

Cortical Laminar Necrosis in Computed Tomography Scan: A Case Report

Necrosis laminar cortical en tomografía computarizada: presentación de caso

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Summary

Introduction: Cortical laminar necrosis is an imaging term used to describe gyriform hyperdense lesions in the cortical region that can be appreciated on the CT (Computed Tomography). The etiology of this finding involves brain energy depletion, which can be derived from multiple pathologies, that mainly lead to hypoxia or metabolic alterations. **Case report:** the case of a 3-month-old female patient is presented; she was taken to the emergency department with a 1-week clinical manifestations consisting of thermal rises, dyspnea, cyanosis coldness in extremities, tachypnea, tachycardia and edema. An echocardiography diagnosed myocarditis of probable viral origin. Subsequently, the patient presented cardiac arrest and prolonged shock that required cardiopulmonary resuscitation and admission to neonatal ICU. A cerebral CT scan taken 1 week after these events evidenced imaging findings compatible with cortical laminar necrosis. **Discussion:** There are no epidemiological descriptions of this finding, however, an increase has been theorized due to the high survival rates of patients suffering from ischemic hypoxic pathologies. Although findings of cortical laminar necrosis are typical of MRI, they can also be seen on CT scan, where they typically show as a subtle cortical hyperdensity, and the most striking findings are associated to a worst prognosis. **Conclusion:** Disclosure of these type of radiological images is necessary in order to promote more accurate prognoses in patients in whom these findings are appreciated.

Resumen

Introducción: Necrosis laminar cortical es un término imaginológico que se usa para describir lesiones de localización cortical, hiperdensas de distribución giriforme, en tomografía computarizada (TC). La etiología de este hallazgo involucra depleción de energía cerebral, la cual puede derivarse de múltiples patologías, que conllevan principalmente a hipoxia o alteraciones metabólicas. **Presentación de caso:** Es el caso de una paciente femenina de 3 meses de edad quien fue llevada a urgencias de la institución de los autores con un cuadro clínico de 1 semana de evolución consistente en elevación de la temperatura, disnea, cianosis, frialdad en extremidades, taquipnea, taquicardia y edema. Por medio de ecocardiografía se diagnosticó miocarditis de probable origen viral. Posteriormente, la paciente sufrió paro cardiorrespiratorio y choque prolongado que requirió reanimación cardiopulmonar e ingreso a UCI neonatal. Una TC cerebral tomada 1 semana después de estos eventos evidenció hallazgos compatibles con necrosis laminar cortical. **Discusión:** No hay descripciones epidemiológicas sobre este hallazgo; sin embargo, se ha teorizado un incremento en su frecuencia debido a la alta tasa de supervivencia de pacientes que padecen patologías hipóxico-isquémicas. A pesar de que los hallazgos de necrosis laminar cortical son descritos para la resonancia magnética (RM), también se pueden apreciar en la TC, donde típicamente se evidencian como una alta densidad cortical sutil, y los hallazgos más llamativos están relacionados con un peor pronóstico. **Conclusión:** Es necesaria la divulgación de este tipo de imágenes radiológicas, con el fin de promover la realización de pronósticos más acertados en pacientes en quienes se aprecien estos hallazgos.

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Introduction

Cortical laminar necrosis, also known as pseudolamellar necrosis, is an imaging term that describes hyperdense, gyriformly distributed cortical lesions on high signal magnetic resonance imaging (MRI) T1-enhanced computed tomography (CT) scans in the subacute or chronic phase of brain damage in some foci involving the cerebral cortex and white matter.

This finding is caused by depletion of brain energy, which results in necrosis of a particular cortical lamina. *Cortical laminar necrosis and pseudolamellar necrosis* are terms used classically interchangeably despite the difference in their histological meaning (1). The predominant cause is hypoxia, metabolic disorders such as hypoglycemia, intoxications, encephalopathies hypoxic-ischaemic and renal or hepatic failure (2).

The objective of this publication is to describe the case of a patient 3-month-old, who was seen with CT scans compatible with cortical laminar necrosis secondary to cardiorespiratory arrest related to myocarditis.

Case report

Female patient, 3 months old, arrives with her parents at consultation in the emergency department with a clinical picture of approximately 1 week of evolution consisting of thermal increases not quantified, concomitant with dyspnea and cyanosis, no history of importance; on physical examination he found tachypnea, tachycardia, edema and coldness in the extremities.

