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Validation of the Spanish-language version of Mini-Addenbrooke's Cognitive Examination as a dementia screening tool☆

Validación de la versión española del *Mini-Addenbrooke's Cognitive Examination* para el cribado de demencias

Dear Editor:

The number of people who seek medical care due to cognitive symptoms has increased significantly in recent years, mainly due to the increase in the population's life expectancy. Cognitive assessments are essential in the differential diagnosis of dementia,[1] since they contribute to treatment decision-making, which will affect the quality of life of patients and their families.

Quick cognitive screening tests are especially useful in our setting due to long waiting lists and limited resources that do not allow specialists to administer more thorough neuropsychological tests.

Addenbrooke's Cognitive Examination III (ACE-III)[2] has recently been validated in Spanish.[3] Both the current and previous versions of this test are widely used in memory units and dementia research centres around the world.[4] The ACE-III is known for its ability to detect dementia and differentiate between dementia subtypes.[5] However, its use is not as widespread as one might like since it takes 15-20 minutes to administer.

Hsieh et al.[6] have developed and validated the Mini-Addenbrooke's Cognitive Examination (M-ACE), a brief version of the ACE-III. These authors reduced the original version using Mokken scaling[7] and administered the new version to patients with Alzheimer-type and frontotemporal dementia and to healthy controls. The Mini-Mental State Examination (MMSE) was used as the gold standard.[8]

The M-ACE includes 5 items (orientation to time, semantic fluency, clock face drawing, immediate recall, and delayed recall) with a maximum score of 30. Maximum administration time is approximately 5 minutes. In the original validation study, scores $\leq$ 25/30 were identified as the cut-off point for dementia with both high sensitivity (85%) and specificity (87%). The M-ACE was found to be more sensitive than the MMSE and showed a less pronounced ceiling effect.

We have studied the psychometric properties of this new version of the ACE-III in our population using the same methodology applied by its authors and the complete sample recently gathered for the ACE-III validation study.

We selected items from the original questionnaire that are included in the M-ACE and created a new score. Of the 175 subjects comprising the sample, 92 were cognitively healthy controls (age: 77.0 $\pm$ 6.4 years; education: 8.4 $\pm$ 5.8 years) and 83 were patients (age: 78.4 $\pm$ 6.8 years; education: 7.4 $\pm$ 4.7 years) diagnosed with different types of dementia in mild stages: Alzheimer disease (46 patients, 55.4%), vascular dementia (4, 4.8%), mixed dementia (9, 10.8%), dementia associated with Parkinson's disease (11, 13.3%), Lewy body dementia (6, 7.2%); frontotemporal dementia (5, 6%), alcoholic dementia (1, 1.2%), and atypical parkinsonism with dementia (1, 1.2%). All participants were at least 65 and they were recruited from neurology departments at Hospital Clínico San Carlos in Madrid and Hospital de la Santa Creu i Sant Pau in Barcelona.

Our sample was significantly older and had a lower educational level than the sample in the study by Hsieh et al.[6] In the reliability analysis, the scale showed high internal consistency (Cronbach α = 0.828).

Results on the M-ACE were compared to those on the ACE-III and MMSE; clinical diagnosis of dementia was used as the factor for determining cut-off points (Fig. 1). With an area under the curve (AUC) = 0.94, M-ACE scores $\leq$ 16/30 were identified as the cut-off point for dementia with high levels of sensitivity (86.7%) and specificity (87.0%). This means that the M-ACE achieves better discrimination indices than the MMSE (AUC = 0.91; score $\leq$ 24/30; sensitivity = 88.0; specificity = 78.3) and the ACE-III (AUC = 0.92; score $\leq$ 65/100; sensitivity = 83.1; specificity = 80.4) (Table 1).

The M-ACE demonstrated a high diagnostic ability, with values above 85% for discrimination between healthy controls and subjects with mild dementia. The optimal cut-off point was 16.5, although a slightly higher point (17.5)

☆ Please cite this article as: Matias-Guiu J, Fernandez-Bobadilla R. Validación de la versión española del *Mini-Addenbrooke's Cognitive Examination* para el cribado de demencias. Neurología. 2016;31:646—648.

