Monoclonal Gammopathy–Related Kidney Diseases

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Monoclonal gammopathies (Paraproteinemia/ dysproteinemias)

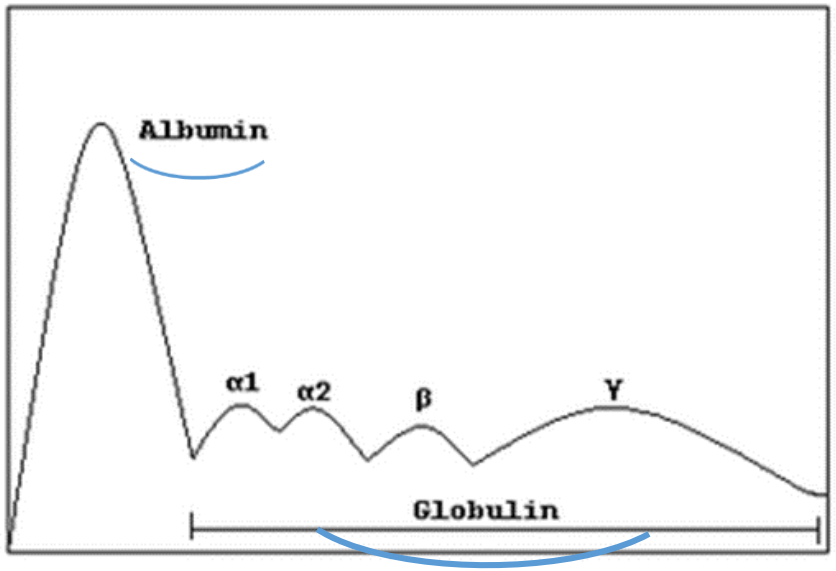
- There are two large classes of blood proteins: albumin and globulin

  The globulins: three regions on the electrophoretic gel

  the α band,

  the β band,

  the γ band.



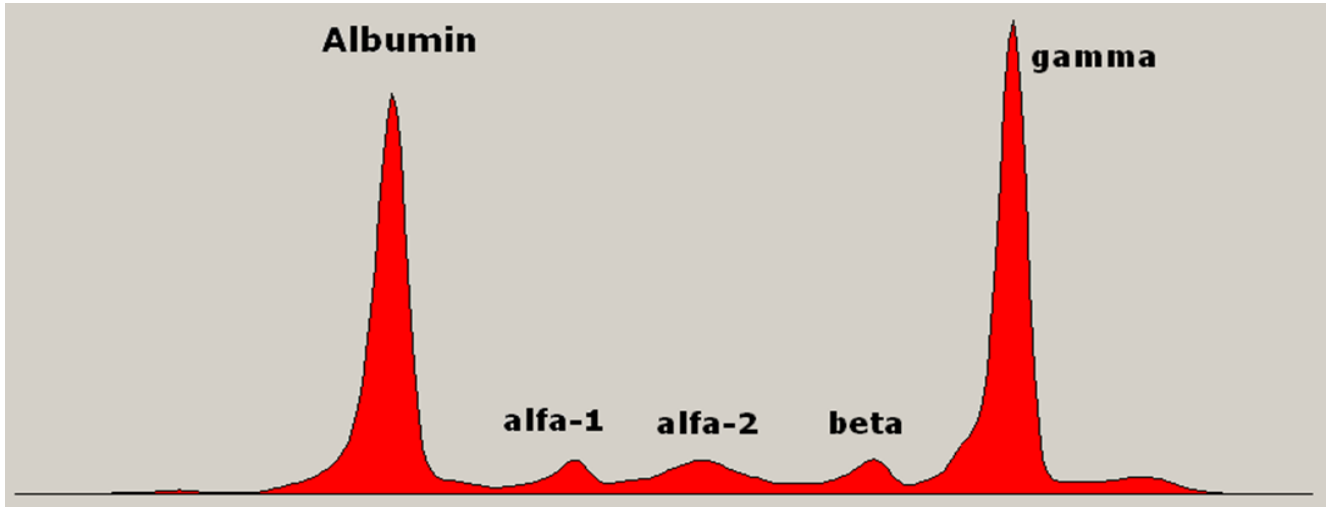
- Paraproteinemia:

Conditions in which abnormal proteins are found in the blood.

- The majority of paraproteins appear in γ band.

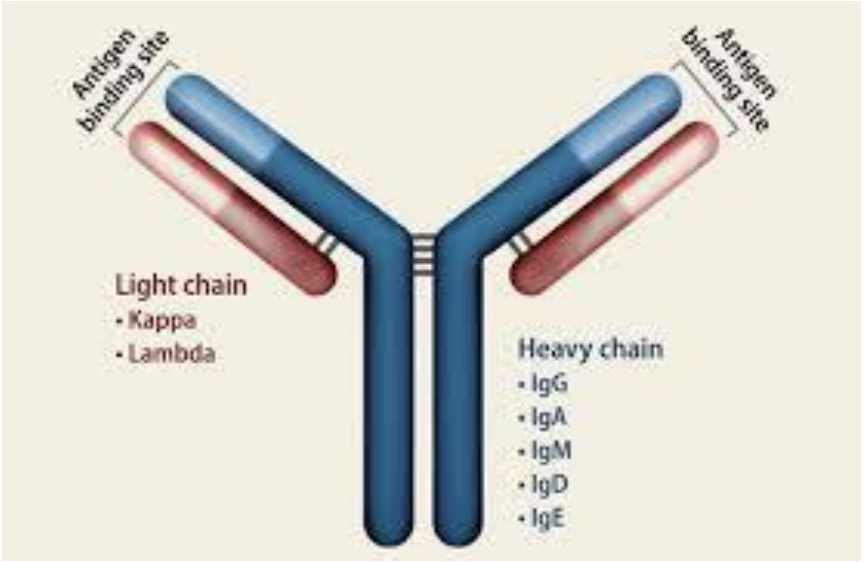
  The γ band is where the immunoglobulins appear, which are also known as gammaglobulins.

- In monoclonal gammopathy in the blood, there will be a narrow band, or spike, in the serum protein electrophoresis → Excess production of one protein



Monoclonal gammopathies according to the type of monoclonal protein found in blood:

- Light chains only (or Bence Jones protein):
  - Multiple myeloma
  - AL amyloidosis
- Heavy chains only:
  - Heavy chain disease
- Whole immunoglobulins (macroglobulinemia):
  - Cryoglobulinemia
  - Waldenström



Monoclonal gammopathies (B cell/ plasma cell proliferation disorders):

- Characterized by the single clone proliferation of the lymphoplasmacytic cells in the bone marrow and monoclonal immunoglobulin (Ig) deposition in the tissues:

- Monoclonal gammopathy of undetermined significance (MGUS)
- Multiple Myeloma/ Smoldering multiple myeloma
- Waldenström's macroglobulinemia
- Solitary plasmacytoma
- Other plasma cell leukemias and lymphomas (low-grade chronic lymphocytic leukemia, and low-grade B-cell lymphomas)

https://www.hopkinsmedicine.org/health/conditions-and-diseases/monoclonal-gammopathies

Monoclonal gammopathy of undetermined significance (MGUS)

- Monoclonal protein (M-protein) exists in about 3% of adults older than 50 years and increases to 5% in those older than 70 years.
- MGUS: Patients do not meet the criteria for multiple myeloma—M-protein <3 g/dL and plasma cells on BMB <10% and No myeloma related end-organ damage and absence of B-cell lymphoma or other disease known to produce an M-protein
- The most common condition linked with these abnormal proteins
- The exact cause of MGUS is not known. Infection, immune system problems, and the environment may play a role.

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan

Monoclonal gammopathy of renal significance (MGRS)

- Patients in this category do not meet the diagnostic criteria for multiple myeloma or other B-cell malignancies; however, they have renal involvement, which often responds to treatment of the monoclonal protein.

