Glomerulonephritis Associated with Bacterial Infection

The Emerging Role of Staphylococcus

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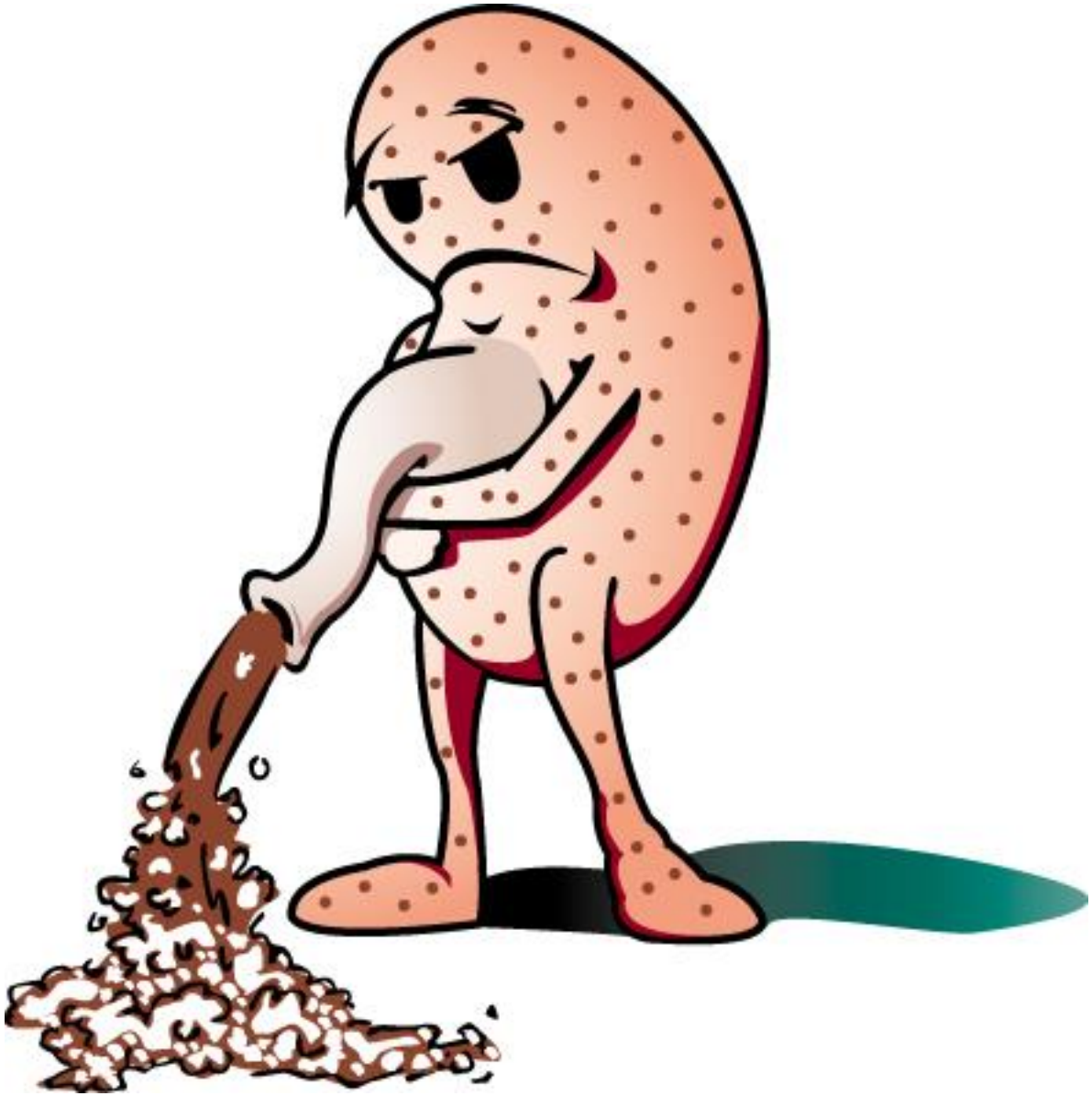
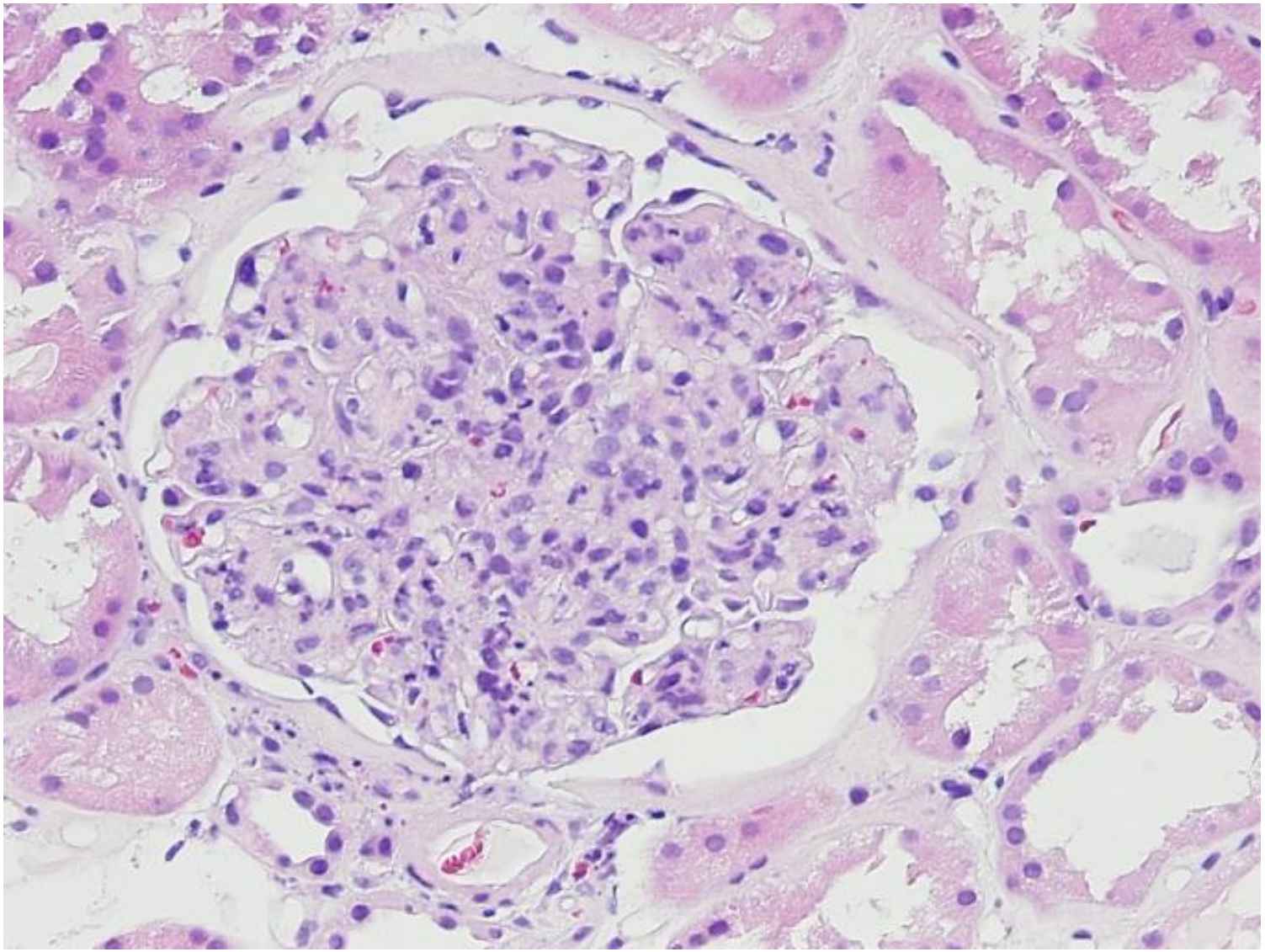


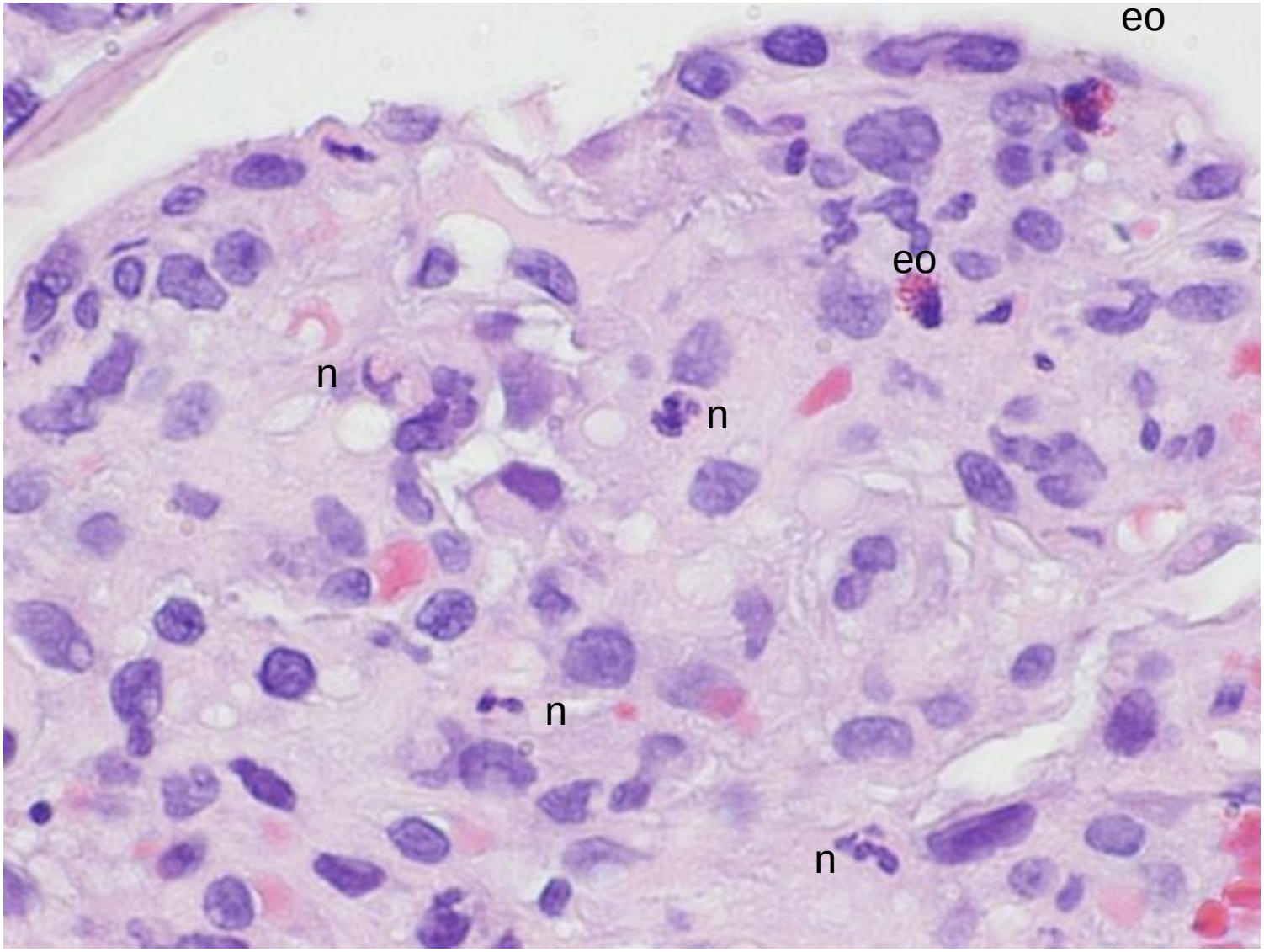
Clinical history (Case #1)

- 60-year-old Caucasian male with insulin dependent DM, diabetic neuropathy, CAD, HTN, obesity.
- Presented with gangrene of left great toe.
- Blood and wound cultures positive for methicillin sensitive Staphylococcus aureus.
- Zosyn and vancomycin were started.
- After two days, amputation of the left great toe was performed.
- After surgery, he became oliguric (425 ml per day) and over 6 days, serum cr. increased from 1.1 mg/dl to 3.8 mg/dl.
- Active urine sediment, Urine pr/cr 3.2, serum complement levels normal

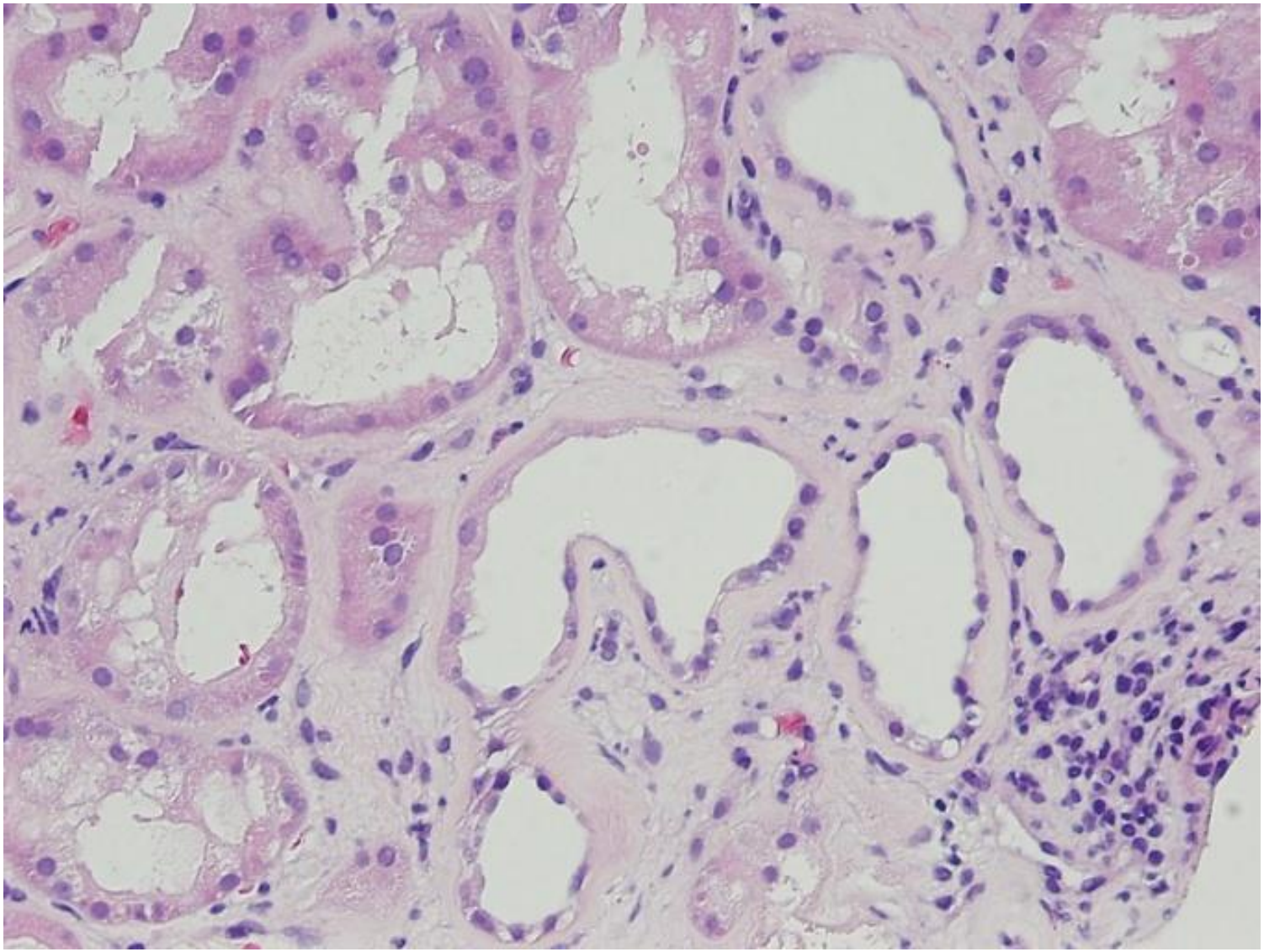
Glomerular endocapillary hypercellularity



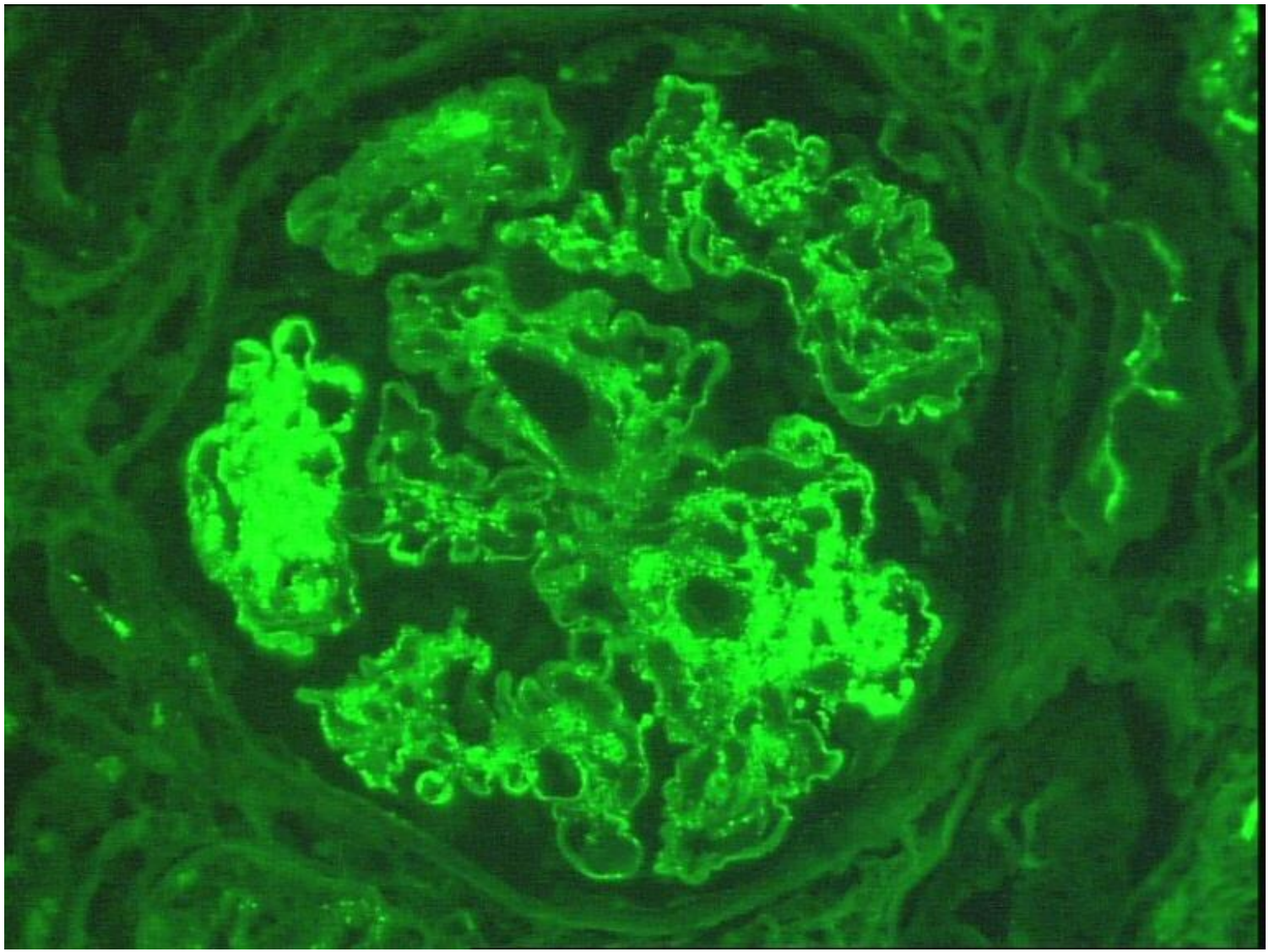
Glomerular capillaries contain inflammatory cells.
Note neutrophil granulocytes [n] and eosinophil granulocytes [eo].



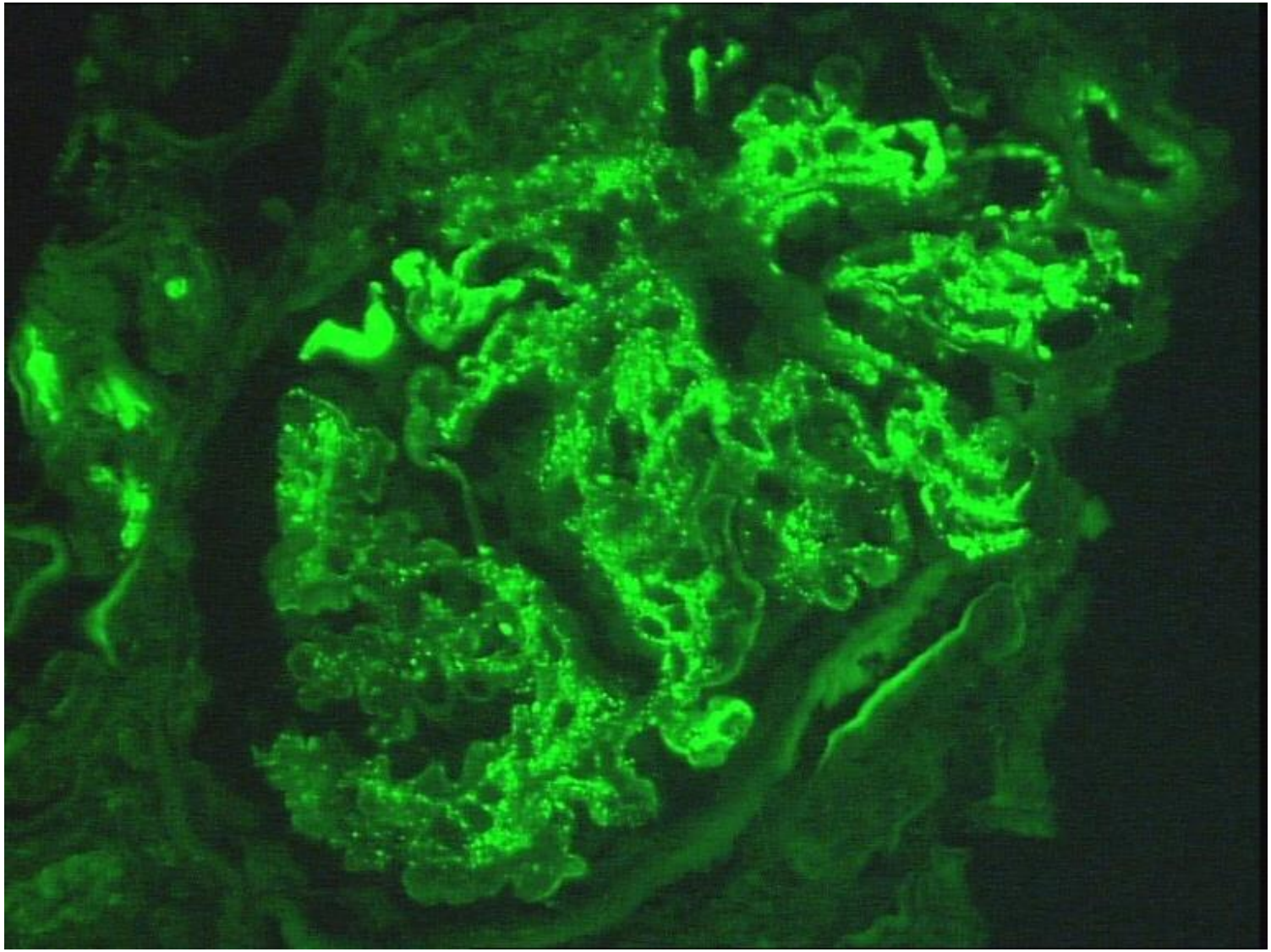
Acute tubular necrosis, affected tubules are lined by flat, irregular epithelium



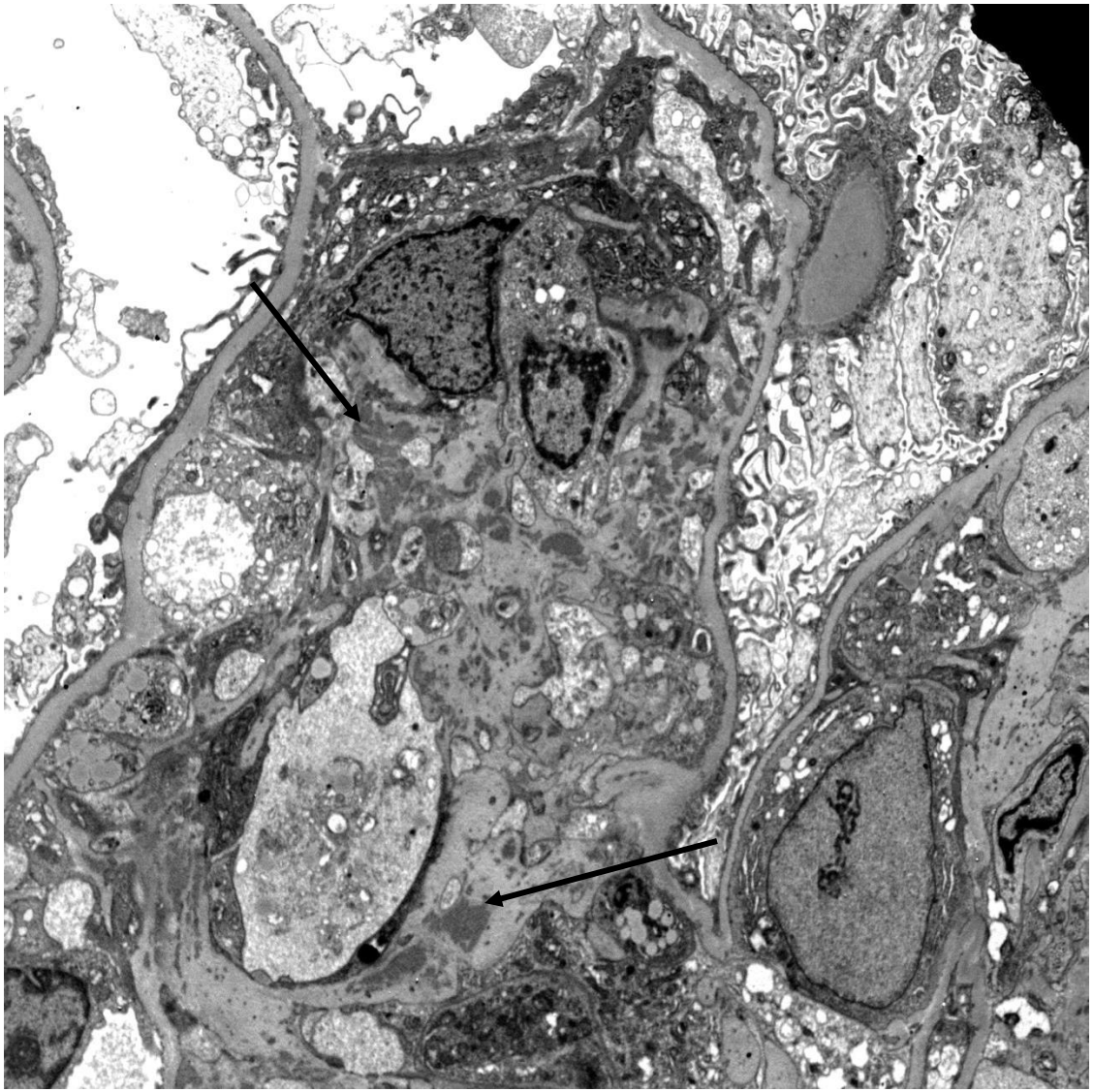
Strong primarily mesangial granular staining for IgA



