

CLINICAL INFORMATION

Postoperative visual loss due to conversion disorder after spine surgery: a case report



Dailson Mamede Bezerra^{a,b,c,*}, Eglantine Mamede Bezerra^d,
Antonio Jorge Silva Junior^d, Marco Aurélio Soares Amorim^a,
Denismar Borges de Miranda^{e,f}

^a Centro de Ensino e Treinamento Dr. José Quinan, Goiânia, GO, Brazil

^b Defesa Profissional da Sociedade de Anestesiologia do Estado de Goiás (2015/2016), Goiânia, GO, Brazil

^c Universidade Estadual Paulista Júlio de Mesquita Filho (FMB/Unesp), Faculdade de Medicina, Botucatu, SP, Brazil

^d Hospital Adventista de Belém, Serviço de Anestesiologia, Belém, Pará, Brazil

^e Pontifícia Universidade Católica de Goiás (PUC/GO), Goiânia, GO, Brazil

^f Universidade Federal de Goiás (UFG), Goiânia, GO, Brazil

Received 1 February 2015; accepted 3 March 2015

Available online 15 September 2016

KEYWORDS

General anesthesia;
Blindness;
Conversion disorder;
Laminectomy;
Decubitus ventral

Abstract

Background and objective: Patients undergoing spinal surgeries may develop postoperative visual loss. We present a case of total bilateral visual loss in a patient who, despite having clinical and surgical risk factors for organic lesion, evolved with visual disturbance due to conversion disorder.

Case report: A male patient, 39 years old, 71 kg, 1.72 m, ASA I, admitted to undergo fusion and discectomy at L4-L5 and L5-S1. Venoclisis, cardioscopy, oximetry, NIBP; induction with remifentanil, propofol and rocuronium; intubation with ETT (8.0 mm) followed by capnography and urinary catheterization for diuresis. Maintenance with full target-controlled intravenous anesthesia. During fixation and laminectomy, the patient developed severe bleeding and hypovolemic shock. After 30 min, hemostasis and hemodynamic stability was achieved with infusion of norepinephrine, volume expansion, and blood products. In the ICU, the patient developed mental confusion, weakness in the limbs, and bilateral visual loss. It was not possible to identify clinical, laboratory or image findings of organic lesion. He evolved with episodes of anxiety, emotional lability, and language impairment; the hypothesis of conversion syndrome with visual component was raised after psychiatric evaluation. The patient had complete resolution of symptoms after visual education and introduction of low doses of antipsychotic, antidepressant, and benzodiazepine. Other symptoms also regressed, and the patient was discharged 12 days after surgery. After 60 days, the patient had no more symptoms.

* Corresponding author.

E-mail: dailsonbezerra@yahoo.com.br (D.M. Bezerra).

Conclusions: Conversion disorders may have different signs and symptoms of non-organic origin, including visual component. It is noteworthy that the occurrence of this type of visual dysfunction in the postoperative period of spinal surgery is a rare event and should be remembered as a differential diagnosis.

© 2015 Sociedade Brasileira de Anestesiologia. Published by Elsevier Editora Ltda. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

PALAVRAS-CHAVE

Anestesia geral;
Cegueira;
Transtorno
conversivo;
Laminectomia;
Decúbito ventral

Perda visual conversiva em pós-operatório de cirurgia de coluna: relato de caso

Resumo

Justificativa e objetivo: Pacientes submetidos a procedimentos cirúrgicos espinhais podem evoluir com perda visual pós-operatória. Apresentamos quadro de perda visual bilateral total em paciente que, apesar de apresentar fatores de risco clínicos e cirúrgicos para lesão orgânica, evoluiu com distúrbio visual conversivo.

