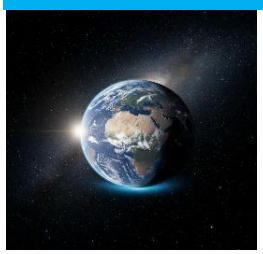
Journal of Education, Health and Sport. 2026;88:68331.
eISSN 2391-8306.

https://doi.org/10.12775/JEHS.2026.88.68331



Journal of Education, Health and Sport. eISSN 2450-3118

Journal Home Page
https://apcz.umk.pl/JEHS/index

CZAPLINSKI, Mieszko, KRÓL, Maria, BRUSKA, Natalia Marta, BILYK, Andrii, PUSTUŁA, Paweł, PATALONG, Mikołaj, BŁASZKOWSKI, Bartłomiej, KOŁODZIEJ, Przemysław, WIĘCKOWSKA, Katarzyna and WOLSKI, Adam. When the Kidney Looks Like Thyroid: Thyroidization of the Kidney as a Histological Pattern and Its Potential Clinical Correlation - A Scoping Review. Journal of Education, Health and Sport. 2026;88:68331. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2026.88.68331

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 15.01.2026. Revised: 10.02.2026. Accepted: 14.02.2026. Published: 17.02.2026.

When the Kidney Looks Like Thyroid: Thyroidization of the Kidney as a Histological Pattern and Its Potential Clinical Correlation - A Scoping Review

Mieszko Czapliński[1], ORCID https://orcid.org/0009-0003-8052-4129

E-mail: 194058@uck.gda.pl

[1] University Clinical Centre in Gdańsk, Medical University of Gdańsk, ul. Dębinki 7, 80-952 Gdańsk, Poland

Maria Król[2] ORCID https://orcid.org/0000-0003-0068-7837
E-mail mari.m.krol@gmail.com
[2] Lower Silesian Centre of Oncology, Pulmonology and Hematology in Wrocław, Plac Ludwika Hirszfelda 12, 53-413 Wrocław, Poland

Natalia Marta Bruska[1], ORCID: https://orcid.org/0009-0008-5749-874X
E-mail: bruska.nataliax@gmail.com
[1] University Clinical Centre in Gdańsk, Medical University of Gdańsk, ul. Dębinki 7, 80-952 Gdańsk, Poland

Andrii Bilyk[3], ORCID: https://orcid.org/0009-0001-5020-1113
E-mail: andrzej.bilyk02@gmail.com
[3] Samodzielny Publiczny Zakład Opieki Zdrowotnej MSWiA w Gdańsku. ul. Kartuska 4/6, 80-104 Gdańsk, Poland

Paweł Pustuła[4], ORCID https://orcid.org/0009-0003-4494-1080
E-mail p.pustula@icloud.com
[4] Family Medicine Center, Medical University of Gdańsk, Dębinki 7, 80-211 Gdańsk, Poland

Mikołaj Patalong[5], ORCID https://orcid.org/0000-0003-4230-7521
E-mail patalongmikolaj@gmail.com
[5] Individual Medical Practice Mikołaj Patalong, ul. Zabrzańska 26, 41-907 Bytom, Poland

Bartłomiej Błaszkowski[6], ORCID https://orcid.org/0009-0001-9898-2908
E-mail: B.blaszkowski46@gmail.com
[6] F. Ceynowa Specialist Hospital in Wejherowo 10 Dr. Alojzy Jagalski Street 84-200 Wejherowo Poland

Przemysław Kołodziej[7], ORCID https://orcid.org/0009-0003-5725-2372
E-mail przemyslaw.i.kolodziej@gmail.com
[7] Individual Medical Practice Przemysław Igor Kołodziej, ul. Alojzego Felińskiego 30/9 41-923 Bytom, Poland

Katarzyna Więckowska[8], ORCID https://orcid.org/0009-0002-4233-1927
E-mail: katarzyna.wieckowska@o2.pl
[8] Individual Medical Practice Katarzyna Więckowska, Deszczowa 18 40-318 Katowice

Adam Wolski[9], ORCID: https://orcid.org/0009-0005-1969-0640
E-mail: adam00wolski@gmail.com
[9] Gdańsk Medical University, Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk, Poland

Corresponding author:
Mieszko Czapliński, E-mail: 194058@uck.gda.pl

Abstract

Background: thyroidization of the kidney is a histological pattern characterized by atrophic renal tubules filled with eosinophilic, colloid-like casts, imparting a resemblance to thyroid follicles. Although traditionally regarded as a nonspecific marker of chronicity in renal disease, its biological significance, clinical correlations, and potential prognostic value remain poorly defined.

Aim: to map and synthesize the existing literature on renal thyroidization, focusing on histomorphology, proposed pathophysiological mechanisms, disease associations, and reported clinical correlations.

