2

Lumbar Puncture: Techniques, Complications and CSF Analyses

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1. Introduction

Lumbar puncture (LP) is one of the well-known ancillary procedures in clinical neurology performed for a variety of functions such as spinal anesthesia, intrathecal administration of drugs, myelography, obtaining cerebrospinal fluid (CSF) samples and measuring pressure since more than 100 years ago. The question that who was really the discoverer of LP is still under debate. Most authors assume that it was Heinrich Irenaeus Quincke (1842-1922) a German internist who was introduced the procedure to medicine in 1891, however, some authors mentioned the American neurologist James Leonard Corning (1855-1923) as the first one who performed LP using birds quills in 1885 (Frederiks et al., 1997; Dakka et al., 2011).

The technique was passed on through generations of physicians to the next but the rules are the same. Quincke's aim on performing LP was to treat patients with hydrocephalus. In 1898 August Bear's assistant attempted to administer a spinal anesthetic to Dr. Bear which was never completed because the syringe did not fit the already implanted spinal needle. However, continued CSF leakage through the dural puncture site was the cause of post lumbar puncture headache (PLPH), discussed later in this monograph (Raskin, 1990).

2. Before beginning the procedure

Before performing LP it is important to think cautiously about the indications and the required diagnostic studies. Patient should be examined especially for papilledema; and cranial CT scans or MRI should be reviewed carefully. Obtaining a cranial CT scan before performing LP in all patients if possible is mandatory especially in patients with papilledema, loss of consciousness, focal neurologic deficit, new seizure onset, and inability to visualize optic fundi (Kastenbauer et al., 2002; Roos, 2003).

In adults with suspected meningitis, clinical features can be used to differentiate between patients with abnormal findings in their CT scans of the head. Clinical features at baseline that are associated with an abnormal findings in CT scans of the brain were an age of at least 60 years, immunocompromised patients and a history of CNS disease (Hasbun et al., 2001). A cranial MRI is an important diagnostic procedure when patient had symptoms and signs of a lesion in posterior fossa. Both CT scan and MRI should be reviewed especially for

finding mass or signs of raised intracranial pressure (RICP) in posterior fossa and quadrigeminal cistern. Physician should carefully consider on the indications of the procedure and do other studies which are essential to perform simultaneously like measuring serum glucose and CSF pressure.

Patients should be carefully informed; explaining the procedure to the patient/ care giver/ next of kin completely and signing an informed consent for back pain, leg pain, headache, bleeding, infection, and death are important legal issues. To prevent hemorrhage platelet count and International Normalized Ratio (INR) should be more than 50,000 and less than 1.5, respectively (Table 1).

Full neurological examination especially for papilledema, hemiparesis and focal neurologic deficits
Cranial CT and/or MRI in all patients with papilledema, focal neurologic deficit, recent epileptic episodes
Obtain cranial MRI in all patients with posterior fossa lesions
Check for visualization of quadrigeminal cistern and fourth ventricle especially in patients with bacterial meningitis
Correct bleeding diatheses such as thrombocytopenia and INR>1.5
Careful consideration for indications or contraindications of LP
Obtaining informed consent from the patient/care giver for the procedure

Table 1. Steps prior to lumbar puncture.

3. Technique of the procedure

Experience will teach us the importance of meticulous of each technique. With a thorough knowledge of the contraindications, regional anatomy and rationale of the technique, and adequate prior skill, lumbar puncture can be carried out safely and successfully. Lack of knowledge may lead to a higher mortality and morbidity rate. It was mentioned that the most important elements of procedural competency are the cognitive aspects and anatomy plays a major role in this domain (Boon et al., 2004). There are two different positions for performing LP. For diagnostic LP, the left lateral recumbent position (for right-handed physicians) is preferred. Patient's knee and neck are flexed to overcome the lumbar lordosis that narrows the interspace between adjacent spinous processes and lamina. Sometimes they are in seated position and only the neck is fully flexed. Ill patients are unable to sit up and for pressure measurement the patient should be in recumbent position. In preterm infants it was suggested that the sitting or modified lateral recumbent, without knees-to-chest position, which results in less hypoxemia with the neck maintained in the neutral position is better than the classic position (Weisman et al., 1983).

Proper positioning is critical to success the procedure and the coronal plane of the trunk should be on right angle to the floor, one hip should stay exactly above the other, and the back should be close to the edge of bed because of pouring CSF in to the tube is easier (Fig.1). Inward rotation of the patient makes it difficult to obtain the CSF therefore it is necessary to place a pillow under the ear of the patient in lat recumbent position (Fig.2). In one study it was shown that fluoroscopy guided lumbar puncture may decrease the frequency of traumatic LP from 10.1% to 3.5% (Eskey et al., 2001). However, the patient

charges for standard lumbar puncture and fluoroscopy-guided lumbar puncture were $393 and $559, respectively. Ultrasound machines with linear array transducers can also localize spinal interspaces noninvasively, in 2-3 minutes, exclusive of the time for setting up the ultrasound machine (Peterson et al., 2005).

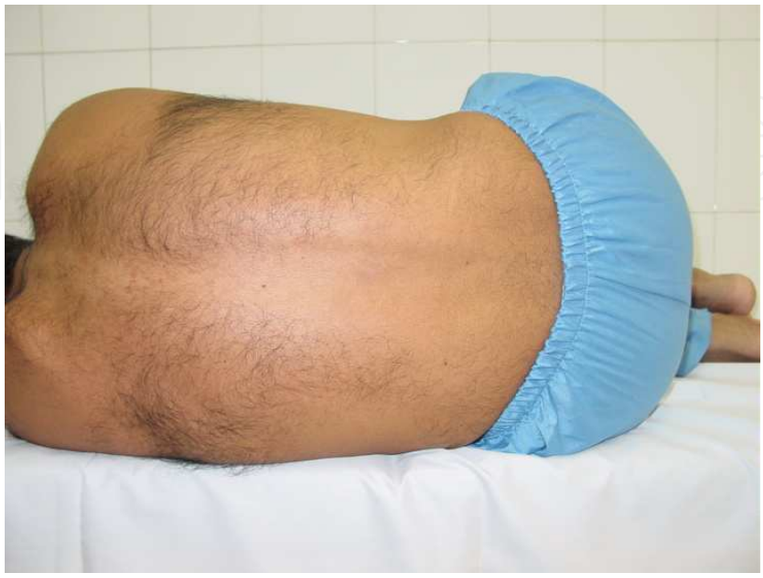

Fig. 1. Proper positioning of the patient for lumbar puncture. The pelvis is vertical and close to the edge of the table.

