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Acute kidney injury with active urinary sediment analysis, a positive ANCA test and hypocomplememtemia: A tough situation

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CASE REPORT

A 55-year-old man was hospitalized for severe sepsis with undefined starting point and an acute oliguric renal dysfunction.

His medical history revealed an active smoker (20 to 30 cigarettes/day), with chronic bronchitis, diabetes *mellitus* type 2 under dietary control, five years of controlled hypertension, dyslipidemia and an estimated alcohol consumption of 100g/day. He was regularly treated and controlled with ramipril and hydroclorothyazide and he regularly took nonsteroidal anti-inflammatory drugs for low back pain.

Two months before admission, the patient went to the hospital for acute gastroenteritis and he was discharged with symptomatic therapy. One month later, he presented to the emergency room with complaints of asthaenia, anorexia, weight loss (10 kg in two months) and a new onset of dyspnoea on exertion. One week before admission, the dyspnoea and cough with increased bronchial production worsened. A purpuric and pruritic rash appeared on the lower extremities. He had no fever. He denied therapeutic changes.

The patient returned to the emergency room. He was febrile, with hypotension and tachycardia. He presented signs of dehydration and oliguria. There were wheezing sounds in the lower third of both lung fields. Cardiac auscultation showed rhythmic sounds and systolic murmur in the aortic focus (III/VI). He had a distended abdomen with ascites without pain and some purpuric lesions on the lower limbs that did not disappear with digital pressure.

The serum analysis showed: anaemia (8.9 g/dl normocytic normochromic), leukocytosis, neutrophilia, thrombocytopenia (91000) and no eosinophilia; no schistocytes were found; PCR was 91 mg/L. He presented normoglicaemia, acute renal failure (urea of 188 mg/dl and creatinine of 3.17 mg/dl, with a basal value of serum creatinine 0.85 mg/dl one month later), hyperkalemia (K^+ – 5,5), DHL – 324, hypoalbuminemia (2.32 g/dl) with total protein in normal range.

Urinalysis showed erythrocyturia (› 25/field), leucocyturia (› 25/field) and proteinuria 200 mg/dl; there was no eosinophiluria nor urinary dysmorphic erythrocytes. The kidney ultrasound was normal. Blood gas analysis showed a metabolic acidosis with normal gap and respiratory failure.

Abdominal ultrasound showed splenomegaly and ascites. There was no consolidation image on chest radiography. Blood and urine cultures were collected and the patient began taking fluids and empirical antibiotic treatment (doxycycline and amoxicillin- clavulanic acid), subsequently changed according to antimicrobial susceptibility. The initial response was unfavourable, with maintained haemodynamic instability, respiratory distress and oliguria with need of haemodynamic, ventilator and renal replacement

support (SLED – sustained low efficiency dialysis) at the Intensive Care Unit.

The remaining study conducted revealed:

Blood cultures: Streptococcus agalactiae;

Urine culture was sterile;

Serology for HCV was positive; it was negative for HBV and HIV;

A type II cryoglobulinemia (mixed cryoglobulinemia) that contain IgA, IgG and IgM and a vestigial monoclonal IgM/Kappa with rheumatoid factor activity;

Electrophoresis of serum proteins showed a monoclonal peak, however, electrophoresis of urinary proteins disclosed monoclonal proteins.

The immunological study revealed: IgA 489 mg/dl (114-457 mg/dl), IgG 1636 mg/dl (793-1590), IgM 158

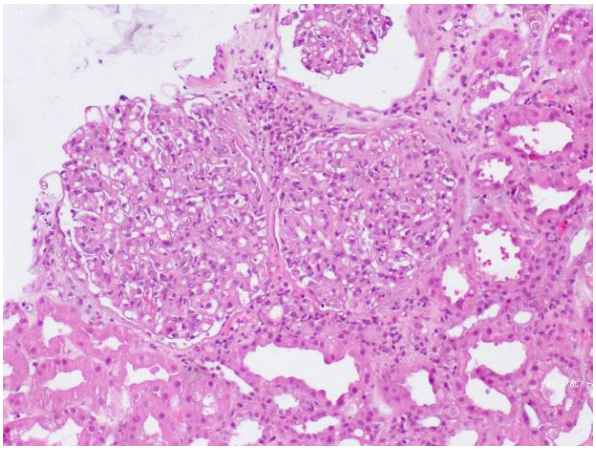

Figure 1

Renal cortex with three bulky glomeruli with mesangial and endocapillary proliferative features. Fibrinoid necrosis and crescents are absent (H&E, x100).

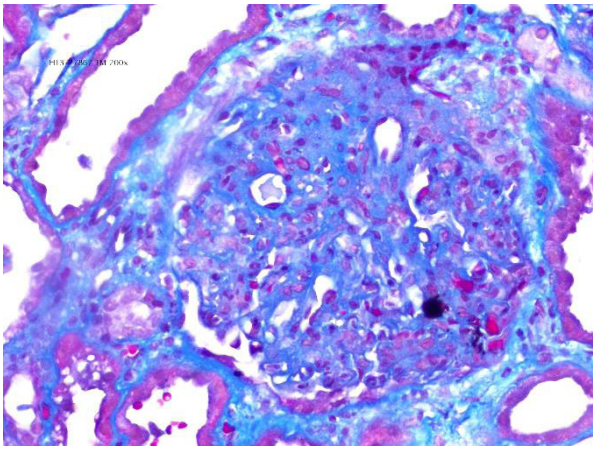

Figure 3

Proliferative aspects with sharp expansion and mesangial proliferation with limited participation of neutrophils (Masson's thricrome, x200).

