

Figure 1 T2-weighted coronal sections of MRI scan showing enlarged parotid glands with small millimetric hyperintense images that may correspond to small cysts or acini, plus a more prominent one (1.3 cm) with a lobular aspect in the right parotid gland.

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J. Porta-Etessam*, A. Sanpedro, D. di Capua,
C. García-Pérez-Cejuela, R. García-Ramos

Servicio de Neurología, Hospital Clínico Universitario San Carlos, Madrid, Spain

*Corresponding author.

E-mail: jporta@yahoo.com,
mporta@caminos.recol.es. (J. Porta-Etessam).

Caudal nucleus haemorrhage due to dental anaesthesia

Hemorragia de núcleo caudado por anestesia dental

Dear Editor:

The most common causes of intraparenchymatous cerebral haemorrhage are arterial hypertension (HT), coagulopathy,

amyloid angiopathy, cerebral malformations, tumours and strokes with hemorrhagic transformation. However, there are other less common aetiologies in which haemorrhage has a specific trigger, such as substances with sympathomimetic effects on the cardiovascular system; consequently, there have been cases caused by diet pills containing phenylpropanolamine,¹ by amphetamines and methylphenidate² for the treatment of attention deficit disorder and by pseudoephedrine^{2,3} in nasal decongestants.

The purpose of our letter is to present the case of a patient who suffered a cerebral haemorrhage, probably related to dental anaesthesia.

The patient was a 41-year-old female, with no cardiovascular risk factors. The only relevant personal history was that she was in treatment with paroxetine for depressive syndrome.

Two days before being seen by our department, she underwent a dental intervention for the extraction of several teeth, in which nerve block anaesthesia was applied in both mental foramina using a solution of 90mg lidocaine hydrochloride monohydrate and 18mg noradrenaline tartrate. She immediately suffered sudden headache and vomiting. Her arterial pressure at that time was 140/70mmHg. Due to the persistence of headache, she decided to go to the emergency service. The only finding on the neurological and physical examination was neck rigidity. Neuroimaging studies were performed (computed tomography scan and brain magnetic resonance) (figs. 1 and 2), revealing haemorrhage in the head of the left caudate nucleus. This was open to the ventricles, with minimal dilatation of the temporal horns. Laboratory analyses, electrocardiogram and chest radiograph were normal. Coagulopathy was ruled out by a normal coagulation study, and angiography showed no vascular malformations that would justify a cerebral haemorrhage.

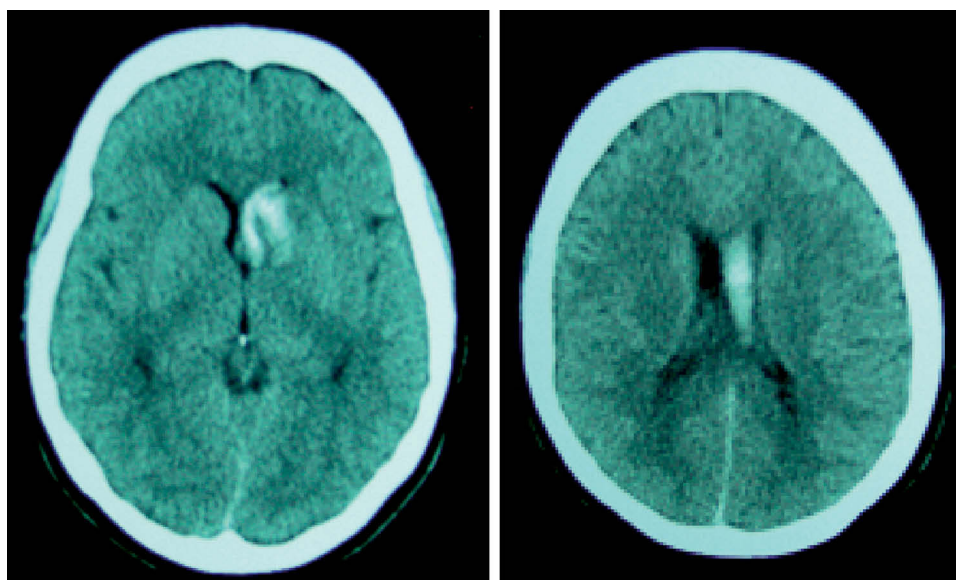


Figure 1 Cranial computed tomography scan. Haemorrhage can be seen in the head of the left caudate nucleus, open to ventricles and with minimal dilation of the temporal horns.