Material and methods: we conducted a scoping review of the available literature describing thyroidization of the kidney in human renal pathology. Two databases were screened - PubMed and Scopus. Eligible sources included original studies, case series, case reports, and reviews that addressed histological features, mechanisms, or clinical contexts of thyroidization. Supporting information was extracted from relevant textbooks widely recognized as authoritative on the field of renal pathology. Data were charted across predefined domains including underlying renal disease, extent and distribution of thyroidization, associated histological changes, and any reported clinical or prognostic implications.

Results: the literature associates thyroidization with chronic tubulointerstitial damage, mostly in chronic kidney disease of diverse etiologies. The pattern reflects tubular atrophy with proteinaceous casts, accompanied by interstitial fibrosis and chronic inflammation. Direct clinical correlations are rarely assessed systematically; however, the presence of thyroidization

is usually interpreted as a marker of long-standing, irreversible injury. Reports suggesting links with disease duration, recurrent infection, or poor renal outcome remain largely descriptive.
Conclusions: thyroidization of the kidney is a reproducible and visually distinctive histological pattern that functions primarily as a morphological signpost of chronicity. Its potential value as a semi-quantitative marker of disease duration, prior injury burden, or prognosis has not been studied. Future work integrating digital pathology, quantitative morphometry, and clinicopathological correlation may clarify whether thyroidization carries information beyond that conveyed by established chronicity indices.

Keywords: thyroidization, kidney pathology, chronic kidney disease, tubulointerstitial injury, histological patterns

1. Introduction:

Renal pathology relies heavily on pattern recognition, where certain morphologic constellations serve as indicators of underlying disease mechanisms or temporal stages of injury [1]. The kidney cell – nephron is morphologically and functionally divided into 2 regions – glomerulus, where filtration takes place and tubules where the resulting urine is condensed and the water is regained [2]. Each part of nephron is subject to different diseases, and these diseases have different histopathological pictures. Glomerulopathies are numerous and visually distinct, and with the help of additional immunohistochemical or electron-microscopic techniques they can be differentiated [1]. In case of tubular diseases, they too have histopathological patterns that may indicate the origin of the disease affecting the tubules. One such pattern is thyroidization of the kidney [3].

Despite being mentioned in scientific literature, thyroidization remains an underexplored phenomenon. It is typically labelled as “nonspecific” and interpreted as evidence of long-standing tubulointerstitial damage, yet the biological processes leading to its formation and its possible clinical implications are seldom discussed in depth.

Given renewed interest in refining histological markers of chronic kidney disease [4] a reappraisal of classical histological patterns such as thyroidization is timely. This scoping review aims to map the existing knowledge on renal thyroidization, propose directions of further studies on that topic, and explore whether this pattern may have underrecognized clinicopathological relevance.

2. Methods (review design):

Because this work is a scoping review, it does not have to be conducted in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses guidelines [5]. Since it is not a systematic review, no need for PROSPERO registration [6] arises.

A literature search was designed in order to capture publications describing thyroidization of the kidney. The search queried 2 electronic databases: PubMed [7] and Scopus [8]. These databases were chosen because of their universal character, vast expanse of sources available within them and the user-friendliness of search interfaces. Each time, appropriate queries were entered into the search window of the database. There were 4 basic queries listed below:

Q1 - “tubular thyroidization”

Q2 - “thyroidization”

Q3 - “thyroidization” AND “kidney”

Q4 - “thyroidization” AND “renal”

In order to ensure that no publications were missed due to spelling variants, there were three additional queries:

Q5 - “thyroidisation”

Q6 - “thyreoidisation”

Q7 - “thyreoidization”

Such a database search returned the following numbers of results (Table 1):

Table 1

	PubMed	**Scopus**
Q1	12	12
Q2	22	27
Q3	17	20
Q4	17	17
Q5	2	7
Q6	0	0
Q7	0	0

The results were then subject to manual screening, based on the eligibility criteria; those publications that fulfilled the criteria were included in this work, adding to the reference list. Sources were deemed eligible if they:

- describe thyroidization of the kidney in human renal biopsy or nephrectomy specimens,
- provide histological characterization, pathophysiological interpretation, or clinical context,
- are original research articles, case series, case reports, or narrative reviews.

10 authors independently screened titles and abstracts, followed by full-text assessment. Discrepancies were resolved by consensus. Animal-only studies and abstracts without full text were excluded from this work. Relevant textbook chapters were screened manually to identify additional sources and information about the phenomenon of thyroidization [9], [10].

