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Spinal Anaesthesia and Neurological Complications: A Brief Report

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Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

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ABSTRACT

Spinal anaesthesia is widely performed for lower abdominal, inguinal, and lower extremity operations in routine practice, with a low complication rate. Neurological deficits are rare but significant complications of spinal anaesthesia. These complications have a wide spectrum varying from mild lumbar pain to serious paraplegia, and almost all time anesthesiologists are held responsible for this. However, most of these complications can be prevented by some precautions such as detailed preoperative evaluation of the patients, better anatomic and pharmacologic knowledge, adequate technical support, compliance with routine guidelines of the procedure, and close postoperative monitoring. In this review, the definitions, risk factors, clinical findings, diagnostic and therapeutic approaches, and preventive factors of the neurological complications associated with spinal anaesthesia were aimed to discuss with the relevant literature.

Keywords: Complication; neurological deficitis; spinal anaesthesia.

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1. INTRODUCTION

Spinal anaesthesia (SA) is a common form of anaesthesia, which has been used for more than one century [1]. This central regional block technique is widely performed for various surgical conditions regarding the lower abdomen, inguinal region and lower extremities.

SA has many advantages for both anaesthesiologists and patients. First of all, it is a simple and inexpensive method, and does not require invasive monitoring procedures during surgery. Also, pulmonary, cardiac and haematological risks are minimalized especially in high-risk patients because of the continuation of spontaneous breathing, preservation of some reflexes such as coughing and swallowing, suppression of surgical stress response, providing postoperative analgesia, and early mobilization and nutrition after surgery [2]. However, it is not a complication-free procedure, and carries a low complication rate of 1/100 000 [3]. Although post-spinal headache and hypotension are the most common complications related to SA, various conditions from lumbar pain to death have been reported to date [4,5]. Among those, neurological complications, such as cauda equina syndrome, paraplegia, paresthesia, and radiculopathy, are extremely rare, with an estimated rate of 0.003% [6].

In this paper, the etiopathogenesis, diagnosis and management of these rare and catastrophic complications of SA were aimed to discuss with the relevant literature.

1.1 General Information on SA

SA is generally applied for three main reasons, including surgery of lower abdomen and lower extremities, distinguishing the autonomic nervous system diseases from the organic diseases, and pain treatment for several conditions such as mesenteric ischemia, acute pancreatitis, and vascular diseases of the lower limbs. This technique is performed by the injection of local anesthetic agents and additive agents to the subarachnoid space, and is characterized by temporary sensory, motor, and sympathetic blocks. Both spinal and epidural methods are also known as neuraxial anesthesia, and can be applied as a one-time injection or intermittent bolus/continuous infusion through a catheter.

1.2 Local Anaesthetic Agents

Many local anaesthetic (LA) agents are used for SA in routine practice. These drugs cause a temporary nerve blockage starting at the application site. Although their exact mechanism is not clear, it has been suggested that LA agents close the sodium channels in the nerve cell by interacting with specific receptors [7]. LA drugs in routine use can be classified into three groups according to their concentrations in cerebrospinal fluid; hypobaric (tetracaine and dibucaine), isobaric (bupivacaine, lidocaine and tetracaine), and hyperbaric (mixed solution of bupivacaine or lidocaine with 5-8% glucose). LAs are also classified as amino esters (cocaine, benzocaine, procaine, amethocaine, chloroprocaine, tetracaine, novocaine) and amino amides (prilocaine, lidocaine, bupivacaine, mepivacaine, etidocaine, ropivacaine, levobupivacaine), according to their biochemical structures.

The ester-type LAs are brought down by plasma pseudocholinesterase and liver esterases, and therefore their local and systemic effects are shorter than the amid-type LAs [8]. All LA agents are not used for subarachnoid block. Lidocaine is one of the ester-type LA agents. However, use of this LA agent intrathecally is avoided due to transient neurological symptoms [9]. Bupivacaine, levobupivacaine, and ropivacaine are among the most used LA agents for SA. Although adrenaline is widely added to LAs in order to prolong the duration of drug effects and gradually rise the peak of blood concentrations due to the vasopressor property in minor surgical interventions, this agent is not added intrathecally due to fear of vascular insufficiency of the spinal cord which may result in various severe ischemic-based neurological conditions such as cauda equina syndrome and anterior spinal artery syndrome [10].

