

Smaller but denser: postmortem changes alter the CT characteristics of subdural hematomas

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Abstract

Purpose The aim of this study was to investigate if (1) the volume of subdural hematomas (SDH), midline shift, and CT density of subdural hematomas are altered by postmortem changes and (2) if these changes are dependent on the postmortem interval (PMI).

Materials and Methods Ante mortem computed tomography (AMCT) of the head was compared to corresponding postmortem CT (PMCT) in 19 adults with SDH. SDH volume, midline shift, and hematoma density were measured on both AMCT and PMCT and their differences assessed using Wilcoxon-Signed Rank Test. Spearman's Rho Test was used to assess significant correlations between the PMI and the alterations of SDH volume, midline shift, and hematoma density.

Results Mean time between last AMCT and PMCT was 109 h, mean PMI was 35 h. On PMCT mean midline displacement was decreased by 57 % ($p < 0.001$); mean SDH volume was decreased by 38 % ($p < 0.001$); and mean hematoma density was increased by 18 % ($p < 0.001$) in comparison to AMCT. There was no correlation between

the PMI and the normalization of the midline shift ($p = 0.706$), the reduction of SDH volume ($p = 0.366$), or the increase of hematoma density ($p = 0.140$).

Conclusions This study reveals that normal postmortem changes significantly affect the extent and imaging characteristics of subdural hematoma and may therefore affect the interpretation of these findings on PMCT. Radiologists and forensic pathologists who use PMCT must be aware of these phenomena in order to correctly interpret PMCT findings in cases of subdural hemorrhages.

Keywords Forensic radiology · Postmortem computed tomography · Head CT · Subdural hematoma · Virtopsy

Introduction

Over the past 15 years, postmortem computed tomography (PMCT) has become an important tool in forensic death investigations [1–3]. PMCT images differ from clinical, ante mortem CT (AMCT) images, and feature a number of normal postmortem findings which must be distinguished from true pathology such as posterior sedimentation, gas formation, or tissue decomposition [4, 5]. In the brain, these findings also include an overall decreased attenuation of the brain, loss of the gray–white junction, and effacement of sulci and gyri [6].

Today, radiologists and forensic pathologists involved in postmortem imaging are generally aware of such normal postmortem findings. However, the effect of postmortem changes on *pathologic* findings is poorly investigated. In our practice, we noted a striking difference between AMCT and PMCT of the head after cranio-cerebral injury. Key findings, such as subdural hematomas (SDH) and midline shift seem to decrease after death and appear to be smaller

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on PMCT than on clinical AMCT images of the same cases. Our observation was supported by a recent case report which describes a decrease of both SDH volume and midline shift from clinical CT to PMCT in two cases [7]. However, both our case based observations and published case reports offer insufficient evidence to draw generalized conclusions regarding effect of postmortem changes on the appearance of SDH on PMCT.

The aim of this study was to investigate if (1) the volume of subdural hematomas (SDH), the displacement of the midline, and the density of subdural hematomas are altered by postmortem changes and (2) if these changes are dependent on the postmortem interval (PMI).

Materials and methods

Study population

The department of the public prosecutor approved the study. For this retrospective study we searched the institutional picture archive and communication system (PACS) for all cases where both clinical (ante mortem) and post-mortem CT datasets of the head were available. During the evaluated period from November 2011 to July 2013 a total 91 potentially suitable cases were identified. Of these 91 cases, all cases featuring SDH and midline shift on AMCT were included in the study population ($n = 23$). The other 67 cases featured no SDH. Of the 23 cases with SDH, four cases were excluded from the final study population. Exclusion criteria were open skull fracture ($n = 1$), medical interventions between AMCT and PMCT ($n = 2$), and metal artifacts ($n = 1$). The final study population included 19 subjects (14 male, 5 female; mean age 67 years (range 23–92 years)). All subject enrolled in this study had been hospitalized after the occurrence of the SDH and had died in hospital care. Investigators were blinded to medical history and individual case circumstances for this evaluation.

