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Focal Segmental Glomerulosclerosis in Côte d'Ivoire: Epidemiological, Clinical and Pathological Aspects

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Abstract

Focal segmental glomerulosclerosis (FSGS) is characterized histologically by hyalinosis and sclerosis of glomeruli associated or not with podocyte involvement. The objective of our work was to clarify the epidemiological aspects and histological variants of FSGS in Côte d'Ivoire. **Materials and Methods:** This was a descriptive retrospective study, conducted from January 2015 to December 2019 using the renal biopsy registers (RB) of the Pathological Anatomy and Cytology departments of the Teaching Hospital of Cocody and Bouake in collaboration with the Nephrology Services of Côte d'Ivoire and the sub-region. The biopsies underwent conventional histopathology and/or immunofluorescence techniques. The parameters analyzed were: frequency, age, gender, proteinuria, biopsy indications and histological aspects and the different correlations between histological aspects and socio-demographic characteristics. **Results:** FSGS represented 58.1% (n = 104) of glomerular nephropathies. The average age of patients was 32.1 ± 13.3 years, with extremes of 13 and 70 years. The sex ratio was equal to 1. Nephrotic syndrome (68.9%), chronic renal failure (14.3%) and acute renal failure

(10.1%) were the main indications for renal biopsy (RB). The mean proteinuria at the time of diagnosis was 4 ± 3.7 g/24 h. It was massive (3.5 g/24 h) in 42.3% of patients. FSGS was primary in 29.8% (n = 31) and secondary in 70.2% (n = 73) of patients, of which 27.9% (n = 35) was due to HIV. According to the Columbia classification, 62.5% NOS type was found; 23.1% collapsing type; 7.7% tip lesion type; 4.8% cell type and 1.9% perihilary type. **Conclusion:** FSGS is a complex heterogeneous entity. It affects young people in our context with a homogeneous gender distribution. Understanding its histogenesis is essential for optimal patient management.

Keywords

Kidney, Glomerulus, Histology, FSGS, Côte d'Ivoire

1. Introduction

Focal segmental glomerulosclerosis (FSGS) is one of the leading causes of kidney failure worldwide. It is histologically characterized by hyalinosis and sclerosis of the glomeruli reaching only one sector of the glomerulus (segmental involvement) and/or only a certain percentage of the glomeruli (focal involvement); associated or not with podocyte involvement [1] [2]. The segmental lesion results in collapse of the glomerular basal membrane with a characteristic synechia between the parietal epithelium and the Bowman capsule [1]. Podocyte involvement is characterized by hypertrophy with erasure of the pedicels and sometimes severe involvement with ultrastructural abnormalities [3]. It is manifested by a very often severe nephrotic syndrome, greater resistance to treatment with corticosteroids and faster progression to end-stage renal failure [1]. FSGS is one of the main causes of nephrotic syndrome, accounting for 40% of cases in adults and 20% in children [4]. Initially, FSGS was classified as primitive (idiopathic) or secondary, and this distinction had therapeutic and prognostic implications [3]. Since 2004, the Columbia classification describes five histological variants: perihilar, vascular pole (tip lesion), cellular, collapsing and NOS (non-specific) [3] [5]. Accurate data on the incidence and prevalence of FSGS are difficult to obtain due to significant racial and geographic differences [4]. The increased incidence of FSGS was objectified in studies in North America and Europe. In Africa, the overall prevalence of FSGS is highly variable with no significant difference [6]. In Côte d'Ivoire, renal biopsy is practically at a beginning stage, the pathological aspects of this pathological entity are unknown. To better understand the prevalence and histopathological characteristics of hyalinosis.

2. Materials and Methods

It was a cross-sectional, descriptive and analytical study carried out in the Department of Pathological Anatomy and Cytology (PAC) of the Teaching Hospital of Cocody and Bouake in collaboration with the departments of Nephrology

