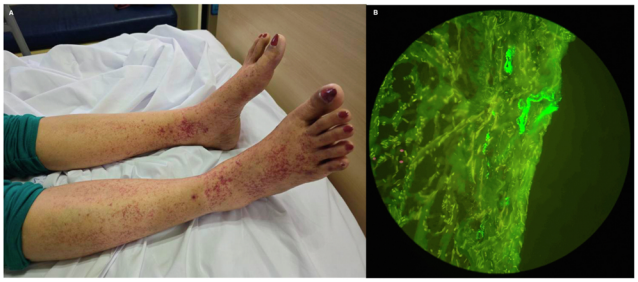
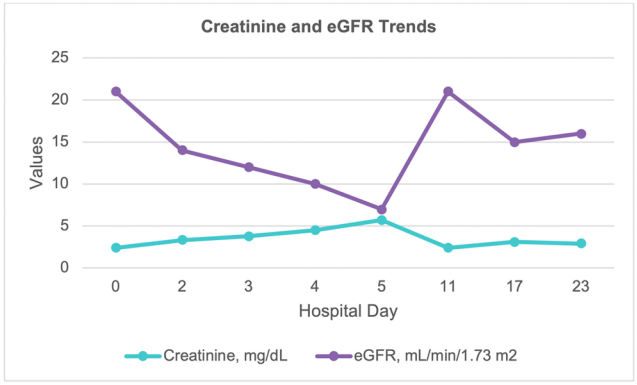
Introduction: Immunoglobulin A nephropathy (IgAN) is a common cause of glomerulonephritis that can progress to end-stage renal disease (ESRD). IgA vasculitis, a systemic small vessel vasculitis, is a recognized secondary cause of IgAN. Although rare, rapidly progressive glomerulonephritis (RPGN) can occur in the context of IgA vasculitis, with drug-induced immune responses implicated in some cases.
Methods: A 74-year-old Filipino female with no known comorbidities initially presented with flu-like symptoms two weeks prior to admission. She later developed hypogastric pain and dysuria, for which she was treated with cefuroxime 500 mg tablet twice daily for two days for urinary tract infection (UTI). Despite treatment, her symptoms worsened, progressing to generalized abdominal pain and vomiting, prompting admission. She was treated with ceftriaxone 2 g intravenously daily and was referred to nephrology for elevated creatinine (2.4 mg/dL, eGFR 21 mL/min/1.73 m2). Two days later, she developed non-blanching, non-palpable, non-pruritic, painless petechial and macular lesions on her lower extremities, accompanied with arthralgia and altered sensorium. Laboratory results revealed progressive renal dysfunction (creatinine 5.15 mg/dL, eGFR 8 mL/min/1.73 m2), proteinuria, hematuria, and oliguria, suggestive of RPGN. Renal biopsy was indicated but was deferred due to financial constraints. Ceftriaxone was discontinued, renal replacement therapy was initiated, and pulse methylprednisolone therapy followed by oral prednisone was given. A skin punch biopsy with direct immunofluorescence was done instead which confirmed IgA vasculitis.





Results: This case illustrates an exceptionally rare presentation of RPGN secondary to IgA vasculitis in an elderly patient, with atypical non-palpable skin lesions rather than the characteristic palpable purpura. The temporal association with cephalosporin use raises the possibility of a drug-induced immune response, complicating the diagnostic process. Despite timely intervention, the patient remains hemodialysis-dependent to date.
Conclusion: Although rare, IgA vasculitis can progress to severe life-altering RPGN. In this case where renal biopsy was not feasible, a skin biopsy provided critical diagnostic insight. While not a substitute for renal biopsy, skin biopsy may be a valuable tool in selected vasculitis cases especially in resource-limited settings. Early intervention is critical to prevent irreversible renal damage and to improve long-term outcomes.

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WCN26-338

CLINICAL FEATURES AND TREATMENT OF RENAL ABSCESS IN CHILDREN

(Article No. 106174)

