Comparison between Adding Intravenous Neostigmine and Atropine versus Intravenous Hydrocortisone for Conservative Treatment of Postdural Puncture Headache after Spinal Anaesthesia for Elective Caesarean Section

A Thesis

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بسم الله الرحمن الرحيم

قالوا

سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم

صدق الله العظيم

سورة البقرة الآية: ٣٢

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INTRODUCTION

Post-dural puncture headache (PDPH) is a common adverse effect following spinal anesthesia, occurring in 2.5-40% of patients.It typically begins within 2 days but may be delayed for as long as 2 weeks and almost resolves spontaneously within a few days.

The classic symptoms of PDPH consist of photophobia, nausea and vomiting, neck stiffness, tinnitus, diplopia, and dizziness. The headache is usually severe, throbbing,frontal in origin with radiation to occipital area and exacerbated by sitting or standing. The positional nature of the headache and dramatic improvement on assuming the supine position the standard diagnostic criterion for this condition [(1)]

Variability In the incidence of PDPH is affected by many factors such as age, gender, needle type, size, operation type and number of attempts for dural puncture. In general, PDPH is more common in young women, particularly in pregnancy.

The pathophysiology of PDPH has not been fully described.It is well known that the puncture in the dura allows cerebrospinal fluid (CSF) to leak from the subarachinoid space, resulting in a decreased of CSF volume and pressure. This CSF volume loss may cause a downwards

pull on pain-sensitive structures resulting in headache. Alternatively, the loss of CSF may cause an increase in blood flow, resulting in arterial and venous vasodilatation and PDHP.

A third explanation involves the role of substance P and the regulation of neurokinin-1receptors (NK1R) [(2)]

The aim of management of PDPH is to replace the lost CSF, seal the puncture site and control the cerebral vasodilatation. Management options may be non invasive including psychological, supportive, posture, abdominal binder and therapeutic agents as desmopressin acetate, adrenocorticotrophic hormone, caffeine, sumatriptan, corticosteroids and neostigmine. The exact mechanism of action of **corticosteroids** in relieving PDPH is not clear. Anti-inflammatory action of corticosteroids at dural puncture site may be responsible for their analgesic action in PDPH. It also favors the reabsorption of CSF from the extradural space and thus leads to increased CSF volume. Both these mechanisms may be interacting in combination to produce therapeutic effects in PDPH [(1)]

Neostigmine is a parasympathomimetic agent that has been investigated for use as an adjunct analgesic agent. A number of studies have investigated the interthecal, epidural, caudal and intra-articular routes of administration of this agent. As well as the addition of neostigmine to local

anesthetics used for brachial plexus block and intravenous regional anesthesia. [5] Neostigmine causes analgesia by inhibiting the breakdown of acetylcholine in the dorsal horn and spinal meninges. Acetylcholine may cause analgesia through a direct action on spinal cholinergic muscarinic MI and M3 receptors and nicotinic receptor subtypes and indirectly through stimulation of the release of nitric oxide in the spinal cord. [5]

Several invasive approaches have also been suggested for management of PDPH such as epidural blood patches, epidural saline epidural dextran, (epidural, interthecal and parentral opioid), fibrin glue, interthecal catheter and surgical closure of the dural perforation [3].

Post-dural puncture headache

Post dural puncture headache is aknown complication of spinal anaesthesia.

In the last 50 yr, the development of fine gauge spinal needles and needle tip modification, has enabled a significant reduction in the incidence of post dural puncture headache. Though it is clear that reducing the size of the dural perforation reduces the loss of CSF, there are many areas regarding - the pathogenesis, treatment and prevention of PDPH that remain contentious. (1)

Pathophysiology of PDPH:

The pathophysiology of PDPH has not been fully described. There are two possible explanations. First, the lowering of CSF pressure through the dural perforation causes traction on the intracranial structures in the upright position. These structures are pain sensitive, leading to the characteristic headache. Secondly,the loss of CSF produces a compensatory venodilatation.(17) The Monro– Kellie doctrine hypothesis, states that the sum of volumes of the brain, CSF, and intracranial blood is constant. The consequence of a decrease in CSF volume is a compensatory increase in blood volume. The venodilatation is then responsible for the headache. (17)

The spinal dura mater is a tube extending from the foramen magnum to the second segment of the sacrum. It contains the spinal cord and nerve roots that pierce it. The dura mater is a dense, connective tissue layer made up of collagen and elastic fibres. The classical description of the spinal dura mater is of collagen fibres running in a longitudinal direction. (8)

This had been supported by histological studies of the dura mater. (9)

Clinical teaching based upon this view of the dura recommends that a cutting spinal needle be orientated parallel rather than at right angles to these longitudinal dural fibres. Orientating the needle at right angles to the parallel fibres, it was said would cut more fibres. The cut dural fibres, previously under tension, would then tend to retract and increase the longitudinal dimensions of the dural perforation, increasing the likelihood of a post spinal headache. (9)

Clinical studies had confirmed that PDPH was more likely when the cutting spinal needle was orientated perpendicular to the direction of the dural fibres. However, recent light and electron microscopic studies of human dura mater have contested this classical description of the anatomy of the dura mater. (10)

These studies describe the dura mater as consisting of collagen fibres arranged in several layers parallel to the surface. Each layer or lamellae consist of both collagen and elastic fibres that do not demonstrate specific orientation.[11] The outer or epidural surface may indeed have dural fibres arranged in a longitudinal direction, but this pattern is not repeated through successive dural layers.

