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MedPeer Publisher
Abbreviated Key Title: MedPeer
ISSN : 3066-2737
homepage: https://www.medpeerpublishers.com

Clinico-pathological Characteristics and Outcomes of Extracapillary Glomerulonephritis: A Retrospective Analysis of a 9-Case Series in a Moroccan Military Hospital

DOI: 10.70780/medpeer.000QGNF

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ABSTRACT

Introduction: Extracapillary glomerulonephritis (ECGN), or rapidly progressive glomerulonephritis (RPGN), is a major nephrological emergency associated with severe renal and vital prognosis, defined by rapid deterioration of renal function and the presence of glomerular crescents. Its classification is based on immunofluorescence (IF) (Type I anti-GBM, Type II immune complex, Type III pauci-immune/ANCA+). Prognosis depends on early diagnosis and immunosuppressive treatment. This study describes the characteristics and outcomes of ECGN in our center.
Methods: A retrospective single-center study was conducted at the Moulay Ismail Military Hospital in Meknes (Morocco). Nine consecutive adult patients diagnosed with ECGN between January 2022 and December 2024 were included. Descriptive analysis of demographic, clinical, biological, immunological, histological, therapeutic, and outcome data was performed.
Results: Nine patients (4 men, 5 women), mean age 68.9 years. The predominant etiology was pauci-immune ECGN (ANCA+) in 7 patients (78%; 4 Granulomatosis with Polyangiitis (GPA), 3 Microscopic Polyangiitis (MPA)/limited Vasculitis), followed by anti-Glomerular Basement Membrane (anti-GBM) antibody disease (Type I) in 2 patients (22%). Pulmonary-renal syndrome was frequent (56%). Initial renal failure was very severe: mean creatinine 670 µmol/L (75.9 mg/L), mean eGFR 7.2 mL/min/1.73m² (CKD-EPI), anuria in 33%. Renal biopsy (7/9 patients) showed an average of 66% crescents. Induction therapy included corticosteroids (9/9) and cyclophosphamide (8/9). No patients received plasma exchange

(unavailability). Initial hemodialysis was required for 44%. Outcomes included 3 early deaths (33%, sepsis/respiratory distress), 4 progressions to ESKD on chronic dialysis (44%), 1 CKD stage 3b (11%), and 1 complete remission (11%). The combined rate of death or ESKD was 78%.

Conclusion: This series illustrates the severity of ECGN in our setting, affecting elderly individuals with very severe initial presentation and a predominance of pauci-immune forms. The high morbidity and mortality observed, despite treatment with corticosteroids and cyclophosphamide, highlight the potential impact of therapeutic limitations, notably the absence of plasma exchange. Early diagnosis and access to the full range of therapeutic resources are crucial to improve prognosis.

KEYWORDS

Extracapillary glomerulonephritis, Rapidly progressive glomerulonephritis, ANCA, Anti-GBM antibody, Vasculitis, Acute kidney injury, Renal biopsy, Morocco.

MAIN ARTICLE

Introduction

Extracapillary glomerulonephritis (ECGN), also known as rapidly progressive glomerulonephritis (RPGN), represents a severe anatomo-clinical entity, constituting a diagnostic and therapeutic emergency in nephrology [1, 2, 3]. It is clinically characterized by a rapid deterioration of renal function, typically a fall in glomerular filtration rate (GFR) of more than 50% within weeks to months [1], and the characteristic histological presence of cellular or fibrocellular crescents occupying Bowman's space in more than 50% of glomeruli [4, 5, 6]. Crescent formation results from severe endocapillary inflammation leading to glomerular basement membrane (GBM) ruptures and the proliferation of parietal and inflammatory cells [7, 8, 9].

