بسم الله الرحمن الرحيم

MONA MAGHRABY

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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MONA MAGHRABY

Addition of Neostigmine and Atropine to Conventional Management of Postdural Puncture Headache after Cesarean Section: A Randomized Controlled Study

Thesis

Submitted for Partial Fulfillment of Master Degree in Anesthesia, Intensive Care and Pain Management

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List of Abbreviations

Abb.	Full term
ASRA	*American Society of Regional Anesthesia and Pain Medicine*
CNS	*Central nervous system*
CSF	*Cerebrospinal fluid*
CT	*Computed tomography*
DBP	*Diastolic blood pressure*
EBP	*Epidural blood patch*
HR	*Heart rate*
LAST	*Local anesthetics systemic toxicity*
PDPH	*Post dural puncture headache*
SBP	*Systolic blood pressure*
SD	*Standard deviation*
SpO2	*Oxygen saturation*
SPSS	*Statistical package for social sciences*

INTRODUCTION

POst dural puncture headache (PDPH) is a known side effect following spinal anesthesia occurring in 2.5-40% of patients, it typically starts after 2 days but may be delayed up to 2 weeks and almost resolves spontaneously within a few days.

Classic symptoms of PDPH consist of photophobia, nausea, vomiting, neck stiffness, tinnitus, diplopia and dizziness ***(Pearce, 1994)***.

The headache is usually severe throbbing frontal in origin with radiation to occipital area and increased by sitting or standing. The position nature of the headache and dramatic improvement on assuming the supine position are the standard diagnostic criteria for this position ***(Pearce, 1994)***.

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

The pathophysiology of PDPH has not been fully described, it is known that the dural puncture leads to CSF leakage from the sub arachnoid space results in decreasing in the volume and pressure of the CSF, this volume loss may cause a downwards pull on pain-sensitive structures which leads to headache.

Alternatively CSF loss may cause an increase in blood flow resulting in arterial and venous vasodilatation and so PDPH could happen ***(Arevalo-Rodriguez et al., 2016)***.

A third explanation includes the role of the substance P and the regulation of neurokinin-1receptors (NKIR) ***(Gorelick and Zych, 1987).***

The goal of management of PDPH is to replace the loss of CSF, heal the site of dural puncture and control the cerebral vasodilatation, Management options may be non-invasive including physiological, supportive, posture, abdominal binder, and also therapeutic agents such as desmopressin acetate, adrenocorticotrophic hormone, caffeine, sumatriptan, steroids, and our drug neostigmine ***(Turnbull and Shepherd, 2003)***.

Neostigmine is a parasympathomimtic agent that has been investigated for us as an adjunct analgesic agent, A number of studies have investigated the intrathecal, epidural, caudal, and intraarticular routes of administration of this agent, in addition to the involvement of neostigmine with local anesthetics for regional blocks and intravenous regional anesthesia ***(Habib and Gan, 2006)***.

Neostigmine analgesic role is due to inhibition of acetylcholine breaking down in the dorsal horn and spinal meninges, Acetylcholine may cause analgesia through a direct action on spinal cholinergic muscarinic M1 AND M3 receptors

and nicotinic reports subtypes and indirectly through stimulation of nitric oxide release in the spinal cord ***(Habib and Gan, 2006)***.

Many invasive approaches have also been used for management of PDPH like saline placebo, epidural blood patches, epidural saline, epidural dextran, fibrin glue, intrathecal catheter up to surgical closure of the dural perforation ***(Turnbull and Shepherd, 2003)***.

AIM OF THE WORK

The aim of the study is to know if the neostigmine and atropine can improve PDPH treatment when added to conventional management after cesarean sections.

Chapter 1

ANATOMY OF SPINAL ANASETHIA

Anatomy relevant to subarachnoid blockade:

The spinal cord is continous with the brain stem proximally and terminates distally in the conus medullaris as the filum terminale (fibrous extension) and the cauda equina (neural extension). This distal termination varies from L3 in infants to the lower border of L1 in adults due to the differential growth rates between the bony vertebral canal and the central nervous system (CNS) ***(Miller et al., 2014).***

The spinal canal has a protective sheath composed of three layers. From the outside to the inside they are: dura mater, arachnoid and pia mater ***(Figure 2)***. These membranes concentrically divide the vertebral canal into three distinct compartments: the epidural, subdural, and subarachnoid spaces ***(Snell, 2007).***

The epidural space contains fat, epidural veins, spinal nerve roots, and connective tissue. The subdural space is a potential space between the dura and the arachnoid and contains a serous fluid. The subarachnoid space is traversed by threads of connective tissue extending from the arachnoid mater to the pia mater. It contains the spinal cord, dorsal and ventral nerve roots, and cerebrospinal fluid (CSF). The subarachnoid space ends at the S2 vertebral level ***(Huang, 2011).***

Lumbosacral CSF has a constant pressure of approximately 15 cm H2O, but its volume varies according to the patient because of differences in body habitus and weight. It is estimated that CSF volume accounts for 80% of the variability in peak block height and regression of sensory and motor blockade ***(Miller et al., 2014).***

The nerve root is the main site of action for spinal anesthesia. In spinal anesthesia the concentration of LA in CSF is thought to have minimal effect on the spinal cord itself ***(Mulroy, 2002).***

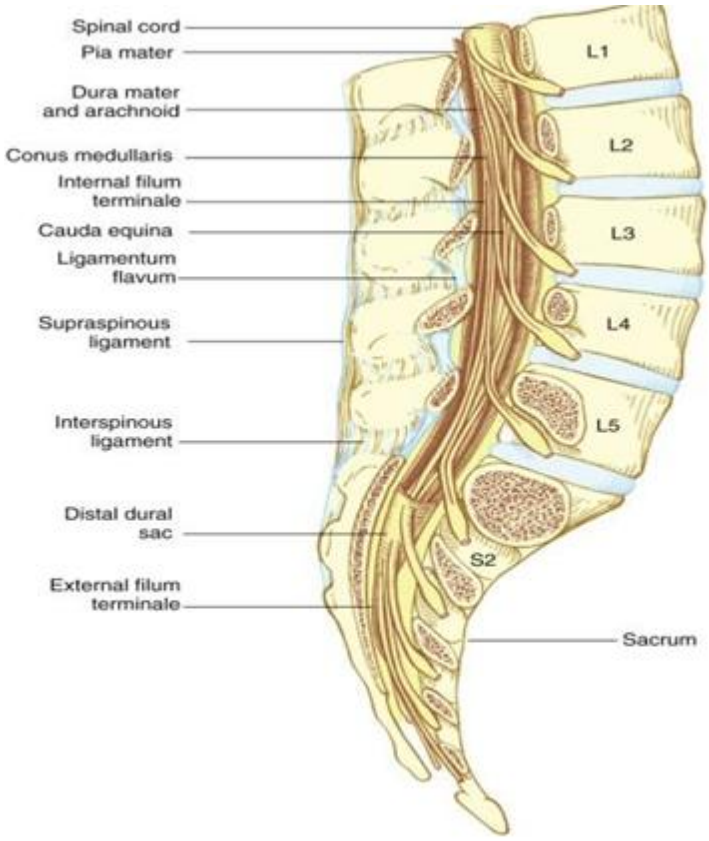

Figure 1: Longitudinal section of spinal cord ***(Reina et al., 2000).***