Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Changes in Renal Function Following Emergency Percutaneous Nephrostomy and Factors Influencing Outcomes: A Retrospective Comparative Study

Yuanxiao Li
The Affiliated Hospital of Qingdao University
Yunduo Fan
The Affiliated Hospital of Qingdao University
Wei Jiang
jiangwei8866@qdu.edu.cn

The Affiliated Hospital of Qingdao University

Research Article

Keywords: Percutaneous Nephrostomy, Renal Function, Hydronephrosis, Cystatin C

Posted Date: November 24th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11412676/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Changes in Renal Function Following Emergency Percutaneous Nephrostomy and Factors Influencing Outcomes: A Retrospective Comparative Study

Yuanxiao Li[#1], Yunduo Fan[#2], Wei Jiang[1*]

[1]Department of Nephrology, The Affiliated Hospital of Qingdao University, Qingdao, China

[2]Department of Urology, The Affiliated Hospital of Qingdao University, Qingdao, China

[*]Corresponding author: Wei Jiang, MD, Department of Nephrology, The Affiliated Hospital of Qingdao University, Qingdao, China (jiangwei866@qdu.edu.cn)

#Contributed equally.

Abstract

Background: Although emergency percutaneous nephrostomy (PCN) is a standard procedure for relieving hydronephrosis, postoperative renal functional changes are poorly characterized. This study was designed to assess renal function before and after PCN and to explore the influencing factors.

Methods: A retrospective analysis was conducted on clinical data from 130 patients who underwent emergency PCN for hydronephrosis at the Affiliated Hospital of Qingdao University over the past 5 years. Data collected included preoperative and short-term postoperative parameters.

Results: Among the 130 patients, 74 cases were classified into the significant short-term renal function recovery group, while the remaining 56 cases were assigned to the non-significant recovery group. Statistically significant differences between the two groups were observed in preoperative age, cystatin C levels, and the prevalence of hypertension, while logistic

regression further confirmed age and cystatin C as significant risk factors.

Conclusion: For emergency PCN, the degree of preoperative renal pelvis anterior-posterior diameter (APD) showed no correlation with short-term postoperative changes in renal function, whereas advanced age and elevated cystatin C levels may be considered risk factors for short-term functional changes.

Keyword: Percutaneous Nephrostomy; Renal Function; Hydronephrosis; Cystatin C

Introduction

Acute hydronephrosis is a common condition in urology, predominantly caused by urinary tract obstruction. Emergency decompression to relieve hydronephrosis prior to assessment and definitive surgical intervention can prevent further loss of nephrons and deterioration of renal function (1). Common decompression methods include double-J stent (DJS) placement or percutaneous nephrostomy (PCN). There remains considerable debate regarding which approach is more suitable for individual patients (2). Overall, DJS placement not only imposes higher demands on the patient, including the need to tolerate anesthesia, but also requires greater expertise from the operator and more specialized equipment. Moreover, the procedure may fail due to ureteral occlusion or stenosis during stent insertion. Postoperative confirmation of stent positioning also entails additional radiation exposure. In contrast, PCN presents lower technical and patient-specific requirements, along with a higher success rate of placement. Owing to its simplicity, safety, and efficacy, PCN is often preferred as the initial clinical intervention.

PCN is commonly employed to relieve obstruction and infection of the pyelocaliceal system (3). As a minimally invasive and well-tolerated procedure, it is typically performed

under various imaging guidance modalities—such as computed tomography (CT) and ultrasonography—to establish a temporary urinary diversion by connecting the renal pelvis or calyx to an external drainage catheter via a percutaneous tract. It also serves to create access for subsequent potential interventions or adjuvant therapies (4). Nowadays, PCN is widely used in numerous clinical scenarios, providing essential support for the diagnosis and treatment of related diseases (5). Nevertheless, the vast majority of PCN procedures in clinical practice are performed emergently to decompress an obstructed kidney and preserve renal function (6).

Current research on PCN predominantly focuses on procedural techniques and associated complications (7-11), while relatively limited attention has been paid to postoperative renal function changes and their potential influencing factors. In routine clinical practice, we have observed that a considerable proportion of patients exhibit minimal improvement or even continued deterioration in renal function during the short-term postoperative period. This phenomenon underscores the necessity of investigating the potential for renal functional recovery following PCN.

