

Opioid - Free Anaesthesia for Microvascular Decompression of Trigeminal Nerve: A Case Report

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Abstract

Trigeminal neuralgia (TN) causes a neuropathic pain in the territory of the distribution of the trigeminal nerve that can become a disabling factor for daily life. Sometimes it is refractory to medical and interventional treatment and may require surgery. We present the case of a 67-year-old man with TN of 10 years of evolution scheduled for microvascular decompression under an opioid - free anaesthesia (OFA) technique.

Keywords: Opioid - Free Anaesthesia; Trigeminal Neuralgia; Neuropathic Pain; Ketamine; Magnesium Sulphate

Introduction

The annual incidence of TN is about 4 to 5 in 100,000. In many patients with TN, the pharmacological treatment (carbamazepine - of choice - and oxcarbazepine more frequently but also other drugs such as lamotrigine and even opiates) and the interventional techniques (radiofrequency of the Gasserian ganglion) are sometimes ineffective, requiring surgery (microvascular decompression) for the relief of their symptoms. On the other hand, OFA techniques associated with locoregional blocks are indicated in patients with certain chronic pain syndromes and in chronic opioid consumers or addicted who are candidates for surgical treatment as is the case of the patient in question.

Case Report

We present the case of a 67 year-old man, 75 kg of weight and 170 cm of height with a history of allergy to quinolones, arterial hypertension, dyslipidemia and 10-year history of TN in dermatomas corresponding to V1 and V2 that was causing daily pain and inability to perform activities of daily living. He had undergone 3 ineffective radiofrequencies of the Gasser ganglion and was on following medications- Atorvastatin, Lercanidipine, Lorazepam, Lacosamide, Carbamazepine, Lamotrigine, Fentanyl patches, Amitriptyline and combination of Oxycodone -Naloxone.

A nuclear magnetic resonance (NMR) showed compression in the cisternal path of the trigeminal nerve caused by the right superior cerebellar artery. He was scheduled for microvascular decompression by right retrosigmoid craniotomy. The patient was classified as ASA-II anaesthetic status.

Upon his arrival at the operating room, he was premedicated with Omeprazole 40 mg, Midazolam 3 mg, Dexamethasone 16 mg and a pre-incisional bolus of 3g of Magnesium Sulfate (40 mg/kg) followed by 10 mg/kg/h (total dose: 4500 mg). The antibiotic prophylaxis consisted of Cefazolin 2 gr in anesthetic induction plus 1 gr at 3 hours after the first dose.

Fluid therapy was based on the administration of 1 litre of 0.9% SS. The intraoperative monitoring consisted of ECG (DII, V5), invasive and non-invasive TA measurement, SpO₂, capnography, anesthetic depth (SedLine Brain Function Monitoring), diuresis and temperature. A plantar venous pump was placed. He was maintained under mechanical ventilation with a mixture of oxygen/air (40/60) % in SIMV/AUTOFLOW (synchronized intermittent mandatory ventilation/AUTOFLOW) mode.

General anesthesia was induced with 200 mg of Propofol and 35 mg of Ketamine. Neuromuscular block was established with 75 mg of Rocuronium. The orotracheal intubation was uneventful. A preincisional scalp block of peripheral cranial nerves (Frontal, Auriculotemporal and Occipital major and minor) was performed with 240 mg of Ropivacaine.

The maintenance was carried out with propofol in perfusion guided by monitoring of anesthetic depth (Sedline), Rocuronium in bolus (20 mg/20 minutes) and 150 mcg/kg x h of ketamine. Infusion of Esmolol (100 - 10 mcg/kg x min) was administered with Nicardipine bolus (200 - 300 mcg) for hemodynamic control of TA.

Approximately 45 minutes before the end of the operation, acetaminophen 1g, ketorolac 30 mg and haloperidol 1.5 mg were administered. At closure, the wound was infiltrated with 0.25% Bupivacaine with adrenaline 1/200000.

At the end of the surgery, sugammadex (400 mg) was administered and the patient was extubated in the operating room without complications, hemodynamically and ventilatory stable, without neurological focus (Glasgow Coma Score = 15) and without pain (VAS = 0). The intraoperative diuresis was approximately 1500 ml. He was transferred to the Postoperative Intensive Care Unit (PICU) for immediate postoperative management, which was uneventful. He could be discharged to the hospitalization plant the day after admission at the PICU.

Note that during his stay in PICU, the patient only required analgesia with acetaminophen 1 gr/8h and Dexketoprofen 50 mg/12h. in addition to his usual treatment with Carbamazepine and Lacosamide (Amitriptyline was not given because of not having the parenteral formula) without needing opioids. The patient was discharged home 10 days after the intervention, pain - free and without opioid drugs prescribed (only with Lacosamide, Carbamazepine, Lamotrigine and Amitriptyline).

