Diagnosis of pulmonary infarction in post-mortem computed tomography and post-mortem magnetic resonance imaging - a technical note.

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Abstract

Pulmonary thromboembolism may be accompanied by pulmonary infarction. Even though pulmonary thromboembolism (PTE) is a frequently found cause of death at autopsy, pulmonary infarction accompanying PTE is a less common finding and may therefore easily be misinterpreted as infectious or cancerous lung disease. Appearance of pulmonary infarction in post-mortem imaging and acquisition parameters helping to identify pulmonary infarctions are not described yet. Based on a case of a 50-year-old man who died due to PTE and presented pulmonary infarction we suggest using a pulmonary algorithm in post-mortem computed tomography combined with post-mortem magnetic resonance imaging of the lungs using conventional T1 and T2 weighted sequences.

Introduction

We present post-mortem imaging and autopsy results of a 50-year-old man who died from right cardiac failure due to PTE. Post-mortem imaging comprised whole body unenhanced post-mortem computed tomography (PMCT) and unenhanced post-mortem magnetic resonance imaging (PMMR) of the chest prior to forensic autopsy. PM-imaging revealed pulmonary findings suspicious of pulmonary infarction.

Methods and results

Post-mortem imaging (PMCT and PMMR)

The interval between death and PMCT was 5 hours. Whole body unenhanced PMCT imaging was performed as part of the routine forensic examination using a Siemens Somatom Definition AS 64. Tube voltage was 140 kV, caredose, slice thickness 1 mm, increment 0.7 mm, a rotation time of 0.5 s and a field of view of 570 mm. Soft tissue algorithm and bone tissue algorithm were used. The thorax was reconstructed using a specific pulmonary algorithm with a field of view of 332 mm.

PMMR of the thorax was conducted immediately after PMCT. Body temperature at the time of scanning was 23 °C. For PMMR (Philips Achiva 3T) imaging the body was placed in an artifact-free body bag. The applied sequences were T2-weighted sequences (T2_Dixon_tra; slice thickness: 3mm, GAP: 0.3mm, TR: 3957ms, TE: 90ms, NSA: 2 and T2_Dixon_cor; slice thickness: 3mm, GAP: 0.3mm, TR: 3957ms, TE: 90ms, NSA: 2) as well as T1-weighted sequences (T1_Dixon_tra; slice thickness: 3mm, GAP: 0.3mm, TR: 702ms, TE: 7.39ms, NSA: 1 and T1_Dixon_cor; slice thickness: 3mm, GAP: 0.3mm, TR: 702ms, TE: 7.44ms, NSA: 1). Dixon technique was chosen according to our standard PMMR-protocol allowing to obtain water only, fat only as well as in-phase and out-of-phase images in a single acquisition. PMMR examination time was 25 minutes. All images (PMCT and PMMR) were viewed and evaluated in a PACS (Sectra Workstation IDS7, Version 17.1.16.3569, Linköping/Sweden) by a board-certified radiologist experienced in post-mortem imaging.

Postmortem imaging results

PMCT and PMMR showed three wedge-shaped, pleura-based lesions in the lung periphery (one lesion in the upper right lobe, approx. 29 mm x 22 mm x 31 mm, and two in the lower right lobe, approx. 25 mm x 21.5 mm x 26 mm and 40 mm x 29 mm x 38 mm, respectively). Lesions appeared mainly hyperdense with smaller hypodense areas in PMCT (Fig. 1). In PMMR lesions appeared mainly hypointense to isointense containing focal hypointense areas in T1w and mainly hyperintense with focal hypointense areas in T2w in-phase images (Fig. 1). Appearance of pulmonary lesions in Dixon water only, fat only and out-of-phase images is shown in Table 1. Additionally, homogeneous material of intermediate signal intensity within the large and small pulmonary arteries of both lungs compatible with PTE was found in T2w PMMR [1].

