Pontine infarct during deep brain stimulation surgery as the cause of postoperative hemiparesis

Zulfiqar Ali, Hemanshu Prabhakar¹, Navid Wani²

Sir,

Deep brain stimulation (DBS) of subthalamic nucleus and globus pallidus is increasingly being used for treatment of Parkinson’s disease (PD) and other movement disorders. The procedure involves the stereotaxic placement of an implantable electrode with four active contacts within the target. This is followed by tunnelling of the external cables of the electrodes, which are connected to an externally implanted pulse generator. A wide array of complications can occur during this procedure. Complications reported in literature are related to the device, the procedure, the electrical stimulation and the spread of current.[1,2]

Of particular concern to the anaesthesiologists are the perioperative complications; some of them, in our experience, were unusual. We report a case where a patient undergoing DBS for PD developed altered sensorium intraoperatively as a result of a lacunar infarct in the pons.

A 60-year-old male patient, weighing 62 kg was scheduled for DBS procedure. His past medical history was unremarkable. The first part of the procedure, that is, electrode placement, was done under monitored anaesthesia care. In the operation theatre, routine monitoring included 5-lead electrocardiogram, non-invasive blood pressure, pulse oximetry and respiratory rate. Supplemental oxygen was administered via nasal prongs at a flow rate of 2 litres per minute. The patient was positioned supine on the operating table in a stereotactic frame with 10° head up tilt. Following placement of electrodes on the left side, as the electrodes were being placed on the right side, the patient was suddenly uncooperative. He stopped following commands from the neurologist and became restless. The procedure was abandoned and patient was shifted for computed tomography (CT) of head. The scan revealed a large pneumocephalus but no operative site haematoma or any other abnormality [Figure 1a].

On the second postoperative day, the patient developed hemiparesis on the left side. Meanwhile, his sensorium improved and he started following commands. A repeat CT scan was performed which now revealed right midbrain small lacunar infarct and reduced pneumocephalus [Figure 1b]. Later on, the patient recovered in the intensive care unit.

The various adverse effects of DBS related to the surgical procedure, the implanted device and electrical stimulation of areas adjacent to the target have been described in literature.[3] Some of these include intracerebral haematoma, ischaemic stroke, seizures, lead migration, erosion and breakage, dyskinesias, dysarthria, mood changes, Horner’s syndrome, etc. Of particular concern to the anaesthesiologists are the perioperative complications. Altered sensorium as a result of pneumocephalus is a known entity.[4] The clinical pattern depends on the territory involved in the pons. Pontine infarcts are known to produce a variegated clinical picture ranging from sensory to motor symptoms.

Figure 1: (a) A non-contrast computed tomographic scan of head showing large bifrontal haematoma and artifacts due to electrodes, (b) A right midbrain small lacunar infarct (white arrow)

Department of Neuroanaesthesiology, Sheri Kashmir Institute of Medical Sciences, Srinagar, ²Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, ¹Department of Neuroanaesthesiology, Neurosciences Centre, All India Institute of Medical Sciences, New Delhi, India

Address for correspondence:
Dr. Zulfiqar Ali, Department of Neuroanaesthesiology, Sheri Kashmir Institute of Medical Sciences, Srinagar - 190 010, Jammu and Kashmir, India.
E-mail: zulfiqaraliii@yahoo.com
Features like dysarthria, ataxia, vertigo, dizziness, tetraparesis, pseudobulbar palsy, etc., may be seen. Our case illustrates altered mental status coinciding with the developing pneumocephalus and infarction in the pons.

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Venous air embolism during scalp dissection in a case of cerebellar haemangioblastoma

Sir,

Venous air embolism (VAE) is the entrainment of air from the operative field or other communication with the environment into the venous vasculature producing systemic effects. We report a case of VAE during scalp dissection in a patient undergoing craniotomy in sitting position for cerebellar haemangioblastoma. A 17-year-old, 40 kg, American Society of Anaesthesiologists physical status I, male patient presented with gait instability for 1 year and headache in occipital region for 9 months. The patient was diagnosed to have cerebellar haemangioblastoma [Figure 1] and was posted for excision in sitting position. There was no history of any respiratory or cardiovascular abnormality. His routine blood investigations, electrocardiogram, and chest X-ray were within normal limits. The patient was premedicated with intramuscular glycopyrollate 0.2 mg 1 hour before induction of anaesthesia. A general endotracheal anaesthesia with fentanyl, propofol, sevoflurane, vecuronium and nitrous oxide and oxygen (2:1 ratio) was used. Central venous cannulation (right subclavian vein) and intraarterial cannulation was performed following induction of anaesthesia. The patient was preloaded with 1 litre of crystalloid solution to ensure a central venous pressure of 8 mmHg, before the patient was made seated. The vital parameters of the patient remained unremarkable after sitting position. But soon after the surgeons started dissection of the scalp and pericranium, a sudden fall in end tidal carbon dioxide (EtCO₂) from 34 mmHg-20 mmHg and blood pressure from