NEURO IMAGE

VOLUME 21, NUMERO 2
DECEMBRE 2005

NEURORADIOLOGY
MUHC–MNH

VOLUME 21, NUMBER 2
DECEMBER 2005

POSTCARDS
FROM THE EDGE

IN THIS ISSUE …

Monastery of Agia Triada in Meteora, Thessaly, a region in central Greece.
It was also used for the James Bond film, "For Your Eyes Only"

IMAGES FROM PAST & PRESENT

I had the pleasure of travelling to Greece late September and I took many pictures, some have been used for the background of this issue. In Rhodes, I could not walk by this small monument representing two seahorses without standing there for a photograph. So many years of imaging hippocampi in memory!

I would also like to invite you to go to our website: http://www.mni.mcgill.ca/neuroimage/index.html there you can read the rest of this issue of NeuroImage; and you can also see previous issues.

NEURO-IMAGE

VOLUME 2, NUMÉRO 4 — NOVEMBRE 1985
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HÔPITAL NEUROLOGIQUE DE MONTRÉAL
MONTREAL NEUROLOGICAL INSTITUTE

The father of magnetick philosophy

William Feindel, m.d.

William Gilbert (1544-1603) studied medecine at Cambridge University and took up practice in London, eventually becoming President of the Royal College of Physicians and the Physician to Queen Elizabeth I.

After some twenty years of systematic experimental observations, Gilbert in 1600 published a book on the magnet in which he gathered all the knowledge at that time as well as his own observations concerning magnetic phenomena. He correctly viewed the earth as a giant magnet and by making a **terella** or small earth from naturally occurring magetic stone (lode stone) he was able to test in practice his theories of «magnetick vigour» and the «orbe of virtue» or the limit of the magnetic field around a lode stone. He showed that many substances — gold, silver, lead, glass had no magnetic properties and he disproved the long standing myth that garlic and diamonds neutralized a magnet's power to attract iron by making «an experiment with 70 excellent diamonds, in the presence of many witnesses». In another observation, he examined the variations from the meridian which the navigators had noted and the deflection from the horizontal known as declination. His instructions for this experiment were quite specific. «Fix a slender iron wire of 3 digits length through a round cork, so that the cork may support the iron in water. Let this water be in a good sized glass vase or bowl». The idea was to allow the wire to float in the middle of the liquid and freely point to indicate the magnetic field. He futher argued that this «magnetic cocktail» could be used to determine latitude and magnetic variation.

1. William Gilbert (1544-1603)

Gilbert was fully aware of the importance of understanding magnetism in every day commerce and navigation. He was the first in England to support the Copernican idea of the planetary system. He clearly distinguished magnetism from the phenomenon of static electricity. His works served as a significant base from which all later scientific research on magnetism could be advanced. He made these extensive observations over the years

(Continued on page 2)

An earlier version of NeuroImage (November 1985)

Best regards

Saluti affettuosi

Amicalement

此致

Herzliche Gruesse

Namaste

As-salaam alaykum

O Genki De

Bäst Hälsningar

Cordialmente

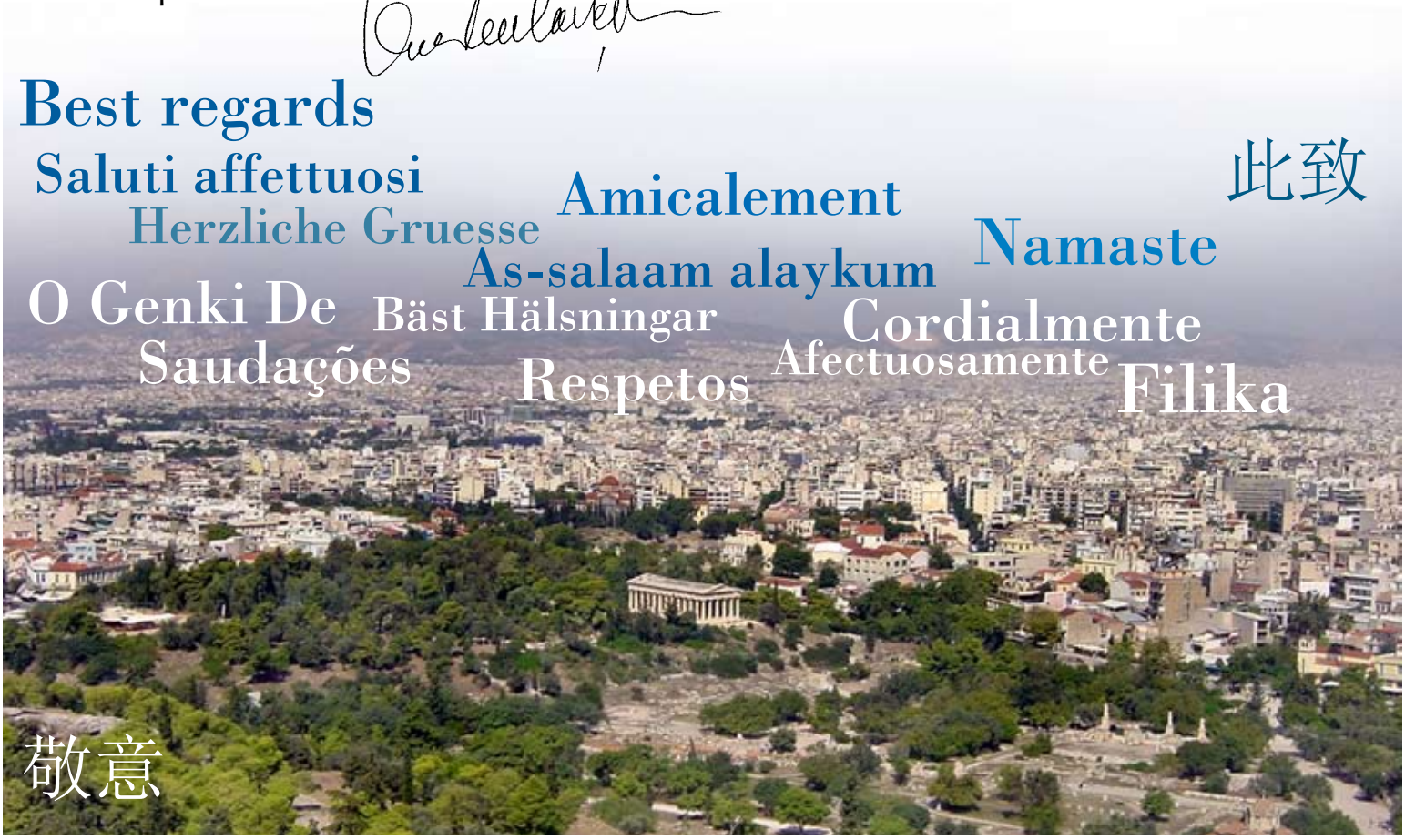
Saudações

Afectuosamente

Respetos

Filika

敬意



PILOCYTIC ASTROCYTOMA OF THE ADULT

Drs David M. Miller and Marie-Christine Guiot

CLINICAL HISTORY

Mr. H. is a 42 year old left handed male who suffered a witnessed generalized seizure lasting 3-4 minutes. His past medical history was significant for ethanol abuse as well as being followed at an outside institution for the past three years for a seizure disorder. At the time of admission, his sole prescription medication was dilantin. Physical exam was notable only for a right parietal soft tissue hematoma. Neurological examination was normal. Blood tests obtained in the Emergency Room revealed no abnormality.

IMAGING FINDINGS

Initial non-enhanced CT scan of the brain (Fig. 1a,b) demonstrated a 4.2 x 3.8cm left occipitoparietal cystic lesion without significant mass effect. Apart from a right parietal soft-tissue hematoma there was no acute intracranial traumatic injury identified. Following the injection of contrast (Fig 2a,b), there is identification of a 1 cm. enhancing nodule at the medial aspect of the lesion.

On MRI (Fig. 3), the left occipitoparietal lesion is again noted with a homogeneous medial enhancing nodule again noted. There was no enhancement identified along the wall of the cyst. Of further note is that the signal intensity of the cystic component differs from that of clear fluid representing proteinaceous material (Fig. 3b).

