ELSEVIER

Seminars in
ULTRASOUND
CT and MRI

Role of Postmortem CT in the Forensic Evaluation of Hemopericardium

Laura Filograna, MD, PhD,* Luca Pugliese, MD,* Adolfo D'Onofrio, MD,* Rosaria Meucci, MD,* Michael John Thali, MD,† and Roberto Floris, MD, PhD*

Hemopericardium (HP) is defined as the accumulation of blood in the pericardial sack. In a clinical setting, prompt identification of the presence of HP is of huge importance, because HP can result in pericardial tamponade. While echocardiography remains the most appropriate method for the evaluation of pericardial effusions in the clinical setting, postmortem imaging computed tomography (PMCT) is a valuable instrument for detecting the presence of HP and in evaluating its significance in causing mechanical impairment of cardiac activity and finally death.

In this article, the actual knowledge on PMCT imaging findings related to HP are reported, with particular attention to the assessment of its significance with relation to the forensic diagnosis of the cause of death. According to the present work, the diagnosis of pericardial tamponade due to HP might be considered one of the critical fields of investigation where classical autopsy may fail and where PMCT imaging may offer its most important aids.

Semin Ultrasound CT MRI 40:79-85

Introduction

Hemopericardium (HP) is the accumulation of blood in the pericardial sack. The HP occurs when an intrapericardial vessel or heart chambers rupture into the pericardium, due to an injury or even spontaneously. The accumulation of blood or other fluids in the pericardium may cause pericardial tamponade (PT).

Whereas this finding is frequently observed at autopsy (Fig. 1), it represents undoubtedly a challenge for the pathologist. In fact, in presence of HP, it is mandatory to assess its ethiopathological relevance in determining the death.

The pathophysiologic mechanism of tamponade is the rapid or slow increase of the intrapericardial pressure that leads to heart chambers compression; thus to the decrease of cardiac inflow and outflow, with reduction of blood pressure.

As is the case in clinical settings as well as in postmortem imaging, computed tomography (CT) is a valuable instrument for detecting the presence of HP and in evaluating its significance in causing mechanical impairment of cardiac activity and finally death.

In the last decades, postmortem imaging has experienced widespread acceptance in forensic settings. The modern imaging techniques have been demonstrated to enhance the potentials of traditional postmortem investigation or even to replace classical autopsy in the determination of cause and manner of death.[1-4]

Recently, some authors have assessed the value of postmortem CT (PMCT) in forensic investigations regarding cases with HP.[5-9]

In this review, after a brief introduction on general aspects of HP and antemortem CT characteristics of pericardial effusion, we examine the role of PMCT in the forensic evaluation of HP. Some interpretational keys are proposed for the postmortem assessment of HP, based on its PMCT characteristics, particularly for the understanding of its significance with respect with the cause of death.

*Department of Diagnostic and Interventional Radiology, Molecular Imaging and Radiotherapy, PTV Foundation, "Tor Vergata" University of Rome, Rome, Italy.

†Department of Forensic Medicine and Imaging, Institute of Forensic Medicine, University of Zurich, Zurich, Switzerland.

Address reprint requests to Laura Filograna, MD, PhD, Department of Diagnostic and Interventional Radiology, Molecular Imaging and Radiotherapy, PTV Foundation, "Tor Vergata" University of Rome, Viale Oxford 81, 00133, Rome, Italy. E-mail: laura.filograna@gmail.com

https://doi.org/10.1053/j.sult.2018.10.005

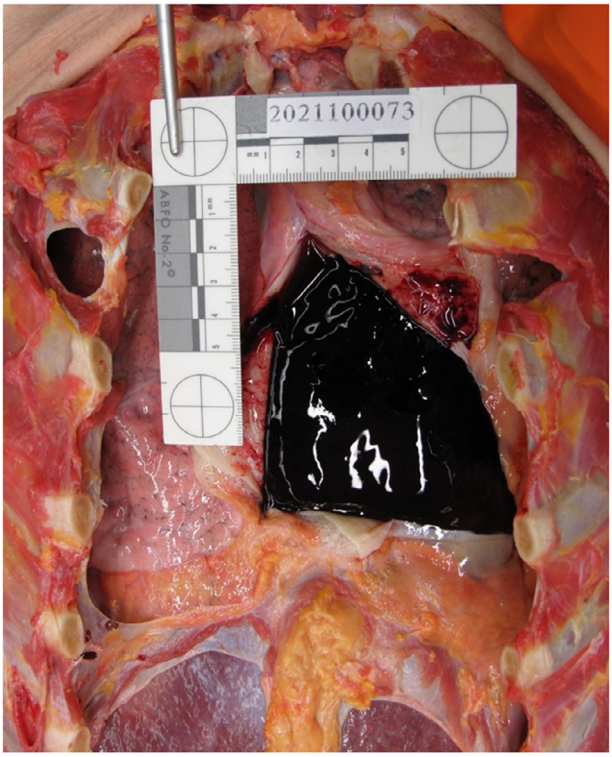

Figure 1 Autopsy photograph of the chest, frontal view after chest cage and pericardial sack opening for macroscopic inspection in a case of postmortem imaging computed tomography (PMCT) documented Hemopericardium (HP). The image shows the presence of HP with the evidence of "clotted" blood on the epicardial surface.

HP: Clinical aspects and antemortem CT imaging characteristics

As defined previously, HP is the accumulation of blood in the pericardial sack. HP may originate from numerous pathologic conditions potentially leading to natural death. These natural death-related etiologies include rupture into the pericardial sack of the ascending aorta by an aneurism or following Stanford type A dissection and adventitia disruption. Heart-related causes of HP include myocardial wall rupture caused by inflammatory alterations in myocarditis, postinfarction pathology or neoplastic disease, both primary and secondary.[10] However, HP may have also a traumatic origin due to sharp or blunt injury, following gunshot wounds to the chest, or during medical procedures when a scalpel or a catheter perforate erroneously the myocardium.[11-14]

In the clinical setting, prompt identification of the presence of HP is of major importance, particularly if the blood accumulation in the pericardial sac is rapid, because HP can result in PT. PT is a life-threatening condition that occurs when the intrapericardial pressure exceeds the cardiac diastolic pressure so that heart chamber filling is impaired. As a consequence, the diagnosis of PT can be based not only on the simple presence of fluid-like blood in the pericardial sac, but also on certain signs, symptoms, and clinical findings. Symptoms of PT for example, include tachypnea and dyspnea, and signs include hypotension and pulsus paradoxus, (inspiratory drop in systolic blood pressure of 10% or 10 mmHg), distension of jugular veins, tachycardia, and a quiet precordium. Generally, the primary diagnostic method for HP is echocardiography as this technique can be quickly performed at the bedside. The echocardiographic diagnosis of PT relies on certain gray scale and color-Doppler criteria.[15,16]

Secondary modalities such as CT, magnetic resonance imaging (MRI) of the heart[15,17] are not generally used for the diagnosis of PT. However, a CT examination performed because of a different indication might incidentally reveal a pericardial effusion.

In the literature, there are clinical reports of in vivo CT findings related to the presence of a potentially fatal pericardial effusion. These include pericardial effusion, dilatation of the superior vena cava (SVC) (with a diameter similar to or greater than that of the adjacent thoracic aorta), compression of the coronary sinus of the inferior vena cava (IVC) (with a diameter greater than twice that of the adjacent abdominal aorta) and of the pulmonary trunk, bowing of the interventricular septum, flattened heart sign (FHS), distension of the hepatic and renal veins, periportal edema and reflux of contrast into the azygos vein and IVC.[10,17,18-26]

Singularly considered, these findings are not specific for an imminent PT, because they may be encountered also in other pathological disorders; however, the simultaneous presence of some of them may highly suggest this life-threatening condition.

All the above mentioned CT findings indicative of PT are explained by the effects of the increase of intrapericardial pressure on the cardiac chambers and the intrapericardial vessels that are compressed, and on the venous vascular system afferent to the heart that are distended and by the obstacle to the cardiac chambers inflow of the blood and consequently of the contrast media. A brief explanation of the pathophysiological mechanisms causative for some of these findings follows.