and Pediatric Nephrology of Abidjan and Bouaké as well as those of the Teaching Hospital of Sylvanus Olympio (Togo), the Danka Hospital (Guinea Conakry), the TAMBA Clinic (Togo) and the Nura Clinic (Ouagadougou). Data were collected from January 2015 to December 2019 from the kidney biopsy registry. Two biopsy fragments were performed and routed separately. One was fixed in a vial containing Alcohol-Formalin-Acetic Acid (AFA), for study in optical microscopy (paraffin inclusion, microtome cut and haematoxylin eosin stain, Masson trichrome, Schiff's periodic acid and Jones' silver plating.). The other fragment was placed in a compress soaked in physiological serum and rapidly frozen in liquid nitrogen at −176˚C or fixed in liquid honey or Michel's liquid for direct immunofluorescence study. Serial sections of 5 microns were made with cryostat and spread on desylannised slides. Incubation of the slices was done using Dako's antihuman Rabbit polyclonal antibodies, directed against immunoglobulin chains (IgG, IgA, IgM), complement fractions (C3, C1q), heavy chains (α, μ, γ) and light chains (κ and λ), and fibrinogen. The observation was made with a fluorescence microscope equipped with a source of UV radiation and excitation filters. A dark room was used to avoid neutralization of Ag-Ac reactions by UV rays of natural light. The fluorescence was observed and classified according to the intensity of staining from 0 to 3. Included in the study were all patients with histologically diagnosed FSGS from renal biopsy with at least three non-sclerosed glomeruli with or without renal biopsy immunofluorescence. The parameters studied were epidemiological (frequency, age, gender, country of origin, occupation, history) clinico-biological (indications, proteinuria and RB techniques) and histopathological (RB topography, elementary lesions and histological variants, and immunofluorescence data). The data was entered from the Microsoft Excel software of Windows 10 (Microsoft Corporation, Redmond, WA, USA) and analysed in Epi-info 7.2. Chi-square and variance analysis were used to study the correlations of histological variants with the parameters studied. The results were considered statistically significant for a probability value $p < 0.05$.

3. Results

3.1. Socio-Demographic Data

Of the 179 renal biopsy cases examined over the study period, 104 patients had Focal segmental glomerulosclerosis (FSGS). This entity represented 58.1% of renal biopsies and 59.4% of glomerular nephropathy. The annual frequency was 20.8 cases/year. Samples were taken from Côte d'Ivoire (84.6%), Togo (6.7%), Burkina Faso (4.8%) and Guinea-Conakry (3.9%). Patients had an average age of 32.1 ± 13.3 years (13 and 70 years). Children under the age of 15 accounted for 6.8% (n = 6) and 2.9% of subjects were over the age of 60. There were as many men as women (sex-ratio = 1).

3.2. Clinico-Biological Characteristics

The indications of biopsy in our work were: nephrotic syndrome in 65.5% of pa-

tients (n = 78), followed by renal failure in 24.4% of patients (n = 29), rapidly progressive glomerulonephritis (RPGN) in 5% of patients (N = 6), hematuria in 3.4% of cases (N = 4) and nephritic syndrome in 1.7% of patients (N = 2). The mean proteinuria at the time of diagnosis was 4 ± 3.7 g/24 h with extremes of 0.2 g/24 h and 21.6 g/24 h. Proteinuria was nephrotic amplitude (≥3 g/24 h) in 50% of patients (N = 52).

3.3. Types of Variants and Correlations

The incidence of the NOS variant was 62.5% of the cases (N = 65) followed by the collapsing variant (26%), tubular pole (tip lesion) (5.8%) and hypercellular and perihilar variant with respectively 3.8% and 1.9%. Histological variants are represented in Figure 1. The statistical relationships between gender (p = 0.88) and different age groups (<15 years, p = 0.74; [16 - 59 years], p = 0.63; ≥60 years, p = 0.17) and histological variants were not significant. Nephrotic syndrome was most observed in NOS (80%), Tip (83.3%), and cellular (75%) variants compared to collapsing (59%) and perihilar (50%) variants. Renal failure was found in the cellular variants (50%), collapsing (44.4%) and Tip lesion (33.3%). No significant association was found between histological variants and biopsy indications rapidly progressive glomerulonephritis (p = 0.29); hematuria (p = 0.34); nephritic syndrome (p = 0.68). However, there was a significant association with nephrotic syndrome (p = 0.02) and kidney failure (p = 0.03).

