Scientific Research Publishing

Open Journal of Nephrology, 2023, 13, 174-194
https://www.scirp.org/journal/ojneph
ISSN Online: 2164-2869
ISSN Print: 2164-2842

Focal Segmantal Glomerulosclerosis: Epidemiological, Clinico-Biological, Pathological, Etiological, Therapeutic and Evolutionary Profiles in Dakar

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How to cite this paper: Gandzali Ngabe, P.E., Bonkano Baoua, D., Lengani, A.H.Y., Yattara, H., Kama Yatte, A., Loumingou, R., Tall, L., Ka, E.F., Niang, A. and Diouf, B. (2023) Focal Segmantal Glomerulosclerosis: Epidemiological, Clinico-Biological, Pathological, Etiological, Therapeutic and Evolutionary Profiles in Dakar. *Open Journal of Nephrology*, **13**, 174-194.
https://doi.org/10.4236/ojneph.2023.132017

Received: March 29, 2023
Accepted: June 27, 2023
Published: June 30, 2023

Abstract

Introduction: Focal Segmental Glomerulosclerosis (FSGS) corresponds to a clinicopathological syndrome, manifested by generally abundant proteinuria associated with hyaline deposits on part of certain glomeruli and sparing other glomeruli, with effacement of the pedicels. The general objective was to determine the prevalence of FSGS, and to give its profiles; epidemiological, clinical, biological, pathological, etiological, therapeutic and evolutionary of FSGS. **Materials and Methods:** This is a retrospective analytical study over a period of six years extending from January 1, 2010 to December 31, 2015 patients aged 16 or over who were hospitalized or received consultations during the study period for primary or secondary segmental and focal hyalinosis. Patients whose records were incomplete or unusable were not included in the study. **Results:** We have 16.54% with 158 cases of FSGS out of 6945 patients received and/or hospitalized. Of the 955 kidney biopsies distributed, the incidences of HSF were; 10.15%; 14.04%; 15%; 17.64%; 20.11%; 19.58% respectively in 2010; 2011; 2012; 2013; 2014 and 2015, *i.e.* an annual increase of around 1.25%. Renal-type edemas were found in 93.3%, the first reason for hospitalization. And ninety-six people had impaired kidney function, or 61%. The average of 24-hour proteinuria was 6.4 ± 3.69 g/24 hours. The extremes were 0.37 and 18.50 g/24h. Patients had nephrotic proteinuria in 84.86%. Non-specific FSGS or NOS (Not Other Specificities) was found in 62 cases or

39.24%, collapsing FSGS in 48 cases or 30.40%. FSGS with found causes was associated with fibrosis in 5/35 cases. Collapsing FSGSs followed by NOS FSGSs were the most corticosteroid-resistant. The evolution of the FSGS reveals that every 8 months the proteinuria decreases by half. **Conclusion:** Segmental and focal hyalinosis requires histological confirmation and the epidemiological, clinico-biological, etiological, therapeutic and evolutionary profiles depend on the histological (pathological) type. Other works on the risk factors for occurrence and the contribution of electron microscopy in the primary and secondary diagnosis of segmental and focal hyalinosis are desired.

Keywords

Proteinuria, Corticotherapy, Focal Segmantal Glomerulosclerosis, Senegal

1. Introduction

Focal Segmental Glomerulosclerosis (FSGS) type glomerulonephritis corresponds to a clinicopathologic syndrome, and is characterized by generally abundant proteinuria associated with hyaline deposits on part of certain glomeruli and sparing other glomeruli, with effacement of the pedicels. Hyaline, defined as a smooth glassy material, results from glomerular infiltration of plasma proteins [1] [2] [3]. The Columbia classification describes five variants or classes of FSGS: the FSGS of the urinary pole (tip lesion), the collapsing form, the cellular form, the perihilar or vascular form and the form with hypercellularity. FSGS is usually steroid-dependent or resistant, it is responsible for renal failure in most cases [1] [2] [3].

The diagnosis of clinically evoked FSGS requires histological confirmation, and it can be primary or secondary [1] [2] [3].

Segmental and focal hyalinosis is the first glomerular lesion in Africans [4] [5].

The clinical and biological complications are similar to those of the nephrotic syndrome.

The prevalence of FSGS was 52% in a series of renal biopsies over a period from 1993 to 2001 in Dakar [5]. At this time kidney biopsies were read in France. Since 2009, renal biopsy activity has intensified, and its reading is now done in Senegal by a pathologist specializing in renal pathology [5].

Based on this knowledge, this study was conducted with the following objectives:

- general:
  - To determine the prevalence of FSGS.
- specific:
  - To give the epidemiological, clinical, biological, pathological, therapeutic and evolutionary profiles of FSGS.

2. Patients and Methods

2.1. Study Framework

This work was carried out in the nephrology department of the Aristide Le Dan-

tec Hospital and the anatomopathology department of the Grand Yoff General Hospital in Dakar. These hospitals are level III public health establishments.

The relevant nephrology service is the main nephrology reference service in the country.

The service includes:

- A hospital sector with a capacity of 13 beds;
- A hemodialysis sector with a capacity of 14 posts;
- A peritoneal dialysis sector;
- Nephrology consultations take place three times a week (Monday, Thursday and Friday);
- Renal biopsy punctures are performed twice a week in the ultrasound room of the radiology department.

2.2. Type and Period of Study

This is a retrospective analytical study over a period of five years extending from January 1, ^{2010} to December 31, 2015.

2.3. Inclusion Criteria

Included were the patients aged 16 or over who were hospitalized or seen in consultation during the study period for histologically proven primary or secondary segmental and focal hyalinosis.

2.4. Non-Inclusion Criteria

Patients whose records were incomplete or unusable were not included in the study.