The classification of ECGN, based on immunofluorescence (IF) findings from renal biopsy, is fundamental for etiological orientation and therapeutic strategy [6]. Type I is mediated by anti-GBM antibodies (linear deposits), Type II by immune complexes (granular deposits), and Type III, termed pauci-immune (absence or paucity of immune deposits), is predominantly associated with ANCA (Anti-Neutrophil Cytoplasmic Antibody)-associated vasculitis [10]. ANCA, by activating neutrophils, cause inflammation and necrosis of small vessels, particularly glomerular capillaries [11].

The renal and vital prognosis of ECGN is severe without early and appropriate immunosuppressive treatment [1], with frequent progression to end-stage kidney disease (ESKD) [1]. Morbidity and mortality remain significant even with treatment, related to the disease itself and iatrogenic complications [12]. While the epidemiology (incidence 2-

7/million/year, peak age 50-70 years, pauci-immune predominance in the West) [1, 7, 13] and management are well codified by international guidelines [1, 14], epidemiological and outcome data may vary across regions and populations. In Morocco, a few series have been published, mainly from civilian university hospitals [15, 16, 17], showing a non-negligible frequency (8-11%) and possible etiological particularities, such as a significant proportion of lupus nephritis in some series [15]. A specific study on pulmonary-renal syndrome (PRS) was conducted in another military hospital [18], highlighting the need for additional local data.

The objective of our study was to describe the epidemiological profile, clinico-biological and histological characteristics, therapeutic modalities, and outcomes of a series of patients with ECGN managed in the Nephrology Department of the Moulay Ismail Military Hospital (HMMI) in Meknes.

Materials and Methods

- **Study Type and Setting**

  This was a retrospective, descriptive study conducted in the Department of Nephrology and Hemodialysis at HMMI in Meknes, Morocco.

- **Study Population and Period**

  Consecutive inclusion of all adult patients (≥ 18 years) hospitalized for ECGN/RPGN between January 2022 and December 2024 (N=9).

- **Inclusion Criteria**

  Clinical presentation of rapidly progressive renal failure AND histological confirmation by percutaneous renal biopsy (PRB) showing crescents OR strong diagnostic presumption (clinical presentation + positive anti-GBM Ab or ANCA serology) in case of contraindication or refusal of PRB.

- **Exclusion Criteria**

  Unexploitable medical records (no cases).

- **Data Collection**

  Retrospective data collection from medical records using a standardized form : demographic data, medical history, clinical presentation (renal/extra-renal symptoms, diuresis), laboratory tests (urinalysis, serum creatinine [mg/L and µmol/L], eGFR [CKD-EPI], 24h proteinuria [g/24h], CRP [mg/L], Hb [g/dL], MCV, MCHC), immunology (ANCA [IFI, ELISA anti-PR3/MPO], anti-GBM Ab), PRB histology (number of

glomeruli, % crescents, IF), treatments (induction [MP, CYC], maintenance [AZA, RTX], PE, initial HD), outcomes (follow-up, vital/renal status, final diagnosis).

- **Operational Definitions**

  ESKD was defined by the need for chronic HD. Chronic Kidney Disease (CKD) was defined according to KDIGO criteria (GFR < 60 mL/min/1.73m² for > 3 months). Complete remission was defined as dialysis independence, significant improvement/stabilization of renal function, and disappearance of signs of activity. Anuria was defined as urine output < 100 mL/24h.

- **Statistical Analysis**

  Descriptive analysis (mean ± SD, median [min-max], counts n, percentages %). Software: Microsoft Excel (Version 2019).

- **Ethical Considerations**

  The study adhered to the ethical principles of Helsinki, and patient anonymity was maintained. Formal ethics committee approval was not deemed necessary for this retrospective study on anonymized data, according to institutional practice.

