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Idiopathic Focal Segmental Glomerulosclerosis, Nephrotic Syndrome and Steroid Psychosis: A Case Report in Enugu, Nigeria, and Review of Literature

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Abstract **Background:** Focal segmental glomerulosclerosis (FSGS) is a common cause of nephrotic syndrome in adults in Sub-Saharan Africa. The nephrosis usually responds well to steroid therapy but alternative therapy may be needed, following steroid toxicity. **Aim:** We thus report a case of idiopathic FSGS that manifested as nephrotic syndrome that initially responded to steroid therapy but later was controlled with Azathioprine following steroid-induced psychosis. **Findings:** Patient was a 32-year old man who developed features of nephrotic syndrome and was found to have biochemical evidence of hypoalbuminemia, low density lipoprotein hypercholesterolemia and nephrotic proteinuria. Renal biopsy histology showed features of FSGS. He was commenced on oral steroid therapy, hematenics, an antiplatelet, a proton pump inhibitor and loop diuretics. Though the edema regressed he developed acute psychosis. The steroid was discontinued, while antipsychotic therapy and Azathioprine were added. The features of psychosis resolved. Nephrosis also remitted. **Conclusion:** This case report of FSGS that presented as nephrotic syndrome in an adult in Enugu, Nigeria-one of the four cases of steroid-induced psychosis we observed in five years-shows that steroid-induced psychosis is rare in this area and could be addressed by withdrawing the steroid and instituting antipsychotic therapy. It further shows that the nephrosis in idiopathic FSGS could also respond well to isolated Azathioprine therapy in our setting.

Keywords: *focal segmental glomerulosclerosis, nephrotic syndrome, steroid-induced psychosis, Azathioprine, enugu, Nigeria*

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1. Introduction

There is increasing incidence of nephrotic syndrome (NS) associated with focal segmental glomerulosclerosis (FSGS) in Sub-Saharan Africa. [1] Nephrotic syndrome comprises a clinical entity that is characterized by edema, marked proteinuria, low density lipoprotein (LDL) hypercholesterolemia, and hypoalbuminemia.

The causes of NS are myriads. In adult black people, they include FSGS, membranoglomerulonephritis (MN) and idiopathic variants, among others [2,3] Irrespective of etiology, NS results from inflammatory damage to glomerular filtration barrier. Although proteinuria is the hallmark in patients with FSGS, only a fraction of them would have overt NS. [4]

The treatment of FSGS associated with NS includes steroid and, sometimes, use of other immunosuppressants. [5] Steroid toxicity, including neuropsychiatric involvement, is not uncommon and may necessitate the discontinuation of steroid and the incorporation of other immunosuppressants. [6]

In a resource-poor setting such as ours, most cases of NS are not routinely backed up with renal histological diagnosis. [7] As a result, most management outlines adopted are usually empirical. Even when renal histology is done it is usually limited to Light Microscopic analysis. Overall, management outcomes and choices of medications might be suboptimal. This has prompted us to document this case report of idiopathic FSGS associated with NS and evident steroid toxicity that showed remarkable remission of nephrosis following alternative use of Azathioprine therapy.

2. Case Presentation

The patient was a 32-year old man, a native of Enugwu-Ezike, Igboeze North LGA, Enugu State, who presented in the Medical Out-Patient (MOP) clinic of University of Nigeria Teaching Hospital, Enugu, Nigeria. He complained of recurrent body swelling of 7 years' duration. The swelling was first noticed on the legs and face, gradually progressed to the thighs, abdomen, scrotum and upper limbs and was worse in the morning but regressed

as the day progressed. The urine was also noticed to be frothy. There was a gradual decrease in urine frequency but no change in urine color; he has no dysuria or passage of blood in urine.

