Turk J Anaesth Reanim 2015; 43: 199-201 DOI: 10.5152/TJAR.2015.50023

Case Report

Late Local Anaesthetic Toxicity After Infraclavicular Block Procedure

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Abstract

Local anaesthetic (LA) toxicity is the most fatal complication of peripheral nerve block techniques. Accidental intravascular application or use of doses above the safety range are the most common cause of toxicity. Bupivacaine is a long-acting LA frequently used for long procedures or those associated with significant post-procedural pain. Fatal central nervous system and cardiovascular system toxicity are described. In this paper, we reported a young patient who showed LA toxicity symptoms 7 h after an infraclavicular peripheral block.

Keywords: Infraclavicular block, local anaesthesia, drug toxicity

Introduction

Local anaesthetic (LA) toxicity, a major complication that causes mortality in peripheral nerve block application, is frequently used in outpatient surgeries (1). Toxicity is often caused by accidentally administering LA to the systemic circulation or the application of LA over the safety limit levels. It has been reported that toxicities resulting from LAs with high cardiotoxicity, such as bupivacaine and etidocaine, lead to treatment-resistant malign arrhythmias, asystole and mortality (1). Central nervous system and cardiovascular system toxicities of bupivacaine have been known for a long time (2, 3). Toxicity symptoms usually emerge because of the inadvertent intravascular injections, rapid systemic absorption, or overdose medication (4). In this case presentation, we present a patient who developed LA toxicity with nonspecific symptoms 7 h after infraclavicular block administration for palmar and dorsal flap revision on the right hand.

Case Presentation

A 23-year-old patient weighing 86 kg and identified with ASA I risk was admitted to the emergency room because of an occupational accident and urgently operated under general anaesthesia for nerve and tendon repair of the right hand. After 3 weeks, elective palmar and dorsal flap revision was planned. Accompanied with ultrasonography (USG), infraclavicular block administration was planned for the patient. After the patient was provided with the detailed information regarding surgical and anaesthetic procedures, the patient's written consent was obtained. As pre-medication, 2 mg midazolam was intravenously administered before the block application. The patient was placed in the supine position and following electrocardiography, peripheral O_2 saturation (SpO_2) and non-invasive blood pressure monitorization, the patient's head was turned to the opposite side of the region on which the block was performed. After disinfection with povidone iodine, the injection point was determined as the intersection of the clavicle and coracoid process as suggested by Klaastad et al. (5). During the block, same doses of 7.5 mL of 0.5% bupivacaine (Bustesin® 0.5%), 7.5 mL 2% prilocaine (Priloc® 2%) prepared in two different 20 mL syringes and 30 mL of LA mixture containing 5 mL of physiological saline solution were applied. We used 100-mm long 20 gauge (G) (Ultraplex, Braun®, Germany) needles compatible with USG. Ultrasound probe was placed right next to the injection site and 1 cm below the clavicle. During the block, Siemens® Sonoline G20 USG (Germany) and 10–18 MHz linear probes were used. Immediately after the imaging of the axillary artery and cords, the stimulation needle with the probe (via in plane technique) was uniplanarly directed to the back of the axillary artery. By means of a neurostimulator, the site of the needle was confirmed by observing the rhythmic contraction movements of the hand and wrist. After aspirating to avoid intravascular injection and ensuring that there was no bleeding, through intermittent aspiration, a drug combination was injected to a total of 30 mL. LA distribution around the cord and axillary artery was observed with

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Received : 12.06.2014
Accepted : 16.09.2014
Available Online Date : 16.02.2015

USG during LA administration. Adequate anaesthesia for surgery occurred at the 20th minute. During the 95-min long operation, vital signs remained stable, respiratory distress was not observed, and additional analgesia was not required. At 3 h after surgery, the patient was evaluated in his bed in the PRC unit. Without any complications and complaints up to this stage, i.e., at 5 h after surgery (7 h after the block), the patient developed dizziness and fatigue followed by slurred speech, nystagmus and tremor. The patient was conscious. Upon developing bradycardia (heart rate: 30–35/min), hypotension (75/48 mmHg), respiratory distress and involuntary arm movements, the patient was admitted to the intensive care unit. The patient had SpO_2: 93%, respiratory rate: 36/min, blood pH: 7.35, pO_2: 124 mmHg, pCO_2: 29 mmHg and methemoglobin: 0.6%. To cease tremors and involuntary movements, 4 mg midazolam was intravenously administered. Considering LA toxicity, the patient was administered a total of 600 cc of 20% lipid solution. Methemoglobinemia was excluded because the methemoglobin value was within normal limits in blood gas analysis. The patient was intravenously administered a total of 1.5 mg atropine and 0.3 mg adrenaline after re-developing bradycardia at 12 h after ICU admission. The patient was then discharged to the PRC unit because the patient had been haemodynamically stable and conscious during his 24-h follow-up.

