

Clinical and Radiographic Recovery from Postpartum Cerebral Angiopathy with Conservative Management

Rahul Khera^{1,2*}, Emitseilu K Iluonakhamhe³ and Tiffany R. Chang⁴

¹University College of Medical Sciences and GTB Hospital, New Delhi, India

²Observer, Division of Neurocritical Care, The University of Texas Medical School at Houston, USA

³Emitseilu K. Iluonakhamhe, Neurocritical Care Fellow, The University of Texas Medical School at Houston, USA

⁴Tiffany R. Chang, Assistant Professor, Department of Neurology & Neurosurgery, The University of Texas, USA

Abstract

A 31 year old woman presented with severe frontal headache and blurry vision about 6 days after her normal delivery. Her blood pressure was 193/111 mmHg and her liver and renal function tests and coagulation studies was normal. Her CT Head showed bifrontal subarachnoid hemorrhage (SAH) and MR Angiogram showed characteristic "beaded" appearance of multiple branches of the middle and anterior cerebral arteries bilaterally. The patient was treated with nimodipine and normal saline infusions. Her repeat MRA after one week showed resolution of SAH with normal anterior, middle, and posterior cerebral arteries. The patient was diagnosed with Postpartum cerebral angiopathy.

Keywords: Postpartum Cerebral Angiopathy; RCVS; MRI; CT; Headache

Introduction

Postpartum cerebral angiopathy (PCA) is a form of reversible cerebral vasoconstriction syndrome (RCVS) which occurs in women, most often within days to a few weeks of delivery [1]. We present a case of postpartum angiopathy in a patient within one week of her normal delivery. This case report highlights that the patient improved, both clinically and radiographically, 8 days after her initial presentation with conservative treatment measures.

Case Presentation

The patient is a 31 year-old gravida 4, para 4 Hispanic woman who presented with severe frontal headache, blurry vision, and nausea. She had an uncomplicated spontaneous vaginal delivery 6 days prior. She had a past medical history of anxiety, was not taking any medications, and there was no history of alcohol, tobacco or illegal drug use. At initial presentation, she was afebrile with a blood pressure of 193/111 mmHg. She was alert and oriented to time, place, and person. Her eyes were open and she was following commands with full strength in all four extremities. Her pupils were 3 mm and equally reactive to light and extra ocular movements were intact. Her initial labs demonstrated white blood cell count 11,400, hemoglobin 11.9 g/dl, platelet count 244,000 normal renal and liver function, and normal coagulation studies. Urinalysis was negative for protein (Table 1).

Computed tomography (CT) of the head was performed which showed a moderate amount of diffuse bifrontal subarachnoid hemorrhage (SAH). There was no evidence of parenchymal hemorrhage or ventricular hemorrhage (Figure 1). CT angiogram showed areas of vessel irregularity in the left M2 segment of the middle cerebral artery and right anterior cerebral artery; a fetal right posterior cerebral artery was noted and the neck vessels were normal (Figure 2a and 2b).

Magnetic resonance (MR) imaging of the brain confirmed bilateral subarachnoid hemorrhages (Figure 3) and MR Angiogram showed characteristic "beaded" appearance of multiple branches of the middle and anterior cerebral arteries bilaterally (Figure 4). MR Venogram showed patent deep cerebral veins and major venous sinuses.

Blood pressure was lowered to the normal range with intravenous labetalol. She was started on nimodipine and normal saline to maintain

euvolemia. Over the next two days, her headache markedly improved and her neurologic exam remained normal. Serial imaging showed no further progression of hemorrhage. She was discharged to home in stable condition. One week after her initial presentation, she followed up with her primary care physician in clinic with complaints of only a mild headache. Repeat CT brain and MRA at that time showed resolution of SAH with normal anterior, middle, and posterior cerebral arteries.