3. Results:

Thyroidization is characterized by atrophic renal tubules with flattened or attenuated cuboidal epithelium distended by dense, eosinophilic, colloid-like protein intratubular casts, creating a resemblance to thyroid follicles on routine haematoxylin–eosin staining [3]. The pattern is not disease-specific and may coexist with a wide range of tubular, glomerular, or vascular lesions. It typically involves distal tubules and collecting ducts. The pattern is commonly associated with interstitial fibrosis of tubular basal membranes (especially well seen in the special stains for fibrous material, like Jones silver methenamine stain [11]). and with chronic inflammation. Special stains usually demonstrate the proteinaceous nature of the casts [12], while immunohistochemical studies suggest derivation from filtered or secreted proteins rather than true thyroglobulin [13]. The most frequently encountered proteins in the casts are hyaline and uromodulin (the so-called Tamm-Horsfall protein casts [14]), but there are many more possible proteins that can build such casts; again, additional stains and immunohistochemical staining are invaluable aids in the diagnostic process, enabling the differentiation of proteins and sometimes the identification of disease that causes the casts to form [15]. Despite the fact that already in 1960s tubular abnormalities were studied in the electron microscopy [16], ultrastructural data are sparse but support advanced epithelial degeneration and impaired tubular transport. Histopathology is needed to find thyroidization; nevertheless, cytological techniques can provide some diagnostic clues of its occurrence [17].

There are several different theories that try to explain the creation of such patterns. The proposed mechanisms are not mutually exclusive; in fact, they may coexist. The first mechanism claims that the cause of thyroidization is chronic tubular injury, inflammation and

atrophy - long-standing ischaemia, inflammation, or obstruction leads to epithelial simplification (flattening) and loss of resorptive capacity; renal tubular injury contributes to severity and chronicity of the disease process due to various mechanical, immunological and metabolic mechanisms [18]. As for proteinaceous cast accumulation, reduced tubular flow and impaired reabsorption promote intraluminal protein precipitation [19]. The recurrent infection obstruction, or inflammation, particularly in chronic pyelonephritis and reflux nephropathy, due to repeated injury may favour cast formation and tubular remodelling [20]. Interestingly, there are some theories that the Tamm-Horsfall protein not only is a result of above-mentioned processes, but it also can contribute to further development and intensification of tubular atrophy and chronic renal disease either directly or indirectly through facilitating reflux uropathy and pyelonephritis [21]. Importantly, thyroidization appears to represent an end-stage morphological adaptation of distorted tubules rather than an active disease process/disease entity per se, and is rather associated with chronicity than with acute tubular injuries, where other histological phenotypes are more common and thyroidization was not reported in this kind of diseases [22].

Across the literature thyroidization has been reported in association with numerous medical conditions, the majority of them leading to chronic kidney disease and the decrease in renal function. It is noteworthy that thyroidization, although predominantly occurring in chronic diseases, is not restricted to them - some acute kidney injuries can also lead to tubular thyroidization (e.g. gasoline poisoning). The entities where thyroidization was reported to be found are enumerated in **Table 2**.

Table 2

Disease/Clinical situation	**Histopathological picture**	**Reference**
Antiphospholipid syndrome	subendothelial fibrosis and luminal arteriolosclerosis of renal vessels, mesangiolysis, focal cortical atrophy, C3 and IgM deposits in glomeruli, membranaceous glomerulitis, tubular thyroidization with eosinophilic casts, typically the inflammation is not pronounced	[23], [24], [25]
Systemic Lupus Erythematosus (SLE)	Thrombotic microangiopathy (TMA), fibrous intimal hyperplasia (FIH), fibrocellular arterial occlusion (FAO), focal cortical atrophy (FCA), tubular	[26], [27], [28]

Table 2		
	thyroidization; often coexistence with APSN and its histopathological image	
MELAS Syndrome	Interstitial fibrosis, dense inflammatory infiltrate, tubular atrophy, thyroidization, prominent vascular lesions with intimal fibrosis and hyaline arteriolosclerosis, glomerular abnormalities	[29]
APOL-1 nephropathy risk variants	less obsolescent glomerulosclerosis, more solidified/atrophic glomerulonephritis, more thyroidization-type tubular atrophy, more microcystic tubular dilatation (as opposed to other patients who didn't have *APOL-1* risk alleles).	[30]
Xanthogranulomatous pyelonephritis	diffuse granulomatous inflammatory infiltrate with xanthomatous macrophages, giant cells; loss of cortico-medullary differentiation, glomerular sclerosis, tubular atrophy and thyroidization, interstitial fibrosis, chronic inflammation, blood vessels thickening	[31]
Vesicoureteral reflux screening after kidney transplantation	Interstitial fibrosis, tubular atrophy, mononuclear cell infiltrates, thyroidization	[32]
COVID-19, Fabry disease, kidney transplant	Tubular thyroidization – due to complicated clinical history of the patient one cannot certainly establish due to which clinical entity it occurred.	[33]
Kidney transplantation	Thyroidization found in 16 out of 213 renal allograft biopsies and sometimes coexisting with tubulointerstitial nephritis, low-capacity urinary bladders, vesicoureteral reflux, UTIs	[34]
Gasoline poisoning due to i.v. administration	Marked congestion, sclerotic glomeruli, thickened and hyalinized vessel walls with hyaline deposition in arterioles, tubular thyroidization, interstitial fibrosis with diffuse lymphoplasmacytic inflammatory infiltrate	[35]

In addition, tubular thyroidization was reported in analgesic nephropathy, hydronephrosis, nephronophthisis if preceded by pyelonephritis [36].

