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Vascular depression with melancholic symptoms: response to electroconvulsive therapy

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The concept of vascular depression includes cases of late-onset depression, with cerebral infarction observed in imaging modalities and cardiovascular risk factors, with cognitive disabilities and poor response to antidepressant drugs. A case of a 71 year-old woman with no psychiatric background and with vascular risk factors who was re-admitted to a psychiatric hospitalization unit for melancholic depression with psychotic symptoms is presented. The magnetic resonance imaging showed a subcortical lacunar lesion and mild leukoaraiosis lesions and the neuropsychological examinations showed mild cognitive impairments. In the previous admissions the symptoms were refractory to several psychopharmacology strategies, responding only to electroconvulsive therapy (ECT). In this admission she received ECT, with remission of the symptoms, and was included in an ECT maintenance program. The vascular depression concept would explain the special features and outcome of this case.

Key words:
Vascular depression. Late-onset depression. Electroconvulsive therapy. Executive functions.

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Depresión vascular con síntomas melancólicos: respuesta a terapia electroconvulsiva

El concepto de depresión vascular hace referencia a casos de depresión de inicio tardío, con infartos cerebrales en técnicas de imagen y factores de riesgo cardiovascular, con déficit cognitivos y pobre respuesta a fármacos antidepresivos. Se presenta el caso de una mujer de 71 años, sin antecedentes psiquiátricos y con factores de riesgo vascular, que reingresa en una unidad de hospitalización psiquiátrica por depresión melancólica con síntomas psicóticos. La resonancia magnética de la paciente mostró un infarto lacunar subcortical y una ligera leucoaraiosis y presentó déficit cognitivos en las pruebas neuropsicológicas. En los ingresos previos fue refractaria a psicofármacos, respondiendo únicamente a terapia electroconvulsiva (TEC). En este ingreso se aplicó TEC, con remisión de la sintomatología, incluyéndose en programa de TEC de mantenimiento. El concepto de depresión vascular podría explicar las especiales características y evolución del caso.

Palabras clave:
Depresión vascular. Depresión de inicio tardío. Terapia electroconvulsiva. Funciones ejecutivas.

INTRODUCTION

A little more than one decade ago, Krishnan (1995) expressed the hypothesis of arteriosclerotic depression[1] and Alexopoulos (1997) introduced the concept of vascular depression[2], when he proposed that cerebrovascular disease could predispose, precipitate or perpetuate depression in the geriatric age. He indicated the features that would be characteristic of it: cardiovascular risk factors, neurological focality, neuropsychological disorders, hypersignal intensities or silent infarcts in the MRI, late onset, absence of affective family background and, among the clinical characteristics, greater frequency of endogenicity data and significant disability as well as possible alteration of the executive function (depression-executive dysfunction syndrome[3]). He proposed a common pathophysiology for post-stroke depression, vascular depression and depression-executive dysfunction syndrome, based on the disruption of the prefrontal subcortical circuitry, with alteration of the monoaminergic neurotransmission circuits.

There are many references to the frequent refractoriness to psychopharmacological treatment in these patients. That is why ECT has arisen as an alternative[4], there being data on its effectiveness in patients resistant to or with intolerance to antidepressant drugs, similar to those of patients without alterations in the neuroimage[5], although subsequent works have not confirmed this superiority[6]. The ECT does not

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produce long-term cognitive deficiencies, this aspect improving parallelly with the depressive syndrome[7]. However, interictal delirium occurred more frequently. No advance of the cerebrovascular disease has been demonstrated in relationship with the hemodynamic changes during the ECT, nor appearance of changes in the structural neuroimage[8], this being considered a safe and effective technique in these patients[9].

