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Evaluation of the Biochemical Subtypes of Amyloidosis without Immunohistochemistry according to the Recommendations of the International Amyloidosis Nomenclature Committee: A Multicenter Study in Dakar Involving 41 Cases

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Abstract

Introduction: Amyloidoses are a heterogeneous group of diseases characterized by extracellular deposits of insoluble fibrillar proteins in tissues. In sub-Saharan Africa, the epidemiology remains poorly understood, and the diagnosis of biochemical subtypes remains challenging in our clinical setting. The objective of our study was to determine the biochemical subtypes based on observed phenotypes, in accordance with international recommendations. **Methods:** This is a multicenter study combining a retrospective clinical review of hospital archives and an aggregation of cases documented in the scientific literature. All cases of histologically confirmed amyloidosis in patients over 16 years of age from January 1990 to October 2024 within level-3 national centers

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in Dakar were screened. Subtypes were systematically stratified into Confirmed (via immunohistochemistry/immunofluorescence), Probable (via predictive clinical phénotype and etiology guidelines), or Indeterminate categories. **Results:** A total of 41 cases were identified, including 18 men and 23 women (male-to-female ratio 0.78). The mean age was 59.93 ± 14.89 years. Regarding data sources, 36 cases (87.80%) were compiled from direct hospital archives and 5 cases (12.20%) were integrated from peer-reviewed publications. The most common manifestations were renal (75.61%, n = 31), cardiac (48.78%, n = 20), and gastrointestinal (39.02%, n = 16). The histological diagnosis was established via renal biopsy in 22 patients (53.65%), salivary gland biopsy in 13 patients (31.70%), skin biopsy in two patients (4.87%), bronchial biopsy in one patient (2.44%), and combined salivary-subcutaneous adipose tissue biopsy in three patients (7.31%). Definitive subtyping was Confirmed in only 3 cases (2 cases of AL and 1 case of AA). Based on phenotypes according to international guidelines, the remaining cases were classified as Probable AA in 20 patients, Probable AL in 12 patients, Probable senile ATTR in 2 patients, Probable beta-2-microglobulin in 2 patients, and Indeterminate in 2 patients. Sensitivity analysis excluding literature cases demonstrated no significant shift in subtype distribution. Chronic infectious or inflammatory etiologies underlaid all 21 AA cases, with tuberculosis alone accounting for 17.07% (n = 7) of the entire cohort. All AL amyloidosis cases were secondary to multiple myeloma. The 36-month survival rate was 60%, with age being the only factor statistically associated with death (p = 0.023). **Conclusion:** The incidence of amyloidosis in our region has been steadily increasing over the past decade. However, immunophenotyping remains highly inaccessible. Renal manifestations are the most common.

Keywords

Amyloidosis, Biochemical Subtypes, Sub-Saharan Africa, Senegal, Survival Analysis

1. Introduction

Amyloidoses represent a heterogeneous group of diseases defined by the extracellular deposition of insoluble fibrillar proteins in tissues, sharing common staining properties, a fibrillar structure under electron microscopy, and a spatial conformation known as beta-pleated [1]. They can be hereditary or acquired, localized or systemic.

The diagnosis is historical and histological, based on the detection of amyloid deposits using specific stains. Biochemical subtypes are ideally determined by immunohistochemistry and/or immunofluorescence using specific antibodies [1] [2]. Since 2022, forty-two amyloid proteins have been identified by the Nomenclature Committee of the International Amyloidosis Society [3], each with its own clinical characteristics [4].

The epidemiology of amyloidosis varies by geographic region. Transthyretin

amyloidosis (ATTR) and immunoglobulin light-chain amyloidosis (AL) [5]-[8] are more common in developed countries, whereas AA amyloidosis is thought to be predominant in developing countries and/or the Mediterranean region [7]-[10]. In sub-Saharan Africa, a 2012 systematic review of the literature showed an incidence ranging from 0.28% to 0.57% in autopsy series dating back to 1975 [10].

Due to inadequate technical facilities, immunohistochemistry and immunofluorescence are rarely performed in our setting. Thus, the overall objective of our study was to determine the biochemical subtypes based on the observed phenotypes, in accordance with international recommendations, while explicitly evaluating diagnostic certainties through standardized classification.

