Fenoldopam and Renal Function After Partial Nephrectomy in a Solitary Kidney: A Randomized, Blinded Trial

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OBJECTIVE To test the hypothesis that fenoldopam administration ameliorates ischemic injury, preserving the glomerular filtration rate and serum creatinine postoperatively after partial nephrectomy in patients with a solitary kidney.

MATERIALS AND METHODS Fenoldopam is a short-acting dopamine-1 receptor agonist that might provide renal protection during ischemic stress. A total of 90 patients with a solitary functioning kidney who were undergoing partial nephrectomy were randomized to fenoldopam or placebo in a double-blind protocol. The patients assigned to fenoldopam received an infusion rate of 0.1 μg/kg/min for 24 hours. The effect of fenoldopam on renal function was assessed by comparing the groups on the change in glomerular filtration rate from baseline to the third postoperative day (primary outcome) and on the change in serum creatinine over time (secondary outcome).

RESULTS Of the 90 enrolled patients, 77 provided analyzable data (43 in fenoldopam and 44 in placebo group). Fenoldopam (vs placebo) did not reduce the mean percentage of change in the glomerular filtration rate from baseline to the third postoperative day ($P = .15$), with an estimated ratio of means of 0.89 (95% confidence interval 0.69-1.09) for fenoldopam vs placebo. The postoperative serum creatinine in the 2 groups changed at comparable rates from postoperative day 1 to 4 (group-by-time interaction, $P = .72$) after adjusting for baseline creatinine, with no difference in the mean serum creatinine over time ($P = .78$).

CONCLUSION Fenoldopam administration did not preserve renal function in the clinical setting of renal ischemia during solitary partial nephrectomy, as evidenced by changes in the glomerular filtration rate or serum creatinine. UROLOGY 81: 340–346, 2013. © 2013 Elsevier Inc.

Acute kidney injury (AKI) and renal failure remain important causes of perioperative morbidity and mortality. Kheterpal et al[1] found that 2%-25% of cardiovascular surgery patients experience AKI and that patients who did had 2-5 times the risk of postoperative mortality. In patients undergoing general surgical procedures, the incidence of AKI (defined as an increase in serum creatinine >2 mg/dL or AKI necessitating dialysis) was 1%. The patients developing AKI experienced an eightfold increase in 30-day mortality.

Fenoldopam is a short-acting dopamine-1 receptor agonist that decreases systemic vascular resistance while simultaneously increasing renal blood flow in patients with normal renal function and those with chronic renal failure.[2] It is distinguished from dopamine by being solely a selective dopamine A_1 receptor agonist and lacks any β- or α-receptor activity. During the past decade, fenoldopam has been used as an off-label therapy in the setting of cardiac, liver, vascular, and renal transplantation surgery and in the intensive care unit to ameliorate AKI.

A canine model demonstrated the ability of fenoldopam to augment renal blood flow to the kidneys and other organs.[3] This increase in renal blood flow has been described to be proportionately greater to the outer medullary area of the kidney, which might result in a renal preservation effect in the anatomic portion of the kidney at greatest risk of ischemia.[4]

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An animal model of ischemic nephropathy suggested fenoldopam might attenuate ischemia/reperfusion injury.[5] Data from an in vitro study supported the idea of preconditioning with fenoldopam to induce heme oxygenase-1 as a method to protect kidneys against cold storage injury.[6]

In a review of 16 randomized studies of 1290 patients with, or at risk of, AKI, fenoldopam therapy appeared to reduce the development of acute tubular necrosis, the requirement for renal replacement therapy, and overall patient mortality.[7] The investigators reported that a larger randomized controlled trial to support their conclusion was necessary because of meta-analysis limitations. Another meta-analysis, restricted to cardiovascular surgery patients, concluded that fenoldopam reduced the need for postoperative renal replacement therapy and reduced in-hospital mortality.[8] In contrast, a more recent review of AKI, that focused on early acute tubular necrosis, did not find fenoldopam to be an effective method for preventing acute renal failure.[9]

Along with the small sample sizes, another difficulty with the previous randomized trials was that they did not necessarily restrict enrollment to very-high-risk patients, diluting their ability to identify a putative benefit of fenoldopam prophylaxis for AKI. One group at especially high risk of perioperative kidney injury is patients with an isolated kidney who require partial resection. These patients have little reserve, because they have only 1 kidney before surgery; thus, an alternate kidney cannot compensate for surgical injury. We, therefore, tested the primary hypothesis that 24 hours of perioperative fenoldopam administration preserves the mean glomerular filtration rate (GFR) during the first 3 postoperative days in such patients. Our secondary hypothesis was that fenoldopam would ameliorate the mean postoperative increase in serum creatinine.

