Letter to the Editor: Risks Of Neuraxial Anesthesia/Analgesia In Diabetic Patients

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Abstract

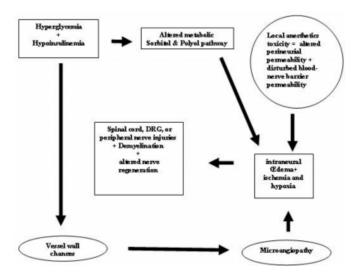
TO THE EDITOR

I read with interest the case report by Poupat KU et al ($_1$) published in the Internet Journal of Anesthesiology. In fact, the authors reported two cases of paraplegia following epidural analgesia. The authors focused the cause of this paraplegia on spinal cord ischemia without referring to multiple risk factors in their patients especially diabetes mellitus. Spinal cord involvement in diabetes mellitus has been known since the 1930s ($_2$). Few articles reported mild to severe neurological complications following neuraxial anesthesia/analgesia in diabetic patients ($_3$, $_4$, $_5$).

Evidence of spinal cord abnormalities in diabetes mellitus comes from electrphysiological (prolonged central conduction time)(₃), radiological studies (reduced mean spinal cross-sectional area in diabetic patients)($_{4}$) and necropsies (similar microangiopathy in diabetic peripheral neurves and spinal cord $(_{2},_{6})$. In addition, the observation that electrical spinal-cord stimulation is ineffective in relieving neuropathic pain in diabetic patients with severe loss of vibration and joint-position sense is suggestive of spinal cord involvement in the disease process (6). This would suggest that the same pathogenic mechanisms in diabetic patients may involve the peripheral nerves and the spinal cord.(2) In addition, dorsal root ganglia (DRG) may be obvious targets in diabetes mellitus by virtue of their barrier properties, high metabolic requirements and blood flow $(_7)$. In diabetes mellitus probably two mechanisms may cause peripheral or central neural edema-ischemia, the microangiopathy (vascular hypothesis) and the altered polyol sorbitol pathway (metabolic hypothesis) (3, 7). Furthermore, local anesthetics toxicity may also lead to intraneural edema and ischemia $(_2)$. The addition of both mechanisms may result in transient or permanent nerve injury in diabetic patients (Fig 1).

Figure 1

Figure 1: Simplified scheme illustrating the mechanism of peripheral nerve, spinal cord, DRG involvement in diabetes mellitus and the relation of local anesthetics toxicity.



Al-Nasser B (3) reported a case of post-epidural bilateral lower limb paresthesia and pain in a diabetic patient. Other authors (4,5) reported conus medullaris injury and lower limb deficit following spinal anesthesia or epidural analgesia in diabetic patients. They suggested that nerve injury may be related to local anesthetics or epinephrine toxicity on diabetic nerve fibers along with other factors such as perioperative position, associated alcohol neuropathy, and perioperative hypotension $(_{3,4,5})$. Although it is difficult to link between ischemia - edema produced by local anesthetics toxicity and that seen in diabetic myelopathy and radiculopathy probably the addition of both mechanisms may cause the emergence of transient or permanent nerve injury after neuraxial anesthesia. Poupat KU et al (1) did not consider the role of local anesthetics toxicity, diabetic myelopathy/radiculopathy, and alcohol neuropathy as possible risk factors that contributed to paraplegia in their

patients.

CONCLUSION

Neuraxial anesthesia/analgesia is safe, but patients with diabetic myelopathy/radiculopathy associated with other risk factors such as perioperative position, perioperative hypotension, alcohol intake and heavy smoking may present a higher risk to neuraxial local anesthetics toxicity. This may lead to post –operative transient or permanent nerve injury and neuropathic pain. In addition, literature is poor and studies are needed to determine whether neuraxial anesthesia/analgesia in diabetic patients with spinal cord and DRG involvement needs reduced local anesthetic volume and concentration.

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Peripher Nerv Syst. 1996;1:119-30.

References

1. Keyuri U. Popat, Thuy Ngyugen, Alicia M. Kowalski, Mary D. Daley, James F. Arens, Dilip Thakar: Postoperative Paraplegia after Nonvascular Thoracic Surgery. The Internet Journal of Anesthesiology. 2004. Volume 8 Number 1. 2. Giladi N, Turezkite T, Harel D. Myelopathy as a complication of diabetes mellitus. Isr J Med Sci. 1991 27: 316-9. 3. Al-Nasser B Toxic effects of epidural analgesia with 0.2% ropivacaine in a diabetic patient. J Clin Anesth 2004 16 : 220-23 4. Lena P, Teboul J, Mercier B, Bonnet F. Motor deficit of the lower limbs and urinary incontinence following peridural anesthesia Ann Fr Anesth Reanim. 1998;17(9):1144-7 5. Waters JH, Watson TB, Ward MG.Conus medullaris injury following both tetracaine and lidocaine spinal anesthesia. J Clin Anesth. 1996 8:656-8. 6. Eaton SE, Harris ND, Rajbhandari SM, Greenwood P, Wilkinson ID, Ward JD, Griffiths PD, Tesfaye S. Spinalcord involvement in diabetic peripheral neuropathy. Lancet. 2001 Jul 7;358: 35-6.(letter) 7. Zochodne DW. Is early diabetic neuropathy a disorder of the dorsal root ganglion? A hypothesis and critique of some current ideas on the etiology of diabetic neuropathy.J

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