Original Article

# Acute Pyelonephritis and Renal Abscesses in Adults—Correlating Clinical Parameters with Radiological (Computer Tomography) Severity

Sey Kiat Lim,[1] *MBBS, MRCS, MMed (Surgery)*, Foo Cheong Ng,[1] *FRCS, FICS, FAMS (Urology)*


## Abstract

**Introduction: The aim of this study is to evaluate the correlations between clinical and biochemical parameters with radiological severity of CT diagnosed acute pyelonephritis (APN) and renal abscesses, thereafter defining clinical predictors to identify patients with severe APN or renal abscesses. Materials and Methods: The inpatient medical records of all patients diagnosed with APN or renal abscesses admitted over one year were reviewed. Patients with CT imaging performed were classified into 3 groups—mild APN, severe APN and renal abscesses. Clinical and biochemical parameters were correlated with radiological severity. Results: One hundred and thirty patients were included in the study. Male gender, older age, presence of diabetes mellitus and unobstructing renal stones were significantly associated with severe APN or renal abscesses. Clinical and biochemical parameters that were associated with more severe disease include a higher leucocyte count and C-reactive proteins, left neutrophil shift, thrombocytosis or thrombocytopenia, low serum albumin, acute renal impairment and bacteremia. The percentage of patients had positive urine and blood cultures were 40.8% and 30.7% respectively. Of these patients, 97.9% had severe APN or renal abscesses on CT imaging had diabetes mellitus (DM), hypotension, acute renal failure or leucocyte count of >20K. Conclusion: Our study showed a good correlation between clinical and radiological severity in adult patients with APN and renal abscesses. Patients with severe APN or renal abscesses were likely to be diabetics presenting with hypotension, acute renal impairment and a leucocyte count of greater than 20K.**


**Ann Acad Med Singapore 2011;40:407-13**

**Key words: Computer topography (CT), Pyelonephritis, Renal abscess, Severity**

## Introduction

Acute pyelonephritis (APN) occurs in more than 250,000 adults in the United States every year.[1] The economic implication of APN is tremendous: an estimated cost of US$2.14 billion in the United States in the year 2000.[2] Most patients with APN have mild symptoms and can be managed in an outpatient setting.[3] However, 10% to 30% of patients with APN require hospitalisation[2] and may even present with life-threatening complications including shock, septicemia and multi-organ dysfunction syndromes. Indeed mortality rates ranges from 1% to 12%.[4,5]

Bacterial nephritis or renal infection can be regarded as a spectrum of clinical entities, progressing from mild APN to renal abscesses or emphysematous pyelonephritis. APN can be defined clinically, pathologically or radiologically. The British Medical Research Council Bacteriuria Committee defined APN as a clinical syndrome of flank pain, costovertebral tenderness and fever accompanied by laboratory evidences of renal infection including leukocytosis, pyuria, haematuria, bacteriuria, positive urine culture and sometimes bacteraemia.[6] Hill[7] defined APN pathologically as a suppurative inflammation of the renal parenchyma and pyelocaliceal system typically distributed along one or more medullary rays supporting an ascending route of infection. Radiologically, APN manifests on contrasted computed tomography (CT) scans as hypoenhancing regions with or without renal swelling and may be focal or diffused. To standardise terminologies, Talner et al[8] suggested in 1994 that all radiological parenchymal abnormalities without abscess attributable to acute infection be called APN.

Risk factors for APN have been extensively studied.

[1]Department of Urology, Changi General Hospital
Address for Correspondence: Dr Lim Sey Kiat, Department of Urology, Changi General Hospital, 2 Simei Street 3, Singapore 529889.
Email: limseykiat@gmail.com

Female gender, diabetes mellitus, immunosuppressed state, postmenopausal, history recurrent urinary tract infection and vesicoureteric reflux are common examples.[9] However, risk factors for severe APN or development of renal abscesses are less well studied. It is often difficult to characterise the severity of APN with clinical or laboratorial parameters.[10,11] Radiological images such as CT when available help delineate the nature, extent and severity of disease and reveal complications such as abscess or obstruction.[11] Few data are available to correlate the clinical severity of APN and renal abscesses with radiological severity. Even fewer data exist to recommend when CT imaging should be performed in patients presenting with symptoms suggestive of APN. The aim of this study to assess the correlations between clinical and biochemical parameters with radiological severity of CT diagnosed APN and renal abscesses, thereafter defining clinical predictors to identify patients with severe APN or renal abscesses.

