Case report

Cortical laminar necrosis following myocardial infarction

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

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

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
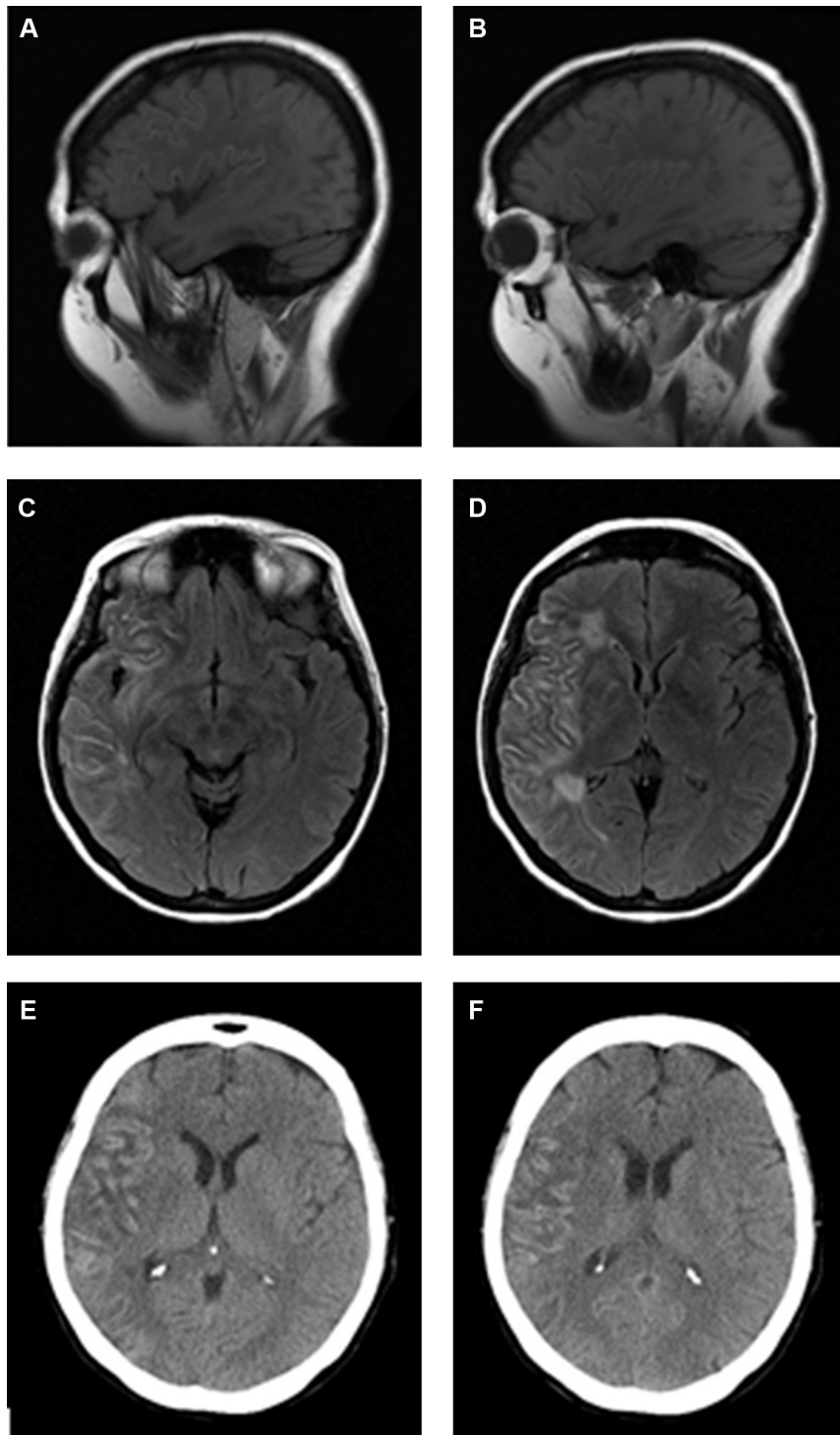
Fig. 1 – Neuroimaging of cortical laminar necrosis. Sagittal T1-weighted (A and B) and axial Fluid Attenuated Inversion Recovery (C and D) MRI sequences performed at 11 days from stroke onset showed curvilinear increased signal intensity following the gyral pattern of the cortex of the right fronto-insular 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.

