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Case Report

Internal Medicine Section

Bilateral Renal Cortical Necrosis and Reversible Cerebral Vasoconstriction Syndrome in a Case of Severe Postpartum Haemorrhage

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ABSTRACT

Bilateral Renal Cortical Necrosis (BRCN) is a rare but serious condition characterised by the ischaemic necrosis of both kidneys' cortical tissues. Concurrently, Reversible Cerebral Vasoconstriction Syndrome (RCVS) manifests as a transient constriction of cerebral blood vessels. The fusion of these two rare entities within the context of severe Postpartum Haemorrhage (PPH) poses a unique diagnostic and therapeutic dilemma for healthcare practitioners. This case study documents the intricate and severe complications experienced by a 32-year-old primiparous woman following abruptio placenta with Intrauterine Foetal Death (IUFD). The patient presented with massive vaginal bleeding, leading to an emergency Lower Segment Caesarean Section (LSCS) where bilateral uterine artery ligation and bilateral internal iliac artery ligation were performed to control haemorrhage. Despite initial interventions, the patient's condition deteriorated, leading to signs of shock, haemoperitoneum, sepsis, Acute Kidney Injury (AKI), and cerebral complications. The medical journey unfolded with an urgent re-laparotomy, draining haemoperitoneum and conducting a subtotal hysterectomy. This case highlights the critical importance of a multidisciplinary approach involving obstetrics, nephrology, and intensive care, in managing such complex postpartum complications. Vigilant monitoring, collaborative decision-making, and meticulous postoperative care played pivotal roles. The successful outcome was attributed to early recognition, aggressive supportive measures, and timely interventions, including haemodialysis. The challenges faced underscore the need for accessible obstetric care, awareness about antenatal complications, and community-focused initiatives. Further research is warranted to unravel the intricate connections between PPH, RCVS, and associated complications, enhancing our understanding and improving patient outcomes in similar critical cases.

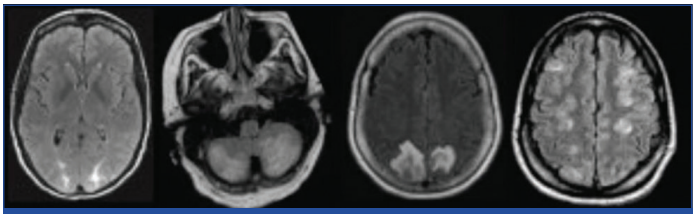
Keywords: Haemoperitoneum, Haemodialysis, Injury, Intrauterine foetal death, Preeclampsia

CASE REPORT

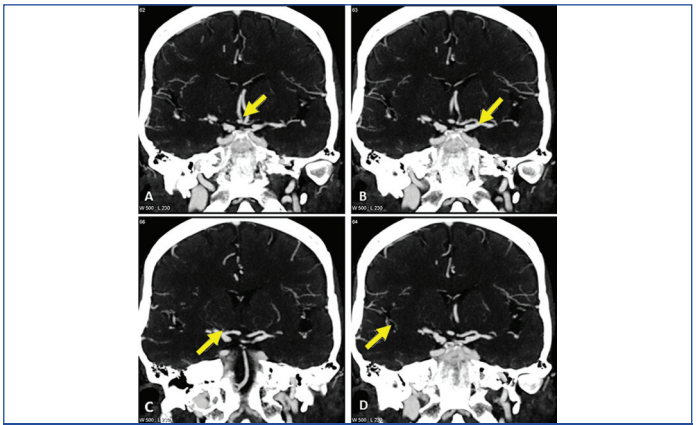
A 32-year-old primiparous woman with a history of previous abortion was diagnosed as a case of abruptio placenta with Intrauterine Foetal Death (IUFD), presenting with massive vaginal bleeding (about 2000 mL blood loss). An emergency Lower Segment Caesarean Section (LSCS) was performed. During the procedure, bilateral uterine artery ligation and bilateral internal iliac artery ligation were executed to control the haemorrhage. Postoperatively, the patient developed signs of shock. Intravenous fluids were administered, and noradrenaline support was initiated along with blood transfusions.

Despite initial interventions, her condition deteriorated. Abdominal distension raised suspicions of haemoperitoneum, confirmed by abdominal and pelvic ultrasound, revealing moderate to gross free fluid in the abdomen and pelvis. An urgent re-laparotomy was performed to drain 1.2 litres of haemoperitoneum and a subtotal hysterectomy was carried out. Postoperatively, she developed sepsis and septic shock, necessitating mechanical ventilatory support and multiple inotropic agents. Laboratory investigations revealed Acute Kidney Injury (AKI) with elevated urea (58.7 mg/dL), creatinine (2.41 mg/dL), and metabolic acidosis (pH 7.06, HCO_3 9.1), hypokalaemia (K 3.23 mmol/L) and a markedly elevated lactate level (48 mmol/L) reflecting severe tissue hypoxia. Following the initial complications, she underwent two cycles of Slow Low-Efficiency Dialysis (SLED). She was diagnosed as stage 3 AKI with Bilateral Renal Cortical Necrosis (BRCN) secondary to Postpartum Haemorrhage (PPH).