CT data acquisition and image reconstruction

AMCTs of the head had been performed on several different CT scanners by different manufacturers, using different scanning protocols, each according to the individual hospital's protocols. All PMCTs (except two) had been performed on a 128-slice Somatom Definition Flash Dual Source CT scanner (Siemens Healthcare, Forchheim, Germany). Scan parameters were as follows: 120 kVp; automatic dose modulation (CARE Dose4D, Siemens Healthcare, Forchheim, Germany); 0.6 mm collimation. Two PMCTs were performed on a Somatom Sensation Open CT scanner (Siemens Healthcare, Forchheim, Germany) during maintenance service of the other scanner. Scan parameters were as follows: 120 kVp;

300 mAs; 1.2 mm collimation, using a dedicated head field of view. PMCT image reconstruction was performed with a slice thickness of 0.6 mm in increments of 0.4 mm using soft tissue and bone-weighted tissue kernel in all cases [8].

Data analysis

Midline shift and attenuation of the hematoma were evaluated using a PACS workstation (IDS7, Version 14.3.5.136, Sectra, Linköping, Sweden). Attenuation was measured by placing a caliper in the hematoma on both AMCT and PMCT. Each ROI was placed in the middle of the area of interest (i.e. the core of the hematoma) and covered more than one pixel as indicated by the literature (Fig. 1) [9]. In this study each ROI had a diameter of at least 6 mm to account for heterogeneous hematoma composition. Midline shift was measured in millimeters at the level of the maximal displacement of the septum pellucidum from the midline [10, 11] (Fig. 1). SDH volumes were quantified manually using dedicated segmentation software (Amira 5.4.1, Visage Imaging, Berlin, Germany), based on the protocol by Ampanozi et al. [12, 13] (Fig. 2). Note: if multiple AMCTs were available from one individual, the last AMCT before death was used for measurements of hematoma density, midline shift, and SDH volumetry. All measurements were performed by one radiologist (4 years of professional experience). All cases underwent subsequent medico-legal autopsy.

Statistical analysis

All statistical analyses were performed using the open-source statistics software SOFA (Statistics Open For All, Version 1.3.3, Paton-Simpson & Associates Ltd, Auckland, New Zealand) and SPSS (Version 17.0, IBM, Chicago, USA). Shapiro–Wilk Test was used to assess the normality of distribution. Wilcoxon–Signed Rank Test for non-parametric paired values was used to assess differences between ante mortem and postmortem attenuation of the hematoma (i.e. hematoma density), midline shift, and SDH volumes. The time intervals between the occurrence of the SDH and the last AMCT before death, between the last AMCT and death, between death and PMCT (i.e. PMI), and between AMCT and PMCT were documented. Spearman's Rho Test was used to assess significant correlations between the PMI and the alterations of midline shift, SDH volume, and hematoma density. A p value of <0.05 was defined as statistically significant.

Results

Mean time interval between trauma and the last AMCT before death was 10.7 h, mean time interval between AMCT and

Fig. 1 Comparison between subdural hematoma with concomitant midline shift on AMCT (a) and PMCT (b): CT-density of SDH increases from AMCT (76 HU) to PMCT (99 HU) while midline shift decreases from 14.2 to 7.0 mm