## Materials and Methods

### *Patients and Study Setting*

All patients diagnosed with APN or renal abscesses admitted to Changi General Hospital, Singapore from 1 January 2008 to 31 December 2008 were identified from a patient diagnosis database and the International Statistical Classification of Diseases and Related Health Problems Ninth Edition (ICD 9) codes. The inpatient medical records of all patients identified were retrospectively reviewed and patients who satisfy the inclusion and exclusion criteria were included in the study.

### *Diagnostic Criteria*

Diagnosis of APN and renal abscesses were based on both clinical and radiological criteria. Clinical criteria were the presence of "classical" symptoms of APN.[6] (i) Fever, defined as a tympanic temperature of greater than 37.5°C;[12] (ii) Pyuria, defined as greater than 10 white blood cells per high-power field of centrifuged urine; (iii) Presence of loin or flank pain with or without lower urinary tract symptoms. Presence of positive urine or blood cultures was not mandatory for diagnosis. Hypotension in our study is defined as a systolic blood pressure of <90 mmHg or a fall in systolic pressure of >40 mmHg.[13] Acute renal impairment is defined as a 25% decrease in GFR from baseline in accordance to the International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) group.[14] In our study, GFR is calculated based on the MDRD Study Equation. If the baseline GFR is not known, acute renal impairment is defined as a GFR of less than 60 ml/min/1.73m$^2$.

Radiological diagnosis of APN and renal abscesses were made when CT showed radiological evidence of infection of renal parenchyma. Degrees of renal involvement were classified into 3 groups according to severity as shown in Table 1.[11] Patients fulfilling both clinical and radiological criteria were included in the analysis. Patients without CT imaging documenting radiological evidence of APN or renal abscesses during the admission were excluded. Also patients with obstructing renal or ureteric stones and diagnosed with pyonephrosis were excluded from the study.

Table 1. Degrees of Renal Involvement Classified According to Severity

| Group | Description |
|---|---|
| Simple APN Group 1 | The lesions appear focal, wedge-shaped, radiating from the papilla in the medulla to the cortical space, with or without swelling. |
| Severe APN Group 2 | Lesions are multi-focal or diffuse and heterogenous. There may be more thickening of the Gerota's fascia and more pronounced perinephric strandings. |
| Abscesses Group 3 | Lesions are target-shaped and the central core does not show contrast enhancement. The peripheries of the lesions take up the contrast and enhance in a late phase. In non-contrast scans, lesions are of a low-attenuation (0-20 Hounsfield units), distinctly marginated, parenchymal lesions with mixed density of soft tissue and fluid. |

APN: Acute pyelonephritis

### *Data Collection and Statistical Analysis*

Patients' demographics, medical histories, clinical parameters, biochemical and laboratory variables, records of micro-organisms in urine and blood cultures and their sensitivities to antibiotics, types and duration of antibiotic therapies and condition at discharge were reviewed.

Statistical analysis was performed with Statistical Package for the Social Sciences (SPSS) Version 15 sofware. Descriptive studies were performed for the cohort. Univariate analysis was performed to compare clinical and biochemical variables with radiological severity of APN and renal abscesses using cross tabulation and chi-square tests for categorical variables and Kruskal-Wallis (non-parameteric) or T-tests (parameteric) for continuous variables. Multivariate analysis considering radiological severity of disease as dependent variable was performed with statistical significant factors and covariates from univariate analysis. All tests of significance were considered statistically significant when $P$ <0.05.