Materials and Method

Clinical Materials

We retrospectively collected clinical data from 130 patients who underwent emergency PCN for hydronephrosis at the Affiliated Hospital of Qingdao University over the past 5 years. Data collected included preoperative and short-term postoperative parameters such as age, gender, renal pelvis anterior-posterior diameter (APD), presence of diabetes mellitus (DM) and hypertension, serum creatinine (SCr), estimated glomerular filtration rate (eGFR), total

cholesterol (TC), free fatty acids (FFA), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), albumin, uric acid (UA), cystatin C, blood urea nitrogen (BUN), urea/creatinine ratio, sodium (Na^+), and potassium (K^+) levels. To mitigate potential confounding effects from compensatory adaptations in the contralateral kidney following PCN, which may obscure accurate assessment of renal functional changes in the affected kidney (12-13), short-term postoperative SCr and eGFR levels were specifically selected as primary outcome measures. The inclusion criteria were as follows: 1. patients undergoing ultrasound-guided PCN for hydronephrosis or urinary obstruction; 2. availability of complete clinical data before and after the procedure; 3. age ≥18 years at the time of intervention; 4. absence of postoperative complications (e.g., hemorrhage, shock, drainage tube blockage, or dislodgement) with maintained urinary patency.

In terms of grouping, there is currently no relevant research on post-PCN grouping. Therefore, we referred to studies on acute kidney injury (AKI) recovery and used eGFR as the criterion for classifying changes in renal function (14-15). We first selected short-term eGFR after PCN. Subsequently, we calculated the short-term renal function change in patients after percutaneous nephrostomy using the formula: (postoperative eGFR - preoperative eGFR) / preoperative eGFR × 100%. Patients with a result of this ratio > 50% were classified into the significant short-term renal function recovery group (Group 1). Additionally, considering the potential for eGFR improvement, patients with a short-term renal function recovery ≤ 50% were also assigned to Group 1 if they met either of the following criteria: preoperative eGFR < 60 mL/min/1.73m² and postoperative eGFR > 60 mL/min/1.73m², or preoperative eGFR < 90 mL/min/1.73m² and postoperative eGFR > 90 mL/min/1.73m². The remaining patients

were classified into the non-significant short-term renal function recovery group (Group 2).

In our study, all eGFR values were calculated from the SCr levels of the corresponding period using the Chronic Kidney Disease Epidemiology Project Initiative (CKD-EPI) equation (16).

$eGFR = 141 \times min(\kappa/Scr, 1)^{\alpha} \times max(\kappa/Scr, 1)^{-1.209} \times 0.993^{Age} \times (Gender\ Coefficient) \times (Race\ Coefficient)$

Parameters: 1.κ (kappa): Female: 0.7 & Male: 0.9; 2.α (alpha): Female: -0.329 & Male: -0.411; 3. Gender Coefficient: Female: 1.018 & Male: 1.0; Race Coefficient: Black: 1.159 & Non-Black: 1.0.

Statistical Methods

Statistical analysis was performed using SPSS software (version 31.0). Continuous variables with normal distribution were expressed as mean ± standard deviation ($\bar{X} \pm SD$), while non-normally distributed continuous variables were presented as median (interquartile range) [M (Q1, Q3)]. Categorical variables were reported as number (percentage) or frequency count. Difference Analysis and logistic regression analysis were employed for statistical modeling, with a P-value <0.05 considered statistically significant.

Results

Based on short-term renal function changes, 74 patients (56.92%) were classified into the significant short-term renal function recovery group (Group 1), while the remaining 56 patients (43.08%) were assigned to the non-significant short-term renal function recovery group (Group 2). No statistically significant differences were observed between the two groups in terms of preoperative gender, renal pelvis APD, presence of DM, SCr, eGFR, TC,

FFA, TG, LDL-C, HDL-C, albumin, UA, BUN, urea/creatinine ratio, Na^+ or K^+ levels (Table 1). Advanced age, comorbid hypertension, and elevated cystatin C levels were identified as potential risk factors for short-term renal function changes after PCN in both difference analysis and univariate regression analysis. The specific findings were as follows: 63.23 ± 14.15 vs. 69.05 ± 14.44 ($p = 0.023$); 17 (22.97%) vs. 24 (42.86%) ($p = 0.016$); and 1.41 [1.12, 1.97] vs. 2.04 [1.51, 3.25] ($p < 0.001$). Subsequent binary logistic regression analysis further validated the findings. Although hypertension was not confirmed as a significant risk factor (OR = 1.628, p = 0.258), the other two indicators remained consistent with the univariate screening results: age (OR = 1.039, p = 0.011) and cystatin C (OR = 2.033, $p < 0.001$) (Table 2). Partial results are presented in a forest plot (Figure 1).