Table 1 Cut-off points for a diagnosis of mild dementia.

Score	M-ACE			Score	MMSE		Score	ACE-III	
	SEN	SPE	PPV (NPV)		SEN	SPE		SEN	SPE
19/20	96.4	64.1	70.8 (95.2)	27/28	97.6	40.2	68/69	90.4	72.8
18/19	92.8	75.0	77.0 (92.0)	26/27	96.4	55.4	67/68	86.7	75.0
17/18	92.8	82.6	82.8 (92.7)	25/26	89.2	68.5	66/67	85.5	77.2
16/17	86.7	87.0	85.7 (87.9)	**24/25**	88.0	78.3	**65/66**	83.1	80.4
15/16	80.7	89.1	87.0 (83.7)	23/24	78.3	87.0	64/65	81.9	83.7
14/15	75.9	92.4	90.0 (80.9)	22/23	71.1	92.4	63/64	80.7	84.8

The cut-off point found to be optimal is shown in bold.
NPV: negative predictive value; PPV: positive predictive value; SEN: sensitivity; SPE: specificity.

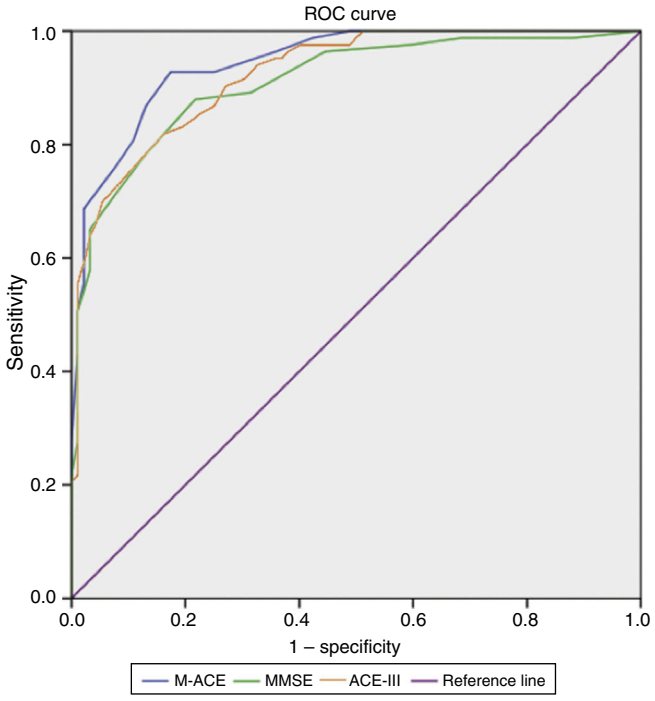

Figure 1 ROC curve for discriminating between patients with mild dementia and healthy controls.

would probably be recommendable to increase sensitivity, since this is the main purpose of a screening test. This cut-off point is below 25, the proposed threshold in the study of the English-language version. However, the original study only included patients with Alzheimer-type and frontotemporal dementia, who were also younger and more educated.

In conclusion, our study is the first to apply the M-ACE to a Spanish-speaking population, and it demonstrates the usefulness of this scale as a cognitive screening test. The short time required to administer the M-ACE suggests that this tool may be useful in centres with a greater care load or in less specialised centres. In addition, the higher sensitivity and specificity of the M-ACE compared to the ACE-III supports using the former even when the original long form is also administered. The M-ACE would therefore serve 2 purposes in neuropsychological assessment: it is a screening tool, especially when combined with the ACE-III, and also a short neuropsychological test since it includes the domains assessed by the ACE-III (attention, language, memory, verbal fluency, and visuospatial fluency).

Conflicts of interest

The authors have no conflicts of interest to declare.

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Cerebrospinal fluid hypotension as a cause of cortical vein thrombosis☆

Hipopresión de líquido cefalorraquídeo como causa de trombosis de una vena cortical

Dear Editor:

Intracranial hypotension syndrome (IHS) is due to imbalances in the production and absorption of cerebrospinal fluid (CSF). It is mainly caused by CSF leakage owing to structural weakness of the meninges alone or in combination with local trauma to the spinal dura mater. Symptoms appear when pressure drops below 65 mm H_2O.[1]

We present the case of a patient with CSF hypotension who experienced a complex partial seizure with secondary generalisation which led to a diagnosis of cortical vein thrombosis.