There was no history of use of mercury-containing soap or cream, snake bite or bee sting in the past. He has no high risk behavior for human immunodeficiency virus infection. There was no history of body swelling in childhood or early school years. He could not remember if there was a prior sore throat. He also denied history of skin rash, jaundice or use of injections from local health providers. There was no tiredness, anorexia, nausea, vomiting, coke-colored urine or history of blood transfusion. He was not a known hypertensive or diabetic patient. He was the 1st in a family of 5 offsprings. There was no family history of renal disease. He was single, used alcohol occasionally, about 2 bottles of beer a week, but did not use tobacco in any form.

The patient appeared chronically ill-looking and has anasarca. He was mildly pale, has fluffy hair and mild leuconychia. There was no significant peripheral lymphadenopathy. His weight was 89.3kg, height 1.73m and body mass index 30.0kg/m^2.

His pulse rate was 64beats/min, regular and of normal volume. His blood pressure was 130/80mmHg; JVP was not raised. Apex beat was at the 5th left intercostal space in the mid-clavicular line; heart sounds heard were S1 and S2. There was no murmur or crepitation. His respiratory rate was 22 cycles per minute. The breath sound was vesicular.

Abdomen was distended; gross ascites was demonstrable by fluid thrill. Liver, spleen and kidneys were not enlarged.

He was conscious and alert, oriented in time, place and person. There was no sign of meningeal irritation. There was no asterixis or any other neurological deficit.

A provisional diagnosis of nephrotic syndrome was made.

He was commenced on tab Frusemide 120mg BD and worked up for renal biopsy while still on out-patient clinic visits.

Urinalysis, full blood count (FBC), 24-hour urine protein, serum electrolytes, urea and creatinine (SEUC), serum calcium, serum phosphate, fasting serum lipid profile, serum protein, liver function tests, prothrombin time, International Normalized Ratio (INR), renal ultrasound scan, chest X-Ray (CXR), stool microscopy, viral screens, fasting blood sugar and renal biopsy were done. The results are displayed on Table 1, Table 2 and "Result 3", while the renal biopsy film is shown in Figure 1.

A diagnosis of nephrotic Syndrome secondary to FSGS was made.

His problems were nephrotic proteinuria, hypoalbuminemia, hypercholesterolemia (dominantly LDL), anemia, sepsis and fluid retention.

The patient was commenced on the following treatment: dietary changes (high biologic value protein 1g/kg/day, low potassium diets, avoidance of polysaturated fats), tab Prednisolone 60mg morning daily, cap Omeprazole 20mg daily, tab Fluvastatin XL 80mg nocte, tab Warfarin 5mg nocte, tab Lisinopril 2.5mg daily, tab Frusemide 120mg BD, tab Metolazone 5mg daily, tab Fersolate 200mg tds, tab Folic acid 5mg daily, tab Ascorbic acid 200mg tds, tab Multivite I tds, tab Ciprofloxacine 500mg Bd x 10 days.

Follow-up:

First visit

After 24 days of being on the above regimen he was brought to the MOP Clinic. He was said to be behaving abnormally – praying a lot, claiming a call to priesthood, hearing from his dead father, and seeing things that were not there. He has insomnia but was not violent. His drugs had been discontinued 5 days before this visit when he presented in a peripheral hospital on account of abnormal behavior. A history of a similar behavioral change 2 years before presentation was also obtained from his relative; it was also admitted by same informant that the behavioral change was preceded by use of some medications administered to the patient in a peripheral hospital for complaint of body swelling. One of the medications was taken about twenty tablets at a time once a day by patient. The name of the medications the relative could not tell. Patient was confused but cooperative; he has no waxy flexibility or asterixis. The peripheral edema disappeared. He has moon face, but no pallor. His pulse was 108/min and regular. BP was 130/90mmHg.

Urinalysis, SEUC, 24-hour urine protein and serum protein were re-evaluated. The results are shown in Table 1 and Table 2. Estimated glomerular filtration rate (eGFR) was 99mls/min/1.73m^2 (MDRD).