2.5. Data Collection

The analysis of the files was carried out using pre-printed sheets comprising various elements, also from the indication sheet for the renal biopsy puncture and their reports. Due to the retrospective nature, we did not need an ethics committee, received no funding and report no conflicts of interest.

2.6. Settings

For each patient included, the following parameters were studied:

- Socio-demographic: age, gender and occupation.
- Anamnestics:
  -Reasons for admission.
  -Medical and surgical history.
  -Lifestyle: taking phytotherapy, alcohol, tobacco.
- Clinical parameters which included functional signs, general signs and physical signs.
- Biological parameters in blood and urine.
- Radiological parameters:

Based on ultrasound of the urinary tract which assessed the size, structure, corticomedullary differentiation of the kidneys, the existence or not of pyelocaliciel dilation.

- Histological parameters:

For each patient included, the type of FSGS, the tubulo-interstitial and vascular repercussions and the results of the immunofluorescence study were collected.

- Therapeutic and evolutionary parameters:

The parameters can be: favorable; corticosensitivity, complete remission, partial remission.

Unfavorable: corticoresistance, corticodependence and complications related to the nephrotic syndrome and treatment, relapse and death.

2.7. Definitions of Operational Variables

Corticosensitivity: Normalization of proteinuria after one month.

Complete remission: Decrease in proteinuria below 0.3 g/24h.

Partial remission: Drop in proteinuria to 50%, below 3 g per 24 hours

Corticoresistance: Persistence of proteinuria after four months of well-conducted corticosteroid therapy.

Steroid dependence: Reappearance of proteinuria after decreasing the dose of corticosteroid therapy.

Relapse: Reappearance of proteinuria above 3 g per 24 hours, 3 months after stopping corticosteroid therapy.

HBP: systolic arterial pressure greater than 120 mmhg and/or diastolic pressure greater than 90mmhg.

2.8. Statistical Analyzes

Data entry was carried out using Excel year 2013 and EPI Infos Version 3 software.

Descriptive statistics were presented in the form of diagrams, tables and graphs. Quantitative variables were summarized on average with standard deviation (SD) and extremes. Qualitative variables were presented as proportions with percentages.

The comparison between the groups (the two main types of FSGS) was made on the basis of anthropometric, biological and echographic parameters requiring the Chi square test for the percentages and the Student test for the means of the quantitative variables.

A value of $p < 0.05$ was considered statistically significant, and $p < 0.01$ very significant.

3. Results

3.1. Prevalence and Incidence

The number of segmental and focal hyalinosis listed was 158 cases out of 6945

patients received and/or hospitalized. Of the 955 kidney biopsies performed, they are distributed as follows: 138, 114, 160, 170, 179, 194, respectively in 2010, 2011, 2012, 2013, 2014 and 2015, the prevalence was 16.54%

For the annual incidences see Table 1 below. That is an annual increase of approximately 1.25%.

Age

The mean age of the patients was 33.07 ± 13.8 years with extremes of 16 and 81 years and the median of 28 years.

Figure 1 below is the distribution of patients by 10-year age group.

3.2. Genre

There were 114 men or 72.2% and 44 women or 27.8% or a sex ratio of 2.59.

3.3. Personal History

Arterial hypertension was the most found antecedent for the rest, see Table 2 below.

Table 1. Incidence of FSGS.

Year	Number	Percentage %
2010	14	10.15
2011	16	14.04
2012	24	15
2013	30	17.64
2014	36	20.11
2015	38	19.58

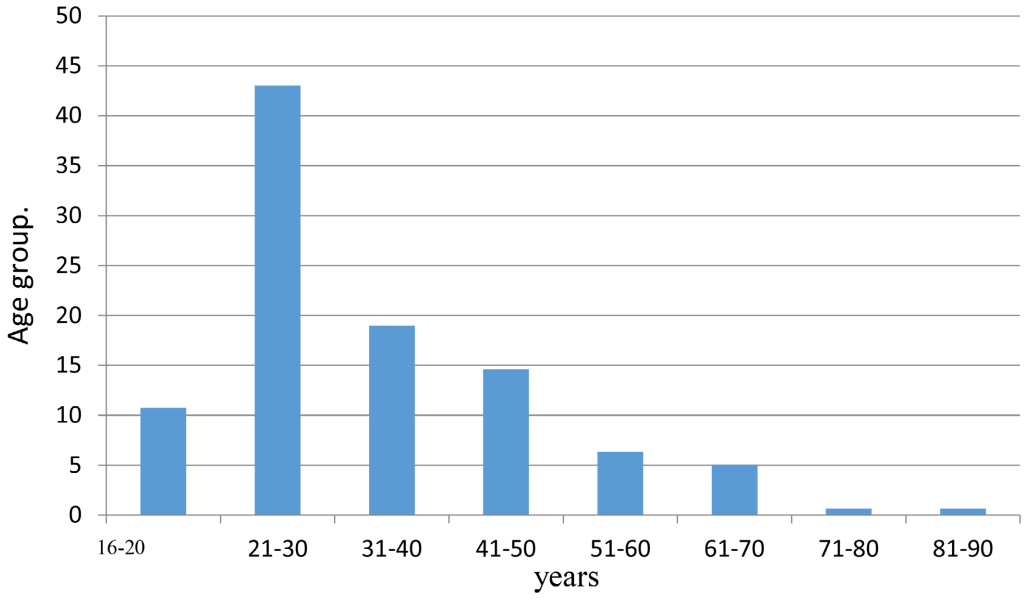

Figure 1. Distribution of patients by 10-year age group.

3.4. Family History

Among the 158 patients collected, no family history of HSF was found.

3.5. The Lifestyle

The risk factors found in lifestyle are shown below (Table 3).

3.6. Clinics

3.6.1. Reasons for Consultation

Renal-type edema was the most common reason.

Here are the other reasons for consultation (Table 4).

Table 2. Distribution of patients according to medical history.

