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Post-mortem CT and MRI diagnosis of acute cerebral hemorrhage in a putrefied corpse

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ARTICLE INFO	A B S T R A C T
Keywords: Brain hemorrhage Post-mortem magnetic resonance imaging (PMMR) Post-mortem computed tomography (PMCT) Putrefaction	A 61-year-old man was found dead on the terrace in front of his house. The body showed external signs of advanced (Grade 4) putrefaction. Routine post-mortem computed tomography (PMCT) was conducted prior to the forensic autopsy showing a radiological alteration index (RAI) of 100. Due to a suspicious hyperdense brain lesion seen in PMCT, additional post-mortem magnetic resonance imaging (PMMR) of the head was performed for research purpose. Compared to PMCT, the lesion appeared considerably more detailed in PMMR with a slightly hyperintense and focal hypointense signal in T2w images and an isointense and focal hyperintense signal in T1w images. Autoptic assessment of the brain lesion was considerably complicated due to softening and partial liquefaction of the brain. Considering post-mortem imaging and autopsy results, the lesion was determined as an acute brain hemorrhage. Case findings indicate that PMMR may be superior to autopsy in terms of diagnosis and

localization of cerebral hemorrhage in putrefied brains.

Case circumstances

A 61-year-old man was found dead on the terrace of his house. The body showed signs of advanced putrefaction and was infested with maggots. Surroundings of red fluid, a broken wine bottle, and glass as well as two kitchen knives were discovered on the terrace floor near the body (Fig. 1A). Forensic medical external examination of the body at the scene revealed several skin defects at the left cervical and thoracic regions (Fig. 1B). The scenery was suspicious for homicide and the prosecutor ordered a forensic autopsy.

Forensic examination and imaging results

Based on the condition of the body, the post-mortem interval was estimated to be several days to weeks. Police investigations determined the date of death 11 days before finding, based on neighbor's testimonies, recorded smartphone messages, e-mails, and unchecked mail in the letterbox. The man was living alone. Medical records of the decedent showed that the man suffered from hypertension. Overall, criminalistic investigations did not reveal any hints of third-party involvement. Neighbors who spoke to the man one day before his suspected death reported no abnormity in his behavior.

In the man's house, the police found an open attic door. The door's springs and closing mechanism were broken causing the door to fall unbridled immediately after attempts to close it (Fig. 1C). After several attempts the door could be closed but fell spontaneously after a few seconds. The edge of the door was 175 cm above the ground. Hair residues were found on the outside of the attic door near the closing mechanism. Further forensic hair analysis was not conducted.

Routine whole-body post-mortem computed tomography (PMCT) was conducted before forensic autopsy (Siemens Somatom Definition AS 64; Tube voltage: 140 kV; slice thickness head: 0.75 mm; increment 0.5 mm; rotation time: 0.5 s; field of view head: 290 mm; soft tissue algorithm and bone tissue algorithm). PMCT diagnostics were conducted using 2D image reconstructions. Due to suspicious intracranial findings at PMCT, post-mortem magnetic resonance imaging (PMMR) of the head (Philips Achiva 3 T; axial and coronary T2-weighted sequences (slice thickness: 4 mm, Gap: 1 mm, TR: 3000 ms, TE: 80 ms) and T1-weighted sequences (same thickness and gap, TR: 450 ms, TE: 8 ms)) was conducted immediately after PMCT. At the author's forensic institution, whole body PMCT is conducted routinely in each case. PMMR is not conducted routinely but only for research purposes after PMCT.

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Received 10 August 2023; Received in revised form 1 November 2023; Accepted 16 November 2023 Available online 19 November 2023 2665-9107/© 2023 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/bync-nd/4.0/). In PMCT, the post-mortem radiological alteration index (RAI) was 100 (overall gas grade III) indicating severe putrefaction gas formations [1]. PMCT showed a y-shaped skull fracture at the median frontal and parietal portion of the skull canopy (Fig. 2A). Moreover, PMCT revealed the partial collapse of the brain hemispheres and large amounts of intracranial gas. In both brain hemispheres, pronounced on the right side, a hyperdense, sharply defined, approx. $8 \times 3.5 \times 6$ cm lesion was visible (Fig. 3 A-C). In PMMR, the brain lesion appeared inhomogeneous with a slightly hyperintense and focal hypointense signal in T2w images and an isointense and focal hyperintense signal in T1w images (Fig. 3 D-F). Dimensions of the lesion measured in MR images were approx. the same as in PMCT. Based on PMMR imaging the hemorrhage was localized at the basal ganglia and thalamic regions.

Forensic autopsy assessment of the head, thorax, and abdomen was limited due to advanced putrefaction. The Grade of external putrefaction (GEP) was 4 according to the standardized classification of Maujean et al. [2]. Body height was 185 cm. The autopsy (Fig. 2B) confirmed the skull fracture seen in PMCT. After opening of the scull cavity, the brain appeared softened and partially liquefied. Hence, it was not possible to remove the brain manually as a whole from the skull cavity. Softening and liquefaction resulted in the leaking of brain portions downwards following gravitation. Due to knowledge of post-mortem imaging findings, all brain portions were collected in a linen sheet that was held under the opened skull (Fig. 4A) and put in a cup with formalin-buffered fixation fluid. After 2 weeks, formalin-fixated brain tissue remains were dissected (Fig. 4B). Within brain tissue remains, a dark red area of $10 \times 6 \times 4$ cm in size was recognizable (Fig. 4C), appearing to be an acute cerebral hemorrhage in histology. It was not possible to determine an exact anatomic localization of the hemorrhage within the brain remnants due to brain tissue softening and deforming before fixation.

As far as assessable, no relevant thoracic or abdominal traumatic lesions or relevant natural preconditions were present at the autopsy. Dissection of the cervical and thoracic parts showed that skin defects did not continue to the deeper soft tissue. Cervical vessels, airways, and surrounding soft tissue showed no signs of trauma, which led to the interpretation that skin defects were caused by maggot myiasis.

Toxicological analyses in muscle showed a blood alcohol concentration of 0.30‰ and Oxazepam (< 13 μ g/kg), Duloxetin (258 μ g/kg), and Mirtazapine (< 30 μ g/kg) in non-toxic concentrations. Altogether, toxicological analyses did not indicate relevant intoxication.

The final forensic report ruled cerebral hemorrhage as a cause of death. Neighbor's reports on no abnormal behavior of the decedent on the day before his suspected death indicated an acute hemorrhage, for a hemorrhage older than a few hours would probably have caused noticeable neurological symptoms.

Case discussion

In the present case, the simultaneous occurrence of brain hemorrhage and skull fracture suggested a connection of findings. The trauma that caused the skull fracture may have caused the cerebral hemorrhage. However, the location of the hemorrhage at the basal ganglia and thalamic region as assessed in PMMR was rather typical for a hypertensive hemorrhage. Such a hemorrhage of natural origin could have caused a fall to cause the skull fracture. The location of the skull fracture at the canopy was not typical for an ordinary fall to the ground. Skull fracture may also be explainable by a hit on the head with an object by a third person. However, police investigations could not find any hints to third-party involvement. Based on the findings inside the man's house an accidental hit on the head by the broken attic door as the cause for skull trauma and cerebral hemorrhage seemed likely. However, this assumption cannot be proved and it is not to be excluded that the skull fracture and the cerebral hemorrhage may even have occurred unrelatedly.

Putrefied corpses pose a challenge to forensic pathologists because the judgment of regular anatomical structures as well as pathologic or



Fig. 1. Finding situation of the body (A). Notice a broken wine bottle, glass fragments from a broken drinking glass, two knives, and red-brownish stains on the ground nearby the putrefied corpse. External examination of the body revealed maggot infestation and several skin defects at the left cervical and upper thoracic region (B). C shows a broken attic door that was suspected to have hit the man on the head, causing the skull fracture.



Fig. 2. Y-shaped fracture (yellow arrows) of the median frontal and parietal portion of the skull canopy shown at PMCT 3D reconstruction (A) and autopsy (B).



