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Nephrotic Syndrome Management in Pregnancy: A Diagnostic and Therapeutic Challenge in a Resource-Limited Setting—A Case Report

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Abstract

Nephrotic syndrome is a rare condition in pregnancy. This case report highlights the challenging management of nephrotic syndrome in a pregnant woman in a resource-limited hospital. **Case presentation**: We report the case of a 35-year-old grand multiparous woman, at 19 weeks + 3 days of gestation, treated for pulmonary tuberculosis in 2018, who presented in 2025 with a 3-week history of lower limb swelling that progressed progressively to the abdomen, also associated with early morning face and neck swelling, fatigue, and occasional breathing difficulties. Urinalysis showed 2+ proteinuria. Chest X-ray revealed atelectasis of the left upper lobe. Management included furosemide for 1 week. Further laboratory investigations at 20 weeks of gestation revealed: urine dipstick of 3+ proteinuria, severe hypoalbuminemia, elevated serum urea and creatinine, hyperlipidemia, and stage 2 nephropathy consistent with nephrotic syndrome. Obstetrical ultrasound showed an intrauterine singleton pregnancy, homogenous hepatomegaly, and ascites of moderate quantity. Treatment instituted involved mainly human albumin infusions, fluid restriction, and anticoagulants. Hospitalization stay was marked by improvement of symptoms, and the patient was discharged on day 15 on cardio aspirin, iron, and potassium iodide tablets. The patient, due to reported financial constraints, did not keep her appointment one week later. The patient re-

portedly developed respiratory difficulties at about 28 weeks gestation for which she was hospitalized in the initial health facility; cesarean section was performed in the course of hospitalization, complicated by both fetal and maternal demise within a 48-hour interval. **Conclusion:** In resource-limited contexts, nephrotic syndrome in pregnancy is a medical challenge with high mortality.

Keywords

Nephrotic Syndrome, Pregnancy

1. Introduction

Nephrotic syndrome is characterized by peripheral edema, severe proteinuria, hypoalbuminemia, and is often associated hyperlipidemia [1]. It is a rare condition in pregnancy. The incidence of primary nephrotic syndrome in pregnancy is around 0.012% - 0.025% [2]. It can be primary or secondary and is usually diagnosed in the second or third trimester, accompanied by severe hypoalbuminemia with or without impaired kidney function [1] [2]. The cause of nephrotic syndrome must be established by ruling out all possible secondary causes, especially in pregnancy with preeclampsia, which can also occur with proteinuria [1] [3]. In context of nephrotic syndrome, the incidence of cesarean sections and preterm deliveries is higher than in healthy women [1] [2]. Among nephrotic syndromes, membranous nephropathy (MN) is the most favorable in terms of outcomes, whereas membranoproliferative glomerulonephritis (MPGN) and focal and segmental glomerulosclerosis (FSGS) expose patients to a higher degree of both materno-fetal and renal adverse events [2]. Thus, the aim of this work is to highlights the diagnostic and management challenges of this pathology in low-resource settings.

2. Case Presentation

This was a 35-year-old G7P6006, LNMP = 20/07/2024, EDD = 27/04/2025, GA (upon hospitalization (04/12/2024)) = 19 wks + 3 days, housewife.

Presented at the Yaounde University Teaching Hospital (YUTH) with a 3-week history of lower limb swelling that progressed progressively to the abdomen, also associated with early morning facial and neck swelling, fatigue, and occasional breathing difficulties. This prompted her to consult at the Ebolowa Regional Hospital (ERH), where the following investigations were done.

1) *Urine dipstick = 2++ proteinuria.*
2) *Chest X-ray = atelectasis of the left upper lobe.*
3) *ECG = normal.*

For which she was prescribed furosemide, injectables followed by tablets (2 tablets x^2/day) for 1 week. The evolution was marked by incomplete resolution of symptoms. A second consultation was done at ERH where the following investi-

gations were carried out.

