INTRODUCTION

Idiopathic intracranial hypertension (IIH) was first described in 1893. Patients with IIH have a classical picture of headache, papilledema, and a raised cerebrospinal fluid (CSF) pressure of more than 25 mmHg. It is more common in women than in men (3:1) with the highest incidence seen in obese women of reproductive age group (20-45 years). The most common presenting symptoms are manifestations of generalized intracranial hypertension, normally headache and visual obscuration. Diplopia, pulsatile tinnitus, nausea and vomiting may be present in about 50% of patients, with neck/back/shoulder pain or radicular pain less frequent.[1] The major risk of IIH is visual loss, which may be permanent despite medical therapy. It seems that the symptoms worsen during pregnancy in 50% of the patients and usually resolve postpartum.[2]

CASE REPORT

A 45-year-old female, weighing 70 kg presented with complaints of headache, menorrhagia and pain in the abdomen was admitted to our hospital. She was posted for transcervical resection of the endometrium and patient had a past history of headache and blurring of vision 2 years ago and was diagnosed to have IIH and was on tablet acetazolamide 250 mg every 6 h. She had a history of lumbar CSF drainage twice. Patient had a history of hypertension and was on tablet atenolol once daily. On pre-operative examination, pulse was 70/min and blood pressure was under control. Other general and systemic examination was normal. All other investigations were within normal limits. Computer tomography brain was normal and magnetic resonance imaging revealed partially empty sella and no pituitary mass lesion, cerebral hemisphere and ventricles were normal. Fundus examination revealed chronic papilledema, optic nerve pallor and perimetry showed increased size of blind spot. Neurologist advised preoperative CSF pressure measurement and drainage if pressure is high. CSF manometry was performed in the lateral decubitus position and CSF pressure was more than 250 mm H2O with normal cytological and biochemical profile. 20 mL of CSF was drained with a 22 gauge Quincke’s needle. After
drainage the CSF pressure was 150 mm H₂O. Patient was posted for surgery after 2 days.

On the day of surgery, injection glycopyrrolate (0.2 mg), injection midazolam (2 mg) and injection fentanyl (200 μg) were given intravenously as pre-medication. Anaesthesia was induced with propofol 175 mg and tracheal intubation was facilitated with vecuronium 5 mg injection plain lignocaine 2% 5 ml was given to attenuate pressor response. Anaesthesia was maintained with oxygen, nitrous oxide, propofol infusion, and vecuronium. Intra-operatively injection mannitol 1 g/kg was given. Monitoring included pulse oximetry, electrocardiogram (ECG), noninvasive blood pressure noninvasive blood pressure (NIBP), ETCO₂, urine out-put and temperature. At the end of surgery neuro-muscular block was reversed with injection neostigmine 3.5 mg and glycopyrrolate 0.4 mg. She was shifted to post-operative recovery room and monitored for 48 h with pulse oximetry, ECG, NIBP, urine out-put, and temperature. She was completely asymptomatic at discharge.

DISCUSSION

IIH was first described by Quinke in 1893 and was called “serous meningitis.” IIH is caused by dural sinus thrombosis, a reduction in corticosteroid therapy, hormonal imbalance, vitamin A toxicity, anabolic corticosteroids, long-term tetracycline, hormonal contraceptives, lithium, and pregnancy. Theories of IIH pathophysiology include increased venous sinus pressure, decreased spinal fluid absorption, increased spinal fluid secretion, increased blood volume and brain oedema. The various treatment modalities used in patients include corticosteroids, acetazolamide, diuretics, repeated lumbar puncture and surgery. The goals of treatment of IIH involve a reduction of intracranial pressure to control symptoms and prevent pressure on the optic nerve and optic meninges, preserving vision. Serial lumbar punctures have a success rate of 30-40% when used alone. Generally, up to 30 ml of fluid is withdrawn to lower the intracranial pressure to normal. Our patient had a history of IIH since 2 years, was on tablet acetazolamide and showed response to medical therapy. Lumbar puncture was carried out twice to improve symptoms. In our patient, 20 cc of CSF was removed to keep the CSF pressure within normal limits.

From the literature, it has been found that IIH is more common in females in the age group 20-45 years. Women with more than 10% over their ideal body weight are 13 times more likely to develop IIH. Our patient was having a body mass index of >30 kg/m². The mechanism proposed is that central obesity raises intra-abdominal pressure, which increases intra-pleural pressure and cardiac filling pressure, which in turn impedes venous return from the brain and leads to increased intracranial venous pressure, and increased intracranial pressure.

