Reversible posterior leukoencephalopathy syndrome after spinal anesthesia

Satoru Takeuchi · Kimihiro Nagatani ·
Naoki Otani · Hiroshi Nawashiro · Katsuji Shima

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Dear Editor,

We read with interest the recent article, by Yamada SM, Kitagawa R, Teramoto A (Neurol Sci 32:165–168, 2011) a case of reversible posterior leukoencephalopathy syndrome with acute hypotension. The authors reported a rare case of reversible posterior leukoencephalopathy syndrome (RPLS), who demonstrated hypotension when her symptoms occurred. The patient had undergone cholecystectomy 4 days prior to presentation and had been receiving ropivacaine via an epidural catheter. Her symptoms were mild headache and blurred vision with hypotension. Brain magnetic resonance imaging (MRI) demonstrated compatible findings with RPLS, and magnetic resonance angiography showed severe vasoconstriction of the cerebral arteries. The authors hypothesized that arterial endothelial injury resulting from toxic drug exposure might result in transudation of fluid from blood vessels causing vasogenic brain edema and RPLS. We agree with the author's opinions and wish to provide further comment. Several studies have suggested a possible pathogenic relationship between cerebrospinal fluid (CSF) hypovolemia, secondary to inadvertent dural puncture, and RPLS [1, 2], and the potential mechanisms of RPLS after spinal analgesia were suggested as venous stagnation due to venous engorgement or impaired drainage of deep venous system due to a functional stenosis of the vein of Galen entering the straight sinus produced by brain sagging [3]. These conditions may increase venous pressure with passive overdistension of cerebral vessels, leading to

interstitial extravasation of proteins and fluids in verte-brobasilar vascular territory, which may cause the typical MRI signs of RPLS [1]. The diminished volume of CSF can also collapse the ventricle, and this traction may cause mechanical stimulation on the arterial wall and potentially induce vasospasm [4, 5]. Based on these findings, we suggest that CSF hypovolemia should be considered a cause of RPLS, and more careful investigation for the presence of pachymeningeal enhancement and brain sagging [6] is required, particularly in RPLS patients after spinal anesthesia, as the characteristics of headache may be changed by anesthesia.

References

1. Pugliese S, Finocchi V, Borgia ML, Nania C, Della Vella B, Pierallini A, Bozzao A (2010) Intracranial hypotension and PRES: case report. *J Headache Pain* 11:437–440
2. Ho CM, Chan KH (2007) Posterior reversible encephalopathy syndrome with vasospasm in a postpartum woman after postdural puncture headache following spinal anesthesia. *Anesth Analg* 105:770–772
3. Savoirdo M, Minati L, Farina L, De Simone T, Aquino D, Mea E, Filippini G, Bussone G, Chiapparini L (2007) Spontaneous intracranial hypotension with deep brain swelling. *Brain* 130:1884–1893
4. Mercieri M, Mercieri A, Paolini S, Arcioni R, Lupoi D, Passarelli F, Pinto G, Celleno D (2003) Postpartum cerebral ischaemia after accidental dural puncture and epidural blood patch. *Br J Anaesth* 90:98–100
5. Arutiunov AI, Baron MA, Majorova NA (1974) The role of mechanical factors in the pathogenesis of short-term and prolonged spasm of the cerebral arteries. *J Neurosurg* 40:459–472
6. Schievink WI (2006) Spontaneous spinal cerebrospinal fluid leaks and intracranial hypotension. *JAMA* 295:2286–2296

S. Takeuchi (✉) · K. Nagatani · N. Otani · H. Nawashiro · K. Shima

Department of Neurosurgery, National Defense Medical College, 3-2 Namiki, Tokorozawa, Saitama 359-8513, Japan
e-mail: drne776@ndmc.ac.jp