



Traumatic axonal injury revealed by postmortem magnetic resonance imaging: A case report

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ABSTRACT

In forensic investigations, it is important to detect traumatic axonal injuries (TAIs) to reveal head trauma that might otherwise remain occult. These lesions are subtle and frequently ambiguous on macroscopic evaluations. We present a case of TAI revealed by pre-autopsy postmortem magnetic resonance imaging (PMMR).

A man in his sixties was rendered unconscious in a motor vehicle accident. CT scans revealed traumatic mild subarachnoid hemorrhage. Two weeks after the accident he regained consciousness, but displayed an altered mental state. Seven weeks after the accident, he suddenly died in hospital. Postmortem computed tomography (PMCT) and PMMR were followed by a forensic autopsy.

PMMR showed low-intensity lesions in parasagittal white matter, deep white matter, and corpus callosum on three-dimensional gradient-echo T1-weighted imaging (3D-GRE T1WI). In some of these lesions, T2*-weighted imaging also showed low-intensity foci suggesting hemorrhagic axonal injury. The lesions were difficult to find on PMCT and macroscopic evaluation, but were visible on antemortem MRI and confirmed as TAIs on histopathology.

From this case, it can be said that PMMR can detect subtle TAIs missed by PMCT and macroscopic evaluation. Hence, pre-autopsy PMMR scanning could be useful for identifying TAIs during forensic investigations.

1. Introduction

Diffuse traumatic white matter injuries caused by shear stress to axons were first suggested about some six decades ago [1,2]. Studies of primates by Gennarelli et al. [3] and of humans by Adams and Graham [4–9] describe diffuse axonal injury (DAI), caused by high-magnitude angular or rotational acceleration, rendering the victim unconscious from the time of injury, typically without definitive structural changes seen on computed tomography (CT) scan. This distinct clinicopathological entity was considered to be widespread and irreversible [4,7]. In addition, based on animal studies, milder, reversible forms of DAI have been suggested to exist [10–12]. Traumatic axonal injury

(TAI) is now recognized to appear in a spectrum of severity [13] from concussion and other reversible injuries to the most severe form of DAI [14,15].

Forensic pathologists are sometimes called on during postmortem examinations to assess the possibility of TAIs [13], especially when it is suspected as being the only factor responsible for the victim's brain dysfunction and sometimes the sole cause of death [16]. As TAIs are considered the result of acceleration-deceleration force, their presence could suggest the mechanism of injury [4,17,18]. Extensive systematic sampling of the brain is necessary for medico-legal evaluation of TAIs, as their macroscopic appearance is frequently subtle and ambiguous, making it difficult to distinguish them from other pathology, such as

Abbreviations: β-APP, beta-amyloid precursor protein; CT, computed tomography; DAI, diffuse axonal injury; DWI, diffusion-weighted imaging; FA, flip angle; FLAIR, fluid attenuated inversion recovery; FSE, fast spin echo; GRE, gradient echo; 3D GRE, 3-dimensional gradient-echo T1-weighted imaging; MRI, magnetic resonance imaging; PMCT, postmortem computed tomography; PMMR, postmortem magnetic resonance imaging; SE, spin echo; TAI, traumatic axonal injury; TE, echo time; TR, repetition time; T1WI, spin-echo T1-weighted imaging; T2WI, spin-echo T2-weighted imaging; T2*WI, T2 star weighted imaging

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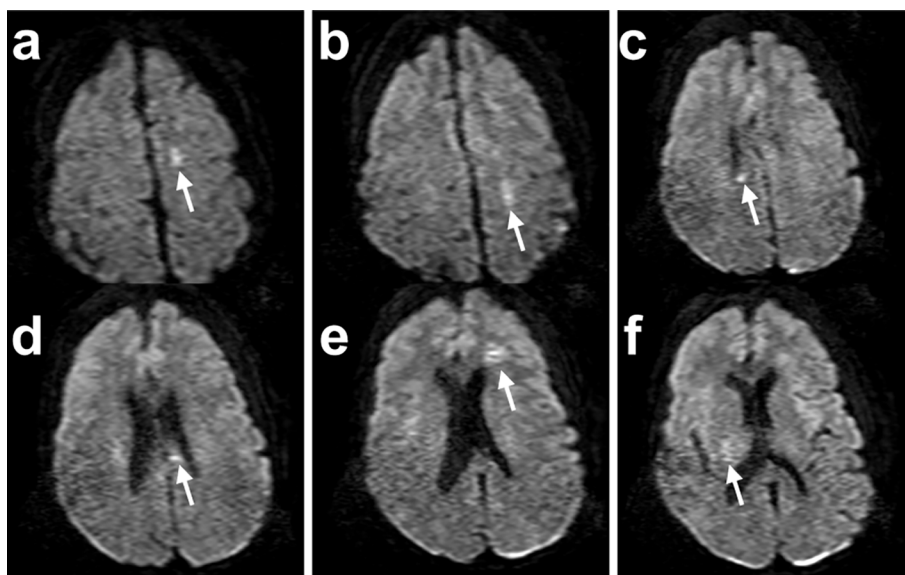


Fig. 1. Antemortem magnetic resonance images of the victim scanned 2 weeks after his injury. Axial diffusion-weighted images (b: 1000 s/mm^2) are shown. High-intensity lesions, whose distribution is consistent with typical traumatic axonal injury, are apparent in the left parasagittal white matter (a, b), left and right corpus callosum (c, d), deep white matter around the anterior horn of the left lateral ventricle (e), and the right posterior internal capsule (f).

ischemic brain injury [13,19].