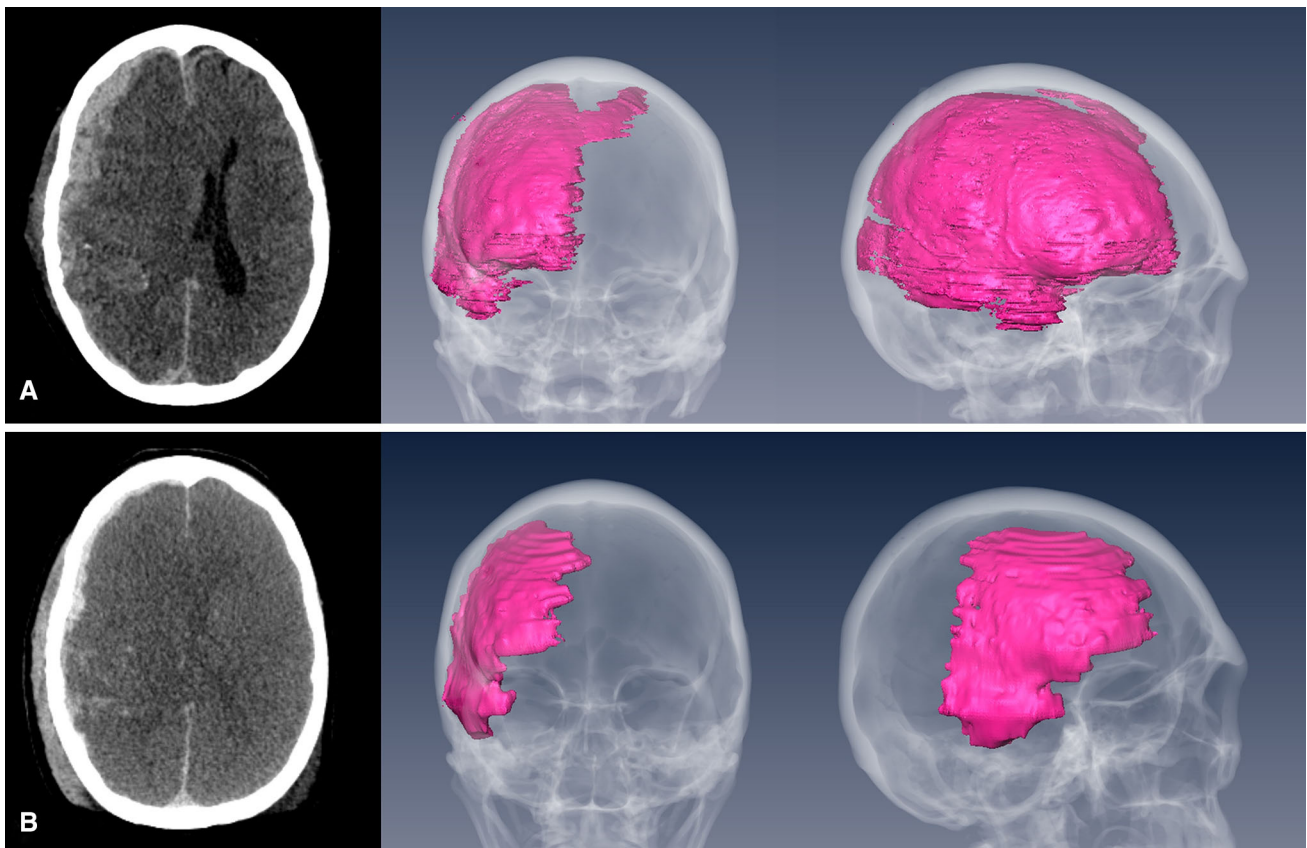
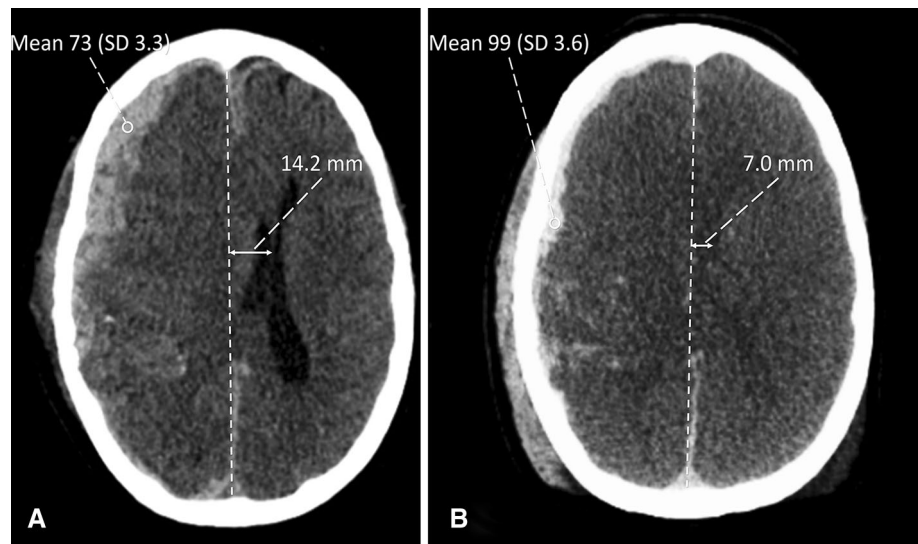


