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Echerichia coli Infection—Associated Glomerulonephritis in a Kidney Transplant Patient: A Case Report

Meryem Benbella[1,2], Zaineb Kaouiri[1,2], Imane Saidi[1,2], Naima Ouzedoun[1,2], Tarik Bouattar[1,2]

[1]Department of Nephrology-Dialysis-Renal Transplantation, Ibn Sina University Hospital in Rabat, Rabat, Morocco
[2]Faculty of Medicine and Pharmacy in Rabat, Mohammed V University, Rabat, Morocco
Email: benbellameryem@gmail.com, Zaineb.kaouiri @gmail.com, imanesaidi12@yahoo.com, Ouzedoun.naima@hotmail.fr, tbouattar@hotmail.fr

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Abstract

Post infectious Glomerulonephritis (PIGN) in renal allograft is a rare entity. Only a few Cases have been described in the literature. *The post streptococcal glomerulonephritis* is the classic example in native kidney. A wide variety of organism has been associated with PIGN in renal allograft such as *Staphylococcus*, *Cytomegalovirus* and *Polyomavirus*. We describe one case of Infection associated glomerulonephritis due to *Echericha Coli*, developed 5 years after kidney transplantation, in 47 years old female patient. The Clinical presentation was characterized by a peripheral edema and high blood pressure, and biological tests showed a nephrotic syndrome, an acute kidney injury, a consumption of Complement fractions. The renal biopsy revealed a diffuse endocapillary cell proliferation with preponderant deposits of C3. Total recovery was achieved 4 months after *Methyprednisolone* pulse and *Cyclophasphamid* with antimicrobial treatment.

Keywords

Glomerulonephritis, *Echerichia coli*, Kidney Transplant

1. Introduction

Following Kidney Transplant, recurrent and *de novo glomerulonephritis* are possible causes of renal dysfunction.

Recurrent g*lomerulonephritis* occurs much more frequently than De novo, but the exact prevalence remains unknown, in 1% - 12% [1], due to the lack of allograft biopsies, and also to unknown initial kidney disease.

Any glomerulonephritis can recur. Segmental and focal hyalinosis, membranous

nephropathy and atypical UHS are the most reported ones.

On the other hand, membranous nephropathy and HCV-associated membranoproliferative glomerulonephritis are the most frequent types of de novo glomerulonephritis. Rare cases of minimal change nephropathy and crescentic glomerulonephritis in *Alport's syndrome* have been described.

Postinfectious acute glomerulonephritis (PIGN) is one of the most rarely reported *de novo glomerulonephritis* with only 18 cases previously described [2]. It's due to an immunological process that affects the kidney after or during an ongoing infection (Infection—Associated glomerulonephritis).

2. Case Report

We report the case of a 47-year-old North African woman with end-stage renal disease (ESRD) due to unknown kidney nephropathy. She underwent hemodialysis for 11 years, and received the first kidney transplantation from her sister in 2017.

We used *thymoglobulin* as immunosuppressive induction, associated with *Tacrolimus*, mycophenolate and prednisone. Baseline serum creatinine was 0.9 mg/dl with normal urinalysis.

On March 2023, she is hospitalized for fever and dysuria. On admission, blood pressure was 155/85 mmHg with peripheral edema and the temperature was 38.5˚C with macroscopic hematuria.

Biological tests showed a leukocytosis (WBC 13,500 per cmm), acute kidney injury with serum creatinine at 3.5 mg/dl, a nephrotic syndrome with a serum albumin 25 g/l, urin protein excretion of 4 g/day, and a consumption of C3 and C4. Cryoglobulins were absent.

Urinalysis showed severe hematuria with 1200 red blood cells (RBC), 150 WBC, and urine culture was positive to *Echerichia coli*. Intravenous *Ceftriaxone* was given for 10 days.

The graft biopsy, performed 7 days after fever appeared, revealed in the light microscopic examination, a diffuse endocapillary cell proliferation in all glomeruli (**Figure 1**), an extracapillary proliferation in 3 glomeruli (**Figure 2**), with a preponderant deposit of C3 in direct fluorescence (**Figure 3**).

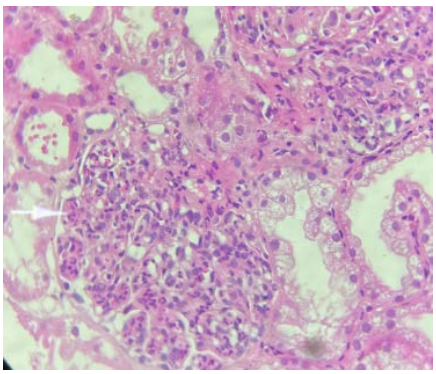

Figure 1. Light microscopy ×40 (Hematoxylin eosin). Diffuse endocapillary cell proliferation and neutrophil exudation.

