



Myocardial Contrast Defect Associated with Thrombotic Coronary Occlusion: Pre-Autopsy Diagnosis of a Cardiac Death with Post-Mortem CT Angiography

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We report the case of a female who died of suspected acute myocardial infarction. Post-mortem CT angiography (PMCTA) was performed with intravascular contrast infusion before the standard autopsy, and it successfully demonstrated the complete thrombotic occlusion of a coronary artery and also a corresponding perfusion defect on myocardium. We herein describe the PMCTA findings of a cardiac death with special emphasis on the potential benefits of this novel CT technique in forensic practice.

Index terms: Post-mortem radiology; CT angiography; Coronary thrombosis; Myocardial perfusion

INTRODUCTION

With the continuous refinements of CT technology, coronary CT angiography has been widely accepted as a powerful noninvasive tool for the assessment of coronary artery disease (CAD) (1). Accordingly, in recent years, the use of CT in autopsy practice (post-mortem CT; PMCT) is being increasingly used to guide the subsequent forensic dissection as a complimentary examination (2, 3). A significant obstacle for widespread use of native PMCT as a standard pre-autopsy protocol, however, is its failure to yield detailed information concerning the vascular structures, particularly in natural cardiovascular deaths. Hence, to address this drawback, PMCT with intravascular

contrast infusion (PMCT angiography; PMCTA) has been introduced, and it has shown promising results for the evaluation of vascular structures, including coronary arteries (4-8). Recently, although various clinical CT techniques have enabled the simultaneous evaluation of myocardial perfusion, as well as coronary morphology, for determining treatment strategies, however, there are very few reports showing how these techniques can be translated to a post-mortem setting using radio-opaque contrasts dedicated to forensic application (9).

Therefore, we report a case of a female who died of suspected myocardial infarction. We performed PMCTA using oily contrast and herein describe the CT findings with the special emphasis on the potential role of PMCTA in forensic medicine. To the best of our knowledge, this is the first report to describe the PMCTA findings concerning a coronary occlusion and myocardial perfusion defect in a patient who died of suspected acute and complete coronary occlusion, using oily contrast. This report followed Declaration of Helsinki for studies with human individuals.

CASE REPORT

A 59-year-old female was admitted to the hospital with sudden onset of acute chest pain. Coronary angiography

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showed focal significant stenosis in the distal segment of the right coronary artery (RCA), and a stent was then successfully placed at the site of narrowing. However, a newly developed vascular dissection was found in the proximal RCA at the end of the procedure, extending to the aortic arch and thoraco-abdominal aorta. Although new stents were implanted in the proximal and middle RCA to restore the true lumen, complete thrombotic occlusion developed in the entire RCA. The patient then underwent immediate aortic hemi-arch replacement and a bypass graft

using saphenous vein after the successful resuscitation of ventricular fibrillation. Nevertheless, she progressed to refractory cardiogenic shock and died 7 days after surgery.

Forensic examination was decided under the suspicion of acute myocardial infarction caused by thrombotic occlusion of the RCA associated with iatrogenic dissection during coronary intervention, and PMCTA was performed before standard autopsy dissection. Following the standardized protocol, cannulation of the right femoral vessels was performed using two cannulas with a diameter of 16-Fr for