MATERIAL AND METHODS

With institutional review board approval and written consent, we enrolled 90 patients with a solitary kidney who were scheduled for partial nephrectomy from November 2002 to April 2010. We enrolled patients undergoing partial nephrectomy of a solitary functioning kidney. Solitary kidney status resulted from various causes, including contralateral nephrectomy, a congenitally solitary kidney, or nonfunctioning atrophic kidney. The exclusion criteria were a history of current renal disease beyond the diagnosis of renal malignancy, insulin-dependent diabetes mellitus, myocardial infarction, congestive heart failure, or a major perioperative complication that would potentially affect postoperative renal function (eg, myocardial infarction, congestive heart failure, pulmonary embolus, massive hemorrhage, and refractory hypotension).

Protocol

General anesthesia was induced with sodium thiopental or propofol, fentanyl, and a nondepolarizing muscle relaxant. Anesthesia was maintained with isoflurane and 60% nitrous oxide and fentanyl. Surgery was performed with the patient in the lateral flex surgical position using open or laparoscopic techniques.

The patients were randomly assigned to fenoldopam or placebo using computer-generated codes that were maintained in sequentially numbered opaque envelopes until shortly before anesthetic induction. Drug administration was fully double-blinded. The patients assigned to fenoldopam were given an infusion of the drug at rate of 0.1 μg/kg/min beginning just after the induction of general anesthesia. The placebo group was given comparable volumes of saline. The study infusion continued for 24 hours.

Intraoperative fluid management consisted primarily of lactated Ringer's solution and limited colloid or blood transfusion to maintain the blood pressure within 20% of the individual preoperative baseline values, the central venous pressure at 12-16 mm Hg, or appropriate hematocrit. Intravenous mannitol (12.5 g) was given after anesthetic induction, before renal artery clamping, on renal artery unclamping, and before the emergence from anesthesia—for a total dosage of 50 g; 40 mg of furosemide was given intravenously after solitary kidney reperfusion. Surface hypothermia was used during renal vascular occlusion of the solitary kidney during open nephrectomy, but the patients were kept normothermic.

Patients who were undergoing open partial nephrectomy received preoperative thoracic epidural anesthesia for postoperative pain unless contraindicated or refused by the patient. Thoracic epidural dosing of 5 μg/mL fentanyl and 0.0625% bupivacaine was initiated intraoperatively at 5-8 mL/h with programmed boluses after renal resection was completed. Epidural analgesia continued postoperatively for 3-4 days. Patients undergoing laparoscopic partial solitary nephrectomy received intravenous patient-controlled analgesic narcotics.

Measurements

The baseline characteristics and details of the surgical and anesthetic management were recorded.

GFR studies were performed preoperatively and on postoperative day (POD) 3. The GFR was measured using the method devised by Israelit et al.[10] A 25-μCi dose of ^{125}I-iothalamate was administered as a subcutaneous injection without epinephrine. Marker equilibration proceeded until a total minimum of 200 mL urine was discarded. The urine was then collected, and plasma concentrations of ^{125}I-iothalamate were determined in bracketed blood samples. The GFR was calculated by the mean value of the clearance equation: GFR = UV/P, where U represents the milliliters of urine, V indicates the urine flow in mL/min, and P indicates the counts/min/mL plasma. The final GFR results were corrected to the standard body surface area. When a ^{125}I-iothalamate GFR test could not be obtained for logistical reasons (usually related to laboratory availability) either preoperatively or on POD 3, the GFR was estimated using the Modification of Diet in Renal Disease (MDRD) formula of Levey et al.[11]

Statistical Analysis

Our intended primary analysis was to assess the effect of fenoldopam vs placebo on the GFR at POD 3 with an analysis of covariance adjusting for the baseline GFR. However, because the intervention-by-baseline GFR interaction using GFR at POD 3 as the outcome was significant ($P = .006$), the analysis of covariance was not valid. We, therefore, used the GFR percentage of change from baseline to POD 3 as the primary

