Transient ipsilateral lower limb paresis after axillary brachial plexus block- a diagnostic dilemma

Abstract

We describe the occurrence of transient ipsilateral lower limb paresis following uncomplicated ultrasound-guided axillary brachial plexus block. We were unable to establish and explain this incidental occurrence despite systematic elimination of major causes how an uncomplicated axillary brachial plexus block can cause ipsilateral lower limb paresis.

Keywords: axillary brachial plexus block complication, oxymetazoline, acute reversible cerebral vasoconstriction syndrome, TIA

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Introduction

The axillary brachial plexus block is one of the most commonly used regional anaesthesia technique for anaesthesia and analgesia of forearm, wrist and hand surgery. The proximity of the terminal nerves of the brachial plexus to the axillary artery makes identification of the landmarks consistent (axillary artery) equally for both the nerve stimulator and the surface based ultrasound-guided techniques. The success rate of block is known to be the highest when all four nerves (median, ulnar, radial and musculocutaneous) are blocked especially ultrasound guided where lower volume of local anesthetic agent is used. The block has the advantage of being performed away from the pleura and central neuraxial structures but block failure, bleeding and hematoma, infection, nerve damage and a rare inadvertent vascular injection leading to local anesthetic toxicity are known to occur.1

In this case report, we describe transient ipsilateral lower limb paresis which followed an uncomplicated ultrasound guided axillary brachial plexus block and discuss possible reasons for this unusual manifestation.

Case report

44 year ASA II man weighing 75kg and height 170cm was scheduled for excision of a symptomatic ganglion on the dorsum of left wrist under regional anaesthesia. He was seen in the anaesthesia clinic two weeks prior to surgery and was noted to have hypertension on repeated measurement and he was treated with amloidipine 5 mg. He also suffered from recurrent headache for 2 weeks which was frontal, bilateral, throbbing and severe in nature that waxed and waned. Routine blood tests, electrocardiogram and chest radiograph were otherwise normal. His current medications included oxymetazoline nasal spray 2 puffs daily for allergic rhinitis. The patient underwent routine left axillary brachial plexus block and standard monitoring was applied. The baseline vital parameters included pulse rate 65/min, blood pressure 125/85mm Hg and Spo2 100%. Intravenous access was secured with 20G canula. Patient was prepared for an axillary brachial plexus block aseptically and placed supine with abduction of the arm at 90° in relation to the shoulder.

All 4 nerves (median, ulnar, radial and musculocutaneous) were blocked under real time ultrasound guidance. A total of 20ml of 1.5% lignocaine was injected using 5ml aliquots for each nerve after negative aspiration for blood. Fifteen minutes after the block, patient experienced gradual loss of temperature, sensory and motor sensation and inability to abduct the arm. Few minutes later, the patient complained of throbbing headache and inability to move the left lower limb. Left leg examination revealed complete loss of motor power (0/5) and sensory loss. Contralateral upper and lower limb motor power and sensations were intact. Hemodynamic parameters were stable (pulse rate 80/min, respiratory rate 16/min, blood pressure 160/110mmHg and Spo2 100%). He also exhibited dysarthria. Patient denied any symptoms of circumoral numbness, tingling or tinnitus. The surgery was postponed.

Patient was transferred and monitored in the post anaesthesia care unit. The patient was examined by a neurologist and an urgent MRI of brain was arranged which revealed no obvious acute pathology. His hemodynamic parameters remained stable. Two hours later, a repeat neurological examination by the same neurologist revealed continuing partial weakness of left lower limb (4/5) but sensations returned and reflexes were normal. Headache resolved over a period of two hours. The patient was admitted to a ward. 24 hours later, ultrasound and echocardiogram of carotid artery and repeat MRI brain were performed and reported as normal. Partial motor weakness of the left lower limb completely resolved over the next 24 hours and patient was discharged home. Patient has formally agreed for us to share the facts for teaching and research purposes.

Discussion

This case report describes transient ipsilateral lower limb paresis which occurred after an uncomplicated ultrasound guided axillary brachial plexus. We were very surprised to witness this event considering no anatomical connection between axillary brachial
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Normal or near-normal cerebrospinal fluid analysis (protein level

Angiography (DSA, CTA, or MRA) documenting multifocal segmental cerebral artery vasoconstriction

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Diagnostic criteria for RCVS

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38

32

Table 1

Diagnostic criteria for RCVS.12

Table 1

| S. No | Diagnostic criteria for RCVS |
|-------|-----------------------------|
| 1     | Angiography (DSA, CTA, or MRA) documenting multifocal segmental cerebral artery vasconstriction |
| 2     | No evidence of aneurysmal subarachnoid hemorrhage |
| 3     | Normal or near-normal cerebrospinal fluid analysis (protein level <80mg%, leukocytes <10mm<sup>3</sup>, normal glucose level) |
| 4     | Severe, acute headaches, with or without additional neurologic signs or symptoms, Reversibility of angiographic abnormalities within 12weeks of symptom onset. If death occurs before the follow-up studies |
| 5     | are completed, autopsy rules out such conditions as vasculitis, intracranial atherosclerosis, and aneurysmal subarachnoid hemorrhage, which can also manifest with headache and stroke |

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Conclusion
This case posed a diagnostic dilemma in managing a patient who developed unrelated anatomical transient neurological deficit after axillary brachial plexus block. We describe the processes involved in excluding the major causes of transient ipsilateral paralysis of the lower limb following axillary plexus but the true aetiology remains a mystery.

Acknowledgements
None.

Conflict of interest
The author declares no conflict of interest.

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