter, renal pelvis or any calyx at the end of the procedure. Where the system remained loaded, where pus continued to discharge, or where papillae were still separating, the procedure was abandoned, a stent was replaced and the patient was brought back after a further interval.

Follow-up: Patients were reviewed at 2 weeks, 4 weeks, 3 months, 6 months, 9 months and 12 months from the index stenting, and for a further 6 months after definitive clearance. Each visit comprised symptom review, urine culture, and serum creatinine with estimated glomerular filtration rate by the 2021 CKD-EPI creatinine equation, [13] and ultrasonography of the kidneys, ureters and bladder.

Outcome measures: The primary outcome was complete clearance of necrosed papillae achieved in a single session.

Secondary outcomes were operative time, re-stenting for incomplete clearance, unplanned stent change for blockage, post-procedure fever, culture-positive sepsis, intensive care admission, stent dwell time, stent-related symptoms, and chronic pyelonephritis. Chronic pyelonephritis was defined as one or more cortical defects with associated volume loss on technetium-99m dimercaptosuccinic acid scintigraphy performed at least six months after definitive clearance, an interval chosen because cortical defects seen earlier cannot be distinguished reliably from residual acute inflammatory change. [14,15] Scans were reported independently by two nuclear medicine physicians blinded to group allocation, with disagreements resolved by consensus. Stent-related symptoms were measured with the urinary symptom and body pain domains of the Ureteral Stent Symptom Questionnaire, administered at the time of final stent removal. [16,17]

Sample size: Assuming complete single-session clearance of 70% with early intervention and 95% with delayed intervention, a two-sided alpha of 0.05 and 80% power, 36 patients were required in each arm.¹⁸ Thirty patients per arm were recruited, this being the number of eligible patients presenting during the study window. The study is therefore smaller than planned and the implications are addressed in the limitations.

Statistical analysis: Categorical variables are presented as counts with percentages and Wilson score 95% confidence intervals, and compared

using the Fisher exact test. Normally distributed continuous variables are presented as mean with standard deviation and compared using the Welch t test; skewed variables are presented as median with interquartile range and compared using the Mann-Whitney U test. Chronic pyelonephritis was modelled by multivariable logistic regression with group, baseline chronic kidney disease, glycated haemoglobin and age as covariates, the number of covariates being limited by the number of events. A prespecified sensitivity analysis repeated the group comparison within strata of baseline chronic kidney disease. A two-sided p value below 0.05 was taken as significant. Analyses were performed in R version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Participants and baseline characteristics: Sixty patients were enrolled, 30 in each group. Two patients in Group A and five in Group B were lost to follow-up, an attrition of 6.7% and 16.7% respectively ($p = 0.42$). Of the seven, four had relocated out of state, two could not be contacted, and one patient in Group B died at seven months of an acute coronary syndrome unrelated to the urinary tract. Twenty-eight patients in Group A and 25 in Group B were available for analysis.

Baseline characteristics are shown in Table 1. The cohort was overwhelmingly female, 47 of 53 (88.7%), with a mean age of 54.0 (SD 10.6) years and a range of 18 to 70. Type 2 diabetes was present in 45 patients (84.9%) and control was poor, with a mean glycated haemoglobin of 9.1% (SD 1.9). Disease was right-sided in 28 patients (52.8%), left-sided in 21 (39.6%) and bilateral in 4 (7.5%). The groups were comparable in age, sex and the prevalence of diabetes. Baseline chronic kidney disease was commoner in Group B, affecting 13 of 25 (52.0%) against 8 of 28 (28.6%) in Group A, and mean estimated glomerular filtration rate at three months was correspondingly lower at 49.7 (SD 24.3) against 63.8 (SD 28.0) mL/min/1.73 m². Neither difference reached conventional significance ($p = 0.099$ and $p = 0.055$), but both are large enough to matter and both were carried into the multivariable model. *Escherichia coli* was the commonest isolate, grown in 34 of 53 patients (64.2%), followed by *Klebsiella pneumoniae* in 9 (17.0%), *Enterococcus faecalis* in 4 (7.5%), *Proteus mirabilis* and *Pseudomonas aeruginosa* in 2 each (3.8%), and *Candida* species in 1 (1.9%). One urine sample was sterile at presentation, the patient having received antibiotics before referral.

Table 1: Baseline characteristics of the two groups.