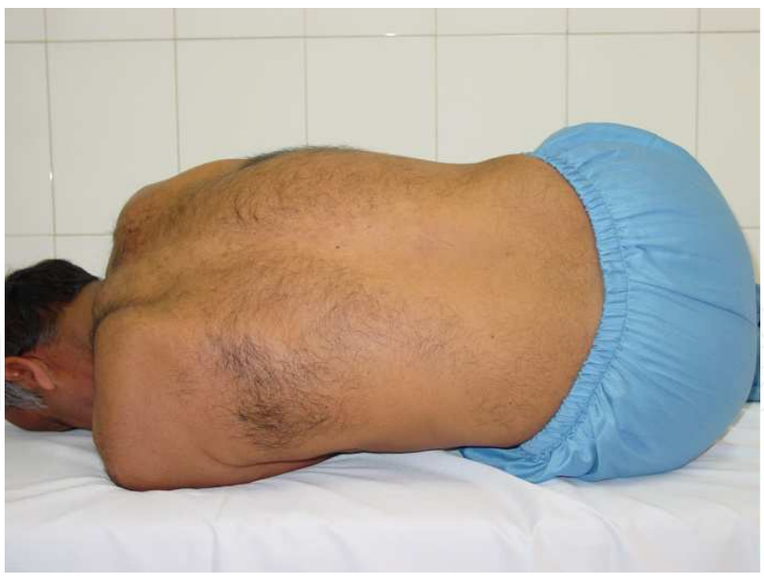

Fig. 2. Incorrect positioning of the patient due to inward rotation of the upper shoulder.

For the safety of both patient and physician the procedure should be done under sterile conditions. At least physician should wear sterile gloves, eye glasses and a mouth mask. Wearing a gown especially for patients with meningitis or meningoencephalitis is considered mandatory. The area should be cleaned and sterilized with 10% povidone iodine

(Betadine) and draped with a sterile towel with a whole in the middle which is large enough to visualize and find the correct place for insertion of the needle (Fig.3). It is better to anesthetize only the subcutaneous tissue of the intervertebral space by 1% to 2.5% lidocaine; however, experienced physicians especially in thin patients do not need it. Warming of the analgesic by rolling the lidocaine vial between the palms may diminish the burning sensation after cutaneous infiltration of the drug.

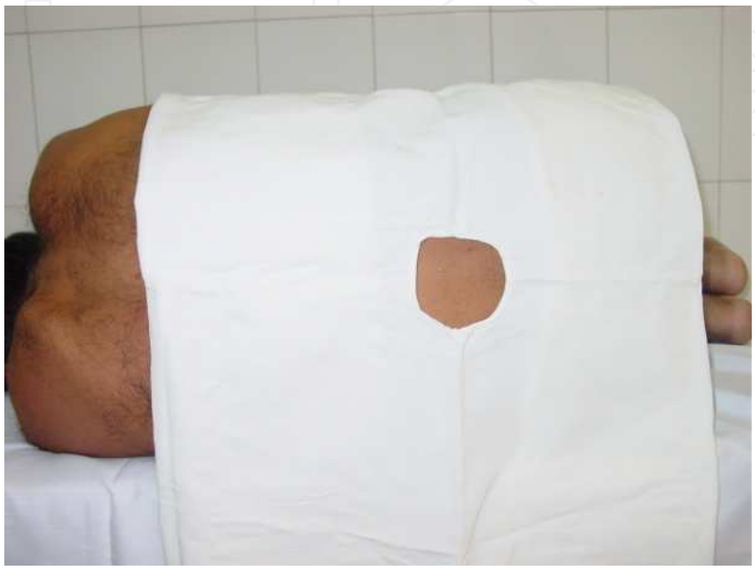

Fig. 3. Correct draping of the patient.

The best intervertebral space is L4-L5 but one space lower (L5-S1) or higher (L3-L4) may be used. In 94% of individuals conus medullaris will extend in to the L1-L2 interspace. In the remaining 6% it extends to the L2-L3 interspace (Fishman, 1992b). The possibility of injury in to the spinal cord will increase with performing the procedure at the L1-L2 or L2-L3 interspaces. In newborns and infants it is safer to use the lowest interspace. The needle is passed horizontally, parallel to the floor. Lumbar puncture is performed by inserting an 18 to 25 gauge needle. The smaller the gauge number, the bigger the diameter of the needle. The size of needle with gauge number 18 indicates that the diameter of the needle is 1/18 inch and for the gauge number 25 indicates 1/25 inch. For diagnostic collection of CSF, a larger gauge needle (18, 19 or 20 standard gauge needles; 22 G, 3.5-cm long needle for neonates; 20 G, 5-cm long needle for children) should be used. Finding the L4-L5 interspace in recumbent position is not very difficult by placing the index finger on the superior part of the upper iliac crest, then drawing an imaginary line between to upper and lower crests (Tuffier's line) and put the thumb of the same hand (for right handed individuals use the left hand and vice versa) in the interspace that the Tuffier's line is crossed with (Boon, Abrahams et al., 2004).

The standard needle for LP is Quincke needle with a bevel sharp tip at the end of the needle. The smaller the diameter the lowest risk for post lumbar headache but the procedure, obtaining CSF and measuring pressure will be more difficult. The bevel head of the needle must be parallel to the longitudinal fibers of the dura mater. It should be inserted at the superior aspect of the inferior spinous process, aimed to the umbilicus (15° cephaled).

