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Challenge of Managing a Recurrent Ischemic Stroke In a Septuagenarian Presenting With Laminar Cortical Necrosis on Brain MRI

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Cortical laminar necrosis; Recurrence stroke; Hypoglycemia; Status epilepticus

1. Abstract

1.1. Introduction: Cortical laminar necrosis (CLN) corresponds to neuronal ischemic remodeling secondary to hypoxia. In the past, it was underdiagnosed and unrecognized. Nowadays, thanks to the accessibility of MRI, it is now possible to make the diagnosis of CLN in tropical environments.

1.2. Objective: The authors identify through this case illustration, the clinical profile and the characteristics of the cerebral lesion on encephalic imaging. The interest of this observation is to evaluate the predictive factors of occurrence and the impact on the vital and functional prognosis of this patient victim of a recurrent of stroke.

1.3. Observation: Septuagenarian hospitalized for a recurrence of severe ischemic stroke complicated by cortical laminar necrosis in the context of hypoglycemia with status epilepticus.

1.4. Conclusion: Ischemic stroke exposes you to the risk of occurrence of laminar necrosis, especially if it is a recurrence. The prognosis is pejorative, hence the importance of urgently identifying the predictive factors and the urgent use of appropriate early aggressive treatment in order to avoid the risk of occurrence of cerebral hypoxia.

2. Introduction

Laminar cortical necrosis corresponds to neuronal ischemia associated with a glial reaction and laminar deposition of lipid-rich macrophages. It occurs following cerebral hypoxia, with damage to the deep layers of the cortex [6, 7]. The clinical picture is dominated by altered consciousness, sometimes by motor or sensory deficit [9]. The diagnosis is based on brain magnetic resonance imaging (MRI) to detect and monitor its evolution. On brain MRI, early cytotoxic edema causes a high signal with low apparent diffusion coefficient (ADC) in the affected cortical area. Cortical enhancement can also be observed, after two weeks of development in general. The intrinsic increase in T1 signal is the most specific imaging feature. Curvilinear T1 hyper signals signaling laminar necrosis become more evident 3 to 5 days after the stroke, after two weeks in general, with peak intensity around one month. They fade slowly, most often within three months or more [2, 8]. It can sometimes remain visible for more than a year after the stroke [13]. This high T1 signal is thought to be due to the accumulation of denatured proteins in dying cells and/or lipid-laden macrophages. It is important to note that it does not represent the presence of hemorrhage or calcium. T2-weighted images show increased signal [12]. The prognosis is poor, with high morbidity and mortality. There is no specific curative treatment, it mainly depends on the etiology [1].

2.1. Objectives

In Côte d'Ivoire, no description in the literature has been reported and very few African studies published on this case. The authors, through this case illustration in a patient presenting cortical laminar necrosis and a permanent neurological deficit following a status epilepticus, hypoglycemia and a recurrent ischemic stroke, would like to discuss the clinical, neuroradiological presentation and possible mechanisms leading to cortical laminar necrosis in this patient, as well as the implications of prompt and aggressive

treatment in similar cases.

Observation

2.2. Identity and History of the Patient

This involved K.A., a 71-year-old right-handed patient with high blood pressure who had already been hospitalized in 2020 for an ischemic stroke of undetermined etiology with right hemicorporeal motor sequelae (Rankin score 2). She had poor compliance with secondary prevention treatment. She was admitted in July 2023, in the neurology department of the teaching Hospital of Cocody-Abidjan for a sudden onset of awareness impairment.

2.3. History of the Disease

Interviewing those close to her reported that 5 days before her admission to the neurology department, in a non-febrile and atraumatic context, she had incoherent speech followed by a status epilepticus with right focal onset with secondary generalization. The clinical course is marked by the persistence of epileptic seizures with a high blood pressure at 160/100 mmHg and severe hypoglycemia at 0.28 g/l. During her transfer, she would have benefited from an intravenous (IV) infusion of 10% glucose serum and nicardipine using an electric syringe pump at a dose of 1 mg/h as an emergency. Within 48 hours after emergency treatment, the absence of epileptic seizures and recovery of consciousness were noted.