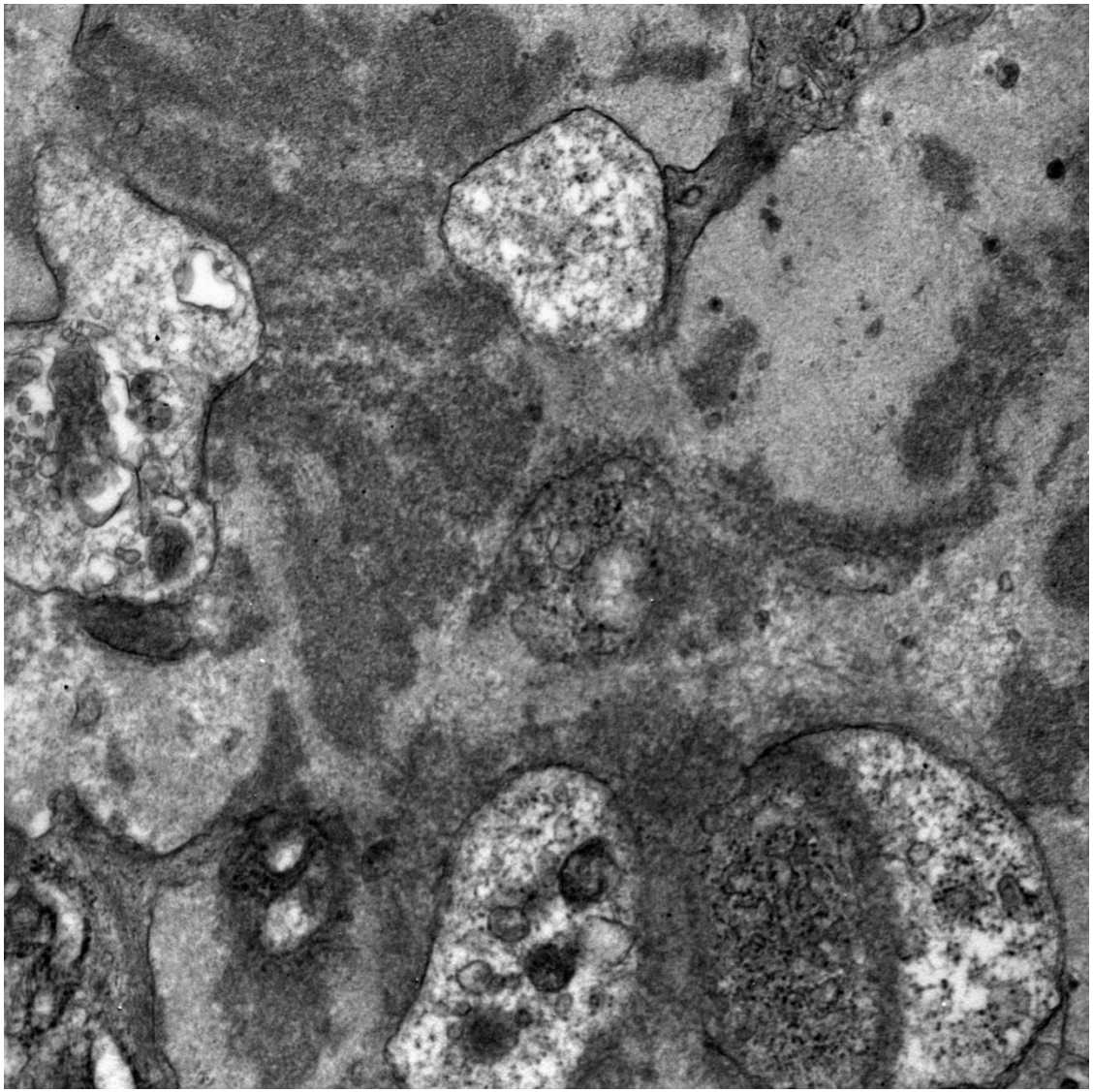
Immune complex deposits also stain for C3



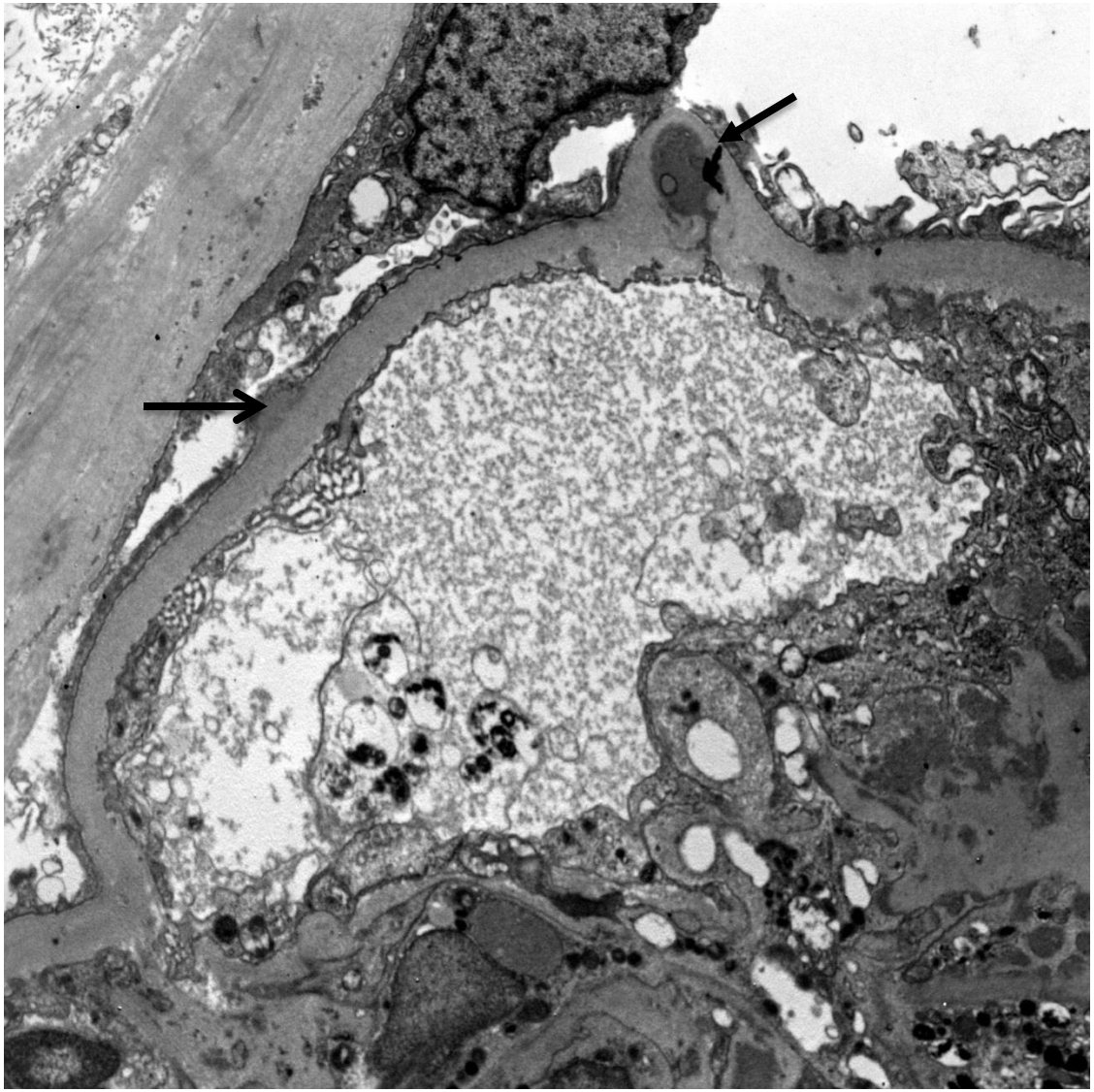
Expanded mesangium with electron dense immune-type deposits (arrows)



Mesangial electron dense immune-type deposits



Few scattered immune-type deposits along the glomerular capillary loops, slightly thickened GBM



Diagnosis

- Active endocapillary proliferative glomerulonephritis with IgA and C3-containing immune complex deposits, consistent with *Staphylococcus aureus* infection associated glomerulonephritis (SAIGN).
- Moderate diabetic nephropathy with mainly diffuse diabetic glomerulosclerosis.

Follow-up

- Infection was successfully treated, renal function slowly improved
- Follow-up after 4 years and 5 months:
  - Serum creatinine stable at 2.0 mg/dl.
  - Blood pressure controlled.
  - Lasix daily, with low salt diet

Bacterial infections and glomerulonephritis

Post-infectious /post-streptococcal glomerulonephritis

"Post" – after infection resolves with or without anti-microbial treatment

Latent period of 1 to 4 weeks with normal state of health

Acute onset of glomerulonephritis

Glomerulonephritis with active ongoing infection

Staphylococcus infection associated glomerulonephritis (usually IgA codominant or dominant)

Glomerulonephritis with other persistent infections

- Endocarditis
- Deep seated abscesses
- Shunt nephritis

Table 3. Infectious agents (109 patients)

Infectious agent[a]	No. of Patients (%)
Staphylococcus	50 (46)
Streptococcus[b]	17 (16)
E. coli	5 (5)
Pseudomonas	2 (2)
Acinetobacter	1 (1)
Serratia marcescens	1 (1)
Proteus	1 (1)
Klebsiella	1 (1)
Enterobacter cloacae	1 (1)
Candida	1 (1)
Unknown	37 (34)

[a]In seven patients, cultures grew two or three bacteria (staphylococcus and *E. coli* in three; staphylococcus and enterococcus in one; staphylococcus and *S. marcescens* in one; *E. coli* and enterococcus in one; and staphylococcus, proteus, and pseudomonas in one).

[b]Including five patients with infection by enterococci (group D streptococci).

Nasr SH, et al. Post-infectious glomerulonephritis in the elderly. J Am Soc Nephrol. 2011;22:187-195.

Staphylococcus infection associated glomerulonephritis (SIAGN)

- In developed countries, the incidence of post-streptococcal GN has declined because of successful treatment of acute streptococcal infections.
- Staphylococcal infection-related glomerulonephritis is on the rise because
  - 1. Emerging drug-resistant strains of Staphylococcus and both nosocomial and community-acquired staphylococcal infections.
  - 2. The growing elderly population with increasing prevalence of comorbidities, such as diabetes and morbid obesity
- Infection is ongoing at the time the glomerulonephritis is diagnosed. <u>Therefore the term “post”-infectious is a misnomer and should not be used.</u>
- IgA-dominant or co-dominant immune complexes are commonly seen, posing a diagnostic pitfall with idiopathic IgA nephropathy (and Henoch-Schönlein purpura).

Characteristics of staphylococcus infection-associated GN (SIAGN)

- Culture-proven, ongoing Staphylococcus aureus infection (blood cultures are frequently negative) – commonly done first after the biopsy.
- Concomitant glomerulonephritis with acute kidney injury.
- Hematuria and proteinuria.
- Renal biopsy: diffuse mesangial and intracapillary hypercellularity (sometimes crescents and necrosis) frequently with ATN.
- IgA and C3 containing glomerular immune complexes by immunofluorescence.
- Mesangial, frequently also peripheral glomerular capillary electron dense immune-type deposits by electron microscopy (subepithelial humps may or may not be present – seen in approximately 30% of cases).

Diagnostic difficulties in SIAGN

- Bacterial culture results are unavailable in a large percentage of patients despite characteristic clinical presentation and with kidney biopsies showing histologic typical features.
- Most patients are elderly with multiple co-morbidities, often treated empirically with multiple antibiotics. Cultures may become negative.
- Blood cultures are negative in a large percentage of these cases. Cultures from the site of infection need to be taken.
- Also, clinical features of infection, in elderly patients can be subtle

Post-streptococcal versus SIAGN

Post-infectious /post-streptococcal glomerulonephritis

"Post" – after infection resolves with or without anti-microbial treatment

Latent period of 1 to 4 weeks with normal state of health

Acute onset of glomerulonephritis, usually supportive treatment; sometimes steroid treatment may help.

Biopsy:

LM: Proliferative GN

IF: C3 dominant granular deposits with or without IgG

EM: subepithelial humps, some mesangial deposits

Staphylococcus infection associated glomerulonephritis (IgA dominant)

Infection is active and ongoing when the glomerulonephritis develops

Therapy: Antibiotics, sometimes requires amputation or debridement of infected area. Avoid steroids!

Glomerulonephritis will resolve after infection is treated, provided underlying chronic kidney injury is not severe.

Biopsy:

LM: Variable degree (frequently mild) of glomerular proliferative changes;

IF: C3 and IgA dominant deposits.

EM mesangial and glomerular capillary deposits, sometimes humps

Common associations with SIAGN

- Predisposing Co-morbidities
  1. Diabetes Mellitus
  2. Post-surgery
  3. Post-trauma
  4. Prosthetic heart valves
  5. Intravenous drug use
  6. Hepatitis C
  7. Underlying malignancy

- Sites of Infection
  1. Diabetic foot ulcers
  2. Cellulitis
  3. Pneumonia
  4. Endocarditis
  5. Infected surgical sites
  6. Osteomyelitis.
  7. Visceral abscess
  8. Septic arthritis
  9. Infected pace-maker, heart valves, indwelling catheters, iv lines
  10. Infected abdominal mesh
  11. Dental infection
  12. Amputated limbs in diabetic patients

Experience at Ohio State University Medical Center

- During the period 2004 to 2016, out of 9,092 native kidney biopsies, 78 (0.94%) had culture proven SIAGN.
- At least 41 additional biopsies with typical histologic features and clinical presentation of SIAGN, but no documented culture results.

