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Renal Cortical Necrosis Complicating Postpartum Hemorrhage. Use of Tranexamic Acid: Solution or Problem? Case Report

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Abstract **Case Report**

Renal cortical necrosis associated with postpartum hemorrhage is a rare obstetric complication that can lead to end-stage renal disease. We report a case of a 37-year-old woman, admitted for preeclampsia complicated by in utero fetal death, having presented postpartum hemorrhage complicated by a retroplacental hematoma (RPH). She was treated with tranexamic acid to control bleeding. She subsequently presented a severe acute kidney injury (AKI) requiring hemodialysis with, on computed tomography (CT), the presence of bilateral renal cortical necrosis. A renal biopsy showed cortical necrosis lesions associated with glomerular and vascular thrombotic microangiopathy. There was no recovery of renal function, requiring long-term hemodialysis. Renal cortical necrosis, although rare, can occur in some cases of postpartum hemorrhage. Tranexamic acid would be involved and its use should be done with suspicion. The CT scan is an important diagnostic tool. Renal prognosis is poor.

Keywords: Renal cortical necrosis, postpartum hemorrhage, tranexamic acid, CT scan.

INTRODUCTION

Bilateral renal cortical necrosis (CN) complicating obstetric hemorrhage seems to have disappeared [1]. It was in former time an important cause of acute kidney injury (AKI), usually associated with catastrophic obstetric emergencies [2,3]. Currently, it is rare, especially in resource-rich countries where it is only responsible for 1 to 2% of all AKI cases [3]. It is the result of prolonged renal ischemia or diffuse microvascular damage. However, we report a new case of cortical necrosis complicating postpartum hemorrhage with administration of tranexamic acid.

Our aim is to remind clinicians of this rare but catastrophic complication.

CASE PRESENTATION

A 37-year-old woman from North African was admitted to the Maternity Department for preeclampsia complicated by in utero fetal death and retroplacental hematoma (RPH) in a 38-week pregnancy, with antecedents of gestational hypertension and diabetes since 6 years treated with metformin, initially, and then with insulin since she got pregnant. This was the 4th pregnancy with a history of 3 cases of in utero fetal death.

The delivery was done vaginally, after episiotomy, complicated by hemorrhage with difficult hemostasis requiring the administration of 3g of tranexamic acid.

The clinical examination showed high blood pressure of 170/100mmHg treated with Nicardipine and angiotensin converting enzyme inhibitor (ACE inhibitor), a heart rate of 90 beats/minute, a respiratory rate of 18 cycles/minute. The abdominal examination noted diffuse abdominal pain. The remainder of the examination didn't show anything particular**.**

Immediate postpartum laboratory data showed a HELLP syndrome made up of anemia of 6.6 g/dl) and thrombocytopenia of 85,000/mm3, requiring transfusion of packed red blood cells and fresh frozen plasma, hepatic cytolysis with ALAT of 1045IU/l, and elevated LDH (4022 IU/l). Hemostasis was disrupted (TP of 50%). One day after delivery, the checkup showed acute anuric renal failure (creatinine of 77 mg/l versus 15 mg/l

before delivery) complicated by hyperkalemia (6.5 mmol/l) requiring emergency hemodialysis.

An abdominal CT scan showed a moderately large peritoneal effusion, kidneys of normal size, regular contours with no obstacle or detectable renal pyelocalicial dilatation, with hypodense cortex non enhanced after injection of the iodinated contrast agent, in favour of a renal cortical necrosis (figures 1 and 2).

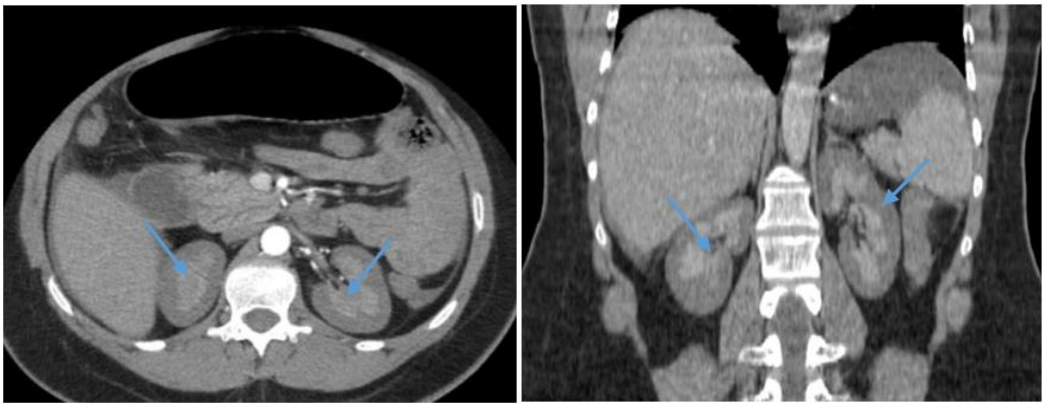

Figure 1 and 2: Images showing hypodense cortex which remained non enhanced in the 2 kidneys

Given the non-recovery of renal function after one month, a renal biopsy was performed, concluding that there were cortical necrosis lesions associated with glomerular and vascular thrombotic microangiopathy.

Currently the patient has chronic end-stage renal failure requiring long-term hemodialysis.

DISCUSSION

The Renal cortical necrosis was first described in 1883 by Friedlander, and since then, it has been reported sporadically [4]. It remains a significant potential complication of postpartum hemorrhage even in resource-rich countries [5]. It can be difficult to diagnose, due to the similarity of symptoms compared with other types of acute kidney injury. Patients with cortical necrosis show sudden onset oliguria or anuria, following an obstetric catastrophe [6], as it is the case for our patient.

According to the literature, the use of tranexamic acid with doses over 2 g would be involved in the development of renal cortical necrosis with severe microangiopathy lesions [7]. In our case, the patient received 3g of tranexamic acid to control obstetric hemorrhage. In addition to tubular necrosis, she presented glomerular and vascular thrombotic microangiopathy. The hypothesis incriminating tranexamic acid as an aggravating factor in our case cannot be excluded. However, a randomized controlled trial including more than 20,000 participants showed no difference in AKI between participants assigned to tranexamic acid compared to placebo [8].

The diagnosis of renal cortical necrosis is often confirmed by an imaging test, such as computed tomography angiography (CTA), which reveal hypoechoic or hypodense areas in the renal cortex [5]. A kidney biopsy can provide, as in our case, the most concise information to conduct the diagnosis.

No specific treatment was proven efficient to better manage this health problem. 30 to 50% of patients develop end-stage renal disease, while others have shown partial recovery of renal function [9]. In our case, there was no recovery of renal function and the patient is treated with a long-term hemodialysis.

CONCLUSION

Although renal cortical necrosis is rare, it remains a possible complication of postpartum hemorrhage. The use of tranexamic acid would be involved, catalysing the occurrence of micro-thrombotic damage. The CT scan is an important tool for the diagnosis. Currently, there is no specific treatment and renal prognosis is still poor.

Conflict of interest: No conflict of interest declared by the authors.

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