Dear Editor,

I would like to comment on the “Lipid to the rescue!” first case report published in the July-September 2013 SALG Patient Safety Update.

Apart from the presumed diagnosis of “brief episode of LA toxicity”, another differential diagnosis should be kept in mind in the event of hypotensive/bradycardic events in response to regional anaesthesia (HBE). HBE commonly associated with regional anaesthesia with epinephrine in an awake to mildly sedated patients.

Possible aetiologies of HBE: β1-agonist effects of exogenous epinephrine and activation of the Bezold-Jarisch reflex. This reflex occurs when the combination of decreased venous return (actual or relative) and increased sympathetic tone (endogenous or exogenous) leads to forceful contraction of a near-empty left ventricle, with consequent parasympathetically mediated arterial vasodilation and bradycardia.¹

Clinically, HBE is unpredictable, typically occurring 61 ± 18 mins after block placement and often heralded by light-headedness or nausea. Whether the incidence is different in patients under general anaesthesia or a combined technique is unknown.² Prophylactic Metoprolol may have a place in reducing the incidence of HBE.

Such events are described in the literature in response to central neuraxial blocks or regional anaesthesia with variable severity and outcomes.²,³

In our case, the fact that the LA was injected under US guidance, negative aspirations demonstrated, good LA spread seen, patient didn’t demonstrate LAST signs/symptoms, it was a very effective block, its resistance to treatment with glycopyrrolate and partial response to epinephrine and finally quick resolution of the event all point out that it can also be explained by the HBE.

Yours sincerely,

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References: