Rev Bras Anestesiol. 2016;66(3):321–323

Sociedade Brasileira de Anestesiologia

REVISTA BRASILEIRA DE ANESTESIOLOGIA

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

CLINICAL INFORMATION

Aphonia after shoulder surgery: case report

Carlos Alberto da Silva Soares Moreno*, **Sara Fonseca**

Hospital São João, Porto, Portugal

Received 8 August 2013; accepted 19 September 2013
Available online 23 November 2014

KEYWORDS
Block;
Interscalene brachial plexus;
Aphonia;
I-Gel;
Choking;
Recurrent laryngeal nerve

Abstract In this case report we highlight the uniqueness of aphonia as, to the best of our knowledge, cases of aphonia related to interscalene brachial plexus block (IBPB) are not described in the literature. Although hoarseness is a common complication of IBPB, aphonia is not. Therefore, we think it is important to publicize the first case of aphonia after IBPB, which may have arisen only because of a recurrent laryngeal nerve chronic injury contralateral to the IBPB site.

PALAVRAS-CHAVE
Bloqueio;
Plexo braquial interescalênico;
Afonia;
I-Gel;
Engasgamento;
Nervo laríngeo recorrente

Afonia após cirurgia do ombro: relato de caso

Resumo Relativamente a este relato de caso destacamos a sua singularidade, uma vez que não se encontram descritos na literatura, tanto ou quanto os autores puderam investigar, casos de afonia após uma anestesia combinada com bloqueio do plexo braquial via interescalénica (BPBI). Embora a rouquidão seja uma complicação frequente do BPBI, a afonia não o é. Desse modo, pensamos ser importante dar a conhecer o primeiro caso de afonia após o BPBI, que na opinião dos autores surgiu apenas por causa de uma lesão crônica do nervo laríngeo recorrente contralateral ao local do BPBI.

Background and objective

Shoulder surgery is the main indication of the interscalene brachial plexus block (ISBPB).[1] Although safe and effective, this technique is not free of complications.[2–4]

Currently, there is a growing interest in the use of ultrasound guidance for regional anesthesia procedures such as

* Corresponding author.
E-mail: carlosmoreno_10@msn.com (C.A.S.S. Moreno).

http://dx.doi.org/10.1016/j.bjane.2013.09.014

peripheral nerve blocks (PNB), but its superiority to neurostimulation is still controversial.[5] Recent studies suggest that ultrasound can improve the effectiveness of peripheral nerve block compared with neurostimulation. However, there is no evidence that its use can decrease the number of complication, such as nerve injury or systemic toxicity of local anesthetics.[5,6] The Concomitant use of ultrasound and neurostimulation is a common, reliable, and valid practice for PNB.

Since the 1990s, the supraglottic airway devices are often used for airway management in patients undergoing general anesthesia. It is a safe device, although some complications associated with its use have been described.[7]

The aim of this study was to analyze the first case of aphonia described in literature, which emerged after combined anesthesia (ISBPB and balanced general anesthesia [BGA]) in shoulder arthroscopy to correct rotator cuff syndrome.

Case report

Female patient, 52 years old, 65 kg; scheduled for right shoulder arthroscopic rotator cuff repair. Personal history of controlled hypertension medicated with losartan 50 mg day^{-1}, physical status ASA II (American Society of Anesthesiologists, ASA classification); no surgical history and/or relevant anesthetics. Physical examination and preoperative tests (blood count, biochemistry, blood clotting tests, and electrocardiogram) showed no changes.

Combined anesthesia was proposed: ISBPB under mild sedation and BGA. After obtaining informed consent, standard monitoring (electrocardiogram, pulse oximetry, noninvasive blood pressure) and supplemental oxygen (3 L min^{-1}) via nasal cannula, sedation with IV midazolam (2 mg) and ISBPB were performed: Echoplex® Vygon needle 50 mm 22G guided by ultrasound (Sonosite M-Turbo®) and neurostimulation (Plexygon® 7501.31, Vygon). After ultrasound identification, and through brachial plexus neurostimulation (forearm muscle contraction, motor response up to 0.36 mA, pulse duration of 0.1 ms, and 2 Hz of frequency), infiltration was performed with ropivacaine 0.5% (30 mL), under visualization. Sensory block level 2 and MBS 1 (Modified Bromage Scale) were obtained after 20 min. BGA was then performed: IV fentanyl 2 $\mu g\,kg^{-1}$ and IV propofol 2.5 mg kg^{-1}; the supraglottic device I-Gel 4 introduction was uneventful; maintenance performed with desflurane 6% in oxygen/air (40%/60%). Concomitant analgesia performed with IV paracetamol 1 g and IV parecoxib 40 mg. Surgery lasted 80 min. There were no anesthetic or surgical complications. Emergence from anesthesia and removal of I-Gel 4 were uneventful. Transport to post-anesthesia care unit (PACU) was performed with the patient spontaneously breathing, without respiratory distress.

In the PACU, with the patient fully awake, inability to make sounds associated with ptosis, miosis, and enophthalmos were observed. Neurological examination revealed no other changes, and there were no hemodynamic or respiratory changes too. The initial reversal of symptoms occurred two hours after arrival in the PACU, but the hoarseness persisted. The patient was transferred to the orthopedic ward seven hours after ISBPB, with MBS 0 and hoarseness. On the first postoperative day (POD 1), the patient was evaluated by the Acute Pain Functional Unit (APFU), already showing complete clinical reversal of neurological disorders. During this consultation, a review of the clinical conditions presented in the first hours after surgery was performed, with confirmation of no sound transmission/vocalization and any other associated neurological symptoms. The patient was discharged on POD 2.

