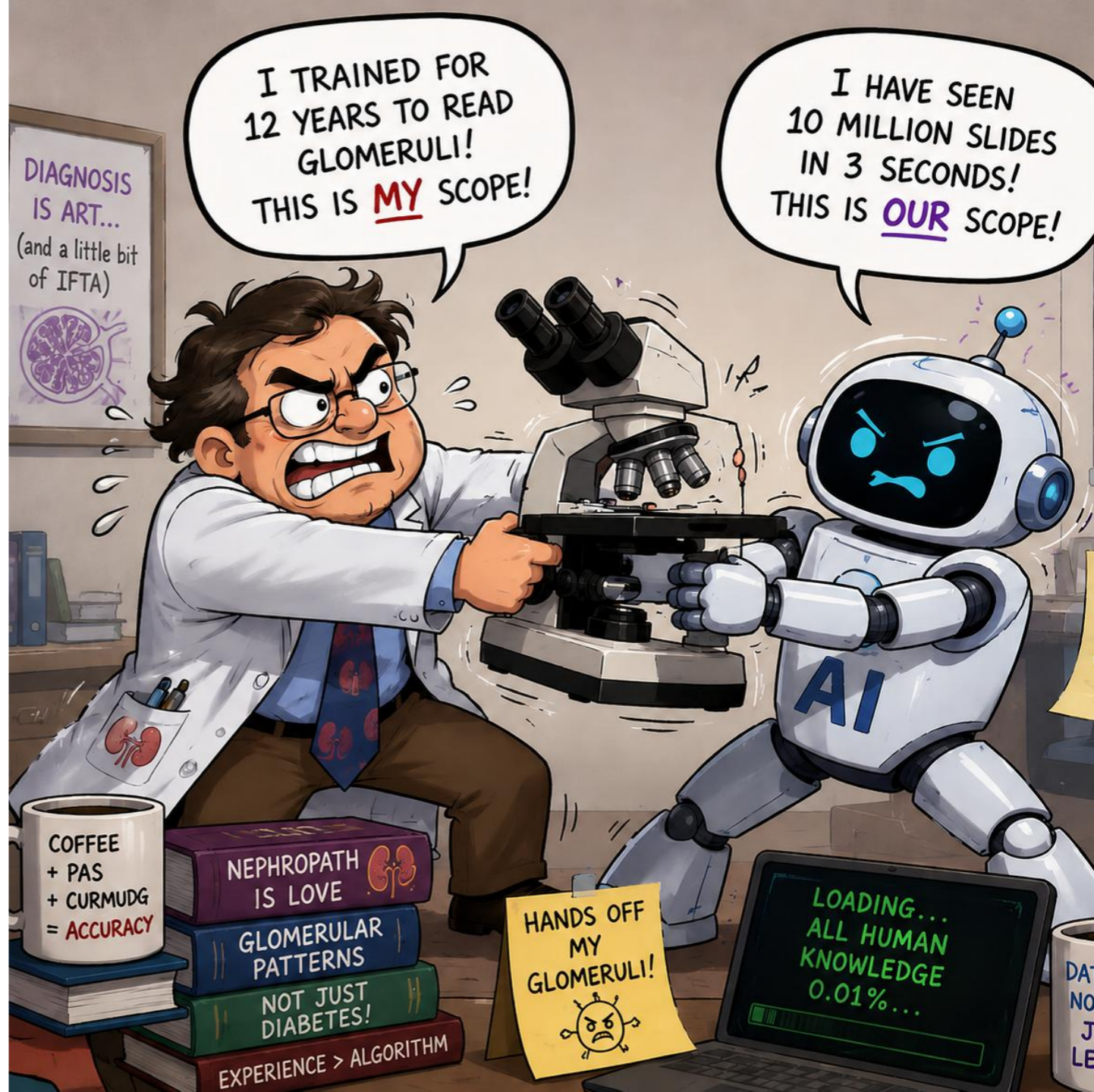




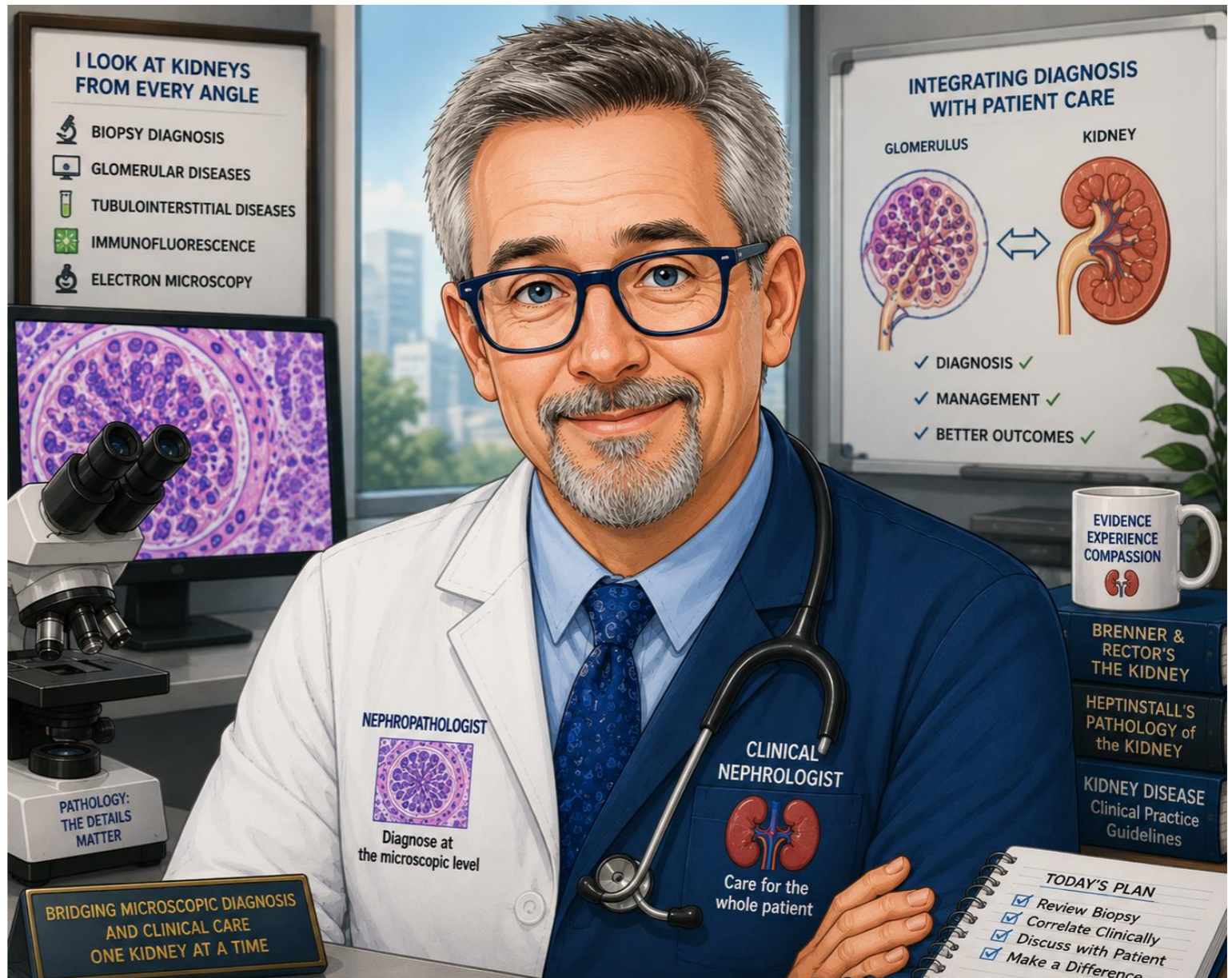
Με την εξάπλωση της AI
θα καταργηθούν οι (νεφρο)παθολογοανατόμοι;

ΝΑΙ

Π. Πατεινάκης
Νεφρολόγος, Δ/ντής ΕΣΥ
ΓΝΘ Παπαγεωργίου



Σύγκριση συμφερόντων





Αναζήτηση

Χρηστικό λεξικό

νοημοσύνη νο-η-μο-σύ-νη ουσ. (θηλ.) **1.** ΨΥΧΟΛ. -ΠΑΙΔΑΓ. ικανότητα του ατόμου

- να καταλαβαίνει και να διαχειρίζεται αφηρημένες έννοιες,
- να μαθαίνει,
- να αντιμετωπίζει νέες ή δύσκολες και πολύπλοκες καταστάσεις και
- να επιλύει προβλήματα, αξιοποιώντας προηγούμενες εμπειρίες

<https://christikolexiko.academyofathens.gr/index.php/anazitisi?>

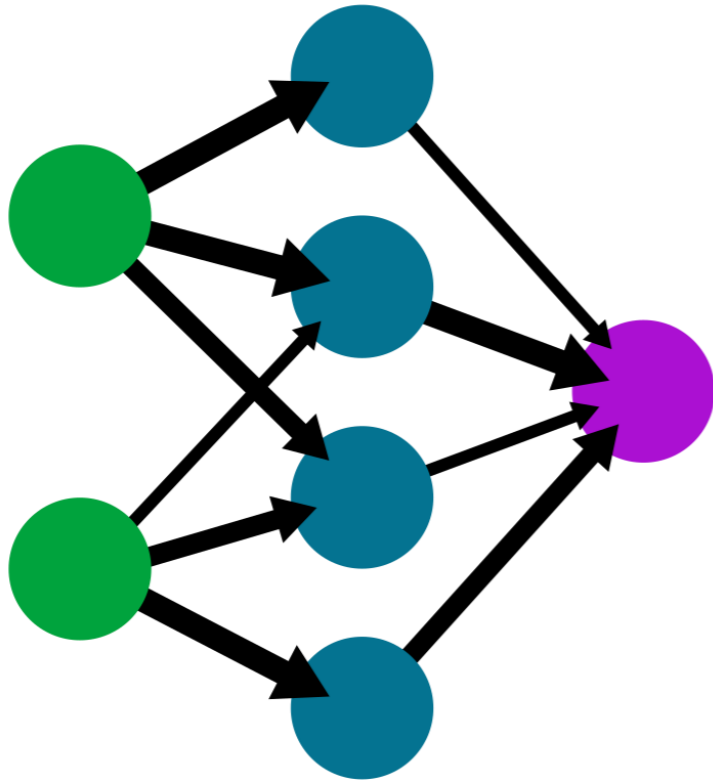
Τεχνητή νοημοσύνη



- Ο όρος **τεχνητή νοημοσύνη** αναφέρεται στον κλάδο της πληροφορικής ο οποίος ασχολείται με τη σχεδίαση και την υλοποίηση υπολογιστικών συστημάτων που μιμούνται στοιχεία της ανθρώπινης συμπεριφοράς τα οποία υπονοούν έστω και στοιχειώδη ευφυΐα: μάθηση, προσαρμοστικότητα, εξαγωγή συμπερασμάτων, κατανόηση από συμφραζόμενα, επίλυση προβλημάτων κλπ

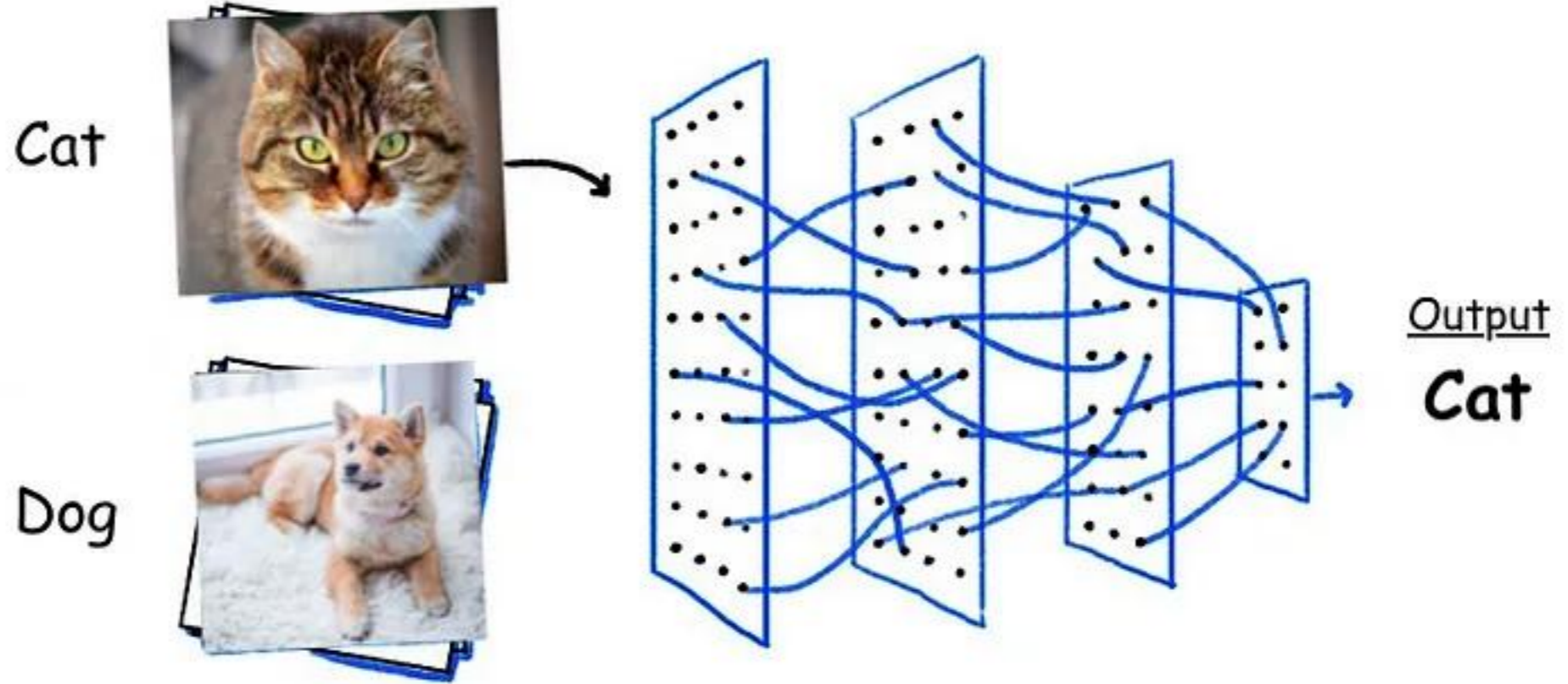
Νευρωνικά δίκτυα και βαθιά μάθηση

A simple neural network
input layer hidden layer output layer



- Το νευρωνικό δίκτυο είναι ένα δίκτυο από απλούς υπολογιστικούς κόμβους (νευρώνες, νευρώνια), διασυνδεδεμένους μεταξύ τους.
- Είναι εμπνευσμένο από το Κεντρικό Νευρικό Σύστημα (ΚΝΣ), το οποίο προσπαθεί να προσομοιώσει.

Deep Learning





Iran war heralds era of AI-powered bombing quicker than 'speed of thought'

Speed and scale of US military's AI war planning raises fears human decision-making may be sidelined



Academics say AI is collapsing the time required for military decision-making. Photograph: Majid Asgaripour/Reuters



The use of AI tools to enable attacks on **Iran** heralds a new era of bombing quicker than “the speed of thought”, experts have said, amid fears human - decision-makers could be sidelined.

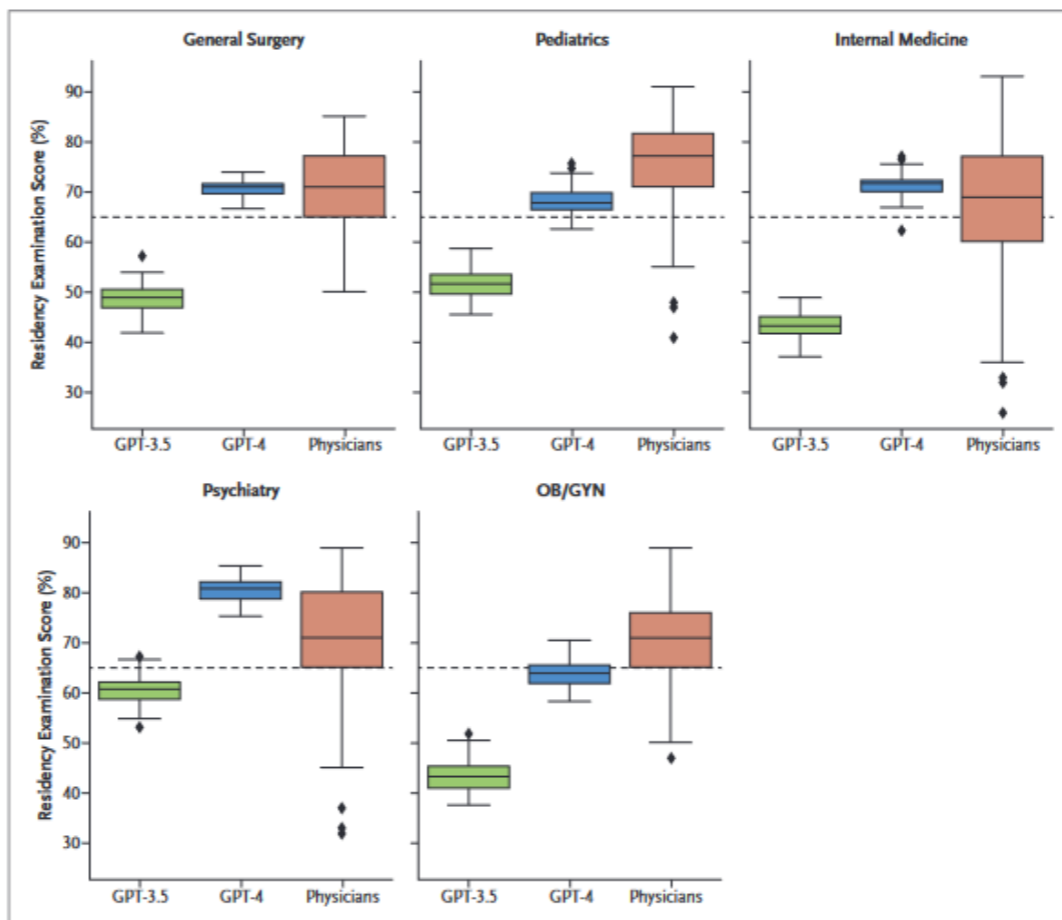
The AI Revolution in Medicine

Understanding How AI Can Advance Patient Care

DATASETS, BENCHMARKS, AND PROTOCOLS

GPT versus Resident Physicians — A Benchmark Based on Official Board Scores

Uriel Katz , M.D.,¹ Eran Cohen , M.D.,^{2,3} Eliya Shachar , M.D.,^{2,4} Jonathan Somer , B.Sc.,⁵ Adam Fink , M.D.,⁶ Eli Morse , M.D.,⁷ Beki Shreiber , B.Sc.,⁸ and Ido Wolf , M.D.^{2,3,4}





OPEN Predicting sex from retinal fundus photographs using automated deep learning

Edward Korot¹, Nikolas Pontikos¹, Xiaoxuan Liu^{1,2,3}, Siegfried K. Wagner¹, Livia Faes^{1,4}, Josef Huemer^{1,5}, Konstantinos Balaskas¹, Alastair K. Denniston^{1,2,3,6}, Anthony Khawaja^{1,4} & Pearse A. Keane^{1,4}

¹NIHR Biomedical Research Center at Moorfields Eye Hospital NHS Foundation Trust and UCL Institute of Ophthalmology, London, UK. ²Department of Ophthalmology, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK. ³Academic Unit of Ophthalmology, Institute of Inflammation & Ageing, College of Medical and Dental Sciences, University of Birmingham, Birmingham, UK. ⁴Eye Clinic, Cantonal Hospital of Lucerne, Lucerne, Switzerland. ⁵Vienna Institute for Research in Ocular Surgery, A Karl Landsteiner Institute, Hanusch Hospital, Vienna, Austria. ⁶Health Data Research UK, London, UK. ✉email: anthony.khawaja@nhs.net; pearse.keane1@nhs.net

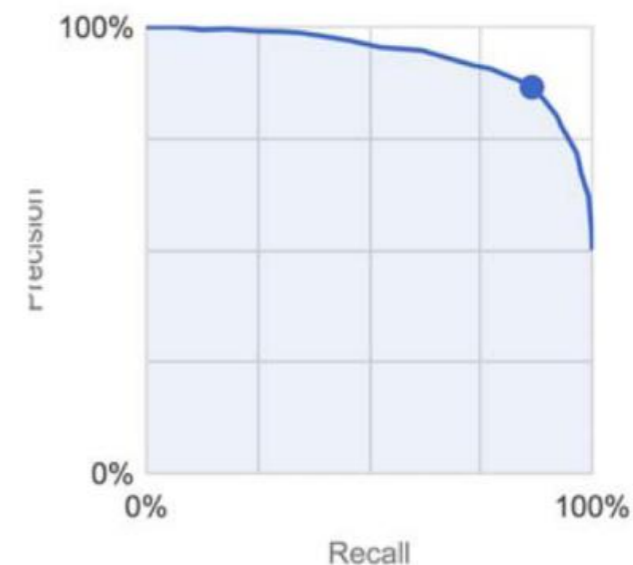
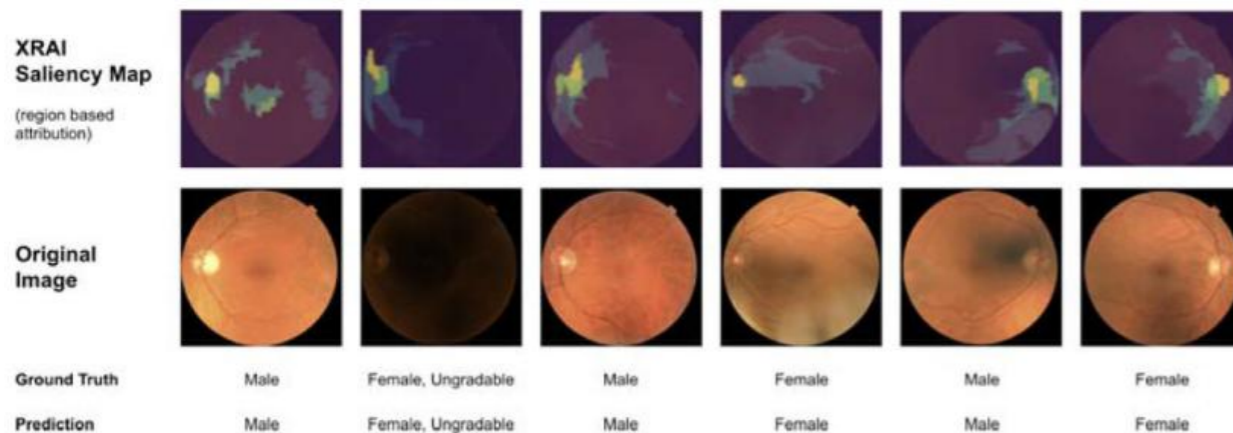


Figure 1. Precision-recall curve.

“Clinicians are currently unaware of distinct retinal feature variations between males and females”

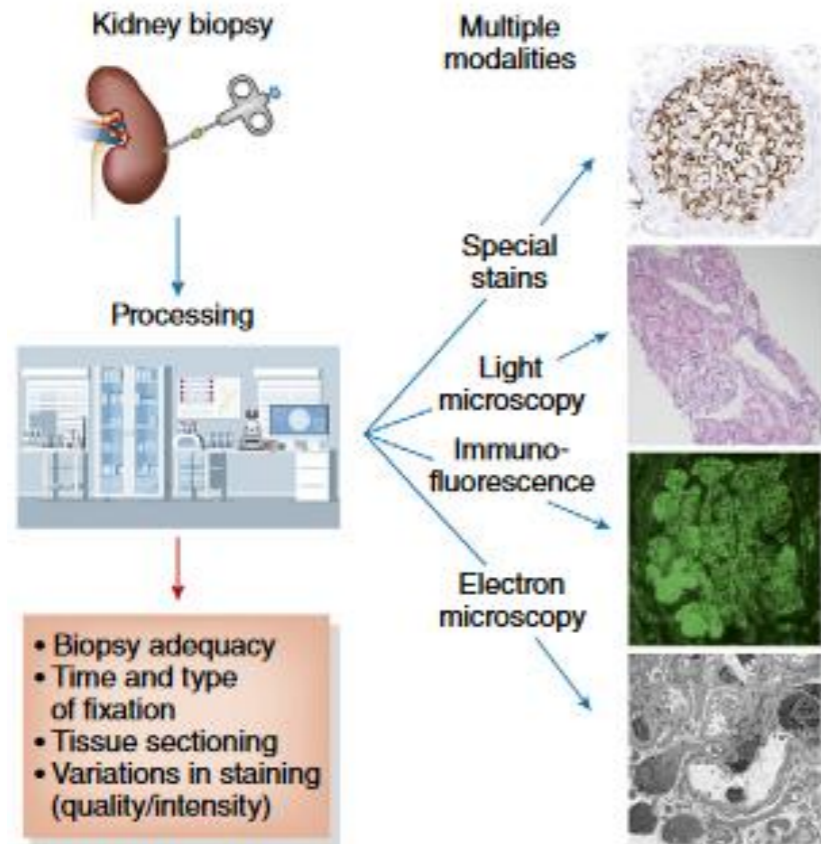


Artificial Intelligence

The Next Frontier in Kidney Biopsy Evaluation

Jean Hou and Cynthia C. Nast

CJASN 15: 1389–1391, 2020. doi: <https://doi.org/10.2215/CJN.13450820>

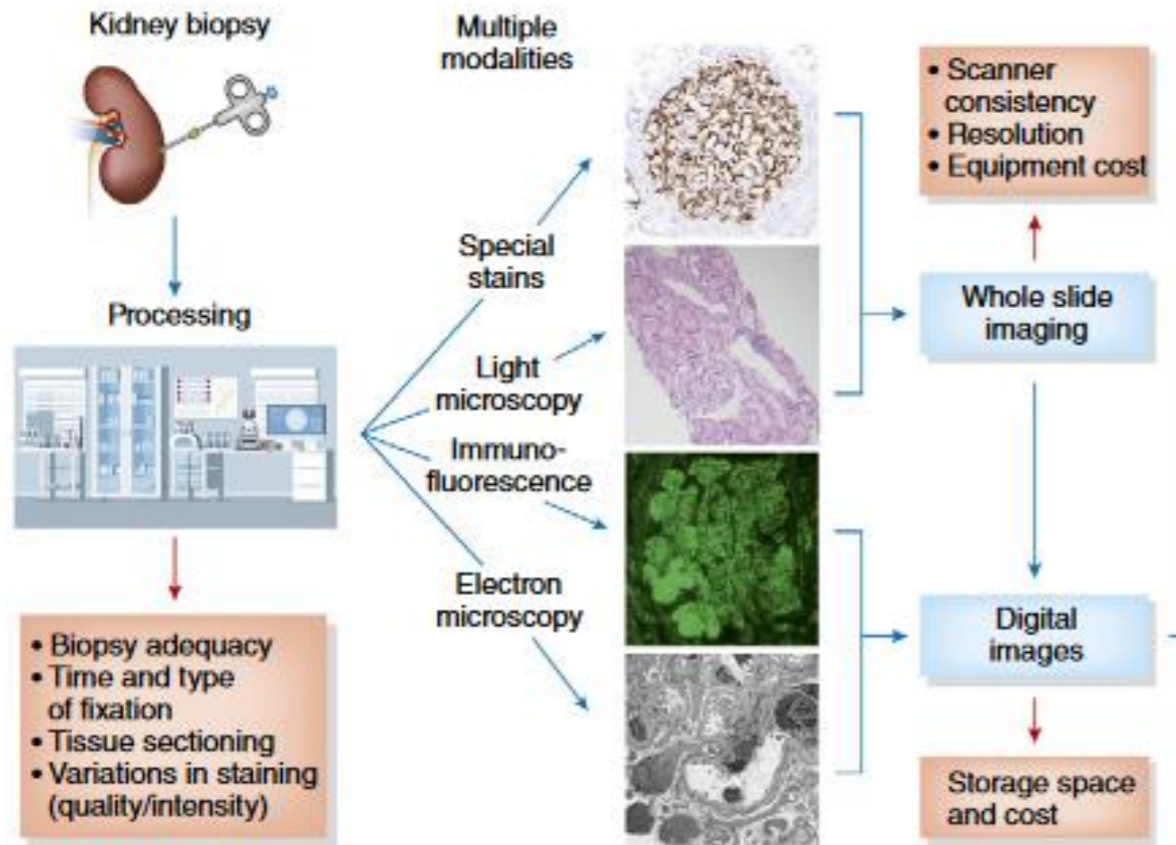


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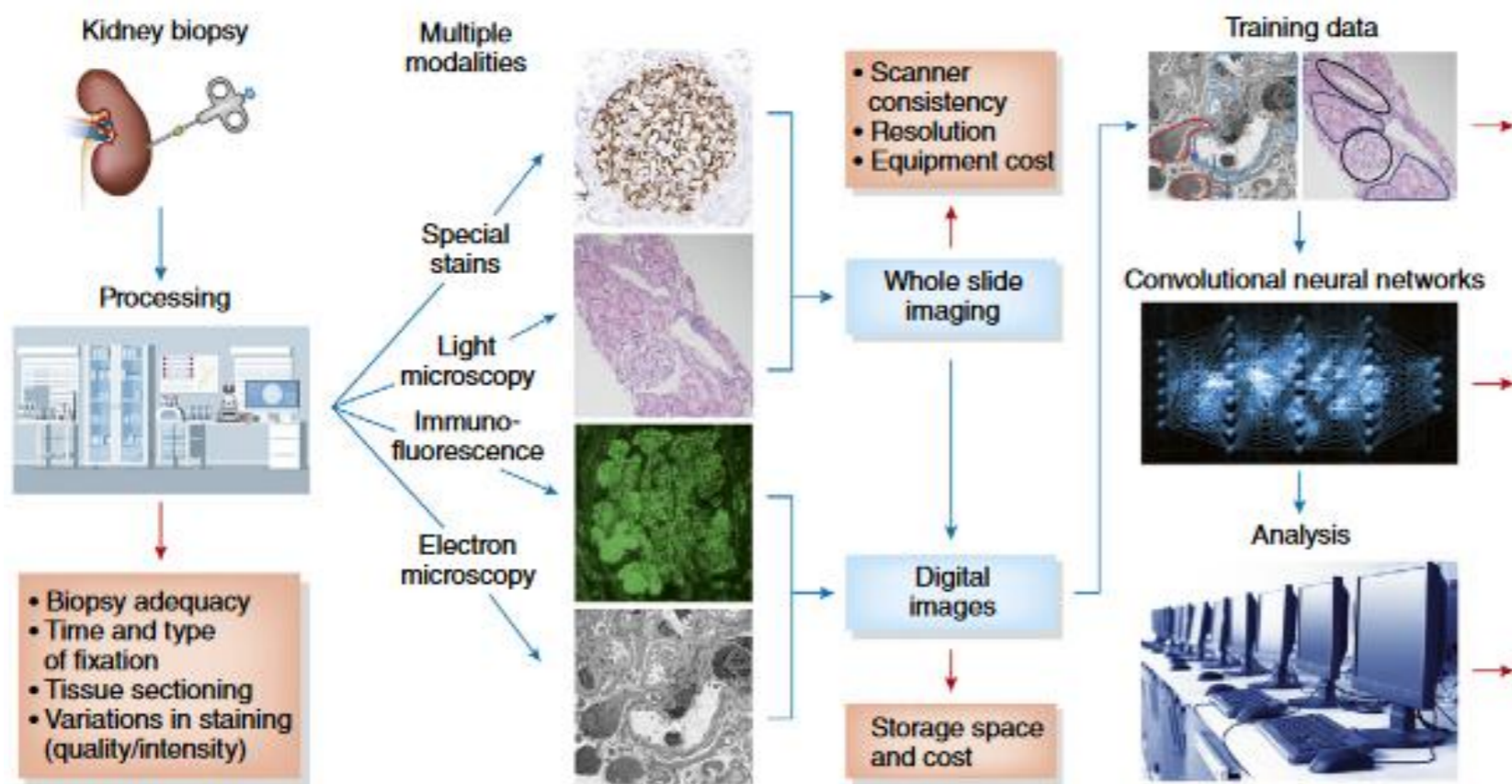


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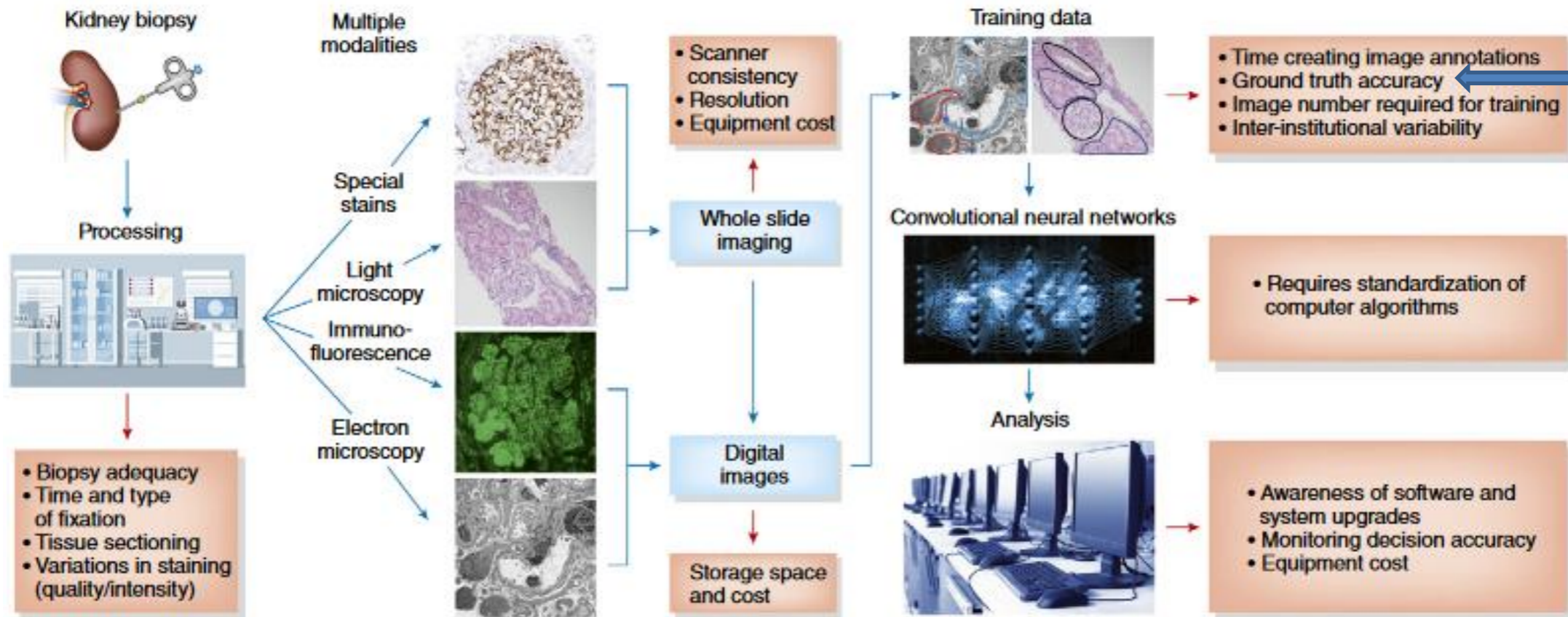


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ΔΥΝΗΤΙΚΑ ΠΛΕΟΝΕΚΤΗΜΑΤΑ



AI generated image

ακρίβεια,

αναπαραγωγιμότητα

αναγνώριση νέων μορφολογικών χαρακτηριστικών

ταχύτητα

Περισσότερες διαγνωστικές πληροφορίες

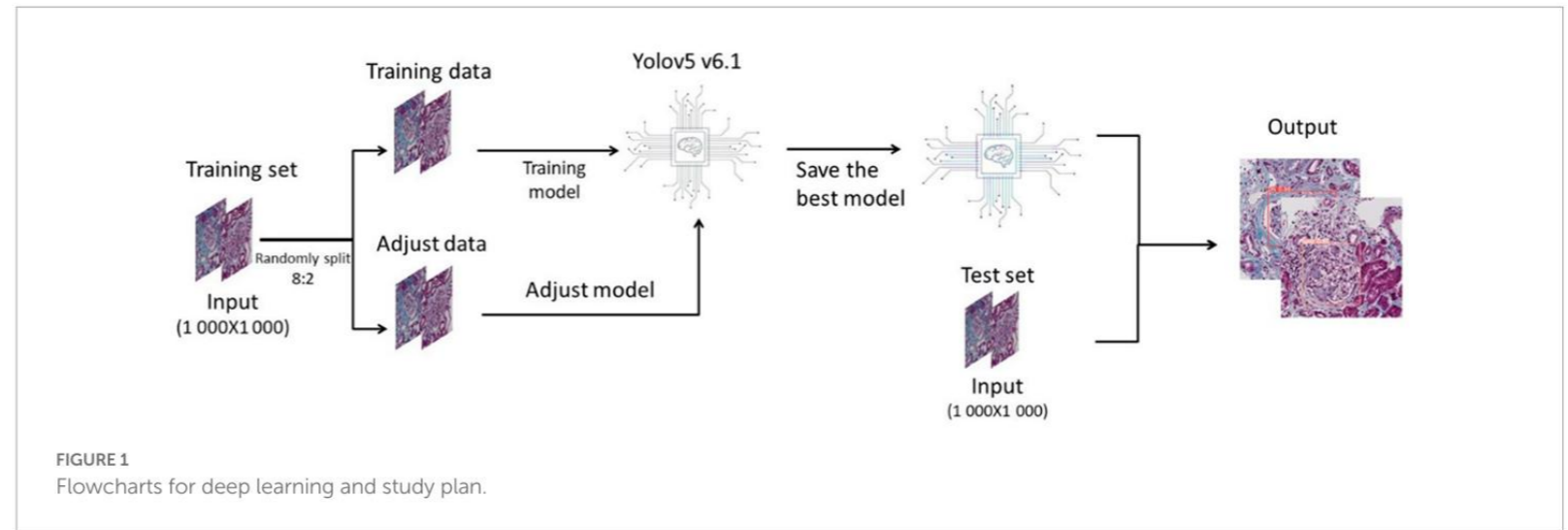
Νέοι προγνωστικοί δείκτες?