2.4. Clinical Examination

The neurological examination carried out on admission revealed a new episode of epileptic seizure complicated by status epilepticus without recovery of consciousness. The Glasgow coma scale was estimated at 13/15. The presence of a spastic pyramidal syndrome of the right side of the body and a secondarily generalized right focal comitial syndrome were highlighted.

2.5. Paraclinical Explorations and Treatments

The brain CT scan was in favor of a cerebral infarction of the superficial territory of the left middle cerebral artery (Figure 1). The EEG revealed a generalized slow pattern with an anterior predominance in the fronto-centro-temporal region. No metabolic abnormalities were diagnosed. The patient is therefore treated for vascular epilepsy of structural origin with an emergency protocol for status epilepticus with clonazepam at a dose of 0.05 mg/kg slowly intravenously over 2 min for a weight of 65kg. Background antiepileptic treatment with sodium valproate, acetyl salicylic acid and atorvastatin 20 mg through the nasogastric tube are started. On the fifth day of hospitalization, we noted the sudden appearance of left hemiplegia with a Glasgow coma scale which dropped from 13/15 to 10/15. Brain MRI was performed urgently in a T1-weighted spin echo sequence (Figure 2), without (Figure A) then with injection of contrast product (Figure B), and a T2-weighted spin echo sequence (Figure 3), in T2 Flair (Figure C) and in b1000 diffusion (Figure D). It made it possible to describe a spontaneous and linear fronto-parieto-temporal hyperintensity with a well-systematized gyriform enhancement during the injection of gadolinium, as well as areas of bi-hemispheric hyperintensity in T2 Flair on the left taking the fronto-parieto-temporal region. right temporo-insular and left frontoparietal appearing in b1000 diffusion hyperintensity on the right and in diffusion hypointensity on the left suggestive of a diagnosis of left cortical laminar necrosis with extensive ischemic recurrence at the expense of the superficial territory of the right middle cerebral artery.

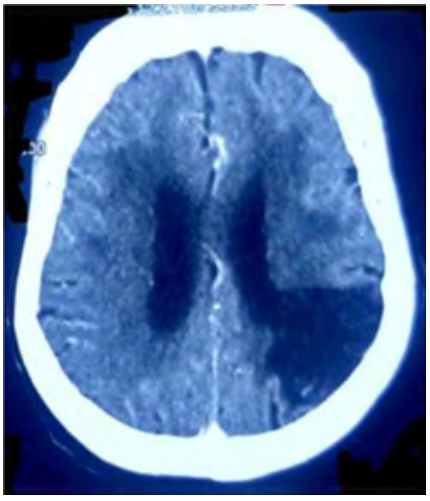

Figure 1: Axial cranioencephalic CT scan with injection of iodinated contrast product showing a well-systematized area of left parietal cortico-subcortical hypodensity with ectasia of the posterior horn of the ipsilateral lateral ventricle.