1.3 Anatomy and Procedure

Medulla spinalis contains spinal cord and nerve roots, and ends at the lower edge of the L1 vertebrae in men and in the middle of L2 vertebrae in women. Spinal cord is surrounded by three membranes called as pia mater (innermost membrane), arachnoid and dura mater (outermost membrane). Subarachnoid space is placed between the arachnoid and pia mater membranes, and contains spinal nerves and cerebrospinal fluid. Subarachnoid space ends at the level of sacral 2 vertebrae in adults. On the other hand, there are significant anatomical and physiological differences between adults and neonates/children. At birth, spinal cord and dural sac terminate at L3 and S3 vertebraes, respectively, are the end points of

the subarachnoid space. Adult level is not reached until second year of life. Therefore, a low approach (L 4-5 or L 5-S 1) is used to avoid damage to spinal cord. In addition, newborns have a narrow subarachnoid space and low cerebrospinal fluid pressure [11].

Patient position is very important in regional anesthesia, and directly affects the success of the procedure. Lateral decubitus and sitting positions are the most common positions in this technique. Lateral decubitus position is usually preferred in combined spinal-epidural, caudal (infants) and unilateral blocks, while sitting position is frequently used in centraland epidural blocks. Prone and trendelenburg positions are less frequently used methods.

In SA, LA solution is injected into the subarachnoid space of lumbar 3-4 or 4-5 vertebrae (directly into the cerebrospinal fluid) via a spinal needle (Fig. 1). The effect of LA drug is mainly on spinal nerve roots and the dorsal root ganglia. During SA, depending on the location of surgical site, the desired anesthesia level can be obtained by applying different block types including saddle-type, low-level, high-level, and single-sided (hemiblock). Anesthesia depth and block level may also vary according to the patient characteristics and properties of LA as well as type of technique. It is very important that the patient's vital signs and respiratory values should be closely monitored during the operation. In addition, patient's general condition is consistently evaluated by verbal warnings.

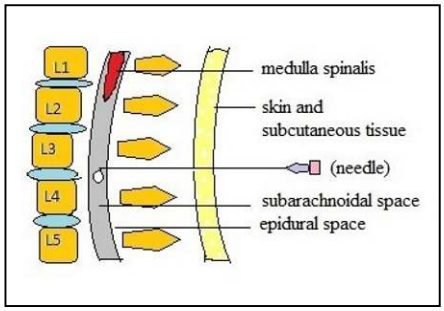

Fig. 1. Schematic illustration of the application of SA

1.4 Neurological Complications of SA

The effects of LA drugs and procedure on the organ systems and potential complications should be well known for the successful anesthetic management, as well as enough anatomic, pharmacologic and technical knowledge. The complications associated with SA can be divided into two groups: Early and late complications (Table 1).

Table 1. Early and late complications of SA

Early	Late
Needle fracture, subdural/epidural haematoma	Headache
Hypotension, bradycardia	Lumbar pain
Nausea, vomiting	Urinary retention
Systemic toxic reactions	Meningitis, meningismus
Cardiac arrest	Neurological sequelae
Apnea	Infection and abscess

Among all complications in SA, the incidence of neurological deficits is extremely low, and was mostly reported as single case or small cases series in the literature [3,6,8]. However, its real incidence can be thought to be higher than known because there may be a greater number of unreported cases, probably due to the legal problems [6]. Various types of neurological complications including transient back and lower limb pain, transverse myelitis, anterior spinal artery syndrome, intracranial subdural hematoma, chronic arachnoiditis, and cauda equina syndrome (CES) have been documented to date [12,13]. Many factors such as direct trauma to the cord, hematoma or abscess at the application site, LA-induced cytotoxicity, spinal cord ischemia due to hypotension, co-existing tumors and arteriovenous malformations, anatomic abnormalities of the spinal cord (e.g. narrow spinal canal), previous spinal surgery play role in the etiopathogenesis of the neurological injury (Table 2) [12-14].