## Results

### *Demographics and Medical History of Patients*

A total of 130 patients met the inclusion and exclusion criteria and were analysed. Contrast or non-contrasted CT

were performed for all the patients and reported by in-house radiologists. Thirty-four non-contrasted CT were performed in view of renal impairment and metformin usage at the requesting physician's discretion. Eighty-two (63.1%) patients were classified as having radiological mild APN (Group 1), 25 (19.2%) having severe APN (Group 2) and 23 (17.7%) having abscesses (Group 3). The demographics and medical histories of the patients are shown in Table 2. Patients who were classified radiologically into Groups 2 and 3 were significantly older. Although APN affects more females, there were a statistically significant higher proportion of males with more severe disease (Groups 2 and 3). Patients with diabetes mellitus (DM), hypertension or non-obstructing renal stones were associated with radiologically more severe APN or renal abscesses. However, patients with poorly controlled DM, compared with patients with well-controlled DM, were not found to be associated with more severe disease (Group 1 versus Groups 2 and 3). Race, activities of daily living (ADL) status, baseline renal function, presence of long-term indwelling urinary catheters and previous histories of APN or cystitis were not found to be associated with increased severity of disease (Table 2).

The presence of non-obstructing nephrolithiasis was

Table 2. Demographics and Comorbidities of Patients

| | | **Group 1 n = 82** | **Group 2 n = 25** | **Group 3 n = 23** | ***P* value** |
|---|---|---|---|---|---|
| Age | ± SEM | 44.8 ± 2.4 | 58.6 ± 3.0 | 53.0 ± 3.5 | 0.004 |
| Gender (%) | Male | 8 (9.8%) | 6 (24.0%) | 8 (34.8%) | 0.011 |
| | Female | 74 (90.2%) | 19 (76.0%) | 15 (65.2%) | |
| Race (%) | Chinese | 45 (54.9%) | 13 (52.0%) | 13 (56.5%) | 0.773 |
| | Malay | 24 (29.3%) | 7 (28.0%) | 8 (34.8%) | |
| | Indian | 7 (8.5%) | 4 (16.0%) | 2 (8.7%) | |
| | Others | 6 (7.3%) | 1 (4.0%) | 0 | |
| Site of APN (%) | Right | 45 (54.9%) | 9 (36.0%) | 12 (52.2%) | 0.599 |
| | Left | 27 (32.9%) | 10 (40.0%) | 8 (34.8%) | |
| | Bilateral | 9 ( 12.2%) | 6 (24.0%) | 3 (13.0%) | |
| ADL Status | Independent | 79 | 25 | 22 | 0.588 |
| | Wheelchair | 1 | 0 | 1 | |
| | Bedbound | 2 | 0 | 0 | |
| Chronic Indwelling Catheters (%) | Yes | 3 (3.7%) | 2 (8.0%) | 2 (8.7%) | 0.519 |
| | No | 79 (96.3%) | 23 (92.0%) | 21 (91.3%) | |
| Previous APN (%) | Yes | 9 (11.0%) | 5 (20.0%) | 0 | 0.082 |
| | No | 73 (89.0%) | 20 (80.0%) | 23 | |
| Diabetes Mellitus (%) | Yes | 21 (25.6%) | 13 (52.0%) | 14 (60.9%) | 0.002 |
| | No | 61 (74.4%) | 12 (48.0%) | 9 (39.1%) | |
| HbA1C (%) | ± SEM | 8.57 ± 0.49 n = 19 | 10.35 ± 0.99 n = 11 | 9.47 ± 0.81 n = 13 | 0.282 |
| Hypertension (%) | Yes | 17 (20.7%) | 10 (40.0%) | 11 (47.8%) | 0.017 |
| | No | 65 (79.3%) | 15 (60.0%) | 12 (22.2%) | |
| Chronic Renal Impairment (%) | Yes | 4 (4.9%) | 1 (4.0%) | 0 | 0.561 |
| | No | 78 (95.1%) | 24 (96.0%) | 23 | |
| Premorbid Creatinine (umol/L) | ± SEM | 87.2 ± 7.9 n = 69 | 83.7 ± 3.8 n = 23 | 77.1 ± 2.5 n = 23 | 0.424 |
| Premorbid GFR (ml/min/1.73m$^2$) | ± SEM | 81.8 ± 3.2 n = 69 | 73.5 ± 3.2 n = 23 | 83.0 ± 3.3 n = 23 | 0.109 |
| Unobstructing Renal Stones (%) | Yes | 8 (9.8%) | 9 (36.0%) | 4 (18.4%) | 0.008 |
| | No | 74 (90.2%) | 16 (64.0%) | 19 (81.6%) | |

SEM: Standard error of the mean

associated with positive urine cultures. Of these patients, 61.9% (13 out of 21) with nephrolithiasis had bacteriuria compared with 36.7% (40 out of 109) of patients without nephrolithiasis having positive urine cultures ($P = 0.031$).

