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ORIGINAL ARTICLE

Differentiation of hemopericardium due to ruptured myocardial infarction or aortic dissection on unenhanced postmortem computed tomography

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Abstract The aim of the study was to evaluate unenhanced postmortem computed tomography (PMCT) in cases of non-traumatic hemopericardium by establishing the sensitivity, specificity and accuracy of diagnostic criteria for the differentiation between aortic dissection and myocardial wall rupture due to infarction. Twenty six cases were identified as suitable for evaluation, of which ruptured aortic dissection could be identified as the underlying cause of hemopericardium in 50% of the cases, and myocardial wall rupture also in 50% of the cases. All cases underwent a PMCT and 24 of the cases also underwent one or more additional examinations: a subsequent autopsy, or a postmortem magnetic resonance (PMMR), or a PMCT angiography (PMCTA), or combinations of the above. Two radiologists evaluated the PMCT images and classified each case as “aortic dissection”, “myocardial wall rupture” or “undetermined”. Quantification of the pericardial blood was carried out using segmentation techniques. 17 of 26 cases were correctly identified, either as aortic dissections or myocardial ruptures, by both readers. 7 of 13 myocardial wall ruptures were identified by both readers, whereas both readers identified correctly 10 of 13 aortic dissection cases. Taking into account the responses of both readers, specificity was 100% for both causes of hemopericardium and sensitivity as well as accuracy was higher for aortic dissections than myocardial wall ruptures (72.7% and 87.5% vs 53.8% and 75% respectively). Pericardial blood volumes were constantly higher in the aortic dissection group, but a statistical significance of these differences could not be proven, since the small count of cases did not allow for statistical tests. This study showed that diagnostic criteria for the differentiation between ruptured aortic dissection and myocardial wall rupture due to infarction are highly specific and accurate.

Keywords Virtopsy · Forensic radiology · Hemopericardium · Postmortem computed tomography (PMCT) · Aortic dissection · Myocardial infarction

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Introduction

Pericardial tamponade is a cause of death that is encountered in forensic institutes because it leads to sudden and possibly unexpected death. As such, it is investigated by forensic pathologists to exclude an unnatural mode of death.

Postmortem computed tomography (PMCT) has found its established place in postmortem forensic examinations [1]. There are institutes where PMCT is being used as a triage tool to exclude cases with positive evidence of natural death from subsequent, more extensive medicolegal investigations [2]. Although it is currently not considered a substitute for medicolegal autopsy, there are undoubtedly cases where forensic autopsy will only confirm the PMCT findings without adding case-relevant information [3]. Moreover, when considering minimally invasive histological and toxicological sampling [4, 5], PMCT gains in potentially depicting what can only be considered natural causes of death [6].

Alternative possibilities for differentiating between the hemopericardium due to myocardial wall rupture and ruptured aortic dissection would be PMCT angiography (PMCTA) and/or postmortem magnetic resonance imaging (PMMR). Although PMCTA can provide a definite diagnosis regarding the cause of a pericardial tamponade and depict the exact localization of blood extravasation, fewer forensic institutes have access to PMCTA than to unenhanced PMCT due to the latter's cost. A very low-cost alternative for PMCTA has been recently proposed by Schweitzer et al. [7]. Even fewer forensic institutes have dedicated -or even access to- magnetic resonance equipment. This is why the ability to diagnose the cause of pericardial tamponade correctly on unenhanced PMCT is essential.

Pericardial tamponade can have various natural death-related pathological conditions as etiologies, such as post-infarction rupture of the myocardial wall, aortic rupture secondary to an aneurysm, aortic dissection with subsequent rupture of the adventitia, rupture of the myocardial wall in the course of myocarditis, or neoplastic diseases (primary or metastatic) [8]. Pericardial blood extravasation of traumatic origin may also be encountered after gunshots, medical intervention due to catheters or scalpel action, or blunt or sharp force injury to the chest [9–12]. The presence of pericardial effusion, particularly hemopericardium, is a finding that is difficult to miss on PMCT. In the postmortem setting, hemopericardium can be present with or without sedimentation [13] and with or without forming a so-called "target" or "armored heart" sign [14]. According to Watanabe et al., hemopericardium can appear either as double band ("armored heart" or "target sign"), single band or with a horizontal level [14]. Filograna et al. described the conditions that should be fulfilled in cases of hemopericardium for diagnosing a fatal cardiac tamponade [15]. Although trauma and its results are easily recognized on PMCT, it is unknown whether the various atraumatic causes of the hemopericardium can be differentiated on unenhanced PMCT.

