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Letter to the Editor

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Letter to the Editor: Commentary on White Cord Syndrome: A Reperfusion Injury Following Spinal Decompression Surgery (*Korean J Neurotrauma* 2022;18:380–386)

Byung-Jou Lee

Department of Neurosurgery and Neuroscience, and Radiosurgery Hybrid Research Center, Inje University Ilsan Paik Hospital, Inje University College of Medicine, Goyang, Korea

▸ See the article "White Cord Syndrome: A Reperfusion Injury Following Spinal Decompression Surgery" in volume 18 on page 380.

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Address for correspondence:
Byung-Jou Lee
Department of Neurosurgery and Neuroscience, and Radiosurgery Hybrid Research Center, Inje University Ilsan Paik Hospital, Inje University College of Medicine, 170 Juhwa-ro, Ilsanseo-gu, Goyang 10380, Korea.
Email: lbjguni@hanmail.net

ORCID iDs
Byung-Jou Lee
https://orcid.org/0000-0002-4030-3618

Conflict of Interest
The author has no financial conflicts of interest.

Dear Editor,

Thank you for the opportunity to review the paper "White Cord Syndrome: A Reperfusion Injury Following Spinal Decompression Surgery."[13]

Spinal cord reperfusion injury is defined as a sudden expansion of the compressed cord after decompression surgery and an acute increase in the blood supply to the cord, leading to disruption of the blood-spinal cord barrier and eventually causing acute neurologic deterioration.[4,8] Preferred diagnostic criteria for spinal cord reperfusion injury include (1) severe preoperative spinal cord compression, (2) surgical decompression, and (3) motor and sensory dysfunction occurring within 3 hours postoperatively. Furthermore, it should not include postoperative surgical disease that may require additional treatment and surgery, such as hematoma, residual cord compression lesion, direct intraoperative cord trauma, and spine displacement.[2,6,8] The postoperative magnetic resonance imaging (MRI) study is most helpful in diagnosing spinal cord reperfusion injury, and it shows a high signal change indicating an expanded cord compared to that before surgery and cord swelling/edema in the T2 weight image without extrinsic pathology.[9,11] 'White cord syndrome' was first described in 2013 by Chin et al.,[3] considering a high intramedullary signal change, an imaging characteristic of spinal cord reperfusion injury in a T2 weighted sagittal image of a postoperative MRI study. According to Seichi et al.,[12] among 114 patients who underwent laminoplasty for cervical myelopathy, only 4 (3.5%) had symptomatic postoperative magnetic resonance changes. Additionally, another study reported that the incidence rate of cord reperfusion injury was 2%–5.7% in cervical decompression surgery and 14.5% in thoracic decompression, which was higher in thoracic decompression than in cervical decompression.[5,19] The mechanism of cord injury due to reperfusion after decompression is not yet fully understood. However, a widely acceptable pathophysiology of spinal cord reperfusion injury is that, after decompression surgery, abrupt cord expansion and a sudden increase in blood supply trigger an inflammatory cascade, releasing oxygen free radicals, which injure nerve cells with a borderline status and cause permanent neurological damage. Other etiologies include: altered perfusion and grey matter dislodged

by internal recoil of spinal structures after decompression, small artery or anterior spinal artery occlusion, microthrombi, and direct injury to blood flow.[16,17] Many previous studies have been conducted to understand the mechanism underlying the relationship between decompression and spinal cord reperfusion injury. Yang et al.[18] reported that surgical decompression increased the expression of inflammatory cytokines in an animal model of chronic severe spinal cord compression. Subsequent studies also reported that decompression increased inflammatory cytokines 1.5–2 fold and caused neurological deficits.[14] An additional rodent study analyzed the structural changes of the spinal cord according to changes in blood flow after decompression. In the 8-oxoG DNA staining, which reveals the degree of reactive oxygen species and DNA damage repair, a large amount of 8-oxoG DNA was observed in decompressed rats, suggesting that many reactive oxygen species were affected. Ultimately, the results of this study demonstrated that reperfusion after surgical decompression causes persistent oxidative damage to nerves.[7] Then, when a spinal cord reperfusion injury occurs during decompression, there is not much we can do to manage it. However, based on the mechanism of occurrence of reperfusion injury, steroids with an anti-inflammatory effect can be used preferentially. In fact, the use of steroids in clinical studies reduced the neurological deficit caused by reperfusion injury.[15] The dose of steroid used for spinal cord reperfusion injury has not been determined, but the dose used for traumatic cord injury, which has been widely studied, is generally used. Results from previous studies indicate that the mean artery pressure (MAP) goal should be increased by at least 10 mmHg when somatosensory evoked potentials are decreased in spinal cord injury,[1,17] and the current recommendation for blood pressure management after spinal cord injury is that MAP be maintained at 85–90 mmHg for 5–7 days.[10] Despite the low incidence of spinal cord reperfusion injury, complications caused by this injury are catastrophic, and therefore spinal surgeons make efforts to reduce its risk. A known effective method is the use of preoperative remote ischemic preconditioning. This is done in 3 cycles of upper right limb ischemia for 5 minutes with 5-minute intervals of reperfusion between each cycle.[5]

Accordingly, this paper is valuable in that the study reports on reperfusion injury that can occur very rarely after cervical decompression, and because it recommends a management method for when reperfusion injury occurs. However, I would like to comment on the diagnostic judgement of spinal cord reperfusion injury. As mentioned above, it is known that reperfusion injury can occur when decompression is performed rapidly in a state of "severe" cord compression. The mechanism underlying this condition is presumed to involve the exacerbation of inflammatory factors such as free radicals or cytokines, which spread to the injury site by perfusion, causing damage to nerve cells in the cord penumbra site. However, in case 1, there is a change in the cord signal on the MRI, but it is not severely compressed; hence, it is necessary to explain why this case is that of a spinal cord reperfusion injury. I hope my comment will be helpful toward the authors' research.

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