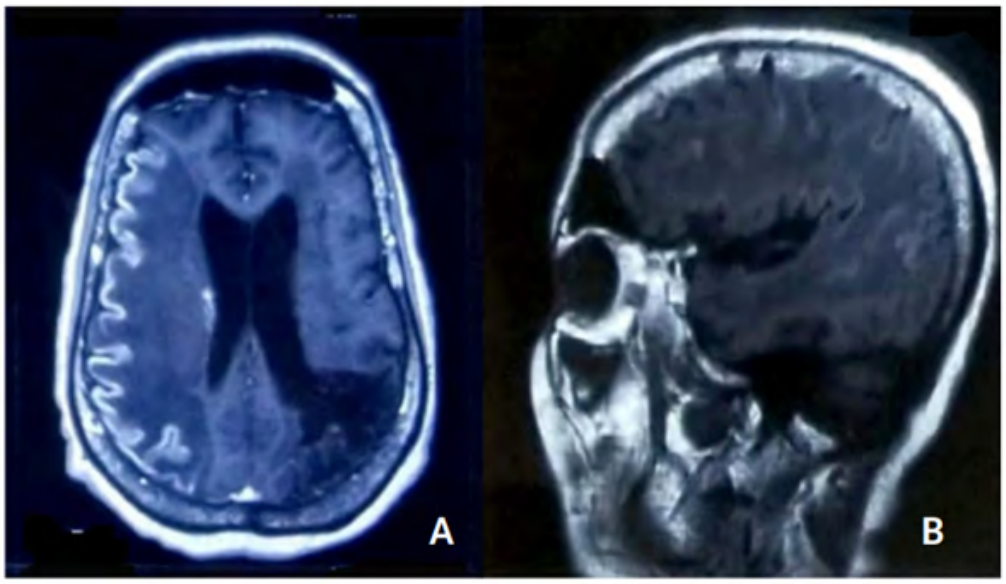

Figure 2: Brain MRI in T1-weighted spin echo sequence in sagittal section without injection of contrast product (A) showing areas of diffuse subcortical hypo-signals with a curvilinear right fronto-parieto-temporal cortical hyper-signal, in axial section with injection of the contrast material (B) showing right fronto-parieto-temporal gyriform contrast enhancement.

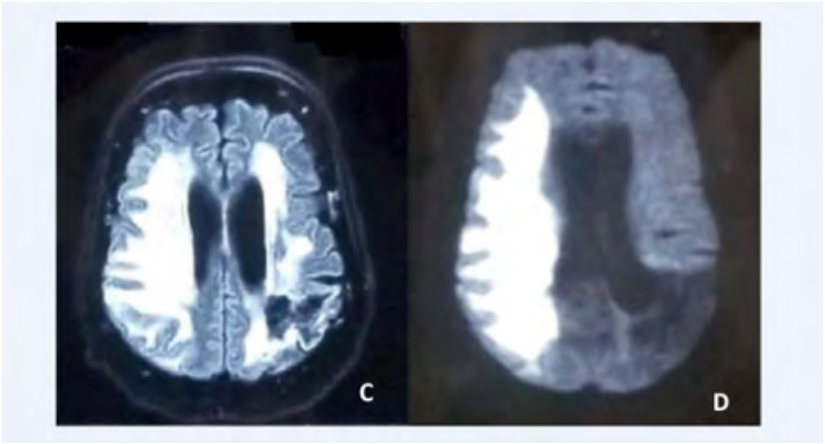

Figure 3: Brain MRI in T2- weighted echo sequence axial section (C) of bi hemispheric hypersignal areas in T2 Flair taking the right fronto-parieto-temporo-insular and left fronto-parietal regions, appearing in b1000 diffusion hypersignal on the right and hypersignal diffusion on the ledt (D).

3. Discussion

Laminar cortical necrosis corresponds to neuronal ischemia associated with a glial reaction and laminar deposition of lipid-rich macrophages. It occurs following cerebral hypoxia, with damage to the deep layers of the cortex. The gray matter, more vulnerable than the white matter, can be affected in isolation, defining selective neuronal necrosis [15]. It preferentially affects layers III and V. Depending on the intensity of the damage, the necrosis of the cortex is more or less extensive and is associated with the presence of macrophages, gliosis and vascular neogenesis [10]. Cortical laminar necrosis is because neurons in the cortex are much more metabolically active than glial cells or adjacent white matter; Selective gray matter vulnerability may be due to higher metabolic demand and denser concentrations of receptors for excitatory amino acids released after an anoxic-ischemic event. The distribution of the damage tends to favor the banks and the base of the furrows rather than the gyral crests. In the chronic phase, subcortical white matter involvement is limited and characterized by iron deposition rather than encephalomalacia, as is the case in cerebral infarctions [7, 14]. Cortical laminar necrosis has been described as associated with intoxication, hypoglycemia, ischemia, prolonged arterial hypotension, status epilepticus, cardiorespiratory arrest, renal or hepatic dysfunction, immunological causes, as in anti-phospholipid syndrome or lupus, and described during encephalitis [4]. A case has been reported in a tropical environment linked to a snake bite [15]. In our case, although the patient presented repeated convulsive attacks, it would be difficult to confirm the cause-effect relationship between cortical laminar necrosis and ischemic stroke, especially since left cortical laminar necrosis appeared in the territory contralateral to the right cortical cerebral infarction. The epileptogenic focus, the acute cortical edema, and the subsequent ischemic lesion in relation to the secondary hemodynamic and metabolic changes can be incriminated as predictive factors in the

