International Journal of Gerontology 18 (2024) 263–267
https://doi.org/10.6890/IJGE.202410_18(4).0012

International Journal of Gerontology

journal homepage: http://www.sgecm.org.tw/ijge/

Original Article

Differences between Clinical Presentation in Acute Pyelonephritis and Acute Renal Infarction, a Single-Center Study

Kuang-Wei Chou [a], Chun Chen [b], Kuo-Song Chang [c,d,e], Pang-Yen Chen [b,f,g], Yen-Chun Lai [h *], Yu-Jang Su [c,d,e,f *]

[a] *Division of Internal Medicine, Emergency Department, MacKay Memorial Hospital, Taipei, Taiwan,* [b] *Division of Disaster Medicine, Emergency Department, MacKay Memorial Hospital, Taipei, Taiwan,* [c] *Department of Nursing, MacKay Junior College of Medicine, Nursing, and Management, Taipei, Taiwan,* [d] *Toxicology Division, Emergency Department, MacKay Memorial Hospital, Taipei, Taiwan,* [e] *Department of Medicine, MacKay Medical College, New Taipei City, Taiwan,* [f] *Department of Nursing, Yuanpei University of Medical Technology, Hsinchu, Taiwan,* [g] *Institute of Biomedical Engineering, National Tsing Hua University, Hsinchu, Taiwan,* [h] *Department of Anesthesiology, Taipei Medical University Hospital, Taipei, Taiwan*

ARTICLE INFO

Accepted 16 April 2024

Keywords:
pyelonephritis,
infarction,
visceral pain

SUMMARY

Introduction: Acute pyelonephritis and acute renal infarction have several of the same aspects of symptoms and presentation easily be misidentified. This study aimed to find the difference in clinical presentation between renal infarction and acute pyelonephritis.
Methods: We retrospectively searched the database in MacKay Memorial Hospital from Jan 1, 2011, to Sep 30, 2021. Twenty cases of acute renal infarctions (ARI) and 20 of 1600 cases of acute pyelonephritis (APN) were obtained. We analyze the visiting time, the initial vital signs, the clinical symptoms, and the laboratory data. Both Student's t-test and chi-square test were used in the statistical analyses, and a p-value of < 0.05 indicated a statistically significant difference.
Results: In laboratory exam, hematuria and pyuria are more common in APN group (hematuria 80% vs. 40%, $p = 0.01$, pyuria 75% vs. 15%, $p < 0.001$). More patients presented with abdominal pain in ARI group (70% vs. 30%, $p = 0.001$). There are 20% of the APN group has bilateral flank pain and none of the ARI group has bilateral flank pain ($p = 0.035$).
Conclusion: ARI is male-predominant, and APN is female-predominant. APN patients have 2-fold hematuria and 5-fold pyuria than ARI. The ARI patients presented more abdominal pain and bilateral flank pain than APN patients. A relatively small number of APNs enrolled in this study. Maybe a multi-center study is a better way to have enough sample numbers to explain these findings well.

1. Introduction

Acute pyelonephritis (APN) is a common complication of urinary tract infection and is usually resolved by antibiotic treatment. Acute renal infarction (ARI) is an uncommonly seen disease resulting from disruption or occlusion of the kidney's blood flow. Without adequate treatment, renal infarction will lead to acute renal injury, end-stage renal disease (ESRD), and even death.[1,2] APN and acute renal colic are more commonly seen than renal infarction since they are all characterized by flank pain, hematuria, or pyuria. We can usually diagnose APN based on clinical presentation, abdominal or flank pain, blood test, and urine test. However, renal infarction is hard to diagnose simply based on these clinical presentations. It needs more image studies such as contrast computed tomography or angiography to confirm the diagnosis.

Clinically there are some symptoms that may mimic renal infarction due to similar presentation. In the presentation, ARI often presented with abrupt onset of abdominal or flank pain, vomiting, and fever. In laboratory data, the most common elevated is seral lactate dehydrogenase (LDH) accounting for 86.5% of cases and 56.5% of cases having elevated D-dimer values, respectively.[3]

This study aimed to find the differences in the clinical course, presentation of the visceral pain, the location of abdominal or flank, back pain, and the pain pattern between renal infarction and APN. If we could find some clinical hints of renal infarction, clinical physicians may arrange an advanced image study for suspicious renal infarction to make the diagnosis earlier. Thus, physicians do not misdiagnose the renal infarct as APNs. The earlier detection of renal infarction, the lower risk of the subsequent comorbidity will happen.

