Oral case presentations

<u>OC-1</u> Renal biopsy in children – element of surprise

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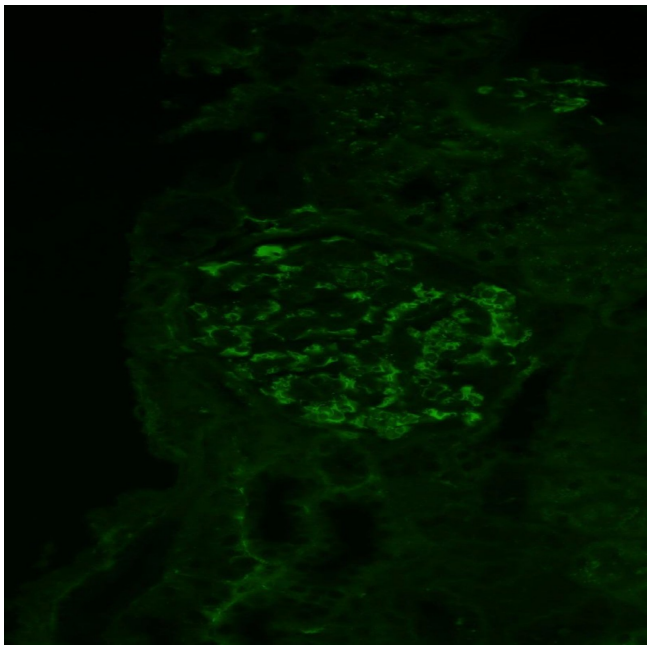
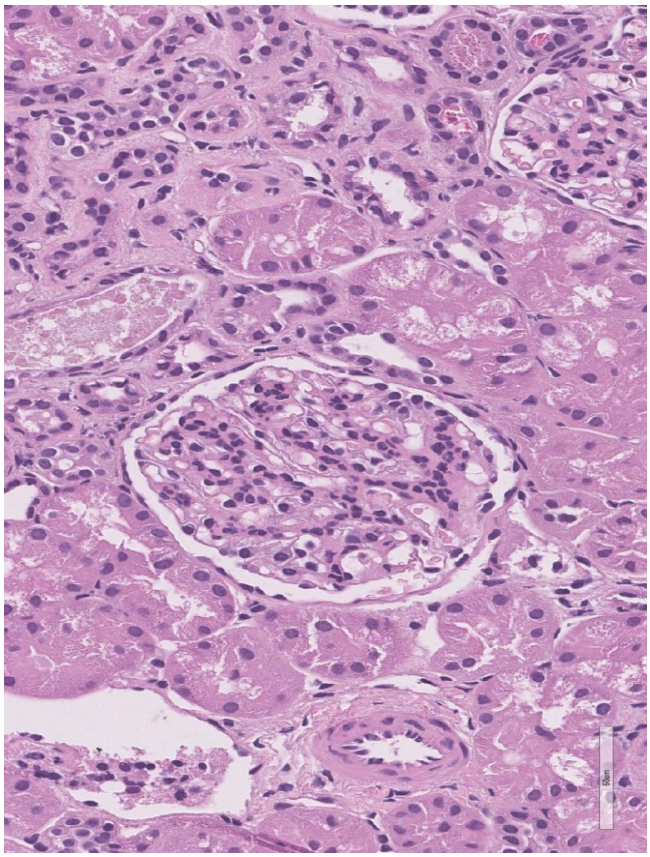
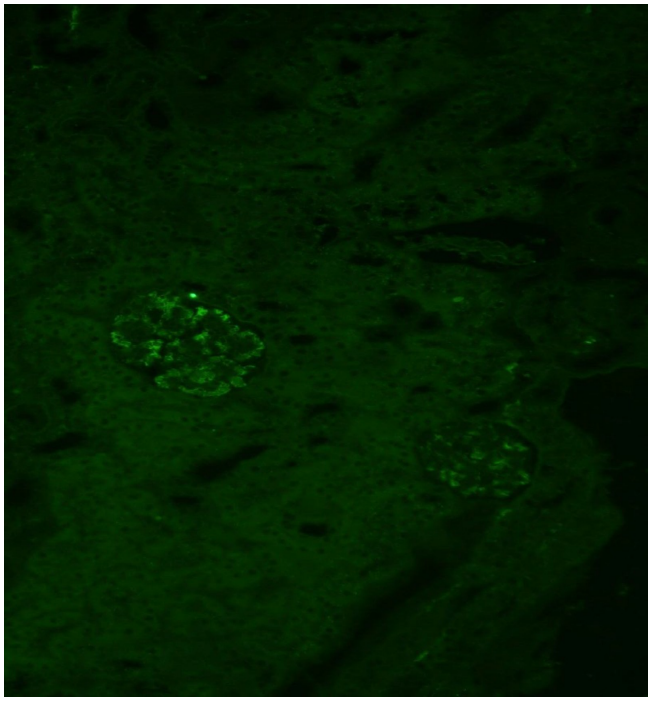
Introduction. Full-house glomerulonephritis (FHN) is a biopsy finding where all types of immunoglobulins (IgA, IgG, IgM) and complement components (C1q, C3) are deposited in the glomeruli, mimicking the pattern seen in lupus nephritis (LN). It can occur without systemic lupus erythematosus (SLE) or positive autoantibodies (non-lupus FHN) and it can present as nephrotic syndrome or a rapidly progressive glomerulonephritis. Treatment is challenging, but may involve immunosuppressants such as steroids, cyclophosphamide, or calcineurin inhibitors.