4. Limitations & Discussion:

This study is limited by the choice of two databases. Perhaps more valuable entries could be found if we screened more databases, expanding the corpus of publications to be included and analysed. While being aware of this, we tried to limit the effect of this limitation by choosing the most relevant databases in the field of medicine; their vast coverage of articles should guarantee that at least the most relevant publications in the field were not missed.

While renal pathology is strongly based on the histopathological patterns when it comes to recognising disease entities under the microscope, the extent of microscopic changes does not always correlate with the severity of symptoms. Already in 1960s [37] and in 1980s [38] the researchers tried to correlate the clinical and histopathological pictures of renal diseases, yet with mixed results. Direct clinicopathological correlations are infrequently addressed in the literature. In most reports, thyroidization is interpreted qualitatively as a marker of chronicity and irreversibility, analogous to global glomerulosclerosis or advanced interstitial fibrosis. Only anecdotal and largely case-based associations are described between thyroidization and disease duration, history of recurrent urinary tract infection, or obstructive uropathy. Quantitative assessment of the extent or density of thyroidized tubules is not routinely performed, and no studies to date have evaluated its independent association with renal function decline, response to therapy, or patient-centred outcomes. Nevertheless, the consistent linkage of thyroidization with advanced tubulointerstitial remodelling (a common factor in the majority of diseases from **Table 2**) suggests potential roles as a semi-quantitative indicator of cumulative tubular injury, a morphological footprint of prior obstructive or infectious insults, or a complementary descriptor within existing chronicity indices used in renal pathology.

From a practical diagnostic perspective, recognition of thyroidization reinforces the interpretation of renal injury as long-standing and largely irreversible. Its presence should prompt careful evaluation for coexisting features of chronic kidney damage, including interstitial fibrosis, tubular atrophy, and global glomerulosclerosis, and may help contextualize active lesions seen elsewhere in the biopsy. Considering the differential diagnoses, there are some rare disease entities that mimic thyroidization, yet are not connected with chronic tubular atrophy. It is possible (although rare) for thyroid follicular carcinoma to metastasise into kidneys [39] [40] – in such case the follicles seen in kidney specimens are real thyroid follicles. In order to complicate things further, kidney neoplasms themselves can mimic thyroid follicular patterns despite not having anything in common with tubular atrophy and thyroidization, nor having common points with the thyroid gland. An extremely rare variant of kidney neoplasm

called follicular thyroid-like carcinoma of the kidney is also a differential diagnosis when thyroid-resembling follicles are encountered in renal specimens [41]. Immunohistochemical investigation directed against thyroid-specific proteins, renal proteins and Tamm-Horsfall protein can help to solve the diagnostic dilemma.

Clinically, thyroidization serves as a histological clue to prior disease mechanism, particularly recurrent infections, vesicoureteral reflux, chronic obstruction, chronic inflammation with or without auto-immunity, or genetic diseases pertaining kidney, even when clinical picture is asymptomatic or the patient' documentation is incomplete. In transplant or native kidney biopsies with mixed acute and chronic features, prominent thyroidization may support a guarded prognosis and inform expectations regarding chronic course of disease, its rather longer duration, limited reversibility/irreversibility and therapeutic response.

Several valuable research ideas can be pursued based on this review. The first of these is the standardisation of thyroidization. There is currently no consensus definition or grading system for thyroidization, limiting reproducibility across studies and diagnostic reports. The notion of thyroidization is largely observer-dependent, and the inter-observer discrepancies in various domains of pathology are widely known and proven in the scientific literature [42] [43] [44]. A standardisation system that could reduce this inter-observer discordance would advance the research on this topic. Following the issue of standardization comes the issue of quantification. The extent of thyroidization has not been systematically measured; if a grading scale based e.g., on the percentage of specimen thyroidization (something similar to reporting system used in prostate biopsies, where the percentage of tumour in the biopsy specimen is reported [45]) were made, the precision of observation and the further reduction of interobserver error would be achieved. Such investigations would be purely theoretical unless they were correlated with established markers of chronic kidney disease severity. Rigorous studies linking thyroidization with clinical history, renal function trajectories, or outcomes are lacking. If such correlations were carried out, more could be found out about its diagnostic relevance and potential uses.

