Focal Myelomalacia and Syrinx Formation after Spinal Anaesthesia

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Abstract

Spinal cord injury is an uncommon complication of post epidural lumbar anaesthesia with acute and delayed progressive neurological complications described. Few cases of post spinal anaesthesia syrinx formation have been reported in literature. We report an interesting case of focal myelomalacia with syrinx formation occurring post lumbar anaesthesia in a thirty five year old female undergoing open cholecystectomy.

Introduction

Spinal cord injury due to incorrect identification of the lumbar space is an uncommon complication of lumbar puncture. Lumbar epidural anaesthesia is easily performed and usually considered safe therefore it is extensively used in orthopaedic, urology, gynaecology and other surgeries. However this procedure rarely cause acute and delayed progressive and sometimes devastating neurological complications. Spinal anaesthesia is a procedure where lidocaine or its analogue are introduced into the subarachnoid space. It has several known but rare complications such as epidural or spinal subarachnoid hemorrhage and arachnoiditis. Few cases of post spinal anaesthesia syrinx formation have been reported in literature. We report a patient who suffered focal injury to the spinal cord secondary to injection of local anaesthetic agent during attempted spinal anaesthesia using the lumbar approach.

Case Report

A 35 year old healthy woman was scheduled for open cholecystectomy for multiple gallstones under spinal anaesthesia with lignocaine. She had no significant past medical history and her preoperative general and neurological examination was normal. In the operation theatre under normal vital status patient underwent lumbar puncture in left lateral recumbent position with pencil tip needle in a single prick at the level of L1-L2. She experienced severe shooting pain and tingling in both lower limbs radiating downwards, immediately after the introduction of spinal LP needle. Three hours post surgery with the wearing off effect of the anaesthetic drug, patient noticed complete loss of sensation in her left lower limb and lower anterior abdominal wall below the level of umbilicus with complete inability to move the limb. On the 3rd postoperative day patient developed urinary retention when catheter removal was attempted following which patient had to be re catherized. Her bladder symptoms improved over the next 3 months and sensorimotor complaints improved partially over a period of 2 months, with patient being able to walk independently with limping and complaints of frequency, straining while micturition and sensation of incomplete voiding persisting till date. Examination revealed weakness of left foot dorsiflexors (3/5), plantarflexors (4/5), knee (4/5) and hip (4/5). Ninety percent loss of pain and temperature sensation and 50% loss of touch, vibration and joint position sense were found below D11 on left side. Deep tendon reflexes were absent in left lower limb and normal in other limbs. Left planter was extensor and right was not elicitable. Rest of the neurological examination was normal.

Routine biochemical investigations were normal. MRI of thoracolumbar spine showed small cystic cavity noted in left aspect of distal conus medullaris at L5 vertebral level showing isointense signal intensity to the CSF on all sequences. Nerve conduction studies in both lower limbs was normal. EMG was suggestive of neurogenic pattern of involvement of left lower limb from L2-S1.

Discussion

The exact frequency of neurological sequelae after lumbar epidural anaesthesia is difficult to estimate from published data. However available data suggests an estimated frequency of neurological complications related to lumbar epidural anaesthesia of 1 in 10,000 procedures, with serious neurological deficits even rarer, approximately 0.4-0.6/10,000 procedures. Lumbar puncture is a blind procedure. Factors associated with increased risk of neural injury from spinal anaesthesia have been reviewed by several authors. The needle tip often traumatizes the posteriorly placed venous plexus in the epidural space and if continued forward after entering the subarachnoid space, may also damage anteriorly placed blood vessels (a so called traumatic tap). There are numerous possible mechanisms of trauma to the neural structures such as nerve roots and the spinal cord; they include spinal cord ischemia, the toxicity of anesthetic preparation, or direct needle trauma. A traumatic needle induced lesion at conus level can cause a severe disturbance of the intramedullary microcirculation that could lead to the formation of a rod shaped cavity in the central region of conus -epiconus. Probably in our case a needle injury to the conus first caused edema (no imaging in acute phase available) and then intramedullary ischemic damage that spread upwards until it reached the epiconus and this could explain the fact that, after the disappearance of the edema a distal motor deficit appeared that corresponded to a lesion of the epiconus, since a lesion confined to the conus would only induce sensory symptoms of the ‘saddle anaesthesia” type. If the LP is done at higher intervertebral spaces which overlie the spinal cord, missing the sub-arachnoid space would mean hitting the cord. Furthermore, an LP done with the intention of giving spinal anaesthesia will have an additional damaging effect on the cord by way of

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drug volume related pressure effect as well as chemical injury. Complications such as arachnoiditis, infectious spondylodiscitis and development of arachnoid cyst have been observed to have developed gradually after the acute insult. The arachnoiditis in turn may cause polyradiculopathy and syrinx within the cord. Syringomyelia associated with spinal arachnoiditis is explained by two probable consequent mechanisms: the meningeal inflammatory process leads to severe arachnoid scar formation and vessel constriction, spinal cord ischemia, necrosis and cyst development; once the cavity is formed, it can break into the surrounding subarachnoid space, and CSF can enter the cyst, thus forming a syrinx which may extend upwards. Even if the cyst does not break out into the subarachnoid space, the syrinx may extend upwards because CSF may be forced into it through the perivascular spaces. Most previous reports of lumbar puncture related spinal cord injury have noted irreversible cord damage evidenced by persistent neurological deficits.

Gentle slow insertion of the needle must be an essential component of every lumbar puncture with the needle being inserted no further than is necessary to ensure freeflow of CSF. Though spinal cord damage can occur after careful needle insertion, it is more likely with a heavy handed approach.

We also recommend that every patient with a history of low backache must have a careful neurological examination to rule out subtle motor or sensory deficits. Although preanaesthesia MRI of lumbar spine is not advisable in every patient, but a history suggesting lumbar canal stenosis should be taken into account, because it may be contraindication to central neuraxial blockade.

References