



REVISTA BRASILEIRA DE ANESTESIOLOGIA

Publicação Oficial da Sociedade Brasileira de Anestesiologia
www.sba.com.br



CLINICAL INFORMATION

Cardiac arrest after epidural anesthesia for a esthetic plastic surgery: a case report



Larissa Cardoso Pinheiro^{a,*}, Bruno Mendes Carmona^a,
Mário de Nazareth Chaves Fascio^a, Iris Santos de Souza^a,
Rui Antonio Aquino de Azevedo^a, Fabiano Timbó Barbosa^b

^a Centro de Ensino e Treinamento do Serviço de Anestesia do Ophir Loyola, Belém, PA, Brazil

^b Universidade Federal de Alagoas (UFAL), Maceió, AL, Brazil

Received 3 January 2015; accepted 23 March 2015

Available online 15 September 2016

KEYWORDS

Cardiac arrest;
Epidural anesthesia;
Resuscitation after
spinal anesthesia

Abstract Cardiac arrest during neuraxial anesthesia is a serious adverse event, which may lead to significant neurological damage and death if not treated promptly. The associated mechanisms are neglected respiratory failure, extensive sympathetic block, local anaesthetic toxicity, total spinal block, in addition to the growing awareness of the vagal predominance as a predisposing factor. In the case reported, the patient was 25 years old, ASA I, scheduled for a esthetic lipoplasty. After sedation with midazolam and fentanyl, epidural anesthesia in interspaces T12-L1 and T2-T3 and catheter insertion into inferior puncture were performed. The patient remained in the supine position for 10 min. Then, she was placed in the prone position, developing asystolic cardiac arrest 20 min after the completion of neuraxial blockade. The medical team immediately placed the patient in the supine position and began cardiopulmonary resuscitation. Spontaneous circulation was achieved after twenty minutes of resuscitation. We discuss in this report the exacerbated vagal response as the main event mechanism. The patient's successful outcome emphasizes the importance of anaesthetic monitoring by anesthesiologists, prompt recognition and treatment of rhythm changes on the electrocardiogram.

© 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

Parada cardíaca;
Anestesia peridural;
Reanimação após
peridural

Parada cardíaca após peridural para cirurgia plástica estética: relato de caso

Resumo A parada cardíaca durante anestesia neuroaxial é um evento adverso grave, que pode ocasionar sequelas neurológicas importantes e morte se não tratada em tempo hábil. Os mecanismos associados são insuficiência respiratória negligenciada, bloqueio simpático extenso, toxicidade por anestésicos locais, raquianestesia total, além da crescente consciência da predominância vagal como fator predisponente. No caso reportado, a paciente tinha 25 anos

* Corresponding author.

E-mail: lari.cardoso@hotmail.com (L.C. Pinheiro).

e estado físico ASA I e foi programada para lipoplastia estética. Após sedação com midazolam e fentanil, foi feita anestesia peridural nos espaços T12-L1 e T2-T3 e inserção de cateter na punção inferior. A paciente foi mantida em decúbito dorsal horizontal durante 10 minutos. Em seguida, foi posicionada em decúbito ventral, evoluiu com parada cardíaca em assistolia 20 minutos após o bloqueio do neuroeixo. A equipe médica imediatamente colocou a paciente em decúbito dorsal e iniciou as manobras de ressuscitação cardiopulmonar. O retorno da circulação espontânea foi obtido após 20 minutos de reanimação. É discutida neste relato a resposta vagal exacerbada como principal mecanismo causal do evento. O sucesso do desfecho da paciente em questão ressalta a importância da vigilância do anestesiológico, e do pronto reconhecimento e tratamento de mudanças de ritmo no eletrocardiograma.

© 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

Thoracic epidural anesthesia for cosmetic breast surgery, or combined breast and abdomen, provides satisfactory results both peri- and postoperatively. The use of a small number of drugs, early awakening, amnesia, and the possibility of hospital discharge within 24 hours make thoracic epidural anesthesia an excellent technique for this type of surgery, with a higher percentage of survival compared to general anesthesia.¹ This technique showed a reduction in postoperative stress and on systemic sympathetic response, with consequent reduction of adverse cardiac events.^{2,3} The incidence of cardiopulmonary arrest (CPA) during neuraxial blockade is associated with good outcomes.^{4,5} Although several factors may lead to CPA during epidural anesthesia, increasing evidence suggests the vagal predominance as a common mechanism.⁶ In this report we discuss a case of cardiac arrest associated with exacerbated vagotonic response.