Differentiation between aortic dissection and myocardial wall rupture in cases of hemopericardium can be important because it could not only increase the accuracy in death statistics and subsequently lead to better health strategies [16] but also facilitate familial counseling and the eventual detection of preventable fatalities [17].

Differentiating between ruptured aortic dissection and post-infarction myocardial wall rupture in cases of hemopericardium on unenhanced PMCT can be challenging. The characteristic aortic dissection signs on unenhanced PMCT were recently described in the literature [18]; these can be absent, impeding correct diagnosis. On the other hand, the current consensus is that recent myocardial infarction is not directly depicted on unenhanced PMCT [19, 20]. However, hemorrhagic infiltration of the myocardial tissue is expected in cases of myocardial wall rupture. We hypothesize that this hemorrhagic infiltration would be indicated by inhomogeneities of the myocardium (primarily hyperdensities due to extravasated blood), thus posing an indirect sign of a myocardial wall rupture. Accordingly, hyperdensities of the adjacent epicardial adipose tissue could also be an indirect sign of extravasated blood permeating a ruptured myocardial free wall.

Empirically, greater hemopericardial volumes were encountered in cases of ruptured aortic dissections than in myocardial wall ruptures. Therefore, another goal of the present study was to evaluate a hypothetically different volume of the pericardial effusion in cases of aortic dissection and myocardial rupture.

We hypothesize that the differential diagnosis for hemopericardium could be based on signs of aortic dissection or on indirect findings, such as the hemorrhagic infiltration of the myocardial wall in cases of infarction with subsequent rupture, the infiltration of the epicardial adipose tissue in cases of myocardial wall rupture and the volume of blood within the pericardial sack.

The aim of the study was to evaluate unenhanced PMCT in cases of assumedly non-traumatic hemopericardium by establishing the sensitivity, specificity and accuracy of diagnostic criteria for the differentiation between aortic dissection and myocardial wall rupture due to infarction.

Materials and methods

The institutional database was retrospectively searched for cases of hemopericardium. Cases with PMCT imaging and a definite diagnosis of the cause of hemopericardium (ruptured myocardial wall or aortic dissection) were included. The definite diagnosis succeeded for most of the cases with one or more additional examinations: a subsequent autopsy, or a PMMR, or a PMCTA, or combinations of the above. The presence of thoracic trauma, blunt or sharp force injury (except for cardiopulmonary resuscitation efforts related injuries), cardiac medical devices and catheters, and advanced decomposition [21] led to the exclusion of cases from the study group. The presence of hemothorax was also an exclusion criterion because a causal relation to the hemopericardium and, consequently, an alteration of the initial volume of the pericardial blood could not be ruled out. Since comparing the pericardial blood volumes between the cases was a research question, the exclusion of these cases was inevitable.

Study sample

Twenty-six ($n = 26$) cases were identified as suitable for evaluation (19 males, mean age 59.2 years, age range 32–88 years, and 7 females, mean age 74.6 years, age range 59–96 years). Aortic dissection and intrapericardial aortic rupture could be identified as the underlying cause of hemopericardium in 50% (13/26) of the cases, and myocardial rupture was recognized as a post-infarction complication in 50% (13/26). All cases underwent a whole-body PMCT examination. Twenty-one cases were autopsied, 8 of which underwent additional imaging (PMMR = 4, PMCTA = 4) prior to autopsy. The remaining cases underwent the following imaging modalities: 2 cases had only a PMMR examination, and 1 case had a PMMR examination followed by a PMCTA. Two cases did not receive any further exam because the diagnosis was definite by unenhanced PMCT [18]. The final forensic reports were evaluated for all cases. Resuscitation efforts were noted for all cases.