2. Patients and Methods

2.1. Study Design and Data Sourcing Protocol

We conducted a multicenter study at various Level 3 national centers in Dakar, including 16 medical departments and 3 departments of pathological anatomy. We screened all cases of amyloidosis confirmed by histology in patients over 16 years of age from January 1990 to October 2024.

Case recruitment was performed through two separate pathways:

1) Direct Hospital Record Review: A retrospective review of patient files from outpatient clinics and inpatient wards was performed using a pre-designed data collection form.

2) Scientific Literature Aggregation: An online literature review was conducted in PubMed, ScienceDirect, and African Journals OnLine (AJOL) using the keywords: *amyloidosis*, *amyloid*, *Dakar*, *and black*. A manual check of bibliographical references was performed. Out of 8 published case reports identified from Senegal, 3 were excluded because they were already captured within the hospital databases or lacked primary variables. To prevent double-counting or duplicating data from patients, deduplication was performed by cross-referencing patient initials, sex, age at diagnosis, year of admission, and the reporting hospital center.

2.2. Taxonomical Classification and Variable Definitions

To account for the limited access to immunophenotyping, all biochemical subtypes were systematically reclassified into three distinct diagnostic certainty categories based on the recommendations of the International Amyloidosis Society Committee [3] [11]:

1) Confirmed Subtype: Formally established in the presence of positive immunohistochemistry and/or immunofluorescence using specific antibodies.

2) Probable Subtype: Assigned in the absence of typing, when a patient met all presumptive epidemiological, clinical phenotype, and etiological criteria according to international recommendations.

3) Indeterminate Subtype: Categorized when histologically proven amyloidosis lacked any prescriptive phénotypic or etiological evidence to support a specific

biochemical protein.

The presumptive international criteria used to assign a Probable subtype were:

1) AA Amyloidosis: Nephrotic syndrome or chronic kidney disease, combined with an active or long-standing history of a chronic infectious disease (e.g., tuberculosis, leprosy) or a well-documented chronic autoinflammatory/autoimmune condition (e.g., rheumatoid arthritis).

2) AL Amyloidosis: Nephrotic syndrome, restrictive cardiomyopathy, macroglossia, or peripheral neuropathy, occurring in the presence of a serum/urinary monoclonal gammopathy or a clonal plasma cell population in the bone marrow.

3) Senile ATTR Amyloidosis: Patients aged 75 years or older presenting with predominant restrictive cardiomyopathy, in the complete absence of a detectable monoclonal gammopathy or family history of amyloidosis.

4) Genetic ATTR Amyloidosis: Sensorimotor axonal neuropathy or cardiomyopathy associated with autonomic dysfunction and a confirmed family history of hereditary systemic amyloidosis.

5) Beta-2-Microglobulin Amyloidosis: Long-term hemodialysis patients (on dialysis for more than 5 years) presenting with carpal tunnel syndrome, joint pain, or destructive arthropathy.

Visceral organ involvement was formally attributed to amyloidosis if tissue biopsy confirmed amyloid material or if clinical, biological, and non-invasive imaging findings were strongly suggestive of organ dysfunction as defined by international consensus guidelines, even in the absence of site-specific histological confirmation.

2.3. Statistical Analysis and Survival Assessment

Statistical analysis was performed using Sphinx and SPSS software. Quantitative variables are presented as means ± standard deviations; qualitative variables as counts and percentages. Survival analysis was conducted using the Kaplan-Meier method to calculate the cumulative survival rate over time. Right-censoring was applied at the date of last known follow-up or at the end of the 36-month observation window. To manage the high rate of patients lost to follow-up (43.90%), a sensitivity analysis was performed by treating them as censored at their last contact date. Due to the limited number of documented deaths (n = 7), multivariate Cox regression analysis was treated as strictly exploratory, and no definitive long-term prognostic conclusions were drawn. Ethical clearance was obtained from the institutional boards, data were anonymized, and a waiver of written consent was granted for this retrospective chart review and literature public data analysis.