Results

- Nine patients (4 men, 5 women) were included. The mean age was 68.9 ± 11.1 years (median 67 years). Detailed individual characteristics are presented in Table 1.
- **Etiological Characteristics :**

  The final diagnoses were: Pauci-immune ECGN (Type III, ANCA+) in 7 patients (78%), including 4 cases of GPA and 3 cases of MPA or limited vasculitis. Anti-GBM Ab disease (Type I) concerned 2 patients (22%).
- **Clinical and Biological Presentation :**

  Pulmonary-renal syndrome was the most frequent presentation (5/9, 56%) (Table 2). Other extra-renal involvements included ENT (n=1) and digestive (n=1) signs. Initial renal failure was very severe: mean serum creatinine 670 ± 225 µmol/L (75.9 ± 25.5 mg/L), mean eGFR 7.2 ± 3.1 mL/min/1.73m². Anuria was present in 3 patients (33%). Proteinuria was sub-nephrotic (mean 0.68 g/24h). An inflammatory syndrome (mean CRP 164 mg/L) and normocytic anemia (mean Hb 9.1 g/dL) were constant
- **Histological Data :**

A PRB was performed in 7 patients (78%). The mean number of glomeruli was 15 ± 4. The mean percentage of crescents was 66.1 ± 20.1%. IF confirmed 5 cases of Type III and 2 cases of Type I.

- **Treatment :**

  Induction therapy included Methylprednisolone (MP) pulses for all (100%) and Cyclophosphamide (CYC) pulses for 8 patients (89%). Critically, no patient (0%) received **plasma exchange (PE)** due to technical unavailability. Initial hemodialysis was required for 4 patients (44%). Maintenance therapy (Azathioprine (AZA) or Rituximab (RTX)) was initiated in 3 patients (Table 3).

- **Outcomes and Prognosis :**

  Outcomes at last follow-up were : 3 early deaths (33%) (< 3 months, respiratory/infectious cause), 4 patients (44%) progressed to End-Stage Kidney Disease (ESKD) requiring chronic Hemodialysis (HD). One patient (11%) had CKD Stage 3b, and only one patient (11%) achieved complete remission. The combined rate of death or ESKD was 78% (7/9) (Table 4).

Discussion

Our retrospective study, although based on a small sample size (N=9), describes a population with ECGN exhibiting distinct characteristics within the context of a Moroccan military hospital. The high mean age (69 years) contrasts with the median age reported in other Moroccan series, such as Marrakech (41 years) [15] or Rabat (45 years) [18], but aligns more closely with Western cohorts where AAV predominates in the elderly [19, 20]. ECGN did not appear to be sex-linked in our series (slight female predominance), similar to others [23], whereas some Moroccan series noted a male predominance [15, 18]. The clear predominance of pauci-immune ANCA+ forms (78%) is, however, similar to findings in Rabat (78% pauci-immune) [18] and international literature [23, 24, 25], differing from the Marrakech series where lupus was a major cause [15].

The extreme severity of the initial presentation is a salient point : very low mean eGFR (7.2 mL/min/1.73m²) and a high rate of initial anuria (33%). Anuria is recognized as a very poor renal prognostic factor in ECGN [26, 27]. This severity is comparable to the Rabat series (eGFR 5 mL/min, anuria 28%) [18] and meets the inclusion criteria for the most severe patients in international trials like PEXIVAS [32]. This presentation, a known poor prognostic factor [10], suggests potentially delayed diagnosis [27] or particular intrinsic disease aggressiveness.

Management with corticosteroids and cyclophosphamide aligns with standard induction regimens [1, 14]. However, the fundamental difference and major challenge lie in the complete absence of plasma exchange (PE). This situation contrasts not only with international recommendations positioning PE as essential therapy for anti-GBM disease [1, 29] (used in Rabat [18]), but also with the historical and current debated role for the most severe ANCA-Associated Vasculitis (AAV) [1, 14, 22, 30, 31, 32]. The value of PE in these severe forms was highlighted over 20 years ago [30].

Faced with this constraint, our strategy focused on maximal and rapid pharmacological immunosuppression. It is particularly noteworthy that for our 2 anti-GBM disease cases, this approach, although incomplete without PE, likely helped control alveolar hemorrhage and ensure their survival through the acute phase. Indeed, neither experienced fatal pulmonary complications. Although their renal outcome was ESKD – a frequent outcome even with PE [29, 30] – the fact that these patients are still alive today on chronic hemodialysis attests to the medical team's effort in managing these critical situations by maximizing the efficacy of accessible treatments and ensuring intensive overall care.

