Supplemental Table 1: Some examples of proposed terminology for use in the diagnosis of MIg-associated renal diseases. The hematologic disease should be based on the diagnostic hematologic criteria available at the time of kidney biopsy after discussion with the hematologist.

- Cast nephropathy (myeloma kidney), (no designation of the hematologic disease required because this diagnosis is MM-defining)
- AL-amyloidosis, multiple myeloma-associated
- AL-amyloidosis, plasmacytoma-associated
- AL-amyloidosis, B-cell lymphoma-associated
- Light chain deposition disease, smoldering multiple myeloma-associated
- Light chain deposition disease, B-cell lymphoma-associated
- Light chain deposition disease, MGRS-associated
- Cryoglobulinemic glomerulonephritis, Waldenström macroglobulinemia-associated
- Cryoglobulinemic glomerulonephritis, chronic lymphocytic leukemia-associated
- C3-glomerulonephritis, MGRS-associated
- Crystal storing histiocytosis, B-cell lymphoma-associated
- Light chain proximal tubulopathy, MGRS-associated
- Immunotactoid glomerulopathy, lymphoplasmacytic lymphoma-associated
- Proliferative glomerulonephritis with MIg deposits, MGRS-associated
- Thrombotic microangiopathy, MGRS-associated
- Proliferative glomerulonephritis with MIg deposits, with no demonstrable serum/urine M-protein
- Light chain deposition disease, hematological evaluation pending

