**Abstract**

**Objectives**

To evaluate the frequency of total-body CT and MR features of postmortem change in in-hospital deaths.

**Materials and methods**

In this prospective blinded cross-sectional study, in-hospital deceased adult patients underwent total-body postmortem CT and MR followed by image-guided biopsies. The presence of PMCT and PMMR features related to postmortem change was scored retrospectively and correlated with postmortem time interval, post-resuscitation status and intensive care unit (ICU) admittance.

**Results**

Intravascular air, pleural effusion, periportal edema, and distended intestines occurred more frequently in patients who were resuscitated compared to those who were not. Postmortem clotting was seen less often in resuscitated patients ($p = 0.002$). Distended intestines and loss of grey-white matter differentiation in the brain showed a significant correlation with postmortem time interval ($p = 0.001$, $p < 0.001$). Hyperdense cerebral vessels, intravenous clotting, subcutaneous edema, fluid in the abdomen and internal livores of the liver were seen more in ICU patients. Longer postmortem time interval led to a significant increase in decomposition related changes ($p = 0.026$).

**Conclusions**

There is a wide variety of imaging features of postmortem change in in-hospital deaths. These imaging features vary among clinical conditions, increase with longer postmortem time interval and must be distinguished from pathologic changes.
Introduction

Hospital autopsy rates today are as low as 0–5%, having decreased from a rate of 30% or higher in the 1990s. [1–3] This low rate is alarming, since one in five autopsies show major discrepancies between antemortem and postmortem diagnoses despite improved diagnostic testing. [4] A possible cause for this decline may be the invasiveness of the conventional autopsy procedure. [5] To provide a less invasive alternative to conventional autopsy, imaging based autopsy methods were developed, primarily in forensic medicine. These modern autopsies include total-body postmortem CT (PMCT) and MR (PMMR), sometimes combined with CT angiography (PMCTA) and image-guided biopsies. [6–8]

More recently the imaging autopsy is steadily emerging in clinical radiology and there is a growing number of diagnostic studies analyzing the performance of the noninvasive (imaging only) and minimally invasive autopsy (imaging with angiography and / or biopsies). [9–11] Combined PMCT, PMCTA and image-guided biopsies appear most sensitive in diagnosing cause of death, however more clinical studies are needed to accurately determine the diagnostic value of the imaging autopsy. [10, 11] In forensic centers access to MR scanners is often limited, so PMCT is most commonly performed. In hospitals, MRI is more widely available, and its high performance to visualize organ parenchyma and soft tissues make PMMR a valuable addition to PMCT.

Postmortem imaging is not the same as imaging the living. Directly after death various chemical and physical processes affect the body in ways that can change PMCT and PMMR features of organs and soft-tissues. These processes can generally be divided into gravity dependent changes (including sedimentation of blood and livor mortis; also known as lividity or hypostasis), decomposition (including putrefaction), rigor mortis (muscle stiffness) and algor mortis (cooling of the body).

Livor mortis is caused by blood settling in the dependent parts of the body due to gravity. Livores can be observed both internally, on imaging and autopsy, and externally upon visual inspection. External livores manifest as dark bluish (or livid) areas of the skin within several hours after death. Internal livores are noted as increased attenuation or signal changes of the dependent areas of organs. The combination of postmortem leakage of cell membranes and subsequent increased osmolality of the interstitial fluid, together with the effect of gravity leads to accumulation of fluids in dependent areas, such as the subcutaneous fat, thoracic cavity and abdominal cavity. [12–14]

Decomposition consists of many processes that cause organic material to break down into simpler forms of matter. It includes putrefaction, autolysis and insect and animal predation. Putrefaction leads to gas formation, it is found intravascular in an early decomposition stage and in more advanced stages also in soft tissues and organ parenchyma.

Rigor mortis leads to muscle contraction after death that results in muscle stiffness. Rigor mortis is caused by cessation of synthesis of adenosine triphosphate (ATP). ATP is consumed in muscle fibers to separate actin and myosin filaments. Directly after death ATP is still present in the muscle, but it is consumed in the first hours after death. When the ATP reserves are depleted, actin and myosin filaments cannot separate anymore. This state lasts until decomposition leads to the breakdown of actin and myosin filaments. The speed of this process depends on temperature: both the time until rigor mortis starts and reaches its maximum and the time until rigor mortis recedes are longer in colder bodies. [15–18]

Algor mortis can affect tissue contrast on PMMR images. There is a wide variability of T1 values due to higher sensitivity of T1 to temperature differences. [19] T2 values are less temperature dependent.
Radiologists need in-depth understanding of these processes for correct acquisition and interpretation of PMCT and PMMR scans. The aim of this study is to evaluate the frequency of total-body CT and MR features of postmortem change in in-hospital deaths.

