

CASE REPORT

Ischaemic optic neuropathy in a blunt abdominal trauma patient with profound shock: a case report and concise review of possible risk factors

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Abstract

Ischaemic optic neuropathy and resulting visual loss is a known perioperative complication. In the intensive care unit (ICU), however, it is rarely seen. We present a case of ischaemic optic neuropathy and describe factors associated with this condition, found in the current literature. Our patient who was admitted and treated in the ICU of Medical Center Haaglanden in The Hague, is a 33-year-old male, who sustained blunt abdominal trauma resulting in liver rupture. He underwent multiple operations, was mechanically ventilated in the prone position with high positive end-expiratory pressure (PEEP) received large amounts of fluids and was treated with vasoactive drugs. He developed blindness because of ischaemic optic neuropathy (ION). We believe that the ION was caused by a combination of risk factors. Current anaesthetic literature describing ION after surgery indicates several independently associated risk factors for this rare complication. In our patient these were male sex and the large volume of estimated blood loss. Further review of the literature suggests massive fluid resuscitation, hypotension and the use of vasopressors as possible risk factors, but these are not proven to be independently associated with ION.

Introduction

Ischaemic optic neuropathy (ION) and resulting visual loss is an uncommon perioperative complication. Incidence after spinal fusion surgery is estimated from 0.017% to 0.36%.^{1,2} In the intensive care unit (ICU) it is rarely seen. We present a patient who developed profound shock after blunt abdominal trauma and respiratory failure requiring mechanical ventilation in the prone position with high positive end-expiratory pressure (PEEP). He developed blindness during his ICU stay. We reviewed the current literature to describe possible pathogenetic factors leading to decreased oxygen delivery to the optic nerve, causing ION.

Case report

A 33-year-old male with a history of alcohol abuse was admitted to the emergency department after a motorcycle accident. He sustained blunt abdominal trauma and was diagnosed with liver rupture. In severe hypovolaemic shock, he underwent an emergency laparotomy, having systolic blood pressures of around 60 mmHg. He was initially treated with perihepatic packing and after 24 hours the injured liver segments V, VI and VII were resected. The patient was admitted to the ICU and in the following period he was mechanically ventilated, with a maximum PEEP of 20 mmHg. Mechanical ventilation was performed in the prone position for 29 hours. In the first 24 hours, the patient was resuscitated with 21.8 litres of fluids, including 19 units of packed red blood cells, 10 units of fresh frozen plasma and 2 units of thrombocytes. For 5 days, he was supported with a dopamine infusion, with a maximum dosage of $25 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, and a dobutamine infusion, with a maximum dosage of $10.4 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. The target mean arterial pressure was $> 65 \text{ mmHg}$. Furthermore, the patient was treated with continuous veno-venous haemofiltration while he developed anuria. He underwent multiple procedures, including an ileostomy because his surgical treatment was complicated by intestinal obstruction and an intra-abdominal abscess. When sedation was stopped and the patient regained the ability to communicate, he complained of blindness. Ophthalmologic examination revealed bilateral oedema of the optic disc, supporting the diagnosis of optic nerve neuropathy. Computed tomography excluded any cerebral pathology. At the end of his hospital stay, the patient had no light perception and started on a rehabilitation program.

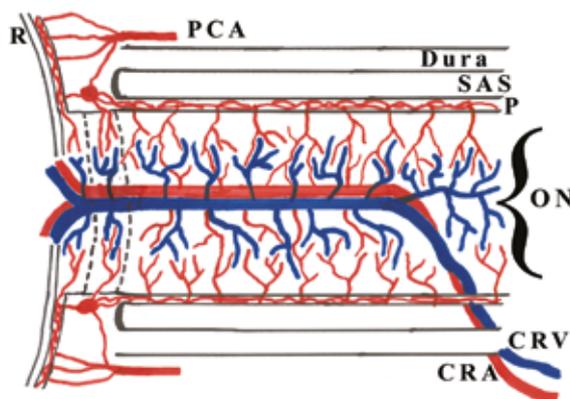
Discussion

In 1973, Drance et al. proposed shock-induced ION as a causal factor of glaucoma and visual loss.³ In 1988, Chelluri was the first to report of visual loss in a patient treated in the ICU, after

cardiac arrest.⁴ He suggested that high levels of PEEP together with a low systemic filling pressure can cause intraocular pressure build-up and decreased perfusion pressure leading to ischaemia of the optic nerve. Most of our knowledge is from case reports like these. There are no controlled studies on ION and the underlying pathology is poorly understood. The intraocular and retrobulbar segments of the optic nerve are supplied by the posterior ciliary artery and have a less abundant vascular supply than the intracranial segments, but the specific mechanism and location of the vascular insult is not known.² Figure 1 is a schematic representation of the optic nerve vascular supply.

ION is the most common diagnosis in perioperative visual loss in non-ophthalmologic surgery.^{5,6} It is particularly associated with spine surgery in the prone position. Possible risk factors for ION after this type of surgery were evaluated in 2012 by the Postoperative Visual Loss Study Group in a case-control study of 80 affected patients matched with 315 control subjects.⁷ This is the only study in which risk factors were systematically evaluated. The authors suggest venous congestion plays an important role in the aetiology of ION, because they found obesity (with elevated intra-abdominal pressure in the prone position), long anaesthetic duration and the use of a Wilson frame (with the head positioned lower than the heart) to be independent risk factors. Furthermore, they found male sex, lower percentage of colloid administration (of total non-blood fluid infusion) and greater estimated blood loss as independent risk factors. Considering venous congestion, one may expect patient tilt to be of influence, but the authors were not able to study this variable.

Figure 1. Representation of the optic nerve vascular supply. In our patient there was ischaemia of the optic nerve head, which is the region of the optic nerve directly behind the retina. The main source of blood supply is from the posterior ciliary artery. The central retinal artery gives off no branches in this region



R = retina; PCA = posterior ciliary artery; SAS = subarachnoid space; P = pia mater; ON = optic nerve; CRV = central retinal vein; CRA = central retinal artery

Blindness in the ICU is a rare complication. The aetiology of ION in this setting remains unknown, and current literature consists of case reports and expert opinions. It is suggested that severe hypotension causes local ischaemia around the optic nerve head, leading to ION.⁸ However, in the previously mentioned study the Postoperative Visual Loss Study Group did not find an independent effect of low blood pressures.

In 2000, Cullinane et al. reviewed 350 trauma patients who received massive volume resuscitation (>20 litres infused in the first 24 hours), of which nine patients developed ION.⁹ They suggest that the combination of massive resuscitation and prone positioning (for acute respiratory distress syndrome) will increase the risk of ischaemia through capillary leakage and pressure build-up around the optic nerve. They also suggest the inflammatory response plays an important role in the pathogenesis of ION. This remains to be investigated.

Concerning vasoactive drugs, Lee et al. presented a series of four patients who developed ION within one month of each other.¹⁰ They suggest the use of vasopressors might play an important role. All four patients at their ICU received treatment with norepinephrine together with one or more other vasoactive agents. However, since many ICU patients receiving vasoactive drugs do not develop ION, the contribution to its pathogenesis remains unclear. Our patient was treated with dopamine and dobutamine. In a study by Huemer et al., dopamine had no effect on optic nerve head blood flow in healthy volunteers.¹¹ The effect of dobutamine on ocular blood flow has never been investigated. In review: our patient had a history of alcohol abuse, which is not described as a risk factor in the current literature, although in the previously mentioned case series by Lee et al., all four patients had a history of alcohol abuse.¹⁰ Furthermore, our patient was of male gender and suffered massive blood loss, two risk factors shown to be independently associated with ION in current anaesthetic literature. The effects of other suggested risk factors, such as high levels of PEEP, large amounts of fluid infusion and the use of vasoactive drugs, remain unclear. This is reflected in a recent randomised controlled trial in which ICU patients with severe acute respiratory distress syndrome (ARDS) were ventilated in the prone position.¹² The mean duration was four sessions, with 17 hours per session. There were no reports of visual loss. Based on these data, the prone position is less likely to be an independent risk factor. In our patient, there could be other factors of importance, such as systemic inflammation and immunosuppression.¹³ Which role they play is not known.

Conclusion

The aetiology of ION in the ICU setting is a matter of debate. After reviewing our case, although some risk factors for ION were present (male sex, large volume of estimated blood loss), it is not clear why this particular patient developed ION. Most ICU patients with shock, polytransfusion and prone mechanical ventilation do not develop ION.

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