1) *Urine dipstick = 3+ proteinuria.*

2) *Blood tests:*

a) *Albumin = 14.3g/l (38-48) ↓↓*

b) *Urea = 0.60 g/l (0.15-0-42 g/l) N.*

c) *Creatinine = 17.2 mg/l (6-11 mg/l).*

d) *Alanine aminotransferase (ALAT) = 0.2, Aspartate aminotransferase (ASAT) = 14.4.*

3) *Obstetrical ultrasound: intrauterine singleton pregnancy at 20 weeks, homogeneous hepatomegaly, ascites of moderate quantity.*

a) *Note: HIV and hepatitis B/C serology negative.*

Prompting her referral to the YUTH for further management. She had a poorly followed-up pregnancy with first ANC at 17 weeks 5 days, baseline BP = 106/53 mmHg, weight = 60 kg, treated for pulmonary tuberculosis in 2018 for a period of 6 months. No past history of streptococcal throat infections. Upon arrival at our service, she complained of asthenia. On physical examination**,** she was conscious and ill-looking, with anasarca. Vital signs: BP = 138/79 mmHg, PR = 95 b/m, RR = 22 breaths/min. Anthropometric, weight = 68.4 kg, pale, anicteric sclera, pupillary edema. Chest exam: S1/S2 heart sounds audible with holosystolic murmur 2/6, bilateral lower lung field crackles. Abdomen was distended, ascites present with fluid thrill, no palpable abdominal masses, uterine height difficult to appreciate due to ascites. AC = 100 cm, bilateral pedal pitting edema. The diagnosis (made with consultation of nephrologists): Nephrotic syndrome in pregnancy at 19 weeks 3 days' gestation based on anasarca, elevated proteinuria, hypoalbuminemia.

The following laboratory investigations were requested and revealed:

1) *Renal ultrasound = right kidney = 126 mm, left kidney = 130 mm, hyperechogenic kidney with corticomedullary differentiation, nephropathy stage II.*

2) *Spot proteinuria = 4.9 g/l N ≤ 0.14.*

3) *Creatinemia =1.90 g/l.*

4) *C-reactive protein <10 mg/l.*

5) *protein/creatinine ratio = 4.49 (greater than 3).*

6) *Lipid profile: elevated total cholesterol = 2.32 g/l ↑, low HDL = 0.10 g/l ↓ elevated triglycerides = 5.81 g/l↑.*

7) *Serum electrolytes (sodium, potassium, chloride) were normal.*

8) *Full blood count: Hemoglobin = 7.5 g/dl, White blood cells = 4430/mm^3, Platelets = 196,000/mm^3.*

9) *Blood group O, rhesus positive.*

Renal biopsy or immunological testing was unavailable.

The treatment instituted included:

Hospital admission for stabilization, fluid restriction, salt-free diet, human albumin infusion 20% 200 mg/L per 24 hrs (diluted in 100 cc of normal saline and administered over 4 hrs), enoxaparin 4000 IU injection/24 hrs, omeprazole 40 mg

injection/24 hrs.

The prescribed compression stockings were not available.

1. Maternal and fetal outcome

On day 10 of hospitalization, the patient was still on the treatment plan as mentioned previously.

She lost 3 kg, Weight = 65.3 kg (68.3 on entry). Her lungs were clear on auscultation. The abdominal circumference was 99 cm (100 on entry), uterine height = 21 cm. The fetal monitoring showed the presence of a regular fetal heart.

The control laboratory investigations revealed: severely low albuminemia = 11.4g/l, the urea value at the upper limit = 0.72 g/l, and significantly elevated creatinine = 22.9 mg/l.

Treatment added was: aspirin 100 mg tab, 1 tab daily, iron and folic acid 80/5 mg, 1 tablet daily.

2. During her hospitalization, the patient presented significant difficulties affording prescribed medications. She did not receive corticosteroids due to fetal concerns.

On day 15 of hospitalization, the patient had taken four albumin infusions and 5 days of low-dose aspirin. This was associated with a favorable clinical evolution. The patient's edema regressed, her renal function parameters improved, no preeclampsia signs were observed, and fetal growth remained appropriate for gestational age.

Therefore, the patient was discharged at 21 weeks of gestational age on enoxaparin, potassium iodine tablets, iron tablets, low-dose aspirin, and given an appointment in 1 week. On discharged, the patient was informed about the chronic nature of her kidney disease and the importance of close monitoring of her pregnancy at the university teaching hospital. Multidisciplinary follow-up by both obstetricians and nephrologists was planned.

The patient did not attend her scheduled follow-up appointment. While not succeeding in reaching her directly, her husband was contacted by telephone. During the call, he reported that, at 28 weeks + 3 days, the patient complained of respiratory difficulties and had one episode of convulsion. She was rushed to the closest health care facility at the Ebolowa District Hospital (EDH). She underwent an emergency cesarean section and was subsequently hospitalized in the reanimation unit. The precise sequence of events and medical management remains unclear due to the lack of records from the facility. The newborn baby died 24 hours after birth, and the patient died 48 hours after the surgery.

