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White cerebellum sign – radiological indicator of poor prognosis

Příznak bílého mozečku – radiologický indikátor špatné prognózy

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Dear Editor,
Herein, we present a rare case of a white cerebellum sign (WCS), a radiological indicator of poor prognosis, observed in a 23-month-old child following severe traumatic brain injury (TBI). WCS is characterized by widespread hyperdensity in the infratentorial region, including the cerebellum, thalamus, and brainstem, with concomitant hypodensity in the supratentorial region on CT. This rare but critical finding is associated with severe anoxic-ischemic brain injury and poor clinical outcomes [1,2].

A 23-month-old female patient was brought to the emergency department after a fall from a height of approximately 5 meters. Upon admission, the patient was intubated, and the Glasgow Coma Scale (GCS) score was 4 (E1M2Ve). Physical examination revealed a mid-dilated nonreactive left pupil, right periorbital edema, and superomedial deviation of the right eyeball, consistent with IV cranial nerve involvement (Fig. 1B). CT imaging demonstrated widespread brain edema, traumatic subarachnoid hemorrhage, bilateral temporal fractures extending to the foramen magnum, and a depressed right frontal fracture. The peri-mesencephalic and basal cisterns were obliterated, indicating severe intracranial hypertension (Fig. 1A, B).

Emergency surgery was performed, including a right frontotemporoparietal decompressive craniectomy. Intraoperatively, significant cerebral edema, dural laceration,

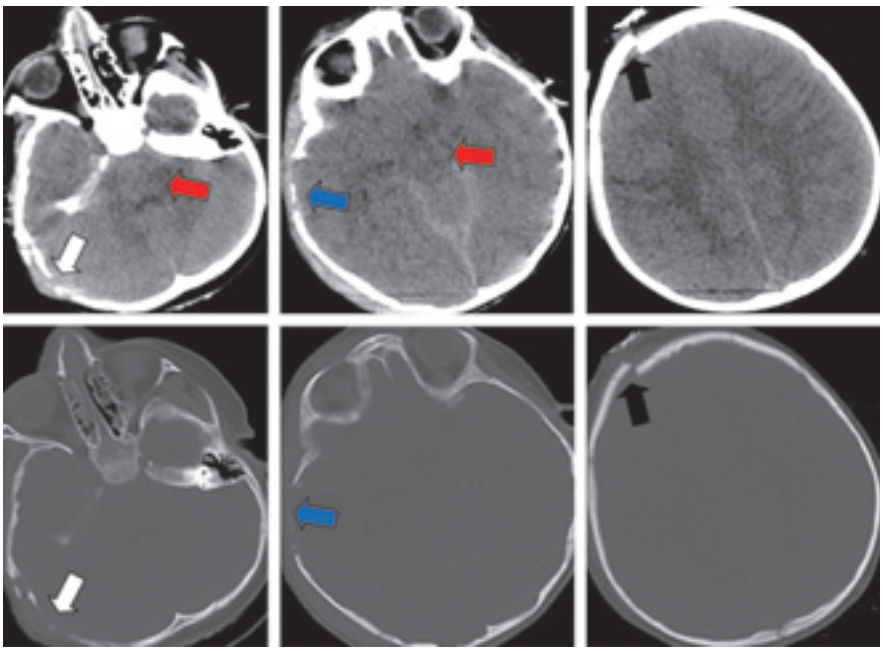

Fig. 1A. Axial pre-operative CT images of the same section, displayed in the brain (tissue) window (upper panel) and bone window (lower panel). The white arrow indicates the occipital fracture, the blue arrow points to the temporal fracture, the black arrow shows the displaced frontal fracture line, and the red arrow marks the obliterated brainstem cisterns. Widespread cerebral edema and traumatic subarachnoid hemorrhage are evident on the brain window.

Obr. 1A. Axiální předoperační CT snímky stejného řezu, zobrazené v mozkovém (tkáňovém) okně (horní panel) a kostním okně (spodní panel). Bílá šipka označuje okcipitální zlomeninu, modrá šipka ukazuje na temporální zlomeninu, černá šipka ukazuje na posunutou frontální zlomeninu a červená šipka označuje obliterované cisterny mozkového kmene. Na mozkovém okně jsou patrné rozsáhlý cerebrální edém a traumatické subarachnoidální krvácení.