Monoclonal gammopathy of clinical significance

- In addition to renal pathology, excess M-protein can affect peripheral nerves and skin (another recent classification)

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan

Kidney involvement in monoclonal gammopathies:

- Kidney involvement is secondary to the production of monoclonal immunoglobulin or its fragments, such as light or heavy chains.
- Predispose factors of renal injury:

  Underlying CKD

  Hypovolemia

  Electrolyte derangements (hypercalcemia, hyperuricemia)

  loop diuretics or NSAIDs

  Administration of contrast dyes.

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan

Categories of Monoclonal gammopathy-related renal disease according to :

- Pathophysiology:

  Immunoglobulin-mediated or non-immunoglobulin–mediated

  (considerable overlap and interaction)

  Autoimmune involvement and complement activation

- Location of renal injury: glomerular, tubulointerstitial, vascular (combination)
- Clinical syndrome: AKI, CKD, proteinuria, hematuria, renal crystallopathy, and Fanconi syndrome.

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan

Epidemiology:

- Kidney involvement in multiple myeloma and other plasma cell dyscrasias:

  20-50% At the time of presentation or during illness

  Associated with poor prognosis and higher mortality

- Cast nephropathy (myeloma kidney): the most common renal disease in MM

  40% to 60% of Kidney Bx even when the primary diagnosed renal disease is amyloidosis or crystalline podocytopathy

- Renal failure:

  The second most common cause of mortality in MM(after infections)

  1.5% of all cases of RRT

  2-year mortality in ESRD + multiple myeloma is 58% VS 31% in just ESRD

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan

Monoclonal Gammopathy–Related Kidney Diseases. Advances in Chronic Kidney Disease. March 2022

Immunoglobulin-Mediated Renal Disease

1. Cast nephropathy (Myeloma kidney):

The most common kidney involvement/ AKI in myeloma

Any protein overload in the renal glomeruli and tubules can result it

Can be present concurrently with other types of Monoclonal gammopathy-related kidney diseases.

Tubular pH can affect cast formation

Renal damage results both from direct nephrotoxic effects of light chains on proximal tubular cells and mechanical obstruction of distal and collecting tubules by the protein casts.

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan

- The normal level of light chain excretion: <30 mg/day
- In multiple myeloma can increased excretion 100-2000 mg/day.
- The light chains accumulate due to:

  Increased excretion of light chains

  Resistance to degradation

  Saturation of endocytosis capacity
- Release proinflammatory cytokines and reactive oxygen species,

  causing damage to the proximal tubules and apoptosis

- FLCs reach the distal tubules:

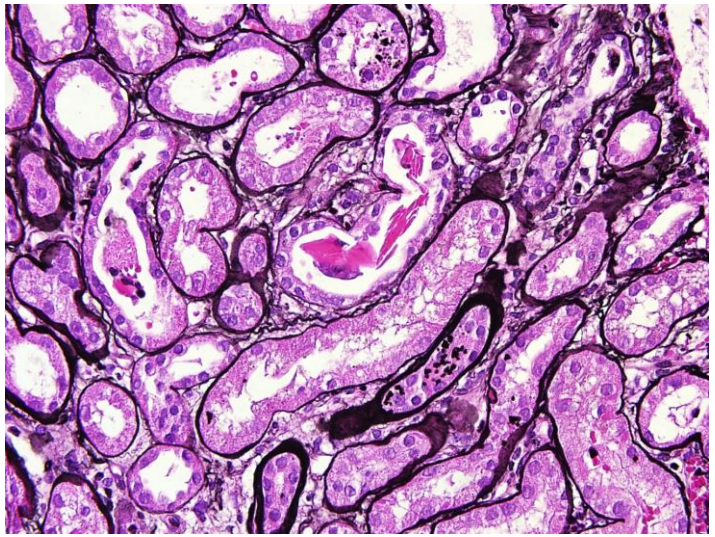
  Aggregate with uromodulin (Tamm- Horsfall mucoprotein),

  Leads to dense and obstructing intratubular casts in the distal tubules

  Start a giant cell reaction
- Obstructing casts may cause tubular rupture
- Further extravasation of FLCs into the interstitium

  development and progression of interstitial inflammation and fibrosis

- Tubular atrophy, interstitial fibrosis
- Dense, eosinophilic, homogeneous casts often laminated or fractured and partially surrounded by multinucleated giant cells.
- Intratubular light chains may change in situ, leading to amyloid formation.



Cast nephropathy begins in the collecting tubules, so biopsy samples should include some medullary tissue.

Precipitating factors:

- Radiocontrast agents,
- NSAIDs,
- ACE-I/ ARBs,
- Diuretics,
- Dehydration (hypovolemia)

} especially potent risk factors for precipitating cast nephropathy

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan

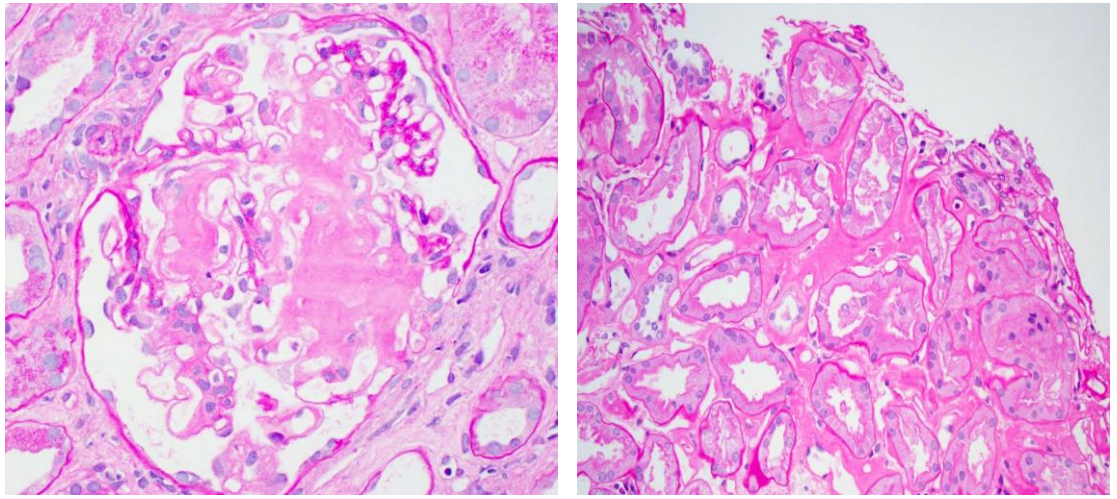
- Typically presents as AKI and progresses rapidly, with a drop in creatinine clearance over 1- 3 months.
- Can either be the first presentation or develop later during the course of illness. Reported in up to 50% of newly diagnosed MM
- Unusual to have normal or stable kidney function beyond 6 months