Fig. 3. Unenhanced PMCT (A: axial view, B: coronary view, C: sagittal view) with intracerebral hyperdense lesion (yellow arrows in A, B, and C) as well as unenhanced PMMR (D: axial T2w, E: coronary T2w, F: axial T1w) with an inhomogeneous slightly hyperintense (yellow markings in D and E) and focal hypointense (red arrows and star in D and E) appearance in T2w images and mainly isointense (yellow arrows in F) and focal hyperintense (blue arrows in F) appearance in T1w images.

traumatic lesions is often difficult or impossible [3,4]. This is especially true for the brain since putrefaction results in brain softening, liquefaction, and discoloration [3,5]. In such conditions, it may be difficult to remove the brain as a whole from the scull cavity at autopsy. Often, it is only possible to remove softened single brain parts or collect liquefied

brain parts that run out of the opened skull cavity. Hence, in putrefied brains classification of regular anatomical structures as well as diagnosis of traumatic injuries or pathologic lesions is usually considerably more difficult or impossible [6]. The putrefied brain is held together by the meninges as long as it remains within the unopened skull. Thus, by using

Fig. 4. Softened and partially liquefied brain within the skull cavity at autopsy (A). Collected brain tissue portions were collected in a sheet, fixated for 2 weeks (B), and dissected afterward. Dissection showed a large, sharply defined dark red area (yellow arrows) appearing to be a rather fresh cerebral hemorrhage.

imaging techniques such as PMCT or PMMR the brain can be assessed as a whole although being softened or partially liquefied [7-12]. Tschui et al. demonstrated that PMCT and PMMR might aid in the forensic diagnosis of putrefied brains. Their study showed that PMMR was superior to autopsy regarding the classification of regular anatomical brain structures such as grey and white matter, ventricular system, basal ganglia, and brain stem. This was especially true for highly softened or liquefied brains [7]. Post-mortem imaging offers the advantage of putrefied brain evaluation before opening the skull. In their study, Tschui et al. did not present cases with confirmed brain pathology such as cerebral hemorrhage. Several publications demonstrated the possibility of detecting acute cerebral hemorrhage with PMCT in putrefied brains [13–15]. To the best of our knowledge, the presented case is the first depiction of a confirmed cerebral hemorrhage in a putrefied brain with PMMR. In the presented case, brain hemorrhage appeared considerably more detailed in PMMR compared to PMCT. Since the putrefied brain is often collapsed, it is hard to identify specific anatomic brain structures in PMCT. In contrast, PMMR allows for a far more detailed depiction of anatomical structures even of the putrefied brain. Moreover, the more detailed presence in different image weightings such as T1 and T2 may allow for a rough age estimation of hemorrhage too, comparable to clinical MRI. In the living, MR signal appearance of intracranial hemorrhage depends on the levels of hemoglobin degradation over time. In clinical MRI, acute hemorrhage aged less than 24 h appears isointense to hyperintense in T1 weighted images and hyperintense in T2 weighted images. From one to three days, the T1w hemorrhage signal remains the same while the T2w signal appears hypointense. Hemorrhage older than 2 weeks appears hypointense in both T1 and T2w [16,17]. So far, there are no PMMR studies investigating signal behavior of cerebral hemorrhages of different ages. In addition, it is unknown how putrefaction alters the MR signal behavior of cerebral hemorrhage. If one considers PMMR signal behavior the same as in the living, T1w and T2w signal appearance indicates an acute hemorrhage in the present case. To learn about the signal behavior of cerebral hemorrhage in fresh as well as putrefied copses systematic post-mortem imaging studies are needed.

Conclusion

The presented case is the first depicting PMMR findings of a cerebral hemorrhage in an advanced putrefied brain. Case findings indicate that PMMR may be superior to autopsy in terms of diagnosis and localization of cerebral hemorrhage in putrefied brains.

Compliance with ethical standards

Conflict of Interest: All authors declare no conflict of interest. This study received no funding.

This manuscript does not contain any studies with live human participants or animals.

The usage of the acquired data was approved by the local ethics committee.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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R. Ringger et al.

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