2. Materials and methods

2.1. Study design and data collection

We retrospectively searched the database in MacKay Memorial Hospital from Jan 1, 2011, to Sep 30, 2021; we reviewed the charts of all inpatients above 18 years old. We used the International Classification of Disease (ICD) code to search for renal infarction and APN diagnoses. ICD-9 Code 593.81: Vascular disorders of kidney and ICD-10 code N28.0: Ischemia and kidney infarction, as the diagnosis of renal infarction. ICD-9 590.10 and ICD-10 N10: acute pyelonephritis as the

* Corresponding author. Emergency Department, MacKay Memorial Hospital, No. 92, Sec. 2, Zhongshan N. Rd., Taipei 10449, Taiwan.
E-mail address: yjsu.md@msa.hinet.net (Y.-J. Su)
Yen-Chun Lai M.D. and Yu-Jang Su M.D. shared the corresponding authors.

diagnosis of APN. Due to the ICD-9 and ICD-10 code transition, we used the ICD-9 code before 2015 and ICD-10 since 2016 for database searching.

Patients who came from the emergency department with the diagnosis of renal infarction or APN were enrolled. Patients who were complicated with emphysematous pyelonephritis and renal abscess were excluded. Renal infarction was defined by single or multiple wedge-shaped perfusion defects in the kidney shown on the computed tomography (CT) image. After a rigorous review of images and chart records, 20 cases of renal infarctions were enrolled in this study. By ICD code search, 1600 cases of APN were obtained, and we randomly chose 20 cases of them compared to renal infarction. There are 86% of 1600 APN cases were female. The randomization is made by the collection of every 80th case of the ranking list. The diagnosis of urolithiasis was made by the discharge diagnosis list made by the nephrologist. These patients have gone through image studies during hospitalization.

2.2. Data analysis

We use statistical software (IBM SPSS Statistics for Windows, version 20.0; IBM Corp.) to analyze the emergency department (ED) visiting time, the initial vital signs, the clinical symptoms, including the pain location, nausea, vomiting, hematuria or pyuria, the past history and the lab data including white blood cell count (WBC), blood creatinine, C-reactive protein (CRP), LDH, length of stay and outcome. Hematuria and pyuria were diagnosed by microscopic evaluation (RBC $\geq$ 3 /HPF or WBC $\geq$ 5 /HPF).

Both unpaired Student's t-test and chi-square test were used in the statistical analyses, and a p-value of < 0.05 indicated a statistically significant difference. The Institutional Review Board (IRB) of MacKay Memorial Hospital (IRB No. 22MMHIS026e) approved the study.

3. Results

3.1. Demographic characteristics

Demographic data of acute renal infarcts and pyelonephritis are described in Table 1. In the renal infarction group, the patient's age ranged from 18 to 71 years old with a mean $\pm$ standard deviation (SD) = 48.3 $\pm$ (14.6). The gender of the renal infarction group is more male (male to female is 3:1). On the other hand, in the APN group, age ranged from 23 to 80 years old with a mean $\pm$ SD = (49.4 $\pm$ 15.9), and the gender is mainly in female (male to female is 1:4). There is no difference in ED presenting time. 3 (15%) in renal infarction and 1 (5%) in APN were presented due to trauma.

The record of fever was by the data of admittance of these patients entering the emergency department, and above 37 °C. The mean body temperature is higher in the APN group (37.7 $\pm$ 0.9 °C), whereas there is a relatively normal temperature in the renal infarction group (36.8 $\pm$ 0.8 °C). The mean heart rate is also higher in the APN group (103.6 $\pm$ 18.8 bpm). No significant difference was noted in blood pressure. 20% of renal infarction has atrial fibrillation and none in APN group by the first electrocardiogram (ECG) in the emergency department. On the contrary, more patients have urolithiasis in the APN group (40% vs. 5%). Both groups have a similar percentage of nausea, vomiting, and past history, including diabetes mellitus, congested heart failure, and hypertension.

Table 1
Demographic data and comparison in renal infarction and pyelonephritis.