Because of this clinical picture, myocarditis was suspected, probably of viral origin. Echocardiography confirmed the diagnosis of severe myocarditis. As a consequence of this pathology, the patient developed cardiorespiratory arrest, accompanied by prolonged shock, for which she was given cardiopulmonary resuscitation and transferred to the neonatal intensive care unit (ICU) to be stabilized.

One week after her admission to the neonatal ICU, and as a result of the depressed state of consciousness and the time she spent in cardiorespiratory arrest, she underwent a brain CT scan to evaluate possible injuries resulting from prolonged hypoxia due to cardiorespiratory arrest (Figures 1 and 2). CT showed multiple areas of high density that follow a linear gyriform pattern in the cortical region, located in frontal, occipital and mainly parietal lobes; bilateral, with right predominance. At the same time, low density areas were observed perilesionally, mainly in the right parietal lobe. These findings are compatible with anoxic encephalopathy related to cortical laminar necrosis with white matter lesion resulting from cardiorespiratory arrest and prolonged shock caused by cardiomyopathy.

After two weeks in the neonatal ICU, without favorable neurological evolution, the patient suffered a second cardiorespiratory arrest that did not respond to CPR and died.

Discussion

Cortical lamellar necrosis is a specific type of cortical damage that usually results from widespread and critically long-lasting depletion of brain energy, as can occur in anoxic encephalopathy. Among the most frequently attributed etiologies are hypoglycemia, epileptic status, immunosuppressive therapy (cyclosporine A and tacrolimus), intoxications, antineoplastic therapy (metrotexate and vincristine), systemic lupus erythematosus, Moyamoya's disease, correction of hyponatremia, citrulinemia, congenital aplasia cutis and compromised renal or hepatic function; other less common etiologies must also be taken into account: viral infections, such as dengue virus encephalitis (2).

In the review of literature no epidemiological description of this imaging finding was found; however, an increase in its frequency has been perceived as a consequence of the progression and diffusion of cardiopulmonary resuscitation techniques, given that they result in an increase in patients surviving with sequelae of hypoxic-ischaemic encephalopathy (3). A series of cases in pediatric patients described by Niwa and collaborators, found that the causes, in order of

frequency were: acute myocardial infarction, Moyamoya's disease, hypoxic-ischaemic lesion, meningoencephalitis and shaken baby syndrome (1).

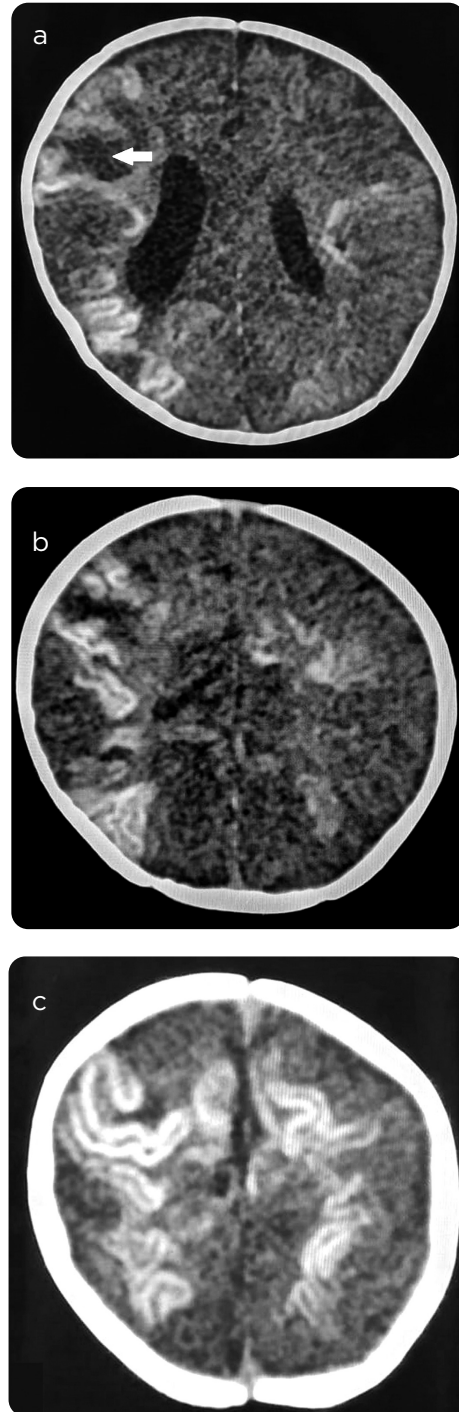


Figure 1. Axial cut CT showing areas of high density with gyriform pattern of predominantly right bilateral involvement compatible with cortical laminar necrosis. a) The arrow points to low perilesional density in the area with greatest involvement of the white substance.

