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Renal Polycystic Disease during Pregnancy: A Case Report at the Departmental University Hospital Center of Ouémé, Benin

Ingrid Olowo[1,2*], Moufalilou Aboubakar[2,3], Patrice Dangbèmey[2,3], Mathieu Ogoudjobi[1,2], Tchimon Yéa Sètchégnon Vodounhè[2,4], Christiane Tshabu Aguèmon[2,4], Josiane Angélique Tonato Bagnan[2,3]

[1]Departmental University Hospital of Oueme Plateau, Porto-Novo, Benin
[2]Faculty of Health Sciences, University of Abomey-Calavi, Abomey-Calavi, Benin
[3]Mother and Child University Hospital Center Lagoon of Cotonou, Cotonou, Benin
[4]University Clinic of Gynecology and Obstetrics of National University Hospital Center Hubert Koutoukou Maga, Cotonou, Benin
Email: *olowoingrid@yahoo.fr, moufaliloua@yahoo.fr, ddpatous78@yahoo.fr, drogoumath@yahoo.fr, tchimonvod@yahoo.com, Tshabuchristiane@yahoo.fr, angelinetba@yahoo.fr

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Abstract

Renal polycystic disease is a genetic condition that may be revealed or complicated during pregnancy. We report the case of a 24-year-old primigravida admitted to the Departmental University Hospital Center of Ouémé for progressive dyspnea in the context of pregnancy at 35 weeks of amenorrhea, associated with a large abdominal mass, ascites, and pleural effusion. Clinical and paraclinical investigations initially suggested Demons-Meigs syndrome secondary to an ovarian cyst. Cesarean section associated with surgical exploration allowed the diagnosis of renal polycystic disease to be established. This case highlights the diagnostic and therapeutic challenges of renal polycystic disease during pregnancy, as well as its maternal and fetal implications.

Keywords

Autosomal Dominant Polycystic Kidney Disease, Pregnancy, Pseudo-Meigs Syndrome, Renal Failure, Cesarean Section

1. Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited renal disorder, with an estimated prevalence of 1 in 400 to 1 in 1000 live births [1]. It is characterized by the progressive development of multiple bilateral renal cysts, leading to renal enlargement and progression to chronic renal failure,

chronic kidney disease (CKD), hypertension, and hepatic cysts. The onset of clinical symptoms often coincides with the reproductive years [2]. In pregnant women, ADPKD may remain asymptomatic or may be revealed by severe maternal complications such as hypertension, anemia, renal failure, and, more rarely, respiratory complications due to mechanical compression [3]-[5].

Pregnancy represents a particular situation that may worsen the course of the disease because of hemodynamic, hormonal, and anatomical changes, and may complicate the differential diagnosis with other abdominal or gynecological conditions [6]. We report a case of ADPKD revealed during the third trimester of pregnancy, initially misdiagnosed as an ovarian pathology. The aim is to describe this case and to analyze the diagnostic and therapeutic challenges encountered.

2. Case Report

This was a 24-year-old woman, a reseller by occupation, primigravida nulliparous (G1P0), referred on December 9, 2025, from a peripheral health center for dyspnea of undetermined etiology during a 35-week pregnancy, associated with anemia and an abdominal mass suspected to be of ovarian origin. She had no known past medical history and had attended two regular antenatal care visits, with up-to-date tetanus vaccination.

Symptoms had been evolving for approximately three months, characterized by progressive abdominal distension associated with diffuse abdominal pain, early satiety, asthenia, and dyspnea worsening in the supine position. An obstetric ultrasound performed at 21 weeks of gestation revealed an ongoing pregnancy associated with a large right-sided cystic mass measuring 190 × 158 mm, interpreted as a serous ovarian cyst. An undocumented medical treatment was then initiated.

Subsequently, the clinical course was marked by worsening dyspnea over the past month, associated with significant weight loss, without hematuria, fever, or jaundice, prompting referral to a specialized center.