3. Results

3.1. Descriptive and Recruitment Data

Forty-one cases were identified. This total reflects a single, verified master dataset

comprised of 36 cases (87.80%) from direct hospital consultations and 5 cases (12.20%) from the aggregated literature review. The incidence of amyloidosis has been steadily increasing over the past decade. Our cohort consisted of 18 men and 23 women, yielding a male-to-female ratio of 0.78. The mean age at diagnosis was 59.93 ± 14.89 years (range: 24 to 83 years). Patients were primarily recruited from nephrology (39.02%, n = 16) and internal medicine (31.71%, n = 12) (Table 1). No family history of amyloidosis was found.

Table 1. Distribution of amyloidosis cases by referring specialty.

Specialty	Number (n)	Percentage (%)
Nephrology	16	39.02
Internal Medicine	12	31.71
Cardiology	4	9.76
Clinical Hematology	4	9.76
Pathological anatomy	3	7.32
Rheumatology	1	2.44
Total	**41**	**100.00**

The most common reasons for consultation were lower extremity edema (60.98%, n = 25) and diffuse pain (39.02%, n = 16) (Table 2). Amyloidosis was systemic in 68.29% (n = 28) and localized in 31.71% (n = 13). The histological diagnosis was made based on a renal biopsy in 22 patients (53.65%), a salivary gland biopsy in 13 patients (31.70%), a skin biopsy in two patients (4.87%), a bronchial biopsy in one patient (2.44%), and via combined salivary gland and abdominal subcutaneous fat biopsy in three patients (7.31%) [12].

Table 2. Distribution of amyloidosis cases by reason for consultation.

Reasons for Consultation	Number of Cases (n)	Percentage (%)
Edema of the Lower Extremities	25	60.98
Asthenia	19	46.34
Diffuse Pain	16	39.02
Dyspnea	13	31.71
Deterioration in General Condition	11	26.83
Dry Mouth	11	26.83
Weight Loss	5	12.20
Abdominal Pain	5	12.20
Cough	2	4.88
Vomiting/Diarrhea/Dysphagia	3	7.32
Other (Skin Lesions, Lymphadenopathy, Paresthesia)	4	9.76

3.2. Clinical Presentation and Organ Involvement

Renal manifestations were reported in 31 cases (75.61%). Their presentation was dominated by a glomerular nephropathy syndrome (64.51%, n = 20), presenting as an impure nephrotic syndrome in 12 cases [13]. Chronic kidney disease was present in 51.61% (n = 16) of these patients, hypertension in 25.80% (n = 8), and microscopic hematuria in 58.06% (n = 18). Renal ultrasound revealed kidneys of normal size in 18 cases (58.06%), small kidneys in 2 cases (6.45%), and renal parenchymal structural changes in 3 cases (9.67%) **(Table 3)**.

Table 3. Clinical and laboratory characteristics of renal manifestations.

Renal Manifestations	Total (n = 31)	Percentage (%)
Glomerular Nephropathy Syndrome	20	64.51
- Pure	08	25.80
- Mixed/Impure	12	38.70
Undetermined Presentation	11	35.48
Chronic Kidney Disease/Failure		
- Present	16	51.61
- Absent	15	48.39
Renal Ultrasound Findings		
- Normal/Retained Size	18	58.06
- Small Size	02	6.45
- Renal Structural Changes	03	9.67
- Nephromegaly	01	3.22

Cardiac manifestations were observed in 20 cases (48.78%), symptomatic in 13 cases (65%), and dominated by dyspnea. Electrocardiogram (ECG) performed in 34 cases was abnormal in 30 patients, dominated by microvoltage in 44.12% (n = 15). Echocardiography performed in 21 patients revealed a suggestive pattern in 12 patients (57.14%), showing restrictive cardiomyopathy with predominant septal hypertrophy in 11 cases (52.38%) **(Table 4)** [14]. Cardiac MRI performed in 5 cases was helpful in 3 patients. No endomyocardial biopsies were performed.

Table 4. Clinical and paraclinical characteristics of cardiac manifestations.