The thyroidization pattern has not yet been explored using contemporary tools such as digital image analysis, machine learning, or spatial proteomics. Currently the digital informatics technologies using deep learning are ubiquitous in all domains of medicine, permeating radiology, but also pathology [46] [47]. An increasing number of pathology specialists are occupied with creating and employing artificial intelligence in their work, and there is an ever-increasing body of publications devoted to explaining these technologies to a wider medical audience [48]. The striking follicle-like morphology of thyroidized tubules makes this pattern

particularly amenable to automated detection using convolutional neural networks in whole-slide imaging. Integration of such approaches may allow large-scale, unbiased assessment of thyroidization and its relationship to clinical renal outcomes.

We propose that tubular thyroidization be reconsidered as a distinct end-stage tubular phenotype rather than a purely descriptive curiosity. A simple, reproducible grading system (e.g. absent, focal, multifocal, diffuse) could be incorporated into routine reporting and tested for prognostic relevance. Furthermore, in the future, standardized reporting or grading of thyroidization could enhance communication between pathologists and clinicians and facilitate integration into prognostic models, especially if validated through quantitative or digital pathology approaches.

5. Conclusions:

Thyroidization of the kidney is a classical, yet understudied histological pattern that signals chronic tubulointerstitial injury. While traditionally regarded as a nonspecific marker of chronicity, the existing literature suggests that it may represent a reproducible end-stage tubular phenotype shared across multiple (mostly chronic) tubular renal disease entities. Thyroidization is important when it comes to differential diagnostics, as some of its differentials are neoplastic and correlate with worse survival prognosis. There is a lack of systematic studies, meta-analyses and randomised control trials that would examine in detail the diagnostic and clinical usefulness of thyroidization and correlate it with clinical outcomes. Reframing tubular thyroidization as a quantifiable and potentially informative morphological feature may certainly open new ways for clinicopathological correlation, prognostic stratification, and digital-based pathology analysis, contributing not only to medical advances and the patient well-being, but also to the development of pathology-related information technologies. Systematic studies are required to determine whether this distinctive pattern conveys information beyond that provided by established renal indices. The field has until now certainly not been exhausted, and there is still a lot of work to be done – these certainly are not the last words written on the tubular thyroidization.

Supplementary files: not applicable.

Author Contributions: Conceptualization and methodology: M.C..; formal analysis and investigation: M.C.; resources: M.C., M.K., N.B., P.P., A.B., M.P., A.W., P.K., B.B., K.W.; data curation: M.C., M.K., N.B., P.P., A.B., M.P., A.W., P.K., B.B., K.W.; writing—original draft preparation: M.C., M.K., N.B., P.P., A.B., M.P., A.W., P.K., B.B., K.W.; writing—review

and editing: M.C., M.K., N.B., P.P., A.B., M.P., A.W., P.K., B.B., K.W.; visualization: M.C.; supervision, M.C. All authors have read and agreed to the published version of the manuscript.

Funding: no external funding was obtained.

Institutional Review Board Statement: not applicable.

Informed Consent Statement: not applicable.

Data Availability Statement: the study is a review; thus, no new data were created while creating this publication; existing data used in this work can be found in PubMed and Scopus databases; further inquiries can be directed at the corresponding author.

Use of generative AI: no generative AI was used while writing this work.

Acknowledgments: not applicable.

Conflicts of Interest (Disclosure): the Authors declare no conflicts of interest.

References:

1. Qasim, H., Abu Shugaer, M., Dibian, S., Ktaifan, M., Khattab, K., Leoni, M. L. G., & Varrassi, G. (2025). Patterns of glomerular injury: Histopathological classification and clinical correlation. *Cureus*. https://doi.org/10.7759/cureus.91728

2. Brown A. L., Jr (1966). The structure of the nephron. *The Medical clinics of North America, 50*(4), 927–935. https://doi.org/10.1016/s0025-7125(16)33140-6

3. Lusco, M. A., Fogo, A. B., Najafian, B., & Alpers, C. E. (2016). AJKD Atlas of renal pathology: Tubular atrophy. *American Journal of Kidney Diseases*, *67*(6), e33–e34. https://doi.org/10.1053/j.ajkd.2016.04.007

4. Trevisani, F., Floris, M., Cinque, A., Bettiga, A., & Dell'Antonio, G. (2023). Renal Histology in CKD Stages: Match or Mismatch with Glomerular Filtration Rate? In *Nephron* (Vol. 147, Issue 5, pp. 266–271). S. Karger AG. https://doi.org/10.1159/000527499

5. Tricco, AC, Lillie, E, Zarin, W, O'Brien, KK, Colquhoun, H, Levac, D, Moher, D, Peters, MD, Horsley, T, Weeks, L, Hempel, S et al. PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. Ann Intern Med. 2018,169(7):467-473. doi: 10.7326/M18-0850

6. Schiavo, J. H. (2019). PROSPERO: An International Register of Systematic Review Protocols. *Medical Reference Services Quarterly*, *38*(2), 171–180. https://doi.org/10.1080/02763869.2019.1588072

7. U.S. National Library of Medicine. (n.d.). *PubMed*. National Center for Biotechnology Information. https://pubmed.ncbi.nlm.nih.gov/ Accessed 22nd December 2025.