There are two types of atraumatic needles, Whitacre pencil-point needle which is available since 1951 (Hart et al., 1951) and Sprotte needle since 1979 with a duller tip and a small oval opening proximal to the tip (Fig.4) (Sprotte et al., 1987). It should be pointed out that Sprotte needle is a modification of the Whitacre needle with larger laterally placed opening. Because of its dull head it needs an introducer which should not shear the dura, therefore it is technically more difficult to use, however, majority of physicians use Quincke needle nowadays. When using Quincke needle the flat portion of the bevel tip should be in line with and parallel to the longitudinal fibers and the hub's notch of the stylet should point to the lateral side of the patient (Evans, 1998).

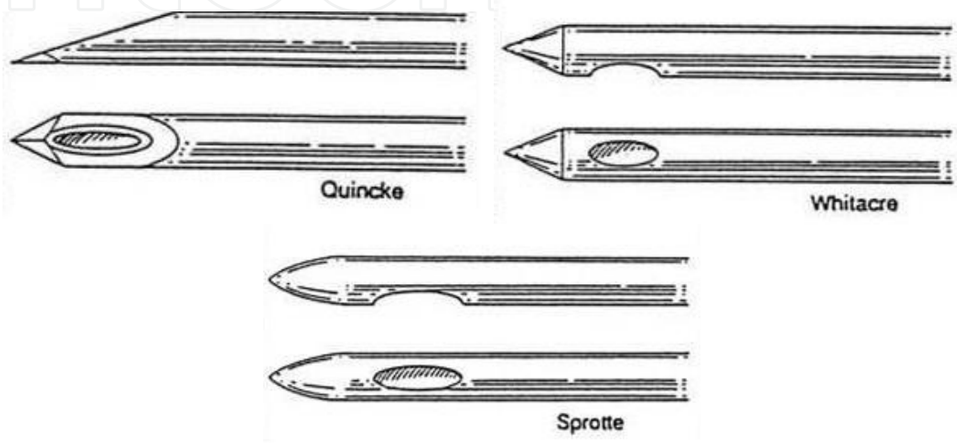

Fig. 4. Three types of spinal needles: Quincke, Whitacre and Sprotte.

When the needle pierces the supraspinous ligament and ligamentum flavum a sudden yielding sensation is often felt often referred by clinicians as a "pop". After entering the ligamentum flavum (1cm thick), the stylet should be removed each 2 mm, however, if the fluid is not appeared in the hub, stylet should be replaced again and the needle rotates 90 degrees, till the bevel head be in the way of CSF. Then, if the fluid does not appear again, the needle should pierce a few millimeters more and a second "pop" may be felt again. If the procedure fails again, the needle should be withdrawn almost under the skin where it can possible to redirect the needle without making another painful needle insertion. The stylet should always be removed slowly from the needle in order to avoid sucking a nerve root in to the lumen and/or causing radicular pain.

When the fluid appears in the hub, a three-way stopcock will be fixed at the hub and a manometer will be attached. A slow raise in the CSF pressure may indicate the obstruction of the tip of the needle with meningeal membranes or nerve roots. Normal CSF pressure in adults is 100-180 mmH_2O (8-14 mmHg) and 30-60 mmH_2O in children (Ropper et al., 2009). Sometimes a firm abdominal pressure, with the help of an assistant and rapid rise and fall in the pressure indicate the free flow of the CSF (Fishman, 1992b). Aspiration of the CSF by a syringe may increase the risk of subdural hemorrhage or root herniation through the opening of the needle and it is not recommended routinely. The rationale for using a stylet when inserting a non-cutting needle (Sprotte) into the subarachnoid space is the possibility of introducing a small piece of epidermoid tissue in to the subarachnoid space and increasing the risk of development of an epidermoid tumor. Reinserting the stylet before

removing the needle may prevent a thread of arachnoid membrane to be withdrawn and CSF leakage will be shorter. Therefore the stylet should always be reinserted before the needle is withdrawn especially in non-cutting needles. Patient may remain flat for 4-24 hours and can take fluid liberally (Roos, 2003). The "dry tap" is almost always due to improper placement of the needle especially when the needle is placed far laterally than to the obliteration of the subarachnoid space by a compressive lesion in the cauda equina or lumbar arachnoiditis (Ropper and Samuels, 2009).

4. Bed rest after the procedure

There is not any firm recommendation for bed rest after LP and there is also no preference how to lie after the procedure on the side, supine or on the face. Spriggs *et al.* (Spriggs et al., 1992) performed a randomized study to assess the immediate effect of mobilization with 4 hours bed rest on the incidence of PLPH. They did not found any difference between the mobile (54 cases) and bed rest (56 patients) groups in the incidence of PLPH (32% versus 31%, respectively). The report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology (Evans et al., 2000) recommendation noted that Class 1 evidence shows no benefit for the prevention of PLPH after 24 hours bed rest. In our department we ask patient to be at bed for one hour and after that they can walk at their will but we recommend them to be at rest during the remainder of the day. This is a successful practice till now in our department.

5. Transient complications

One of the most frequent transient complications is transient stabbing pain in the territory of sensory nerve root(s) due to contact of the spinal root with the needle. However, pain may be revealed with patient repositioning. In one study it was occurred in 13% of patients during LP (Dripps et al., 1951) but permanent motor or sensory loss is extremely rare (Dahlgren et al., 1995). It seems to be that analgesia is underused in infants and children, however, it should be mentioned that sometimes it takes less than an hour to be efficacious (Baxter et al., 2004). In case of severe nerve injury, pain may persist for a longer time. In the seated position, a drainage headache may be felt and the risk of faint is higher in this group of patients. Men are more sensitive to pain and may show a vasovagal syncope especially when they are seated. Infusion of intravascular normal saline before starting the procedure may decrease the risk of this problem (Table 2). Dysfunction of the 3rd to 8th cranial nerves has been reported in many case reports but they are transient and reversible complications probably secondary to the traction of the nerve over the cranium (Broome, 1993; Lybecker et al., 1995a).