Medical background	Frequency	Percentage
Hypertension	25	15.82
Edema	19	12.02
Infections	7	4.43
Diabetes	4	2.53
Sickle cell disease	4	2.53
Systemic lupus erythematosus	2	1.26
Eczema	1	0.63
Rheumatoid purpura	1	0.63

Table 3. Representation according to lifestyle.

Way of life	Frequency	Percentage (%)
Phytotherapy	81	51.27
Without risk factors	59	37.36
Not specified	10	6.32
Tobacco	5	3.16
Alcohol	2	1.26
Heroin	1	0.63
Total	158	100

Table 4. Representation according to the reasons for consultation.

Reasons for hospitalization	Frequency	Percentage
Joint deformities + pruritus	1	0.95
Long term fever	2	1.9
Hypertension	2	1.9
Macroadenopathies + follicular papules	1	0.95
Kidney type edema	98	93.35
Dirty broth urine	1	0.95

3.6.2. Weight

Sixty-one patients, or 38.60%, had their weight reported in the files. The average weight was 70.95 ± 10.79 kg.

3.6.3. Diuresis

The mean diuresis was 1550 ± 569.97 ml/24h with extremes of 0 to 3000 ml/24h. And the patients had normal diuresis in 74.1%. The diuresis parameters are shown below.

3.6.4. Arterial Pressure

Patients had normal blood pressure in 51.9%. The average SBP was 130.5 ± 8.5 mm hg and that of the DBP was 77.27 ± 9 mm hg. Eighty-nine patients had hypertension, *i.e.* 56.32%, see Table 5 below.

3.7. Paraclinics in the Blood

3.7.1. Hemoglobin

The average hemoglobin was 11.05 ± 2.63 g/dl and the extremes 5.1 g/dl and 17 g/dl. Fifty-eight patients were anemic, *i.e.* 36.7%.

3.7.2. Albuminemia

Mean albuminemia was 19.09 ± 9.14 g/l with extremes of 2 and 47 g/l.

Eighty-one people had hypoalbuminemia, *i.e.* 51.26%.

3.7.3. Protidemia

The mean protein level was 50.84 ± 11.94 g/l with extremes of 30 and 83 g/l.

Seventy-five people had hypoprotidemia, *i.e.* 47.46%.

3.7.4. Urea

The average urea was 0.70 ± 0.83 g/l. And the extremes of 0.11 and 4.93 g/l. 49 people had hyperuremia, *i.e.* 31.01%.

3.7.5. Creatininemia

Mean serum creatinine was 33.74 ± 51.69 mg/l, and extremes from 4 to 394 mg/l. And ninety-six people had impaired kidney function, or 61%. And Figure 2 below represents a distribution of patients by stage of kidney disease.

Table 5. Representation according to blood pressure.

YOUR	Frequency	Percentage
Normal hypertension	82	51.9
Hypertension grade 1	41	25.97
Hypertension grade 2	3	1.9
Hypertension grade 3	1	0.63
Not specified	31	19.6
Total	158	100

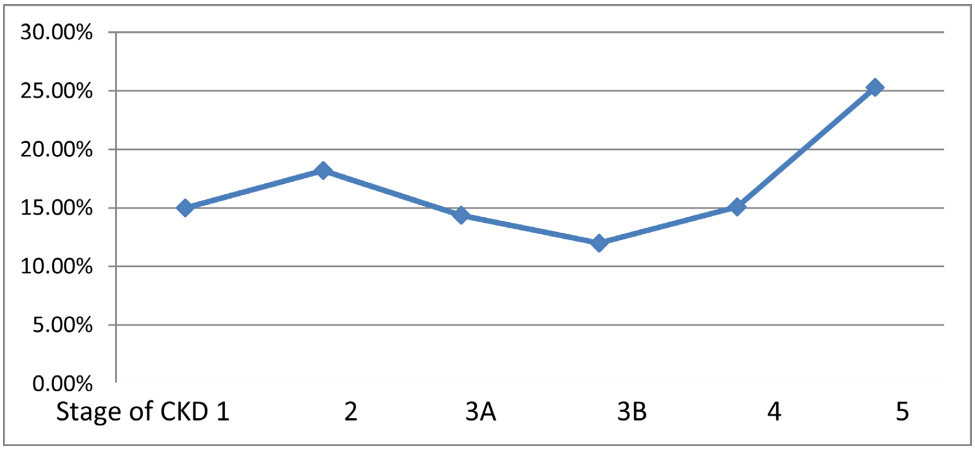

Figure 2. Distribution of patients by stage of renal disease according to CKD-EPI.

3.7.6. Calcemia

Mean serum calcium was 80 ± 13.06 mg/l with extremes of 60 and 95 mg/l. Ten people had dosed serum calcium, *i.e.* 6.32%.

3.7.7. HDL Cholesterol

The mean HDL cholesterol was 0.37 ±0.13 g/l with extremes of 0.21 and 0.57 g/l.

3.7.8. LDL Cholesterol

The average LDL cholesterol was 3.02 ± 1.5 g/l with extremes of 0.83 and 5.04 g/l.

3.7.9. Serologies

Fifty-three patients or 33.54% had retroviral serology among which one patient or 1.88% had positive serology and the other patients (52) or 98.11% had negative serology.

Forty-seven patients or 29.74% had done the AgHbs and 4 patients or 8.51% or had the AgHbs positive.

Thirty-seven patients or 23.41% had anti-HCV Ab and one patient or 3.7 had positive anti-HCV Ab.

3.8. In the Urine

3.8.1. Proteinuria

The average of 24-hour proteinuria (PU/24 hours) was 6.4 ± 3.69 g/24 hours, and the extreme was from 0.37 to 18.50 g/24h. The median was 5.9 g/24h. Patients had nephrotic proteinuria in 84.86%.

Proteinuria according to slices is shown below (**Table 6**).