Table 1. Results of investigations

Investigation	At presentation	First clinic visit (24 days)	Second clinic visit (2 months)
URINALYSIS			
Specific gravity	1.025	1.025	1.020
pH	Acidic	Acidic	Acidic
Protein	+++	+++	++
Leucocytes	6-8/hpf	1-2/hpf	0-2/hpf
Blood	0/hpf	0/hpf	0/hpf
FULL BLOOD COUNT			
PCV	31%		35.7%
RBC	3.5x10^{12}/l		
Hb	10.5g/dl		11.9g/dl
MCHC	33.4%		
MCV	87.7fl		
WBC	11,300cell/ml		
Neutrophils	71%		
Lymphocytes	27%		
Basophils	1%		
Esinophils	1%		

Table 2. Results of investigations

Investigation	At presentation	First clinic visit (24 days)	Second clinic visit (2 months)
URINALYSIS			
Specific gravity	1.025	1.025	1.020
pH	Acidic	Acidic	Acidic
Protein	+++	+++	++
Leucocytes	6-8/hpf	1-2/hpf	0-2/hpf
Blood	0/hpf	0/hpf	0/hpf
BLOOD CHEMISTRY			
24-hour urine protein	7.0g	5.8g	2.8g
24-hour urine creatinine	1435mg		
SEUCr			
Sodium	136mmol/l	138mmol/l	140mmol/l
Potassium	5.3mmol/l	5.1mmol/l	4.6mmol/l
Bicarbonate	20mmol/l	21mmol/l	22mmol/l
Urea	6.6mmol/l	6.1mmol/l	5.7mmol/l
Creatinine	93µmol/l	98µmol/l	100µmol/l
Chloride	101mmol/l	101mmol/l	102mmol/l
Calcium	2.7mmol/l		
Phosphate	1.8mmol/l		
eGFR	129mls/min/1.73m^2	99mls/min/1.73m^2	97mls/min/1.73m^2
Fasting Serum Lipid Profile			
Total cholesterol	18.8mmol/l		
HDL-C	3.2mmol/l		
LDL-C	14.3mmol/l		
VLDL-C	1.3mmol/l		
Triglyceride	2.8mmol/l		
Serum protein	4.8g/dl	5.1g/dl	5.8g/dl
Serum albumin	1.7g/dl	1.3g/dl	2.3g/dl
Serum globulin	3.1g/dl	3.8g/dl	3.5g/dl
FBS	75mg/dl		
LIVER FUNCTION TESTS			
Bilirubin(total)	17.1		
Direct bilirubin	8.6		
Alkaline phosphatase	37		
Aspartate transaminase	22		
Alanine transaminase	12		
Prothrombin time(pt)	10.0seconds		
Control (pt)	11.1seconds		
INR	0.9		

An impression of steroid-induced psychosis and Cushing's syndrome in nephrotic syndrome due to FSGS was made.

Prednisolone was reduced to 40mg in the morning daily for 1 month, then to 20mg daily for another one month, then to 10mg on alternate day x 1 month. He was commenced on Azathioprine 50mg daily. The aim was to taper and stop the Prednisolone and to continue Azathioprine on account of the steroid-induced psychosis. He was continued on hematinics.

Psychiatric Unit reviewed and placed him on Resperidone

Second clinic visit 2 months later:

Patient had stabilized and the abnormal behavior had disappeared. The moon face and peripheral edema had also disappeared. He has no pallor; his pulse was 90/min and regular. Bp was 126/86mmHg.

Urinalysis, SEUC, 24-hour urine protein, Hb and serum protein were re-assessed. The results are also displayed on Tables 1 and 2. eGFR was 97mls/min/1.73m^2 (MDRD).

His 24-hour urine protein, 2.8g, down from 7.0g, the value recorded two months previously, showed a partial remitting nephrotic process. The Prednisolone 10mg on alternate day was given for 1 month and stopped. The patient was continued on Azathioprine 50mg daily, tab Fersolate 200mg tds, tab Multivite I tds, tab Folic acid 5mg daily and advised to be attending clinic monthly, with monthly SEUC, urinalysis, 24-hour urine protein, Hb evaluation. Patient was still on clinic follow-up at the time of writing this Case Report.