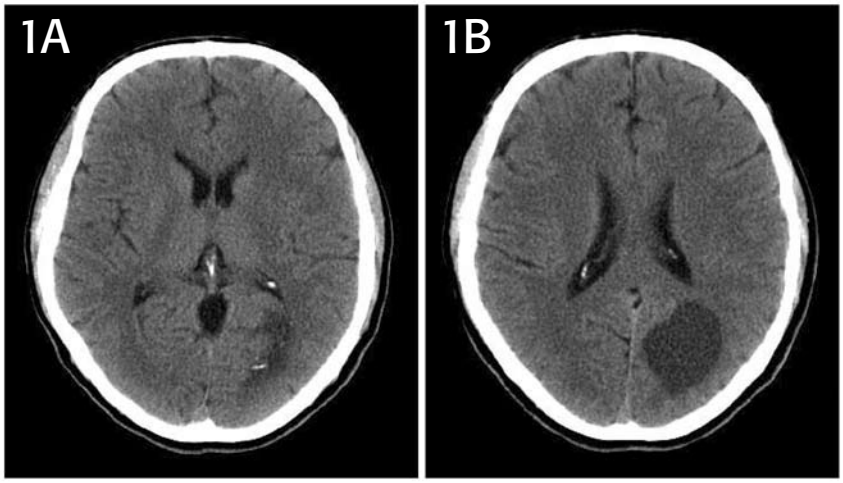


Figure 1: A, *Unenhanced axial CT scan image at the level of the basal ganglia demonstrates a left occipital hypodense focus with a possible nodule at the medial aspect* ***B,*** *Unenhanced image slightly cephalad to (a) shows a well defined left parietoccipital cystic lesion that is slightly hyperdense to CSF.*

DISCUSSION

In the pediatric population, the pilocytic astrocytoma is both the most common cerebellar neoplasm as well as the most common glioma overall- constituting 10% of all pediatric astrocytomas[3]. The quoted incidence rate within the adult population varies across studies, with 0.49 per million per year the incidence quoted in a recent British study specifically interested in adult pilocytic astrocytomas (Bell, 2004).

One of the earliest features of pilocytic astrocytomas in the adult to be recognized was how the distribution of lesion location within the brain differed from the pediatric population. In children, the cerebellum was the most frequent site of tumor involvement (67%). In adults, however, Bell et al.[1] found that five of the ten patients over 30 with pilocytic astrocytomas had supratentorial lesions and the remaining five had cerebellar lesions. Similarly, Burkhard et al[2] found supratentorial involvement in 55% of adult patients. As would be expected from the diverse location of the lesions, the clinical presentation is varied depending on the site of the tumor.

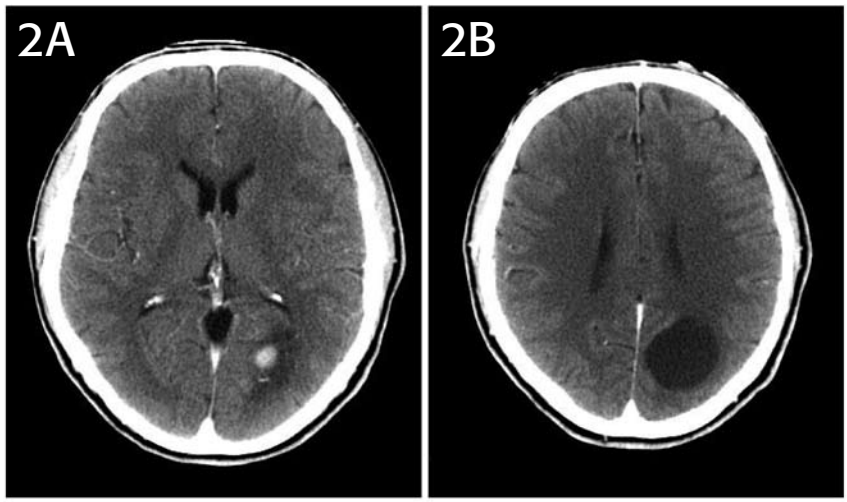


Figure 2: a,b *Contrast-enhanced CT matched images to Figure 1 demonstrates homogeneous enhancement of the medial mural nodule with no significant enhancement of the cystic component.*

The classic imaging manifestation of cerebellar and cerebral pilocytic astrocytoma is that of a cyst-like mass with an enhancing mural nodule and is seen in approximately two-thirds of cases. The cyst wall can

be either enhancing or non-enhancing. Less frequently, there can be a necrotic mass with a central non-enhancing zone or a predominantly solid mass with minimal or no cyst-like component. The precise cause for the often-noted enhancement of the cyst wall is not well understood and does not seem to correlate with aggressiveness of the lesion or prognosis.

Macroscopically, as would be anticipated from the imaging features described above, these tumors are typically well-circumscribed cyst-like masses with a discrete mural nodule. Histologically, these lesions demonstrate a 'biphasic pattern'. Rosenthal fibers as well as microcysts are often present. It should also be noted that the relative contribution of the loose and compact tissue components is highly variable within different tumors. While macroscopically well circumscribed, there may in fact be microscopic invasion into surrounding brain parenchyma, but this has not been shown to affect long-term prognosis.