Supplemental figures and legends

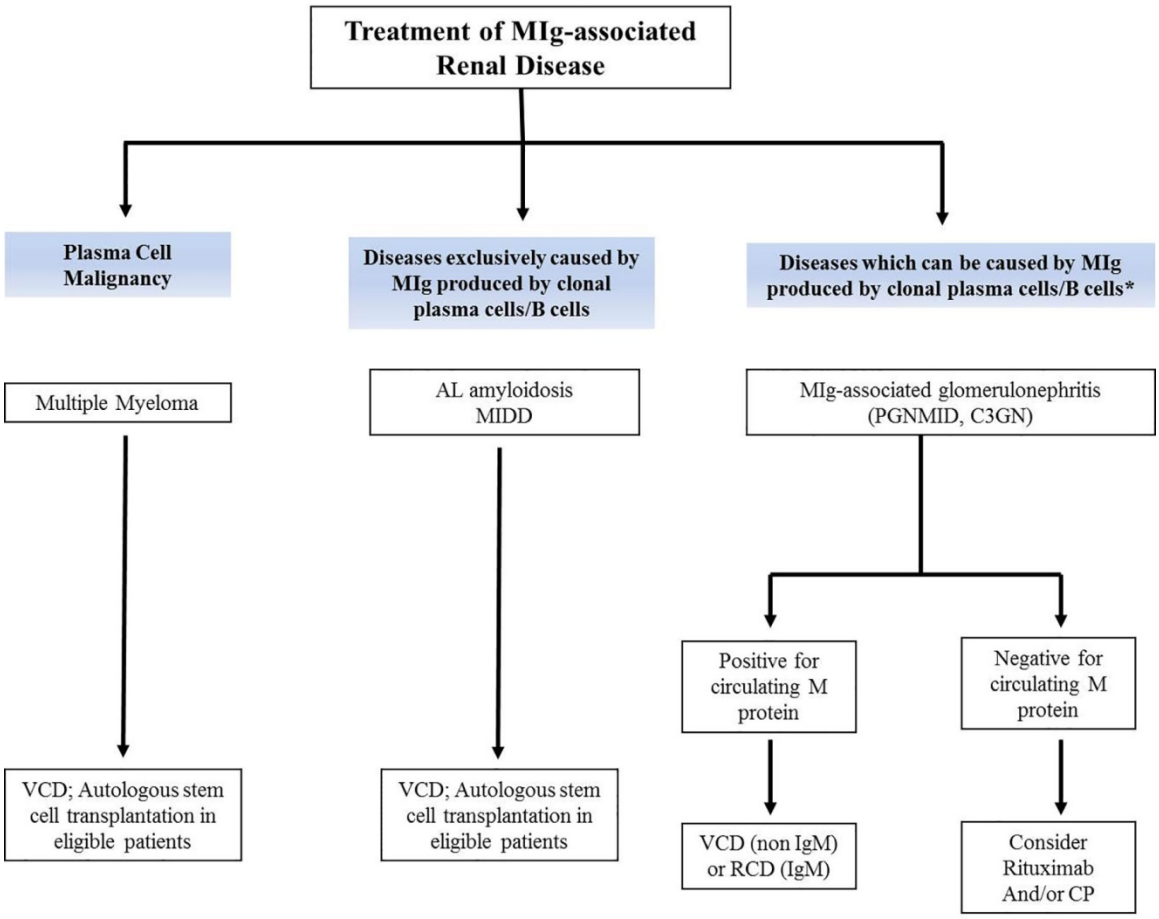

Figure 1: Suggested approach to the treatment of MIg-associated renal disease. We recommend VCD for patients with multiple myeloma and acute kidney injury; in patients without renal injury, VRD is typically preferred. We suggest VCD based regimen for a non-IgM MIg (as these are usually related to a plasma cell clone) or RCD based regimen for IgM MIg (as these are usually related to a B lymphocyte clone). For patients with no detectable serum or urine M-protein we suggest a cyclophosphamide or rituximab based regimen. It is also important to exclude other etiologies that may be causing the renal lesion. It should also be pointed out that this figure represents a basic treatment algorithm. Treatment of MIg-associated renal disease is evolving with newer therapeutic options on the horizon. While the basic algorithm may be applied to rare diseases such as LCPT, CSH, crystalglobulinemia and TMA, there are no large studies available to recommend specific treatment guidelines and in each case treatment should be tailored to the underlying hematologic disease. *In these disorders, data to support therapy against the neoplastic plasma cell or B lymphocyte clone are limited. Abbreviations: VRD, bortezomib,

lenalidomide, dexamethasone; VCD, bortezomib, cyclophosphamide, dexamethasone; RCD, rituximab, cyclophosphamide, dexamethasone; CP, cyclophosphamide.