**Materials and methods**

**Study protocol**

This study was undertaken as part of the *Minimally Invasive Autopsy* (MIA) study. This is a prospective single center cross-sectional study in a tertiary referral hospital comparing diagnostic performance of conventional autopsy and MIA. Approval of the Erasmus MC Institutional Review Board and Ethics Committee was obtained; the study was filed with the Netherlands National Trial Register. Patients aged 18 years and older who died in the Erasmus University Medical Center were eligible for inclusion, if written informed consent was obtained from next-of-kin for MIA and CA of at least the torso.

Exclusion criteria were (suspected) unnatural COD, body size exceeding diameter of 16 inches in supine position (limitation for PMMR), known or suspected “high-risk” infected bodies (tuberculosis, hepatitis B and C, human immunodeficiency virus, methicillin-resistant

**Table 1. Postmortem imaging protocol.**

| Scans area | Coils | Sequence | TR/TE/ TI (ms) | Slice width (mm) | FOV (cm) | Matrix | Number of slices | Coverage per section (cm) | Number of sections | Scan time per section (s) |
|------------|-------|----------|----------------|------------------|----------|--------|------------------|--------------------------|----------------------|-------------------------|
| Head-Pelvis | Body   | FLAIR FSE T1w | 2320/9.5/ 963 | 4.0 | 48 | 384x320 | 50 | 20.0 | 5–8 | 174 |
| Head-Pelvis | Body   | STIR FSE T2w | 12000/41/120 | 4.0 | 48 | 288x224 | 50 | 20.0 | 5–8 | 168 |
| Thorax | 8-channel | 3D fs FSPGR T1w | 3.3/1.2/ 14 | 1.6 | 40 | 256x256 | 212 | 33.9 | 1 | 153 |
| Thorax | 8-channel | 2D STIR FSE T2w | 11200/94/120 | 2.0 | 40 | 256x256 | 170 | 34.0 | 1 | 359 |

**Table 2. Image-guided biopsy protocol.**

| Organ | Targets aimed at |
|-------|------------------|
| Brain | Normal parenchyma and suspected pathology |
| Lungs | Both lungs, normal parenchyma and suspected pathology |
| Heart | Left ventricle: lateral wall, apex, normal myocardium and suspected pathology. Right ventricle: on indication |
| Kidneys | Both kidneys, normal parenchyma and suspected pathology |
| Spleen | Sub-capsular parenchyma and suspected pathology |
| Liver | Normal parenchyma and suspected pathology |
| Other | Region of interest |

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Table 3. Scoring list of PMCT and PMMR features of postmortem changes in the brain.

| Postmortem imaging feature                          | Category                     | Postmortem process               |
|-----------------------------------------------------|------------------------------|----------------------------------|
| 1. Hyperdense superior sagittal sinus               | Gravity dependent changes    | Blood sedimentation              |
| 2. Hyperdense veins                                 | Gravity dependent changes    | Blood sedimentation              |
| 3. Hyperdense arteries                              | Gravity dependent changes    | Blood sedimentation              |
| 4. Thickened / irregular falx                       | Gravity dependent changes    | Blood sedimentation              |
| 5. Liquid in paranasal sinus                        | Decomposition                | Cell wall leakage                |
| 6. Intracranial air                                 | Decomposition                | Putrefaction                     |
| 7. High T1 signal basal ganglia                     | Algol mortis                 | Temperature change               |
| 8. Low T2 signal basal ganglia                      | Algol mortis                 | Temperature change               |
| 9. Diffusion restriction                            | Algol mortis                 | Temperature change               |
| 10. Insufficient suppression of liquor on Flair     | Algol mortis                 | Temperature change               |
| 11. Loss of grey-white matter differentiation       | Miscellaneous                | Cerebral hypoxia                 |
| 12. Sulcal effacement                               | Miscellaneous                | Cerebral hypoxia                 |
| 13. Compression cisterns                            | Miscellaneous                | Cerebral hypoxia                 |
| 14. Cerebellar tonsillar herniation                 | Miscellaneous                | Cerebral hypoxia                 |

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Table 4. Scoring list of PMCT and PMMR features of postmortem changes in the thorax.