Autopsy and Histology

Autopsy was conducted immediately after imaging by board-certified forensic pathologists according to the Recommendation of the Committee of Ministers to Member States of Europe on the harmonization of medico-legal autopsy rules [2] and included external and internal examination with opening of all three body cavities and dissection of all organs**.** During autopsy tissue samples of the lung as well as tissue samples from all other internal organs were collected for histologic examination. Histology staining included hematoxylin and eosin (H&E) and iron (Fe) staining. Histologic examinations and adjudication were performed by board-certified forensic pathologists. Toxicological examinations were not commissioned by the prosecutor.

Autopsy and histology findings

External examination showed signs of upper venous congestion with livid discoloration of the face, neck and upper chest as well as a visible engorgement of the jugular veins. Internal examination revealed thromboembolic occlusion of the large and small

pulmonary arteries of both lungs, partially attached to the vessel walls. Wedge-shaped lesions seen in PMCT and PMMR in the upper and lower lobe of the right lung periphery were identified as pulmonary infarction with surrounding hemorrhage.

H&E staining of the wedge shaped lung lesions showed central infarcted necrotic lung tissue and a surrounding hemorrhagic rim. Iron staining revealed iron positive macrophages at the edge of necrotic lung tissue.

Furthermore, autopsy showed concentric hypertrophy of the heart (heart weight 520 g; 180 cm body length, 81 kg body weight) and an asymmetric heart configuration with a hypertrophic right ventricle and round configuration of the apex. The large pulmonary arteries were dilated and showed plaques indicative of pulmonary hypertension.

The manner of death was assessed as a natural death, the cause of death was determined as right cardiac failure due to PTE.

Discussion

PMCT is widely used and accepted as a complementary examination to forensic autopsy in routine forensic examination [3, 4]. Over the last 10 years, PMMR has been introduced as an additional tool in post-mortem imaging mainly feasible for diagnosis of soft tissue and organ pathology [5, 6]. Acute PTE is a frequently found natural cause of death at autopsy [7]. However, pulmonary infarction accompanying PTE is a less common finding. Pulmonary infarction presents mostly in patients with PTE and reduced cardiac function or pre-existing pulmonary disease While PTE has been demonstrated in PMCT and PMMR [5, 8–12], pulmonary infarction has neither been shown nor discussed in PM-imaging.

Pulmonary infarction may present similarly to other pulmonary findings such as lung cancer and infectious lung disease in CT imaging [13]. In clinical CT pulmonary

infarction is often described as wedge-shaped opacities located at the periphery of the lung [14], also known as “Hampton’s hump” [15]. Hyperlucent focal areas may be present within the infarction [16]. Pneumonia, cancerous pulmonary nodules or septic emboli (Fig. 2) may also appear as pleura based, wedge shaped, inhomogeneous lesions in clinical CT [17–19]. Distinction between these entities may therefore be challenging or impossible [13]. Regarding cancerous pulmonary nodules, PMCT is comparable to ante-mortem images in the early post-mortem interval [20]. In our case we observed wedge-shaped, pleura-based lesions at the lung periphery mainly appearing hyperdense with smaller hypodense areas in PMCT (Fig. 1). Thus, similar appearance of pulmonary infarction in clinical CT and PMCT may be assumed. However, since pulmonary infarction may not be distinguishable from infectious or cancerous lung diseases, PMCT alone may not be enough to strengthen the diagnosis of pulmonary infarction and additional PMMR of the lungs may be useful.

Combined appearance of PTE and wedge shaped lesions in post-mortem imaging may hint to acute pulmonary infarction. Post-mortem clots in MRI usually show signs of sedimentation due to gravitation with compartments of lower density containing fibrin and compartments with higher density containing erythrocytes. The structure of vital clots like PTE appears homogenous, with intermediate signal intensity on T2w images, in direct contact with the vessel wall and situated within a hypointense layer of sedimented erythrocytes [21]. Older pulmonary infarction may not be accompanied by clear signs of PTE since previous acute PTE may present as strings and webs of connective tissue [22] in the lumen of the pulmonary arteries, or may be complete remnant free. In clinical contrast-enhanced CT-imaging, these strings are connected to the vessel wall on both sides, surrounded by contrast media [23, 24]. As contrast-

enhanced imaging is not a routine tool in post-mortem imaging, the diagnosis of chronic PTE will likely be established during autopsy.

