Letters to Editor

This reminds us that lignocaine, which is considered as the least toxic local anaesthetic, can also induce cardiotoxicity after topical application.

We remember a 75-year-old man after coronary revascularization who was extubated early. However, on the first post-operative day, he developed shortness of breath due to left lower lobe atelectasis with haemoglobin saturation 92% on oxygen via face mask. He was indicated to bronchoscopy to remove the mucous plug. Topical anaesthesia was induced by transtracheal injection of 2 ml 4% lidocaine and by application of 10 puffs of 10% lidocaine spray. Each puff was 10 mg. During insertion of the bronchoscope, the patient coughed. The bronchoscopist therefore applied additional five puffs of lidocaine spray, i.e. the total dose was 230 mg (3 mg/kg; maximum dose according to the Summary of Product Characteristics 20 puffs) being administered during 10 min. Shortly after the additional doses, the patient lost consciousness and, on the electrocardiogram monitor, slow and wide QRS complexes were suddenly seen. The patient was immediately intubated and connected to a ventilator. Bradycardia was treated with atropine but without any obvious effect. Cardiac pacing via epicardial leads was therefore commenced, and it quickly restored normal heart rate and blood pressure. The patient was sedated with intravenous midazolam to perform the bronchoscopy. Shortly after the procedure, he regained consciousness and was extubated because he breathed sufficiently. The pacer could also be switched off. Further clinical course was uneventful and without troponin elevation.

We think the event was due to the systemic effect of lidocaine on the brain (loss of consciousness) and heart (bradycardia unresponsive to atropine with widening of QRS complexes due to sodium channel block) after rapid absorption from the site with a rich blood supply. This conclusion is confirmed by close temporal relationship between lidocaine application and development of toxicity and by rapid subsiding of the heart block. The extreme sensitivity to lidocaine could be explained by a diminished binding capacity of blood proteins (serum albumin level after extracorporeal circulation 40–50 g/L) and possibly by reduced hepatic clearance.

Lidocaine in the form of jelly, gargle, spray or packs is usually applied on mucous membranes during fibreoptic bronchoscopy or transoesophageal echocardiography. Sometimes, it is used in high doses.

Lidocaine not so innocent: Cardiotoxicity after topical anaesthesia for bronchoscopy

Sir,

We have read with great interest the letter by Nath et al. on neurotoxicity caused by absorption of lignocaine from nasal sponges in a 26-year-old male with epistaxis.[1] This reminds us that lignocaine, which is considered as the least toxic local anaesthetic, can also induce cardiotoxicity after topical application.

We remember a 75-year-old man after coronary revascularization who was extubated early. However, on the first post-operative day, he developed shortness of breath due to left lower lobe atelectasis with haemoglobin saturation 92% on oxygen via face mask. He was indicated to bronchoscopy to remove the mucous plug. Topical anaesthesia was induced by transtracheal injection of 2 ml 4% lidocaine and by application of 10 puffs of 10% lidocaine spray. Each puff was 10 mg. During insertion of the bronchoscope, the patient coughed. The bronchoscopist therefore applied additional five puffs of lidocaine spray, i.e. the total dose was 230 mg (3 mg/kg; maximum dose according to the Summary of Product Characteristics 20 puffs) being administered during 10 min. Shortly after the additional doses, the patient lost consciousness and, on the electrocardiogram monitor, slow and wide QRS complexes were suddenly seen. The patient was immediately intubated and connected to a ventilator. Bradycardia was treated with atropine but without any obvious effect. Cardiac pacing via epicardial leads was therefore commenced, and it quickly restored normal heart rate and blood pressure. The patient was sedated with intravenous midazolam to perform the bronchoscopy. Shortly after the procedure, he regained consciousness and was extubated because he breathed sufficiently. The pacer could also be switched off. Further clinical course was uneventful and without troponin elevation.

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Lidocaine in the form of jelly, gargle, spray or packs is usually applied on mucous membranes during fibreoptic bronchoscopy or transoesophageal echocardiography. Sometimes, it is used in high doses.
Ameer et al. described a 55-year-old white man who received 1131 mg of lidocaine (17.4 mg/kg) prior to diagnostic bronchoscopy. This dose produced a peak plasma concentration of 7.1 μg/ml within 0.67 h and levels above 1.5 μg/ml were sustained for 4 h after the procedure, but without any undesired effects. The absence of toxicity could be caused by alpha-1-acid glycoprotein elevation due to the patient's lung cancer. Labedzki et al. measured serum lidocaine concentrations during topical spray anaesthesia in patients undergoing diagnostic bronchoscopy. The mean total dose of lidocaine of 480–720 mg led to concentrations between 1.9 and 7.4 μg/ml. Repeated doses may therefore easily induce toxic levels. Day et al. notified of a healthy 19-year-old college student volunteer in a clinical research program undergoing a bronchoscopy who died as a result of acute lignocaine toxicity.

If we add a case report of a cardiac arrest immediately following intraurethral administration of lidocaine as topical anaesthesia for cystourethroscopy in an 87-year-old man, it is obvious that lidocaine does not differ too much from other local anaesthetics characterized by Jöhr as wonderful drugs but also dangerous toxins. Extreme care and close monitoring of patients is therefore always warranted when topical lidocaine anaesthetic is employed.

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