	Renal infarction	Pyelonephritis	p value (2-tailed)
Gender			< 0.001*
Male:Female	15:5	4:16	
Age (years old)	48.3 $\pm$ 14.6	49.4 $\pm$ 15.9	0.841
Time of presentation			0.260
Day (7:00–15:00)	5 (25)	10 (50)	
Evening (15:00–23:00)	10 (50)	7 (35)	
Night (23:00–next 7:00)	5 (25)	3 (15)	
Trauma history	3 (15)	1 (5)	0.292
Body temperature (°C)	36.8 $\pm$ 0.8	37.7 $\pm$ 0.9	0.010*
Heart rate/minute	81.1 $\pm$ 12.8	103.6 $\pm$ 18.8	0.001*
Systolic blood pressure (mmHg)	139.8 $\pm$ 24.2	123.4 $\pm$ 23.9	0.066
Diastolic blood pressure (mmHg)	77.2 $\pm$ 16.1	69.2 $\pm$ 13.8	0.162
Atrial fibrillation	4 (20)	0 (0)	0.018*
Nausea or vomiting	6 (30)	7 (35)	0.736
Diabetes mellitus	5 (25)	2 (10)	0.212
Heart failure	2 (10)	0 (0)	0.147
Hypertension	8 (40)	4 (20)	0.168
Urolithiasis	1 (5)	8 (40)	0.008*
White blood cell count/micro-L	12875.0 $\pm$ 4044.1	12515 $\pm$ 6473.5	0.851
Creatinine (mg/dl)	1.1 $\pm$ 0.2	2.1 $\pm$ 2.0	0.040*
C-reactive protein (mg/dl)	6.2 $\pm$ 6.7	12.1 $\pm$ 10.8	0.040*
Hematuria	8 (40)	16 (80)	0.010*
Pyuria	3 (15)	15 (75)	< 0.001*
Medical treatment	20 (100)	17 (85)	0.072
Drainage	0 (0)	0 (0)	0.999
Surgical intervention	0 (0)	5 (25)	0.017*
Shock	1 (5)	1 (5)	0.814
Length of stay (days) median $\pm$ SD	6.0 $\pm$ 3.6	6.0 $\pm$ 7.3	0.530
Intensive care unit	4 (20)	0 (0)	0.035*
Mortality	0 (0)	0 (0)	

Data described as n (%), and mean $\pm$ SD (standard deviation). * Indicates significant statistical difference.

3.2 Laboratory data analysis

The D-dimer value of ARI group is available in 12 cases with a mean ± standard deviation (SD) of 1990.0 ± 1813.5 ng/ml and the LDH is available in 6 cases with a mean ± SD as 782.7 ± 720.5 U/L. There is only one D-dimer data as 3360 ng/ml in APN group and no data record in LDH values of APN group.

In laboratory exam, hematuria and pyuria are higher in APN group (hematuria 80% vs. 40%, p = 0.01, pyuria 75% vs. 15%, p < 0.001) shown as Figure 1. There are no significant differences in WBC, creatinine, and CRP. Besides, concurrent urolithiasis was noted in 40% of the APN group and only 5% in the ARI group (p = 0.008).

Most cases of the two groups received only medical treatment without surgical intervention. Five patients (25%) of the APN group received ureterorenoscopy due to urolithiasis. On the other hand, none of the ARI group needed surgical intervention (p = 0.017). Twenty percent of the ARI group were admitted to the intensive care unit (ICU), and none of the APN group needed ICU admission.

3.3. Comparisons in visceral pain between renal infarction and pyelonephritis

To differentiate the visceral pain pattern, we compared the two groups of abdominal pain, flank pain, and back pain, whether it is unilateral (left side or right side) or bilateral.

Table 2 showed a comparison of visceral pain presentation in renal infarction and pyelonephritis. More patients presented with abdominal pain in RI group (70% vs. 30%, p = 0.001), especially left abdominal pain (50% to 20%, p = 0.047). No significant right abdominal pain between the two groups (p = 0.376).

There are 20% of the APN group has bilateral flank pain and none of the ARI group has bilateral flank pain (p = 0.035). Only a few cases were presented with back pain (2 and 1 cases out of 20 in ARI and APN group), and all of them are right back pain.