Cardiac Manifestations	Total (n = 20)	Percentage (%)
Presentation		
- Asymptomatic	07	35.00
- Symptomatic (Dyspnea/Heart Failure)	13	65.00
Electrocardiogram (ECG) (n = 34)		
- Normal	04	11.76
- Abnormal	30	88.24

Continued

*Microvoltage	15	44.12
*Conduction/Rhythm Disorders	15	44.12
Echocardiogram Findings (n = 21)		
- Normal	09	42.86
- Restrictive Cardiomyopathy with Hypertrophy	11	52.38
- Granular/Shiny Myocardium Appearance	01	4.76

Gastrointestinal manifestations were observed in 16 patients (39.02%); macroglossia was present in 2 cases (4.87%). Musculoskeletal manifestations were found in 13 patients (31.70%), dominated by polyarthritis (n = 8) **(**Figure 1**)**. Neurological manifestations were found in 9 patients, presenting as sensorimotor polyneuropathy in 8 cases (19.51%) and carpal tunnel syndrome in 2 cases (4.88%) **(**Figure 2**)**. Dermatological signs were present in 3 cases (7.31%), including periorbital hematomas (n = 2). Pulmonary manifestations were observed in 12 cases (29.26%).

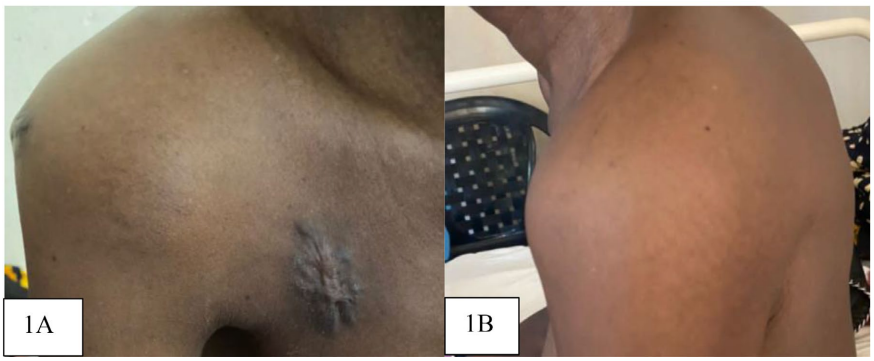

Figure 1. Shoulder strap sign during AL amyloidosis (A: frontal view, B: lateral view).

Figure 2. Carpal tunnel syndrome.

3.3. Etiological Profile and Stratified Subtype Distribution

Definitive immunophenotyping was successfully performed in only 3 cases, establishing a Confirmed diagnosis of AL amyloidosis in 2 cases (4.87%) and Confirmed AA amyloidosis in 1 case (2.43%). The remaining 38 patients were strati-

fied into Probable or Indeterminate categories based on phenotype-etiology alignment.

Neoplastic disease (multiple myeloma) underlaid 34.14% (n = 14) of cases, followed by chronic infections in 24.39% (n = 10) and rheumatic/autoimmune diseases in 24.39% (n = 10). Tuberculosis alone accounted for 17.07% (n = 7) of cases (Table 5).

Table 5. Distribution of cases according to the different etiologies of amyloidosis.

Etiological Categories	Specific Diseases	Number (n)	Percentages (%)
Neoplastic	Multiple Myeloma	14	34.14
Infectious	Tuberculosis/Leprosy	08	19.51
Autoimmune	Rheumatoid Arthritis/Lupus	05	12.19
Autoinflammatory	Crohn's Disease /SPA/FMF/DADA2	04	9.76
Mixed/Other	RA + Tuberculosis/ Sarcoidosis /ARF	04	9.76
Undetermined	-	06	14.63
Total		41	100.00

Note: **SPA:** Ankylosing Spondylitis, **FMF:** Familial Mediterranean Fever, **DADA2:** Adenosine Deaminase 2 Deficiency, **ARF:** Acute Rheumatic Fever, **RA:** Rheumatoid Arthritis.

By correlating diagnostic certainty categories with biochemical types, our single dataset yielded: Confirmed AL in 2 cases, Confirmed AA in 1 case, Probable AA in 20 cases, Probable AL in 12 cases, Probable senile ATTR in 2 cases, Probable beta-2-microglobulin in 2 cases, and Indeterminate amyloidosis in 2 cases (Table 6). To meet the reviewers' request, an analysis of sensitivity was performed: excluding the 5 literature-derived cases resulted in a cohorte of 36 hospital cases, within which the sub-type proportions remained statistically unchanged (AA: 52.77%, AL: 33.33%, ATTR: 5.55%, B2M: 5.55%, Indeterminate: 2.77%), proving that literature integration did not skew the local epidemiological landscape.