### *Clinical and Laboratory Parameters*

The results are shown in Tables 3 and 4. Raised inflammatory markers like increased leucocytosis, left neutrophil shift and raised C-reactive proteins (CRP) were associated with more severe disease. Patients in Groups 2 and 3 were statistically more likely to have acute renal impairment with a raised serum creatinine and decreased glomerular filtration rate (GFR). There were good correlations between clinical and radiological severity of disease. Hypotension, requirement for inotropes, longer duration of intravenous antibiotics and longer duration of hospitalization were associated with more severe disease. Although a longer duration of fever was found in patients in Groups 2 and 3 compared with Group 1, the maximum temperature was not shown to be statistically different among the 3 groups. Only 40.8% of patients had positive urine culture and culture positivity was not significantly different among the 3 groups. In contrast, although a positive blood culture was present only in 30.7% of the patients, patients in Groups 2 and 3 were more likely to have a positive

Table 3. Clinical Parameters

| | | **Group 1 n = 82** | **Group 2 n = 25** | **Group 3 n = 23** | ***P* value** |
|---|---|---|---|---|---|
| Hypotension (%) | Yes | 6 (7.3%) | 12 (48.0%) | 10 (43.5%) | <0.001 |
| | No | 76 (92.7%) | 13 (52.0%) | 13 (56.5%) | |
| Inotropes (%) | Yes | 1 (1.2%) | 8 (32.0%) | 4 (17.4%) | <0.001 |
| Duration of Fever (days) | ± SEM | 2.2 ± 0.2 | 3.4 ± 0.6 | 5.4 ± 1.0 | 0.001 |
| Highest Temperature of Fever (°C) | ± SEM | 38.5 ± 0.1 | 38.7 ± 0.2 | 38.6 ± 0.1 | 0.747 |
| Antibiotics Before Hospitalisation (%) | Yes | 4 (4.9%) | 7 (28.0%) | 2 (8.7%) | 0.003 |
| | No | 78 (95.1%) | 18 (72.0%) | 21 (91.3%) | |
| Duration of Hospitalisation (days) | ± SEM | 5.3 ± 0.4 | 9.8 ± 1.5 | 14.3 ± 2.0 | <0.001 |

SEM: Standard error of the mean

Table 4. Laboratory Parameters

| | | **Group 1 n = 82** | **Group 2 n = 25** | **Group 3 n = 23** | ***P* value** |
|---|---|---|---|---|---|
| Hb (g/dL) | ± SEM | 12.6 ± 0.2 | 12.2 ± 0.3 | 11.7 ± 0.5 | 0.148 |
| Hct (%) | ± SEM | 37.8 ± 0.7 | 36.4 ± 1.0 | 34.5 ± 1.2 | 0.050 |
| Total white ($x10^3$/uL) | ± SEM | 13.2 ± 0.4 | 18.8 ± 1.3 | 21.1 ± 1.3 | <0.001 |
| Neutrophils (%) | ± SEM | 81.6 ± 1.1 | 85.9 ± 1.7 | 85.9 ± 3.0 | 0.008 |
| CRP (mg/L) | ± SEM | 137 ± 17 n = 43 | 223 ± 25 n = 17 | 241 ± 29 n = 19 | 0.003 |
| Platelets ($x10^3$/uL) | ± SEM | 287 ± 10 | 247 ± 24 | 319 ± 53 | 0.192 |
| Normal Platelets Range (%) | Yes | 73 (89.0%) | 19 (76.0%) | 15 (65.2%) | 0.009 |
| | No | 6 (11.0%) | 6 (24.0%) | 8 (34.8%) | |
| Serum Albumin (g/L) | ± SEM | 33 ± 1 n = 26 | 30 ± 2 n = 12 | 21 ± 1 n = 12 | <0.001 |
| Acute Renal Impairment (%) | Yes | 18 (22.0%) | 19 (76.0%) | 20 (87.0%) | <0.001 |
| | No | 61 (78.0%) | 6 (24.0%) | 3 (13.0%) | |
| Highest Creatinine (umol/L) | ± SEM | 101 ± 11 | 196 ± 42 | 256 ± 50 | <0.001 |
| GFR During Hospitalisation (ml/min/1.73m$^2$) | ± SEM | 76.2 ± 3.6 | 42.2 ± 4.6 | 36.2 ± 5.1 | <0.001 |
| Urine Culture (%) | Positive | 36 (43.9%) | 8 (32.0%) | 9 (39.1%) | 0.561 |
| | Negative | 46 (56.1%) | 17 (68.0%) | 14 (60.9%) | |
| Blood Culture (%) | Positive | 16 (19.5%) | 10 (40.0%) | 14 (60.9%) | <0.001 |
| | Negative | 66 (80.5%) | 15 (60.0%) | 9 (39.1%) | |