4. Discussion

4.1. Acute renal infarction is male predominant, and acute pyelonephritis is female predominant

Our result showed that the male-to-female ratio was about 1:4 in the APN group while 3:1 in the acute renal infarction (ARI) group. As we all know, urinary tract infections (UTIs) are more common in females due to shorter urethras and distance to the anus. As a complication of an ascending urinary tract infection, the incidence of APN is expected to be higher in females. Christopher CA's study found acute pyelonephritis was 15 to 17 cases per 10,000 females and 3 to 4 cases per 10,000 males in the United States annually.[4] In studies of renal infarction, some articles showed males are dominant, and others led to the opposite results. Among these studies, there was no statistically significant difference in gender.[5–8] Therefore, there is currently no evidence that men or women are more prone to renal infarction. Hence, it was controversial to rely on sex for differential diagnosis between APN and renal infarction. However, our study showed significant differences in gender (p value < 0.001).

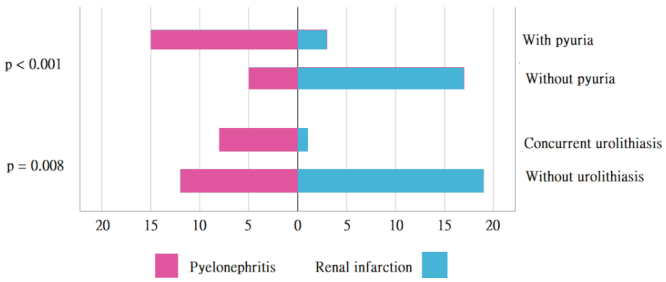

Figure 1. In laboratory exam, hematuria and pyuria are higher in APN group. Besides, concurrent urolithiasis was noted in 40% of the APN group and only 5% in the ARI group (p = 0.008).

4.2. Patients with acute pyelonephritis are 0.9 °C higher in body temperature than acute renal infarction, and 23 beats per minute faster in heart beats than acute renal infarction

Although clinical presentations vary widely, APN classically presents as a triad of flank pain, fever, and nausea or vomiting. Most patients of APN have a fever, which is a sign of systemic inflammation, while fever is present occasionally in acute renal infarction. Acute renal infarction presents with abdominal or flank pain, nausea or vomiting, and fever is present in about only one-third of patients.[9] That's why the average body temperature of patients with acute pyelonephritis is 0.9 °C higher than that of patients with acute renal infarction (37.7 vs. 36.8, p = 0.010). We also found that the heart rate in the APN group was an average of 22.5 beats per minute faster than in the acute renal infarction group (103.6 vs. 81.8, p = 0.001). It may be related to the higher body temperature in patients with APN; as the body temperature rises by one degree Celsius, the heart rate will increase by about seven beats per minute.[10] The lower blood pressure may also cause a compensatory increase in heartbeat. Besides, studies have shown that females had higher heart rates than males.[10,11] The APN group has the majority of women, while the acute renal infarction group mainly impacted males, which may also be one of the reasons for the higher heartbeats in acute pyelonephritis than in acute renal infarction.

4.3. Concurrent urolithiasis in pyelonephritis is 8-fold that of acute renal infarction with urolithiasis

In many studies, atrial fibrillation was the crucial risk factor for renal infarction.[5–7] The cause of renal infarction includes thromboembolism, hypercoagulable state, renal artery dissection, and renal trauma. In our study, 20% of patients diagnosed with renal infarction had atrial fibrillation, while none of the patients diagnosed with APN had atrial fibrillation (p = 0.018). Our result is compatible with previous studies.[12] APN is usually caused by obstructive uropathy, structural abnormalities, prostatic hypertrophy, or urolithiasis.[13] Our result also showed that pyelonephritis patients have a much higher

Table 2
Comparison of visceral pain presentation in renal infarction and pyelonephritis.

	Renal infarction N (%)	Pyelonephritis N (%)	p value (2-tailed)
Right abdominal pain	4 (20)	2 (10)	0.376
Left abdominal pain	10 (50)	4 (20)	0.047*
Abdominal pain	14 (70)	6 (30)	0.001*
Right flank pain	8 (40)	13 (65)	0.113
Left flank pain	5 (20)	9 (45)	0.185
Bilateral flank pain	0 (0)	4 (20)	0.035*
Flank pain	13 (65)	18 (90)	0.058
Right back pain	2 (10)	1 (5)	0.548
Left back pain	0 (0)	0 (0)	0.999
Back pain	2 (10)	1 (5)	0.548

* Indicates significant statistical difference.

percentage of urolithiasis than renal infarction patients (40% vs. 5%, p = 0.008). It is proper to arrange image studies for patients with acute pyelonephritis with urolithiasis, especially in suspicion of sepsis or septic shock.[14]