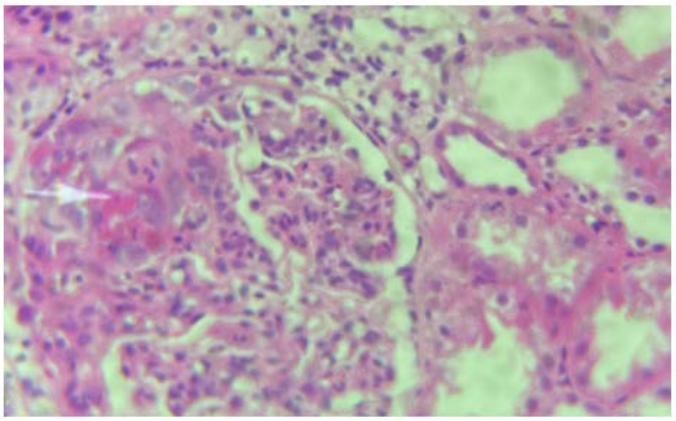

Figure 2. Light microscopy (Hematoxylin eosin ×40): Extra capillary proliferation.

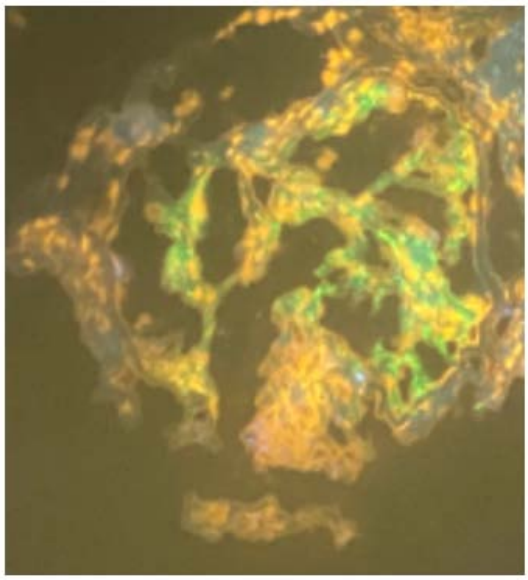

Figure 3. (Direct Fluorescence) intense staining of C3.

The patient was treated with one intravenous pulse of *Methylprednisolone* and *Cyclophosphamid.*

The serum creatinine normalized after 3 months; Hematuria disappeared after 2 months. The complement was normalized after 10 weeks.

Proteinuria slowly decreased and had completely disappeared 4 months later.

The particularity of our clinical case, in addition to the rarity of the entity, is the favorable evolution contrary to the data in the literature.

3. Discussion

The immunosuppressive regimen of the organ transplant recipients increases the rate of infection complications. Both bacterial and viral infections are very common and may have indirect effects on the patient and graft. However, despite the high incidence of post-transplant infections, PIAGN is a rare entity. It's an unusual cause of renal allograft dysfunction.

This could be due the ongoing immunosuppressive therapy that decreases antibody production or simply to the lack of biopsies during such events.

The few cases of PIGN in renal transplant reported in the literature were secondary to a wide variety of organisms; G. Moroni and al. described 3 heterogeneous types due to *Echerichia coli* bacteremia, a skin abscess, and cholangitis [3]. Some cases secondary to *Staphylococcus aureus* [4] [5] [6], *Cytomegalovirus* [2] *and polyomavirus* [7] were also reported.

Regarding glomerulonephritis (GN) and bacterial infections, 2 different entities should be considered: a postinfectious GN with usually a latent period, and infection-associated GN developed in same time of an active ongoing infection. Our Patient developed the GN in the same time of an *Echerichia coli* pyelonephritis; Troy. J *et al.* described one case of PIGN in renal graft, 3 days after dysuria empirically treated with *levofloxacin* [2].

In 2003, a new form of PIGN characterized by IgA dominant immunocomplex deposit has been described [8]. In kidney transplant, two cases of graft dysfunction related to IgA dominant PIGN were reported, the first one is a *staphylococcus* Infection-Associated GN developed 18 years post Kidney transplantation [4], the second one reported 5 years after kidney transplantation, was an *Influenza A* secondary PIGN [3].

PIGN usually presents as a nephritic syndrome with C3 hypocomplementemia; as for our patient who developed a nephrotic syndrome with a C3 but also C4 hypocomplementemia. The renal allograft biopsy found a diffuse proliferative Glomerulonephritis with extensive neutrophil infiltrate into the glomerular capillaries. Direct Immunofluorescence typically shows dominant staining for C3 predominantly mesangial, and less intensely for IgG, IgM and C1q.

Unlike the typical form of *post-streptococcal* GN, there is no established method in treating PIGN in renal transplant recipients. Some authors speculate a potential benefit of short course of intravenous corticosteroid; others use plasma exchange associated to steroid when crescents are present [6]. Despite that treatment, graft loss occurs in up to 60% of cases [9]. Some papers consider that using an immunosuppression can increase the risk of septicemia, especially in forms with active ongoing infection associated GN [10].

In our case, we decided to associate an antimicrobial therapy with Corticosteroid and intravenous *Cyclophosphamid* because of the presence of crescents in the graft biopsy. Complete recovery was achieved 4 months later.

4. Conclusion

PIGN is surely a rare entity in renal graft patients, but any transplant clinician should know of its possible occurrence after or during any ongoing infection.

Renal biopsy needs to be considered during any graft dysfunction because of the amount of information it can provide.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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