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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

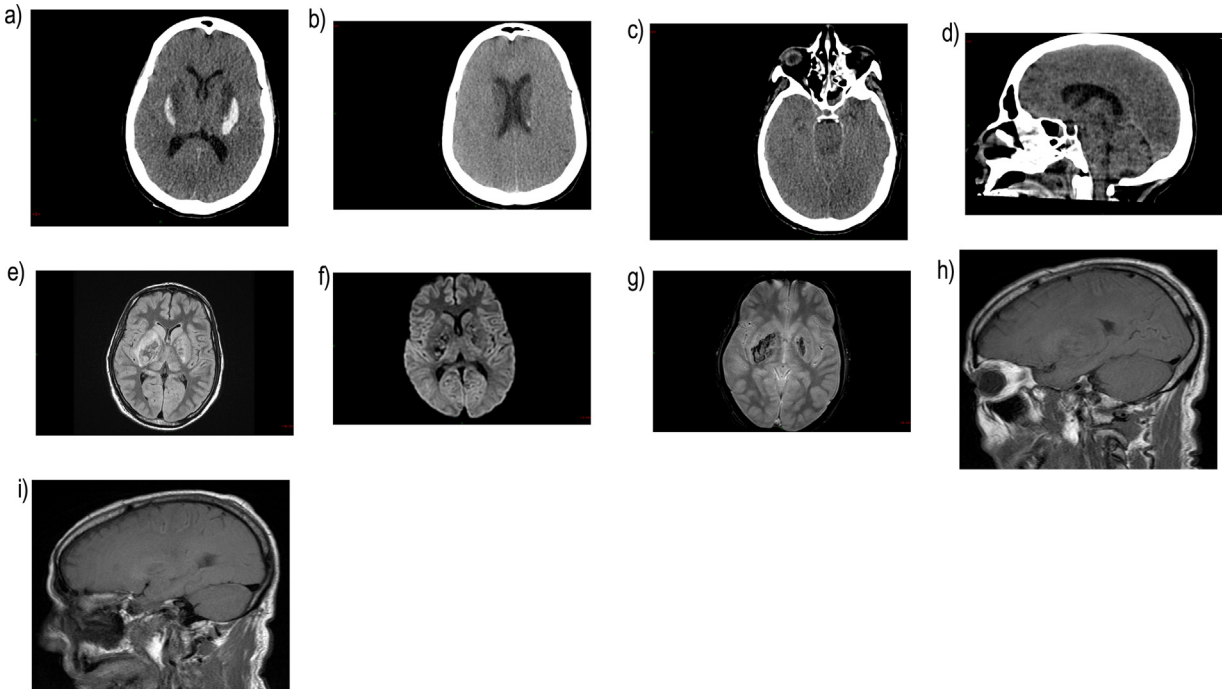

Fig. 1 – *Case 1*: Non-enhanced CT at day 2 after CA shows: bilateral hemorrhagic infarct at the external and posterior part of both putamen and profound hypodensity of the anterior part of both putamen, of both pallidi and caudate nuclei (CN) (a), petechial hemorrhage in the corpus of the right CN (b) 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).

MR-angiography (MRA) did not reveal underlying cerebrovascular pathology. The patient died 1 day later.

The background in the two patients was similar. In both cases there was no medical history, the cardiovascular and the coagulation profile were normal.

4. Discussion

Previous pathological studies have reported bilateral symmetric foci of hemorrhagic necrosis of nuclear groups in the BS and/or within the BS tegmentum, related to hypotensive shock or to CA [3–6].

On the other hand, hemorrhagic transformation of bilateral and symmetric infarct of BG have been reported in isolated cases on CT at day 24 after a shock of unknown origin and on MRI at day 26 after CA [2–7]. In the present cases these lesions were diagnosed earlier, on day 2 and 6, indicating that these lesions can appear soon after the CA.

In only one case bipallidal hemorrhages were observed on CT at day 7 [1].

Finally, a pathological study of 9 cases following intractable hypotension in 8 cases and CA in 1, reported symmetrical hemorrhagic necrosis of the BG (3/9 cases), of the BS tectum (1/9), and in both the BG and BS (5/9) [8].