| Postmortem imaging feature                          | Category                     | Postmortem process               |
|-----------------------------------------------------|------------------------------|----------------------------------|
| 1. Hyperdense aortic wall                           | Gravity dependent changes    | Blood sedimentation              |
| 2. Sedimentation of blood aorta                     | Gravity dependent changes    | Blood sedimentation              |
| 3. Sedimentation of blood large vessels             | Gravity dependent changes    | Blood sedimentation              |
| 4. Livores heart                                    | Gravity dependent changes    | Livor mortis                     |
| 5. Sedimentation of blood heart                     | Gravity dependent changes    | Blood sedimentation              |
| 6. Livores lung                                      | Gravity dependent changes    | Livor mortis                     |
| 7. Groundglass opacification                         | Gravity dependent changes    | Livor mortis                     |
| 8. Increased density dependent areas skin and subcutis | Gravity dependent changes  | Livor mortis                     |
| 9. Edema dependent areas subcutis                   | Decomposition / Gravity dependent | Cell wall leakage / Livor mortis |
| 10. Intravascular air                               | Decomposition                | Putrefaction                     |
| 11. Gas formation heart                             | Decomposition                | Putrefaction                     |
| 12. Gas formation myocardium                        | Decomposition                | Putrefaction                     |
| 13. Susceptibility artifacts heart (gas)            | Decomposition                | Putrefaction                     |
| 14. Pericardial effusion                            | Decomposition                | Cell wall leakage                |
| 15. Pleural effusion                                | Decomposition                | Cell wall leakage                |
| 16. Gas formation lung parenchyma                   | Decomposition                | Putrefaction                     |
| 17. T2 signal decay from subependocardial to subendocardial | Decomposition | Rigor mortis                     |
| 18. Dilated vena cava inferior                      | Miscellaneous                | Loss of blood pressure           |
| 19. Postmortem clotting large vessels               | Miscellaneous                | Clotting                         |
| 20. Collapse large vessels                          | Miscellaneous                | Loss of blood pressure           |
| 21. Dilated heart                                   | Miscellaneous                | Loss of blood pressure           |
| 22. Dilated right atrium                            | Miscellaneous                | Loss of blood pressure           |
| 23. Postmortem clotting heart                       | Miscellaneous                | Clotting                         |
| 24. Collapse of aorta                               | Miscellaneous                | Loss of blood pressure           |
| 25. Postmortem clotting aorta                       | Miscellaneous                | Clotting                         |
| 26. Dilated vena cava superior                      | Miscellaneous                | Loss of blood pressure           |
| 27. Gas formation subcutaneous areas                | Decomposition                | Putrefaction                     |
| 28. Liquid trachea / bronchi                        | Decomposition                | Cell wall leakage                |

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| Postmortem feature               | Category            | Postmortem process |
|---------------------------------|---------------------|--------------------|
| 1. Intestinal sedimentation     | Gravity dependent   | Sedimentation      |
| 2. Livores liver                | Gravity dependent   | Livor mortis      |
| 3. Sedimentation gall bladder   | Gravity dependent   | Sedimentation      |
| 4. Livores spleeney             | Gravity dependent   | Livor mortis      |
| 5. Livores kidneys              | Gravity dependent   | Livor mortis      |
| 6. Free air                     | Decomposition       | Putrefaction       |
| 7. Fluid in the abdomen         | Decomposition       | Cell wall leakage  |
| 8. Gas in the intestinal wall   | Decomposition       | Putrefaction       |
| 9. Distended intestines         | Decomposition       | Putrefaction       |
| 10. Gas liver parenchyma        | Decomposition       | Putrefaction       |
| 11. Air liver vessels           | Decomposition       | Putrefaction       |
| 12. Gas bile ducts              | Decomposition       | Putrefaction       |
| 13. Periportal edema            | Decomposition       | Cell wall leakage  |
| 14. Gas spleen parenchyma       | Decomposition       | Putrefaction       |
| 15. Gas kidney parenchyma       | Decomposition       | Putrefaction       |
| 16. Intravascular air           | Decomposition       | Putrefaction       |
| 17. Collapse aorta              | Miscellaneous       | Loss of blood pressure |
| 18. Collapse vena cava          | Miscellaneous       | Loss of blood pressure |
| 19. Dilated vena cava           | Miscellaneous       | Loss of blood pressure |