Methods. The patient aged 3 years and 6 months, known to have Nephrotic Syndrome since February 2025, treated with Prednisone (35 mg/day), was admitted on 15.04.25 to a local hospital with headache, eyelid edema, diplopia and vomiting. Subsequently, the patient developed convergent strabismus and complained of frontal headache, which is why an ophthalmological consultation was recommended, which revealed papillary edema and a cerebral angiography-CT was performed, where a thrombus was suspected in the superior sagittal sinus. During the hospitalization, in the Pediatrics department of the local hospital, the patient received pulse therapy with Methylprednisolone. The patient had been taking daily treatment with Prednisone since the onset of Nephrotic Syndrome and an emergency transfer to the Nephrology department was decided, presenting edema, cushingoid face, hypertension.

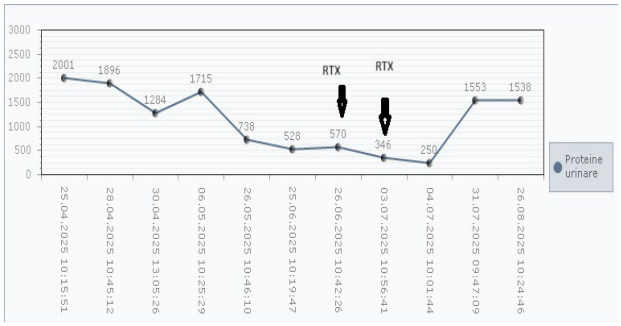
Results. Considering the duration of corticosteroid treatment without response on proteinuria, it is classified as a corticosteroid-resistant nephrotic syndrome and treatment with cyclosporine is initiated and corticosteroid tapering is started according to the IPNA 2023 guideline. The antibodies for AAN where negative and a renal biopsy is performed, which directs the diagnosis to a non-lupus "full-house" nephropathy.







Anticoagulant treatment is also initiated in therapeutic doses, with good evolution, antihypertensive treatment with ACE inhibitors. In the dynamics, the persistence of nephrotic-range proteinuria and microscopic hematuria is noted, which is why treatment with Rituximab is initiated and the tapering of Cyclosporine. The range-nephrotic proteinuria persisted, so it was associated Mycofenolate Mofetil in the therapeutic plan.



Conclusions. Diagnosing nephrotic syndrome is not difficult, but it is also necessary to detect the underlying disease. Kidney biopsy is the gold standard in establishing the ethiology of cortico resistant nephrotic syndrome and adapting an appropriate treatment.

OC-2 An Unusual Scenario of 'Three Sisters': Exploring Alport's Syndrome in Women“ A Case Series

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Introduction. Alport Syndrome is a genetic disorder caused by mutations in type IV collagen genes, essential for the structural integrity of the glomerular basement membrane, inner ear, and eyes. The X-linked form, due to *COL4A5* mutations on chromosome Xq22, accounts for about 85% of cases and is typically more severe in males. Females, often labeled as asymptomatic "carriers," may still experience significant renal impairment. Less commonly, autosomal recessive or dominant forms linked to *COL4A3* or *COL4A4* mutations (on chromosome 2q36) can also cause progressive disease in females. Diagnosis relies on genetic testing and, when needed, kidney biopsy showing glomerulosclerosis and interstitial fibrosis. This presentation aims to emphasize the importance of recognizing Alport Syndrome in females and the role of genetic and clinical evaluation, even when family history or extrarenal signs are absent.