8. Scopus preview - Scopus - Welcome to scopus. (n.d.). https://www.scopus.com/home.uri?zone=header&origin=sbrowse Accessed 22nd December 2025.

9. Jennette, J. C., Olson, J. L., Silva, F. G., & D'Agati, V. D. (Eds.). (2015). *Heptinstall's pathology of the kidney* (7th ed.). Wolters Kluwer.

10. Tisher, C. C., & Brenner, B. M. (Eds.). (1994). *Renal pathology with clinical and functional correlations* (2nd ed.). Lippincott Williams & Wilkins.

11. Golberg, M., Kobos, J., Clarke, E., Smędra, A., Zagacki, D., Wróbel-Roztropiński, A., & Żytkowski, A. (2023). Concise overview of selected metal-based stains: Application in morphology. In *Translational Research in Anatomy* (Vol. 33). Elsevier GmbH. https://doi.org/10.1016/j.tria.2023.100265

12. Cathro, H. P., Shen, S. S., & Truong, L. D. (2018). Diagnostic histochemistry in medical diseases of the kidney. *Seminars in diagnostic pathology*, *35*(6), 360–369. https://doi.org/10.1053/j.semdp.2018.10.001

13. Nadasdy, T., Laszik, Z., Blick, K. E., Johnson, D. L., & Silva, F. G. (1994). Tubular atrophy in the end-stage kidney: a lectin and immunohistochemical study. *Human pathology*, 25(1), 22–28. https://doi.org/10.1016/0046-8177(94)90166-x

14. Micanovic, R., Lafavers, K., Garimella, P. S., Wu, X. R., & El-Achkar, T. M. (2020). Uromodulin (Tamm-Horsfall protein): Guardian of urinary and systemic homeostasis. *Nephrology Dialysis Transplantation*, *35*(1), 33–43. https://doi.org/10.1093/ndt/gfy394

15. Dvanajscak, Z., Cossey, L. N., & Larsen, C. P. (2020). A practical approach to the pathology of renal intratubular casts. In *Seminars in Diagnostic Pathology* (Vol. 37, Issue 3, pp. 127–134). W.B. Saunders. https://doi.org/10.1053/j.semdp.2020.02.001

16. Flume, J. B. et al. "An Electron Microscopic Study Of Tubular Lesions in Human KIidney Biopsy Specimens." *The American journal of pathology* vol. 43 (6) (1963): 1067-1087. No DOI available. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1949774/

17. Rosa, M. (2011). Cytomorphology of kidney "thyroidization." In *Diagnostic Cytopathology* (Vol. 39, Issue 7, pp. 508–509). https://doi.org/10.1002/dc.21468

18. Liu, B. C., Tang, T. T., Lv, L. L., & Lan, H. Y. (2018). Renal tubule injury: a driving force toward chronic kidney disease. In *Kidney International* (Vol. 93, Issue 3, pp. 568–579). Elsevier B.V. https://doi.org/10.1016/j.kint.2017.09.033

19. Hoyer, J. R., & Seiler, M. W. (1979). Pathophysiology of Tamm–Horsfall protein. *Kidney International*, *16*(3), 279–289. https://doi.org/10.1038/ki.1979.130

20. Sahutoglu, T., & Perazella, M. A. (2025). Update on acute tubulointerstitial nephritis: Clinical features, immunologic insights, and diagnostic and treatment approaches. *Kidney International Reports*, *10*(6), 1643–1656. https://doi.org/10.1016/j.ekir.2025.03.050

21. Andriole, V. T. (1985). The role of Tamm-Horsfall protein in the pathogenesis of reflux nephropathy and chronic pyelonephritis. *Yale Journal of Biology and Medicine*, *58*(2), 91–100. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2589891/

22. Wen, Y., Yang, C., Menez, S. P., Rosenberg, A. Z., & Parikh, C. R. (2020). A Systematic Review of Clinical Characteristics and Histologic Descriptions of Acute Tubular Injury. *Kidney International Reports*, *5*(11), 1993–2001. https://doi.org/10.1016/j.ekir.2020.08.026

23. Turrent-Carriles, A., Herrera-Félix, J. P., & Amigo, M. C. (2018). Renal involvement in antiphospholipid syndrome. In *Frontiers in Immunology* (Vol. 9, Issue MAY). Frontiers Media S.A. https://doi.org/10.3389/fimmu.2018.01008

