ASEPTIC MENINGITIS FOLLOWING SPINAL ANAESTHESIA- TWO CASE REPORTS
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ABSTRACT: Meningitis after spinal and epidural anaesthesia is rare but serious complication which requires early diagnosis and treatment. We report two cases of probable chemical meningitis, which developed within few hours after subarachnoid block. CSF pattern was suggestive of chemical meningitis. Both the patients recovered next day without any neurological sequelae. Meningitis should always be considered as a differential diagnosis in patients having post spinal headache, convulsions and changes in mental status. Despite thorough knowledge and practice of aseptic technique, aseptic meningitis can occur.

INTRODUCTION: Meningitis after spinal and epidural anaesthesia is rare but serious complication which requires early diagnosis and treatment. Despite thorough knowledge and practice of aseptic technique, aseptic meningitis can occur. We report two cases of aseptic meningitis following subarachnoid block probably due to the drug used for intrathecal injection.

CASE1: A 55years old male was scheduled for hemorrhoidectomy. He was chronic smoker for past 35 years (pack years 35). No relevant drug history. His past medical history was unremarkable. Preoperative physical examination revealed Height of 170cms, Weight of 72kgs, afebrile, Pulse Rate (PR) of 88/min, Blood Pressure(BP) of 120/70 mmHg. Chest x-ray revealed COPD changes, other investigations were within normal limits.

Patient was premedicated with Inj. ranitidine 50mg, ondansetron 4mg, midazolam 1mg intravenously (IV), fifteen minutes prior to regional anaesthesia. Patient was preloaded with ringer lactate(RL) 750ml. supplemental oxygen was given through nasal prongs at 3L/min. PR, BP, O2 saturation and electrocardiogram were continuously monitored. Patient was allowed to lie down in left lateral position. The area for subarachnoid block was prepared using povidone iodine, spirit and draped in sterile fashion. Infiltration of the skin was done with lignocaine 1% at L3-L4 interspinous space. Lumbar puncture was done using a 25G Quinckes needle, Bupivacaine 0.5% heavy 12.5mg was injected intrathecally. Patient was repositioned supine. A T10 sensory level was obtained and patient was put in lithotomy for surgery. Patient received a total of 1000ml of crystalloid throughout the surgery. Intraoperative period was uneventful. Patient was awake, alert and haemodynamically stable when transferred to recovery room. His vitals were monitored continuously.
CASE REPORT

After 6 hours of surgery, patient (Pt) complained of non-positional headache which was associated with nausea, vomiting, photophobia. Pt was irritable, confused and had mild neck rigidity. No other focal neurological deficits were noted. Provisional diagnosis of meningitis was made, and lumbar puncture was done for CSF analysis. CSF was cloudy. Patient was treated with empirical antibiotics (Inj. Ceftriaxone 2g IV 12th hourly) for meningitis. Laboratory analysis of CSF shows glucose 48mg/dl, protein 35mg/dl, WBCs 225/cu mm, with neutrophils 15%, lymphocytes 85%. AFB, gram stains and culture were negative. Cultures of blood, urine and local anaesthetic solution of same batch were negative for bacterial pathogens. Renal function test (RFT), Liver function test(LFT) and serum electrolytes were within normal limits. Next day afternoon, after 24 hours of surgery, patient was conscious, oriented with no neurological sequelae.

CASE2: On the same day in OBG OT of our institution, a 28 year old primigravida with 38 wks of gestational age, was posted for emergency caesarean section for fetal distress. There was no significant antenatal history. No history of leaking membranes. Her past medical history was unremarkable. Preoperative examination revealed Ht 145cms, Wt 48kgs, afebrile, PR 98/min, BP 130/70mmHg. Mild pedal edema was present bilaterally. CVS and other systemic examination were normal. Blood and urine investigations were within normal limits. Patient was premedicated with Inj. ranitidine 50mg and Inj. Metoclopramide 10mg intravenously. O₂ supplementation was given through nasal prongs 3L/min. Patient was preloaded with ringers lactate 500ml. PR, BP, O₂ saturation and ECG were continuously monitored. Patient was allowed to lie down in left lateral position. The area for subarachnoid block was prepared using povidone iodine, spirit and draped in sterile fashion. Infiltration of the skin was done with lignocaine 1% at L3-L4 interspinous space. Lumbar puncture was done using a 25G Quincke's needle, Bupivacaine 0.5% heavy 10mg injected intrathecally. Patient was repositioned supine with left lateral tilt of 15degrees. A T6 sensory level of anaesthesia was obtained. 20U oxytocin infusion was given after extraction of baby. Total of 1500ml of crystalloids was administered intraoperatively. Intraoperative period was uneventful. Patient was awake, alert and haemodynamically stable when transferred to recovery room. Her vitals were monitored continuously.