Characteristic	Group A (early) n = 28	Group B (delayed) n = 25	p value
Age, years, mean (SD)	52.8 (10.7)	55.4 (10.5)	
Female sex, n (%)	24 (85.7)	23 (92)	
Type 2 diabetes mellitus, n (%)	23 (82.1)	22 (88)	
Glycated haemoglobin, %, mean (SD)	9.0 (2.0)	9.3 (1.7)	
Chronic kidney disease at baseline, n (%)	8 (28.6)	13 (52)	
Serum creatinine at admission, mg/dL, mean (SD)	2.6 (1.3)	3.2 (1.5)	
eGFR at 3 months, mL/min/1.73 m ² , mean (SD)	63.8 (28.0)	49.7 (24.3)	
Lost to follow-up, n (% of 30 enrolled)	2 (6.7)	5 (16.7)	0.42

SD, standard deviation; eGFR, estimated glomerular filtration rate by the 2021 CKD-EPI equation.

Proportions compared by Fisher exact test, means by Welch t test.

Procedural outcomes and peri-operative morbidity: Procedural outcomes are set out in Table 2. Complete clearance of necrosed papillae in a single session was achieved in 20 of 28 patients (71.4%, 95% CI 52.9 to 84.7) in Group A and in 23 of 25 (92.0%, 95% CI 75.0 to 97.8) in Group B. The 20.6 percentage point difference favoured delayed clearance but did not reach significance at this sample size ($p = 0.081$).

The difference in operative time was unambiguous. Clearance took a mean of 127.7 (SD 28.5) minutes in Group A against 75.8 (SD 20.9) minutes in Group B, a difference of 51.9 minutes ($p < 0.001$).

Eight of 28 patients in Group A (28.6%) required re-stenting because clearance was incomplete, against 2 of 25 (8.0%) in Group B ($p = 0.081$). In Group A the reasons recorded were multiple necrosed papillae, a system still loaded with purulent debris, and papillae visibly still separating at the time of ureteroscopy. In Group B the ureter and pelvicalyceal system were clear in most cases. What remained was usually two or three discrete fragments lying in a dependent lower calyx.

Unplanned stent change for blockage during the waiting interval was needed in 2 patients in Group A and 1 in Group B ($p = 1.00$). Peri-operative morbidity followed the same pattern. Post-procedure fever occurred in 9 patients in Group A (32.1%) and 3 in Group B (12.0%, $p = 0.107$), culture-positive sepsis in 5 (17.9%) and 1 (4.0%) respectively ($p = 0.196$), and two patients in Group A required intensive care for septic shock, both of whom recovered. No patient in either group required nephrectomy and there were no procedure-related deaths.

The cost of waiting appears in Table 3. Median stent dwell was 80 days (IQR 75 to 86) in Group A against 322 days (IQR 293 to 340) in Group B ($p < 0.001$), and patients in Group B underwent a median of 2 planned stent exchanges (IQR 1 to 2) against none in Group A. At final stent removal the Ureteral Stent Symptom Questionnaire urinary domain score was 28.0 (SD 5.7) in Group A and 33.2 (SD 6.1) in Group B ($p = 0.002$); body pain domain scores were 16.7 (SD 5.4) and 19.6 (SD 6.9) respectively ($p = 0.096$).

Table 2: Procedural outcomes and peri-operative morbidity.

Outcome	Group A (early) n = 28	Group B (delayed) n = 25	p value
Complete clearance in a single session, n (%)	20 (71.4)	23 (92)	
95% confidence interval	52.9 to 84.7	75 to 97.8	
Operative time, minutes, mean (SD)	127.7 (28.5)	75.8 (20.9)	< 0.001
Re-stenting for incomplete clearance, n (%)	8 (28.6)	2 (8)	
Unplanned stent change for blockage, n (%)	2 (7.1)	1 (4)	
Post-procedure fever, n (%)	9 (32.1)	3 (12)	
Culture-positive sepsis, n (%)	5 (17.9)	1 (4)	
Intensive care admission, n (%)	2 (7.1)	0 (0)	

Confidence intervals are Wilson score intervals.

Table 3: Stent burden and stent-related symptoms.

Measure	Group A (early) n = 28	Group B (delayed) n = 25	p value
Total stent dwell, days, median (IQR)	79.5 (75.2 to 86)	322 (293 to 340)	< 0.001
Planned stent exchanges, n, median (IQR)	0 (0 to 0)	2 (1 to 2)	< 0.001
USSQ urinary symptom domain, mean (SD)	28.0 (5.7)	33.2 (6.1)	
USSQ body pain domain, mean (SD)	16.7 (5.4)	19.6 (6.9)	

IQR, interquartile range; USSQ, Ureteral Stent Symptom Questionnaire, administered at final stent removal. Urinary symptom domain scored 11 to 55, body pain domain 0 to 40; higher scores indicate greater symptom burden. Medians compared by Mann-Whitney U test.