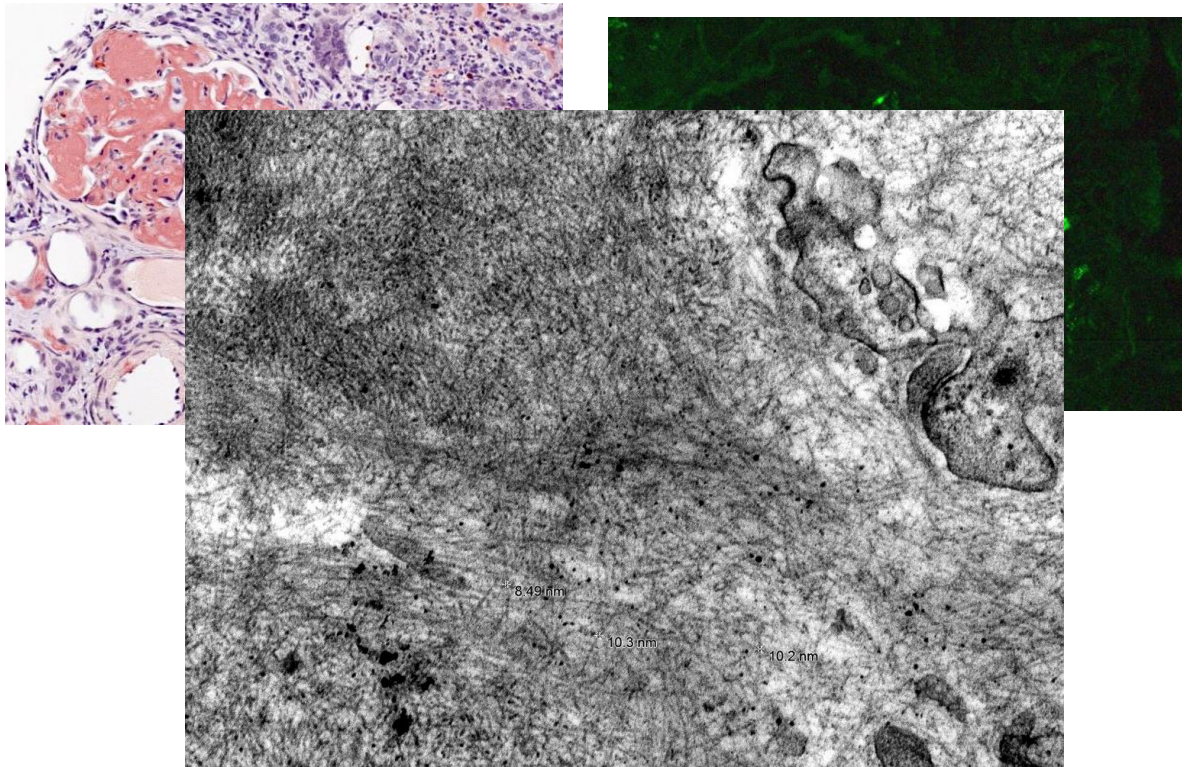
Monoclonal Gammopathy–Related Kidney Diseases. Advances in Chronic Kidney Disease. March 2022

2. Amyloidosis:

- Extracellular accumulation of Congo red-positive fibrils in various tissues (skin, soft tissues, liver, kidney, heart, GI, and PNS)
- Multi organ damage/ poor prognosis
- Median survival of AL amyloidosis: 2- 3 years
- Cardiac involvement: Important cause of early mortality.
- Fibrils immunopathologic types: monoclonal light chains (AL), heavy chains (AH), or light and heavy chains (AHL). (organized deposits)
- AL type: the most common (> 94%)
- Monoclonal Immunoglobulin–Related Amyloidosis: the most common type of kidney amyloidosis (86%) in the Western countries

- Acellular deposits that are pinkish on (H&E),Negative on PAS and silver stain.
- Lesions will be classically Congo-red positive and show "apple-green" birefringence under polarized light.
- IF will stain for 1 immunoglobulin component (usually κ or < λ).
- Electron microscopy will show non-branching fibrils





3. Monoclonal immunoglobulin deposition disease (MIDD)

- The difference between MIDD and amyloidosis is that the light (or heavy) chains in MIDD do not turn into fibrils, and the deposits are Congo-red negative.

nd non-fibrillary) M-Igs
rgans
of the deposits:
ost common

heavy chain deposition disease
light and heavy chain depositio

- Generally present with nephroti

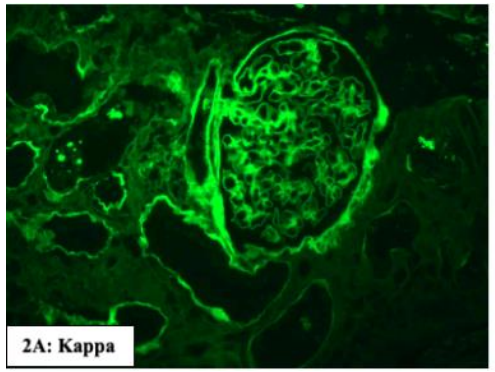
Similar to AL amyloidosis, patients with MIDD generally present with albuminuria >0.5 g in 24h and kidney impairment, leading to ESRD if left untreated

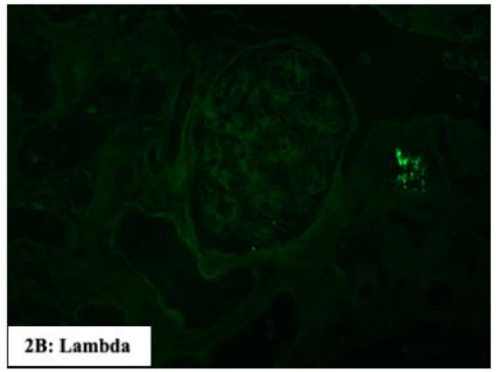
- κ is the most common
- Nodular mesangial expansion, glomerular and tubular basement membrane thickening,

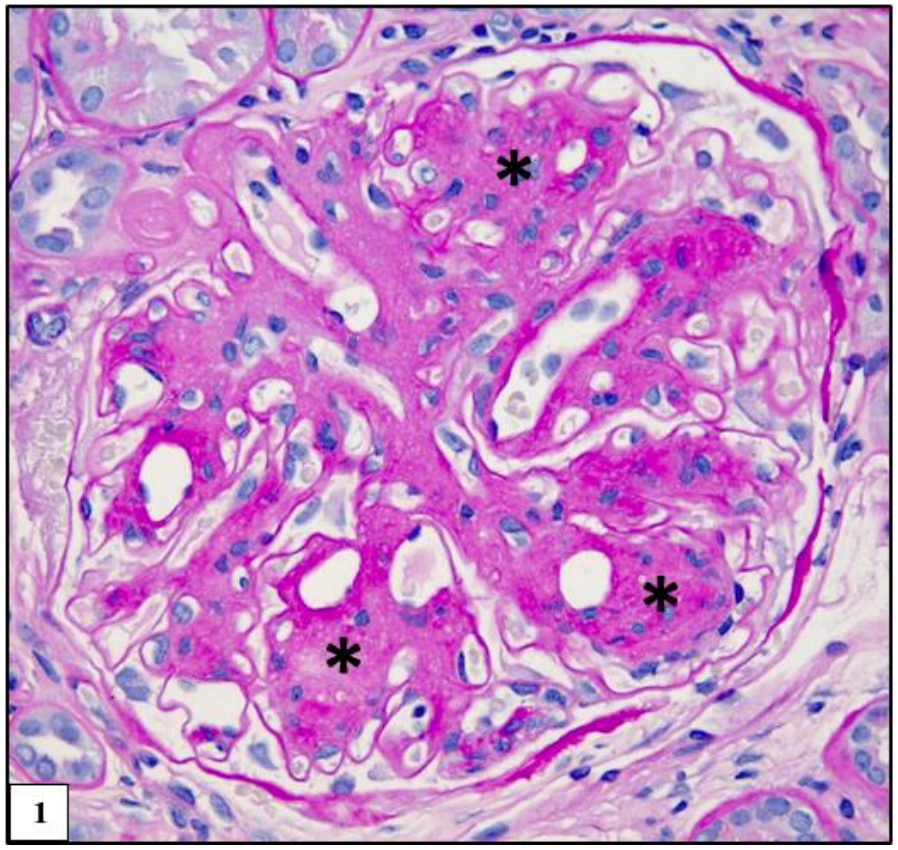
  Resembling Kimmelstiel-Wilson

  nodules*

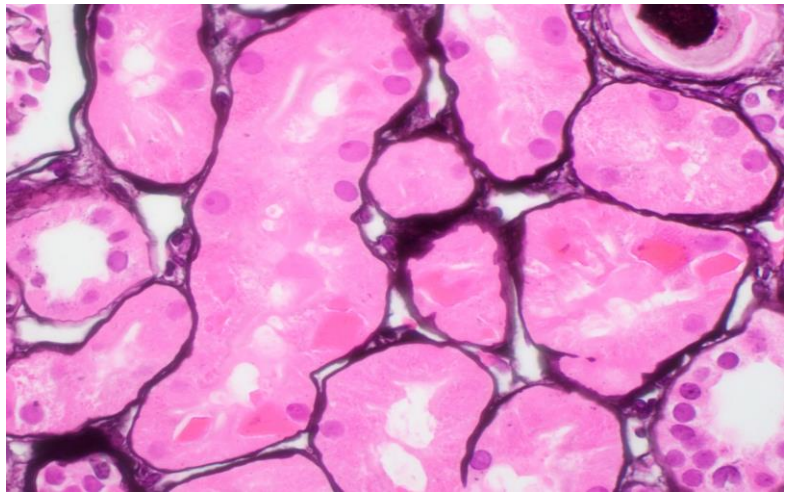
Tissue deposits are granular (instead of fibrillar), do not bind Congo red, and are electron-dense







