Accidental Intravascular Injection of Local Anesthetic?

To the Editor—I read with great interest the recent case report detailed by Loubert et al.1 I respectfully disagree with their conclusion of this being a case of local anesthetic toxicity. Presuming, based on their case description, that only 5 ml local anesthetic was injected into a blood vessel and minimal peripheral uptake occurred from the previous injections, a maximum of 75 mg lidocaine was inadvertently injected intravascularly.1 This amount of local anesthetic is unlikely to produce complications of peripheral nerve blocks. The patient, assumed from her American Society of Anesthesiologists status of I to be normotensive, has a documented blood pressure of 280/130 mmHg during the described symptoms.1 Hypertensive encephalopathy has been seen with diastolic readings of as low as 100 mmHg in patients without preexisting hypertension.4 As blood pressure exceeds the threshold of cerebral autoregulation, a hyperperfusion situation exists that may disturb the blood-brain barrier and cause cerebral edema.5 The resultant cerebral edema can lead to symptoms not dissimilar to those described by the patient in question.6 In cases of autoregulatory failure, the rate of blood pressure elevation is pivotal in the pathogenesis of both hypertensive encephalopathy and reversible posterior leukoencephalopathy syndrome.6 A rapid increase in blood pressure, from the alleged intravascular epinephrine, was no doubt present in the case report.1 Neuroimaging, although not performed in this case, may have revealed cerebral edema.7 When cerebral edema is primarily localized to the posterior cerebral hemispheres and is coupled with the clinical picture of restlessness, confusion, altered consciousness, seizures, or coma, a diagnosis of reversible posterior leukoencephalopathy syndrome should be entertained.7 With reversible posterior leukoencephalopathy syndrome, a complete recovery is typically seen after blood pressure is controlled and stabilized.5 It seems that the rapid onset and offset of symptoms in this case would likely correlate with epinephrine, not lidocaine or bupivacaine, serum levels. Patient symptomatology paralleled the elevation and subsequent normalization of the recorded blood pressures. In summary, I propose the intravascular epinephrine provided a positive stress test to the patient’s blood-brain barrier and that the concomitantly intravenously administered local anesthetic may have been an inert bystander.

References


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References


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In Reply—We thank Drs. Brull et al., Shankar, and Nelson for responding to our case report of accidental intravascular injection of local anesthetic and epinephrine during ultrasound-guided perivascular axillary block.¹

The suggestions provided by Dr. Brull’s group for improved safety during ultrasound-guided axillary block seem reasonable. The large case series of axillary blocks recently published by Dr. Brull et al. bears witness to their experience of significantly reduced (but not completely eliminated) rates of accidental intravascular injection with the adoption of ultrasound guidance compared with the blind transarterial or neurostimulator-guided techniques used and taught until recently at their institution.² Further large case series such as theirs, or the establishment of a complication registry will be needed to quantify the relative safety benefits of various preblock precautions and ultrasound-guided approaches to axillary blockade (including perivascular vs. perineural injection). However, there seems to be little doubt that future improvements in block safety lie in the optimal application of ultrasound training and imaging, and technical advances including echogenicatraumatic needles specifically designed for regional anesthesia.

To Dr. Shankar, the problems we wished to highlight in our case report include modification of anatomical relations by injection of local anesthetic leading to migration of the needle tip into a blood vessel, and the existence of small, compressible, low-flow veins that are difficult to detect with even the most sophisticated ultrasonic equipment, experienced operators, and careful scanning techniques. These problems may be mitigated by technical and educational improvements, but we wished to emphasize that continued adherence to traditional safety rules such as fractionated injection is necessary even in the ultrasound age of regional anesthesia. Blaming ultrasound guidance for the complication we present in our report would constitute in our opinion a misinterpretation of the events we related.

Dr. Nelson brings up the interesting point that 75–100 mg lidocaine would not be expected to result in the neurologic symptoms presented in our report, and proposes the alternative diagnosis of hypertensive encephalopathy or reversible posterior leukoencephalopathy secondary to the epinephrine in the block solution. Although we agree that the dose of lidocaine administered intravenously was relatively small (due to fractionated injection with ultrasonographic confirmation), we believe the time course of our patients’ symptoms (minutes, rather than days for the other evoked diagnostic possibilities) are more consistent with a high but transient peak concentration of lidocaine, possibly potentiated by the epinephrine in the solution.³

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References


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