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Case report

Hemorrhagic infarct of basal ganglia in cardiac arrest. CT and MRI findings. 2 cases

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ABSTRACT

We report the CT and MRI findings in two cases of hemorrhagic infarct of the basal ganglia (BG), following out-of-hospital cardiac arrest (CA).

In case 1, Brain-CT realized at day 2 showed bilateral and almost symmetric hemorrhagic infarct of the BG and infarct of the tectum of the mesencephalon. In case 2, MRI realized at day 6 showed hemorrhagic infarct of both lenticular nuclei on T2 GE images.

In both cases there was no medical history and the cardiovascular and the coagulation profile were normal.

In these cases, the lesions are observed earlier than reported in a few previous radiological cases. Similar lesions have been reported in pathological studies.

These lesions seem occur early after CA. Reperfusion is probably responsible for the hemorrhagic transformation. The reason why some patients present either BG or brainstem infarct or both remains unclear.

Bilateral and symmetric hemorrhagic infarct of the BG, especially of the Lenticular nuclei, and infarct of the dorsal pons and mesencephalic tegmentum seem to be a characteristic feature of profound and prolonged hypotension or of CA.

1. Introduction

Symmetrical hemorrhagic necrosis of the basal ganglia (BG), of the lower cranial nerve nuclei, the tegmentum of the pons and the mesencephalon subsequent to prolonged and severe hypotension or to cardiac arrest (CA) have been described pathologically.

Only isolated cases of hemorrhagic infarct of the BG have been reported in imaging studies [1,2].

We report the CT and MRI findings in two cases of hemorrhagic infarct of the BG, following CA. In one case it was associated with infarct of the tectum of the BS.

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2. Case 1

A 54-year-old woman presented with sudden chest pain, dyspnea, loss of consciousness and cardiorespiratory arrest (no flow: 10 min; low flow: 10 min). She was successfully resuscitated and transferred to our ICU 4 h after onset. She was comatose with Glasgow coma score (GCS) of 3 after sedation (midazolam and sufentanyl), *the limbs were flaccid*, with fixed and dilated pupils. The brainstem reflexes were absent.

Initial vital signs were temperature 33.6 °C, blood pressure 125/90 mmHg, pulse 94/min. Physical examination was otherwise unremarkable.

Electrocardiogram showed normal sinus rhythm.

The relevant biological data on admission were as follows: arterial pH 7.08, $PaCO_2$ 26.5 mmHg, (Sp O_2 and Fi O_2: 100%) blood bicarbonate 3.8 mmol/L, blood lactate 6.38 mmol/L, serum creatinine 103 μmol/L, blood urea nitrogen 0.1 mmol/L and, leukocytosis (22 500/mL), LDH 352 UI/L, CK 923 UI/L.

Coronarography performed at admission showed non stenotic coronary lesions. Non-contrast Brain-CT at day 2 revealed symmetric hypodensities of the BG with hemorrhagic components and hypodensity of the tectum of the mesencephalon (Fig. 1a–d). The following days the neurological state was unchanged. The patient died on day 6.

3. Case 2

A 64-year-old man was found unconscious and pulseless lying on the floor in the street. He was transferred to our ICU 3 h after cardio-pulmonary resuscitation (no flow: unknown; low flow: 10 min). At admission, he was comatose with GCS of 3 after sedation *(midazolam and sufentanyl)* and with dilated non-reactive pupils. The brainstem reflexes were absent.

Initial vital signs were temperature 33.4 °C, blood pressure 130/80 mmHg, pulse 90/min. Physical examination was otherwise unremarkable.

Electrocardiogram showed normal sinus rhythm.

The relevant biological data on admission were as follows: arterial pH 7.13, $PaCO_2$ 57 mmHg, blood bicarbonate 9 mmol/L, blood lactate 5.66 mmol/L, serum creatinine 122 μmol/L, blood urea nitrogen 0.1 mmol/L and leukocytosis (22 500/mL), LDH 352 UI/L, CK 923 UI/L.