The overall prognosis observed remains particularly grim (78% death or ESKD). Our early mortality (33%) appears markedly higher than reported in Rabat (11%) [18] or in the EUVAS and PEXIVAS trials (~10-15% at 1 year) [12, 32]. Of great concern, none of our initially dialysis-dependent patients (0/4) recovered renal function, whereas the Rabat series reported recovery in 21% [18], and PE is suspected to improve this early recovery [31]. The overall remission rate was also much lower in our series (11% vs 44% in Rabat [18]). These discrepancies, particularly regarding early mortality and renal recovery (see Table 5), strongly suggest that the absence of PE was a major aggravating factor in our series.

The limitations of our study are evident: N=9, retrospective, single-center. Comparison with other series reinforces the hypothesis of the impact of PE unavailability. However, this "real-world" description highlights the concrete challenges of ECGN management, while also underscoring the team's ability to manage acute survival through optimized immunosuppression in a potentially resource-limited setting.

Conclusion

ECGN in our series presents as a severe disease in elderly patients, with very poor initial renal function and a predominance of pauci-immune ANCA+ forms. The observed renal and vital prognosis is concerning (78% death/ESKD) despite treatment with corticosteroids and cyclophosphamide. The unavailability of plasma exchange appears as a major therapeutic limitation that likely contributed to these outcomes. Nevertheless, rigorous application of

available immunosuppressive treatments allowed control of acute systemic complications and ensured the survival of very severe patients. Prognostic improvement requires earlier diagnosis and access to the full recommended therapeutic arsenal. Prospective multicenter Moroccan studies are essential to better define the local epidemiology, prognostic factors, and the impact of available therapeutic strategies in our national context.

TABLES :

Table 1 : Summary Characteristics of the 9 Patients with ECGN

ID	Age / Sex	Étiology (Type / Marker)	Init Créat (µmol/ L)	Init eGFR (ml/min//1.73m2)	Anuria	PRB (% Cresc.)[1]	PE Received	Init HD	Final Statut
1	76 / H	Pauci (ANCA-PR3+)	556	9	No	Yes (54%)	No	No	Deceased
2	55 / F	Anti-GBM (Ac+)	891	4	Yes	Yes (88%)	No	Yes	ESKD / HD
3	88 / H	Anti- GBM (Ac+)	1015	4	Yes	Yes (91%)	No	Yes	ESKD / HD
4	84 / H	Pauci (ANCA-MPO+)	485	10	No	Yes (42%)	No	No	Deceased
5	64 / F	Pauci (ANCA-PR3+)	706	5	No	Yes (62%)	No	No	Deceased
6	62 / F	Pauci (ANCA-MPO+)	830	4	No	Yes (80%)	No	No	CKD 3b
7	58 / F	Pauci (ANCA-PR3+)	406	10	No	No[2]	No	No	ESKD / HD
8	66 / H	Pauci (ANCA-MPO+)	794	6	Yes	No[2]	No	Yes	ESKD / HD
9	67 / F	Pauci (ANCA-PR3+)	344	12	No	Yes (46%)	No	No	Rémission

Notes / Abbreviations:

ID: *Patient Identifier;* ***M****: Male;* ***F****: Female;* ***Pauci****: Pauci-immune;* ***Anti-GBM:*** *Anti-GBM Antibody Disease;* ***Ab+:*** *Antibody Positive.*

Init Creat*: Initial Serum Creatinine;* ***Init eGFR****: Initial Estimated Glomerular Filtration Rate (CKD-EPI).*

Init Anuria*: Urine output < 100 mL/24h at admission.*

PRB*: Percutaneous Renal Biopsy; % Cresc.: Percentage of glomeruli with crescents.*

PE Received*: Plasma Exchange received.*

Init HD*: Hemodialysis required initially.*

Final Status*: At last follow-up; ESKD/HD: End-Stage Kidney Disease on Hemodialysis;*

CKD 3b*: Chronic Kidney Disease Stage 3b.*

[1] For the 7 patients who underwent PRB. [2] PRB not performed ***(small kidneys)****.*

Table 2 : Initial Clinical Presentation (N=9)

Variable	Count (n)	Percentage (%)
Rénal signs		
- Anuria (<100 mL/24h)	3	33.3%
- Hématuria (Urinalysis ≥ 3 +)	7/8[1]	87.5%
- Protéinuria (Urinalysis ≥ 1 +)	8/8[1]	100%
Pulmonary signs		
- Pulmonary Rénal Syndrome (PRS)[2]	5	55.6%
Other Extra-Rénal signs		
- ENT Involvement	1	11.1%
- Digestive Involvement	1	11.1%

[1] Urinalysis data available for 8 patients.
[2] Defined by the association of renal signs and pulmonary involvement (cough, fever, hemoptysis/DAH).

Table 3 : Initial Therapeutic Management (N=9)

Treatement	Count (n)	Percentage (%)
Induction		
- Corticosteroids (MP Pulses)	9	100%
- Cyclophosphamide (IV Pulses)	8	88.9%

- Plasma Exchange (PE)	0	0%
Renal Replacement Therapy		
- Initial Hemodialysis	4	44.4%
Maintenance (initiated)		
- Azathioprine or Rituximab	3	33.3%

Table 4: Final Patient Outcomes (N=9)

Final Status	**Count (n)**	**Percentage (%)**
Deceased	3	33.3%
ESKD / Chronic HD	4	44.4%
CKD non-dialysis (Stage 3b)	1	11.1%
Complete Remission	1	11.1%
Combined Death or ESKD	7	77.8%

Table 5 : Comparison of ECGN Characteristics and Outcomes : HMMI Meknes Series vs. Other Cohorts and References

Characteristic	**HMMI Meknes Series (N=9, ECGN)**	**HMIMV Rabat Series (N=18, PRS) [18]**	**EUVAS Cohorts (AAV, Various**	**PEXIVAS Trial (N=704, Severe AAV)[1]**	**KDIGO 2021 (Recommandations)**	**"Classic Cohorts" / General Literature**
Population	Adults, Military	Adults, Military (PRS only)	Adults (AAV)	Adults, Severe AAV (eGFR<50 ou DAH)	Adults & Children	Variable (Adults++)
Mean / Median Age	68.9 yrs (Médian 67)	Médian 45 yrs	~50-65 yrs	~60-65 yrs	Peak often 50-70 yrs	Peak 50-70 yrs

Predominant Etiology	78% Pauci (ANCA+), 22% Anti-GBM	78% Pauci (incl. ANCA-neg), 17% Anti-GBM, 6% Lupus	Majority Pauci (ANCA+)	100% Pauci (ANCA+)	Pauci frequent ; Anti- GBM rare ; Type II variable	often>50% Pauci-immune
Initial Severity			Variable	High (by definition)	Variable	Variable
- Mean eGFR (ml/min)	7.2	5.0	Variable (often <20 in renal trials)	< 50 (by definition)	Variable	Variable
- Dialysis at admission	44%	78%	Variable (e.g., ~20% in RITUXVAS)	~50% (in some analyses)	Variable	Variable (e.g: 20-40%)
- Anuria at admission	33%	28%	Less frequent	Not specified globally	Poor prognostic factor	Less frequent (severity factor)
- Diffuse Alveolar Hemorrhage (DAH)	56% (PRS)	100% (by definition)	Variable (e.g., ~10-20%)	Possible inclusion criterion (~20%?)	Potential PE indication if severe	Variable (e.g., 10-30% in AAV)
Induction Treatment						
- Corticosteroids + CYC/RTX	Yes (Standard)	Yes (Standard)	Yes (Standard, shift towards RTX)	Yes (Standard)	Yes (Recommanded)	Yes (Historical/Current Standard)
- Plasma Exchange (PE)	NO (0%) - Unavailable	YES (33%) (Severe DAH/Adv. RF)	Variable (used in some trials/centers for	YES (PE arm, ~50%) vs NO (Control arm, ~50%)	YES (Recommended Anti-GBM); YES (Discussed/Reco	Variable (depends on severity/center/era)

			severe cases)		mmended Severe AAV²)	
Outcomes / Prognosis					Variable, depends on type/severity/Tx	Variable
Mortality (early/1 yr)	33% (< 3 mo)	11%	~10-15% at 1 yr	~10-15% at 1 yr (?)	High risk if Tx delayed/inadequate	Variable (e.g., 10-20% at 1 yr)
- ESKD / HD Dependence	44% ((early/mid-term)	~61% (final)	~15-25% at 1-5 yrs (Severe AAV)	~20-25% at 1 yr (?)	High risk if Tx delayed/inadequate	Variable (e.g., 20-40% at 5 yrs for AAV)
- Death OR ESKD (combined)	78% (early/mid-term)	~72% (final)	Variable (e.g., ~30-40% at 5 yrs?)	~30% (at 7 yrs, no difference PE vs non-PE)	Very high without appropriate Tx	Variable (e.g., ~40-50% at 5 yrs?)
Renal Recovery (if init HD)	0% (0/4)	21% (3/14)	Variable / Not systematically reported	| Not reported as primary endpoint (but PE seemed to improve short-term)	Possible but difficult if anuria/chronic lesions	Variable

Notes / Abbreviations *(Common to both tables):*
ECGN*: Extracapillary Glomerulonephritis;* ***RPGN****: Rapidly Progressive Glomerulonephritis;* ***AAV****: ANCA-Associated Vasculitis;* ***Anti-GBM****: Anti-Glomerular Basement Membrane;* ***PRS****: Pulmonary-Renal Syndrome;* ***HMMI:*** *Moulay Ismail Military Hospital;* ***HMIMV****: Mohammed V Military Instruction Hospital;* ***eGFR****: Estimated Glomerular Filtration Rate;* ***HD****: Hemodialysis;* ***CYC****: Cyclophosphamide;* ***RTX****: Rituximab;* ***MP****: Methylprednisolone;* ***PE****: Plasma Exchange;* ***ESKD****: End-Stage Kidney Disease;* ***CKD****: Chronic Kidney Disease;* ***DAH****: Diffuse Alveolar Hemorrhage.*
[1] ***PEXIVAS****: Trial on severe AAV (eGFR<50 or DAH). Compared PE vs non-PE. Primary endpoint: Death or ESKD long-term (median 7 yrs).*
[2] ***KDIGO & PE for AAV****: Recommendation for creat > 500 µmol/L, dialysis or severe DAH, mentioning PEXIVAS results (no benefit on primary composite endpoint long-term).*
[3] ***EUVAS:*** *European Vasculitis Study Group. Data from large observational cohorts and clinical trials (e.g., Flossmann 2011 [12], MEPEX, CYCLOPS, RITUXVAS [21] trials...). Figures are indicative and vary by trial/period.*
[18]: Reference to Benbria S. thesis (Rabat, 2021).

ACKNOWLEDGEMENTS :

The authors declare no conflicts of interest.

