

Letter to the Editor

Convulsive crisis by local anesthetic during the placement of CVC: A purpose of a case[☆]

Crisis convulsiva por anestésico local durante la colocación de CVC: a propósito de un caso

Dear Editor,

Systemic toxicity due to local anaesthetics (LAs) is a rare, but dangerous, complication. Its incidence ranges from 0.2% to 0.01%.¹

In the heart, local anaesthetics increase the recovery period after repolarisation and suppresses automaticity and duration of action potentials in the His-Purkinje system.² In the central nervous system (CNS), it increases the threshold for electrical excitability.³ Classification: Group I, low potency and short duration (procaine); Group II, intermediate potency and duration (lidocaine, mepivacaine); Group III, high potency and long duration (tetracaine, bupivacaine).⁴

Toxic effects appear when the maximum safe dosage is exceeded^{5,6} or due to accidental intravascular injection. Liver failure, kidney failure and/or age factors that predispose to toxicity. Immediate toxicity (<60 s) suggests intravascular injection of the anaesthetic versus delayed toxicity (1–5 min). Prodromal symptoms (tinnitus, agitation, metallic taste, dysarthria, perioral numbness, confusion, blurred vision and dizziness) may precede seizures, CNS depression and coma, respiratory arrest, hypotension, ventricular arrhythmias and cardiac arrest. Lidocaine is metabolised in the liver to monoethylglycinexylidide and glycinexylidide. It has a peak effect at 2–5 min and a duration of action of 1–3 h. Its half-life is 7–30 min in healthy individuals and 115 min in individuals with heart failure, uraemia or cirrhosis. No dose adjustment is necessary in patients with kidney failure. However, glycinexylidide (an active metabolite) is eliminated via the kidneys, and its accumulation in severe kidney failure could cause neurotoxicity. The maximum dose of lidocaine is 300 mg, and a dose of 6.4–14.2 mg/kg is considered that may induce seizure. Patients who receive local anaesthesia to the head or neck are at higher risk of CNS toxicity.

A 42-year-old woman with no known drug allergies was a former parenteral drug addict. Personal history: hypertension (HTN); chronic kidney disease (CKD) stage 5D, secondary to chronic tubulointerstitial nephropathy; human immunodeficiency virus (HIV) infection (CD4+ 658); hepatitis C virus (HCV) infection (viral load <20 copies/mL); anxiety-depressive disorder. She had undergone surgery several times with no incidents of note. Weight: 42 kg, height: 160 cm (body mass index [BMI]: 16.4 kg/m², lean body mass index: 10.5 kg/m², body fat index: 5.9 kg/m²), aspartate aminotransferase (AST): 30 mg/dl, alanine aminotransferase (ALT): 16 mg/dl, alkaline phosphatase (AP): 97 mg/dl, gamma-glutamyl transferase (GGT): 152 mg/dl, total protein: 5.9 g/l, albumin: 2.85 g/dl and cholinesterase was normal.

The patient was scheduled for implantation of a tunneled central venous catheter (TCVC). Her complete blood count and clotting parameters were normal. The patient was placed in the supine decubitus position and peripheral vein is canulated, then her vital signs (ECG, blood pressure and pulse oximetry) were monitored. Subcutaneous lidocaine 2% (10-ml ampoule) was infused in the patient's right Sedillot's triangle in the tunnel area. As the patient experienced persistent pain in the area handled, it was decided to infuse an additional half ampoule. During the procedure, there was no resistance to infusion and aspiration performed prior to administration of local anaesthetic did not show any blood reflux at any time. During administration of the second (half) ampoule, the patient started to experience a sensation of dry mouth and paraesthesia in the right corner of the mouth, followed by forced extension and rigidity of the limbs with internal rotation and sudden loss of consciousness. Examination revealed anisocoria, blood pressure 200/90 mmHg, heart rate 135 bpm and no evidence of desaturation. Subsequently, the patient started to have generalised seizures which stopped following

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administration of diazepam 5 mg/IV. TCVC implantation was suspended, and the patient was transferred to the ICU for cardiac and neurological monitoring. During her stay, her ECG tracing showed no abnormalities, and a CT scan was performed with no pathological findings. After 24 h observation, the patient remained haemodynamically stable with no need for ventilation or vasoactive drugs, her anisocoria disappeared and her HR and BP normalised.

Our patient presented a delayed toxic response to lidocaine with CNS involvement (paraesthesia, seizures and anisocoria) probably due to several causes: (1) the patient's low weight and high dose of lidocaine (total dose administered: 300 mg; a seizure-inducing dose for this patient: 268–596 mg), and (2) low body fat with a BMI of 16.4 kg/m² (fat tissue index 5.9 kg/m²).

According to various publications, lipid solutions could be used as an effective antidote in LA toxicity.⁷ Lidocaine is a fat-soluble anaesthetic and the patient had low body fat; this could be why lower doses have a higher likelihood of toxicity. The only unexplained matter was the patient's anisocoria which resolved spontaneously. Regarding cardiotoxicity, the patient had sinus tachycardia with no increase in BP. She later underwent TCVC implantation using mepivacaine as an LA without incident. Mepivacaine is metabolised at a rate of 99% in the liver in less toxic products with a milder vasodilating effect than lidocaine, giving it a better safety profile.

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Letter to the Editor

Tuberculous interstitial nephritis: A difficult diagnosis that requires a high clinical suspicion[☆]

Nefritis intersticial tuberculosa, un diagnóstico difícil que precisa de una alta sospecha

Dear Editor,

Tuberculosis remains a global public health problem, especially in developing countries. Somewhat more than half of cases (55%) occur in Asia, followed by Africa (31%), with a

lower prevalence in the Mediterranean area (6%), Europe (5%) and Latin America (3%).¹ Regarding forms of presentation, pulmonary tuberculosis remains the most common. Among

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