Discussion

Of the systemic reactions developed against LA agents, 99% of the cases result from the high blood levels of the drug. This occurs in applications, such as epidural block and peripheral nerve block, in which LA is required in high volume and concentration (6). Because of high blood concentrations, which result from inadvertent intravascular injections, rapid systemic absorption, or overdose medication, central nervous system and cardiovascular toxicity symptoms generally emerge within 0–5 min (4). In the systemic toxicity of LA, drug delivery rate and dose, patient's acid–base balance, comorbidities, age and various factors, such as pregnancy, also play a role (6). In this case, clinical symptoms emerged after 7 h, and symptoms suggesting toxicity were mild. We have not found a case with LA toxicity that demonstrated symptoms in such a late period in the literature review that we conducted. The patient did not present any systemic disease that could cause these clinical symptoms.

The frequency of convulsions observed in the peripheral nerve block-induced CNS toxicity is reported as two for each 1000 applications (7, 8). In the early period of LA-induced CNS toxicity, numbness around the mouth, metallic feeling on the tongue, tinnitus and dizziness can be observed, and in the late period, blurred vision, loss of consciousness and muscle contractions can be observed. In contrast, in the later period, convulsions and respiratory arrest may occur. Hypoxia, hypercapnia and acidosis facilitate the formation of convulsions. Hypercapnia causes a greater amount of LAs being transferred to the brain because of the increase in cerebral blood flow. In cardiovascular system toxicity, in early period tachycardia and hypertension; in late period bradycardia, hypotension and myocardial depression; and in the advanced period cardiac arrest may occur. Because of the direct effect of LA on the vascular smooth muscle and myocardium, bradycardia and vasodilatation emerges, and this situation may result in cardiovascular collapse.

Since the first use of USG on peripheral nerve block in 1978, advances in technology and the development of portable USGs have resulted in the increasing number of studies conducted on this subject. The advantages of the use of US in regional anaesthesia are direct imaging of nerves, monitoring anatomical structures, being able to monitor the needle, dose reduction thanks to monitoring LA's distribution and increasing the patient's comfort by decreasing the number of needle guidance (9, 10). In addition to these general advantages, Gurkan et al. (11) stated that peripheral nerve block applications are possible in cases where either neurostimulation or the paresthesia technique are difficult or impossible to administer when anatomical cue points cannot be determined because of a surgery, trauma, obesity, etc. Once the position of the needle in USG in our case was confirmed, drug distribution was appropriately observed. Although LA agent (a total of 56.25 mg bupivacaine, 225 mg prilocaine) was used in low doses and during the 7-h period after the block application, the patient did not have any problems, in late period inexplicit toxicities were observed. Involuntary movements that may have been convulsive were prevented after midazolam. Lipid infusion was administered for LA toxicity, and bradycardia and hypotension were treated. We thought that these late symptoms resulted from LA's absorption by the small vessels around the brachial plexus.

Conclusion

It should be noted that after a peripheral block, possible LA toxicity may be observed in the late period, clinical symptoms may be obscure, and the patients should be carefully watched in the postoperative period.

Informed Consent: Written informed consent was obtained from patient who participated in this case.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - H.İ.; Design - H.İ., Z.B.T.; Supervision - Z.B.T., H.İ.; Funding - A.K., M.B.; Materials - H.İ.; Data Collection and/or Processing - H.İ.; Analysis and/or Interpretation - Z.B.T., M.B.; Literature Review - H.İ., Z.B.T.; Writer - E.H., A.K.; Critical Review - E.H., A.K.; Other - H.İ., Z.B.T.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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