SPORADIC MULTIPLE MENINGIOMAS – A RARE CAUSE OF SECONDARY EPILEPSY

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ABSTRACT

We present the case of a 60 year-old woman diagnosed with meningiomatosis (multiple sporadic meningiomas), in the absence of neurofibromatosis type 2. The cerebral computerized axial tomography (CT scan) revealed six expansive lesions (one isodense and five spontaneously hyperdense partially or completely calcified), which at the cerebral magnetic resonance imaging (MRI) examination showed typical features of meningiomas.
Case particularity: the very low incidence of sporadic multiple meningiomas and a rare cause of secondary epilepsy.

Key words: multiple meningiomas, secondary epilepsy

INTRODUCTION

Meningiomas are defined as tumors that arise contiguously to the meninges. They are most commonly found at the surface of the brain and, in rare cases, they may have an intraventricular or intraosseous location.

Meningiomatosis (multiple meningiomas) referes to two or more meningiomas with different intracranial locations (1). They are diagnosed with a frequency ranging from 0.58% to 4.5% on CT scans (2).

They are mostly associated with neurofibromatosis type 2 (NF2) but can rarely occur as cases of sporadic meningiomas (3).

Secondary epilepsy is considered to be the most common clinical presentation of patients with intracranial meningiomas (4).

CASE REPORT

We report the case of a 60-year-old left handed female, with a history of type-2 diabetes mellitus and arterial hypertension, diagnosed with multiple meningiomas (in 2000) and with a right middle cerebral artery ischemic stroke (2003), recovered with sequelae (left hemiparesis and elements of aphasia).

She was previously diagnosed with secondary epilepsy (2000), was treated accordingly with 2 antiepileptic drugs, but was admitted to our department following left partial motor seizures and left Todd paresis.

The neurological examination revealed some pathological signs: left central facial palsy, a left predominantly brachial hemiparesis, left hemihypoesthesia, right crural paresis, Babinski sign bilaterally,

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absent osteotendinous reflexes bilaterally and residual elements of Broca's aphasia.

The clinical general examination did not reveal signs of neurofibromatosis type 2.

Native and contrast enhanced cerebral CT scan showed multiple, partially or completely calcified expansive lesions (one isodense and five spontaneously hyperdense). The lesions were located as follows: two frontobasal – one on the left lesser wing of the sphenoid bone (2/1.5 cm), the other one right paramedian (3.2/3 cm) in close contact with the falx cerebri, one right frontoparietal (1.8/1.6 cm) and one right parietal lesion (1.7/1.3 cm), both in contact with the internal table of the skull and two high right frontal lesions (2/1.7 cm and 2/1.9 cm), also in contact with the internal table of the skull (Figs.1, 2 and 3).

The cerebral CT scan also showed a right fronto-parietal cortico-subcortical hipodense lesion, consistent with the sequellar ischemic stroke in the territory of the right middle cerebral artery.

The patient also underwent a cerebral contrast enhanced MRI examination that showed multiple expansive, well circumscribed, partially or completely calcified lesions, situated in the same locations and with the same dimensions as described on the cerebral CT scan, all in contact with the meninges.

Comparing the MRI images (2011-2013), the presence of the 6 meningiomas and the sequellae of the ischemic stroke are demonstrated, the examinations performed in 2013 showing an enhancement in size of the meningiomas, even if their location and characteristics remained unchanged.

The EEG examination showed bilateral, mostly central, slow theta waves, with a frequency of 5-6 Hz/sec, with frontal delta waves and bilateral frontoparietal discharges of slow theta waves (Fig.4).