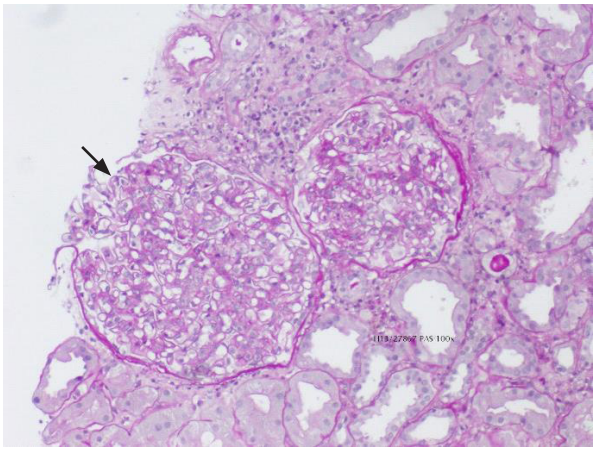

Figure 2

Proliferative aspects and presence of double contours (black arrow) are underlined with PAS staining. Scarce neutrophils can also be identified (PAS, x100).

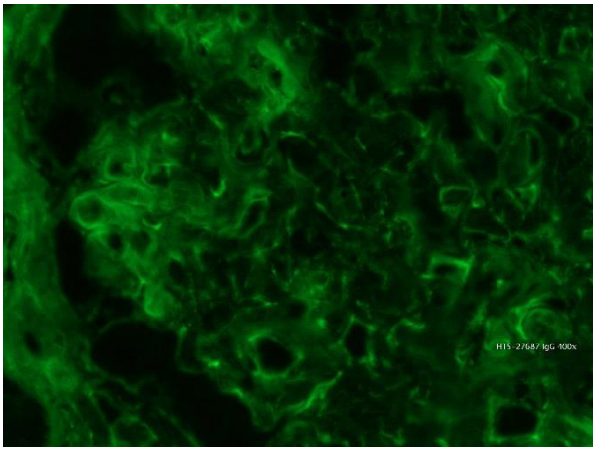

Figure 4

Immunofluorescence (x400) showing pseudolinear IgG deposits in the glomerular basement membrane.

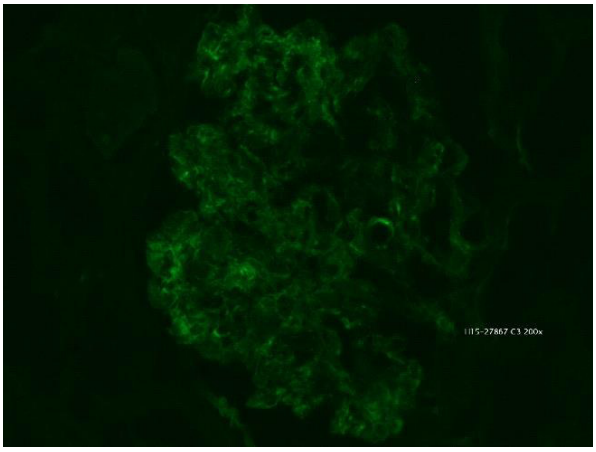

Figure 5

Immunofluorescence (x200) showing peripheral pseudolinear deposits of C3.

mg/dl (29-226), low C3 – 36 mg/dl (81-167) and C4 – 5 mg/dl (11-42 mg/dl); rheumatoid factor 37 UI/ml (< 14); ANA 1/80, speackled, ANCA 1/160 (< 1/20), anti-PR3 112.7; Anti-MPO and anti-MBG were normal.

A kidney biopsy was performed.

Diagnosis: Membranoproliferative glomerulonephritis.

COMMENTS

From an academic point of view, the case poses as differential diagnoses the proliferative glomerulonephritis, namely glomerulonephritis pauci-immune ANCA+, post-infectious glomerulonephritis, membranoproliferative glomerulonephritis (type I, cryoglobulinemic and type II)[1,2].

Apart from the prognostic value of defining the type of proliferative glomerulonephritis, it is important to guide through the morphology which additional tests must be requested and treatment. So, for practical purposes, renal biopsy is of great help to differentiate between infectious and non- infectious causes of kidney damage. In a patient with rapidly progressive renal failure with active urinary sediment and ANCA+, the biopsy is important to distinguish between small vessel vasculitis and proliferative glomerulonephritis associated with infections. This distinction allows to decide the most appropriate therapy for this particular case, since the alternatives are diametrically opposed depending on whether the aetiology is infectious or vasculitic ANCA+ [3-5].

After the kidney biopsy, a trans-oesophageal echocardiography was done and showed a severe aortic stenosis with multiple vegetations adjacent to the valves (the largest measuring 0.6 x 0.5 cm).

He had persistent fever, increased inflammatory parameters, haemoglobin drop and multi-organ dysfunction, notwithstanding the antibiotic therapy aimed at the isolated agent and optimization of organ support measures. In a thoracic, abdominal and pelvic CT there was suggestive evidence of a little area with splenic infarction, probably caused by an infectious systemic embolization.

The patient died with multiple organ dysfunction and refractory shock to all measures instituted.

Conflict of interest statement: None declared.

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