Discussion

PCA can present with headache, focal deficit, visual disturbance, encephalopathy and seizure [2]. This disorder is largely characterized by the multifocal vasoconstriction of cerebral arteries associated

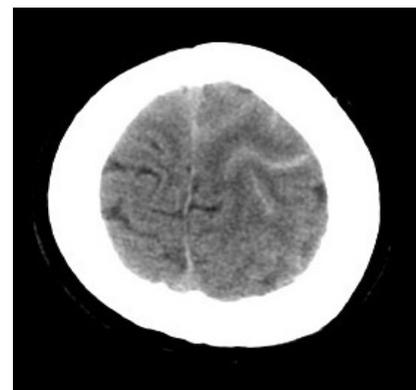


Figure 1: Brain CT showing diffuse bifrontal subarachnoid haemorrhage.

*Corresponding author: Rahul Khera, Division of Neurocritical Care, The University of Texas Medical School at Houston, USA 6431 Fannin Street, Houston, TX 77030, USA, Tel: 214-648-3111; E-mail: rkhera9@yahoo.com

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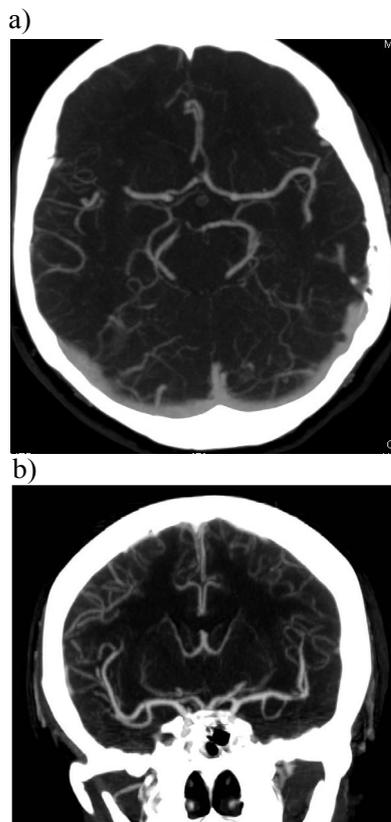


Figure 2: CT angiogram showing areas of vessel irregularity in the left M2 segment of Middle Cerebral Artery (a) and right anterior cerebral artery (b).

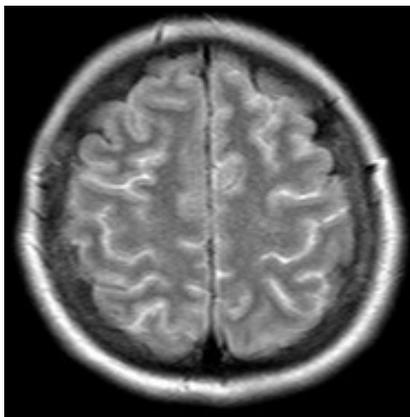


Figure 3: MRI Brain with Flair sequence showing bilateral subarachnoid hemorrhages.

with subsequent vaso-occlusive phenomena. Patients may have uncomplicated pregnancies or they may have pre-existing conditions such as coagulopathy, migraine headaches, gestational hypertension and autoimmune disease which may contribute [2]. Pertinent drug history should always be taken as vasospasm has been reported in patients taking medications for migraine [3]. Vasospasm is thought to be the underlying vascular pathology, although the exact molecular mechanism of PCA is unknown.

In addition to the symptoms above, patients may present with transient neurologic deficits, ischemic stroke, intracerebral hemorrhage,

or SAH. PCA can also present rarely with arterial dissection [4,5]. Peripartum SAH occurs during the postpartum period in more than half of patients and is less likely to be aneurysmal than in the non-pregnant population [6]. As PCA may result in severe neurological complications, -early neuroimaging with a vascular imaging study (e.g. CT, MR, or conventional angiogram) should always be considered [2]. Doppler ultrasound of the carotids could also be done. In a 5 year follow up study on 112 patients with dolichocarotids (carotid tortuosity, kinking or coiling) found on doppler, 24% patients had a TIA while 14% had an ischemic stroke on the side opposite to the dolichocarotid [7]. Dolichocarotid is more common in patients with non-ischemic dilated cardiomyopathy and patients having arterial hypertension [8].