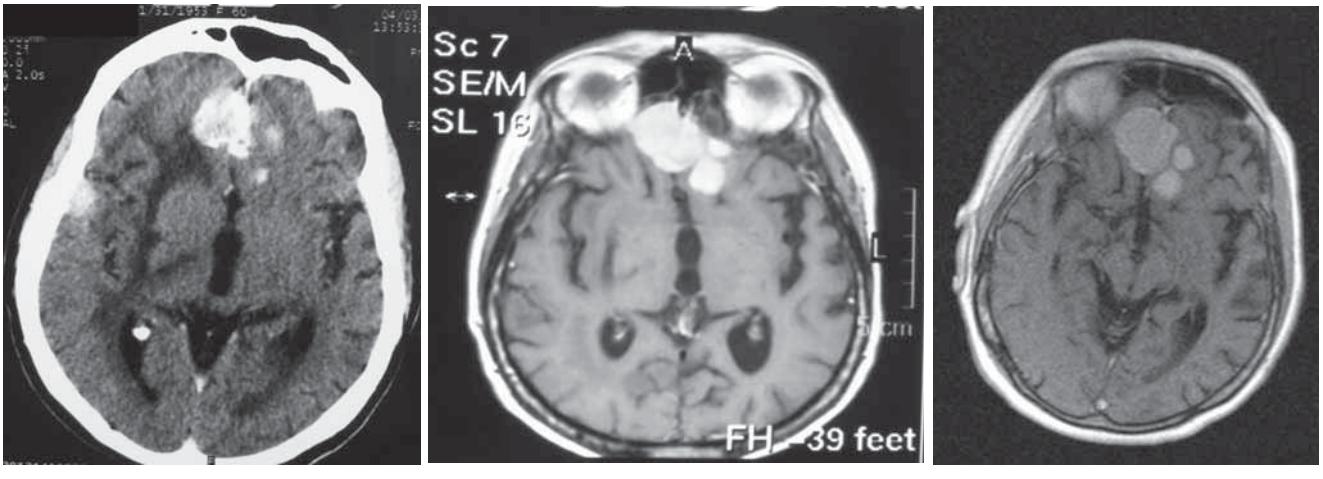
DISCUSSION

The cerebral CT and MRI findings corroborated with the physical examination were consistent with the diagnosis of sporadic multiple meningiomas. Due to the large number of lesions and their particular locations, neurosurgery is not a recommended treatment, but if the size of the meningiomas increases it could be taken into consideration.

The parietal meningiomas are the cause of the patient's epileptic seizures, but the fronto-parietal cortico-subcortical stroke sequelae could also be a possible cause in their absence (epilepsy was diagnosed before the stroke).

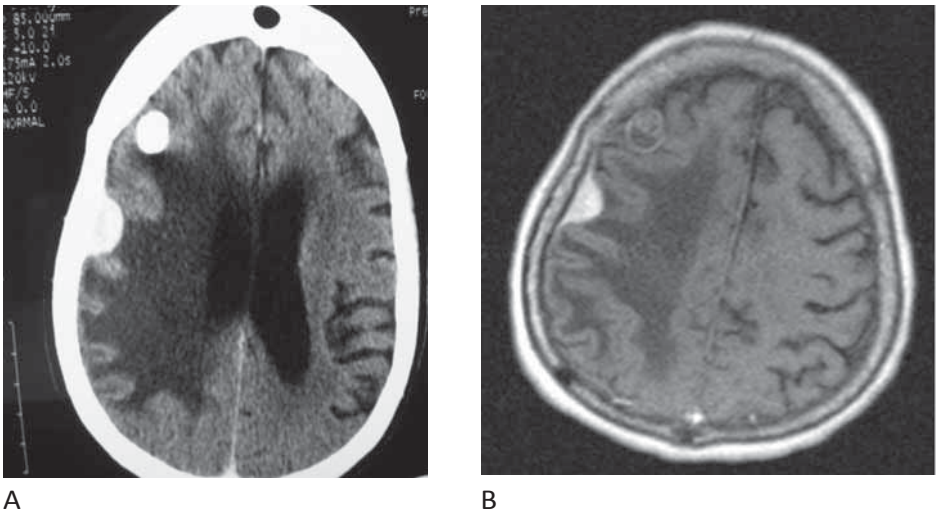
CONCLUSION

Multiple meningiomas, in the absence of neurofibromatosis type 2, represent a rare finding and should be considered as a diagnosis in patients with seizures and bilateral neurological signs. Patient's follow-up is necessary including clinical examination and cerebral imaging due to the possible enlargement of the lesions. Neurosurgical treatment should be considered if the meningioma's size or number enhance and the neurological signs and symptoms are worsening.