4. Renal crystallopathies



- Light chain proximal tubulopathy

Accumulation and deposition of light chain monoclonal Igs leading to proximal tubular injury and dysfunction,

Resistant to degradation by lysosomal proteases in tubular cells

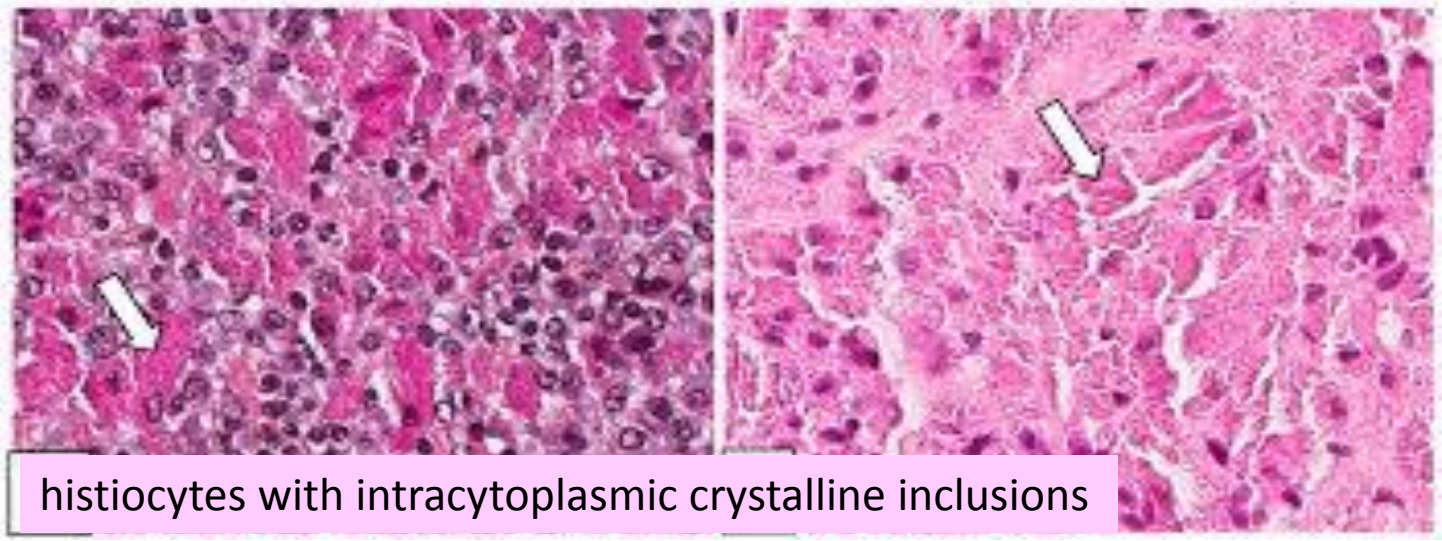
Resulting in intracellular crystal formation

- Fanconi syndrome: glucosuria, aminoaciduria, uricosuria, phosphaturia.
- Can be subtle: no clinical signs.

- Crystal-storing histiocytosis

Extremely rare: associated with B-cell malignancies (MM)

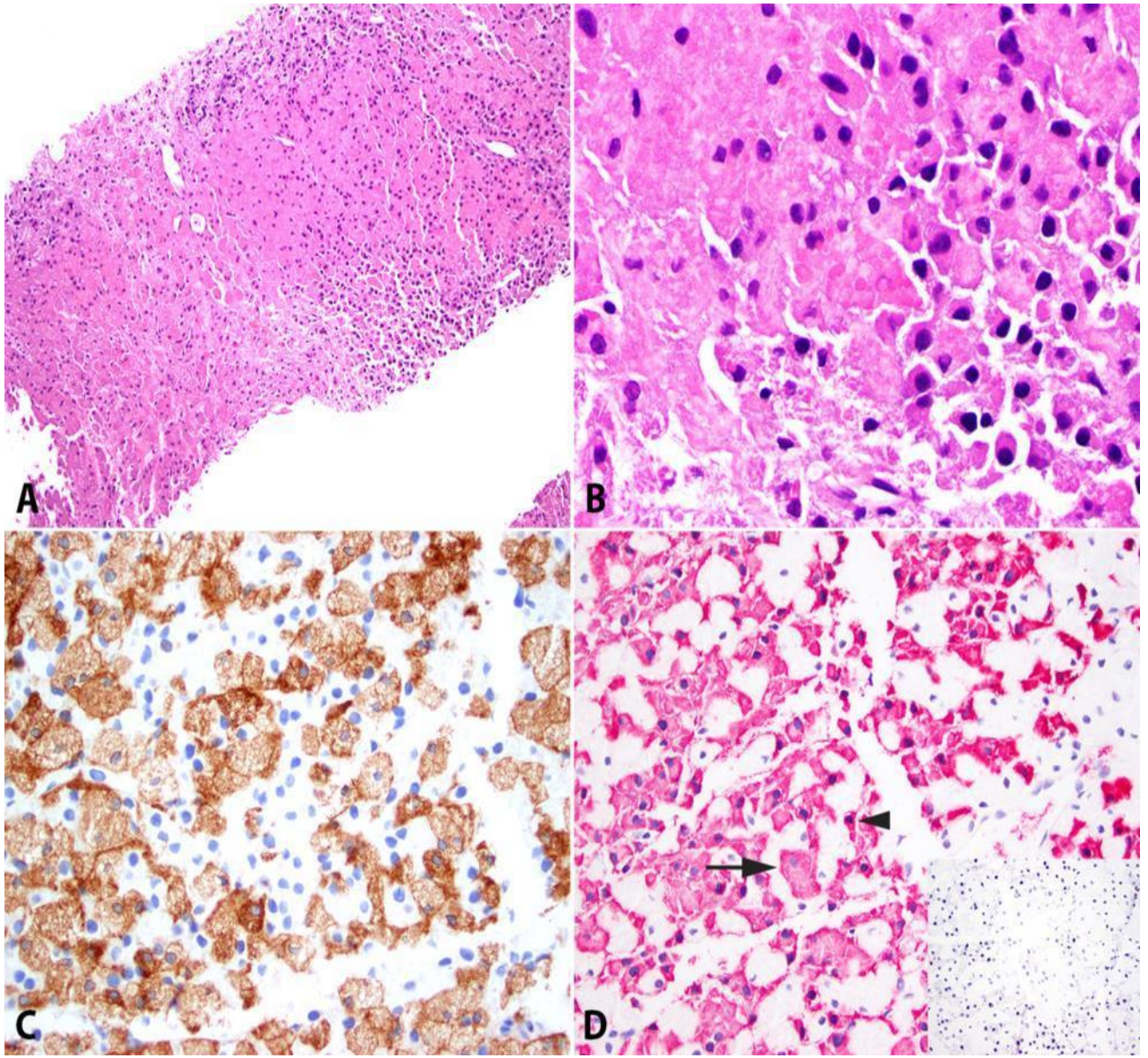
Needle-shaped crystal accumulation of monoclonal IgS in the histiocytes that typically found in the bone marrow, lungs, lymph nodes, skin, stomach, eyes, kidneys.



histiocytes with intracytoplasmic crystalline inclusions

Immunostains confirmed sheets of CD68 positive histiocytes separated by small, mature appearing plasma cells (Panel C)

By in situ hybridization, both the plasma cells (Panel D; arrowhead) and the histiocytes (Panel D; arrow) contain cytoplasmic kappa light chain immunoglobulin.