The differential diagnosis of a sudden severe headache includes aneurysmal subarachnoid hemorrhage, reversible cerebral vasoconstriction syndrome, arterial dissection, cerebral venous thrombosis, pituitary apoplexy, intracranial hemorrhage, ischemic stroke, reversible posterior leukoencephalopathy, spontaneous intracranial hypotension, and central nervous system infections [9].

It is important to distinguish PCA from primary cerebral vasculitis. Patients with primary cerebral vasculitis may have a milder headache with gradual clinical progression; they may also present with focal deficits, stroke, seizures, and radiological features common to PCA. Although both conditions are associated with “beading” on angiography, the hallmark of PCA is the reversibility of this finding with time [10]. Initial vascular imaging in PCA may be negative at the time of symptom onset [11]. Management of primary cerebral vasculitis may include lumbar puncture, brain biopsy, and immunosuppressive therapy.

Different strategies have been used for the management of PCA varying from conservative to aggressive. PCA has been managed with volume expansion using normal saline solution along with albumin [12]. Calcium channel blockers are often used in the management of vasospasm. Nimodipine (60 mg orally every 4 hours) in addition to hypervolemic therapy has been shown to decrease cerebral ischemia [10]. In patients who have a lack of tolerance to nimodipine [13,14] or who do not respond to therapy, intra-arterial (IA) nicardipine has been shown to improve cerebral blood flow [15], especially in patients with distal vessel vasospasm on cerebral angiography [16]. Nicardipine may also be a more potent vasodilator than nimodipine [17]. Proximal vessel vasospasm can be treated with IA nicardipine and/or angioplasty [16]. Angioplasty may result in greater improvement in radiographic vasospasm compared with IA nicardipine alone [17], and has been shown to improve cerebral blood flow in the management of symptomatic vasospasm. Transcranial Doppler may be considered as

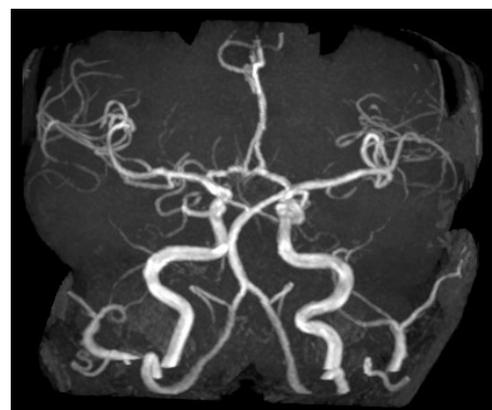


Figure 4: MR angiogram showing beading of multiple middle cerebral artery and anterior cerebral artery branches.

Age	31
Race	Hispanic
Past medical history	Gravida 4, para 4 Anxiety
Social history	Negative for alcohol, smoking, or illicit drug use
Blood pressure	193/111
Urinalysis	pH 6.0, specific gravity 1.003, trace blood, negative ketones, negative protein
Urine pregnancy	Negative
Urine toxicology	Negative
Blood urea nitrogen	19 m/dL
Creatinine	0.8 mg/dL
White blood cell count	11,400
Hemoglobin	11.9 g/dl
Platelet count	244,000

Table 1: Baseline patient characteristics.

a non-invasive method of monitoring for vasospasm and response to therapy [18,19].

Our patient was managed conservatively with intravenous fluids, blood pressure control, and oral calcium channel blocker. She completely recovered without any neurologic deficit. Other patients have been reported to have persistent neurological deficits despite radiographic resolution of vasospasm [20]. In a review of 18 patients with PCA, around 50% had a full neurological recovery, 28% had residual neurological symptoms, and in-hospital mortality was 22% [2]. Although patients should be monitored subsequent pregnancies, recurrent PCA is uncommon [18,21]. It is unknown why some patients have complete recovery while others have long term complications, but it is possibly related to the extent of the initial injury and the patient's response to treatment.

Conclusion

Postpartum cerebral angiopathy may lead to nonaneurysmal subarachnoid hemorrhage during the peripartum period. In some cases, patients may experience a complete recovery with conservative treatment including intravenous hydration, blood pressure control, and calcium channel blockers.

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