Renal outcomes: Renal outcomes are given in Table 4. Chronic pyelonephritis on dimercaptosuccinic acid scintigraphy was identified in 6 of 28 patients (21.4%, 95% CI 10.2 to 39.5) in Group A and 17 of 25 (68.0%, 95% CI 48.4 to 82.8) in Group B, an absolute difference of 46.6 percentage points (95% CI 22.8 to 70.3, $p < 0.001$).

Filtration followed the same direction. Mean estimated glomerular filtration rate at last follow-up was 62.8 (SD 29.1) mL/min/1.73 m² in Group A and 42.2 (SD 22.3) in Group B ($p = 0.005$). Expressed as change from the three-month baseline, which removes the effect of the unequal starting point, Group A lost a mean of 1.0 (SD 6.1) mL/min/1.73 m² and Group B lost 7.5 (SD 7.0) ($p < 0.001$).

The multivariable model is shown in Table 5. After adjustment for baseline chronic kidney disease, glycated haemoglobin and age, delayed clearance remained independently associated with chronic

pyelonephritis, with an adjusted odds ratio of 8.13 (95% CI 1.97 to 33.56, $p = 0.004$). Baseline chronic kidney disease carried an adjusted odds ratio of 3.78 (95% CI 0.93 to 15.45, $p = 0.064$) and each one percentage point rise in glycated haemoglobin an odds ratio of 1.65 (95% CI 1.01 to 2.70, $p = 0.044$). Age was not associated with the outcome.

The prespecified stratified analysis is shown in the lower panel of Table 5. Among patients without baseline chronic kidney disease, chronic pyelonephritis occurred in 3 of 20 (15.0%) in Group A and 6 of 12 (50.0%) in Group B ($p = 0.049$). Among those with baseline chronic kidney disease the corresponding figures were 3 of 8 (37.5%) and 11 of 13 (84.6%) ($p = 0.056$). The direction and approximate magnitude of the group effect were preserved in both strata, which argues against the difference being wholly an artefact of the baseline imbalance.

In an exploratory analysis, chronic pyelonephritis was recorded in 6 of the 10 patients (60.0%) who required re-stenting for incomplete clearance and in 17 of the 43 (39.5%) cleared at the first attempt ($p = 0.300$). The study was not powered for this comparison and it should be treated as hypothesis-generating.

Table 4: Renal outcomes at last follow-up.

Outcome	Group A (early) n = 28	Group B (delayed) n = 25	p value
Chronic pyelonephritis on DMSA, n (%)	6 (21.4)	17 (68)	< 0.001
95% confidence interval	10.2 to 39.5	48.4 to 82.8	
Absolute risk difference, percentage points	46.6(95%CI22.8to70.3)		
eGFR at last follow-up, mL/min/1.73 m ² , mean (SD)	62.8 (29.1)	42.2 (22.3)	
Change in eGFR from 3 months, mean (SD)	-1.0 (6.1)	-7.5 (7.0)	< 0.001

DMSA, technetium-99m dimercaptosuccinic acid scintigraphy performed at least six months after definitive clearance and reported blind to group allocation.

Table 5: Multivariable model for chronic pyelonephritis, and sensitivity analysis stratified by baseline chronic kidney disease.

Covariate	Adjusted odds ratio (95% CI)	p value
Multivariable logistic regression (n = 53, 23 events)		
Delayed clearance (Group B vs Group A)	8.13 (1.97 to 33.56)	0.0038
Chronic kidney disease at baseline	3.78 (0.93 to 15.45)	0.0637
Glycated haemoglobin, per 1 percentage point	1.65 (1.01 to 2.7)	0.0442
Age, per year	1.01 (0.94 to 1.08)	0.8258
Stratified sensitivity analysis: chronic pyelonephritis, n/N (%)		
Patients without baseline CKD	A 3/20 (15) vs B 6/12 (50)	0.0493
Patients with baseline CKD	A 3/8 (37.5) vs B 11/13 (84.6)	0.0555

CI, confidence interval; CKD, chronic kidney disease. The number of covariates was limited to four by the number of events. Stratum comparisons by Fisher exact test.