We also observed that in Group 1, 3 patients (4.05%) exhibited an initial increase followed by a decrease in SCr levels, along with an initial decline followed by an improvement in eGFR after PCN, which differed from the consistent downward trend in SCr and upward trend in eGFR seen in other patients. Furthermore, owing to variations across patients in the frequency and timing of postoperative creatinine measurements, the rebound trend noted in Group 1 may potentially be more pronounced than reflected in the current observations. This suggests that relying solely on immediate postoperative renal function to predict short-term changes after PCN may be unreliable. The changes in SCr and eGFR of two representative patients are illustrated in Fig 2 and Fig 3.

Table 1. Demographic and Clinical Characteristics by Two Groups

Variable	Group 1 (n=74)	Group 2 (n=56)	P value

Gender, n (%)			0.925
Male	43(58.11)	33(58.93)	
Female	31(41.89)	23(41.07)	
Age (year)	**63.23±14.15**	**69.05±14.44**	**0.023**
DM, n (%)	9(12.16)	10(17.86)	0.363
Hypertension, n (%)	**17(22.97)**	**24(42.86)**	**0.016**
TC (mmol/L)	4.08[3.45, 5.11]	3.94[3.25, 5.14]	0.561
FFA (mmol/L)	0.44[0.32, 0.63]	0.44[0.34, 0.60]	0.714
TG (mmol/L)	1.20[0.88, 1.76]	1.10[0.84, 1.70]	0.547
LDL-C (mmol/L)	2.26[1.86, 3.15]	2.23[1.72, 3.00]	0.638
HDL-C (mmol/L)	1.14±0.35	1.10±0.36	0.522
Albumin (g/L)	38.33±7.83	38.17±9.07	0.916
UA (μmol/L)	384.00[275.75, 497.00]	389.50[299.50, 566.50]	0.214
BUN (mg/dL)	14.75[6.86, 27.72]	19.79[10.15, 28.24]	0.089
SCr (μmol/L)	240.42[116.28, 630.33]	349.50[158.88, 606.38]	0.364
eGFR (mL/min/1.73m²)	21.60[6.88, 55.30]	15.00[7.65, 39.60]	0.220
Urea/Creatinine Ratio	15.02[10.15, 18.81]	15.63[12.35, 18.59]	0.401
Cystatin C (mg/L)	**1.41[1.12, 1.97]**	**2.04[1.51, 3.25]**	**＜0.001**
K^+ (mmol/L)	4.32[3.89, 5.05]	4.49[4.10, 5.09]	0.420
Na^+ (mmol/L)	138.90[134.80, 140.38]	137.95[134.85, 141.55]	0.976
Renal Pelvis APD (cm)	2.70[1.70, 4.20]	3.00[1.80, 4.95]	0.396
Laterality			0.841

Unilateral, n(%)	53(71.62)	41(73.21)
Bilateral, n(%)	21(28.38)	15(26.79)

Abbreviations: DM, Diabetes Mellitus; TC, Total Cholesterol; FFA, Free Fatty Acids; TG, Triglycerides; LDL-C, Low-Density Lipoprotein Cholesterol; HDL-C, High-Density Lipoprotein Cholesterol; UA, Uric Acid; BUN, Blood Urea Nitrogen; SCr, Serum Creatinine; eGFR, estimated Glomerular Filtration Rate; K+, Potassium; Na+, Sodium; APD, Anterior-Posterior Diameter.

*Statistically significant values are identified in boldface.

*For bilateral cases, the APD is the sum of the two sides.

