Ischaemic optic neuropathy after spinal fusion

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Purpose: We report a case of ischaemic optic neuropathy which occurred after prolonged spine surgery in the prone position in an obese, diabetic patient.

Clinical features: The patient was a 44-yr-old, 123 kg, 183 cm man for decompressive laminectomy and instrumented fusion of the lumbar spine. Anaesthesia was induced with thiopentone, fentanyl and succinylcholine and maintained with nitrous oxide, oxygen, isoflurane and a fentanyl infusion. He was positioned prone on the Relton-Hall frame and had an uneventful intraoperative course. Estimated blood loss was 3,000 ml. He was taken to the surgical intensive care unit (SICU) and the trachea was extubated 3.5 hr later. He had no pulmonary or haemodynamic problems and went to a regular nursing floor in the morning. He was discharged home on postoperative day #5. He telephoned his surgeon on postoperative day #7 to say that his vision had been blurry since surgery. His visual acuity was decreased, and on examination, he had a bilateral papillary defect, optic swelling and a splinter haemorrhage in the right eye. Magnetic resonance imaging (MRI) scan of the head and orbits detected no other abnormality. Based on this examination, he was felt to have bilateral ischaemic optic neuropathy and treated conservatively. By postoperative day #47, his visual acuity was greatly improved and near normal. Careful review of possible contributing factors suggests that the cause of the ischaemic optic neuropathy was venous engorgement.

Conclusion: This patient developed ischaemic optic neuropathy from a prolonged interval in the prone position of the Relton-Hall frame, which may be related to venous engorgement.

Objectif : Nous rapportons un cas de neuropathie optique ischémique consécutive à une chirurgie rachidienne prolongée en décubitus ventral chez un obèse diabétique.

Éléments cliniques : Un patient de 44 ans, pesant 123 kg et mesurant 183 cm était opéré pour une laminectomie décompressive avec ostéosynthèse instrumentale de la colonne lombaire. Le thiopental associé à du fentanyl et de la succinylcholine a servi à induire l'anesthésie, qui a été entretenue avec du protoxyde d'azote, de l'oxygène, de l'isoflurane et une perfusion de fentanyl. Il était immobilisé en décubitus ventral sur un cadre de Relton-Hall et l'opération s'est déroulée sans incident. On a estimé la perte sanguine à 3 000 ml. Il a été transféré à l'unité des soins intensifs (USI) et extubé 3,5 h plus tard. Il ne présentait aucun problème respiratoire et hémodynamique et on l'a ramené dans son unité de soins le lendemain matin. Il était libéré le cinquième jour. Par téléphone, il avisait son chirurgien le septième jour que sa vision était embrouillée depuis l'intervention. Son acuité visuelle avait diminué et à l'examen, il présentait une lésion papillaire bilatérale, de l'oedème du nerf optique et une hémorragie linéaire de l'oeil droit. L'imagerie par résonance magnétique de la tête et des orbites ne montrait aucune autre anomalie. Cet examen suggérait une neuropathie optique ischémique bilatérale qui fut traitée de façon conservatrice. Le 47^e jour postopératoire, son acuité visuelle s'était améliorée considérablement et était presque redevenue normale. Parmi les facteurs contributaires possibles cette neuropathie ischémique, il faut retenir l'engorgement veineux.

Conclusion : À la suite d'une immobilisation prolongée en décubitus ventral sur un cadre de Relton-Hall, ce patient a présenté une neuropathie optique par ischémie causée vraisemblablement par engorgement veineux.

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ISCHAEMIC optic neuropathy (ION) is the most frequently reported cause of postoperative visual loss after general Anaesthesia.¹ Temporal arteritis, giant cell arteritis, arteriosclerosis, hypertension, diabetes, polycythemia vera, sickle cell disease, syphilis, narrow angle glaucoma, atherosclerotic vascular occlusive disease, emboli, cardiopulmonary bypass and collagen vascular disorders have been cited as contributing factors to loss of visual acuity after general anaesthesia.²⁻⁷ Hypotension, anaemia, or both have been identified in the literature as the main causes of ION, but it has been reported without these factors.^{1,6,7} We report a patient who underwent prolonged spinal fusion surgery on a Relton-Hall frame who experienced postoperative ION, despite the lack of prolonged hypotension or significant anaemia.