Our patient was a 40-year-old Ukrainian woman with a history of episodic migraine with aura beginning in adolescence. She had no other personal or family history of interest, epilepsy risk factors, oral contraceptive use, or prothrombotic risk factors. The patient visited our hospital due to constant headache which was more intense than her usual migraine episodes, and had different characteristics. She described the headache as oppressive and holocranial, and reported nausea and vomiting. Pain worsened when standing, sitting, and during Valsalva manoeuvers, and lessened in the decubitus position. Over time, pain became continuous and interfered with our patient's nightly sleep. A MRI scan revealed diffuse dura mater enhancement and increased size of the pituitary gland and the proximal cervical and intracranial venous plexi; these findings were all compatible with CSF hypotension. Two weeks later our patient experienced a self-limiting episode lasting 10 minutes and featuring paraesthesias of the right hand which progressed to the right side of her upper lip. A few hours later, she reported disorientation, paraesthesia, and loss of strength which progressed proximally from her right hand to her right forearm. On arriving at the emergency department, she displayed language impairment, right limb paresis, and psychomotor agitation. At the emergency department our patient experienced a self-limiting generalised tonic-clonic seizure lasting 2 minutes with no recurrences. The results of the neurological examination conducted after the post-critical phase were normal, and a new cranial CT scan revealed no new changes since the previous one. An EEG revealed slow activity in the left temporoparietal region. An additional MRI scan showed T1 hyperintensity and T2 hypointensity of a left parietal superior cortical vein at the level of the parasagittal convexity, compatible with cortical venous thrombosis. In addition, adjacent sulci were hyperintense on FLAIR, which suggested subarachnoid haemorrhage. Likewise, the signs of CSF hypotension seen in the previous MRI scan remained visible (Figs. 1 and 2). Symptoms improved significantly with conservative treatment (hydration, caffeine, intravenous corticosteroids, and antiepileptics). We decided not to administer anticoagulants due to the risk of bleeding associated with CSF hypotension.

Although it is not infrequent in clinical practice, headache secondary to CSF hypotension is still underdiagnosed.[2]

Orthostatic headache is the typical and most frequent manifestation of CSF hypotension, but we should never forget other less frequent, more severe symptoms, such as cerebral venous thrombosis. Cerebral venous thrombosis should be suspected in patients with CSF hypotension presenting altered headache patterns or focal neurological

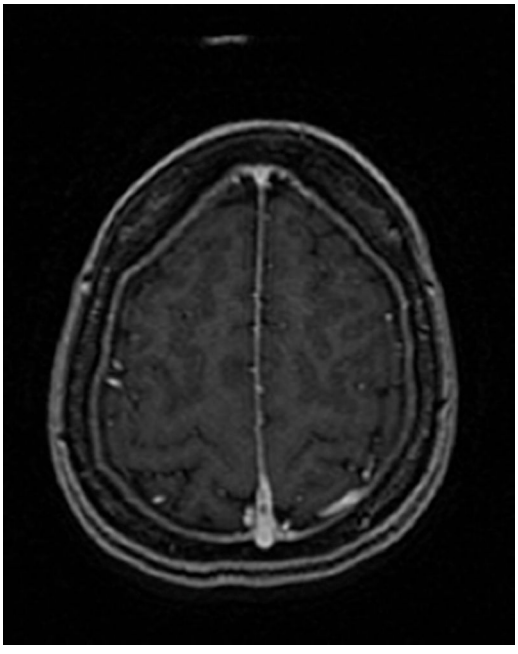

Figure 1 T1 hyperintensity (Ax FSE T1 + GD) in a left parietal superior cortical vein at the level of the parasagittal convexity, compatible with cortical venous thrombosis.

☆ Please cite this article as: Pérez Pérez A, Calvo Porqueras B, Porta Etessam J, Jorquera Moya M. Hipopresión de líquido cefalorraquídeo como causa de trombosis de una vena cortical. Neurología. 2016;31:648—649.