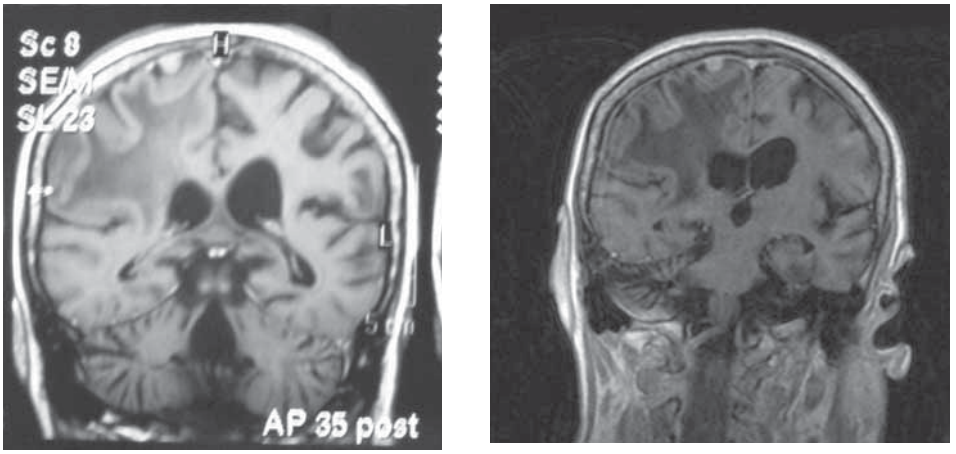
A B C

FIGURE 1. *A. CT scan (2013), B. Cerebral MRI (2011) C. Cerebral MRI (2013) – showing the frontobasal lesions, and in figure 1A also the right parietal meningioma*



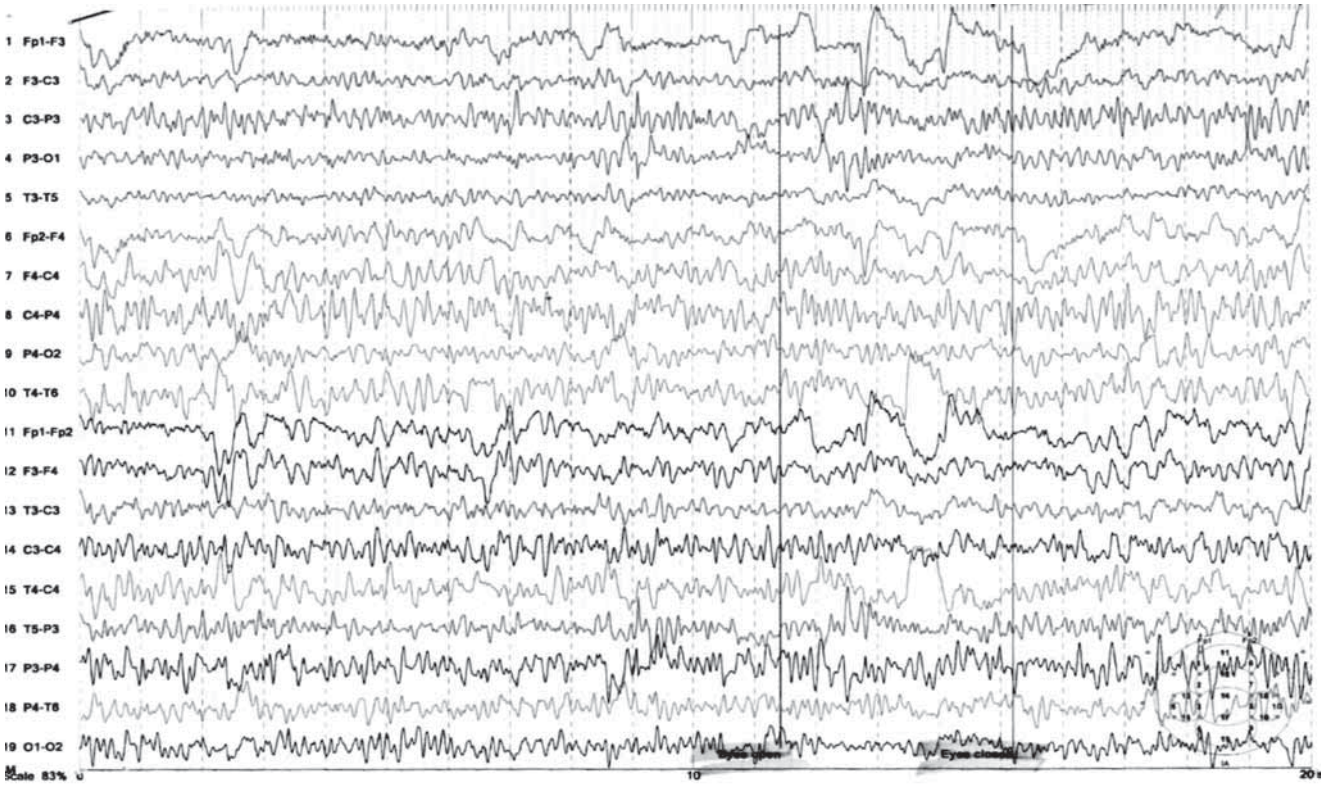
A B C D

FIGURE 2. *A. Cerebral CT scan (2013) B. Cerebral MRI (2013) C. Cerebral MRI (2011-Sagital section)*
D. Cerebral MRI(2013 – Sagital section)– showing the fronto-parietal and parietal meningiomas and the fronto-parietal cortico-subcortical stroke sequelae (Fig. 2.A, 2.B)