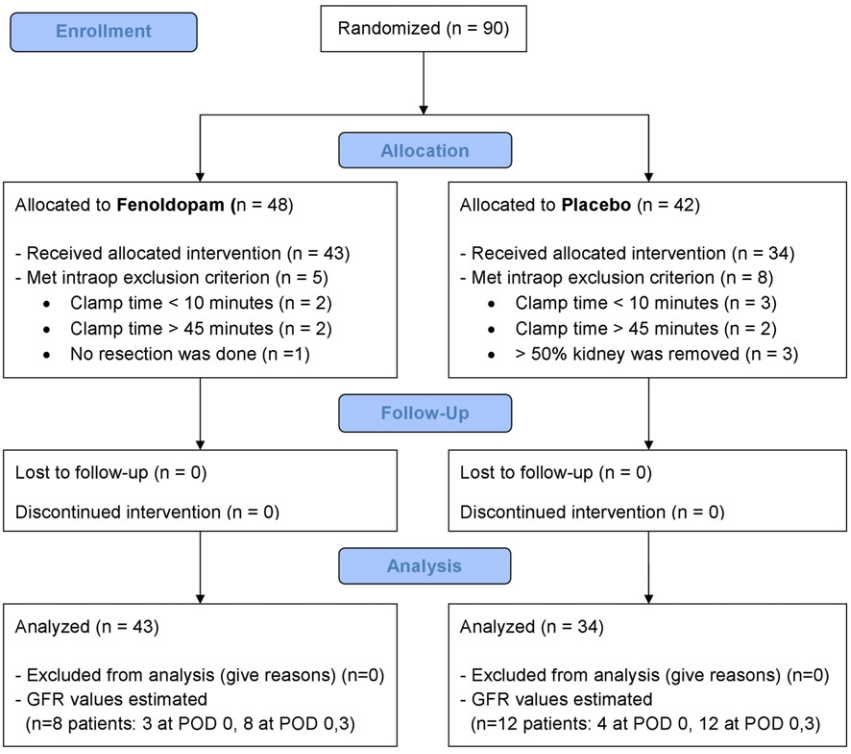

Figure 1. Consort study flow diagram. GFR, glomerular filtration rate; POD, postoperative day. (Color version available online.)

outcome and assessed the fenoldopam effect using the Wilcoxon rank-sum test. The results are reported as the mean ± standard deviation (SD) for each group and interim-adjusted confidence intervals for the difference in mean values.

In the secondary analyses, we assessed the effect of fenoldopam on creatinine over time (immediately postoperatively and on PODs 1-4) and the interaction between the fenoldopam effect on the percentage of change in GFR and several baseline and intraoperative variables. A linear mixed effects model was used to assess the main effect of fenoldopam on the postoperative log-transformed (base 2) serum creatinine and the fenoldopam-by-time interaction, adjusting for baseline serum creatinine. We, a priori, planned to further assess the effect of fenoldopam on serum creatinine at each point, regardless of the interaction, adjusting for multiple comparisons using the Holm-Bonferroni method.[12] Finally, we compared the randomized groups using the RIFLE classification system for AKI in the postoperative phase.[13]

We also assessed the interaction between the effect of fenoldopam and 3 a priori-specified factors on the primary outcome of the percentage of change in GFR from baseline to POD 3 using analysis of covariance: clamp time (>20 vs <20 min), amount of resection (>20% vs <20%), and baseline serum creatinine (>1.5 vs <1.5 mg/dL). Given a significant interaction, Bonferroni's correction for testing within factor levels was made.

The trial was designed to enroll a maximum of 111 patients to have 90% power at the 0.05 significance level to detect a difference of ≥20 in the mean percentage of change in GFR from baseline to POD 3, assuming a SD of about 30, such as was observed in our pilot data. Similarly, we had 90% power to detect a ≥25% reduction in the mean serum creatinine over time (PODs 1-4), assuming a log-scale SD of 0.50 at each point (our pilot data SD estimates ranged from 0.45 to 0.60). Calculations included plans for interim analyses at each one third of the target enrollment to assess the efficacy and futility using group sequential methods and a γ-spending function ($\gamma = -4$ for efficacy and -1 for futility). Although 3 analyses were planned, the chosen spending function was flexible; thus, interim analyses could be conducted at any point during the trial if necessitated for logistics or other reasons, as designated by the study's executive committee.