Imaging parameters

Postmortem imaging was carried out in the supine position, approximately 2–81 h postmortem. Imaging was performed on a 128-slice CT scanner (SOMATOM Definition Flash, Siemens Healthineers, Erlangen, Germany). Imaging parameters were set as follows [22]: tube voltage 120 kVp, slice collimation 128 × 0.6 mm. All scans were performed using the Siemens CARE Dose 4D (CARE dose 4D, Siemens Healthineers, Erlangen, Germany) automatic dose modulation software. PMCT image reconstructions of the thorax and abdomen were performed with a slice thickness of 1.0 mm and an increment of 0.6 mm using the soft-tissue and lung window with a soft and hard kernel, respectively.

PMCTA was performed by injecting iodinated contrast medium solution in the arterial and venous vessels, followed by the scanning protocol of Flach et al. [22].

PMMR imaging was performed using a 3.0-T MR unit (Achieva 3.0 T TX, Philips, Best, The Netherlands) after each case was scanned by unenhanced PMCT. Each evaluated case underwent a standard cardiac protocol using a 16-element phased-array coil that included a 4-chamber view (T2w) and several short axis views (T1w, T2w, fat saturated T2w steady-state-inversion recovery (STIR), PDw (proton density weighted) and T2*w sequences), all of which had 3 mm slice thicknesses. Additionally, axial sequences of the entire chest were obtained (T1w, fat saturated T2w using spectral attenuated inversion recovery (SPAIR)) with a slice thickness of 5 mm and an axial T2w sequence aligned to the pulmonary arteries with a slice thickness of 1.5 mm [23].

For radiological assessment, a multi-modality workstation was used (Syngo.via, Version VB10A, Siemens Healthcare GmbH, Erlangen, Germany).

Autopsy

Autopsies were performed by two forensic pathologists, at least one of whom was board certified, with the assistance of an autopsy technician. The cranial, thoracic, and abdominal cavities were opened and organs were dissected. Histological and toxicological samples were preserved for further examination in all cases.

Retrospective PMCT reading

Two radiologists with significant experience in forensic imaging (seven and eight years, respectively) reviewed the cases independently and were blinded both to the cause of hemopericardium and to each other. They classified each case as “aortic dissection”, “myocardial wall rupture” or “undetermined”. The findings and signs on which they relied to establish the diagnosis were recorded. Cases with signs of aortic dissections, including a medially dislocated calcification, an intimal flap or a double sedimentation (all described as characteristic PMCT signs of aortic dissections [18]), were classified as aortic dissections. If none of the above-described signs were depicted, heterogeneities of the myocardial wall and hyperdensities of the epicardial adipose tissue (Figs. 1 and 2) were assessed. These cases were classified as myocardial wall rupture.

Regarding the PMCT appearance of the hemopericardium, the presence of the “target” sign or “armored heart” sign, as described in the literature, and the presence of sedimentation lines [14] were also noted.

Segmentation of the pericardial blood

Quantification of the blood was carried out by a computer scientist with a background in biomedical engineering using segmentation techniques. For this, Amira® (Version 5.4.1, Visualization Sciences Group, Bordeaux, France, and Zuse Institute Berlin, Berlin, Germany) was used. Prior to segmentation, the dataset was resampled to a slice thickness of 4 mm, thus reducing the time required for the segmentation of each dataset without sacrificing too much accuracy. Segmentation was performed manually using the masking functionality of Amira. The investigator performing the segmentation was blinded to the details of the study.

The accuracy of the virtually estimated pericardial blood volume was tested in a previous study [24].