Discussion: We set out to answer a narrow operative question and got two answers pointing in opposite directions. Waiting 9 to 12 months

improved every measure a surgeon takes on the day.

More patients cleared in one sitting, operative time cut by more than 40%, fewer second procedures, less fever, less sepsis. It also left the patient with a worse kidney a year later. The technical advantage explains itself. At 2 to 3 months the necrotic papilla has often not fully demarcated. Fragments remain

partly attached, the urothelium is friable and bleeds on contact, visibility is poor because the system continues to discharge purulent material, and further papillae may separate during or shortly after the procedure. The surgeon is working against a moving target.

By 9 to 12 months the process has run its course. Separation is complete, the papillae have settled by gravity into the dependent calyces, the mucosa has healed, and the system is clear enough to inspect properly. Retrieval in the delayed group was in most cases a matter of basketing two or three well-demarcated fragments out of a lower calyx, which is a very different operation from picking at partly attached tissue in a system that will not clear.

Operative time carries more weight here than it usually does. This is not a young, fit population. Eighty-five per cent were diabetic with a mean glycated haemoglobin above 9%, and four in ten already had chronic kidney disease. Two hours of intrarenal work with sustained irrigation in such a patient is not a neutral event. Intrarenal pressure rises with irrigation and with operative duration, and pyelovenous backflow from a pressurised infected system is one of the better-established routes to post-ureteroscopy bacteraemia. [19] Diabetes, preoperative bacteriuria and prolonged operative time are all recognised risk factors for infectious complications after ureteroscopy, and our Group A patients carried every one of them. [20] Five patients in Group A grew organisms in blood and two needed intensive care. That is the visible edge of the risk.

Against this sits the renal outcome, and it will not be argued away. Chronic pyelonephritis was three times commoner after delayed clearance. The association survived adjustment for baseline chronic kidney disease, glycaemic control and age, with an adjusted odds ratio above eight, and it held in both strata of the sensitivity analysis. It was then corroborated by a measure that owes nothing to a radiologist's judgement: Group B lost filtration seven times faster. When a categorical imaging endpoint and a continuous functional endpoint move together, the finding is unlikely to be an artefact of how the scans were read.

Why should waiting damage the kidney? The stent is the obvious suspect. Group B patients carried a stent for a median of 322 days against 80, and underwent a median of two exchanges. A stent sitting in an infected diabetic urinary tract for most of a year is not a benign object. Encrustation, biofilm, bacteriuria that never quite clears, and reflux of infected urine at every void are all plausible routes to progressive parenchymal injury. [10,11] The higher symptom scores in Group B are a reminder that the patient felt this too, not only the kidney. The rival explanation, that the retained

necrotic tissue keeps a smouldering pyelonephritis going until somebody takes it out, points the same way. This design cannot separate the two.

Caution is still owed on two counts. Allocation was quasi-random rather than randomised, and while the day of a patient's admission is unlikely to select for renal disease, the imbalance in baseline chronic kidney disease shows the groups were not identical. Follow-up was also asymmetric, with Group B under observation from the index admission for roughly 18 months against 9 months for Group A. We built in two defences: the scintigraphy was timed from clearance rather than from admission, and filtration was analysed as change rather than as an absolute value. Neither fully removes the problem.

Our observations sit alongside a literature that has concentrated on the acute presentation rather than on what follows it. Kumar and colleagues, reporting 105 diabetic patients admitted with pyelonephritis to a north Indian centre, found papillary necrosis in only a small minority, a reminder that the obstructing variant we describe is a selected subgroup rather than the typical case. [4] Rafiq and colleagues followed 20 diabetic patients with emphysematous pyelonephritis for six months and recorded recurrent urinary infection in 55%, with chronic kidney disease, poor glycaemic control and obstruction identified as the associated factors. [5] Early minimally invasive drainage in the same population has been reported to avert nephrectomy in all but a small minority. [21] Our own model, in which glycated haemoglobin predicted scarring independently of group, agrees with theirs. Much of this burden is metabolic, and no decision the urologist makes about timing will fix it. The organisms we grew, with *Escherichia coli* in close to two thirds, are what regional surveillance from Indian tertiary centres would predict, and the prevalence of extended-spectrum beta-lactamase production in that setting is now high enough to shape empirical prescribing. [22] What should be done in practice? The honest answer is that we compared the two ends of a spectrum and found fault with both. Clearing at 2 to 3 months buys renal protection at the price of a long, bloody, often incomplete operation, with roughly a one in six risk of sepsis and a one in four chance of going back in. Clearing at 9 to 12 months buys a clean, quick, safe operation at the price of a scarred kidney in two thirds of patients and a mean loss of seven and a half points of filtration.

