

## CLINICAL IMAGE

# The white cerebellum sign

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## Case presentation

A 78-year-old man presented to the emergency room (ER) after an out-of-hospital cardiac arrest. His medical history included diabetes mellitus, iron-deficiency anaemia, vascular dementia and an ischaemic stroke of the left hemisphere. He was found unresponsive in his wheelchair in his care home for an unknown amount of time. Upon arrival of the ambulance the first registered rhythm was asystole. Return of spontaneous circulation was achieved after 10 minutes.

Physical examination in the ER revealed a SatO<sub>2</sub> of 92% with 40% supplemental oxygen via mechanical ventilation, diffuse bilateral rhonchi, a heart rate of 102 beats/min, blood pressure of 100/60 mmHg and a basal body temperature of 37 °C. He had fixed, dilated pupils, absent cornea reflexes and a Glasgow Coma Scale score of three. Based on the electrocardiogram and echocardiography findings, myocardial infarction was deemed unlikely and thus acute coronary angiography was not performed. Due to ensuing hypotension, cerebral and thoracic computed tomography (CT), as part of the routine diagnostic work-up, were postponed. The following day, a thoracic CT scan showed no abnormalities. The non-enhanced cerebral CT scan revealed diffuse oedema with effacement of sulci, loss of grey and white matter differentiation, compression of the lateral ventricles and mild hyperdensity of the cerebellum ('white cerebellum sign', *figure 1*). Enhanced cerebral CT angiography confirmed progressive cerebral oedema and significant reduction in the intracerebral arterial blood circulation. Brain stem reflexes were persistently absent. Due to this extensive brain damage and poor prognosis, life-prolonging treatment was ceased. The patient passed away shortly after.



**Figure 1.** White cerebellum sign. Coronal [A] and sagittal [B] non-enhanced cerebral CT scan showing hyperattenuation of the cerebellum relative to cerebral hemispheres

## Discussion

White cerebellum sign is an infrequently described, yet ominous finding in neuroradiology. On cerebral CT imaging it is seen as hypoattenuation of the cerebral hemispheres sparing infratentorial structures and leading to the characteristic lighter aspect of the cerebellum and/or the brain stem.<sup>[1,2]</sup> It is often interchangeably mentioned with 'reversal sign', a finding in which there is diffuse hypodensity of the cortical grey matter relative to the adjacent white matter.<sup>[1-4]</sup> The white cerebellum sign and reversal sign may occur together, however have also been separately reported.<sup>[1,3,5]</sup> The exact pathophysiological mechanism of white cerebellum sign is unclear.<sup>[4,5]</sup> One proposed mechanism suggests redistribution of cerebral blood flow to the posterior circulation, due to cerebral oedema in hypoxic-ischaemic events.<sup>[4,5]</sup> It has been described in cases of severe head trauma, perinatal asphyxia or asphyxia, drowning, hypothermia, status epilepticus, cerebral infections and post-anoxic encephalopathy.<sup>[1,4,6]</sup> White cerebellum sign is an important diagnostic marker that may aid in therapeutic and prognostic decision-making as it is indicative of severe and often irreversible brain injury with poor neurological outcome.<sup>[4-6]</sup>

Nevertheless, cessation of life-prolonging therapies should not be solely based on this marker. In our case, due to the subtle nature of the white cerebellum sign, we additionally conducted cerebral CT angiography to confirm compromised cerebral arterial blood flow and in light of persistent absent brain stem reflexes, treatment was withdrawn.

## Disclosure

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