Table 6. Distribution of cases according to biochemical subtypes and certainty levels.

Biochemical Subtype	Certainty Category	Number (n)	Cohort Percentage (%)	Age Mean (Years)	Sex (M/F)
AA Amyloidosis	Confirmed (n = 1)/Probable (n = 20)	21	51.22	53.60	9/12
AL Amyloidosis	Confirmed (n = 2)/Probable (n = 12)	14	34.14	68.70	10/4
Senile ATTR	Probable	02	4.88	72.00	2/0
Beta-2-M (B2M)	Probable	02	4.88	55.50	2/0
Indeterminate	Indeterminate	02	4.88	57.00	1/1
Total		**41**	**100.00**	**59.93**	**18/23**

3.4. Therapeutic and Survival Outcomes

Therapeutic management was entirely symptomatic and etiology-directed: chemotherapy for multiple myeloma in 34.14% (n = 14), conventional immunosuppressants or corticosteroids in 51.21% (n = 21), and antituberculosis therapy in 17.07% (n = 7). Dialysis was required for 14.63% (n = 6) of patients.

Regarding patient follow-up, 12 patients (29.27%) are currently active, 18 patients (43.90%) were lost to follow-up, 4 patients (9.76%) were transferred out, and 7 patients (17.07%) died. The causes of death were heart failure (n = 2), septic shock secondary to catheter-related infections (n = 2), acute respiratory failure (n = 1), and unknown causes (n = 2). The 36-month cumulative survival rate was 60% (Figure 3). In the exploratory multivariate analysis, only advanced age was statistically associated with mortality (p = 0.023), with a survival rate of approximately 75% for patients under 80 years of age, whereas all patients over 80 years died during the follow-up period. Gender, biochemical certainty sub-types, and organ involvement did not show a statistically significant association with survival within this exploratory framework.

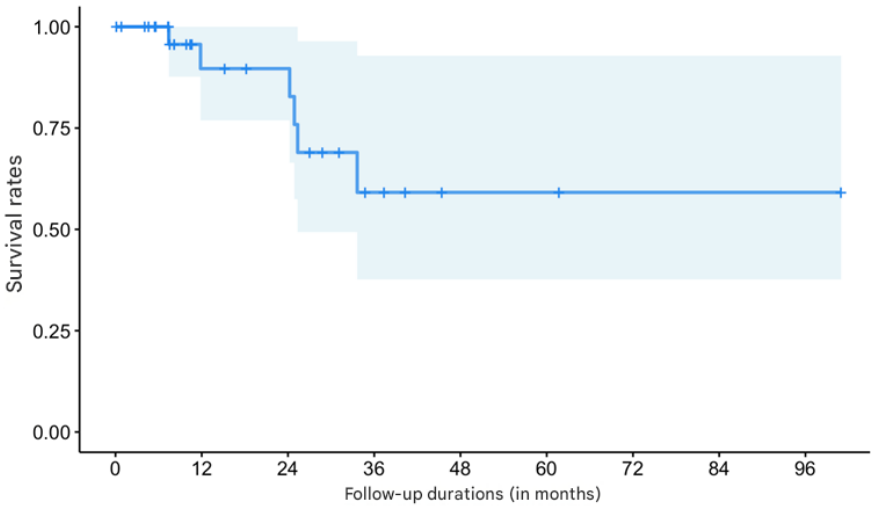

Figure 3. Overall patient follow-up curve.

4. Discussion

4.1. Methodological Justification and Classification Certainty

The primary strength of this study lies in its multi-centric design over a 34-year inclusion span, providing the largest series documented in Senegal. However, the lack of widespread immunophenotyping represents a significant real-world challenge, reflecting a structural limitation shared by many sub-Saharan tertiary centers. This is primarily due to the fact that immunohistochemistry was only introduced in 2021 at the National Public Pathology Center, combined with a frequent shortage of specific reagents, the lack of cryostats for immunofluorescence, and high out-of-pocket costs for patients [10]. To avoid diagnostic overinterpretation, our systematic reclassification into Confirmed, Probable, and Indeterminate sub-

types aligns with standard recommendations from the International Amyloidosis Nomenclature Committee [3] [11]. By formalizing these definitions, we minimize classification error risks while capitalizing on highly specific clinical phénotypes and etiological links to characterize our cohort.