4.4. Pyelonephritis had 2-fold hematuria and 5-fold pyuria than acute renal infarction

APN is mainly caused by bacterial infection of the kidney parenchyma. It usually originates from the upstream infection via the lower urinary tract. It clinically results in flank pain, nausea, elevated urine white cells (pyuria), and red cells (hematuria).[1] Although there are few cases of APNs having no pyuria, over three-fourths of APNs, have pyuria.[2] Unless there is coexisting bacterial infection, pyuria should be less in ARI than pyelonephritis. On the other hand, renal infarction results from an acute disruption of renal blood flow.[15] Hematuria can be caused by various conditions, such as trauma, glomerular disease, infection, and even post-renal disorders. It is most frequently the result of nephrolithiasis, pyelonephritis, benign prostatic hypertrophy, or malignancy.[15] Both ARI and APN may result in hematuria. In the APN group, 40% of patients are comorbid with urolithiasis, which causes hematuria. Thus, in our study, pyelonephritis had a 2-fold hematuria percentage (p = 0.010) than ARI.

A review analysis reported that 32–90% of renal infarction patients have abdominal pain.[5] In our study, 70% of renal infarction cases have abdominal pain, 50% have left-side abdominal pain, and only 30% of APNs have abdominal pain. on the contrary, APN is mainly manifested by flank pain.

In Fernando's study, 62 patients of renal infarction from a single center have no left or right statistical difference.[8] Korzets's study also showed no difference between left and right side predominance in renal infarcts.[16] Another large review article showed the left side predominant but no statistically significant difference.[6] In anatomy, the abdominal aorta is slightly left to the midline, thus the length of the right renal artery is mild longer than the left renal artery. Theoretically, we may conclude that the resistance of the right renal artery is more than the left side, which means the right kidney is more likely to happen ischemia or infarction.[17] In our study, the presentation of right flank pain in ARI patients is 2-fold that of the left flank pain.

Prior studies suggest that serum LDH may elevate 6.9-fold higher than the normal upper limit and it helps physicians to raise the concern of renal infarction.[18,19] In the retrospective data of our study, we found that LDH data is available in 25% of RI cases and none of the APN patients have LDH data. The common practice is only suspected RI cases; we check LDH for this patient as a method of differential diagnosis.

4.5. Management, outcome, and prognosis

Urological procedures were often performed for the treatment of APNs with obstructive uropathy, which include urinary lithotripsy, extracorporeal shock wave lithotripsy (ESWL), ureteroscopic stone removal, and ureterolithotomy.[20] Other procedures are percutaneous nephrostomy, clean intermittent catheterization, ureteroneocystostomy, nephrectomy, and percutaneous pus drainage. It explains the result of our study shows that 25% patients of in the APN group need surgical intervention, while none of the ARI patients received surgery (25% vs. 0%, p < 0.017).

Acute kidney injury is often associated with ARI. A delayed diagnosis of ARI may cause impaired renal function or even death.[20,21] The incidence of acute kidney injury ranges from 0% to 60% in different studies of ARI.[12,16,20,22] Most patients spontaneously recovered from acute kidney injury, although about 7% developed persistent renal impairment.[23] The benefits of revascularization are evaluated by the time since the onset of ischemia, the size of infarcted kidney parenchyma, the kidney function, and the degree of occlusion evaluated by computed tomography angiography (CTA) in most patients.

The treatments of ARI include open surgery, anticoagulation, and percutaneous endovascular therapy (thrombolysis, thrombectomy with or without angioplasty, or stent placement). Aortic dissection patients extending into the renal artery or a traumatic renal artery occlusion may receive surgical intervention.[24–27] The occlusion of the renal artery results in a release of renin, which develop an elevation of blood pressure during the first week of infarction. It may subside over time unless the patient has underlying hypertension. Thus, angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) are the preferred treatment choice in the absence of acute kidney injury.

Patients of ARI usually have underlying morbidity and mortality conditions, such as atrial fibrillation and diffuse atherosclerosis. The renal emboli patients often suffer from cerebral infarction and ischemia bowel disease due to embolization.[12,20,21] The 30-day mortality was 11.4% in a previous study of forty-four cases of renal infarction in patients with atrial fibrillation.[20] Thrombolytic agents are the most commonly used treatment for ARI. Therefore, patients were often admitted to ICU to observe the bleeding side effects of thrombolytic treatment. It explains our result that more ARI patients were admitted to ICU compared with pyelonephritis patients (20% vs. 0%, p < 0.035) in our study.