Brain MRI at day 6 showed, associated with diffuse hypoxic brain damage on FLAIR and diffusion images (Fig. 1e–f) and hemorrhagic lesions of both LN on T2 GRE images (Fig. 1g). T1 sagittal paramedian right and left images show the light hyperintense signal in both lenticular nuclei (Fig. 1h and i), indicating the subtle hemmorhagic component, highlighted by the ''blooming effect'' on T2 GRE images (Fig. 1g). MRI and

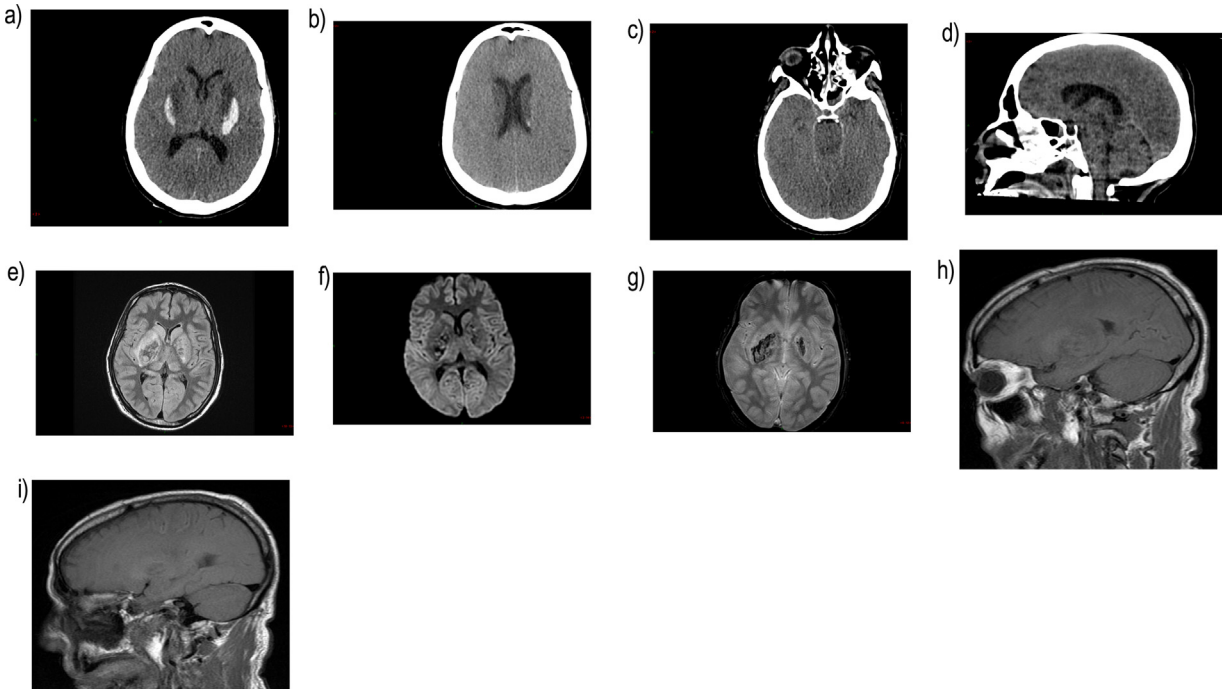

Fig. 1 – *Case 1*: Non-enhanced CT at day 2 after CA shows: bilateral hemorrhagic infarct at the external and posterior part of both putamen and profound hypodensity of the anterior part of both putamen, of both pallidi and caudate nuclei (CN) (a), petechial hemorrhage in the corpus of the right CN (b) and marked hypodensity of the tectum of the mesencephalon (c), well visualized on Sagittal reconstruction (d).
Note the presence of global edema with no-visualization of the sulci and cisterns. No midline shift.
***Case 2*: MRI at day 7 shows on FLAIR a hyperintense signal of both lenticular nuclei (LN) and caudate nuclei (CN) with an heterogeneous aspect of both LN. Furthermore the cerebral cortex and the thalami have a hyperintense and homogeneous signal (e).**
On diffusion sequence (f) heterogeneous signal of both LN is related to the hemorrhagic component well identified on T2 GRE sequence (g). T1 parasagittal images show subtle hyperintense signal in both LN, related to hemorrhagic components (right: h; left: i), highlighted by the ''blooming effect'' on T2 GRE image (g).

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