Results 3

Renal Biopsy: done on 23/02/2011. Histology film is shown in Figure 1.

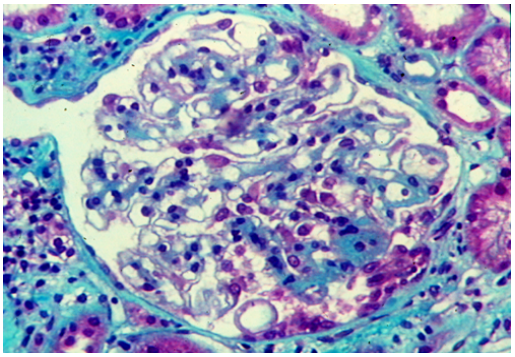

Figure 1. FSGS (X400 magnification, Jone's silver stain)

It showed, on gross examination, 2 cylindrical fragments, each 1cm in length. Microscopic examination: The sections showed renal parenchyma with 8 glomeruli, 2 of which were totally sclerotic. There was no associated hypercellularity. The uninvolved area of the tuft showed mesangial matrix thickening. Many tubules had calcified masses and oxalate crystals plugging their lumen. The interstitium showed mild fibrosis. Histologic diagnosis of focal segmental glomerulosclerosis (FSGS) was made.

Renal ultrasound scan: This showed that both kidneys have increased echogenicity, right kidney measuring 11.0 × 4.9cm and left kidney 12.4 × 4.9cm.

CXR: It showed normal findings.

Stool microscopy: This revealed no abnormalities.

Viral screens: HIV, HBsAg and HCV were all negative.

3. Discussion

Nephrotic syndrome is a clinical entity that is characterized by proteinuria > 3.5g/day and is marked by edema, hyperlipidemia, hypoproteinemia and other metabolic derangements. Many clinicians, in practice, refer to "nephrotic range" proteinuria as nephrotic syndrome regardless of whether there are manifestations of the full syndrome because, according to them, the latter are consequences of proteinuria. [8] However, isolated massive proteinuria may occur without other features of NS. Diabetic nephropathy, membranoglomerulonephritis (MN), minimal change glomerulonephritis (MCGN), FSGS, and membranoproliferative (MPGN) are some of the causes of NS and these diagnoses are based on renal biopsy histology.

MCGN is more uncommon than MPGN in adult Nigerian population. [9] However, varied reports in Nigeria have shown that FSGS is the commonest cause of NS in adults. [9,10,11] In contrast, FSGS and MN are the most common causes of NS in adults worldwide. [12]

In our report, renal biopsy result showed that our patient has FSGS. There were no features to pinpoint idiopathic FSGS in this patient as supportive electron microscopy was not available in our center. It was also not specified the variant of FSGS the patient has, from among a). tip lesion, b). perihilar lesion, c). cellular variant, d). collapsing variant, and e). FSGS NOS (not otherwise specified. [13] MCGN and FSGS are thought to belong to two points in the same spectrum, each representing a distinct point in the progression of the same disease. [14,15,16,17]

Some diseases, including infections, may be associated with the etiology of FSGS and nephritic process. Our index patient has sepsis which could also be explained by reduced immunity observed in NS. [18] Hypertension may not be present at onset, as this is seen in about 13% to 50% of all cases of FSGS. [5,19,20] Majority of FSGS patients present with normal or mildly elevated renal function. FSGS occurs more in black race, [21] is commoner in men and typically is observed in adults between 18 and 45 years. [5,22] This was the classical presentation in our patient: there was no prodromal illness prior to the onset of the illness and hypertension was not a feature. Renal function as evidenced by eGFR 129mls/min/1.73m^2 was normal, although the kidneys showed increased echogenicity, suggesting that our index patient has an early stage of FSGS. However, with disease progression, renal function would become impaired and may lead to end stage renal disease. [5,23]