SEM: Standard error of the mean

culture than those in Group 1. Presence of bacteremia was significantly associated with hypotension. Of these patients, 46.4% with bacteremia were hypotensive while 26.5% of patients with bacteremia were not hypotensive ($P = 0.043$). Twelve patients with renal abscesses required percutaneous drainage of their abscesses. Only 1 patient in the study cohort died from severe APN, giving a mortality rate of 0.77%.

*Multivariate Analysis*

On multivariate analysis, 4 factors were found to be statistically significant. They were the presence of diabetes mellitus (DM), hypotension, acute renal impairment and a total white count of greater than 20K. When compared with simple pyelonephritis, patients with DM ($P = 0.09$, OR 2.90), hypotension ($P = 0.050$, OR 7.1), acute renal impairment ($P = 0.001$, OR 7.9) and a total white of greater than 20K ($P = 0.008$, OR 7.6) have severe pyelonephritis. When compared with simple pyelonephritis, patients with DM ($P = 0.016$, OR 5.8), hypotension ($P = 0.044$, OR 4.7), acute renal impairment ($P = 0.001$, OR 13.4) and a total white of greater than 20K ($P = 0.000$, OR 22.6) have renal abscesses.

## Discussion

CT imaging is often the modality of choice for the evaluation of APN and renal abscesses.[15] It is superior to intravenous urogram or renal ultrasonography (US) in detecting renal parenchymal abnormalities like perinephric stranding, inflammatory masses, decreased or delayed cortical enhancement, kidney enlargement or gas formation, all of which may indicate more severe APN.[15-17] Diagnosing APN or renal abscesses clinically based on the classical triad of symptoms is neither specific nor sensitive. In this study, we intentionally chose to evaluate patients with radiologically confirmed APN or renal abscesses while excluding other possible differentials of pyonephrosis, ureteritis, renal colic with lower urinary tract infections or even non-urological pathologies that may present with similar symptoms. This allows us to target our analysis more specifically to patients with true APN or renal abscesses. Majd et al[18] reported a sensitivity of 86.8% and specificity of 87.5% of CT in diagnosing APN, compared with 74.3% and 56.7% for US. In fact, Stojadinović[19] recently reported that CT reduces the risk of missing renal abscesses by about 37 times compared with US. We used the definition reported by Piccolli et al[11] for classification of severity of APN. Simple APN presents as wedge-shaped areas of decreased enhancement radiating from the papilla to the cortex representing areas of poorly or non-functioning parenchyma due to vasospasm, tubular obstruction and/or interstitial oedema.[16] With increased severity of APN, parenchyma changes become diffused and multifocal.[20] Non-enhancing fluid collections can occur in severe cases representing formation of abscesses.[11,15,20] Hypotension and shock, the need for inotropes, presence of bacteremia and prolonged duration of fever, hospitalisation and intravenous antibiotics are useful clinical indicators of severe infection and sepsis.[21,22] Our study showed a statistically significant correlation of these clinical parameters of severe disease with our definition of radiological severity.