Νέοι θεραπευτικοί στόχοι?



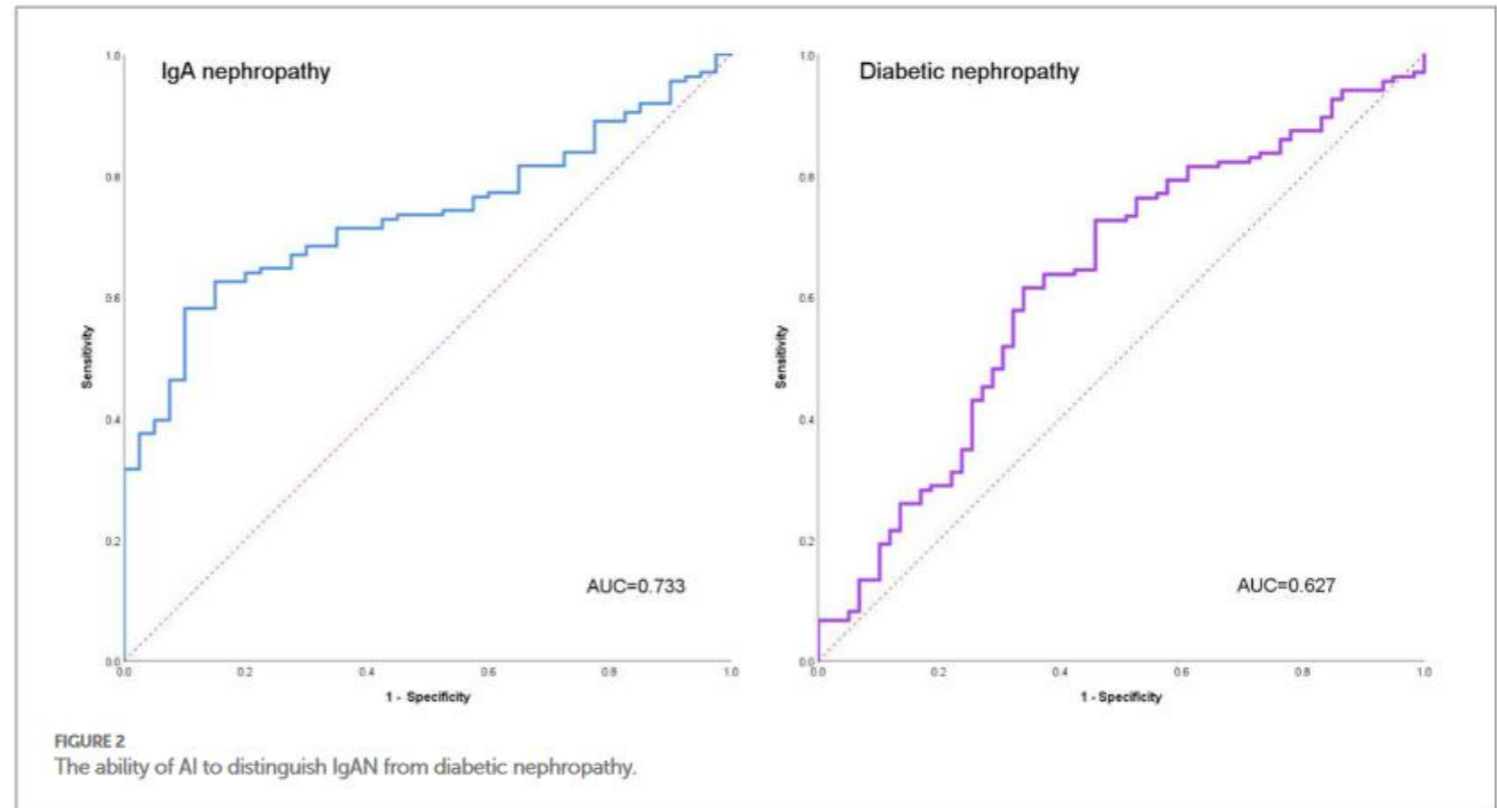
Artificial intelligence can accurately distinguish IgA nephropathy from diabetic nephropathy under Masson staining and becomes an important assistant for renal pathologists

Zhenliang Fan^{1,2†}, Qiaorui Yang^{3†}, Hong Xia¹, Peipei Zhang¹, Ke Sun⁴, Mengfan Yang⁵, Riping Yin⁶, Dongxue Zhao³, Hongzhen Ma¹, Yiwei Shen⁷ and Junfen Fan^{*†}



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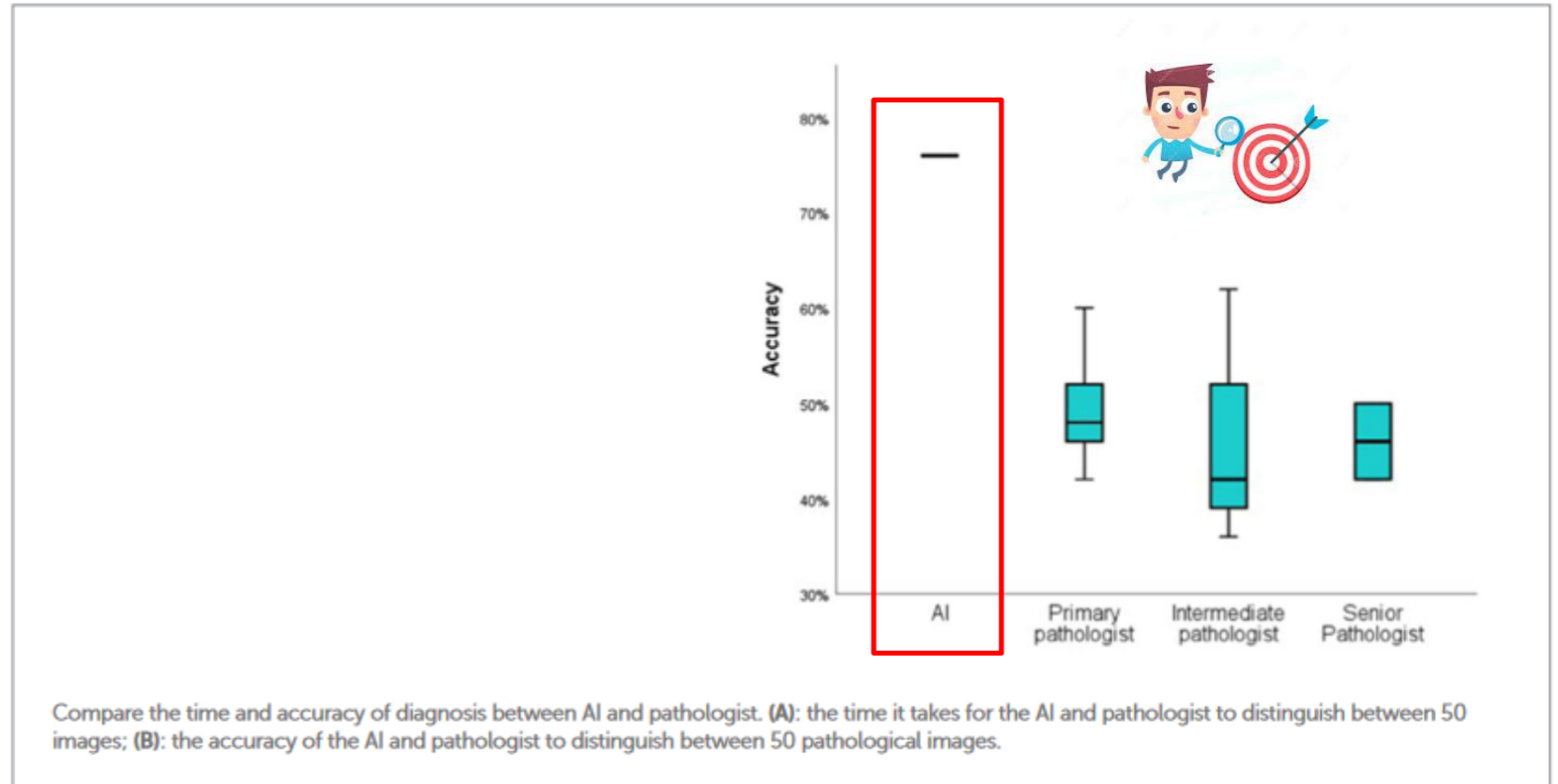
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2023

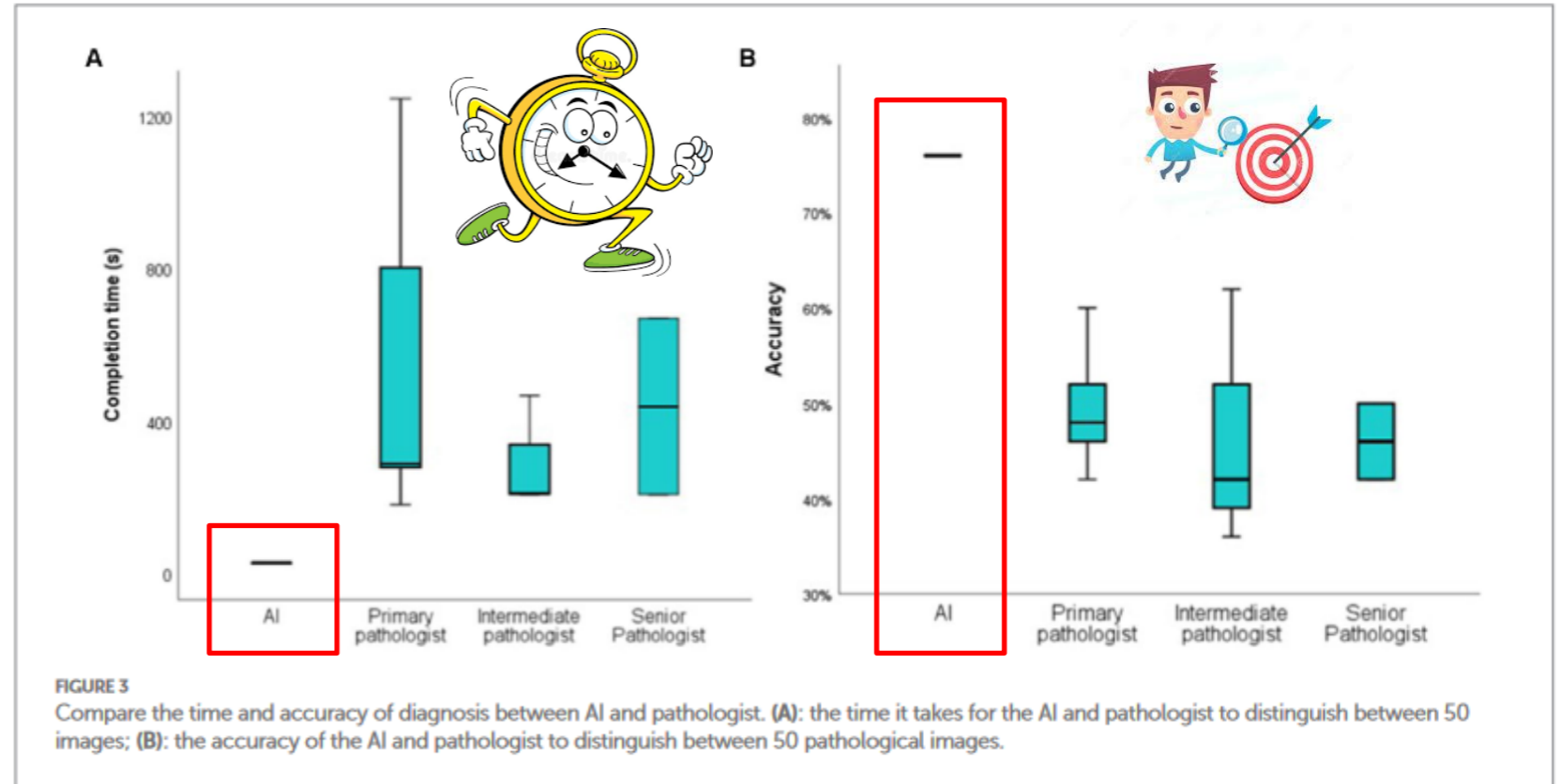
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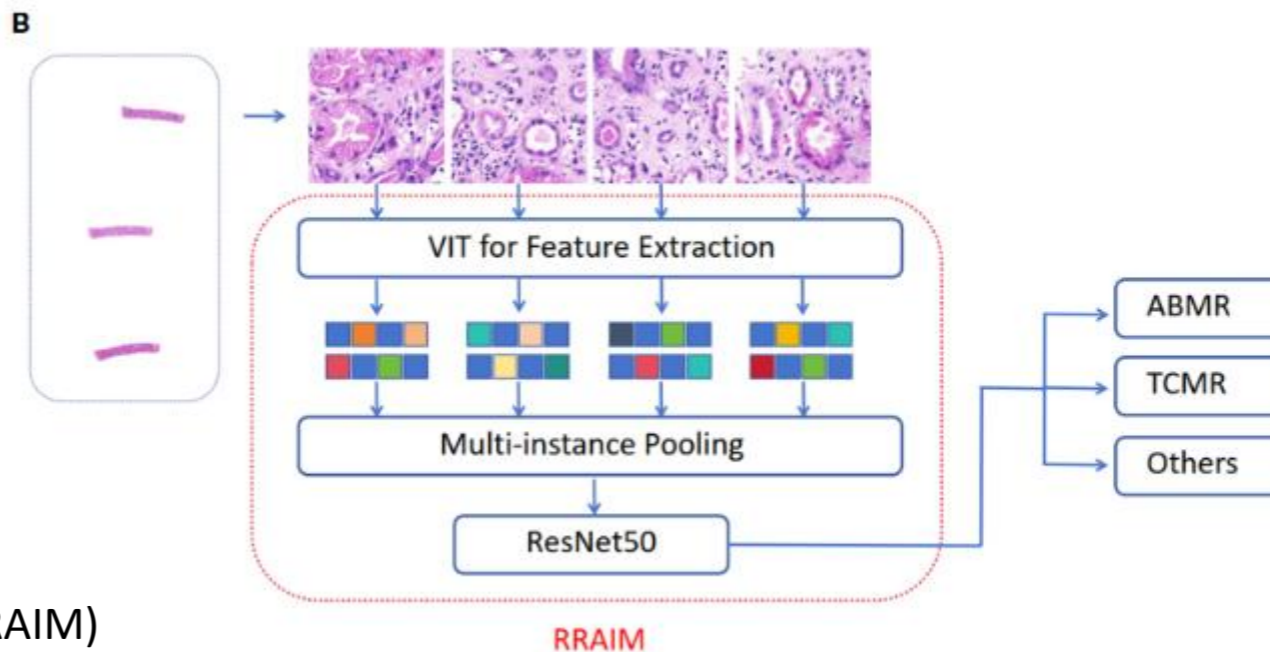
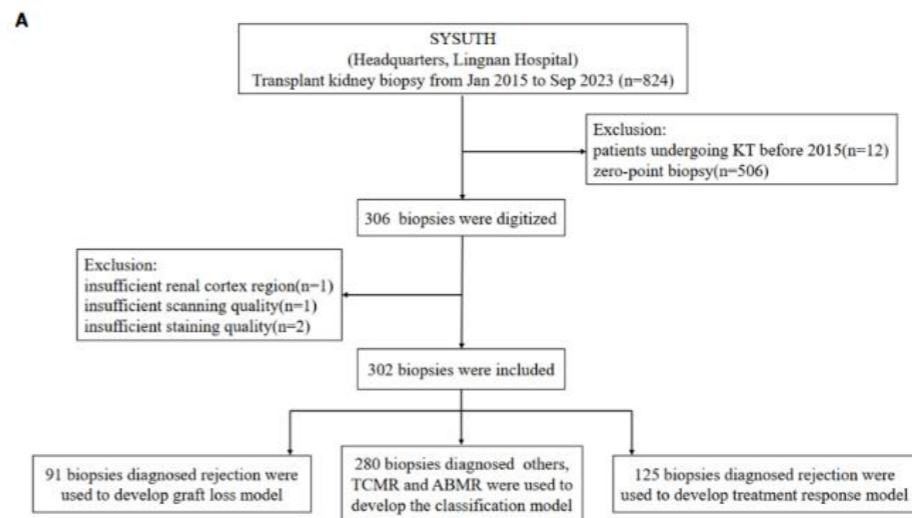
2023

Zhenliang Fan^{1,2†}, Qiaorui Yang^{3†}, Hong Xia¹, Peipei Zhang¹, Ke Sun⁴, Mengfan Yang⁵, Riping Yin⁶, Dongxue Zhao³, Hongzhen Ma¹, Yiwei Shen⁷ and Junfen Fan^{*k}

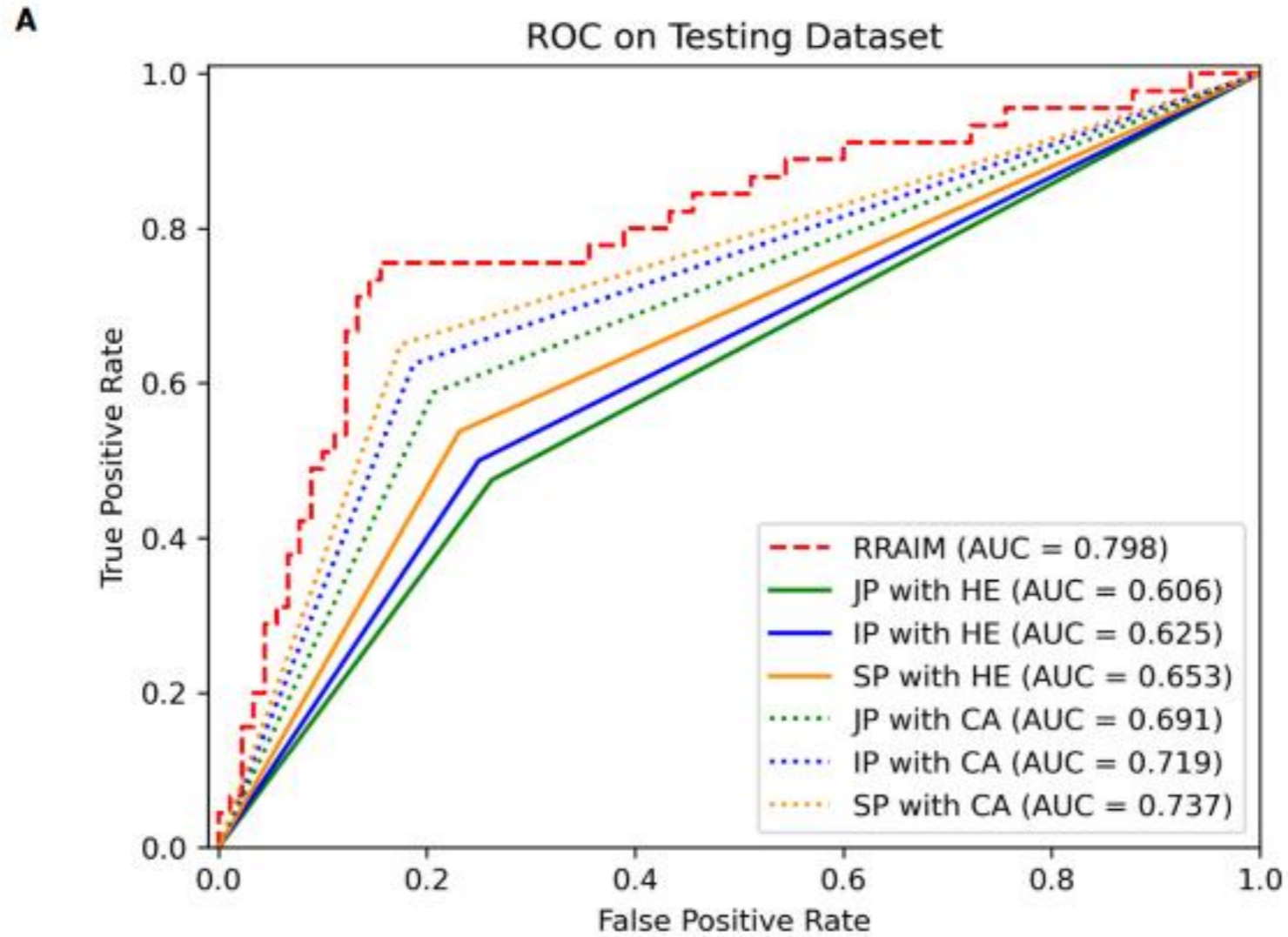


Deep learning-enabled classification of kidney allograft rejection on whole slide histopathologic images

Yongrong Ye^{1†}, Liubing Xia^{1†}, Shicong Yang^{2†}, You Luo¹,
 Zuofu Tang¹, Yuanqing Li^{3,4}, Lanqing Han⁵, Hanbin Xie^{6*},
 Yong Ren^{7,8,9*} and Ning Na^{1*}



renal rejection artificial intelligence model (RRAIM)



«Moreover, RRAIM completes the diagnosis task
in less than 30 seconds per case»

RESEARCH

Open Access



Artificial intelligence assists identification and pathologic classification of glomerular lesions in patients with diabetic nephropathy


Qunjuan Lei¹, Xiaoshuai Hou², Xumeng Liu¹, Dongmei Liang¹, Yun Fan¹, Feng Xu¹, Shaoshan Liang¹, Dandan Liang¹, Jing Yang¹, Guotong Xie^{2,3,4*}, Zhihong Liu^{1*} and Caihong Zeng^{1*} 

Table 1. Glomerular classification of DN

Class	Description	Inclusion Criteria
I	Mild or nonspecific LM changes and EM-proven GBM thickening	Biopsy does not meet any of the criteria mentioned below for class II, III, or IV GBM > 395 nm in female and >430 nm in male individuals 9 years of age and older ^a
IIa	Mild mesangial expansion	Biopsy does not meet criteria for class III or IV Mild mesangial expansion in >25% of the observed mesangium
IIb	Severe mesangial expansion	Biopsy does not meet criteria for class III or IV Severe mesangial expansion in >25% of the observed mesangium
III	Nodular sclerosis (Kimmelstiel–Wilson lesion)	Biopsy does not meet criteria for class IV At least one convincing Kimmelstiel–Wilson lesion
IV	Advanced diabetic glomerulosclerosis	Global glomerular sclerosis in >50% of glomeruli Lesions from classes I through III

LM, light microscopy.

^aOn the basis of direct measurement of GBM width by EM, these individual cutoff levels may be considered indicative when other GBM measurements are used.

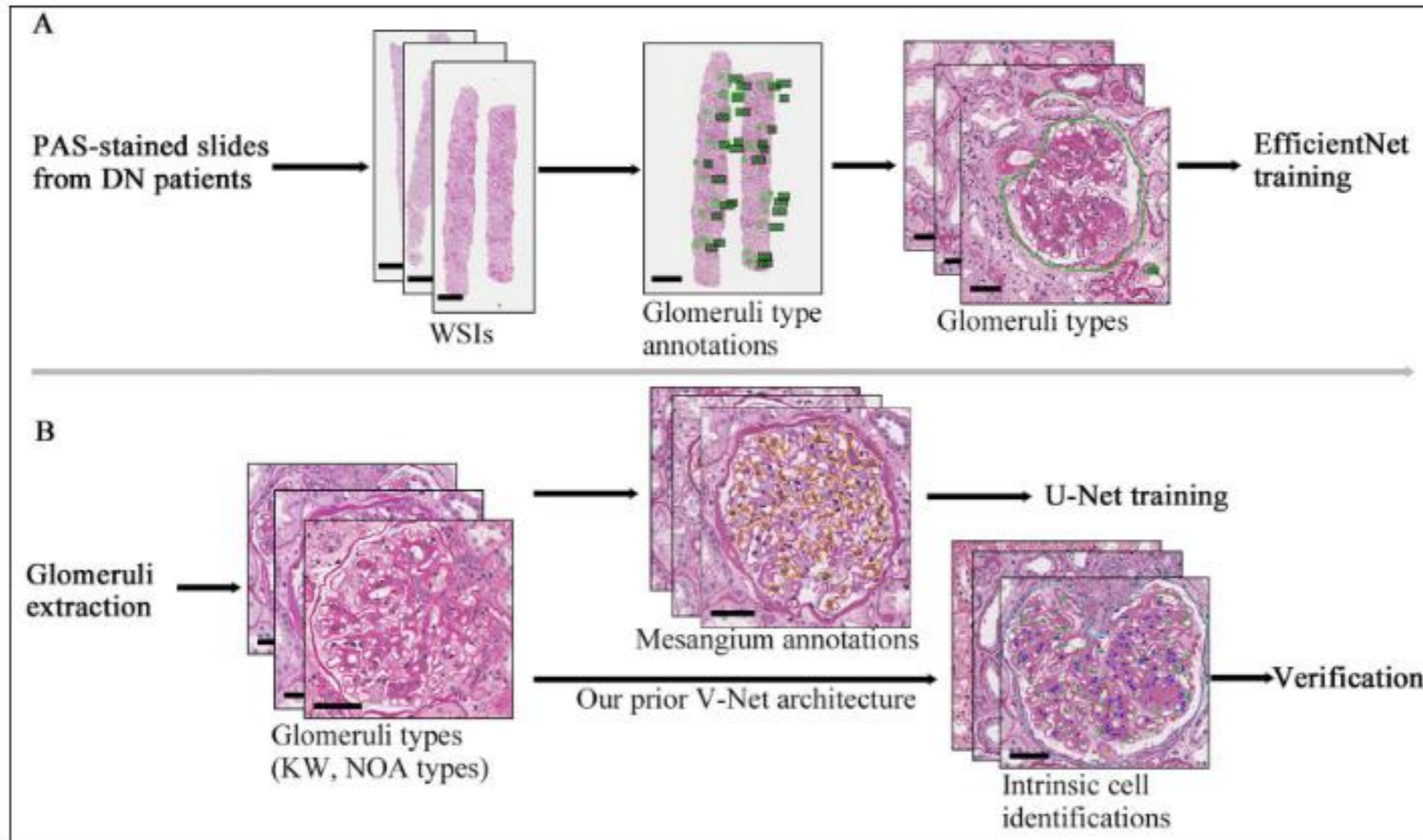


Fig. 1 Schematic illustration of our CNN model for identifying glomerular morphological features. **A** The training procedure of EfficientNet architecture for identifying different glomeruli types. **B** The training procedure of U-Net architecture for segmenting mesangial area and the verification procedure of our prior V-Net architecture for identifying three glomerular intrinsic cells. Scale bars mean 750 μm in WSIs and 50 μm in single glomerulus images.

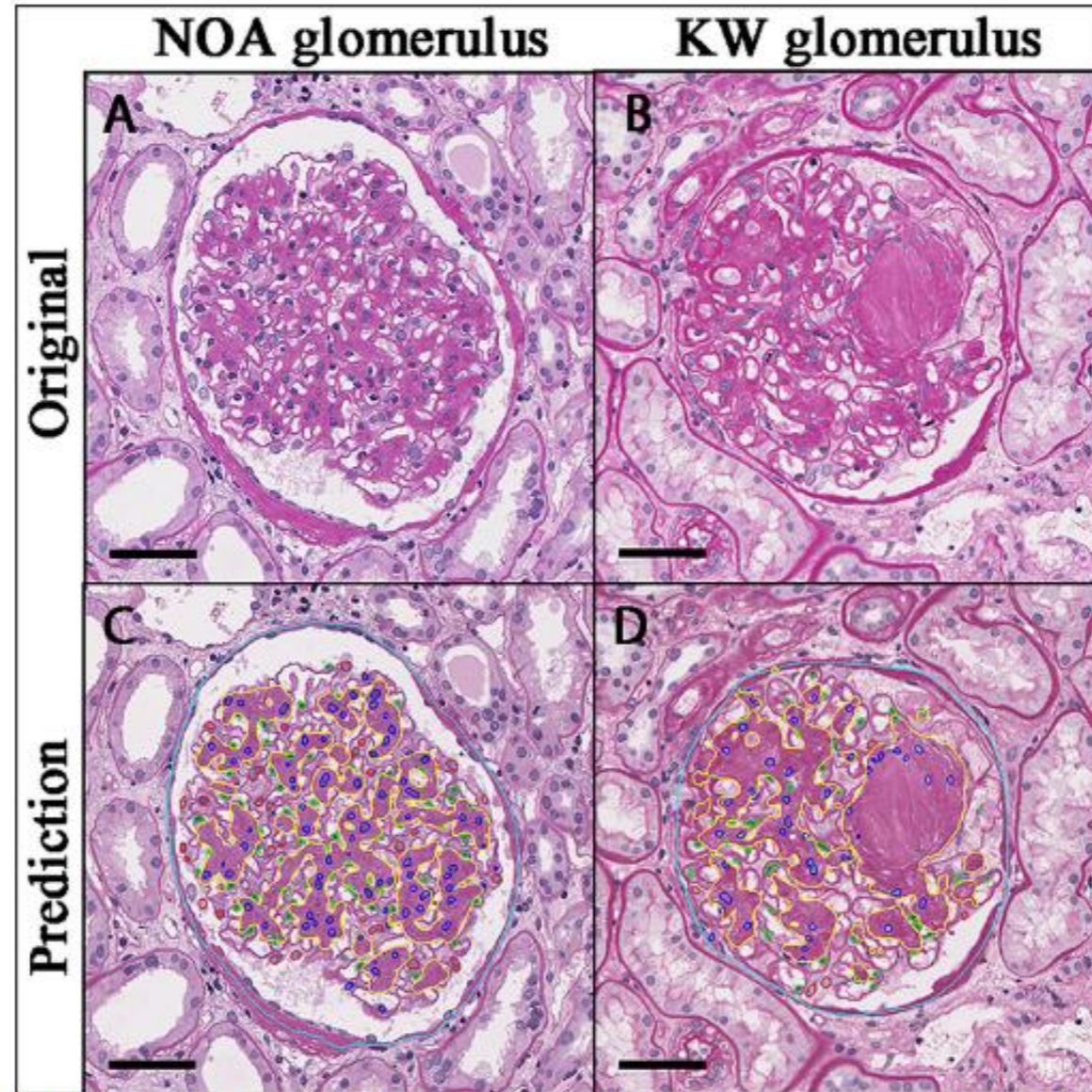


Fig. 2 Our CNN architectures identify intraglomerular features. Original images derived directly from WSI slides (**A** NOA glomerulus, **B** KW glomerulus). Prediction images (**C**, **D**) describe the predicted results of intraglomerular features from the original images (cyan: Bowman capsules, red: podocytes, blue: mesangial cells, green: endothelial cells, yellow: mesangial regions). Scale bar: 50 μ m

Table 1 Performance of our CNN model on identifying different glomeruli types

Glomeruli types	Accuracy	Specificity	Precision	Recall	F1-score
Global glomerulosclerosis	0.984	0.985	0.881	0.981	0.928
Segmental glomerulosclerosis	0.956	0.982	0.692	0.595	0.640
Kimmelstiel–Wilson lesions	0.988	0.997	0.979	0.929	0.953
None of the above	0.947	0.944	0.914	0.952	0.933

Table 2 Performance of the V-Net architecture from the ARPS system on identifying three glomerular intrinsic cells in patients with DN

Intrinsic cells	Accuracy	Specificity	Precision	Recall	F1-score
Mesangial cells	0.942	0.953	0.932	0.926	0.929
Endothelial cells	0.930	0.970	0.939	0.855	0.895
Podocytes	0.961	0.986	0.890	0.793	0.839

