

hypertensive crisis, an acute episode of decompensated diabetes, or a bout of arthritic pain, although these episodes may last less time. There must be many other terms for this type of migraine which would more accurately reflect its nature, that is, symptom exacerbation in addition to chronicity.

However, this term is already well established and will therefore be very difficult to change. Unfortunately, *migraña crónica* is not a neologism; neologisms usually become accepted with time, especially in scientific settings. Rather, we find ourselves reinforcing a sort of false friend that should, at the very least, be unmasked.

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The 'three-hour effect' constitutes procrastination in thrombolytic stroke treatment[☆]



En la trombólisis del ictus el «efecto 3 horas» es procrastinación

Dear Editor:

It was with great interest that we read the study by Iglesias Mohedano et al.¹ addressing the factors associated with in-hospital delays in administering intravenous thrombolysis to patients with acute ischaemic stroke in a tertiary hospital. Curiously enough, one of the crucial factors associated with treatment delay in the multivariate analysis was onset-to-door time: the sooner patients arrive at the hospital, the longer they have to wait to receive thrombolysis once the CT study has been completed. The authors cite 2 articles dated 2011 and 2012 mentioning this phenomenon, which they call the '3-hour effect'.^{2,3} However, this 3-hour effect is a function of the therapeutic window and should now be called the '4.5-hour effect', or the '6/8-hour effect' in the case of endovascular revascularisation.

In 2005, we proposed the term 'procrastination', that is, putting off a task unnecessarily and with no justification,⁴ a very typical practice possibly resulting from laziness, which is probably not the case here, or reflecting the complexity and risks associated with a pending decision, as occurs with thrombolysis. Informally alerting our neurologists to the dangers of procrastination led to significant improvements, as we found some months later.⁵ However, not all

tertiary hospitals seem to be aware of procrastination and this faulty practice is still frequent: CT-to-needle time was longer in patients with shorter onset-to-door time, at least until the publication of the study by Iglesias Mohedano et al.¹ Specific emphasis should be placed on avoiding unnecessary delays, which can still be observed even after 20 years of thrombolytic stroke treatment. Even for patients within the therapeutic window, the sooner the treatment, the better.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Brain lesions in a long-term kidney transplant recipient: Primary cerebral lymphoma or cerebral toxoplasmosis?^{☆,☆☆}



Lesiones cerebrales en trasplante renal de larga evolución, ¿linfoma cerebral primario vs. toxoplasmosis cerebral?

Introduction

Infection with the obligate intracellular protozoan parasite *Toxoplasma gondii* is asymptomatic in more than 90% of the immunocompetent adult population.¹ Cerebral toxoplasmosis (CTx) mainly affects untreated HIV-immunocompromised patients with a CD4+ T cell count <100 mm³. It is an extremely rare finding in other patients except for transplant recipients.^{2,3} Toxoplasmosis after solid organ transplantation is generally diagnosed after the first month post-procedure. It is more frequent in heart transplant recipients and rarely affects kidney transplant recipients.⁴ We present the case of a patient with cerebral toxoplasmosis 18 years after transplantation.

Clinical case

Our patient was a 74-year-old woman with a history of arterial hypertension, obesity, and severe chronic kidney disease secondary to nephroangiosclerosis and analgesic nephropathy. She underwent transplantation of the right kidney in 1996 and was treated for acute rejection with OKT3. Since then, she has been treated with ciclosporin, prednisone, and mycophenolic acid as immunosuppressants. The patient was assessed by the neurology department due to acute-onset symptoms of dysarthria and right hemiparesis with spontaneous recovery. While hospitalised, she experienced 3 similar episodes that remitted with levetiracetam and increasing the dose of corticosteroids.

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^{☆☆} This study was presented in poster format at the 66th Annual Meeting of the Spanish Society of Neurology.

A cranial computed tomography (CT) scan showed diffuse hypodense lesions with effacement of convexity sulci on the right frontal-parietal-temporal area and the left frontoparietal area, as well as multiple subcortical hyper-enhanced lesions. A brain MRI showed vasogenic oedema in the subcortical frontoparietal area and left posterior temporal area, plus 3 subcortical nodules (Fig. 1). The electroencephalography revealed no abnormalities. Results for tumour markers and a CT scan of chest, abdomen, and pelvis were normal. The positron emission tomography (PET) revealed that lesions were hypermetabolic and suggestive of malignancy.

A brain biopsy was performed. The first histopathological study yielded non-specific results consisting of presence of lymphoproliferative infiltrate. We requested a second assessment of the same sample, which revealed trophozoites; we diagnosed CTx. The PCR test for *T. gondii* was positive.

The patient showed good clinical and radiological progress (Fig. 2) after starting treatment with pyrimethamine and clindamycin.

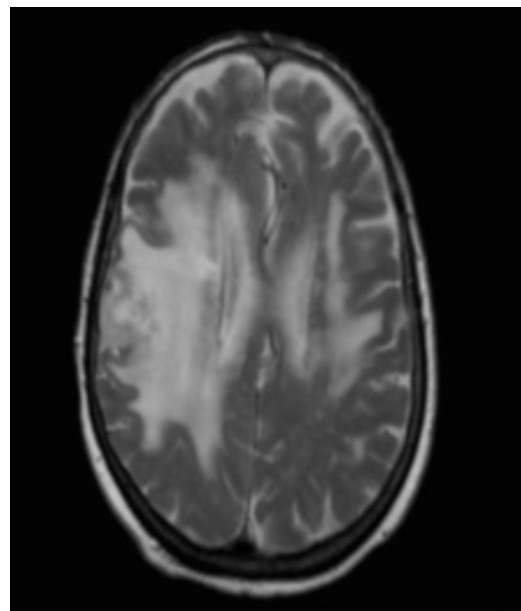


Figure 1 Brain MRI T2-weighted sequence. Hyperintense lesions on the right temporoparietal area and the left temporal area with associated vasogenic oedema.