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| Table 6. Patient demographics. |
|--------------------------------|
| Patient demographics (n = 100) |
|--------------------------------|
| Men/women (n)                  | 62/38                |
| Mean age (SD, min-max)         | 62.7 (±13.0, 25–92)  |
| Mean PTI (SD, min-max)         | 22.6 (±15.4, 3.1–71.5) |
| PRS (n)                        | 43                   |
| Mean image acquisition time    | MRI: 59 minutes, CT: 3–4 minutes |
| Hospital ward                  | n                   |
| ICU                            | 38                  |
| ER                             | 15                  |
| Internal medicine / gastroenterology | 11               |
| Oncology                       | 8                   |
| Neurology                      | 6                   |
| Thoracic surgery               | 5                   |
| Hematology                     | 5                   |
| Pulmonology                    | 5                   |
| General surgery                | 4                   |
| Gynecology/urology             | 3                   |
| Antemortem imaging             | Not available (n)   |
| Brain                          | 65                  |
| Thorax                         | 30                  |
| Abdomen                        | 31                  |
| CT (n)                         | 26                  |
| MR (n)                         | 5                   |
| CT and MR (n)                  | 4                   |
| SD = standard deviation; PTI = postmortem time interval (hours); PRS = post-resuscitation status; ICU = intensive care unit; ER = emergency room; CT = computed tomography, MR = magnetic resonance |

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Staphylococcus aureus, multi-drug resistant Acinetobacter), and open abdominal wounds that could not be completely closed or taped to prevent leakage of body fluids.

All cases underwent total-body PMCT and PMMR followed by biopsies under CT (torso) or stereotactic guidance (brain) according to standardized protocols (Tables 1 and 2). Total scan time was approximately 60 minutes for PMMR and 10 minutes for PMCT. First PMMR was performed on a 1.5T scanner (Discovery MR450, GE Healthcare, Milwaukee, Wisconsin USA) and consisted of scans from the head to the pelvis (legs were omitted because of MR scanner availability). Directly after PMMR, PMCT was performed on a dual-source CT scanner (SOMATOM Definition Flash, Siemens Healthcare Forchheim, Germany) and consisted of scans from head to feet. Standardized CT-guided biopsies (12 Gauge) were taken from

| PMCT and PMMR features of brain (n = 100) | PMCT | PMMR |
|-----------------------------------------|------|------|
| Loss of grey-white matter differentiation | 85%  | 85%  |
| Hyperdensity superior sagittal sinus    | 96%  | NA   |
| Hyperdensity veins                      | 54%  | NA   |
| Sulcal effacement                       | 44%  | 41%  |
| Hyperdensity Willis’ circle and cerebral arteries | 35%  | NA   |
| Liquid paranasal sinuses                | 32%  | 32%  |
| High T1 signal basal ganglia and thalamus | NA   | 32%  |
| Thickened / irregular aspect falx       | 20%  | NA   |
| Intracranial air cerebral vasculature   | 8%   | 1%   |

PMCT = postmortem CT; PMMR = postmortem MR; NA = not assessable

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| PMCT and PMMR features of thorax (n = 100) | PMCT | PMMR |
|------------------------------------------|------|------|
| Heart and large vessels                  |      |      |
| Air heart                                | 44%  | 22%  |
| Sedimentation of blood heart             | 62%  | 84%  |
| Dilatation right atrium and ventricle    | 25%  | 25%  |
| Pericardial effusion                     | 26%  | 27%  |
| T2 signal decay epicardial               | NA   | 12%  |
| Postmortem clotting heart                | 4%   | 8%   |
| Air pericardial space                    | 4%   | 2%   |
| Air coronaries                           | 18%  | 12%  |
| Hyperdense aortic wall                   | 90%  | NA   |
| Sedimentation in blood vessels           | 71%  | 88%  |
| Postmortem clotting vessels              | 25%  | 38%  |
| Intravascular air                        | 31%  | 8%   |
| Collapse of thoracic aorta               | 30%  | 29%  |
| Lungs                                   | PMCT | PMMR |
| Internal livores                         | 86%  | 85%  |
| Pleural effusion                         | 31%  | 38%  |
| Liquid trachea / main bronchi            | 78%  | 78%  |

PMCT = postmortem CT; PMMR = postmortem MR; NA = not assessable

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heart, lungs, liver, kidneys, spleen, and additional biopsies were taken from radiologically suspected pathology. All biopsies were stained with hematoxylin and eosin (H&E) and when requested by the pathologist additional stains were performed.

**Scoring**

We composed a scoring list of PMCT and PMMR features of postmortem change (Tables 3–5). The features that were included were based on our radiological expertise [9, 10] and were supplemented with features from published postmortem imaging studies. [5, 12, 20–51]

All cases were retrospectively and independently scored by a radiologist (ACW; board-certified with 10 years of clinical expertise in postmortem imaging) and a researcher (IMW with 3 years of expertise). When available, clinical information and antemortem scans were reviewed. Specific clinical conditions were scored; including intensive care unit (ICU) admittance and post-resuscitation status (PRS).

PMCT and PMMR features were–if possible–categorized to a specific chemical and/or physical process; 1. gravity dependent changes; 2. decomposition; 3. rigor mortis; 4. algor mortis. Features that could not be classified to any of these four processes were labeled to a miscellaneous category.

**Statistical analyses**

We recorded percentage of male/female cases, mean age at death, and mean postmortem time interval (PTI) including standard deviations. PTI was defined as the time from death to the start of MR scanning. For each case, we calculated the frequency of PMCT and PMMR
Fig 1. Postmortem imaging features of the brain. (A) PMCT: Symmetrical hyperdense cerebral arteries (arrowheads). (B) PMCT: Hyperdensity in the dependent cerebral veins and sagittal sinus (arrowheads). (C)
features. Fisher’s exact test was used for the association between specific clinical conditions (ICU and PRS) and frequencies of PMCT and PMMR features. Linear discriminant analysis was used to evaluate the correlation of PTI and PMCT and PMMR features. ANOVA was used to test the correlation of PTI and a combination score for all decomposition and all gravity dependent changes. The inter-observer agreement was calculated using kappa statistics (agreement <0.2: poor, 0.2–0.4: fair, 0.4–0.6: moderate, 0.6–0.8: good, and 0.8–1.0: very good). Furthermore we calculated inter-observer agreement for the group of pathological mimics (those postmortem changes that were most likely to be confused with real pathologic changes) and a group of postmortem changes that does not correspond to a pathologic process with similar radiological features.

Results

We scanned 100 cases from January 2012 to December 2014. The mean age was 62.7 (±13.0), 62% were male (Table 6). Inter-observer agreement was very good, with a kappa of 0.84 for PMCT and 0.83 for PMMR. The kappa score for the group with pathological mimics was 0.79 for PMCT and 0.76 for PMMR, the kappa score for the non-pathologic mimics was 0.89 for PMCT and 0.88 for PMMR.

Total-body CT and MR features of postmortem change—general overview

PMCT and PMMR features of different organs and observed frequencies are presented in Tables 7–9.

**Brain.** Sedimentation led to increased attenuation of the posterior sagittal sinus (96%), cerebral veins (54%) and cerebral arteries (35%) in a symmetric distribution (Fig 1A and 1B). Putrefactive gas in the brain was seen in only a few cases (8%). Liquefaction was not observed. PMMR showed high T1 signal of the basal ganglia in one third of cases (Fig 1C). Effacement of sulci (Fig 1D) and loss of grey-white matter differentiation was seen in the majority of cases (85%) (Fig 1E and 1F).

**Heart and large vessels.** The right atrium and ventricle were dilated in 25% (Fig 2A). The thoracic aorta showed clotting in 38% of cases and was detected best on PMMR (Fig 2B). Air in the heart chambers was seen in 44% (Fig 2C and 2D). No air was observed within the myocardium. T2 signal decline in the myocardium from the epicardial to endocardial regions was seen in 12% (Fig 2A). We observed a collapse of the thoracic aorta in 30% (Fig 2E). Sedimentation of blood was often present in the heart (84%) (Fig 2A) and large thoracic vessels (Fig 2F). The thoracic aortic wall showed increased attenuation in a majority of cases (90%) (Fig 2G). The abdominal aorta was collapsed in 67% and the abdominal vena cava in 53% (Fig 2H). Air in the vertebral venous plexus was seen less frequently (11%), and usually in cases with extensive intravascular air.

**Lungs.** Livor mortis affected the lungs frequently (86%); it appeared as areas with increased density or high T2 signal in the dependent areas of the lungs. In these parts of the lung it is challenging to distinguish livores from pneumonia (Fig 3A–3I) or other interstitial diseases. Liquid in the trachea and bronchi was very common (78%). Pleural effusion was seen in only 38% of cases.

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Liver, spleen, kidneys, gallbladder, pancreas, adrenals. Internal livores of the spleen and kidney were noted by two layers of different T1 and T2 signal reflecting blood settling in the parenchyma. In the liver, three layers can be seen: an upper layer with small amounts of putrefactive gas, a middle layer with intermediate signal and a lower layer that together with the middle layer reflect settling of blood (Fig 4A–4F). Gravity can cause sedimentation of the gallbladder content and this is best seen on PMMR as vertical signal gradients. Livor mortis in organ parenchyma (spleen 31%, kidneys 6% and liver 74%) was also best depicted on PMMR and presented as different layers of T1 and T2 signal. In general, livores of the organ parenchyma were not clearly detectable on PMCT. Periportal edema was found on PMMR in 27% (Fig 5A). Putrefactive gas in the liver vasculature was seen on PMCT in 37% (Fig 5B). The imaging features of the pancreas and adrenal glands were not notably affected by postmortem change.