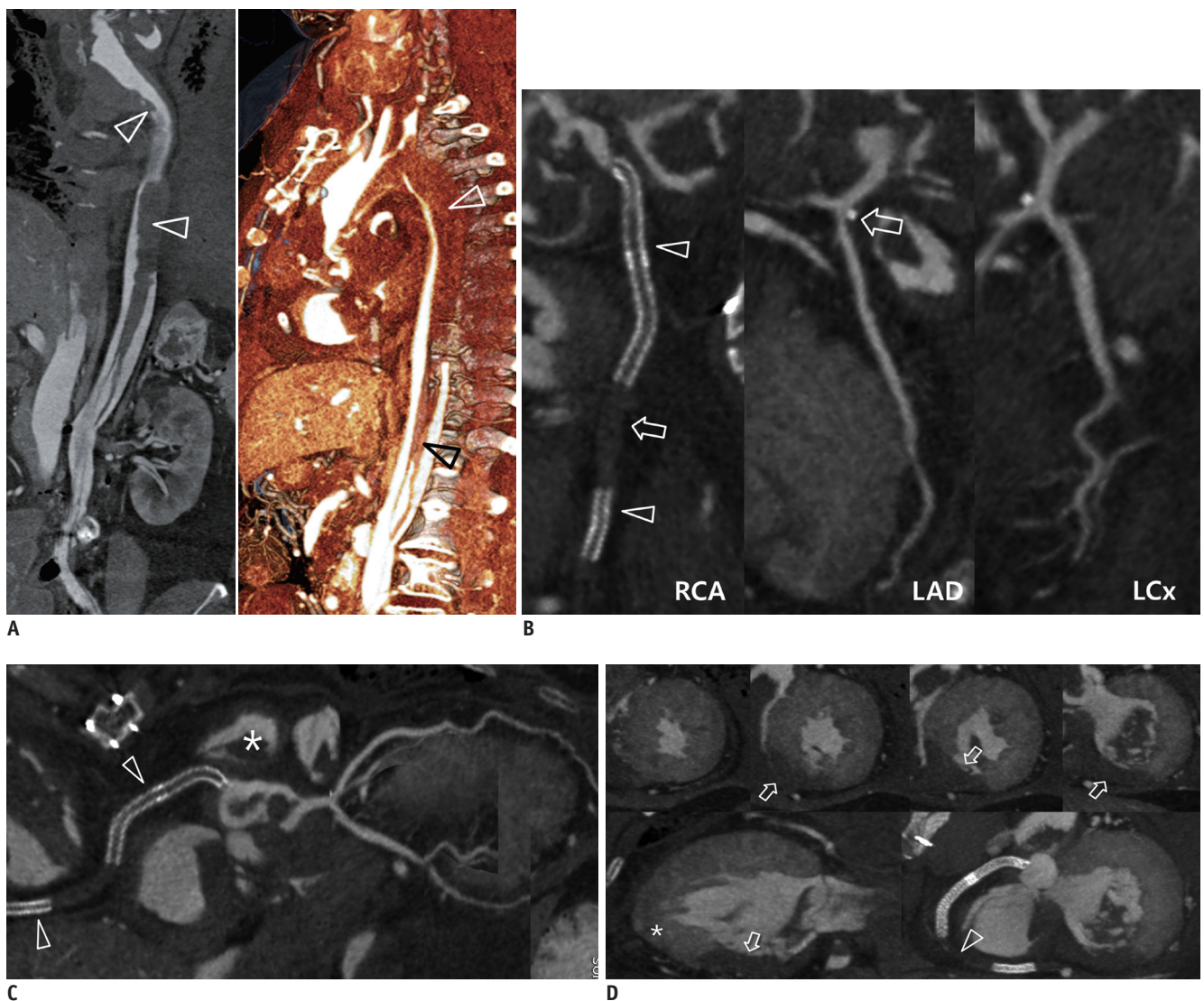


Fig. 1. Post-mortem CT angiography and pathologic specimen of 59-year-old female who died of suspected myocardial infarction. **A.** Curved multiplanar reformation (MPR, left) and three-dimensional volume rendering image (right) demonstrate aortic dissection with intimo-medial flap and thrombosed false lumen (arrowheads) from aortic arch extending to left common iliac artery. Autopsy confirmed ascending aortic replacement due to previous type A aortic dissection. **B, C.** Curved MPR (**B**) and medial axial reformat (**C**) images of coronary tree show total thrombotic occlusion of stents (arrowheads) in right coronary artery (RCA) and RCA itself (arrow), and also depict focal mixed plaque (arrow) in proximal left anterior descending artery (LAD) and normal left circumflex artery (LCx). Note postmortem clots (*) in pulmonary artery. **D.** Multiple short-axis views of left ventricle (LV) show localized transmural perfusion defects (arrows) in inferior and inferoseptal walls of mid and basal LV, sharply demarcated from normal myocardium showing diffuse contrast enhancement (upper). Perfusion defects correspond to thrombosed RCA (arrowhead) territory with apical sparing (*) clearly depicted on two-chamber view (lower column).

