



Contents lists available at ScienceDirect

Taiwanese Journal of Obstetrics & Gynecology

journal homepage: www.tjog-online.com

Research Letter

Intracranial hypertension associated with postpartum hemorrhage

Bo-An Chen^{*}, Chuo-Yu Lee

Department of Neurology, Mackay Memorial Hospital, Taiwan

ARTICLE INFO

Article history:

Accepted 26 January 2019

Dear Editor,

Headaches are common throughout the postpartum period; it can be primary or secondary. Most headaches are primary (i.e., migraine, and tension-type headache), while secondary headaches account for less than 10% of cases and increase in incidence in the last trimester and postpartum period [1]. The common causes of secondary postpartum headaches include intracranial hypertension (IH), eclampsia, reversible cerebral vasoconstriction syndrome, cerebral venous thrombosis (CVT), pituitary apoplexy, and post-dural puncture headache. Severe anemia can also produce IH and may be life-threatening, but it is treatable; neurological deficit can be reversible with timely treatment.

This is the first report of postpartum IH as a complication of postpartum hemorrhage (PPH). A previously healthy 38-year-old woman, gravida 5 para 0; who had an artificial abortion, 3 spontaneous abortions, and an intrauterine pregnancy at 40 + 5 weeks was admitted for labor induction. After vaginal delivery, primary PPH due to uterine atony and cervical laceration occurred immediately, followed by delayed PPH caused by retained placenta. Her serum hemoglobin level decreased from that at baseline, from 12.8 g/dL to 7.1 g/dL within 36 h, and to 5.8 g/dL 6 days after delivery. She reported having a headache after delivery, with characteristics of increased intracranial pressure (IICP), which was triggered by lying down and limited to bilateral lateral gazing on neurological examination. A cerebrospinal fluid (CSF) examination showed an opening pressure of up to 27 cm of water with normal constituents. Her body mass index before pregnancy was 19.3 kg/m², and she gained 14 kg before delivery. She denied any previous

history of anemia, autoimmune disorders, polycystic ovary syndrome, eclampsia, and illegal drug use or vitamin A exposure. A series of work-ups for IICP included a magnetic resonance (MR) scan of the brain and MR venography (Fig. 1). Her serum pituitary gland-secreted hormone level, fundoscopy result, and serum iron/ferritin levels were normal. The treatment strategy included a prescription of acetazolamide, blood transfusion, and removal of retained placenta. Her headache subsided gradually without any neurological sequelae.

The hint that prompted us to investigate for a secondary etiology was the new onset of postpartum headache and IH in a non-obese woman. Several conditions may produce IH, such as CVT, central nerve system infection, mass lesion, anemia, and vitamin A overdose. In anemia-related IH, most cases of anemia are attributed to iron deficiencies [2], while a few cases presented with hemolytic anemia [3], sickle cell disease [4], and Fanconi anemia [5]. The pathophysiology for the development of IH in anemic patients remains unclear. Some authors hypothesized an increase in the amount of CSF produced in response to anemia, while other theories suggest the possibility of altered cerebral hemodynamics and increased cerebral permeability to compensate for anemia and hypoxia. The mainstream therapy is treatment of anemia, and other therapies include acetazolamide, optic nerve sheath fenestration, and CSF shunting.

In conclusion, postpartum hemorrhagic anemia can produce IH. A series of examinations to exclude other secondary IHs is an indispensable part of the diagnosis. Treatment of the underlying anemia plays a crucial role and timely treatment can prevent long-term sequelae.

^{*} Corresponding author. Department of Neurology, Mackay Memorial Hospital, No. 92, Sec. 2, Zhongshan N. Rd, Zhongshan Dist, Taipei City, 104, Taiwan.
E-mail address: s71238@msn.com (B.-A. Chen).