*Conversion factors for units: serum creatinine in mg/dL to μmol/L, ×88.4; cystatin C in mg/dL to μmol/L, ×0.075; blood urea nitrogen in mg/dL to mmol/L, ×0.357; uric acid in mg/dL to μmol/L, ×59.5.

Table 2 Logistic Regression Analysis for Risk Factors

Variable	**Univariate analysis**		**Multivariate analysis**	
	OR (95% CI)	**P value**	**OR (95% CI)**	**P value**
Male (versus female)	1.034(0.511-2.093)	0.925		
Age	**1.029(1.004-1.056)**	**0.025**	**1.039(1.009-1.069)**	**0.011**
DM	1.570(0.591-4.169)	0.365		
Hypertension	**2.515(1.179-5.363)**	**0.017**	1.628(0.700-3.783)	0.258
TC	0.916(0.676-1.241)	0.570		
FFA	0.487(0.121-1.964)	0.312		
TG	0.903(0.555-1.467)	0.680		
LDL-C	0.894(0.607-1.315)	0.569		
HDL-C	0.723(0.270-1.937)	0.519		
Albumin	0.998(0.957-1.040)	0.915		
UA	1.002(1.000-1.004)	0.107		
BUN	1.013(0.987-1.040)	0.321		
SCr	1.000(0.999-1.001)	0.893		

eGFR	0.989(0.976-1.004)	0.144		
Urea/Crea Ratio	1.022(0.972-1.074)	0.394		
Cystatin C	**1.971(1.339-2.903)**	**<0.001**	**2.033(1.358-3.044)**	**<0.001**
K+	1.082(0.728-1.608)	0.696		
Na+	1.014(0.949-1.084)	0.674		
Renal Pelvis APD	1.096(0.955-1.256)	0.192		
Bilateral (versus unilateral)	0.923(0.424-2.010)	0.841		

* Statistically significant values are identified in boldface.

* With the significant short-term renal function recovery group (Group 1) as the reference standard.

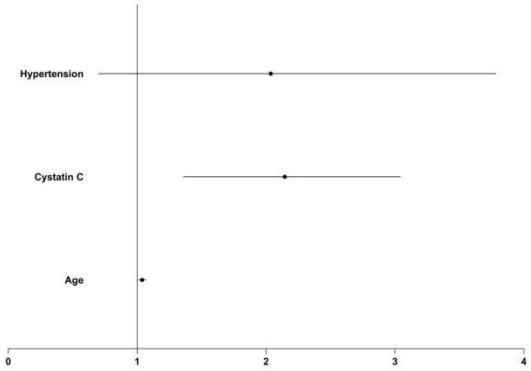

Fig 1. Forest plot of the binary logistic regression results: hypertension *(OR 1.628, 95% CI 0.700 to 3.783; p = 0.258); cystatin C (OR 2.033, 95% CI 1.358 to 3.044; p < 0.001) and age (OR 1.039, 95% CI 1.009 to 1.069; p = 0.011)*

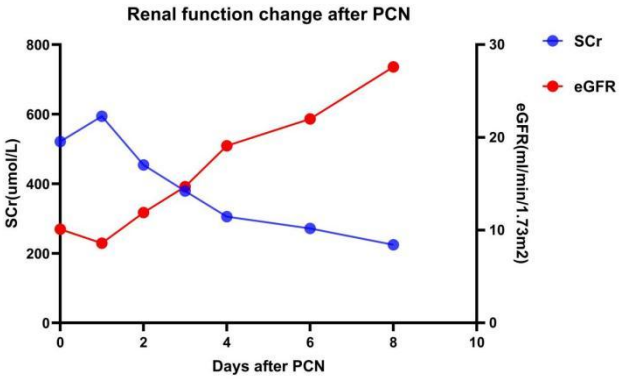

Fig 2. One representative patient showing an initial increase followed by a decrease in SCr, and an initial decrease followed by an increase in eGFR after PCN.