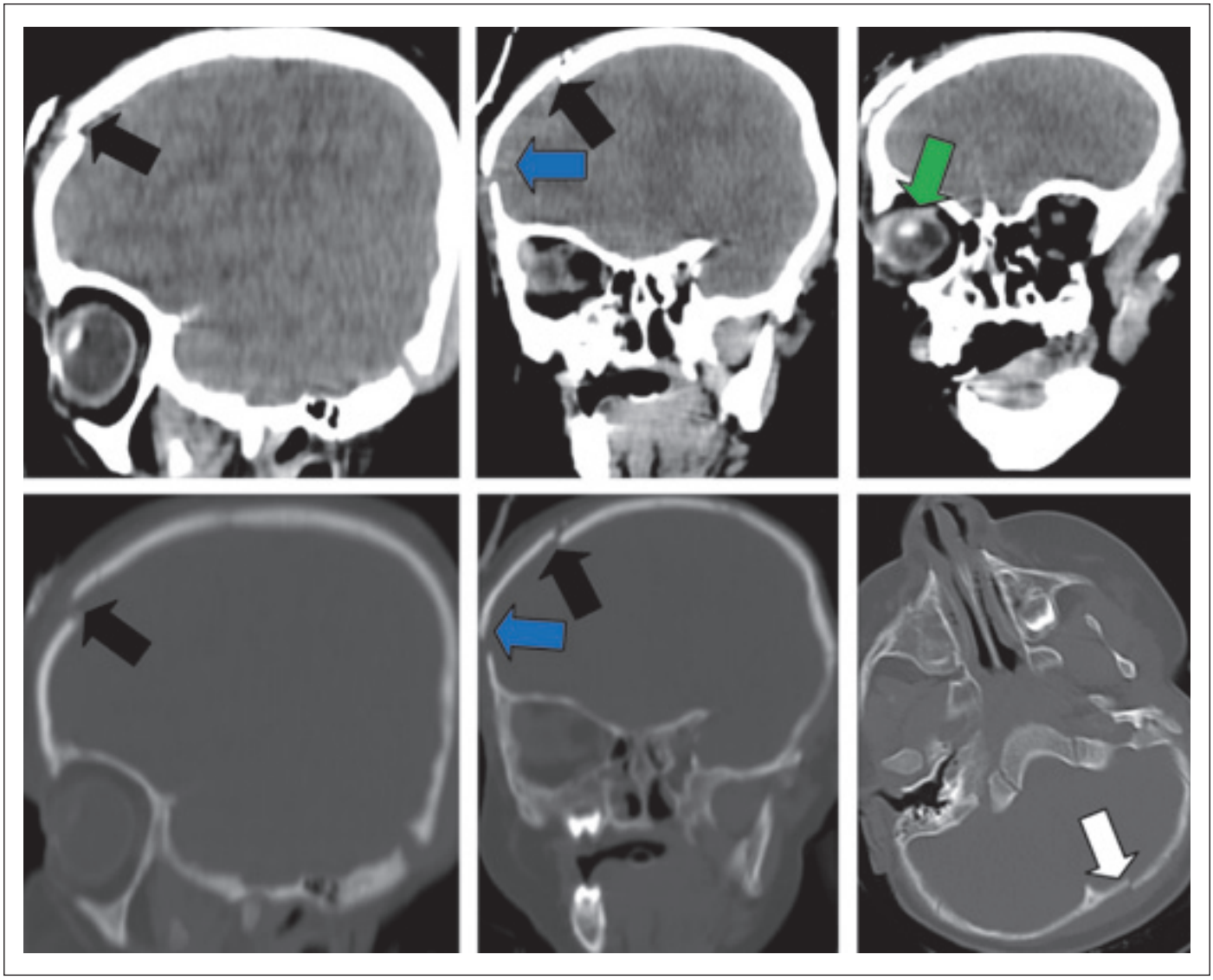

Fig. 1B. Pre-operative CT images of the same anatomical level displayed in the brain (tissue) window (upper row) and bone window (lower row). In each row, images are shown from left to right as sagittal, coronal, and axial sections. The black arrow indicates the displaced frontal fracture, the blue arrow points to the temporal fracture, the white arrow shows the occipital fracture line, and the green arrow marks the IV cranial nerve involvement.

Obr. 1B. Předoperační CT snímky stejné anatomické úrovně zobrazené v mozkovém (tkáňovém) okně (horní řada) a kostním okně (spodní řada). V každé řadě jsou snímky zobrazeny zleva doprava jako sagitální, koronální a axiální řez. Černá šipka označuje posunutou frontální zlomeninu, modrá šipka ukazuje na temporální zlomeninu, bílá šipka ukazuje na okcipitální zlomeninu a zelená šipka označuje postižení IV. hlavového nervu.

and brain contusion were observed. Post-operative CT revealed the presence of WCS, with marked hyperdensity in the cerebellum and brainstem, alongside diffuse hypodensity in the cerebral hemispheres (Fig. 2). Despite aggressive medical and surgical interventions, the patient's neurological status remained poor, and she died after a period of intensive care due to severe hypoxic-ischemic brain injury. The patient ultimately succumbed to her injuries despite maximal supportive treatment in the intensive care unit.