The FHS[22] occurs when the increase of the intrapericardial pressure is so high to cause during diastolic phase a transient reversal of the transmural left ventricular pressure. In these conditions a change in the contour of the anterior aspect of the heart characterized by a flattening of the anterior wall of the right ventricle with decrease of the cardiac anteroposterior diameter is observed. Right-sided chambers are in fact more prone to compressive deformity because they are thinner and thus more compliant than the left-sided ones.

The bowing of the interventricular septum is considered another CT finding that can be detected in PT. It is related to the echocardiographic evidence of paradoxical motion of the septum frequently observed in patients with signs of PT.[24-26]

Compression by pericardial effusion of the intrapericardial structures such as the coronary sinus, the pulmonary trunk, and the intrathoracic tract of the IVC in its anterior aspect are among the most common consequences of a critically increased intrapericardial pressure. The obstacle of the cardiac inflow is at the origin of hepatic and renal veins distention and of the enlargement of the SVC.

HP: Postmortem detection significance and PMCT imaging characteristics

HP is a relative frequent finding detected at autopsy as it is due to various causes leading to natural or traumatic (including iatrogenic) death.

The autopsy evidence of HP being potentially the cause of death creates many diagnostic challenges for the pathologist. Part of the problem is that the diagnosis of PT is mainly clinical and based on some antemortem signs and symptoms and instrumental data, many of which are often unavailable for the pathologist performing autopsy. The simple detection at autopsy even of a large HP does not imply that the death was due to PT.

In fact, the scientific literature is not clear about the amount of blood in the pericardial sac that would cause PT. Consequently, in traumatic deaths with the autopsy evidence of a HP of large amount, the cause of death is often attributed to PT. In general, about 200 mL of clotted blood seems to be sufficient. Often, 300-400 mL of blood is found in pericardial sac, and some authors report even greater volumes.[27,28] However, it should be considered that in traumatic cases a pericardial defect might allow blood to escape from the sac, or also may represent the entry for blood to pass into or out of the pericardial sac. This is especially true if cardiopulmonary resuscitation maneuvers had been performed. Most importantly, if clinical and instrumental data are not available, other traumatic or ischemic myocardial injuries or different coexisting potentially fatal pathologic condition (ie, extention to the coronary vessel of a ruptured Stanford type A aortic dissection) should be carefully considered, and the diagnosis of PT as the cause of death questioned.

Echocardiography remains the most appropriate method for the evaluation of pericardial effusions in the clinical setting. But in postmortem investigations, CT has largely retained its crucial role worldwide. PMCT is the perfect technology for a rapid and effective examination of the whole body of the deceased with a particular ability to detect skeletal injuries and for a first discrimination of natural deaths from deaths necessitating deeper forensic investigations.

Even in the clinical setting, CT techniques offer some important advantages over other imaging methodologies such as echocardiography. For example, one of the most important advantages of CT is the ability to visualize the entire body volume of the chest, due to the larger field of view inherent in CT. This offers the possibility of detecting associated alterations of the lungs, mediastinum and other thoracic structures.[10] Moreover, one of the classical drawbacks of echocardiography, the variability related to the operator, is less of a problem in CT.

Moreover, echocardiography has some intrinsic limitations, related to the high rate of false-positive findings caused by confounding adjacent pathologic alterations simulating pericardial effusion such as lower lobe atelectasis and pleural effusions.

Furthermore, the advantages offered by CT also include the potential of depicting with more detail the intrinsic characteristics of pericardial effusion, including density values as well as other characteristics.

This opportunity has been exploited particularly in the postmortem setting, where some PMCT characteristics of HP have been identified.

In 2004, Shiotani et al.[29] were the first authors to describe a specific PMCT finding related HP in acute aortic dissection patients, not previously documented in clinical imaging. This HP appearance was named "hyperdense armored heart" (HAH) and consists of stratification of the HP into two double density concentric rings, the denser in contact with the epicardial surface (Fig. 2). In addition to this sign, Shiotani et al.[4,8,30] identified stratification of the HP as a fluid-fluid level (FFL) (Fig. 3).