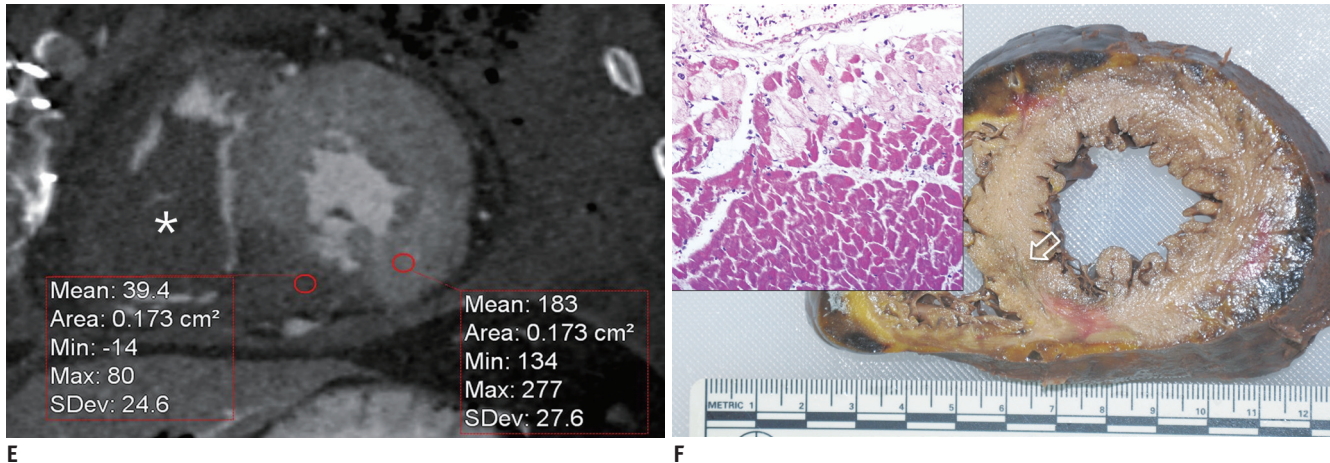


Fig. 1. Post-mortem CT angiography and pathologic specimen of 59-year-old female who died of suspected myocardial infarction.
E. Density measurements in short axis image demonstrate considerable contrast enhancement in normal myocardium, that can be attributed to compact filling of oily contrast agent in micro-capillary system through patent LAD and LCx coronary arteries. Note huge postmortem clot (*) in right ventricle. **F.** Gross and microscopic specimens of heart. Cut surface shows subtle mottling with yellow-tan softening in damaged inferoseptal wall (arrow), which has typical microscopic changes of coagulation necrosis with focal interstitial infiltrate of neutrophils, indicative of acute myocardial infarction (left upper). LAD = left anterior descending artery, LCx = left circumflex artery

the artery and 18-Fr for the vein. PMCTA was then carried out on a 128-slice multidetector CT system (Somatom AS plus, Siemens Healthcare, Erlangen, Germany). Scanning was conducted from the vertex to the upper thigh with tube voltage of 120 kVp and tube current of 210 mAs using a 0.625 mm collimation. A pressure-controlled perfusion device (Virtangio, Fumedica AG, Maquet, Muri, Switzerland) was used to inject 1200 mL of a mixture of esters of polyiodinated fatty acid (Angiofil, Fumedica AG, Muri, Switzerland) and paraffin oil at the injection rate of 800 mL/min (5).

Post-mortem CT angiography at the arterial phase (a retrograde injection of the contrast mixture into the arterial system through the femoral artery) demonstrated an aortic dissection with a contrast-filled true lumen and thrombosed false lumen (Fig. 1A). Furthermore, multiplanar reconstructed (MPR) images of the coronary tree showed a normal contrast-filled lumen of the left anterior descending and circumflex artery with their smaller branches. However, MPR images of the RCA clearly demonstrated complete thrombosis of os to distal segment, and also depicted complete occlusion of both stents implanted in proximal and distal segment, respectively (Fig. 1B, C). In addition, the short-axis view of the heart showed diffuse contrast enhancement of the left ventricular (LV) myocardium; interestingly, it also depicted a transmural contrast defect localized in the inferior and inferoseptal myocardium of the middle and basal LV that corresponds to the RCA territory (Fig. 1D, E).