White cerebellum sign was first described in patients with hypoxic brain injury and is often referred to as the "reversal sign" due to the inversion of expected white and gray matter densities [1]. It is commonly seen in conditions such as birth asphyxia, drowning, smoke inhalation, epilepsy, bacterial meningitis, and hypoxia secondary to cardiac arrest [1,2]. The pathophysiology involves extensive cerebral edema leading to loss of white-gray matter differentiation, intracranial hypertension, and selective hypoperfusion of supratentorial structures while infratentorial structures maintain perfusion [2–4]. This results in the characteristic hyperdensity of the cerebellum relative to the edematous supratentorial tissue.

In pediatric patients, WCS has been reported predominantly in cases of child abuse, near-drowning, and severe hypoxia. Kin Han et al. analyzed 20 pediatric cases with WCS, identifying hypoxic events in 45% of cases, traumatic etiologies in 10%, and infections in 5% [1]. Overall prognosis was extremely poor, with one-third of patients suc-

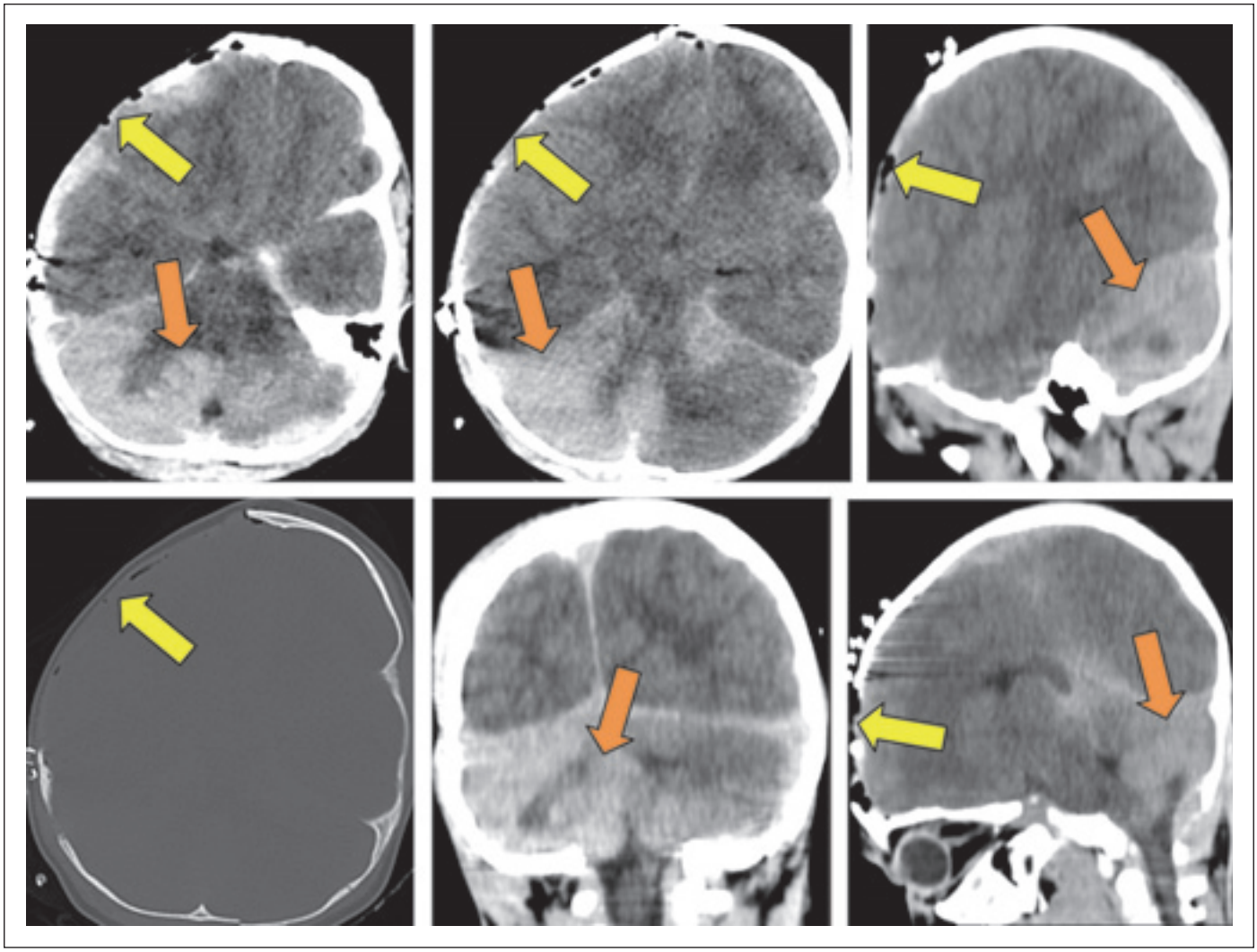

Fig. 2. Post-operative CT images demonstrating the white cerebellum sign. The upper row shows brain (tissue) window images, with axial sections in the first and second panels and a sagittal section in the third panel. The lower row includes an axial bone window image in the first panel, followed by coronal and sagittal brain (tissue) window images in the second and third panels, respectively. The yellow arrow indicates the decompression area, while the orange arrow highlights the diffuse hyperdensity in the infratentorial region.

Obr. 2. Pooperační CT snímky, které znázorňují příznak bílého mozečku. Horní řada zobrazuje snímky mozkového (tkáňového) okna, s axiálními řezy v prvním a druhém panelu a se sagitálním řezem ve třetím panelu. Dolní řada zahrnuje axiální snímek kostního okna v prvním panelu, následují koronální a sagitální snímek mozkového (tkáňového) okna v druhém a třetím panelu. Žlutá šipka označuje oblast dekomprese, zatímco oranžová šipka zvýrazňuje difuzní hyperdenzitu v infratentoriální oblasti.

cumbing to their injuries and the remaining cases exhibiting severe neurological sequelae, including diffuse cerebral atrophy and cystic encephalomalacia [2].

The radiological hallmark of WCS is best identified on non-contrast CT, which reveals the paradoxical hyperdensity of infratentorial structures against a diffusely hypodense cerebral cortex. Differential diagnosis includes conditions such as severe hypoglycemia, mitochondrial encephalopathies, and other metabolic disorders [5–7]. The presence of obliterated basal cisterns, as seen in our patient, further correlates with a fatal outcome [2].