According to her husband, the mentioned primary cause of death was cardiac/respiratory arrest, and the secondary cause was probable kidney injury from nephrotic syndrome in pregnancy.

3. Discussion

This case underscores the complexity of the management of nephrotic syndrome during pregnancy and the severity of the complications associated with this life-

threatening pathology.

In resource-limited settings, the first expected challenges are linked to the diagnosis. Lack of renal biopsy and immunological tests and the overlap with preeclampsia often lead to delays in diagnosis and misclassification [1]. Obviously, nephrotic syndrome is mainly a collection of clinical symptoms and biological modifications, consisting of peripheral edema, severe proteinuria, hypoalbuminemia, and often accompanied by hyperlipidemia [2] [3]. These symptoms and abnormal signs were found in this patient and promptly associated with nephrotic syndrome. In fact, the patient was initially seen and managed at the regional hospital of Ebolowa, which is a referral hospital in the south region of the country. She was appropriately referred to the university teaching hospital for specialized care by a team of obstetricians and nephrologists. This reflects a well-functioning referral system and accurate clinical appreciation by the practitioners on the frontline.

The nephrotic syndrome in pregnancy is usually diagnosed in the second or third trimester, with clinical features of peripheral edema and fatigue and severe hypoalbuminemia and/or impaired renal function [1]-[3]. According to this description, in this case, the patient had her diagnosis at 19 weeks of gestation and she experienced generalized edema and severe hypoalbuminemia.

Nephrotic syndrome in pregnancy can be precipitated by preeclampsia, type 2 diabetes mellitus, systemic lupus erythematosus (SLE) or secondary to a prior tuberculosis [1]. In this case, the patient's clinical picture made the initial diagnosis of pre-eclampsia less likely, as this condition generally requires both hypertension and proteinuria after 20 weeks [2]-[4]. The patient was admitted at 19 weeks with symptoms that had been developing for 3 weeks and no abnormalities in her blood pressure before or during her hospitalisation. The subsequent development of hypertension at 28 weeks suggests the late onset of pre-eclampsia superimposed on a pre-existing glomerular disorder [1] [2] [4].

Lupus nephritis can be considered for this patient, as it can cause nephrotic syndrome in early pregnancy; however, it may be ruled out due to the absence of systemic symptoms of lupus such as rash, arthralgia, polyseritis and clinical history [1] [5].

Furthermore, diabetic nephropathy was ruled out in the absence of diabetes or associated complications.

In this case, the past history of pulmonary tuberculosis can suggest a tuberculosis-related glomerulopathy, such as secondary amyloidosis despite, there were no signs of active tuberculosis, and the patient had been declared completely cured [1]. Despite that, Renal amyloidosis should be part of the differential diagnosis for proteinuria, especially in case of nephrotic syndrome [1] [6].

Indeed, in the absence of biopsy or serological tests, the initial nephrotic syndrome may be presumed to be due to primary glomerulopathy or possible amyloidosis secondary to previous pulmonary tuberculosis, or secondary to undiagnosed immune-mediated kidney disease. As shown by Verma *et al.*, the majority of nephrotic syndrome episodes are classified as unexplained or primary [1].

The cause of nephrotic syndrome must be established by ruling out all possible secondary causes, especially in pregnancy [3]. In this case, diabetes was excluded, but neither serum amyloid A (protein SAA), nor immunological nor histological diagnosis was available.

One of the aims of early detection and treatment is to prevent long-term complications to the kidneys and improve maternal and fetal outcomes [2] [3]. In this case, despite the good diagnostic performance, there was a medication access problem due to the patient's financial constraint. In developing countries, lack of universal drug coverage can significantly impact the capacity of patients to afford their treatment [7].

Pregnancy-related acute kidney injury (AKI) in young women worldwide is a factor that exacerbates the morbidity and mortality rates of mothers and neonates [4] [8] [9].