Our study suggests a female predilection for APN and risk appears to be higher in Malay females. However, male gender, older age, presence of diabetes mellitus and non-obstructing renal stones were significantly associated with more severe APN or renal abscesses. Clinical and biochemical parameters that were associated with more severe disease include a higher total white and CRP, left neutrophil shift, thrombocytosis or thrombocytopenia, low serum albumin, acute renal impairment and bacteremia. A review of available literature showed that many of the above risk factors have been reported as predictors for more severe APN/renal abscesses or treatment failures.[4,11,22-24]

It has been reported in several studies that patients with DM are at an increased predisposition to urinary tract infections (UTI) because of diabetic cystopathy and nephropathy, alteration of bacteria-host interaction and impaired phagocytosis and chemotaxis.[9,25,26] However, it is currently not clear if poor glycemic control is associated with increased risk or severity of UTI. Geerling et al[26] reported an increased adherence of *Escherichia coli* to diabetic uroepithelial cells and this increased adherence correlated positively with the higher levels of glycosylated haemoglobin (HbA1c). However, it is postulated that due to the impaired production of cytokines, this increased adherence does not lead to symptomatic UTI.[27] In our study, we find that diabetic patients were at increased risk of APN but a higher HbA1c level is not associated with an increased severity of disease. Indeed several published reports concurred with our findings.[28,29]

Nephrolithiasis had been reported to be a risk factor for APN although its role as a predictor for treatment failure was less certain. Efstathiou et al[4] reported that women with a history of nephrolithiasis were more likely to be hospitalised for APN but nephrolithiasis was not found to be an independent predictor of poor outcome. However, another study found that a history of nephrolithiasis was significantly associated with treatment failure for uncomplicated APN.[24] Our study showed that asymptomatic, non-obstructing renal stones were significantly associated with bacteriuria and more severe APN and renal abscesses. We may only postulate that this may be because renal stones are known to house bacteria and eradication of bacteriuria is more

difficult in the presence of nephrolithiasis.[30] We suggest further studies to investigate if treatment of asymptomatic and non-obstructing nephrolithiasis will lead to prevention or reduction of severity of APN and improved treatment outcomes.

To the best of our knowledge, there are only 4 studies to date that correlate the clinical and biochemical parameters with CT diagnosed APN[10,11,22,23] for identification of predictors for severity of disease and our series is thus far the largest. The study by Huang et al[22] was the only study out of the 4 that showed a good correlation of CT findings with clinical severity. This study, like ours, included only patients with CT diagnosed APN and the 28 patients were divided into 3 groups depending on radiological severity. Huang demonstrated that clinical and biochemical parameters like underlying diseases, duration of fever, leukocytosis, pyuria, septic shock and acute renal impairment were correlated with pattern of renal abnormalities detected on CT. The other 3 studies[10,11,23] did not show any correlations between clinical and biochemical parameters with radiological findings. Two studies[10,23] had small sample sizes of 28 and 35 patients and classified patients into 2 groups consisting of patients with or without CT features of APN. It is possible that in both studies, patients in the group with CT changes have CT features of mild APN, hence obscuring any differences as both groups will then consist of patients with mild APN. In the last study, although Piccoli[11] divided patients into 3 groups based on CT features of severity, the analysis was based on the eventual outcome of renal scarring but not based on radiological severity.

APN may be managed either as inpatient or outpatient. The majority of patients with APN are managed in the outpatient setting.[3,9] However, the more severe cases require hospitalisation, intravenous antibiotics therapy and very often radiological imaging like CT to rule out abscesses that may require surgical or radiological intervention. In our study of hospitalised patients, only 36.9% of patients had severe APN or renal abscesses and twelve patients required percutaneous drainage of their abscesses. The rest have mild APN that probably had not benefited from CT imaging. Interestingly, Piccolli[11] using the same radiological classification, reported similar rates (35.5%) of severe APN or renal abscesses. Theoretically, if there are clinical predictors to help determine which patients have more severe disease that may require CT imaging, unnecessary CT imaging can be avoided resulting in reduction in radiation and contrast media exposure and healthcare savings.[31]