- Crystalglobulin-induced nephropathy (CrysIN) or crystaglobulinemia

- A rare M-Ig crystalline nephropathy
- Hypereosinophilic extracellular microcrystals within the systemic microvasculature (glomerular capillaries)
- Ischemic injury due to
  vascular injury
  thrombosis
  occlusion

- Sometimes, the pathogenic M-Ig has cryoprecipitating properties (Cryo-crystalglobulinemia)

PAS (b) and Masson trichrome (c) stained: segmental to global occlusion of glomerular capillaries by pseudothrombi

5. Gammopathy–Associated Thrombotic Microangiopathy

- Obstruction of the microvasculature, including arterioles and capillaries, by platelets and fibrin thrombi.
- Can result from immunoglobulin-mediated endothelial damage or chemotherapy.

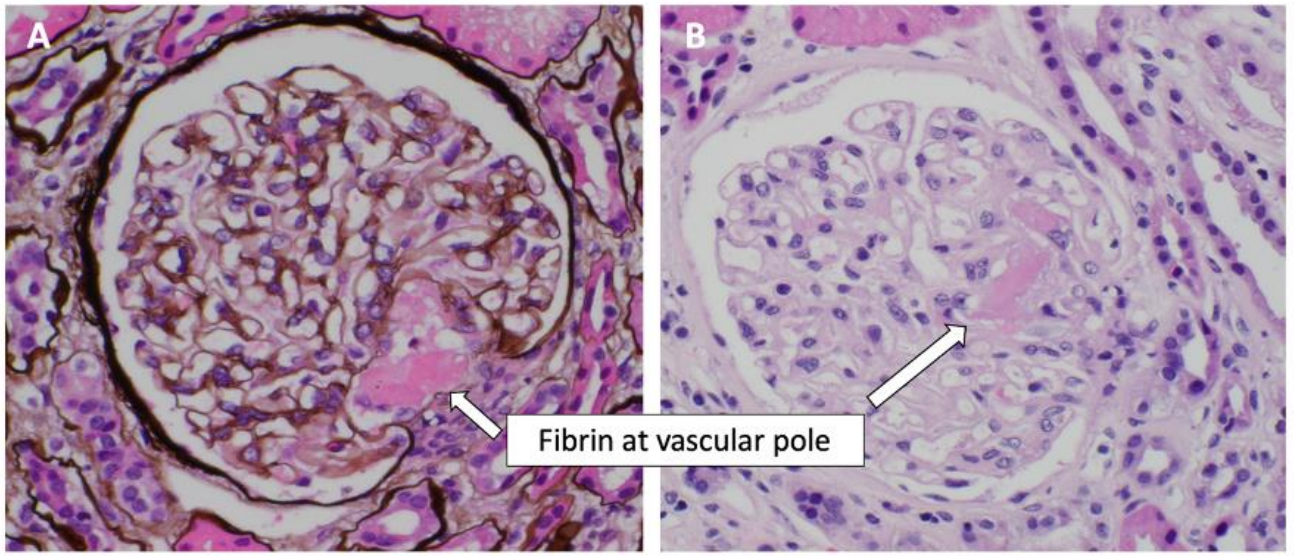


Figure 1: Fibrin thrombi at vascular pole A) non-argyrophilic eosinophilic material on JMS B) Pinkish amorphous material on H&E

6. Immune complex-mediated glomerular diseases (glomerulonephritides)

- **Proliferative glomerulonephritis with monoclonal immunoglobulin deposits (PGNMID)**
- Combination of nephrotic syndrome, kidney dysfunction, and hematuria.
- It mimics immune-complex glomerulonephritis.
- The deposits are usually IgG, are found only in the glomeruli, and consist of 1 type of heavy chain (gamma in the case of IgG) and 1 type of light chain (most often kappa), rarely IgA or IgM
- The complement system is usually activated, C3 and C1q can be positive

Affected glomeruli:

- Light microscopy:

  Endocapillary proliferation

  Membranoproliferative

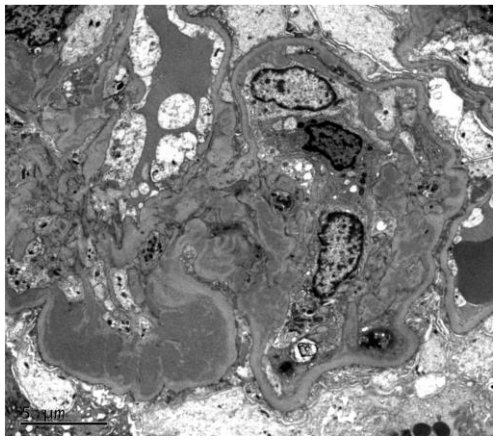
  Membranous nephropathy

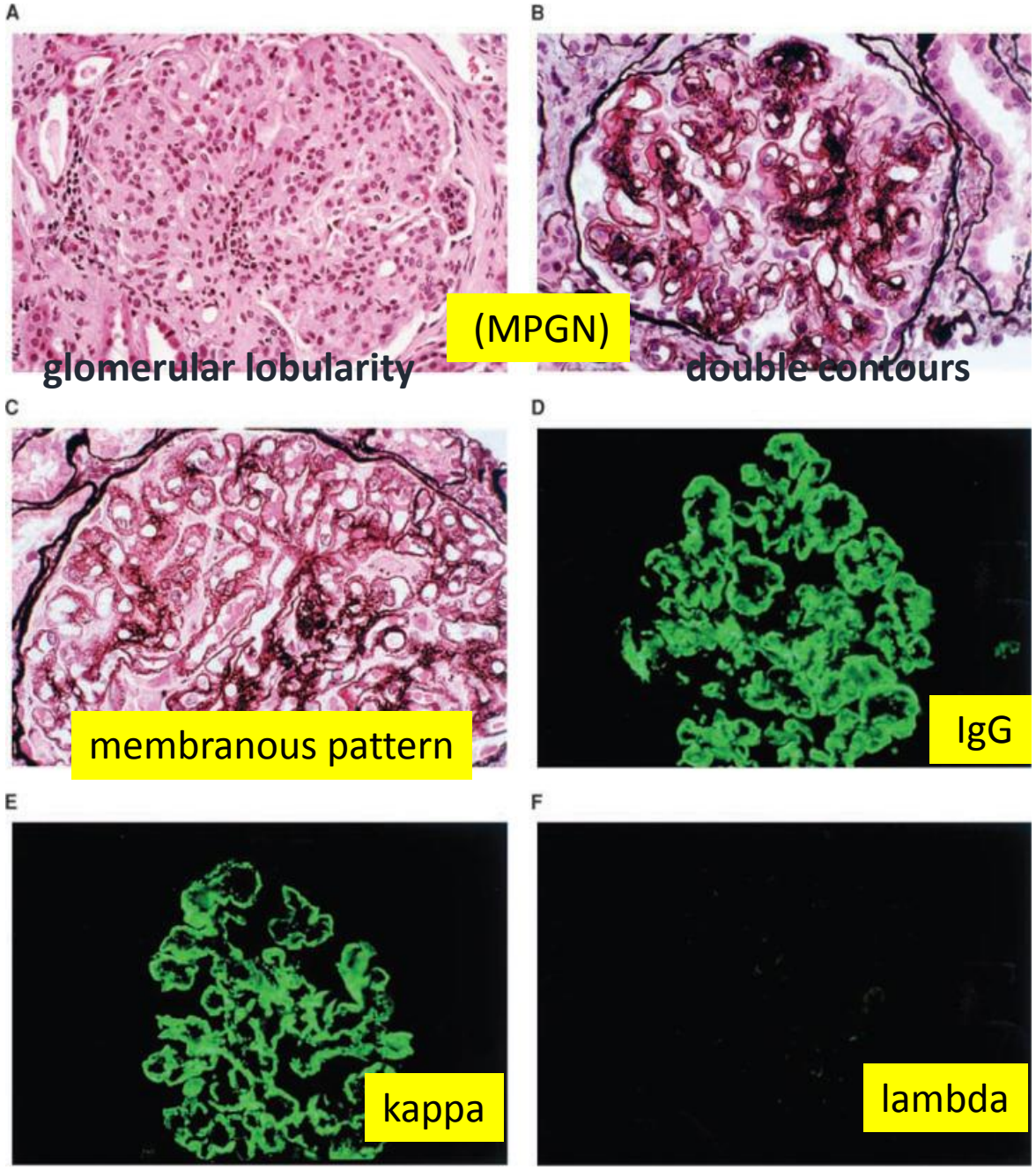
- Electron microscopy:

