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Case report

Cortical laminar necrosis following myocardial infarction

Simona Lattanzi *, *Mauro Silvestrini*, *Leandro Provinciali*

Neurological Clinic, Department of Experimental and Clinical Medicine, Marche Polytechnic University, Ancona, Italy

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ABSTRACT

The cortical laminar necrosis (CLN) is a permanent injury characterized by the selective delayed necrosis of the cerebral cortex, mainly of the third layer, and usually greater in the depths and sides of the sulci than over the crest of the gyri. The damage involves all cellular components – either neurons, glia cells and blood vessels – and results in a focal cortical band of pan-necrosis detectable in late sub-acute or chronic stages of reduced energy supply to the brain. The CLN has been described in different conditions as hypoxia, hypoglycemia and status epilepticus. At brain CT or MR scans it appears with pathognomonic highly hyperdense or T1-hyperintense lesions following the gyral anatomy of the cerebral cortex. We reported a case of CLN associated to myocardial infarct and discussed the underlying mechanisms.

1. Introduction

The cortical laminar necrosis (CLN) is a specific type of cortical damage which usually results from generalized and critically long-lasting cerebral energy depletion as in anoxic encephalopathy. Other etiologies like hypoglycemia, status epilepticus and immunosuppressive chemotherapy have been involved. We reported a case of CLN associated to myocardial infarction and discussed the pathogenic mechanisms.

2. Case description

A 48-year old Caucasian woman presented to the Emergency Department, after a 2 days history of central chest pain, because of the sudden onset of left-sided weakness and hypoesthesia. Her past medical history was unremarkable unless the smoking habit (about 15 cigarettes a day for 15 years). Electrocardiogram demonstrated the ST elevation in lead I and aVL with reciprocal ST segment depression in lead V1, V2, V3, V4; biochemical analysis showed the troponin-I level of 30.54 ng/ml and CK-MB of 82.7 ng/ml. The brain CT scan disclosed an area of hypodensity, loss of gray-white matter differentiation and effacement of sulci in the right sylvian region. Neurological examination showed left hemiparesis and hemihypoesthesia associated to dysarthria (National Institutes of Health Stroke Scale [NIHSS] score = 12). Postero-lateral myocardial infarction and acute ischemic stroke were diagnosed. Coronary angiography revealed an atheroma occluding the left circumflex coronary artery; echocardiogram showed postero-lateral and apical akinesia

* *Corresponding author at*: Neurological Clinic, Department of Experimental and Clinical Medicine, Marche Polytechnic University, Via Conca 71, 60020 Ancona, Italy Tel.: +39 071 5964438; fax: +39 071 887262.
E-mail address: alfierelattanzisimona@gmail.com (S. Lattanzi).

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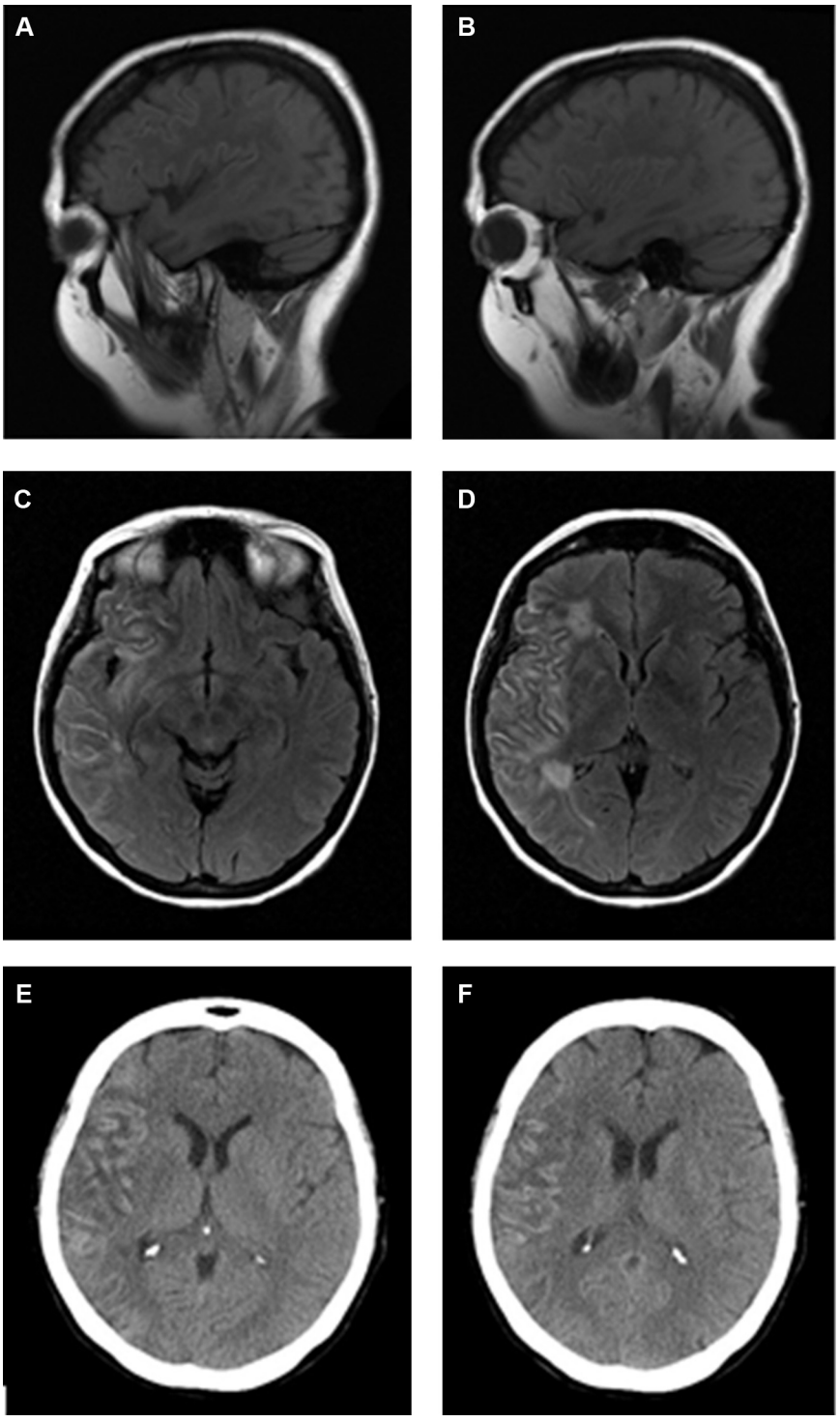

Fig. 1 – Neuroimaging of cortical laminar necrosis. Sagittal T1-weighted (A and B) and axial Fluid Attenuated Inversion Recovery (C and D) MRI sequences performed at 11 days from stroke onset showed curvilinear increased signal intensity following the gyral pattern of the cortex of the right fronto-insular and temporo-parietal ischemic regions. Brain CT scans (E and F) performed on day 22 displayed gyriform cortical hyperdensities in the ischemic area, prominently in fronto-insular and temporal lobes.