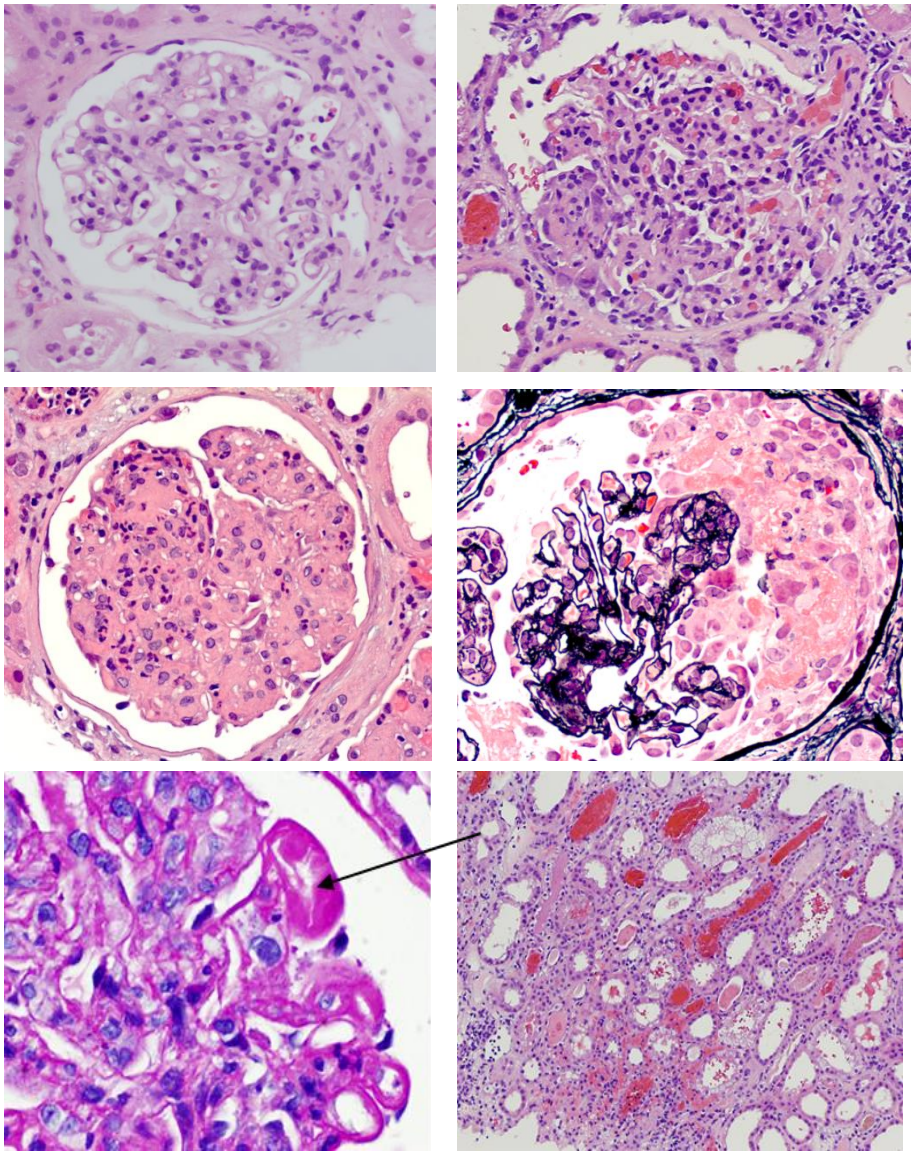
Table 1. Clinicopathologic characteristics of the 78 cases of culture positive Staphylococcal infection associated glomerulonephritis from 2004 to 2016 at the Ohio State University Medical Center.

Clinicopathologic features	n	%
Age (years)	55 +/- 12.1 (21-91)	
Ethnicity		
Caucasian	74	95%
African American	3	3.8%
Asian	1	1.2%
Gender		
Males	61	78%
Females	17	22%
Diabetes mellitus	32	41%
ANCA positive	9/41	22%
Hepatitis C positive	22	28%
Staphylococcal strain		
MRSA	42	59%
MSSA	17	27%
MRSE	3	1.20%
MSSE	2	1.20%
Staph strain unknown	7	11%
Mixed bacterial infection	7	9%
Blood culture positive	39	50%
Local wound culture positive	43	55%
Both cultures positive	4	5%
Low C3	19 of 64	30%
Low C4	9 of 64	14%
Both C3 and C4 low	9 of 64	14%
Purpuric lower extremity skin rash	16	20.5%
Nephrotic range proteinuria	35 of 73	48%
Type and site of infection		
Endocarditis	18	21%
Bacteremia	10	14%
Osteomyelitis, arthritis	17	22%
Leg ulcers, cellulitis	17	22%
Pneumonia	6	8%
Others	10	13%
Infected abdominal mesh	1	1%
Post-surgical site infection	1	1%
Visceral abscess	6	8%
Urinary tract infection	2	3%

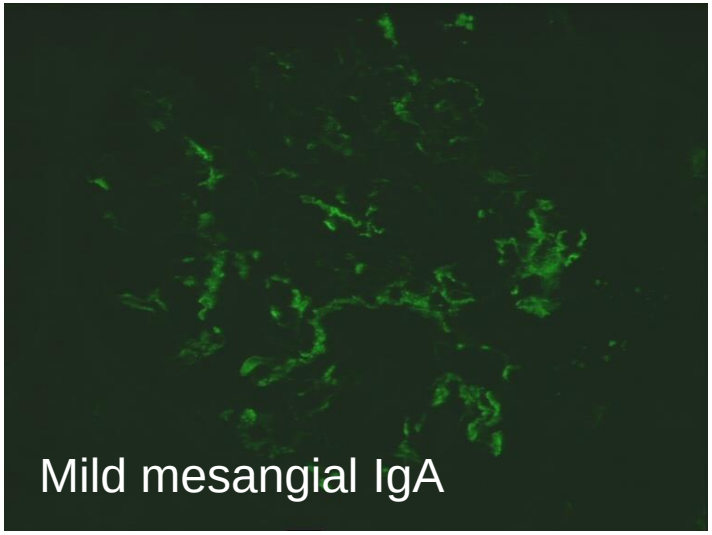
Satoskar A et al: CJASN 12:39-49,2017

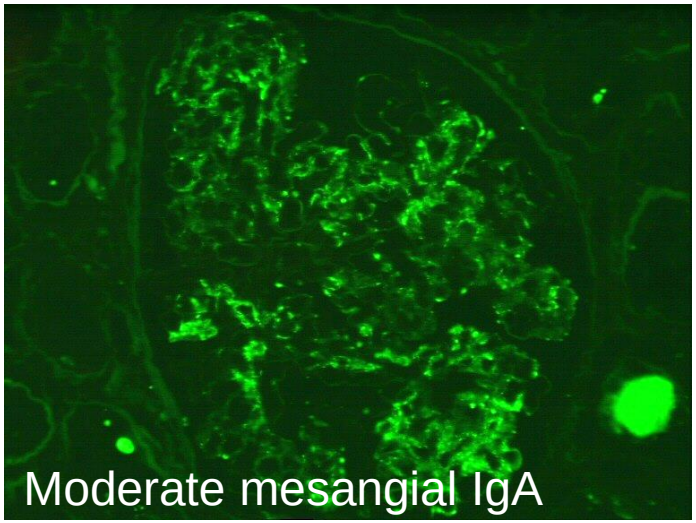
Morphology of SIAGN: Light Microscopy

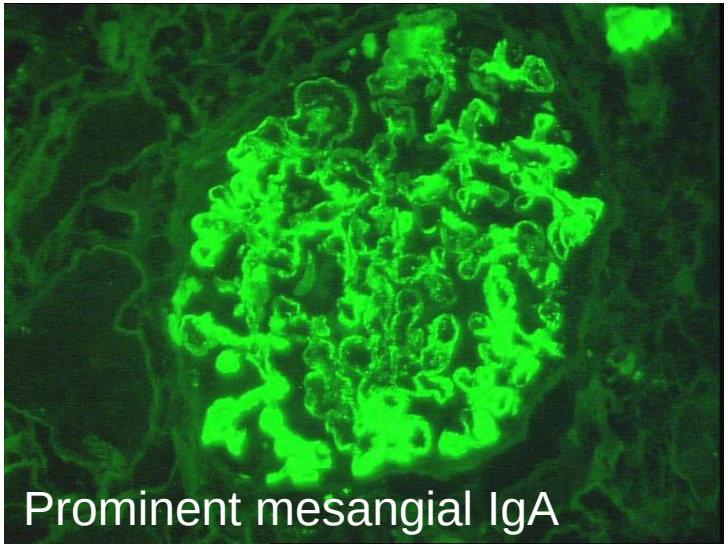
- Spectrum of glomerular changes.
- ATN
- RBC casts

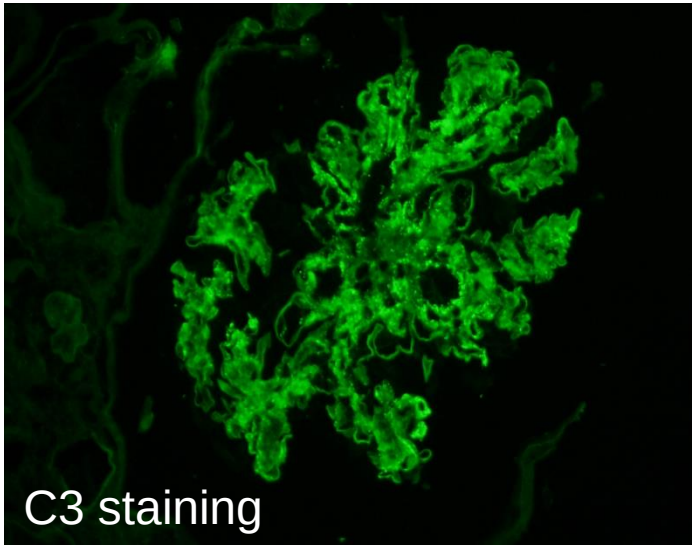


Immunofluorescence: IgA of variable intensity. C3 usually bright



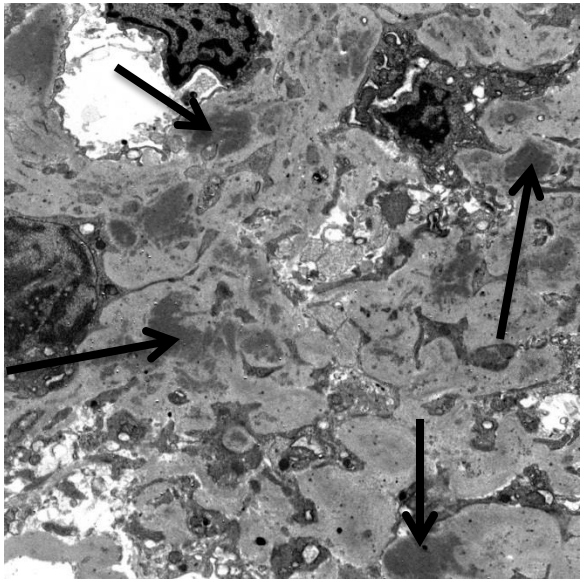




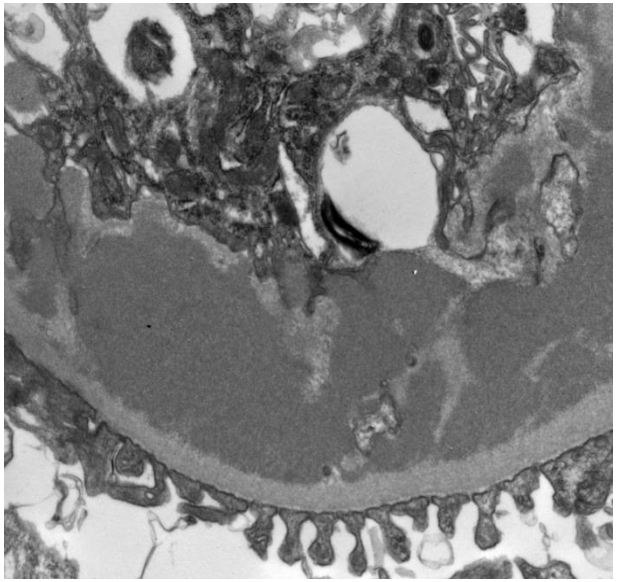


Electron microscopy

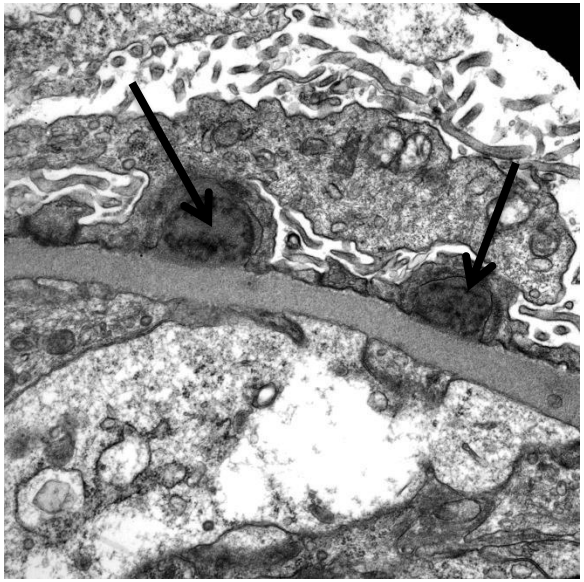
"Humps" may or may not be seen.



Mesangial deposits
(always present)



Subendothelial deposits
(sometimes large)



Subepithelial "humps"
(24% of cases at OSU)

Based on morphology alone, differentiating SIAGN from IgA nephropathy is difficult

Morphologic Differential Diagnosis of IgA Nephropathy and SIAGN

		IgAN	SIAGN
LM	Mesangial Hypercellularity	0-3+	0-3+
	Endocapillary Hypercellularity	0-2+	0-3+
	Crescents	0-2+	0-2+
IF	IgA	2-3+	1-3+ (rarely absent)
	C3	0-2+	1-3+
	C1q	0	0-1+
	IgG	0-2+	0-1+
EM	Mesangial Deposits	2-3+	2-3+
	Subendothelial Deposits	0-2+	0-3+
	Subepithelial “Humps”	0	0-2+

Unreliable: Renal biopsy findings are quite similar

Clinical Differential diagnosis of SIAGN and IgA nephropathy

Patient Characteristic	SIAGN	Primary IgA nephropathy
Age	Usually older (50-80 years)	Usually younger (20-30 years)
History of infection	antecedent or coexistent	Occasionally (30 to 40% after URI)
Latent period	Several weeks, ongoing infection	May be "Synpharyngitic" 1 to 2 days
Microbes	Staphylococcus, about 70% MRSA	May be bacterial or viral
Site of infection	Skin infection, infected leg ulcers in diabetic patients, deep –seated abscesses, post-surgical infections	Upper respiratory tract infections (URI)
Gross hematuria at presentation	50 to 60% of cases	40% (more frequent in children)
Proteinuria	Frequently nephrotic range	Usually mild <1 g/24 hours
Serum complement	Normal or low end of normal range	Normal
Acute kidney injury	Common	Uncommon
Comorbidities	Common (diabetes, drug addiction, chronic devastating diseases, obesity)	Uncommon

If you have a patient with AKI, heavy proteinuria, hematuria and some evidence of infection (other than upper respiratory tract) and particularly if the patient has comorbidities, such as diabetes, morbid obesity, cancer, etc. and the renal biopsy diagnosis is IgA nephropathy, seriously consider ***staphylococcus infection*** in the background

Case History (Case #2)

- 51-year-old Caucasian male
  - Right wrist pain and swelling
  - MRI 2 weeks prior to presentation: tenosynovitis.

