Postoperative tension pneumocephalus following cerebral aneurysm surgery in supine position without prior lumbar drainage

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Abstract

The occurrence of tension pneumocephalus in neurosurgeries done in the supine position is scarcely reported. We present a case of 57-year-old man who developed tension pneumocephalus postoperatively, following cerebral aneurysm surgery, in supine position, where lumbar drainage before clipping surgery was not done. The patient's neurological status deteriorated rapidly, characterized by convulsion and unresponsiveness to external stimuli, 1 h following the uneventful surgery. Immediate computed tomography scan revealed bi-frontal tension pneumocephalus. Long duration of surgery and cerebrospinal fluid loss were assumed to be the causative factors. The patient was treated immediately with frontal drill hole evacuation for intracranial air, which saved the patient from a life threatening complication.

Key words: Cerebral aneurysm surgery, cerebrospinal fluid loss, tension pneumocephalus

INTRODUCTION

Pneumocephalus, characterized by air in the cranial cavity, is a common occurrence following posterior fossa craniotomies performed in a sitting position,[1,2] but development of tension pneumocephalus is uncommon. This severe complication puts the patient's life at risk, by producing a mass effect and abnormal neurological signs, which requires immediate treatment. Few cases have been reported as a complication of head trauma, trans-spheniodal, spinal, cranial (posterior fossa, supratentorial) surgeries, or even after accidental dural puncture.[1,3] However, tension pneumocephalus after neurosurgical procedure in the supine position and without prior lumbar puncture has been very scarcely reported.[4,5] We report a case, where an adult patient developed tension pneumocephalus in an immediate postoperative period, after surgical clipping of anterior communicating artery aneurysm, in the supine position, without prior lumbar drainage.

CASE REPORT

A 57-year-old male patient, weighing 60 kg and 165 cm in height, was referred to our institute with a past history of sudden onset severe headache, nuchal rigidity, and diplopia 15 days back. At the time of presentation, central nervous system examination revealed a conscious, oriented patient with normal higher function, without diplopia or neck rigidity. There was no loss of power in all four limbs, 4/5 in both the upper limbs, and 4/5 in both the lower limbs as per Medical Research Council (MRC) rating. His sensory system, other cranial nerves,
brain-stem function appeared normal. His other systems were within the normal limit. His vitals were also within the normal limit (pulse rate: 70/min, regular; blood pressure: 124/76 mm Hg, respiratory rate: 14/min). Laboratory investigations (routine hematological; liver and kidney function tests; serum electrolytes; coagulation profile), chest X-ray, electrocardiogram (ECG) were normal. Magnetic resonance imaging of brain showed gross subarachnoid hemorrhage (SAH) and intraventricular hemorrhage (IVH) but no focal lesion of parenchyma or any midline shift [Figure 1]. Cerebral angiography revealed an anterior communicating artery aneurysm, which was thought to be the cause of SAH and IVH. According to Hunt and Hess classification, he was considered grade II. The patient was posted for clipping of anterior communicating artery aneurysm through the pterion (right) approach. An informed consent was taken for surgery, anesthesia, and postoperative care in ICU.

The patient was premedicated with tablet alprazolam 0.5 mg orally at the night before operation. On the day of surgery, after establishing intravenous (IV) line with 18G cannula, the patient received injection (Inj) midazolam 2 mg, Inj ondansetron 6 mg, and Inj glycopyrrolate 0.2 mg. General anesthesia was induced by Inj fentanyl (2 µg/kg) and thiopentone (5 mg/kg) following preoxygenation and intubation was done with size 8.5 flexometallic endotracheal tube (Sheridan/SPIRAL-FLEX®, Hudson RCI) using Inj rocuronium (0.6 mg/kg). Anesthesia was maintained with O₂:N₂O (40:60), propofol (infusion at 100-150 µg/kg/min) and intermittent bolus of Inj vecuronium and Inj fentanyl. Mannitol (1 g/kg) was infused to provide optimum brain relaxation. For invasive monitoring, central venous catheter was placed via the right subclavian vein and intra-arterial catheter was placed at left radial artery. Intraoperative monitoring included five lead ECG, invasive blood pressure, SpO₂, end tidal carbon dioxide, central venous pressure, urine output, core temperature, bispectral index, and neuromuscular monitoring. Keyhole-type craniotomy was done and surgery proceeded uneventfully in the supine position and haemodynamics remained stable throughout the operative period, which lasted for 6 h. N₂O was discontinued before dural closure. At the end of the surgery, neuromuscular blockade was reversed when the TOF ratio was 40% with 0.5 mg Inj glycopyrrolate and 2.5 mg neostigmine. The trachea was extubated once the TOF ratio was 90%, respiratory effort was adequate, and patient was obeying commands. He was shifted to the postoperative recovery room.