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Introduction: To investigate the clinical characteristics and treatment strategies of renal abscesses in children.
Methods: A total of 5 cases of pediatric renal abscess admitted to the Department of Pediatrics, General Hospital of Ningxia Medical University from January 2022 to December 2024 were included into study. A retrospective analysis was conducted on general information, clinical manifestations, laboratory tests, imaging findings, treatment plans, and outpatient follow-up results of these 5 children.
Results: ①Among the 5 children, 3 were male and 2 were female, with ages ranging from 3 to 13 years. The clinical manifestations of the 5 children were primarily characterized by recurrent fever, with some children presenting with abdominal pain, vomiting, and urinary tract irritation signs. One child (Child 2) exhibited tenderness on renal percussion, while no other positive physical signs were observed. ②Before treatment, all 5 children had elevated peripheral blood white blood cell count (WBC), C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR). Two children (Child 2, 5) had increased urinary leukocytes, and one child (Child 5) had a positive urine culture, with the pathogen identified as *Escherichia coli*. ③After admission, all 5 children underwent color Doppler ultrasound of the urinary system, enhanced CT of the kidneys, or MRI. Among them, left renal abscess was found in 3 children (Child 2, 3, 5), and right renal abscess in 2 children (Child 1, 4). Upper pole abscess was present in 3 children (Child 1, 3, 5), and lower pole abscess in 2 children (Child 2, 4). Three children had renal abscess diameters less than 3 mm (Child 1, 3, 4), while 2 children had diameters greater than 3 mm (Child 2, 5). Three children exhibited hydronephrosis (Child 2, 3, 4). Color Doppler ultrasound of the urinary system showed hypoechoic masses within the renal parenchyma with unclear borders, with diameters ranging from 1.7 to 5.0 cm. MRI of the kidneys revealed patchy abnormal signals within the renal parenchyma, with high signal intensity on diffusion-weighted imaging (DWI) and unclear borders; enhanced scanning showed heterogeneous enhancement of the lesions. ④All children received conservative treatment. Initial treatment involved intravenous infusion of broad-spectrum antibiotics (piperacillin-tazobactam, meropenem, or ertapenem) for anti-infection therapy. However, the therapeutic effect was unsatisfactory, leading to an upgrade in antibiotic treatment. ⑤After discharge, all children continued oral antibiotic therapy for more than 14 days. Follow-up within 2 weeks after discharge showed complete resolution of renal abscesses without renal scar formation. No recurrence was observed during the outpatient follow-up period after discontinuation of medication.
Conclusion: The use of broad-spectrum antibiotics for the treatment of pediatric renal abscesses has proven to be effective and can be recommended as the first-line therapeutic approach.

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WCN26-436

DE NOVO IDIOPATHIC NODULAR GLOMERULOSCLEROSIS IN A KIDNEY TRANSPLANT PATIENT

(Article No. 106175)

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Introduction: Chronic Kidney Disease is a condition characterized by a progressive and irreversible decrease in kidney function that can be caused by different etiologies, among which are primary or secondary glomerular diseases (1). Within renal replacement therapies; Transplantation is the modality that provides the greatest benefit in terms of quality of life and mortality. Renal graft survival can be affected not only by rejection and infections but also by relapse in the graft of a

glomerular disease that affected the native kidney or otherwise, a de novo onset (2)(3). A study of 1505 transplant patients between 1988-1997 with a diagnosis of confirmed glomerular disease documented that recurrence was the third most common cause of allograft loss at 10 years, after chronic rejection and death with a functioning allograft (4). It should be noted that live transplantation has better HLA compatibility and therefore greater graft survival, however increased rates of primary glomerulonephritis recurrence have been noted in this group of patients (5).

Nodular glomerulonephritis is a histological lesion pattern characterized by the presence of hyaline deposits in the mesangial matrix with a nodule-like distribution; It also shows glomerular vascular involvement, and its usual clinical manifestation may be complete nephrotic syndrome or significant proteinuria. This type of injury is closely related mainly in patients with Diabetes Mellitus and smokers with a high rate of smoking activity. The de novo appearance of this type of lesion in the post-transplant period would constitute a primary form, whose presentation would be infrequent, corresponding to only 0.45% in the series published to date (6).