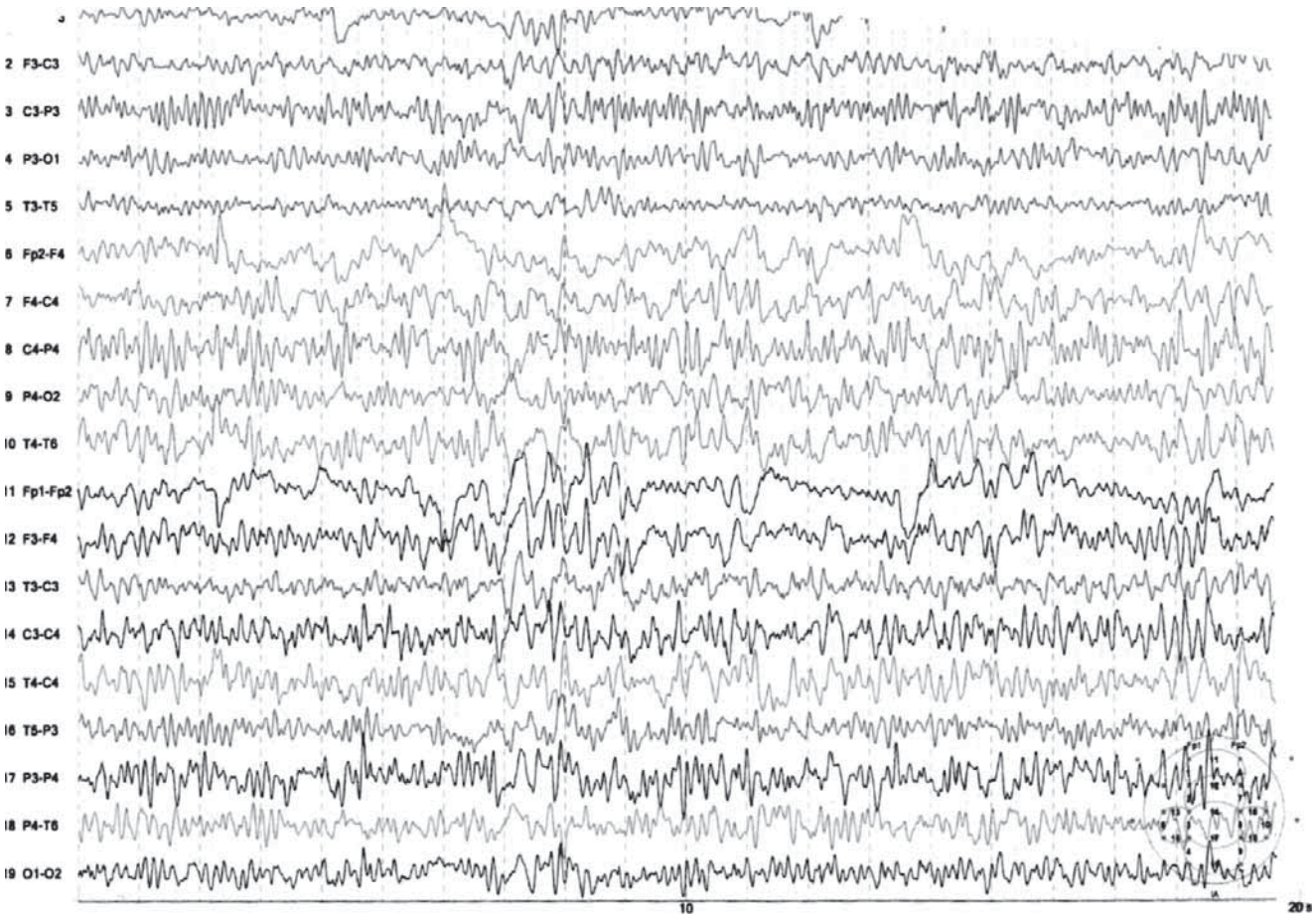


A B

FIGURE 3. *A Cerebral MRI (2011) and B (2013) – coronal sections showing the two high frontal meningiomas and also the fronto-parietal cortico-subcortical stroke sequelae*



A



B

FIGURE 4. A, B *– EEG: bilateral, mostly central, slow theta waves, with a frequency of 5-6 Hz/sec, with frontal delta waves and bilateral frontoparietal discharges of slow theta waves.*

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