

CASE REPORT

Co-Existence of Transient Neurological Symptoms Along With Post Dural Puncture Headache (PDPH): A Case Report

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SUMMARY

Regional anaesthesia is well known for complications. Major neurological sequelae after central blockade although rare but can be devastating for the patient and the anaesthetist. Coexistence of transient neurological manifestations along with post dural puncture headache has not been reported in literature. A case with the features of both is presented, although the exact cause for the coexistence could not be ascertained. Also, this patient did not respond to the usual doses of ACTH.

INTRODUCTION

Transient neurological symptoms (TNS) first described in 1993, also referred to as transient radicular neuritis (TRN) are characterized by back pain radiating through the legs with or without sensory or motor deficit, appearing after resolution of spinal block and resolving spontaneously within several days. The incidence is 11.9% with hyperbaric LidocaineTM and 1.3% with hyperbaric bupivacaine. The condition has also been reported with the use of subarachnoid tetracaine, mepivacaine and procaine. The incidence is highest in outpatients due to early ambulation especially after surgery in the lithotomy position and lowest amongst patients in positions other than lithotomy¹.

Post dural puncture headache (PDPH) is defined as severe headache appearing in first three days or even up to a week after dural puncture. Incidence varies between 1-37% in different studies. The various factors influencing the incidence are age, gender, needle size, multiple dural punctures, needle bevel direction and previous history of PDPH.² Symptomatic therapy includes hydration, non-opioid

analgesics, laxatives, intravenous or oral caffeine and other maneuvers e.g. tight abdominal binders and forced fluid intake.³ In resistant cases other useful alternatives include epidural blood patch, epidural or intravenous saline infusions, steroids and ACTH. Coexistence of PDPH with TRN has not been reported in literature. We hereby report a case in which both co-existed in a patient.

CASE REPORT

A 45 years old female, weighing 60kg, ASA Grade 1, had to undergo total abdominal hysterectomy for fibroid uterus. Preanaesthetic assessment did not reveal any other abnormality. In the operating room, monitors for ECG, SpO₂, heart rate and non-invasive blood pressure were attached and spinal anaesthesia was given by midline approach with 25G Quincke needle in L2 L3 space. There was no difficulty during needle placement or resistance at the time of drug injection and the patient did not complain of any pain or paresthesiae. The procedure was completed in a single attempt. Surgery was conducted in supine position and lasted for three hours in a smooth fashion.

Same day in the evening the patient presented with frontal and occipital headache with postural variation. She also complained of numbness around her right knee and weakness in her right leg along with pain radiating to her right leg. She felt inability to bear weight fully on that leg. There was a loss of patellar reflex (right). No deficit was noted in her left lower limb. A diagnosis of PDPH with TRN on the right side was made. Physiotherapy and vitamin B complex in the form of Inj. Neurobion forte™ was started to relieve TRN, and for PDPH she was advised an analgesic containing caffeine 3 times a day, tight abdominal binder, strict bed rest, plenty of fluids and lots of tea and coffee. Severity of headache did not decrease. She was also administered a dose of dexamethasone 8 mg IM stat repeated after 12 hours but still no relief was obtained. Eventually on the third postoperative day, it was decided to use ACTH 80u intramuscularly with which she had 40% relief only. The drug was repeated 12 hours later. Relief was still not adequate. Two equivalent doses at an interval of 12 hours on fourth day gave her 80% relief. The neurological deficit gradually improved and the patient was discharged on eighth post operative day.

DISCUSSION

Post spinal anaesthetic sequelae manifesting as peripheral nerve symptoms such as motor weakness, hypaesthesia, paresthesia and nerve root involvement resulting in peripheral neuropathy have been linked to the anaesthetic.⁴ Also, features such as numbness, tingling, heaviness or burning sensations have been noted in patients after spinal anaesthesia with any local anaesthetic.⁵ Though the information about specific factors affecting their occurrence is limited yet spinal anaesthesia, ambulatory surgery, lithotomy position and obesity all have been incriminated to predispose to features of neurological sequelae.⁶ Neurological complications directly related to spinal anaesthesia may be caused by trauma, ischemia, infection or neurotoxicity. Though controversial, but local anesthetic solutions administered in clinical concentrations do not cause nerve damage,⁷ prolonged exposure and/or high concentrations of local anaesthetic solutions at the

spinal roots have been shown to result in permanent neurological deficit.⁸ The incidence is more with lithotomy position due to reduced tissue perfusion and increased vulnerability of the nerve fibers. Lithotomy is a risk factor in patients who receive Lidocaine and not in patients who receive bupivacaine and tetracaine. The increased incidence of neurological sequelae in lithotomy position could be due to the potential stretching of cauda equina which caused nerve fibers to be exposed to a hyperbaric solution.⁹

None of the above mentioned risk factor existed in our patient. Though the incidence of TRN is less with bupivacaine but it can occur without any predisposing factor. The only explanation could have been prolonged exposure of nerve fibres to local anaesthetics. Since the introduction of spinal anaesthesia, headache has remained a well recognized complication. A single treatment with ACTH may offer an alternative therapy for PDPH.¹⁰ Gupta S. reported relief in PDPH in 83.3% cases by single intramuscular dose of 1ml (60 units/ml) while the rest of the patients in his study required a 2nd dose after 24 hours to provide complete pain relief. None of the patients in their study required any further aggressive management.¹¹ Mechanism of action of ACTH is not known. Possible mechanism of action is that it stimulates the adrenal cortex to secrete more cortisol, androgens and mineralocorticoids. These steroids due to their salt and water retention prove beneficial.¹² ACTH can be used intramuscularly as well as intravenously in a dose of 1.5u/kg diluted in 250ml normal saline. It can also be given as an infusion.¹³

Usually patients respond to a single shot and sometimes a second dose is needed for PDPH. All patients respond well and get adequate relief but our patient required four doses and still, the relief was inadequate. Its use was considered as her headache did not respond to routine therapy. Why our patient did not respond to the usual routine dosages of ACTH is not well understood. It may well be just an individual variation in response. Refractory headache to ACTH therapy may need ambiguous treatment

Diathermy Malfunction May Increase the Patient's Body Temperature without Leading to Burn

with alternative therapy with epidural blood patch which may prove beneficial in such cases.

A correlation of PDPH with transient radicular neuritis has not been reported. The common factor in the occurrence of both could be needle size and needle bevel direction. In our case, surgery had been performed in supine position and bupivacaine had been used so the exact predisposing factor could not be ascertained. Also, symptoms of PDPH coexisted. She got fully relieved for symptoms of TRN but not for PDPH. The pathogenesis in our case could be attributed to concentration dependent neurotoxicity of local anaesthetics. This patient developed the features of both simultaneously. Different studies and case reports ascribe transient neurological manifestations (TNM) after neuraxial anaesthesia, at least in part to Lidocaine, bupivacaine.¹⁴ In fact, nerve conduction block could be expression of a reversible toxic effect and TNM may be a moderate expression of this toxicity.¹⁵

We suggest that during administration of spinal anaesthesia meticulous attention should be given to avoid direct needle trauma and intra neural injection of local anaesthetics which is the most common cause of neurological sequelae. Also adequate care should be taken for proper positioning. A set protocol for aggressive management of PDPH and TRN may be followed if these sequelae do happen.

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