The results are presented as the mean ± SD or percentages unless otherwise indicated. SAS, version 9.2, software (SAS Institute, Cary, NC), R statistical software, version 2.7.2 (R Foundation for Statistical Computing, Vienna, Austria), and East 5 software (Cytel, Cambridge, MA) were used for the analyses and graphics.

RESULTS

Of 90 randomized patients, 13 (5 fenoldopam, 8 placebo) met the postrandomization intraoperative exclusion criteria, leaving 77 patients for the analysis (Fig. 1). The baseline and intraoperative covariables were well-balanced between the randomized groups (Table 1). The mean ± SD ischemia time was 22 ± 8 minutes for fenoldopam and 23 ± 7 minutes for placebo; the renal resection fraction was 23% ± 14% and 25% ± 12% for the 2 groups. GFR was directly measured in 74% of the patients and estimated in the remainder.

Owing to slower than expected enrollment, interim analyses were conducted at 29, 63, and 77 patients. No futility or efficacy boundary for the primary outcome of the percentage of change from the baseline GFR to POD

Table 1. Summary of demographic, baseline and intraoperative factors

Variable	Fenoldopam (n = 43)	Placebo (n = 34)
Demographic data		
Age (y)	59 ± 10	59 ± 9
Height (cm)	172 ± 10	177 ± 8
Weight (kg)	92 ± 19	92 ± 17
Body mass index (kg/m^2)	31 ± 6	29 ± 5
Male sex*	33 (79)	29 (85)
White race	42 (98)	31 (91)
Baseline		
GFR (mL/min)†	59 ± 19	59 ± 19
Sodium (mmol/L)*	139 ± 2	140 ± 2
Potassium (mmol/L)*	4 ± 0	4 ± 1
Chloride (mmol/L)*	102 ± 2	102 ± 2
Carbon dioxide (mmol/L)*	25 ± 3	26 ± 3
Anion gap (mmol/L)*	11 ± 2	11 ± 3
BUN (mg/dL)*	18 [16, 22]	20 [17, 24]
Creatinine (mg/dL)	1.2 [1.0, 1.4]	1.2 [1.0, 1.4]
Glucose (mg/dL)*	94 [86, 110]	93 [84, 98]
Calcium (mg/dL)*	10 [9, 10]	10 [9, 10]
HR (beats/min)	78 ± 14	73 ± 13
Systolic blood pressure (mm Hg)	140 ± 26	140 ± 18
Diastolic blood pressure (mm Hg)	80 ± 8	80 ± 11
Right partial nephrectomy (%)*	22 (52)	19 (56)
Intraoperative		
Angle of bed (°)†	33 ± 7	34 ± 8
Estimated blood loss (mL)	350 [200, 525]	300 [150, 438]
Total IVF (mL)*	4942 ± 1571	4992 ± 1473
Urine (mL)*	500 [362, 802]	540 [389, 915]
Kidney removed (%)‡	23 ± 14	25 ± 12
Duration of case (min)*	323 ± 52	338 ± 64
Total clamp time (min)*	22 ± 8	23 ± 7

BUN, blood urea nitrogen; GFR, glomerular filtration rate; HR, heart rate; IVF, intravenous fluids.

Data presented as mean ± SD, median [quartile 1, quartile 3], or n (%).

* One to three data points missing.

† Six to seven data points missing.

‡ Four data points missing.

3 was crossed at any of the analyses. However, at 77 patients, the conditional power, defined as the probability of finding a statistically significant result if the current trend were to continue for the remainder of the trial, was only 28%. Thus—and because of the slow enrollment—the executive committee decided to stop enrollment at 77 patients. At the last analysis, the P value boundary for efficacy and futility was $P < .010$ and $P > .302$, respectively.