  - Purpura on legs progressed to burning erythematous rash on legs, thighs, buttocks, lower abdomen, arms.
  - Abdominal pain – X-ray – Colonic distention
  - Patient was started on steroid (Medrol Dosepak – methylprednisolone)

Laboratory Data on Admission

Serum creatinine: 0.9 mg/dl on admission

Urinalysis: Trace blood, no protein, no WBCs, no RBCs, few granular casts

WBC count: 9,200/µl
Differential: 85% PMNs, 7.2% Lymphs, 5.9% Monos ESR: 43 mm

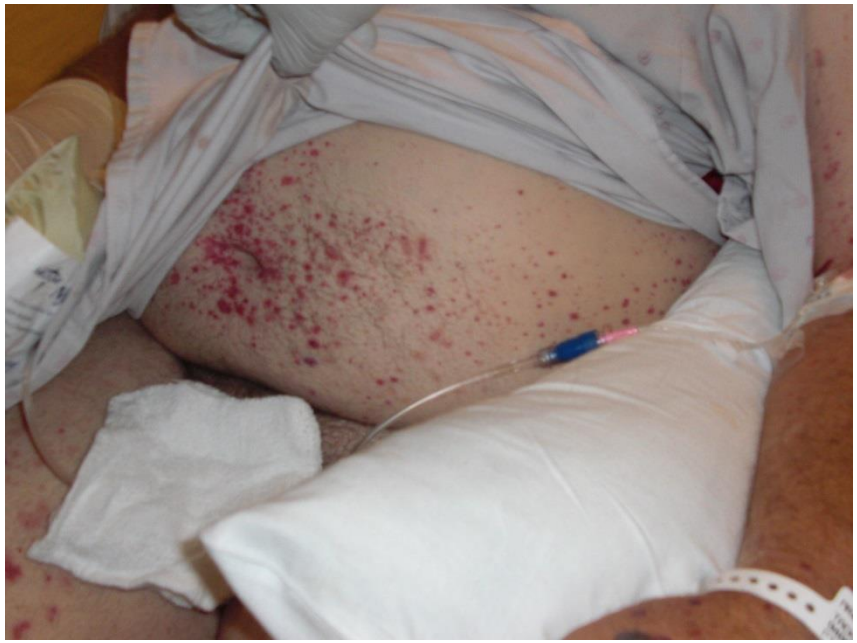
Blood cultures negative

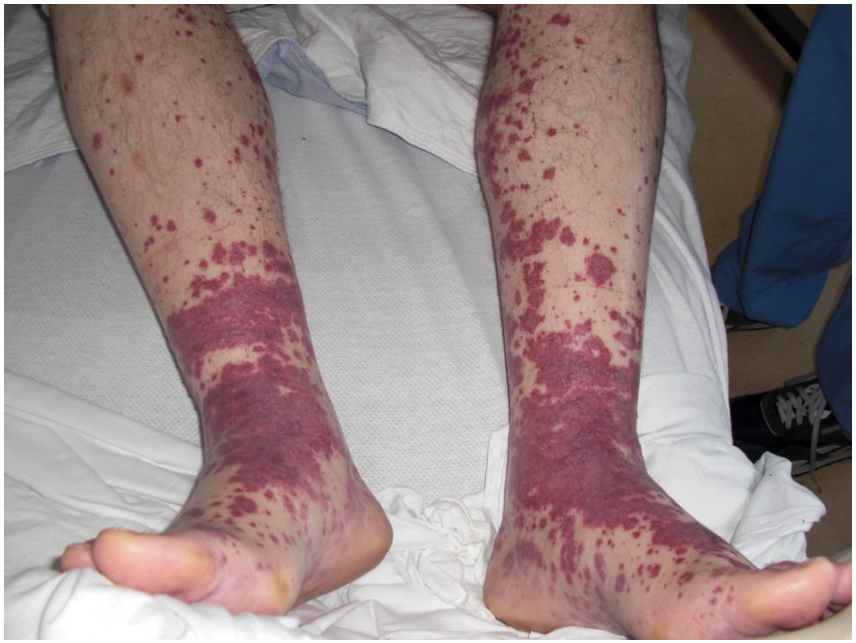
Arthrocentesis

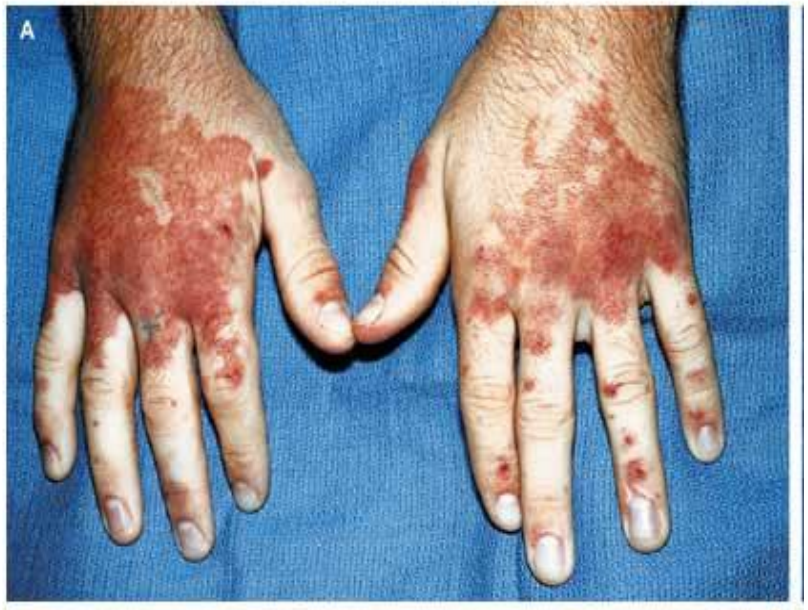
- Unfortunately, only scant fluid obtained, no cell count available
- No crystals by polarized light

Additional Labs

- ANCA – Negative
- ANA – Negative
- RF – < 20
- Total IgG – 452
- Total IgA – 302
- Hepatitis B, C – Negative
- Lyme Abs - Negative
- Serum immunofixation - negative