Neurological complications can be transient or permanent. Transient neurological symptoms (TNS) are characterized by post-anesthetic lower limb pain or dysesthesia and tingling, and typically develop within 6-36 hours after procedure [1]. TNS can be occur unilaterally or bilaterally, and may persist for one week. This complication is not rare, and can be observed in up to 37% of the patients who underwent regional anaesthesia [15]. Needle-induced trauma, perivertebral muscle spasm, early mobilization of the patient, irritation of dorsal root ganglia, stretching of the sciatic nerve, and LA toxicity are among the possible causes of TNS. The incidence of TNS is mostly seen in lidocaine use compared with the other LAs [13,16]. In cases of mild or moderate symptoms, non-steroidal inflammatory drugs are sufficient in

the treatment. However, in patients with severe symptoms, an immediate MRI evaluation is needed for determining the main cause. MRI can show the edema at spinal cord, nerve roots or muscle, hematoma, and abscess [17]. Spinal hematoma is a rare but serious complication of regional anesthesia, and often develops in patients who use anti-coagulant drugs [18]. Therefore, all patients should be questioned in terms of anti-coagulant drug use in the preoperative evaluation. The patient's coagulation status should be optimized at the time of regional catheterization, and the level of anticoagulation must be carefully monitored during the procedure. All anesthesiologists must know the general rules of the anesthetic management of patients receiving antithrombotic, anticoagulant, or thrombolytic therapy, which have been recommended by the American Society of Regional Anesthesia and Pain Medicine (ASRA) [19]. The other risk factors for spinal hematoma are personal or family history of coagulation disorders, severe liver and kidney disease, liver dysfunction and preeclampsia with thrombocytopenia, spinal cord diseases such as medullary canal stenosis and ankylosing spondylitis, hypertension and alcohol use [17]. However, no etiological factor can be identified in approximately one third of the patients who develop spinal hematoma following SA, which is called as idiopathic [20]. The management of spinal hematomas should be carried out in coordination with neurosurgery and radiology departments. Hematomas causing severe symptoms should be immediately decompressed while conservative approach with closed monitoring may be preferred for small hematomas or those causing symptoms [21].

Development of spinal abscess is also a dangerous complication of SA, and may cause neurological symptoms from TNS to severe neurological sequels. Diabetes, chemotherapeutic agents, infection of the surrounding area, and ignoring the rules of asepsis and antisepsis are the risk factors for this complication. The treatment is radiology-guided or surgical drainage, with using wide spectrum antibiotics. On the other hand, the patients with TNS due to paravertebral muscle spasm can be treated with simple analgesics, muscle relaxants, warm compress and trigger-point injection [15,22].

Anterior spinal artery syndrome, characterized by weakness, pain and decreased thermal sensation in the lower extremities, is another severe complication of SA [23]. Prolonged hypotension following SA has been reported in most cases to cause spinal cord ischemia or thrombosis of the anterior spinal artery [14,24,25]. Iatrogenic trauma, patient position and vasoconstrictor agents are the other risk factors for developing anterior spinal artery syndrome. The patients with accompanying atherosclerotic disease are at high risk of this terrible complication [24].

Cauda equina syndrome (CES), characterized by polyradicular symptomatology including lumbar and bilateral lower limb pain, loss of sensation in the anal region, motor and sensory loss of lower extremities, dysfunction of bladder and descending colon, and erectile dysfunction, is caused by the compression of the lumbosacral nerve roots located in the dural sac [26]. Risk factors for the development of CES are similar to the other neurological complications of SA, including direct trauma of the needle, hematoma, infection, narrow canal, spinal tumors, disc herniation, neurotoxicity due to high dose LA, hyperbaric 5% lidocaine, and dibucaine.

Table 2. The facilitating risk factors for the development of neurological complications of SA

Procedure/practitioner-related factors	Patient-related factors
Trauma due to the needle	Spinal cord ischemia due to hypotension
Paresthesia during needle placement	Subdural/epidural haematoma (anti-coagulant use)
Pain during injection of local anesthetic	Co existing spinal tumors and AVMs
Multiple attempts to perform a block	Anatomic abnormalities (e.g. medullary canal stenosis and ankylosing spondylitis)
Paravertebral muscle spasm	Previous spinal surgery
LA-induced neurotoxicity	Position of the patient (e.g. stretching of the sciatic nerve)
Inexperience of practitioners	Early mobilization of the patient
Subdural/epidural abscess	

Paresthesia during needle placement, pain during injection of local anesthetic, and multiple attempts to perform a block are also reported as associated risk factors for neurological injury [8]

Chronic Adhesive arachnoiditis is also one of the serious complications of regional anaesthesia, which is more frequently secondary to epidural anaesthesia than SA. It usually results from accidentally dural puncture during epidural anaesthesia. This rare condition is caused by fibrosis and adhesion of the arachnoid membrane due to chronic inflammation, and may occur up to six months after caudal block [27]. Chronic arachnoiditis has a non-specific symptomatology including back pain, radiating pain to the lower extremities, urinary symptoms, and sensory impairment, and therefore can be diagnosed by various imaging methods such as computed tomography, magnetic resonance, and myelography in combination with detailed physical examination.