24. Barbhaiya, M., Taghavi, M., Zuily, S., Domingues, V., Chock, E. Y., Tektonidou, M. G., Erkan, D., & Seshan, S. v. (2024). Efforts to Better Characterize "Antiphospholipid Antibody Nephropathy" for the 2023 ACR/EULAR Antiphospholipid Syndrome Classification Criteria: Renal Pathology Subcommittee Report. *Journal of Rheumatology*, *51*(2), 150–159. https://doi.org/10.3899/jrheum.2022-1200

25. Pons-Estel, G. J., & Cervera, R. (2014). Renal involvement in antiphospholipid syndrome. *Current rheumatology reports*, 16(2), 397. https://doi.org/10.1007/s11926-013-0397-0

26. Silvariño, R., Sant, F., Espinosa, G., Pons-Estel, G., Solé, M., Cervera, R., & Arrizabalaga, P. (2011). Nephropathy associated with antiphospholipid antibodies in patients with systemic lupus erythematosus. *Lupus*, 20(7), 721–729. https://doi.org/10.1177/0961203310397410

27. Domingues, V., Chock, E. Y., Dufrost, V., Risse, J., Seshan, S. v., Barbhaiya, M., Sartelet, H., Erkan, D., Wahl, D., & Zuily, S. (2022). Increased risk of acute and chronic microvascular renal lesions associated with antiphospholipid antibodies in patients with systemic lupus erythematosus: A systematic review and meta-analysis. In *Autoimmunity Reviews* (Vol. 21, Issue 10). Elsevier B.V. https://doi.org/10.1016/j.autrev.2022.103158

28. Shah, R., Brodsky, S. V., Hebert, L., Rovin, B. H., Nadasdy, T., & Satoskar, A. A. (2018). Zonal cortical scarring and tubular thyroidization in kidney biopsies of patients with SLE-histologic indicator for antiphospholipid antibodies. *Lupus*, 27(14), 2236–2244. https://doi.org/10.1177/0961203318809177

29. Piccoli, G. B., Bonino, L. D., Campisi, P., Vigotti, F. N., Ferraresi, M., Fassio, F., Brocheriou, I., Porpiglia, F., & Restagno, G. (2012). Chronic kidney disease, severe arterial and

arteriolar sclerosis and kidney neoplasia: on the spectrum of kidney involvement in MELAS syndrome. *BMC nephrology*, 13, 9. https://doi.org/10.1186/1471-2369-13-9

30. Larsen, C. P., Beggs, M. L., Saeed, M., Ambruzs, J. M., Cossey, L. N., Messias, N. C., Walker, P. D., & Freedman, B. I. (2015). Histopathologic findings associated with APOL1 risk variants in chronic kidney disease. *Modern Pathology*, *28*(1), 95–102. https://doi.org/10.1038/modpathol.2014.92

31. Prasad, P., Sudha, S., Niranjan, G., Verma, R., & Vimal, J. K. (2025). Xanthogranulomatous Pyelonephritis in the Tropics. *Discoveries*, *13*(3), e212. https://doi.org/10.15190/d.2025.11

32. Akioka, Y., Chikamoto, H., Horita, S., Yago, R., Tanabe, K., Yamaguchi, Y., & Hattori, M. (2009). Screening of vesicoureteral reflux in pediatric patients with kidney transplantation showing non-specific interstitial fibrosis and tubular atrophy with interstitial Tamm-Horsfall protein deposits in protocol allograft biopsy. *Clinical transplantation*, 23 Suppl 20, 2–5. https://doi.org/10.1111/j.1399-0012.2009.01000.x

33. Mahoney, R., Lee, G. K., Zepeda, J. P., Gabriel, C., Hall, K., Edwards, R., & Kimonis, V. (2021). Severe manifestations and treatment of COVID-19 in a transplanted patient with Fabry disease. *Molecular Genetics and Metabolism Reports*, 29. https://doi.org/10.1016/j.ymgmr.2021.100802

34. Ito, S., Kobayashi, A., Tsuchiya, T., Moriyama, Y., Kikuchi, M., Deguchi, T., & Yamaguchi, Y. (2009). Thyroidization in renal allografts. *Clinical transplantation*, *23 Suppl 20*, 6–9. https://doi.org/10.1111/j.1399-0012.2009.01001.x

35. Bubalo, P., Nestic, M., Martinovic, S., Bakovic, M., Mayer, D., & Mihic, A. G. (2024). Death by accidental intravenous administration of gasoline. *International journal of legal medicine*, 138(4), 1315–1321. https://doi.org/10.1007/s00414-024-03181-8

36. Laberke, H. G., Klingebiel, T., & Quack, G. (1983). A contribution to the morphology and pathogenesis of thyroid-like lesions in the kidney. *Pathology, research and practice*, *176*(2-4), 284–296. https://doi.org/10.1016/S0344-0338(83)80018-1