The duration of time to diagnose may play a vital role in the outcome of ARI patients. In a study of 22 patients with segmental renal infarction, a trend of better recovery of renal function was noted in the early diagnosis group (mean time to diagnosis 76.4 hours) compared with the late diagnosis group (mean time to diagnosis 126 hours).

Antibiotics should be administered empirically in the early phase of the treatment of APN when the culture result of causative microorganisms is not yet revealed. The most common pathogen was *Escherichia coli* (67.0%), followed by *hemolytic Streptococci*.[28]

5. Conclusion

Our study showed significant differences by gender in both APN and ARI patients. Acute renal infarction is male predominant, and acute pyelonephritis is female predominant. Acute pyelonephritis is 0.9 °C higher than acute renal infarction, which may illustrate the infection condition. The heart rate of acute pyelonephritis is also higher than ARI. There were 20% of patients diagnosed with renal infarction who had atrial fibrillation. Acute pyelonephritis patients have a much higher percentage of urolithiasis than renal infarction patients and had 2-fold hematuria and 5-fold pyuria than acute renal infarction. Urological procedures were often performed for the treatment of APNs. In the APN group, 25% of patients needed surgical intervention, while none of the ARI patients received surgery. The ARI patients were often admitted to ICU to observe bleeding side effects due to thrombolytic agents. In clinical practice, the physicians must to review the risk factors, such as atrial fibrillation, urolithiasis and accurate medical history in an emergency room at the first approach to the patient. To enlarge the number of cases enrolled is better to explain these findings if multi-center studies in the future.

6. Limitation

Confinement in the number of acute renal infarcts, we gather

the same number of pyelonephritis causing a relatively small number of APNs enrolled in this study. This leads to bias to have the suitable presentation of APNs by way of these 20 patients. This is the major limitation. Second, maybe a multi-center study is a better way to have enough sample numbers to achieve a good explanation of these findings.

Funding source

There's no funding in this article.

Conflict of interest statement

No.

