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Case Study: Postpartum Hemorrhage Leading to Renal Infarction

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Introduction

A young healthy patient without medical comorbidities developed bilateral renal infarcts after a significant postpartum hemorrhage with associated hypotension. This case is one of a few reported incidents in which significant blood loss associated with a vaginal delivery was sufficient to cause this abnormality. In this case the patient was asymptomatic and the infarcts were found incidentally. This fact begs the question that if this sequalae of hemorrhage may occur more often than detected, as CT scans are not routinely performed on postpartum patients.

Patient presentation

A 19-year-old G1P0 with insignificant past medical history was admitted at 39w1d gestation for induction of labor after having category 2 fetal heart tones. Her induction was unremarkable aside from bradycardia in the second stage of labor requiring a vacuum-assisted delivery and episiotomy. Immediate postpartum course was complicated by a hemorrhage with a total estimated blood loss of 1450 cc. Her hemoglobin dropped from 12.1 g/dL to 5.7 g/dL—a drop in hemoglobin that was unexplained by the amount of blood loss that preceded this change. A CT scan was obtained to rule out retroperitoneal bleeding as a potential reason for the discrepancy. The CT showed a wedge-shaped hypoattenuation concerning for multiple renal infarcts vs pyelonephritis. Urine culture did not indicate the presence of a urinary tract infection, nor did the patient have flank or suprapubic pain indicative of pyelonephritis.

Nephrology was consulted regarding these findings. Recommendations included obtaining urinalysis and kidney doppler studies which were normal. Urology was consulted and recommendations were to follow urinary output and check post-void residual volumes to ensure absence of urinary retention— all of which remained within an acceptable range. Hematology was consulted and concurred that wedge infarcts in kidneys were likely not of thromboembolic origin and given the patient's negative history of thromboembolism, recommended against a thrombophilia workup. Etiology was suspected to be due to hypoperfusion during episodes of significant hypotension that occurred as a result of the patient's significant hemorrhage. The patient had been symptomatic at the time of and shortly after the hemorrhage, reporting some mild dizziness. Her lowest recorded blood pressures being 90s/50s mmHg.

The patient received a total of 4 units of pRBCs and a standard 30 mu of IV Pitocin immediately after delivery, a single dose of intramuscular methergine, and oral methergine for 48 hours. Her Hgb ultimately stabilized at 9.1 g/dL.

Throughout her postpartum period the patient's main complaint was only dizziness during the periods of hypotension, but she never experienced flank pain or urinary symptoms. Since the patient's creatinine remained normal the patient did not require nephrology follow up. She was discharged home on postpartum day # 4 in stable condition.

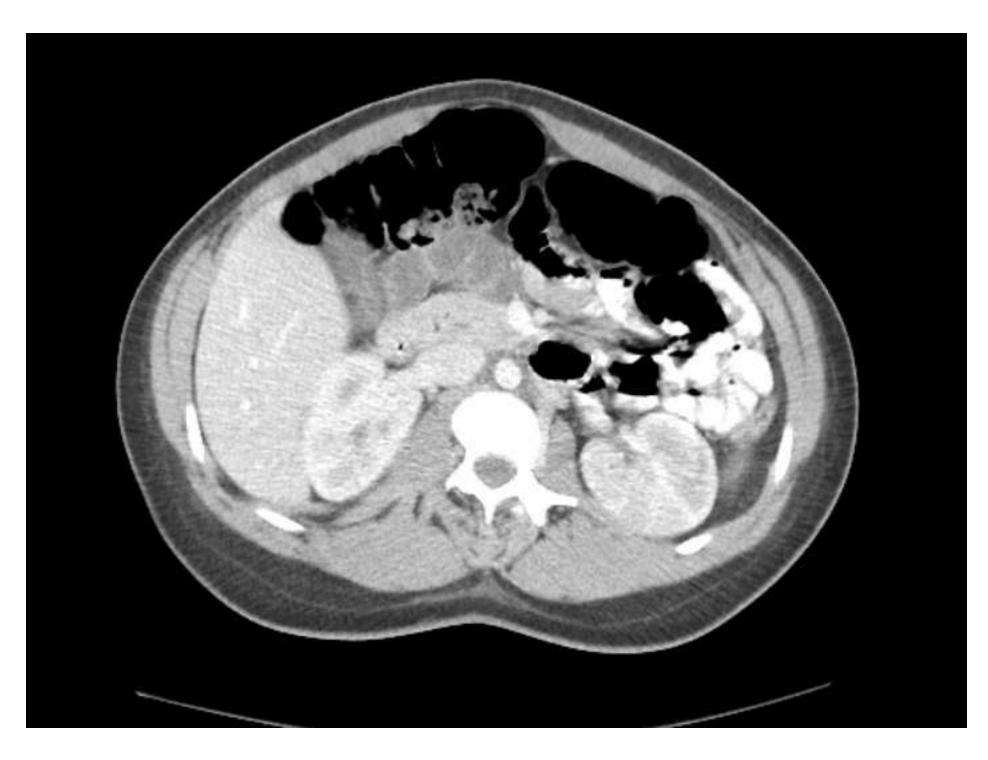




Figure 1&2: Wedge-shaped areas of hypoattenuation involving the left renal parenchyma, concerning for pyelonephritis. Alternatively, this finding can be seen in the setting of multiple renal infarcts.

Discussion

Postpartum hemorrhage is not a causative event typically associated with acute renal infarction. The most commonly cited risk factors for renal infarction include cardiovascular disease, renovascular injury, and hypercoagulability. Other risk factors include autoimmune dysfunction, history of prior cerebral infarctions, recent trauma, infection, or cocaine abuse¹—none of which were applicable to this patient. Given that the majority of patients who suffer from acute renal infarction have significant comorbidities that led to renal infarction,⁴ the risk of long-term outcomes is primarily studied in these patients. There are limited data regarding long-term outcome for young, healthy patients who suffer from acute renal infarction without significant laboratory abnormalities at the time of diagnosis. There have been rare case reports associating a postpartum hemorrhage with a renal infarction,² but given its rarity it is not a complication typically considered by obstetric providers in the setting of a hemorrhage. The fact that this sequelae can result from a postpartum hemorrhage warrants extrapolation to the conclusion that other causes of acute blood loss can have the same impact on renal perfusion leading to infarction. This also underscores the importance of aggressive resuscitation and replacement of blood loss in a timely manner to maintain adequate perfusion and volume status in the setting of acute blood loss anemia and hemodynamic instability.

Acute renal infarction in this instance was discovered incidentally upon CT imaging looking for retroperitoneal hemorrhage. This introduces the possibility that this sequelae of postpartum hemorrhage may be more common than previously thought, as there could be other asymptomatic patients who suffer from undetected renal infarction. Review of this case further indicates that acute hemorrhage in the postpartum period complicated by significant hypotension may indeed be sufficient cause for acute renal infarction. Evidence such as this may prompt clinicians in the future to have greater suspicion for this sequela of postpartum hemorrhage if a patient suddenly develops signs of acute kidney injury without alternative explanation.

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