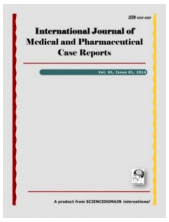

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Which is the Best Treatment in High Suspicion Embolic Stroke of Undetermined Etiology? A Case Report

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Authors' contributions

This work was carried out in collaboration between all authors. Author MGD wrote the draft of the manuscript. Authors MGD, AS, RG, EM, ES, SC designed the figures, managed literature searches and contributed to the correction of the draft. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aims: Presence of aphasia or cortical stroke on neuroimaging suggests an embolic etiology of the stroke. However sometimes it is difficult to discover the source of the emboli doing the decision of the optimal treatment a challenge.
Presentation of Case: 75-year old female was admitted on February 2007 due to a sudden left hemiparesis. Cranial MRI showed two right parietal cortical ischemic lesions. Cardiological and neurovascular studies were normal. Despite the high suspicion of embolic etiology, the patient was discharged home with clopidogrel and low molecular weight heparin (LMWH). Transesophageal echocardiography was later made and showed a minor atherosclerosis of the aortic artery. Patent foramen ovale and atrial aneurism were discarded. LMWH was stopped. On April 2007, the patient was admitted due to global aphasia and right hemiparesis. Cranial CT showed a cortical subacute ischemic stroke in the left middle cerebral artery territory. A complete etiological study (including systemic study) was normal. Anticoagulation was initiated. The patient has remained stable since

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then (mRS 3: moderate disability).
Discussion: The American and European guidelines do not include the possibility of highly probable embolic stroke of undetermined cause. In these patients, anticoagulation treatment might increase the possibility of intracranial hemorrhages. On the other hand, antiplatelet treatment does not prevent the recurrence of embolic events with devastating consequences.
Conclusion: This case illustrates the controversy about antithrombotic treatment when the etiology remains unknown in highly suspicious embolic stroke. Even if the source of stroke remains unknown, an oral anticoagulation should be carefully considered in these patients.

Keywords: Embolic stroke; anticoagulation therapy; recurrent stroke; undetermined stroke.

1. INTRODUCTION

The presence of aphasia or cortical stroke on the neuroimaging suggests and embolic etiology of the stroke. However sometimes it is difficult to discover the source of the emboli making the decision of the optimal treatment a challenge. The presence of an embolic source such as atrial fibrillation is an indication for anticoagulation. The absence of such an embolic source will be an indication for antiplatelet treatment.

2. PRESENTATION OF CASE

A 75-year old female was admitted on the 11th of February 2007 due to a sudden left hemiparesis. The patient had asthma, familiar dyslipidemia and a recent diagnosis of high blood pressure, treated with enalapril and budesonide. Neurological status in Emergency Department showed a left facio braquio (1-2/5) crural (2/5) hemiparesis and hypoestesia (National Institute Health Stroke Scale (NIHSS) of 11). Cranial CT was normal. Intravenous (IV) fibrinolysis was performed and clinical improvement was seen (NIHSS of 2 at 12 h). Cranial MRI showed two right parietal cortical ischemic lesions (Figs. 1A and B). Transthoracic echocardiography and Holter were normal. Transcranial and carotid duplex echography, and intra and extracranial CT angiography were normal (Fig. 1C). Despite the high suspicion of embolic etiology, and due to the previous results the patient was discharged home with clopidogrel and nadroparin (Fraxiparin 0.6 cc Sc/d). Transesophageal echocardiography was later made and showed left ventricle hypertrophy, and a minor atherosclerosis of the aortic artery. This excluded soft or complicated plaques of the aortic arch. Patent foramen ovale and atrial aneurism were also discarded. Low molecular weight heparin (LMWH) was stopped.

On the 2nd of April 2007, the patient was admitted due to language disturbance. Neurological status showed a global aphasia, right hemianopia, right braquio (3/5) crural (2/5) hemiparesis and previous left arm paresis (4+/5). Cranial CT showed a cortical subacute ischemic stroke in the left middle cerebral artery territory (Fig. 1D). IV thrombolysis was rejected and anticoagulation was initiated. A repeated holter and intra and extracranial CT angiography were normal. A complete blood analysis (including serology, immunology, lupus anticoagulant and anticardiolipin antibodies) was normal and was repeated at 3 months with normal levels of protein S, protein C, antithrombin III and factor V Leyden. A prothrombotic state was excluded. On a later date a complete systemic study was performed in order to exclude tumoral pathology. A control cranial MRI in August 2007 showed a bilateral chronic ischemic stroke (Fig. 1E and F). The patient has remained stable since then (mRS 3: moderate disability).