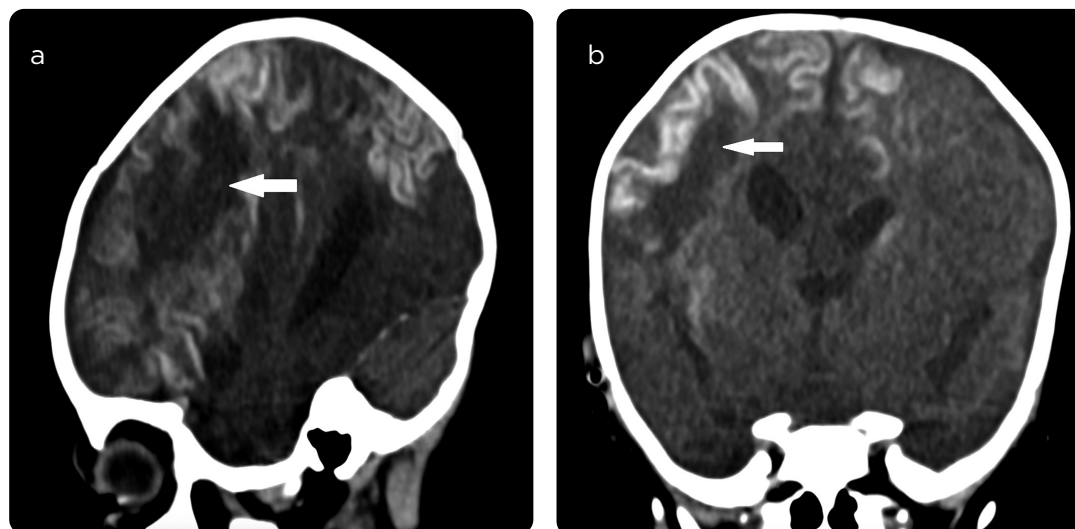


Figure 2. CT scan, sagittal and coronal sections. High density cortical areas with gyriform pattern compatible with cortical laminar necrosis. a) The arrow points to the low perilesional density due to the associated involvement of the white substance (arrows).

This pattern of injury is one of the imaginary manifestations of hypoxic-ischaemic encephalopathy. The histological studies show that the grey substance is more susceptible to ischemic necrosis than white, due to hypoperfusion, and the third layer of the crust is the most vulnerable, with usually more damage on the sides and depths of the grooves, which on the ridges of the circumvolutions (4).

The proposed mechanisms for cortical laminar necrosis are cytolysis, necrosis and edema followed by resorption and phagocytosis of the necrotic material, resulting in the presence of lipid-laden macrophages (5), which has been correlated with the imaging findings in MRI so particular to this type of necrosis. The theory that cortical laminar necrosis is related to areas of hypoperfusion has been proposed, in that it overlaps in its pathological findings with borderline cerebral infarctions; however, the underlying physiopathological mechanisms of this radiological finding have not been clarified (6). It should be noted that the brain lesions produced are known thanks to studies from of autopsies, but few descriptions have been published about the findings in neuroimaging (7).

In MRI, cortical laminar necrosis is characterized by high signal areas in T1-weighted images located in the cortex, which gives it its characteristic linear appearance. As for the T2/FLAIR sequences, their images show intermediate or high signal where the lesions are seen in sequences with T1 information (5). In most cases, only a high subtle cortical density is seen on CT (8). The high cortical density is first seen typically after 2 weeks, peaks after 1 to 2 months and usually resolves after 6 months. The finding of cortical laminar necrosis on tomography is usually subtle; however, in the most severe cases of this pathology, much more notable images are evident, a circumstance that has associated this finding with a worse prognosis (4). In this particular case, the lesions seen on CT are compatible with poor prognosis laminar cortical necrosis, described as being located in the high-density gyriform cortical region

Conclusion

A case of cortical laminar necrosis diagnosed by CT scan has been presented in a 3-month-old patient with myocarditis, complicated by cardiorespiratory arrest and prolonged shock, who died shortly after a

second cardiorespiratory arrest. In our environment it is a little known imaging finding, which should be disseminated to facilitate an appropriate diagnosis and prognosis according to the radiological findings and the condition of the patient.

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