

CASE REPORT

Local Anaesthesia Systemic Toxicity (LAST) – A Stroke Mimicker

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ABSTRAK

Ketoksikan sistematik bius lokal (KSBL) adalah satu kejadian yang jarang berlaku tetapi boleh mengancam nyawa dan terjadi selepas suntikan blok pada saraf perifer. Kejadian ini mungkin berpunca dari pelbagai faktor termasuk pilihan ubat, teknik pemberian ubat atau faktor risiko pesakit sendiri. Kami melaporkan satu kes seorang wanita berusia 55 tahun yang menghidap pening kepala, percakapan telor dan lumpuh kesemua tangan dan kaki selepas beliau menerima suntikan campuran ubat lignocaine dan hidrokortison pada sendi di bahu kanan. Pemeriksaan neurologi menunjukkan ton otot lemah dan skala kekuatan otot kaki dan tangan 0/5. Kejadian-kejadian toksik ini mungkin disebabkan oleh suntikan yang tidak disengajakan ke dalam salur darah arteri atau lapisan dura pada saraf atau penyerapan ubat dari tisu berdekatan. Laporan kes ini menceritakan tentang suntikan ke dalam sendi bahu yang boleh menyebabkan KSBL yang mungkin tidak dikenalpasti kerana ia boleh menyerupai strok.

Kata kunci: bius lokal, dalam sendi, lidocaine, sendi bahu, strok, suntikan

ABSTRACT

Local anaesthesia systemic toxicity (LAST) is an uncommon and a potentially life-threatening event that develops after peripheral nerve block. The cause may be multifactorial and may include the choice of drug, technique of block and individual patient risk factors. We report a case of a 55-year-old female who developed slurring of speech and quadriplegia after receiving a mixture of lignocaine and hydrocortisone through an intra-articular injection to the right shoulder. Neurological examination revealed hypotonia and absence of power (0/5) in all limbs. These toxic events may have resulted from an accidental intra-arterial

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or dural cuff injection of local anaesthesia or absorption from surrounding tissues. This case report demonstrated that an intra-articular injection in the shoulder may cause LAST and may be under-recognized as it can mimic stroke.

Keywords: intra-articular, injection, lidocaine, local anaesthesia, shoulder joint, stroke

INTRODUCTION

Lignocaine is the most frequently used drug in almost all medical specialties especially in the Emergency Department (ED) (Torp & Simon 2018). Local anaesthesia systemic toxicity (LAST) is a potentially life-threatening event that may develop following peripheral nerve block using lignocaine (Barrington & Kluger 2013). The cause may be multifactorial and may include the choice of drug, technique of block and individual patient risk factors. The existing guideline on LAST was written based on animal and human studies and case reports (Neal et al. 2010). The present case report highlights an interesting case of LAST mimicking an acute stroke after an intra-articular lignocaine injection which has not been reported, to date.

CASE REPORT

A 55-year-old lady presented to the ED with dizziness, numbness of tongue and mouth, progressively developing slurring of speech and quadriplegia. The symptoms began to develop ten minutes after she had an intra-articular injection to the right shoulder containing a mixture of 200mg 2% lignocaine and 1ml hydrocortisone for

her supraspinatus tendinopathy at the Orthopaedic Clinic. Her body weight was 60kg. The physician confirmed that no blood was aspirated prior to anaesthetic injection. On further history, she had no previous history of allergy to drugs and food. In ED, her blood pressure was 144/80 mmHg, heart rate was 69 beats/minute and respiratory rate was 20 breaths/minute, saturation in room air was 99% and temperature was 37°C.

On clinical examination, she was alert. Her speech was significantly slurred and she was unable to open both eyes and mouth. Both pupils were normal and reactive to light. Neurological examination revealed hypotonia, power of 0/5 and normal reflexes for all four limbs. The extensor plantar responses were normal and no clonus noted. The Hoover's test was not performed. Cranial nerve examination and other components of neurological assessment were unremarkable. Cardiorespiratory function was intact. There was no documented hypoxia. An urgent head computed tomography scan & spine magnetic resonance imaging showed no abnormality. The nerve conduction study was also insignificant. She was admitted for supportive care which included rehabilitation and

neurological monitoring. Serum level of lignocaine test was not done. Her slurred speech was resolved after six hours and the neurological function regained full recovery of over a period of nine days.

DISCUSSION

LAST is suspected when a patient develops abnormal neurological and cardiovascular symptoms or when cardiac arrest ensues following a local anaesthetic (LA) agent administration. The incidence of LAST for peripheral nerve block is rare and reported in only nearly 10/10,000 patients in 2009 (Christie et al. 2014). Lignocaine toxicity can occur up to 1 in 2000 for peripheral nerve block, depending on the type of block (Torp & Simon 2018). To date, intra-articular injection was not reported to cause LAST.

Besides classic neurological complication of LAST such as circumoral numbness and seizure, unusual CNS presentations were discussed. Despite careful drug dosage, isolated unresponsiveness without respiratory and hemodynamic depression has been reported after dental surgery using lignocaine (Hayaran et al. 2017). In this case, a mixture of 2% lignocaine and adrenaline was infiltrated into the inner surface of the lower lip and vestibule. Several cases of transient locked-in syndrome were reported following stellate ganglion block where the patients remained conscious and able to blink their eyes despite being quadriplegic and having respiratory depression (Chaturvedi & Dash 2010). Accidental intra-arterial

injection and absorption from the surrounding tissues were thought to cause CNS toxicity. In the present case, the patient demonstrated transient locked-in syndrome with stable cardiorespiratory function after administration of lignocaine into the right shoulder. A rapid central neural blockade may happen during the intra-articular shoulder injection. It may be due to an inadvertent injection of local anaesthetic in the dural cuff which travels retrogradely into the subepineural space and subsequently into C4 to T8 spinal cord levels. Prolonged block in this patient was likely due to slow spread of drug through epidural fat into the intradural space (Arcas-Bellas et al. 2009; Finucane & Tsui 2007). Given the absence of any objective findings to explain the patient's neurological deficit, one should consider functional disorder such as conversion disorder. Functional magnetic resonance imaging identifies abnormal brain activity during patient's examination (Smith et al. 2007). Other differentials were high cervical cord injury and pontine stroke.

The cornerstone of the treatment for LAST is to prompt an effective airway management (Christie et al. 2014; Neal et al. 2010). In the present case, the patient was stable throughout admission without ventilation support. Lipid emulsion may be considered in the event of arrhythmia, prolonged seizure or progressive toxicity. It restores cardiac contractility by withdrawing the lipid content in lignocaine. Nonetheless, Hayaran et al. (2017) administered lipid emulsion

in an unconscious patient without cardiac compromise. This differs from Chaturvedi and Dash (2010) when they resuscitated a hemodynamically unstable patient according to ACLS protocol without lipid emulsion institution. This did not happen in our case as lipid emulsion was not favoured in view of a stable cardiac status. We believed that the patient suffered from acute central neural blockade following LA injection into the shoulder joint. Lack of experience and no diagnostic test to confirm the diagnosis left us perplexed. There was not enough evidence to determine if early lipid emulsion administration can halt the progress of the disease or shorten duration of symptoms.

CONCLUSION

The present case illustrated a rare manifestation of LAST after an intra-articular injection in the shoulder. The diagnosis is challenging. Thus, early recognition of clinical symptoms as well as prompt treatment, is essential to prevent the progression of the disease.

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