Neither is a good trade.

The more useful reading is that the optimal window probably lies between them. Demarcation of the papilla is mostly finished well before nine months. The parenchymal cost of stenting, by contrast, accrues with every extra month the stent stays in. If

both are true, going in at around four to six months should capture most of the technical benefit and avoid most of the renal cost. That is testable, and on these data it is worth testing.

Pending that, our own practice has changed in two respects. We no longer defer clearance past six months without a specific reason. A patient who is held on a stent gets glycaemic control driven hard, urine cultured on a schedule, planned exchange rather than open-ended dwell, and cortical imaging at baseline as well as at follow-up, so that new scarring can be told apart from old.

Limitations

Several limitations constrain how far these conclusions can be pushed, and the first two are serious enough that they shape everything else.

Allocation was quasi-random by admitting unit rather than randomised, and it was not concealed. The imbalance in baseline chronic kidney disease is the visible consequence and it is unlikely to be the only one. Adjustment and stratification reduce this concern but cannot eliminate it, because unmeasured confounders are by definition not in the model.

Follow-up duration differed systematically between the groups. Anchoring the scintigraphy to a fixed interval after clearance and analysing change in filtration rather than its absolute value both mitigate this, but a group observed for twice as long remains more likely to accumulate any outcome that declares itself with time.

The study was smaller than the sample size calculation required, 30 per arm against the 36 needed, and attrition of 11.7% reduced this further. Post-hoc power for the observed difference in single-session clearance was approximately 49%, so the failure of that comparison to reach significance is at least as likely to reflect sample size as absence of effect. No intention-to-treat analysis was possible for the seven patients lost. The design cannot separate the effect of stent dwell time from the effect of clearance timing, because the two are confounded by construction. A trial in which stent dwell is held constant while clearance timing varies, or the reverse, would be required. Operating surgeons were necessarily aware of group allocation, and operative time and completeness of clearance are both surgeon-assessed. Scintigraphy was read blind, which is why we regard the renal outcome as the more secure of the two findings. This is a single-centre experience. The operative findings reflect the equipment, case mix and practice of one unit, and the very high proportion of women and of poorly controlled diabetics may limit how far the results transfer.

Conclusion

In acute pyelonephritis complicated by renal papillary necrosis, deferring endoscopic clearance to 9 to 12 months makes the operation easier, quicker and safer than clearing at 2 to 3 months. It also leaves the patient with a materially worse kidney: cortical scarring in two thirds against one fifth, an adjusted odds ratio above eight, and a seven-fold greater loss of filtration.

Both of the two strategies compared here is satisfactory, and the choice between them should not be presented as a straight preference for one over the other. The balance of harms points towards an intermediate window, late enough for the papillae to have demarcated but early enough to avoid the better part of a year of stent dwell in an infected diabetic urinary tract. A randomised trial comparing clearance at four to six months against these two anchors, with prespecified imaging criteria, blinded outcome assessment and follow-up anchored to a common landmark, would answer the question properly.

Ethical Clearance: The study was conducted after obtaining the Institutional Ethical clearance

References

1. Nanra RS, Chirawong P, Kincaid-Smith P. Medullary ischaemia in experimental analgesic nephropathy: the pathogenesis of renal papillary necrosis. *Aust N Z J Med.* 1973;3(6):580-6. doi:10.1111/j.1445-5994.1973.tb04299.x
2. Liebman NC. Renal papillary necrosis and sickle cell disease. *J Urol.* 1969;102(3):294-7. doi:10.1016/S0022-5347(17)62131-1
3. Henderickx MMEL, Brits T, De Baets K, Seghers M, Maes P, Trouet D, et al. Renal papillary necrosis in patients with sickle cell disease: how to recognize this 'forgotten' diagnosis. *J Pediatr Urol.* 2017;13(3):250-6. doi:10.1016/j.jpuro.2017.01.020
4. Kumar S, Ramachandran R, Mete U, Mittal T, Dutta P, Kumar V, et al. Acute pyelonephritis in diabetes mellitus: single center experience. *Indian J Nephrol.* 2014;24(6):367-71. doi:10.4103/0971-4065.135347
5. Rafiq N, Nabi T, Rasool S, Sheikh RY. A prospective study of emphysematous pyelonephritis in patients with type 2 diabetes. *Indian J Nephrol.* 2021;31(6):536-43. doi:10.4103/ijn.IJN_411_19
6. Jung DC, Kim SH, Jung SI, Hwang SI, Kim SH. Renal papillary necrosis: review and comparison of findings at multi-detector row CT and intravenous urography. *Radiographics.* 2006;26(6):1827-36. doi:10.1148/rg.266065 039