Post-Lumbar Puncture Headache (PLPH)
Cerebral (brain) herniation
Infection
Backache
Subdural or epidural hematoma
Subarachnoid hemorrhage
Implantation of epidermoid tumor
Root irritation/Radicular pain

Table 2. Potential complications of lumbar puncture.

6. Post–Lumbar Puncture Headache (PLPH)

Post-lumbar puncture headache (PLPH) is the most frequent complication (40%) after performing diagnostic LP (Evans, 1998). Headache worsens 15 minutes after sitting or standing and improves within 15 minutes after lying down in the bed and it should be accompanied with one or more of symptoms such as neck stiffness, tinnitus, hypacusia, photophobia and nausea (Dakka, Warra et al., 2011). Tourtellotte *et al*.(Tourtellotte et al., 1972) reported that the average frequency of PLPH after diagnostic LP (excluding myelography and pneumoencephalography), obstetric spinal anesthesia and non-obstetric spinal anesthesia was 32%, 18%, 13%, respectively. In patients received special measures to prevent headache, the average frequency of PLPH in the above groups was decreased to 6.0%, 6.2% and 5.5%, respectively.

PLPH is more frequent in young individuals, thin women (Kuntz et al., 1992) and patients with previous headache (Evans, Armon et al., 2000). It occurs two times more in women than in men (Kuntz, Kokmen et al., 1992) and most of the increased frequency has been seen in child bearing age (18 to 30 years old) (Tourtellotte, Henderson et al., 1972). The frequency is lower in children less than 14 years (Bolder, 1986) and adults more than 60 years (Tourtellotte, Henderson et al., 1972). In a prospective study of 239 patients, pain was located within the region innervated by the trigeminal nerve in 49% of the patients, within the occipital and/or suboccipital region in 11%, and within the combined trigeminal/occipital region in 39% (Vilming et al., 1998). Temporal region is the most frequent place. The intensity of associated symptoms was positively correlated to PLPH severity. However, the classic feature of the headache is a bilateral generalized throbbing or pressure sensation which is increased with sneezing, sitting, coughing or straining especially 48-72 hours after the procedure. Associated symptoms were experienced by 85% of patients, nausea in 73% and dizziness in 60% being the most frequently reported. Those symptoms are more present during the upright position. It may be associated with neck stiffness, nausea and vomiting. Quite rarely there are unilateral or bilateral 6th palsy even at times without headache (Ropper and Samuels, 2009). The pain is revealed after lying down again in the bed (Vilming and Kloster, 1998; Roos, 2003). In particular, PLPH is a constant headache appearing or worsening significantly when standing and resolving or improving significantly upon lying down, however, the severity of the headache has not been defined completely (Lybecker et al., 1995b).

Muller *et al.*(Muller et al., 1994) in a double blind placebo controlled study showed that only 2 out of 42 patients, using Sprotte needle, had PLPH, backache or orthostatic hypotension comparing to the 15 patients out of 48 remaining patients whom experienced Quincke needle for LP. Convincing Class 1 evidence shows that the thicker Quincke needle is associated with more PLPH (Lybecker et al., 1990) from 70% when using 16-19 G to 12% when using 24-27 G needle (Evans, Armon et al., 2000). In another study it was shown that the frequency of PLPH will decrease from 18.9% after using Quincke needle 22 G comparing to 2.3% of PLPH with a 27 G needle. The frequency of PLPH after using atraumatic needles with the same gauges were 6.7% and 0.4% respectively (Carson et al., 1996). It is consistent with the theory of CSF leakage from the puncture site and intracranial hypotension due to presence of a whole in the dura. Thinner needles are harder and more difficult to use, unsatisfactory for measuring CSF pressure and sometimes it requires a syringe to drain CSF, however, it is more useful in performing spinal epidural anesthesia and lumbar

myelography. Actually needles thinner than 20 G may not be practical until a small amount of fluid is needed.

There is Class 1 evidence that in the case of parallel insertion of the bevel head of Quincke needle with longitudinal dural fibers rather than perpendicular insertion may be associated with a lesser incidence of PLPH because the dural fibers run parallel to the long axis of the spine (Flaatten et al., 1998). When the bevel head is perpendicular to the dural fibers more of them will be severed and the risk of PLPH is 50% more in this group of patients comparing to the parallel group. Norris *et al.* in their study reported that fourteen of 20 women in the group in which the needle bevel was perpendicular to dural fibers developed a moderate to severe headache, whereas only five of 21 in the group in which the needle bevel was parallel to dural fibers did so (Norris et al., 1989).

The incidence of PLPH is lesser when the stylet reinserts before withdrawing the needle. Strupp *et al.* in a randomly assigned trial in 600 patients reported that the incidence and severity of PLPH was decreased from 16% to 5% after replacement the stylet (Strupp et al., 1998). They used Sprotte needle for performing LP in their 600 patients. Despite there is not any clear evidence for reinserting stylet before withdrawing a Quincke needle but it seems that it is safer to insert the stylet in all types of needles. The main disadvantage of Sprotte spinal needle is that it is technically more difficult to use than Quincke standard needle. Sprotte needle needs an introducer to insert up to the dura. It should not be pierce dura; therefore, the best results with atraumatic needles are depended in the experience of the physician. Sometimes it is not possible to perform LP with Sprotte needle and the physician has to change into the Quincke needle (Jager et al., 1993). It is possible that Sprotte needle will be damaged during the procedure because it needs more pressure and the sensation of cutting dura is different from the Quincke standard model (Benham, 1996).

The "pencil point" non-cutting needles of Whitacre or Sprotte had dull tips and an oval opening just proximal to the tip in contrast to the Quincke classic needles. Contrary to common use of those needles by anesthesiologists, many neurologists never heard anything about such needles and only 2% of neurologists use such needles (Evans, Armon et al., 2000). There are some reports confirming the use of small gauge non-cutting needle for spinal anesthesia especially in patients prone to PLPH (Halpern et al., 1994).

