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Topical Transnasal Sphenopalatine Ganglion Block as Management for Postdural Puncture Headache. Where Does It Fit? A Case Report and Literature Review

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Abstract

Debilitating and sometimes life-threatening, Postdural Puncture Headache (PDPH) remains simple to recognize but challenging to treat and understand its physiopathology and consequences. With the aim of better understand the potential contribution of topical transnasal Sphenopalatine Ganglion Block (SPGB) in Central Venous Thrombosis (CVT) as a treatment of PDPH, we reviewed the literature and describe a case of CVT after PDPH managed with SPGB. A 68-year-old male patient, American Society of Anesthesiologists physical status 2 underwent an uneventful Transurethral Resection of the Prostate (TURP) under sedation and spinal anesthesia. Neither personal, nor familiar risk factor for thrombosis was referred. On the fourth postoperative day, the patient was treated with SPGB due to PDPH. Following six days, referring diplopia, he was diagnosed with CVT evidenced by Magnetic Resonance Angiography (MRA). After twenty-two days of anticoagulation and complete resolution of his symptoms, the patient continues been monitoring by neurology with no recurrence so far. Compared with Epidural Blood Patch (EBP) for treatment of PDPH, SPGB showed faster headache relief, lesser expensive, safer and better tolerated without associated complications besides a proposed causal relationship between SPGB and CVT. Well conducted trials should be developed in order to stablish where SPGB fits: protector or risk factor, or only the trigger to manifest clinical signs of a thrombus already inside of cerebral veins.

Keywords: Postdural puncture headache; Sphenopalatine ganglion block; Epidural blood patch; Central venous thrombosis

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Introduction

Debilitating and sometimes life-threatening, Postdural Puncture Headache (PDPH) remains simple to recognize but challenging to treat and understand its physiopathology and consequences [1,2]. With a rate of 10% to 30% [3], the orthostatic headache presenting within 5 days after spinal anesthesia guides to the diagnosis of PDPH [4], that can be associated with nausea, vomiting, vertigo, diplopia, tinnitus, hypoacusis, neck stiffness, back pain, cranial nerve palsies and subdural hematoma [1,5-7]. Due to the common benign course of its entity, only the minority of patients need treatment [3].

The Monro-Kellie-Abercrombie doctrine advocates an equilibrium among brain, blood volume and Cerebrospinal Fluid (CSF) inside of skull [3]. Hence, reduction or increase of either element leads to adjustment in the other two [3]. In the PDPH as CSF decreases, its volume and pressure also do so, driving to increase in the intracranial arterial and venous volume and descent of the brain and brainstem structures [1]. This cerebral vasodilation associate with distortion and stretching of the veins are nociceptive stimulus, which are sensed as PDPH that is aggravated in erect posture [3,8].

Since the 1960s the gold-standard treatment for PDPH is autologous Epidural Blood Patch (EBP) [9]. Despite it's been an invasive procedure that may complicate with facial nerve palsies, back pain, permanent spastic paraparesis, infection, cauda equina syndrome, meningitis [1,10] and subdural hygroma [3], it acts in all physiopathology ways of PDPH. Epidural blood patch promotes compression of the thecal sac that increases intracranial pressure [11], stops CSF leakage and by an inflammatory and reflex *via* relives cerebral vasodilation [12].

On the other hand, although first described in 1908 [13] and only applied to heal PDPH in 2001 [14], topical transnasal Sphenopalatine Ganglion Block (SPGB) is a non-invasive procedure

related to shorter headache relief compared to EBP, with transient and mild adverse effects, such as bitter taste, nostril burning and discomfort, and oropharyngeal numbness [1,15], besides the suggested possibility to favor Central Venous Thrombosis (CVT) [16]. The hypothesized mechanism of action of SPGB is to prevent these parasympathetic ganglia activity after CSF hypotension. It impairs cerebral vasodilation *via* discontinuing the release of acetylcholine, nitric oxide and vasoactive intestinal peptide into dural blood vessels. Additionally, inhibiting the headache amplification, it hinders neurogenic inflammation with decrease of plasma protein extravasation that would trigger trigeminal nociceptors [17].

With the aim of better understand the potential contribution of SPGB in CVT as a treatment of PDPH, we reviewed the literature and describe a case of CVT after PDPH managed with SPGB.

Case Presentation

A 68-year-old male patient, American Society of Anesthesiologists physical status 2 due to hypothyroidism, Benign Prostate Hyperplasia (BPH), and hyperuricemia taking levothyroxine 112 mcg, doxazosin 2 mg and alopurinol 200 mg underwent Transurethral Resection of the Prostate (TURP) under sedation and spinal anesthesia with isobaric bupivacaine 0.5% 15 mg and fentanyl 25 mcg using a 27-gauge Quincke spinal needle. His peri- and post-operative period were uneventful, and he was discharged the next day. Neither personal, nor familiar risk factor for thrombosis was referred.

The patient returned on the fourth postoperative day with complaints of frontal and occipital headache with orthostatic character associated with tinnitus, hypoacusis and neck stiffness, which had started and worsening progressively one day before. A diagnosis of PDPH was made, and a SPGB was performed with 5 ml of bupivacaine 0.25% without epinephrine *via* cotton-tip applicators placed bilateral intranasally until they reach the posterior wall of the pharynx for 5 min. Immediately after the procedure, referring significant improvement of all symptoms the patient was release from care. Every two days he was contacted by phone.