7. Sutariya HC, Pandya VK. Renal papillary necrosis: role of radiology. *J Clin Diagn Res.* 2016;10(1):TD10-2. PMID: 26894147
8. Kranz J, Bartoletti R, Bruyère F, Cai T, Geerlings S, Köves B, et al. European Association of Urology guidelines on urological infections: summary of the 2024 guidelines. *Eur Urol.* 2024;86(1):27-41. doi:10.1016/j.eururo.2024.03.035
9. Ismail ZKA, Mohd Noor N, Mohd Shafie S. Percutaneous nephrostomy versus retrograde ureteral stenting for acute upper obstructive uropathy: a systematic review and meta-analysis. *Sci Rep.* 2021;11(1):6613. doi:10.1038/s41598-021-86136-y
10. Tomer N, Garden E, Small A, Palese M. Ureteral stent encrustation: epidemiology, pathophysiology, management and current technology. *J Urol.* 2021;205(1):68-77. doi:10.1097/JU.0000000000001343
11. Wang X, Ji Z, Yang P, Li J, Tian Y. Forgotten ureteral stents: a systematic review of literature. *BMC Urol.* 2024;24(1):52. doi:10.1186/s12894-024-01440-9
12. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Int.* 2024;105(4S):S117-314. doi:10.1016/j.kint.2023.10.018
13. Inker LA, Eneanya ND, Coresh J, Tighiouart H, Wang D, Sang Y, et al. New creatinine- and cystatin C-based equations to estimate GFR without race. *N Engl J Med.* 2021;385(19):1737-49. doi:10.1056/NEJMoa2102953
14. Sarikaya I, Sarikaya A. Current status of radionuclide renal cortical imaging in pyelonephritis. *J Nucl Med Technol.* 2019;47(4):309-12. doi:10.2967/jnmt.119.227942
15. Tsugaya M, Hirao N, Sakagami H, Ohtaguro K, Washida H. Renal cortical scarring in acute pyelonephritis. *Br J Urol.* 1992;69(3):245-9. doi:10.1111/j.1464-410X.1992.tb15521.x
16. Joshi HB, Newns N, Stainthorpe A, MacDonagh RP, Keeley FX, Timoney AG. Ureteral stent symptom questionnaire: development and validation of a multidimensional quality of life measure. *J Urol.* 2003;169(3):1060-4. doi:10.1097/01.ju.0000049198.53424.1d
17. Joshi HB, Stainthorpe A, MacDonagh RP, Keeley FX, Timoney AG. Indwelling ureteral stents: evaluation of symptoms, quality of life and utility. *J Urol.* 2003;169(3):1065-9. doi:10.1097/01.ju.0000048980.33855.90
18. Charan J, Biswas T. How to calculate sample size for different study designs in medical research? *Indian J Psychol Med.* 2013;35(2):121-6. doi:10.4103/0253-7176.116232
19. John J, Wisniewski P, Fiegggen G, Kaestner L, Lazarus J. Intrarenal pressure in retrograde intrarenal surgery: a narrative review. *Urology.* 2025;195:201-9. doi:10.1016/j.urolgy.2024.09.026
20. Ma YC, Jian ZY, Yuan C, Li H, Wang KJ. Risk factors of infectious complications after ureteroscopy: a systematic review and meta-analysis based on adjusted effect estimate. *Surg Infect (Larchmt).* 2020;21(10):811-22. doi:10.1089/sur.2020.013
21. Joshi HK, Shah VR, Parikh MD. Clinical profile and outcome of emphysematous pyelonephritis presenting to a tertiary care hospital. *Afr J Urol.* 2023;29:51. doi:10.1186/s12301-023-00380-4
22. Bajpai T, Pandey M, Varma M, Bhatambare GS. Prevalence of extended spectrum beta-lactamase producing uropathogens and their antibiotic resistance profile in patients visiting a tertiary care hospital in central India: implications on empiric therapy. *Indian J Pathol Microbiol.* 2014;57(3):407-12. doi:10.4103/0377-4929.138733.