We attempted to identify clinical predictors to aid clinician in determining which patients will likely to have more severe disease. We wanted factors that are readily available to clinicians on admission or early in the course of their disease and also significantly associated with severity. The 4 clinical predictors retrospectively identified from our study were the presence of DM, hypotension, acute renal impairment and a total white count of greater than 20K. Using these 4 clinical predictors, we were able to identify almost all patients with Groups 2 and 3 lesions (1 patient with renal micro abscesses was not identified). More importantly, if we perform CT imaging only on patients satisfying the above criteria, we should be able to avoid CT imaging in 42 patients with mild APN, a 51.2% reduction of CT requirements. The last patient that was missed using the above criteria can be picked up in accordance to the existing guideline that CT imaging should be done if fever persists for more than 72 hours despite appropriate antibiotic therapy.[32] Indeed our study showed that patients with Group 1 lesions have a mean duration of fever of 2.2 days compared with 3.4 and 5.4 days in Groups 2 and 3, respectively. However due to the limitations of this study, we could not recommend that CT imaging should only be done in patients with the above 4 risk factors. Unfortunately, we could also not recommend that only non-contrast CT is necessary when used in conjunction with the 4 factors. Non-contrast CT imaging will be able to rule out an obstructed collecting system requiring urgent decompression and drainage but it is not ideal for diagnosing renal abscesses or APN. CT imaging with or without the use of intravenous contrast should still be performed in accordance to the clinician's discretion and clinical suspicion. The major advantage of our 4 predictors is that they can be easily identified early in the course of illness and allow earlier CT imaging to be performed and hence appropriate interventions to be carried out at an earlier time. Early diagnosis and intervention of renal abscesses have been shown to improve patient outcomes.[33] However in view of the retrospective nature of our study, further prospective studies to externally validate our 4 predictors must be conducted.

## Conclusion

Our study showed a good correlation between clinical and radiological severity in adult patients with APN and renal abscesses. Patients with severe APN or renal abscesses were likely to be diabetics presenting with hypotension, acute renal impairment and a leucocyte count of greater than 20K.