It has been suggested that HAH sign was caused by a fibrination process induced by the beating movements on blood cells adhering to the epicardium which results in the high density inner ring and to serum along the pericardium constituting the low density outer ring.[4,9,29-31] This hypothesis might suggest that when on PMCT a stratification of HP as FFL is found, the heart did not beat sufficiently for inducing a fibrination process in terms of time or in terms of effectiveness. Moreover, Yamaguchi et al.[6] postulated the hypothesis that when a FFL stratification of HP is found, this evidence

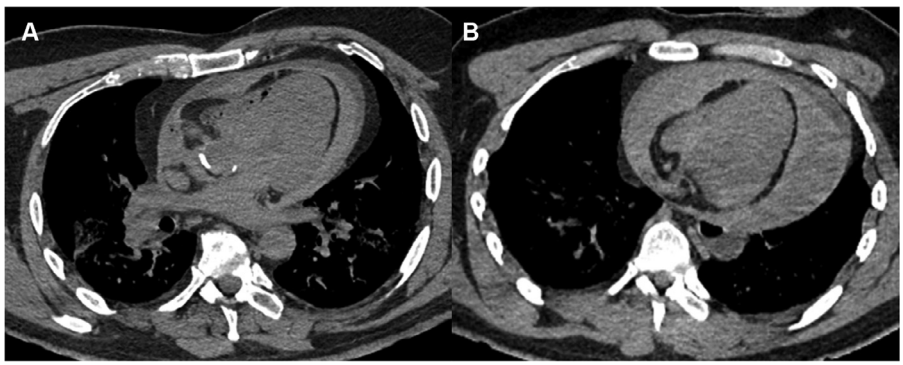

Figure 2 PMCT axial images (A, B) at the cardiac level show a double density stratification of the HP showing hyperdense armored heart (HAH), with the denser ring on the epicardial surface. The image (A) shows the inner ring homogeneous in thickness, but thinner than in (B), where it appears slightly inhomogeneous in thickness and density.

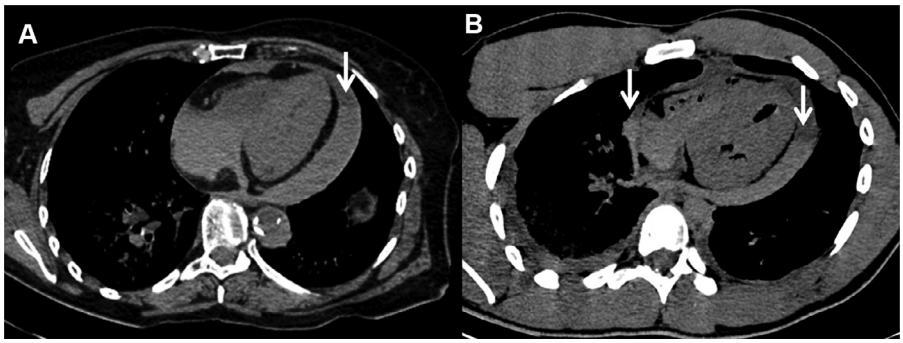

Figure 3 The PMCT axial images (A, B) at the cardiac level show double density stratification of the HP as a fluid-fluid level (arrows), with the denser fluid level in dependent regions of the pericardial sac.

might mean that the pericardial effusion originated after death.

Other findings identified on PMCT that are highly suggestive for an imminent PT in clinical setting were systematically investigated in a study of Filograna et al.[9] In their study, the authors analyzed PMCT images of 14 cases with a clear diagnosis of PT due to HP in the presence of an intact pericardium. Thirteen of 14 cases had HAH, FHS, and other signs of compression of intrapericardial structures such as the coronary sinus and/or the pulmonary trunk. The previously mentioned other findings related to venous system congestion were variably detected. Thus as in postmortem examinations, the most in vivo CT findings suggestive for PT that can be identified include: FHS (Fig. 4), narrowing of coronary sinus and pulmonary trunk (Fig. 5), distension of the IVC, hepatic and renal veins (Fig. 6).

PMCT findings related to HP: Relevance in the postmortem diagnosis of the cause of death due to PT

The autopsy detection of HP does not necessarily indicate that the death was caused by PT.

Even the autopsy finding of a large HP does not definitively indicate that a PT occurred. The postmortem diagnosis of PT can be extremely challenging.