A subsequent Magnetic Resonance Imaging (MRI) of the brain done after two days [Table/Fig-1] revealed acute infarcts involving the parietal, posterior temporal, occipital and right frontal cerebral lobes, with multifocal intracranial stenosis of cerebral vessels associated with preeclampsia raising concerns about Reversible Cerebral Vasoconstriction Syndrome (RCVS) [Table/Fig-2]. Meanwhile, Two-Dimensional (2D) echocardiography demonstrated global hypokinesia

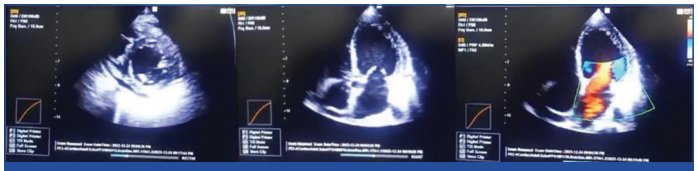


[Table/Fig-1]: Vasogenic oedema (28%). Subcortical crescent-shaped T2- hyper-intense lesions consistent with the posterior reversible encephalopathy syndrome are seen on Fluid-attenuated Inversion Recovery (FLAIR).

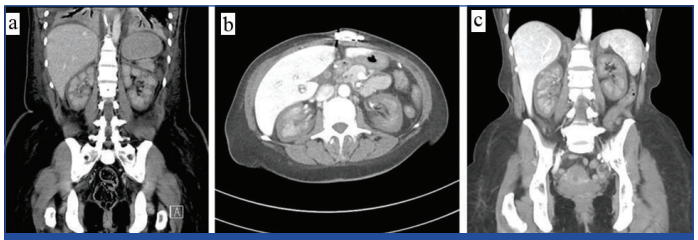


[Table/Fig-2]: Computed Tomography (CT) shows multifocal intracranial stenosis (arrows).

of the left ventricle, moderate mitral regurgitation, mild to moderate tricuspid regurgitation, mild left ventricular systolic dysfunction, and an ejection fraction of 45% [Table/Fig-3]. Furthermore, a Contrast-enhanced Computed Tomography (CECT) of the abdomen and pelvis exhibited non enhancement of the renal cortex, reversal of corticomedullary differentiation in the kidneys bilaterally and positive reverse rim sign, indicating BRCN [Table/Fig-4 a-c].



[Table/Fig-3]: A 2D echocardiography demonstrating global hypokinesia.



[Table/Fig-4]: a) Reversal of normal corticomedullary differentiation with hyperattenuating renal medulla and hypoattenuating renal cortex (reverse rim sign) compatible with renal cortical necrosis. Background of bilateral lower lobe consolidation, anasarca and ascites; b,c) Reversed corticomedullary differentiation in the kidneys bilaterally in keeping with acute cortical necrosis. Moderate volume free fluid throughout the abdomen, most prominent in the pelvis and perihepatic regions. Periportal oedema and mild hepatomegaly.

As her condition remained critical, further investigations unveiled complications. An overdistended gall bladder with mild wall oedema raised concerns about acalculous cholecystitis, possibly aggravated by her critical state. Simultaneously, the presence of mild free fluid in the peritoneal cavity indicated persistent haemoperitoneum and also the imaging studies revealed bilateral moderate pleural effusion, adding to the respiratory challenges.

Throughout treatment, she underwent 11 cycles of haemodialysis, responded well to the therapy, and received continuous monitoring, adaptive treatments, and comprehensive care. Also, supportive measures were provided, including ventilatory support and antibiotics for sepsis, including inj. piperacillin-tazobactam 2.25 g i.v. qid 1-1-1-1 for 14 days, inj. metronidazole 500 mg i.v. bd 1-0-1 for 14 days empirically. After this, she developed ventilator-associated pneumonia with Endotracheal Tube (ET) culture sensitivity reports showing acinetobacter initially and enterobacter, *E.coli* later in the course of her hospital stay. It was treated with nebulisation with colistin 75 mg cba/colistin base activity inhalational bd 1-0-1 for 14 days, inj. meropenem 500 mg i.v. bd 1-0-1 for 14 days and meticulous fluid management. She showed significant improvement. However, due to daily haemodialysis, the patient developed dyselectrolytemia (hypervolemic hypotonic hyponatremia), corrected with slow continuous ultrafiltration and 3% sodium chloride (NaCl). The patient had a few episodes of generalised tonic-clonic seizures secondary to dyselectrolytemia and was placed on levetiracetam to prevent further such episodes. Later in the course of the hospital stay, the patient developed sepsis secondary to ventilator-associated pneumonia and intensive care acquired weakness (critical illness myopathy/polyneuropathy) for which the patient was given antibiotics such as nebulisation with colistin 75 mg cba/colistin base activity inhalational bd 1-0-1 for 14 days, inj. meropenem 500 mg i.v. bd 1-0-1 for 14 days based on culture and sensitivity profile. The patient gradually improved and was weaned off the ventilator, mobilised with near normal recovery of power in bilateral upper limbs and lower limbs. She was discharged and referred to a transplant surgeon for renal transplantation/renal replacement therapy.