More recently, Opeskin [9] reported the simultaneous occurrence of hemorrhagic necrosis in the BG and BS related to CA in 3 cases.

Finally brain damage after profound and prolonged hypotension or CA follows 3 patterns: symmetrical hemorrhagic necrosis of the BG alone, of the dorsal BS alone and of the BG and dorsal BS.

Such symmetric lesions located in the end-zone distribution of the lenticulo-striate arteries for the BG and of the perforating arteries for the BS probably result from terminal zone ischemia. Reperfusion seems to be responsible for the hemorrhagic transformation.

These bilateral and symmetric hemorrhagic infarcts seem to be a specific feature of hypotensive shock and CA.

The reason why some patients present with either BG or BS infarct or both remains unclear.

These findings are rarely objectified in the literature. During the period between March 2010 and June 2012,150 consecutive adult patients resuscitated from an out-of-hospital CA (OHCA) were admitted to our center. Among them, 70 patients without cardiac cause underwent Brain CT and/or MRI. In only 2 cases (2.8%) these lesions were observed and are reported here.

The current cases show unusual radiological findings: CT scan showed evidence of infarct of the tectum of BS, non-reported before, probably because in previous reports Brain-CT were performed with lower performance devices than those actually used. On the other hand, the hemorrhage of the BG in our case was demonstrated on T2 GRE MRI sequence, highly sensitive for subtle hemorrhagic transformation of infarcts.

This hemorrhagic pattern is quite different from that of usual hematoma.

A recent review of the literature studying simultaneous bilateral basal ganglia hemorrhage demonstrated that this entity is extremely rare, and only 53 cases have been reported between 1978 and 2014. Hematomas are usually not perfectly symmetric. The most frequent location is putaminal (71%) and the most common cause is hypertension [10,11]. Bilateral BG hemorrhage or hemorrhagic necrosis can occur in methanol poisoning [12], after head trauma [13], and have been reported in isolated cases in diabetic ketoacidosis [14], hyperglycemic hyperosmolar syndrome [15], fungal infection [16], during the reversible cerebral vasoconstriction syndrome (RVCS) [17] and after lightning strike [18].

Furthermore, cerebral hematomas, without associated subarachnoid hemorrhage (SAH), are an uncommon cause of CA.

In two recently reported large cohorts of 3710 [19] and 2716 patients with OHCA [20], cerebral hematoma without SAH was the cause of CA respectively in 0.13% and 0.3%.

In a Japanese cohort of 124 patients with OHCA, 2 patients presented with cerebellar hemorrhage (1.6%) [21]. The discrepancy between the reported incidence of these series may be related to the higher incidence of fatal cerebral hemorrhages in the Japanese population.

Different mechanisms of cardiac arrest in the setting of brain hemorrhage have been hypothesized: cardiac dysrhythmia caused by extreme surge of endogenous catecholamines [22], instantaneous respiratory arrest secondary to medullary respiratory center lesions from mass effect and increase intracranial pressure, finally seizures related to brain hemorrhage can be responsible of cardiac arrhythmia. of cardiac arrest in the setting of brain hemorrhage have been hypothesized: cardiac dysrhythmia caused by extreme surge of endogenous catecholamines [22], instantaneous respiratory arrest because secondary to medullary respiratory center lesions from mass effect and increase intracranial pressure, finally seizures related to brain hemorrhage can be responsible of cardiac arrhythmia [23,24]

5. Conclusion

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

Conflict of interest

None declared.

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None declared.

REFERENCES

[1] Finelli PF. Bilateral hemorrhagic infarction of the pallidum. J Comput Assist Tomogr 1984;8(1):125–7.
[2] Fujioka M, Okuchi K, Miyamoto S, Sakaki T, Hiramatsu K, Tominaga M, et al. Changes in the basal ganglia and thalamus following reperfusion after complete cerebral ischaemia. Neuroradiology 1994;36(8):605–7.