In a context of nephrotic syndrome in pregnancy, treatment is usually symptomatic [1]-[3]. Management of minimal change disease includes initial and follow-up therapy for frequent relapsing and steroid-dependent conditions [1] [2] [6]. Primary glomerulonephritis may require the administration of immunosuppressive drugs, such as mycophenolic acid and cyclophosphamide, whose teratogenic risk is known and should therefore be replaced before gestation [1] [2] [10]. Monoclonal antibodies, such as anti-CD20 rituximab, should be avoided because of their transplacental transport in the last trimesters of pregnancy, which can induce transient B cell depletion in fetuses [10]. Conversely, short half-life corticosteroids, such as prednisone and methylprednisolone, are considered a valid alternative because of their inactivation by placental 11β-hydroxysteroid dehydrogenase type 2 that prevents steroid adverse effects on the fetus [1] [2]. However, this patient did not received corticosteroids. This choice was made due to the absence of active inflammation, as evidenced by the absence of fever, constitutional symptoms, and elevated inflammatory markers such as CRP or leukocytosis. and considering potential risks such as gestational diabetes and fetal growth restriction with prolonged use, along with these drugs availability limited in this setting.

In this case, although the patient did not receive corticosteroids, clinical stabilization was observed after adding aspirin. Aspirin will improve microvascular blood flow by inhibiting platelet aggregation [11]. On the other hand, low-dose aspirin likely promotes prostacyclin release and helps reduce systemic inflammation that exacerbates the signs and symptoms of nephrotic syndrome and limits endothelial injury [11].

Diuretics are usually avoided in pregnancy and may be adopted in nephrotic syndrome refractory to immunosuppressive therapy under strict control to avoid volume depletion and AKI risk. 20% albumin intravenous infusions can also be done [2]. In this case, the patient did not receive diuretics but received albumin intravenous infusions. Albumin infusion helps restore plasma oncotic pressure, reducing oedema and improving renal perfusion.

Nephrotic syndrome is also characterized by a hypercoagulable state that must

be treated when the albumin level is lower than 2 g/dl. Low molecular-weight heparin is a valid choice for both prophylaxis and treatment of thromboembolic complications during pregnancy and postpartum, because it does not cross the placenta [1] [11]-[13]. In this case, the patient received Enoxaparin every day since her admission. However, it was noted that her initial clinical improvement was observed after adding aspirin to the treatment with enoxaparin and albumin. This aligns with authors attesting that combining antithrombotic therapy may improve outcomes [1] [2].

This temporary relief of symptoms supports the theory that vascular dysfunction and volume depletion were the main contributors to the initial decompensation of the patient [1] [2].

Despite this improvement, the patient did not attend her follow-up appointment.

Upon discharge from hospital, the patient was advised about the chronic nature of her kidney condition and the importance of close monitoring of her pregnancy at the university teaching hospital. She was advised to attend follow-up visits, with the first examination scheduled for one week after her discharge from hospital. However, the patient and her family did not live in the capital, where the university teaching hospital is located, and could not afford to stay there. She had to return to her region with her husband, a six-hour drive from the capital. Due to socio-economic constraints, limited access to specialised care and a possible lack of understanding of the seriousness of the disease, the patient was then lost to follow-up.

Her husband reported a deterioration of her condition at 28 weeks of gestation, exactly 7 weeks after discharge. The delayed communication and limited information only lead to some hypotheses. The possible lack of adherence to treatment due to financial difficulties, the lack of corticosteroid therapy to address the immune aspect of the disease, the likely persistent proteinuria, and the possible lack of early detection of complications. Cardiac output is at its highest in the third trimester of pregnancy and can exacerbate nephrotic syndrome [14]. The hypercoagulable state is more pronounced during this trimester of pregnancy, increasing the likelihood of pulmonary embolism [13]. Hypertensive disorders occur most often in the third trimester [4].

According to her husband, the patient experienced respiratory distress and seizures. This may be associated with the complications of nephrotic syndrome [1] [2] [8]. The patient was reportedly dead on day two post-emergency caesarian section, and the newborn baby on day one. At 28 weeks of gestation, the fetus is viable but severely premature, and in low settings, the mortality rate is high [15].

This patient was managed and operated on in a secondary health care facility. In this low-resource setting, the technical platform was insufficient. The lack of a maternal critical unit in non-referral facilities makes complications arising at these hospitals difficult to manage, increasing maternal deaths [9].

4. Conclusion

Nephrotic syndrome in pregnancy is a severe condition with both maternal and fetal risks, especially in low-resource settings. Pregnant women with nephrotic syndrome can be managed successfully by a collaborative team of obstetricians and nephrologists with careful clinical and biological monitoring. In order to improve the maternal-fetal prognosis, quality care must be accessible to all, complications must be prevented and rapidly resolved, and the management of critical conditions must be carried out diligently in facilities with adequate technical resources.

Conflicts of Interest

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

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