On admission, the patient's general condition was poor. Vital signs were as follows: blood pressure 136/91 mmHg, temperature 37˚C, heart rate 95 beats per minute, and oxygen saturation 95% on room air. The mucous membranes were moderately pale. Abdominal examination revealed a markedly distended, tense, and shiny abdomen with shifting dullness and an abdominal circumference of 110 cm. The uterus was difficult to assess clinically, as were fetal heart sounds.

Pleuropulmonary examination showed tachypnea at 32 breaths per minute, decreased vocal fremitus on the left, absence of vesicular breath sounds, and right basal dullness, suggestive of pleural effusion. Laboratory investigations revealed anemia with a hemoglobin level of 8.6 g/dL, white blood cell count of 6.5 G/L, platelet count of 152 G/L, and C-reactive protein level of 6 mg/L. Preoperative workup showed normal liver function but impaired renal function, with blood urea nitrogen at 1.54 g/L and serum creatinine at 115 μmol/L.

Abdominal ultrasound performed at admission demonstrated ascites associated with right pleural effusion, leading to a preoperative suspicion of pseudo-

Meigs syndrome in a 35-week + 4-day pregnancy.

Given the maternal respiratory distress, a cesarean section was indicated. The procedure was performed under general anesthesia via a midline infra- and supraumbilical incision. At laparotomy, several large cystic formations were identified, the largest originating from the right kidney. During attempted exteriorization, the main cyst ruptured with spillage of fluid, requiring aspiration. Multiple cystectomies were performed to allow immediate abdominal decompression. A transverse lower-segment hysterotomy then enabled delivery of a female newborn with Apgar scores of 6/8/10 and a birth weight of 1500 g. The left kidney, liver, and other abdominal organs appeared macroscopically normal.

Postoperative course was marked by worsening renal failure with fluid overload and transient oliguria, requiring three sessions of hemodialysis. Clinical and biological evolution was subsequently favorable. At discharge, on postoperative day 12, renal function was progressively improving, and the patient was referred for long-term follow-up in a specialized nephrology clinic. The newborn was managed in the neonatal care unit for prematurity and low birth weight, with a favorable short-term outcome. Histopathological examination of the surgical specimens confirmed polycystic kidney disease with no evidence of malignancy (Figures 1-3).

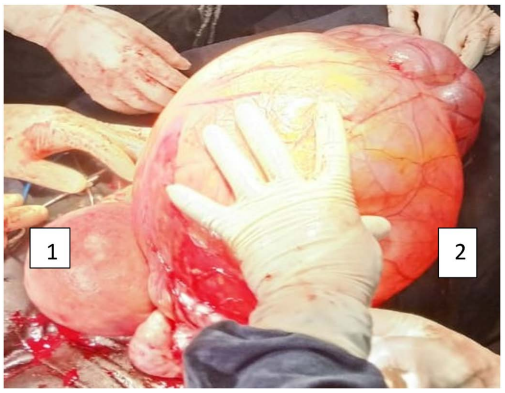

Figure 1. Renal polycystic. 1: Externalized uterus; 2: Largest cyst.

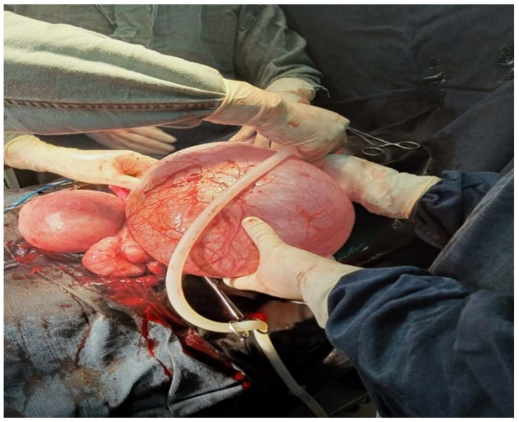

Figure 2. Aspiration of the largest cracked cyst.