With advances in neuroradiology, some TAIs can be detected by CT [20–22], although magnetic resonance imaging (MRI) with advanced sequences is considered more sensitive for identifying TAIs [20–24]. Also, antemortem MRI is not always performed—and even if performed, the scans are not always available to pathologists. If post-mortem magnetic resonance imaging (PMMR) could reveal TAIs, it would help guide the neurohistopathological examination, especially in patients with no antemortem imaging studies. Although PMMR has been conducted in some forensic institutes and departments worldwide [25–29], to the best of our knowledge there are no reports on whether it can detect TAIs. We describe a case in which pre-autopsy PMMR, which was useful for detecting subtle TAIs.

2. Case report

A right-hand-drive car driven by a man in his sixties was hit on the right side by a medium-sized truck. Both vehicles were traveling approximately 40 km/h. The man was thrown free from his safety belt and he hit the left-side window. He was found unconscious but with a heartbeat. He was immediately transferred to the hospital, where CT scans revealed mild traumatic subarachnoid hemorrhage and mesenteric injuries. CT did not provide any evidence of TAIs. Open abdominal surgery was performed to control bleeding from the mesenteric injuries.

Two weeks after the accident, his consciousness returned but he remained disoriented. He could say words when addressed but the words were meaningless. He could not remain sitting by himself, and so remained bedridden. The Glasgow coma scale was E4V3M5. Non-enhanced brain MRI was performed at this point. The scan protocol included the following: T2-weighted imaging-fast spin echo (T2WI-FSE), with TE 105 ms, TR 5000 ms, 5 mm slice thickness; T1-weighted imaging-spin echo (T1WI-SE), with TE 12 ms, TR 535 ms, 5 mm slice thickness; diffusion weighted imaging (DWI), with b 1000 s/mm^2 , TE 120 ms, TR 5000 ms, 5 mm slice thickness; and T2*-weighted imaging-gradient echo (T2*WI-GRE), with flip angle (FA) 20° , TE 15 ms, TR 670 ms, 5 mm slice thickness. DWI revealed small diffusion-restricted lesions in the parasagittal white matter, deep white matter, corpus callosum, and internal capsule, compatible with cytotoxic edema caused by TAIs (Fig. 1). T2WI (although acutely affected by a motion

artifact caused by the patient's movements) showed high-intensity areas in these lesions, which is also compatible with TAIs (Fig. 2a, b). In contrast, no lesions were visible on T1WI (Fig. 2c, d). T2*WI showed a small, low-intensity focus in a deep white matter lesion around the frontal horn of the left ventricle (Fig. 2e, f), suggesting a lesion with small iron depositions (i.e., a hemorrhagic lesion) because T2*WI is highly sensitive to magnetic substances. Five weeks after the accident, bilateral subdural hygromas had gradually increased. Bilateral burr hole openings were made and drainage was performed but with no change in his mental status.

Seven weeks after the accident, he experienced sudden oxygen desaturation, followed by death. No resuscitation was performed according to previously signed advance directive. To confirm the cause of death and its causal relation to the initial traffic accident, the cadaver was brought to our department for forensic autopsy. Prior to autopsy, postmortem computed tomography (PMCT) and PMMR were performed.

PMCT (12 h after death), performed with a 16-row multidetector CT (Eclis; Hitachi, Ltd., Tokyo, Japan), was undertaken with the following conditions: 0.625 mm collimation, 120 kV tube voltage, 200 mA tube current, 1 rotation per second. PMCT showed one low-density area in the deep white matter lateral to the anterior horn of the left lateral ventricle (Fig. 3). No other lesions suggesting TAIs were found.

Following the CT scan, the cadaver was preserved at 4°C . PMMR was performed 29 h after death using a 1.5 T scanner (Intera Achieva; Philips, Amsterdam, The Netherlands). Scanning room temperature was approximately 22°C . The rectal temperatures measured before and after MRI were 7.5°C and 8.0°C , respectively. A 16-channel head/neck coil was used for brain scanning. The brain scan was performed under the following conditions: T2WI-FSE: TE 100 ms, TR 4500 ms, 5 mm slice thickness; T1WI-SE: TE 12 ms, TR 600 ms, 5 mm slice thickness; T2*WI-GRE: FA 18° , TE 23 ms, TR 650 ms, 5 mm slice thickness; DWI: b 1000 s/mm^2 , TE 75 ms, TR 3000 ms, 5 mm slice thickness; three-dimensional gradient-echo T1-weighted imaging (3D-GRE): FA 30° , TE 6.4 ms, TR 25 ms, 1.2 mm slice thickness. The images were evaluated by two board-certified radiologists.

3D-GRE showed low-intensity areas in the right and left corpus callosum, left parasagittal white matter, left deep white matter, and right internal capsule (Fig. 4), which corresponded to the lesions shown

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