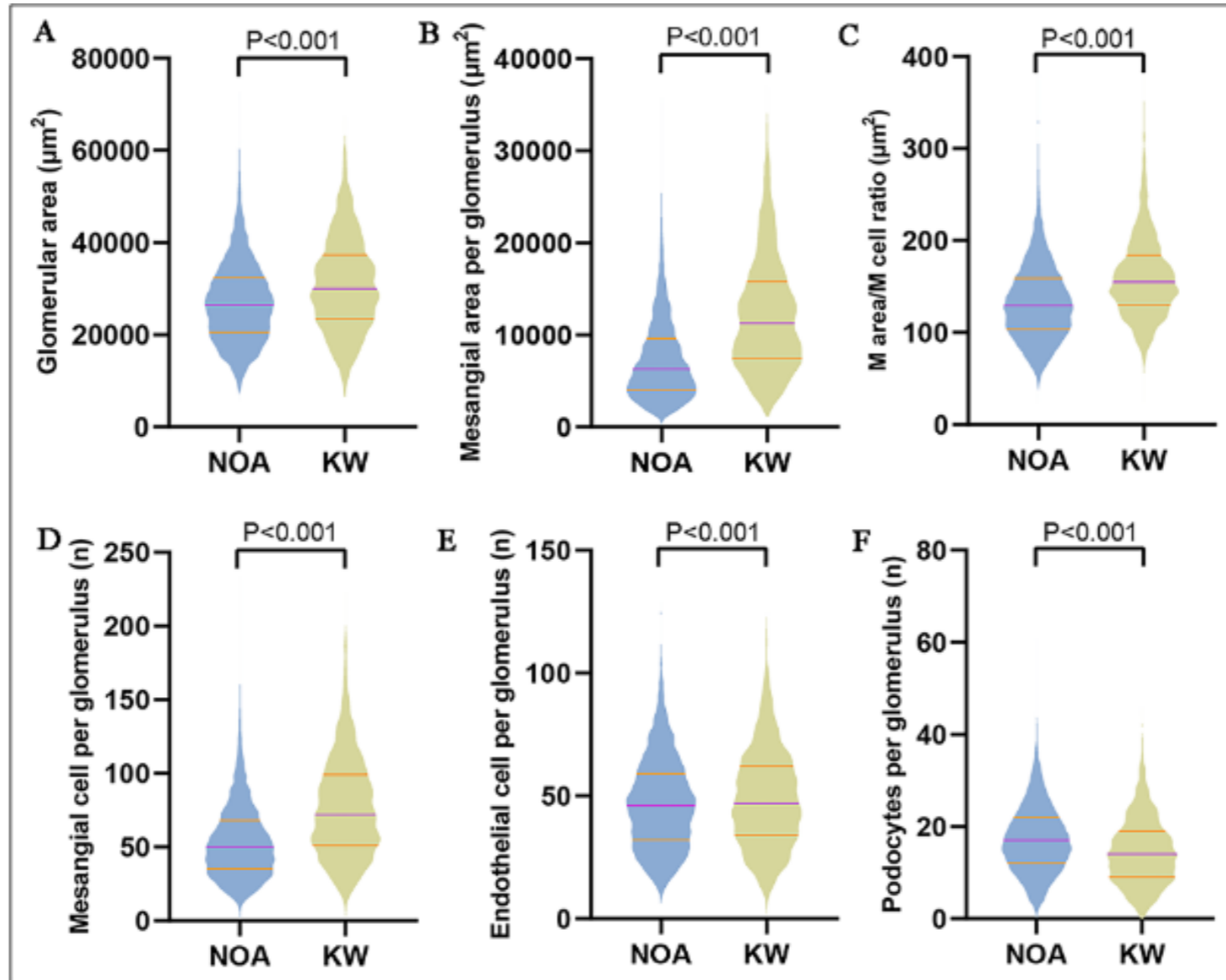


Fig. 3 Violin plots depict the distribution of each intraglomerular feature between NOA and KW glomeruli from a pooled 11,188 midsection glomerular image (NOA: 8181, KW: 3007). M: mesangial, E: endothelial, P: podocytes. The purple line: the median value, the orange line: the interquartile range. Inter-group comparisons were performed by Mann Whitney U test



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Extending the landscape of omics technologies by pathomics

[Roman D. Bülow](#), [David L. Hölscher](#), [Ivan G. Costa](#) & [Peter Boor](#) 

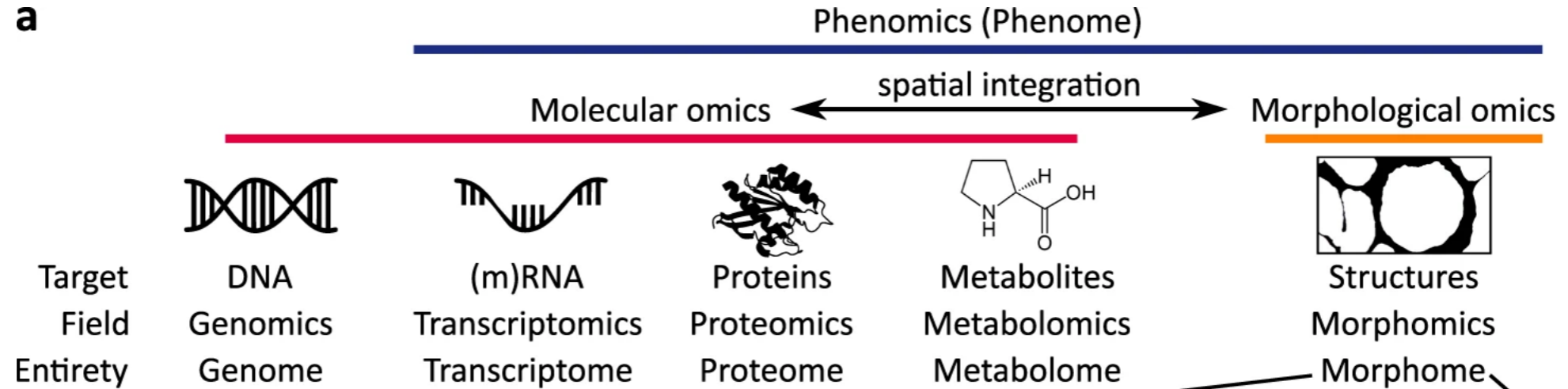
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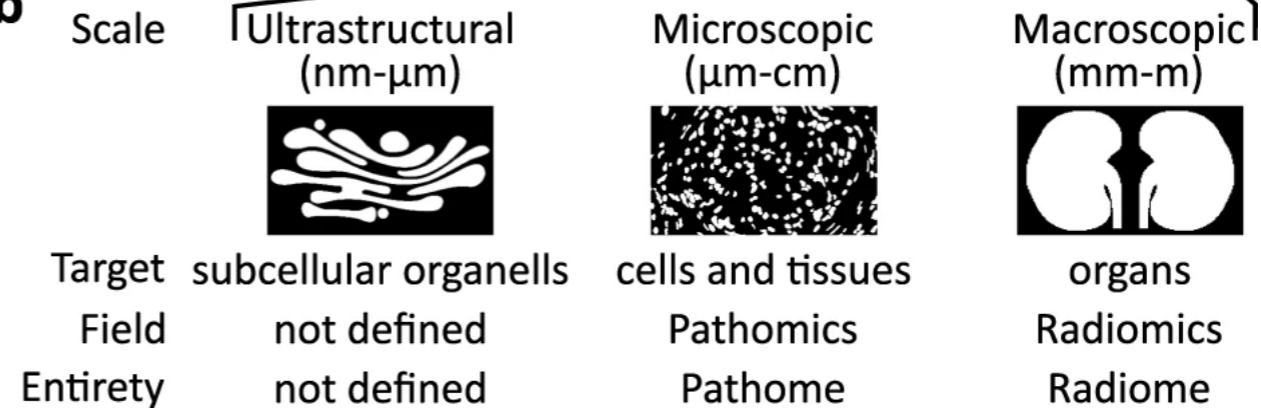
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a



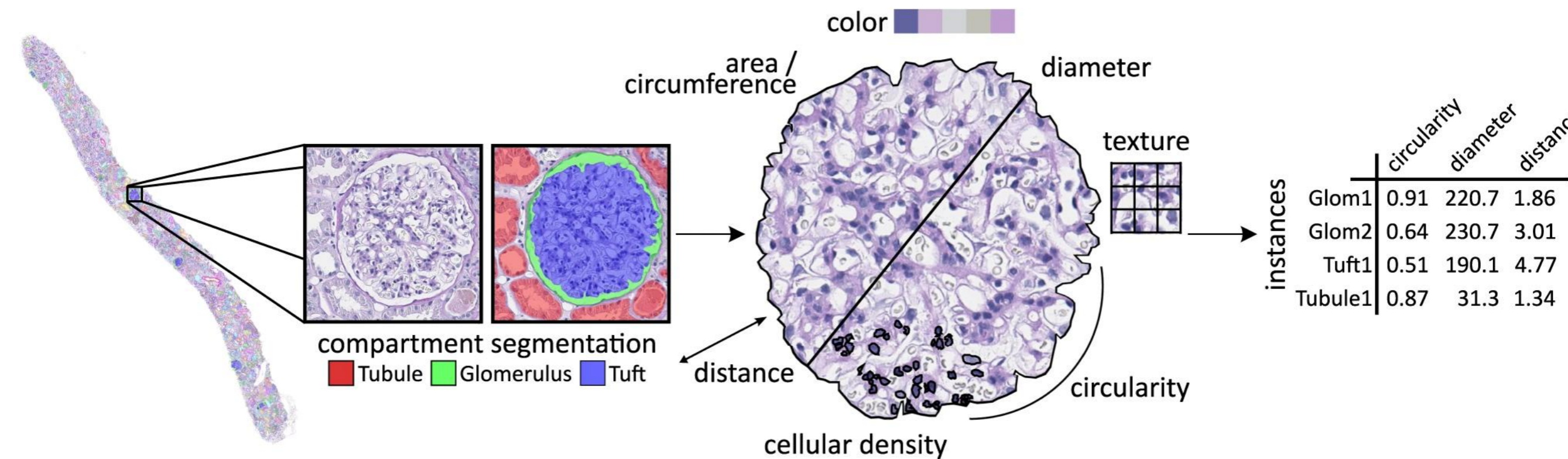
b



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Table 3 The consistency between the CNN-based and pathologist-based pathologic classes

Cohen's kappa value	CNN-based classes					
	Internal application subset			External application subset		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Pathologist 1	0.475	0.457	0.529	0.667	0.585	0.640
Pathologist 2	0.688	0.722	0.688	0.655	0.588	0.423
Pathologist 3	0.653	0.655	0.654	0.699	0.618	0.741
Total	0.604	0.587	0.624	0.663	0.592	0.602

Model 1 was the CNN-based classification derived from combination of percent GS, the presence of KW lesions with the optimal cutoff values of average mesangial area; Model 2 derived from combination of percent GS, the presence of KW lesions with the optimal cutoff values of mesangial area/mesangial cell ratio; Model 3 derived from combination of percent GS, the presence of KW lesions with the optimal cutoff values of mesangial area fraction

We also assessed the Cohen's kappa values **between three experienced pathologists in 65 cases**, which are **0.773** (pathologist 1-pathologist 2), **0.797** (pathologist 1-pathologist 3), and **0.754** (pathologist 2-pathologist 3), respectively.

Διαγνωστική ακρίβεια

It is really confusing!!!

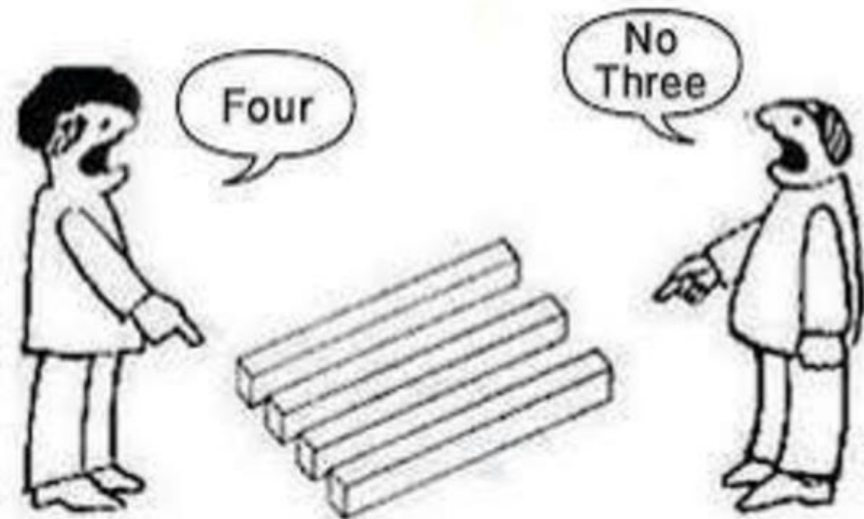


Table 1. Intraclass correlation and κ -values for all parameters

Parameter	Statistical Method	Overall ^a	Level of Experience	
			New or Moderate ^a (n=15)	High ^a (n=19)
Class III/IV	Intraclass correlation	0.39	0.33	0.43
Active and/or chronic	κ	0.36	0.27	0.38
Segmental or global	κ	0.39	0.27	0.46
Endocapillary proliferation	Intraclass correlation	0.46	0.42	0.50
Influx of inflammatory cells	Intraclass correlation	0.32	0.19	0.45
Swelling of endothelial cells	Intraclass correlation	0.46	0.40	0.52
Extracapillary proliferation	Intraclass correlation	0.57	0.42	0.71
Type of crescent	κ	0.46	0.40	0.55
Wire loops	Intraclass correlation	0.35	0.27	0.41

^aExcellent, >0.8; good, 0.6–0.8; moderate, 0.4–0.6; poor, <0.4.

Ιατρικές ειδικότητες που θα αντικατασταθούν από τεχνητή νοημοσύνη

Η πιο πιθανή εξέλιξη δεν είναι ότι η τεχνητή νοημοσύνη θα «αντικαταστήσει» πλήρως τους γιατρούς, αλλά ότι θα αυτοματοποιήσει μεγάλο μέρος της δουλειάς σε ορισμένες ειδικότητες. Οι πιο ευάλωτες είναι όσες βασίζονται:

- στην ανάλυση εικόνων ή δεδομένων,
- σε επαναλαμβανόμενες διαδικασίες,
- σε πρωτόκολλα,
- και λιγότερο στη χειρωνακτική παρέμβαση ή στη μακροχρόνια σχέση με ασθενείς.

Ιατρικές ειδικότητες που θα αντικατασταθούν απο τεχνητή νοημοσύνη

Ειδικότητες με τη μεγαλύτερη έκθεση στην AI

1. Ακτινολογία

Radiology

Η AI είναι ήδη πολύ καλή σε:

- ανάλυση ακτινογραφιών, CT, MRI,
- εντοπισμό όγκων ή αιμορραγιών,
- αυτόματη δημιουργία αναφορών,
- προτεραιοποίηση επειγόντων περιστατικών.



medical specialties most likely to be replaced by ai

2. Pathology

Pathology

Digital pathology is increasingly AI-friendly because tissue slides are image-based.

AI can:

- identify cancerous cells,
- quantify biomarkers,
- screen normal slides,
- reduce diagnostic workload.



medical specialties most likely to be replaced by ai

Human pathologists still matter for:

- rare diseases,
- nuanced interpretation,
- multidisciplinary tumor boards,
- legal responsibility.



MedFoundationHub: A Lightweight and Secure Toolkit for Deploying Medical Vision Language Foundation Models

Xiao Li^a, Yanfan Zhu^a, Ruining Deng^b, Wei-Qi Wei^c, Yu Wang^c, Shilin Zhao^c, Yaohong Wang^d, Haichun Yang^c, and Yuankai Huo^a


^aVanderbilt University, Nashville TN 37235, USA

^bWeill Cornell Medicine, New York, NY 10021, USA

^cVanderbilt University Medical Center, Nashville TN 37232, USA

^dUT MD Anderson Cancer Center, TX 77030, USA

✦ Επισκόπηση AI

Vision-Language Foundation Models (VLFMs or VLMs) are advanced AI systems trained on vast datasets of paired images and text. By merging a visual encoder with a Large Language Model (LLM), they map visual data and human language into a shared embedding space, allowing the AI to "see," describe, and interact with images or videos.  NVIDIA +1

ABSTRACT

Recent advances in medical vision-language models (VLMs) open up remarkable opportunities for clinical applications such as automated report generation, physician copilots, and uncertainty quantification. Despite their promise, medical VLMs raise serious security concerns. These include the risk of Protected Health Information (PHI) exposure, data leakage, and vulnerability to cyberthreats, concerns that are especially critical in hospital environments. Even when adopted for research or non-clinical purposes, healthcare organizations must exercise caution and implement safeguards. To address these challenges, we present MedFoundationHub, a graphical user interface (GUI) toolkit that: (1) enables physicians to manually select and use different models without programming expertise, (2) supports engineers in efficiently deploying medical VLMs in a plug-and-play fashion, with seamless integration of Hugging Face open-source models, and (3) ensures privacy-preserving inference through Docker-orchestrated, operating system agnostic deployment. MedFoundationHub requires only an offline local workstation equipped with a single NVIDIA A6000 GPU, making it both secure and accessible within the typical resources of academic research labs. To evaluate current capabilities, we engaged board-certified pathologists to deploy and assess five state-of-the-art VLMs (Google-MedGemma3-4B, Qwen2-VL-7B-Instruct, Qwen2.5-VL-7B-Instruct, and LLaVA-1.5-7B/13B). Expert evaluation covered colon cases and renal cases, yielding 1,015 clinician-model scoring events. These assessments revealed recurring limitations, including off-target answers, vague reasoning, and inconsistent pathology terminology.

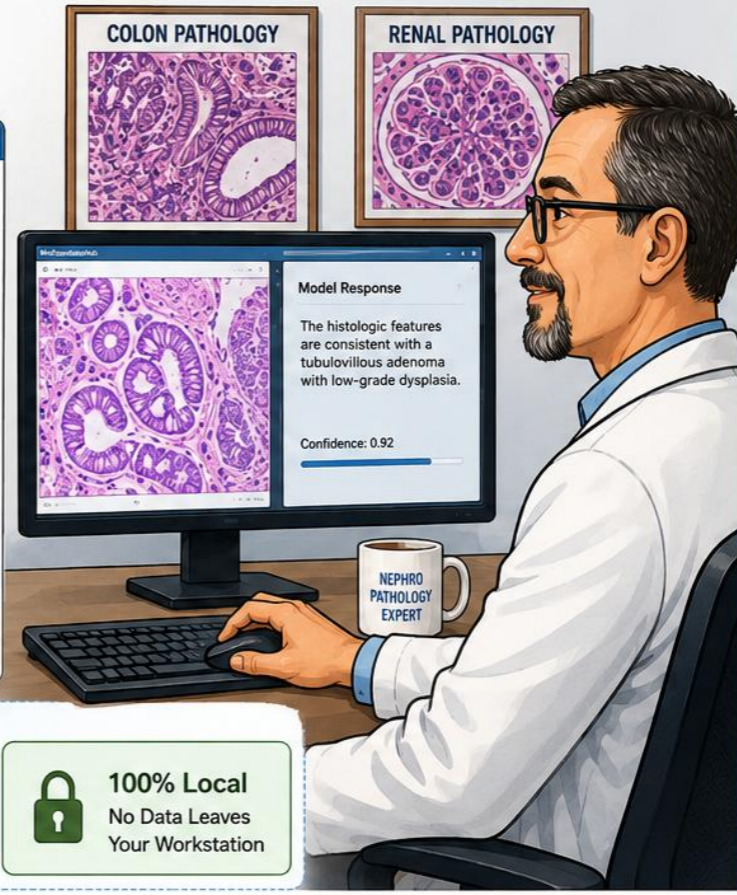
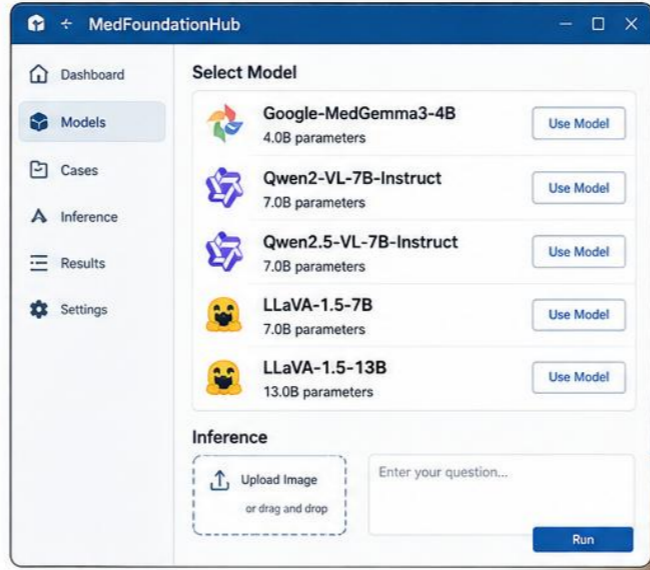
MedFoundationHub

A GUI toolkit for effortless deployment and evaluation of Medical Vision-Language Models

For Physicians
Manually select and use different models without programming expertise.

For Engineers
Efficiently deploy medical VLMs in a plug-and-play fashion, with seamless integration of Hugging Face open-source models.

Privacy-Preserving & Secure
Privacy-preserving inference through Docker-orchestrated, OS-agnostic deployment.



Expert Evaluation Study

Board-Certified Pathologists

Colon Cases

Renal Cases

Models Evaluated

- Google-MedGemma3-4B
- Qwen2-VL-7B-Instruct
- Qwen2.5-VL-7B-Instruct
- LLaVA-1.5-7B
- LLaVA-1.5-13B

Evaluation Summary

1,015

Clinician-Model Scoring Events

MedFoundationHub: A Lightweight and Secure Toolkit for Deploying Medical Vision Language Foundation Models

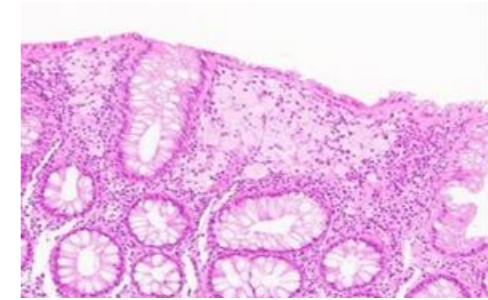
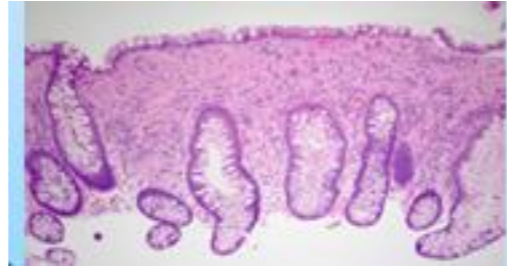
Xiao Li^a, Yanfan Zhu^a, Ruining Deng^b, Wei-Qi Wei^c, Yu Wang^c, Shilin Zhao^c, Yaohong Wang^d, Haichun Yang^c, and Yuankai Huo^a

^aVanderbilt University, Nashville TN 37235, USA

^bWeill Cornell Medicine, New York, NY 10021, USA

^cVanderbilt University Medical Center, Nashville TN 37232, USA

^dUT MD Anderson Cancer Center, TX 77030, USA



Score	Definition
0	No answer
1	Wrong answer
2	Partially correct answer
3	Correct answer with wrong reasoning
4	Correct answer with correct reasoning

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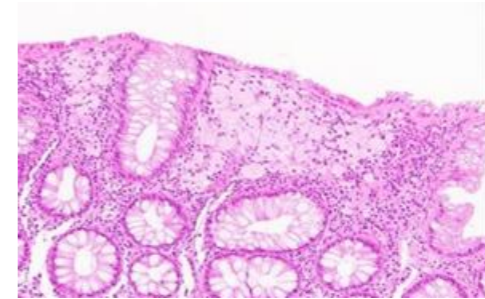
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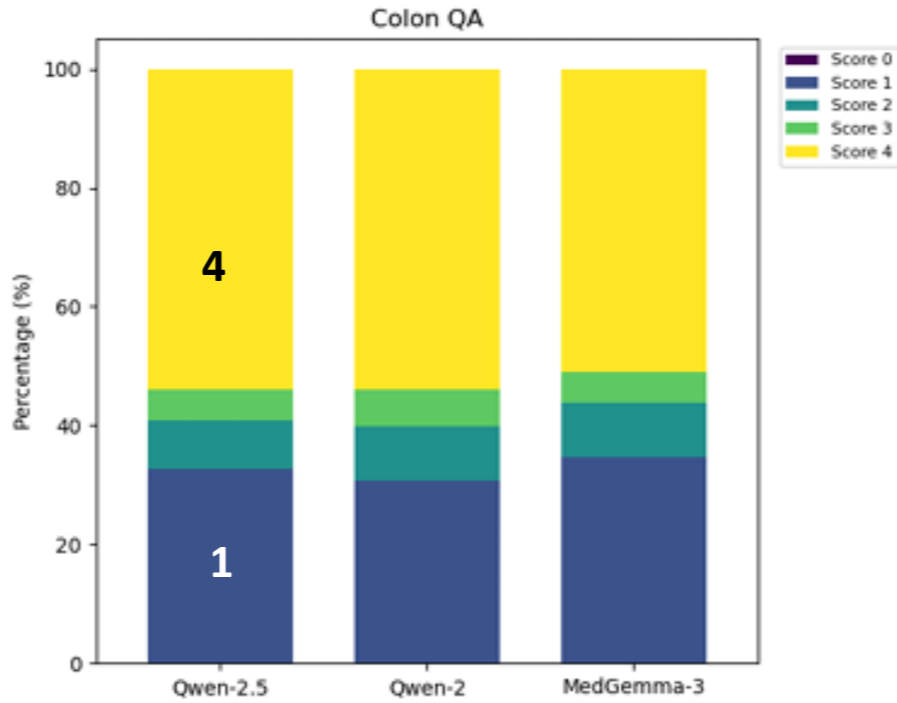
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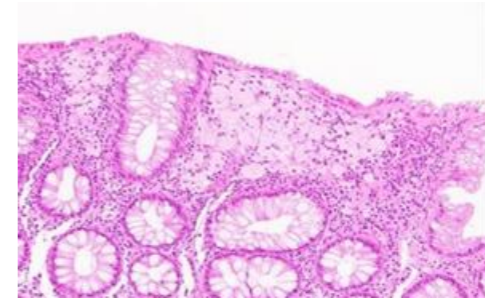
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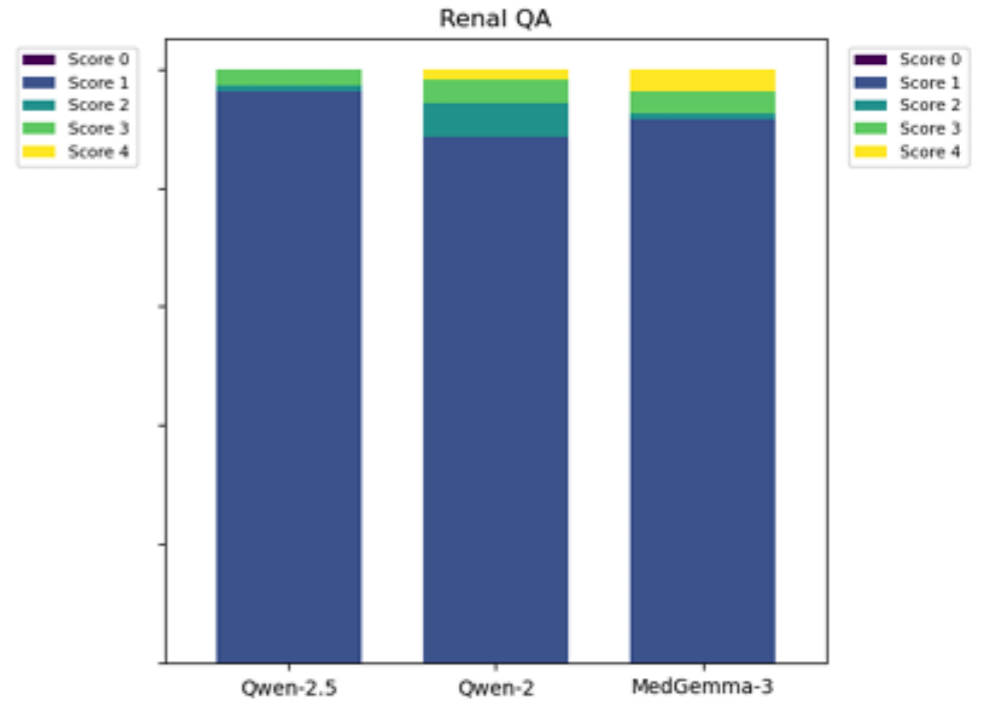
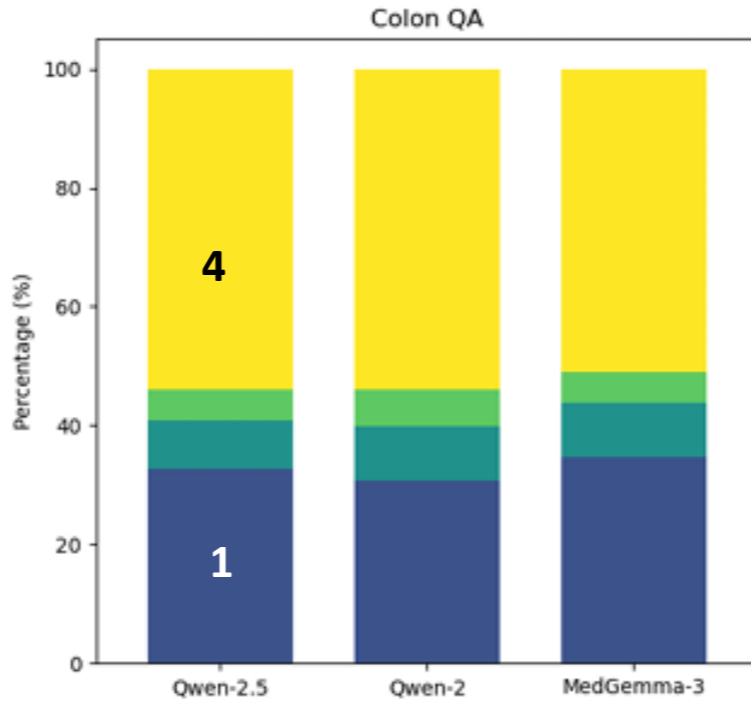
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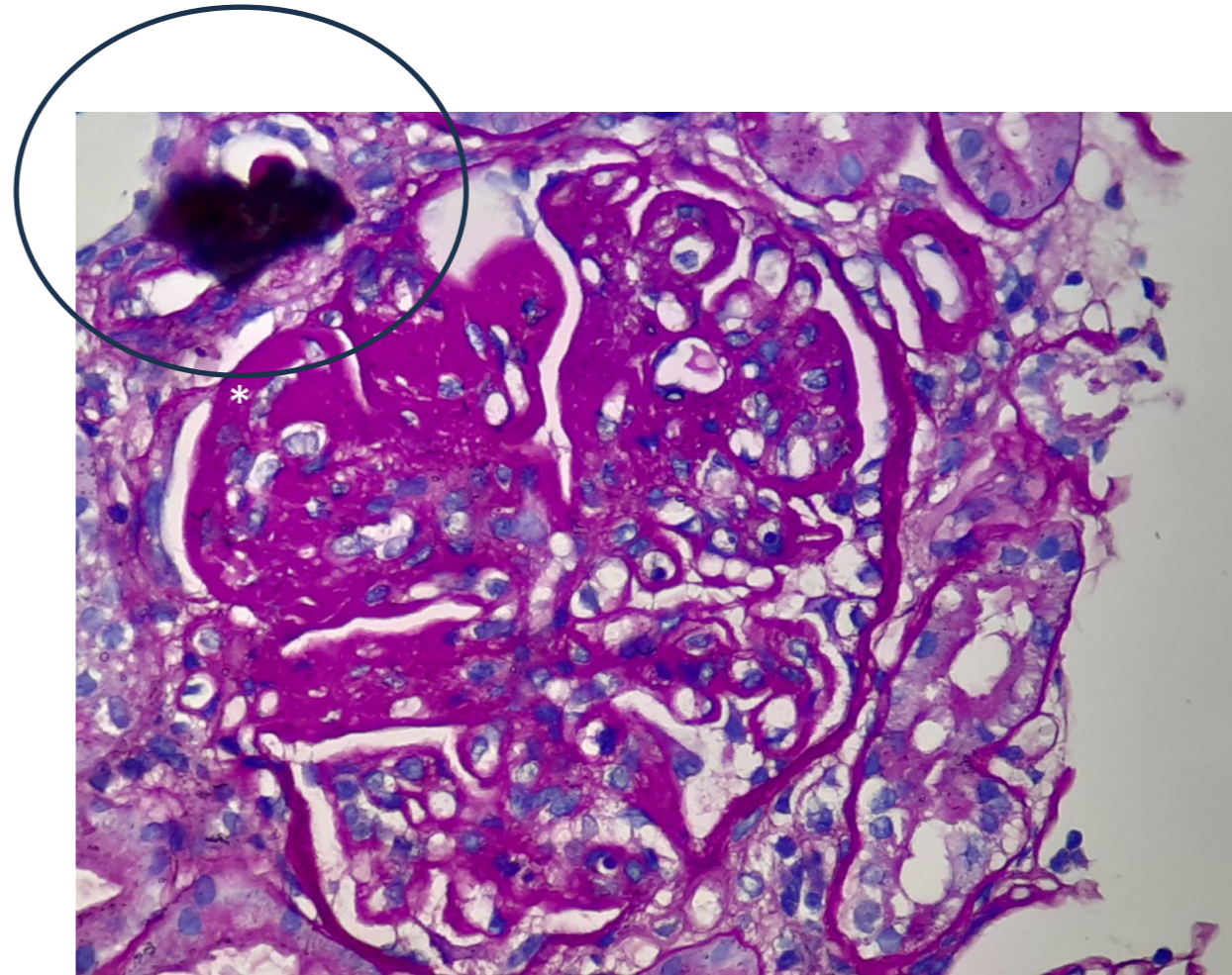
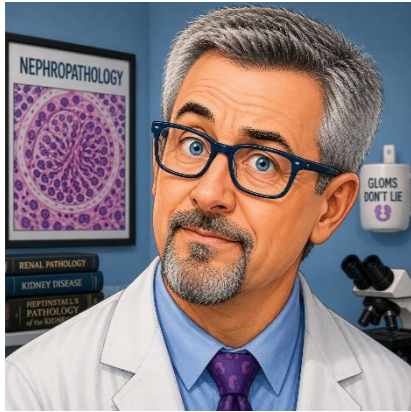




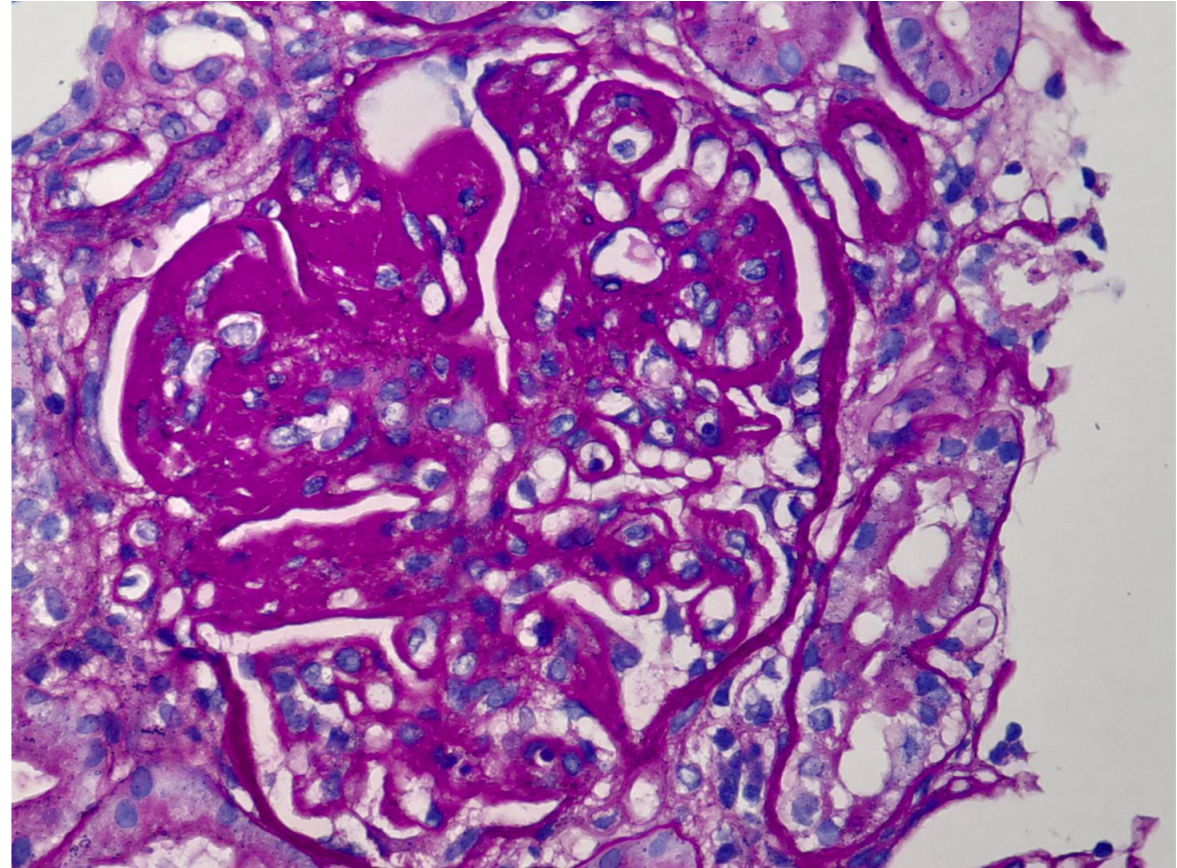
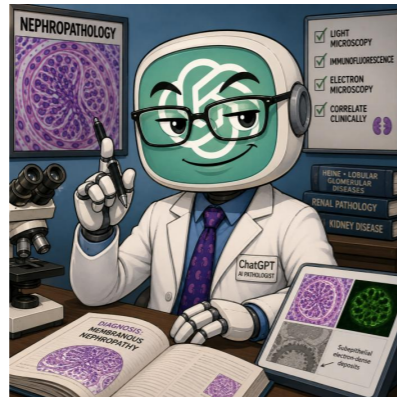
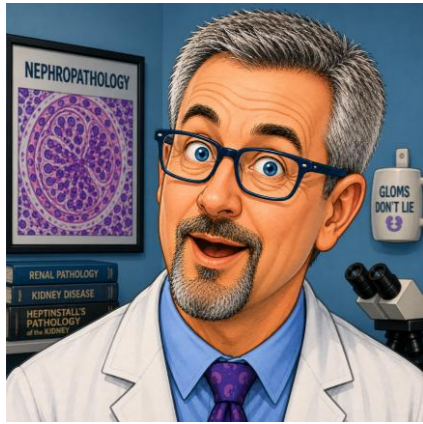
ChatGPT

5/2026

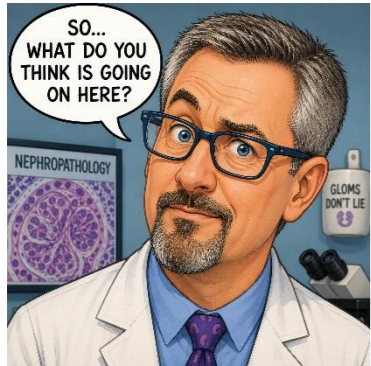
*could you remove the black smear on
the upper left corner
and restore the background
accordingly?*



could you remove the black smear on the upper left corner and restore the background accordingly?