The main indications of renal biopsies were nephrotic syndrome (65.5%), followed by renal failure (27.9%), rapidly progressive glomerulonephritis (RPGN) (5%), hematuria (3.4%) and nephritis syndrome in 1.7% of patients. Nephrotic syndrome was more common in NOS variants (80%), followed by Tip (83.3%),

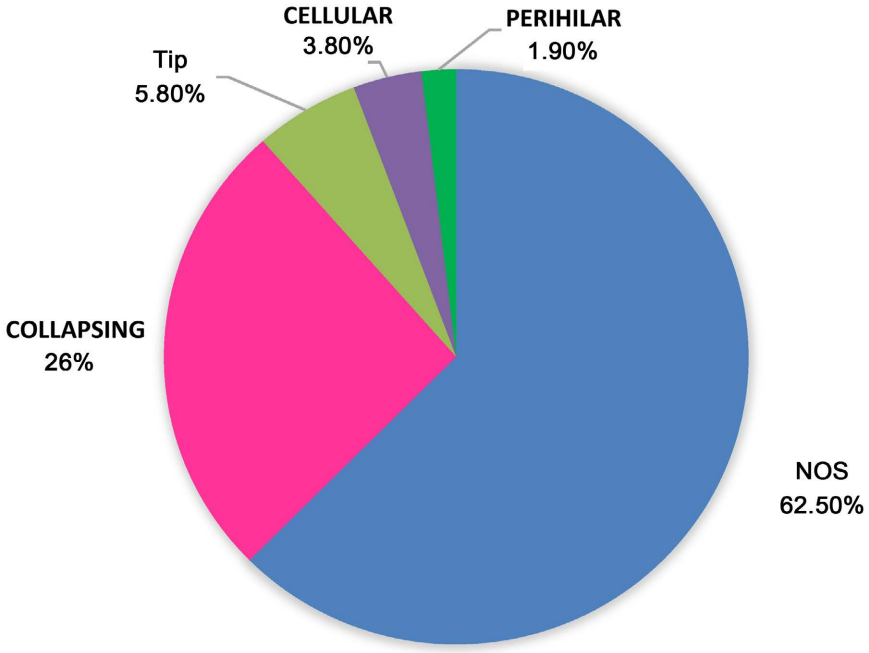

Figure 1. Distribution of histological variants.

and cellular (75%) compared to collapsing (59%) and perihilar (50%) variants. Renal failure was found in the cellular variants (50%), collapsing (44.4%) and Tip lesion (33.3%). Mean proteinuria was estimated at 4 ± 3.7 g/24 h with extremes of 0.2 g/24 h and 21.6 g/24 h. Proteinuria was nephrotic (≥3 g/24 h) in 50% (n = 52) of patients. No significant association was found between histological variants and biopsy indications (rapidly progressive glomerulonephritis (p = 0.29); hematuria (p = 0.34); nephritic syndrome (p = 0.68). However, nephrotic syndrome (p = 0.02) was associated with renal failure (p = 0.03).

3.4. Characteristics between Histological Variants and Elementary Lesions

The mean number of glomeruli per biopsy was 21.5 ± 17.3 and a median of 16.5 (extreme: 2 and 105). Glomerular sclerosis was found in 20.9% of cases and 79.1% of glomeruli were permeable. Interstitial fibrosis was present in 72.1%. Tubular atrophy accounted for 57.7% of cases. Fibrosis was moderate to severe in 21.1% (N = 21) and minimal in 51% (N = 53). However, 27.9% of patients (N = 29) had no interstitial fibrosis. A statistically significant association was found between histological variants and sclerosed glomeruli (p = 0.01) interstitial fibrosis (p = 0.01) and tubular atrophy (p = 0.01).

Among FSGS patients, 29.8% (N = 31) had primary FSGS and a secondary cause was identified in 70.2% (N = 73) of patients, of which 27.9% (N = 29) had HIV followed by genetic, hemodynamic and toxic causes in 18.3%, 16.3% and 7.7% of patients respectively. At immunofluorescence a mesangial deposition of IgM and C3 together was noted in 7.9% (N = 5) of cases, that of IgM, IgG and C3 together represents 4.8% (N = 3). However, isolated entrapment of IgM, IgA and IgG was observed in 20.6% (N = 13), 19.1% (N = 12) and 17.5% (N = 11) of subjects, respectively. It was associated with an isolated deposit of C3 (28.6%, N = 18) and C1q (4.8%, N = 3) and a deposit of the light chains Kappa and Lambda in respectively 4.8% (N = 3) and 1.6% (N = 1). Biopsy immunofluorescence showed no statistically significant differences with histological variants.