Regarding pulmonary infarction Bray et al. [25] state that appearance of pulmonary infarction in clinical magnetic resonance imaging varies according to the accumulated aging blood within the alveoli and its signal characteristics. Therefore, blood in hyperacute (less than 24 hours) pulmonary infarction areas appears hyperintense in T2w and hypointense in T1w whereas the lesions appear hyperintense in T1w in the acute interval (up to 1 week). In the presented case infarction areas appeared mainly hyperintense with hypointense components in T2w and hypointense to isointense containing small focal hypointense areas in T1w PMMR (Fig. 1). Based on the above-mentioned characteristics this PMMR signal intensity corresponds to an infarction age slightly over 24 hours. This is in accordance with the known case circumstances as well as autoptic and histologic findings. It can thus be assumed that in a short post-mortem interval, characteristics of pulmonary infarction in PMMR correspond to those in clinical MRI and may be applied for diagnosis and age determination of pulmonary infarction. Literature regarding pulmonary findings in PMMR is scarce. To the best of our knowledge, there is no literature describing scanning protocols in PMMR for depiction of pulmonary infarction. Some of the faced difficulties in PMMR are rapid changes in signal intensity in a post-mortem interval due to sedimentation, decomposition as well as cooling of the corpse [26–28]. Knowing the temperature of the body before scanning is of great importance, as the image contrast decreases for both T1w and T2w images for lower body temperatures. At a body temperature below 20°C on T2w and below 10°C on T1w images, a visible decrease in image contrast was observed [26].

Due to our in-house standard we used Dixon technique [29] for PMMR imaging. The benefit of Dixon technique lies in the simultaneous generation of fat and water suppressed images from a single acquisition. The Dixon in-phase sequences correspond to T1w/T2w images seen in spin-echo sequences. However, fat and water suppressed images are not crucial for identification of pulmonary infarctions. Since the appearance of pulmonary infarction in PMMR seems to be similar to clinical MRI, it is mainly dependent on the accumulated blood and its age characteristics. Therefore, conventional T1 and T2 weighted spin-echo sequences will suffice.

Conclusion

Pleura based wedge shaped lesions in unenhanced PMCT may indicate pulmonary infarction but lesions may not be distinguishable from other pulmonary pathologies. Additional PMMR of the lungs using conventional T1 and T2 weighted sequences may aid in diagnosis and age determination of pulmonary infarction at a short post-mortem interval.

Tables

	In-phase	Out-of-phase	Fat only	Water only
T1w	iso- to hypointense	Iso- to hyperintense	lesion not visible	hyperintense
T2w	Iso- to hyperintense	Iso- to hyperintense	lesion not visible	hyperintense

Table 1
Appearance of pulmonary infarction in PMMR in T1w and T2w Dixon sequences