The amount of spinal fluid removed is not a risk factor for PLPH (Kuntz, Kokmen et al., 1992). There is not any Class 1 evidence confirming the effect of duration of recumbency in the bed after LP for preventing PLPH one century after Bier's recommendation (Bier, 1899) for staying in the bed at least for 24 hours (Corbett et al., 1983; Spriggs, Burn et al., 1992). However, many physicians still advise their patients staying in the bed for some hours. The role of psychological factors in the development of PLPH appears to be minimal. The frequency of PLPH without any awareness of the procedure was the same as in patients who were aware of the procedure (Raskin, 1990). The majority of headaches is self limited and will be resolved in 5 days. Bed rest is the simplest way of treatment. The patient stays on recumbent position and liberally takes fluids with a simple analgesic like acetaminophen, codeine, caffeine benzoate. Parenteral use of analgesics such as intravenous caffeine sodium benzoate by inactivating brain adenosine receptors may cause vasoconstriction (Raskin, 1990) and decreasing CSF pressure. Intramuscular NSAIDs such as piroxicam or diclofenac may be effective.

Injection of 20-30 ml of saline making an epidural saline patch may cause an increase in epidural pressure and stop headache abruptly (Bart et al., 1978; Baysinger et al., 1986). In refractory cases an epidural blood patch or epidural saline close to the site of puncture will be useful (Evans, Armon et al., 2000). The best place for administration of blood patch is the same interspace level of LP but in case of any problem it is better to use one space lower than the puncture site. In multiple punctures the lowest interspace is the best one (Evans, 1998). The recommended amount of the blood is 15-20 ml with a rate of 0.5 ml/sec. to achieve maximal benefit it would be better that the patient stays in decubitus position for 1-2 hours. The reported success rate is about 95% and it should not be earlier than 24 hours after LP (Tarkkila et al., 1989). Prophylactic epidural patch does not recommend and does not prevent PLPH (Berrettini et al., 1987). Epidural injection of blood may form a gelatinous tamponade over the dural hole that prevents CSF leakage and immediately stop headache (Raskin, 1990). Currently the cost of blood patch in the United States is about $1500 each time (Dakka, Warra et al., 2011). There is not any evidence for using oral or intravenous fluids to diminish headache after LP (Table 3).

Bed rest: Majority of patients are being improved in two days
Intravenous caffeine benzoate sodium (500 mg intravenous infusion)
Caffeinated beverages
Epidural blood patch
Epidural saline injection

Table 3. Treatment of post-lumbar puncture headache (PLPH).

7. Cerebral herniation

Another important and not commonly occurred complication of LP is cerebral (brain) herniation due to asymmetric increase in cranial vault or highly severe symmetrically increase in intracranial pressure. Theoretically, when the CSF is under increased pressure and there is an obstruction to the free flow between the supratentorial space and the thecal sac surrounding the spinal cord, removing spinal CSF under these circumstances would decrease the pressure below the tentorium cerebelli and allow the temporal lobes to be herniated downward, impinging on the brainstem. Increased intracranial pressure without the obstruction of CSF circulation usually does not have this catastrophic effect, for instance, in pseudotumor cerebri or acute bacterial meningitis (ABM) (Archer, 1993). In ABM brain herniation occurs in 5% of patients in which accounts for a significant proportion of deaths due to ABM (Joffe, 2007).

Rebaud *et al.* (Rebaud et al., 1988) in their study in a group of children with ABM reported that 86% had raised intracranial pressure (RICP). Intracranial hypertension defined as increased mean pressure more than 15 mmHg. Totally they analyzed intracranial pressure in 14 children with Glasgow Coma Scale (GCS) less than 7. The mean opening pressure in survivors and nonsurvivors were 14 ±6 and 55 ±25 mmHg, respectively. In another study Minns and his colleagues showed that 33 out of 35 patients with ABM had RICP (Minns et al., 1989). Those studies showed that RICP is common in ABM. This is attributed to hyperemia, impaired CSF outflow resistance and finally cerebral edema which is inflammatory in the earlier stages of the disease (Saez-Llorens et al., 2003). In most studies

brain herniation defined as either pathologic confirmation at autopsy or at least two signs of herniation including loss of consciousness, asymmetric unresponsive pupillary dilatation, irregular respiration and/or respiratory arrest and finally decorticate/ decerebrate response or complete flaccidity (Rennick et al., 1993).

Korein *et al.* in a collection of 418 patients reported that only 1.2% of patients had brain herniation after LP (Korein et al., 1959; Joffe, 2007). It has been pointed out that regardless of ICP, LP may hasten herniation in patients with RICP only if brain shift is present. Sometimes it is a gradual process because of ongoing CSF leakage from the LP site or cerebral vascular engorgement and edema after LP (van Crevel et al., 2002). It should be reminded that herniation *after* LP does not necessarily means herniation *caused* by LP. It seems that LP cause herniation in patients with papilledema but it was not definitely approved (van Crevel, Hijdra et al., 2002).

There is strong evidence that when herniation occurs in ABM, it most often occurs shortly after an LP. Joffe in his review reported that 79% of cerebral herniation was occurred during the first 12 hours after LP, half of them in the first 3 hours (Joffe, 2007). The reported odds ratio for herniation occurring in the first 12 hours after the LP is 32.6 (95% CI, 8.5 to 117.3) (Rennick, Shann et al., 1993). Neuroimaging can provide the structural information from which pressure data must be inferred (Gower et al., 1987). Those findings include lateral shift of midline structures, loss of the suprachiasmatic and basilar cisterns, obliteration or shift of the fourth ventricle, obliteration of the superior cerebellar and quadrigeminal cisterns with sparing of the ambient cisterns. Fourth ventricle and quadrigeminal cistern should be visualized and open on CT; otherwise LP should not be done (Fig.5).