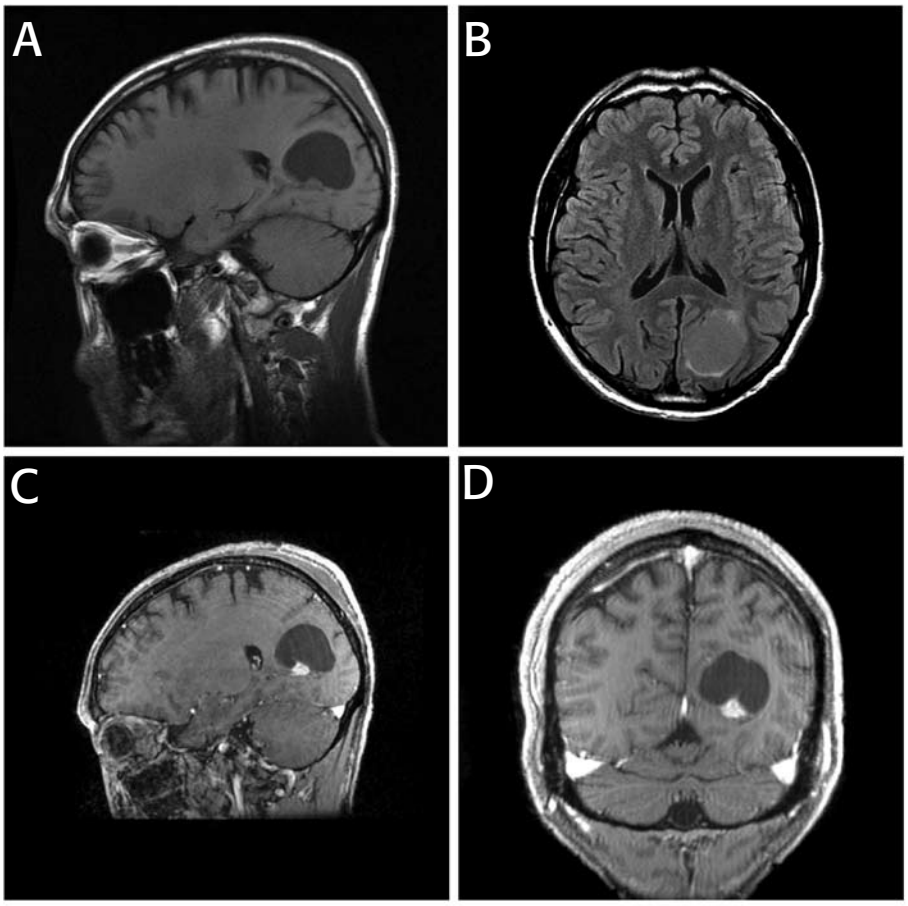


Figure 3: A, *Sagittal T1 pre-contrast.* ***B,*** *FLAIR image demonstrates that contents of cyst are hyperintense to CSF.* ***C,*** *Sagittal T1-post gadolinium injection demonstrates homogeneous enhancement of the mural nodule.* ***D,*** *Coronal T1-post gadolinium.*

THERAPY AND PROGNOSIS

Surgical management is the treatment of choice for pilocytic astrocytomas. Total resection of the mural nodule is considered curative. Treatment of the surrounding cyst is somewhat controversial: studies have failed to demonstrate any significant outcome difference with or without resection of the cystic component. There is excellent prognosis with surgical resection- the 10-year survival rate is up to 94% and a quoted 20-year survival rate of 79%[3].

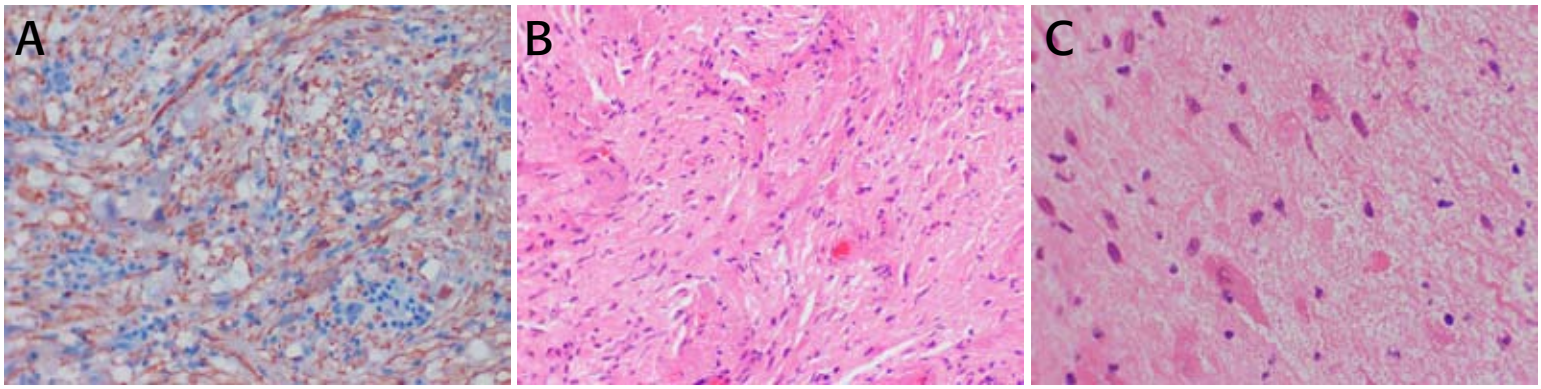


Figure 4: ***A,*** *Loose microcystic pattern* ***B,*** *Dense fibrillary background* ***C,*** *Rosenthal fibers*

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Cerebral Amyloid Angiopathy Related Hemorrhage Masquerading as SAH

Drs Kevin Petrecca, Grant Linnel, Marie-Christine Guiot, John Richardson & Denis Melançon

CASE REPORT

We present the case of a 77 year old man who presented with acute paresis of the left lower face and arm. The patient was otherwise neurologically intact. One week prior to admission he sustained a fall from his own height striking his occiput.

A plain CT revealed acute blood in the right central sulcus and inferior frontal sulcus (Fig. 1A). The presumptive diagnosis was vasospasm secondary to a traumatic subarachnoid hemorrhage (SAH). A diagnostic cerebral angiogram, performed to exclude an underlying vascular abnormality, was normal. The patient was started on Nimodipine resulting in a significant improvement in symptoms. One week later he developed more widespread symptoms including right arm weakness. A second plain CT revealed more extensive hemorrhage with acute blood in the right superior frontal sulcus, right precentral sulcus and left central sulcus (Fig. 1B).

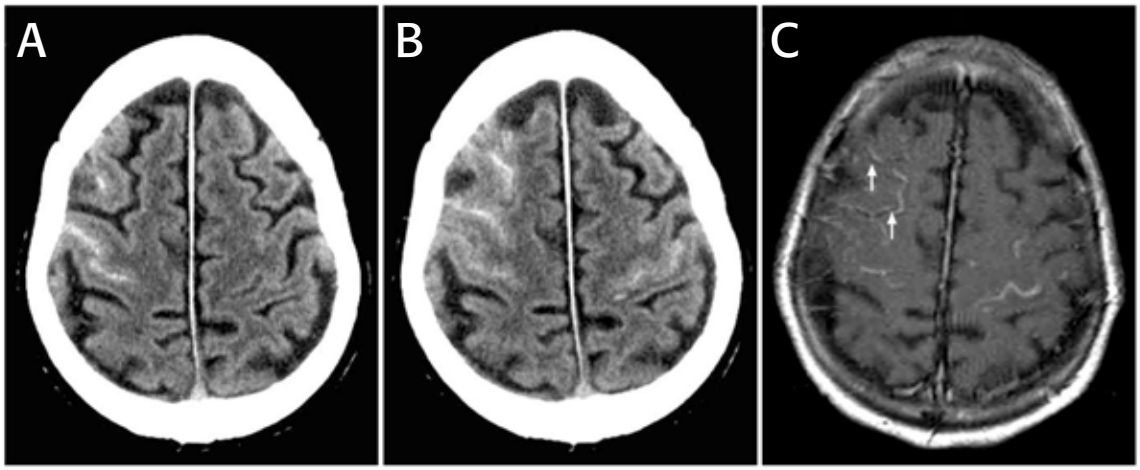

***Figure 1. A**, CT plain. **B,** CT plain. C, T1-weighted MR. Arrows indicate hyperintense signals restricted to the cortex.*

The systemic work-up of a vasculitis was negative. A T1-weighted MRI (Fig. 1C) showed hyperintense signals corresponding to the CT images; however, the hyperintense signals appeared, in certain regions, to be cortical and not extracortical (Fig. 1C, arrows). An open left frontal biopsy was performed for diagnosis. The pathology revealed β-amyloid deposition in blood vessel walls consistent with cerebral amyloid angiopathy.

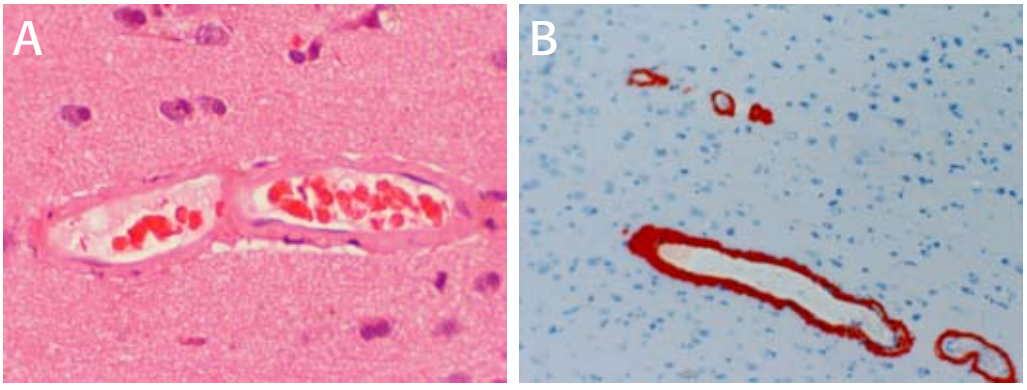

***Figure 2. A,** H&E stain showing a thickened vessel wall. **B**, Blood vessel immunolabelled with an anti-β-amyloid antibody revealing abundant β-amyloid deposition in the vessel wall.*

DISCUSSION

Cerebral amyloid angiopathy (CAA) is a common cause of primary spontaneous intracerebral hemorrhage. The pathogenesis of CAA involves β-amyloid deposition in the media and adventitia of cortical and leptomeningeal arteries, arterioles, capillaries and less often veins (1). As a result, vessels become more brittle and thus more susceptible to minor trauma and changes in blood pressure. As such, CAA-related hematomas are of cortical/subcortical origin; however, they are not restricted to these regions as they typically extend deeply into white matter. Here we present an unusual case of a CAA-related hemorrhage that is strictly confined to the cortex mimicking subarachnoid hemorrhage on plain CT.

REFERENCE

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Intracranial Dissemination of Glioblastoma Multiforme in Adults

Caterina Chiara Bianchi M.D., Donatella Tampieri M.D. F.R.C.P.C.,
Marie Christine Guiot M.D., Jeffery Hall M.D. M.Sc F.R.C.S.(C)

Glioblastoma multiforme (GBM) is usually a single lesion; it tends to recur locally after treatment. However multifocal glioblastoma multiforme has been reported to occur both spontaneously or following treatment of a solitary one.

In multifocal glioblastoma multiforme the lesions present usually close to the primary tumour site, dissemination through the central nervous system occurs rarely. We present a case of a primary multifocal intracranial GBM in a 62-year-old patient.

CASE DESCRIPTION

This is a 62-year-old female patient who was admitted in October 2005, at our hospital with progressive execution function impairment and memory problems occurring over the past two years. The magnetic resonance exam (MRI) done in June 2004 was reviewed and found negative. The symtomatology had particularly worsened in the last month. The neurological examination was completely unremarkable except for a slight ataxic gait. The present MRI demonstrated an heterogeneous space occupying lesion located in the genu of the corpus callosum extending to the anterior aspect of its body and rostrum. The lesion had predominantly hypointense signal in T1 and hyperintense in T2 with a few areas of necrosis. Some of the lesions strongly enhanced following gadolinium injection, while one lesion in the left parietal paramedien area did not enhance. **(Fig1 A,B,C,D)** The lesion was associated with surrounding vasogenic edema and causing severe mass effect resulting in compression of the frontal horns of the lateral ventricles. A multitude of small additional lesions were also noted within the cerebral hemispheres and leptomeningeal spaces. **(Fig1 E,F)**