Figures

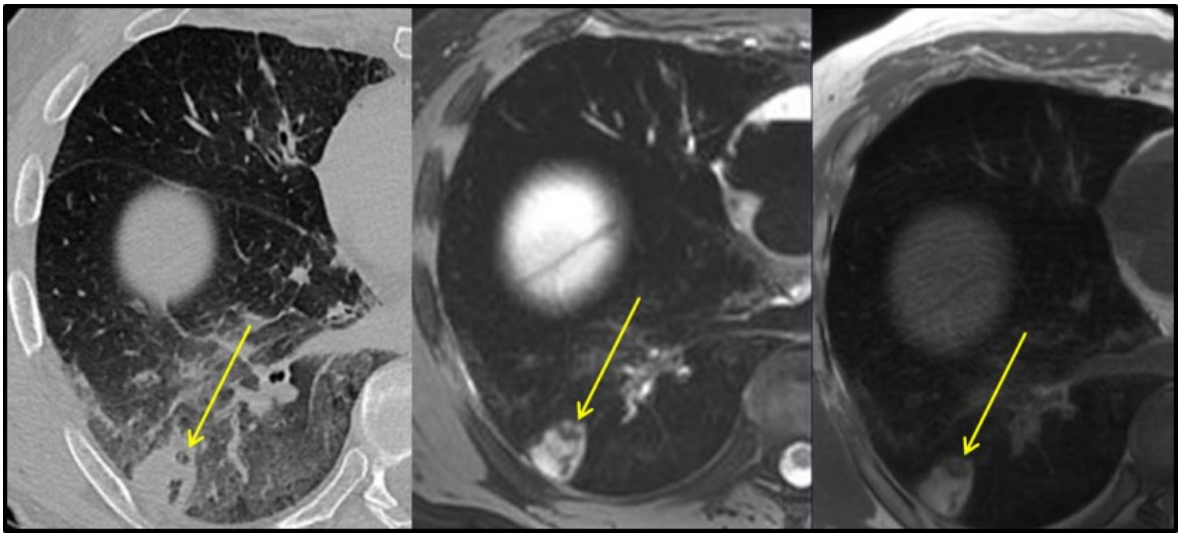

Fig. 1:
Axial PMCT (left image), axial in-phase T2w PMMR (middle image) and axial in-phase T1w PMMR (right image) with exemplary wedge shaped lesion (yellow arrows) in the right lower lobe of the lung.

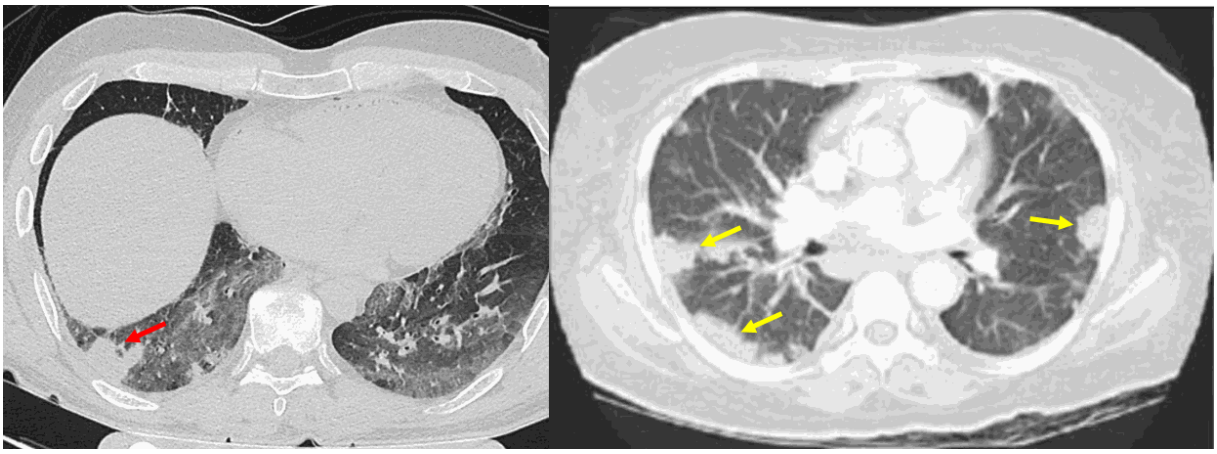

Fig. 2:
Depiction of undistinguishable wedge shaped pleura based lesions in PMCT and clinical CT. The left image shows unenhanced PMCT with pulmonary infarction (red arrow). The right image shows multiple, pleura based lesions due to septic emboli (yellow arrows) [19].

Compliance with Ethical Standards

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

The authors declare that they have no conflicts of interest:

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