Case report. In this case series, we present three female patients diagnosed with Alport Syndrome, symbolically referred to as the “Three Sisters” due to their shared diagnostic and clinical challenges.

Case 1: A 54-year-old woman with no known renal history presented with elevated creatinine (2.59 mg/dL), hyperuricemia, hematuria, and proteinuria (3.5 g/24h). Imaging showed nephrocalcinosis and poor corticomedullary differentiation. Over two years, creatinine rose to 5.67 mg/dL. A *COL4A4* mutation was identified, confirming Alport Syndrome. She progressed to end-stage kidney disease and is on dialysis.

Case 2: A 53-year-old woman with chronic kidney disease and microscopic hematuria had renal cortical cysts. Initially suspected of medullary sponge kidney, genetic testing revealed a *COL4A3* mutation. Renal function remains stable (creatinine 1.62 mg/dL; proteinuria 198 mg/24h).

Case 3: A 23-year-old pregnant woman (36 weeks) presented with hypertension and heavy proteinuria (13 g/24h), suspected of preeclampsia. After a premature cesarean section, nephritic-range proteinuria persisted. Biopsy and genetic testing confirmed Alport Syndrome. Under treatment (ACE inhibitors, statins, SGLT2 inhibitors), her kidney function is stable (eGFR >75 ml/min/1.73 m²; UACR <200 mg/g).

Conclusion. Alport Syndrome should be suspected in females with hematuria and/or proteinuria, regardless of family history or extrarenal signs. Autosomal forms may affect women significantly. Early diagnosis through genetic testing and biopsy is

essential for guiding management and preserving renal function.

OC-3 C1q nephropathy in a patient with relapsed multiple myeloma after autologous stem cell transplantation

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Introduction: C1q nephropathy is a pattern of glomerulonephritis characterized by predominant mesangial C1q deposition but with other histological features resembling lupus nephritis, although no extrarenal disease. C1q nephropathy is a poorly understood and controversial entity with distinctive immunopathologic features: dominant or co-dominant immunofluorescence staining for C1q, mesangial electron dense deposits. The prevalence of C1qN has been estimated from 0.2% to 16%. Clinical presentations vary from asymptomatic urinary anomalies and macroscopic hematuria to nephritic syndrome and nephrotic syndrome.

Case report. Female patient, 44 years old, presented with peripheral edema, arterial hypertension and diabetes mellitus and nephrotic syndrome with proteinuria range 10 grams in 24 h urine sample with preserved renal function. Five years previously she was diagnosed with multiple myeloma. Initially treated according to the CTD protocol, and then according to the VTD protocol. After that, she was treated with autologous stem cell transplantation. The disease was in remission with lenalidomide in maintenance therapy. During the treatment of multiple myeloma, DM type 2 was diagnosed and insulin therapy was introduced. Clinical analysis of the etiology of nephrotic syndrome did not reveal pathological lymphadenopathy or solid tumors. Tumor markers were normal, as were viral markers. In the findings of serology, a positive liver profile was obtained, which was laboratory compatible with PBC (primary biliary cirrhosis) of the liver. A liver biopsy was performed, but PBC was ruled out pathohistologically. Lupus serology was negative. A kidney biopsy was then performed and a pathohistological finding of C1q nephropathy was obtained. On light microscopy there was focal mesangial cell proliferation in some glomeruli. In the immunofluorescence, granular accumulation of C1q 4+ was found, while kappa, lambda, IgG, IgM, IgA and C3 were negative. There was no any other significant finding in the kidney biopsy. In the finding of electrophoresis of serum proteins by immunofixation, an M peak was obtained in the gamma region. The finding of flow cytofluorometry of the bone marrow aspirate was not indicative of lymphoproliferative hematological diseases. Pathohistological findings of bone biopsy with immunohistochemical tests proved relapse of multiple myeloma.

Outcome. She was treated with corticosteroids and daratumumab. Clinical and laboratory partial remission of nephrotic syndrome was achieved.

Conclusion: The association between nephrotic syndrome and multiple myeloma has been reported in various glomerulopathies, but not with C1q nephropathy in literature.