Fenoldopam did not significantly affect the percentage of decrease in GFR from baseline to POD 3, with mean ± SD reduction of 28% ± 38% for fenoldopam and 39% ± 28% for placebo ($P = .15$; Fig. 2A). The mean decrease in GFR was an estimated 11% less for fenoldopam than for placebo (interim-adjusted 95% confidence interval 9% more, 31% less). The mean postoperative serum creatinine (in the mixed effects model collapsed over time) did not differ between the randomized groups, with an estimated ratio of geometric mean of 0.96 (interim-adjusted 95% confidence interval 0.78-1.19) for fenoldopam versus placebo ($P = .64$), adjusting for the baseline serum creatinine. The treatment effect estimates at each point are provided in Table 2, none of which was statistically significant. Furthermore, no interaction was found between the randomized group and time on the mean postoperative serum creatinine (immediate postoperatively through POD 4, $P = .72$; Fig. 2B). We compared the randomized groups for the change in kidney function from baseline through the postoperative period as measured using the RIFLE classification system. No difference was found for either the change in GFR from baseline to POD 3 ($P = .23$) or in the maximal increase in serum creatinine from baseline through POD 4 ($P = .79$).

Although the fenoldopam effect on the percentage of change in GFR did not depend on the clamp time ($P = .18$) or the amount of resection ($P = .20$), it did appear to depend on the baseline serum creatinine concentration being >1.5 or <1.5 mg/dL (borderline interaction, $P = .011$). Fenoldopam appeared to be protective when the serum creatinine was <1.5 mg/dL ($P = .019$) but appeared to be harmful when the baseline serum creatinine was ≥1.5 mg/dL ($P = .09$). However, neither effect was statistically significant after adjusting for the interim analyses and multiple comparisons.

For our main analyses (see previous paragraphs), 26% of the GFR measurements at POD 3 were estimated using the MDRD equation, because the measured GFR at POD 3 was unavailable for those patients. We conducted 2 sensitivity analyses to assess the robustness of our methods: 1 using the Chronic Kidney Disease Epidemiology Collaboration formula to estimate the missing GFR values[14] and another estimating all POD GFR values at POD 3 using the MDRD equation. In both cases (data not shown), the results were very similar, and the conclusions were the same as for our primary analysis.

COMMENT

In a randomized and blinded trial of partial nephrectomy in patients with a solitary kidney (n = 77 usable data), fenoldopam infusion for 24 hours did not improve postoperative renal function compared with saline placebo.

Our trial differs from previous evaluations of fenoldopam in restricting the study population to patients at risk of AKI by virtue of their requiring partial resection of a solitary functioning kidney. Consistent with their risk, the serum creatinine had nearly doubled to 2.5 ± 1.5 mg/dL by POD 4. Similarly, the GFR decreased from 60 ± 18 to 40 ± 24 mL/min. These changes from baseline values classified these patients to be at "risk" or to have encountered "injury" according to the RIFLE classification system for the degree of AKI during partial

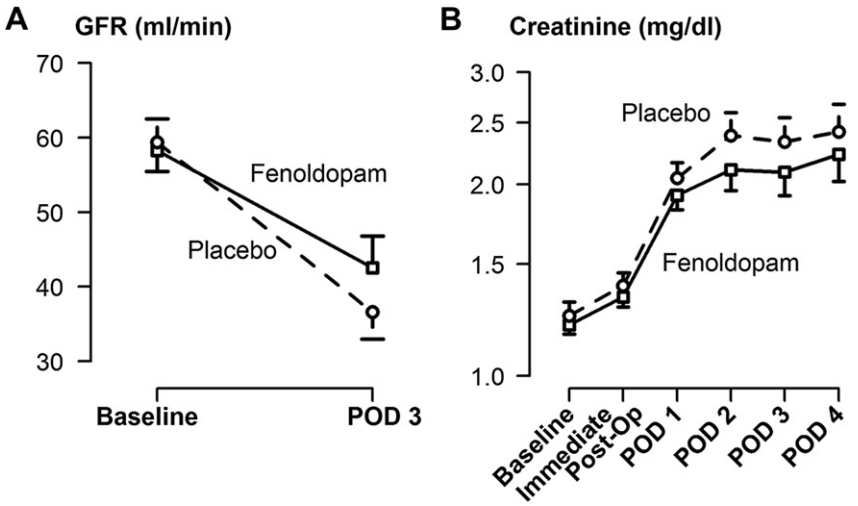

Figure 2. Plot of **(A)** mean and standard error of glomerular filtration rate (GFR) at baseline and postoperative (POD) 3; and **(B)** geometric mean and standard error of serum creatinine during postoperative (Post-Op) period.