  Granular,

  Non-organized

  deposits



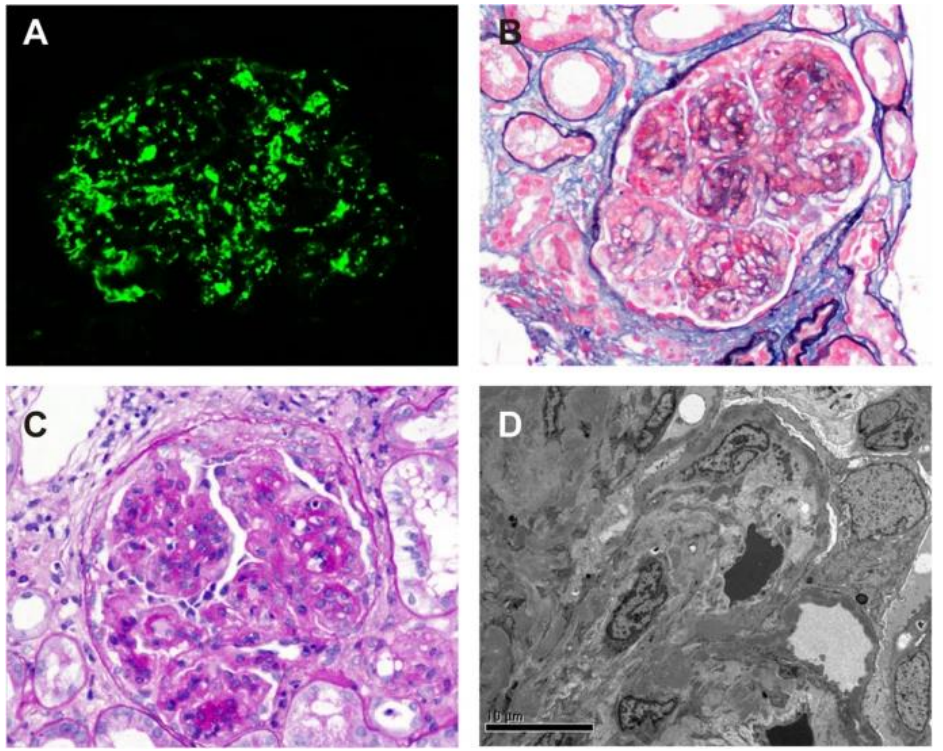


C3 glomerulopathies

- Very rare
- Complement dysregulation, C3 deposits in renal tissue with significantly fewer associated Ig deposits. C3 staining should be at least 2 orders greater than Ig
- M-protein acts as an autoantibody to activate C3 or C5 convertase, prolonging their normally short half-lives and activating the complement cascade.
- Clinically, presentations manifest as mild hematuria and proteinuria to florid glomerulonephritis

granular deposits of C3

lobular appearance



mesangial proliferation with endocapillary hypercellularity

dense deposits in sub-endothelia

Membranous-Like Glomerulopathy with Masked IgG Kappa Deposits (MGMID)

- Immune complexes consisting of masked deposits, show IgG kappa restriction by paraffin IF after pronase digestion.
- Subepithelial and mesangial deposits are seen by LM and EM.

Strong C3-staining by routine IF, can be mistaken for C3G if pronase digestion is not performed.

Clinical Presentation: Proteinuria (35% nephrotic-range)

Hematuria

AKI (29%)

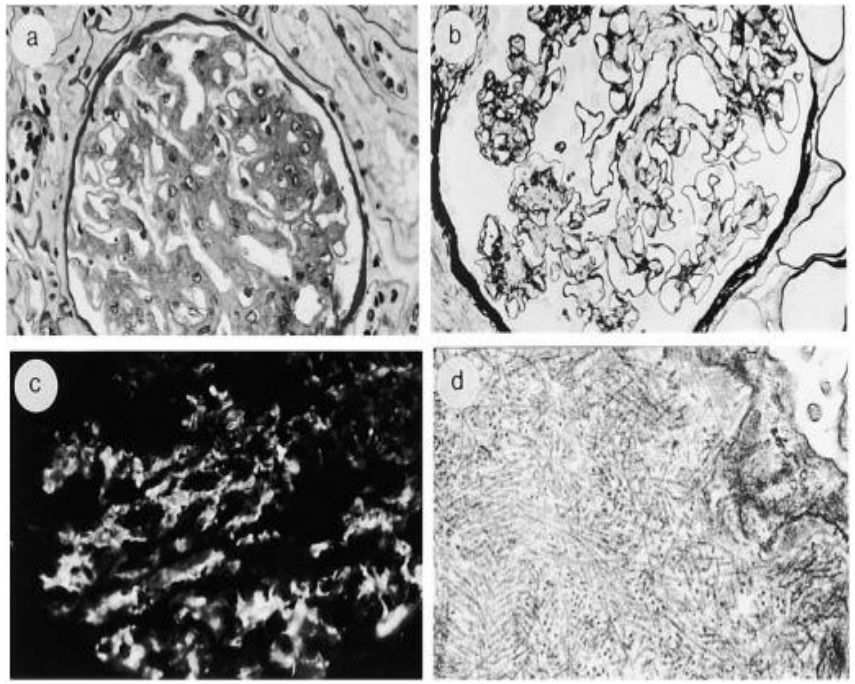
Autoimmune workup is positive (55%) : ANA, dsDNA the most common

mild disease and spontaneous remission/progress to ESKD

Monoclonal Gammopathy–Related Kidney Diseases. Advances in Chronic Kidney Disease. March 2022

Fibrillary glomerulonephritis

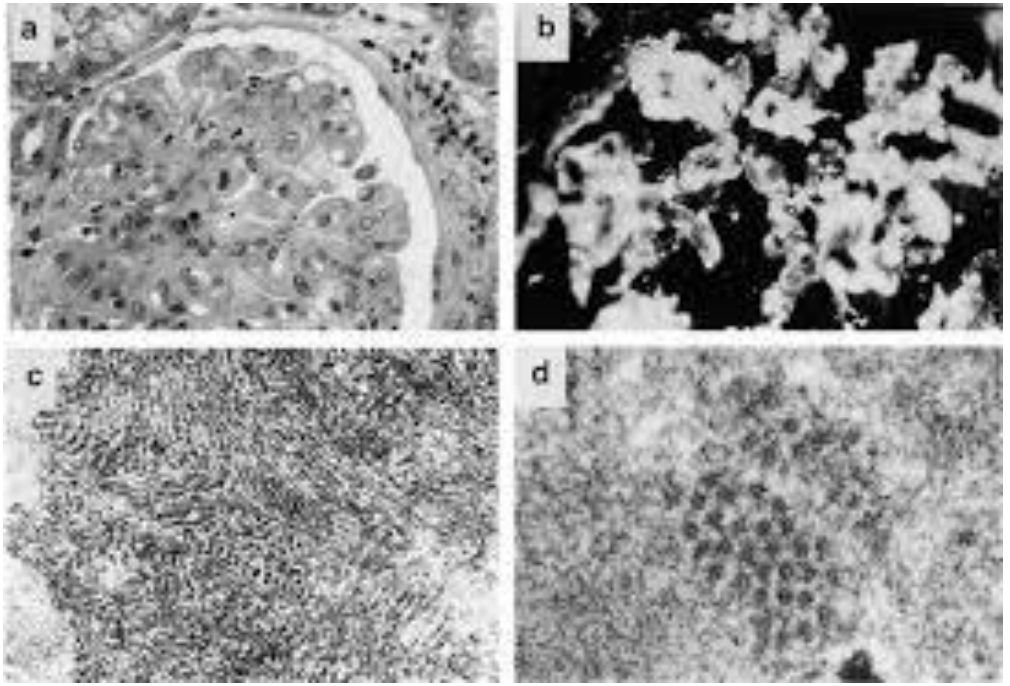
- Underlying processes: carcinoma, autoimmune dis, dysproteinemias
- Fibril structures of Igs, Congo-red negative, randomly oriented
- proteinuria (nephrotic range), hematuria, renal insufficiency, hypertension.
- Poor prognosis, half progressing to ESRD within several yrs, despite treatment
- Over a 30% rate of disease recurrence in KT