DISCUSSION

The present case study examines an extraordinary clinical scenario where a 32-year-old pregnant woman experienced a rare confluence of complications following abruptio placenta with IUFD. The subsequent development of BRCN, RCVS, AKI, haemoperitoneum, and sepsis showcases the intricate challenges faced by healthcare providers in managing such complex cases.

Despite meticulous surgical measures after placental abruption, this patient's clinical course took a dire turn. The development of haemoperitoneum indicated persistent bleeding, leading to a re-laparotomy and subtotal hysterectomy. Such aggressive surgical interventions, although imperative, pose additional risks, potentially contributing to the multiorgan dysfunction observed here. On a broader scale, addressing placenta previa and abruption-related perinatal mortality necessitates a community-focused approach. This includes providing accessible obstetric care with facilities for blood transfusion and iron infusion, along with raising awareness about antenatal care and associated risks in vaginal bleeding during pregnancy [1]. A similar case series reported that all patients had severe PPH and developed rapid onset of AKI, requiring haemodialysis. Diagnosis of renal cortical necrosis was performed four to 33 days following delivery. All patients received tranexamic acid, with a mean cumulative dose ranging from two to 11 g. Despite treatment, none of the patients recovered normal kidney function at six months postpartum [2].

The present case then escalated to stage 3 AKI with BRCN secondary to PPH. AKI in the context of obstetric complications is multifactorial, involving hypovolemia, sepsis, and possibly nephrotoxic medications used in critical care [3]. Severe PPH can present similar features to thrombotic microangiopathy, including thrombocytopenia, elevated lactate dehydrogenase levels, anaemia with identifiable schistocytes, and AKI [4,5]. Moreover, the requirement for SLED reflects the severity of renal impairment and underscores the challenges in managing fluid balance and electrolyte homeostasis. A study conducted among women in labour found that low haematocrit values were associated with the development of renal dysfunction in patients with massive obstetric bleeding. The most frequent causes of postpartum AKI were Haemolysis, Elevated Liver enzymes and Low Platelets (HELLP) syndrome, preeclampsia, PPH, and gestational hypertension [6].

The patient's cerebral complications manifested as acute infarcts with intracranial stenosis of cerebral vessels which is a hallmark of RCVS. The occurrence of RCVS postpartum is exceptionally rare, emphasising the complexity of this case. The mechanisms underlying RCVS remain elusive, possibly involving hormonal fluctuations, endothelial dysfunction, or vasospasm triggered by abrupt changes in blood flow during the peripartum period [7,8]. The presence of severe headaches should alert clinicians to the possibility of cerebral vasospasm, urging timely neuroimaging to guide management. A study reported that prevalence of RCVS in the postpartum period was 11.9%. Of these, 52.7% patients had haemorrhage. The rates of Intracerebral Haemorrhage (ICH) and Subarachnoid Haemorrhage (SAH) were 51.6% and 10.7%, respectively [9]. A similar case report describes a 35-year-old patient who presented with headache after an uncomplicated pregnancy and vaginal delivery. The patient was initially diagnosed with preeclampsia, but subsequent Magnetic Resonance Arteriography (MRA) revealed multifocal vascular narrowing, leading to a diagnosis of RCVS. The study highlights that symptoms of RCVS can mimic or coexist with preeclampsia, and early intracranial imaging such as MRA can aid in timely diagnosis and appropriate management [10]. In another case report, Emilio L et al., emphasised the importance of considering RCVS in the differential diagnosis for postpartum patients presenting with intracranial haemorrhage, as it can be confused with other serious neurological conditions and also to avoid delays and unnecessary interventions in RCVS cases

[11]. The amalgamation of rare complications, ranging from BRCN to RCVS, necessitates careful consideration, and collaborative interventions from various medical specialities.

CONCLUSION(S)

The present case highlights the intricate nature of managing a multitude of rare complications stemming from severe PPH. The favourable outcome observed in this instance can be attributed to an interdisciplinary approach, underscoring the significance of collaborative efforts across various medical specialities. The crucial elements contributing to the patient's recovery encompassed the early identification of complications, implementation of proactive supportive measures, and the prompt initiation of interventions, notably haemodialysis. The challenges encountered underscore the importance for improved access to obstetric care, heightened awareness regarding antenatal complications, and community-oriented healthcare initiatives. Further research is warranted to elucidate the intricate pathophysiological mechanisms linking PPH and RCVS, enhancing our understanding and improving patient outcomes.

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