The increasing use of PMCT imaging in forensic investigations and the recognized advantages offered in postmortem examinations are hopeful indications that proper and confident diagnoses of cause of death due to PT are becoming available.

In contrast, in the forensic literature, there are few studies that investigate the use of PMCT in cases of HP[4,13,14,30-35] and even fewer on the possibility of employing this postmortem imaging technique for diagnosis of PT due to HP.[30,31,33]

Recently, in fact, the efforts of the forensic community have been focused on the possibility of using postmortem, static evidence to reflect the in vivo occurrence of dynamic, clinical conditions through the use of PMCT. This is a major advantage potentially offered by noninvasive postmortem imaging techniques especially since all the in vivo signs of an imminent HP cannot be revealed at autopsy, after body opening.

In 2014, Filograna and her group investigated the forensic relevance of PMCT imaging in cases of HP to determining the cause of death due to HP. The authors examined 15 cases with evidence of HP on PMCT performed before autopsy. The study population was divided in 2 groups: the first was constituted by deaths due to PT, and the second due to hemorrhage.

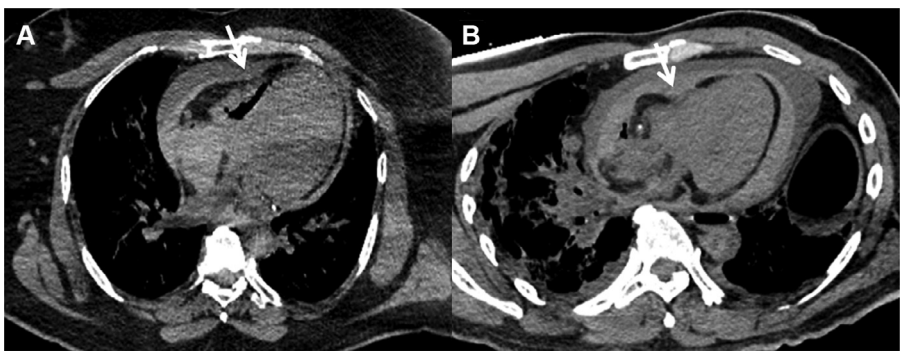

Figure 4 The PMCT axial images (A, B) at the cardiac level show a flattening of the anterior wall of the right ventricle (flattened heart sign) with decrease of the cardiac anteroposterior diameter (arrows).

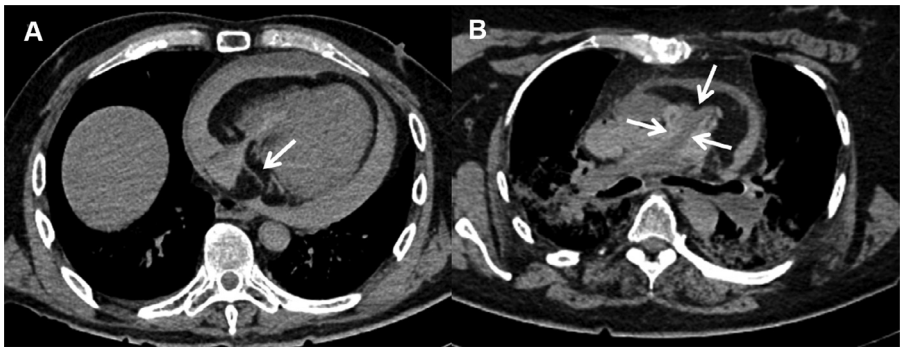

Figure 5 The PMCT axial images (A, B) at the cardiac level show narrowing of coronary sinus (A) and pulmonary trunk (B) (arrows).

The aim was to evaluate autopsy cases with certain diagnoses of PT due to HP and determine which of the above mentioned antemortem CT findings of an imminent PT can be considered on PMCT as indicative for a death due to PT. The final objective was to create a reference for determining the cause of death in autopsy cases with HP where clinical and instrumental antemortem data are not available and/or when other coexistent pathologic alterations may have caused or concurred to cause the death. In the study, with the exception of one case, with respect to deaths due to HP, HAH was always associated with FHS. The authors conclude that the combination of PMCT findings of HAH and FHS and the consideration of circumstantial and clinical data could suggest the postmortem diagnosis of PT.