References

1. Belyayeva M, Leslie SW, Jeong JM. Acute Pyelonephritis. In: *StatPearls*. StatPearls Publishing; February 28, 2024.
2. Song HK, Shin DH, Na JU, Han SK, Choi PC, Lee JH. Clinical investigation on acute pyelonephritis without pyuria: a retrospective observational study. *J Yeungnam Med Sci.* 2022;39(1):39–45. doi:10.12701/yujm.2021.01207
3. Zhang ZG, Liu XM. Clinical characteristics of patients with acute renal infarction: an analysis of 52 patients in a single center. Article in Chinese. *Beijing Da Xue Xue Bao Yi Xue Ban.* 2019;51(5):863–869. doi:10.19723/j.issn.1671-167X.2019.05.012
4. Czaja CA, Scholes D, Hooton TM, Stamm WE. Population-based epidemiologic analysis of acute pyelonephritis. *Clin Infect Dis.* 2007;45(3):273–280. doi:10.1086/519268
5. Antopolsky M, Simanovsky N, Stalnikowicz R, Salameh S, Hiller N. Renal infarction in the ED: 10-year experience and review of the literature. *Am J Emerg Med.* 2012;30(7):1055–1060. doi:10.1016/j.ajem.2011.06.041
6. Eren N, Gungor O, Kocyigit I, et al. Acute renal infarction in Turkey: a review of 121 cases. *Int Urol Nephrol.* 2018;50(11):2067–2072. doi:10.1007/s11255-018-1979-6.
7. Oh YK, Yang CW, Kim YL, et al. Clinical characteristics and outcomes of renal infarction. *Am J Kidney Dis.* 2016;67(2):243–250. doi:10.1053/j.ajkd.2015.09.019
8. Caravaca-Fontán F, Pampa Saico S, Elías Triviño S, et al. Acute renal infarction: Clinical characteristics and prognostic factors. *Nefrologia*. 2016;36(2):141–148. doi:10.1016/j.nefro.2015.09.015
9. Saeed K. Renal infarction. *Int J Nephrol Renovasc Dis.* 2012;5:119–123. doi:10.2147/IJNRD.S33768
10. Kirschen GW, Singer DD, Thode HC Jr, Singer AJ. Relationship between body temperature and heart rate in adults and children: A local and national study. *Am J Emerg Med.* 2020;38(5):929–933. doi:10.1016/j.ajem.2019.158355
11. Quer G, Gouda P, Galarnyk M, Topol EJ, Steinhubl SR. Inter- and intra-individual variability in daily resting heart rate and its associations with age, sex, sleep, BMI, and time of year: Retrospective, longitudinal cohort study of 92,457 adults. *PLoS One.* 2020;15(2):e0227709. doi:10.1371/journal.pone.0227709
12. Domanovits H, Paulis M, Nikfardjam M, et al. Acute renal infarction. Clinical characteristics of 17 patients. *Medicine (Baltimore).* 1999;78(6):386–394. doi:10.1097/00005792-199911000-00004
13. Bethel J. Acute pyelonephritis: risk factors, diagnosis and treatment. *Nurs Stand.* 2012;27(5):51–56; quiz 58. doi:10.7748/ns2012.10.27.5.51.c9334
14. Johnson JR, Russo TA. Acute pyelonephritis in adults [published correction appears in N Engl J Med. 2018 Mar 15;378(11):1069. doi: 10.1056/NEJMx180009]. *N Engl J Med.* 2018;378(1):48–59. doi:10.1056/nejmcp1702758
15. Willis GC, Tewelde SZ. The approach to the patient with hematuria. *Emerg Med Clin North Am.* 2019;37(4):755–769. doi:10.1016/j.emc.2019.07.011
16. Korzets Z, Plotkin E, Bernheim J, Zissin R. The clinical spectrum of acute renal infarction. *Isr Med Assoc J.* 2002;4(10):781–784.
17. Bemanian S, Motallebi M, Nosrati SM. Cocaine-induced renal infarction: report of a case and review of the literature. *BMC Nephrol.* 2005;6:10. doi:10.1186/1471-2369-6-10
18. London IL, Hoffsten P, Perkoff GT, Pennington TG. Renal infarction: Elevation of serum and urinary lactic dehydrogenase (LDH). *Arch Intern Med.* 1968;121(1):87–90. doi:10.1001/archinte.121.1.87
19. Chu PL, Wei YF, Huang JW, Chen SI, Chu TS, Wu KD. Clinical characteristics of patients with segmental renal infarction. *Nephrology (Carlton).* 2006;11(4):336–340. doi:10.1111/j.1440-1797.2006.00586.x
20. Hazanov N, Somin M, Attali M, et al. Acute renal embolism. Forty-four cases of renal infarction in patients with atrial fibrillation. *Medicine (Baltimore)*. 2004;83(5):292–299. doi:10.1097/01.md.0000141097.08000.99
21. Lessman RK, Johnson SF, Coburn JW, Kaufman JJ. Renal artery embolism: clinical features and long-term follow-up of 17 cases. *Ann Intern Med*. 1978;89(4):477–482. doi:10.7326/0003-4819-89-4-477
22. Huang CC, Lo HC, Huang HH, et al. ED presentations of acute renal infarction. *Am J Emerg Med*. 2007;25(2):164–169. doi:10.1016/j.ajem.2006.06.010
23. Bae EJ, Hwang K, Jang HN, et al. A retrospective study of short- and long-term effects on renal function after acute renal infarction. *Ren Fail*. 2014;36(9):1385–1389. doi:10.3109/0886022X.2014.947514
24. Ouriel K, Andrus CH, Ricotta JJ, DeWeese JA, Green RM. Acute renal artery occlusion: when is revascularization justified? *J Vasc Surg.* 1987;5(2):348–355. doi:10.1067/mva.1987.avs0050348
25. Haas CA, Dinchman KH, Nasrallah PF, Spirnak JP. Traumatic renal artery occlusion: a 15-year review. *J Trauma*. 1998;45(3):557–561. doi:10.1097/00005373-199809000-00024
26. Haas CA, Spirnak JP. Traumatic renal artery occlusion: a review of the literature. *Tech Urol.* 1998;4(1):1–11.
27. Bouttier S, Valverde JP, Lacombe M, Nussaume O, Andreassian B. Renal artery emboli: the role of surgical treatment. *Ann Vasc Surg.* 1988;2(2):161–168. doi:10.1016/S0890-5096(06)60800-5
28. Kim SW, Lee JY, Park WJ, Cho YH, Yoon MS. Antibiotic sensitivity to the causative organism of acute simple urinary tract infection for recent 3 years. *Korean J Infect Dis.* 2000:32(5):380–387.