The exact pathophysiological mechanism of WCS remains unclear, but several hypotheses have been proposed. One explanation suggests that elevated intracranial pressure disrupts the deep venous drainage of the supratentorial brain, leading to differential perfusion of the infratentorial structures [3,4,7]. Another hypothesis involves selective vulnerability of the cerebral hemispheres to ischemia due to differences in metabolic demands. Additionally, the posterior circulation is thought to remain functional longer than the anterior circulation in cases of global ischemia, thereby preserving cerebellar perfusion [7]. Harwood-Nash et al. suggested that the WCS could be related to an evolutionary "diving reflex" that preferentially protects the brainstem and cerebellum under conditions of severe hypoxia [7].

The treatment approach for WCS primarily focuses on symptomatic management. Strategies include reducing intracranial pressure through controlled hyperventilation, osmotic diuretics such as mannitol, and targeted temperature management [8]. In some cases, decompressive craniectomy can provide temporary relief, but long-term outcomes remain unfavorable. Literature suggests that early aggressive management can improve survival

in select cases; however, most patients with WCS experience severe and irreversible neurological impairment.

While most cases of WCS lead to poor outcomes, a few reports indicate that some patients with WCS due to post-meningitic encephalopathy have shown significant improvement with appropriate treatment [9]. This suggests that certain reversible mechanisms may contribute to the radiological appearance of WCS in specific scenarios. However, in traumatic and hypoxic cases, the presence of this sign is almost invariably associated with severe morbidity and mortality.

Recognizing the WCS is crucial for both radiologists and clinicians, as it signifies severe hypoxic brain damage with minimal likelihood of recovery [1,2]. Early identification allows for timely prognostication and informs decision-making regarding the appropriateness of continued intensive care [2,10].

Given the grim prognosis associated with this radiological finding, WCS may also be useful in medico-legal cases, particularly in suspected child abuse or non-accidental trauma [6,10]. Furthermore, understanding the underlying pathophysiology of WCS can help in developing novel neuroprotective strategies aimed at mitigating secondary brain injury [3,4,7].

In conclusion, WCS remains a rare but significant imaging finding associated with catastrophic brain injury. Its presence on CT should prompt consideration of profound hypoxic injury and carries a dismal prognosis [1,2]. This case underscores the importance of early radiological recognition, guiding clinical expectations and management strategies. Further research is necessary to elucidate the precise mechanisms underlying WCS and to explore potential neuroprotective strategies that may mitigate the impact of this devastating condition.

Conflict of interest

The authors declare they have no potential conflicts of interest concerning drugs, products, or services used in the study.

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