Relato de caso: Masculino, 39 anos; 71 kg; 1,72 m; ASA I, admitido para realização de artrodese e discectomia em L4-L5 e L5-S1. Venoclise, cardioscopia, oximetria, PANI; indução com remifentanil, propofol e rocurônio; intubação com TOT 8,0 mm seguida por capnografia e diurese por sondagem vesical. Manutenção em anestesia venosa total alvo-controlada. Durante fixação e laminectomia, evoluiu com importante sangramento e choque hipovolêmico. Após 30 minutos obteve-se hemostasia e estabilidade hemodinâmica com infusão de noradrenalina, expansão volêmica e hemoderivados. Na UTI, evoluiu com confusão mental, fraqueza em membros e perda visual bilateral. Não foi possível identificar achados clínicos, laboratoriais ou de imagem para lesão orgânica. Evoluiu com episódios de ansiedade, labilidade emocional e distúrbio de linguagem; foi aventada hipótese de síndrome conversiva com componente visual após avaliação psiquiátrica. Apresentou melhoria total de sintomas visuais após educação e introdução de baixas doses de antipsicótico, antidepressivo e benzodiazepínico. Houve regressão dos demais sintomas com alta no décimo segundo dia pós-operatório. Encontrava-se assintomático após 60 dias.

Conclusões: Distúrbios conversivos podem apresentar diversos sinais e sintomas de origem não orgânica, incluindo componente visual. Destaca-se que a ocorrência desse tipo de disfunção visual no pós-operatório de cirurgias espinhais é evento raro e deve ser lembrado como diagnóstico diferencial.

© 2015 Sociedade Brasileira de Anestesiologia. Publicado por Elsevier Editora Ltda. Este é um artigo Open Access sob uma licença CC BY-NC-ND (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Introduction

Patients undergoing spinal surgery may rarely experience postoperative visual loss.¹ The etiological factors related to these lesions have been described mostly as organic, with objective identification of injury through clinical examination and imaging tests.²⁻⁶

However, there are situations in which the identification of an organic cause for the visual loss is not possible.⁷ In these situations, the deficit is described as being "functional". This term is intended to combine the following conditions: conversion disorder (somatoform), factitious and simulation disorder.^{7,8} Compared with factitious and simulation disorders, the conversion disorder symptoms are unintentional. However, often this distinction is difficult, requiring the experienced psychiatric staff expertise.

This report aims to draw attention to the inclusion of visual loss from conversion disorder as rare differential

diagnosis in cases of postoperative visual loss. In such cases, the presence of a high level of suspicion in addition to neuro-ophthalmological examination able to exclude the presence of positive signs for organ damage is critical for early diagnosis and treatment.

Case report

Male patient, 39 years, 71 kg, 1.72 m, presenting with history of radiculopathy and persistent motor deficit in the left lower limb, was admitted for fusion and disectomy at both levels L4-L5 and L5-S1.

In the pre-anesthetic evaluation, he had no comorbidities, allergies or previous surgeries. The patient denied continued use of medication, and laboratory tests were normal. Magnetic resonance imaging (MRI) showed extrusive foraminal herniation to the left at L4-L5 and L5-S1.

After venous puncture with extracath 16G and monitoring with cardioscope (DII and V5), pulse oximetry,

neuromuscular blocking monitor, and noninvasive pressure, induction of anesthesia was performed with remifentanil ($0.5 \text{ mcg} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), lidocaine 2% without vasoconstrictor (2 mL), propofol ($4 \text{ mcg} \cdot \text{mL}^{-1}$), and rocuronium (50 mg). Endotracheal intubation by direct laryngoscopy with 8.0 mm uncuffed tube. Maintained in total intravenous anesthesia with target controlled propofol ($2\text{--}2.5 \text{ mcg} \cdot \text{mL}^{-1}$) and remifentanil ($0.05\text{--}0.5 \text{ mcg} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). Additional monitoring included capnography and catheterization after anesthesia induction.

The patient was placed in the prone position on Wilson's support with face resting on mop without direct compression of the ocular globes, with periodic review of the position throughout the surgery.

During pedicle fixation and laminectomy, a large quantity of bleeding was seen. Fluid replacement was initiated with crystalloids associated with infusion of aminocaproic acid (5 g for 1 h followed by 1 g.h $^{-1}$) until the end of surgery. The patient progressed with hemorrhagic hypovolemic shock (heart rate 150 bpm and blood pressure $60 \times 40 \text{ mmHg}$). With the administration of crystalloids (8500 mL), two units of packed red blood cells, and infusion of noradrenaline ($0.1\text{--}0.3 \text{ mcg} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) there was resolution of symptoms after 30 min. Surgical procedure of 6 h duration and total blood loss estimated at 2000 mL. Satisfactory hemostasis was obtained at the end of surgery. Central venous access and invasive blood pressure were made only after the procedure due to the prone position during the intraoperative period.

