

Light chain deposition disease with low glomerular proteinuria and multiple myeloma: If you search you find

Light Chain Deposition Disease (LCDD) with low proteinuria is rare and represents a diagnostic challenge. Only 14 patients with biopsy proven renal LCDD and low proteinuria have been, in fact, reported in a recent multicentre retrospective study,¹ confirming that this form is likely underdiagnosed.

An immigrant 65 years old Moldovan male patient, was referred to our Nephrology Unit for CKD of unknown origin (creatinine 350 $\mu\text{mol/L}$, proteinuria 0.8 g/day, anaemia (Haemoglobin [Hb] 9 g/dL), hypocalcemia). At the admission, WBCs and platelets count, blood calcium and phosphorous were normal, Hb 9 g/dL, creatinine 358 $\mu\text{mol/L}$, proteinuria 0.85 g/day and gamma globulins were reduced (9%). ANA, ANCA and complement were negative, serum

free light chains (sFLCs) increased (kappa 136 mg/L, lambda 30 mg/L), with high kappa/lambda ratio (4.53). Urinary monoclonal FLCs kappa (Bence Jones proteinuria) were present and serum immunoglobulins were slightly reduced. Renal biopsy showed, mesangial and arteriolar FLCs kappa and lambda positive deposits and Congo Red and Thioflavine negative deposits. Immunofluorescence was negative for IgG, IgM, IgA, C1q, C3 and fibrinogen. Glomerular, mesangial, glomerular basal membrane and perivascular FLCs kappa positive dense deposits allowed the diagnosis of LCDD (Figure 1).

Medullary osteobiopsy found 16% of clonal immunophenotype kappa plasma cells allowing the diagnosis of Multiple Myeloma (MM) with LCDD.

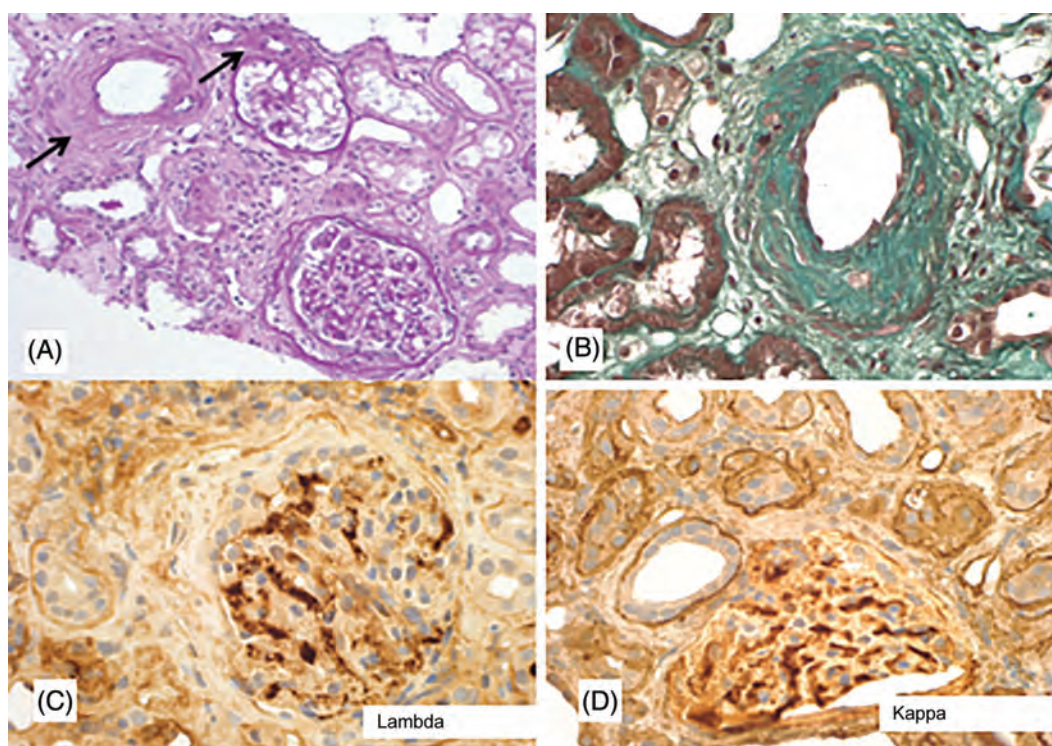


FIGURE 1 One sclerotic glomerulus, 2 with PAS positive mesangial deposits and duplication of Bowman capsule in one. (A) Arterial thickening of the parietal wall of two arteries (arrows). No sign of inflammation. PAS staining $\times 20$; (B) Artery with deposit of amorphous material, Masson trichrome staining $\times 40$. Positivity for Lambda light chain antibody in glomerular deposits (C) $\times 40$ and prevalence for Kappa light chain antibody (D) $\times 40$. Congo red and Thioflavin-T were negative (not shown)

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No osteolytic lesions were present at the CT scan and echocardiogram was normal.

Treatment with Bortezomib/Dexametasone (2.52 mg/40 mg) was able to induce after 25 days a remarkable reduction of sFLCs kappa, the urinary FLCs disappeared and after 3 months the treatment was interrupted. The patient was then found suitable for autologous bone marrow transplant.

Approximately 50% of patients with MM experiences acute kidney injury (AKI) or CKD and severe AKI requiring dialysis generally occurs in 1–3% of these patients with reports in up to 12%.²

LCDD with low or no glomerular proteinuria has been reported only in 1% of all the renal biopsy proven diagnoses. Due to the CKD of unknown origin that our patient presented at the admission in our Unit, renal biopsy was performed allowing the diagnosis. In the absence of significant proteinuria, in fact, paraproteinemias are not, generally, fully studied.

This case confirms the need of an accurate diagnostic workup for monoclonal gammopathy, including the determination of FLCs. The need of renal biopsy in patients with CKD of unknown origin, even without significant proteinuria, becomes therefore crucial for LCCD diagnosis. Early recognition of MM and its complications is extremely important, given the positive renal outcome following the timely clone-directed therapy in addition to the autologous stem cells' transplant.³

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Very large abscesses of lower limbs by *Nocardia farcinica* requiring surgical management in patient with minimal change disease under chronic steroid treatment

1 | CASE REPORT

A 70-year-old Caucasian male was admitted to general medicine unit for swelling and pain in the right leg associated with fever in the last month. The medical history evidenced biopsy-proven minimal change disease (MCD) complicated by nephrotic syndrome treated with 1 mg/kg/day of prednisone for 16 weeks with subsequent tapering and partial proteinuria remission (from 10 to 3 g/day). During steroids tapering, due to significant increment of proteinuria, prednisone dose was re-increased (0,8 mg/kg/day).

At admission, the physical examination revealed a marked swelling and painfulness of the right lower limb and mild erythema of the

anterior region of the left leg. Laboratory blood tests showed: white-blood-cell-count 7720/ μ L, ferritin 3967 ng/mL, C-reactive-protein 6.5 mg/dL and negative procalcitonin. The ultrasonography (US) of the right lower limb showed diffuse multiple abscesses. A US-guided aspiration of one of the lesions was performed. Gram stain of the aspirated pus revealed Gram-positive filamentous bacilli with branching. The isolated strain was subjected to 16S rRNA sequence analysis that showed 100% match with *Nocardia farcinica*. An empirical treatment with intravenous imipenem 500 mg/QID was started. Based on the result of microbiological study, moxifloxacin 400 mg/QD was added. Magnetic Resonance Imaging (MRI) of lower limbs demonstrated multiple large confluent abscesses in the whole right