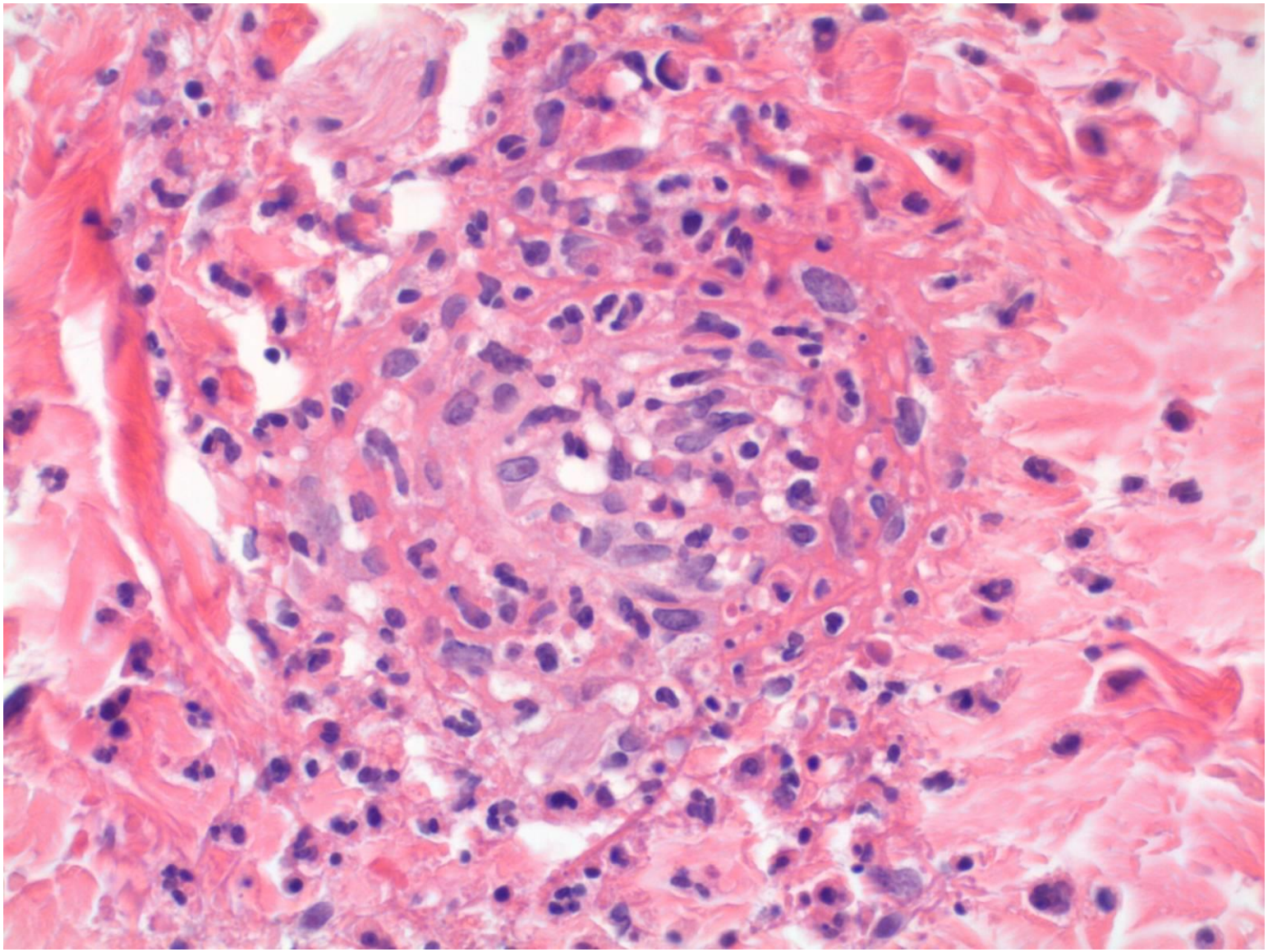




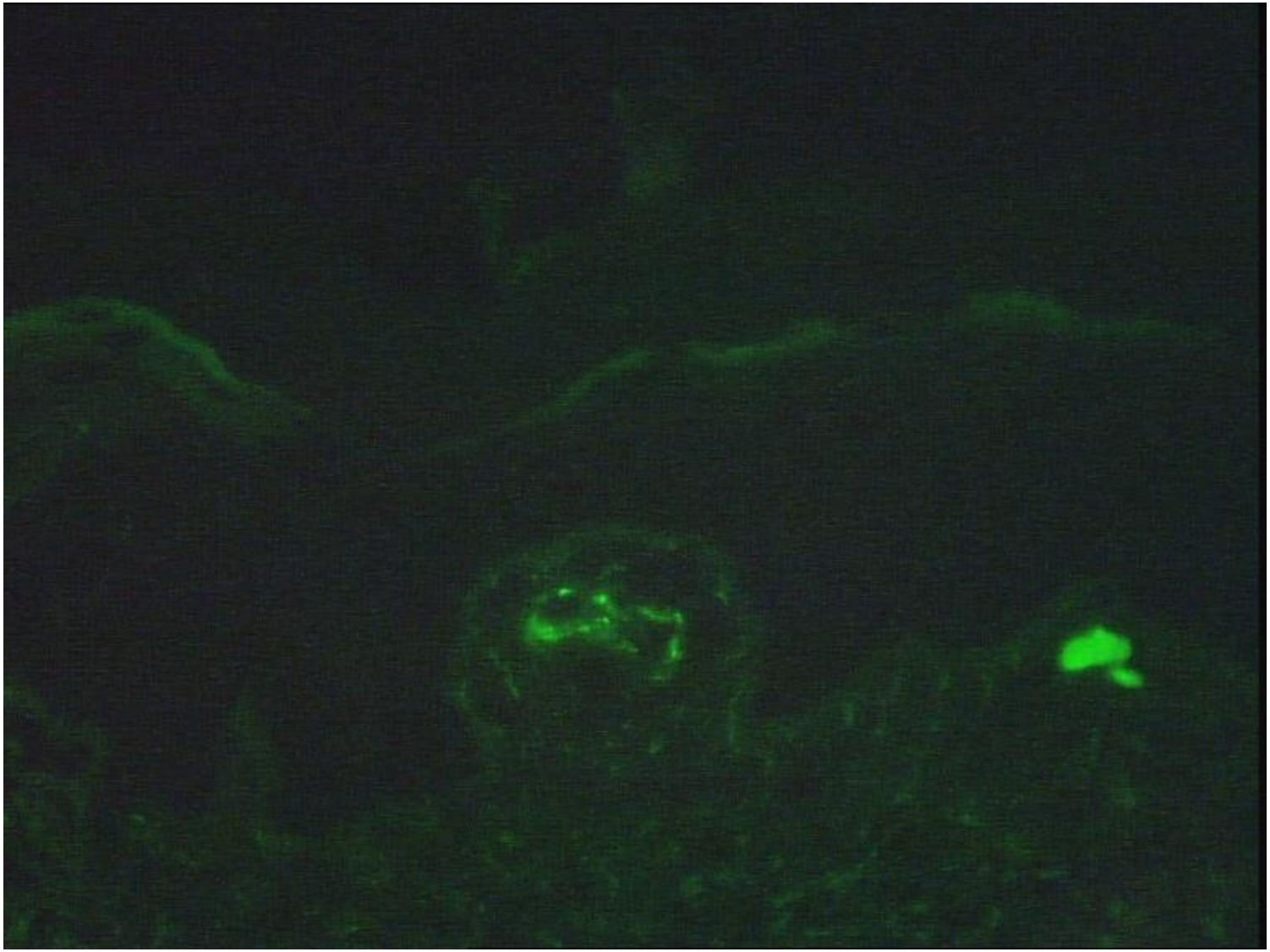


Rash - Palpable Purpura

Skin Bx - Leukocytoclastic vasculitis



Skin Bx immunofluorescence staining – IgA in arteriolar walls

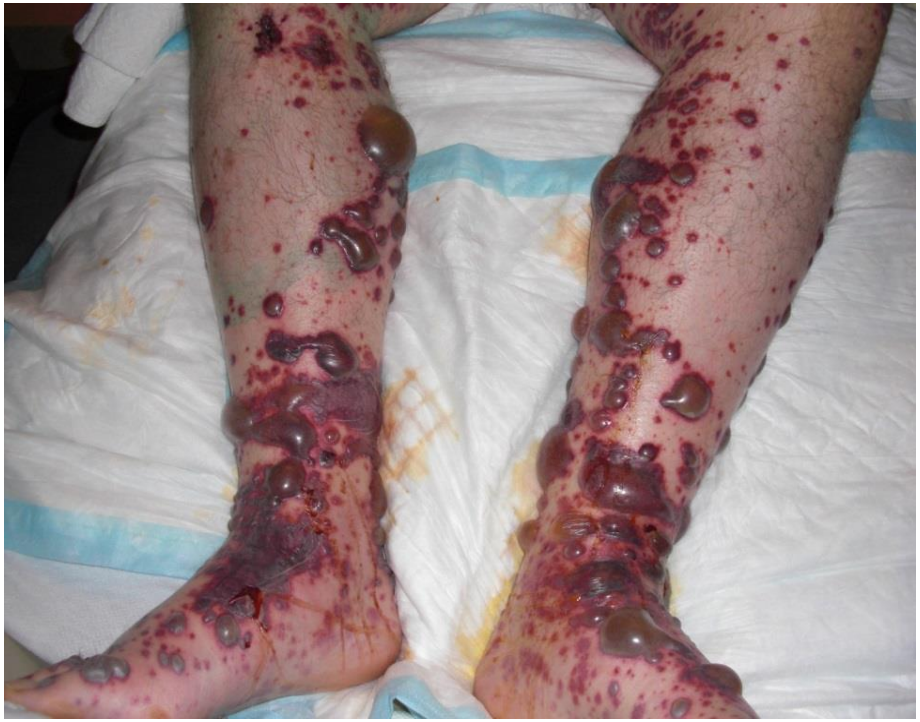


Presumptive Diagnosis: Henoch Schönlein Purpura (IgA vasculitis)

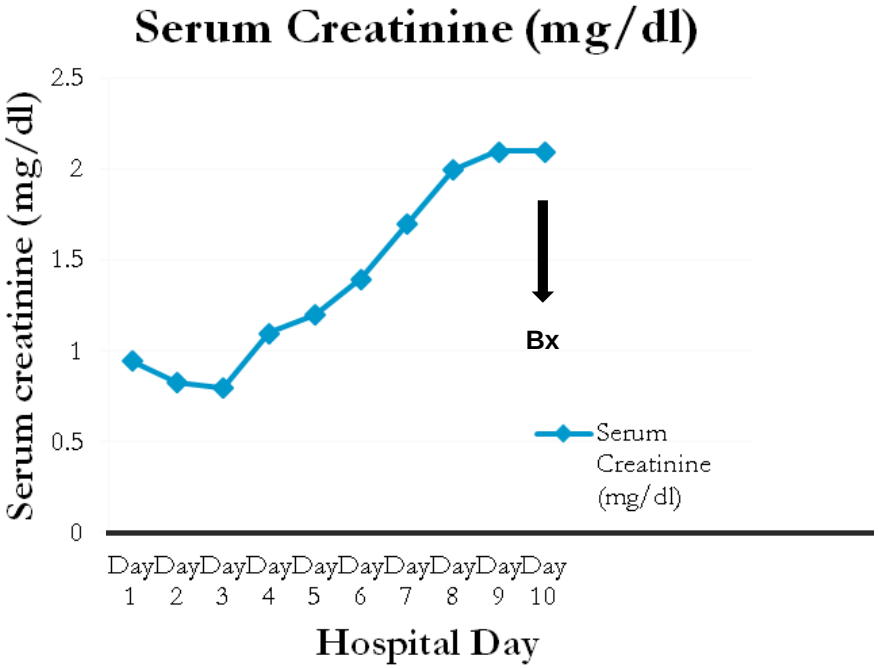
- Findings not in favor of HSP:
  - **Monoarticular arthritis**
  - Persistence and worsening of symptoms despite high dose steroids
  - Joint pain preceded skin rash
  - Adult onset presentation

Hospital Day 4

- Progression of rash to multiple bullous lesions on upper and lower extremities



Renal function worsened



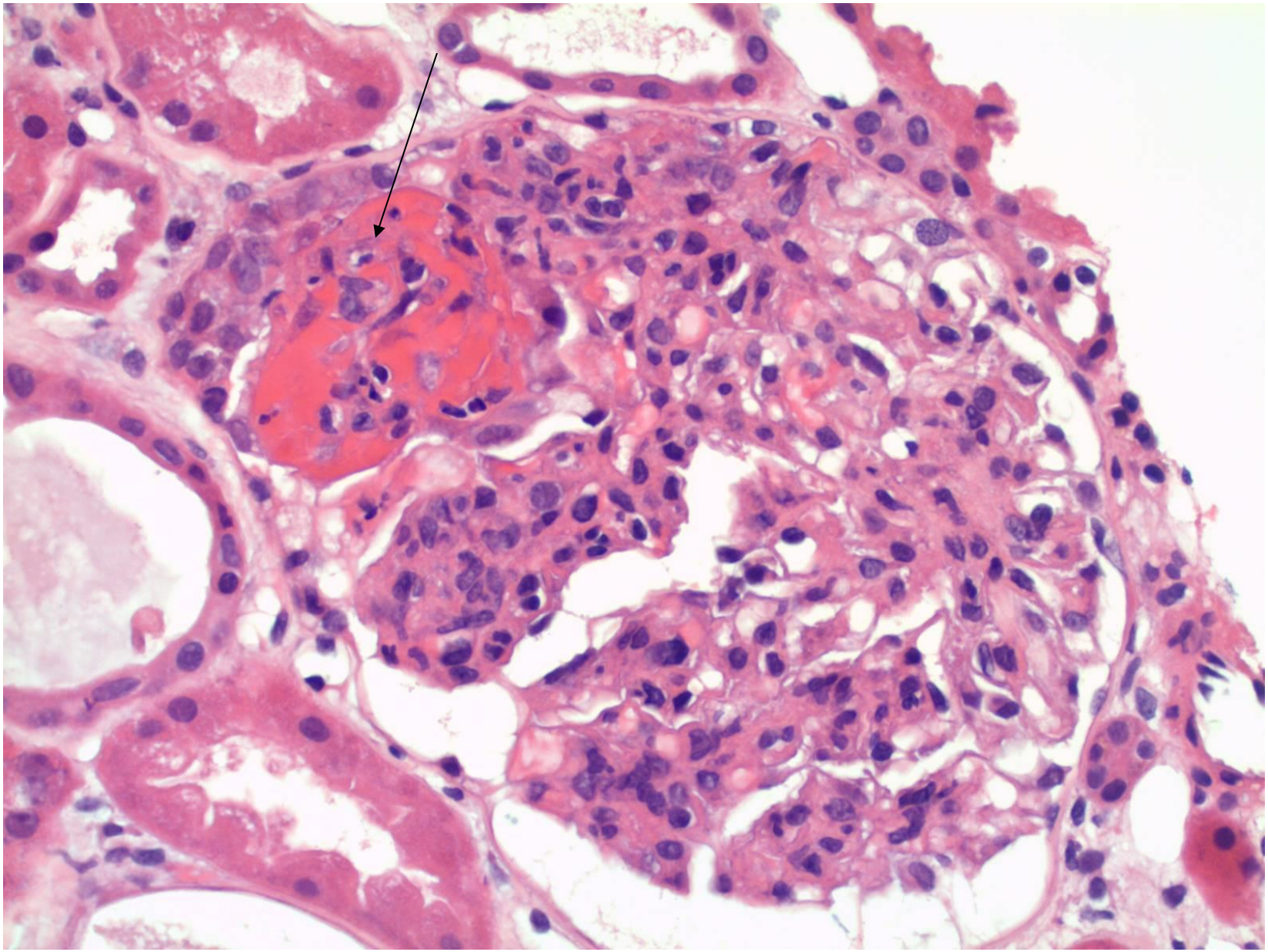
Urine output fell, proteinuria, microscopic hematuria appeared.

Patient became febrile.

Systolic blood pressure less than 100 mm Hg.