REFERENCES :

1. Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases. Kidney Int. 2021 Oct;100(4S):S1-S276. doi: 10.1016/j.kint.2021.05.021.
2. Collard HR, Schwarz MI. Diffuse alveolar hemorrhage. Clin Chest Med. 2004 Sep;25(3):583-92, vii. doi: 10.1016/j.ccm.2004.04.007.
3. Zycinska K, Wardyn KA, Zielonka TM, Otto M. The role of ANCA and anti-GBM antibodies in pulmonary-renal syndrome due to Wegener's granulomatosis. J Physiol Pharmacol. 2007 Nov;58 Suppl 5(Pt 2):839-46.
4. Jennette JC, Thomas DB. Crescentic glomerulonephritis. Nephrol Dial Transplant. 2001;16 Suppl 6:80-2. doi: 10.1093/ndt/16.suppl_6.80.
5. Churg J, Sobin LH. Renal Disease: Classification and Atlas of Glomerular Diseases. New York: Igaku-Shoin; 1982. (Adjusted reference for Churg & Sobin classification)
6. Jennette JC, Falk RJ, Andrassy K, et al. Nomenclature of systemic vasculitides. Proposal of an international consensus conference. Arthritis Rheum. 1994 Feb;37(2):187-92. doi: 10.1002/art.1780370206. (Reference for nomenclature/classification)
7. Le Hir M, Haas C, Marino M, Ryffel B. Podocyte bridges between the tuft and Bowman's capsule: an early event in experimental crescentic glomerulonephritis. J Am Soc Nephrol. 2001 Nov;12(11):2071-4. doi: 10.1681/ASN.V12112071.
8. Le Hir M, Haas C, Marino M, Ryffel B. Prevention of glomerulonephritis induced by anti-glomerular basement membrane (GBM) antibody in TNF-deficient mice. Lab Invest. 1998 Dec;78(12):1625-31.
9. Bonsib SM. Glomerular basement membrane discontinuities. Scanning electron microscopic study of acellular glomeruli. Am J Pathol. 1985 Jun;119(3):357-60.
10. Noel LH. [Renal pathology atlas]. Med Sci (Paris). 2008;24 Spec No 3:27-49. French.
11. Jennette JC, Falk RJ. Pathogenesis of antineutrophil cytoplasmic autoantibody-mediated disease. Nat Rev Rheumatol. 2014 Aug;10(8):463-73. doi: 10.1038/nrrheum.2014.103.
12. Flossmann O, Berden A, de Groot K, et al; European Vasculitis Study Group (EUVAS). Long-term patient survival in ANCA-associated vasculitis. Ann Rheum Dis. 2011 Mar;70(3):488-94. doi: 10.1136/ard.2010.137778.
13. Watts RA, Mahr A, Mohammad AJ, Gatenby P, Basu N, Flores-Suarez LF. Classification, epidemiology and clinical subgrouping of antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis. Nephrol Dial Transplant. 2015 Sep;30 Suppl 1:i14-22. doi: 10.1093/ndt/gfv022.
14. Hellmich B, Sanchez-Alamo B, Schirmer JH, et al. EULAR recommendations for the management of ANCA-associated vasculitis: 2022 update. Ann Rheum Dis. 2024 Jan 2;83(1):30-47. doi: 10.1136/ard-2023-224798.
15. El Fehmi L. Glomérulonéphrites extracapillaires : profil épidémiologique, clinique, thérapeutique et évolutif. [MD Thesis]. Marrakech: Cadi Ayyad University, Faculty of Medicine and Pharmacy; 2020. N°90. French.
16. Bouabid J, Zemraoui N. Biopsie rénale dans la région d'Agadir : indication et résultat (A propos de 70 cas). [Poster presentation]. Congrès de la Société Marocaine de Néphrologie; 2018. French.
17. Ait Lahcen Z, Laouad I. Néphropathies glomérulaires : profil épidémiologique, thérapeutique et évolutif au CHU Mohammed VI de Marrakech. [MD Thesis]. Marrakech; 2013. French.