Methods: Clinical case: A 24-year-old male patient from Soledad (Atlántico, Col) presented renal disease at the age of 16 with subnephrotic proteinuria with clear renal failure, in addition to being congenital monorenal without prior knowledge and since then with renal replacement therapy, so the etiology of CKD was unknown. He denied a history of smoking, or metabolic pathologies, with a normal weight and BMI. Subsequently, there was the appearance of hypertension, for which pharmacological management was initiated with beta-blocker, central alpha agonist, ARB II. He entered the transplant protocol in September 2018 and was transplanted in April 2024, his donor was cadaverous, with cold ischemia time of 17 hours and hot ischemia of 50 minutes. Induction treatment was performed with rabbit antithymocyte immunoglobulin plus methylprednisolone pulses and subsequently initiation of maintenance with mycophenolate, prednisolone and tacrolimus. Early on, he began to present elevation of azotes without an apparent cause, for which a first renal biopsy was performed with an official report given by BANFF category 3: suspected (borderline) of cell rejection, in the same way a change of therapeutic scheme was carried out suspending mycophenolate and initiation of azathioprine, other infectious etiologies causing graft dysfunction were ruled out. Since then, his renal function was established at G3aA1T, but progressively he began to present a significant increase in proteinuria in the subnephrotic range to a maximum value of 2655 mg/24h, nephrotic syndrome, infectious and autoimmune pathologies and dysproteinemias were ruled out. For this reason, a second biopsy was performed in September 2024, finding findings of other changes not considered by acute or chronic rejection, with findings of interstitial fibrosis and tubular atrophy grade II considered as BANFF category: 5 and 6, but within the histopathological findings there is expansion of the mesangial matrix, associated with mesangial hypercellularity with positivity for IgG and C3, so it is required to complement with electron microscopy study for Discard organized deposits, see image 1. Electron microscopy highlights the presence of nodular areas of electrondense material without characteristics of immune complexes. There is no organized material that suggests fibrils, thyroglobulin or amyloid, there is podocyte effacement of 70% of the capillary surface, consistent with nodular pattern glomerulosclerosis and secondary podocytopathy, image 1. To optimize antiproteinuric therapeutic management, ARA II was changed from losartan to irbesartan and iSGLT2 was initiated. The results obtained were important, in the control the reduction of almost 50% of proteinuria and stabilization of azoates stands out. See Table 1

	12/09/24	11/10/24	12/11/24	13/12/24	13/01/25	12/02/25	13/03/25
Hb	10,7	10,9	11,6	12,3	12,7	12,9	12,5
Leucos	4,1	4,2	5,5	7,4	7,6	6,3	6,9
Plaquetas	192	200	224	219	222	236	289
Glucosa mg/dl	100	102	109	117	97	99	125
Proteinuria 24h (mg)	1532				1848	2655	1320
Creatinina	2,1	1,8	1,5	1,8	2,1	1,8	2
TFG CKD-EPI	49		66	50	49	50	50
HbA1c		4,9					

Table 1. Main follow-up paraclinics.

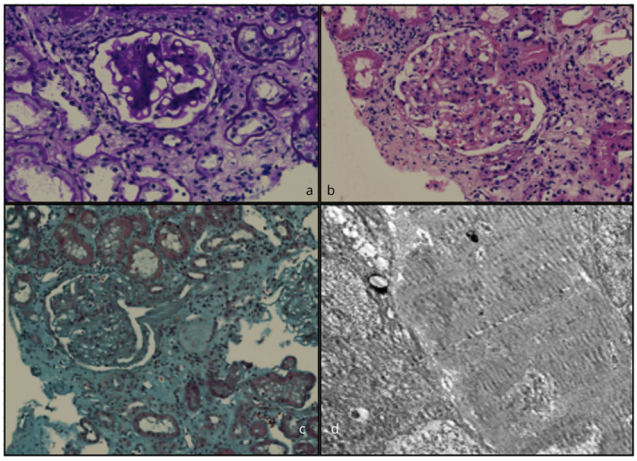

Image 1: Microphotographs. a) PAS staining, b) hematoxylin-eosin staining, c) trichrome staining, d) electron microscopy. The observed mesangial expansion of a-c stands out. In d, the absence of organized electron deposits stands out.

Results: Discussion: Nodular glomerulosclerosis is a pattern of histological lesion that was classically associated with diabetes and was first described in 1936 by Kimmelstiel and Wilson (7). Nodular glomerulosclerosis is also associated with various entities, so it is important to make an adequate clinical-pathological correlation (8). Among other factors associated with this pathology are obesity and smoking, described in 1999 and 2002 (9)(10). In the same way, other entities such as membranoproliferative glomerulonephritis, glomerulopathies associated with dysproteinemia, fibronectin-associated glomerulonephritis, type III collagen glomerulopathy, hypoxic or ischemic pathologies and cystic fibrosis should be ruled out, although it is highlighted that the reports are extremely infrequent, corresponding to 0.45-0.5% of biopsies and scarcely reported in meta-analyses around 95 cases (11). Due to the impact on its correlation with various cardiovascular risk factors, the nomenclature of non-diabetic metabolic nodular glomerulosclerosis has been proposed. This condition is more frequent in men than women, with an average age of 60 years, with progressive renal dysfunction and findings in urinalysis given by proteinuria and microhematuria. It may also present with nephrotic syndrome or subnephrotic proteinuria associated with microhematuria (12). As for the largest reports in the medical literature on idiopathic nodular glomerulosclerosis (IDG) in native kidneys, the one by Yang et al. stands out, which presents 122 cases in a period recorded from 1999 to August 2023. In this registry, most were men, the average age was 62 years, most were smokers, obese and with high blood pressure. 60% had nephrotic syndrome (13). As for the appearance of post-transplant glomerular disease, it can be divided into recurrent or de novo, with recurrences of primary glomerulopathies being more frequent in renal transplantation than de novo appearance. The clinical expression is similar to that presented in native kidneys and the appearance of these has a negative impact on graft survival (14). Among the most common recurrent glomerulopathies in kidney transplantation are focal segmental glomerulosclerosis, IgA nephropathy, membranous nephropathy, and membranoproliferative glomerulosclerosis (2). In the few cases reported worldwide, there is the reported Mohamed Nasreen in a 35-year-old kidney transplant patient in whom he had the appearance of GNI 3 years after transplantation with loss of the kidney graft, who underwent a second kidney transplant with a new recurrence of GNI from the second transplant with subsequent progression to renal failure. evidencing the relevance and severity of this pathology (15).

