Case Report

Suspected Pulmonary Embolism Postcesarean Section in a Patient with Autosomal Dominant Polycystic Kidney Disease

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Abstract

Autosomal dominant polycystic kidney disease (ADPKD) is the most common genetically inherited kidney disease worldwide. It is however relatively underdiagnosed in Africans because its diagnosis is often incidental. During pregnancy, ADPKD is associated with increased risk of preeclampsia and venous thromboembolism. The case of a 33-year-old lady incidentally diagnosed with ADPKD during pregnancy is presented. She developed preeclampsia at term and had cesarean delivery of twins. She however suffered cardiopulmonary arrest postoperatively and this created a treatment dilemma because therapeutic anticoagulation which was the primary treatment for her suspected pulmonary embolism was absolutely contraindicated if the actual cause of her collapse was ruptured cerebral aneurysm which was also a feature of ADPKD. We decided to resuscitate aggressively and perform an urgent cranial computed tomography which ruled out intracranial hemorrhage. We then commenced anticoagulation and she made an excellent recovery. This case illustrates the importance of a timely multidisciplinary approach to patient management.

Keywords: Anticoagulation, cardiopulmonary arrest, cerebral aneurysm, polycystic kidney disease, postpartum collapse, pulmonary embolism

Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is the most common genetically inherited kidney disease worldwide, affecting 1:400–1:1,000.[1,2] It is relatively underdiagnosed among Sub-Saharan Africans.[1] In pregnancy, women with ADPKD are at increased risk of hypertension and preeclampsia.[3] They also have increased risk of thromboembolism due to venous compression by the enlarged cystic kidneys.[4] Intracranial hemorrhage due to rupture of a berry aneurysm is also a possibility.[5]

We present our management of a 33-year-old G3P1^{+1} with ADPKD in pregnancy who suffered cardiopulmonary arrest from suspected pulmonary embolism 2-h postcesarean section for preeclampsia at term.

Case Report

A 33-year-old G3P1^{+1} was diagnosed with ADPKD and twins in index pregnancy during a routine ultrasound scan at 18 weeks. She had no prior problems. Pregnancy progressed uneventfully until 37 weeks + 4 days when her blood pressure spiked to 200/100 mmHg with 1 + proteinuria on the dipstick. She had no headaches, blurred vision, right hypochondrial/epigastric pain, or reduction in urine output. She perceived normal fetal movements. A diagnosis of preeclampsia at term in a patient with twin gestation and ADPKD was made. She was counseled and consented for stabilization and cesarean delivery.

Stabilization was commenced with oral labetalol 200 mg stat for acute blood pressure control and thereafter maintained on

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200 mg twice daily. She had magnesium sulfate for seizure prophylaxis. Preoperative full blood count and renal and liver function tests were within the normal limits. Clotting profile was also normal (international normalized ratio [INR] = 1.0; partial thromboplastin time with kaolin (PTTk) = 35.0 s). Her baseline preoperative blood pressure was 150/100 mmHg. She subsequently had cesarean section under the subarachnoid block (height of block, T6) and was delivered of live male twins, weighing 2.58 kg and 2.93 kg, respectively, both with Apgar scores of 10 at both 1 and 5 min. The cesarean section was uncomplicated, lasted 55 min, with estimated blood loss of 400 ml. Intraoperatively, her blood pressure ranged between 135–170 and 70–100 mmHg. Her vital signs were otherwise stable. Immediate postoperative blood pressure was 150/100 mmHg. She was transferred to the postnatal ward in clinically stable condition.

About 2 h postoperatively, she developed sudden-onset respiratory distress and subsequently had a witnessed cardiopulmonary arrest. Cardiopulmonary resuscitation was immediately commenced with return of spontaneous circulation after about 1 min of resuscitation. She had no prior history of prolonged immobilization, chest pain, orthopnea, calf pain, leg swelling, headaches, vomiting, or limb weakness. There was no bleeding from the operation site and lochia was normal. Immediate postresuscitation vital signs were blood pressure: 156/109 mmHg, pulse rate: 133 beats/min, respiratory rate: 28 cycles/min, and oxygen saturation 68% on intranasal oxygen!