After 8 hours of surgery, patient developed headache, nausea and photophobia. Patient was drowsy, irritable and confused on awakening, had mild neck rigidity. No other focal neurological deficits were present. Provisional diagnosis of meningitis was made, and lumbar puncture was done for CSF analysis. CSF was cloudy. Patient was treated with empirical antibiotics (Inj. Ceftriaxone 2g IV 12thhrly) for meningitis. Laboratory analysis of CSF showed glucose 46mg/dl, proteins 120mg/dl, WBCs 1800/cu mm wit neutrophils 65%, lymphocytes 35%. AFB, gram stain and culture was negative. Blood sugar was 130mg/dl. Urine analysis was normal. Cultures of blood, urine and local anaesthetic solution of same batch were negative for bacterial pathogens. RFT, LFT and serum electrolytes were within normal limits. After 24 hours of surgery, patient was conscious, oriented with no neurological sequelae.

DISCUSSION: Meningitis following spinal anaesthesia is rare. The epidemiological evidence shows that the frequency of post lumbar puncture meningitis is no greater than in ordinary population¹. Furthermore, there were no cases of CSF infection in the classic prospective study on patients undergoing 10098 spinal anaesthetics, performed by Drip's and Vandam².
Earlier chemical/aseptic meningitis was not an uncommon complication. By 1947, more than 100 cases had been described in literature and an incidence of 0.26% was quoted in a summary report of approximately 46000 spinal anaesthetics. However the incidence declined dramatically during the 1950s after a series of studies that had implicated chemical contamination of the CSF as the cause. Since then cases have been reported sporadically. A retrospective review of 4767 consecutive spinal anaesthetics in 1997 by Terese T, Horlocker et.al, did not report any case of meningitis. A study of neurologic complications of 603 consecutive continuous spinal anaesthetics (CSA) in 1997 by Horlocker showed one case of aseptic meningitis following CSA.

Aseptic meningitis is a benign neurological complication which usually presents within 24 hours after spinal anaesthesia and is characterized by fever, nuchal rigidity and photophobia. Microscopic examination of CSF is characterized by polymorphonuclear leucocytosis, bacterial CSF cultures are negative. Aseptic meningitis requires only symptomatic treatment and usually resolves. It may be caused by either chemical irritation or viral infection. In Chris Newton study, CSF analysis typically gives a profile of lymphocytic pleocytosis (usually less than 1000cells/mm³), slightly elevated protein level and normal glucose level, which is similar to the composition of CSF in our first case. Harding explains CSF presentation of aseptic meningitis with more polymorphs than lymphocytes; these findings are similar to the composition of CSF in our second case report.

Aseptic meningitis has been associated with any foreign substance within the intrathecal space. Introduction of subarachnoid contaminants such as the scrub solutions, or surgical glove powder might occur during the lumbar puncture or primary contamination of equipment and anaesthetic drugs. Other cases are believed to be caused by pyrogens contained in the anesthetic solution, blood or other body proteins introduced at the time of lumbar puncture. Less commonly aseptic meningitis is caused by systemic drug administration like NSAIDs, H₂ blockers widely used in anaesthesia), trimethoprim, sulfadiazine etc. Both the cases received Inj. ranitidine as prophylaxis against acid secretion, so also did all cases who were subjected to anaesthesia, but had no untoward effect.

Our patients suffered most probably from chemical meningitis. The conclusion is based on following points: rapid onset of illness, CSF biochemistry, lack of organisms in CSF, lack of positive blood or CSF culture and rapid recovery.

Patients were started on empirical antibiotics because failure to isolate a bacterial pathogen doesn’t disprove a bacterial etiology as Gram stain and cultures may be negative in up to 10% cases of bacterial meningitis. Also pts had received pre-operative ciprofloxacin 500mg IV, single dose. Our cases bear resemblance to the case reported by Harding, etal and Chris Newton. In our view the most likely source was local anaesthetic solution given intrathecally, because both the patients received same batch of bupivacaine, which were used in two different operation theatres of our institution. Patients who had received other batch of bupivacaine on the same day, in the same OT, and also had skin prepared in same fashion with povidone-iodine, spirit solution did not have any complication. After withdrawing that particular batch of bupivacaine, no further case was reported. Since we rely on our manufacturers for proper preparation of spinal ampoules, using the drug manufactured by reputed pharmaceuticals assumes importance.

CONCLUSION: Anesthesiologist should assume grave responsibility when he injects a foreign material into subarachnoid space, also he should be familiar with the possible sources of
contamination and with the means to prevent them. Meningitis after spinal and epidural anaesthesia is rare but serious complication. Early diagnosis and treatment will prevent serious neurological complications.

REFERENCES: