

Amaurosis after spine surgery: survey of the literature and discussion of one case

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Abstract Postoperative vision loss (POVL) associated with spine surgery is a well known, albeit very rare complication. POVL incidence after spinal surgery ranges from 0.028 to 0.2%; however, due to the increase in number and duration of annual complex spinal operations, the incidence may increase. Origin and pathogenesis of POVL remain frequently unknown. A 73-year-old patient presented with lumbar disc herniation with associated neurological deficits after conservative pre-treatment at a peripheral hospital. Known comorbidities included arterial hypertension, moderate arterial sclerosis, diabetes mellitus type 2, mildly elevated blood lipids and treated prostate gland cancer. During lumbar spine surgery in modified prone position the patient presented with an acute episode of severe hypotension, which required treatment with catecholamines and Trendelenburg positioning. Three hours postoperatively, a visual loss in the right eye occurred, resulting in a complete amaurosis. Antihypertensive medication, arteriosclerosis and intraoperative hypotension are possible causes for the POVL. Intraoperative administration of catecholamines and Trendelenburg positioning for treatment of systemic hypotension might further

compromise ocular perfusion. In patients with comorbidities compromising arterial blood pressure, blood circulation and microcirculation, POVL must be considered as a severe postoperative complication. It is recommended to inform patients about such complications and obtain preoperative informed consent regarding POVL. Any recent modification of antihypertensive medication must be reported and analysed for potential intraoperative hemodynamic consequences, prior to spine surgery in prone position.

Keywords Amaurosis · Blood pressure · Spinal surgery · Prone position · Postoperative vision loss (POVL)

Introduction

Postoperative vision loss (POVL) associated with spine surgery in prone position is a well known yet very rare complication [1, 2]. The exact origin and pathogenesis are still unknown in many cases [3, 4]. The most common ophthalmologic diagnosis associated with POVL is ischemic optic neuropathy (ION) [5, 6]. Other causes include central retinal artery occlusion (CRAO), cortical blindness as well as unknown aetiology [5, 6]. A practice advisory for perioperative visual loss associated with spine surgery is provided e.g. by the “American Society of Anesthesiologists” [7].

Survey of literature

There are several reports of vision impairment after surgery in prone position or cardiac surgery. The incidence of POVL after non-ocular surgeries has been reported as 0.002% among all surgeries and reaches 0.2% among cardiac and spine surgeries [8–11]. Due to an increase in number and duration of annual complex spinal operations,

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POVL incidence may increase [1, 6, 12–14]. In a recently published retrospective population-based American study with data of 4,728,815 patients over the years 1997–2002, the overall incidence of visual disturbance after spine surgery was 0.094% [15]. In addition to collections of up to 93 cases from several groups, there are a number of single case reports. Within these case reports, 27.3% refer to thoracic [16–18], 18.2% to lumbar [19–21] and 54.5% to cervical surgery [5, 22–25].

Similar reports are given after orthopaedic and general surgery [26–29]. Patil et al. performed a multivariate multicenter analysis based on diagnosis codes and reported that spine surgery had the highest rates of POVL, with 0.28% for scoliosis correction and 0.14% for posterior lumbar fusion. Paediatric patients (<18 years) and elderly patients (>84 years) were 5.8 times and 3.2 times, respectively, more likely than patients 18–44 years of age to develop non-ION, non-CRAO visual loss after spine surgery. Patients with peripheral vascular disease (OR = 2.0), hypertension (OR = 3), and those who received blood transfusions (OR = 2.2) were more likely to develop non-ION, non-CRAO vision loss after spine surgery. ION was present in 0.006% of patients. Hypotension (OR = 10.1), peripheral vascular disease (OR = 6.3) and anaemia (OR = 5.9) were the strongest risk factors identified for the development of ION [30]. An overview of the published studies of POVL after spine surgery is shown in Table 1.

Case

Special history

A 73-year-old patient presented with radicular pain and sensorimotor deficits was diagnosed with a large

mediolateral lumbar disc herniation at lumbar level 4/5 and a caudally displaced sequester that compressed the right-sided root of the nerve L5. The patient was treated conservatively as an in-patient with physiotherapy and analgesics for over 6 weeks. Lack of improvement led to indication for discectomy and the patient was transferred to our department for surgical treatment.

General history

The patient presented with the following comorbidities: arterial hypertension, generalised arteriosclerosis, type 2 diabetes mellitus, obesity (BMI 31 kg/m²) and radiation therapy for prostate gland cancer 3 years ago. His medication was NORVASC® 10 mg (Amlodipine, Besylate), ATACAND PLUS® 16 mg (combination of Candesartan and Hydrochlorothiazide), BELOC-ZOK® 50 mg (Metoprolol), MODURETIC® (Hydrochlorothiazide 50 mg and Amilorid HCl 5 mg), ASPIRIN CARDIO® 100 mg (Acetylsalicylic acid), AMARYL® 2 mg (Glimerid). He had no history of angina pectoris or other cardiac symptoms and the patient's general physical condition was appropriate for his age.

The day before surgery, the patient underwent preoperative anaesthetic evaluation. Blood pressure was 130/80 mmHg and haemoglobin level was 143 g/l with a haematocrit of 0.38. The indication for surgery was verified by the responsible surgeon and affirmed with written consent, including information about the risks of a POVL. The only drugs given on the day of surgery were oral midazolam 7.5 mg, metoprolol and candesartan.

Surgery

Standard monitoring was established in the OR consisting of non-invasive blood pressure monitoring, ECG and

Table 1 Overview of the published studies of POVL after spine surgery [15, 56, 34, 4, 13, 57, 38, 36, 6]

Study	Number of POVL-patients	Incidence of POVL	Comments
Retrospective studies			
Patil et al. [15] 2008	4,452	0.094% (4,452/4,728,815)	1993–2002 US population analysis, ICD based
Chang and Miller [56] 2005	4	0.03% (4/14,102)	John Hopkins
Warner et al. [34] 2001	8	0.07% (8/11,492)	Mayo clinic
Roth and Barach [4] 2001	1	0.09% (1/1,100)	University of Chicago
Stevens et al. [13] 1997	7	0.20% (7/3,450)	Pooled data from 3 centres
Survey studies			
Delattre et al. [57] 2007	17	–	28 French orthopaedic spine centres
Lee et al. [38] 2006	93	–	ASA POVL-registry
Cheng et al. [36] 2000	24	–	AANS survey
Myers et al. [6] 1997	37	–	Scoliosis Research Society (60/400 responses)

arterial oxygen saturation (SaO_2). Blood pressure was 130/85 mmHg and heart rate 85 s^{-1} . Anaesthesia was induced with intravenous thiopental (4 mg/kg) and fentanyl (1 $\mu\text{g}/\text{kg}$). Succinylcholine (1 mg/kg) was used for neuromuscular relaxation. Anaesthesia was maintained with sevoflurane (0.5–1.5 MAC). Following endotracheal intubation, 10 min later, the patient developed hypotension of 80/45 mmHg that was treated with bolus doses of phenylephrine (100 μg) and ephedrine (10 mg). There was no change in heart rate. Blood pressure then stabilised at 100/50 mmHg. After prone positioning, oculo-bulbar pressure was carefully avoided. Shortly thereafter blood pressure dropped again to 75/40 mmHg. Despite bolus doses of phenylephrine and ephedrine, blood pressure remained at 85/40 mmHg. Therefore, bolus doses of epinephrine (10 μg) were repetitively injected during the following 2 h. The head of the patient was lifted and repositioned every 15 min to avoid prolonged pressure on the ocular bulbs. After flavectomy and during dissection of the sequester in the recessus beside the medialised nerve L5, hypotension recurred and the patient was put into a Trendelenburg position to improve cardiac filling. Surgery was interrupted for approximately 10 min and then the patient was again positioned horizontally. Total blood loss was less than 100 ml. Intravenous fluids provided were 3,100 ml of Ringer's lactate and 0.9% NaCl. Postoperatively, the patient was positioned supine and extubated. The patient was immediately conscious and showed no neurological abnormality except for the pre-existing sensorimotor deficits of nerve L5 on the right side.

Postoperative sequel

Three hours after surgery the patient complained to a nurse of blurred vision in both eyes, more pronounced on his right side. Blood pressure was 130/85 mmHg. No physician was informed. Then the patient was transferred to the neurosurgical ward for further treatment. There he reported once more a reduction of vision in both eyes, worse on his right side. Nurses misinterpreted these symptoms as an effect of the eye ointment as no external problem was observable. Six hours after surgery, the nurses informed the surgeon who diagnosed a complete vision loss of the right eye and a reduction of vision of the left eye. Vital parameters were in a normal range and blood pressure was 140/80 mmHg. The right eye showed no visual reaction to any direct light stimulus though the left eye reacted promptly. Pupils were both of a middle diameter. Vision in the left eye was about 60%. The ophthalmologist diagnosed a complete occlusion of central ophthalmic artery on the right side and a partial occlusion on the left side. An antithrombotic therapy with low-molecular-weight heparin was judged superior to a lytic therapy.

Further sequel and final outcome

An ultrasound examination of the carotid artery showed no stenosis or existing plaques in the carotid bifurcation on either side. There was no evidence of any embolic event. A thoracic CT scan showed no sign of pulmonary embolism. Laboratory values 1 day after surgery for haemoglobin and haematocrit were 117 g/l and 0.32 l/l, respectively. Reduced vision in the left eye was probably pre-existing and improved with corrective lenses. The complete amaurosis of the right eye remained unchanged. In the follow-up period of 3 years with neurological and ophthalmologic examinations at 2, 6 months, 1, 2 and 3 years after surgery, the ophthalmologic findings were unchanged. The neurological examination showed that the preoperative existing deficits of nerve L5 had completely regressed.

The complete patient's history was carefully reinvestigated after the incident. It became apparent that the anti-hypertensive medication had been modified in the peripheral hospital, just before the patient's transfer into our clinic. The patient's initial medication consisted of hydrochlorothiazide 50 mg and amilorid 5 mg, captopril 12.5 mg and atenolol 100 mg combined with chlorthalidone 25 mg. Two days before surgery, the patient's antihypertensive treatment had been supplemented with a calcium antagonist (amlodipine), a beta-blocker (metoprolol) and the ACE inhibitor had been replaced by an AT-II antagonist (candesartan).

Discussion

The combination of extensive antihypertensive medication, arteriosclerosis, intraoperative hypotension and catecholamine treatment [31] is the most probable cause of POVL in this patient. Supplementation of antihypertensive medication 2 days before surgery was certainly an aggravating factor. During this 2-day period, the blood pressure might have been in a normal range for a 40-year-old man but not for a 73-year-old who is used to high blood pressure in daily life. The 3 h delay prior to visual loss is not possible to enlighten definitely, most probable causes are the missing documentation and/or the lack of awareness of the responsible nurses. Other authors report release of hallucinations and visual loss as first manifestations of postoperative unilateral blindness [32]. A greater awareness of the patient's first reported visual impairment by the nurses may have improved his chances of a better outcome [33].

Several groups have undertaken previous retrospective investigations of POVL: an ex post facto study was performed by the Mayo Clinic, Rochester, from 1986 to 1998 [34]. Vision loss was considered present in this investigation if any part of the visual field was affected. Initial database screening found 405 cases of new-onset vision

loss and visual changes in 410 cases; 216 of these patients regained full visual acuity within 30 days. In this study, only 1 per 125,234 patients undergoing non-cardiac surgery developed vision loss persisting for longer than 30 days. The researchers concluded that vision loss and blindness after surgery and anaesthesia are still very rare events.

The same group reported other postoperative visual changes such as blurred vision, which was reversible in most cases [33, 35]. In another extended survey, a questionnaire focussing on intraoperative factors that may predispose patients to perioperative vision loss was sent to current members of the American Association of Neurological Surgeons/Congress of Neurological Surgeons, Section on Disorders of the Spine and Peripheral Nerves [36]. Out of 290 returned surveys, 22 surgeons reported 24 patients with visual loss after spine surgery. Although many of these patients had probable causative factors for POVL (e.g. hypotension, low haematocrit level, coexisting disease), some did not ($n = 8$) [3, 4, 36, 37], suggesting the necessity of high vigilance for evolving POVL even in the absence of risk factors.

In 2006, the “American Society of Anesthesiologists” POVL-Registry did an analysis of spine surgery cases with POVL showing ION in 83 out of 93 spine surgery cases [38]. The mean age of the patients was 50 ± 14 years, and most patients were relatively healthy. Mayfield pins supported the head during surgery in 16 out of 83 cases. The mean anaesthetic duration was 9.8 ± 3.1 h and the median estimated blood loss was 2.0 l (range 0.1–25 l). Bilateral disease was present in 55 patients, with complete visual loss in the affected eye(s) in 47 cases. Patients with ION had significantly longer anaesthetic duration, more blood loss, were more frequently positioned with Mayfield pins and had more bilateral POVL compared to the remaining 10 cases with CRAO ($P < 0.05$). This suggests a different aetiology for ION and CRAO. The conclusion was that ION was the most common cause of POVL after spine surgeries reported while most of the affected patients in this study were relatively healthy. Blood loss of over 1,000 ml or anaesthetic duration of over 6 h was reported in 96% of these cases. The authors recommended that for patients undergoing prolonged spine surgery in the prone position, the risk of POVL should be discussed preoperatively with the patients.

Intraoperative visual-evoked potential monitoring has been proposed [30], but has not yet proven to be effective.

The aetiology of POVL is probably multifactorial [39]. However, patients with an extensive blood loss resulting in hypotension and anaemia along with prolonged operative durations [29] may be more at risk to develop visual disturbances. An acute anaemic state may have an additive or

synergistic effect with other factors such as medical comorbidities leading to visual disturbances [11, 40].

Different hypotheses for the pathophysiological causes of POVL were discussed and investigated. Even without surgery, ION is a common cause of visual loss in the elderly population and shows certain risk factors like hypertension, nocturnal hypotension, diabetes mellitus, atherosclerosis and small cup in the optic disc [41, 42], which should be taken into account in spinal surgery patients. In addition to patient positioning during surgery [43–46], surgery duration and loss of blood [47] seem to be important risk factors [48]. Other factors such as micro-embolisms [49], decrease of haematocrit [47] or poor oxygen saturation [50], hypocapnia [50] and prolonged external ocular compression during surgery are also described [51]. These factors may occur as a single or multifactorial cause [39, 52, 53].

Our hypothesis is that in the reported patient, POVL was caused by an unusually low intraoperative blood pressure where the perfusion pressure was too low for the arteriosclerotically altered vessels and effector organs. Missing information regarding the very recent alteration of anti-hypertensive medication was an additional factor.

Conclusion

In patients with cardiovascular comorbidities such as arterial hypertension, decreased blood circulation and microcirculation, severe postoperative complications such as POVL should be taken into account while planning surgical treatment [38]. Adequate transfusion guidelines should be established for at-risk patients. Unacceptable values of haemoglobin and haematocrit should be corrected preoperatively (e.g. by substituting iron and erythropoietin) and monitored during surgery to avoid intraoperative anaemia in at-risk patients. The blood pressure of patients with predisposing diseases should be kept within normal limits. To avoid POVL as a devastating complication, it is imperative that anaesthesiologists understand contributing factors and prevention strategies [54, 55].

It is recommended for spine surgeons to obtain preoperative informed consent regarding POVL. Since visual loss following spine surgery may be reversible in the early stages, awareness, evaluation and prompt management of this rare but potentially devastating complication is critical [15]. Therefore, we chose the following recommendations from the literature to be regarded by surgeons and anaesthesiologists.

Recommendations according to the literature [1, 7, 15]:

- Avoid direct pressure on the globes
- Avoid perioperative hypotension

- Avoid perioperative anaemia
- Consider 10 degrees of reverse Trendelenburg during prone surgery
- Lower transfusion threshold to keep haematocrit above 30% in at-risk patients
- Avoid infusions of large amounts of crystalloid
- Consider staging long spinal surgeries (above 8 h)
- Maintain mean arterial pressure at patient's baseline
- Avoid changes in any perfusion-related medication shortly before surgery
- Perform a postoperative visual exam as early as possible in at-risk patients

At-risk patients are defined as patients who suffer from a pre-existing cardiovascular disease and additional metabolic diseases, where a prolonged duration of surgery in prone position (e.g. >2 h) or an increased blood loss is expected.

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