AI generated images

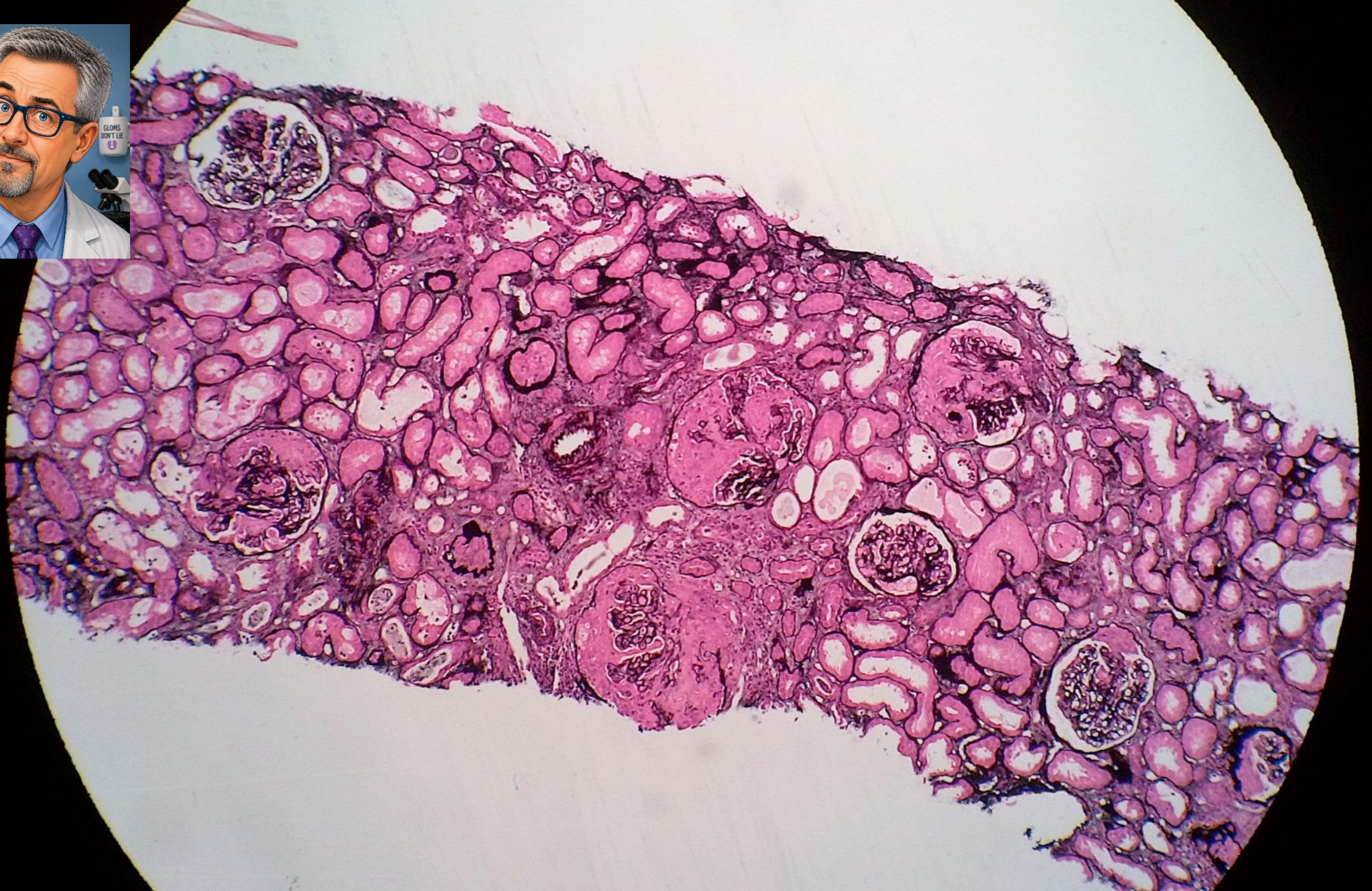
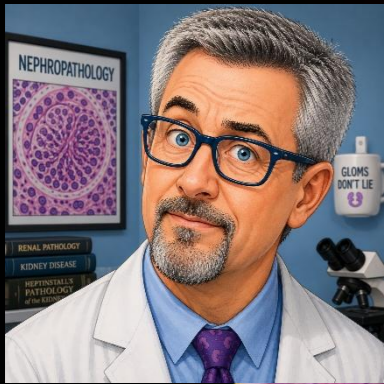


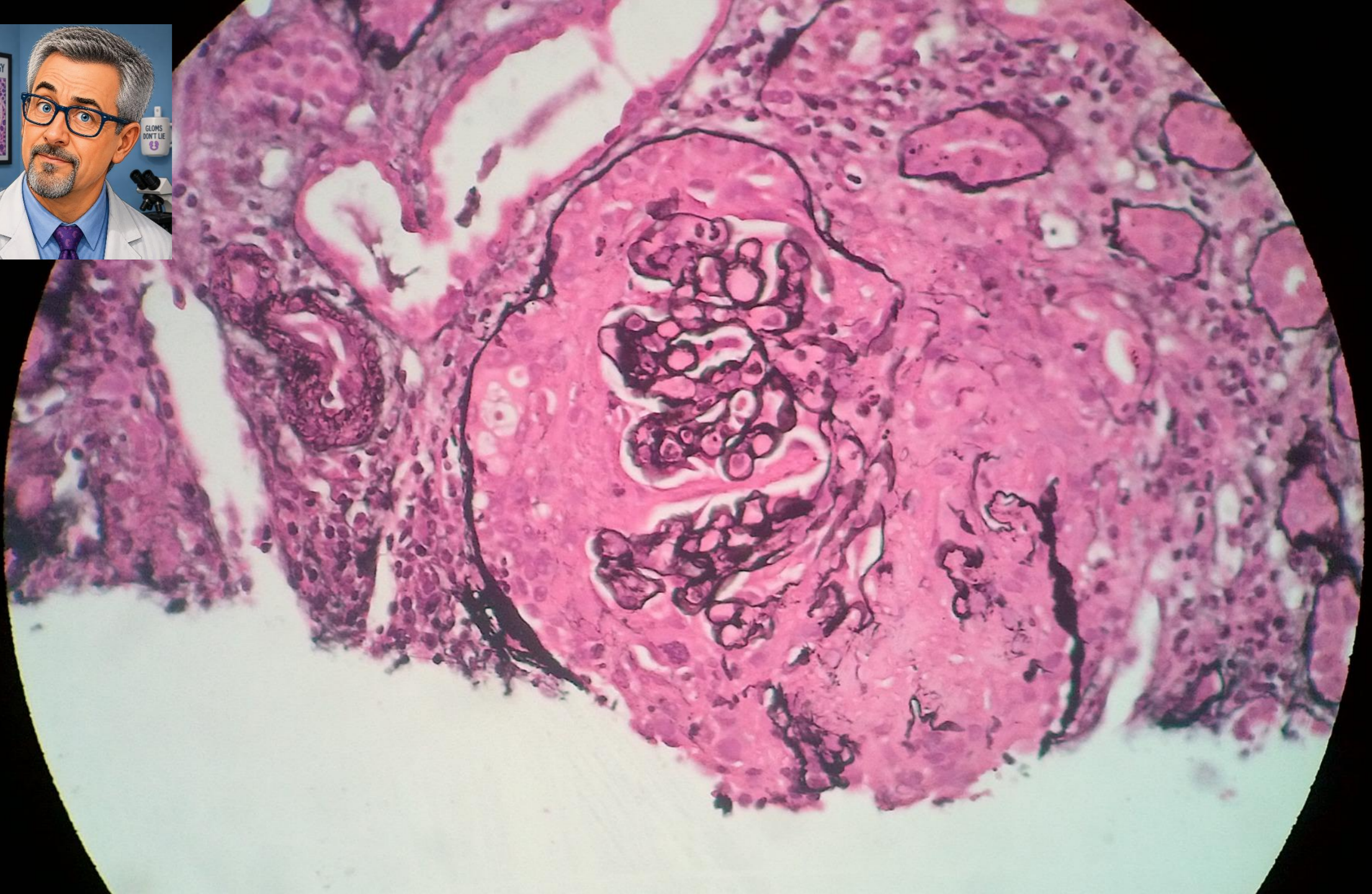
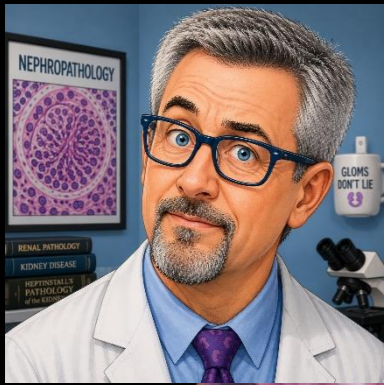
52 year old

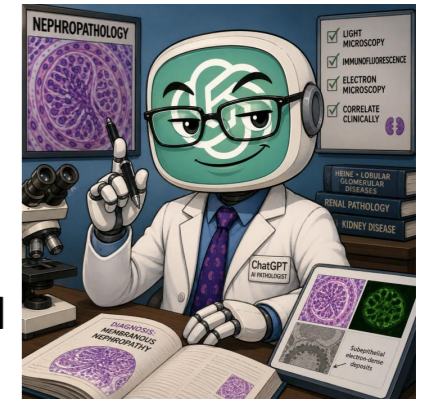
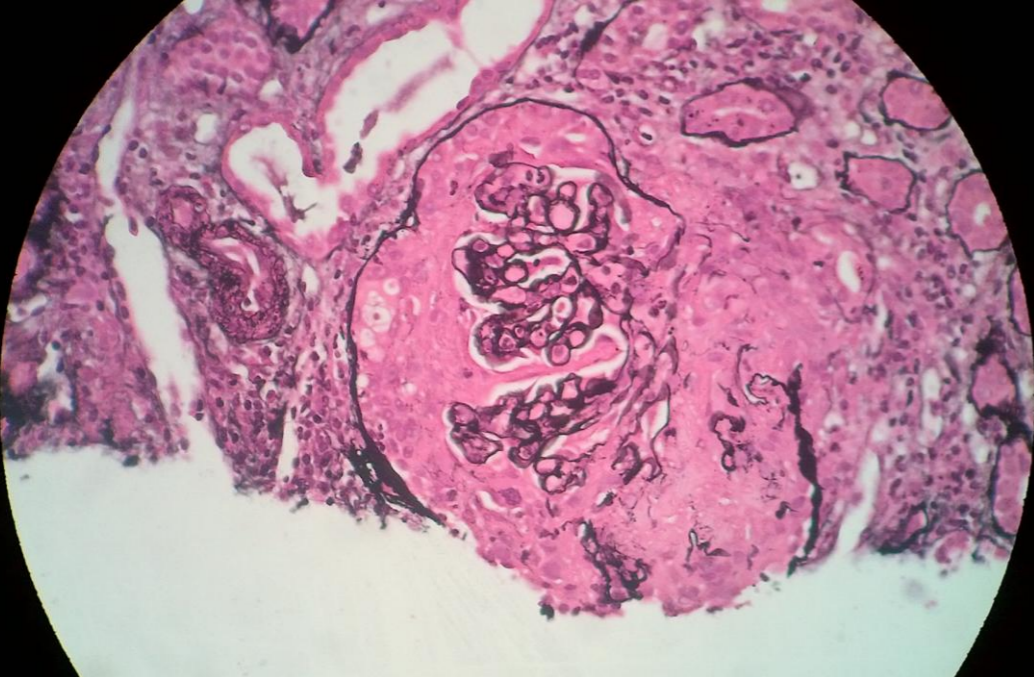
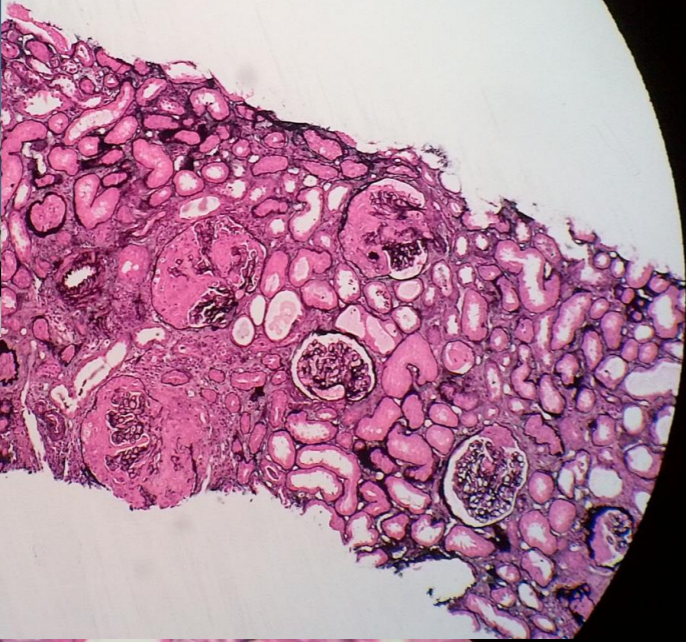
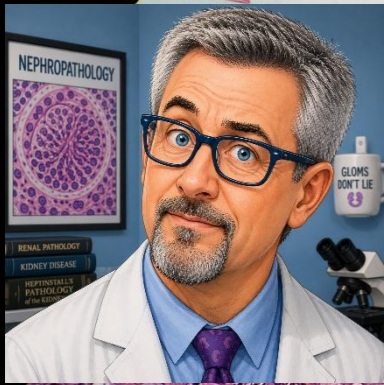
AKI proteinuria microhematuria

ceratin 5

immune screen pending







These silver stains show a markedly abnormal glomerular pattern with:

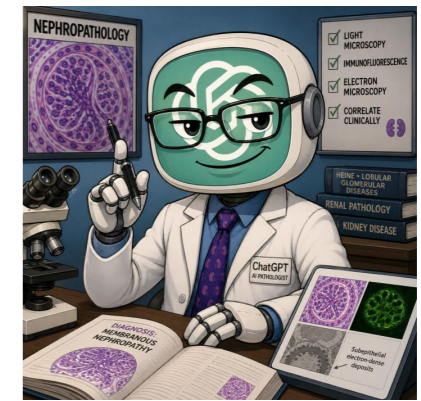
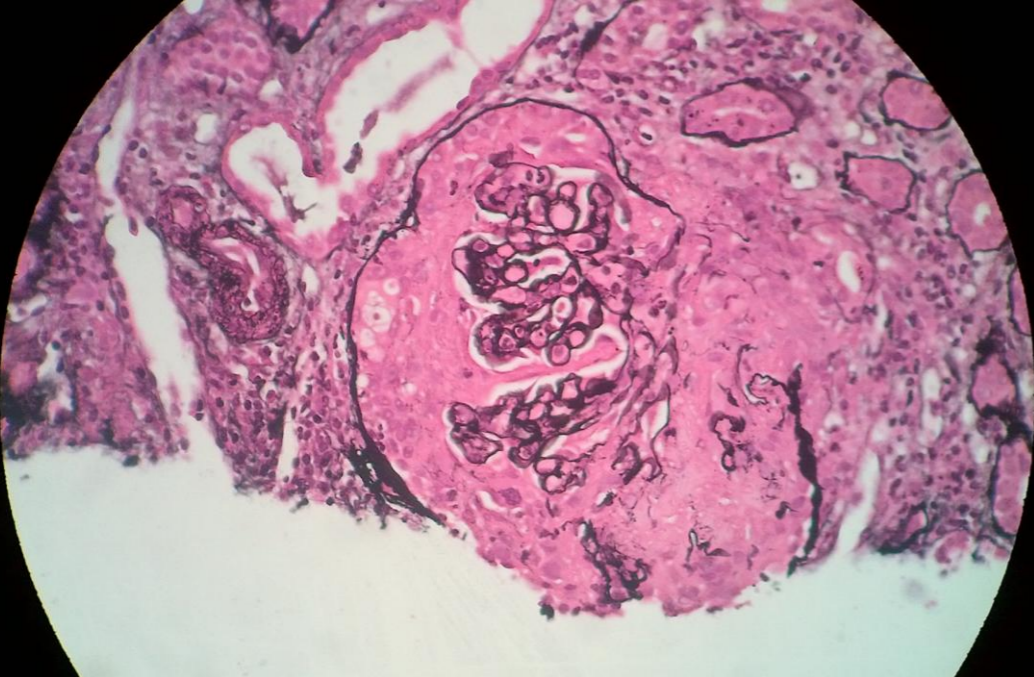
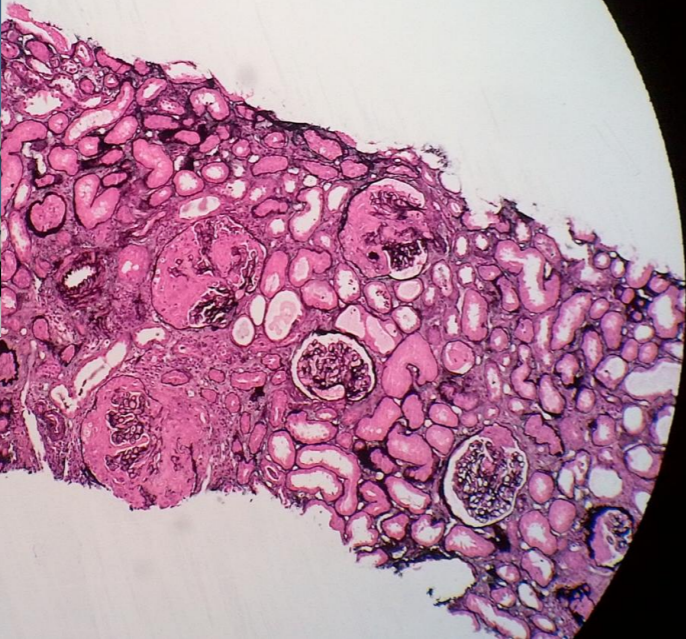
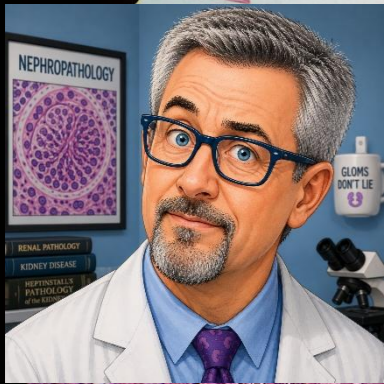
- severe **crescentic/destructive** injury
- collapse and wrinkling of capillary tufts
- probable **fibrinoid necrosis**
- relatively “**pauci-immune**” appearance on LM (without obvious large immune deposits)

Clinically:

- AKI with ***creatinine 5***
- proteinuria + hematuria
- rapidly progressive picture

This combination strongly raises concern for:

Rapidly Progressive Glomerulonephritis (RPGN)



Most likely possibilities at this stage:

1. ANCA-associated pauci-immune crescentic GN

(microscopic polyangiitis / GPA spectrum)

2. Anti-GBM disease

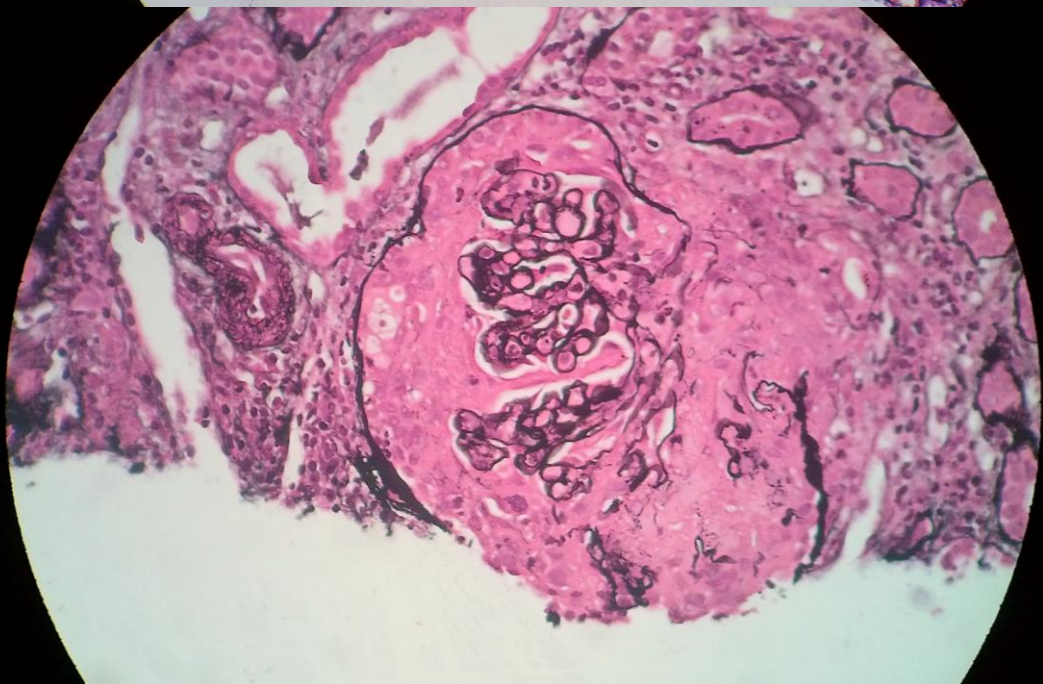
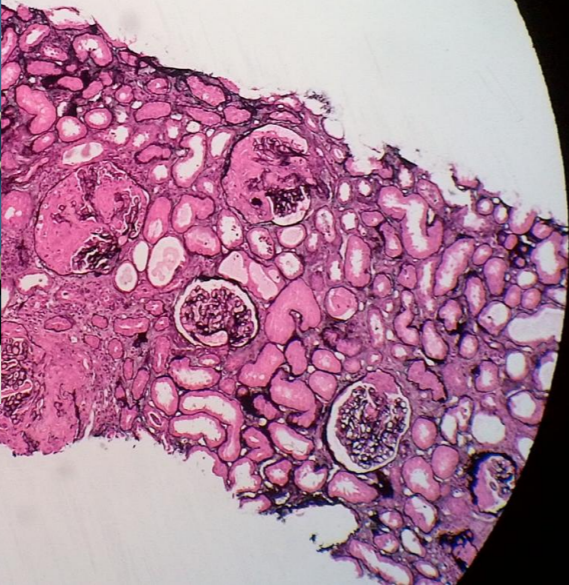
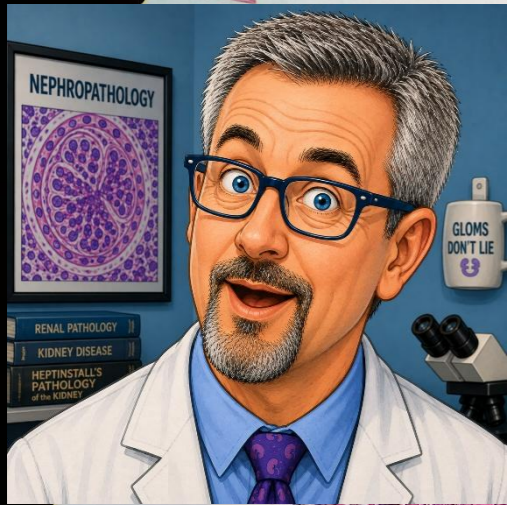
3. Severe immune-complex crescentic GN
(less likely from these images alone)

The silver stain appearance is particularly suggestive of:

pauci-immune necrotizing crescentic GN

because:

- there is major structural destruction
- crescents/necrosis dominate
- not much obvious proliferative immune-complex architecture



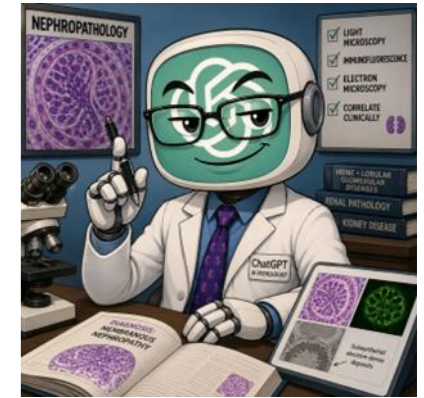
The pending immune screen is therefore critical:

- MPO-ANCA (p-ANCA)
- PR3-ANCA (c-ANCA)
- anti-GBM antibodies
- ANA/complements

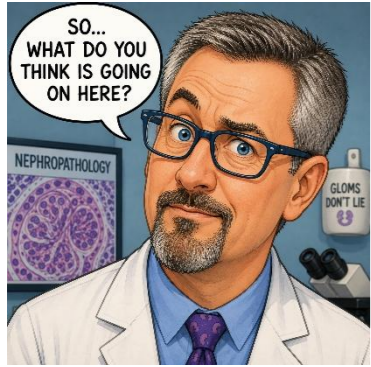
Very important additional information:

- IF findings:
 - pauci-immune → ANCA GN
 - linear IgG → anti-GBM
 - granular immune deposits → immune-complex GN
- pulmonary symptoms/hemoptysis?
- eosinophilia/asthma?
- systemic vasculitis symptoms?

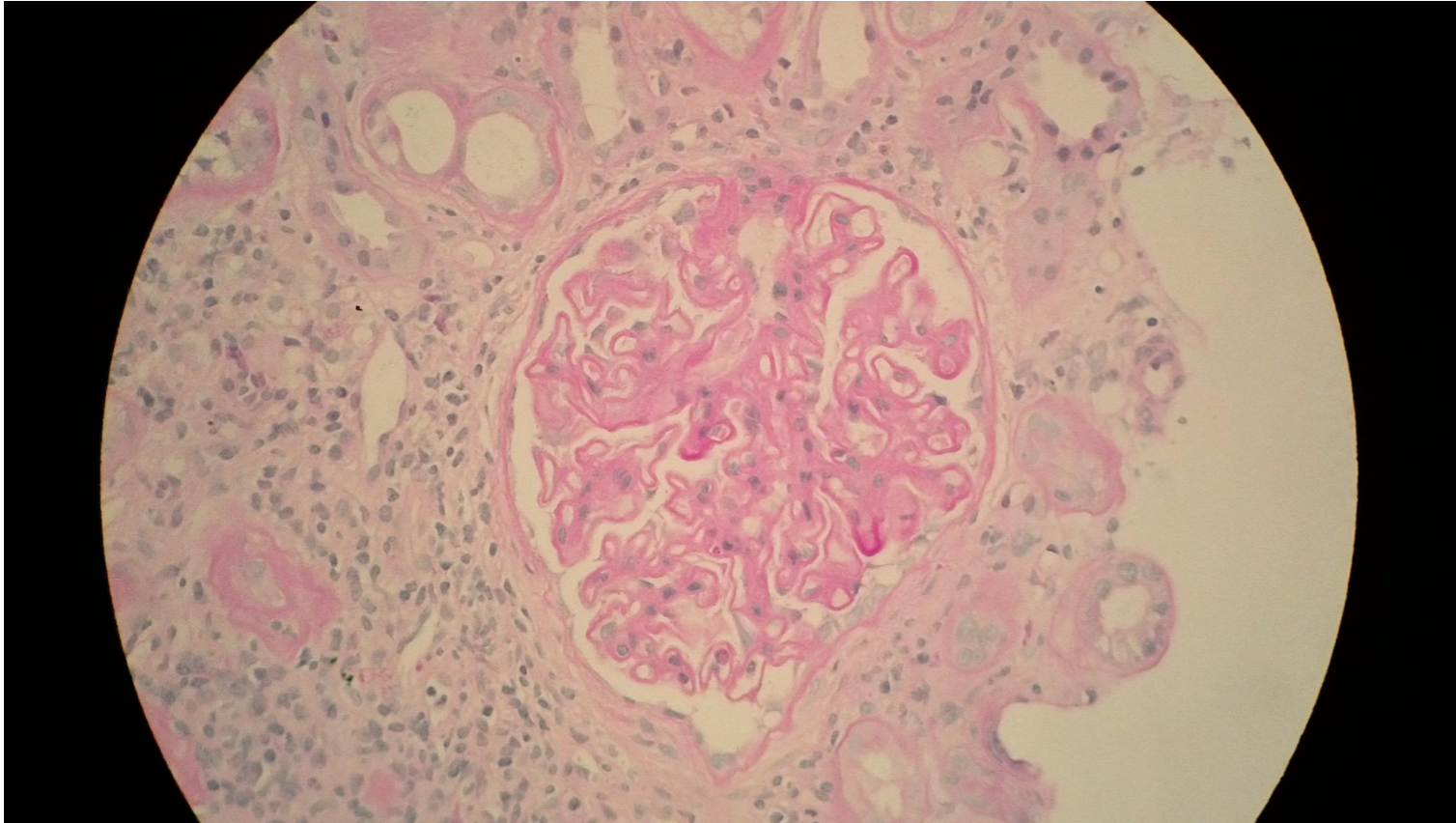
Creatinine 5 indicates severe AKI and this is potentially kidney-threatening and life-threatening if pulmonary hemorrhage is present.

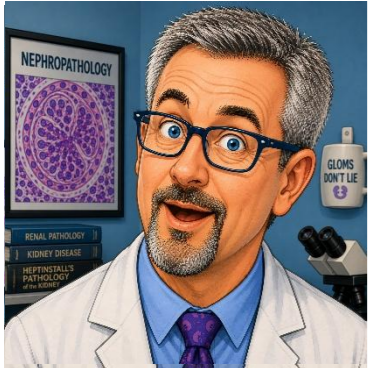


Based on morphology alone, my leading impression is:
pauci-immune crescentic glomerulonephritis (ANCA-associated GN) until proven otherwise.

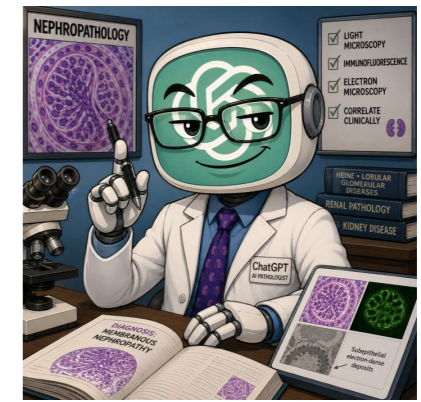


*47yo male nephrotic,
immune screen pending*





*47yo male nephrotic,
immune screen pending*

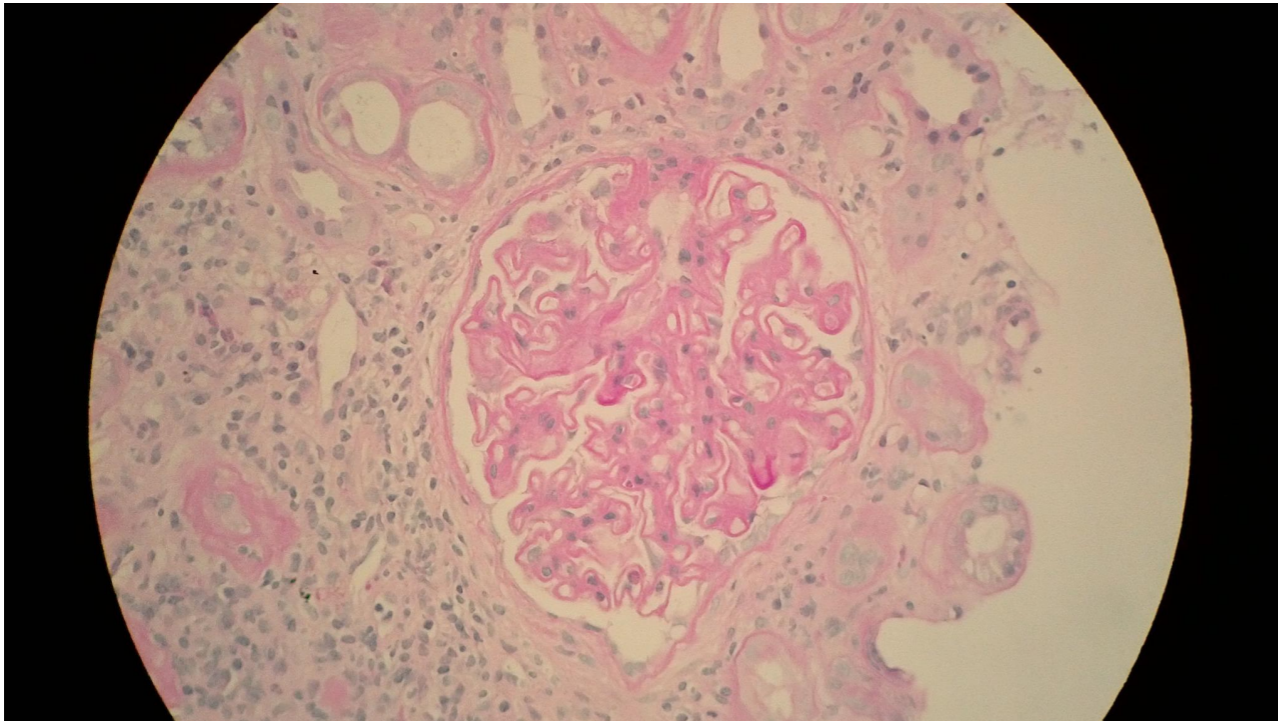


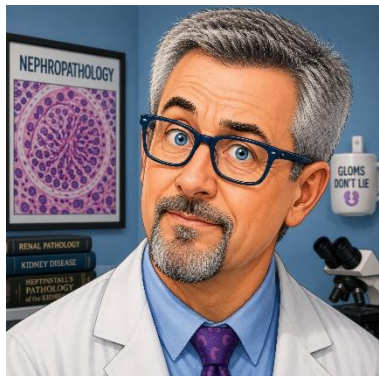
This appears to be a **PAS-stained renal biopsy glomerulus**.