3.8.2. Public Housing

Forty-one patients or 25.94% underwent HLM, among which 10 patients or 24.39% had hematuria and 18 patients or 43.9% had leukocyturia.

3.8.3. Ultrasound

Kidney size

In 88.74% of cases, kidney size was normal.

The following is the representation according to the size of the kidneys of the patients (Table 7).

3.8.4. Histological

Histological types

Non-specific FSGS or NOS (Not Other Specificities) was found in 62 cases or 39.24%, collapsing FSGS in 48 cases or 30.40%, 6 cases of tubular TIP FSGS or 3.79%, 5 cases of cellular FSGS *i.e.* 3.16% and 2 cases of vascular or hilar FSGS *i.e.* 3.16%.

Figures 3-7 below represent the histological types of FSGS.

Table 6. Representation according to 24-hour proteinuria.

PU/24H	Frequency	Percentage
Less than 3	18	11.39
3 to 9	75	47.46
10 and more	26	16.45
Not specified	39	24.70
Total	158	100

Table 7. Representation according to the size of the kidneys.

Ultrasound	Frequency	Percentage
Atrophy	15	9.49
Big size	4	2.53
Normal	134	84.81
Not specified	5	3.16
Total	158	100

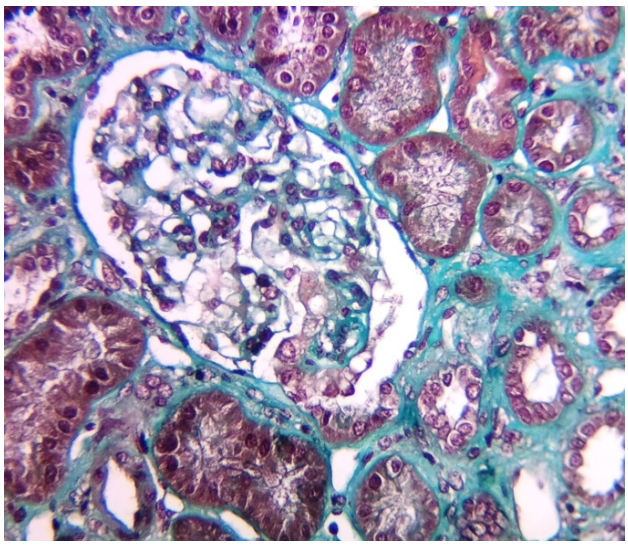

Figure 3. TIP FSGS Trichrome de Masson Gros × 250 (Lesion adhering to Bowman's capsule at the start of the proximal tube).

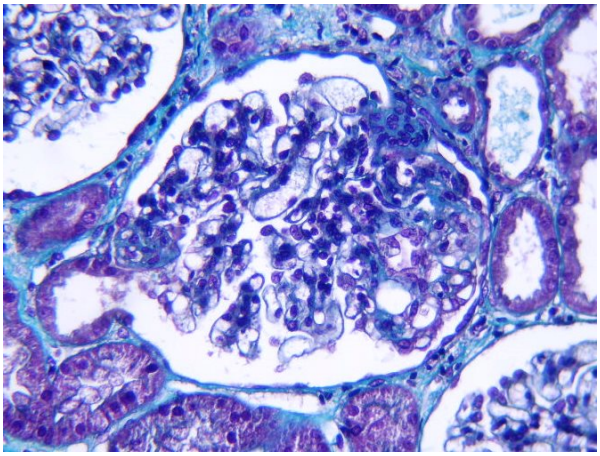

Figure 4. VASCULAR FSGS Trichrome de Masson Gros × 250 (Lesion adherent to Bowman's capsule at the vascular level)

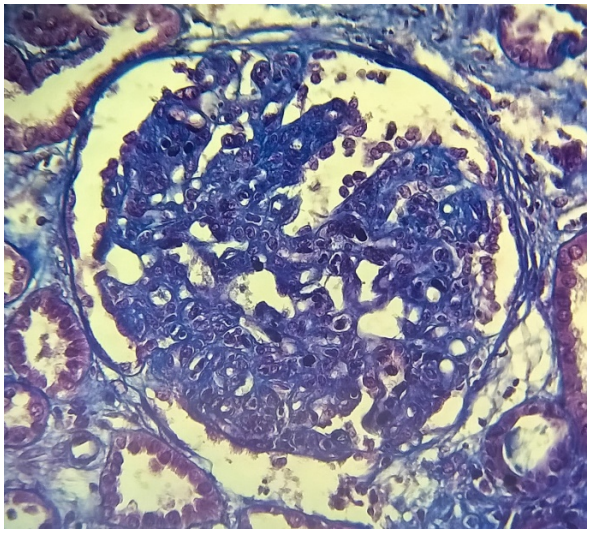

Figure 5. FSGS NOS Trichrome de Masson Gros × 250 (Capillary Wall Retraction with Podocyte Hyperplasia)

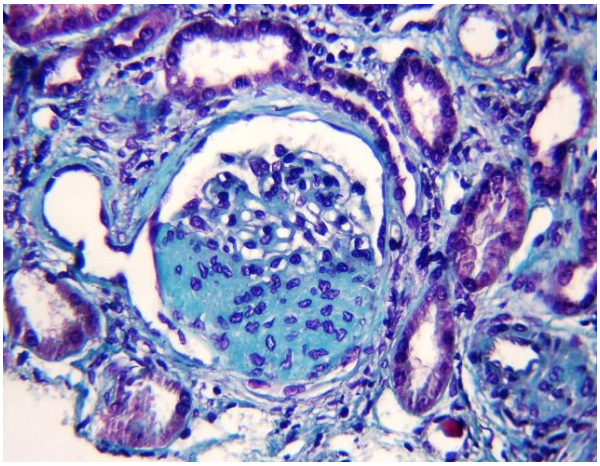

Figure 6. COLLAPSANTE FSGS Trichrome de Masson Gros × 250 (collapsed hyaline deposits of half of the capillaries).