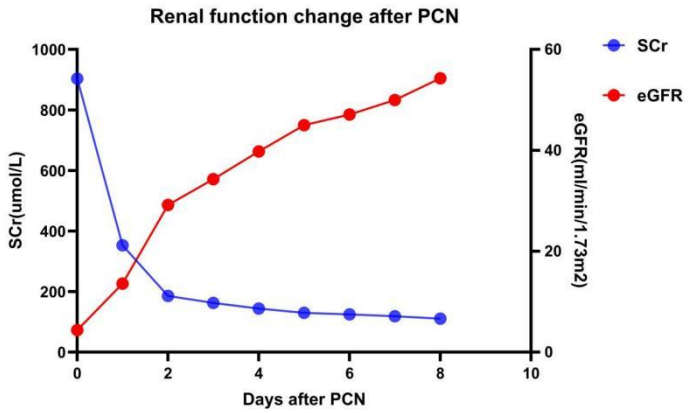

Fig 3. The other representative patient demonstrating a continuous decline in SCr and a sustained increase in eGFR after PCN.

Discussion

In our study, both univariate screening and subsequent logistic regression identified age and cystatin C as statistically significant risk factors for short-term renal function changes following PCN. Firstly, advanced age is a well-established risk factor for the development and progression of many diseases. Previous studies have shown that glomerular filtration rate declines every decade, and renal function gradually decreases with age (17). Even in the absence of age-related comorbidities, aging is associated with structural and functional changes in the kidneys (18). Some studies have reported that total kidney volume decreases by approximately 16 cm^3 per decade, with a more pronounced decline after the age of 60 (19). At the microscopic level, aging is accompanied by glomerulosclerosis, hardening of small intrarenal vessels, tubular atrophy, interstitial fibrosis, and a reduction in nephron number (20). Therefore, age may be a critical determinant of renal function. As individuals grow older, the functional reserve of the kidneys diminishes, resulting in a reduced capacity for rapid recovery after renal injury.

Cystatin C, a low-molecular-weight protein, plays important roles in cellular regulation, proliferation, adhesion, apoptosis, lipid metabolism, and immune responses. It is produced at

a constant rate, freely filtered by the glomeruli, and subsequently reabsorbed and metabolized by proximal tubular cells. Unlike creatinine, it is not influenced by muscle mass, giving it a significant advantage as a marker of renal function (21). Furthermore, previous studies have reported that cystatin C levels vary with age due to physiological declines in kidney function associated with aging (22). Therefore, we suggest that in this study, the identification of cystatin C as a risk factor for insignificant short-term renal function recovery after PCN is also related to age. The lack of significant statistical difference in preoperative creatinine levels further highlights the superior utility of cystatin C in assessing renal function and predicting its changes.

We also noted that although the presence of hypertension did not show a statistically significant difference in the subsequent logistic regression analysis, univariate screening using the chi-square test revealed a significant disparity between the two groups. This discrepancy may be attributed not only to the influence of age on hypertension but also to the bidirectional relationship between declining renal function and the development of hypertension (23). It is well established that hypertension contributes to renal impairment primarily through activation of the renin-angiotensin-aldosterone system (RAAS), thereby affecting renal function (24). Conversely, renal issues such as hydronephrosis can also lead to elevated blood pressure. The underlying mechanisms are complex, involving increased activity of the RAAS and renal sympathetic nerve activity. Additionally, oxidative stress, nitric oxide deficiency in the affected kidney, and subsequent resetting of the tubuloglomerular feedback mechanism may play crucial roles in the development and persistence of hypertension (25). We propose that hypertension not only impacts renal function but also diminishes functional renal reserve,

resulting in a reduced capacity for short-term recovery of renal function following injury.

We observed that in a subset of patients, SCr levels initially increased and then decreased, accompanied by a transient decline followed by an improvement in eGFR shortly after PCN. This phenomenon may be attributed to ischemic reperfusion injury (IRI) following the partial restoration of blood supply to the previously compressed renal cortex after relief of hydronephrosis. The exact mechanisms underlying renal IRI remain incompletely understood; however, the predominant explanation involves the production of reactive oxygen species upon reperfusion, leading to tissue damage via oxidative stress. This is further exacerbated by associated inflammatory responses, collectively contributing to the transient postoperative rise in SCr levels (26). It is noteworthy that research specifically focused on renal IRI following the relief of hydronephrosis remains limited. Most studies concentrate on scenarios such as kidney transplantation, shock, postoperative revascularization, and renal artery stenosis or occlusion. In contrast, the progression of renal injury due to hydronephrosis is relatively slow. Although animal studies have suggested that hydronephrotic rats exhibit increased susceptibility to renal IRI (27), the IRI resulting from decompression of obstructed kidneys is likely to be limited and reversible.

