



Management of neurologic complication after regional anaesthesia – what to tell the patient?

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Complications of regional anaesthesia has been recognised from very long time. Fortunately serious complication are rare. Safe, effective practice of neuraxial anaesthesia requires a detailed knowledge of potential complications, their incidence and risk factors associated with their occurrence. The incidence of complication were higher for spinal than for epidural anaesthesia. 1

The most frequent complications of regional anaesthesia are **post dural puncture headache (PDPH), backache, transient neurological symptoms, total spinal anaesthesia, spinal or epidural hematoma, meningitis, arachnoiditis and cardiac Arrest** (1, 2).

The mechanism of PDPH is thought to be persistent leakage of cerebrospinal fluid (CSF) through the dural defect at a rate faster than that of CSF production. The transdural leak leads to decreased CSF volume and pressure.

The diagnosis is basically clinical, usually presents 48-72 hrs after the procedure, typically bilateral, fronto – occipital extending up to neck and shoulders. Pain is described as dull or throbbing; usually associated with nuchal stiffness and backache. The hallmark of PDPH is that it is postural in nature. It often subsides during supine position and may be associated with malaise, photophobia, nausea, vomiting and cranial nerve palsies (3).

Subdural hematoma is rare but is most severe complication of PDPH.

Backache is a frequent complaint of neuraxial anaesthesia. Although incidence is high but neuraxial anaesthesia may not be the sole cause. The frequency of backache is approximately similar after spinal or general anaesthesia. Although backache is usually benign, it may be an indication of more serious complications like epidural abscess, spinal hematoma or syndrome of transient neurologic symptoms.

Total spinal anaesthesia can happen when there is unintentional intrathecal administration of local anaesthetics during epidural or caudal anaesthesia. The onset is usually rapid. Patient exhibits signs of cardiovascular collapse in the form of severe hypotension, bradycardia and respiratory insufficiency.

Epidural or spinal haematoma is a rare, but potentially disastrous complication of central neuraxial blocks. The incidence of such hematomas has been estimated to be about 1:150,000 for epidural blocks and 1:220,000 for spinal anaesthetics. Spinal hematoma is particularly catastrophic as it may go unnoticed until there is permanent neurologic compromise. Patient usually present with sudden new onset sharp back and leg pain with numbness, weakness, bladder and bowel dysfunction.

When spinal hematoma is suspected, neurologic imaging (MRI and CT scan) and neurologic consultation should be immediately obtained. Good neurological recovery is seen in patients who have undergone surgical decompression within 8–12 hours.

Epidural abscess is a serious complication after neuraxial block. The incidence varies from 0.015% to 0.7% according to different studies. Although epidural abscess is uncommon, early diagnosis and treatment is paramount. Symptoms of epidural abscess usually begin several days after neural block, sometimes after months, include back pain, fever, malaise, signs of cord compression including sensory changes, flaccid paralysis followed by spastic paralysis, elevated blood leukocytes count, elevated cerebrospinal fluid protein and leukocytes.

The management of epidural abscess involve, drainage of the abscess and eradication of the microorganism as the basic principles of therapy. Surgical therapy is the treatment of choice in the overwhelming majority of cases. Rapid surgical intervention is not only needed to minimize neurological damage, but also for controlling sepsis. Duration of antimicrobial treatment is usually 4–6 weeks for epidural abscess.

Dural puncture may be a risk for infection of subarachnoid space. Exact mechanism by which bacteria reaches to the central nervous system may not be known but the infectious source may be exogenous (e.g., contaminated equipment or medication) or endogenous (a bacterial source in the patient seeding to the needle or catheter site). Microorganisms can also be transmitted via a break in aseptic technique, and indwelling catheters may be colonized from a superficial site (skin) and subsequently serve as a wick for spread of infection from the skin to the epidural or intrathecal space. The symptoms appear hours to days after anaesthesia, sometimes onset time may be up to one month. Initial clinical presentation are fever and headache, with backache with emesis, classical sign of meningism and lithargy. CSF is usually turbid with raised leukocytes, proteins and low glucose concentration. In great majority of cases the causative organism is alpha-haemolytic streptococcus. Lumbar puncture aids diagnosis. Give appropriate antibiotics early,

which will usually be before the causative or its sensitivity is established. Use of steroid is debatable but recommended for community acquired meningitis.

Arachnoiditis, another rare complication of neuraxial-anaesthesia may appear as transient nerve root irritation, caudaequina, and conusmedullaris syndromes.⁴ It may show its presence later as radiculitis, clumped nerve roots, fibrosis, scarring dural sac deformities, pachymeningitis, pseudomeningocele, and syringomyelia, etc., all associated with arachnoiditis. Regarding regional anaesthesia in the neuraxis, arachnoiditis has resulted from epidural abscesses, traumatic punctures (blood), local anaesthetics, detergents, antiseptics or other substances unintentionally injected into the spinal canal. Patients usually presents with pain in the lower back, dysesthesia and numbness not following the usual dermatome distribution.

The incidence of cardiac arrest during regional anaesthesia varies in different studies and it ranges from 1.5–6.4/10000 cases.⁵ Theories regarding the mechanism by which neuraxial block contributes to cardiac arrest involve a circulatory aetiology.

Initially sedation was speculated to have contributed to many of the cardiac arrests during spinal anaesthesia. Another likely cause could be decrease in preload associated with neuraxial block resulting in a shift in cardiac autonomic balance toward the parasympathetic system leading to bradycardia.

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