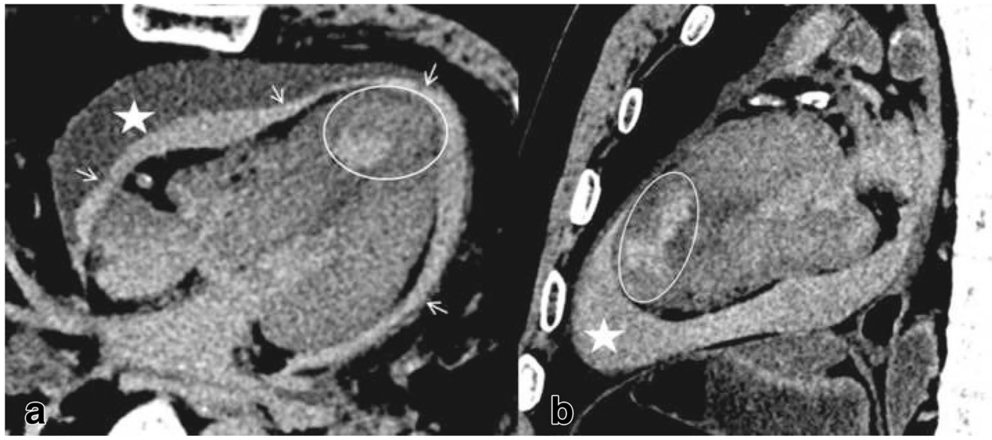

Fig. 1 Inhomogeneity of the myocardium apically (*white circles*). Note the pericardial effusion (*asterisk*) with a hyperdense inner ring (*arrows*), forming the so called "target sign".

Statistical evaluation

To allow calculations of sensitivity, specificity and accuracy for each actual pathology, negatives (false and true) were all cases with incorrect or undetermined diagnosis. For example, by calculating the sensitivity, specificity and accuracy for myocardial wall rupture, true negatives were those cases that the reader characterized as "aortic dissection" or "undetermined", and they were truly no myocardial wall rupture cases. False negatives were considered those cases which the reader characterized as "aortic dissection" or "undetermined", and they were indeed myocardial wall ruptures. Moreover, for the sensitivity, specificity and accuracy calculations, the two cases without any additional examination (autopsy, PMMR or PMCTA) were excluded (Table 1). Statistical tests were performed with SPSS (IBM SPSS Statistics for Windows, IBM Corp., Version 23.0.0.2, Armonk, N.Y., USA). Categorical variables were described as frequencies and percentages, whereas continuous variables were described as the means and standard deviations (SD) and/or ranges. To assess normality, the Shapiro-Wilk test was used. The Mann Whitney-U test was used for comparisons between independent samples. The Kappa value was used to evaluate agreement between the readers. Results with a p value less than 0.05 were considered statistically significant.

Results

The target sign was present in 24 cases, 11 of which also presented sedimentation. In the remaining 2 cases, sedimentation but no target sign was noted.

In total, 17 of the 26 cases were correctly identified as either aortic dissections or myocardial ruptures by both readers. This indicates a moderate agreement (Kappa value 0.465) between readers 1 and 2. Moreover, there was no case in which both readers were wrong. Table 1 provides an overview of the results for each of the readers and for both readers for the myocardial wall ruptures and the ruptured aortic dissection cases.

Ruptured myocardial wall

Each reader independently identified correctly 10/13 ruptured myocardial wall cases. A total of 7/13 of the myocardial wall ruptures were identified by both readers. Only 1 case was misinterpreted by Reader 1 as a myocardial wall rupture. This is interpreted by a specificity of 90.9% for diagnosing myocardial wall rupture for Reader 1 and 100% for Reader 2 (Table 1). However, the sensitivity of both readers (76.9%) in depicting a myocardial wall rupture was lower than the

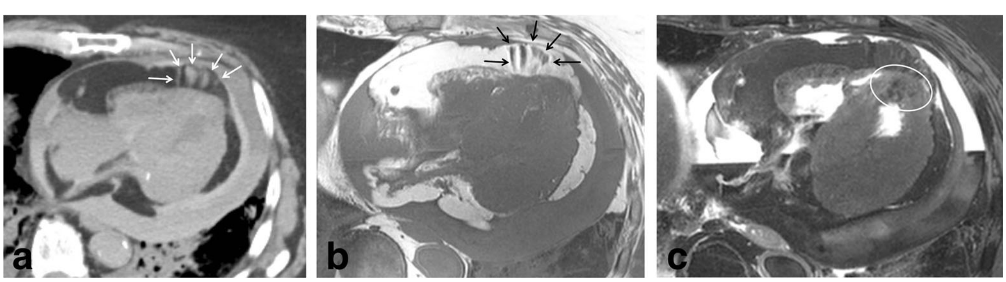

Fig. 2 **a** Inhomogeneities (hyperdensity) of the epicardial adipose tissue on PMCT (*white arrows*, note the significant quantity of epicardial adipose tissue, which allows for the identification of the hyperdense area apically), and on **b** the T1 sequence on PMMR (*black arrows*). **c** The T2 PMMR sequence confirms the presence of a myocardial wall rupture (*encircled*) adjacent to the epicardial fat finding