Furthermore, previous studies have indicated a certain correlation between the degree of hydronephrosis and renal cortical thickness before decompression and the subsequent changes in renal function (28). However, existing research has primarily focused on the medium to long term functional changes following surgical correction of congenital hydronephrosis in children. In contrast, there remains a relative lack of studies investigating the impact of preoperative hydronephrosis severity on short-term renal function changes after PCN in adults.

In this study, we assessed the degree of hydronephrosis using renal pelvis APD measured via preoperative ultrasound. Our analysis revealed no statistically significant association between this parameter and short-term renal function changes following PCN. This finding is consistent with our routine clinical observations, wherein some patients with severe hydronephrosis were found to maintain relatively normal renal function on renal dynamic imaging.

As a retrospective study, it is inherently subject to unavoidable selection bias. Furthermore, this research was conducted at a single center with a limited sample size. Additionally, the relatively narrow range of preoperative parameters available may reduce the robustness of the statistical analysis.

Conclusions

For emergency PCN, the degree of preoperative renal pelvis APD showed no correlation with short-term postoperative changes in renal function, whereas advanced age and elevated cystatin C levels may be considered risk factors for such short-term functional changes.

Author Contributions

Yuanxiao Li conducted data analysis and drafted the manuscript. Yunduo Fan conducted data collection and assisted with clinical management. Wei Jiang critically revised the manuscript for important knowledge content.

Funding

This research was supported by grants from the National Natural Science Foundation of China [82370724], the Qingdao Key Health Discipline Development Fund and the Qingdao Key Clinical Specialty Elite Discipline project.

Ethics Statement

This study was approved by the Ethics Committee of The Affiliated Hospital of Qingdao University and conducted in accordance with the ethical standards of the 1964 Declaration of Helsinki and its later amendments. The number is QYFY WZLL 42258.

Consent

Written informed consent was obtained from all participants. Clinical trial number: not applicable.

Conflicts of Interest

The authors declare no conflicts of interest.

Acknowledgements

N/A.

Data Availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declaration of Generative AI in Scientific Writing

The authors did not use generative AI or AI-assisted technologies in the development of this manuscript.

Reference

1. Chaabane W, Praddaude F, Buleon M, Jaafar A, Vallet M, Rischmann P, et al. Renal functional decline and glomerulotubular injury are arrested but not restored by release of unilateral ureteral obstruction (UUO). Am J Physiol Renal Physiol. 2013 Feb 15;304(4):F432-439.

2. Pietropaolo A, Seoane LM, Abadia AAS, Geraghty R, Kallidonis P, Tailly T, et al. Emergency upper urinary tract decompression: double-J stent or nephrostomy? A European YAU/ESUT/EULIS/BSIR survey among urologists and radiologists. World J Urol. 2022;40(7):1629–36.

3. Regalado SP. Emergency Percutaneous Nephrostomy. Semin Interv Radiol. 2006 Sept;23(3):287–94.

4. Farrell TA, Hicks ME. A review of radiologically guided percutaneous nephrostomies in 303 patients. J Vasc Interv Radiol JVIR. 1997;8(5):769–74.

5. Li AC, Regalado SP. Emergent Percutaneous Nephrostomy for the Diagnosis and Management of Pyonephrosis. Semin Interv Radiol. 2012 Sept;29(3):218–25.

6. Yoo MJ, Bridwell RE, Inman BL, Henderson JD, Long B. Approach to nephrostomy tubes in the emergency department. Am J Emerg Med. 2021 Dec;50:592–6.

7. Zagoria RJ, Dyer RB. Do's and don't's of percutaneous nephrostomy. Acad Radiol. 1999 June;6(6):370–7.

8. Florido C, Herren JL, Pandhi MB, Niemeyer MM. Emergent Percutaneous Nephrostomy for Pyonephrosis: A Primer for the On-Call Interventional Radiologist. Semin Interv Radiol. 2020 Mar;37(1):74–84.

9. Dagli M, Ramchandani P. Percutaneous nephrostomy: technical aspects and indications. Semin Interv Radiol. 2011 Dec;28(4):424–37.