Stomach, intestines, abdominal cavity. Sedimentation in the stomach and intestines was seen in only a few cases (15%). Fluid in the abdomen was present in 35%. Bowel distension (14%), gas in the intestinal wall (8%) and free abdominal air (7%) were less common features (Fig 5C–5F).

Soft tissues. On PMCT superficial internal livor mortis was manifested as increased densities of the dependent subcutaneous areas (37%). Putrefactive gas in subcutaneous tissue was not observed.

Total-body CT and MR features of postmortem change—in relation to clinical conditions and postmortem time interval

Intensive care unit admittance. In our cohort 38/100 patients died in the ICU. Livores of the liver was seen significantly more often in ICU patients than in non-ICU patients (92% vs. 62%, p = 0.001) (Table 10). High T1 signal of the basal ganglia was significantly less frequently observed in ICU patients (44% vs. 13%, p = 0.001).

Post-resuscitation status. Forty-three patients underwent unsuccessful resuscitation just prior to death. Pleural effusion (p<0.001) and periportal edema (p = 0.001) were seen significantly more often in patients that had undergone resuscitation (Table 10). Postmortem clotting occurred significantly less frequently in patients that had underwent resuscitation (p = 0.002). Intravascular air (both arterial and venous) was visible in 58% of patients and more frequently present in PRS patients than in non-PRS patients (72% vs. 47%, p = 0.013).

Postmortem time interval. The mean PTI was 23.0 (±15.6) hours. PTI showed a significant correlation with internal livores of the lungs (p = 0.038), distended intestines (p = 0.001) and loss of grey-white matter differentiation in the brain (p<0.001) (Table 11). PTI showed a significant correlation with postmortem changes related to decomposition (p = 0.026).

Discussion

This is the first study evaluating the frequency of PMCT and PMMR features of postmortem change in a large cohort of adult patients. Similar imaging studies on postmortem change in
fetuses and neonates have been published. [13, 52]. We observed a wide variety of PMCT and PMMR features of postmortem change. Particularly livor mortis and decomposition have great impact on the imaging features. Algor mortis and rigor mortis lead to only minor changes. Our results indicate that PMCT and PMMR appear to be complementary for correct interpretation of postmortem changes. Some changes are more clearly seen on PMMR such as livores of organ parenchyma or blood clotting, while others such as the presence and distribution of putrefaction air is better noted on PMCT.

Clinical conditions may influence imaging features of postmortem change. Importantly, postmortem changes may mimic or even mask real pathological changes related to the cause of death: e.g. gravity causes sedimentation of blood contents within the first hours after death. On PMCT the upper (plasma) and lower layer (blood cells) shows decreased and increased attenuation respectively. As a result the upper part of the aortic wall shows relatively high attenuation compared to the plasma content and may mimic aortic wall hematoma (Fig 2G).

Bacterial infections can speed decomposition processes and increase gas and fluid formation in the body. Hypovolemia causes the heart cavities and vessel lumen to decrease in size.

Medical treatments can also change imaging features; e.g. intravascular lines and surgical wounds can be accompanied by air in the surrounding soft tissues and bloodstream.

Resuscitation can cause rib fractures, pneumothorax, lung contusions, hemothorax, and intravascular air. We found that the majority of PRS patients showed significantly more intravascular air as opposed to non-PRS patients, suggesting that air was introduced during resuscitation. [12, 27, 53, 54] Intravascular air after resuscitation is caused by pneumatization of dissolved gas in the blood as a result of compression and expansion of vessels and direct mechanical force to the chest allowing air from the lungs to enter the bloodstream. [55, 56] Likewise resuscitation attempts may introduce free abdominal air that should not be confused with free air caused by intestinal perforation. [13] Pleural effusion, periportal edema, and distended intestines were also more frequently observed after resuscitation. Postmortem clotting occurred less often in PRS patients and we hypothesize this is caused by anti-coagulation given during resuscitation attempts. [12, 27, 53, 54]

Lack of oxygenation in the brain was noted by loss of grey-white matter differentiation, edema, swelling of the brain and effacement of sulci. [23, 57, 58] These features involve the entire brain and are symmetrical. [14, 23] Patients with elevated intracranial pressure prior to death may show similar features, and comparison to antemortem scans is recommended. In living patients a dense-artery-sign in the cerebral arteries is often asymmetric and indicative for cerebral ischemia, a postmortem mimic of this sign is usually symmetrical and is non-pathological (Fig 1A). The cessation of cardiac output and fall in blood pressure causes the arterial wall to collapse directly after death. [30, 59] This change may obscure an aortic aneurysm or dissection. Within 2 hours after death blood clots form in the heart and large vessels.