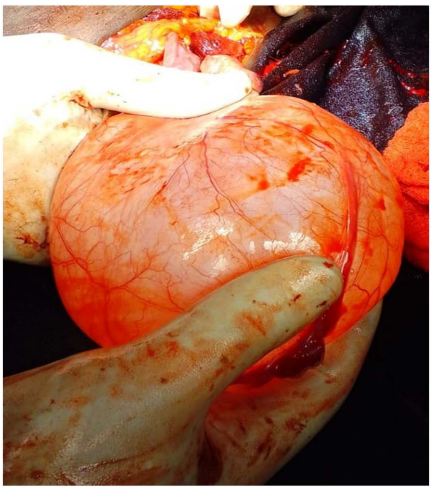

Figure 3. Smallest cyst.

3. Discussion

ADPKD is most often diagnosed in adulthood and may remain asymptomatic for a long period [1] [7]. Pregnancy may reveal or exacerbate the disease due to the hemodynamic, hormonal, and mechanical changes it induces [4] [5]. In our case, the clinical presentation was dominated by massive abdominal distension and severe dyspnea, initially suggesting an ovarian pathology.

Clinical symptoms of ovarian cysts generally include progressive abdominal enlargement, nonspecific abdominal discomfort, vaginal bleeding, and symptoms related to compression of adjacent organs, such as constipation, early satiety, vomiting, and urinary frequency [8]. Differential diagnosis with a giant ovarian cyst or pseudo-Meigs syndrome is common, particularly when imaging is performed in the context of advanced pregnancy [9].

At 21 weeks of gestation, distortion of anatomical landmarks due to the gravid uterus and the absence of systematic renal evaluation contributed to masking the renal origin of the cystic lesions, leading to an erroneous interpretation in favor of an ovarian cyst.

The decision to perform multiple cystectomies during cesarean section is not a standard approach in the management of ADPKD. However, in our case, the large volume of cysts causing major abdominal compression and maternal respiratory distress, combined with preoperative diagnostic uncertainty, justified this intervention in an emergency setting. The expected benefits were rapid abdominal decompression and respiratory improvement, despite the potential risks of worsening renal function [4] [10].

The most frequently reported maternal complications during pregnancy associated with ADPKD include hypertension, anemia, and renal failure [11]. In our patient, anemia and impaired renal function were already present at admission. The indication for postoperative hemodialysis was based on clinical and functional criteria—particularly fluid overload and persistent respiratory distress—rather than on initial biological parameters alone.

From a fetal perspective, maternal ADPKD is associated with an increased risk of prematurity and intrauterine growth restriction [11]-[13], as illustrated by the low birth weight observed in our case. Recent data confirm that pregnancy in women with autosomal dominant polycystic kidney disease remains high-risk, especially in the presence of preexisting renal impairment or hypertension [12]-[14]. Current recommendations emphasize close multidisciplinary monitoring and individualized obstetric decision-making to limit maternal and fetal morbidity. This highlights the importance of multidisciplinary management involving obstetricians, nephrologists, anesthesiologists, and neonatologists in our case.

Long-term consequences of pregnancy-related maternal complications are common in ADPKD. Genetic screening is recommended, as pregnancy has a significant impact on long-term outcomes in women with ADPKD [2]. Regular follow-up of disease progression is essential to promptly identify potential complications, including hypertension, abnormal cardiac valves, hepatic and pancreatic cysts, as well as recurrent urinary tract infections.

4. Conclusion

Autosomal dominant polycystic kidney disease may present atypically during pregnancy and mimic a gynecological disorder. Rigorous clinical assessment and careful interpretation of imaging studies are essential for early diagnosis. Management should be multidisciplinary and individualized to reduce maternal and fetal morbidity.

Ethical Considerations

Ethical and professional principles were respected, including the patient's free and informed consent, confidentiality, and anonymity.

Conflicts of Interest

The authors declare no competing interests.

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