A Case of Postpartum Cerebral Angiopathy

Keiko Kochi¹, Takao Hidaka¹, Kuniaki Yasoshima¹, Kenji Yoneda², Kazunori Arai² and Masanori Kurimoto³

1Department of Obstetrics & Gynecology, Kurobe City Hospital, Japan
2Department of Radiology, Kurobe City Hospital, Japan
3Department of Neurosurgery, Kurobe City Hospital, Japan

Introduction

The incidence of ischemic stroke during pregnancy and postpartum is very low; however, it could be a serious event for mothers and infants. Once it does occur, many concerns arise about the safety of the mother and fetus in relation to common diagnostic tests and therapies. Brain scanning might reveal pathological results in spite of a normal neurological examination. With neurological examination and brain scanning, it may be possible to diagnose and treat severe complications that may otherwise result in maternal mortality [1].

Postpartum Cerebral Angiopathy (PCA) is a reversible clinical-radiological syndrome, characterized by the acute onset of severe headache, focal neurologic deficits, and reversible cerebral segmental vasoconstriction [2-6]. It is diagnosed by angiography, which demonstrates multifocal segmental narrowing in large and medium-sized cerebral arteries, with a similar appearance to vasculitis [2-8]. The process is generally self-limiting; with the resolution of angiographic abnormalities within 4-12 weeks and typically complete resolution of symptoms [1,4]. However, owing to its association with both infarction and hemorrhage, PCA does carry a risk of morbidity and mortality [2,9].

Patients with PCA generally presented the acute onset of severe headache; however, we present a rare case of PCA involving presentation with paresis but without headache.

Case Report

A 34-year-old woman, gravid 1, para 0, was admitted to our hospital for pregnancy-induced hypertension. At 39 weeks of gestation, her blood pressure was 158/86 mmHg, and she presented headache. She had no previous history of heart disease, migraine, diabetes mellitus, or collagen-vascular diseases. At bed rest, although her blood pressure normalized, her headache persisted. On the fourth admission day, a cerebral Computed Tomography scan (CT) was performed, with no abnormal findings, and she is currently well without headache or paresis.

Figure 1: MRI 4 hours after onset
Both T2-weighted and FLAIR imaging showed a hyperintense area (arrows) in the right frontal-parietal lobe. Findings are consistent with acute ischemic stroke.

Figure 2: MRA 2 days after onset
MRA performed 2 days after onset: occlusion of the bilateral proximal middle cerebral arteries (arrows).

*Corresponding author: Keiko Kochi, 1108-1 Mikkaichi, Kurobe-shi, Toyama 938-0031, Japan, Tel: +81-765-54-2211; Fax: +81-765-54-2962; E-mail: yonezawa@med.kurobe.toyama.jp

Received January 15, 2014; Accepted February 10, 2014; Published February 17, 2014

Citation: Kochi K, Hidaka T, Yasoshima K, Yoneda K, Arai K et al. (2014) A Case of Postpartum Cerebral Angiopathy. Reprod Syst Sex Disord 3: 130. doi: 10.4172/2161-038X.1000130

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Hemorrhage [2,9]. Evaluations of both the incidence and risk of stroke period are associated with an increased risk of stroke and cerebral and mortality. The American Maternal Mortality Collaborative placed used to treat postpartum hemorrhage [5,6]. Ducros et al. reported intake of vasoconstrictors, mostly ergot alkaloids, which are commonly first week after delivery [5]. In 50-70% of cases, it is associated with the disease mechanism in eclampsia [4,8,14-16]. It is unclear whether vasospasm, vasculitis, or another mechanism or to segmental and multifocal arterial constriction and dilatation [4,5,8].

A diagnosis of PCA is made with angiography. It demonstrates multifocal segmental narrowing and dilatation (string of beads) in large and medium-sized cerebral arteries [2-8]. Ducros et al. reported that noninvasive angiography (MRA or CT angiography) was 80% sensitive in their series compared with the gold standard of catheter angiography [5]. Brain MRI may show areas of T2/FLAIR hyperintensity at any location, especially in watershed areas between vascular territories [8]. By definition, the process is generally self-limiting, with the resolution of angiographic abnormalities within 4-12 weeks and typically complete resolution of symptoms [2,5,14]. However, the first angiogram, including MRA and CT Angiography (CTA), regardless of the modality, may be normal if performed very early, within 4-5 days of the onset of symptoms; therefore, if the first MRA or CTA is normal, a second angiogram a few days later may be diagnostic [3,5].

In our case, she was diagnosed as PCA, without headache on the basis of the following: 1) cerebral angiography, including MRA, showed multi-segmental areas of narrowing and dilatation of arteries supplying the hemisphere, 2) exclusion of systemic inflammatory disorders such as infectious vasculitis and collagen-vascular diseases, and 3) resolution of both MRA abnormalities and paresis in only a few months.

Cerebral arterial vasoconstriction can be seen in the postpartum period. The obstetric neurologic literature has described a similar entity, given various labels [5,6,8,14], including PCA, postpartum angiopathy, postpartum angitis, and puerperal vasospasm. This syndrome is probably still underdiagnosed or goes unnoticed, in cases for obstetricians to recognize this particular disorder. We should suspect RCVS in patients with thunderclap headache, with or without other neurologic deficits. However, we should be also aware that PCA could occur only with neurologic deficits, differing from RCVS in non-pregnant cases.

References