In patients with FSGS proteinuria is the hallmark. [4,5] Variable values of proteinuria are observed in FSGS: in some as low as less than 1.0g/day yet in some as high as 50.0g/day. [5,23] Characteristically, this proteinuria is nonselective. [5,23] In 25% to 75% of patients with FSGS, microscopic hematuria is observed. [5,24] Our index patient has proteinuria of 7.0g/day although it was not determined whether it was selective or nonselective. Paradoxically, he has no hematuria. Edema, biochemical evidence of hypoalbuminemia, LDL hypercholesterolemia, nephrotic proteinuria, renal biopsy histologic evidence of FSGS, HIV seronegative status, biochemical evidence of absence of diabetes mellitus, absence of prodromal disease, in our index patient, all confirmed a diagnosis of idiopathic FSGS.

The clinical evolution of idiopathic FSGS is variable and, as a result, its management is tasking. [5,25] The management of FSGS entails nonspecific and nutritional aspects, as well as non-immunosuppressive and specific components. Still empirical has remained the specific treatment modality. [5] Nonetheless, emphatic steroid therapy and immunosuppressants for the induction phase of remission and subsequently some agents for maintenance of remission in patients with idiopathic FSGS are recommended. [26,27] In this case report, our patient received steroid for induction and achieved a level of remission before manifesting steroid toxicity that included acute psychosis, warranting tapering and discontinuation of the steroid and replacing it with Azathioprine

It is pertinent that the patient is counselled and monitored for drug toxicity as the steroid therapy may last for a long time. Duration of therapy is usually dependent on the treatment response, as assessed by the presence, absence or declining evidence of edema, serum creatinine, serum albumin, 24-hour urine protein, and serum lipid levels. [5,28] Disappearance of edema in our patient, marked decline in 24-hour urine protein from 7.0g at onset to about 2.0g, and reduction in serum LDL showed he has a substantial level of remission of the FSGS.

The neuropsychiatric complications of steroid therapy are variable and common; they include anxiety, irritability, impaired cognition, depression, mania, psychosis, and suicidality. [6] These may necessitate aggressive and early psychiatric intervention. [29] Our index patient was reviewed and co-managed by our Psychiatric Unit with good resolution of psychosis.

Studies have shown that acute steroid-induced psychosis required tapering or discontinuation of the steroid, treatment with antipsychotics or with lithium. [30] Our patient received steroid treatment but it was discontinued because he developed acute psychosis at onset of administration. In this, the steroid toxicity was not from long-term use; yet it had to be tapered, or at best, was discontinued.

It has been demonstrated that long courses of steroid, Azathioprine and/or cyclophosphamide produced complete remission in 66% of nephrotic syndrome in subjects with FSGS, and a good measure of renal survival. [5,31] In this, remission was defined as protein excretion <0.2g/day – 0.3g/day, partial response 0.2g/day – 0.35g/day, or >50%

reduction in baseline 24-hour urine protein. [5,31] Our patient has therapeutic response to steroid therapy, albeit also having steroid psychosis. Azathioprine therapy, we used here to replace steroid, continued to show therapeutic response as evidenced by resolution of edema and decline of 71% in proteinuria. This observation tends to suggest that our patient probably would be among the small group of FSGS that responds to isolated immunosuppressant therapy.

4. Conclusion

This case report of FSGS that presented as nephrotic syndrome in an adult in Enugu, Nigeria, one out of four cases of steroid-induced psychosis we observed in five years, shows that steroid-induced psychosis is rare in this area, but could be addressed by withdrawing the steroid and instituting antipsychotic therapy. It further shows that the nephrosis in FSGS could also respond well to isolated Azathioprine therapy in our setting.

Competing Interests

The authors declare no competing interest.

Authors' Contributions

The author has read and agreed to the final version of this manuscript.

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