Table 2. Effect of fenoldopam on mean serum creatinine over time

Measurement Point	Ratio of Geometric Mean Fenoldopam vs Placebo (95% CI)*†	*P* Value†
Overall	0.98 (0.79-1.19)	.78
Immediate postoperatively	1.01 (0.80-1.29)	.87
POD 1	0.99 (0.78-1.25)	.89
POD 2	0.94 (0.74-1.19)	.48
POD 3	0.93 (0.73-1.18)	.43
POD 4	0.95 (0.75-1.21)	.61

* 95% Confidence intervals adjusted for interim analysis ($z = 2.58$).

† Linear mixed effects regression model on log-transformed (base 2) serum creatinine adjusting for baseline serum creatinine; treatment-by-time interaction nonsignificant ($P = .72$).

solitary nephrectomy.[13] Nearly all our patients thus experienced a clinically important reduction in kidney function, providing a good opportunity to demonstrate protection by fenoldopam.

Fenoldopam, however, did not, on average, significantly reduce perioperative AKI. Although it remains possible that a larger study would demonstrate a statistically significant benefit, it seems unlikely that the benefit would be clinically important. Thus, our results suggest that clinicians and investigators should focus on other renal protective strategies.

An interesting feature of our results was an interaction between the baseline serum creatinine and the protective effect of fenoldopam. Specifically, there appeared to be a benefit when baseline serum creatinine was <1.5 mg/dL, but fenoldopam appeared to be harmful when the baseline creatinine was greater. However, this was not a preplanned analysis, and there is little physiologic basis for expecting effect modification on the basis of the creatinine level. Most likely, the observation—which was unexpected and unplanned—was spurious. The effect of fenoldopam did not depend on either the fraction of kidney resected or cross-clamp time of the solitary kidney.

The GFRs were directly measured preoperatively and on the POD 3 in 74% of patients; 26% of the values were estimated from serum creatinine using the well-established MDRD equation.[11] To the extent that the calculations under- or overestimated the actual GFR, our ability to detect a beneficial effect of fenoldopam would be degraded. However, it seems unlikely that measurement error was the primary explanation for our generally negative results. The conclusions were unchanged and results very similar when we conducted a sensitivity analysis using the Chronic Kidney Disease Epidemiology Collaboration formula to estimate the missing GFR values.[14] The latter formula has been shown to be more accurate than the MDRD equation for patients with normal renal function. However, our patients had serious renal insufficiency, making the original MDRD equation appropriate. The results were also very similar (and conclusions unchanged) from a sensitivity analysis using the MDRD equation for all patient values at POD 3.

A more serious problem was that the study was stopped before crossing our a priori futility boundary. However, the results were extremely close to the futility boundary at 2 sequential interim analyses, and the conditional power estimates using a variety of assumptions indicated very little chance of identifying a statistically significant (or clinically important) benefit of fenoldopam even if the trial were continued to completion. It, thus, seems unlikely that enrolling additional patients would have substantively altered our conclusions. However, previous work has suggested that fenoldopam might[15] or might not[16] provide renal protection for critical care patients with AKI, although it does not appear to ameliorate contrast-induced nephropathy.[17,18]

The present study had several limitations. The number of patients diagnosed with solitary renal cell carcinoma who present as eligible for partial nephrectomy was limited

and resulted in a study enrollment period of 8 years. Initially, all cases were managed with open surgery (total with open surgery, n = 72). However, by study end, nearly all approached patients were scheduled for laparoscopic surgery (total n = 5). A consequence beyond the surgical technique was that surface hypothermia could not be used in the laparoscopic cases. We infused 0.1 μg/kg/min of fenoldopam. This dose increases renal blood flow without much reducing the systemic vascular resistance.[2] It remains possibly that a greater dose would preserve renal function; however, it would likely also increase the risk of hypotension—which itself can damage kidneys. The effect of fenoldopam in the present study was only reported for the immediate postoperative period. Evaluation at 3 or 6 months postoperatively might have demonstrated different findings after the potential renal ischemia recovery time had occurred. Postoperative pain control was switched from epidural to an intravenous patient-controlled opioid. With randomization, both groups should have had comparable time-dependent changes affecting each similarly; thus, this was unlikely to have influenced our overall findings.

CONCLUSIONS

Fenoldopam administration did not preserve renal function, as evidenced by changes in GFR or serum creatinine even in patients undergoing partial nephrectomy of their sole remaining kidney. Efforts to preserve renal function might thus best be directed at other potential prophylactic measures.

