

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

Volume 2 Issue 2 - 2015

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Received: March 11, 2015 | **Published:** April 21, 2015

Abbreviations: TIA, transient ischemic attack; SAH, subarachnoid hemorrhage; PACNS, primary angiitis of central nervous system; RCVS, acute reversible cerebral vasoconstriction syndrome; CNS, central nervous system; MRI, magnetic resonance imaging

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 anaesthetic 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 anaesthetic 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 amlodipine 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 cannula. 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

plexus and central nervous system. Onset of neurological deficit anatomically unrelated and distant from the injection site with relatively low volume of 1.5% lidocaine posed challenging diagnostic dilemma. The attending neurologist suggested it as a functional disorder of central nervous system related to anxiety in the absence of any focal signs. Anxiety induced functional central nervous system disorder are more common in younger females without conventional vascular risk factors and may affect the non-dominant side more often than the dominant side.

Isolated sensory symptoms are more common presentation and tend to be stereotyped and recurrent. Generalized shaking of the body, tachycardia and hyperventilation may be observed. Anxiety can cause it harder to move certain muscles but rarely causes paralysis.² They are precipitated by emotional or psychosocial stressors. We considered seeking psychiatry opinion but clinical evidence was not sufficient to warrant such advice. We considered further to exclude other reasons such as inadvertent local anaesthetic intravascular injection, adverse events related to the local anaesthetic agent, transient ischemic attack (TIA), subarachnoid hemorrhage (SAH), primary angiitis of central nervous system (PACNS) and acute reversible cerebral vasoconstriction syndrome (RCVS). An accidental intravenous injection of local anaesthetic agent is unlikely because the block was done under real time ultrasound guidance with no evidence of aspiration of blood prior to injections. There was absence of light-headedness, tinnitus, confusion or circumoral numbness, no signs of CNS excitatory phenomenon (shivering, myoclonus, tremors, and sudden muscular contractions) or signs of CNS depression (respiratory depression and arrest).³

This patient demonstrated no signs or symptoms of impending systemic toxicity of local anaesthetic agent. Lidocaine administration is associated with several adverse events in up to 6.3% in patients.⁴⁻²⁶ These adverse events are said to be more common in patients with acute myocardial infarction, congestive heart failure, and liver disease and are recorded within the first hour of therapy. The adverse events range from nervous system side effects such as yawning, restlessness, excitement, agitation, dizziness, blurred vision or double vision, muscle twitching, confusion, tinnitus, drowsiness, vertigo, paresthesia, seizures and respiratory depression and arrest, lightheadedness, apprehension, euphoria, sensation of heat, cold or numbness, twitching, tremors, convulsions, and unconsciousness. Rare cases of seizures and transient neurologic deficit related to lidocaine have been reported. We cannot completely rule out an adverse event related to lidocaine but sequence of events in our do not fit into these events.

TIA usually manifests with sudden onset of symptoms and includes unilateral paresis, speech disturbances and monocular blindness which are maximal initially and then gradually recovers within minutes.^{27,28} Headache is not a primary complaint. TIA

may sometimes begin without loss or reduction of central nervous system neuron functions such as loss of vision, hearing or limb power. This patient suffered initial headache followed by confusion but ipsilateral limb paresis lasted for more than 24hours. Although ultrasound of carotid arteries performed 24hours later was reported as normal and did not reveal any plaque, TIA could not be completely excluded. Subarachnoid hemorrhage may present with a sudden onset severe headache. It is usually associated with neck stiffness, focal neurological deficit and depressed level of consciousness. Brain scan will be associated with features of intracranial hemorrhage, cerebral oedema and hydrocephalus.²⁹ SAH was ruled out in this case because the headache was recurrent, there was no neck stiffness and the brain scan was normal.

Primary angiitis of central nervous system (PACNS) is a rare form of vasculitis of unknown cause. Constant, progressive and dull headache is most frequent initial symptoms and focal neurological symptoms are rare at onset of symptoms of headache.³⁰ It is visualized as diffuse multiple small infarcts on MRI scan in 90-100% of patients.³¹ A sudden onset headache with focal neurological deficit and normal MRI rules out the diagnosis of PACNS. We considered acute reversible cerebral vasoconstriction syndrome (RCVS). RCVS is characterized by severe headache with or without other acute neurological symptoms. It may be caused by diffuse segmental constriction of cerebral arteries that resolves spontaneously within 3months.³² Manifestations are also attributed to a transient disturbance of the regulation of cerebral arterial tone. RCVS peak occurrence is at around 40years of age with a female preponderance.³³ A history of migraine³⁴ and recent exposure to vasoactive agents has been frequently reported.³⁵ The onset is sudden headache which may be associated with neurological deficit. A diagnostic criterion for RCVS³⁶ is included in Table 1.

Brain scans of patients with RCVS may appear healthy,^{32,36} despite the presence of diffuse vasoconstriction on concomitant cerebral angiograms. Although the value of angiography of intracranial vessels is debatable but in retrospect would have been a useful investigation in helping the diagnosis but unfortunately the patient refused this modality of investigation due to its invasive nature as well as cost involved. Loewen et al.,³⁷ reported a case of RCVS associated with the prolonged use of oxymetazoline nasal spray in which recovery was prolonged. Our patient indeed received oxymetazoline nasal spray for rhinitis only for 2weeks prior to the incidence and RCVS lasted for 2days. Oxymetazoline is a selective α 2A-adrenergic receptor agonist used as a topical vasoconstrictor for rhinitis.³⁸ Oxymetazoline can be associated with side effects like rebound congestion, hypertension, palpitations and headaches. We believe that oxymetazoline nasal spray might have caused acute and transient RCVS in this patient but cannot be proven. Patient was advised to discontinue oxymetazoline and there was no further episode of headache, confusion or neurological deficit during 3months review period.

Table 1 Diagnostic criteria for RCVS.¹³

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

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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