On the eleventh postoperative day, he returns to the hospital referring diplopia and a gadolinium-enhanced Magnetic Resonance Angiography (MRA) of the brain evidenced thrombus in the sagittal sinus (Figure 1). Thus, the patient was hospitalized for anticoagulation and enoxaparin 80 mg subcutaneous twice a day was initiated. Following six days, referring 90% of improvement of diplopia he was discharged with apixaban 2.5 mg daily and return in 15 days or at any time if there was an intercurrence.

After taken sixteen days of apixaban and complete resolution of diplopia and others symptoms, the patient keeps in following with the neurology team and there has been no recurrence so far.

Discussion

As first suggested by Schou and Scherb in 1986 the causal association between PDPH and CVT has long been assumed as truth [3,18]. The headache that loses its orthostatic character associated or not with seizures or focal neurological deficit should warn to the development of CVT [3]. Lumbar Puncture (LP) lower intracranial pressure, which induce slowing cerebral blood flow in the veins and dural sinus. Canhao et al. [18] showed an average of 47% reduction of mean Blood Flow Velocity (mBFV) in the Straight Sinus (SS) induced by the LP. This reduction persists in a rate of 29% after 6 h of LP [18]. Even using smaller gauge needle to perform a LP in the described patient compared with Canhao et al. [18], the dropping of mBFV might be induced, presumably with any needle diameter, noticeably at different proportion. This lower blood flow goes favor to clot formation, settled by one of the Virchow's triad components: Stasis.

In this way, hypercoagulability, another Virchow's triad pillar, still remains the major risk factor for CVT [18]. Thus, congenital and acquired prothrombotic conditions such as pregnancy [19], postpartum state [18-21], oral contraceptive use [19,22-26], thrombophilia [22,26], protein C and S deficiency [19], factor V Leiden mutation [19], malignancy [18,27,28], treatment with steroids [22-24,26,29,30] and intrathecal administration of corticosteroids [31] or cytostatics [27] must be investigate at the diagnose of this clinical entity [18]. The descried patient denies any familiar and personal history of thrombotic events and was not taken any medicine that could predispose blood coagulation.

Compared with EBP for treatment of PDPH, SPGB showed faster headache relief, lesser expensive, safer and better tolerated without associated complications [1]. Still, patients managed with EBP had backache radiating to their lower extremities (7.7%), vasovagal reaction (2.6%) and temporary hearing loss (2.6%) lasting no more than a week [1]. Contrasting, Horiguchi et al. [16] suggest worsening stretch downward of cerebral structures favoring CVT by erect posture enabled with headache relief by SPBG. Thus, they proposed causal relationship between treatment of PDPH with SPGB and CVT in two out of 52 patients managed at this way. In our case, in accordance with Horiguchi et al. [16], despite significant headache improvement, he presented with diplopia and was diagnosed with CVT following three days of SPGB. Regardless of SPGB inhibits release of dilated agents at veins of Central Nervous System (CNS),

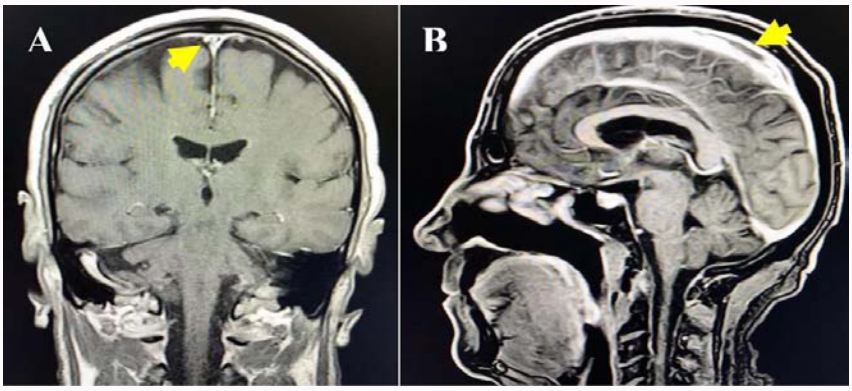

Figure 1: Gadolinium-enhanced MRA of the brain evidencing filling defect in the sagittal sinus (yellow arrow). Sagittal section (A) and coronal section (B).

the descent of brain and brainstem at upright position might promote bigger vasodilation favoring blood stasis and consequently CVT.

However, depending on physiopathology moment that SPGB was done, its intuitive to assume that it can bring distinct outcome. Due to similar presentation and overlap of clinical signs at the beginning [3,32], a misdiagnosis of PDPH may hide an incipient CVT and the SPGB might precipitate loss of orthostatic headache, seizures and/or focal neurological deficit by its cerebral vasoconstriction feature. Hence, as the SPGB reduces the diameter of cerebral veins, the presence of small thrombus inside of them would overt clinical repercussion. Moreover, promoting vasoconstriction of cerebral vessels, SPGB increases blood flow velocity and could prevent the thrombus formation outset by minimizing the blood stasis.

Conclusion

As PDPH is an expected adverse event secondary to LP, a procedure performed a myriad of times a day, its treatment must be safe. Epidural blood patch has its collateral effects well defined, nevertheless SPGB does not.

Once already settled that the blood stasis secondary to vasodilation generated by LP is a CVT risk, it is necessary to stablish where SPGB fits: Protector or risk factor, or only the trigger to manifest clinical signs of a thrombus already inside of cerebral veins. Well conducted trials should be developed to clear these doubts in order to benefit the patients with prompt and correct management.

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