

Co-existence of Transient Neurological Symptoms Along with Post Dural Puncture Headache (PDPH)

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Transient neurological symptoms (TNS) first described in 1993, also referred to as transient radicular neuritis (TRN) is characterized by back pain radiating to the legs with or without sensory or motor deficits, occurring after resolution of spinal block and resolving spontaneously within several days. The incidence is 11.9% with hyperbaric Lidocaine and 1.3% with hyperbaric bupivacaine. The incidence is highest in outpatients due to early ambulation especially after surgery in the lithotomy positions.¹

Post dural puncture headache (PDPH) is defined as headache appearing in 1st 3 days or even up to a week after dural puncture. The factors influencing the incidence are age, gender, needle size, multiple dural punctures, needle bevel direction, previous history of PDPH.² Symptomatic therapy includes hydration, non-opioid analgesics, intravenous or oral caffeine.³ Other alternatives can be epidural blood patch, epidural or intravenous saline infusions, steroids and ACTH. Co-existence of PDPH with TRN has been rarely reported in literature. We here by report a case in which both co-existed.

CASE REPORT

A 45 years old female, weighing 60kg, ASA Grade 1, had undergone total abdominal hysterectomy for fibroid uterus. Preanaesthetic assessment did not reveal any other abnormality. In operation theatre, after putting monitors for ECG, SpO₂, heart rate, blood pressure, spinal anaesthesia was given by midline approach with 25G quincke needle in L₂-L₃ Space. There was no pain / paresthesia / difficulty during needle placement or at the time of drug injection. Surgery was done in supine position and lasted for three hours. There was no undue difficulty in surgical procedure. On the evening of first day, she 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 neurological pain radiating to her right leg. She also could not bear weight fully on that leg. There was loss of right patellar reflex. There was no deficit noted in the left lower limb. A diagnosis of PDPH with TRN of 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 advised a dose of dexamethasone 8 mg i/m stat and one dose later after 12 hours but still no relief was obtained. Eventually, it was decided to use ACTH 80U intramuscularly on 3rd postoperative day with which she had 40% relief only. Since relief was inadequate, so same dose was repeated 12 hours later. Relief was still not adequate. Two more same doses on 4th day at an interval of 12 hours gave her 80% relief. The neurological deficit gradually improved and patient discharged on 8th post operative day.

DISCUSSION

Post anaesthetic sequelae manifesting as peripheral nerve symptoms such as motor weakness, hyperaesthesia, paresthesia and nerve root involvement resulting in peripheral neuropathy has been linked to the anaesthetic.⁴ Also, features such as numbness, tingling, heaviness or burning sensations have been noted.⁵ Though the information concerning factors that affect their occurrence is limited but spinal anaesthesia, ambulatory surgery, lithotomy position and obesity all predispose to features of neurological sequelae.⁶ Neurological complications directly related to spinal anaesthesia may be caused by trauma, ischemia, infection and neurotoxicity. Though controversial, but local anaesthetic solutions administered in clinical concentrations do not cause nerve damage.⁷ However, prolonged exposure and/or high concentrations of local anaesthetic solutions at the spinal roots may 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 reason for increased incidence in lithotomy position could be due to the potential stretching of cauda equina which cause nerve fibers to be exposed to a hyperbaric solution.⁹

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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 one intramuscular dose of 1ml (60 units/ml) while the rest required a 2nd dose of 1ml 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 exactly not known. Possible mechanism of action is that it stimulates the adrenal cortex to secrete more cortisol, androgens, mineralocorticoids. These steroids due to their salt and water retention effect 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 dose and get adequate relief but this patient required 4 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 or else there may be individual variation in response of each patient to ACTH. Refractory headache to ACTH therapy may need ambiguous treatment 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 delineated. 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 intraneural injection of local anaesthetics, alongwith adequate care during positioning.

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