Finally, intracranial subdural hematoma is an extremely rare and life-threatening complication of regional anaesthesia, and limited number of cases have been reported in the literature [28,29]. The majority of the patients develop symptoms such as consciousness disorder, vomiting, hemiplegia and diplopia within one month after SA. Development of neurological sequel or mortality is not rare, and is often associated with delayed diagnosis.

1.5 Management of Neurological Abnormalities

Firstly, all anaesthesiologists must have enough knowledge about the neurological complications and their clinical presentations, and must also know that all these complications may become evident during or months after the procedure [30]. Since delayed diagnosis is the leading cause of death and neurological sequelae, these patients should be immediately evaluated in coordination with other related departments such as radiology, neurology and neurosurgery, and finally the main cause of the neurological disease should be determined.

The patients with mild symptoms such as a resolving paraesthesia can be managed conservatively, but in cases of serious clinical findings such as acute hemiplegia, paraplegia, loss of sphincter control should prompt urgent action. Magnetic resonance imaging is the best radiological method showing the abnormalities within vertebral canal [31]. In the presence of haematoma, decompression within 12 hours of the onset of symptoms is of great importance for the maximised recovery of the patients [20]. As in spinal hematomas, urgent intervention is usually needed for spinal abscess.

Electrophysiological examination is the next diagnostic tool when a decompressive surgery is not immediately indicated. It is fact that an electrophysiological deficit will become clear approximately two weeks after a nerve injury. However, a baseline electrophysiological test within the two days after an injury may reveal preexisting deficits and clarify results of tests two weeks later [1].

Headache is the most frequently seen neurological complication following SA [32]. It is usually postural, and is caused by dural injury. This kind of headaches disappear within 48 hours after the procedure by sufficient fluid intake, bed rest, and simple pain killers such as paracetamol and non-steroidal anti-inflammatory drugs. On the other hand, long-standing headache and/or accompanying neurological findings may be a sign of subdural hematoma, meningitis or intracranial hemorrhage. Therefore, such patients should be closely followed up, and evaluated by lomber punction or advanced radiological tests including CT and MRI. In case of infected conditions, broad-spectrum antibiotics should be immediately given to those patients. Urgent surgical drainage is indicated for abscess formation.

Neuropathic pain following SA is transient in most patients, and usually occurs due to the direct trauma of the needle or adverse effect of local anesthetics. The patients with mild symptoms can be successfully treated with paracetamol or non-steroidal anti-inflammory drugs. However, a multidisciplinary approach is needed for the patients who have severe symptoms. In this respect, pharmacological therapies, physical therapy modalities, cognitive behavioral and psychological treatment and social support should be part of the approach [33]. In the pharmacological treatment of neuropathic pain, pregabalin, gabapentin and tricyclic antidepressants are recommended as the first line drugs. Serotonin/ noradrenaline reuptake inhibitors and tramadol, opioids and lamotrigine are also recommended in the treatment [34].

Other neurological complications such as anterior spinal artery syndrome, cauda equina syndrome, and chronic adhesive arachnoiditis are relatively rare, and clinical suspicion takes an important place in the diagnosis. Thus, sufficient

knowledge regarding these rare complications is of great importance to achive correct diagnosis and to avoid delays in the treatment.

2. CONCLUSION

Although neurological complications of SA are catastrophic, most of those can be prevented with detailed preoperative evaluation of the patients, better knowledge of spinal anatomy, adequate technical equipment, compliance with routine rules of the procedure, and close postoperative monitoring. Delay in diagnosis is the main factor for neurologic sequelae and death. Therefore, a multidisciplinary approach is needed in patients who develop a neurological complication following SA.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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