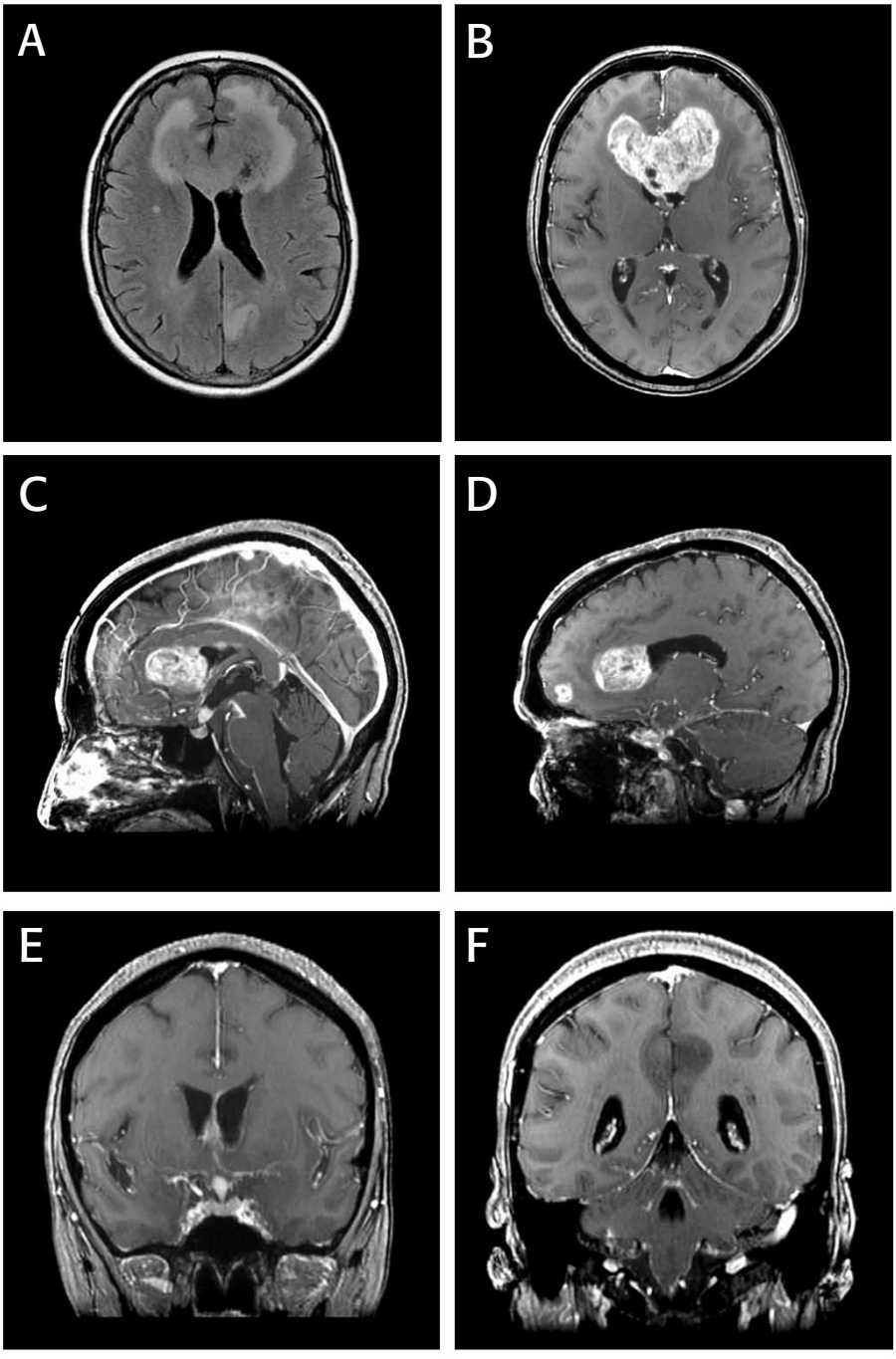

Figure 1. ***A,*** *Axial FLAIR image showing a hyperintense heterogeneous mass crossing the corpus callosum with marked associated vasogenic edema. There is a hyperintense cortical thickening in the left precuneus without edema.* ***B,*** *Axial T1 weighted image with contrast demonstrating enhancement of the lesion.* ***C,*** *Sagittal T1 weighted image with contrast showing small areas of nodular enhancement medially in a subependymal location along the left lateral ventricle. The infundibulum and the hypothalamus are widened and enhancing. There is enhancement of the leptomeninges over the anterior surface of the brainstem and most marked at the level of the interpeduncular fossa.* ***D,*** *Sagittal T1 weighted image with contrast showing an other small enhancing lesion in the right frontal lobe.* ***E,*** *Coronal T1 weighted image with contrast showing the enhancement in the infundibulum.* ***F,*** *Coronal T1 weighted image with contrast showing no enhancement of the lesion in the left precuneus. There is diffuse leptomeningeal enhancement infratentorially.*