There is widespread agreement in the literature that the finding of HAH indicates maintenance of antemortem cardiac beating activity before death.[4,6,9,29-31]

As for FHS, Filograna et al. expressed caution in considering this finding as an absolute expression of in vivo increased intrapericardial pressure. In fact, after death with the interruption of the cardiac activity, the cessation of the blood flow results in progressive diminished pressure of the arterial system and flattening of the heart chambers, especially on the right as shown on PMCT.[36]

These doubts were confirmed by the study of Yamaguchi et al.[6] The authors compared PMCT findings in deaths with evidence of HP respectively with a postmortem against an antemortem origin. The authors found that the finding of FHS did not show a statistically significant difference between the 2 groups.

In 2015, Filograna et al.[9] reviewed PMCT images of 14 cases with HP and intact pericardium where the cause of death determined with autopsy and available antemortem clinical and instrumental records was attributed to PT. The study was focused on other than describing PMCT appearances of HP but also on the research of PMCT signs of increased intrapericardial pressure. A control group with PMCT prior to autopsy was analyzed; it was constituted by 11 cases without relevant pericardial effusion, bleeding, and venous system congestion according to autopsy reports.

In this paper, the authors investigate PMCT imaging all the clinical nonenhanced CT signs of an imminent PT due to HP and the PMCT appearances of the HP. The aim was to determine if the same clinical signs and the trait stratification of HP on PMCT can support the postmortem diagnosis of PT due to HP. The authors found that 13 of 14 cases of PT showed an HAH and compression of the coronary sinus and/or of the pulmonary trunk, 14 of 14 had a FHS, against the control group, where none of these findings was observed, except for a distended or noncompletely collapsed SVC, found in all cases. The value of PMCT for the formulation of the postmortem diagnosis of PT as the cause of death was established by this study, at least in cases with an intact pericardium.

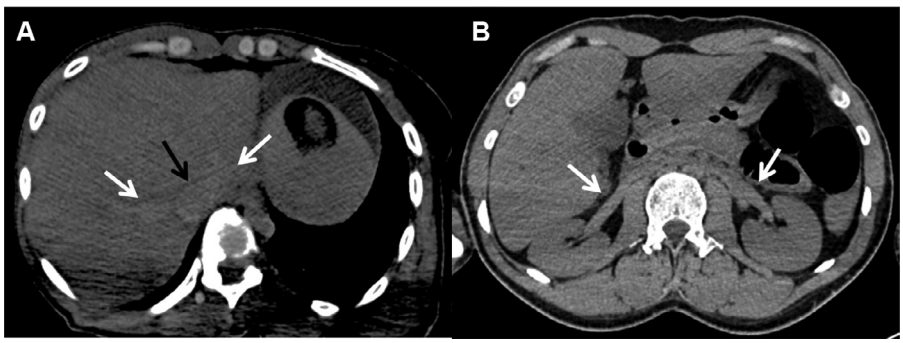

Figure 6 The PMCT axial images (A, B) at the level of the superior abdomen demonstrate distension of the IVC (black arrow), hepatic and renal veins (white arrows). (Color version of figure is available online.)

Conclusions

The postmortem diagnosis of PT due to HP is challenging, particularly if antemortem clinical and instrumental records are not available.

The role of PMCT imaging in forensic examination as a fundamental complementary technique is commonly recognized, so that some authors questioned in some cases the necessity to proceed with a forensic investigation with autopsy after PMCT imaging of the deceased, at least in selected cases.

According to the present work, the diagnosis of PT due to HP might be considered one of the critical fields of investigation where classical autopsy may fail and wee PMCT imaging may offer its most important aids.

In two different works, it was demonstrated that the consideration of PMCT appearances of HP and of indirect signs of an increased intrapericardial pressure might support the postmortem diagnosis of PT.