Neuraxial anaesthesia, spinal or epidural has been used successfully for caesarean section in patients with IIH. Since lumbar puncture for CSF drainage is a therapeutic modality for IIH, there is no indication to withhold spinal anaesthesia in these patients. Bedson and Plaat reported the combined spinal-epidural technique for delivery by caesarean section. Although, dural puncture is contraindicated in patients with increased intracranial pressure resulting from space occupying lesions due to risk of uncal herniation. However, it has been postulated that the uniform swelling and stiffness of the brain in IIH prevents herniation. Aly and Lawther reported a case of uncontrolled IIH successfully managed using an epidural catheter for analgesia in labour and delivery as well as temporary control of intracranial pressure. Abouleish and Ali had given spinal anaesthesia for caesarean section in patient with IIH.

In our patient, both surgeries were carried out simultaneously, so we had no option for regional anaesthesia. We have selected general anaesthesia as nephrectomy was carried out in the lateral decubitus position. If a patient with IIH requires general anaesthesia, the planned approach should minimize the risk of a rise in intracranial pressure associated with intubation, inadequate depth of anaesthesia and extubation. So, we have to take measures to avoid an increase in intracranial pressure (ICP) during the peri-operative period.

Propofol offers a number of pharmacological advantages for total intravenous anaesthesia in patients of raised ICP. It decreases cerebral blood flow and cerebral oxygen consumption and increases cerebrovascular resistance. It could offer cerebral protection. The synthetic short acting opioids like fentanyl lack any significant effect on ICP.

Induction and intubation may aggravate intracranial hypertension. Liberal doses of propofol combined with narcotics to achieve adequate depth of anaesthesia,
mild to moderate hyperventilation, intravenous lignocaine bolus are the measures that prevent dangerous increase in ICP. We induced the patient with (200 µg) fentanyl and propofol and avoided succinylcholine for intubation as muscle fasciculation caused by succinylcholine, increase the intra cerebral blood volume and increase the ICP. Intubation in lighter planes of anaesthesia should be avoided. Atracurium causes histamine release, slightly increases the pulse rate and central system excitation, so we have used vecuronium in our patient. Vecuronium does not alter ICP or CSF dynamics and lack of cerebral effects have made vecuronium a popular choice in patients with raised ICP. Definitive measures used for decreasing the ICP include, mild head elevation, maintain EtCO₂ between 25 mmHg and 30 mmHg, I.V. mannitol, continuous infusion of thiopentone or propofol, avoid hypoxia, hypercarbia, hyperthermia and hypotension.

CONCLUSION

In conclusion, although IIH is rare, there are special considerations for anaesthetic management in patients with this disorder. Even though, these patients have an elevated ICP, anaesthesia does not cause any detrimental effects in patients with IIH. So, we have to take measures to avoid increase in ICP during the perioperative period. Despite the presence of raised ICP in these patients, there is no specific contraindication to either spinal or epidural anaesthetic technique since uncal herniation does not occur in these patients. The main goal is to avoid further increases in ICP.

REFERENCES

1. Aly EE, Lawther BK. Anaesthetic management of uncontrolled idiopathic intracranial hypertension during labour and delivery using an intrathecal catheter. Anaesthesia 2007;62:178-81.
2. Karmaniolou I, Petropoulos G, Theodoraki K. Management of idiopathic intracranial hypertension in parturients: Anesthetic considerations. Can J Anaesth 2011;58:650-7.
3. Sheets C, Peden M, Guy J. Idiopathic intracranial hypertension in a transgender man. J Neuroophthalmol 2007;27:313-5.
4. Soler D, Cox T, Bullock P, Calver DM, Robinson RO. Diagnosis and management of benign intracranial hypertension. Arch Dis Child 1998;78:89-94.
5. Bagga R, Jain V, Das CP, Gupta KR, Gopalan S, Malhotra S. Choice of therapy and mode of delivery in idiopathic intracranial hypertension during pregnancy. MedGenMed 2005;7:42.
6. Abouleish E, Ali V, Tang RA. Benign intracranial hypertension and anesthesia for cesarean section. Anesthesiology 1985;63:705-7.
7. Cenic A, Craen VL, Howard-Lech RA, Lee TY, Gelb AW. Cerebral blood volume and blood flow at varying arterial carbon dioxide tension levels in rabbits during propofol anesthesia. Anesth Analg 2000;90:1376-83.