OC-4 Metastatic neuroendocrine carcinoma of unknown primary site in inguinal lymph nodes complicated with membranous nephropathy: a case report (oral case)

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Introduction. Membranous nephropathy (MN) is one of the most common causes of nephrotic syndrome in adults, with 80% of MN cases being primary and 20% secondary. Primary MN involves the presence of anti-phospholipase A2 receptor antibody (PLA2R) in 70-80% of cases, whereas secondary MN is commonly a result of malignancy, infection or drugs. Neuroendocrine neoplasms (NENs) are uncommon malignant tumors distinguished by fluctuating global incidence rates, exhibiting a general trend of rising frequency. The gastrointestinal tract and lungs are themost common primary sites for NENs, the liver being the most common location for metastases. However, up to 22% of NENs present without an identifiable primary site. Nevertheless, metastasis to the inguinal lymph nodes is rare. Tumor-associated nephropathies can occur in association with both solid tumors and hematologic malignancies. MN is the most prevalent

pathological variant of tumor-associated nephropathies in individuals with solid tumors, but its incidence in patients with neuroendocrine carcinoma is exceedingly uncommon.

Case presentation. We report the case of a 46-year-old man admitted to the hospital with nephrotic syndrome (proteinuria at admission = 10.4 g/24h) and normal renal function (eGFR = 90.8 ml/min/1.73m2). He had no extrarenal symptoms indicative of infection, connective tissue disease, or malignancy. He had been previously healthy and was not receiving treatment with any drugs. Kidney biopsy revealed a MN pattern. Serum anti-PLA2R antibodies were negative. A comprehensive evaluation of secondary causes was conducted. The computed tomography of the abdomen and pelvis showed multiple enlarged left inguinal lymph nodes measuring between 15 mm and 40 mm. A lymph node biopsy was performed and histologic examination showed metastases of a small cell neuroendocrine carcinoma. Despite extensive examination, the primary tumor site could not be detected.

Outcome. The patient was referred to the Oncology Department, where he began a systemic chemotherapy regimen consisting of Carboplatin and Etoposide, administered intravenously, for three consecutive days, every 3 weeks. To date, he has completed four cycles of chemotherapy, resulting in a significant decrease in proteinuria (proteinuria = 4.6 g/24h), with stable renal function.

Conclusion. NEN association with MN is rare, and when present, combined chemotherapy (Carboplatin and Etoposide) represents an effective treatment modality, with positive results on renal function and protein loss.

OC-5 Focal segmental glomerulosclerosis presenting as nephrotic syndrome and acute kidney injury in late pregnancy: a case report (oral case)

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Introduction. Nephrotic syndrome, occurring in 0.012% to 0.025% of pregnancies, is characterized by peripheral edema, severe proteinuria, hypoalbuminemia and often hyperlipidemia. Focal segmental glomerulosclerosis is a common etiology of nephrotic syndrome in the general population.

Case report. We present the case of a 31-year-old female with no significant medical history, who is 29 weeks into an unmonitored pregnancy and was admitted for recent onset of oliguria and anasarca. The patient underwent an uncomplicated urgent cesarean section for imminent preterm labor. Clinical examination revealed normotension, pale skin, alopecia, massive bilateral leg edema and diminished breath sounds in the lung bases. Laboratory findings indicated moderate anemia, azotemia, hyperkalemia, severe nephrotic-range proteinuria, hypoalbuminemia, hyperlipidemia. Imaging studies showed moderate pleural effusion and ascites. We considered the following differential diagnoses for the etiology of nephrotic syndrome: preeclampsia, systemic lupus erythematosus, focal segmental glomerulosclerosis, minimal change disease, membranous nephropathy, ANCA-associated vasculitis and Goodpasture syndrome. Specific diagnostic tests showed negative results for autoimmune antibodies (ANA, anti-dsDNA, p/cANCA, anti-GBM, anti-PLA2R), normal C3, C4 levels, and no viral infections. Renal biopsy sample (Figure. 1) contained a single glomerulus, which showed normal cellularity, a glomerular basement membrane of normal structure and thickness, with no immune deposits and podocytopathy. The suggested histopathological diagnosis was focal segmental glomerulosclerosis.