10. Radecka E, Magnusson A. Complications associated with percutaneous nephrostomies. A retrospective study. Acta Radiol Stockh Swed 1987. 2004 Apr;45(2):184–8.

11. Lewis S, Patel U. Major complications after percutaneous nephrostomy-lessons from a department audit. Clin Radiol. 2004 Feb;59(2):171–9.

12. Anderson RG, Bueschen AJ, Lloyd LK, Dubovsky EV, Burns JR. Short-term and long-term changes in renal function after donor nephrectomy. J Urol. 1991 Jan;145(1):11–3.

13. Rojas-Canales DM, Li JY, Makuei L, Gleadle JM. Compensatory renal hypertrophy following nephrectomy: When and how? Nephrol Carlton Vic. 2019 Dec;24(12):1225–32.

14. Forni LG, Darmon M, Ostermann M, Oudemans-van Straaten HM, Pettilä V, Prowle JR, et al. Renal recovery after acute kidney injury. Intensive Care Med. 2017 June;43(6):855–66.

15. Vijayan A, Abdel-Rahman EM, Liu KD, Goldstein SL, Agarwal A, Okusa MD, et al. Recovery after Critical Illness and Acute Kidney Injury. Clin J Am Soc Nephrol CJASN. 2021 Oct;16(10):1601–9.

16. Levey AS, Stevens LA, Schmid CH, Zhang Y (Lucy), Castro AF, Feldman HI, et al. A New Equation to Estimate Glomerular Filtration Rate. Ann Intern Med. 2009 May 5;150(9):604–12.

17. Beck LH. Changes in renal function with aging. Clin Geriatr Med. 1998 May;14(2):199–209.

18. Hommos MS, Glassock RJ, Rule AD. Structural and Functional Changes in Human Kidneys with Healthy Aging. J Am Soc Nephrol JASN. 2017 Oct;28(10):2838–44.

19. Roseman DA, Hwang SJ, Oyama-Manabe N, Chuang ML, O'Donnell CJ, Manning WJ, et al. Clinical associations of total kidney volume: the Framingham Heart Study. Nephrol Dial Transplant. 2017 Aug;32(8):1344–50.

20. Goyal VK. Changes with age in the human kidney. Exp Gerontol. 1982;17(5):321–31.

21. Spencer S, Desborough R, Bhandari S. Should Cystatin C eGFR Become Routine Clinical Practice? Biomolecules. 2023 July 5;13(7):1075.

22. Evangelopoulos AA, Vallianou NG, Bountziouka VP, Giotopoulou AN, Bonou MS, Barbetseas J, et al. The Impact of Demographic Characteristics and Lifestyle in the Distribution of Cystatin C Values in a Healthy Greek Adult Population. Cardiol Res Pract. 2010 Oct 14;2011:163281.

23. Ma C, Yu B, Zhang W, Wang W, Zhang L, Zeng Q. Associations between aldehyde dehydrogenase 2 (ALDH2) rs671 genetic polymorphisms, lifestyles and hypertension risk in Chinese Han people. Sci Rep. 2017 Sept 11;7:11136.

24. Hong MH, Jin XJ, Yoon JJ, Lee YJ, Oh HC, Lee HS, et al. Antihypertensive Effects of Gynura divaricata (L.) DC in Rats with Renovascular Hypertension. Nutrients. 2020 Oct 29;12(11):3321.

25. Carlström M. Causal link between neonatal hydronephrosis and later development of hypertension. Clin Exp Pharmacol Physiol. 2010 Feb;37(2):e14-23.

26. Chen Y, Yang L. Cellular senescence in renal ischemia-reperfusion injury. Chin Med J (Engl). 2025 Aug 5;138(15):1794–806.

27. Vilskersts R, Vilks K, Videja M, Cirule H, Zharkova-Malkova O, Sevostjanovs E, et al.

Rats with congenital hydronephrosis show increased susceptibility to renal ischemia-reperfusion injury. Physiol Rep. 2020 Nov;8(22):e14638.

28. Sibai H, Salle JL, Houle AM, Lambert R. Hydronephrosis with diffuse or segmental cortical thinning: impact on renal function. J Urol. 2001 June;165(6 Pt 2):2293–5.