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EDITORIAL COMMENT

Preservation of kidney function is a critical outcome for many patients undergoing surgery for renal cortical tumors.[1] It has been well documented that several factors come into play when determining postoperative kidney functional outcomes. Although many of these factors are consider nonmodifiable, such as age, sex, preoperative GFR, and renal nephrometry score, others factors are thought to be modifiable, such as ischemia type and time and volume of resected normal renal parenchyma.[2] Another potential method of improving kidney function outcomes is through the use of renoprotective agents.

It has been hypothesized that kidney injury after partial nephrectomy occurs through an ischemic/reperfusion mechanism. In addition to using renoprotective techniques such cold ischemia and minimal ischemia, surgeons frequently use pharmacologic agents during partial nephrectomy such as mannitol and furosemide (Lasix), which are thought to maximize reperfusion and provide postperfusion diuresis, respectively. Renoprotective agents are also commonly used to minimize AKI in a variety of settings other than kidney surgery. One such agent is fenoldopam, a short-acting dopamine-1 receptor agonist. Fenoldopam has been frequently used "off -label" to ameliorate AKI after cardiovascular surgery and renal transplantation surgery and in the intensive care unit setting.[3,4]

In this study, patients with a solitary functioning kidney undergoing partial nephrectomy were randomized to fenoldopam or placebo to evaluate the effect of a putative renoprotective agent on preserving kidney function in the early postoperative period. This study had a few shortcomings worth mentioning, including premature closure because of difficulties with accrual and a lack of results beyond the initial postoperative period. This was ultimately a negative study,

with no difference found in postoperative GFR between the fenoldopam and placebo group, regardless of the ischemia time or amount of resected tissue. Although the difference was not statistically significant, fenoldopam showed a trend toward causing harm in patients with a baseline serum creatinine ≥1.5 mg/dL ($P = .09$).

Negative studies such as this are frequently not published, which does not mean that they are without merit. The investigators should be praised for their insight and "forward thinking," because this study serves as a basis for future studies exploring renoprotective agents. By selecting patients with a solitary functioning kidney, the investigators chose the best "in vivo" model because no contralateral kidney was present to confound the results. Impressively, they also measured the GFR in nearly three fourths of all the study patients using ^{125}I-iothalamate instead of estimating the GFR using serum creatinine-based formulas. Hopefully, in the near future, there will be more such studies investigating renoprotective agents for use during partial nephrectomy.

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REPLY

I appreciate the editorial comments and agree with the shortcomings stated for this study using a fenoldopam infusion for 24 hours during partial solitary nephrectomy in an attempt to minimize perioperative AKI. Because of the limited number of eligible patients to approach with a solitary kidney it did take several years to conclude. The collaboration with the urologic surgeons involved was vital in being able to complete this clinical study.

Enthusiasm for fenoldopam use in clinical medicine to ameliorate renal injury gained in popularity about a decade ago, but has diminished since. Several early studies included small sample sizes (<40 patients) and were not randomized. A meta-analysis on the effect of fenoldopam in patients from 16 randomized studies involving 1290 patients by Landoni et al[1] in 2007 concluded that fenoldopam reduced the need for renal replacement and mortality in patients with AKI. Although this suggested a positive benefit, the investigators stated there is a need for a large, multicenter, appropriately powered trial to confirm these results. This type of fenoldopam study is still lacking. Another fenoldopam review suggested prophylactic fenoldopam use in cardiovascular surgery patients but not for prophylaxis of contrast nephropathy.[2] Kellum et al[3] provided a recent review of interventions to prevent AKI in randomized controlled studies. This included fenoldopam vs placebo, vs dopamine, and vs other treatments/controls. Overall, the conclusion was that fenoldopam was unlikely to be beneficial to prevent AKI.[3]

Our study used the clinical setting of a patient with a solitary kidney who experienced a defined period of complete interruption of renal blood flow during partial nephrectomy. This created the potential for ischemia/reperfusion AKI. This allowed us to study the effect of a fenoldopam infusion before and after solitary kidney ischemia. Our results have contributed to determining whether fenoldopam has a renal preservation benefit in ischemia/reperfusion AKI. Our conclusion was that fenoldopam did not provide a benefit in this setting.

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References

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