Similarly, it is important to note that the case presented is atypical in terms of demographic profile, which occurs in a young adult and appears after kidney transplantation, which does not have any risk factor associated with the appearance of this pathology. Regarding therapeutic management, it is evident that there is a favorable clinical response with the establishment of ARB II at the maximum tolerated dose, as well as the inclusion of the SGLT2 inhibitor, proving its benefits again. It is known that immunosuppressive drugs such as calcineurin inhibitors and steroids can contribute to the development of post-transplant diabetes, with pathophysiological pathways shared with diabetic nephropathy that lead to glomerular ischemia and overexpression of TGF-B, which may contribute to the appearance of GNI (16).

Conclusion: GNI is a pattern of glomerular lesion that is predominantly seen in men and manifests clinically with nephrotic syndrome or

significant proteinuria. However, its diagnosis represents a challenge, as it requires the careful exclusion of other underlying pathologies, especially diabetic nephropathy and depositional diseases, such as amyloidosis or plasma cell dyscrasias. The precise identification of this entity is essential, since its prognosis may be conditioned by non-traditional risk factors, such as high blood pressure, smoking, and obesity. In the context of kidney transplantation, is an extremely rare entity, but of great clinical relevance due to its potential negative impact on graft survival. The recurrence or de novo appearance of this pathology in the renal graft may be associated with a progressive deterioration of renal function and the eventual loss of the transplanted organ. Therefore, it is essential to maintain a high index of suspicion in the presence of proteinuria or nephrotic syndrome in transplant patients, even in the absence of classic risk factors. The diagnostic approach should be comprehensive, including detailed histopathological studies and exhaustive clinical and biochemical evaluations to rule out secondary etiologies. should be considered within the differential diagnosis of nodular glomerular lesions, especially in non-diabetic patients and in the context of kidney transplantation. Timely detection and appropriate management can contribute to improving the prognosis and quality of life of affected patients. More multicenter studies and case reports are needed to better understand the course, prognosis, and best therapeutic strategies for this rare entity.

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WCN26-1186

UNBOXING A RARE LINK BETWEEN THE KIDNEYS AND THE LUNGS: REVERSAL OF INFECTION RELATED GLOMERULONEPHRITIS IN A PEDIATRIC PULMONARY TUBERCULOSIS PATIENT WITH CORTICOSTEROIDS

(Article No. 106176)

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Introduction: Glomerular presentations are rare in tubercular infections with the most common being IgA nephropathy, membranous nephropathy, amyloidosis, and membranoproliferative disease. Infection related glomerulonephritis is a rare presentation in patients diagnosed with tuberculosis. We hereby report an extremely rare case of pediatric pulmonary tuberculosis who presented with dialysis requiring rapidly progressive renal failure and was subsequently reversed using corticosteroids.
Methods: A 14 year old male presented with a history of fever, cough and weight loss for 4 weeks. Two weeks later he developed progressively decreased urine output and generalized body swelling. On evaluation he was found to have

- High blood pressure -170/100
- Advanced azotemia (serum urea/creatinine 256/7.1 mg/dl)
- Nephrotic syndrome -spot urine protein/creatinine ratio – 4.5 gm/gm , serum albumin- 2.1 gm/dl and deranged lipid profile.
- Active sediments with red blood cells and RBC casts in urine routine analysis.
- Low C3 levels and normal C4 value.
- Anti Nuclear Antibody (ANA), Extractable Nuclear Antigen (ENA) Anti Myeloperoxidase (MPO), Anti Proteinase- 3 (PR-3) and Anti Glomerular Basement Membrane (GBM) were negative.
- High Resolution CT of chest was done in view of chronic fever and cough which showed cavitating lesions in bilateral upper zone and sputum for Acid Fast Bacilli was strongly positive. Ultrasound of whole abdomen was normal.