In 2014,[30] when cases with both intact and nonintact pericardium were analyzed, the simultaneous presence of HAH and FHS signs can be indicative of fatal HP. In 2015,[9] when only cases with an intact pericardium were analyzed and where the diagnosis of PT was clear, the copresence of HAH, FHS, and narrowing of the coronary sinus and/or pulmonary trunk due to compression should be considered PMCT signs highly suggestive for PT due to HP. The question that arises is whether the same last PMCT identified could be found also in nonfatal cases with an intact pericardium, and it is common opinion that lager study populations and further studies are needed.

Nevertheless, as it occurs in clinical setting, and in accordance with Restrepo,[10] we are convinced that, although the CT findings reported in the literature individually detected cannot be considered specific for the diagnosis of a PT, their coexistence can strongly suggest this diagnosis, particularly in a large HP is present.

In conclusion, we maintain that when PMCT is performed and an HP is detected, when antemortem clinical and instrumental data are not available, all the individuated PMCT imaging signs of PT should be carefully searched, for reaching a proper diagnosis of death due to fatal HP.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. Filograna L, Bolliger SA, Spendlove D, et al: Diagnosis of fatal pulmonary fat embolism with minimally invasive virtual autopsy and postmortem biopsy. Leg Med (Tokyo) 12:233-237, 2010
2. Ruder TD, Hatch GM, Thali MJ, et al: One small scan for radiology, one giant leap for forensic medicine. Post-mortem imaging replaces forensic autopsy in a case of traumatic aortic laceration. Leg Med (Tokyo) 13:41-43, 2011
3. Huang P, Wan L, Qin Z, et al: Post-mortem MSCT diagnosis of acute pericardial tamponade caused by blunt trauma to the chest in a motor-vehicle collision. Rom J Leg Med 20:117-122, 2012
4. Filograna L, Hatch G, Ruder T, et al: The role of post- mortem imaging in a case of sudden death due to ascending aorta aneurysm rupture. Forensic Sci Int 228:e76-e80, 2013
5. Gitto L, Serinelli S, Busardò FP, et al: Can post-mortem computed tomography be considered an alternative for autopsy in deaths due to hemopericardium? J Geriatr Cardiol 11:363-367, 2014 Dec. https://doi.org/10.11909/j.issn.1671-5411.2014.04.013
6. Yamaguchi R, Makino Y, Chiba F, et al: Fluid-fluid level and pericardial hyperdense ring appearance findings on unenhanced postmortem CT can differentiate between postmortem and antemortem pericardial hemorrhage. AJR 205:W568-WW77, 2015
7. Ebert LC, Heimer J, Schweitzer W, et al: Automatic detection of hemorrhagic pericardial effusion on PMCT using deep learning - a feasibility study. Forensic Sci Med Pathol 13:426-431, 2017 Dec. https://doi.org/10.1007/s12024-017-9906-1. Epub 2017 Aug 18
8. Ampanozi G, Flach PM, Ruder TD, et al: Differentiation of hemopericardium due to ruptured myocardial infarction or aortic dissection on unenhanced postmortem computed tomography. Forensic Sci Med Pathol 13:170-176, 2017 Jun. https://doi.org/10.1007/s12024-017-9854-9. Epub 2017 Mar 28
9. Filograna L, Laberke P, Ampanozi G, et al: Role of post-mortem computed tomography (PMCT) in the assessment of the challenging diagnosis of pericardial tamponade as cause of death in cases with hemopericardium. Radiol med 2015. https://doi.org/10.1007/s11547-015-0517-1
10. Restrepo CS, Lemos DF, Lemos JA, et al: Imaging findings in cardiac tamponade with emphasis on CT. Radiographics 27:1595-1610, 2007
11. Ruder TD, Ross S, Preiss U, et al: Minimally invasive post-mortem CT-angiography in a case involving a gunshot wound. Leg Med (Tokyo) 12:154-156, 2010
12. Holmes Jr DR, Nishimura R, Fountain R, Turi ZG: Iatrogenic peri-cardial effusion and tamponade in the percutaneous intracardiac intervention era. JACC Cardiovasc Interv 2:705-717, 2009
13. Ebert LC, Schön CA, Ruder TD, et al: Fatal left ventricular rupture and pericardial tamponade following a horse kick to the chest. Am J Forensic Med Pathol 33:167-169, 2012
14. Ebert LC, Ampanozi G, Ruder TD, et al: CT based volume measurement and estimation in cases of pericardial effusion. J Forensic Leg Med 19:126-131, 2012
15. Klein AL, Abbara S, Agler DA, et al: American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr 26:965-1012, 2013
16. Wann S, Passen E: Echocardiography in pericardial dis- ease. J Am Soc Echocardiogr 21:7-13, 2007
17. Oyama N, Oyama N, Komuro K, et al: Computed tomography and magnetic resonance imaging of the pericardium: Anatomy and pathology. Magn Reson Med Sci 3:145-152, 2004
18. Killeen KL, Poletti PA, Shanmuganathan K, et al: CT diagnosis of cardiac and pericardial injuries. Emerg Radiol 6:339-344, 1999
19. Krejci CS, Blackmore CC, Nathens A: Hemopericardium: An emergent finding in a case of blunt cardiac injury. AJR Am J Roentgenol 175:250, 2000
20. Harries SR, Fox BM, Roobottom CA: Azygos reflux: A CT sign of cardiac tamponade. Clin Radiol 53:702-704, 1998
21. Rotondo A, Scialpi M, Catalano O, et al: Periportal lymphatic distension resulting from car- diac tamponade: CT findings and clinical-pathologic correlation. Emerg Radiol 6:85-93, 1999
22. Hernandez-Luyando L, Calvo J, de las Gonzalez Heras E, et al: Tension pericardial collections: Sign of "flattened heart" in CT. Eur J Radiol 23:250-252, 1996
23. Steiner MA, Marshall JJ: Coronary sinus compression as a sign of cardiac tamponade. Catheter Cardiovasc Interv 49:455-458, 2000
24. Doppman JL, Rienmuller R, Lissner J, et al: Computed tomography in constrictive pericardial disease. J Comput Assist Tomogr 5:1-11, 1981
25. Frantz KM, Fishman EK: Hemopericardium leading to cardiac tamponade in the traumatized pediatric patient: Discovery by CT. Clin Imaging 16:180-182, 1992