CLINICAL CASE

The case of a 71 year old female patient is presented. She was widowed one year ago, has no children and is living in a residence. As somatic background, arterial hypertension, hypercholesterolemia and non-insulin dependent diabetes mellitus stand out. No personal nor familial background of mental illness have been identified. At 70 years, coinciding with the disease of her husband, she was admitted due to melancholic delusion, with anhedonia, guilty complex, agitation episodes, anorexia and insomnia, with delusional ideation of ruin, self-referentiality and olfactory hallucinations in additional to suicidal ideation. She was treated with paroxetine 40 mg/day, venlafaxine 300 mg/day, mirtazapine 15 mg/day, risperidone 4.5 mg/day and olanzapine 5 mg/day, without improvement. After, she received six sessions of bilateral ECT with ad integrum recovery. Two months later, she was readmitted with a similar picture, which resolved with 6 sessions of bilateral ECT. Brain MRI was done, and did not show acute lesions, although a lacunar lesion was reported in the left semioval center and minimum lesions of leukoaraiosis in posterior border territories (fig. 1). On discharge, she had significant memory disorders and received a second diagnosis, that of vascular dementia with melancholy. Two months after the discharge, she was readmitted due to a new relapse and was given seven sessions of ECT, that were spaced in an attempt to avoid the unfavorable consequences in the cognitive sphere. After this discharged, she came to the psychogeriatrics consultation where it was evaluated that she had no dementia. Three months later, she was readmitted with a picture of melancholic stupor: with mutism, hypoprosexia or reduced ability to attend or concentrate, bradypsychia, negativism, suspicious attitude, inhibited behavior, with pathological sadness and anhedonia: with delusional ideation of ruin and harm, without awareness of disorder and with basic habits affected (refusal to eat and for cares). The EEG presented a poorly integrated, poly-rhythmic trace with slow and diffuse irritative interference, predominately in the central areas, consistent with vascular etiology. In a new cerebral MRI, no changes were seen regarding the previous study. She was treated with bilateral ECT (eigth sessions, with 225 sec. of total electroencephalographic convulsion) reaching euthymic mood and presenting, as secondary to the ECT, time disorientation and memory difficulties (Mini-mental state exam 29/35[10]). Based on all of the above, she was diagnosed of major melancholic depression of vascular origin (DSM-IVTR: 293.83) and mild cognitive deterioration. On discharge, she was prescribed duloxetine 60 mg/day and quetiapine 100 mg/day, and was scheduled for maintenance ECT. In the neuropsychological evaluation in a clinical interview-remission period, selection of items from the PIEN Barcelona,