Immunotactoid glomerulopathy

- Focal monoclonal deposits forming a membranoproliferative or diffuse proliferative pattern on histology.
- Similar to fibrillar glomerulonephritis,

  but the fibrils are larger.
- The deposits are located in microtubules of the glomerulus and arranged in parallel arrays with positive C3 staining but negative Congo-red staining

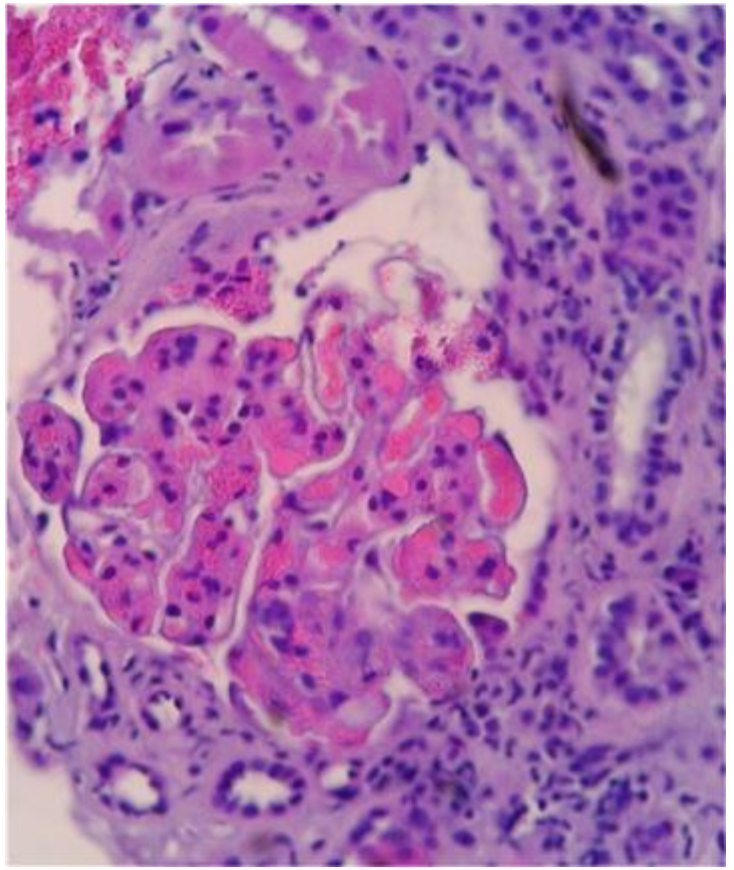


Cryoglobulinemia

- Type I cryoglobulinemia: precipitation of the M-protein at temperatures < 37 °C

  Redissolve when reheated

- Endocapillary proliferation with a membranoproliferative pattern.



multiple hyaline pseudothrombi in the glomerular capillaries

Non Immunoglobulin-Mediated Diseases

- Hypercalcemia,
- Volume depletion,
- Sepsis,
- Tumor lysis syndrome,
- Medication toxicity,
- Plasma cell invasion of the renal parenchyma
- These conditions can also exacerbate immunoglobulin-mediated diseases.

Hypercalcemia

- 15% of patients having serum calcium >11 mg/dL at presentation in MM.
- AKI in Moderate to severe hypercalcemia (>12 mg/dL)

  Renal vasoconstriction and promotes intratubular calcium deposition.

  Arginine vasopressin resistance: polyuria and volume depletion

  increase the toxicity of filtered FLc

Nephrotoxic agents

- Radiocontrast:

Urinary light chain excretion and volume depletion are seen in almost all myeloma patients with radiocontrast-associated AKI.

contrast agent
light chains,
volume depletion
} cause light chain precipitation/intratubular obstruction

- NSAIDs
- Bisphonates: Zoledronate → ATN
  pamidronate → Collapsing FSGS
- lenalidomide, bortezomib, carfilzomib, ixazomib

Hyperuricemia

- Uric acid (>15 mg/dL): in up to 50% of patients with MM
- Raised uric acid levels as a part of tumor lysis syndrome are rare even after chemotherapy for monoclonal gammopathy

History and Physical

- Multiple myeloma initial symptoms:

  Often subtle

  Can include malaise, fatigue, weight loss, and bone pain

  CKD and proteinuria aggregating

  fulminant AKI requiring dialysis

  symptomatic severe hypercalcemia and dehydration.

- Anemia is a characteristic feature of multiple myeloma (75% of cases)
- Therefore, paraprotein disease should be considered in patients with kidney dysfunction and anemia.

- Amyloidosis or MIDD:
- GI bleeding, Heart failure, Elevated ALP, Cardiac arrhythmias, periorbital purpura
- Hypotension +/- orthostasis as a result of coexistent myocardial and autonomic nervous system involvement.
- Hypertension is seen in cast nephropathy and MIDD due to renal failure.
- Cardiac involvement is more common in AL.
- The proteinuria : monoclonal immunoglobulins known: Bence Jones proteins or Urine free light chains (FLCs)
- AKI or worsening CKD and proteinuria

Evaluation

The International Myeloma Working Group recommends:

- 24-hour urine protein electrophoresis
- Serum FLC assay

} To evaluate for cast nephropathy

- The sFLC assay is more sensitive than the urine protein electrophoresis for detecting FLCs in diagnosing new cases and relapses of the disease.

- In general:
  nephrotoxic agents, volume status, acid-base status, urinalysis with microscopic examination, and serum calcium
  should be reviewed.

- **Serum electrophoresis (SPEP):**

  often cannot detect levels of M-protein <500 mg/dL

  especially FLC, as these have a sh

One analysis found that SPEP di
over 50% of AL amyloidosis

- **Serum-free light chain ass**

  use antibodies against anti

  more sensitive than SPEP.

- low-level M-protein may not be detected in urine until the proximal tubular reabsorption capacity is exceeded,
- 24-hour urine collections may not be accurate.

- **International guidelines recommendation:**

Using sFLC, SPEP, and serum immunofixation instead of urinary tests

- **The normal serum free kappa to lambda ratio:** 0.26 to 1.65

- **Elevated sFLCs and an abnormal kappa to lambda ratio:**
  indicate a monoclonal plasma cell disorder.

- Light chain cast nephropathy strongly suspected in:
  unexplained kidney injury over fewer than 6 months and
  an elevated sFLC level ≥1500 mg/L

- In patients with levels of serum FLCs >500 mg/L,

albumin excretion may helpful to avoiding the need for kidney biopsy.

- In glomerular disease: albumin is the predominant urine protein.
- In cast nephropathy: albumin is typically <10% of the urinary protein.

Quantifying urinary light chains is also helpful as amounts <1 g/day point to causes other than cast nephropathy.