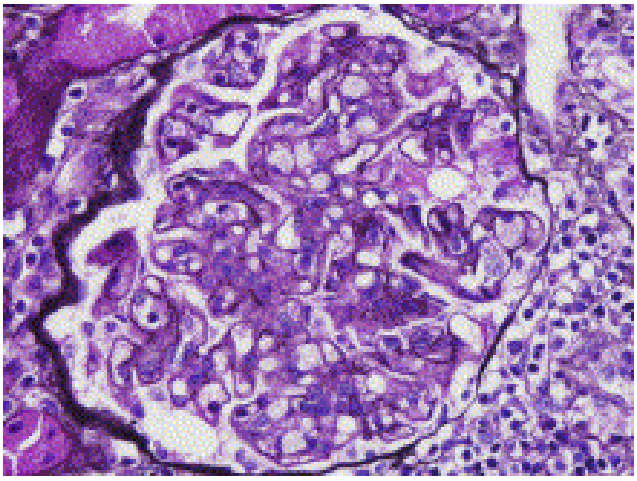

Figure 7. HYPERCELLULAR FSGS Trichrome de Masson Gros × 250 (endocapillary and extracapillary hypercellularity)

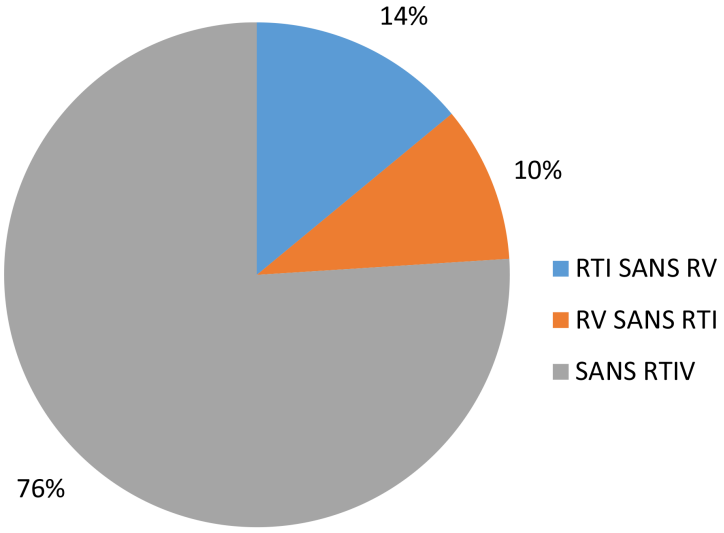

Figure 18. Percentage of FSGS according to tubulointerstitial and vascular repercussions.

Tubulointerstitial and vascular repercussions.

Patients had no tubulointerstitial and vascular repercussions in 76% of cases.

Figure 8 represents the percentage of tubulointerstitial and vascular impact.

3.8.5. Representation According to Vascular Lesions

Specifically, the types of vascular repercussions have been grouped together in Table 8.

3.8.6. Immunofluorescence

Immunofluorescence was performed in 13 patients confers Table 9.

3.8.7. Etiologies

Among the 158 cases of FSGS collected, 123 cases were primary (77.17%) and 35 cases had an etiology, 22.83%. See Table 10 of causes.

Table 8. Representation according to the percentages of vascular lesions.

Vascular lesions	Number	Percentage
Absent	45	35.44
Present non-specific	69	54.34
MAST	1	0.78
Malignant SIN	2	1.57
Benign NAS	10	7.87
Total	158	100

Table 9. Representation according to the percentage of immunofluorescence

Immunofluorescence	Frequency	percentage
Not made	145	91.76
Made without deposits	7	4.43
Made with non-specific deposits	6	3.79
Total	158	100

Table 10. Etiologies of FSGS.

Causes	Workforce	Percentage
HIV	7	39
Sickle cell disease	4	22.2
Diabetes	3	16.7
Lupus	2	11.1
PR	1	5.5
Heroin	1	5.5
Total	18	100

3.9. Treatment and Evolution

3.9.1. Treatment

- Lifestyle and dietary measure

  All the patients benefited from lifestyle and dietary measures.

- Nephroprotective treatment

  Of the 158 patients, 133 patients or 84.17% received nephroprotective treatment with IEC and ARA2.

- Diuretics

  Of the 158 patients, ninety patients or 56.9% were treated with diuretics. Eighty-two patients or 91.1% were treated with loop diuretics alone, then 4 patients or 4.4% were treated with dual diuretics; loop diuretic plus either a potassium-sparing diuretic or a thiazide diuretic.

- Etiological treatment.

  For patients with secondary HSF, the treatment was etiological.

For primary FSGS out of the 158 patients collected for FSGS, 103 received immunosuppressive treatment, *i.e.* 65.18%. Of these 103 patients who received treatment with immunosuppressants; 95 patients received corticosteroid therapy alone, *i.e.* 92.23%, 7 patients, *i.e.* 6.8% due to corticosteroid dependence or corticoresistance, received corticosteroid therapy combined with another immunosuppressant. This immunosuppressive treatment used was either azathioprine in 4.8% (5 patients), or cyclophosphamide in 2% (2 patients). One patient, or 0.97%, had received the 3 immunosuppressants (cyclophosphamide, azathioprine, mycophenolate mofetil) during follow-up at different times (See Table 11).

Corticosteroid therapy was prescribed at a dose of 1 mg/kg/day for an average of 2.8 months in all patients.

Azathioprine was prescribed at a dose of 50 mg twice daily for an average of 11.6 months.

Mycophenolate mofetil was prescribed at a dose of 1 g twice a day for an average of 6 months and cyclophosphamide was prescribed at a dose of 500 mg per square meter for an average of 6 months.

3.9.2. Evolution

The evolution under treatment for twelve months was marked by 12 cases or 7.59% of corticoresistance, 15 cases or 9.49% of corticosensitivity and 4 cases or 2.53% of corticodependence.

