M. M. R. Amorim  
T. Almeida  
A. Barros  
Recife, Brazil  
E-mail: orangeflavio@gmail.com


doi:10.1093/bja/aes084

Convulsions after normal dose of lidocaine: a probable drug interaction

Editor—The case reports by Satsumae and colleagues1 and Pasquier and colleagues2 have addressed the question of sensitivity to local anaesthetics (LAs) with toxicity occurring at low plasma levels. We recently encountered a similar problem in a 74-yr-old woman (ASA II, 60 kg) who presented with generalized myoclonic movement after a brachial plexus block for radius fracture. Her medical history included dyslipidaemia treated with a statin, arterial hypertension with a hypertrophic cardiomyopathy, and atrial fibrillation treated with flecainide. We performed a brachial plexus block using ultrasound for surgery on the radius. After a negative aspiration test, lidocaine 300 mg with epinephrine was injected with repeated negative aspiration and under visual control. The radial nerve was not completely blocked and a further injection was done at the wrist with lidocaine 80 mg with epinephrine again with negative aspiration and ultrasound control. Thus, a total dose of lidocaine with epinephrine 380 mg was injected. After 15 min, the patient presented with general myoclonic movement. A venous blood sample drawn during the myoclonic movements showed a non-toxic lidocaine concentration of 4.2 µg litre⁻¹. The flecainide plasma concentration at that time was 180 µg ml⁻¹. The patient had no neurological disease, and blood glucose was normal. She was treated with 200 ml of Intralipid® 20%. Surgery was possible after 30 min. CT scan and EEG recorded 2 weeks later were normal.

It is generally accepted that toxic effects from lidocaine occur in a conscious subject at a plasma concentration exceeding 5 µg ml⁻¹. For regional blocks, plasma concentrations are typically 3–5 µg ml⁻¹, with toxic levels 6–10 µg ml⁻¹.3 Our case describes seizures after a brachial plexus block with a normal dose of LA. Toxicity is still possible within clinical plasma concentration limits, without intravascular placement of the needle and injection using divided doses and frequent aspiration. A possible explanation is that the needle tip might have been placed within a small vein, so that the negative pressure aspiration resulted in apposition of the vessel wall against the needle. However, the 15 min delay between injection and the onset of symptoms is too long for direct i.v. injection. Therefore, seizures may occur after LAs absorption.1 2 The authors of one of these cases suggested that some patients may have a low tolerance to LA or that the threshold of toxicity varies with factors such as medication, hypercarbia, electrolytes abnormalities, and carnitine deficiency. In our case, the patient did not have electrolyte abnormalities or carnitine deficiency. We initially suspected low tolerance to the LA, but the patient was treated with flecainide. Flecainide is a type 1c anti-arrhythmic agent used for the treatment of supraventricular arrhythmias. It has a relatively narrow therapeutic window.5 Side-effects such as dizziness and visual disturbances, including diplopia, are not uncommon. However, severe neurological complications are rare. There are only four published case reports of serious flecainide-induced nervous toxicity, generalized seizures, and cerebello-myoclonic syndrome. It has been noted that plasma levels of above 700–1000 µg ml⁻¹ are associated with increased likelihood of adverse events.5 Lidocaine is an amide LA which is a class 1B anti-arrhythmic drug affecting nerve axon sodium channels, thus preventing depolarization.6 In our case, both flecainide and lidocaine were within their therapeutic range. An interaction with this anti-arrhythmic agent could potentially explain the LA toxicity observed and care may be needed with this combination of drugs.

Declaration of interest

None declared.

C. Landy*  
E. Schaeffer  
L. Raynaud  
J.-C. Favier  
D. Plancade  
Metz, France  
E-mail: landycb@gmail.com