Risk Scoring System for Prediction of Postdural Puncture Headache Following Spinal Anesthesia

Thesis
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Introduction

Postdural puncture headache (PDPH) was first described by Dr. August Bier in 1898, after experimenting on himself. The incidence of PDPH is up to 25% after spinal anaesthesia, and the main morbidity after PDPH is restriction of activities of daily living (***Lambert et al., 1997).***

PDPH is due to loss of CSF. The subsequent low CSF pressure causes traction on nerve roots and intracranial structures when the patient stands upright (***Lambert et al., 1997).***

Pain after dural puncture is probably due to an increase in cerebral blood flow (CBF). As CSF pressure decreases, CBF increases in order to maintain a constant intracranial volume. Cranial nerve

symptoms such as diplopia and tinnitus may occur along with nausea and vomiting (***Lambert et al., 1997).***

The factors which determine the occurrence of the PDPH can be schematically divided into two categories: non modifiable constitutional risk factors related to the patient, and technical factors involved in the performance of the block ***(Pippa et al., 1995).***

As regard first category it includes age, gender and pregnancy. Among the technical factors; the needle size, the configuration of the needle point, the relationship of needle bevel to longitudinal dural fibers and the angle at which the dura is punctured and type of the analgesic used all proved to be significant ***(Pippa et al.,1995).***

Although most patients can be treated conservatively with fluids, caffeine, bed rest, analgesics, and sumatriptan, it can take anywhere from 1 to 6 weeks for symptoms to resolve spontaneously ***(Candido and Stevens, 2003).***

Aim Of The Work

The aim of this study is to develop a risk scoring system for prediction of Post Dural Puncture Headache following spinal anaesthesia.

Review Of Literature

Chapter One

Pathophysiology And Incidence Of PDPH

More than 100 years have passed since the initial description of PDPH, however, this unique clinical entity still continues to fascinate anaesthetists, and numerous studies on its pathophysiology, prevention, and treatment, have been published ***(Kuczkowski, 2002).***

In 1898 Bier suffered and was the first to report post lumbar puncture headache ***(Raskin, 1990).***

According to the Headache Classification Committee of the International Headache Society, headache after lumbar puncture is defined as “bilateral

headaches that develop within 7 days after a lumbar puncture and disappears within 14 days. The headache worsens within 15 minutes of resuming the upright position, disappears or within 30 minutes of resuming the recumbent position". This definition helps to avoid confusion with migraine or simple headache after lumbar puncture ***(Oslen, 2004).***

The onset of headache after lumbar puncture is usually within 24-48 hours after dural puncture, but contrary to the above definition, it could be delayed by up to 12 days, indicating that the time points in the definition are random ***(Ahmed, 2006).***

There seems to be a considerable variability in the incidence of PDPH, which is affected by many factors such as age, gender, pregnancy, and needle type and size ***(Vallejo, 2000).***

The incidence of PDPH can be as high as 80% if puncture occurs with a 16 gauge needle. However, most recent studies reported incidences of 1% - 3% even with these smaller gauge needles ***(Nafiu, 2007).***

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 fibers. The classical description of the spinal dura mater is of collagen fibers running in a longitudinal direction ***(Turnbull, 2003).***

Clinical teaching based upon this view of the dura recommends that a cutting spinal needle should be orientated parallel rather than at right angle to these longitudinal dural fibers. Orientating the needle at right angles to the parallel fibers, it was said to cut

more fibers. The cut dural fibers, previously under tension, would then tend to retract and increase the longitudinal dimensions of the dural perforation, increasing the likelihood of a post dural puncture headache. Clinical studies had confirmed that post dural puncture headache was more likely when the cutting spinal needle was orientated perpendicular to the direction of the dural fibers. However, recent light and electron microscopic studies of human dura mater have contested this classical description of the anatomy of the dura mater ***(Reina, 2000).***

These studies describe the dura mater as consisting of collagen fibers arranged in several layers parallel to the surface. Each layer or lamellae consists of both collagen and elastic fibers that do not demonstrate specific orientation ***(Fink, 1989).***