4. Discussion

This study examined the clinical and histopathological characteristics of the histological variants of 104 patients with focal segmental glomerulosclerosis (FSGS). This entity represents 58.1% of kidney biopsies. The work carried out by different authors around the world suggests a large variability in the proportion of FSGS from 2.5% to 57% [7] [8]. In Africa, although Lemrabott *et al*. had a relatively high prevalence (48.7%), the majority of authors reported prevalence as variable as in other regions of the world (0.7% to 51.9%) [6] [9]. The average age of the patients in our study is 32.1 ± 13.3 years. This is similar to Lemrabott and al (33.3 years). However, it is lower than the data for Ghali *et al*. (36.3 years) and Stokes (37.7 years). In contrast, Arias and al, as well as Swarnalatha and al, had relatively younger subjects aged 26 and 24.3 years respectively [5] [9] [10] [11]

[12]. There was no gender predominance in this series (sex-ratio = 1). Our data disagree with many studies that were either male-dominated [12] [13] or female-dominated [6]. The NOS variant is the most represented with 62.5% of cases. The proportion of other variants is relatively highly variable with low levels of FSGS Cellular-type (FSGS-CELL) and FSGS Perihilary-type (FSGS-PH) in 3.8% and 1.9% of patients, respectively. This trend was somewhat similar to that of some studies that objected to a predominance of FSGS-NOS with prevalences ranging from 38.7% to 89.1%; (**Table 1** presents the comparative prevalence of variants in different population series) [5] [11] [12] [14] and a prevalence of FSGS-CELL ranging from 0.7% to 3% and FSGS-PH ranging from 0% to 4.8% [5] [11] [14]. It is possible that this variability and difference in variants may be related to population characteristics or environmental factors. It should be noted that the other four variants may evolve into the NOS variant during the progression of disease and increasing chronicity [11]. In this study, the average age of patients with different FSGS variants is substantially the same (29.5 - 34.3 years). However, FSGS-PH subjects are younger with an average age of 20.5 years. The results of this series differ from those of Das and al and Swarnalatha and al which objectify a predominance of FSGS-NOS in young subjects and a high frequency of Collapsing-FSGS in the adult population [12] [13].

The indications of biopsy are preferentially nephrotic syndrome in 65.5% of patients followed by renal failure in 27.9% of patients, and hematuria in 3.4% of patients. These results are consistent with literature data [9]. The NOS, Tip, and cellular variants share common clinical features with nephrotic syndrome and hematuria more common than in collapsing and perihilar variants. Jeroen and al objectified a higher frequency of nephrotic syndrome in the Collapsing, Tip, and Cellular variants with lower frequencies in the Perihilary and NOS variants [5]. The average proteinuria in our study is 4 ± 3.7 g/24 h. Ghali *et al.* noted average proteinuria of 5.67 +/- 4.5 g/24 h. In this series, proteinuria is of nephrotic amplitude (≥3 g/24 h) in 50% of patients and it was in all variants of FSGS with the exception of the Cellular variant. This observation corroborates that of many series, with mean proteinuria of nephrotic amplitudes in all variants of FSGS [10] [13]. In contrast, Arias reported moderate non-nephrotic proteinuria in the

Table 1. Distribution of histological types according to sex.

Histological Variant of FSGS	Our study		Stokes MB (2014)	Swarnalatha G (2015)	Arias LF (2013)	Shakeel S (2014)	D'Agati (2011)
	N	(%)	(%)	(%)	(%)	(%)	(%)
NOS	**65**	**62.5%**	**38.7%**	**62.2%**	**77%**	**89.1%**	**68%**
Collapsing	27	26%	24.9%	4.3%	3.4%	8%	12%
Tip lesion	6	5.8%	26.7%	7.7%	13.7%	1.4%	10%
Cellular	4	3.8%	9.8%	9.4%	1%	0.7%	3%
Perihilar	2	1.9%	0%	11.2%	4.8%	0.7%	7%