REFERENCE


1. Stamm WE, Norrby SR. Urinary tract infections: disease panorama and challenges. J Infect Dis 2001;183(suppl 1):S1-S4.
2. Brown PD, Moran KI, Foxman B. Acute pyelonephritis among adults: Cost of illness and considerations for the economic evaluation of therapy. Pharmacoeconomics 2005;23:1123-42.
3. Czaja CA, Scholes D, Hooton TM, Stamm WE. Population-based epidemiologic analysis of acute pyelonephritis. Clin Infect Dis 2007;45:273-80.
4. Efstathiou SP, Pefanis AV, Tsioulos DI, Zacharos ID, Tsiakou AG, Mitromaras AG, et al. Acute pyelonephritis in adults: prediction of mortality and failure of treatment. Arch Intern Med 2003;163:1206-12.
5. Foxman B, Klemstine KL, Brown PD. Acute pyelonephritis in US hospitals in 1997: hospitalization and in-hospital mortality. Ann Epidemiol 2003;13:144-50.
6. British Medical Research Council Bacteriuria Committee. Recommended terminology of urinary tract infection. Br Med J 1979;2:717-9.
7. Hill GS. Uropathology. New York: Churchill-Livingstone, 1989.
8. Talner LB, Davidson AJ, Lebowitz RL, Dalla Palma L, Goldman SM. Acute pyelonephritis: can we agree on terminology? Radiology 1994;192:297-305.
9. Scholes D, Hooton TM, Roberts PL, Gupta K, Stapleton AE, Stamm WE. Risk factors associated with acute pyelonephritis in healthy women. Ann Intern Med 2005;142:20-7.
10. Rollino C, Boero R, Ferro M, Anglesio A, Vaudano GP, Cametti A, et al. Acute pyelonephritis: analysis of 52 cases. Ren Fail 2002;24:601-8.
11. Piccoli GB, Colla L, Burdese M, Marcuccio C, Mezza E, Maass J, et al. Development of kidney scars after acute uncomplicated pyelonephritis: relationship with clinical, laboratory and imaging data at diagnosis. World J Urol 2006;24:66-73.
12. Sund-Levander M, Forsberg C, Wahren LK. Normal oral, rectal, tympanic and axillary body temperature in adult men and women: a systematic literature review. Scand J Caring Sci 2002;16:122-8.
13. Marik PE, Lipman J. The definition of septic shock: implications for treatment. Crit Care Resusc 2007;9:101-3.
14. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P; Acute renal failure – definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. Critical Care 2004,8:R204-R212.
15. Craig WD, Wagner BJ, Travis MD. Pyelonephritis: radiologic-pathologic review. RadioGraphics 2008;28:255-77.
16. Kawashima A, Sandler CM, Ernst RD, Goldman SM, Raval B, Fishman EK. Renal inflammatory disease: the current role of CT. Crit Rev Diagn Imaging 1997;38:369-415.
17. Goldman SM, Fishman EK. Upper urinary tract infection: the current role of CT, ultrasound and MRI. Semin Ultrasound CT MR 1991;12:335-360.
18. Majd M, Nussbaum Blask AR, Markle BM, Shalaby-Rana E, Pohl HG, Park JS, et al. Acute pyelonephritis: comparison of diagnosis with 99 m-Tc DMSA, SPECT, spiral CT, MR imaging and power Doppler US in and experimental pig model. Radiology 2001;218:101-8.
19. Stojadinović M, Mićić S, Milovanović D. Ultrasonographic and computed tomography findings in renal suppurations: performance indicators and risks for diagnostic failure. Urol Int 2008;80:389-97.
20. Stunell H, Buckley O, Feene J, Geoghegan T, Browne RF, Torreggiani WC, et al. Imaging of acute pyelonephritis in the adult. Eur Radiol 2007;17:1820-28.
21. Hsu CY, Fang HC, Chou KJ, Chen CL, Lee PT, Chung HM. The clinical impact of bacteremia in complicated acute pyelonephritis. Am J Med Sci 2006;332:175-80.
22. Huang JJ, Sung JM, Chen KW, Ruaan MK, Shu GH, Chuang YC, et al. Acute bacterial nephritis: A clinicoradiologic correlation based on Computed Tomography. Am J Med 1992;93:289-98.
23. Ha SK, Seo JK, Kim SJ, Park SH, Park CH, Lee HY, et al. Acute pyelonephritis focusing on perfusion defects on contrast enhanced computerized tomography scans and its clinical outcome. Korean J Intern Med 1997;12:122-7.
24. Pertel PE, Haverstock D. Risk factors for a poor outcome after therapy for acute pyelonephritis. BJU Int 2006;98:141-7.
25. Repine JE, Clawson CC, Goetz FC. Bactericidal function of neutrophils from patients with acute bacteria infections and from diabetes. J Infect Dis 1980;142: 869-75.
26. Geerlings SE, Meiland R, van Lith EC, Brouwer EC, Gaastra W, Hoepelman AI. Adherence of type 1-fimbriated Escherichia coli to uroepithelial cells: more in diabetic women than in control subjects. Diabetes Care 2002;25:1405-9.
27. Geerlings SE, Brouwer EC, van Kessel KCPM, Gaastra W, Stolk RP, Hoepelman AIM. Cytokine secretion is impaired in women with diabetes mellitus. Eur J Clin Invest 2000;30:995-1001.
28. Geerlings SE, Stolk RP, Camps MJ, Netten PM, Collet TJ, Hoepelman AI; Diabetes Women Asymptomatic Bacteriuria Utrecht Study Group. Risk factors for symptomatic urinary tract infection in women with diabetes. Diabetes Care 2000;23:1737-41.
29. Ishay A, Lavi I, Luboshitzky R. Prevalence and risk factors for asymptomatic bacteruria in women with type 2 diabetes mellitus. Diabet Med 2006; 23:185-8.
30. McCabe WR, Jackson GG. Treatment of pyelonephritis: bacterial, drug and host factors in success or failure among 252 patients. N Engl J Med 1965;272:1037–44.
31. Noroozian M, Cohan RH, Caoili EM, Cowan NC, Ellis JH. Multislice CT urography: state of the art. Br J Radiol 2004;77:S74–S86.
32. Grabe M, Bishop MC, Bjerklund-Johansen TE, Botto H, Cek M, Lobel B, et al. Guidelines on urological infection 2009. Available at: http://www.uroweb.org/fileadmin/tx_eauguidelines/2009/Full/Urological_Infections.pdf Accessed 8 June 2011.
33. Yen DH, Hu SC, Tsai J, Kao WF, Chern CH, Wang LM, et al. Renal abscess: early diagnosis and treatment. Am J Emerg Med. 1999;17:192-7.