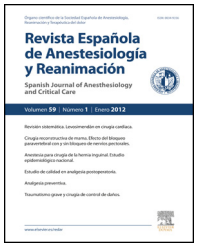
Rev Esp Anestesiol Reanim. 2014;xxx(xx):xxx-xxx

Revista Española de Anestesiología y Reanimación

www.elsevier.es/redar



CASE REPORT

Ammonia encephalopathy and awake craniotomy for brain language mapping: Cause of failed awake craniotomy

G. Villalba Martínez[a,*], J. Fernández-Candil[b], R.M. Vivanco-Hidalgo[c], S.P. Terradas[b], A.L. Jorba[d], R.A. Pérez[b]

[a] *Neurosurgery Department, Hospital del Mar, Barcelona, Spain*
[b] *Anesthesiology and Resuscitation Department, Hospital del Mar, Barcelona, Spain*
[c] *Neurology Department, Hospital del Mar, Barcelona, Spain*
[d] *Neurophysiology Department, Hospital del Mar, Barcelona, Spain*

Received 19 February 2014; accepted 22 September 2014

KEYWORDS
Awake craniotomy;
Ammonia levels;
Ammonia encephalopathy;
Valproic acid;
Insular glioma

Abstract We report the case of an aborted awake craniotomy for a left frontotemporoinsular glioma due to ammonia encephalopathy on a patient taking Levetiracetam, valproic acid and clobazam. This awake mapping surgery was scheduled as a second-stage procedure following partial resection eight days earlier under general anesthesia. We planned to perform the surgery with local anesthesia and sedation with remifentanil and propofol. After removal of the bone flap all sedation was stopped and we noticed slow mentation and excessive drowsiness prompting us to stop and control the airway and proceed with general anesthesia. There were no post-operative complications but the patient continued to exhibit bradypsychia and hand tremor. His ammonia level was found to be elevated and was treated with an infusion of L-carnitine after discontinuation of the valproic acid with vast improvement. Ammonia encephalopathy should be considered in patients treated with valproic acid and mental status changes who require an awake craniotomy with patient collaboration.

PALABRAS CLAVE
Craneotomía con el paciente despierto;
Niveles de amonio;

Encefalopatía por amonio y cirugía con el paciente despierto para mapeo del lenguaje; causa de fracaso de craneotomía despierta

Resumen Se presenta el caso de un paciente con un glioma insulofrontotemporal izquierdo, tratado con levetiracetam, valproato y clobazam. Se realizó una primera cirugía bajo anestesia

* Corresponding author.
E-mail address: gloriavillalbamartinez@gmail.com (G. Villalba Martínez).

http://dx.doi.org/10.1016/j.redar.2014.09.009

Please cite this article in press as: Villalba Martínez G, et al. Ammonia encephalopathy and awake craniotomy for brain language mapping: Cause of failed awake craniotomy. Rev Esp Anestesiol Reanim. 2014. http://dx.doi.org/10.1016/j.redar.2014.09.009

Encefalopatía por amonio;
Ácido valproico;
Glioma insular

general para la exéresis del lóbulo temporal tumoral, y 8 días después se sometió a una cirugía con el paciente despierto para mapeo del lenguaje, bajo sedación consciente con remifentanilo y anestesia local. A la llegada a quirófano, el paciente se encontraba cansado y con cierta bradipsiquia; tras parar la infusión de remifentanilo, y antes de abrir la duramadre, el paciente presentó una disminución del nivel de consciencia con privación respiratoria que requirió intubación endotraqueal y la finalización de la cirugía. En el periodo posoperatorio se apreció bradipsiquia moderada, cansancio y temblor de manos. Fueron detectados niveles altos de amonio en sangre, y la clínica mejoró tras la administración de L-carnitina y la suspensión del valproato. La encefalopatía por amonio, aunque con mínima sintomatología, debería ser considerada en pacientes tratados con valproato que van a ser sometidos a una cirugía bajo sedación, donde se requiere que el paciente colabore.