The follow-up visit was conducted 30 days after discharge in the APFU, noting the absence of neurological abnormalities and appropriate surgical recovery. A more detailed exploration of the patient's history of frequent choking with saliva for more than 10 years, which was associated with intermittent hoarseness was performed.

Discussion

To the best of our knowledge, there is not a case similar to ours described in the literature. Aphonia (complete inability to utter sounds) occurs after bilateral palsy in abduction of vocal folds. The ipsilateral recurrent laryngeal nerve (RLN) is responsible for the motor innervation of the vocal fold muscles. Unilateral RLN injury paralyzes the ipsilateral vocal fold, and the clinical symptoms of dysphonia or hoarseness appear.[8] If the RLN paralysis or injury is bilateral, both vocal folds are paralyzed and aphonia develops.

A surgical cause seems less likely, given the type and location of the surgery. Among other possible causes, we highlight the bilateral blockade of the RLN with ropivacaine, ipsilateral blockade of the RLN in the presence of a prior contralateral RLN injury, larynx region trauma/injury caused by the supraglottic device I-Gel 4, and intraoperative neurological event.

The neurological examination performed in the PACU after the diagnosis of aphonia did not reveal any other changes, only upper limb motor block on the ISBPB side. We emphasize the preservation of the mimic and face muscles, equally photoreactive and symmetric pupils, which makes the occurrence of an intraoperative neurologic event unlikely.

Regarding the use of I-Gel, the most frequently described complications are oropharyngeal pain/inflammation and dysphagia.[9] Rene et al. reported a case of nerve injury with its introduction. The main injured nerve is the lingual, which is only responsible for the tongue sensory innervation.[10] In literature, there are no cases reporting aphonia associated with the use of this type of device. However, with the use of cuffed laryngeal mask, there are case reports of transient hoarseness due to RLN paralysis. Jones et al. justified hoarseness by cuff hyperinflation, with temporary compression of the RLN and vocal folds.[11] Inomata et al. justified transient hoarseness by vocal cord trauma after the LMA cuff hyperinflation, but they also suggested the possibility of vocal folds injury caused by the laryngoscopy performed prior to the respective LMA placement.[12]

We think that aphonia may have been caused by ISBPB. Although the ISBPB on the right is not sufficient to cause hoarseness, as it can only condition the ipsilateral RLN block, with the onset of hoarseness and dysphonia (not aphonia), one might think that there was a local anesthetic leakage to the opposite side of the neck with bilateral blockade of NLR. However, the total volume of local anesthetic used and the

anatomical structures that the local anesthetic would have to cross to reach the contralateral RLN make this hypothesis unlikely. We found no reports in the literature about local anesthetic leakage after contralateral ISBPB.

The clinical history of facility to choke and frequent intermittent hoarseness for more than ten years, which never bothered the patient, suggests the presence of unilateral paralysis of a vocal fold, which prevents the effective airway protective reflexes and enables the development of frequent intermittent hoarseness. The reversal of ISBPB began approximately two hours after the predicted time for motor nerve fibers blockade and for the beginning of aphonia reversal. These data allow aphonia justification as a result of a right ISBPB in a patient with previous chronic injury of left RLN. The absence of respiratory difficulty raises the possibility that the vocal cord paralysis has occurred in abduction, and not in adduction, which allowed spontaneous ventilation.

We consider that the most likely cause for the transient aphonia in our patient was the RLN temporary blockade by ropivacaine, in the presence of a unilateral prior injury of contralateral RLN.

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Winnie AP. Interscalene brachial plexus block. Anesth Analg. 1970;49:455–66.
2. Urmey W, McDonald M. Hemidiaphragmatic paresis during interscalene brachial plexus block: effects of pulmonary function and chest wall mechanics. Anesth Analg. 1992;74:352.
3. Fujimura N, Namba H, Tsunoda K, et al. Effect of hemidiaphragmatic paresis caused by interscalene brachial plexus block on breathing pattern, chest wall mechanics and blood gases. Anesth Analg. 1995;81:962.
4. Joseph L, Seltzer M. Hoarseness and Horner's syndrome after interscalene brachial plexus block. Anesth Analg. 1976; 56:587.
5. Abrahams M, Aziz MF, Fu RF, et al. Ultrasound guidance compared with electrical neurostimulation for peripheral nerve block: a systematic review and meta-analysis of randomized controlled trials. Br J Anaesth. 2009;102:408–17.
6. Hebl J. Ultrasound-guided regional anesthesia and the prevention of neurologic injury: fact or fiction? Anesthesiology. 2008;108:186–8.
7. Gatward JJ, Cook TM, Seller C. Evaluation of the size 4 i-gel airway in one hundred non-paralysed patients. Anaesthesia. 2008;63:1124–30.
8. Crumley R. Unilateral recurrent laryngeal nerve paralysis. J Voice. 1994;8:79–83.
9. Amini S, Khoshfetrat M. Comparison of the intersurgical solus laryngeal mask and the i-gel supralaryngeal device. Anaesthesia. 2010;65:805–9.
10. Renes SH, Zwart R, Scheffer GJ. Lingual nerve injury following the use of an i-gel laryngeal mask. Anaesthesia. 2011;66:220–31.
11. Jones L, Hegab A. Recurrent laryngeal nerve palsy after laryngeal mask airway insertion. Anaesthesia. 1996;51:171–2.
12. Inomata S, Nishikawa T, Suga A, et al. Transient bilateral vocal cord paralysis after insertion of laryngeal mask airway. Anesthesiology. 1995;82:787–8.