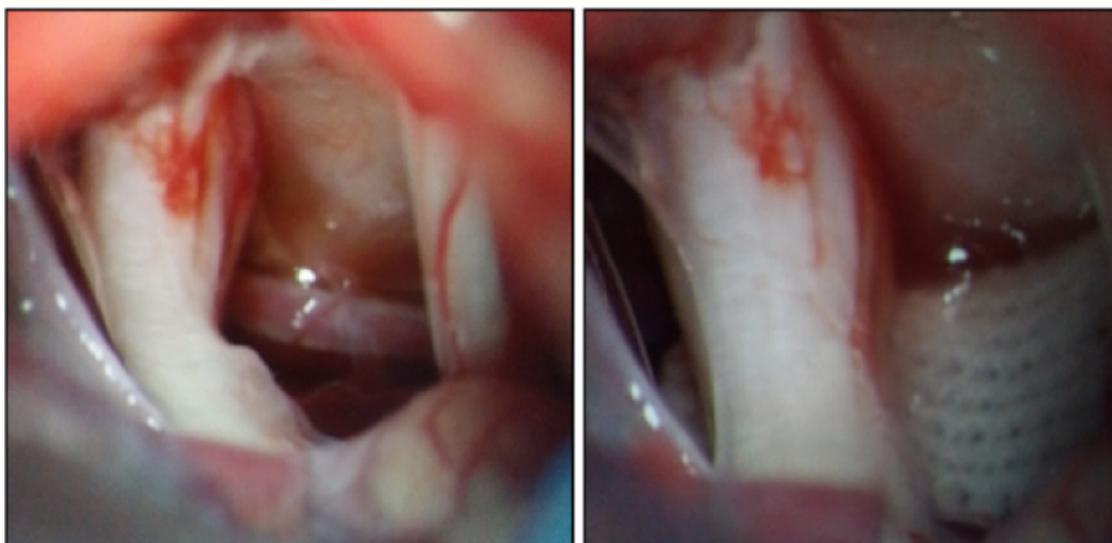


Figure 1: The trigeminal nerve compressed by the right superior cerebellar artery (left) is observed; subsequently (right image); in the image on the left we can see the Teflon band placed by the surgeon separating both structures.

Discussion

TN causes sudden, recurrent, short-lived, and unilateral episodes of pain, most often described as stabbing or “electric shock - like” and sometimes triggered by non-harmful stimuli per se (allodynia), in one or more of the facial dermatomes dependent on the trigeminal nerve [1-3].

This case represents a classic TN, caused by vascular compression. First choice’s treatment is pharmacological, typically with Carbamazepine (200 - 2400 mg/24h), although other drugs may be used, reserving surgery and interventional techniques (e.g. radiofrequency rhizotomy...) for cases refractory to this treatment.

On the other hand, OFA techniques have been performed since 1947 [4]. With the OFA we ensure the clinical principles of all general anesthesia (hypnosis, immobility, autonomic nervous system (ANS) control) avoiding or minimizing the intraoperative administration of opioid drugs in order to keep away from their side effects, some of which (nausea, vomiting, respiratory depression, decreased level of consciousness) can especially put the neurosurgical patient at risk, in which the desirable is an early anesthetic education to assess neurological function avoiding situations that increase intracranial pressure (ICP).

A wide range of OFA regimens adapted to different clinical situations have been used with several drugs (Magnesium Sulphate, Lidocaine, Ketamine, Dexmedetomidine, another adjuvant drugs...) [5]. In this case, a total intravenous anaesthesia (TIVA) was performed with propofol and administering rocuronium as a neuromuscular blocker.

The perioperative approach to analgesia/antinociception was performed with a locoregional block of the peripheral cranial nerves (“scalp block”) with ropivacaine made before the incision plus infiltration of the surgical wound with bupivacaine associated with adrenaline at closure. This technique reduces postoperative pain in the first 12 hours and opioid requirements for more than 24 hours [6-10].

The analgesic and opioid - sparing effect of magnesium sulphate and ketamine is well described [11-20]. Both drugs act mainly as antagonists of the N - methyl - D - Aspartate receptor (NMDAr), which is involved in the development of chronic pain syndromes and states of hypersensitization of central nervous system origin and have demonstrated their effectiveness in the management of neuropathic pain.

Special mention should be made of the use of ketamine in neuroanesthesia: its indications and effects have been reviewed and updated [21,22] and its use in neuroanesthesia is currently not contraindicated since it has been shown that the increase in cerebral blood flow (CBF) and PIC originally associated with its use are due to hypercapnia and mean arterial pressure (MAP) increments, and not due to a ketamine’s direct neurovascular effect [22].

Its intraoperative usefulness has also been demonstrated in “preventive analgesia protocols” and in the management of chronic painful syndromes [22,23]. Haloperidol was administered as a prophylactic measure for nausea - vomiting and for ketamine’s psychomimetic effects. Lidocaine i.v. was not administered for not producing overdosage of local anesthetics. To this day in my hospital we do not have dexmedetomidine for intraoperative use.

The clinical situation of the patient in the immediate postoperative period, with the remission of his neuralgia and the abandonment of opioid treatment shows that opioid-free anesthetic techniques can be useful in the perioperative management of chronic pain syndromes, even in the context of neuroanesthesia.

Conclusion

OFA multimodal anesthetic techniques may be useful in the perioperative management of neurosurgical patients in general and in particular in patients with chronic pain syndromes. In addition, with OFA we avoid the side effects of opioids, which are particularly harmful in this type of patients.

The scalp block is useful to decrease the postoperative consumption of opioid analgesics. All these measures could contribute to an early extubation and to a precise neurological evaluation in the immediate postoperative period of this type of patients.

Declaration of Patient Consent.

The authors certify that they have obtained all appropriate patient consent forms.

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Nil.

Conflicts of Interest

There are no conflicts of interests.

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