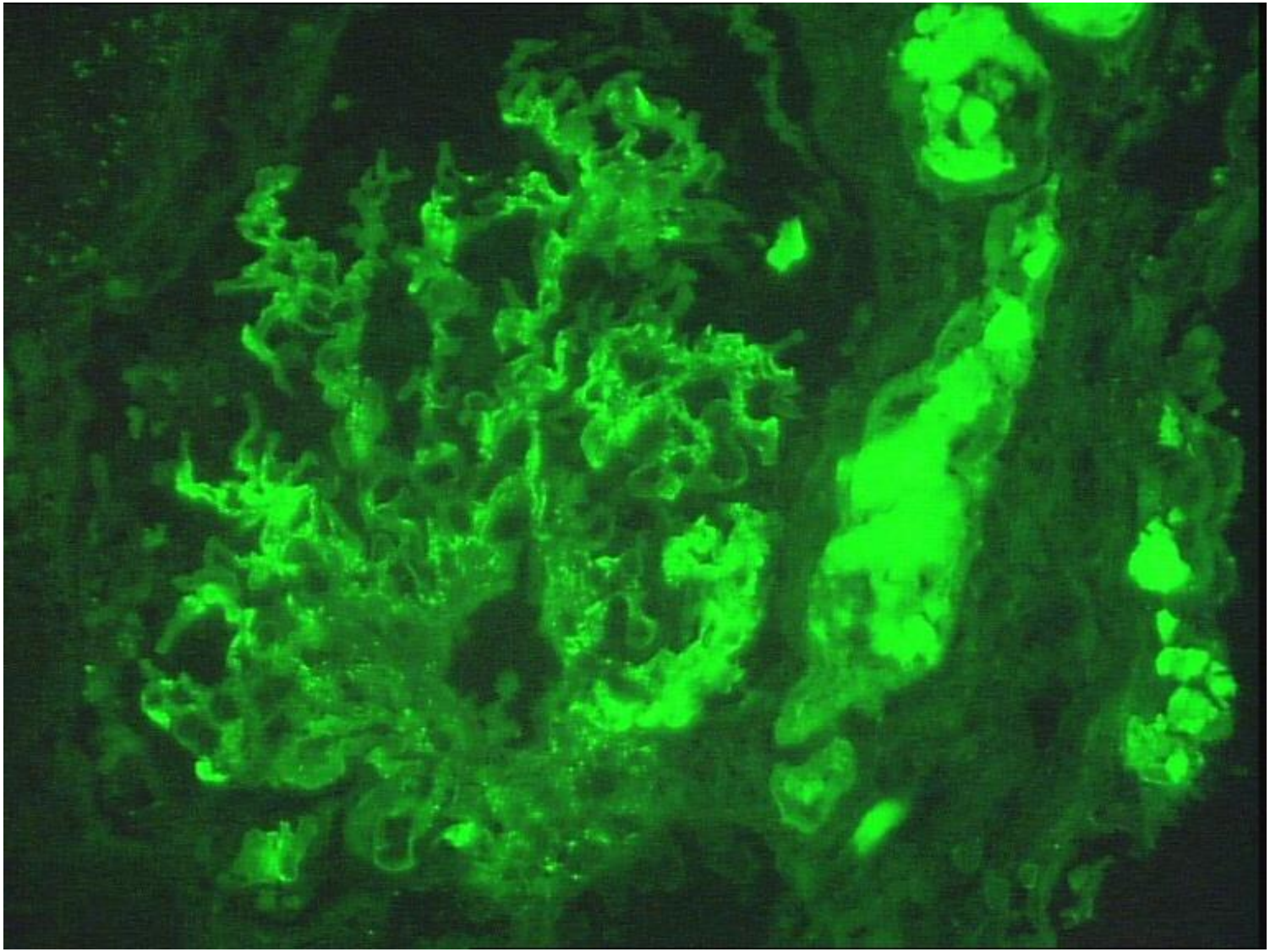
Leukocytosis – WBC 17,900/µl

Segmental necrosis with early crescent formation

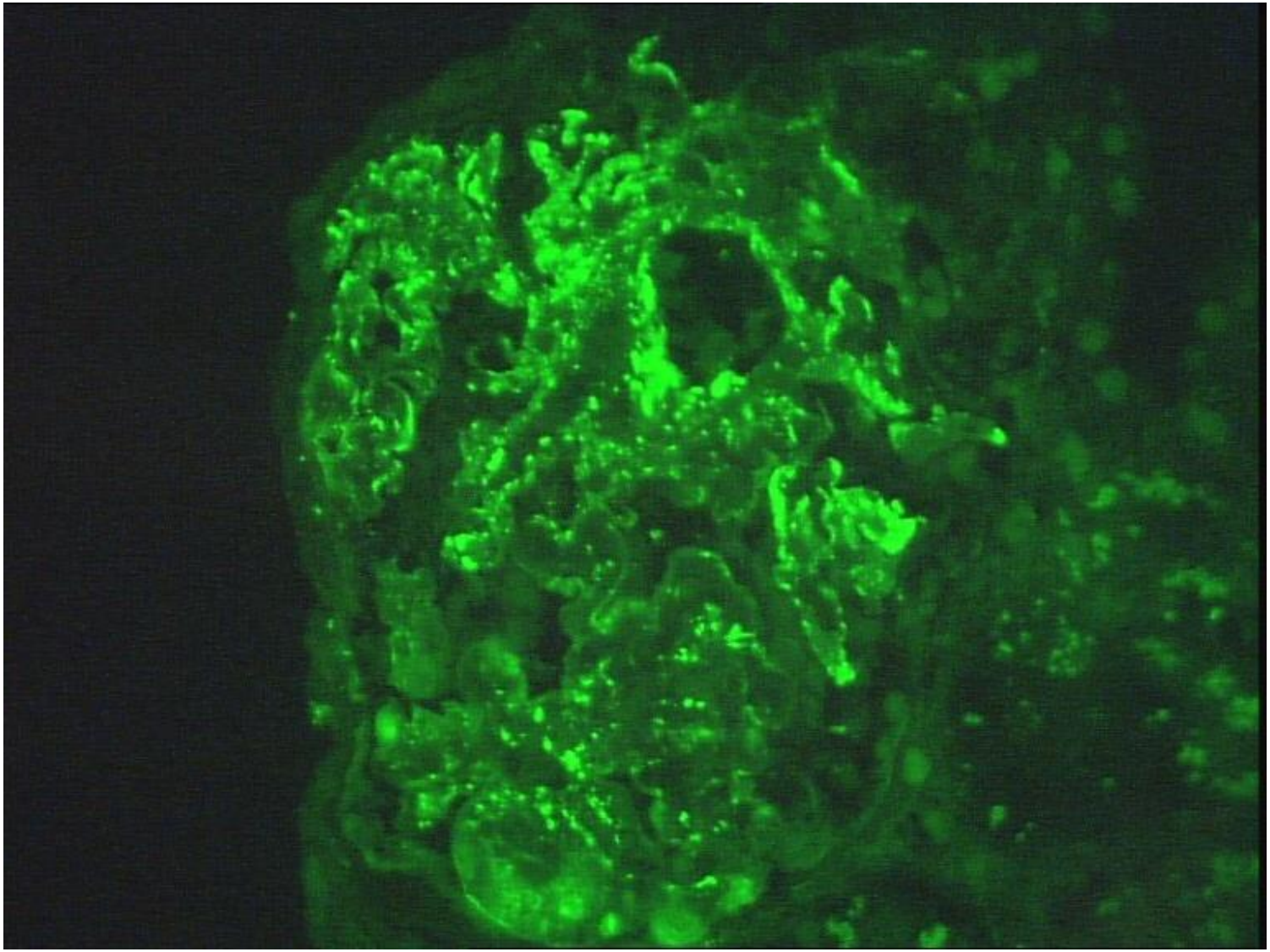


Immunofluorescence panel alb, IgG, IgA, IgM, C1q, C3, Fibrinogen, kappa and lambda

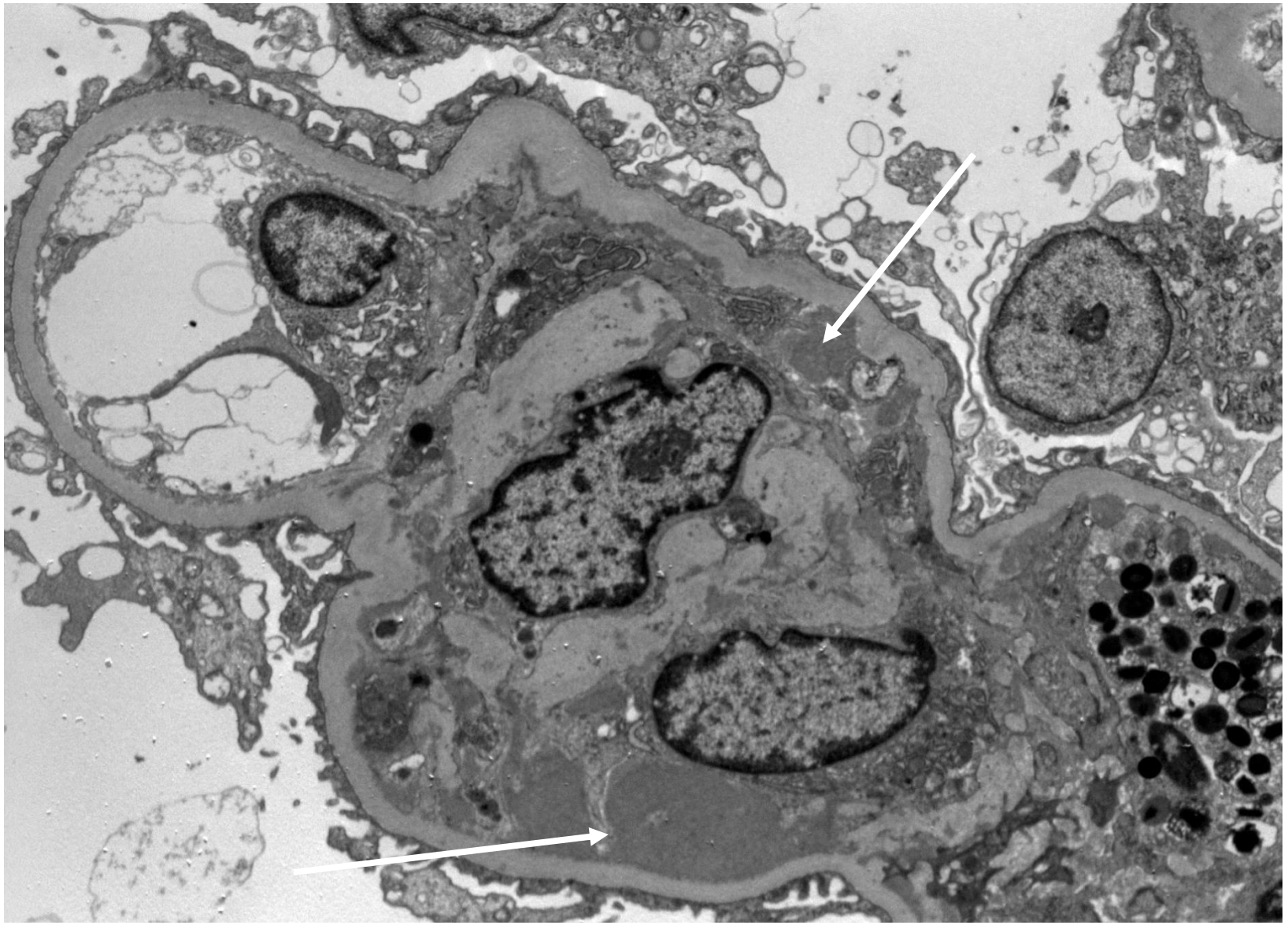
IgA (granular mesangial staining)



C3 (coarse granular mesangial staining)



Electron dense immune-type mesangial deposits



Biopsy Diagnosis

Diffuse proliferative glomerulonephritis with IgA and C3 containing immune complex deposits.

Note: The biopsy findings raise the possibility of IgA-dominant Staphylococcus infection-associated acute glomerulonephritis.

(PROBABLY NOT HENOCH-SCHÖNLEIN PURPURA OR IgA NEPHROPATHY)

- Repeat MRI of the wrist

MRI Results - Extensive osteomyelitis of the distal radius, all carpal bones and 2nd and 3rd proximal metacarpals. Extensive overlying cellulitis, myositis, and fasciitis.

Blood cultures: still negative

Further management and follow-up

- Debridement of the wrist wound
- Cultures from synovium and bone: Methicillin-Sensitive Staphylococcus aureus (MSSA)
- Started on Nafcillin infusion for MSSA – 6 weeks
- Renal function stabilized at serum creatinine of 2.0mg/dl.
- Follow up after completion of antibiotics
  - Serum creatinine decreased to 1.3mg/dl
  - Urinalysis – negative for blood and protein

Comparison of idiopathic HSP and Staphylococcus infection associated glomerulonephritis presenting with HSP-like symptoms.

	Idiopathic Henoch-Schönlein Purpura (HSP)	HSP-like presentation in Staphylococcus infection associated glomerulonephritis
Age	Predominantly affecting children, uncommon in adults	Predominantly affects the elderly population
Other associated co-morbidities	No other co-morbid conditions seen	Patients commonly diabetic, have post-surgical wound complications, alcoholic, history of malignancy, endocarditis, intravenous catheters.
Associated infections	May have recent history of upper respiratory tract infection (bacterial or viral), but usually cleared before onset of HSP vasculitis.	Ongoing infections such as infected diabetic ulcers, infected surgical sites or trauma wounds, endocarditis, pneumonia, visceral abscess, infected intravenous catheters. Infection may be undiagnosed before the onset of ARF. Usually methicillin resistant or sensitive Staphylococcus aureus (MRSA/MSSA), mixed bacterial infections.
Findings on skin biopsy	Leukocytoclastic vasculitis with mild IgA deposits	Leukocytoclastic vasculitis with mild IgA deposits
Findings on kidney biopsy	Focal or diffuse mesangial and intracapillary proliferative glomerulonephritis with or without crescents. Mesangial IgA, C3	Focal or diffuse mesangial and intracapillary proliferative glomerulonephritis with or without crescents. Crescents are usually small and segmental. Mesangial IgA and C3 with/without mild IgG.
Therapy	Supportive management in children. Glucocorticoid treatment only if needed for persistent renal dysfunction.	Active treatment of the infection. Immunosuppression should be avoided.
Outcome	In children, usually self-limited disease and favorable renal outcome. In adults, renal outcome can be poor.	Usually poor renal outcome.

Satoskar A et al: Clin Nephrol 79:302-312, 2013

HSP-like presentation in an elderly or adult

Always consider underlying staphylococcus infection before starting immunosuppression

Renal complications in Infective Endocarditis

- Infective emboli with renal infarcts, abscess.

  - Obstructive emboli coming from the endocardial vegetations can cause focal renal cortical infarcts, abscess.

- Immune-mediated (immune complex) glomerulonephritis

  - Diffuse glomerular endocapillary proliferative lesions, frequently with focal segmental necrotizing lesions, crescents.
  - The term “focal embolic nephritis” implying embolization of infected material to the glomerular capillaries causing necrotizing lesions, is no longer accepted.
  - The focal necrotizing glomerular lesions are believed to be an immunologic phenomenon.
  - Endocapillary proliferative lesions are not always evident; the glomerulonephritis may appear pauci-immune crescentic and necrotizing.

- Acute tubular necrosis

  -Secondary to poor cardiac function, poor renal perfusion and or drug toxicity

- Therapy-induced tubulointerstitial nephritis

  - Multiple potent antibiotics can be nephrotoxic.

Bacterial Endocarditis and Glomerulonephritis

- *Streptococcus viridans* group has been long implicated in endocarditis involving damaged left-sided heart valves. But its frequency has diminished.
- *Staphylococcus aureus* has become the major cause of endocarditis and is frequently seen in intravenous drug abusers with right-sided endocarditis;
- Streptococcus, Bartonella henselae, Enterococcus are other reported pathogens in endocarditis associated glomerulonephritis.
- At our center, 18/78 (21%) patients with Staphylococcus infection-associated glomerulonephritis had endocarditis.

Clinical characteristics of 49 patients with endocarditis-associated GN

Gender/age	
Male:female, *n/n* (%/%)	38/11 (78/22)
Age (years), mean (range)	48 (3–84)
Clinical syndrome n=*47 with data (%)*	n (%)
Acute renal failure	37 (79)
Acute nephritic syndrome	4 (9)
Rapidly progressive glomerulonephritis	3 (6)
Nephrotic syndrome	3 (6)
Predisposing states[a]	
Intravenous drug abuse	14 (29)
Prosthetic cardiac valve	9 (18)
Cardiac valve disease/Intracardiac shunt	6 (12)
Associated conditions	
Hepatitis C	10 (20)
Diabetes mellitus	9 (18)
Coronary artery disease	3 (6)
Chronic obstructive pulmonary disease	2 (4)
Congestive heart failure	1 (2)
Systemic lupus erythematosus	1 (2)
Recent surgery	1 (2)
Prostate cancer	1 (2)
Laboratory data	
Serum creatinine at biopsy (mg/dl), *n*=45	3.8 (1.0–12.0)
Proteinuria (g per day), *n*=18	1.8 (0.5–15)
	n (%)
Hematuria, *n*=37	36 (97)
ANA, *n*=26	
Positive	4 (15)
ANCA (29 tested)	
Positive	8 (28)
C3/C4, *n*=32	
Low C3 only	12 (37)
Low C4 only	1 (3)
Low C3 and C4	5 (16)
Normal C3 and C4	14 (44)