associated to inferior segmental hypokinesia with severe impairment of left ventricular systolic function (estimated ejection fraction 30%), and two pedunculated thrombi in the proximal tract of the ascending aorta. The CT scans confirmed the presence of aortic thrombi (diameter of 15 and 7 mm, respectively) and detected the occlusion of the right internal carotid artery after its origin from the carotid bulb. A comprehensive work-up for thrombophilia turned out to be negative. The brain MRI performed on day 11 from stroke onset showed bright signals on DWI sequences and corresponding low apparent coefficient values in the right sylvian region, as for sub-acute ischemia, and high intensities on T1-weighted and Fluid-Attenuated Inversion Recovery (FLAIR) images (Fig. 1A–D) following the gyral anatomy of the cerebral cortex in the injured area, as for CLN. Gyriform cortical hyperdensities were still evident on brain CT scans performed on days 22 (Fig. 1E and F) and 42, but no longer seen at 4 months. The patient was transferred to the Neurological Rehabilitation Unit two weeks after the stroke onset. Despite the intensive treatment, severe sensory-motor deficits (NIHSS = 10) and disability still lasted at four months.

3. Discussion

Brain is one of the most vulnerable organs to hypoperfusion with the gray matter being much more exposed than the white substance because of its higher metabolic demand and denser concentration of receptors for the excitatory aminoacids which are released after the anoxic ischemic insult and mediate the excitotoxicity. The 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 which becomes detectable in the late sub-acute or chronic stages of reduced energy supply to the brain [1,2]. It has been described in both adult and pediatric age and related to hypoxic and hypoglycemic encephalopathy, status epilepticus (SE), immunosuppressive therapy (cyclosporine A, FK 506 and tacrolimus), antineoplastic treatments (methotrexate and vincristine), meningoencephalitis, CNS lupus erythematosus, Moya-Moya disease, hyponatremia correction and citrullinemia [3–6].

The CLN presents as linear hyperdensity on CT scans and high intensity on T1-weighted and FLAIR images following the gyral anatomy of the cerebral cortex and corresponding to the neuronal damage, the reactive gliosis, the deposition of fat-laden macrophages, the accumulation of denatured proteins in dying cells, and not to hemorrhagic infarction as initially thought [4,5].

Although the etiologies of the CLN may be different, it usually accompanies and results from the generalized, rather than circumscribed, vascular or metabolic insults to the brain. The most common causes are hypoxic-ischemic encephalopathy and watershed infarction as, when mean arterial blood pressure falls below the lower limit of cerebral autoregulation, it compromises the brain metabolism and energy production. When associated to immunosuppressive and antiblastic agents, CLN is thought to be the effect of the acute hypertensive encephalopathy and the direct endothelial or neuronal toxicity [4]. Similarly, in hypoglycemic encephalopathy the cerebral impairment, although it might be more severe in the more active and thus more susceptible regions, is global [7]. The CLN may also complicate SE with a diffuse or, less frequently restricted, localization: even when focal, however, the damage is mainly due to the increase in the cerebral demand for glucose and oxygen, which in turn leads to the hypermetabolic neural necrosis, rather than to the deficit in the cerebral perfusion like in stroke [8].

The main novelties of the reported case lay in either the occurrence of the CLN in the setting of a circumscribed ischemic damage to the brain and the combined conjunction of systemic and local deficits of the vascular supply. The patient developed firstly the myocardial infarction, which was associated to the severe reduction of the left ventricle function, and than the thrombo-embolic ischemic stroke. It is reasonable to assume that the underperfusion of the cerebral infarcted region, due to the impairment of the cardiac activity and presumably lasting over the recanalization of the occluded cerebral artery, resulted in a critically sustained depletion of energy to the ischemic cortex and thus created the appropriate conditions for the laminar necrosis. The hampered brain perfusion due to the occlusion of the cerebral artery – the *local* vascular defect – added to the limited cardiac output due to the myocardial infarction – the *systemic* vascular deficiency – could explain the occurrence of the CLN. It is plausible that neither the reduced cardiac activity nor the cerebral artery occlusion taken alone would have caused it.

4. Conclusion

The CLN usually results from the critically long lasting and diffuse depletion of oxygen or glucose to the brain, but it may also be ascribable to localized vascular abnormality when a systemic perfusion defect coexists, as we reported. Additionally, there is accruing evidence that the CLN could be a negative prognostic index, invariably associated to persistent vegetative state or brain death in anoxic encephalopathy [9]. We could tentatively hypothesize that CLN may be a clue for predicting poor neurological recovery even in localized brain infarct.

Conflict of interest

None declared.

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Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical

Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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