Nineteen patients put on hemodialysis and peritoneal dialysis.

The complications found were: one case of corticosteroid-induced diabetes, one case of femoral neck fracture, 4 cases of urinary infections, 3 cases of skin infections and 1 case of digestive infection.

3.10. Analytical results

3.10.1. Correlation between FSGS and History and Clinic

The P-Value was significant for sex, age and hypertension.

The anamnestic and clinical data correlated to the types of FSGS are shown below (Table 12).

3.10.2. Correlation between FSGS and Biology

The P-Value was significant for serum creatinine and 24-hour proteinuria. See Table 13.

Table 11. Immunosuppressants used.

Molecules	Effective	Percentage
Corticosteroids	95	92.23
Azathioprine	7	4.85
Cyclophosphamide	2	1.94
MMF	1	0.97

3.10.3. Correlation between FSGS and Renal Ultrasound and Associated Histological Lesions

P-values were significant for dedifferentiation, vascular lesions, and tubulointerstitial lesions. The sonographic and histological data correlated to the types of FSGS are shown below (Table 14).

3.10.4. Correlation between HSF Type and Evolutionary Modalities

P-values were not significant for all parameters. See Table 15 below.

Table 12. Segmental and focal hyalinosis correlated with age, sex, herbal medicine, hypertension and renal-type oedemas.

Settings	Total cohort (N = 158)	Types of Primary Segmental and Focal Hyalinosis (N = 123)					Secondary HSF N = 35	P-value
		Cellular N = 5	Vascular N = 2	Collapsing N = 48	Not specific N = 62	Tubular N = 6		
Age	33.08 ± 13.8 (16 - 81)	27.4 ± 9.2 (17 - 38)	20.5 ± 6.36 (16 - 25)	30.8 ± 12.1 (16 - 64)	32.04 ± 13.23 (16 - 68)	23 ± 6.3 (20 - 27)	41.14 ± 0 (22 - 81)	0.0020
Sex % (M/F)	72.2/27.8	100/0	50 /50	77.1/22.9	72.6/27.4	83.3/16.7	60/40	0.0020
Phytotherapy %	56.6	80	50	52.08	48.38	66.66	48.57	0.6
hypertension %	25	0	50	8.33	24.19	0	42.85	0.008
Renal type oedemas.	78.48	60	100	81.25	82.25	100	62.85	1.4

Table 13. FSGS correlated with biological abnormalities.

Settings	**Total cohort (N = 158)**	**Types of Primary Segmental and Focal Hyalinosis (N = 123)**					**Secondary HSF N = 35**	**P-value**
		Cellular N = 5	**Vascular N = 2**	**Collapsing N = 48**	**Not specific N = 62**	**Tubular N = 6**		
Hemoglobin (g/l)	11.05 ± 2.6	12.1 ± 2.3	13.1 ± 0.21	11.2 ± 2.3	11.8 ± 2.5	12.6 ± 3.1	9.1 ± 2.4	0.13
Serum creatinine (mg/l)	33.7 ± 51.6	13.3 ± 6.2	10 ± 4.2	26.58 ± 23.25	19.8 ± 14.9	10.8 ± 7.1	72.4 ± 92.2	0.020
Albuminemia (g/l)	19.09 ± 9.14	16.3 ± 14.5	20.3 ± 0	17.03 ± 7.7	17.4 ± 6.5	27.3 ± 14.3	23.7 ± 10.4	0.11
24 hour proteinuria	6.4 ± 3.6	4.5 ± 2.2	5.6 ± 0.4	7.6 ± 4.1	5.8 ± 3.3	5.3 ± 2.1	6.1 ± 3.54	0.02

Table 14. Segmental and focal hyalinosis correlated with ultrasound and basic histological damage.

Settings	**Total cohort (N = 158)**	**Types of Primary Segmental and Focal Hyalinosis (N = 123)**					**Secondary HSF N = 35**	**P-value**
		Cellular N = 5	**Vascular N = 2**	**Collapsing N = 48**	**Not specific N = 62**	**Tubular N = 6**		
Poor differentiation (%)	18.98	0	50	10.41	14.51	0	42.85	0.014
Glomerular lesions greater than and equal to 50%	25.94	40	50	25	20.96	0	37.14	0.9
Vascular lesions (%)	66.45	80	0	62.5	56.45	33.33	97.14	0.006
Lesions Tubulointerstitial (%)	67.72	100	0	66.66	56.45	16.66	97.14	0.014
Immunofluorescence (%)	8.86	25	0	8.33	6.45	16.66	11.42	0.7

Table 15. Segmental and focal hyalinosis correlated with evolution.

Settings	Total cohort (N = 158)	Types of Primary Segmental and Focal Hyalinosis (N = 123)					Secondary HSF N = 35	P-value
		Cellular N = 5	Vascular N = 2	Collapsing N = 48	Not specific N = 62	Tubular N = 6		
Complete remission (%)	6.96	0	0	2.08	11.29	50	0	0.15
Partial remission (%)	6.96	0	0	8.33	8.06	0	5.71	0.17
Steroid sensitive (%)	10.75	0	0	10.41	11.29	33.33	8.57	0.9
Steroid dependence (%)	3.16	0	0	4.16	4.83	0	0	0.8
Corticoresistance (%)	8.22	0	0	12.5	8.02	0	5.71	0.6

4. Discussion

This work had limitations among which; the lack of biological analyses, the high number of patients lost to follow-up and the difficulty of evaluating the evolution of patients. On the one hand, the fact of not intervening in the patients studied gives a real insight into the management of patients.

4.1. Prevalence and Incidence

Epidemiologically, several publications suggest that the incidence of idiopathic HSF in adults is increasing, as is the case with Davison AM *et al.* [6].