26. Chong HH, Plotnick GD: Pericardial effusion and tamponade: Evaluation, imaging modalities, and management. Compr Ther 21:378-385, 1995
27. Karger B, Niemeyer J, Brinkmann B: Physical activity following fatal injury from sharps pointed weapons. Int J Legal Med 112:188-191, 1999
28. Moritz AR: The pathology of trauma. Philadelphia: Lea & Febiger, 1942
29. Shiotani S, Watanabe K, Kohno M, et al: Postmortem computed tomographic (PMCT) findings of pericardial effusion due to acute aortic dissection. Radiat Med 22:405-407, 2004
30. Filograna L, Thali MJ, Marchetti D: Forensic relevance of post-mortem CT imaging of the hemopericardium in determining the cause of death. Leg Med 16:247-251, 2014
31. Filograna L, Flach PM, Bolliger SA, et al: The role of post-mortem CT (PMCT) imaging in the diagnosis of pericardial tamponade due to hemopericardium: A case report. Leg Med 16:150-153, 2014
32. Shiotani S, Kohno M, Ohashi N, et al: Postmortem intravascular high-density fluid level (hypostasis): CT findings. J Comput Assist Tomogr 26:892-893, 2002
33. Huang P, Wan L, Qin Z, et al: Post-mortem MSCT diagnosis of acute pericardial tamponade caused by blunt trauma to the chest in a motor-vehicle collision. Rom J Leg Med 20:117-122, 2012
34. Burke M, Parsons S, Bassed R: Management of medicolegal natural deaths from hemopericardium or hemothorax using postmortem CT scanning. Forensic Sci Med Pathol 8:367, 2012. https://doi.org/10.1007/s12024-012-9347-9
35. Okuda T, Shiotani S, Kobayashi T, et al: Immediate non-traumatic postmortem computed tomographic demonstration of myocardial intravascular gas of the left ventricle: Effects from cardiopulmonary resuscitation. Spring 2:86, 2013
36. Ishikawa N, Nishida A, Miyamori D, et al: Estimation of postmortem time based on aorta narrowing in CT imaging. J Forensic Leg Med 20:1075-1077, 2013