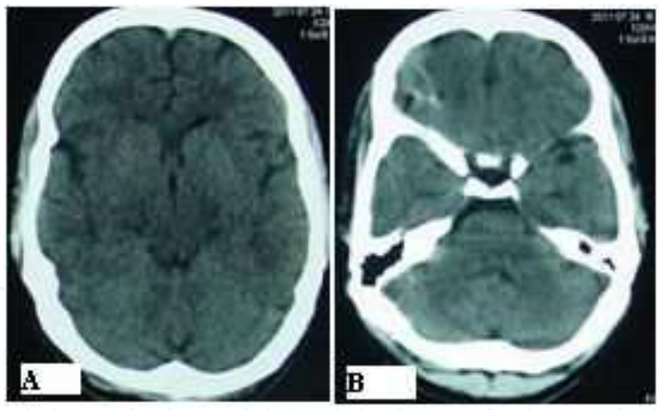

Fig. 5. Oblitration of quadrigeminal cistern (A) and fourth ventricle (B) in a patient with bacterial meningitis.

Only using very thin Quincke needles (26 G) or atraumatic Sprotte needles (22 G) reduce the incidence of herniation but it is unfeasible in practice but the incidence of brain herniation is not decreased by a head-down position (van Crevel, Hijdra et al., 2002). Therefore, a neuroimaging procedure is indicated prior to LP in all patients with papilledema, focal neurologic signs, significant altered level of consciousness or new onset seizure (during the last 24 hours) which are risk factors for abnormal CT scan (Kastenbauer, Winkler et al., 2002), however, very few patients in these studies had ABM. Patients with early signs of uncal or central cerebral herniation which is called "incipient herniation" are most

vulnerable to complete the herniation process. Therefore, it seems that clinical signs and symptoms are the most reliable indicators of brain herniation after LP in the setting of ABM and normal brain CT scan (Joffe, 2007). The overall mortality of cerebral herniation after analysis of 8 articles was 60 out of 107 patients (56%). A good outcome for 51% of survivors (23 of 45) has been shown (Joffe, 2007). CT scan is more available in most emergency setting than MRI. However, CT cannot reliably estimate the risk of cerebral herniation after LP in ABM because of considerable variability in the size of lateral ventricles; decreased CSF absorption, decreased meningeal compliance due to meningeal inflammation and prevention of narrowing of the subarachnoid and ventricular spaces. Finally, a normal CTscan does not necessarily mean that an LP is safe. Avoiding LP also has two major risks. The first one is missing the exact cause of bacterial meningitis and appropriate treatment and the second one is misdiagnosis in some similar patients such as tuberculous meningitis (TBM), cerebral malaria, encephalitis and opportunistic infections in immunocompromised patients. Both consequences will postpone adequate therapy.

In conclusion, about 5% of patients with ABM have cerebral herniation, accounts for 30% of the ABM mortality. Although CT scan can reveal important data on the likelihood of herniation but a normal CT does not necessarily mean that LP is a safe procedure in ABM. Clinical correlation with impending signs of herniation is highly recommended for making decision. When herniation occurs it should be treated aggressively because there are many reports indicating good outcomes. In patients considered with signs of RICP, interventions to control ICP such as mannitol infusion (1gr/kg) and antibiotics should be started before a CT scan obtains and LP should *not* be performed. Hypertonic intravenous urea, mannitol, ventricular drainage, and in two studies, the "Lund protocol" of ICP management had been proposed for treatment of cerebral herniation after LP. The response to mannitol is often rapid. Some authors prescribe mannitol infusion 30-60 minutes before doing the procedure in patients with impending signs of herniation (Roos, 2003) but we do not recommend. Of course, the outcome is not dismal in many patients.

8. Bleeding (epidural or subdural hematoma/ subarachnoid hemorrhage)

Many patients complain back or low back pain after LP. Only simple reassurance and oral analgesics are enough except when there are neurological signs and symptoms concerning the involvement of spinal cord. Interventricular hemorrhage, subarachnoid hemorrhage (SAH) and spinal epidural hematoma have been reported after LP (Lee et al., 2009). Bleeding from small vessels of the ligamentum flavum or rupture of an epidural vein either by a sudden increase in the intra-abdominal pressure impacting on a previously damaged or weakened vein or by mild trauma can also cause spinal epidural hematomas (Gurkanlar et al., 2007). Sometime penetration of Adamkiewicz artery and/or vein is the responsible etiology (Scott et al., 1989). Progressive paraparesis, saddle anesthesia, low back pain and sphincter incontinence are the major neurological findings (Sweasey et al., 1992).

Those symptoms are completed in hours or days. Emergency MR scanning of the spinal cord is the first diagnostic procedure and early surgical decompression is mandatory. While patients with bleeding diathesis are more prone to local bleeding, SAH or intracranial subdural hematoma (uni- or bilateral) are usually seen in healthy individuals. The responsible mechanism is probably due to traction on bridging veins secondary to intracranial hypotension. Subdural hematoma may occur anywhere in the cranial vault but SAH may be presented in the territory of a fragile aneurysm (Vos et al., 1991).

9. Infection

Inadequate sterilization of the skin or a breach in the aseptic technique may induce rare but important complications of LP such as purulent meningitis or disc space infection. However, subcutaneous and/or epidural abscesses are sometimes reported. The most frequent causes are intravenous drug abuse, diabetes mellitus, trauma, alcohol abuse, wound infection, multiple medical illnesses and prior spinal surgery (Kamiyama, 2006). There are different ways to contaminate the skin: contamination of the skin site and subsequent spread along the needle track, by hematological spread, or by intraluminar contamination via a contaminated syringe or contaminated local anesthetic solution (Phillips et al., 2002).

Infectious complications ranging from vertebral osteomyelitis, discitis, epidural abscess, and bacterial meningitis have reported in the literature. In another study it was revealed that in consecutive neuroradiological procedures requiring LP, bacterial meningitis occurred with an incidence of 0.2% soon after the procedure (Domingo et al., 1994). The clinical signs and symptoms of vertebral discitis include pain, local tenderness, irritability, and spinal irritation usually two weeks after the procedure (Bhatoe et al., 1994). Lumbar puncture for analysis of CSF at different interspace other than the original puncture site is mandatory. The process of active degeneration to fusion takes about 6 to 24 months. One case report described retroperitoneal abscess that occurred after dural laceration and penetration of infected CSF to the abdominal cavity (Levine et al., 1982).