One hour after surgery, the patient developed sudden generalized convulsions, a rapid deterioration in the consciousness level, with dilatation of the left pupil. The airway was secured with Gudel’s airway (number 5) and Inj midazolam 4 mg was given to control the convulsions. He maintained an oxygen saturation of 97-99% with 3L oxygen through the facemask. His arterial blood pressure and pulse rate were 120/70 mmHg and 92/min, respectively. An immediate computed tomography (CT) scan of brain done postoperatively, revealed accumulation of air in the bi-frontal subdural space compressing mainly the right sylvian fissure and right lateral ventricle without any apparent midline shift (mount Fuji sign [Figure 2]), indicating a diagnosis of tension pneumocephalus. The patient was taken to the operation theatre immediately for aspiration of pneumocephalus through a frontal drill hole and air came out under pressure. The patient’s sensorium showed marked improvement after the surgery and he started responding to verbal commands. The rest of the postoperative course was uneventful and the patient was discharged 15 days later without any neurological deficit.

DISCUSSION

Tension pneumocephalus may present with deterioration of consciousness with or without lateralizing signs,[6] headache, severe restlessness, generalized convulsions,[7] or focal neurological deficit leading to coma or even
that N₂O played no significant role in our patient, as which was done in the supine position and N₂O was the patient deteriorated 1 h after completion of surgery, Several hypotheses have been implicated in the cardiac arrest. It is always a life threatening condition. Several hypotheses have been implicated in the pathogenesis of tension pneumocephalus: They include nitrous oxide (N₂O) anesthesia, a functional ventriculo-peritoneal shunt, cerebrospinal fluid (CSF) leakage due to lumbar drainage, or dural rent after epidural anesthesia, dural defect creating one-way valve, factors that decrease the brain volume such as surgical decompression, intracranial dehydration, hyperventilation, duration of surgery.

Nitrous oxide has been held responsible in the pathogenesis of tension pneumocephalus by several authors, particularly during sitting posture; although, its role has been questioned by others. We believe that N₂O played no significant role in our patient, as the patient deteriorated 1 h after completion of surgery, which was done in the supine position and N₂O was stopped well before dural closure.

In gross hydrocephalus, a proportionately greater volume of CSF can be drained during surgery and this may suck air inside and can produce pneumocephalus. A functioning ventriculo-peritoneal shunt helps in continuous drainage of CSF in the peritoneal cavity creating space for intracranial air. Both factors were absent in our patient.

Continuous CSF leakage from lumbar puncture or dural rent during epidural anesthesia causes significant CSF loss creating negative pressure space, where air from positive pressure atmosphere rushes into the intracranial space via the dural defect causing tension pneumocephalus. Saito et al. reported a case where tension pneumocephalus developed following lumbar drainage for cerebral aneurysm surgery. Lumbar drainage to facilitate brain retraction is not done routinely for aneurysm surgery in our institution.

Intracranial dehydration or hyperventilation result in a slack brain with the subsequent enlargement of the subdural space and is apt to increase the volume of entrapped intracranial air. These were not used in our patient, in fact, hyper-volemia and high-normal range of blood pressure was maintained to prevent cerebral ischemia (protection against potential vasospasm).

Prolong duration of surgery may lead to continuous CSF loss intraoperatively, which may be a causative factor. The duration of surgery in our case was 6 h, so we assumed it might be a causative factor.

An adult patient developing tension pneumocephalus as a postoperative complication after clipping surgery for ruptured anterior communicating artery aneurysm in supine position without prior lumbar drainage is very rare. The present case helps us to conclude that efforts must be directed to minimize CSF loss during intracranial surgery and dura should be sutured water-tight during craniotomy closure, to prevent the development of tension pneumocephalus and once it has developed, immediate diagnosis and treatment are required to save the life of these patients.

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