18. Benbria S. Syndrome pneumo-rénal : Expérience d'un centre militaire (A propos de 16 cas). [Medical Specialty Thesis Nephrology]. Rabat: Mohammed V University; 2021. French.
19. Quiroga B, Arruza A, Casado S, et al; Spanish Registry of Glomerulonephritis. Crescentic glomerulonephritis: data from the Spanish Registry of Glomerulonephritis. Intern Med J. 2015 Oct;45(10):1037-43. doi: 10.1111/imj.12851.
20. Zink C, Zink O, Mildenberger S, et al. Trends of Renal Diseases in Germany: Review of a Regional Renal Biopsy Database from 1990 to 2013. Clin Kidney J. 2019 Oct 1;12(5):685-695. doi: 10.1093/ckj/sfy128.
21. Gallagher H, Kwan JT, Jayne DR. Pulmonary renal syndrome: a 4-year, single-center experience. Am J Kidney Dis. 2002 Jan;39(1):42-7. doi: 10.1053/ajkd.2002.29869.
22. Lauque D, Cadranel J, Lazor R, et al. Microscopic polyangiitis with alveolar hemorrhage. A study of 29 cases and review of the literature. Groupe d'Etudes et de Recherche sur les Maladies "Orphelines" Pulmonaires (GERM"O"P). Medicine (Baltimore). 2000 Jul;79(4):222-33. doi: 10.1097/00005792-200007000-00003.
23. Jaynul I, Jahan I, Muqeet MM, Rahman MH, Islam MN. Immunohistological Spectrum of Diffuse Crescentic Glomerlonephritis, a Single Center Study in Bangladesh. Clin Pathol Res J. 2019;2(1):1-6.
24. Rampelli SK, Rajesh NG, Srinivas BH, Haridas C, Priyamvada PS, Parameswaran S. Clinical Spectrum and Outcomes of Crescentic Glomerulonephritis: A Single Center Experience. Indian J Nephrol. 2016 Jul-Aug;26(4):252-6. doi: 10.4103/0971-4065.175980.
25. McAdoo SP, Pusey CD. Anti-Glomerular Basement Membrane Disease. Clin J Am Soc Nephrol. 2017 Jul 7;12(7):1162-1172. doi: 10.2215/CJN.01390217. (Updated reference for Anti-GBM)
26. Wu T, Yuan Z, Duan Z, Zhang L, Chen Z. Clinicopathological Features and Prognostic Analysis of 49 Cases with Crescentic Glomerulonephritis. Exp Ther Med. 2019 Nov;18(5):3541-3548. doi: 10.3892/etm.2019.8011.
27. Faye M, Kane Y, Diallo S, et al. Crescentic Glomerulonephritis in a Sub-Saharan Country: Clinical Presentation, Etiological and Evolutive Profile 2017. Nephrourol Mon. 2017 Nov 1;9(6):e59007. doi: 10.5812/numonthly.59007.
28. Harper L, Morgan MD, Walsh M, et al; EUVAS investigators. Pulse versus daily oral cyclophosphamide for induction of remission in ANCA-associated vasculitis: long-term follow-up. Ann Rheum Dis. 2012 Jun;71(6):955-60. doi: 10.1136/annrheumdis-2011-200477.
29. Johnson JP, Moore J Jr, Austin HA 3rd, Balow JE, Antonovych TT, Wilson CB. Therapy of anti-glomerular basement membrane antibody disease: analysis of prognostic significance of clinical, pathologic and treatment factors. Medicine (Baltimore). 1985 Jul;64(4):219-27. doi: 10.1097/00005792-198507000-00001.
30. Guillevin L. [Plasma exchange in systemic diseases: current status]. Reanimation. 2002;11(5):359-65. French. doi: 10.1016/s1624-0693(02)00256-6.
31. Jayne DR, Gaskin G, Rasmussen N, et al; European Vasculitis Study Group. Randomized trial of plasma exchange or high-dosage methylprednisolone as adjunctive therapy for severe renal vasculitis. J Am Soc Nephrol. 2007 Jul;18(7):2180-8. doi: 10.1681/ASN.2007010090.
32. Walsh M, Merkel PA, Peh CA, et al; PEXIVAS Investigators. Plasma Exchange and Glucocorticoids in Severe ANCA-Associated Vasculitis. N Engl J Med. 2020 Feb 13;382(7):622-631. doi: 10.1056/NEJMoa1803537.