4.2. Epidemiological Shifts and Gender Profiles

Globally, amyloidosis is reported to affect both sexes equally, though significant geographic variations exist. While several European and North American registries note a female predominance for AA amyloidosis and a male predominance for AL and ATTR forms [15] [16], reports from North Africa and India frequently describe an overall male predominance [17]-[19]. Our cohort demonstrates a female predominance **(M/F ratio 0.78)**, driven by the distribution of AA amyloidosis **(12 females vs 9 males)**. The mean age at diagnosis of 59.93 years is consistent with data from Brazil (57 years) [20] and France (63 years) [21], but significantly higher than historical series from Morocco (39 years) [17] and Tunisia (48 years) [18]. This shift reflects an increasing life expectancy and the rising diagnosis of multiple myeloma-associated AL forms in our aging population [22] [23].

4.3. Phenotypic Tropism and Etiological Drivers

Renal involvement was the predominant systemic manifestation **(75.61%),** presenting as a glomerular nephropathy syndrome with an impure nephrotic syndrome in 52% of renal cases. This high prevalence is consistent with literature indicating that renal tropism occurs in up to 90% of AA cases and 60% of AL cases [5] [9] [13]. Cardiac involvement, observed in 48.78% of our patients, manifested as a classic restrictive cardiomyopathy with septal hypertrophy, representing a major driver of mortality in AL and senile ATTR sub-types [14].

Etiologically, our findings reveal a co-dominance of neoplastic and chronic infectious/inflammatory diseases. While the incidence of AA amyloidosis has rapidly declined in Western nations due to highly effective cytokine-targeted therapies [24] [25], it remains highly prevalent in developing countries [26]. In our series, chronic infectious etiologies underlaid **all 21 AA cases**, with tuberculosis alone accounting for **17.07%** of the entire cohort. This matches historical reports from India and North Africa where infections remain the primary driver of secondary amyloidosis [17] [19] [27]. Conversely, all AL amyloidosis cases in our series were secondary to multiple myeloma, reflecting a distribution pattern that resembles high-resource country cohorts where hematological malignancies predominate [21] [28], complemented by an autoimmune and autoinflammatory profile typical of African cohorts [29].

4.4. Survival Bounded by Follow-Up Biais

Our cumulative 36-month survival rate of 60% must be interpreted with caution. The exceptionally high rate of patients lost to follow-up (43.90%) represents a common limitation in retrospective sub-Saharan chart reviews, caused by struc-

tural deficiencies in patient tracking systems, financial constraints, or patients returning to rural areas [10]. By treating lost patients as right-censored within our Kaplan-Meier analysis, we minimize structural survival underestimation. The exploratory finding that advanced age remains the sole variable statistically associated with mortality ($p = 0.023$) highlights the vulnerability of elderly patients facing un-typed and non-specifically treated systemic amyloidosis in tropical settings.

5. Conclusion

The incidence of amyloidosis in Senegal has been rising sharply over the past decade. While immunohistochemistry and immunofluorescence remain mostly inaccessible due to high costs and scarcity of reagents, the clinical phénotype combined with etiological screening allows for a reliable classification into Probable biochemical sub-types. AA amyloidosis secondary to chronic infectious diseases like tuberculosis remains a major public health reality in our setting, even as multiple myeloma-associated AL amyloidosis emerges among older subjects. Optimizing diagnostic pathways and increasing access to typed pathology are priority areas to reduce long-term mortality in West Africa.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Merlini, G. and Bellotti, V. (2003) Molecular Mechanisms of Amyloidosis. *New England Journal of Medicine*, **349**, 583-596. https://doi.org/10.1056/nejmra023144

[2] Gertz, M.A. and Kyle, R.A. (1991) Secondary Systemic Amyloidosis: Response and Survival in 64 Patients. *Medicine*, **70**, 246-256. https://doi.org/10.1097/00005792-199107000-00002