perihilar variant [11]. Table 2 summarizes the distribution of epidemiological and clinical data according to the histological variants. The average number of glomeruli per biopsy is 21.5 ± 17.3 with a median of 16.5. This data agrees with that of Arias and al (16.7 ± 12.9; median: 14) [11]. Glomerular sclerosis is found in 20.9% of the included glomeruli and 79.1% of the glomeruli were permeable. The proportion of sclerotic glomerulus in our study is higher than that of Swarnalatha and al (17.2%), but remains much lower than the rate of glomerular sclerosis detected in the series of Das and al (54.1%) [12] [13]. The sclerosis is more severe in the perihilar variant, intermediate in the NOS and Collapsing variants and less severe in the lesion and cellular variants. As in our study, Joroen and *et al.* noted severe sclerosis in the perihilar variant, intermediate in the NOS variants and less severe in the Tip lesion variant. Interstitial fibrosis and tubular atrophy were common in our study with proportions of 72.1% and 57.7%, respectively. These results are consistent with those of Das and al who found high frequencies of fibrosis and tubular atrophy in 87.7% and 90.7% respectively [13]. Fibrosis is moderate to severe in 21.1% (N = 21) and Minimal in 51% of cases (N = 53). However, no interstitial fibrosis was objectified in 27.9% of patients (N = 29). Shakeel and al reported moderate to severe interstitial fibrosis in 14.4% of patients but 16% of these patients had no fibrosis [14]. Table 3 shows the distribution of histopathological data according to FSGS variants.

The proportion of idiopathic FSGS found in our study is 29.8%; despite its predominance as etiology of FSGS, it remains below the Eljouehari and *et al.*

Table 2. Distribution of epidemic-clinical data according to histological variants.

		FSGS-TOTAL			NOS		Collapsing		Tip		Cellular		PH	
		N	%	P	N	%	N	%	N	%	N	%	N	%
		104	**58.1**		**65**	**62.5**	**27**	**26**	**6**	**5.8**	**4**	**3.8**	**2**	**1.9**
Average age		32.1 ± 17.3			31.6 ± 23.7		34.3 ± 12.3		29.5 ± 14.2		34.5 ± 13.3		20.5 ± 6.4	
GENDER	**Female**	52	50		28	43.1%	17	63	3	50%	3	75	1	50
	Male	52	50	0.88	37	56.9	10	37	3	50%	1	25	1	50
≤15		6	5.8	0.74	5	7.7	-	-	1	16.7%	-	-	-	-
[16 - 59]		95	91.3	0.63	58	89.2	26	96.3	5	83.3%	4	100	2	100
≥60		3	2.9	0.17	2	3.1	1	3.7	-	-	-	-	-	-
Average proteinuria		4 ± 3.7			3.7 ± 2.7		4.6 ± 4.9		5.5 ± 7.9		2.8 ± 1.5		3.5 ± 2.4	
Proteinuria ≥3 g/24 h		52	50	0.06	33	50.8	13	48.1	3	50	2	50%	1	50
Nephrotic syndrome		78	65.5	**0.02**	52	80	16	59.3	5	83.3	3	75	2	100
Renal failure		29	24.4%	**0.03**	13	20%	12	44.4%	2	33.3%	2	50%	-	-
RPGN		6	5%	0.71	6	9.2%	-	-	-	-	-	-	-	-
Haematuria		4	3.4%	0.34	2	3.1%	-	-	-	-	2	50%	-	-
Nephritic syndrome		2	1.7%	0.68	2	3.1%	-	-	-	-	-	-	-	-

Table 3. Distribution of histopathological data according to the variants of FSGS.

	FSGS-TOTAL		NOS	Collapsing	Tip	Cellular	PH
	(%)	P	(%)	(%)	(%)	(%)	(%)
	58.1%		61.5%	26%	5.8%	4.8%	1.9%
Glomerulus average number	21.5 ± 17.3		22.7 ± 18.7	21.5 ± 15.9	16.2 ± 7.4	6.7 ± 2.6	27 ± 19.8
Sclerotic glomerulus	20.9%	<0.01	22.7%	15.9%	19.6%	22.2%	29.6%
Interstitial fibrosis	72.1%	<0.01	67.7%	81.5%	83.3%	50%	100%
Tubular atrophy	57.7%	<0.01	56.9%	55.6%	100%	50%	50%

data which noted 35% of idiopathic FSGS [15]. Primary FSGS is a distinct entity and paradoxically is better defined at present as an exclusion diagnosis after the elimination of any other cause of secondary FSGS. It has long been attributed to a presumed factor of permeability to circulation [16] [17]. Recent data suggest that FSGS associated with APOL1 may be another form of primary FSGS [17]. Among the secondary causes identified human immunodeficiency virus (HIV) reached 27.9% of FSGS (39.7% of secondary FSGS) followed by genetic, hemodynamic and toxic causes in 18.3%, 16.3% and 7.7% of cases, respectively. Viruses can act on the podocyte either by direct infection or by the release of inflammatory cytokines that interact with podocyte receptors. HIV-1 directly infects podocytes and tubular epithelial cells [18]. Other viral etiologies may exist but are infrequent, the case of cytomegalovirus infection (CMV) [19].