Fig. 2 Axial CT image and corresponding manual segmentation of a subdural hematoma on AMCT (a) and PMCT (b) (the same case as above): the volume of the subdural hematoma (colored purple on three-dimensional images) has decreased from 120 ml on AMCT to 20 ml on PMCT

death was 74.1 h, mean PMI (i.e. time between death and PMCT) was 35 h, mean time interval between last AMCT and PMCT was 109 h, and mean survival time was 3.5 days (Table 1). Mean midline displacement decreased significantly from AMCT to PMCT by 57 % (mean 4.1 mm; $p < 0.001$);

mean SDH volume decreased significantly from AMCT to PMCT by 38 % (mean 25.8 ml; $p < 0.001$); and mean hematoma density increased from AMCT to PMCT by 18 % (mean 12.8 HU; $p < 0.001$) (Table 2). There was no correlation between the PMI and the decrease of the midline shift

Table 1 Time intervals between events

Case	Trauma/AMCT (h)	AMCT/death (h)	AMCT/PMCT (h)	PMI (h)	Survival (days)
1	0.5	4	18	14	0.2
2	1.0	17	20	3	0.8
3	0.9	4	20	16	0.2
4	1.1	3	24	21	0.2
5	136.0	3	24	21	5.8
6	0.8	19	41	22	0.8
7	1.4	26	46	20	1.1
8	1.2	1	52	51	0.1
9	0.4	2	53	51	0.1
10	1.3	3	56	53	0.2
11	0.8	8	59	51	0.4
12	1.0	19	60	41	0.8
13	44.0	4	66	62	2.0
14	0.6	17	85	68	0.7
15	0.8	69	96	27	2.9
16	2.6	179	213	34	7.5
17	8.0	168	236	68	7.3
18	1.0	379	401	22	15.8
19	0.4	483	502	19	20.1
Mean	10.7	74.1	109.0	34.9	3.5
Median	1.0	17.0	57.5	30.5	0.8

Trauma/AMCT, time interval between trauma and AMCT; AMCT/death, time interval between AMCT and death; AMCT/PMCT, time interval between AMCT and PMCT; PMI, postmortem interval (i.e. time interval between death and PMCT; Survival, time interval between trauma, and death

($p = 0.706$), the reduction of SDH volume ($p = 0.366$), or the increase of hematoma density ($p = 0.140$).

Discussion

This study reveals that normal postmortem changes significantly affect the extent and imaging characteristics of subdural hematoma and may therefore affect the interpretation of these findings on PMCT.

In this study, both SDH volume and midline shift decreased after death by 38 and 57 % respectively. In all cases where midline shift was present on AMCT its extent had decreased on PMCT. The following step-by-step analysis of pathophysiologic and postmortem changes may explain these findings: unilateral cranio-cerebral injuries lead to a unilateral brain swelling and subsequent displacement of the midline to the non-injured hemisphere [10]. After death, failure of cellular sodium pump leads to a general increase of intracellular sodium, and consequentially, to an osmotic influx of water [14]. This postmortem intracellular fluid accumulation in both the injured and the non-injured hemisphere may cause a certain push-back of the midline towards the injured hemisphere, which may result in the apparent normalization of the midline.