Gross pathological examination of the coronary arteries opened longitudinally revealed complete thrombotic occlusion of stents implanted in the false lumen of the proximal and middle segments, causing collapse of the true lumen of the involved segments and thrombosis of the remaining true lumen of the distal segment. On visual inspection of the heart, areas of damage were revealed as subtle mottling with a yellow-tan center. Using light microscopy of sections stained by routine hematoxylin and eosin stains, coagulation necrosis with the loss of nuclei and striations was identified at infarcted area (Fig. 1F).

DISCUSSION

Recently, PMCT has increasingly been used in forensic practice and has proved to be useful as a complimentary tool to guide the following autopsy in selected cases (2, 3). However, to overcome the intrinsic weakness of PMCT; i.e., the very limited information on the vascular structures, intravascular contrast infusion has been introduced for the detailed pre-autopsy CT evaluation of vascular structures, particularly in cardiovascular deaths. Several contrast materials have been developed for the complete filling of vascular system, including corpuscular particles, oily liquids, water-soluble preparations, and casts (4, 7, 8). It has been reported that PMCT complements autopsy, and the incorporation of PMCTA into autopsy practice enhances the diagnostic accuracy of the autopsy report (4, 6). In recent years, a new lipophilic agent, a mixture of Angiofil

and paraffin oil has been introduced and has proved to be effective in the filling of the vasculature with micrometer resolution to the pixel size of 15 μm (10). The advantage of these liquid oils would be that they are retained in the vascular lumen for a considerably longer period without extravasation or penetration into the surrounding tissue during CT (9, 10).

In this case, PMCTA was performed using this lipophilic agent and demonstrated complete thrombotic occlusion of the RCA, which was later assessed by making longitudinal cuts externally across the course of coronary vessels and directly visualizing the lumen during autopsy. Furthermore, as in clinical practice, various image reconstructions, including MPRs, have enabled detailed and objective assessment of the vessel course, plaque composition, and degree of luminal stenosis in this case. Similar to clinical coronary angiography, however, PMCTA is a morphological imaging tool; thus, it cannot define the hemodynamic significance of identified coronary stenosis. Therefore, the demonstration of coronary stenosis alone on PMCTA cannot prove CAD as the cause of death "beyond a reasonable doubt". This is the same background as with autopsy findings. In fact, in the case of sudden cardiac death from coronary occlusion, myocardial infarct cannot be identified as a cause of death with absolute certainty even at autopsy if patients died of immediate causes such as arrhythmia. In such cases, the cause of death is attributed to CAD based on "the balance of probability" if no other lethal pathology is demonstrated (6, 9). In our case, however, a regional defect of myocardial enhancement was also noted, reflecting a non-perfused myocardium with blockade of contrast into myocardial micro-capillaries by coronary occlusion. This may be helpful in identifying culprits in multi-vessel CAD. In contrast to our case, more importantly, we believe this perfusion defect, if it corresponds to a coronary territory, may have an additional benefit for more confident diagnosis of myocardial ischemia associated with CAD as the cause of death, particularly in the case of grossly or pathologically normal-looking myocardium in sudden cardiac death.

There have been several reports describing the potential roles of PMCTA performed with various types of contrast agents and injection techniques. In those cases undergoing angiography with water soluble contrast medium, Roberts et al. (7) reported a "myocardial blush" in some cases, reflecting a normal capillary leakage of water-soluble agent into the myocardial interstitium. Additionally, Morgan et al. (9) noted coronary plaques with localized absence of

blush (perfusion deficit) in the corresponding myocardium. Conversely, Michaud et al. (6), using a lipid-soluble agent not expected to leak from the vessels, described localized enhancement of myocardium probably due to an abnormal leak in the infarcted myocardium, that correlated with the area of infarction. However, no report has described a myocardial contrast defect associated with coronary occlusion using oily contrast as in this case. Although we believe the localized perfusion defect in our case is attributable to a relatively high degree of RCA stenosis, and it was accentuated by "normal blush" of the neighboring myocardium with intensive contrast filling in micro-capillary system through patent coronary arteries (10), we acknowledge that further studies with more cases are needed to evaluate the factors that may potentially influence the myocardial enhancement pattern, including the degree of coronary stenosis, micro-vascular integrity, and infarct age.

We believe this case illustrates the potential benefit of PMCTA for the demonstration of the hemodynamic significance of identified coronary stenosis, providing additional information on the perfusion status of the corresponding myocardium.

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