TO THE EDITOR

In a recently published paper *"Sudden ventricular fibrillation during catheterization due to lidocaine infiltration for local anesthesia in a 53 year old woman"*, Dr. Arefi[1] reports an interesting case who developed cardiopulmonary arrest after lidocaine infiltration in the first setting, and subsequently after a lapse of more than 3 years, the same patient again developed ventricular fibrillation and cardiopulmonary arrest while the femoral artery was being cannulated. Lidocaine was incriminated to have caused ventricular fibrillation in this patient.

The author is to be commended for having successfully managed the dreaded complication on two different occasions. Ventricular fibrillation following the second attempt has attracted the author's attention as evident from the title's selection but the cardiopulmonary arrest following lidocaine infiltration during the first attempt has not been explored or elucidated, leaving the reader with the freedom to draw his own conclusions.

Without adequately addressing certain important facts (as discussed in the following paragraphs), the deduction of the final conclusion incriminating lidocaine for the entire scenario seems inadequate.

It seems that the patient had been taking beta-adrenergic blockers. Knowing very well that beta-adrenergic blockers decrease hepatic blood flow,[2] one can presume that the serum concentration of lidocaine increased beyond the threshold and caused the complication because hepatic blood flow had been hampered by beta-blockers.

The dosage of lidocaine utilized on both occasions fell safely within the recommended range and is therefore unlikely to have caused the complication. Shall we suppose that the patient had a sinus node pathology, Wolff-Parkinson-White syndrome, or impending heart failure not noticed clinically which could have precipitated ventricular fibrillation, because in these conditions the dosage of lidocaine needs to be curtailed[3] to avert toxic plasma levels or the inevitable altered pharmacokinetics of the drug.

Central nervous system (CNS) symptoms are the most common adverse side effects observed with lidocaine administration.[3,4] Toxic levels of local anesthetic probably lead initially to depression of cortical inhibitory pathways, thereby allowing unopposed activity of an excitatory nature. Cardiovascular effects appear later in the form of depression.[5] It appears that in this case, the symptoms of CNS toxicity were not seen which therefore rules out the possibility of lidocaine toxicity. Some patients however exhibit CNS toxicity with relatively low plasma concentrations. This eventuality is attributed to accumulation of the active metabolites, monoethylglycene xylidine and glycine xylidine seen especially in patients

with severe heart and renal failure.[4] This case seemingly had no such pathology.

Toxic levels of lidocaine are seldom seen after local infiltration and are usually encountered after intravenous infusions of long durations, intercostal blocks or inadvertent dural puncture while attempting an epidural block. The latter initiates a total spinal block secondary to an exceedingly large dose of the local anesthetic being injected into the subdural space. The subcutaneous route used in this particular case and the exceedingly slow and erratic absorption via this route rules out the remote possibility of toxic plasma levels especially so when the lidocaine used did not exceed the recommended dosage range permissible for such procedures. It may be added that, although toxicity undoubtedly depends upon the administered dose, nevertheless it depends more upon the serum levels of lidocaine which in turn are largely governed by the rapidity of action.

Lidocaine is very often the drug of choice for acute suppression of ventricular arrhythmias[3] and undoubtedly demonstrates efficacy against ventricular arrhythmias of diverse etiology.[2] The reported ventricular fibrillation in this case can perhaps be accounted for by a possible idiosyncratic reaction or perhaps an altered or an unexpected response of lidocaine at the receptor site. Since lidocaine depresses the S.A. node, it is highly likely that the patient had developed a transient bradycardia in the beginning which eventually terminated in ventricular fibrillation.

Pain is a common finding experienced with insertion of intravenous catheters, and either plain or alkalinized lidocaine is used by clinicians to ameliorate this pain. Alkalinized lidocaine which increases the pH of the solution, decreases H^+ ion concentration and attenuates the ensuing pain of an inserted catheter.[6,7] The reduction in pain associated with catheter insertion would be especially useful in patients prone to vasovagal reactions and in pediatric patients.[7]

Severe respiratory distress associated with hypertension, supraventricular tachycardia, and massive pulmonary edema after Nadbath and retrobulbar blocks have been reported.[8] Shall we presume that while inserting the arterial sheath, the patient developed agonizing pain that initiated a vasovagal response initially appearing in the form of transient bradycardia and finally culminating in ventricular fibrillation?[9]

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