The patient was taken to the intensive care unit (ICU) intubated and receiving norepinephrine ($0.1 \text{ mcg} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). In the immediate postoperative period, he required additional fluid replacement with crystalloids (1000 mL), one packed red blood cells, and maintenance of low-dose norepinephrine. Laboratory tests revealed hypokalemia and hypocalcemia, which were properly corrected. At the end of the first postoperative day, the patient was stable, without vasoactive drug support, and was successfully extubated. As part of the strategy for multimodal analgesia, oral pregabalin (75 mg 12/12 h) and intravenous morphine (10 mg 4/4 h) were initiated.

On the second postoperative day, he showed a slight mental confusion and loss of strength in the upper and left lower limbs. We ordered neurological monitoring and a brain MRI, which showed no anatomical changes that could justify the symptoms. We opted for the suspension of pregabalin and morphine for possible association with cognitive impairment.

On the third day after surgery he had persistent mental confusion, motor weakness and mild anterograde amnesia, complaining of complete visual impairment. Ophthalmological examination denied perception of visual stimuli in both eyes, with ectoscopy, duction, convergence, and retinal mapping unchanged; pupils with photomotor reflection unchanged. Brain magnetic resonance angiography and carotid Doppler showed no changes suggestive of organ damage. Monitoring and physical therapy were started to help motor recovery.

The patient was discharged from the ICU at the end of the third postoperative day with laboratory parameters within normal limits and without significant pain complaints (1–2 in the visual numerical scale). On the fourth

postoperative day he evolved with insomnia, agitation, emotional lability (tearful and anxious), hospital discharge desire, language disorder ("humming" and "infantile" speech), motor deficits maintenance, and persistence total visual deficit.

Behavioral inconsistencies were observed, such as inability to resist the passive movement of limbs, but able to stand and walk with assistance, follow people with eyes, and identify colors of objects. Subjected to psychiatric evaluation, which suggested the hypothesis of visual loss from conversion disorder, he started taken haloperidol (0.5 mg 12/12 h) and escitalopram (5 mg.day $^{-1}$) after family and patient counseling, who did not adhere to the psychotherapy indicated by the team as part of multimodal treatment.

The next day, he achieved full recovery of visual acuity, maintaining intermittent episodes of anxiety and psychomotor agitation (specially at night) along the fifth and sixth postoperative day; so, diazepam (5 mg at night) was added.

He remained hospitalized until the twelfth postoperative day, with gradual recovery of other conversion symptoms. At discharge, the symptoms were restricted to only a mild motor deficit and paresthesia of left lower limb restricted to L4–S1 dermatomes, symptoms reported preoperatively. The patient underwent outpatient monitoring with psychiatry, physiotherapy and surgical team. He remained on continuous use of escitalopram (5 mg.day $^{-1}$). After 60 days of outpatient follow-up, he had no visual, cognitive or motor deficit.

Discussion

Visual loss has been described as a postoperative complication of various surgical procedures, usually with limited recovery.^{9,10} However, its occurrence is rare, with reported incidence of up to 0.2% after spinal surgery.⁸

Probably due to the incomplete knowledge of its etiology, is not always possible to unequivocally identify a causative factor.^{11,12} However, the main organic conditions involved in its pathogenesis are: retinal ischemia,^{2,3} anterior and posterior ischemic optic neuropathy,^{4,5} and cortical blindness.⁶ Visual deficit may be unilateral or bilateral, have varying degrees of severity, and affect indiscriminately both sexes. The main risk factors identified are prolonged surgeries, anemia, hypotension, hypoxia, atherosclerosis, fluid overload, and direct ocular compression.^{13,14} Some medications used perioperatively, such as anticonvulsants and opioids, may lead to visual disturbances. However, in such cases, improvement tends to occur with these drugs discontinuation.¹⁵

Although this patient had risk factors for organic visual loss (bleeding with hypotension, prolonged surgery, and large volume replacement),^{13,14} it was not possible to identify tissue injury in his ophthalmological examination, laboratory tests or imaging. It is noteworthy that some of these changes require days to weeks to be expressed. However, certain aspects of this patient's clinical picture (emotional lability, language impairment, motor weakness unrelated to the surgical level, and vision loss with incongruent particularities) called attention to the hypothesis of conversion syndrome.

The conversion syndrome is characterized by neurological symptoms with no correlation with neurological disease, but it causes discomfort or functional damage to the patient.¹⁶ Patients who are young, female, and with low socioeconomic status are the most susceptible; the estimated incidence in the general population is between 4–12/100,000.¹⁷ Depression, anxiety, and interpersonal conflicts frequently worsen the symptoms, which may include non-epileptic seizures, weakness, paralysis, movement disorders, language disorders, cognitive and sensory symptoms.¹⁸

Among the sensory symptoms, visual loss from conversion disorder is relatively common and may include visual blurring, diplopia, nystagmus, visual field defects, and complete visual loss.^{8,18,19} The diagnosis of visual loss from conversion disorder must be corroborated by ophthalmological examination that demonstrates unequivocally normal visual function, in addition to psychiatric evaluation to discard the presence of factitious or simulation disorder for secondary gains,⁸ as occurred in this case.

Postoperative conversion disorder is an unusual event, but has a good prognosis when diagnosed and treated properly. It has been described after a wide variety of surgical procedures, both in adults and children.^{20,21} Although most often related to general anesthesia, it may also occur after regional anesthesia.²² In this patient, it was an important differential diagnosis, because improvement only occurred with specific treatment for conversion syndrome.

The diagnosis requires a high level of suspicion. Several simple tests can be applied to aid in the differentiation of visual loss due to organic or conversion causes.^{18,22} However, in this patient, the application of these clinical trials was waived by the psychiatry, as the history and overall clinical picture of the patient strongly indicated symptoms of conversion disorder.

Observational studies suggest as a first-line treatment of conversion disorders the patient's education regarding his diagnosis, always seeking to create a therapeutic alliance and adding a multidisciplinary team.^{18,23} In addition, cognitive behavioral therapy and motor physical therapy, the latter in the presence of motor deficit, can bring benefits such as second-line treatment when patient's clarification is insufficient.^{24,25} However, it is not unusual that there is resistance to these conservative measures in severe cases; in this situation, it is suggested as a third-line treatment the use of pharmacological agents. Antidepressants are most commonly used, although there are reports²⁶ on the effective use of other classes of drugs, such as antipsychotics, anticonvulsants, and sedatives.²⁷⁻²⁹

Due to the severity of visual symptoms and refusal to adhere to cognitive behavioral therapy, in this case we chose the immediate start of drug combination with antipsychotic and antidepressant in low doses for immediate effect, and maintenance of antidepressant as mid-term therapy.²⁶⁻²⁹ The total recovery of this patient is consistent with the majority of reports in which there was specific diagnosis and treatment for conversion syndrome.

Postoperative visual loss from conversion disorder after spinal surgery is a clinical case not yet reported. This case report intended to alert professionals involved in the perioperative period of this type of surgery for the possibility of such a rare occurrence. High level of suspicion and involvement of a multidisciplinary team (anesthesiology, neurology,

ophthalmology, psychiatry, psychology, physiotherapy, and nursing) are key to early diagnosis and effective treatment of this type of disease, which generally have a good prognosis.

Conflicts of interest

The authors declare no conflicts of interest.