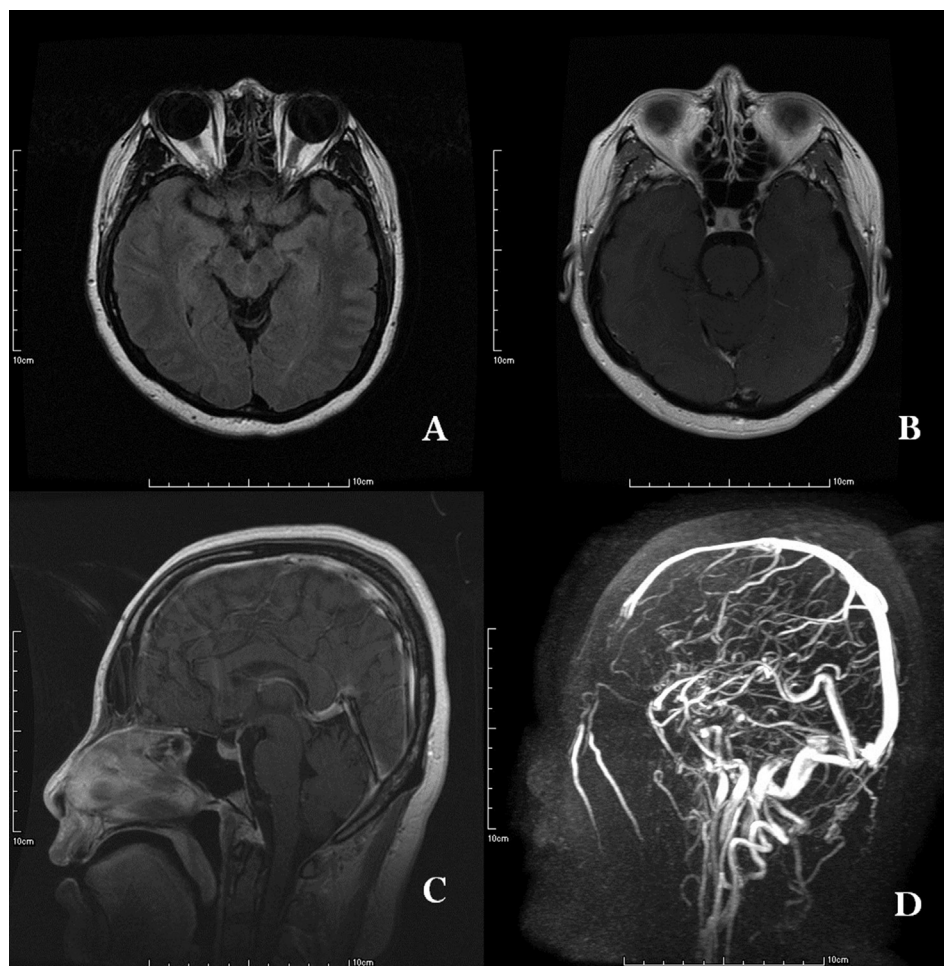


Fig. 1. (A) T2 flair: there is no evidence of structure lesion. (B) and (C) T1 enhancement: pituitary gland, bilateral cavernous sinus are normal, and no abnormal meningeal enhancement. (D) MR venography: there is no evidence of cerebral venous thrombosis.

Conflicts of interest statement

The authors have no commercial associations or sources of support that might pose a conflict of interest.

References

- [1] O'Neal MA. Headaches complicating pregnancy and the postpartum period. *Pract Neurol* 2017;17:191–202.
- [2] Bioussé V, Rucker JC, Vignal C, Crassard I, Katz BJ, Newman NJ. Anemia and papilledema. *Am J Ophthalmol* 2003;135:437–46.
- [3] Vargiami E, Zafeiriou DI, Gombakis NP, Kirkham FJ, Athanasiou-Metaxa M. Hemolytic anemia presenting with idiopathic intracranial hypertension. *Pediatr Neurol* 2008;38:53–4.
- [4] Segal L, Discepolo M. Idiopathic intracranial hypertension and sickle cell disease: two case reports. *Can J Ophthalmol* 2005;40:764–7.
- [5] Tavit B, Karakurt N, Bozkaya I, Culha V, Mehmet Azk F, Tunç B. Pseudotumor cerebri in a Turkish boy with Fanconi anemia. *J Pediatr Hematol Oncol* 2012;34:296–7.