Case report

Female patient, 25 years old, ASA I, scheduled for a esthetic lipoplasty. Preanaesthetic evaluation was performed in office, when she received the proper orientation and gave written informed consent. In the operating room, the patient was monitored with cardioscope, noninvasive blood pressure, and pulse oximetry. After establishing venous access in the left arm with a 20G catheter, midazolam 4 mg and fentanyl 50 mcg were used for sedation.

Double epidural puncture was performed in the T12-L1 and T2-T3 interspaces with a 18G Tuohy needle with an insertion of a 18G epidural catheter in the inferior puncture, uneventfully. In the T12-L1 interspace, a local anaesthetic bupivacaine S75:R25 (*simocaina*) 0.5% with vasoconstrictor (14 mL), morphine 2 mg, fentanyl 50 mcg, and distilled water (3 mL) were administered. In the T2-T3 interspace, a local anaesthetic bupivacaine S75:R25 (*simocaina*) 0.5% with vasoconstrictor (8 mL), fentanyl 50 mcg, and distilled water (2 mL) were administered. There were no complications during the procedures or insertion of catheters.

Subsequently, the patient remained in the supine position for 10 min, at which time a slight decrease in oxygen peripheral saturation from 98% to 92% was observed, which was

reversed with two deep breaths instructed by the attending physician. Then the patient was placed in the prone position, and she even helped herself to move.

About five minutes in the prone position, the patient had a new episode of desaturation (92%), reversed at the request of the anesthesiologist. However, after five more minutes the patient developed cardiac arrest in asystolia noticed by the attended anesthesiologist, who immediately arranged for the patient's repositioning in the supine position and began the cardiopulmonary resuscitation maneuvers recommended by the Advanced Cardiac Life Support (ACLS), with high-quality chest compressions, vasopressor administration, and airway control in a timely manner.

After about 20 min of cardiopulmonary resuscitation, the patient regained spontaneous circulation, and vasoactive drugs were required to ensure hemodynamic stability. In a joint decision, the surgical team and the anesthesiologist opted for the cancelation of the surgical procedure, and the patient was taken to the ICU with the following parameters: HR 120 bpm, sinus rhythm, BP 120 × 70 mmHg, SpO₂ 98%, EtCO₂ 30 mmHg, receiving vasoactive drugs (noradrenaline), sedated (midazolam), and with miotic pupils.

In the ICU, the patient developed oliguria and pulmonary edema, treated with improvement of mechanical ventilation parameters (high PEEP and alveolar recruitment maneuvers) and diuretic. Approximately 24 hours after admission to the ICU, the patient was extubated successfully and maintained the hemodynamic parameters up to a discharge from the ICU to the ward the next day.

Discussion

The frequency, predisposing factors, and outcomes associated with cardiac arrest during neuraxial anesthesia remain undefined.⁵ Auroy et al. found that cardiac arrest during neuraxial anesthesia is associated with good neurological outcomes.⁴

The Neuraxial blockade can reduce perioperative mortality compared to general anesthesia, especially in patients undergoing surgery of moderate to high cardiac risk.⁷ Particularly, the use of thoracic epidural anesthesia could reduce the incidence of perioperative myocardial infarction.⁸

Thoracic epidural anesthesia is an attractive approach to cosmetic surgery that became popular in Brazil.⁹ We opted

for the dual-epidural catheterization puncture due to the need for extensive surgical field coverage, reaching a wide range of dermatomes, in addition to the more uniform distribution of the anaesthetic mass through the two punctures and the possibility of anaesthetic supplementation.¹⁰

Cardiopulmonary arrest (CPA) in epidural anesthesia may be related to the following factors: accidental subarachnoid administration, extensive sympathetic block, myocardial ischemia, respiratory depression secondary to sedation, anaphylactic shock, local anaesthetic poisoning. Conditions not justified by these reasons can be explained by vagal predominance.¹¹

Local anaesthetic poisoning is a likely mechanism of CPA after epidural anesthesia. However, negative aspiration of blood in the syringe were made during the procedure, as well as anaesthetic test dose administration with vasoconstrictor, and there was no change in heart rate, QRS morphology, rhythm, or complexes.