References

- Katz DA, Karlin LI. Visual field defect after posterior spine fusion. *Spine*. 2005;30:E83-5.
- Blauth CJ, Arnold JV, Schulenberg WE, et al. Cerebral microembolism during cardiopulmonary bypass: retinal microvascular studies in vivo with fluorescein angiography. *J Thorac Cardiovasc Surg*. 1988;95:668-76.
- Ozcan MS, Praetel C, Bhatti T, et al. The effect of body inclination during prone positioning on intraocular pressure in awake volunteers: a comparison of two operating tables. *Anesth Analg*. 2004;99:1152-8.
- Johnson LJ, Arnold AC. Incidence of nonarteritic and arteritic anterior ischemic optic neuropathy: a population-based study in the state of Missouri and Los Angeles County, California. *J Clin Neuroophthalmol*. 1994;14:38-44.
- Schobel GA, Schmidbauer M, Millesi W, et al. Posterior ischemic optic neuropathy following bilateral radical neck dissection. *Int J Oral Maxillofac Surg*. 1995;24:283-7.
- Newman MF, Mathew JP, Grocott HP, et al. Central nervous system injury associated with cardiac surgery. *Lancet*. 2006;368:694-703.
- Bruce BB, Newman NJ. Functional visual loss. *Neurol Clin*. 2010;28:789-802.
- Berg KT, Harrison AR, Lee MS. Perioperative visual loss in ocular and nonocular surgery. *Clin Ophthalmol*. 2010;4:531-46.
- Roth S, Thisted RA, Erickson JP, et al. Eye injuries after non ocular surgery; a study of 60,965 anesthetics from 1988 to 1992. *Anesthesiology*. 1996;85:1020-7.
- Shmygalev S, Heller AR. Perioperative visual loss after nonocular surgery. *Anaesthetist*. 2011;60:683-94.
- Kawaguchi M, Hayashi H, Kurita N, et al. Postoperative visual disturbances after nonophthalmic surgery. *Masui*. 2009;58:952-61.
- Grover V, Jangra K. Perioperative vision loss: a complication to watch out. *J Anaesthesiol Clin Pharmacol*. 2012;28:11-6.
- Lee L, Roth S, Todd M, et al. Risk factor associated with ischemic optic neuropathy after spinal fusion surgery. *Anesthesiology*. 2012;116:15-24.
- Apfelbaum JL, Roth S, Connis RT, et al. Practice advisory for perioperative visual loss associated with spine surgery: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Visual Loss. *Anesthesiology*. 2012;116:274-85.
- Toth C. Pregabalin: latest safety evidence and clinical implications for the management of neuropathic pain. *Ther Adv Drug Saf*. 2014;5:38-56.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub; 2013.
- Carson A, Stone J, Hibberd C, et al. Disability, distress, and unemployment in neurology outpatients with symptoms "unexplained by organic disease". *J Neurol Neurosurg Psychiatry*. 2011;82:810-3.
- Stone J, Carson A, Sharpe M. Functional symptoms and signs in neurology: assessment and diagnosis. *J Neurol Neurosurg Psychiatry*. 2005;76:i2-12.

19. Sharpe M, Walker J, Williams C, et al. Guided self-help for functional (psychogenic) symptoms: a randomized controlled efficacy trial. *Neurology*. 2011;77:564–72.
20. Maddock H, Carley S, McCluskey A. An unusual case of hysterical postoperative coma. *Anesthesia*. 1999;54:717–8.
21. Judge A, Spielman F. Postoperative conversion disorder in a pediatric patient. *Paediatr Anaesth*. 2010;20:1052–4.
22. Chen CS, Lee AW, Karagiannis A, et al. Practical clinical approaches to functional visual loss. *J Clin Neurosci*. 2007;14:1–7.
23. Aybek S, Hubschmid M, Mossinger C, et al. Early intervention for conversion disorder: neurologists and psychiatrists working together. *Acta Neuropsychiatr*. 2013;25:52–6.
24. Goldstein LH, Chalder T, Chigwedere C, et al. Cognitive-behavioral therapy for psychogenic nonepileptic seizures: a pilot RCT. *Neurology*. 2010;74:1986–94.
25. Czarnecki K, Thompson JM, Seime R, et al. Functional movement disorders: successful treatment with a physical therapy rehabilitation protocol. *Parkinsonism Relat Disord*. 2012;18:247–51.
26. Voon V, Lang AE. Antidepressant treatment outcomes of psychogenic movement disorder. *J Clin Psychiatry*. 2005;66:1529–34.
27. Oulis P, Kokras N, Papadimitriou GN, et al. Adjunctive low-dose amisulpride in motor conversion disorder. *Clin Neuropharmacol*. 2009;32:342–3.
28. Messina A, Fogliani AM. Valproate in conversion disorder: a case report. *Case Rep Med*. 2010;2010:1–3.
29. Stone J, Hoeritzauer I, Brown K, et al. Therapeutic sedation for functional (psychogenic) neurological symptoms. *J Psychosom Res*. 2014;76:165–8.