She was promptly intubated, transferred to the intensive care unit (ICU), and commenced on mechanical ventilation with continuous cardiorespiratory monitoring. Intravenous labetalol was administered to keep her diastolic blood pressure between 90 and <110 mmHg and intravenous morphine and paracetamol were prescribed for postoperative analgesia. She was reviewed by the hematologist, cardiologist, and pulmonologist. She had urgent complete blood count, clotting profile, 12-lead electrocardiography (ECG), and chest radiograph, which were unremarkable except for sinus tachycardia on ECG.

Pulmonary embolism was strongly suspected. However, anticoagulation was not commenced until after ruling out an intracranial bleed due to a possible ruptured cerebral aneurysm associated with her ADPKD. She had an urgent cranial computed tomography (CT) scan which ruled out intracranial hemorrhage and she was commenced on a therapeutic dose of enoxaparin (Clexane). Invasive monitors (for arterial blood pressure and central venous pressure monitoring) were instituted. Her arterial blood gases were unremarkable.

About 24 h following admission into the ICU, she became fully conscious and responsive. Her blood pressure normalized, and labetalol was discontinued. She was successfully liberated from the mechanical ventilator by 48 h postoperatively. Invasive lines were removed and following sustained clinical improvement, she was returned to the postnatal ward on the 5th day of ICU admission with INR 3.6, and PTTk 59 s. She was discharged home on the 7th postoperative day on subcutaneous enoxaparin 60 mg daily and warfarin tablet 5 mg daily. She was well at the 6-week postnatal clinic review. She was therefore discharged to family planning, hematology, and nephrology. Her babies were also doing fine.

Discussion

ADPKD is characterized by the development and progressive growth of multiple bilateral renal cysts.[6] Cysts can also develop in other organs such as the liver, pancreas, spleen, and arachnoid membranes.[6] In the developed world, the diagnosis of ADPKD is now made as early as the first decade of life due to advancement in gene technology. However, in resource-poor settings, diagnosis is by clinical judgment and renal ultrasound.[1,2,6] Milutinovic *et al*. in their series found 36% of patients with ADPKD to be asymptomatic, normotensive, and without previous problems.[7] This was also the case in our patient.

Women with ADPKD are at increased risk of hypertension and preeclampsia during pregnancy.[3,8] This possibly explains the occurrence of preeclampsia in our patient, although twin gestation was another possible risk factor. Asides this, our patient had an uneventful pregnancy. This is in keeping with findings in previous studies that have suggested that pregnancy in women with ADPKD who are normotensive with normal kidney function often results in favorable outcome.[2,8] That said, compression of the iliac veins and inferior vena cava with possible thrombus formation and pulmonary embolism can be caused by enormously enlarged cystic kidneys, particularly the right.[9] This risk is further compounded by the procoagulant state of pregnancy in pregnant women with ADPKD.

Although pulmonary embolism was strongly suspected in our patient, urgent brain imaging was performed to exclude rupture of a cerebral aneurysm before commencing anticoagulation. Cerebral aneurysms are extrarenal manifestations seen in up to 8% of ADPKD patients.[9] Rupture of these aneurysms resulting in subarachnoid bleed has been described as the most devastating extrarenal complication of ADPKD, often resulting in premature death or disability.[9] A ruptured cerebral aneurysm would contraindicate anticoagulation, hence the need to exclude it in our patient. Although CT-pulmonary angiography could have confirmed pulmonary embolism, it was withheld in our patient because of concerns about intravenous contrast, given her poor clinical state.

In conclusion, ADPKD may be incidentally discovered during pregnancy, especially in resource-constrained settings where genetic counseling and testing are not widely available.[1,2,6] While these pregnancies are fortunately often uneventful, possible complications such as late-onset preeclampsia should be anticipated and expertly managed. In the event of potentially catastrophic complications of ADPKD such as thromboembolism or cerebral hemorrhage, a timely multidisciplinary approach to care with careful determination of the correct diagnosis by maximizing the available resources even in resource-constrained settings can be lifesaving.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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