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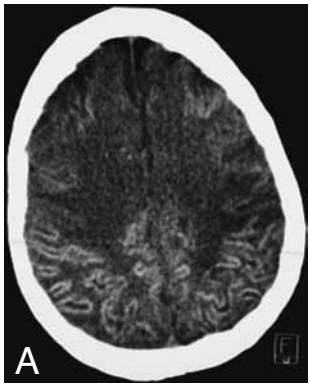
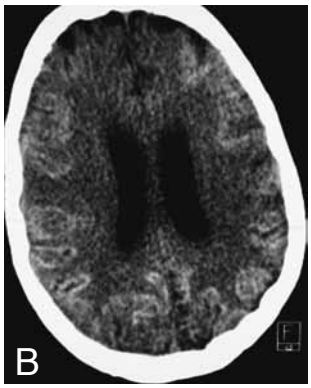
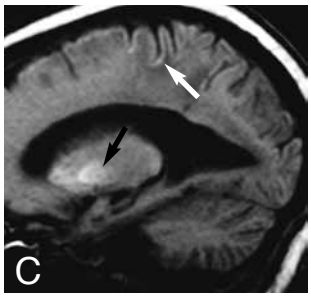
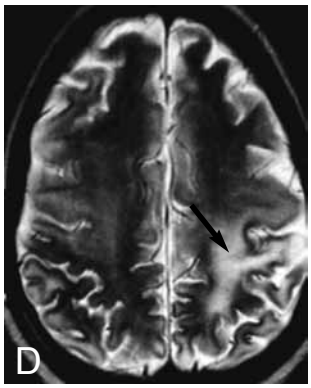
IMAGES IN CLINICAL RADIOLOGY









Typical CT and MRI features of cortical laminar necrosis

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A 47-year-old woman with a prior medical history of SLE and chronic arterial hypertension developed a post-anoxic coma after a prolonged cardiac arrest. She also presented a convulsive status epilepticus.

At admission contrast enhanced CT brain (not illustrated) showed minimal loss of gray-white differentiation in the left sylvian region.

Due to detoriation of the patient a follow-up CT scan was performed two weeks later. This examination showed hyperdensity in a laminar way, following the cortical gyri, mostly in the temporoparieto-occipital region (Fig. A and B). In the left peri-rolandic region this is associated with low-attenuation sub-cortical edema (Fig. B). Low density of the lentiform nuclei was also observed.

Further examination by MRI showed in the same areas thin cortical hyperintensity on sagital T1- and hypo-intense on axial T2-weighted imaging (white arrow in Fig. C). A hyperintense signal in the putamen is seen on T1 weighted imaging (black arrow in Fig. C). T2- and FLAIR- weighted sequence displayed increased signal in the white matter and throughout the subcortical layer, most accentuated in the temporoparieto-occipital region (arrow in Fig. D).

Diagnosis of advanced cortical laminar necrosis with bilateral deep gray nuclei involvement and encephalomalacia as typical manifestation of anoxic-ischemic brain damage was made. Shortly afterwards the patient passed away.

Comment

Several signs of advanced anoxic-ischemic brain damage were observed in our case. The most striking finding is the imaging manifestation of the so called 'cortical laminar necrosis'.

Histological studies demonstrate much more vulnerability of grey matter than white matter to ischaemic necrosis due to hypoperfusion. The third layer of the gray matter is the most vulnerable and the damage is usually greater in the depths and sides of the sulci than over the crest of the gyri.

Cortical laminar necrosis is a specific type of cortical infarction, which usually develops as a result of generalized hypoxia rather than a local vascular abnormality. Depletion of oxygen or glucose as in anoxia, hypoglycemia, status epilepticus, ischemic stroke and less common in immunosuppressive therapy and polychemotherapy has been attributed as an underlying cause of cortical laminar necrosis. A hypoxic insult leads to death of neurons, glia and blood vessels along with degradation of proteins.

When cortical laminar necrosis is seen on CT imaging, it presents as gyriform linear hyperdensity in the superficial cortex, most frequently located in the medial occipital lobes and perirolandic regions. Mostly this is a subtle finding, but the image can be more spectacular in more severe cases. These areas are often associated with cortical hypoattenuation as presentation of oedema.

Cortical enhancement is first seen after 2 weeks, peaks after 1 to 2 months, and is usually resolved after 6 months. Contrast enhancement is present due to disruption of the blood-brain barrier, where loss of neurons and vascular proliferation occur.

Initially thought to be caused by hemorrhagic infarction, histopathological examination has demonstrated the cortical short T1 lesions to occur by neuronal damage, reactive tissue change of glia and deposition of fat-laden macrophages. The close interrelation between the protein concentration and free water content makes MR signal changes more complex.

Early cortical changes usually show low signal intensity on T1-weighted, which could be due to acute ischemic changes (tissue edema).

Usually, cortical high intensity lesions on both T1-weighted and FLAIR-weighted images appear two weeks after the ictus indicating short T1 and long T2 lesions. Proton-density images are even more sensitive than T1-weighted MR images.

Cortical diffusion abnormalities in the early postanoxic period are proven to be associated with poor outcome.

To conclude, cortical laminar necrosis has no pathognomonic features but shows characteristic chronological signal intensity changes, especially seen on T1-weighted, FLAIR and proton-density MR images. On CT imaging the cortical linear hyperdensities are mostly seen in severe hypoxic-ischemic brain damage.

Reference

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