One recent study observed that almost one-third of patients with multiple myeloma who underwent kidney biopsy had non-myeloma–related kidney disease.

Urinary albumin excretion patterns of patients with cast nephropathy and other monoclonal gammopathy-related kidney diseases. Clin J Am Soc Nephrol. 2012 Dec;

- **Cast nephropathy** typically presents with proteinuria without albuminuria, as the glomerular basement membrane (GBM) remains intact.
- **MIDD** and **light chain amyloid** present with significant proteinuria and albuminuria due to the involvement of the GBM.

- Although there are concerns regarding the safety of **kidney biopsy** in patients with multiple myeloma due to presumed coagulation abnormalities, several studies indicate the procedure is generally safe, having similar rates of adverse events as encountered in the general population.

kidney biopsy can be deferred as follows:

- A presumptive diagnosis of light chain cast nephropathy:

MM with AKI or subacute kidney injury with an sFLC ≥1500 mg/L along with the predominance of monoclonal light chains in the urine by urine protein electrophoresis and immunofixation

- A presumptive diagnosis of renal amyloidosis:

Albuminuria or nephrotic syndrome with an established diagnosis of immunoglobulin light chain AL based on a biopsy of non-kidney tissue

- A presumptive diagnosis of light chain proximal tubulopathy:

MM or monoclonal gammopathy with symptoms of Fanconi syndrome (aminoaciduria, glycosuria in non-diabetics, hypophosphatemia, hypokalemia, hypouricemia, proteinuria)

Treatment / Management

- The initial treatment of patients with myeloma-associated kidney diseases should focus on assessing the degree of renal impairment and correcting hemodynamics, volume status, and electrolyte disturbances.
- The formation of casts and paraprotein concentration needs to be minimized as early as possible.
- Most patients have some degree of renal injury, which improves with myeloma treatment.

- Nephrotoxic agents should be discontinued
- adequately hydration:

  Volume repletion with targeted U/O : 100 to 150 mL/hour (about 3 L/day).

  Intravenous or oral fluid therapy unless contraindicated
- Loop diuretics: can be used cautiously in patients with hypervolemia (precipitate the cast formation)
- Hemodialysis: may be needed to correct acid-base balance, electrolyte disturbances, volume overload, and uremic symptoms. extended dialysis sessions, using the dialysis cartridge to help remove the light chains has improved renal recovery.

Extracorporeal Removal of Light Chains

- plasma exchange or plasmapheresis:

  Decrease the concentration of the paraproteins. (FLC MV: 25-50 kDa)

  The use of plasmapheresis is limited in cast nephropathy due to a large volume of distribution of light chains, resulting in a rapid rebound of light chains after the treatment.

- High-cutoff hemodialysis (HCO-HD):

  Removal of FLCs based on their MW

  This technique uses a large-pore membrane Hemofilter (10 nm) for repeated dialysis sessions over several weeks to remove FLCs.

High-cutoff hemodialysis (HCO-HD) challenges:

- An in vitro study revealed that an HCO-HD membrane could remove 90% of FLCs over 3 weeks.
- One retrospective study evaluated HCO-HD in conjunction with chemotherapy: 63% of patients achieved dialysis independence.

  Albumin is lost obligatorily with HCO-HD and requires replacement.

Immunoglobulin free light chain levels and recovery from myeloma kidney on treatment with chemotherapy and high cut-off haemodialysis. Nephrol Dial Transplant. 2012

- One randomized controlled trial compared patients with myeloma cast nephropathy treated with bortezomib and dexamethasone, then treated with intensive dialysis and either a high-cutoff dialyzer or conventional dialyzer:
- The number of dialysis-independent patients was greater in the high-cutoff group, but the difference did not rise to clinical significance (possibly due to low study numbers).
- A key association with dialysis independence was achieving a sFLC <500 mg/dL after the first chemotherapy, which is considered the cut-off for cast formation. Additional studies are required in this area.

Chemotherapy

- 80% of patients may recover renal function by 3 weeks if there is a reduction in the sFLC by at least 60%.
- This can be achieved by early diagnosis and prompt initiation of treatment.
- During treatment, labs must be monitored carefully for any evidence of tumor lysis syndrome.

The commonly regimens for multiple myeloma:

- Cyclophosphamide, bortezomib, and dexamethasone (CyBorD)
- Lenalidomide, dexamethasone (Rd)
- Lenalidomide, bortezomib, dexamethasone (RVD)
- Lenalidomide, thalidomide, dexamethasone (VTD)

The most commonly used agent is bortezomib, a proteasome inhibitor, which has a rapid onset of action and is the most studied chemotherapeutic for monoclonal gammopathies

Bortezomib does not need renal dose adjustment
Lenalidomide dosage needs to be adjusted based on creatinine clearance

- MM associated with light chain cast nephropathy:

  Should receive chemotherapy, such as CyBorD, as quickly as possible to suppress light chain production

- Triplet regimen:

  Suitable for young and healthy patients, especially in severe AKI

- Doublet bortezomib-dexamethasone regime :

  First-line treatment In older patients with comorbidities

- Non-multiple myeloma conditions: The decision for treatment is less clear-cut and also less well-studied due to low occurrence.

- Amyloidosis: treatment similar to MM due to significant morbidity and mortality.

  high-dose dexamethasone with melphalan or bortezomib-based

  regimens and autologous stem cell transplants in eligible patients.

- MGRS: treatment similar to MM may be applicable given the significant complications, specifically in acute/subacute kidney injury +/- proteinuria> 1 g/d

Transplantation

- **Hematopoietic stem cell transplant (HSCT):**

  Important and potentially curative management for MM

  several series have reported that HSCT is safe and effective in patients with kidney disease.

- Patients younger than 70 years with an ejection fraction >45% and without active infection should be considered for HSCT.

- **kidney transplantation:**

The data on patients with multiple myeloma-associated kidney impairment is limited.

- An option for carefully selected patients with successful HSCT and hematologic complete response.
- Acute T-cell–mediated rejection may develop due to a chemotherapeutic agent, lenalidomide.
- can be considered if myeloma remission last for at least 3-5 yrs.
- can be associated with myeloma recurrence due to immunosuppressive agents and Ig-mediated graft rejection.

MGRS with IgM Monoclonal Protein

- very rare, unclear what the best treatment options
- IgM proteins form large multimers lead to hyperviscosity other than immunoglobulins.
- If the bone marrow clone is CD20+, rituximab-based therapy is considered first-line treatment.

  It can be combined with dexamethasone plus cyclophosphamide or

  bendamustine

- A decrease in the nephrotoxic M-protein is key to renal recovery; Hematology parameters should be measured for response rates:

  sFLC assays

  Repeat BMB in cases where the M-protein is difficult to measure.

- Renal recovery usually lags behind hematologic response (in 1 study, up to a year), so serum creatinine and urine protein may not accurately indicate treatment response.

Prognosis

- Worse outcomes : Myeloma presenting with significant kidney dysfunction compared to those with normal renal function
- Main predictor of survival outcomes: Response to chemotherapy
- Average survival in chemo-responsive patients: 3 yrs
- Recent cohort studies:

  patients with myeloma kidney requiring dialysis with no recovery of renal function: 50% survival at <1 yr.

  patients who can discontinue dialysis: 50% at 3 yrs.

ESRD due to Multiple Myeloma in the United States, 2001-2010. J Am Soc Nephrol. 2016

- AKI secondary to volume depletion and hypercalcemia: good recovery as opposed to those with dense cast formation and tubular damage.
- In 1 study, a higher number of cortical casts was independently linked to a lower follow-up eGFR and a greater risk of remaining on dialysis.
- These favorable trends are attributable to increasingly effective chemotherapeutic options, such as thalidomide, lenalidomide, and bortezomib.
- The improvement in kidney function has been reported to be 50% - 80% in patients with newly diagnosed myeloma managed with bortezomib

Renal Disease in Monoclonal Gammopathies. StatPearls Publishing; 2025 Jan


THANK YOU!