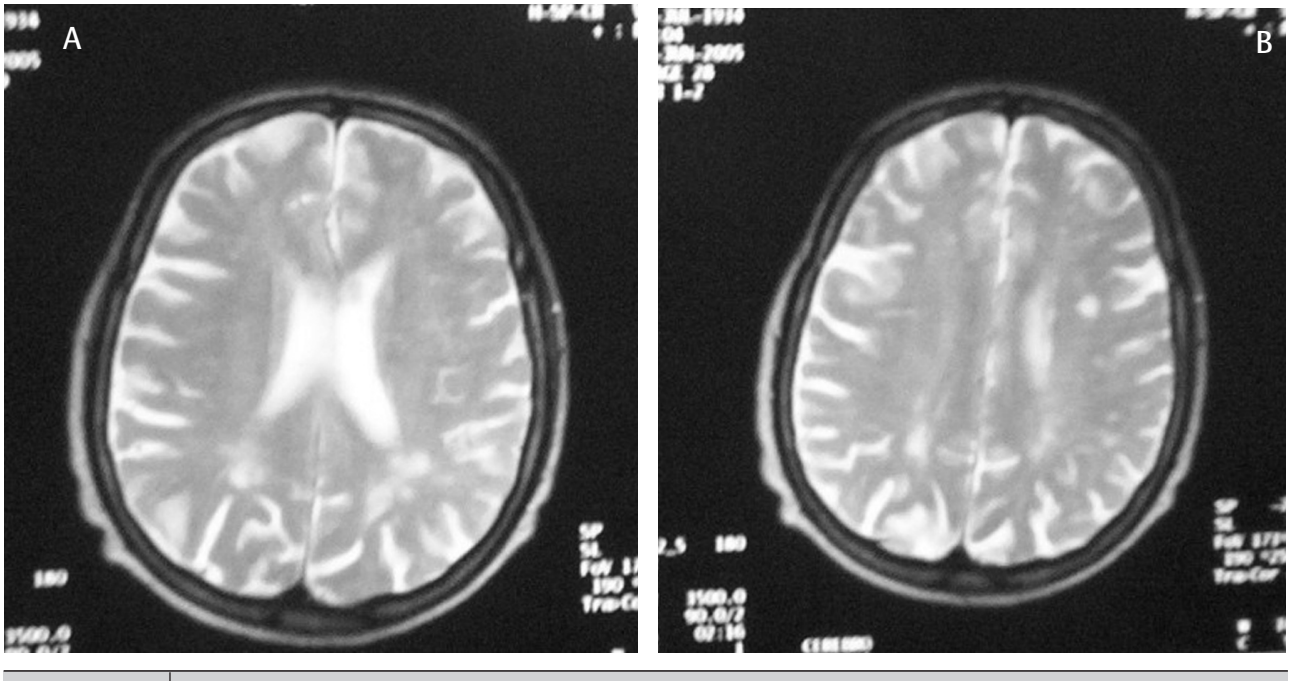

Figure 1 *MRI in T2. The left subcortical lacunar lesion* **(A)** *and leukoaraiosis lesions of the posterior border territories* **(B)**.

WAIS-III and WMS-III, Tower of Hanoi, Rey Complex Figure and Wisconsin Test, a deficit in phonetic evocation was obtained. There was conservation of immediate evocative memory with short-middle term evocative deficit and conserved recognition, executive dysfunction with planning and execution deficit and lack of cognitive flexibility. The patient was aware of the deficits (fig. 2). In the next 8 months, there were no relapses of the depressive symptoms, and she was maintained in the ECT program (with one session per month) and with antidepressant treatment with duloxetine 60 mg/day.

DISCUSSION

The clinical case presented would adapt to the definition of vascular depression proposed by Alexopoulos[2]. The finding of data of cerebrovascular disease in the MRI and existence of executive dysfunction demonstrated in the neuropsychological examination have been associated with poor response to psychopharmacological treatment (in this case with an evolution characterized by recurrent of depressive symptoms). Another characteristic that provides greater interest to the case is that there is a vascular depression with prominent melancholic symptoms. The concurrence of a significant life stressor does not decrease the validity to the hypothesis of organic etiology as it is understood that the cerebrovascular disease would not act as a single precipitating factor but rather facilitating and precipitating the depressive disorder.

This case also contributes a relevant observation regarding the treatment and prognosis, as it shows the efficacy of electroconvulsive therapy in cases of late onset depression with poor therapeutic response and great functional deterioration, thus avoiding therapeutic nihilism[1]. Treatment with ECT not only did not produce cognitive deterioration but produced a significant improvement in a patient who was previously diagnosed of dementia. During the treatment course, she had episodes of delirium that were adequately managed and resolved without complications. The possible relationship of executive deficits with electroconvulsive therapy could be argued, but recent observations have not supported this hypothesis (however, deficits in phonetic verbal fluency have been found)[12]. It is difficult to evaluate the contribution of treatment with duloxetine in the prevention of recurrences when the patient is maintained in a maintenance ECT program.

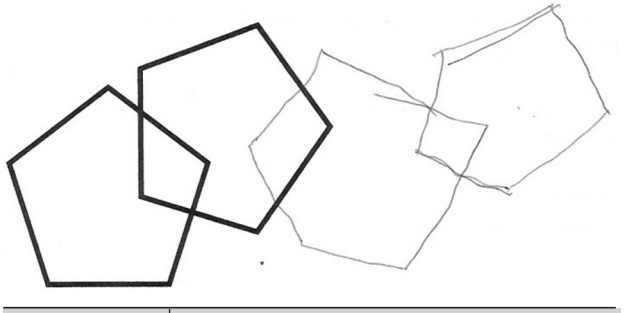

Figure 2 *Pentagons of the MEC. In spite of the correct execution the deficit of planning is observed when making the task.*

This clinical case, with its phenomenological and evolutive features, would support the theoretical construct of vascular depression that has contributed to its understanding and management.

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