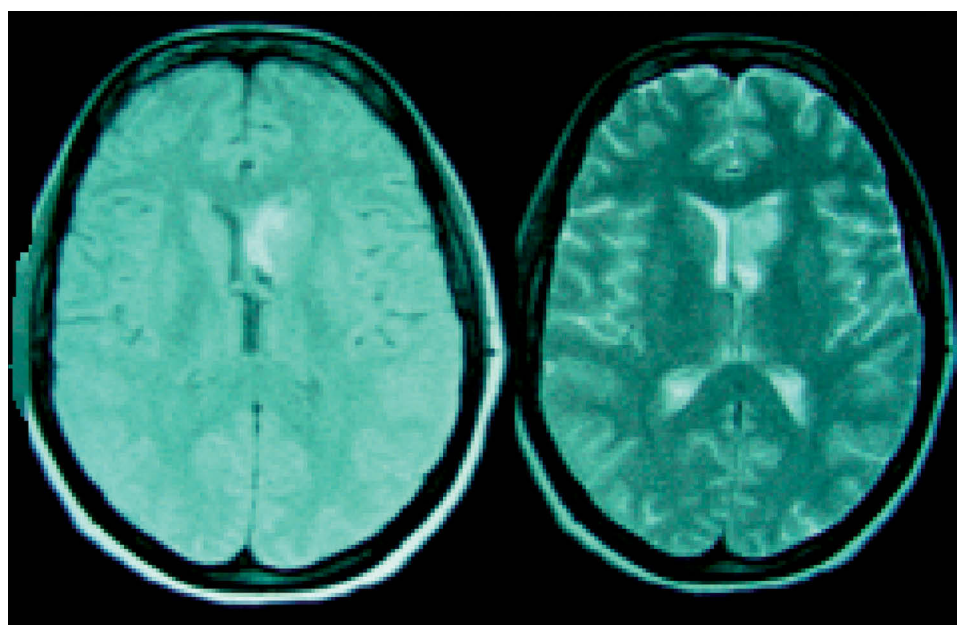


Figure 2 Cranial magnetic resonance scan (T1 and T2 sequences). Hyperintensity can be observed in the head of the caudate nucleus, corresponding to the hemorrhagic lesion in that area.

The outcome was favourable. The patient was asymptomatic at discharge and the neurological examination resulted normal.

Injection into the mental foramen, through which the mental nerve and artery run, might have caused, through the puncture of the latter, vasoconstrictor release into the vascular stream.

Cerebrovascular complications caused by sympathomimetic drugs are due to their systemic effects on the cardiovascular system,² so cerebral haemorrhage is attributed to a sharp

rise in blood pressure. However, our patient did not present high blood pressure at any time. Underlying vascular anomalies were not detected either, so this case could have been caused by a very brief rise in blood pressure or by a direct effect on the blood vessel.

In this regard, there have been reports of reversible cerebral vasoconstriction⁴⁻⁶ associated with the use of drugs and pharmaceutical agents with sympathomimetic activity. We have not found any that have occurred in the contexts of injection of anaesthetic and vasoconstrictor.

Presentations

This case study was presented as a poster communication at the LIX Annual Meeting of the SEN.

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J. González Fernández*, V. García Morales,
J.M. Trejo Gabriel y Galán

*Servicio de Neurología, Hospital General Yagüe, Burgos,
Spain*

*Corresponding author.

E-mail: jimgonfer@hotmail.com (J. González Fernández).