occurrence of this cortical laminar necrosis although being distant from the epileptogenic focus. The hypothesis according to which the necrosis observed in this patient is mainly the consequence of the recurrence of the ischemic stroke is supported by the fact that the necrosis is observed in the vascular territory of the extensive infarction of the right superficial sylvian. However, we do not forget the role of increasing brain vulnerability. The latter could play a predisposing role in the hypoglycemia that the patient had in the occurrence of this cortical necrosis because Christiaens FJ. reports in his study three cases of prolonged hypoglycemia leading to hemiplegia due to unilateral hemispheric lesions of non-vascular cortical topography [5]. These lesions corresponded to a selective neuronal necrosis, confirmed by histopathological examination in one of the patients. [4]. The clinical picture is dominated by awareness impairments, sometimes motor or sensory deficit [9]. Aphasia and homonymous lateral hemianopia were also reported by Donaire A. [7]. However, the hemiplegia and impaired consciousness presented by our patient, as in other case reports illustrated in the literature, make it difficult to link clinical neurological manifestations to cortical necrosis alone. There are most often other concomitant brain lesions such as a brain abscess, a focal epileptogenic focus, or an infarction. Radiologically, MRI remains the gold standard for the diagnosis of cortical laminar necrosis and for the precision of the site and extent of the lesions. Laminar cortical necrosis is characterized by high intensity cortical lesions on T1-weighted and FLAIR images following a gyral distribution, associated with loss of volume of the underlying cortex. The lesions appear in hypersignal on the diffusion sequence and the T2 and T2 Flair weighted sequences from the first 24 hours and during the first three weeks. The T1 sequence shows cerebral edema during the first two weeks and spontaneous cortical and basal ganglia hyperintensity, delayed and transient, two weeks later. Cortical contrast enhancement reflecting the breakdown of the blood-brain barrier can be observed from the first weeks and can persist for up to nine months. [1]. The MRI was done three weeks after the symptomatology because of our working conditions in a tropical environment where the difficulty of accessibility prevails on the one hand and on the other hand the low income of our disadvantaged populations. The price of the brain MRI at 1.5 Tesla is XOF 230,000 or 353 euros, which sets up an economic barrier given the expensive cost of this examination. The abnormalities observed in our patient follow the dynamics of the cortical laminar necrosis lesions described. The most vulnerable and prone areas to CLN are most frequently located in the posterior quadrant of the cerebral hemispheres. Thus, several authors have reported cases with typical locations [6]. The posterior location of the lesions may be linked to the lack of sympathetic innervation of the vessels originating from the basilar artery. This innervation plays a fundamental role in cerebral vascular reactivity which leads to the coupling between cortical activity and cerebral blood flow [3]. Furthermore, experimental animal studies examining vasoreactivity in response to nitric oxide (NO) assessed by cerebral angiography, showed that the vascular response to NO was greater in the posterior circulation than in the anterior circulation. [11]. Considering that basal NO release contributes to the maintenance of vascular tone at rest, it can be hypothesized that the posterior circulation is more dilated under basal conditions and therefore has less capacity to continue vasodilation in the event of a brain abnormality. The consequence of a reduction in cerebral vascular reserve (CVR) and a decoupling between cortical metabolic demand and cerebral blood flow in situations where increased metabolic demands are necessary, such as in the case of status epilepticus. This hypothesis, however, requires confirmation as other human studies evaluating CVR and CO2 responsiveness using transcranial Doppler (TCD) ultrasound have not found significant differences in vasoreactivity between vertebro-basilar and carotid systems. In our observation, brain lesions are not located in the posterior quadrant of the cerebral hemisphere but over the entire extent of the vascular territory of the right middle cerebral artery. Further studies are needed to establish the correlation between the cause and topography of cortical laminar necrosis. The presence of CLN is life-threatening. It is pejorative, with high morbidity and mortality. In our case, it complicates the evolving profile of neurological signs and therefore darkens the clinical picture. There is no specific curative treatment, it mainly depends on the etiology and in our case report, it is ischemic stroke.

4. Conclusion

MRI is the examination of choice for positive diagnosis and for the precision of the extent of the lesions on which the prognosis depends. Aggressive treatment of any situation likely to aggravate cerebral hypoxia constitutes the main preventive means, which implies the fear of all the personnel involved and the parents in the face of any sign of deficit or alteration of consciousness.

5. Funding

Non

6. Conflict of Interest

None

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