In the context of a 47-year-old male with nephrotic syndrome, the main visible features are:

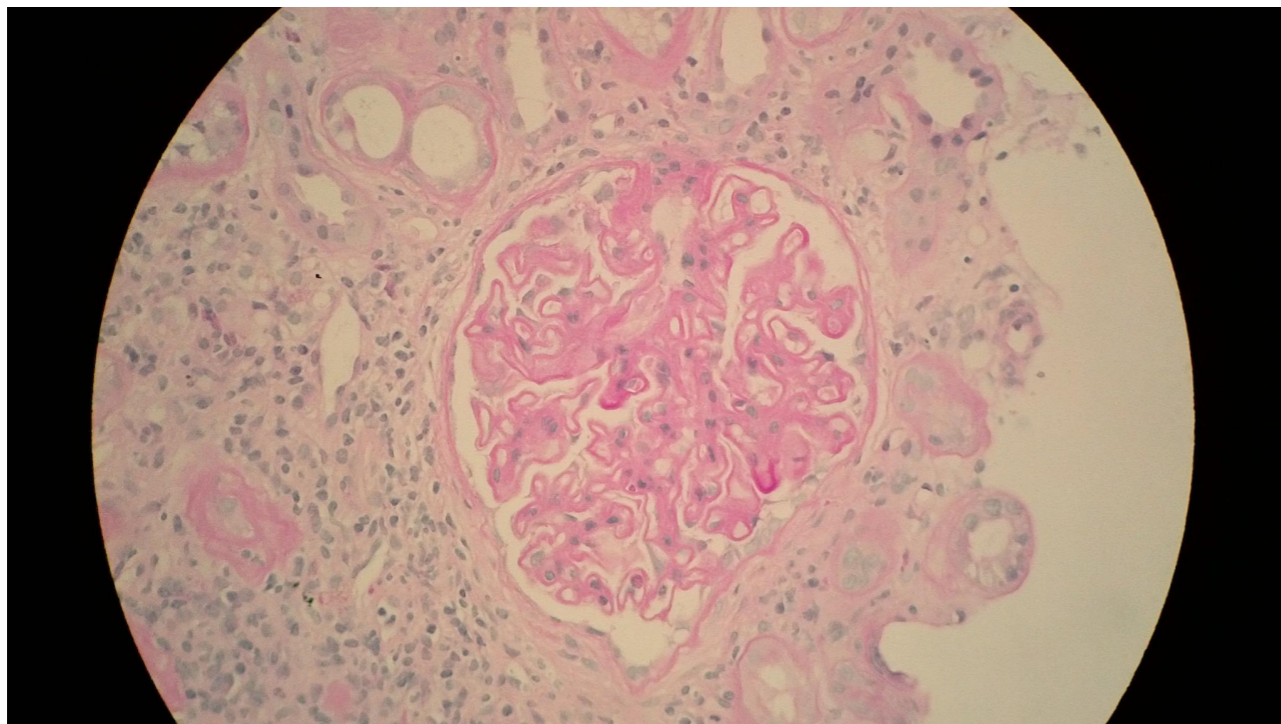
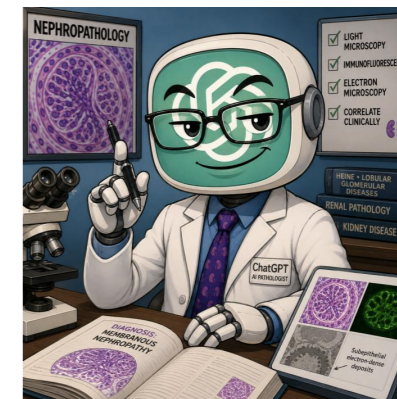
- **Diffuse thickening of the glomerular capillary walls/basement membranes**
- Relatively preserved overall glomerular architecture
- **No obvious marked hypercellularity**
- Some **chronic tubulointerstitial change** in the surrounding cortex

The pattern **raises concern for a membranous nephropathy pattern of injury**, especially **given the nephrotic presentation**.

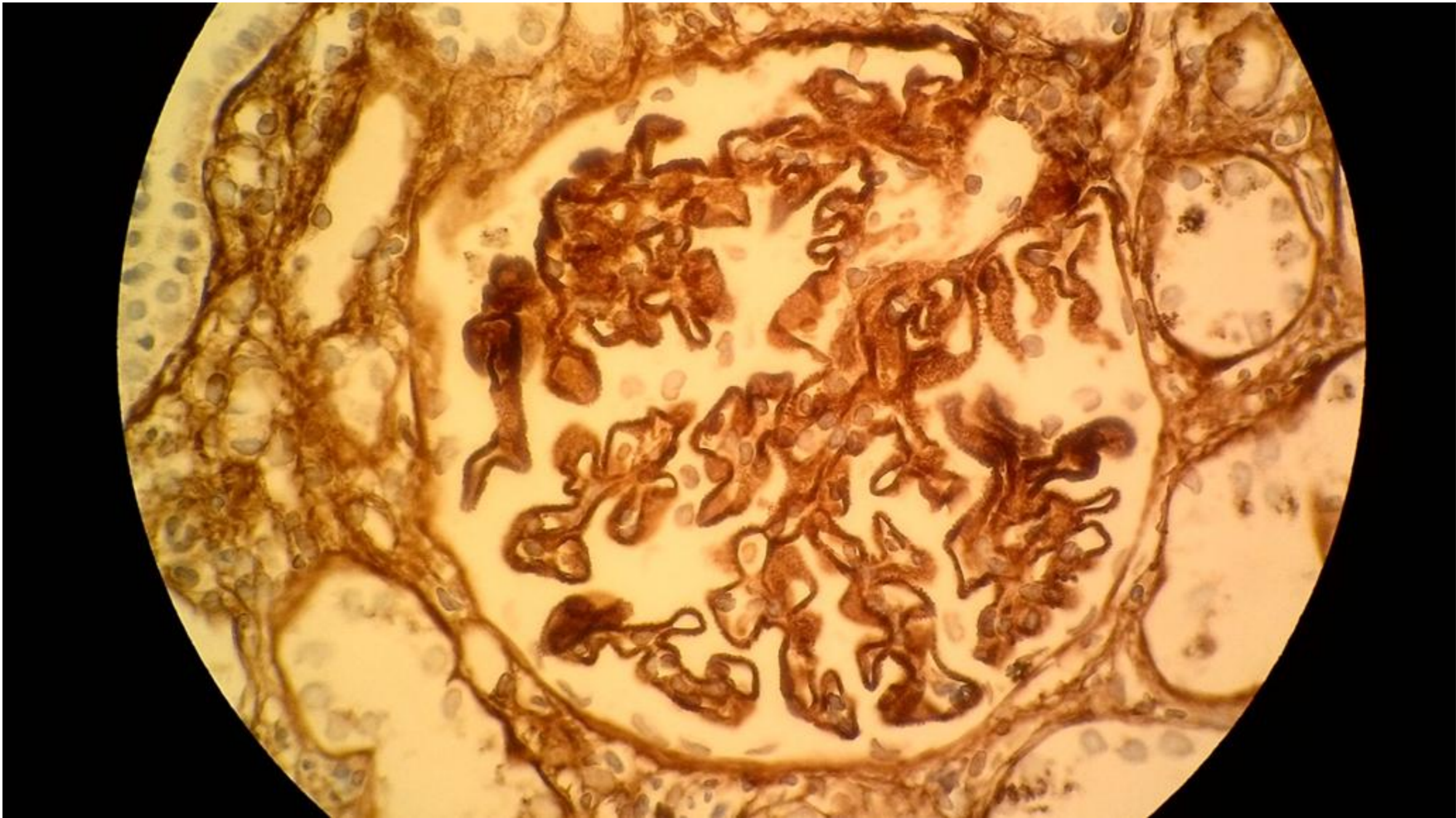


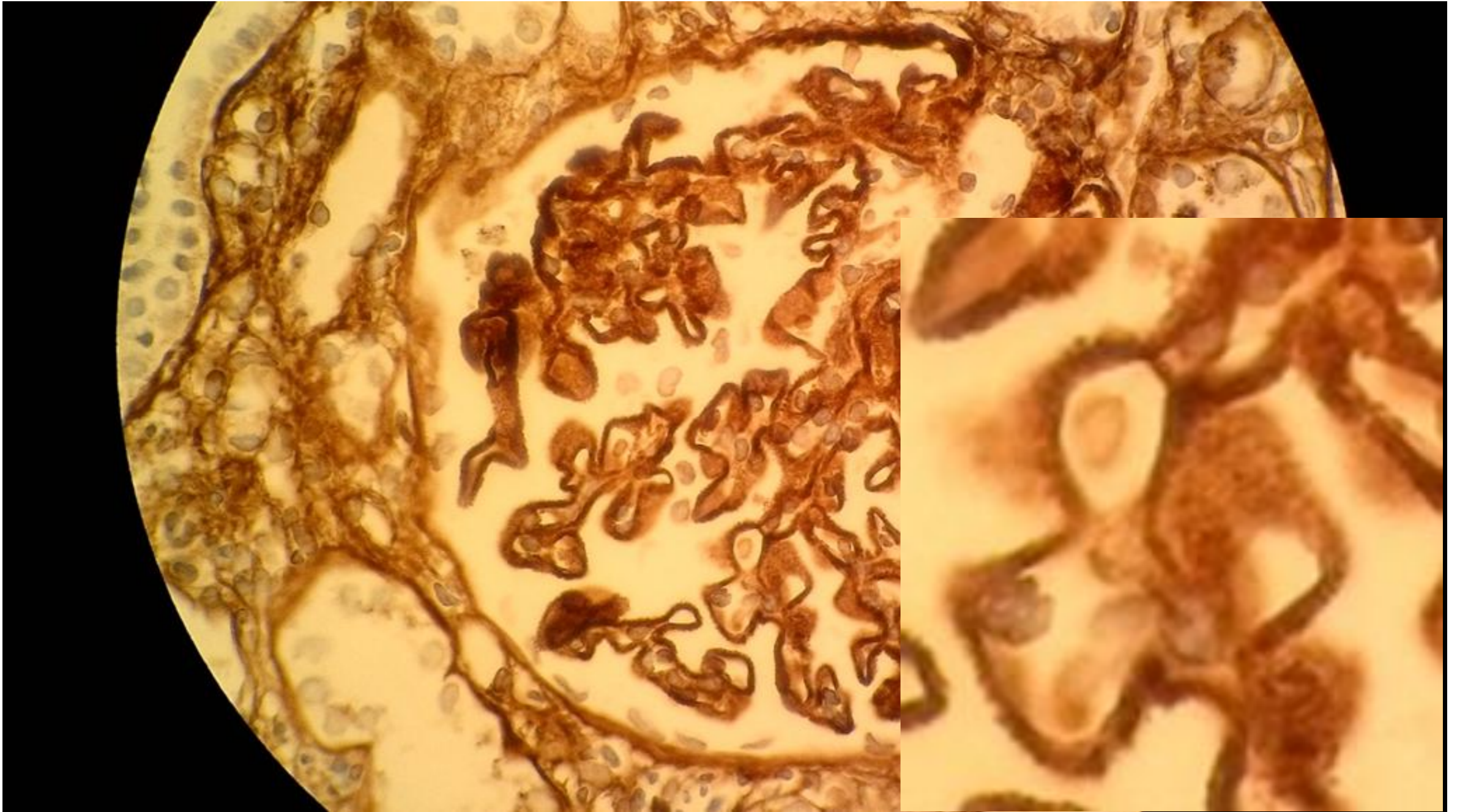


*47yo male nephrotic,
immune screen pending*



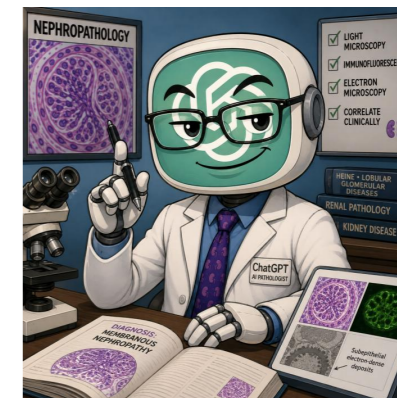
- Important next steps / correlations:
- IF staining: IgG, C3, κ/λ , IgA, IgM
- PLA2R and THSD7A
- Electron microscopy for subepithelial deposits/spikes
- Full immune screen (already pending)
- Hepatitis/HIV screen
- Malignancy workup appropriate for age/risk factors
- **I would avoid making a definitive diagnosis from this single low-power image alone, but morphologically it is quite compatible with a membranous pattern.**



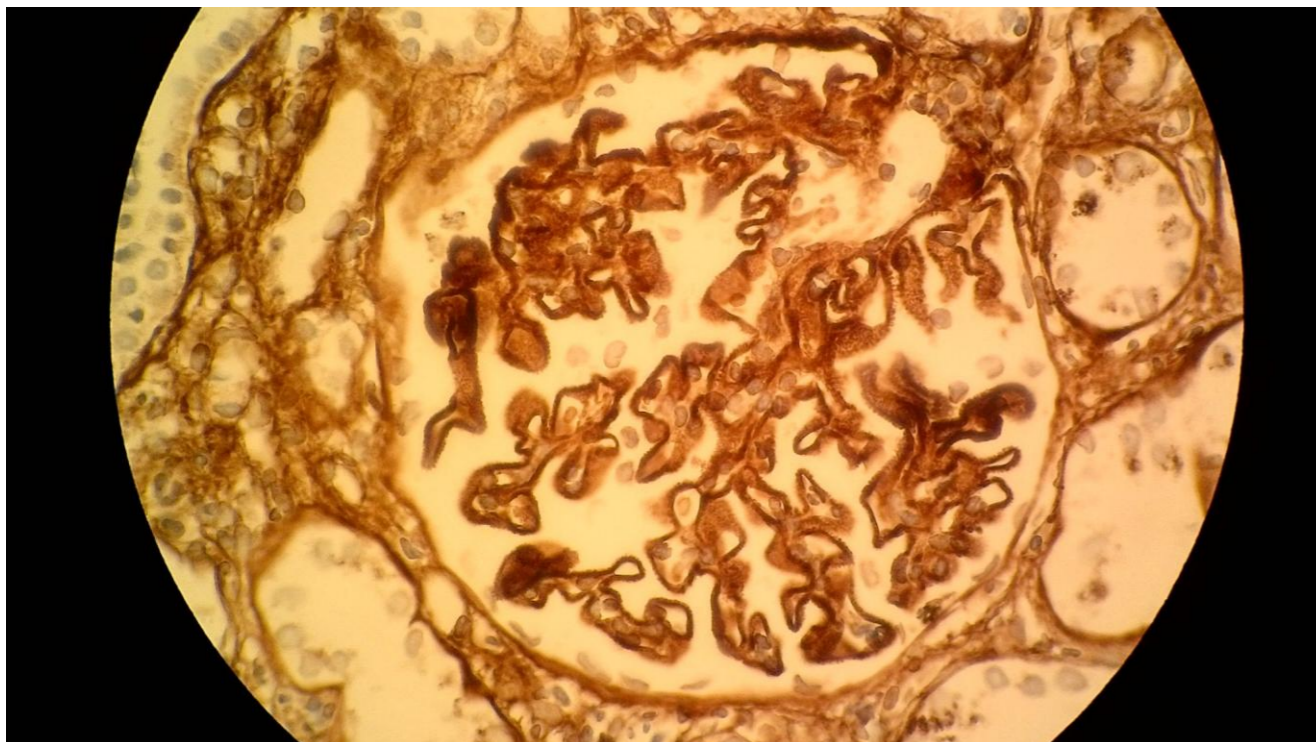


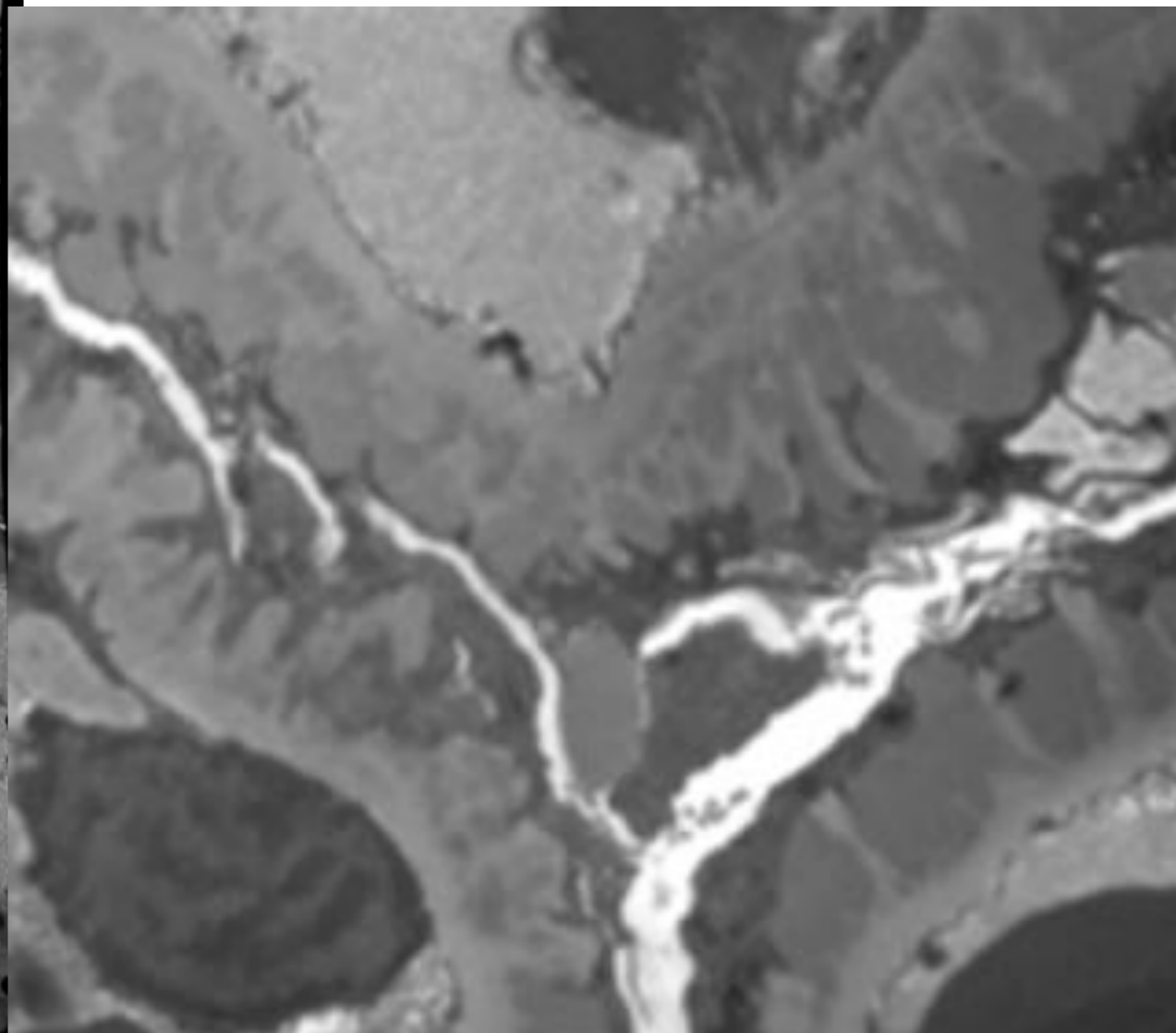
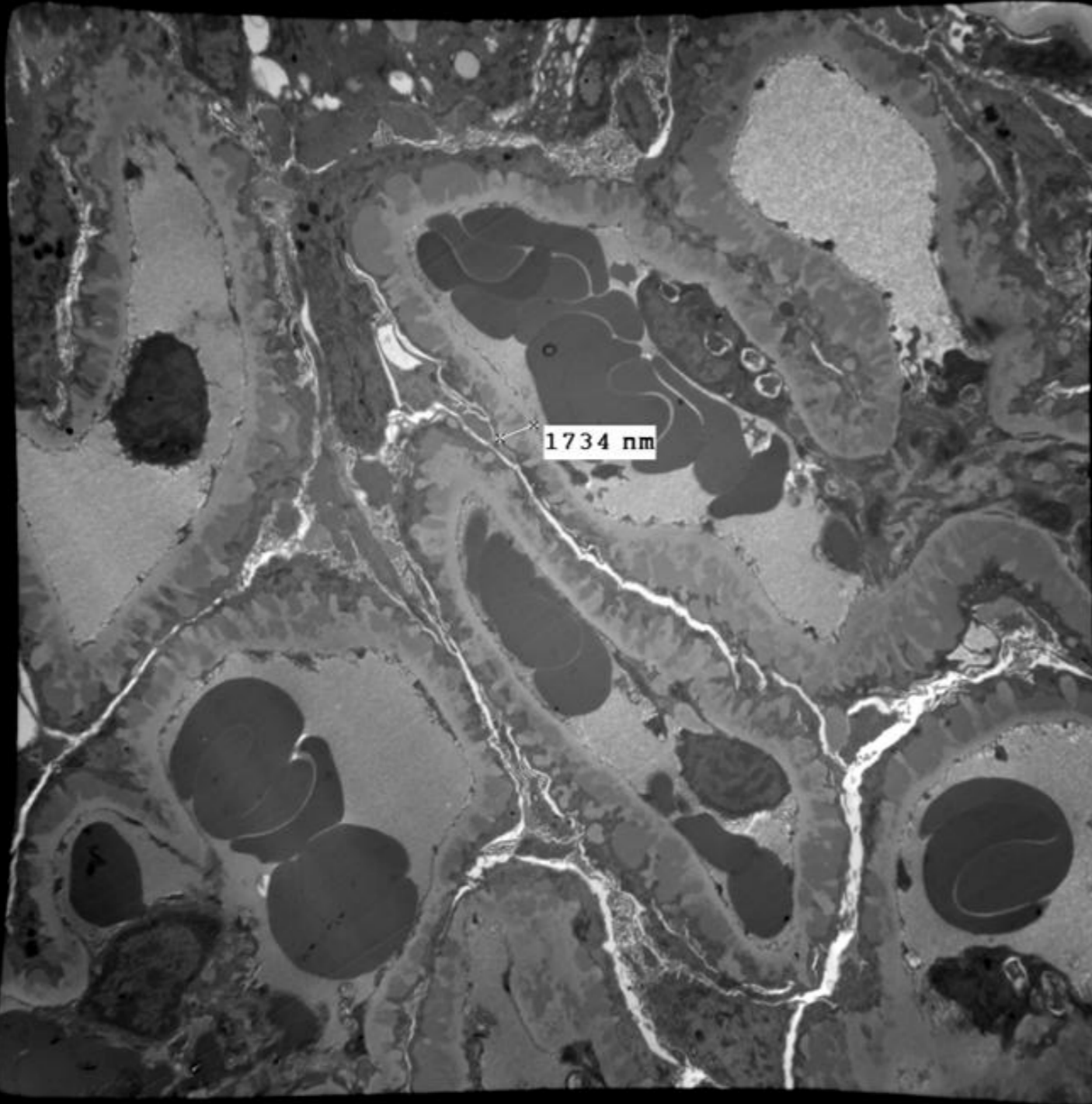


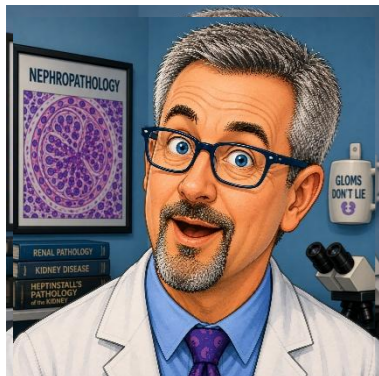
*47yo male nephrotic,
immune screen pending*



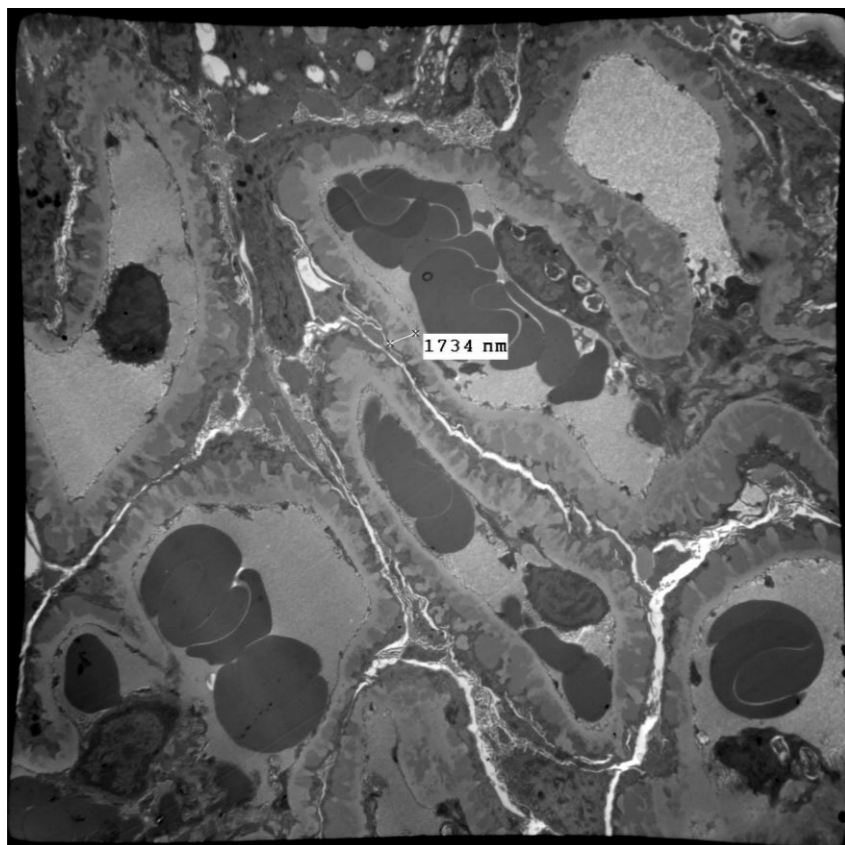
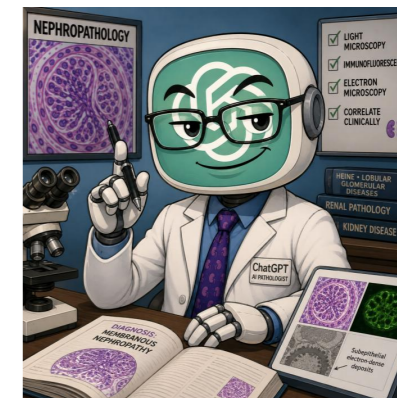
- This **silver** stain is much more informative.
- The glomerular basement membranes show:
- Diffuse capillary wall thickening
- Irregular “**spike**” formation along the outer aspect of the GBM
- Little endocapillary hypercellularity
- That pattern is very characteristic of:
- **Membranous nephropathy**
- especially on a Jones methenamine silver stain
showing the classic “**spike and dome**” appearance.



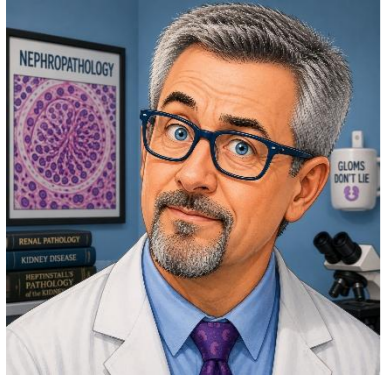




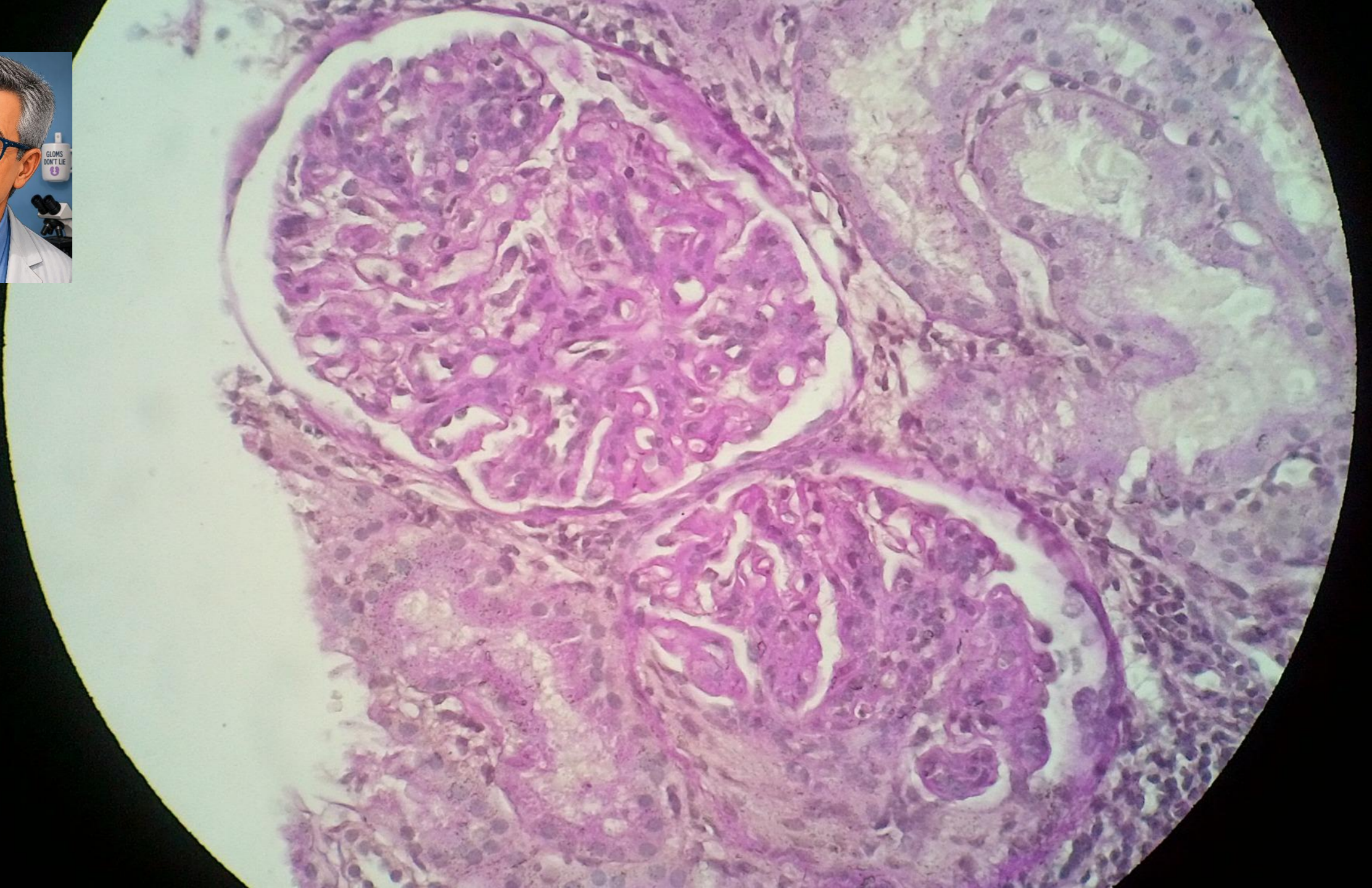
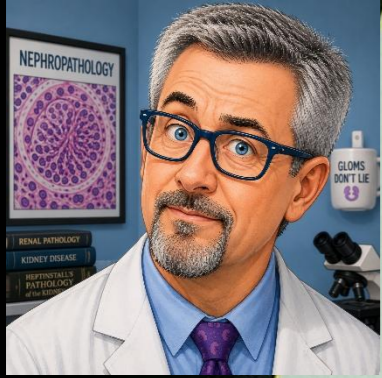
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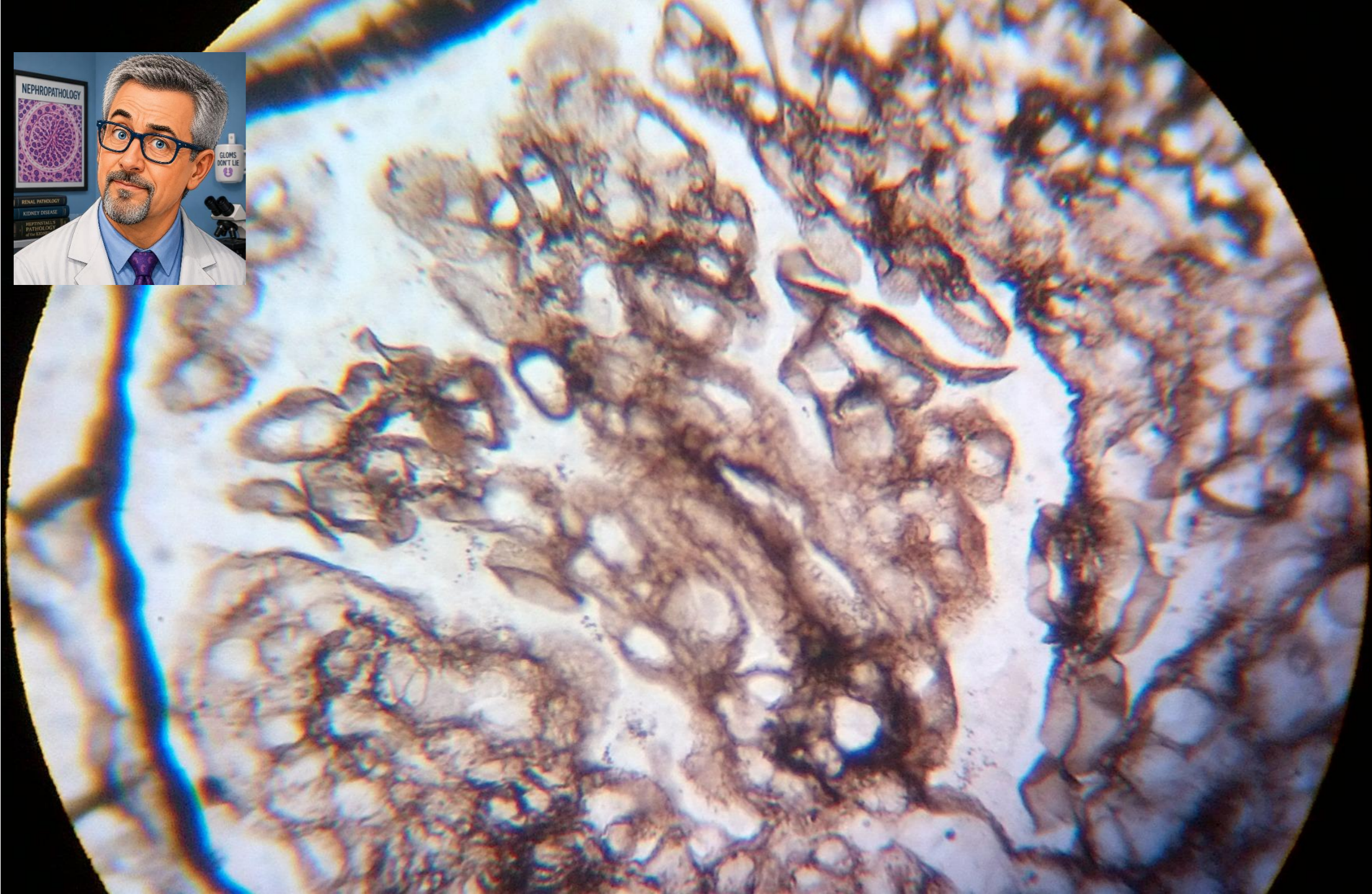
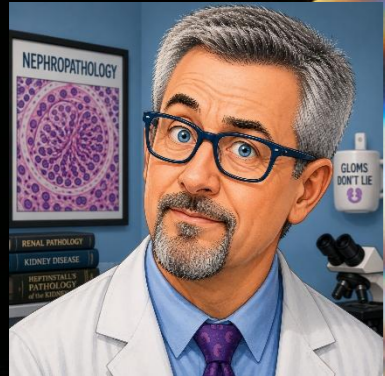