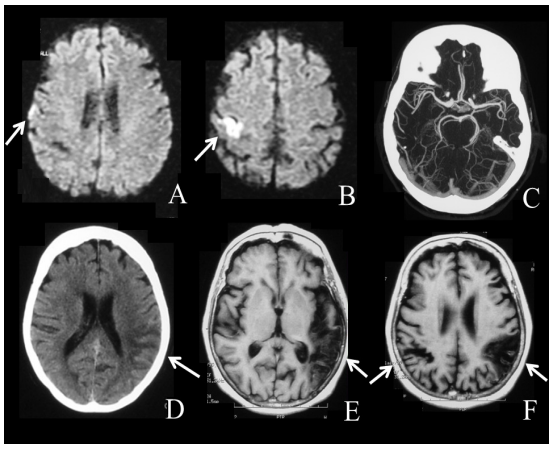

Fig. 1. A and B. Cranial MRI, diffusion sequences showed two right cortical ischemic lesions (white arrows). C. Intracranial CT angiography showed normal vessels. D. Cranial CT on second admission showed a left ischemic stroke (white arrow). E and F. Cranial MRI, T1 sequences showed a chronic bilateral ischemic stroke (White arrows)

3. RESULTS AND DISCUSSION

A sudden onset to maximal neurological deficit, Wernicke´s aphasia or global aphasia without hemiparesis has been described as a classic cardioembolic presentation [1]. In addition, lacunar clinical presentations, a lacunar infarct and multiple lacunar infarcts make cardioembolic origin unlikely [1]. In our case, clinical evolution at first admission with a sudden onset of hemiparesis and a striking clinical improvement made an embolic source the most probable etiology. The presence of cortical stroke in cranial MRI pointed toward this possibility [2]. In this sense determination of biomarkers, such as brain natriuretic peptide o D-Dimer, is a new and promising clinical research line for the diagnosis of embolism in cases of ischemic stroke of undetermined etiology [3]. However, cardiological study excluded atrial defect, thrombus, valvular pathology or atrial fibrillation (AF); and neurovascular study excluded vascular pathology. A systemic evaluation, complete blood analysis and thrombophilic study were normal some months later. So ischemic stroke of unusual cause subtypes was excluded, emphasizing the need to distinguish these infrequent etiologies from other ischemic stroke subtypes, which have a different treatment approach and outcome [4]. In this case, a presumed embolic stroke was managed with antiplatelet treatment and LMWH. Recurrence of a stroke, this time in the contralateral hemisphere, supported the embolic etiology.

The American and European guidelines do not include the possibility of highly probable embolic stroke of undetermined cause. The European Stroke Organization [5] recommends oral anticoagulation after ischemic stroke associated with AF, and in cardioembolic stroke unrelated to AF if the risk of recurrence is high. Anticoagulation should not be used after non-cardioembolic ischemic stroke, except in some specific situations, such as aortic atheromas, fusiform aneurysms of the basilar artery, cervical artery dissection, or patent foramen ovale in the presence of proven deep vein thrombosis or atrial septal aneurysm. American guidelines [6] include atrial fibrillation, acute miocardial infarction, cardiomyopathy, valvular heart disease or prosthetic heart valves as indications for oral anticoagulation, and antiplatelet treatment is recommended in noncardioembolic stroke. The clinical case described here, is not included in other specific conditions. Thus, more clinical trials focusing on this specific issue are highly necessary [7].

4. CONCLUSION

This case illustrates the controversy about antithrombotic treatment when the etiology remains unknown in highly suspicious embolic stroke. In these patients, antiplatelet treatment does not prevent the recurrence of embolic events with devastating consequences. On the other hand, anticoagulation treatment might increase the possibility of intracranial hemorrhages. In these cases, a wide etiological study is mandatory. Even if the source of stroke remains unknown, an oral anticoagulation should be carefully considered in these patients.

CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case repots and accompanying images.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Arboix A1, Alió J. Cardioembolic stroke: clinical features, specific cardiac disorders and prognosis. Curr Cardiol Rev. 2010;6(3):150-61.
2. Rovira A, Grivé E, Rovira A, Alvarez-Sabin J. Distribution territories and causative mechanisms of ischemic stroke. Eur Radiol. 2005;15(3):416-26.
3. Santamarina E, Penalba A, García-Berrocoso T, Delgado P, Quintana M, González-Alujas T, et al. Biomarker level improves the diagnosis of embolic source in ischemic stroke of unknown origin. J Neurol. 2012;259(12):2538-45.
4. Arboix A1, Bechich S, Oliveres M, García-Eroles L, Massons J, Targa C. Ischemic stroke of unusual cause: Clinical features, etiology and outcome. Eur J Neurol. 2001;8(2):133-9.

5. The European Stroke Organization (ESO) Executive Committee; ESO Writing Committee. Guidelines for management of ischemic stroke and transient ischemic attack 2008. Cerebrovasc Dis. 2008;25(5):457-507.
6. Kernan WN, Ovbiagele B, Black HR, Bravata DM, Chimowitz MI, Ezekowitz MD et al. On behalf of the American Heart Association Stroke Council, Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, and Council on Peripheral Vascular Disease. Guidelines for the Prevention of Stroke in Patients with Stroke and Transient Ischemic Attack: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45(7):2160- 236.
7. Hart RG, Diener H-C, Coutts SB, Easton D, Granger CB, O'Donnell MJ, et al for the Cryptogenic Stroke/ESUS International Working Group. Embolic strokes of undetermined source: the case for a new clinical construct. Lancet Neurol. 2014;13(4):429–38.

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