HSF was found in 2.5% - 4% of kidney biopsies in the 1970s, but in that same Chicago population a decade later it was estimated to be 12.2% - 18.7% making it the lesion no longer diagnosed based on biopsies, FSGS increased by 4.0% ± 0.6% [7]. In our work the incidence of FSGS was; 10.15%; 14.04%; 15%; 17.64%; 20.11%; 19.58% respectively in 2010; 2011; 2012; 2013; 2014 and 2015, *i.e.* an annual increase of around 1.25%.

The similarity of the prevalences was found in several studies. See Table 16.

The prevalence of 16.54% in our study was close to Haas M *et al.* in USA [8], Briganti EM *et al.* in Australia [9], and Narasimhan B *et al.* in India [10]. This is because their cohorts are retrospective.

4.2. Age

The average age was 33.07 ± 13.8 years. These results were similar to those of Van Rensburg BWJ *et al.* in South Africa who were 39.6 ± 15 years old although subjects were recruited as young as 13 years old [3].

The oldest patients were found among the non-specific FSGS cases 32.04 ± 13.23 years for the primary types but the secondary HSFs were even older 41.14 ± 0 years.

4.3. Sex

The sex ratio was 2.59 with 114 men for 44 women. In many studies, such as Van Renburg BWJ *et al.*, and GANI TD *et al.*, men were the most affected. [3] [11].

Table 16. Prevalence of FSGS in the series of the literature.

Authors and countries	Prevalence
Haas M *et al.* in USA [8]	18.7%
Briganti EM *et al.* in Australia [9]	16.95%
Narasimhan B *et al.* in India [10]	16.95%
Our study	16.54%

4.4. Antecedents

Among the medical history, hypertension was observed first with 15.82% followed by edema at 12.02%. It was identical to Van Rensburg BWJ *et al.* in South Africa and Narasimhan B *et al.* in India [3] [11].

The majority risk lifestyle found was herbal medicine in 51.26%. The study by GANI TD *et al.* [12] found that phytotherapy would be a cause of HSF in 36.94% of patients.

4.5. Clinics

4.5.1. Renal Type Edemas

The first reason for hospitalization found was edema, *i.e.* 93.3%, this was identical to the work of Van Rensburg BWJ *et al.* in South Africa and Narasimhan B *et al.* in India [3] [10].

4.5.2. Diuresis and Blood Pressure

In 82.91% of cases the diuresis was normal. This is corroborated by the work of Haas *et al.*, Weiss *et al.*, and Detwiler *et al.* [8] [12] [13] in which diuresis was normal in 80% of cases.

And 56.1% of patients had normal blood pressure either less than or equal to 120/80mmhg.

However 8.3% of patients with collapsing HSF had hypertension. The P-Value was 0.008 or $p < 0.01$ or significant. This was corroborated with the work of Haas *et al.* [8], Weiss *et al.*, and Detwiler *et al.* [12] [13].

4.6. Paraclinical

Biology

Serum creatinine

In our work (**Table 17**), the mean for serum creatinine was 33.74 ± 51.69 mg/l, and the extremes of 5.17 and 394 mg/l.

In collapsing FSGS it was superior to cases of FSGS NOS. There were no statistically significant differences. This was identical to the work of Haas *et al.* [8] and Detwiler *et al.* [14].

Sixty-two percent of patients with FSGS described by Detwiler *et al.* [14] and by Weiss *et al.* [12] were more likely to have impaired kidney function, which manifests as elevated serum creatinine. This joins our series in which sixty one percent had altered serum creatinine.

Table 17. Mean serum creatinine in the literature FSGS collapsing versus FSGS NOS.

Authors	collapsing FSGS	NOS FSGS
Detwiler *et al.* USA. [14]	(35 ± 34 mg/l)	13 ± 6 mg/l
Haas *et al.* USA. [15]	38 ± 27 mg/l	19 ± 15 mg/l
Our study	33.74 ± 51.69 mg/l	19.8 ± 14.9 mg/l

Table 18. Mean PU of collapsing FSGS versus NOS FSGS

Authors	collapsing FSGS	NOS FSGS
Haas *et al.* [8]	10 g/24h	4.4 g/24h
Our study	7.6 g/24 hours	5.8 g/24h

This would be a consequence of more collapsed and sclerotic glomeruli than in other forms of FSGS.

24-hour proteinuria (PU/24 hours)

The average PU/24 hours was 6.14 g/24 hours ± 3.69 g/24 hours. The extremes were 0.37 and 18.50.

The 2 cases of vascular FSGS had proteinuria between 3 and 10 g/24 hours, *i.e.* 100% nephrotic PU.

Just as 100% of patients with FSGS TIP, *i.e.* 7 cases, had proteinuria between 3 to 9 g/24h, therefore nephrotic PU.

In collapsing FSGS, the 24-hour proteinuria was 7.6 ± 4.1 with a P-value of 0.02, which is statistically significant (**Table 18**). This was corroborated by the work of Haas *et al.* [8] in which collapsing FSGS had an average proteinuria of 10 g/24h and vascular FSGS 9 g/24h. And the NOS FSGS had an average proteinuria of 4.4 and the cell type had an average proteinuria of 5.5 g/24h. Finally, the cellular form was the least proteinuric in all the studies.

4.7. Ultrasound

In collapsing FSGS 10.4% of the kidneys had poor differentiation. The P Value was at 0.014 which is statistically significant.

However 84.81% of patients with FSGS had normal sized kidneys which probably relates to tubulointerstitial infiltrates, edema, fibrosis and dilated tubules as seen in HIVAN cases [8] [14]-[18].

4.8. Histological

4.8.1. FSGS in General

The prevalence of segmental and focal hyalinosis listed was 158 cases with 123 primary cases or 77.17% and 35 cases with etiology or 22.83%. Among the primary cases, the NOS FSGS type is the most represented 62 cases or 39.24% followed by the collapsing FSGS 48 cases or 30.37%.