Postmortem clots are best detected on PMMR; the clot shows low T2 signal relative to the high T2 signal of the serum. A postmortem clot can often be distinguished from a central pulmonary embolism that shows a more homogeneous high T1 signal (Fig 6A–6I). Other distinctive features of postmortem clots are that they are seen in the dependent areas of the vessel,
Fig 4. Internal livores of the liver and spleen. (A/C) T1w (A) and T2w fs (C) PMMR. Internal livores. In the liver 3 distinct layers (arrowheads) of different T1 signal can be seen and in the spleen 2 layers (arrows). A low T1 signal layer on top, relatively intermediate-to high signal layer in the middle and a low T1 signal layer in the dependent part of the liver. On T2w fs internal livores are not clearly seen. (B/D) T1w (B) and T2w fs (D) PMMR. The spleen shows 2 layers of different T2 signal (arrowheads). T1w show no clear livores in this patient. (E) HE, x100 original magnification. Centrilobular area of the liver with wide sinuses extended by blood (asterisks). (F) HE, x200 original magnification. Congested spleen with lakes of blood (asterisks).

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Fig 5. Postmortem imaging features of the abdomen. (A) T2w PMMR: Periportal edema (arrowhead) and subcutaneous edema (asterisks). (B) PMCT: Putrefactive gas in the liver vessels (arrowhead) and distended stomach (asterisk). (C/ D) PMCT: Gas in the intestinal...
usually fill only part of the lumen and do not expand the lumen. With pulmonary embolism the thrombus follows the blood stream until it reaches a point where the lumen becomes too narrow or the vessel branches. The shape of a postmortem clot is often more irregular than a thrombus (Fig 6C and 6D). If clinically relevant, a CT-guided biopsy may help differentiate between postmortem clotting and pulmonary embolism.

Table 10. Postmortem CT and MR features in relation to clinical conditions.

| Clinical condition                        | Modality* | Yes   | No    | Total | P-value |
|------------------------------------------|-----------|-------|-------|-------|---------|
| Intensive care unit admittance           | PMCT      | N = 38| N = 62| N = 100| 0.133   |
| Hyperdense cerebral arteries             | PMMR      | 17 (45%)| 18 (29%)| 35 (35%)| 0.002   |
| High T1 signal basal ganglia             | PMMR      | 5 (13%)| 27 (44%)| 32 (32%)| 0.098   |
| Postmortem clotting                      | PMMR      | 21 (55%)| 23 (37%)| 44 (44%)| 0.286   |
| Subcutaneous edema                       | PMMR      | 17 (45%)| 20 (32%)| 37 (37%)| 0.133   |
| Fluid in the abdomen                     | PMMR      | 17 (45%)| 18 (29%)| 35 (35%)| 0.098   |
| Livores liver                            | PMMR      | 35 (92%)| 39 (63%)| 74 (74%)| 0.001   |
| Livores spleen                           | PMMR      | 10 (26%)| 21 (34%)| 31 (31%)| 0.507   |
| PRS                                      | PMMR      | N = 43| N = 57| N = 100| 0.002   |
| Pleural effusion                         | PMMR      | 25 (58%)| 13 (23%)| 38 (38%)| <0.001  |
| Periportal edema                         | PMMR      | 19 (44%)| 8 (14%)| 27 (27%)| 0.001   |
| Distended intestines                     | PMCT/PMMR | 9 (21%)| 5 (9%)| 14 (14%)| 0.144   |
| Postmortem clotting                      | PMMR      | 11 (26%)| 33 (58%)| 44 (44%)| 0.002   |
| Dilated right atrium / ventricle         | PMCT/PMMR | 15 (35%)| 10 (18%)| 25 (25%)| 0.063   |
| Intravascular air                        | PMCT      | 31 (72%)| 27 (47%)| 58 (58%)| 0.015   |