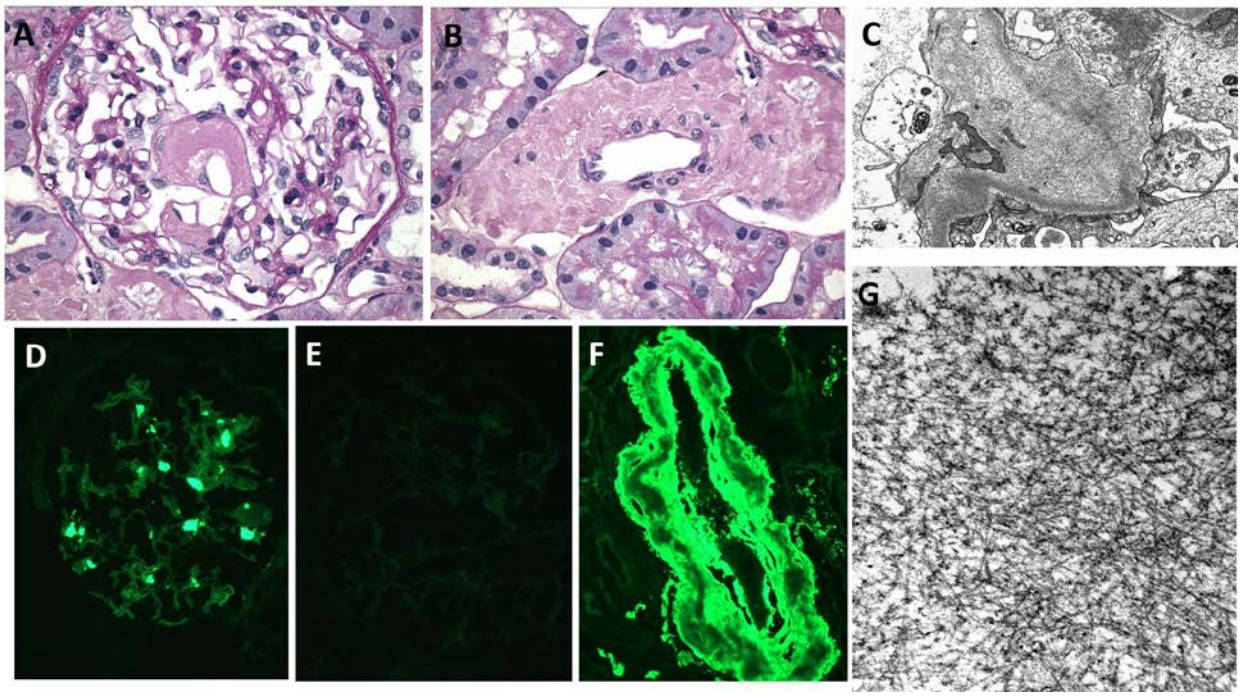

Figure 2: Renal amyloidosis, AL-lambda. A 68 year-old man with history of IgA-lambda serum M-protein and smoldering myeloma develops albuminuria 1.5 g/day, serum creatinine 0.9 mg/dl and orthostatic hypotension. Renal biopsy reveals: A) Segmental mesangial deposits of amorphous PAS-pale material that infiltrates the overlying glomerular basement membranes, Periodic acid Schiff (PAS) x 600; B) Massive PAS-pale deposits infiltrating the media of interlobular arteries, replacing the medial myocytes, PAS x600; C) Randomly oriented fibrils of mean 10 nm diameter within the mesangium and forming spicular projections through the overlying glomerular basement membrane (electron micrograph, x25,000); D) Immunofluorescence showing smudgy positivity for lambda light chain only involving the mesangial regions, x400. There was no staining for IgA, IgG, or IgM (not shown); E) Immunofluorescence staining of the same glomerulus showing negativity for kappa light chain, x400; F) Immunofluorescence staining for lambda light chain involving the arterial media, x200; G) High power electron microscopic view of the randomly oriented amyloid fibrils, mean 10 nm diameter, within the vascular media, x80,000. Congo red stain for amyloid (not shown) was positive in the distribution of the glomerular and arterial wall deposits, confirming a diagnosis of renal amyloidosis, AL-lambda type.

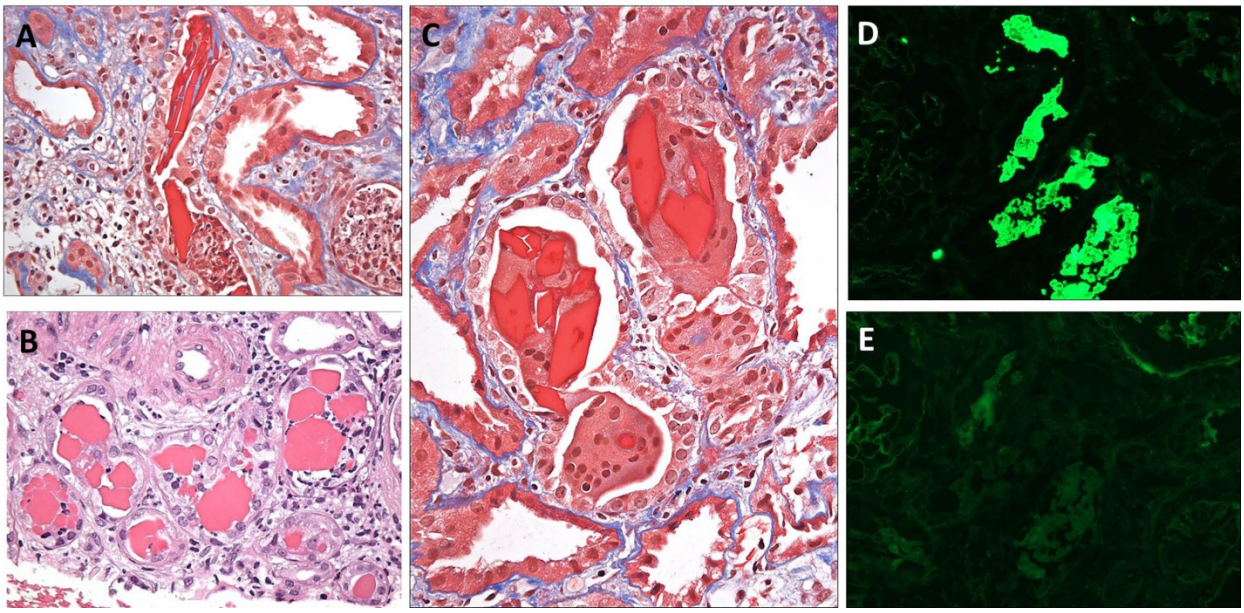

Figure 3: Myeloma cast nephropathy, kappa type. A 65 year-old man with no prior medical history presents with anemia, AKI, back pain, serum creatinine 5.4 mg/dl, 1+ proteinuria, and marked elevation of kappa serum FLCs. Renal biopsy shows: A) Elongated needle-shaped trichrome-red crystals with sharp edges and neutrophil reaction within the distal tubular lumina. The adjacent interstitium is expanded by fibrosis and chronic inflammation (Masson trichrome, x200); B) Hard, brittle brightly eosinophilic tubular casts with geometric shapes and focal adherent mononuclear cells and neutrophils, H&E x400); C) High power view showing distal tubular trichrome-red casts with rhomboidal shapes engulfed by multinucleated giant cells (trichrome, x600); D) Immunofluorescence showing intense staining of the crystalline casts for kappa light only (x400). Stains for IgG, IgM, IgA, C3, and C1 were negative (not shown); E) The same immunofluorescence field stained for lambda light chain is negative in the distribution of the casts (x400).