Table 1 Sensitivity, specificity, and accuracy for each and both readers in diagnosing myocardium wall rupture and aortic dissection. For these calculations, we excluded the two aortic dissection cases that did not have a subsequent examination after PMCT

		Myocardium wall rupture			Aortic dissection	
		yes	no		yes	no
Reader 1	Yes	10 (TP)	1 (FP)	Yes	9 (TP)	0 (FP)
	No (Aorta and undetermined)	3 (FN)	10 (TN)	No (Myocardium and undetermined)	2 (FN)	13 (TN)
	Sens: 76.9% Specif: 90.9% Accuracy: 83.3%			Sens: 81.8% Specif: 100% Accuracy: 91.7%		
Reader 2	Yes	10 (TP)	0 (FP)	Yes	10 (TP)	2 (FP)
	No (Aorta and undetermined)	3 (FN)	11 (TN)	No (Myocardium and undetermined)	1 (FN)	11 (TN)
	Sens: 76.9% Specif: 100% Accuracy: 87.5%			Sens: 90.9% Specif: 84.6% Accuracy: 87.5%		
Both Readers	Yes	7 (TP)	0 (FP)	Yes	8 (TP)	0 (FP)
	No (undetermined*)	6 (FN)	11 (TN)	No (undetermined*)	3 (FN)	13 (TN)
	Sens: 53.8% Specif: 100% Accuracy: 75%			Sens: 72.7% Specif: 100% Accuracy: 87.5%		

*No case was incorrectly identified by both readers. If one reader identified the case correctly and the other did not, the case was classified as undetermined

specificity. Both readers together had an accuracy of 75% in diagnosing myocardial wall ruptures.

Ruptured aortic dissection

Reader 1 correctly diagnosed 9/11 aortic dissections, and Reader 2 correctly diagnosed 10/11; 8 of the aortic dissections were recognized by both readers. The sensitivity of the readers in depicting aortic dissections was 81.8% (Reader 1), 90.9% (Reader 2) and 72.7% (both readers). The specificity for Reader 1 and for both readers was 100%, whereas Reader 2 falsely diagnosed an aortic dissection in 2 cases and thus had a specificity of 84.6%. Both readers had an accuracy of 87.5% in diagnosing aortic dissections.

Pericardial blood volume

The mean estimated pericardial blood volume was 415 ml (SD 191 ml, range 135–1002 ml). Males (mean 466 ml, SD 191 ml) had larger pericardial blood volumes than females did (mean 278 ml, SD 112 ml). This difference proved to be statistically significant ($p = 0.006$).

Myocardial wall rupture was the cause of hemopericardium in 11/19 males (mean blood volume 408 ml, SD 86 ml) and aortic dissection in 8/19 males (mean blood volume 544 ml, SD 266 ml). Resuscitation efforts were documented in 12 of the males (mean blood volume 464 ml, SD 158 ml) but not in 7 (mean 468 ml, SD 252 ml), and there was no statistically significant difference between these groups. Among the 7 males without resuscitation efforts, 4 were caused by myocardial rupture (mean 415 ml, SD 105 ml) and 3 by aortic dissection (mean 539 ml, SD 401 ml).

Among the 7 females, myocardial wall rupture was the cause of hemopericardium in 2/7 (mean blood volume 239 ml, SD 37 ml) and aortic dissection in 5/7 (mean blood volume 294 ml, SD 132 ml). Resuscitation efforts were documented for 2 (mean blood volume 213 ml, SD 0.7 ml) and none for 5 (mean 305 ml, SD 125 ml). The limited number of cases did not allow a statistical evaluation of the differences.

Discussion

This study shows that if the responses of both readers were considered, the specificity was 100%, and both the sensitivity and accuracy were higher for aortic dissections than myocardial wall ruptures (72.7% and 87.5% vs 53.8% and 75%, respectively). Pericardial blood volumes were constantly higher in the aortic dissection group, but the statistical significance of these differences could not be proven because the small count of cases did not allow statistical tests.