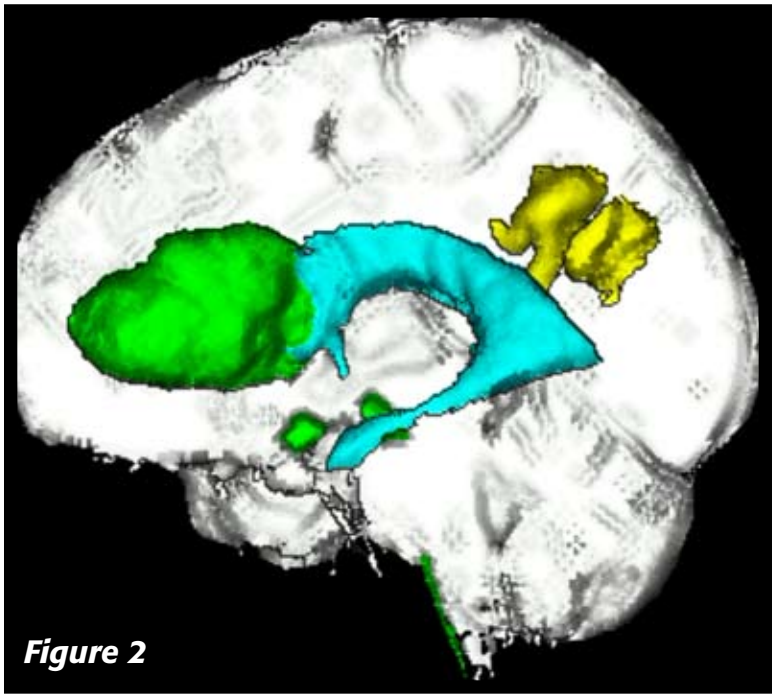

Figure 2

Reconstructed sagittal image of a 3D MR acquisition. Green represents GBM foci; yellow represents lower grade glioma; aqua blue represents ventricles

In consideration of the morphology and of the location of the enhancing lesion the diagnosis of the glioblastoma multiforme was placed. The patient was sent for a stereotactic needle biopsy of the frontal mass which conclude for astrocytoma grade IV (GBM). **(Fig2)**

The histology revealed a tumoral proliferation composed of gemistocytes and elongated cells with fibrillary process. Marked nuclear pleomorphism mitotic figures and numerous apoptotic cells were also seen as well with an area of necrosis and foci of endothelial proliferation.

The immunoistochemistry for GFAP was strongly positive and the proliferation index estimated by MIB-1 was high, reaching at 15% in a large part of the tumour. Immunoistochemistry for P53 was also positive. The patient was then referred for chemotherapy and radiotherapy.

DISCUSSION

Multiple cerebral lesions as the case the Authors described, could represent GBMs, metastases, even if the primary site of malignancy could not be identified; lymphoma because of multifocal pattern of lesion with spread along the corpus callosum, involvement of the infundibulum and the hypothalamus. However, multifocal GBM, even though the hypothalamus is a rare location, could result in this pattern and the cortical abnormality in the left parietal lobe could be compatible with a different stage of the same pathology and could be considered a low grade glioma.

Multiple high-grade gliomas (GBM) have been classified as: A) multicentric if they arise independently in more than one side of the cerebral hemisphere and B) multifocal if they spread from a primary focus to other areas of the brain. However this distinction does not have practical clinical value and gliomas have been categorized as early if they present at the initial diagnosis or late if they present during the treatment[2,3,5]. Dissemination can occur intracranially or throughout the spinal axis and various patterns of spread have been described in the literature[3,4]. In the most recent literature three subtypes of intracranial dissemination based on MRI characteristic have been described; Type I; a single location of GBM is present associated to subependymal or subarachnoid spread at sites distant from the primary tumour location; Type II; a multifocal GBM is present associated to subependymal or subarachnoid spread; Type III, multifocal GBM displays subependymal or subarachnoid spread at sites distant from the primary tumour location.[1] According to this classification the case we describe represents a Type III. To our knowledge no Type III has been yet described in the literature. In particular the characteristics of lesions in different degrees of malignancy as suspected on the MRI is an extremely rare event.

CONCLUSION

In conclusion, multifocal GBM can be diagnosed on the first MRI as we have illustrated in this case.
Because of the subependymal and leptomeningeal spreads, we include this unique case as Type III according to the recent classification proposed by Parsa et al. A focus of lower grade was likely preexisting in this case, making it a different subtype.

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The public library at the ruins of Ephesus

A view of the Aegean Sea from Santorini

The amphitheatre at the ruins of Delphi

A view of the village in Santorini

Mykonos windmills

Volume 21 – number1 – Bibliothèque nationale, ISSN 1180-0844 National Library of Canada, Production – Denis Melançon – Neurikon Inc.
Layout, Graphic Design by J.P. Acco at the Department of NeuroPhotography at the Montreal Neurological Hospital (11-2005)