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Oxidative stress in neurological disease: Is it the cause, consequence, or trigger of a chronic progressive form?☆

Estrés oxidativo en la enfermedad neurológica. ¿Es causa, consecuencia o induce una forma crónica progresiva?

Dear Editor:

We have read with great interest the article published in October 2014 titled 'Oxidative stress in neurological diseases: cause or effect?'[1] and, as we find ourselves in agreement with the concepts expressed by the authors, we would like to present here some pertinent items of interest which have been published in the scientific literature over the past 25 years. Systemic oxidative stress (OS) is basically an imbalance between the production of such oxidants as reactive oxygen species (ROS) and reactive nitrogen species (RNS), and the capacity to neutralise their detrimental effects through both exogenous (diet and medication) and endogenous antioxidants.[2] The affected neural areas present different morphological and metabolic changes; likely, the individual characteristics of each group of neurons is what determines their strengths or weaknesses in the face of OS. Systemic OS, which can be measured in blood analyses, is increased in such entities as Alzheimer disease (AD), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS), chronic vascular encephalopathy (CVE), epilepsy, and Friedreich ataxia, among others.[3—8] In the same way, such clinical diseases as diabetes mellitus (DM), cancer, atherosclerosis, and chronic inflammation of the liver, kidney, and lung have been associated with systemic OS. Since the 1990s, a number of studies have described an association between ROS/antioxidants and AD, PD, and CVE. In addition, it has been shown that systemic OS is not identical in each of these entities. One of the most interesting findings was that OS was significantly lower in patients with both AD and type 2 DM than in patients with either AD or type 2 DM alone. The same was true for cognitive impairment.[9—12] This may be due to hyperinsulinaemia and/or to glycaemia-lowering drugs.[12] In conclusion, systemic OS must be located between the 2 extremes of cause or effect as it may be a factor contributing to pathological metabolic changes in a number of diseases. Reaching a balance between ROS and antioxidants may possibly diminish the risk of progression of these entities. Therefore, an emphasis should be made on the development of pharmacological studies aimed at minimising systemic OS.

Conflicts of interest

The authors declare that they have no conflicts of interest involving specific companies. All of the authors are familiar with the content of the manuscript and have given their permission to publish it. This manuscript has received no funding of any kind.

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Fatal outcome following thrombolysis for stroke secondary to infectious endocarditis☆

Evolución fatal tras trombólisis de un ictus secundario a endocarditis infecciosa

Dear Editor:

Acute stroke leading to intracranial haemorrhage is a rare complication of infectious endocarditis. Unfortunately, the available scientific evidence is limited to small case series; as a result, treatment decisions and recommendations have a low level of evidence. We present the case of a 49-year-old man who developed an intracranial haemorrhage with a fatal outcome after fibrinolytic treatment for infectious endocarditis.

The patient weighed 105 kg and his medical history included allergy to quinolones, dyslipidaemia (under treatment with simvastatin), atrial fibrillation for the past 10 years (treated with ASA 300 mg), bicuspid aortic valve stenosis, and moderate aortic insufficiency. The patient had developed a fever with no focus of infection 4 days previously and was prescribed cefuroxime and common antipyretics by his primary care physician. On the day of admission, he came to the emergency department of our hospital due to presyncope and right hemiparesis, leading to activation of code stroke. Upon examination, he showed a blood pressure of 95/67, a heart rate of 82 bpm, and a temperature of 37.3 °C.

Physical examination, which included cardiac auscultation, revealed a systolic murmur (grade I-II/VI); the pulmonary auscultation, abdominal examination, and the study of the extremities yielded normal results. During the neurological examination, the patient was disoriented and showed global aphasia, dysarthria, right hemiparesis which was more marked in the arm (strength 1/5) than in the leg (strength 3/5), and a NIHSS score of 17.

Following the stroke code protocol, the patient underwent a simple CT scan which showed no pathological findings; CT angiography revealed an occlusion in the distal portion of the M1 segment of the left MCA and perfusion CT, a 40% mismatch.

Since our patient met the criteria for thrombolytic treatment, he received a 9 mg bolus of rtPA at 2 hours and 5 minutes followed by an infusion of 81 mg. Treatment led to an improvement of paresis in the upper limb at 15 minutes and an NIHSS score of 14. During rtPA infusion, our patient began to shiver and his fever peaked at 39.6 °C. As a result, 2 blood samples were drawn for culture and he was administered antibiotics. The patient vomited and showed a deterioration in level of consciousness 30 minutes after rtPA infusion ended; another CT scan revealed multiple small

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