[3] Buxbaum, J.N., Dispenzieri, A., Eisenberg, D.S., Fändrich, M., Merlini, G., Saraiva, M.J.M., *et al.* (2022) Amyloid Nomenclature 2022: Update, Novel Proteins, and Recommendations by the International Society of Amyloidosis (ISA) Nomenclature Committee. *Amyloid*, **29**, 213-219. https://doi.org/10.1080/13506129.2022.2147636

[4] Nuvolone, M. and Merlini, G. (2017) Systemic Amyloidosis: Novel Therapies and Role of Biomarkers. *Nephrology Dialysis Transplantation*, **32**, 770-780. https://doi.org/10.1093/ndt/gfw305

[5] Stankovic, K. and Grateau, G. (2008) AA Amyloidosis. *Néphrologie & Thérapeutique*, **4**, 281-287. https://doi.org/10.1016/j.nephro.2008.02.002

[6] Jaccard, A., Desport, E., Mohty, D. and Bridoux, F. (2015) AL Amyloidosis. *La Revue de Médecine Interne*, **36**, 89-97. https://doi.org/10.1016/j.revmed.2014.08.003

[7] Koumou, G.C.G., Sinomono, D.T.E., Merzouk, S., Kabbali, N., Harmouch, T., Arrayhani, M., *et al.* (2019) Renal Amyloidosis in Nephrology. *Pan African Medical Journal*, **34**, Article 79. https://doi.org/10.11604/pamj.2019.34.79.8722

[8] Grateau, G., Valleix, S. and Callard, P. (2007) Multisystemic Amyloidosis in 2007. *La Revue de Médecine Interne*, **28**, 281-283. https://doi.org/10.1016/j.revmed.2007.01.005

[9] Bziz, A., Rouas, L., Lamalmi, N., Malihy, A., Cherradi, N., Ouzeddoun, N., *et al.* (2015) AA Renal Amyloidosis: Anatomopathological and Clinical Correlations in a Moroccan Series of 30 Cases. *Néphrologie & Thérapeutique*, **11**, 543-550. https://doi.org/10.1016/j.nephro.2015.06.007

[10] Diallo, S., Diouf, B., Ka, F., Niang, A., Pouye, A., Leye, A., *et al.* (2008) AL Amyloidosis in Black Patients: A Report on 4 Cases from Senegal. *Médecine d'Afrique Noire*, **55**, 471-477.

[11] Muchtar, E., Dispenzieri, A., Magen, H., Grogan, M., Buadi, F., Lacy, M.Q., *et al.* (2021) Systemic Amyloidosis from AL to ATTR: General Review on Contemporary Diagnostic Methods. *Blood*, **137**, 2933-2941.

[12] Fatihi, E., Zahiri, K., Hachim, K., Fadel, H., Benghanem, G.M., Sqalli, S., *et al.* (1999) Value of the Biopsy of Accessory Salivary Glands in Amyloidosis. *La Revue de Médecine Interne*, **20**, 759-761. https://doi.org/10.1016/s0248-8663(00)88681-6

[13] Baudin, B. (2013) Biochemical Markers of the Nephrotic Syndrome. *Revue Francophone des Laboratoires*, **2013**, 51-56. https://doi.org/10.1016/s1773-035x(13)72179-8

[14] Isabel, C., Georgin-Lavialle, S., Aouba, A., Delarue, R., Nochy, D., Karras, A., *et al.* (2013) Cardiac Amyloidosis: A Series of 14 Patients, Description, and Prognostic Factors. *La Revue de Médecine Interne*, **34**, 671-678. https://doi.org/10.1016/j.revmed.2013.05.003

[15] Georgin-Lavialle, S. and Grateau, G. (2024) Clinical Aspects of Systemic Amyloidosis in 2024. *Annales de Pathologie*, **44**, 407-413. https://doi.org/10.1016/j.annpat.2024.09.015

[16] Quock, T.P., Yan, T., Chang, E., Guthrie, S. and Broder, M.S. (2018) Epidemiology of AL Amyloidosis: A Real-World Study Using US Claims Data. *Blood Advances*, **2**, 1046-1053. https://doi.org/10.1182/bloodadvances.2018016402

[17] Saba, M., Tohmé, A., Abadjian, G., Haddad, F. and Ghayad, E. (2005) Multisystemic Amyloidosis: Clinical Study of 39 Patients in Lebanon. *La Presse Médicale*, **34**, 640-646. https://doi.org/10.1016/s0755-4982(05)84002-3

[18] Maïz, H.B., Moussa, F.B., Kaaroud, H., Abderrahim, E., Goucha, R., Abdallah, T.B., *et al.* (2001) Renal Amyloidosis: A Study of 420 Cases. *The Journal of Internal Medicine*, **249**, Article 21.

