Abstract

Acute Kidney Injury (AKI) caused by bilateral compression of the ureters by the gravid uterus is a rare complication of pregnancy. We describe the case of a pregnant woman who presented with obstructive AKI, developing severe complications (severe renal hypofunction, fetal growth restriction and urinary tract infections) and requiring invasive treatment. After exclusion of other causes, the diagnosis of AKI caused by bilateral compression of the ureters by the gravid uterus was established and double-J catheters were placed bilaterally with progressive resolution of the condition.

Key-Words: Pregnancy; Acute Kidney Injury; Ureteral Obstruction.

INTRODUCTION

Over the last 50 years, the incidence of Acute Kidney Injury (AKI) during pregnancy has been decreasing abruptly, currently affecting 1 in 20,000 pregnancies in developed countries.1

Regardless of the cause, AKI in pregnancy is an entity associated with significant maternal and fetal mortality and morbidity.2

Obstructive AKI during pregnancy is rare and only 22 cases caused by bilateral compression of the ureters by the pregnant uterus are described in the literature.3–12

The purpose of this paper is to report the case of a pregnancy complicated by AKI, reviewing maternal and perinatal outcomes and alerting healthcare professionals to this rare but potentially fatal situation. The patient has given written informed consent.

CASE REPORT

A 31-year-old pregnant woman, G2P1 (previous uneventful pregnancy), irrelevant personal and familial history, was admitted at 25 weeks’ gestation with contractions. Blood analysis revealed serum creatinine of 22 mg/dl and urea of 350 mg/dl, disclosing AKI. Induction of fetal pulmonary maturity and tocolysis were performed. The patient was apyretic and inflammatory parameters and bacteriologic urinalysis were negative. Renal ultrasound showed severe bilateral hydronephrosis. Cervical evaluation revealed a posterior cervix, 50 percent effaced, 1 cm of dilation. Obstetric ultrasound revealed a fetus in centile 33, polyhydramnios (not quantified) and normal medial cerebral artery’s pulsatility index and peak systolic velocity. Within 24 hours, the clinical condition evolved into oliguria, severe metabolic acidosis, hyperkalemia and volume overload and the patient was admitted to the Intensive Care Unit, where hemodialysis was performed.

Invasive ventilation for severe respiratory distress, caused by pulmonary edema and an iatrogenic hemothorax, was required. Double-J stents were placed to treat the obstructive AKI, with progressive normalization of renal function. Two days later, the patient was extubated and transferred to the obstetric ward.

There was progressive clinical improvement and renal function returned to normal, with serum creatinine...
of 0.75 mg/dL at discharge. Urinalysis on day 10 after admission revealed E. coli and Cefuroxime was initiated. No morphologic anomalies were detected on the ultrasound, TORCH infections were excluded, oral glucose test revealed gestacional diabetes and fetal echocardiography was normal. With the purpose of excluding other causes of post-renal AKI, colposcopy and pelvic MRI were performed, showing no alterations. After discharge, on day 19, clinical surveillance was uneventful, no abnormalities.

The patient returned to the hospital at 34 weeks' gestation, with an acute pyelonephritis. Renal ultrasound revealed bilateral hydronephrosis, more severe on the right side, and an anecogenic content in the right renal pelvis. Empiric therapy with meropenem for ten days was administered, after which the patient started prophylactic nitrofurantoin.

Serial ultrasounds were performed and at 34 weeks' gestation, fetal growth restriction (FGR) with hemodynamic redistribution was diagnosed. As fetal situation remained stable – fetus in centile 2, with centralization of circulation and an amniotic fluid index of 11,7cm - labor was induced at week 37, and a normal delivery of a healthy newborn, Apgar Index of 9/10 and 2200g, took place. At discharge, the patient was asymptomatic and had a normal renal function (serum creatinine of 0,81 mg/dL).

The renogram and Computed Tomography (TC) scan, performed 1 month after delivery, demonstrated right pyonephrosis and compromised function of the left kidney. The patient was proposed to right pyeloplasty and left nephrectomy.

**DISCUSSION**

The definition of AKI in pregnancy is controversial. Some authors claim that the formulas usually used to evaluate kidney injury are not validated in this population. Despite the fact that the 24 hours' clearance of creatinine is considered the gold-standard to calculate glomerular filtration rate (GFR) in pregnancy, the preferred method to diagnose AKI is the elevation of serum creatinine. Thus, creatinine level higher than 1mg/dL or a rapid rise of 0,5mg/dL (in 48 hours) should be evaluated.

Post-renal AKI is caused by obstructions that affect the excretory system bilaterally or unilaterally if the contralateral is non-functioning. This obstruction may be caused by blood clots, stones or stenosis of the ureters, the pyeloureteral junction or the ureterovesical junction. Two other causes are cervical cancer and bilateral compression of the ureters by the pregnant uterus.