Outcome. Initial supportive therapy (albumin, electrolytes, loop diuretic) was ineffective. Due to persistent fluid overload and worsening renal function (creatinine 6.25 mg/dL), renal replacement therapy was initiated via six hemodiafiltration sessions. Pulse therapy with methylprednisolone and cyclophosphamide was started for severe nephrotic syndrome, but the response was inadequate after two cycles. We noted infectious complications (urinary tract infection and bacteremia with MDR *Klebsiella)* related to the nephrotic syndrome and immunosuppressive therapy. The patient remained on immunosuppressive corticosteroids until biopsy confirmed focal segmental glomerulosclerosis. Given the steroid-resistant profile, second-line treatment with cyclosporine was initiated and then tapered gradually. Renal function improved progressively, with significant reduction in proteinuria and normalization of creatinine (0.55 mg/dL) after nine months, accompanied by scalp and body hair regrowth.

Conclusion. This case underscores the complexities of diagnosing nephrotic syndrome in pregnancy, particularly in the setting of acute kidney injury, and highlights the importance of a thorough differential diagnosis [3,4]. Notable aspects of this case include the rare occurrence of focal segmental glomerulosclerosis-induced nephrotic syndrome with acute kidney injury during pregnancy necessitating

aggressive treatment, and reversible alopecia universalis following restoration of renal function.

OC-6 Hidden in plain sight: infectious spondylodiscitis as an underrecognized complication of Hemodialysis

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Introduction. Infectious spondylodiscitis is defined as an infection of the intervertebral disc and adjacent vertebral bodies. It is an uncommon, but potentially fatal complication among patients on maintenance hemodialysis, that already carry a heightened risk of infection due to immune dysfunction associated with the uremic milieu, pre-existing comorbidities such as diabetes mellitus and vascular disease, as well as repeated vascular access procedures. Recurrent bacteremia episodes mainly due to cannulation of the vascular access or the presence of a central venous catheter (CVC), represent the primary pathologic mechanism, often leading to metastatic infections. The most frequent culprits are gram-positive bacteria, principally Staphylococcus aureus, although gram-negative organisms and polymicrobial infections have also been reported. Clinical presentation is often insidious and nonspecific, with persistent back pain as the hallmark symptom, contributing often to a delayed recognition and underdiagnosis. If left untreated, the infection can progress to paraspinal or epidural abscess formation, vertebral collapse and permanent neurological impairments.

Case report. Case 1: A 45-year-old man with end-stage kidney disease and hypertension, recently started on hemodialysis via a temporary femoral central venous catheter (CVC), presented with one week history of severe lumbar pain refractory to analgesics and limited mobility. Initially, acute abdominal emergencies were ruled out. Neurological examination revealed a bilateral, positive Lasegue sign, but no sensory deficits were noted. MRI revealed spondylodiscitis at the L3–L4 level with paravertebral involvement. Neurosurgical consultation recommended conservative treatment. Empiric antibiotic therapy was initiated, the CVC was removed and hemodialysis continued via a functioning AVF. Cardiac ultrasound ruled out concurrent infectious endocarditis. Blood cultures were positive for *Enterococcus faecalis*, and subsequently antibiotics were tailored to sensitivity testing. The patient improved clinically and made a full recovery without neurological sequelae.

Case 2: A 61-year-old man on maintenance hemodialysis for 12 years, with a history of kidney transplant failure and hypertension, presented with a 7-day history of severe back pain and fever following his dialysis sessions. He had longstanding vascular access problems and was dialyzing via a tunneled left internal jugular CVC for the past 3 years. Blood cultures were obtained, empiric antibiotics started, and echocardiography ruled out endocarditis. MRI revealed spondylodiscitis of T9–T10 with a pathological fracture of T9. Cultures grew *Staphylococcus aureus*. Due to a history of multiple vascular access failures and no alternative access options, immediate removal of the CVC was considered unfeasible, and an attempt at access salvage was made. Therapy was adjusted accordingly, alongside the administration of an antibiotic lock. Neurosurgical evaluation recommended conservative treatment. The patient's fever resolved, pain improved, and mobility partially recovered. He continues on antibiotics and is scheduled for a delayed catheter exchange.

Case 3: A 60-year-old woman on chronic hemodialysis for 5 years, with diabetes mellitus, hypertension, multiple prior vascular access failures and recurrent catheter-related bloodstream infections (CRBIs), was using a tunneled right subclavian CVC for 2 years. She presented with a new CRBI and was initiated on empiric antibiotic therapy, awaiting culture and sensitivity results. Subsequently, she developed severe lumbar pain radiating to the upper abdomen, refractory to analgesics. After ruling out acute abdominal emergencies, an MRI confirmed spondylodiscitis and echocardiography revealed tricuspid valve endocarditis. Her CVC was removed and broad-spectrum empiric antibiotic therapy continued, however her condition rapidly deteriorated and she succumbed to septic shock.