Boils et al Kidney Int 87:1241, 2015

Cardiac and bacterial characteristics

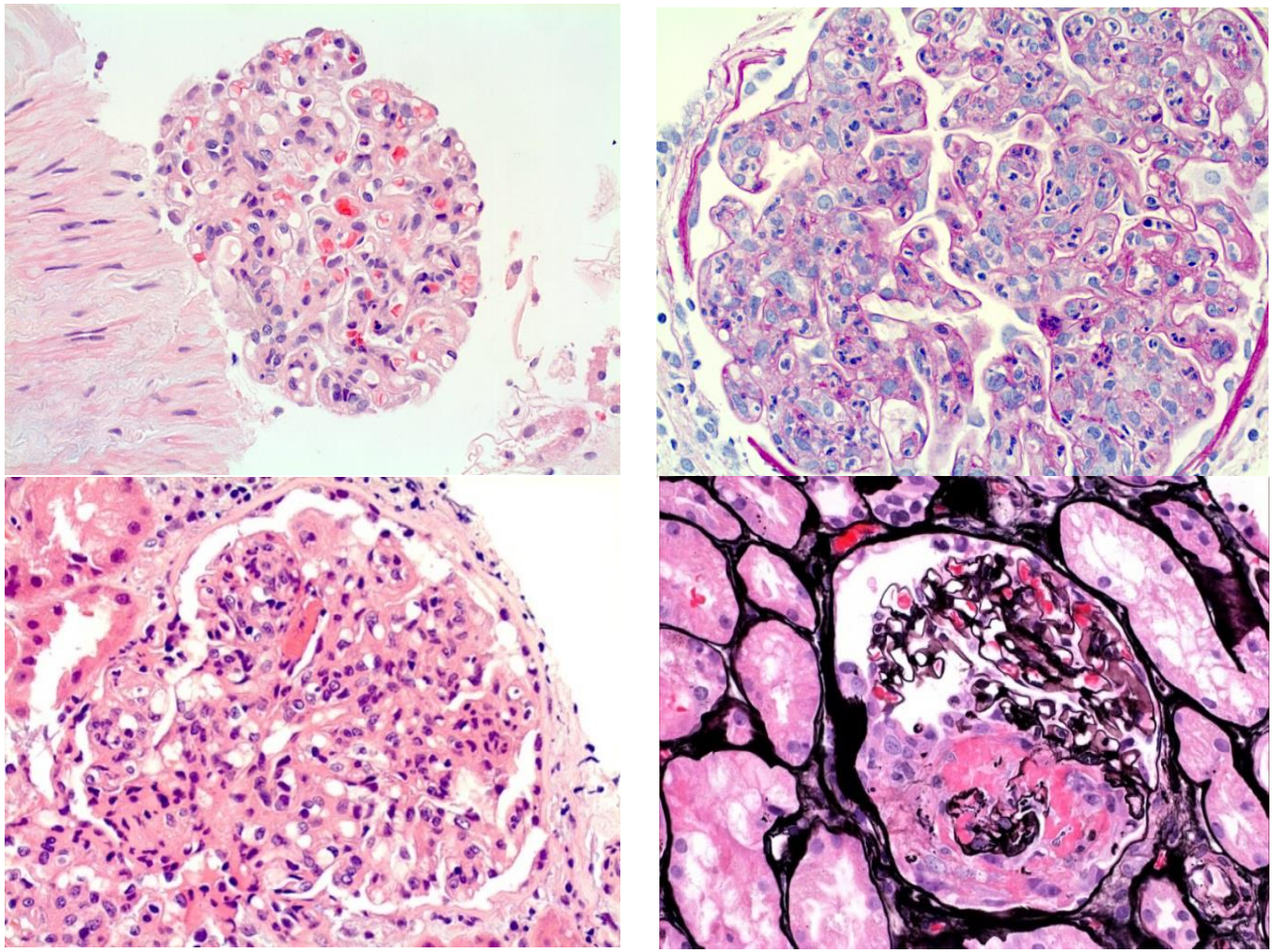
	n (%)
Valve/location[a]	
Tricuspid	18 (43)
Mitral	14 (33)
Aortic	12 (29)
Pulmonic	2 (5)
Chordae tendinae	1 (2)
Culture results	
Positive	44 (90)
Negative	4 (8)
Unknown	1 (2)
Bacterial agent[c]	
Staphylococcus	25 (53)
Streptococcus	11 (23)
Bartonella henselae	4 (8)
Coxiella burnetii	2 (4)
Cardiobacterium hominis	1 (2)
Gemella	1 (2)

Boils et al Kidney Int 87:1241, 2015

Clinical and laboratory data on 34 patients with biopsy proven staphylococcus endocarditis at The OSU

- MRSA: 18
- Diabetic: 5
- IV drug use/Hepatitis C: 23
- Nephrotic: 12
- ANCA+: 7 of 17 tested (41%)
  - 4 anti PR3; 2 pANCA (1 anti-MPO; other unclear), 1 unspecified
- Low serum complement: 9 of 23 tested (39%)
  - Low C3: 8; Low C4: 5; Both C3 and C4 low: 4; Low C4 only: 1

Morphologic patterns in endocarditis associated glomerulonephritis

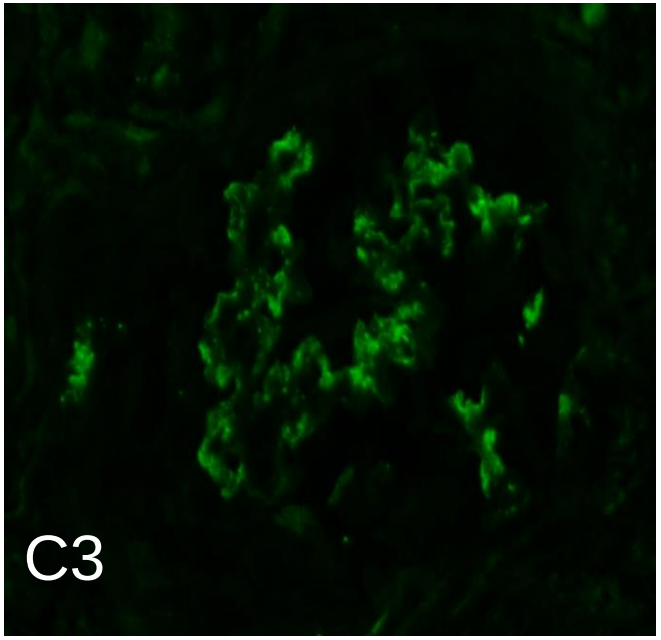


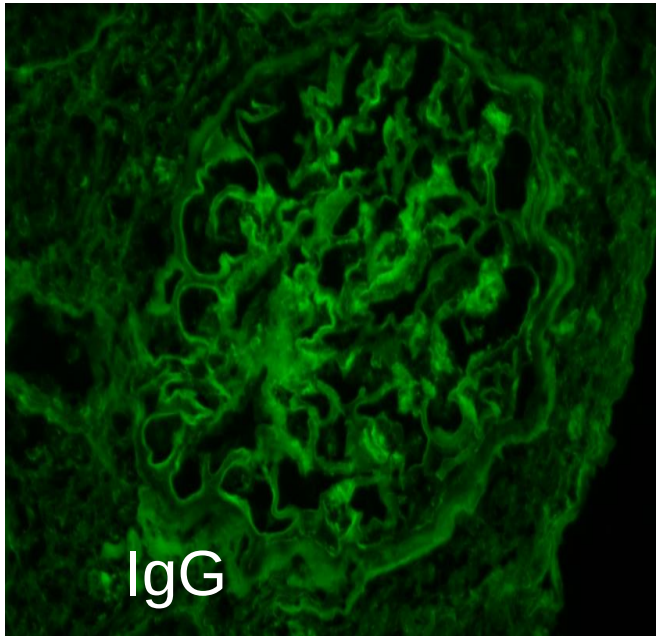
Variable histologic patterns from mild mesangial hypercellularity to prominent intracapillary proliferative glomerulonephritis to crescentic and necrotizing glomerulonephritis

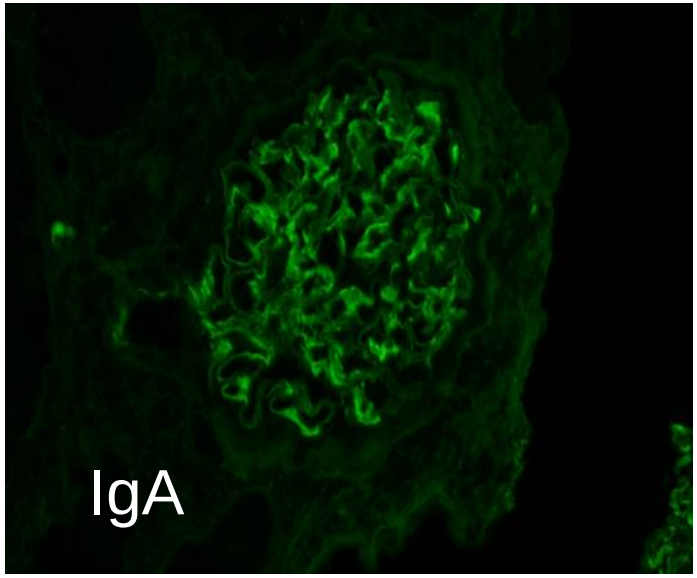
Direct Immunofluorescence (IF)

- Variable intensity and extent of immune complex deposition.
- Granular staining for C3 with or without IgG are seen in the mesangium and/or along the capillary wall. IgA may be seen but, interestingly, glomerular IgA deposits are less common in Staphylococcus endocarditis-associated GN than in GN secondary to staphylococcus infection of other sites.
- C3 may be dominant without significant immunoglobulin deposits.
- Sometimes deposits can be scant to absent, particularly if the patient is ANCA positive.

Immune complex deposits can be mild and may not go hand-in-hand with the severity of the light microscopic features. This case had necrotizing glomerular lesions.



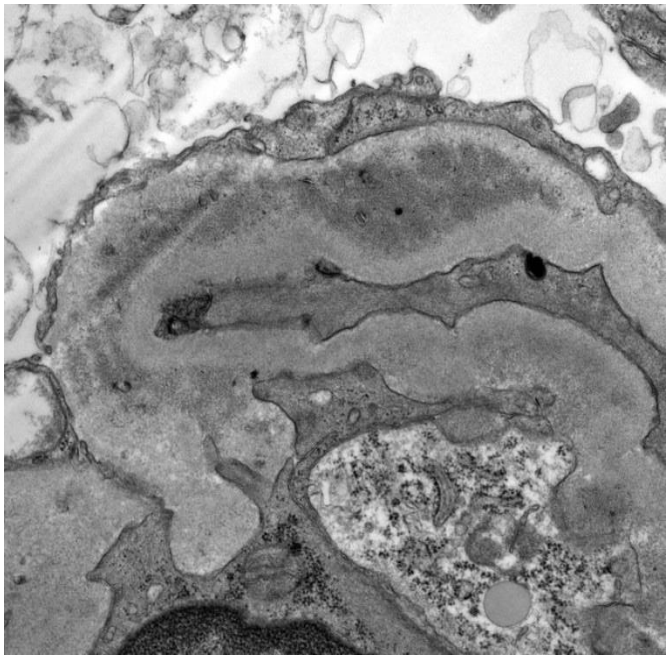


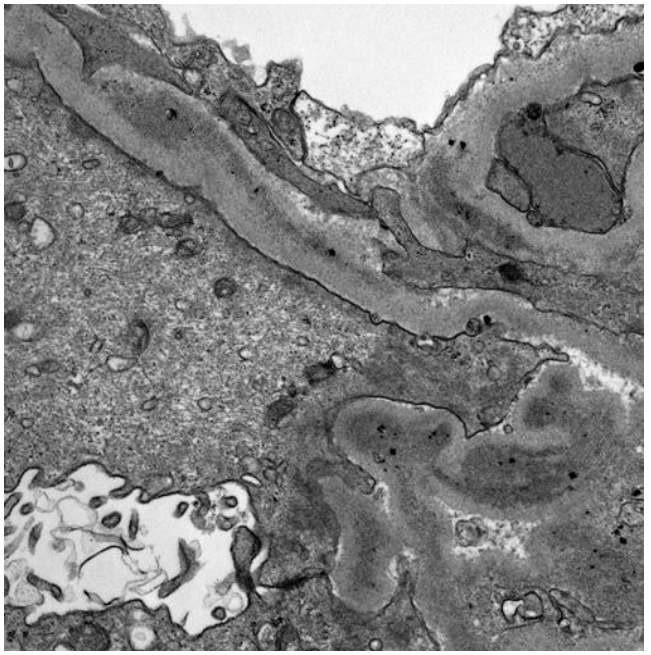


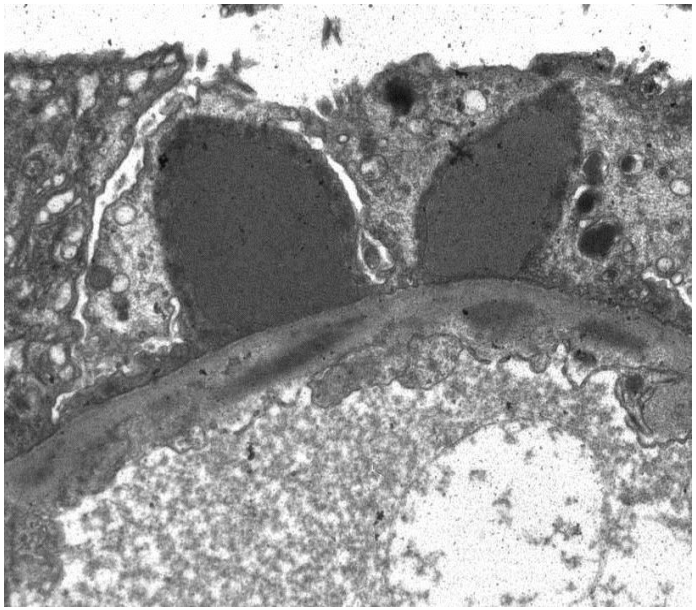
Electron Microscopy

	n (%)
Mesangial deposits	41 (84)
Subendothelial deposits	22 (45)
Subepithelial deposits	17 (35)
Subepithelial ‘humps’	7 (14)
No deposits	5 (10)

Boils et al: Kidney Int 87:1241, 2015







Main morphologic data of 34 biopsies with endocarditis-associated GN at The OSU

- Endocapillary hypercellularity : 20 (59%); 3 also had crescents
- Crescentic and necrotizing GN:14 (41%); 6 had no endocapillary hypercellularity
- Mesangial hypercellularity: 12 (35%)
- Pauci-immune*: 7 (21%); 5 of them crescentic
- Presence of C3: 31 (91%);IgA: 22 (65%); IgG: 15 (44%)
- Humps: 7 (21%)

*: Defined as few or weak deposits by IF and no or rare electron dense deposits by EM

Diagnostic pitfalls

1. ANCA serology can be positive – anti-proteinase 3 (PR3-ANCA) alone or both anti-PR-3 and anti-myeloperoxidase (MPO-ANCA) (dual ANCA positivity).

2. Cryoglobulinemia (usually type III) is common in patients with endocarditis; misdiagnosis as cryoglobulinemic glomerulonephritis (GN) should be avoided.

3. Other forms of proliferative GN (MPGN type I, C3 GN, Lupus nephritis, GN secondary to other infections).

Conclusions

- Staphylococcus infection became the most common cause of infection-associated GN in the US (and probably in most developed countries), particularly in adults and in the elderly
- AKI + Proliferative GN with IgA deposits (frequently with severe proteinuria): Consider infection, in particular staphylococcus infection
- Infection may not be clinically obvious – needs to be carefully looked for.
- Blood cultures may be negative. Local wound cultures important.
- SAIGN should be clearly distinguished from IgA nephropathy/HSP.
- Infection is active, ongoing; therefore avoid the term “post-infectious” (post-staphylococcal) glomerulonephritis
- SAIGN, in particular endocarditis-associated GN, may sometimes mimic pauci-immune crescentic and necrotizing GN
- Treatment of the infection is most important. Immunosuppression should be avoided

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Editors

Bacterial Infections and the Kidney

Springer

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