Recent measurements of dural thickness have also demonstrated that the posterior dura varies in thickness, and that the thickness of the dura at a particular spinal level is not predictable within an individual or between individuals.[10] Dural perforation in a thick area of dura may be less likely to lead to a CSF leak than a perforation in a thin area, and may explain the unpredictable consequences of a dural perforation. [12]

The **CSF volume** in the adult is approximately 150 ml, of which half is within the cranial cavity. The CSF pressure in the lumbar region in the horizontal position is between 5 and 15 cm H2O. On assuming the erect posture, this increases to over 40 cm H2O.[12]

Puncture of the dura has the potential to allow the development of excessive leakage of CSF. Excess loss of CSF leads to intracranial hypotension and a demonstrable reduction - in CSF volume.[17; 18]

The rate of CSF loss through the dural perforation (0.084–4.5 ml s^{-1}) is generally greater than the rate of CSF production (0.35 ml min^{-1}), particularly with needle sizes - larger than 25G [19]

Gadolinium enhanced MRI, in the presence of a post dural puncture headache, frequently demonstrates 'sagging' of the intracranial structures. The MRI may or may not demonstrate meningeal enhancement.(20) The meningeal - enhancement is attributable to vasodilatation of thin walled vessels in response to the intracranial hypotension. Histological studies have confirmed that the vasodilation of meningeal vessels is unrelated to aninflammatory response. [20]

Although the loss of CSF and lowering of CSF pressure is not disputed:

- **Incidence:**

The incidence of PDPH was 66% in 1898. [4] This high incidence was likely attributable to the use of large gauge, medium bevel, cutting spinal needles (needles 5, 6 and 7, Fig. 1). In 1956, with the introduction of 22G and 24G needles, the incidence was estimated to be 11%.[21]

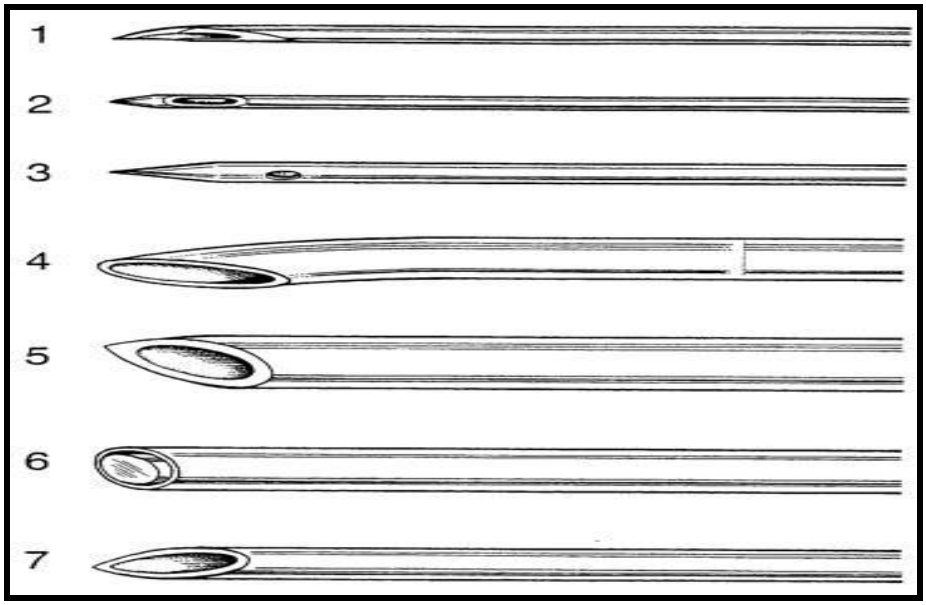

Figure (1): Graphical representations of epidural (needle 4) and spinal needle tip design, 1, 26G Atraucan® Double Bevel Design; 2, 26G Sprotte®Style Pencil Point; 3, 22G Whitacre Style Pencil Point; 4, 16G Tuohy Needle; 5, 17G Barkers Spinal Needle; 6, Large Gauge Spinal Needle; 7, 18G Quincke Needle [22].

Today the use of fine gauge pencil‐point needles, such as the Whitacre and Sprotte® ‐ has produced a greater reduction in the incidence of PDPH, which varies with the type of procedure and patients involved. It is related to the size and design of the spinal needle used (Fig.1; Table 1) the experience of the personnel performing the dural puncture, the age and sex of the patient [23]

Table (1): Relationship between needle size and incidence of post‐dural puncture headache [23]

Needle tip design	Needle gauge	Incidence of post dural Puncture headache (%)
Quincke	22	36
Quincke	25	3–25
Quincke	26	0.3–20
Quincke	27	1.5–5.6
Quincke	29	0–2
Quincke	32	0.4
Sprotte	24	0–9.6
Whitacre	20	2–5
Whitacre	22	0.63–4
Whitacre	25	0–14.5
Whitacre	27	0
Atraucan	26	2.5–4
Tuohy	16	70

Incidence in Obstetrics:

The parturient is at particular risk of dural puncture and the subsequent headache because of their sex, young age, and the widespread application of epidural anesthesia. In