10. Contraindications

Lumbar puncture is an extremely safe procedure when performed by an experienced physician using standard methods and techniques (Table 4). LP is contraindicated in patients with asymmetric increase in cerebral pressure in one part of cranial cavity. It should not be performed when fourth ventricle or quadrigeminal cistern was not visualized. Other contraindications are spinal epidural abscess or subdural empyema because of telescopic herniation of the spinal cord or introducing infection into the spinal subarachnoid space when the abscess or empyema located in lumbar region. Lumbar puncture is also contraindicated in patients on coumadin therapy or platelet count less than 50,000/μL, shock with cardiorespiratory compromise and local superficial infection at the site where LP should be done. When there is thrombocytopenia and strong clinical indication for an LP, particularly in patients whose platelet count is below 20,000 per cubic milliliter, platelets should be transfused just prior to the procedure to reduce the risk of a procedure-related hemorrhage. Nevertheless, the risk of epidural or subdural hematoma should also be diminished before performing LP (Edelson et al., 1974).

Evidence of intracranial mass lesion
Evidence of midline shift on cranial CT/ MRI
Presence of posterior fossa tumor
Platelet count less than 50,000 per cubic milliliter
INR greater than 1.5
Poor visualization of quadrigeminal cistern or fourth ventricle
Known suppurative infection in the lumbar region
Presence of spinal subarachnoid block

Table 4. Contraindications to lumbar puncture.

11. Normal cerebrospinal fluid analyses

After performing LP the gross appearance of CSF should be noted. A complete CSF analysis can assist in the diagnosis and management of many central and peripheral nervous system disorders. Normally the CSF is clear and colorless. One of the strongest indications for urgent LP is suppurative infections of CNS. It should be examined for cells (white and red blood cells), microorganisms (bacteria, fungi, mycobacteria, cryptococcal antigen, herpes and cytomegalovirus DNA), protein and glucose content, gammaglobulines and cytological study for tumor cells. CSF will be changed in all patients with inflammatory, infectious, neoplastic and sometimes degenerative central or peripheral nervous system disorders.

11.1 Pressure and dynamics

An opening pressure should be measured in all patients with altered level of consciousness, papilledema, hydrocephalus and infections of the central nervous system. The upper limit of normal CSF pressure in lateral recumbent position is 110 mmH_2O in infants, 150 mmH_2O in children, 180 mmH_2O in adults and 200-250 mmH_2O in obese individuals (Corbett and Mehta, 1983). Pressure less than 50 mmH_2O indicates intracranial hypotension due to CSF leakage or severe dehydration. CSF must be measured in recumbent position. When it is measured in sitting position, the fluid may rise to the cisterna magna but not to the ventricle level because of presence of negative pressure in the closed cranial cavity. Pressure in this situation is about two times higher than recumbent position (Ropper and Samuels, 2009).

11.2 Cells

During the first month of life CSF contains a small number of leukocytes. Beyond this period, in uninfected CSF the number of leukocyte (WBC) count is zero to five mononuclear cells (monocytes or lymphocytes) per cubic milliliter. CSF should not contain polymorphonuclear (PMN) in the uninfected CSF. However, if the total number of cells is less than five, the presence of one PMN per cubic milliliter is not considered abnormal. Counting leukocytes needs an ordinary chamber but identification needs Wright stain of the centrifuged sediment. A "traumatic tap" will be occurred when a radicular artery or epidural venous plexus is penetrated with the spinal needle and blood is introduced into the spinal fluid. In this situation, to calculate the true number of WBC count in the CSF one WBC per cubic milliliter for every 700 RBC per cubic milliliter is subtracted from the total number of the WBC count in the CSF. In case of traumatic tap in the collection tube, a clot or a thread of blood will be seen in the CSF. When a small amount of blood entered into the CSF, it is possible to collect CSF after dripping for a few seconds but in case of more amounts of blood it is necessary to change the position of needle and enter into the another interspace. The presence of 200 RBC per cubic milliliter cause a ground-glass appearance, 1000-6000 RBC per cubic milliliter imparts a hazy pink to red color and several hundreds of WBC per cubic milliliter (pleocytosis) induces a slight opaque haziness of the CSF color, respectively (Ropper and Samuels, 2009).

To distinguish a traumatic (bloody) tap from SAH the number of RBCs and WBCs remains in the first and it will be diminished in third or fourth tube. In SAH the CSF color remains pink-tinged through all tubes and the supernatant is xanthochromic (yellow-brown) after CSF centrifuge. Xanthochromia will be occurred 2 to 12 hours after SAH and persists for a few weeks. The reason for rapid hemolysis of RBCs in the spinal fluid after SAH is not clear.

In case of multiple punctures of the radicular arteries of different interspaces it is possible that the CSF color became blood tinged due to entrance of RBCs in to the subarachnoid space. Special immunochemical staining and electron microscopy will be helpful for the diagnosis of malignant cells in the CSF (Bigner, 1992).

11.3 Glucose

The concentration of glucose in the CSF depends on the serum glucose. Normal CSF glucose concentration is between 45 and 80 mg/dL when the serum glucose is between 70 and 120 mg/dL or approximately two thirds of serum glucose (about 0.6 to 0.7 serum glucose). CSF glucose range is in parallel with higher level of blood glucose. With marked hyperglycemia the ratio is lower (0.5-0.6) and with marked hypoglycemia the ratio is greater (0.85). Hyperglycemia will mask hypoglycorrhachia, however, CSF glucose less than 45 mg/dL will be considered abnormal (Fishman, 1992a) and values less than 35 mg/dL are always abnormal (Ropper and Samuels, 2009). It takes 2-4 hours after intravenous glucose injection to have equilibrium with CSF glucose. Therefore it is mandatory to measure serum glucose level at the time of LP. In normal individuals CSF/serum glucose ratio is about 0.6 and CSF/glucose ratio less than 0.6 considered to be abnormal.