[19] Chugh, K.S., Datta, B.N., Singhal, P.C., Jain, S.K., Sakhuja, V. and Dash, S.C. (1981) Pattern of Renal Amyloidosis in Indian Patients. *Postgraduate Medical Journal*, **57**, 31-35. https://doi.org/10.1136/pgmj.57.663.31

[20] Fonseca, E.O. da, Filho, P.J.S., Silva, L.E. da and Caldas, M.L.R. (2015) Epidemiological, Clinical, and Laboratory Profile of Renal Amyloidosis: A 12-Year Retrospective Study of 37 Cases. *Journal of Nephropathology*, **4**, 7-12.

[21] Cazalets, C., Cador, B., Mauduit, N., Decaux, O., Ramée, M.-P., Le Pogamp, P., *et al.* (2003) Descriptive Epidemiology of Amyloidosis Cases Diagnosed at Rennes University Hospital from 1995 to 1999. *La Revue de Médecine Interne*, **24**, 424-430. https://doi.org/10.1016/s0248-8663(03)00136-x

[22] Villesuzanne, C. (2018) Epidemiological and Clinical Evolution of Immunoglobulin Light Chain (AL) Amyloidosis. Ph.D. Thesis, Université de Limoges.

[23] Abdelghani, K.B., Barbouch, S., Ounissi, M., Mahfoudhi, M., Moussa, F.B., Goucha, R., *et al.* (2012) Etiological Profile of Amyloidosis in Tunisia among the Elderly. *La Tunisie Médicale*, **90**, 431-436.

[24] Lachmann, H.J., Gillmore, J.D., Wechalekar, A.D., Gibbs, S.D., Pinney, J.H., Rowczenio,

D.M., *et al.* (2012) THU0379 a 20 Year Single Centre Experience of Aa Amyloidosis Demonstrating Changes in Its Epidemiology. *Annals of the Rheumatic Diseases*, **71**, 283-284. https://doi.org/10.1136/annrheumdis-2012-eular.2344

[25] Lane, T., Loeffler, J.M., Rowczenio, D.M., Gilbertson, J.A., Bybee, A., Russell, T.L., *et al.* (2013) Brief Report: AA Amyloidosis Complicating the Hereditary Periodic Fever Syndromes. *Arthritis & Rheumatism*, **65**, 1116-1121. https://doi.org/10.1002/art.37827

[26] Papa, R. and Lachmann, H.J. (2022) Secondary, Inflammatory AA Amyloidosis: International Update and Diagnostic Management. *Frontiers in Immunology*, **13**, Article No. 904351.

[27] Tuglular, S., Yalcinkaya, F., Paydas, S., Oner, A., Utas, C., Bozfakioglu, S., *et al.* (2002) A Retrospective Analysis for Aetiology and Clinical Findings of 287 Secondary Amyloidosis Cases in Türkiye. *Nephrology Dialysis Transplantation*, **17**, 2003-2005. https://doi.org/10.1093/ndt/17.11.2003

[28] Kyle, R.A., Larson, D.R., Kurtin, P.J., Kumar, S., Cerhan, J.R., Therneau, T.M., *et al.* (2019) Incidence of AL Amyloidosis in Olmsted County, Minnesota, 1990 through 2015. *Mayo Clinic Proceedings*, **94**, 465-471. https://doi.org/10.1016/j.mayocp.2018.08.041

[29] Genga, E., Oyoo, O. and Adebajo, A. (2018) Vasculitis in Africa. *Current Rheumatology Reports*, **20**, Article No. 4. https://doi.org/10.1007/s11926-018-0711-y