In 17/19 cases, SDH volumes were smaller on PMCT than on AMCT. There are two principal theories to explain

the general decrease of SDH volume: (1) redistribution and compression of the hematoma by acute brain swelling and (2) degradation and dilution of the hematoma by cerebrospinal fluid [15–17]. It is our opinion that redistribution and compression are critical factors in cases with early death after SDH whereas degradation and dilution being the key to cases with prolonged survival after SDH. In two cases, SDH volumes were larger on PMCT than on AMCT. It is conceivable that in these two cases secondary bleeding occurred in the time between the last AMCT and death. This—clinically feared complication of SDH—would account for an increase in SDH volume from AMCT to PMCT.

The results of this study underline Inokuchi's initial observation that SDHs are smaller and midline displacement less pronounced after death than before death [7]. The implication of this observation is that radiologic findings of a fatal, clinically extensive SDH may be subtle on PMCT. Radiologists and forensic pathologists who are using PMCT should be aware that postmortem changes affect and decrease ante mortem findings in SDH.

AMCT characteristics of hematomas are routinely used for SDH dating: Acute hemorrhages (<3 days old) are hyperdense on CT (80–100 HU) relative to brain parenchyma, subacute hemorrhages (3–14 days) may be hyperdense, isodense, or hypodense, and chronic hemorrhages (>2 weeks) are hypodense to brain parenchyma (<40 HU)

Table 2 Comparison of SDH volume, midline shift, and SDH density on AMCT and PMCT

Case	Vol AM (ml)	Vol PM (ml)	Δ Vol (ml)	MS AM (mm)	MS PM (mm)	Δ MS (ml)	Dens AM (HU)	Dens PM (HU)	Δ Dens (HU)
1	1.2	1.1	-0.1	3.0	0.0	-3.0	68	71	3
2	15.3	2.3	-13.0	2.9	1.2	-1.7	78	88	10
3	168.2	65.1	-103.1	17.9	4.3	-13.6	80	85	5
4	14.5	2.6	-11.9	7.5	1.5	-6.0	52	85	33
5	162.1	114.6	-47.5	10.9	5.8	-5.1	73	91	18
6	0.3	0.0	-0.3	5.0	0.0	-5.0	52	73	21
7	102.0	20.0	-82.0	14.3	4.3	-10.0	74	97	23
8	114.8	66.9	-47.9	6.8	0.0	-6.8	65	70	5
9	159.9	144.7	-15.2	21.9	18.8	-3.1	71	84	13
10	93.9	58.4	-35.5	10.3	7.0	-3.3	68	92	24
11	135.0	34.4	-100.6	7.5	4.2	-3.3	76	96	20
12	2.2	19.8	17.6	1.9	1.5	-0.4	72	91	19
13	116.0	110.5	-5.5	6.7	1.8	-4.9	70	82	12
14	94.3	87.0	-7.3	3.5	0.0	-3.5	82	94	12
15	66.3	56.4	-9.9	0.0	0.0	0.0	73	82	9
16	0.6	1.7	1.1	0.0	0.0	0.0	70	80	10
17	0.2	0.2	0.0	14.8	5.4	-9.4	53	76	23
18	8.0	0.9	-7.1	3.1	4.1	1.0	66	58	-8
19	22.8	0.0	-22.8	0.0	0.0	0.0	72	64	-8
Mean	67.2	41.4	-25.8	7.3	3.2	-4.1	69.2	82.1	12.8
Median	66.3	20.0	-11.9	6.7	1.5	-3.3	71.0	84.0	12.0
Percentage (%)	100.0	61.6	-38.4	100.0	43.4	-56.6	100.0	118.6	18.6

Vol AM, volume of subdural hematoma (SDH) on ante mortem CT; Vol PM, SDH volume on postmortem CT; Δ Vol, difference between SDH volume on AMCT and PMCT; MS AM, midline shift on AMCT; MS PM, midline shift on PMCT; Δ MS, Difference between midline shift on AMCT and PMCT; Dens AM, Density of SDH on AMCT; Dens PM, Density of SDH on PMCT; Δ Difference of SDH Density between AMCT and PMCT

and isodense to cerebrospinal fluid [18]. The hyperdense, bright appearance of acute hematomas is primarily caused by the presence of protein-rich blood clots. Over time, the clots undergo gradual degradation, which results in the hypodense appearance of chronic hematomas [18].