Case report

A 44-yr-old Caucasian man (183 cm, 123 kg, body mass index 36.7 kg·m⁻²) presented for elective decompressive laminectomy with instrumented lumbar fusion for treatment of lumbar canal stenosis and L4-5 foraminal stenosis. He had a medical history of type I insulin-dependent diabetes mellitus since age 14 yr, and at the time of surgery the patient had no end organ impairment related to diabetes. His visual acuity was recorded at 20/20 in both eyes in the recent past, and no diabetic retinopathy was present or recognised. The patient also was morbidly obese and had had an L4-5 laminectomy in the past. He had no known drug allergies. The patient's current medications included lente insulin (35 u AM, 40 u PM) and regular insulin (10 u AM, 20 u PM). Vital signs showed a blood pressure (BP) of 126/74 mmHg, a heart rate (HR) of 72 bpm, a tympanic membrane temperature (T) of 36.3°C, and a respiratory rate of 18 breaths per minute. Physical examination was otherwise unremarkable. The haemoglobin concentration was 15.9 g·dl⁻¹ in the fasting state. Other laboratory values were normal, except for a serum glucose (204 mg·dl⁻¹; normal 65–110 mg·dl⁻¹). The preoperative ECG showed a normal sinus rhythm with nonspecific T-wave abnormalities.

On the morning of surgery, the patient received half of his usual morning dose of lente insulin. Anaesthesia was induced with 4 mg·kg⁻¹ thiopentone, 4 mg·kg⁻¹ fentanyl, and intubation was facilitated with 160 mg succinylcholine mg after 4.5 mg *d*-tubocurarine had been given to prevent fasciculation. Anaesthesia was maintained with a fentanyl infusion at 2 µg·kg⁻¹·hr⁻¹, nitrous oxide 60%, isoflurane 0.4%, and pancuronium. Ventilation was controlled with a tidal volume of 1000 ml at 10 breaths·min⁻¹ resulting in a P_{ET}CO₂ of approx-

imately 30 mmHg. Two large-bore intravenous cannulae, a radial arterial cannula, and a urinary catheter were placed before the patient was positioned prone on the Relton-Hall frame. His neck was maintained in the midline position with moderate flexion, and although the head was slightly dependent, the patient's back was in a neutral position. The forehead was supported without pressure on the orbits. Throughout surgery, his position was repeatedly checked; there was special attention to the brachial plexus, ulnar nerves, axillae, and orbits to detect movement on the frame during the operation. Haemodynamic stability was maintained by titrating an infusion of sodium nitroprusside in preference to inhaled isoflurane concentration to prevent the possible interference with intraoperative monitoring of somatosensory evoked potentials (SSEP). The SSEP monitoring was conducted by the neurology department using the posterior tibial nerve. Except for two five-minute episodes in which the systolic blood pressure decreased to 90 mmHg, SBP was maintained between 95 and 120 mmHg (within 24% below baseline) throughout surgery. His heart rate remained at 60 to 85 bpm. Arterial blood gases and haemoglobin concentration were monitored periodically (nine times) throughout the 12 hr operation. The PaO₂ ranged from 82 to 140 mmHg, and the PaCO₂ from 30 to 35 mmHg. Blood glucose concentration was maintained between 80 and 200 mg·dl⁻¹ with intermittent, small doses of insulin. Intraoperatively, the haemoglobin concentration varied between 13.4 and 9.4 g·dl⁻¹. Surgery lasted for 12 hr because of previous spine surgery, the patient's physical size and the large amount of venous bleeding. Blood loss was estimated at 3,000 ml and was replaced with 8000 ml ringers lactate, 2,000 ml normal saline, 1,000 ml Hespan and three units of packed red blood cells.

After surgery, the patient resumed the supine position and was transported to the SICU with the endotracheal tube in place. In the supine position, there were no anatomical abnormalities of the head or neck although the patient did have some edema of face and eyelids without ecchymosis. On arrival in the surgical intensive care unit (SICU), his vital signs were: BP 155/70 mmHg; HR 91 bpm; tympanic temperature 35.8°C; Hb 9.5 g·dl⁻¹; and serum glucose 244 mg·dl⁻¹. Other laboratory tests were normal. The head of the bed was elevated to reduce mild upper body oedema. Arterial blood gas analysis showed PaO₂ of 167 mmHg and PaCO₂ of 35 mmHg. The trachea was successfully extubated 3.5 hr after arrival in the intensive care unit. After an uneventful night, he was transferred to the regular nursing floor. He was discharged home on postoperative day #5.

On postoperative day #7, he phoned his surgeon to report, for the first time, that his vision had been "blurry" since his surgery. On day #8 he was examined by his ophthalmologist who noted his visual acuity to be 20/200 in each eye compared with 20/20 bilaterally preoperatively. A diagnosis of bilateral anterior ION was made based on the presence of an afferent papillary defect with bilateral optic swelling and a splinter haemorrhage in the right eye. There was no retinopathy consistent with visual changes related to long standing diabetes. A magnetic resonance imaging (MRI) scan of the head, with special attention to the orbital region, was obtained and ruled out a demyelinating disease, such as multiple sclerosis, as a possible cause of the visual changes. There were no additional findings on the MRI. Evaluation on day #47 revealed his vision to be nearly normal.

Discussion

Postoperative ION is the most frequently cited cause of postoperative visual deficit, particularly after open heart surgery, but it has received little attention in the anaesthesia literature until recently.^{1,6,7} Although multiple risk factors for the development of perioperative ION exist, hypotension and anaemia have been identified as the most common causes of postoperative ION.^{1,5,6,8}

During this procedure, the blood pressure was tightly controlled with sodium nitroprusside. The nitroprusside dose ranged between 0–4 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ to keep the systolic blood pressure between 95–120 mmHg in an attempt to decrease blood loss during the surgery. The total dose of nitroprusside was <50 mg. On two occasions the systolic blood pressure decreased transiently to 90 mmHg during periods of minimal surgical stimulation. With adjustment of the nitroprusside dosage, the blood pressure returned to within the target range within five minutes. Although hypotension is a common cause of ION, the transient hypotension in this case should not have been the major contributor to the development of ION.

Anaemia is also a possible contributor to ION. The Hb concentration started at 15 $\text{g}\cdot\text{dl}^{-1}$, and throughout the procedure, the patient received three units of blood. Despite the maintenance of blood pressure between 95–120 mmHg with nitroprusside, the blood loss was heavy for this procedure at three litres. Although the patient did not show signs of decreased oxygen carrying capacity, such as hypotension, tachycardia, decreased urine output, metabolic acidosis or changes in the SSEP, he was transfused with packed red blood cells when the Hb was <10 $\text{g}\cdot\text{dl}^{-1}$ because of continuing blood loss. The lowest Hb during the procedure was 9.4 $\text{g}\cdot\text{dl}^{-1}$ and

did not occur during either brief hypotensive episode. Neither the anaemia nor the hypotensive episodes were associated with any SSEP change. The lowest haemoglobin concentration was promptly corrected with a packed red cell transfusion. Although anaemia is a common cause of ION, to be a major factor in this case there should have been some other sign of poor tissue perfusion, such as metabolic acidosis or end-organ ischemia. Considering the age of this patient and the absence of pre-existing retinopathy, this relative anaemia was probably not the cause of the ION.

While in the operating room, there were no changes in heart rate or $\text{P}_{\text{ET}}\text{CO}_2$. There were no alterations in oxygen saturation, capnography or haemodynamics consistent with fat embolism, nor were there signs of fat embolism after surgery.^{3,4} The patient had insulin dependent diabetes mellitus which is a risk factor for ION.² The blood sugar concentration on the routine lab work was non-fasting at 204 $\text{mg}\cdot\text{dl}^{-1}$, and the patient had well controlled diabetes including a negative Hb A 1-C. The patient's blood sugar in the operating room was maintained <200 $\text{mg}\cdot\text{dl}^{-1}$ with intermittent doses of regular insulin. Although hyperglycaemia may worsen an ischaemic insult, the threshold for this risk is not known. Postoperative hyperglycaemia has been associated with postoperative ION,^{3,4} although this possible explanation for the outcome of this case was not supported by the clinical events.

The head of the patient on the Relton-Hall frame was in a dependent, down-tilt position for nearly 12 hr. A decrease in venous outflow from the cranium, leading to an elevation in local capillary bed stasis has been cited as a contributing factor in postoperative ION.⁹ This position could have decreased perfusion pressure to the capillary bed of the optic nerve head secondary to an elevated central venous pressure and decreased venous outflow. The CVP may have been elevated in the prone position due to compression of the abdomen by the frame and upward shift of the diaphragm. This patient was morbidly obese and, when placed on the Relton-Hall frame, abdominal excursion was restricted by the frame. The heavy surgical bleeding, probably related to compression of the inferior vena cava by increased intraabdominal pressure, is further evidence that CVP could have been elevated by the position on the frame. Although the back was in a neutral position, the head was dependent which increased CVP further.

As a result of increased CVP or venous obstruction, elevation in intra-ocular pressure (IOP) can produce a corresponding decrease in choroidal blood flow by elevating the episcleral venous pressure which acts as the

venous outflow pathway for the aqueous humour.¹⁰ This could result in an elevation of the IOP and further decrease the perfusion pressure to the optic nerve head. The elevation of the central venous pressure could have directly affected optic nerve head perfusion by causing retarded venous drainage through the superior ophthalmic vein into the cavernous sinus due to an elevation of central venous pressure transmitted to the brain. We feel that the prolonged elevations of central venous pressure and increase in intra-ocular pressure resulted in a characteristic arterial occlusion in the posterior ciliary circulation which caused the ION in this patient. Obstruction to venous outflow is considered to be a contributing factor in postoperative ION after head and neck surgery,⁸ and we believe such obstruction is an under-appreciated risk factor for ION in the prone position. Proper positioning of the head should be considered an important issue even in the postoperative period, as it is also a risk factor for ION.¹¹ In addition to hypotension, anaemia and diabetes mellitus, other risk factors which would apply in the postoperative period include excessive weight gain from fluid replacement, microembolism and the use of inotropes, such as epinephrine and amrinone.¹²

In conclusion, we feel that prone positioning, on a Relton-Hall frame, of an obese patient for a long spinal surgery contributed to the development of ION. The Relton-Hall frame may not be the best choice for obese patients scheduled for prone procedures of long anticipated duration, and a frame that allows for the increased abdominal girth may be a better choice.

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References

- 1 Williams EL, Hart WM Jr, Tempelhoff R. Postoperative ischemic optic neuropathy. *Anesth Analg* 1995; 80: 1018-29.
- 2 Miller NR. Anterior ischemic optic neuropathy. In: *Clinical Neuro-Ophthalmology*, 4th ed. Baltimore: Williams & Wilkins, 1982: 212-6.
- 3 Horton JC. Embolic cilioretinal artery occlusion with arteriosclerosis of the ipsilateral carotid artery. *Retina* 1995; 15: 441-4.
- 4 Lieberman MF, Shabi A, Green WR. Embolic ischemic optic neuropathy. *Am J Ophthalmol* 1978; 86: 206-10.
- 5 Newman NJ. Optic neuropathy. *Neurology* 1996; 46: 315-22.
- 6 Brown RH, Schauble JF, Miller NR. Anemia and hypotension as contributors to perioperative loss of vision. *Anesthesiology* 1994; 80: 222-6.
- 7 Lund PE, Madsen K. Bilateral blindness after cardiopulmonary bypass. *J Cardiothorac Vasc Anesth* 1994; 8: 448-50.
- 8 Johnson MW, Kincaid MC, Trobe JD. Bilateral retrobulbar optic nerve infarctions after blood loss and hypotension. A clinicopathologic case study. *Ophthalmology* 1987; 94: 1577-84.
- 9 Bouzaïenne M, Deboise A, Kheyar A, Le Queau F, Lemogne M, Baudoin Lacour S. Cécité bilatérale irréversible après curage cervical fonctionnel bilatéral. A propos d'un cas et revue de la littérature. *Rev Stomatol Chir Maxillofac* 1994; 95: 226-32.
- 10 Hvidberg A, Kessing SV, Fernandes A. Effect of changes in PCO₂ and body positions on intraocular pressure during general anaesthesia. *Acta Ophthalmol* 1981; 59: 465-75.
- 11 Schobel GA, Schmidbauer M, Millesi W, Undt G. Posterior ischemic optic neuropathy following bilateral radical neck dissection. *Int J Oral Maxillofac Surg* 1995; 24: 283-7.
- 12 Shapira OM, Kimmel WA, Lindsey PS, Shahian DM. Anterior ischemic optic neuropathy after open heart operations. *Ann Thorac Surg* 1996; 61: 660-6.