FSGS with etiology was associated with fibrosis in 5/35 cases, *i.e.* 1 case out of 7.

4.8.2. Tubulointerstitial and Vascular Lesions

The collapsing FSGSs had tubulointerstitial lesions in 66.66% and P 0.006 which is statistically significant.

The 62.5% of the collapsing FSGSs had vascular lesions with 0.35 (0.15 - 0.78) and P 0.01 statistically significant.

Weiss *et al.* as well as Detwiler *et al.* had previously shown that collapsing FSGSs were more likely to cause tubulointerstitial lesions [17] [19].

Tubulointerstitial lesions best correlate with the degree of reduction in glomerular filtration rate (GFR) sign severity.

These tubulointerstitial lesions may be a consequence of glomerular injury through disruption of postglomerular circulation, trafficking of tubular proteins with promotion of local cytokine and growth factor, production of inflammatory infiltrates and promotion of fibrogen or again, the formation of obstructions.

4.8.3. Treatment and Evolution

TIP FSGS were the most corticosensitive 33.33% followed by NOS FSGS 11.29% and collapsing FSGS.

There was complete remission in 50% for TIP FSGS and 11.29% for NOS FSGS and 2.08% for collapsing FSGS and no complete or partial remission for vascular and cellular FSGS. The work of Dial *et al.* [5] had found 29.7% complete remission for the primary forms. In our work we specified the evolution of each type of FSGS that can explain the difference.

Corticoresistance was present in 12.5% of cases of collapsing FSGS versus 8.02% in NOS type FSGS. The other primitive types had no cases of corticoresistance. Steroid dependence was present in 4.83% of cases of NOS type FSGS and in 4.16% of collapsing FSGS. The other types of primary FSGS and secondary FSGS had no cases of corticodependency. This was corroborated by the work of Haas *et al.* [8], Weiss *et al.* as well as Detwiler *et al.* [12] [13] who found collapsing FSGS to be more corticoresistant.

The complications found were: one case of corticosteroid-induced diabetes, one case of femoral neck fracture, 4 cases of urinary infections, 3 cases of skin infections and 1 case of digestive infection, *i.e.* 16.66%. MENDOZA *et al.* [16] had also found 17% of cases of infections in the patients followed.

The evolution of the FSGS in our work reveals that every 8 months the proteinuria decreases by half. Similarly, complete remissions were observed in 8 months in PEI *et al.* [20].

Actually, the efficacy of extracorporeal plasma therapy for adult patients with native kidneys with primary FSGS is established. The repeated administrations of retuximab were not associated with an habituation effect and resulted in a high percentage of complete or partial remission comparable to larger pediatric studies. Renal function remained stable during the treatment period. This approach led to an extension of relapse-free survival compared to previous treatment regimens over a median duration of follow-up of 110 months. And the translational study has identified clopidogrel as a promising therapeutic option

for FSGS via network-based bioinformatics analysis. Clopidogrel significantly improved key outcome parameters in the adriamycin FSGS mouse model, including albuminuria, weight loss, and histopathological lesions of the kidney. These data, along with a favorable drug safety profile, endorse clopidogrel as an attractive candidate for drug repositioning and subsequent clinical trial evaluation for patients suffering from FSGS [20] [21] [22].

5. Conclusions

The prevalence of FSGS was 52% in a series of renal biopsies over a period from 1993 to 2001 in Senegal.

Thus our analytical study on histologically proven primitive or secondary segmental and focal hyalinosis has made it possible to prove that:

A prevalence of FSGS found was 16.54 and the incidences of 10.15%; 14.04%; 15%; 17.64%, 20.11%; 19.58% respectively in 2010, 2011, 2012, 2013, 2014 and 2015, *i.e.* an incidence of around 1.25%.

The mean age was 33.07 years ± 13.8 years. The sex ratio was in favor of men 2.59.

Among the medical history, hypertension was observed first with 15.82% followed by edema at 12%. And the risky lifestyle found first was herbal medicine in 51.27%.

At the clinic, renal-type edema was found in 93.3%, the first reason for hospitalization. In 92.14% of cases, the diuresis was normal. In collapsing FSGS, mean proteinuria was 7.5 ± 4.1 g/24h.

We found that the collapsing FSGSs had more severe proteinuria than the other forms, followed by the urinary pole FSGS (TIP), NOS and vascular form.

In collapsing FSGS 18.98% of the kidneys were of poor differentiation. However, 90.5% of patients with HSF, which is the strong majority, had kidneys of normal size as described in HIVAN.

General histology showed:

Of the 158, 123 cases are primary *i.e.* 77.17% and 35 secondary cases *i.e.* 22.83%. FSGS type NOS 62 cases is the most represented 46 cases followed by FSGS collapsing.

Secondary FSGS was associated with fibrosis in 5/35 cases.

The 66.6% of the collapsing FSGSs had tubulointerstitial lesions and the 62.6% of the collapsing FSGSs had vascular lesions.

Therapeutically and evolutionarily, collapsing FSGSs followed by NOS FSGSs were the most corticoresistant. Of the 158 patients recruited for FSGS, 103 received immunosuppressive treatment, *i.e.* 65.18%.

Of these 103 patients who received treatment based on immunosuppression, 95 received corticosteroid therapy alone, *i.e.* 92.23%, 7 patients or 6.8% received corticosteroid therapy combined with another immunosuppressant, either azathioprine or mycophenolate mofetil or cyclophosphamide. One patient, or 1.95%, had received the 3 immunosuppressants during follow-up at different

times.

90 patients out of 158 or 60% were treated with diuretics.

The evolution was interspersed with 12 patients or 7.59% corticoresistant, 15 patients or 9.49% corticosensitive, 4 corticodependent patients.

The evolution of the FSGS reveals that every 8 months the proteinuria decreases by half.

Conflicts of Interest

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

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