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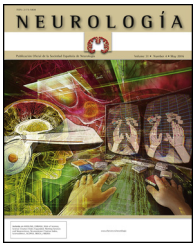

REVIEW ARTICLE

Malignant hemispheric infarction of the middle cerebral artery. Diagnostic considerations and treatment options☆

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Abstract
Introduction: Malignant hemispheric infarction (MHI) is a specific and devastating type of ischaemic stroke. It usually affects all or part of the territory of the middle cerebral artery although its effects may extend to other territories as well. Its clinical outcome is frequently catastrophic when only conventional medical treatment is applied.
Objective: The purpose of this review is to analyse the available scientific evidence on the treatment of this entity.
Development: MHI is associated with high morbidity and mortality. Its clinical characteristics are early neurological deterioration and severe hemispheric syndrome. Its hallmark is the development of space-occupying cerebral oedema between day 1 and day 3 after symptom onset. The mass effect causes displacement, distortion, and herniation of brain structures even when intracranial hypertension is initially absent. Until recently, MHI was thought to be fatal and untreatable because mortality rates with conventional medical treatment could exceed 80%. In this unfavourable context, decompressive hemicraniectomy (DHC) has re-emerged as a therapeutic alternative for selected cases, with reported decreases in mortality ranging between 15% and 40%.
Conclusions: In recent years, several randomised clinical trials have demonstrated the benefit of DHC in patients with MHI. This treatment reduces mortality in addition to improving functional outcomes.

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PALABRAS CLAVE
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Infarto maligno;
Edema cerebral;
Hemicraniectomía descompresiva

Infarto hemisférico maligno de la arteria cerebral media. Consideraciones diagnósticas y opciones terapéuticas

Resumen
Introducción: El infarto hemisférico maligno (IHM) constituye un tipo específico y devastador de ictus isquémico. Usualmente afecta el territorio completo de la arteria cerebral media, aunque a veces involucra además otros territorios, presentando evolución clínica frecuentemente catastrófica, cuando solo se aplica tratamiento médico convencional.
Objetivo: El propósito de esta revisión es analizar la evidencia científica disponible sobre el tratamiento de esta entidad.
Desarrollo: El IHM tiene una morbimortalidad elevada. Clínicamente se caracteriza por deterioro neurológico temprano y síndrome hemisférico severo. Su sello distintivo es el desarrollo de edema cerebral ocupante de espacio, entre el primer y tercer día del inicio de los síntomas. El efecto de masa provoca desplazamientos, distorsiones y herniaciones de las estructuras encefálicas, aún en ausencia inicial de hipertensión endocraneal. Hasta hace pocos años, el IHM era considerado una entidad fatal e intratable, ya que la mortalidad asociada al tratamiento convencional podía superar el 80%. En este contexto desfavorable, la hemicraniectomía descompresiva ha resurgido como una alternativa terapéutica eficaz en casos seleccionados, reportándose un descenso de la mortalidad entre un 15%-40%.
Conclusiones: En los últimos años diversos estudios clínicos aleatorizados han demostrado el beneficio de la hemicraniectomía descompresiva en los pacientes con IHM, la cual no solo ha disminuido la mortalidad sino que también ha mejorado los resultados funcionales.

Introduction

Cerebrovascular disease (CVD) is one of the most frequent causes of death and functional disability worldwide.[1] An epidemiological study conducted in Catalonia which analysed both types of CVD (ischaemic and haemorrhagic) revealed annual incidence rates of 218 and 127 per 100 000 population in men and women, respectively. The crude mortality rate at 28 days after stroke was 36%; 62.5% of these patients died out of hospital.[2]

The clinical consequences of ischaemic brain lesions depend on their extension and the eloquence of the involved parenchyma.[3] Ischaemic injuries range from clinically silent lesions to life-threatening infarctions; as a result, the associated morbidity and mortality varies greatly.[3–5] The in-hospital mortality rate for patients with middle cerebral artery (MCA) infarctions is 17%,[6] while a specific subtype, generically termed 'malignant', has shown a mortality rate of up to 80%.[7] Today, thanks to a multidisciplinary approach to MCA infarction and the advances in critical care, neuromonitoring, neuroimaging, and surgery, mortality has decreased dramatically, with rates between 25% and 40%.[5,7,8]

The concept of 'malignant MCA territory infarction', coined by Hacke et al.[9] in 1996, refers to a specific type of ischaemic stroke which usually affects the entire MCA territory and may also extend to other vascular territories. This type of infarction produces a mass-effect secondary to oedema, mainly cytotoxic, and has a fatal clinical outcome in the majority of cases.[10] It is most commonly caused by an embolic or thrombotic occlusion of the distal internal carotid artery or the main branch of the MCA (M1 segment). These occlusions are rarely recanalised, either spontaneously or after intravenous administration of tissue plasminogen activator.[5,7–9]

Although malignant MCA infarctions account for fewer than 10% of all supratentorial ischaemic strokes,[5,7–9] their huge impact on mortality and quality of life has led researchers to look for new therapeutic strategies. In the last decade, several research groups have shown excellent results in experimental models of ischaemia using moderate hypothermia[10]; these results, however, are yet to be confirmed in a clinical setting.[11–15]

On the other hand, recent randomised studies have underlined the benefits of decompressive hemicraniectomy in terms of survival and functional outcome.[16–19]

The purpose of this review article is to critically analyse the options available for treating malignant hemispheric infarction (MHI).

Pathophysiology of MHI

The pathophysiological substrate of MHI is cerebral oedema, which usually presents between the first and third days after symptom onset.[20] MHI exerts a mass effect which compresses, distorts, and herniates brain structures, resulting in neurological deterioration that may lead to death.[5,7–9] From a pathophysiological viewpoint, severe decreases in cerebral blood flow to the ischaemic territory compromise normal functioning of the Na^+-K^+-ATPase pump in cell membranes,