Introduction

Awake craniotomy is performed in patients with a tumor located in close proximity to eloquent areas of the brain. This procedure needs the collaboration of the patient to successfully map cortical and subcortical structures. Therefore, sedation effects should be absent or negligible at this point.[1]

Different anesthetic strategies have been described to perform the awake craniotomy and language mapping with local anesthetic: IV sedation (IVS) and asleep–awake–asleep (AAA).[2–8] AAA technique consists in general anesthesia induction with laryngeal mask (Mayfield head holder placement, scalp and dura mater incision) followed by total awakening for intraoperative cerebral mapping, and finally general anesthesia with laryngeal mask for the dura mater and scalp closure.

IVS is the usual technique used in our center. It consists of conscious sedation using low doses of an opioid analgesic such as remifentanil with or without propofol, and local anesthesia for the surgical wound and for the regional nerves using bupivacaine 0.25%. Invasive blood arterial pressure, heart rate, oxygen saturation (SpO_2) and end-tidal CO_2($etCO_2$) were recorded at baseline and during all the procedures via nasal cannula.

During the Mayfield head holder placement, scalp and dura mater incision the remifentanil (with or without propofol) infusion is maintained. The surgeons then proceed to map and resect the tumor with an awake and cooperative patient. During closure the remifentanil is reestablished at the same rate or at a higher dose because patient cooperation is not required.

The success of awake surgery is dependent on an adequate selection of patients. Obstructive sleep apnea syndrome (OSAS), obesity, gastroesophageal reflux, chronic cough, dysphasia, impaired consciousness and language barrier have been reported as relative contraindications to awake surgery.[9]

The majority of patients with a low-grade glioma undergoing surgery receive antiepileptic treatment, as seizures occur frequently in these patients.

Ammonia encephalopathy is not a frequent complication derived from the treatment with valproic acid. Patients with normal levels of valproic acid may suffer hyperammonemia, while patients suffering hyperammonemia do not necessarily have to suffer encephalopathy.[10] An association between a daily intake of valproic acid and the appearance and severity of ammonia encephalopathy has not been found. A relation between the duration of the treatment and the ammonia levels has not been found either. Valproic acid, can induce hyperammonemia by acting in the mitochondria of the hepatocyte, inhibiting the carbamoyl phosphate synthetase 1, and decreasing ammonia utilization.[11] Valproic acid also increases renal excretion of carnitine, which reduces fatty acids available for B-oxidation, increasing protein metabolism. In addition, an altered B-oxidation can decrease the ammonia metabolism in the cycle of urea.[12,13]

Symptoms of ammonia encephalopathy can be: lethargy, bradypsychia, decreased level of consciousness, vomiting, focal neurological deficits and even death. A clear correlation between clinical severity and ammonia blood levels has not been obtained. Diagnosis is based on clinical picture, blood test and electroencephalography (EEG). The EEG shows diffused signs of severe encephalopathy, with an irregular, continuous, severe and slowing diffused pattern with a predominance of theta and delta activity.[14–18]

A case of a patient who underwent awake craniotomy for language mapping is reported, in whom the presence of ammonia encephalopathy contributed to the appearance of respiratory depression, a fact that forced the neurosurgeon to abort the language mapping.

Case report

We present a 47 year old male, 87 kg, 180 cm, ASA grade ll, without toxic habits with a brain MRI showing a left frontotemporoinsular tumor suggestive of high-grade glioma. He debuted with a tonic-clonic seizure, followed by partial-complex and generalized seizures. He was treated with levetiracetam (1500 mg/12 h), valproic acid (1000 mg-500 mg-500 mg), and clobazam (10 mg/12 h). Liver function was normal. The preoperative neuropsychological assessment confirmed a slight expressive aphasia, and impaired verbal memory. The patient also complained of fatigue

Please cite this article in press as: Villalba Martínez G, et al. Ammonia encephalopathy and awake craniotomy for brain language mapping: Cause of failed awake craniotomy. Rev Esp Anestesiol Reanim. 2014. http://dx.doi.org/10.1016/j.redar.2014.09.009