37. Risdon, R. A., Sloper, J. C., & De Wardener, H. E. (1968). Relationship between renal function and histological changes found in renal-biopsy specimens from patients with persistent glomerular nephritis. *Lancet* (London, England), 2(7564), 363–366. https://doi.org/10.1016/s0140-6736(68)90589-8

38. Bohle, A., Mackensen-Haen, S., & von Gise, H. (1987). Significance of tubulointerstitial changes in the renal cortex for the excretory function and concentration ability of the kidney: a morphometric contribution. *American journal of nephrology*, 7(6), 421–433. https://doi.org/10.1159/000167514

39. Nath, V., Baliga, M., Lewin, J., Souza, F., & Akhtar, I. (2015). Follicular Thyroid Carcinoma Metastatic to the Kidney: Report of a Case with Cytohistologic Correlation. *Case Reports in Pathology*, *2015*, 1–5. https://doi.org/10.1155/2015/701413

40. Hamdy, O., Shokeir, F. A., Saleh, G. A., Awni, S., & Zaki, Ma. M. A. (2020). Solitary renal metastasis of follicular thyroid carcinoma with cervical nodal deposits. *World Journal of Endocrine Surgery*, *12*(1), 30–33. https://doi.org/10.5005/jp-journals-10002-1276

41. Amin, M. B., Gupta, R., Ondrej, H., McKenney, J. K., Michal, M., Young, A. N., Paner, G. P., Junker, K., & Epstein, J. I. (2009). Primary thyroid-like follicular carcinoma of the kidney: report of 6 cases of a histologically distinctive adult renal epithelial neoplasm. *The American journal of surgical pathology*, 33(3), 393–400. https://doi.org/10.1097/PAS.0b013e31818cb8f5

42. Alexander, N. A., Park, M., Shin, H., Cruz, L., Yang, A., Rosman, I., Fields, R. C., Ansstas, G., Cornelius, L. A., & Chen, D. Y. (2025). Interobserver variability in histopathologic diagnosis of melanocytic neoplasms. *Journal of the American Academy of Dermatology*, S0190-9622(25)03432-2. Advance online publication. https://doi.org/10.1016/j.jaad.2025.12.050

43. de Gordoa, K. S., Daca-Alvarez, M., Rodrigo-Calvo, M., Archilla, I., Lopez-Prades, S., Aguirre, J. J., Alarcón-Molero, L., Jurado, M. C., Canosa, A., Giner, F., González-Lois, C., Jimeno, M., Jurado, I., Machado, I., Martínez-Ciarpaglini, C., Musulen, E., Naranjo, D., Papaleo, N., Peña, C., Rosiñol, Ò., … EpiT1 Consortium (2025). Interobserver variability of histopathological assessment in pT1 colorectal carcinoma. *Histopathology*, https://doi.org/10.1111/his.70043

44. Furness, P. N., Taub, N., Assmann, K. J. M., Banfi, G., Cosyns, J.-P., Dorman, A. M., Hill, C. M., Kapper, S. K., Waldherr, R., Laurinavicius, A., Marcussen, N., Martins, A. P., Nogueira, M., Regele, H., Serón, D., Carrera, M., Sund, S., Taskinen, E. I., Paavonen, T., Tihomirova, T., & Rosenthal, R. (2003). International variation in histologic grading is large, and persistent efforts are needed to improve reproducibility. *American Journal of Surgical Pathology*, *27*(6), 805–815. https://doi.org/10.1097/00000478-200306000-00012

45. *Cancer protocol templates*. College of American Pathologists. (n.d.). https://www.cap.org/protocols-and-guidelines/cancer-reporting-tools/cancer-protocol-templates/ Accessed 27th December 2025.

46. Czapliński, M., Redlarski, G., Kowalski, P., Tojza, P. M., Sikorski, A., & Żak, A. (2025). An Overview of Existing Applications of Artificial Intelligence in Histopathological Diagnostics of Leukemias: A Scoping Review. *Electronics*, *14*(21), 4144. https://doi.org/10.3390/electronics14214144

47. Al-kuwari, H., Alshami, B., Al-Khinji, A., Haider, A., & Arsalan, M. (2025). Automated Detection and Grading of Renal Cell Carcinoma in Histopathological Images via Efficient Attention Transformer Network. *Medical Sciences*, *13*(4), 257. https://doi.org/10.3390/medsci13040257

48. Rashidi, H. H., Hanna, M. G., & Pantanowitz, L. (2025). Introducing an Essential 7-Part Artificial Intelligence Review Series: A Guided Journey Into the Future of Pathology and Medicine. *Modern pathology: an official journal of the United States and Canadian Academy of Pathology, Inc*, *38*(3), 100673. https://doi.org/10.1016/j.modpat.2024.100673