Moreover, resuscitation after circulatory collapse induced by local anaesthetic is described as difficult, prolonged, and refractory to the approaches set out in the ACLS.¹² Increasing evidences support the efficacy of lipid emulsion for use in this context; its use is released by the American Society of Regional Anesthesia after airway management and seizure control.¹³ Literature reports cases in which the rapid institution of extracorporeal membrane oxygenation was related to the return of spontaneous circulation in patients with bupivacaine cardiotoxicity.¹⁴

Intrathecal block is an ever-present risk when large doses of local anaesthetic is accidentally administered into the spinal catheter when it is assumed to be in the epidural space. This possibility was not considered as a possible cause of the cardiac arrest because the anaesthetic administration was uneventful, as it would be expected immediate apnea, loss of consciousness, paralysis, and mydriasis if there were accidental subarachnoid blockade after dural puncture.¹⁵ Although the literature reports this classical presentation, it is present in less than half of the confirmed cases of inadvertent subdural injection, through studies with epidurography.¹⁶

Vasovagal exacerbated response is characterized by an inappropriate combination of bradycardia and paradoxical vasodilation.¹⁷ Studies reinforce that bradycardia and hypotension are the most frequent complications after neuraxial blockade, justified by the blockade of cardioaccelerator fibers (T1-T4), sympathetic stimulation blockade with reduction of inotropic and heart chronotropism, in addition to the parasympathetic cardiac protective reflex (Bezold-Jarisch reflex) triggered by a decrease in venous return and peripheral vascular tone.¹⁸⁻²⁰

Although bradycardia and hypotension are frequent and well tolerated events after epidural anesthesia, immediate treatment is required, considering bradycardia description as an indicator of imminent cardiovascular collapse.^{20,21} Brown et al. reported sudden severe bradycardia and asystole, even a sudden loss of consciousness during patient chat with the anesthesiologist.^{22,23}

Male gender, beta-blockers, ASA physical status I, sensory level above T6, and under-50s age group are factors related to inappropriate vagotonic response.⁶ Besides these, severe pain, anxiety, fear and emotional stress may act as triggers for vagotonic response.^{24,25}

In this case, the patient had three of the aforementioned characteristics, which along with CPA, emphasizes the possibility of vagal predominance as the main causative mechanism. Furthermore, other factors such as sedation, hypoxemia, use of opioids, and positioning change may have contributed.^{16,17}

Jang and Caplan, in different studies, found similar cases of sudden onset of severe bradycardia and heart failure in patients who were hemodynamically stable and well oxygenated.^{11,26}

After the finding of CPA, cardiopulmonary resuscitation maneuvers were initiated and adrenaline was the only drug used. CPA during epidural anesthesia is considered difficult because it decreases coronary perfusion pressure.²⁷ The optimum resuscitation vasopressor should increase coronary perfusion pressure and diastolic pressure in the aortic root, improve coronary and brain blood flow without increasing cellular oxygen demand.²⁸

Adrenaline has been the leading drug therapy in cases of cardiac arrest, although associated with increased myocardium oxygen demand, arrhythmogenic potential, and hypertension after resuscitation.²⁹ While vasopressin offers a theoretical advantage over adrenaline for not increasing the oxygen consumption. However, unlike adrenaline, it has no stimulating effect on the heart.²⁸

Studies performed in Europe found better outcome with vasopressin in cardiac arrest patients with asystole.³⁰ However, regarding the mechanisms of cardiac arrest related to epidural anesthesia, studies are scarce and the effectiveness of adrenaline and vasopressin in this context is unknown.²⁷

Immediate recognition of CPA was essential for the spontaneous circulation return and good neurological outcome of this patient, it stressed the need for anesthesiologist's constant vigilance, the recognition of the pace changing in electrocardiogram, and immediate treatment as pillars for the success of anesthesia.

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Di Lascio JVL, Victoria LGF, Bello CN. Anestesia peridural torácica para cirurgias estéticas de mama ou mama e abdômen. *Rev Bras Anest.* 1988;38:273-6.
2. Freise H, Van Aken HK. Risks and benefits of thoracic epidural anaesthesia. *Br J Anesth.* 2011;107:859-68.
3. Dragana US, Srdjan B, Miomir J. Benefits, risks and complications of perioperative use of epidural anesthesia. *Med Arh.* 2012;66:340-3.
4. Auroy Y, Narchi P, Messiah A, et al. Serious complications related to regional anesthesia. *Anesthesiology.* 1997;87:479-86.
5. Kopp SL, Horlocker TT, Warner ME, et al. Cardiac arrest during neuraxial anesthesia: frequency and predisposing factors associated with survival. *Anesth Analg.* 2005;100:855-65.
6. Pollard JB. Common mechanisms and strategies for prevention and treatment of cardiac arrest during epidural anesthesia. *J Clin Anesth.* 2002;14:52-6.
7. Guay J, Choi P, Suresh S, et al. Neuraxial blockade for the prevention of postoperative mortality and major morbidity: an