Conclusion: Infectious spondylodiscitis is a rare, but serious complication in patients on chronic hemodialysis. Recurrent bacteremia due to their vascular access remains the main risk factor in this population. Early recognition requires a high index of suspicion, and spinal imaging should be promptly pursued in dialysis patients presenting with persistent back pain, even in the absence of fever or other overt signs of infection, to prevent irreversible neurological sequelae.

OC-7 Managing dabigatran overload with intermittent hemodialysis and idarucizumab: case report (oral case)

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Introduction. Dabigatran is a predominantly renally cleared direct thrombin inhibitor with a highly renal-dependent pharmacokinetic profile. Standardised tests for monitoring its activity are lacking, and the sole reversal agent is idarucizumab, but financial

constraints and limited effectiveness in acute kidney injury (AKI) hamper its clinical use. Removal by hemodialysis (HD) may sometimes be the sole reversal strategy. This report presents a case of dabigatran overload associated with AKI.

Case presentation. A 71-year-old man was admitted to the Surgical Intensive Care Unit for urgent surgery due to acute cholangitis and cholecystitis. His physical findings included altered mental status, icterus, hypotension, tachyarrhythmia, dyspnoea, low oxygen saturation, dehydration, prolonged bleeding from puncture sites and oliguria. Clinical history included atrial fibrillation (CHA2DS2-VASc=4), chronic heart failure, hypertension, abdominal aortic aneurysm, hypothyroidism, hyperlipidemia, prostatic hyperplasia, untreated chronic renal disease and previous STEMI with triple aorto-coronary bypass. Chronic treatment included dabigatran 150mg bid, trimetazidine, an ACE inhibitor, beta-blocker, torasemide, statin, levothyroxine, tamsulosin, solifenacin, and diosmin/hesperidin. Ultrasound revealed gallstones in the common bile duct and the gallbladder, aerobillia, and signs of incipient pancreatitis, while the kidneys appeared normal. Bloodwork on admission confirmed stage 3 AKI with mild acidosis, anaemia and thrombocytopenia, no electrolyte imbalance, severe systemic inflammation, signs of hepatic and pancreatic injury and anticoagulation. He was immediately treated with adequate conservative therapy. Cardiologic exam excluded worsening cardiac failure. Dabigatran was promptly discontinued and low-molecular-weight heparin (LMWH) introduced, along with 10mg of vitamin K, tranexamic acid, plasma and human prothrombin complex, but with limited effect on anticoagulation parameters. On day 4, idarucizumab was administered, but the ecarin clotting time (ECT) still suggested a rebound in dabigatran activity. Despite improved hemodynamic status and recovered diuresis, azotemia aggravated, necessitating urgent dialysis. A temporary dialysis catheter was placed in the femoral vein immediately after idarucizumab infusion. On days 5 and 6, two consecutive intermittent predilution hemodiafiltration treatments were performed. ECT between and after dialyses indicated a gradual decline in dabigatran activity. Surgical treatment was postponed. He remained on LMWH at discharge. Dabigatran was reintroduced by the cardiologist two weeks post-discharge. Three months later, the patient underwent a laparoscopic cholecystectomy without complications.

Outcome. HD contributed to dabigatran activity mitigation, even with short, intermittent sessions, as mandated by the anticipated urgent surgery.

Conclusion. HD remains an important option for managing dabigatran overload, even with the availability of idarucizumab. Clearer protocols are needed for cost-effective and safe treatment of dabigatran-associated coagulopathy.

OC-8 ANCA vasculitis and IgA nephropathy in patient with Grave's disease (oral case)

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Introduction. Antineutrophil cytoplasmic autoantibodies (ANCA) are commonly associated with a necrotizing and crescentic glomerulonephritis (GN) that is pauci-immune, with few or no glomerular immune complex deposits. Immunoglobulin A (IgA) nephropathy may also be manifest as a crescentic GN, but it is characterized by mesangial immune complex deposits containing IgA and is rarely associated with myeloperoxidase (MPO)- or proteinase 3 (PR3)-specific ANCA. Graves' disease (GD) is a common autoimmune cause of hyperthyroidism, which is eventually related to the generation of IgG antibodies stimulating the thyrotropin receptor. Clinical manifestations of the disease reflect hyperstimulation of the gland, causing thyrocyte hyperplasia (goiter) and excessive thyroid hormone synthesis (hyperthyroidism). There is no clear association between Grave's disease and immunoglobulin A nephropathy.