[3] Gilles FH. Hypotensive brainstem necrosis: selective symmetrical necrosis of tegmental neuronal aggregates following cardiac arrest. Arch Pathol 1969;88:32–41.
[4] Janzer RC, Friede RL. Hypotensive brain stem necrosis or cardiac arrest encephalopathy? Acta Neuropathol 1980;50 (1):53–6.
[5] Jurgensen JC, Towfighi J, Brennan RW, Jeffreys WH. Symmetric brainstem necrosis in an adult following hypotension: an arterial end-zone infarct? Stroke 1983;14 (6):967–70.
[6] Yamashita M, Yamamoto T. Symmetrical necrosis in the gray matter of the brainstem and spinal cord. Two adult cases of anoxic encephalopathy. Rinsho Shinkeigaku 2003;43(3):113–8.
[7] Inagaki T, Ishino H, Senoh H, Naora C, Iijima M, Sasaki T, et al. A case of bilateral necrosis of the basal ganglia after hypotensive shocks. No To Shinkei 1989;41(5):471–5.
[8] Ng HK. Hypotensive symmetrical hemorrhagic necrosis of the basal ganglia and brainstem. Pathology 1994;26(1):23–7.
[9] Opeskin K, Burke MP. Hypotensive hemorrhagic necrosis in basal ganglia and brainstem. Am J Forensic Med Pathol 2000;21(4):406–10.
[10] Zhao J, Chen Z, Wang Z, Yu Q, Yang W. Simultaneous bilateral hypertensive basal ganglia hemorrhage. Neurol Neurochir Pol 2016;50(4):275–9.
[11] Yang Z, Chen J, Mu J. Simultaneous bilateral basal ganglia hemorrhage. Curr Drug Deliv)2016;(June) [Epub ahead of print].
[12] Sefidbakht S, Rasekhi AR, Kamali K, Borhani Haghighi A, Salooti A, Meshksar A, et al. Methanol poisoning: acute MR and CT findings in nine patients. Neuroradiology 2007;49 (5):427–35.
[13] Vega MB, Hamamoto Filho PT, Machado Cde J, Zanini MA. Traumatic brain injury presenting with bilateral basal ganglia hemorrhage. Neurol Neurochir Pol 2015;49(6):456–9.
[14] Ertl-Wagner B, Jansen O, Schwab S, Sartor K. Bilateral basal ganglion haemorrhage in diabetic ketoacidotic coma: case report. Neuroradiology 1999;41(9):670–3.
[15] Cho SJ, Won TK, Hawang SJ. Bilateral putaminal hemorrhage with cerebral edema in hyperglycemic hyperosmolar syndrome. Yonsei Med J 2002;43(4):533–5.
[16] Verma A. Bilateral basal ganglionic hemorrhage. Arch Neurol 2006;63(3):464.
[17] Westover MB, Cohen AB. Reversible vasoconstriction syndrome with bilateral basal ganglia hemorrhages. J Neuroimaging 2013;23(1):122–5.
[18] Ozgun B, Castillo M. Basal ganglia hemorrhage related to lightning strike. AJNR Am J Neuroradiol 1995;16 (6):1370–1.
[19] Arnaout M, Mongardon N, Deye N, Legriel S, Dumas F, Sauneuf B, et al. Out-of-hospital cardiac arrest from brain cause: epidemiology, clinical features, and outcome in a multicenter cohort. Crit Care Med 2015;43 (2):453–60.
[20] Shin J, Kim K, Lim YS, Lee HJ, Lee SJ, Jung E, et al. Incidence and clinical features of intracranial hemorrhage causing out-of-hospital cardiac arrest: a multicenter retrospective study. Am J Emerg Med 2016;34(12):2326–30.
[21] Inamasu J, Miyatake S, Tomioka H, Nakatsukasa M, Imai A, Kase K, et al. Headache, cardiac arrest, and intracranial hemorrhage. J Headache Pain 2009;10(5):357–60.
[22] Samuels MA:. The brain–heart connection. Circulation 2007;116(1):77–84.
[23] Sörös P, Hachinski V. Cardiovascular and neurological causes of sudden death after ischaemic stroke. Lancet Neurol 2012;11(2):179–88.
[24] Devinsky O, Hesdorffer DC, Thurman DJ, Lhatoo S, Richerson G. Sudden unexpected death in epilepsy: epidemiology, mechanisms, and prevention. Lancet Neurol 2016;15(10):1075–88.