If both readers agreed, the diagnosis was always correct. This was the case in approximately 2/3 of the cases (65.4%, 17/26 cases, or rather 62.5%, 15/24 cases, if the 2 cases without an examination in addition to PMCT were excluded) with pericardial tamponade. It was interesting to note that there was a very high specificity of 90.9% and 100% (which actually means one and no false positives, respectively) for both readers regarding the diagnosis of myocardial wall rupture. This finding can be partly explained by the study methodology: radiologists were instructed to only search for signs of myocardial wall rupture if they could not find any aortic dissection signs. In other words, myocardial wall and epicardial adipose tissue heterogeneities were only looked for in the absence of aortic dissection, thus limiting the number of false positives. Our decision to do so was based on the existence of established criteria for aortic dissection in forensic radiology [18], and therefore the diagnosis of aortic dissection is more justified.

It is noteworthy that signs of aortic dissection can be seen in some cases more distally than the intrapericardial part of the aorta. It is therefore recommended to always critically read the images for signs of dissection of the aorta descendens.

Both cases which underwent only a PMMR as subsequent examination were myocardial rupture cases. The clinical radiology literature is sparse regarding this finding, as it usually leads to death in a short time. Kuroiwa et al. described a case of cardiac rupture after acute myocardial infarction in which myocardial thinning, rupture and epicardial hemorrhage could be diagnosed on PMMR [25].

We noticed that epicardial adipose tissue heterogeneities were only visible if there was an increased quantity of epicardial fat above the myocardial wall rupture site. Furthermore, we could verify that the gas accumulations in the myocardium or the epicardial adipose tissue should not be interpreted as heterogeneities of the tissues and are not a sign of myocardial wall rupture, but rather resuscitation related changes [26].

Males generally had significantly larger volumes of pericardial blood than females did in cases of hemopericardium. Although aortic dissection cases seem to exhibit larger blood volumes in both males and females, the differences could not be tested statistically because of the limited number of cases in each group. The comparison of the blood volumes of males with and without resuscitation efforts did not reveal any statistically significant difference. This result leads us to conclude that resuscitation efforts should not significantly alter the pericardial blood volume. Further research on a larger scale is certainly needed because the number of cases in the present study was limited.

Clearly, there are several limitations of this study that deserve to be addressed. First, no other natural causes of hemopericardium (e.g. myocarditis, aortic aneurysm) were examined because they represent a minority of the cases in a forensic institute and because such cases were not encountered during the database searches. Second, the gold standard for the definite diagnosis of the cause of pericardial tamponade was not only autopsy, which is considered the gold standard in determining the cause of death, but also PMMR and PMCTA. There were also 2 cases that did not have a subsequent examination but did fulfil the criteria of both medially dislocated calcification and a visible intima flap. These 2 cases were also included and were diagnosed correctly by both readers.

We were not able to statistically test the hypothesis that the volume of blood in the pericardium would depend on the pathology that caused it. The small number of cases in each subcategory (male/female, with/without resuscitation efforts) did not allow us to perform statistical tests.

Conclusion

This study showed that diagnostic criteria for the differentiation between ruptured aortic dissection and myocardial wall rupture due to infarction are highly specific and accurate. Even on unenhanced PMCT, the underlying cause of the atraumatic hemopericardium could be established in the majority of cases. This resulted in an improved diagnostic security for atraumatic hemopericardium on unenhanced PMCT.

Key points

1. The aim of the study was to evaluate unenhanced PMCT in cases of hemopericardium by calculating the sensitivity, specificity and accuracy for the differentiation between aortic dissection and myocardial wall rupture.
2. Medially dislocated calcification, intimal flap or double sedimentation were signs of aortic dissections whereas inhomogeneities of the myocardial wall and the epicardial adipose tissue were encountered in myocardial wall rupture cases.
3. 17/26 of the cases (7/13 myocardial wall ruptures and 10/13 ruptured aortic dissections) were correctly identified by both readers.
4. Taking into account the responses of both readers, specificity was 100% for both causes of hemopericardium and sensitivity as well as accuracy was higher for aortic dissections than myocardial wall ruptures.
5. Differentiation between myocardial wall rupture and aortic dissection as causes of hemopericardium is possible on unenhanced PMCT images in the majority of the cases.

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Compliance with ethical standards

Conflicts of interest The authors declare there is no conflict of interest.

Ethical approval Ethical approval was obtained by the Cantonal Ethics Committee of Zurich, Nr. 90–2015.

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