The genetic FSGS was associated with mutations in more than 20 genes coding for a range of molecules that appear critical for podocyte function [16] [17]. For hemodynamic or adaptive FSGS, it would result from a mismatch between the glomerular blood flow and the glomerular filtration surface, resulting in stress, detachment and podocyte loss [3] [20]. Drug-induced FSGS is associated with a short list of drugs, including those that act on the podocyte (pamidronate, interferon alpha) and those that damage the tubulo-interstitium (lithium, cyclosporine and tenofovir) [3] [17].

Immunofluorescence examination of biopsies was performed in 61% (N = 63) of patients and showed mesangial deposition of IgM and C3 together in 7.9% of cases, IgM, IgG and C3 together represent 4.8% however, isolated entrapment of IgM, IgA and IgG were observed in respectively 20.6%, 19.1% and 17.5% associated with an isolated deposit of C3 (28.6%) and C1q (4.8%) and a deposit of light chains (Kappa and Lambda) in respectively 4.8% and 1.6%. Compared to our results Shakeel found 41.1% IgM, 13% C3 with IgA and negative IgG [14].

5. Conclusion

FSGS is a complex heterogeneous entity. It affects young subjects in our context with a homogeneous gender distribution. Understanding its histogenesis is essential for optimal patient management. Because of the diagnostic difficulties in

our region, it would be necessary to establish a subsidy policy for biological balance sheets, essential for the performance of the biopsy, but also for the histological diagnosis.

Conflicts of Interest

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

References

[1] Jotterand, V., Moll, S., Martin, P.Y. and Saudan, P. (2008) Bisphosphonate-Induced Collapsing Focal Segmental Glomerulosclerosis; Two Clinical Cases and Literature Review. *Néphrologie & Thérapeutique*, **5**, 134-138.

[2] Han, M.-H. and Kim, Y.-J. (2016) Practical Application of Columbia Classification for Focal Segmental Glomerulosclerosis. *BioMed Research International*, **2016**, Article ID: 9375753. https://doi.org/10.1155/2016/9375753

[3] D'Agati, V.D., Kaskel, F.J. and Falk, R.J. (2011) Focal Segmental Glomerulosclerosis. *The New England Journal of Medicine*, **365**, 2398-2411. https://doi.org/10.1056/NEJMra1106556

[4] Sim, J.J., Batech, M., Hever, A., Harrison, T.N., Avelar, T., Kanter, M.H., *et al*. (2016) Distribution of Biopsy-Proven Presumed Primary Glomerulonephropathies in 2000-2011 among a Racially and Ethnically Diverse US Population. *American Journal of Kidney Diseases*, **68**, 533-544. https://doi.org/10.1053/j.ajkd.2016.03.416

[5] Stokes, M.B. and D'Agati, V.D. (2014) Morphologic Variants of Focal Egmental Glomerulosclerosis and Their Significance. *Advances in Chronic Kidney Disease*, **21**, 400-407. https://doi.org/10.1053/j.ackd.2014.02.010

[6] Okpechi, I.G., Ameh, O.I., Bello, A.K., Ronco, P., Swanepoel, C.R., *et al*. (2016) Epidemiology of Histologically Proven Glomerulonephritis in Africa: A Systematic Review and Meta-Analysis. *PLOS ONE*, **11**, e0152203. https://doi.org/10.1371/journal.pone.0152203

[7] Gargade, C.B., Khandekar, M.M. and Patil, S.N. (2016) Histomorphologic Analysis in Adult Nephrotic Syndrome: Changing Scenario. *Medical Journal of Dr. D.Y. Patil University*, **9**, Article No. 348. https://doi.org/10.4103/0975-2870.182506

[8] Kaba, M.L., Tolno, A., Bah, M.O., Diakité, F., Bah, A.O., Kourouma, M.L., *et al*. (2016) Lésions histologiques rénales au cours du syndrome néphrotique pur de L'adulte à Conakry. *Mali Médical*, **31**, 1-3.