In this study we found that the CT density of SDH was higher on PMCT than on AMCT in 17/19 cases. Of these 17 cases, 14 had died during the first 3 days after the SDH (i.e. in the acute phase) and 3 had died between the third and 14th day after the SDH (i.e. in the subacute phase). It is important to note that hematoma density increased in both acute and subacute SDH by more than 12 HU (nearly 20 %).

The increased density of SDH on PMCT is a key finding of this study because it indicates that hematomas may appear more acute than they actually are. This impairs our ability to determine the age of a SDH on PMCT. Age determination of SDH with CT is already a challenging task [19]. The fact that subacute hematomas may resume imaging characteristics of acute hematomas after death makes this it even more problematic, especially in cases where AMCT is not available and SDH dating is crucial.

In both cases where the density of SDH was lower on PMCT than on AMCT the victims had survived for more than 2 weeks after the SDH (i.e. in the chronic phase). This means that the protein-rich hematoma had already been degraded to hypodense fluid collection before death.

In this study we found no significant correlation between the PMI and the decrease of SDH volume, the regression of midline shift, or the increased hematoma density. This may indicate that these alterations appear early on after death and are relatively independent from the PMI. This conclusion is supported by the single case in our population where the PMI was only 3 h. In this case, the findings (decrease of midline shift by 59 %, reduction of SDH volume by 85 %, and increase of hematoma density by 13 %) are comparable to the overall findings of the study. However, in view of the small and relatively heterogeneous study population we cannot exclude that a larger cohort would reveal a correlation between the PMI and the decrease of SDH volume and midline displacement.

A number of limitations of this study deserve comment. The principal limitation of this study is that measurements

were performed by only one reader. The authors concede that the use of multiple readers or repeated measurements does fortify the scientific fundament of any study. However, there are a large number of studies which have revealed that measuring physical characteristics—including ROI and distance measurements—on radiologic images yield excellent intra-rater and inter-rater reliability not only on CT [20–22], but across all radiologic imaging modalities [23–26]. It is the opinion of these authors that the excellent reliability counterbalances this limitation. Second, the choice to select the last AMCT before death instead of the first AMCT after the occurrence of the SDH for comparison to PMCT may be questioned. However, in view of the objective of this study—i.e. the analysis of postmortem changes on SDH—the use of the latest available ante mortem data is a prerequisite. Inclusion of earlier AMCTs would inevitably introduce a higher number of potential confounders. A third limitation of this study lies in the small and relatively heterogeneous study population. However, the rare occurrence of suitable cases where both AMCT and PMCT are available was already noted by Inokouchi [7]. In addition, the fact that these postmortem changes of ante mortem pathology were so consistently present *in spite* of the heterogeneity of the study population supports the validity of our results. Nevertheless, additional studies with larger study populations are needed to further elucidate this phenomenon.

Conclusion

This study reveals that normal postmortem changes significantly affect the extent and imaging characteristics of subdural hematoma and may therefore affect the interpretation of these findings on PMCT. Radiologists and forensic pathologists who use PMCT must be aware of these phenomena in order to correctly interpret PMCT findings in cases of subdural hemorrhages.

Key points

1. Postmortem changes in the brain affect the imaging characteristics of subdural hematoma on PMCT.
2. Subdural hematomas are smaller on PMCT than on AMCT and concomitant midline shift is decreased which may impair the accurate interpretation of PMCT findings.
3. The density of acute and subacute subdural hematomas is higher on PMCT than on AMCT, which may affect the dating of subdural hematomas if AMCT is not available.

4. These changes appear early after death and their magnitude seem to be independent of the postmortem interval.

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