In view of increased azotemia he received three sessions of hemodialysis and then underwent an uncomplicated renal biopsy. He was then started on pulse iv steroid (methylprednisolone 500 mg iv once daily for 3 days) followed by oral wysolone 1 mg/kg/day. He was also started on weight based renal modified anti-tubercular therapy – isoniazid, rifampicin, pyrazinamide and ethambutol. Renal biopsy showed diffuse proliferative glomerulonephritis without any crescents and a full house picture on immunofluorescence. He received 2 more hemodialysis sessions following which his urine output started to increase and no further sessions of hemodialysis were required. He was discharged with creatinine of 1.5 mg/dl. On follow up his creatinine became normal and steroid was tapered over the next 6 weeks. His antitubercular drugs were continued throughout the entire course.
Results: We had a case of Infectious Related Glomerulonephritis (IRGN) secondary to primary pulmonary TB. We conclude this because of the timing of the clinical features, the histological features on renal biopsy and the exclusion of alternative aetiologies. Diffuse proliferative glomeurulonephritis with full house pattern on immunofluorescence with ANA negative and low C3 and normal C4 is diagnostic of Infectious Related Glomerulonephritis. IgA nephropathy in association with TB infection is well documented both with and without evidence of intrarenal TB infection. On the other hand, nephrotic nephritic syndrome with non-IgA nephropathy secondary to active extra-renal TB is a very rare occurrence. Only 5 cases of rapidly progressive glomerulonephritis have been reported in adolescent tubercular patients.

This is the first instance of Infection Related Glomerulonephritis occurring secondary to pulmonary tuberculosis in a pediatric population. Strong suspicion should be there specially in patients who present with nephritic nephrotic syndrome. Even more rare is the presentation of dialysis requiring IRGN and these phenotype of patients' respond dramatically with steroids and treatment of the underlying cause, which in this case was anti tubercular therapy.
Conclusion: In conclusion, we present a rare case of immune mediated glomerulonephritis complicating pulmonary tuberculosis in an adolescent. Glomerulonephritis should be strongly considered in patients diagnosed with active tuberculosis who develop renal complications that appear unrelated to the primary focus of tuberculous infection since early initiation of immunosuppression in these patients can complete alter the renal course in these patients.

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WCN26-1479

RAPIDLY PROGRESSIVE IGM-TYPE PGNMID DESPITE SEROLOGIC REMISSION OF LYMPHOPLASMACYTIC LYMPHOMA

(Article No. 106177)

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Introduction: Proliferative glomerulonephritis with monoclonal immunoglobulin deposits (PGNMID) is a rare kidney manifestation of monoclonal gammopathy characterized by glomerular deposition of a single immunoglobulin subclass and light chain. The IgM subtype (IgM-PGNMID) is particularly uncommon, accounting for approximately 10% of all PGNMID cases, and is often associated with B-cell lymphoproliferative disorders. While evidence regarding IgM-PGNMID is accumulating, the clinical course following diagnosis, especially disease progression, remains unclear. We report a case of IgM-PGNMID associated with lymphoplasmacytic lymphoma (LPL) that progressed to nephrotic syndrome despite sustained serologic remission of the underlying LPL, necessitating repeat kidney biopsy.
Methods: A 72-year-old man with a 14-year history of LPL had been treated with corticosteroids and rituximab with a poor response. Hematologic remission was achieved after switching to tirabrutinib three years prior to presentation. However, he developed progressive proteinuria (2.0 g/gCr) with a serum creatinine of 0.9 mg/dL, prompting kidney biopsy.
Results: The biopsy revealed mesangial hypercellularity, double contours of the glomerular basement membrane, and expansion of the subendothelial space. Immunofluorescence showed predominant deposition of IgM and λ light chains. Electron microscopy confirmed non-organized, electron-dense deposits in the mesangial and subendothelial areas, leading to a diagnosis of IgM-PGNMID. Despite ongoing hematologic remission, characterized by low serum IgM levels and no detectable M-protein, the patient's condition rapidly progressed to nephrotic syndrome with declining kidney function, which prompted second kidney biopsy. The repeat biopsy confirmed the progression of interstitial fibrosis and sclerosis without evidence of new-onset glomerulonephritis, reaffirming the diagnosis of IgM-PGNMID. As the LPL was considered well-controlled both serologically and by bone marrow examination, the decision was made to continue tirabrutinib and conservative management, including blood pressure and fluid control. However, three months later, the patient passed away due to a cerebellar hemorrhage.
Conclusion: Serum M-protein is detectable in only approximately 30% of IgM-PGNMID cases. This suggests that a qualitative abnormality of