*Specifies the modality that has the highest detection of the postmortem changes

PMCT = postmortem CT; PMMR = postmortem MR; PRS = post-resuscitation status

Table 11. Postmortem CT and MR features in relation to postmortem time interval.

| Postmortem time interval                  | Modality* | <12 hours | 12–24 hours | 24–48 hours | >48 hours | Total | P-value |
|------------------------------------------|-----------|-----------|-------------|-------------|-----------|-------|---------|
| Intravascular air                        | PMCT      | N = 25    | N = 37      | N = 28      | N = 10    | N = 100| 0.905   |
| Sedimentation blood                      | PMMR      | 14 (56%) | 23 (62%)    | 16 (57%)    | 5 (50%)   | 58 (58%)| 0.416   |
| Loss of grey-white matter differentiation| PMCT/PMMR | 14 (56%) | 33 (89%)    | 28 (100%)   | 10 (100%) | 85 (85%)| <0.001  |
| Distended intestines                     | PMCT/PMMR | 0 (0%)    | 4 (11%)     | 7 (25%)     | 3 (30%)   | 14 (14%)| 0.001   |
| Postmortem clotting                      | PMMR      | 6 (24%)  | 20 (54%)    | 12 (43%)    | 6 (60%)   | 44 (44%)| 0.197   |
| Livores lungs                            | PMCT/PMMR | 20 (80%) | 30 (81%)    | 26 (93%)    | 10 (100%) | 86 (86%)| 0.038   |
| Livores liver                            | PMMR      | 17 (68%) | 27 (73%)    | 25 (89%)    | 5 (50%)   | 74 (74%)| 0.805   |
| Livores spleen                           | PMMR      | 5 (20%)  | 11 (30%)    | 10 (35%)    | 5 (50%)   | 31 (31%)| 0.062   |
| Gravity dependent changes                | PMCT/PMMR | 50%       | 47%         | 51%         | 56%       | 50%    | 0.094   |
| Decomposition                            | PMCT/PMMR | 23%       | 30%         | 29%         | 34%       | 28%    | 0.026   |

*Specifies the modality that has the highest detection of the postmortem change

PMCT = postmortem CT; PMMR = postmortem MR

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In this study we investigated in-hospital deceased adult deaths. The mean PTI was relatively short and bodies were stored in a protected environment after death, PTI seem to have an impact on the occurrence and extent of specific changes.

Autolysis occurs early after death. It leads to significant changes that can be noted at microscopic examination of tissues obtained at biopsy, in particular of the pancreas and adrenal glands. [12, 61] However, in our cohort imaging features of these organs seem less affected by autolysis. Imaging features related to decomposition were seen more frequently. There was tendency to more extensive livores of the lungs with longer PTI and the livores can become so extensive as to completely consolidate the lung parenchyma. In such cases, accurate diagnosis of underlying parenchymal disease can be challenging. In such cases we highly recommend to biopsy both normal and suspected parts of parenchyma to reliably differ postmortem changes from infection (Fig 3A–3I), hemorrhage, or tumor. [13, 14, 62, 63]

The distribution of putrefactive gas also differs with a different PTI, first occurring in the heart cavities and large vessels and with longer intervals in the smaller vessels, organ parenchyma and soft tissues. Putrefactive gas must be differentiated from pathological air collections, such as soft tissue emphysema, free air or gas in the intestinal wall. Putrefactive gas usually has an intestinal origin and travels through the mucosa to the portal veins in the early stage. It may mimic air embolism, however the latter will show a more equal distribution throughout the vascular system. [14] Intestinal bacteria continue to produce gas after death causing bowel distension. The amount of intestinal air significantly increases with longer PTI. This may look similar to a bowel obstruction or paralysis, and should be carefully evaluated. With longer PTI, putrefaction can also lead to formation of subcutaneous air.

Our study had several limitations. We composed a scoring list of postmortem imaging features that may not be complete and some features may be missing. We did not measure body temperature during scanning. Ideally, body temperature should be monitored to allow adaptation of MR scan parameters to temperature variations to achieve optimal tissue contrast. However all bodies were stored at the morgue at a constant temperature of 5 degrees Celsius prior to scanning and the transit time from the morgue to the MR scanner was equal for all cases.

We optimized MR sequences for scanning of cold corpses. Furthermore the scan time was maintained approximately the same for all scanning sessions while the temperature in the scanning room was kept constant.

Conclusion

There is a wide variety of imaging features of postmortem change in in-hospital deaths. These imaging features vary among clinical conditions, increase with longer PTI and must be distinguished from pathologic changes.
Supporting information
S1 File. Dataset PM changes. (SAV)

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