The outer or epidural surface may indeed have dural fibers 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 ***(Reina, 2000).***

The consequences of perforation of the spinal or cranial dura are that there will be leakage of CSF. Neurosurgical experience of dural perforation is that even minor perforations need to be closed, either directly or through the application of synthetic or biological dural graft material. Failure to close the dural perforation may lead to adhesions, continuing CSF leak, and the risk of infection ***(Turnbull, 2003).***

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 ***(Turnbull, 2003).***

In a normal human, production of CSF amounts to 450–500 ml/day (0.3 ml/min). The normal CSF volume is 150 ml and the body generates its entire CSF volume 3 times a day ***(Ronit, 2010).***

The CSF pressure in the lumbar region in the horizontal position is between 5 and 15 cm H_2O. On assuming the erect posture, this increases to over 40 cm H_2O. The pressure of the CSF in children rises with age, and may be little more than a few cm H_2O in early life ***(Turnbull, 2003).***

There is considerable evidence that the PDPH is due to a low CSF pressure consequent upon seepage of CSF through the dural puncture hole and choroid

plexus is unable to secrete sufficient fluid to maintain the CSF pressure ***(Angle, 2003).***

This intracranial hypotension causes subsequent gravity dependent downward sagging of the brain, inducing pain by traction of the anchoring sagittal sinus and its tributary veins and pressure on the tentorium and the large cerebral arteries ***(Vilming, 1997).***

When the patient stands up, or suddenly changes position, there is a sudden reduction of CSF volume below the cisterna magna and a downward movement of brain tissue with displacement and stretching of pain-sensitive structures, such as meninges and vessels, which, in turn, causes a traction headache. The intracranial pressure decreases and intracranial venous distension increases, worsening the headache. A combination of low CSF pressure and resultant

cerebral vasodilatation might have an impact on the reaction to the stretching of vessels. Venous dilatation is known to partially compensate for the loss of intracranial volume and may be responsible for the sensation of pressure that is experienced with post lumbar puncture headache ***(Ronit, 2010).***

The headache that ensues after dural puncture is theorized to be primarily due to loss of CSF from a defect made in the dura with resultant intracranial hypotension. A large defect allows for greater loss of CSF and increases the likelihood of intracranial hypotension and PDPH. When the patient then assumes an upright position there is downward traction on pain sensitive intracranial veins, meninges, and cranial nerves caused by gravity and loss of buoyancy from the reduced CSF pressure. This “sagging” of

intracranial structures has been demonstrated on magnetic resonance imaging ***(Frank, 2008).***

CSF leak after dural puncture has been well documented. Manometric studies have demonstrated that adult subarachnoid pressure is reduced from the normal 5–15 cm H_2O to less than 4 cm H_2O, and the rate of CSF leak is generally greater than CSF production. Support for this theory of PDPH also is present in the fact that intrathecal or epidural injections of saline restore the CSF volume, increase epidural and subarachnoid pressure, and alleviate the headache ***(Frank, 2008).***

Other proposed mechanisms for the pain of PDPH include a resultant dilatation of the cerebral blood vessels as a compensatory mechanism to restore intracranial volume, which may further exacerbate the symptoms ***(Frank, 2008).***

Patients with low CSF levels of the neurotransmitter substance P, which is released with dural puncture, were three times more likely to have PDPH than those with higher levels ***(Clark, 1996).***

Hypersensitivity to substance P with up regulation of receptors is thought to be a causative factor in post-dural puncture as well as other types of headache ***(Frank, 2008).***

Post dural puncture meningitis, despite its rarity, should be on the list of differential diagnosis for any patient who develops headache, neck pain, or both after dural puncture. Similar findings are more frequently due post dural puncture headache, but the seriousness of the former diagnosis requires a high index of suspicion ***(Bear, 2006).***

The presence of fever, altered sensorium, or other neurologic signs can differentiate Post dural