Case report. Male patient, 38 years old, who was diagnosed with Grave's disease 4 years ago with hyperthyroidism and the development of atrial fibrillation. At first, he was treated with thiamazol, which was discontinued due to myelotoxicity, and then with propylthiouracil therapy, regulation of the thyroid hormone status was achieved. In the follow-up, erythrocyturia and hematuria were verified in the patient's urine. In the 24-hour urine tests, proteinuria of the range of 2.0 gr/24h was verified, predominantly albuminuria. An investigation into the etiology of proteinuria was conducted. During hospital monitoring, serum creatinine values increased from 100 to 263 μmol within a week. In the immunological findings, a high titer of MPO antibodies was obtained. Other secondary causes of proteinuria have been excluded. Clinically, extrarenal manifestations of MPO vasculitis were not verified by examination. A kidney biopsy was performed. Pathohistological findings revealed IgA nephropathy, Oxford classification M1E0S1T0C0 with global and segmental sclerosis in glomeruli with mild hyalinosis of arterioles. The immunofluorescence test verified IgA 4+ granular mesangial accumulation.

Outcome. The patient was treated with 6 cycles of pulse therapy with methylprednisolone and cyclophosphamide, with continued therapy with oral corticosteroids, ACE inhibitors, SGLT2 inhibitors, hypolipemic drugs and propylthiouracil. The indicated therapy resulted in a regression of the proteinuria level to 0.3 gr/24h with an improvement in renal function (serum creatinine 190 μmol, GFR 40 ml/min/1.73m2) without other complication. In the control findings of immunology, there was a drop in the MPO antibody

titer. During follow-up, the patient did not develop extrarenal manifestations of MPO vasculitis.
Conclusion: Patients with Graves' disease may develop ANCA-associated vasculitis with the pathohistological presentation of IgA nephropathy. Propylthiouracil use has been associated with ANCA positive pauci-immune glomerulonephritis, but not with IgA nephropathy. Searching the literature, we did not come across a similar described case.

OC-9 Cardio-renal syndrome: what's more important, the heart or the kidneys? (oral case)

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Introduction. Cardio-renal syndrome is a clinical entity that entails a simultaneous disorder of the cardiovascular and renal system, with one system affecting the other. Due to complex underlying mechanisms, high mortality rates and a multi-faceted treatment, it is important to recognize and treat accordingly.
Case report. We present a 75-year-old female patient, smoker, obese, with 2nd-degree HT, dyslipidemia, type 2 diabetes, HFpEF, LBBB, CKD KDIGO G2A1, COPD GOLD III, presenting with fatigue, discrete ankle swelling and chronic diarrhea. The ECG shows Afib with RVR. Lab reports revealed occult hemorrhage, AKI AKIN III, nephrotic range proteinuria, negative urine culture, positive pANCA, and moderate anemia. During the albumin administration patient developed severe cardiogenic APE and cardiac arrest (asystole). Post-resuscitation, she is admitted to ICU (intubated). On cardiac echo EF was 30%, a globally hypokinetic heart, with severe MR and TR. The renal function was on decline. Lung Rx revealed an OTI-associated bronchopneumonia (but without sepsis). The patient frequently develops severe HT crises, entered Afib with RVR, and then cardiogenic APE.
Outcome. Given her worsening kidney function, the medical team decided to initiate emergency hemodialysis. After that, her general state shows slow, gradual improvement in all systems. Her state was stationary for 8 months, until chronic dialysis could no longer be postponed. At present, she is hemodynamically stable, with EF=50%, mild MR, and TR.
Conclusion. A type III cardio-renal syndrome (acute kidney failure gives acute heart failure) with associated lung damage reportedly has a very high mortality rate. A multidisciplinary approach is necessary, as a low eGFR, immune compromise and mechanisms such as diuretic resistance can complicate an already elaborated issue. Emergency dialysis can represent a last resort for these patients, but a close and careful follow-up is mandatory to ensure optimal long-term results.