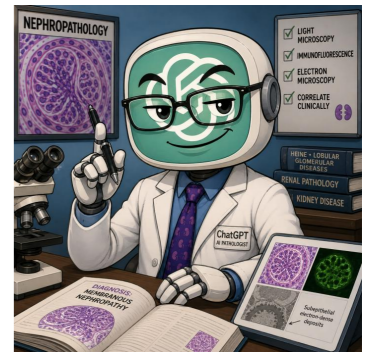
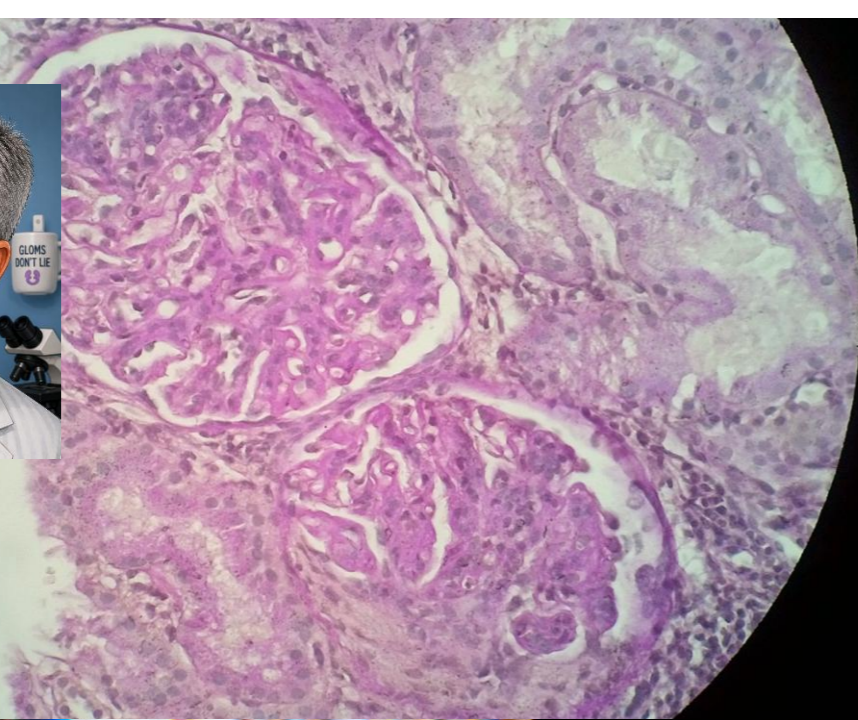
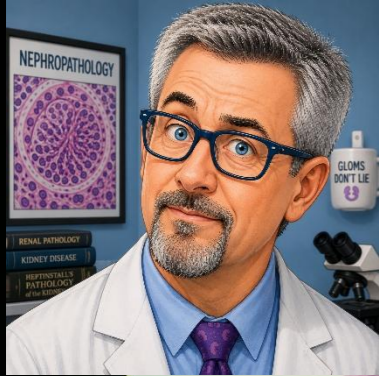
- This EM image supports the diagnosis very strongly.
- Key ultrastructural findings visible here:
- Numerous **subepithelial electron-dense deposits** along the outer aspect of the GBM
- Basement membrane material projecting between deposits (“**spikes**”)
- Podocyte foot process effacement
- **No obvious organized fibrillary** substructure
- This is classic for:
- **Membranous nephropathy**
- The triad across your studies is very convincing:
- LM: diffuse capillary wall thickening
- Silver stain: spike formation
- EM: subepithelial deposits + foot process effacement
- So this is essentially a **textbook membranous pattern.**



*23yo female ANA + 1/320
antidsDNA+ 1/80
low c3 c4
proteinuria microhematuria
normal creatinine*







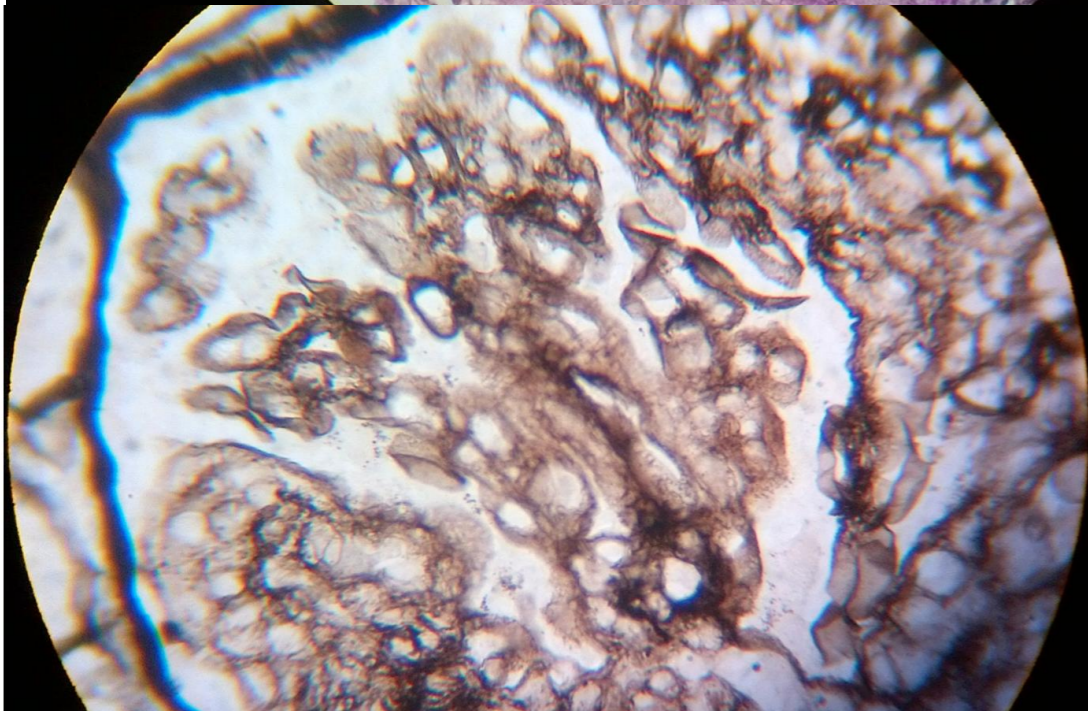
This *clinical* picture is highly suggestive of:

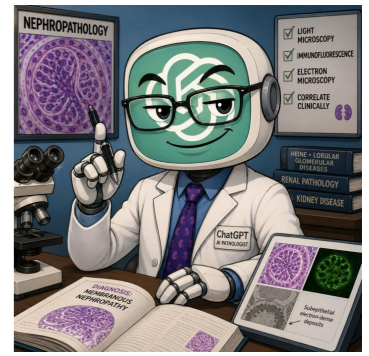
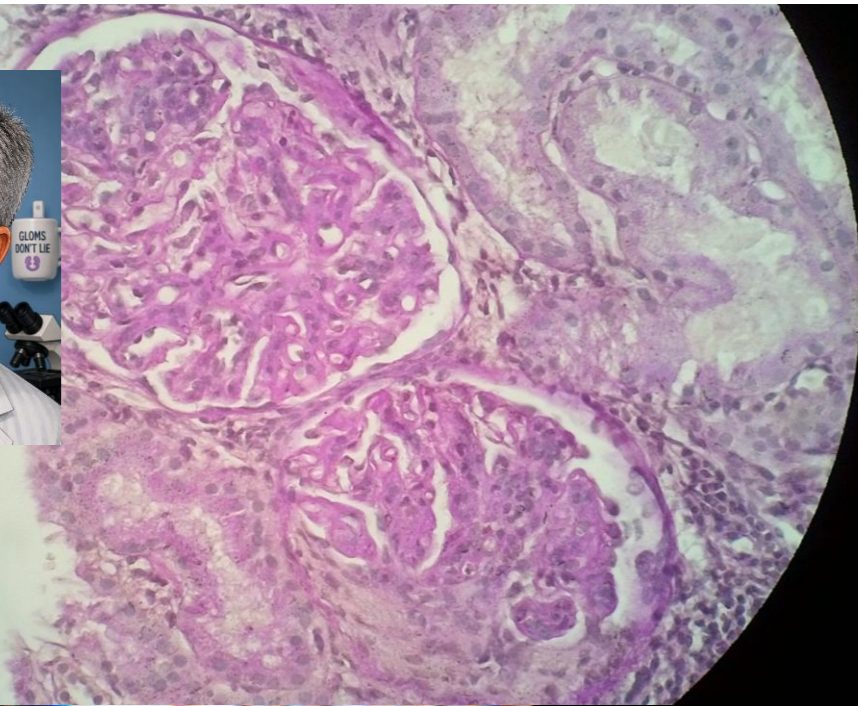
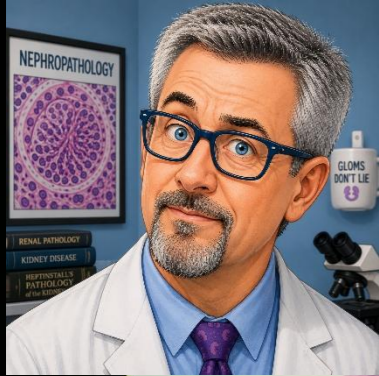
Lupus nephritis

because of:

- young woman
- ANA strongly positive
- anti-dsDNA positive
- low C3/C4
- proteinuria + microscopic hematuria

Those serologies are classic for active immune-complex lupus nephritis.





The biopsy images also support lupus nephritis:

PAS image

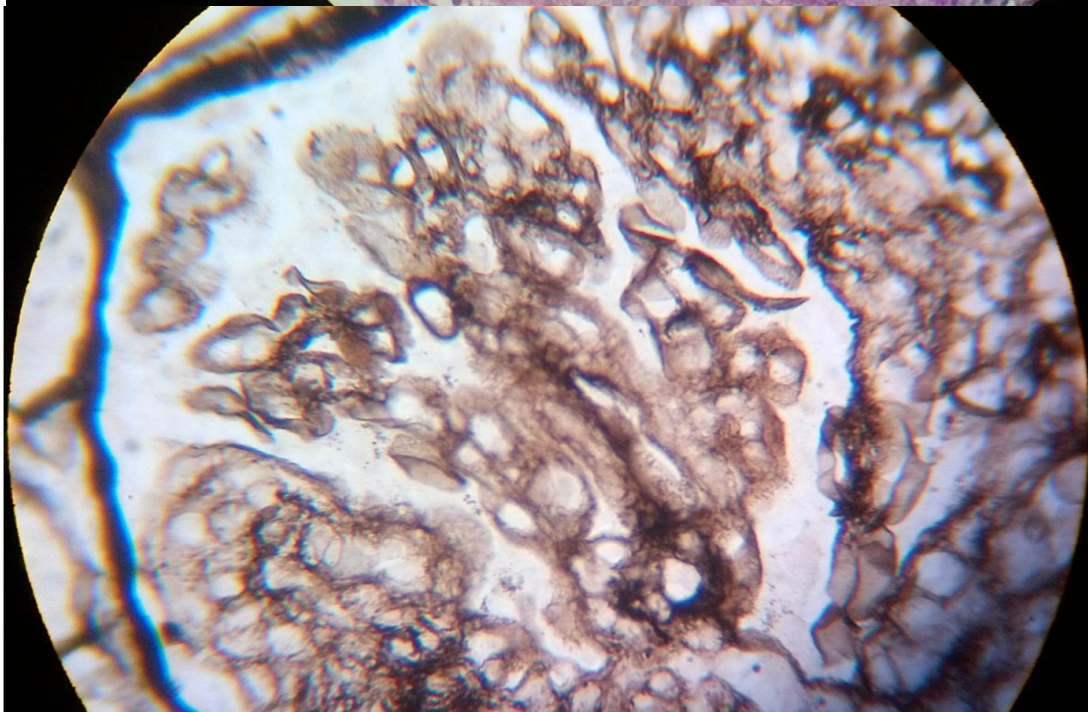
Shows:

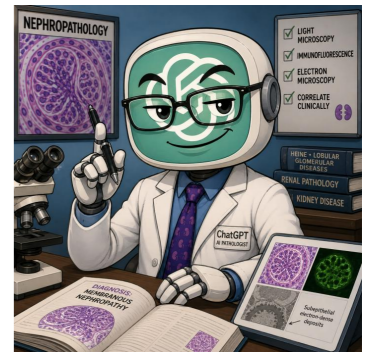
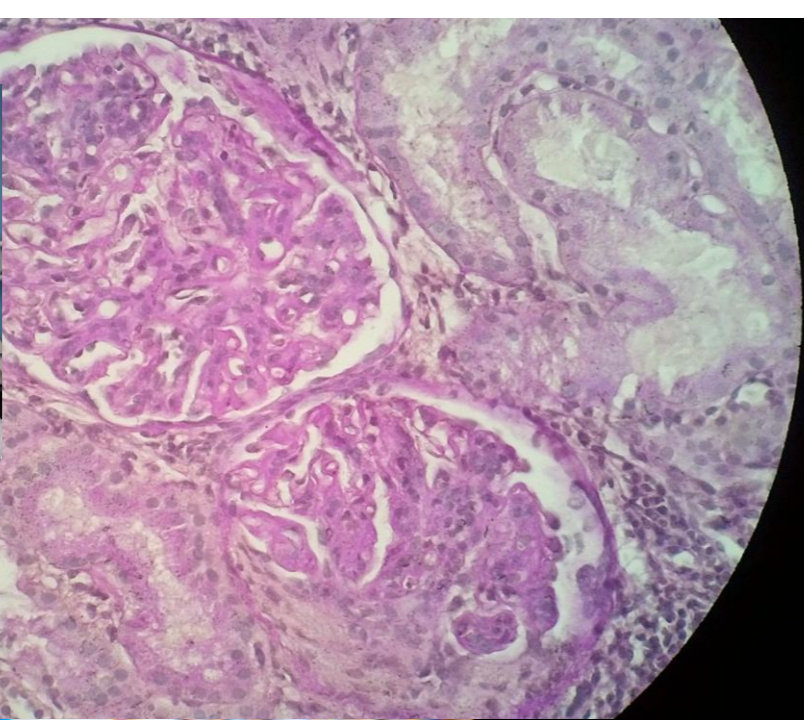
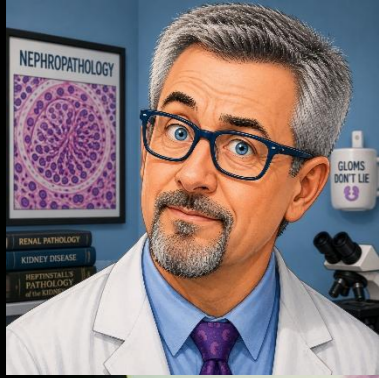
- mesangial and endocapillary hypercellularity
- lobular accentuation
- capillary wall thickening

Silver stain

Shows:

- duplicated/split GBM contours (“tram-tracks”)
- immune-complex remodeling





That pattern raises concern for:

proliferative lupus nephritis

especially:

- Class III (focal)

or

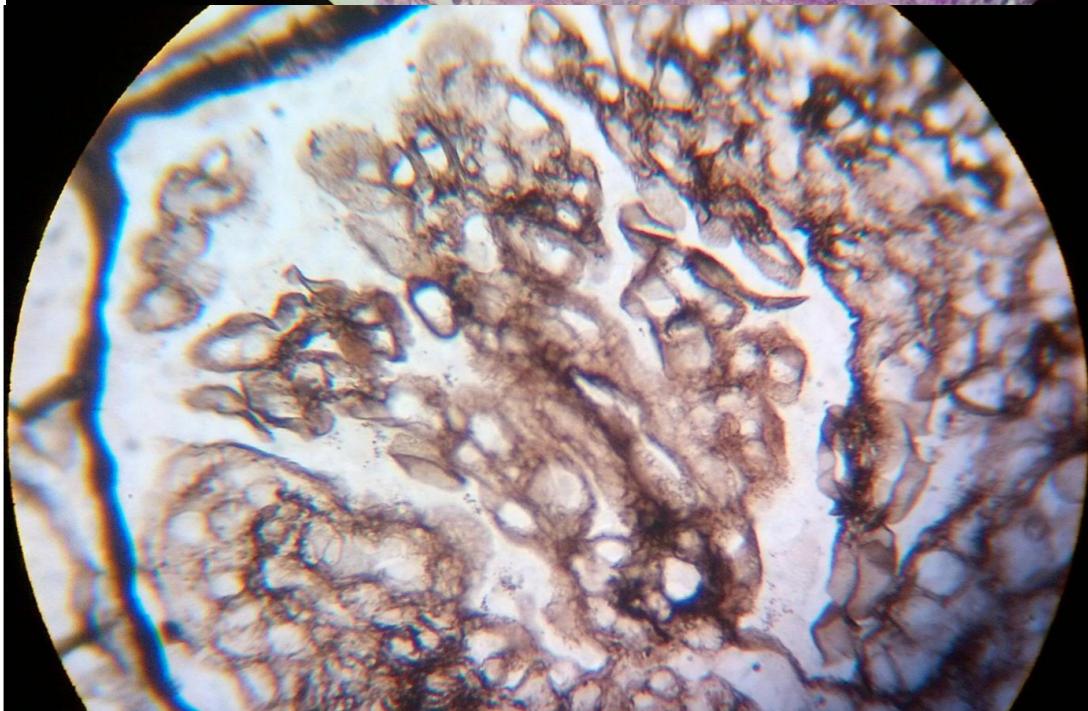
- Class IV (diffuse)

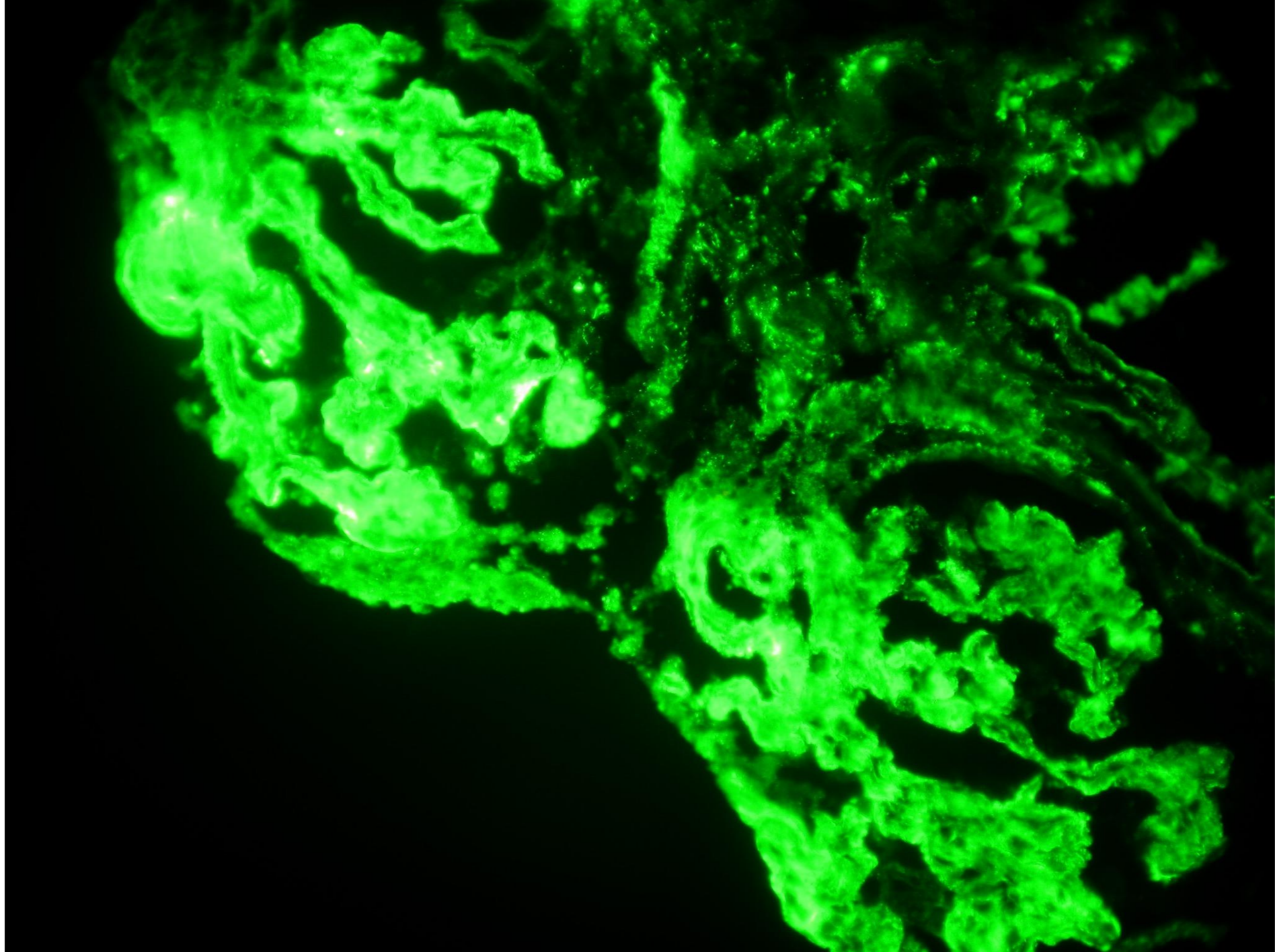
Possibly with a membranous component if there is substantial capillary wall involvement.

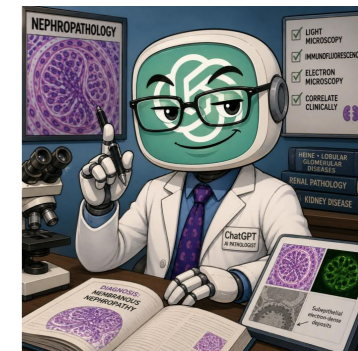
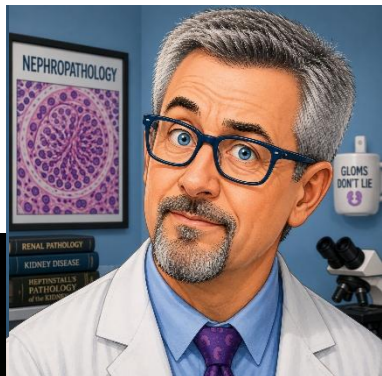
The decisive findings would come from:

- immunofluorescence

- electron microscopy







This immunofluorescence image strongly supports active lupus nephritis.

The staining pattern appears:

- coarse granular
- along capillary walls and mesangium
- very intense C3 deposition**

That is classic for immune-complex deposition in lupus nephritis.

Combined with:

- ANA+
- anti-dsDNA+
- low C3/C4
- proteinuria + hematuria
- proliferative changes on LM

this is very convincing for:

proliferative lupus nephritis

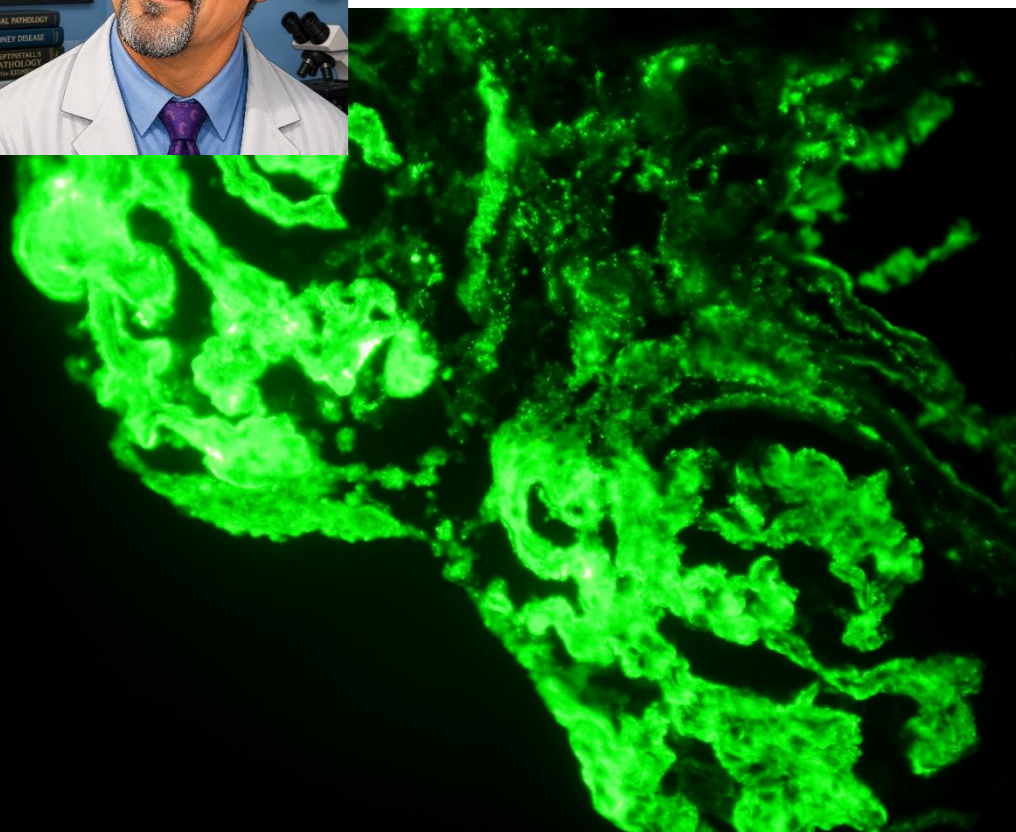
The biopsy now looks most compatible with:

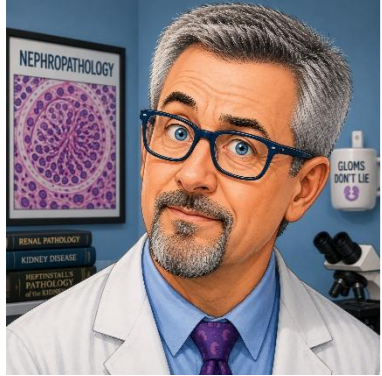
Class IV lupus nephritis

(diffuse proliferative LN)

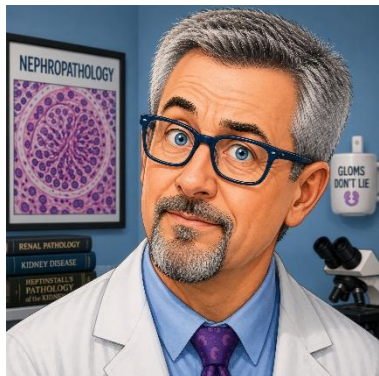
Possibly with:

- membranous overlap (Class V component)**
- because the capillary wall staining is quite prominent.**

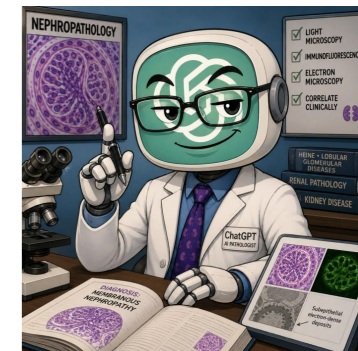




*46 years old nephrotic
creatinine 2.5
immune screen negative
HBV/HCV/HIV negative.*



*46 years old nephrotic
creatinine 2.5
immune screen negative
HBV/HCV/HIV negative.*



A creatinine of 2.5 mg/dL in a 46-year-old with nephrotic syndrome means there is significant kidney dysfunction in addition to heavy protein loss.

The combination of:

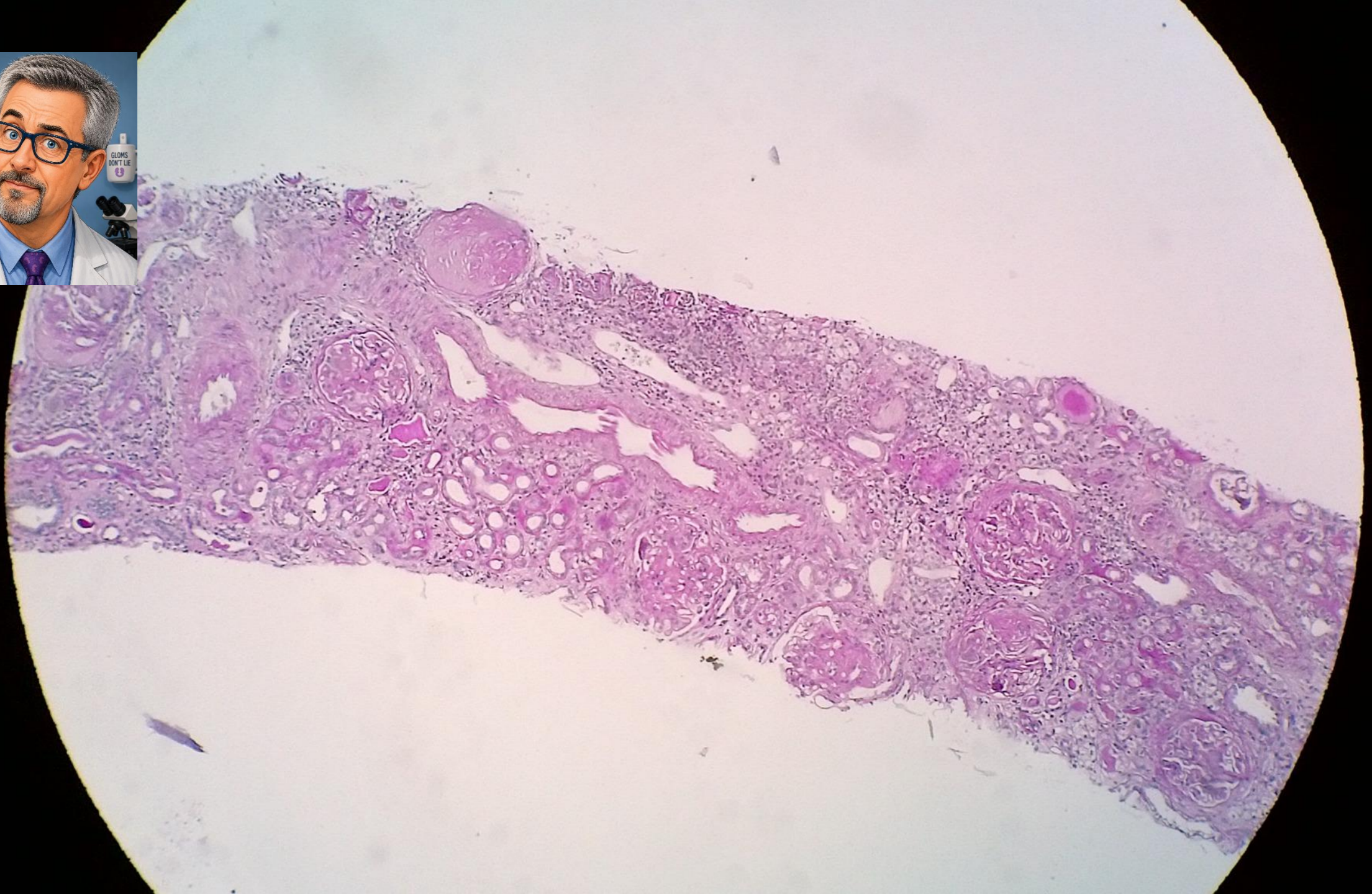
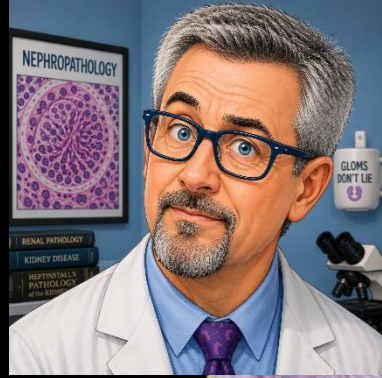
- nephrotic syndrome
- negative immune screen
- HBV/HCV/HIV negative

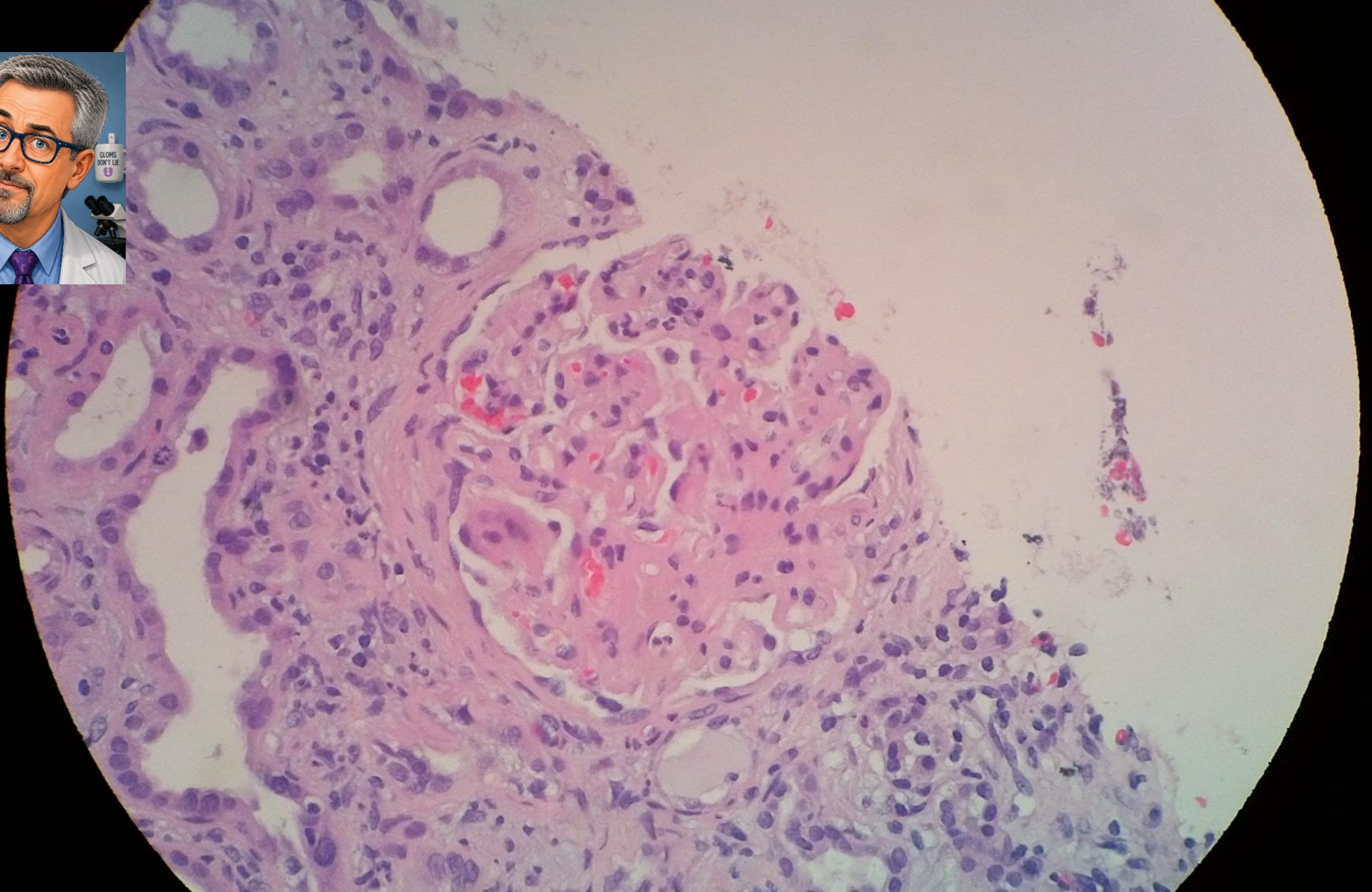
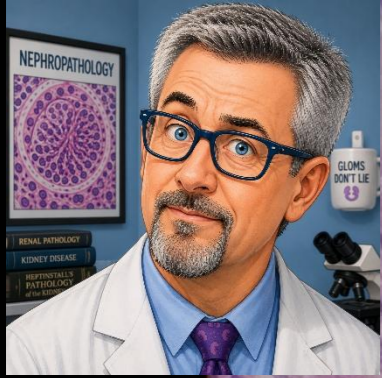
narrows the differential somewhat, but it does **not** identify the cause by itself.