In this case, after excluding other causes of AKI and after a renal ultrasound in which severe bilateral hydronephrosis was observed, the most likely diagnosis was renal obstruction. Ultrasound, colposcopy and pelvic MRI excluded other causes of obstruction such as cervical cancer or bilateral kidney stones. Therefore, the final diagnosis was obstructive AKI caused by bilateral compression of the ureters by the pregnant uterus. Although this mechanical compression rarely causes AKI, it is a known cause of hydronephrosis in pregnancy. This dilation is seen in 89% of pregnancies but it is symptomatic in only 0,2% to 3% of them. Moreover, progesterone also produces relaxation of the smooth muscle of the ureters, contributing to their dilation.

The following are considered risk factors for obstructive uropathy in pregnancy: twin pregnancy, polyhydramnios, obstruction of solitary kidney, kidney stones and stricture of the ureters. In a first pregnancy, the risk is also higher, for greater levels of progesterone. In this case, the patient had no risks factor, as polyhydramnios most likely was explained by the hydroelectolytic imbalance.

The predominant clinical feature of this entity is acute lumbar pain, which mimics renal colic and irradiates to the ipsilateral iliac region. The pain modifies with body position, diminishing with decubitus to the non-affected side. Other symptoms may be nausea, vomiting and shortness of breath.

In our case, the patient presented with recognized complications of severe AKI: oliguria, metabolic acidosi, hyperkalemia and fluid overload. None of the cases described in the literature presented with such severe complications - only one report described lower extremity edema and none mentioned pulmonary edema. Moreover, no cases with such a high value of serum creatinine (22 mg/dL) were reported, the highest value described being 11,6 mg/dL. In order to better support the diagnosis in this case, it would have been important to know the patient’s creatinine level before pregnancy. However, normalization of renal function after placement of double-J stents reinforces the belief that creatinine level was normal before pregnancy.

The criteria to initiate dialysis in a pregnant patient with AKI are similar to those applying to the non-pregnant population: uremic symptoms, metabolic acidosis,
hypermobility, hypercalcemia and or hypervolemia refractory to medical treatment. Lower values have been proposed, with the aim of decreasing the harmful effects of elevated uremia such as polyhydramnios, FGR and premature labor. Some authors consider that dialysis should be prescribed before the onset of uremic symptoms, significant acidosis, hyperkalemia or fluid overload and that the level of urea should be maintained below 100 mg/dl, so that crossing of the placenta and affection of the fetus would be avoided. Both hemodialysis and peritoneal dialysis may be safely performed during pregnancy.

Two treatment options to handle bilateral compression of the ureters by the pregnant uterus have been described: at term, pregnancy termination is indicated, whereas in preterm pregnancy, placement of ureteral stents or nephrostomy tubes should take place (4–13). In some of the earliest cases described in the literature, in which the patients presented polyhydramnios, the chosen treatment to remove excessive amniotic fluid was amnioreduction. However, improvement of renal function in those cases was only temporary and labor induction was often necessary.

In our case, the patient underwent bilateral placement of double-J stents, a procedure considered effective, safe and inexpensive for the treatment of acute hydronephrosis in pregnancy, including the cases in which AKI occurs. Some authors advise the use of prophylactic antibiotics and increase of fluid intake to prevent infection and occlusion of the stent. In fact, urinary tract infection is the most common complication of this procedure but in our case, antibiotics were not prescribed and the placement of the stents probably constituted the main risk factor for the ultimate pyelonephritis.

In the only review on AKI caused by obstructive uropathy by the pregnant uterus, dating to 1996, 18 cases were included and no maternal deaths were registered; however fetal mortality rate was 33%. Several authors claim that AKI in pregnancy increases the risk of preterm labor, low birth weight and neonatal death. In this case, the diagnosis of FGR was established at 34 weeks’ gestation. AKI and the consequent renal disease were probably the leading contributors to this complication, underlining the importance of considering this clinical entity in the differential diagnosis of AKI in pregnancy.

Of the cases reported, only eight present information on the nephrologic and urologic surveillance after birth. In six cases, return to normal renal function occurred. In one a creatinine level of 1.3 mg/dl remained, and in the other a low function of one kidney persisted, and a nephrectomy was performed. In our case, the patient presented with decreased left kidney function after birth. Since we have no information of previous kidney disease and since this complication did not occur in the previous pregnancy, we may consider that decreased renal function persisted as a consequence of the obstruction.

**CONFLICTS OF INTEREST**

None.

**REFERENCES**


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RECEBIDO EM: 18/08/2020
ACEITE PARA PUBLICAÇÃO: 29/03/2021