11.4 Protein

In contrast to the high concentration of protein in the blood (5500 to 8000 mg/dL) the protein concentration of CSF is low. Protein concentration is also diminished in a rostral-caudal direction in the neuroaxis. It reflects a ventriculo-lumbar gradient in the permeability of capillary endothelial cells to protein. The upper ranges of normal cisternal and lumbar CSF protein are 30 and 50 mg/dL in adults, respectively. The choroidal and meningeal perfusion is also increased in bacterial meningitis.

12. Cerebrospinal fluid analysis for bacterial meningitis

Independent of the causative agent of bacterial meningitis, CSF findings in acute bacterial meningitis are often similar. The major determinants of CSF profile are the time duration between performing LP and the onset of infection, severity of the disease, clinical setting in which the infection was acquired and the immune state of the patient (Venkatesan et al., 2009). The opening pressure is typically increased in almost all patients. In 90% of cases the opening pressure is over 200 mmH_2O, in 20% more than 400 mmH_2O and in 15% of patients it is more than 500 mmH_2O (Durand et al., 1993; Venkatesan and Griffin, 2009). Pressure will increase parallel with the progression of the disease and it returns normal with recovery. However, it should be concerned that the normal pressure values are different between different age groups. Minns *et al.* reported that RICP was found in 33 out of 35 infants with bacterial meningitis with a median pressure of 204 mmH_2O. They concluded that RICP is a frequent accompaniment of childhood meningitis and may need treatment in its own right and is an important factor influencing the course and outcome of childhood meningitis (Minns, Engleman et al., 1989).

At the time of first LP in bacterial meningitis, the cell count of CSF is almost always between 1,000 to 10,000 WBC/mm^3; and rarely is less than 100 or greater than 20,000 WBC/mm^3. Findings are remarkably the same, regardless of the type of causative organism. A recent

study in Alberta, Canada revealed that the cellular abnormality in the CSF did not change over the last fifty years (Hussein et al., 2000). In 90-95% of patients PMN accounts for the total WBC count of the CSF and in less than one quarter of cases do PMNs comprise less than 80% of the total leukocyte count (Venkatesan and Griffin, 2009). However, the CSF leukocyte count may be changed during the antibiotic therapy.

The basics for CSF diagnostic studies for meningitis include cell count with differentiation, measuring protein and glucose concentration, Veneral Disease Research Laboratories (VDRL) slide flocculation test, Gram stain and culture for bacterial pathogens. Hypoglycorrhachia in the presence of pleocytosis is usually diagnostic for pyogenic, tuberculous or fungal meningitis. Other studies are not routinely done in many centers especially in countries with poor resources such as latex agglutination for bacterial pathogens involving in the pathogenesis of bacterial meningitis, India ink for Cryptococcal surface antigen, and other fungal culture, Histoplasma polysacharride antigen, viral specific IgG and IgM for mosquito borne viral encephalitis, polymerase chain reaction (PCR) for HIV, Herpes viruses and enteroviruses. Smear for acid fast bacilli and culture for *Mycobacterium tuberculosis* are especially important diagnostic procedures to be done in countries with high incidence of tuberculosis. *Borrelia burgdorferi* antibodies for Lyme disease in endemic region should also be considered.

The CSF glucose content at first LP is usually moderately to severely reduced. In 75% of patients it is less than 50 mg/dL and in 25% of cases it is less than 10 mg/dL (Hussein and Shafran, 2000). For a long time it was assumed that bacteria use glucose in the CSF of patients with bacterial meningitis but the CSF glucose level stays at a subnormal level two weeks after effective treatment of bacterial meningitis. It suggests that polymorphonuclear leukocytes and cells of the adjacent brain tissue may consume glucose in anaerobic way increasing the lactate level of the CSF. Probably an inhibition entry mechanism of glucose into the CSF may be operative. The CSF glucose level may spuriously low, if CSF remains for a long time in room temperature. The normal range for CSF/serum glucose ratio is >0.6 however in another study of 217 patients with confirmed bacterial meningitis, the ratio was less than 0.23 (Spanos et al., 1989). Low glucose values in the CSF have been reported in patients with viral infections such as herpes simplex, zoster and mumps meningoencephalitis (Ropper and Samuels, 2009).

The CSF protein concentration is almost always increased. In more than 80% of patients the absolute value is more than 80mg/dL (Spanos, Harrell et al., 1989). In less than 8% of cases the protein concentration is greater than 1000 mg/dL, is often associated with subarachnoid block (Venkatesan and Griffin, 2009). Schutte *et al.* (Schutte et al., 1998) reported a good correlation between both the GCS and CSF-protein level on admission and the outcome of patients with meningitis was found, with the GCS value being a better prognostic indicator than high CSF protein levels. In their study CSF protein content was five times higher in patients with severe neurological deficit comparing to patients without any detectable deficit. The protein content of CSF in this situation often reaches to 500 mg/dL or more but viral infection with lymphocytic pleocytosis has lesser elevation of protein usually between 50-100 mg/dL; sometimes normal. In patients with meningeal irritation due to parameningeal infections (meningismus) protein value of the CSF is low. Values more than 1000 mg/dL is deeply yellow in color and may clot in the tube because of high content of fibrinogen which is called Froin syndrome.

13. Conclusions

Although collecting and analyzing CSF is an important part of diagnostic and sometimes therapeutic process in the management of various diseases but it needs careful search for contraindications and problems associated with the procedure. A complete description of the procedure and its possible complications to the patient is mandatory and legally important. The experienced physician also estimates how much sample must be obtained and which kind of especial laboratory analysis should be done before starting the procedure. Therefore, prompt delivery of samples to laboratory may reduce the risk of spurious reports.

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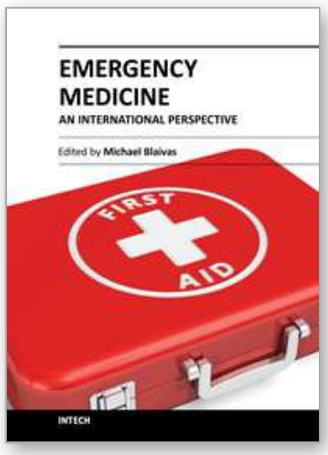

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