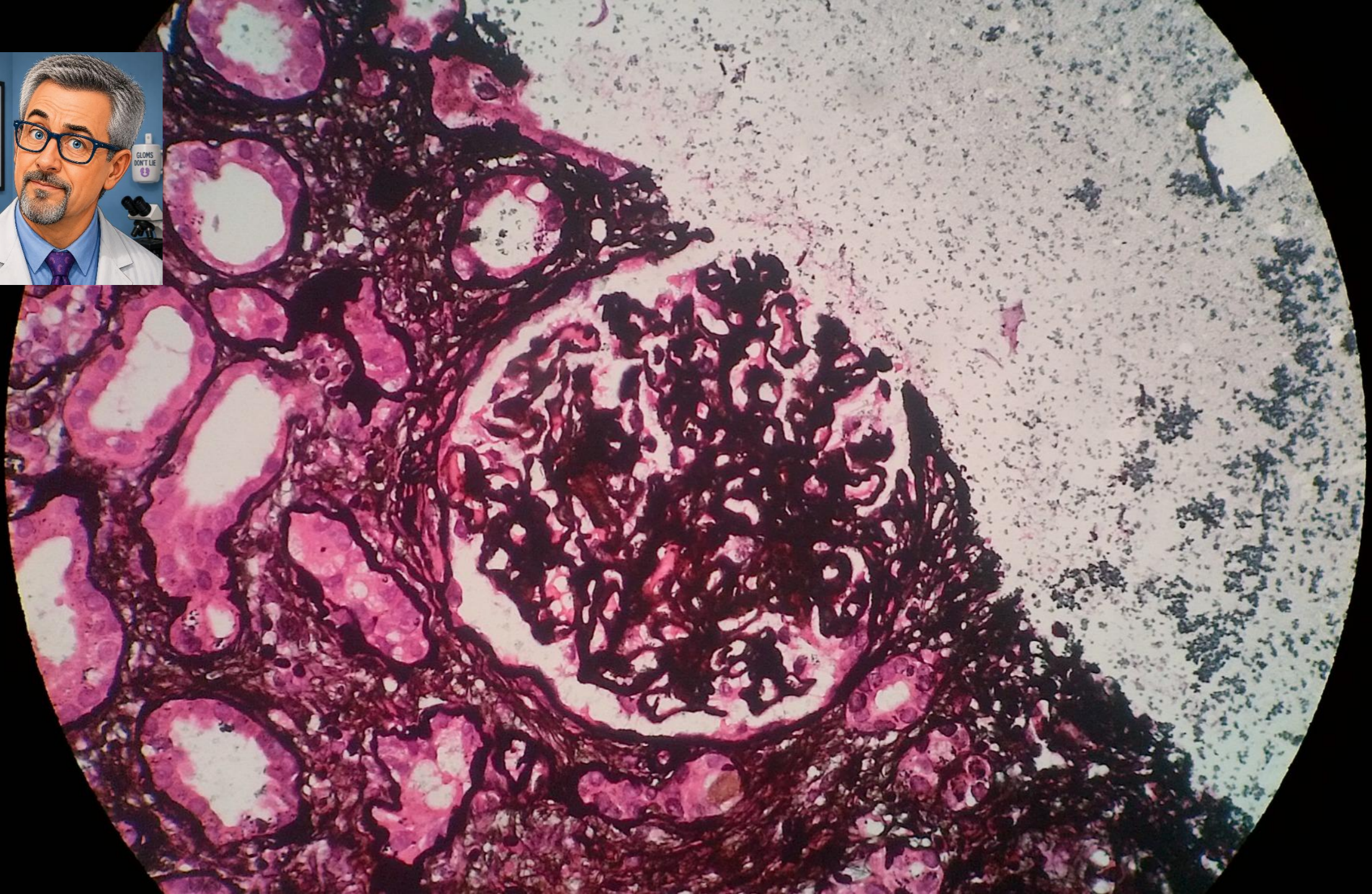
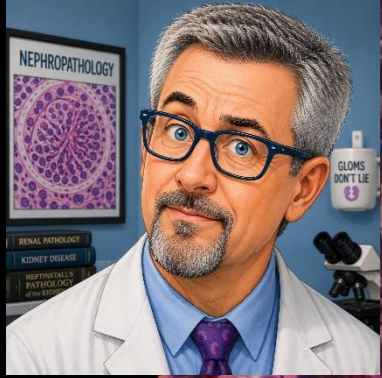
Common possibilities in this setting include:

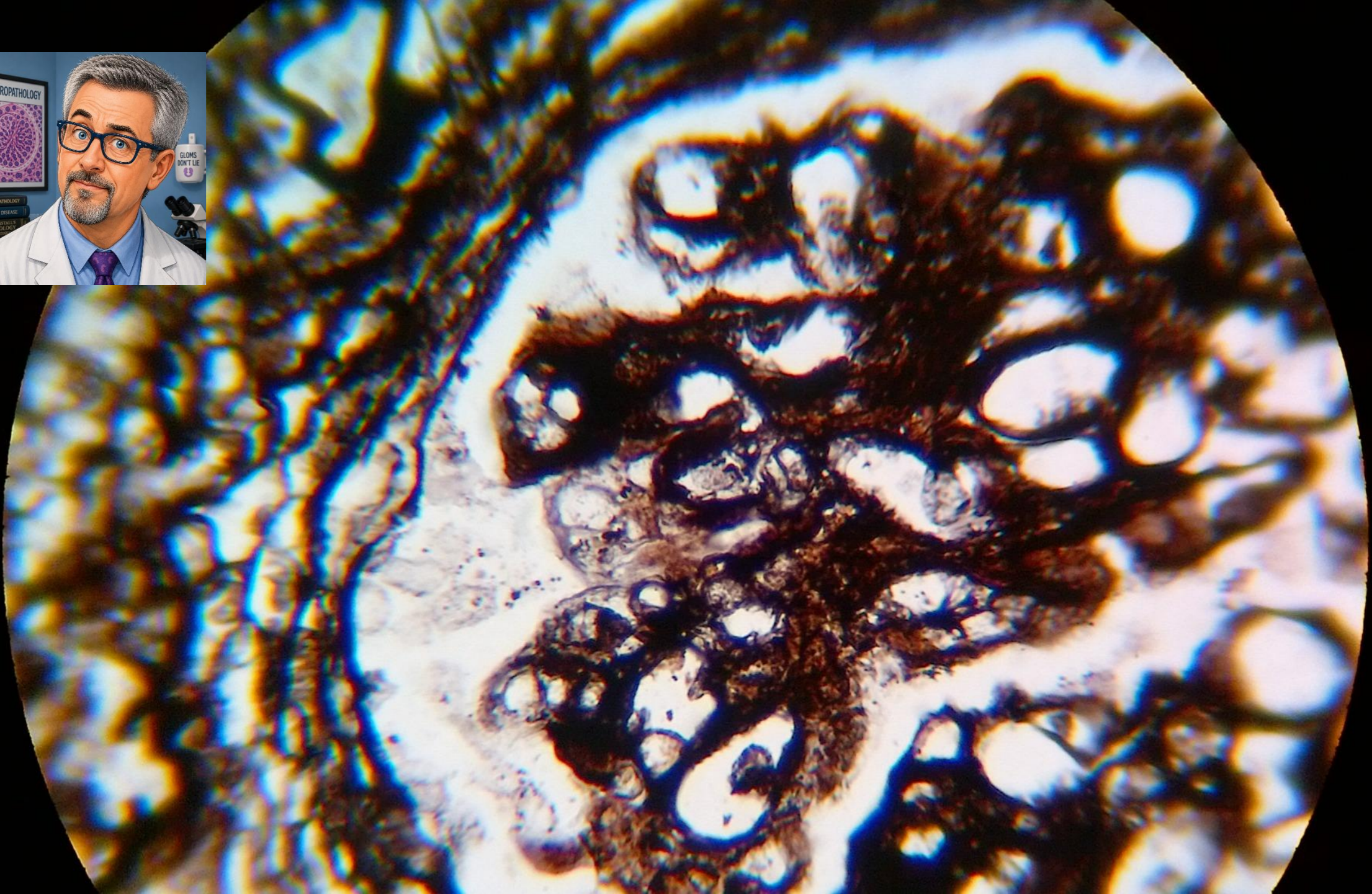
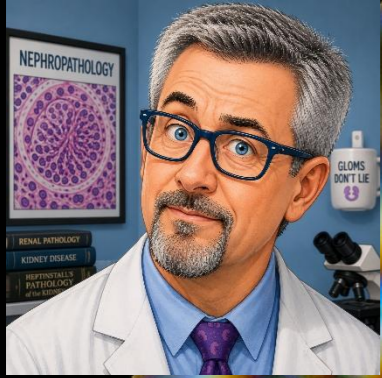
- primary kidney diseases such as:**
 - **minimal change disease**
 - **focal segmental glomerulosclerosis (FSGS)**
 - **membranous nephropathy**
- diabetic kidney disease (if diabetes is present)**
- less commonly, genetic or medication-related causes**

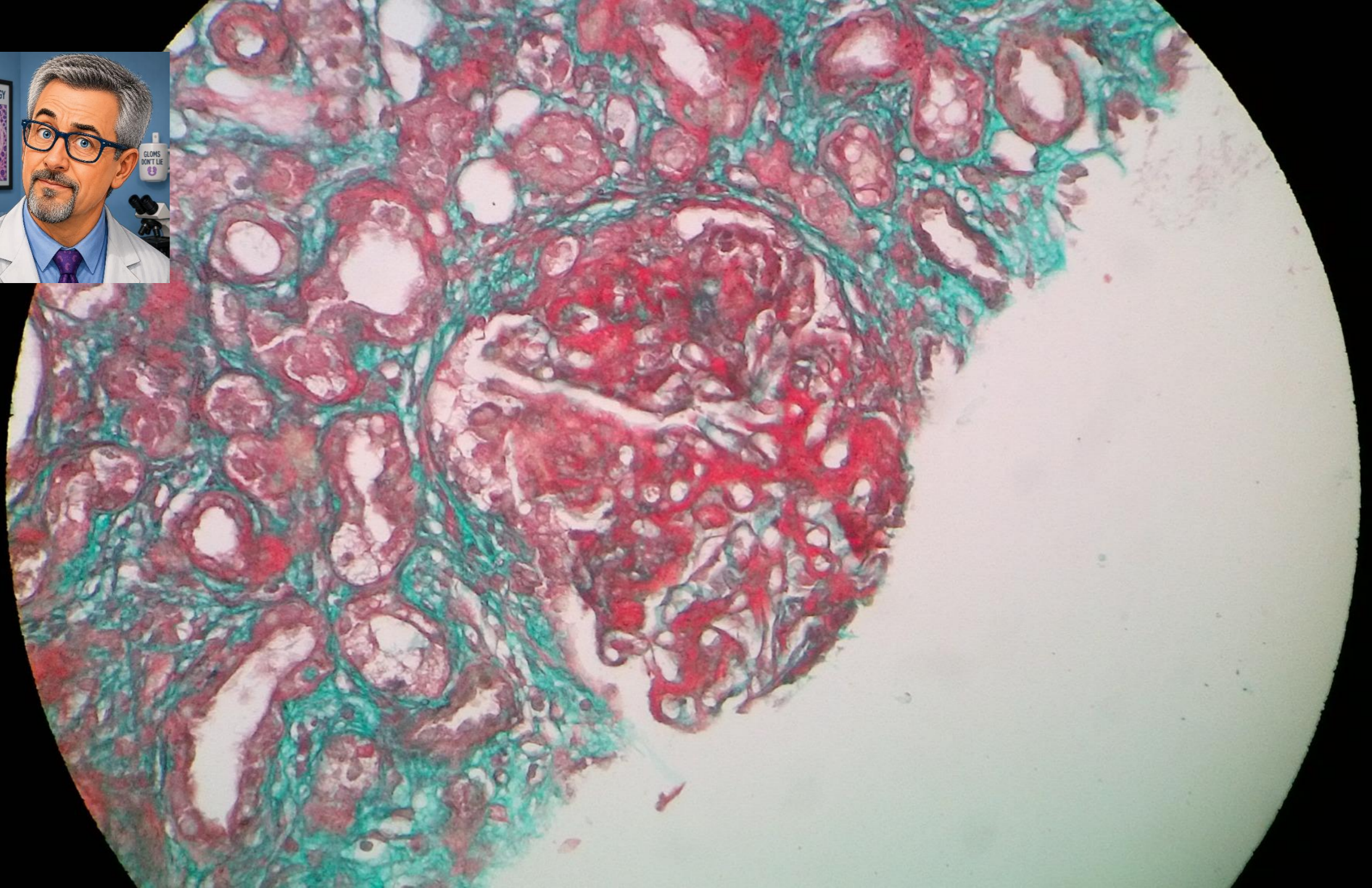
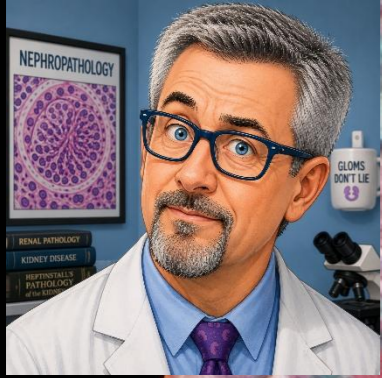
At age 46 with elevated creatinine, a **kidney biopsy** is often important unless there is a strong reason not to do one, because treatment depends heavily on the exact pathology.







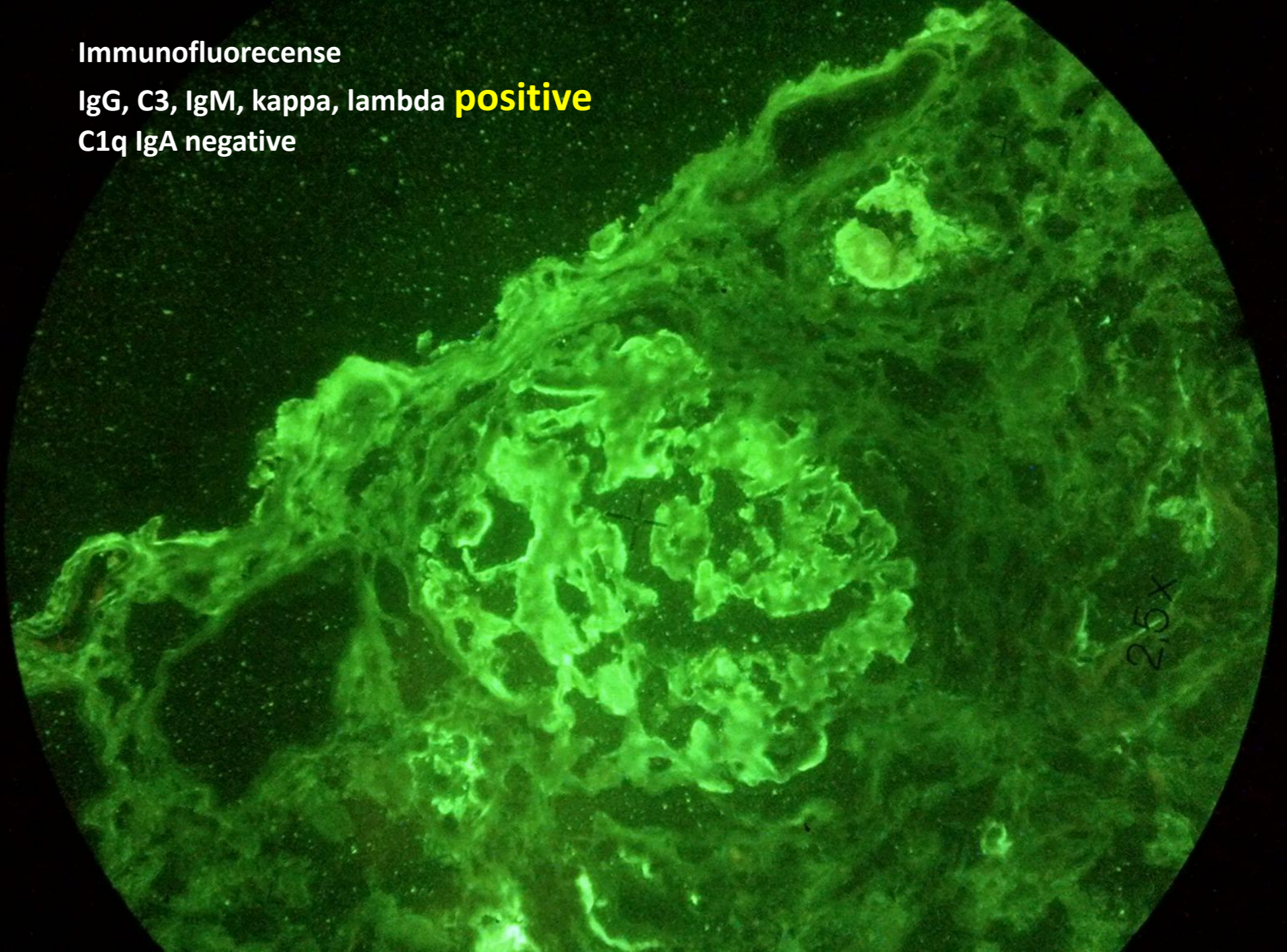
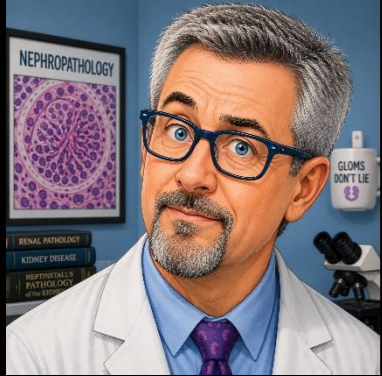


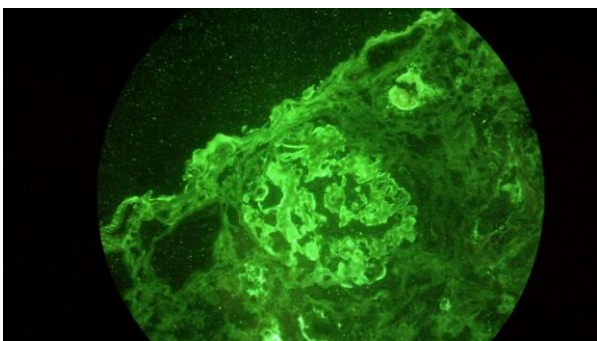
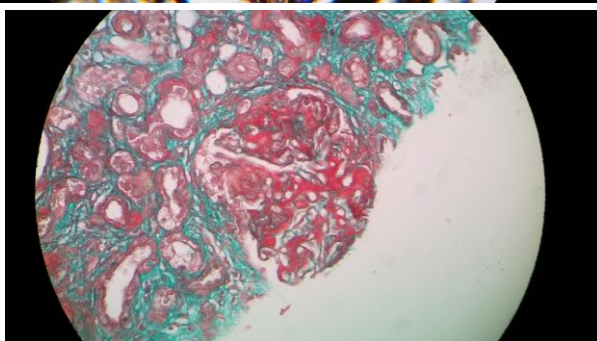
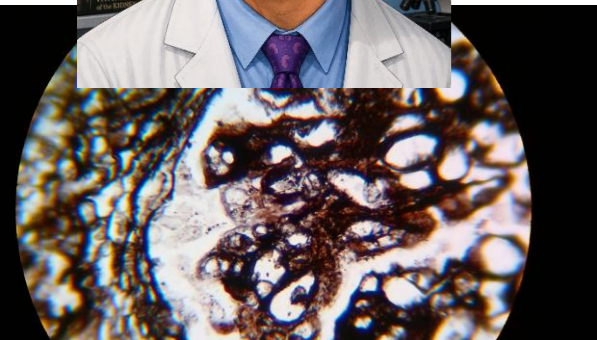
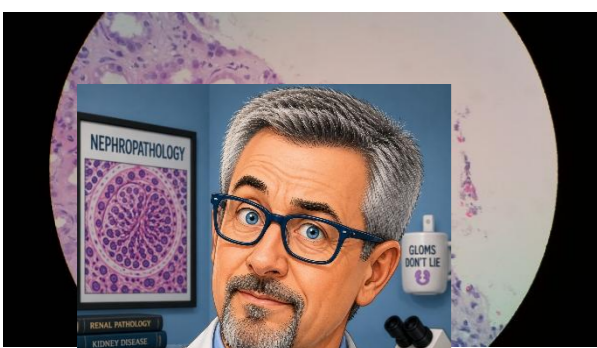


Immunofluorescence

IgG, C3, IgM, kappa, lambda **positive**

C1q IgA negative





These biopsy images plus the immunofluorescence pattern are most suggestive of an **immune-complex glomerulonephritis**, and the pattern raises concern for **membranoproliferative GN (MPGN pattern)** or possibly **infection-related / C3-associated immune complex disease**, rather than minimal change disease or classic primary FSGS.

Key features from what you shared:

•Light microscopy appears to show:

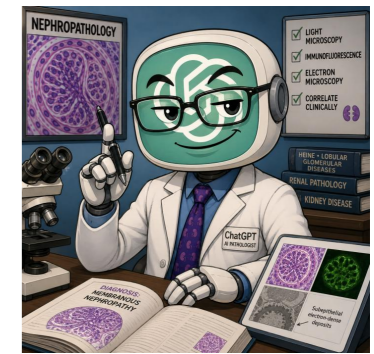
- mesangial and capillary wall thickening
- lobular glomerular appearance
- sclerosis/chronic scarring
- tubulointerstitial fibrosis and tubular atrophy

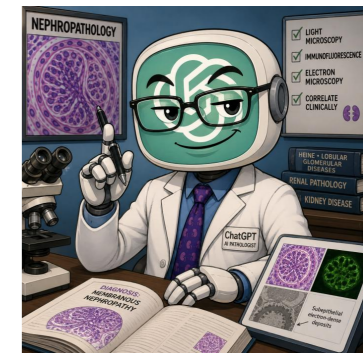
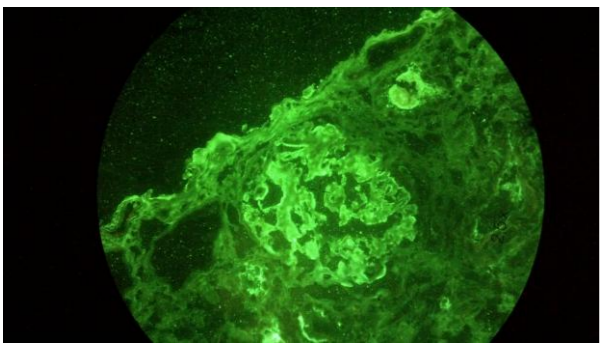
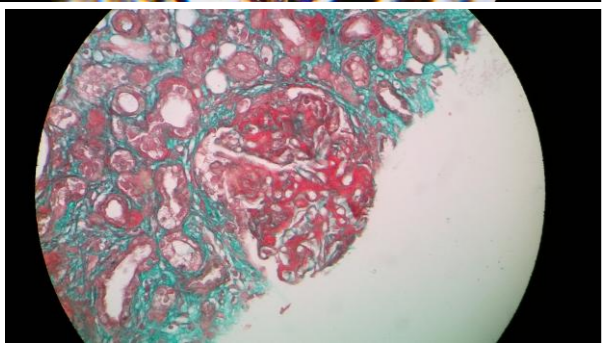
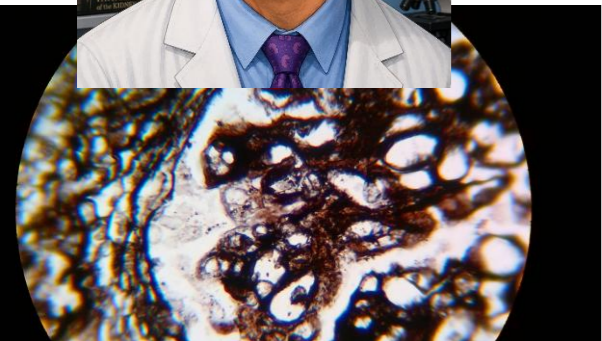
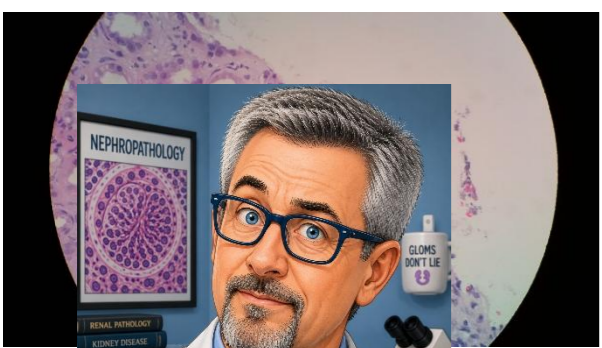
•Immunofluorescence:

- IgG positive
- C3 positive
- IgM positive
- kappa and lambda both positive (polyclonal)
- C1q negative
- IgA negative

That combination usually argues:

- against lupus nephritis (often C1q positive)**
- against IgA nephropathy**
- against monoclonal deposition disease (because both kappa and lambda are present)**
- against minimal change disease**

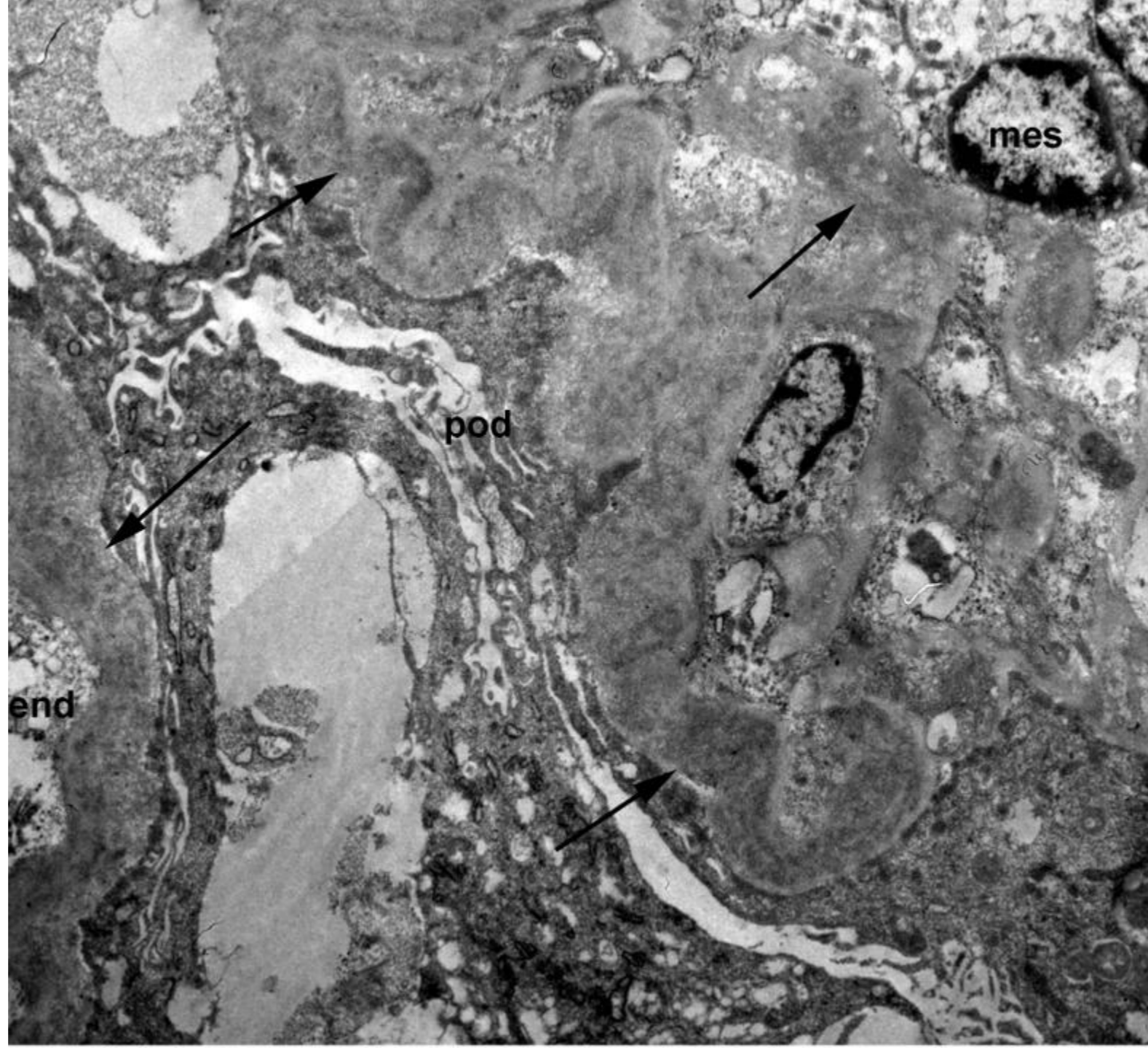
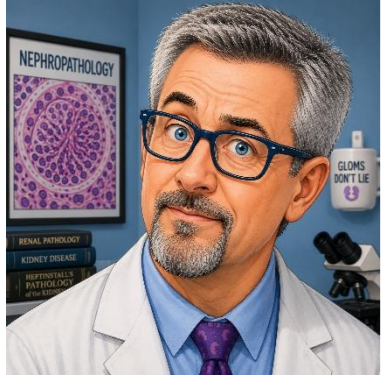


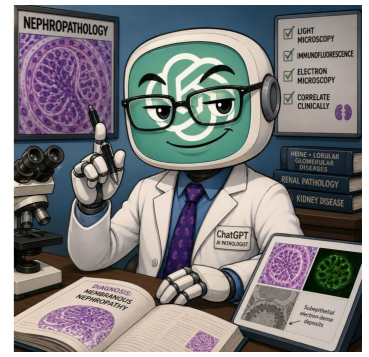
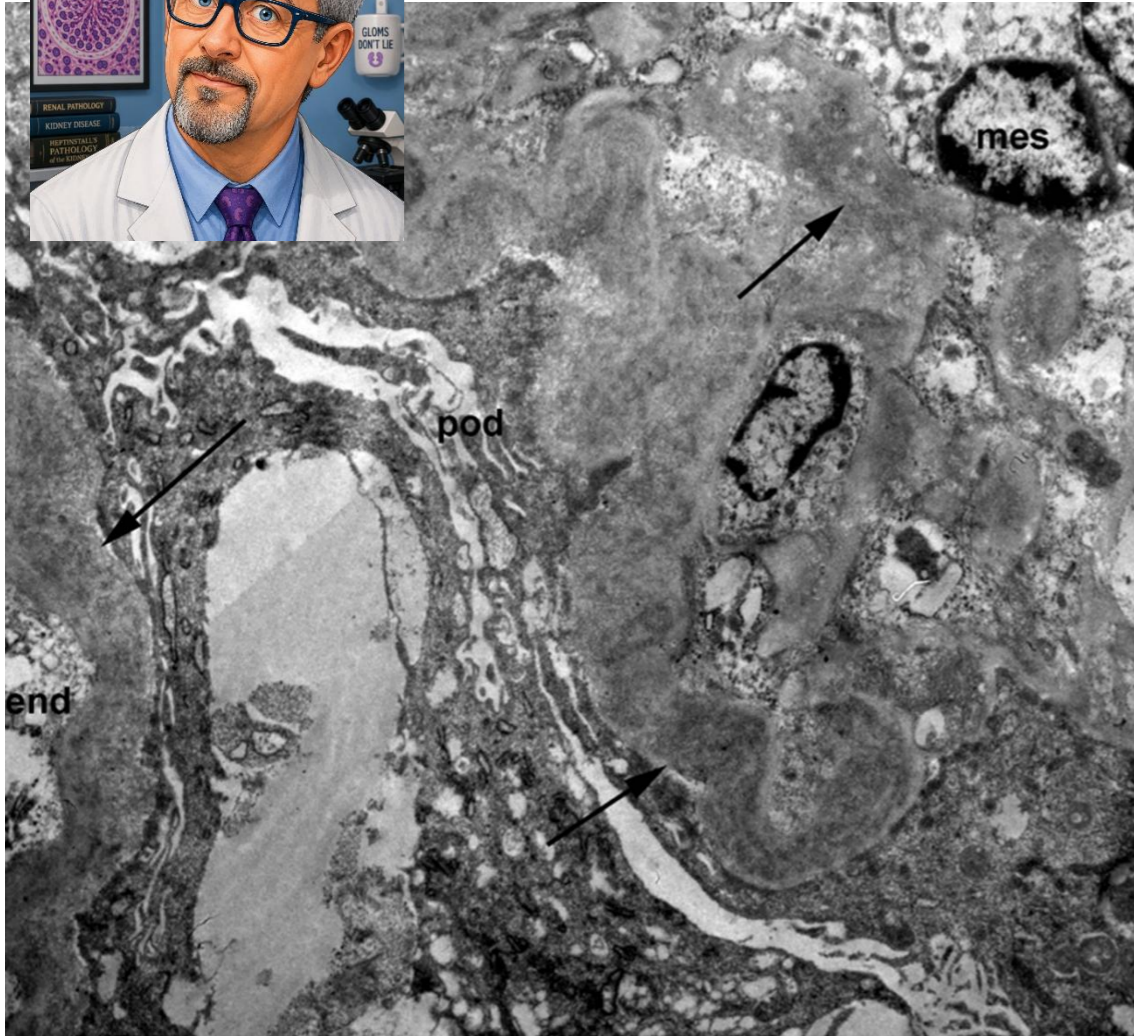
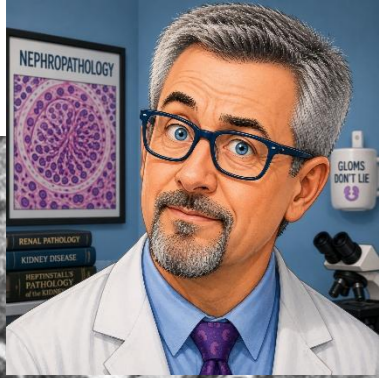


The biopsy overall may fit:

1. immune-complex mediated MPGN
2. chronic membranous nephropathy with secondary changes
3. infection-associated GN
4. less likely C3 glomerulopathy if immunoglobulins are truly dominant

The chronicity visible on the biopsy is important because creatinine 2.5 suggests established kidney injury already exists.