- overview of Cochrane systematic reviews. *Cochrane Database Syst Rev.* 2014;25:CD010108.
8. Stenger M, Fabrin A, Schmidt H, et al. High thoracic epidural analgesia as an adjunct to general anesthesia is associated with better outcome in low-to-moderate risk cardiac surgery patients. *J Cardiothorac Vasc Anesth.* 2013;27:1301–9.
 9. Sperhacke D, Geier KO, Eschiletti JCC. Peridural torácica alta associada ou não à peridural torácica baixa em pacientes ambulatoriais: implicações clínicas. *Rev Bras Anest.* 2004;54:479–90.
 10. Leão DG. Peridural torácica: estudo retrospectivo de 1.240 casos. *Rev Bras Anest.* 1997;47:138–47.
 11. Caplan RA, Ward RJ, Posner K, et al. Unexpected cardiac arrest during spinal anesthesia: a closed claims analysis of predisposing factors. *Anesthesiology.* 1988;68:5–11.
 12. Mercado P, Weinberg GL. Local anesthetic systemic toxicity: prevention and treatment. *Anesthesiol Clin.* 2011;29:233–42.
 13. Neal JM, Bernardis CM, Butterworth JF, et al. ASRA practice advisory on local anesthetic systemic toxicity. *Reg Anesth Pain Med.* 2010;35:152–61.
 14. Weinberg GL. Treatment of local anesthetic systemic toxicity (LAST). *Reg Anesth Pain Med.* 2010;35:2.
 15. Pages-Arroyo E, Pian-Smith MCM. Neurologic complications of neuraxial anesthesia. *Anesthesiol Clin.* 2013;31:571–94.
 16. Hoftman N. Unintentional subdural injection: a complication of neuraxial anesthesia/analgesia. *Anesthesiol Clin.* 2011;29:279–90.
 17. Jang YE, Do SH, Song IA. Vasovagal cardiac arrest during spinal anesthesia for Cesarean section – a case report. *Korean J Anesthesiol.* 2013;64:77–81.
 18. Pereira IDF, Grando MM, Vianna PTG. Análise retrospectiva de fatores de risco e preditores de complicações intraoperatórias dos bloqueios do neuroeixo realizados na Faculdade de Medicina de Botucatu (Unesp). *Rev Bras Anestesiologia.* 2011;61:568–81.
 19. Goertz AW, Seeling W, Heinrich H, et al. Influence of high thoracic epidural anesthesia on left ventricular contractility assessed using the end systolic pressure length relationship. *Acta Anaesthesiol Scand.* 1993;37:38–44.
 20. Imai K, Kayashima K, Yoshino H, et al. Four cases of cardiac arrest during spinal anesthesia. *Masui.* 2012;61:1091–4.
 21. Okutani H, Okutani R, Nakamura T. Asystole developed during total gastrectomy under general anesthesia combined with thoracic epidural anesthesia. *Osaka City Med J.* 2012;58:83–6.
 22. Brown DL, Carpenter RL, Moore DL, et al. Cardiac arrest during spinal anesthesia. *Anesthesiology.* 1988;68:971–2.
 23. Geffin B, Sharpiro L. Sinus brady cardia and asystole during spinal and epidural anesthesia: a report of 13 cases. *J Clin Anesth.* 1998;10:278–85.
 24. Hart PS, Yanny W. Needlephobia and malignant vasovagal syndrome. *Anaesthesia.* 1998;53:1002–4.
 25. Sprung J, Abdelmalak B, Schoenwald PK. Vasovagal cardiac arrest during the insertion of an epidural catheter and before the administration of epidural medication. *Anesth Analg.* 1998;86:1263–5.
 26. Jang YE, Do SH, Song IA. Vasovagal cardiac arrest during spinal anesthesia for Cesarean section: a case report. *Korean J Anesth.* 2013;64:77–81.
 27. Krismer AC, Hogan QH, Wenzel V, et al. The efficacy of epinephrine or vasopressin for resuscitation during epidural anesthesia. *Anesth Analg.* 2001;93:734–42.
 28. Ornato JP. Optimal vasopressor drug therapy during resuscitation. *Crit Care.* 2008;12:123.
 29. Holt NF, Haspel KL. Vasopressin: a review of therapeutic applications. *J Cardiothorac Vasc Anesth.* 2010;24:330–47.
 30. Wenzel V, Krismer AC, Arntz HR, et al. European resuscitation council vasopressor during cardiopulmonary resuscitation study group: a comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation. *N Engl J Med.* 2004;350:105–13.