[9] Lemrabott, A., Keita, I.A., Faye, M., Fall, K., Cissé, M.M., Mbengue, M., *et al*. (2018) Hypertension artérielle secondaire aux glomérulopathies chroniques primitives a Sénégal. *Néphrologie & Thérapeutique*, **14**, Article No. 345. https://doi.org/10.1016/j.nephro.2018.07.215

[10] Ghali, M., Aloui, S., Letaief, A., Hamouda, M., Skhiri, H., Frih, A., *et al*. (2015) A Multicenter Study on Profile of Hypertension in Focal Segmental Glomerulosclerosis in Tunisia. *Annales de Cardiologie et d'Angéiologie*, **64**, 187-191. https://doi.org/10.1016/j.ancard.2015.04.018

[11] Arias, L.F., Jiménez, C.A. and Arroyave, M.J. (2013) Histologic Variants of Primary Focal Segmental Glomerulosclerosis: Presentation and Outcome. *Brazilian Journal of Nephrology*, **35**, 112-119. https://doi.org/10.5935/0101-2800.20130019

[12] Swarnalatha, G., Ram, R., Ismal, K.M., Vali, S., Sahay, M., *et al*. (2015) Focal and

Segmental Glomerulosclerosis: Does Prognosis Vary with the Variants? *Saudi Journal of Kidney Diseases and Transplantation*, **26**, Article No. 173. https://doi.org/10.4103/1319-2442.148772

[13] Das, P., Sharma, A., Gupta, R., Agarwal, S.K., *et al*. (2012) Histomorphological Classification of Focal Segmental Glomerulosclerosis: A Critical Evaluation of Clinical, Histologic and Morphometric Features. *Saudi Journal of Kidney Diseases and Transplantation*, **23**, Article No. 1008. https://doi.org/10.4103/1319-2442.100883

[14] Shakeel, S., Mubarak, M. and Kazi, J.I. (2014) Frequency and Clinicopathological correlations of Histopathological Variants of Idiopathic Focal Segmental Glomerulosclerosis in Nephrotic Adolescents. *Indian Journal of Nephrology*, **24**, 148-153. https://doi.org/10.4103/0971-4065.132003

[15] Eljouehari, M., Flayou, K., Raoundi, O., Benamar, L., Rhou, H., Bayahia, R., *et al*. (2015) Profil anatomopathologique et évolutif de la hyalinose segmentaire et focale primitive idiopathique. *Néphrologie & Thérapeutique*, **11**, Article No. 379. https://doi.org/10.1016/j.nephro.2015.07.352

[16] Fogo, A.B. (2015) Causes and Pathogenesis of Focal Segmental Glomerulosclerosis. *Nature Reviews Nephrology*, **11**, 76-87. https://doi.org/10.1038/nrneph.2014.216

[17] Rosenberg, A.Z. and Kopp, J.B. (2017) Focal Segmental Glomerulosclerosis. *Clinical Journal of the American Society of Nephrology*, **12**, 502-517. https://doi.org/10.2215/CJN.05960616

[18] Bruggeman, L.A., Ross, M.D., Tanji, N., Cara, A., Dikman, S., Gordon, R.E., *et al*. (2000) Renal Epithelium is a Previously Unrecognized Site of HIV-1 Infection. *American Society of Nephrology*, **11**, 2079-2087. https://doi.org/10.1681/ASN.V11112079

[19] Grèze, C., Garrouste, C., Kemeny, J.L., Philipponnet, C., Aniort, J. and Heng, A.É. (2018) Hyalinose segmentaire et focale collapsante secondaire au cytomégalovirus: à propos d'un cas. *Néphrologie & Thérapeutique*, **14**, 50-53. https://doi.org/10.1016/j.nephro.2017.06.002

[20] Kriz, W., Lemley, K.V., Kriz, W. and Lemley, K.V. (2017) Défis mécaniques de la barrière de filtration glomérulaire: adaptations et voie vers la sclérose en plaques. *Pediatric Nephrology*, **32**, 405-417. https://doi.org/10.1007/s00467-016-3358-9