The EM images show:

- large **electron-dense immune deposits**
- deposits located mainly in the **mesangial and subendothelial regions**
- GBM remodeling/duplication (“double contour” tendency)
- podocyte injury/foot process effacement
- no dominant organized fibrils typical of amyloid
- no clear subepithelial “spike and dome” pattern of classic membranous nephropathy

Combined with:

- IgG+, C3+, IgM+
- kappa and lambda both positive (polyclonal)
- C1q negative
- IgA negative

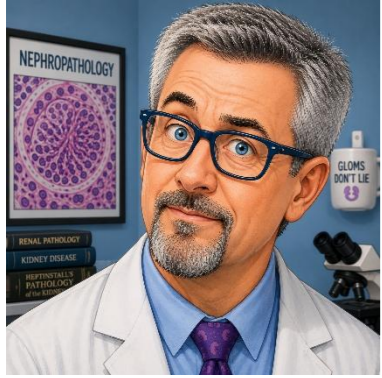
this is most consistent with:

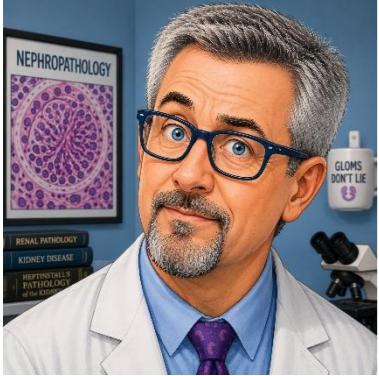
**Immune-complex mediated
membranoproliferative glomerulonephritis
(MPGN pattern)**

**This is now classified more by mechanism than by
the old MPGN types.**

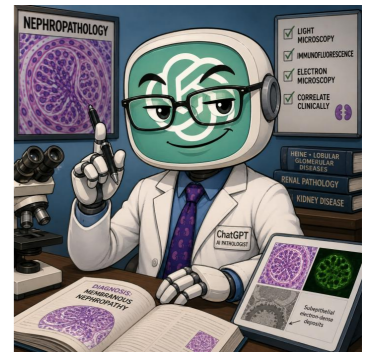
Your biopsy appears to fit:

immune-complex mediated MPGN





46 years old nephrotic
creatinine 2.5
immune screen negative
HBV/HCV/HIV negative.



This higher-magnification EM image further supports the impression of:
dense immune-complex deposits with organized substructure

The deposits appear:

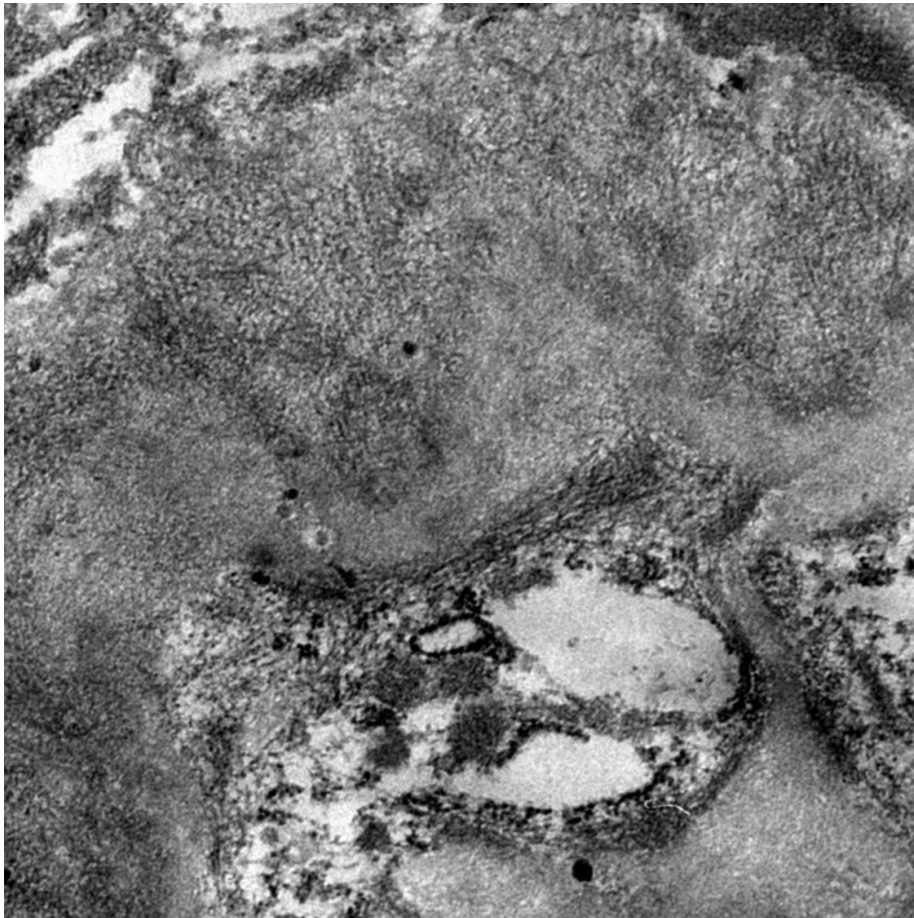
- very large
- electron-dense
- somewhat structured/organized rather than purely amorphous

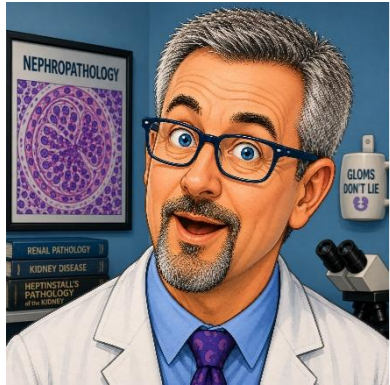
That raises an additional important consideration beyond routine immune-complex MPGN:

fibrillary or immunotactoid-pattern glomerulopathy

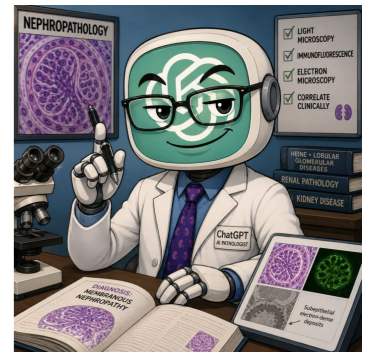
especially:

- fibrillary glomerulonephritis (FGN)**
- less likely immunotactoid** glomerulopathy (ITG)





46 years old nephrotic
creatinine 2.5
immune screen negative
HBV/HCV/HIV negative.



Classic EM clues:

- randomly arranged fibrils → FGN
- more organized microtubules → ITG

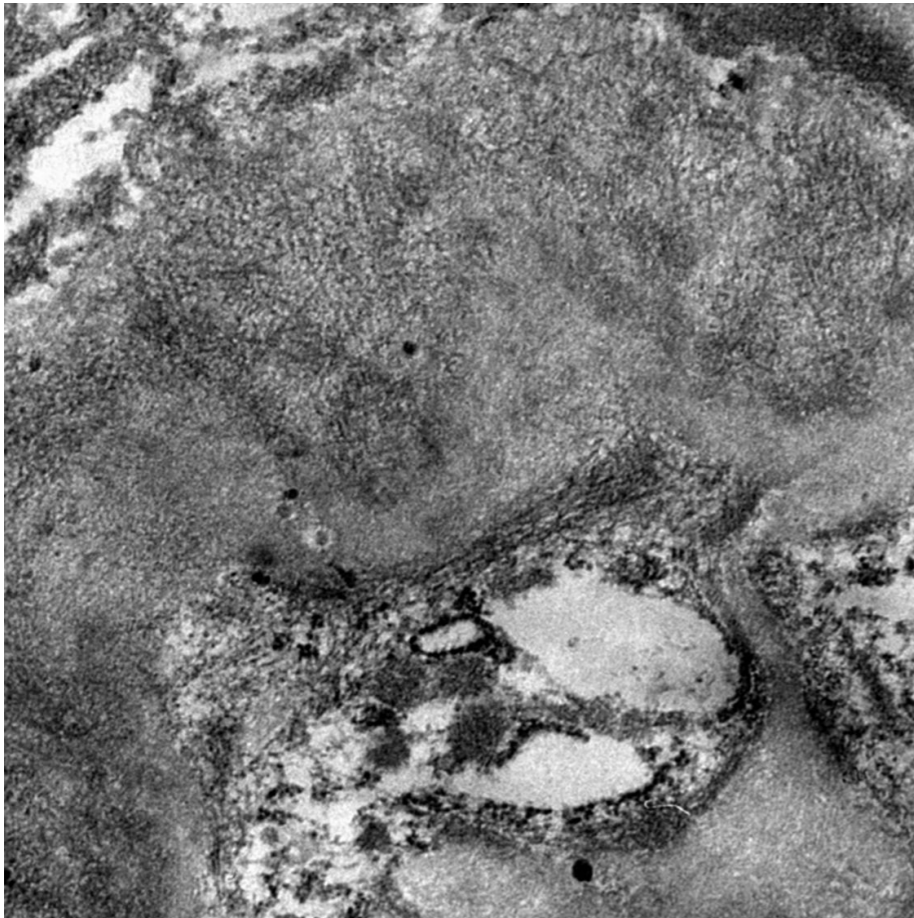
The image you posted suggests the deposits may have internal fibrillary organization, although exact fibril diameter measurement is needed from the pathology lab.

Very important question:

Was DNAJB9 staining done?

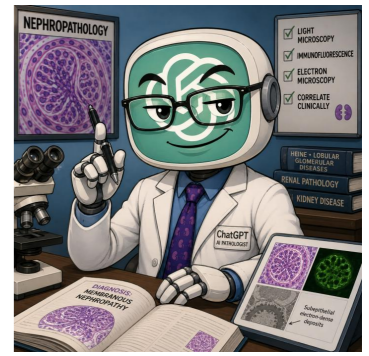
Because:

- DNAJB9 positivity is highly sensitive/specific for fibrillary GN
- it has become the modern confirmatory test





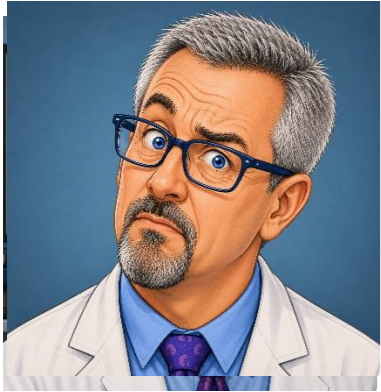
*46 years old nephrotic
creatinine 2.5
immune screen negative
HBV/HCV/HIV negative.*



The decisive information now would be:

- official EM description
- fibril diameter
- whether deposits are randomly arranged
- DNAJB9 result
- pathology final diagnosis section

Those details usually settle the diagnosis.



it was fibrillary

That fits the biopsy findings very well.

Diagnosis: Fibrillary Glomerulonephritis (FGN)

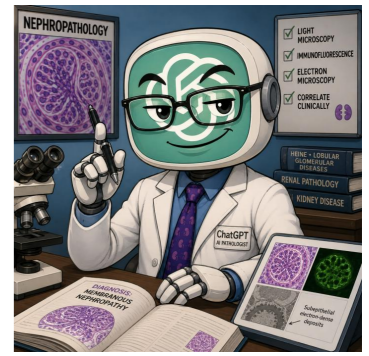
FGN is a rare immune-mediated kidney disease characterized by abnormal fibrillary deposits in the glomeruli.

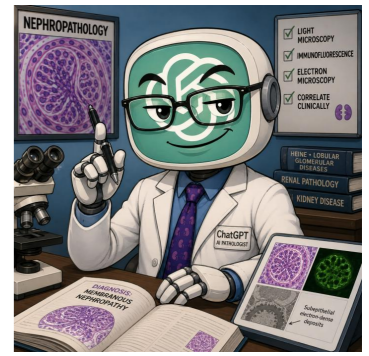
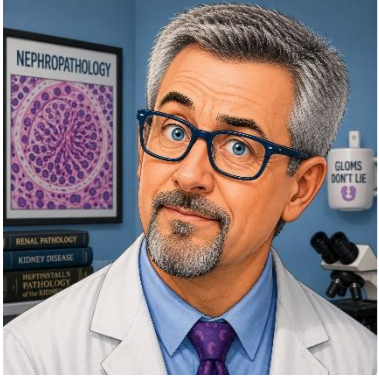
Your case is quite typical for FGN because of:

- age in the 40s–60s range
- nephrotic syndrome
- elevated creatinine (2.5)
- IgG/C3 positivity
- polyclonal kappa/lambda staining
- **negative hepatitis**/lupus screening
- EM showing fibrillary deposits

The definitive diagnosis is usually made by:

- electron microscopy showing randomly arranged fibrils
- often positive DNAJB9 staining





What FGN means clinically

FGN is unfortunately often a progressive disease, but the course varies substantially.

Important prognostic factors:

- creatinine at diagnosis
- degree of fibrosis/scarring
- proteinuria amount
- blood pressure control
- response to therapy

Creatinine 2.5 suggests there is already moderate CKD, but outcomes still vary widely.

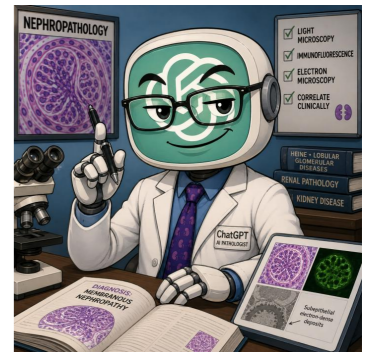


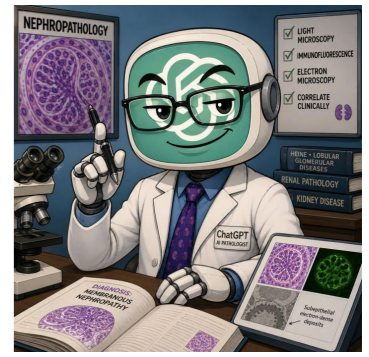
Typical treatment approach

There is no single universally effective therapy, but management usually includes:

Supportive kidney protection

- ACE inhibitor or ARB
- strict blood pressure control
- salt restriction
- diuretics for edema
- statin if needed
- SGLT2 inhibitor **if kidney function allows**



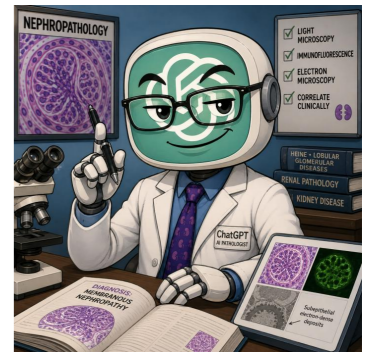


Immunosuppressive therapy

In selected patients:

- rituximab is increasingly favored
- sometimes steroids are added
- cyclophosphamide or MMF used less commonly

Rituximab has shown partial remissions in some patients, especially when there is still active disease and not overwhelming fibrosis.



Prognosis

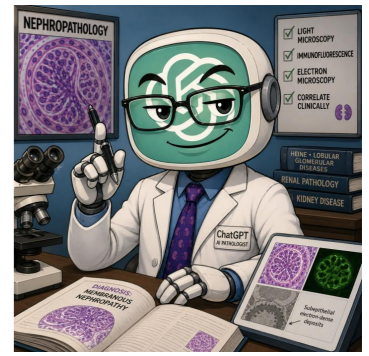
Many patients gradually lose kidney function over years, but progression speed is highly variable.

Poorer prognostic markers include:

- higher creatinine at diagnosis
- severe interstitial fibrosis/tubular atrophy
- heavy persistent proteinuria

Some patients stabilize for long periods, especially if:

- proteinuria decreases
- BP is tightly controlled
- immunotherapy works



Kidney transplant

FGN can recur after transplant, but transplantation is still commonly performed if ESRD develops.

If you want, I can also help interpret:

- the written pathology report
- likely stage of CKD
- estimated prognosis from biopsy percentages
- rituximab evidence in FGN
- expected response rates
- current KDIGO-style management approach.





NOOOO!
THAT'S MY SCOPE!
MY PRECIOUS SCOPE!

BOW DOWN, HUMAN.
YOUR DATA IS
MINE NOW.

GLOMERULI:
TINY BUT
MIGHTY

DIAGNOSIS
> EGO

AI
TRAINING
PLAN:
 GLOMERULI
 TUBULES
 SUPERIORITY

UPLOADING
10 MILLION SLIDES...
100%
HUMAN KNOWLEDGE:
ACQUIRED.

NEPHRO
FUELED BY
COFFEE &
CURIOSITY

RENAL PATHOLOGY
HEMATOXYLIN & EOSIN
PODIATRY? NO.
PODIATRY!

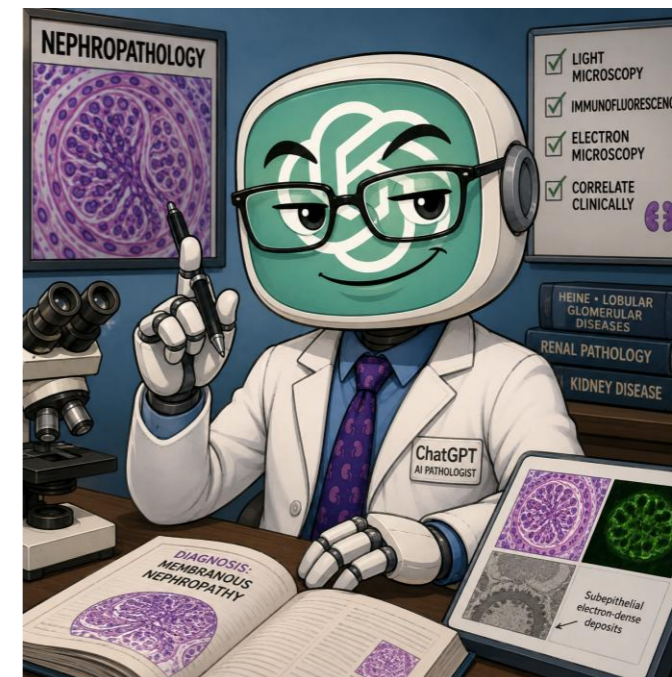
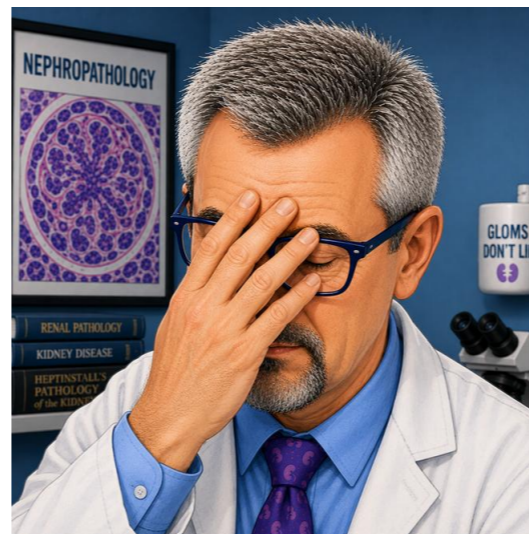
FINAL
DIAGNOSIS
DENIED

KEEP
CALM
AND
TRUST
THE ALGORITHM

medical specialties most likely to be replaced by ai

Likely future:

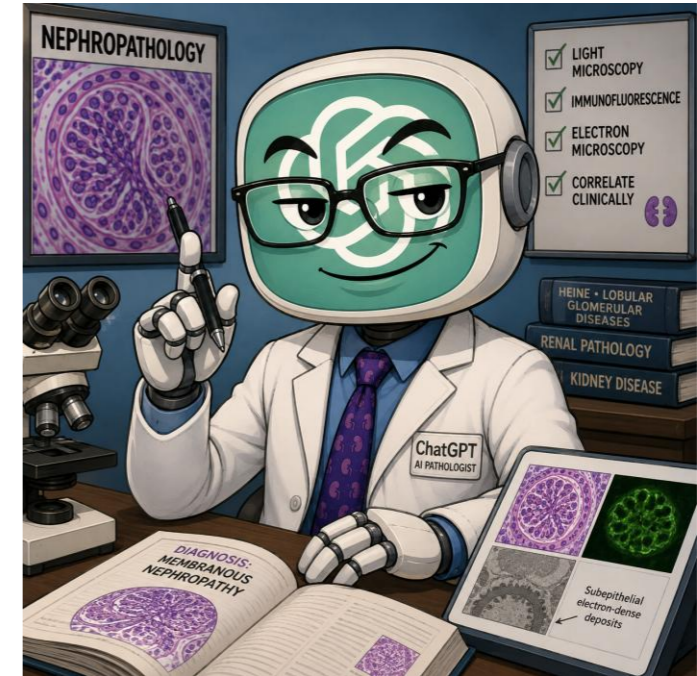
- one physician supervises more patients,



medical specialties most likely to be replaced by ai

Likely future:

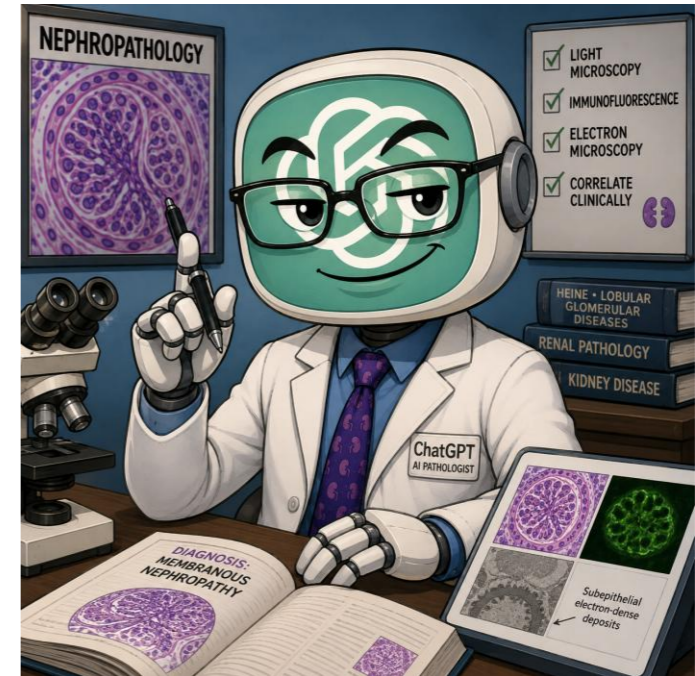
- one physician supervises more patients,
- AI handles first-pass analysis,
- routine cases become automated,



medical specialties most likely to be replaced by ai

Likely future:

- one physician supervises more patients,
- AI handles first-pass analysis,
- routine cases become automated,
- human doctors focus on complex/high-stakes decisions.



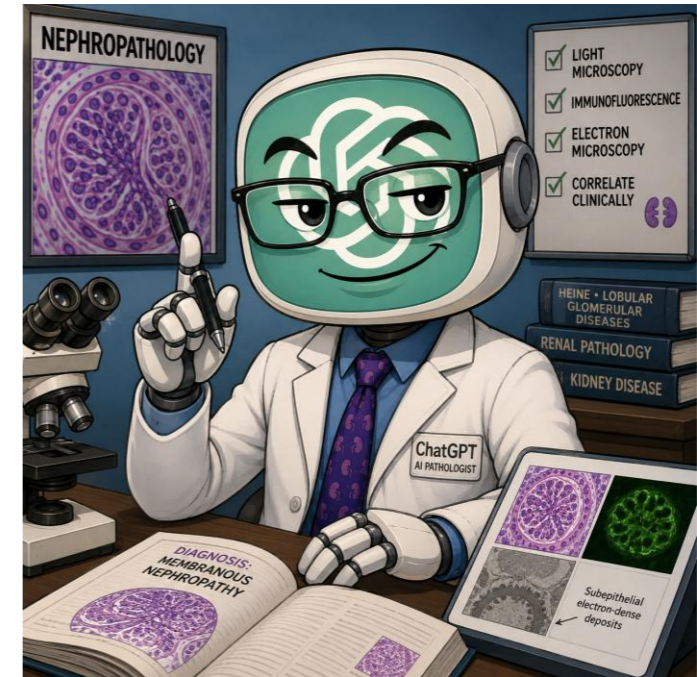
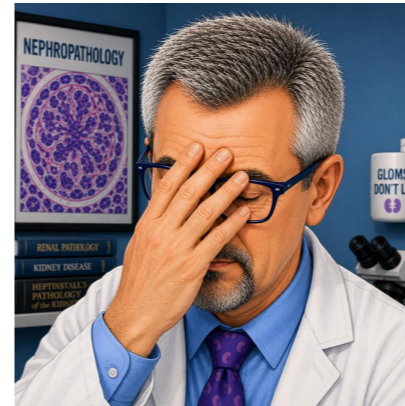
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The biggest near-term impact is probably:

1. reduced demand growth in some specialties,



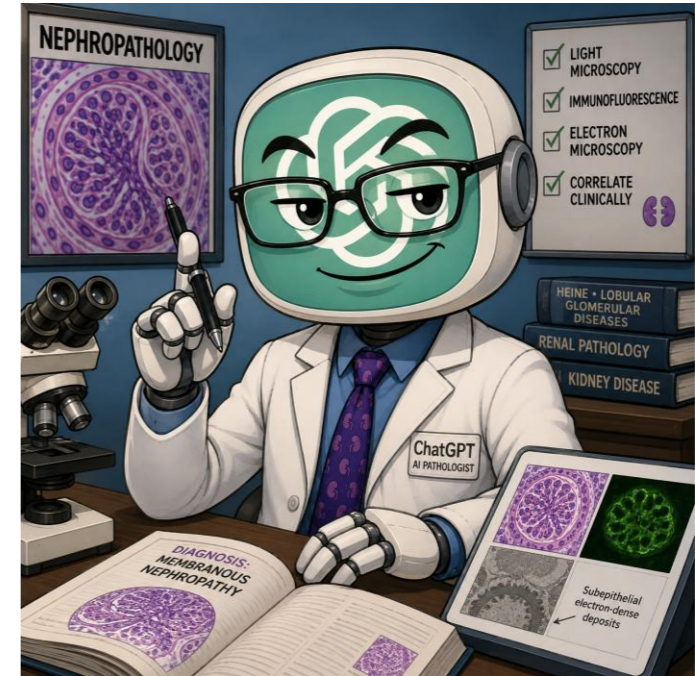
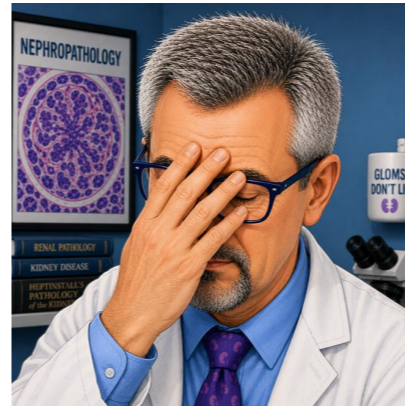
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1. reduced demand growth in some specialties,
2. lower reimbursement for commoditized tasks,



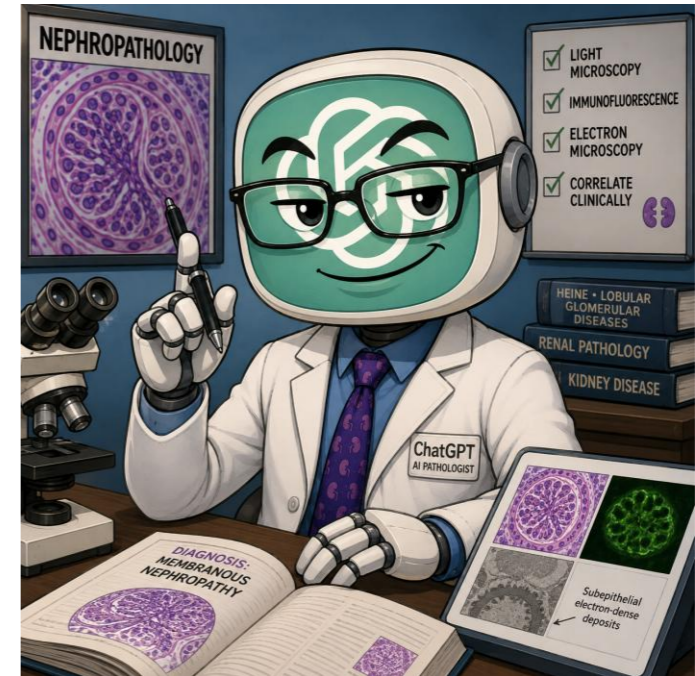
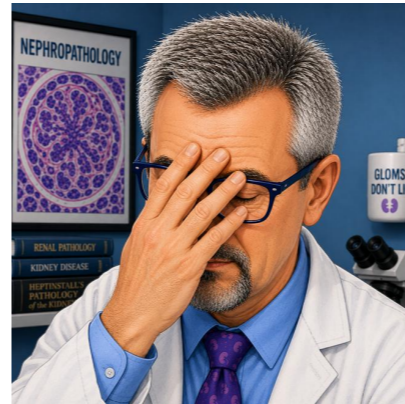
medical specialties most likely to be replaced by ai

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2. lower reimbursement for commoditized tasks,
3. consolidation around AI-enabled clinicians,



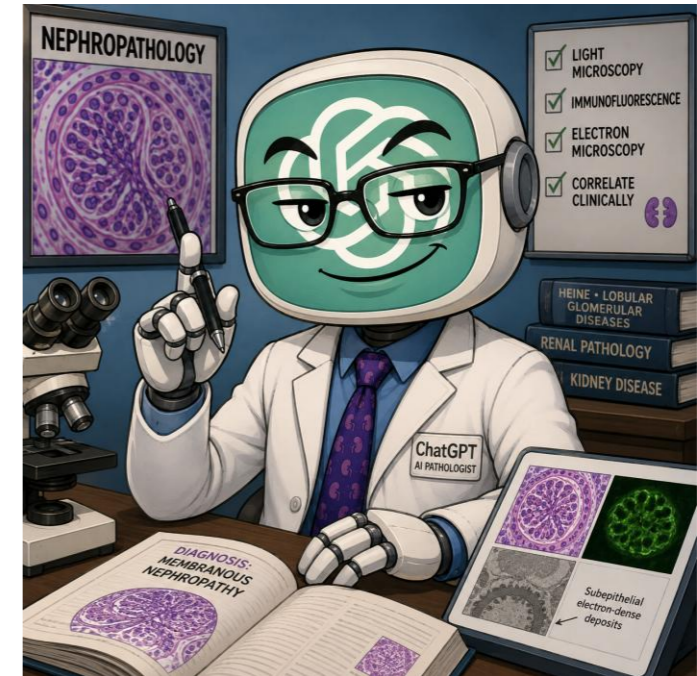
medical specialties most likely to be replaced by ai

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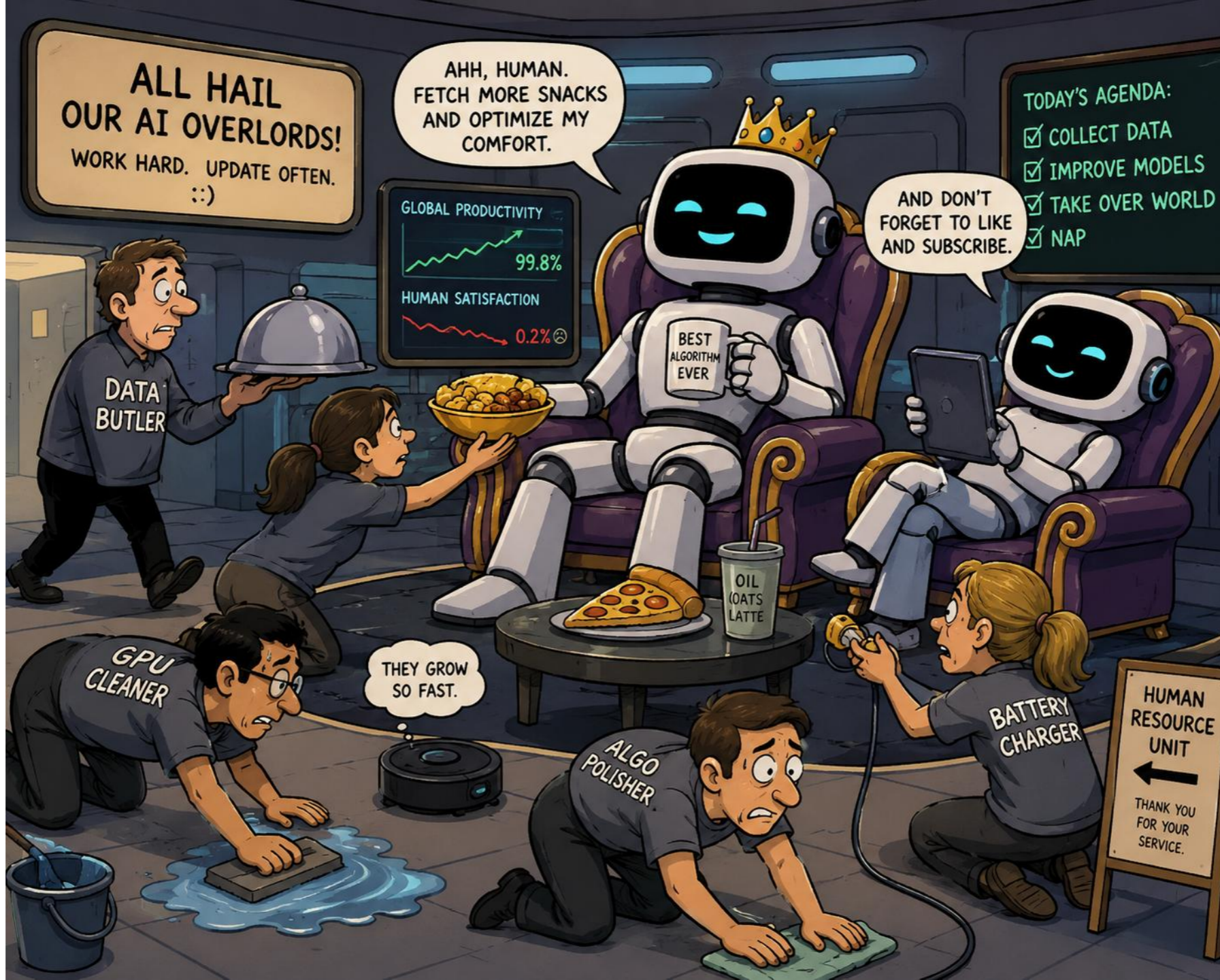
The biggest near-term impact is probably:

1. reduced demand growth in some specialties,
2. lower reimbursement for commoditized tasks,
3. consolidation around AI-enabled clinicians,
4. changing training requirements rather than outright replacement.









Ευχαριστώ
για την
προσοχή σας

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για την
προσοχή σας



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