

# Comparison of volume and attenuation of the spleen between postmortem and antemortem computed tomography

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## Abstract

**Objectives** The purpose of this study is to compare the post-mortem changes in computed tomography (CT) findings between normal spleen, splenic infarct, and splenic tumor infiltration.

**Methods** The institutional review board approved this study, and informed consent was obtained from the next of kin. We studied 63 consecutive subjects who underwent antemortem CT, postmortem CT, and autopsy between February 2012 and December 2013. Postmortem CT was performed within 1678 min after death and was followed by pathological studies. The subjects were divided into three groups based on the pathological findings: normal, splenic infarct, and splenic tumor infiltration. The volume and attenuation of the spleen were compared between antemortem and postmortem CT using paired *t* tests. Gender, age, time elapsed since death, and the causes of death were examined as potential confounding factors of the postmortem changes in volume and attenuation.

**Results** In all groups, the spleen decreased in volume and attenuation increased on postmortem CT compared with

antemortem CT. The postmortem changes in spleen volume and attenuation were not significantly associated with sex, age, time elapsed since death, or causes of death.

**Conclusions** Spleen volume decreased and attenuation increased on postmortem CT compared with antemortem CT in subjects with a normal spleen, splenic infarct, or splenic tumor infiltration. These results should caution us against underestimating the significance of splenomegaly on postmortem CT, misinterpreting reduced splenic volume as the presence of hypovolemic or distributive shock in the subject while alive, and confusing postmortem splenic hyperattenuation with diseases characterized by this finding.

**Keywords** Forensic radiology · Postmortem computed tomography · Postmortem changes · Forensic medicine · Spleen

## Introduction

High-resolution imaging modalities such as computed tomography (CT) and magnetic resonance imaging are increasingly being used as adjuncts to traditional forensic methods in postmortem studies [1–5]. However, clinical radiologists may experience difficulty interpreting postmortem images because there are specific and nonspecific postmortem signs [6]. As guidelines for the diagnosis of postmortem images are now being established worldwide, it is important to understand the appearances of normal changes on postmortem CT [7]. The postmortem CT features of several organs, including the cardiovascular system [8–11], brain [12], thyroid [13], adrenal gland [14], muscle [15], airway [16], and lung [17], have already been described. To our knowledge, however, there are no reports describing the postmortem changes of the spleen between antemortem and postmortem images.

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Therefore, it is difficult to identify postmortem changes in a diseased spleen when the normal changes in the spleen on postmortem CT have not been documented. The objectives of this study were to document the normal changes that occur postmortem in the spleen and to compare these with the postmortem changes in the case of splenic infarct and splenic tumor infiltration. To achieve these objectives, we conducted a quantitative study in and compared splenic volume and splenic attenuation between postmortem and antemortem CT scans obtained in the same subjects.

## Materials and methods

The institutional review board approved this study, and written informed consent was obtained from the subject's next of kin to use the clinical, pathological, and radiographic data in this study.

### Study design and subjects

Consecutive subjects who died from nontraumatic causes in our academic tertiary care hospital and who underwent unenhanced upper abdominal antemortem CT, postmortem CT, and autopsy between February 2012 and December 2013 were retrospectively enrolled. Potential subjects were excluded for the following reasons: age <20 years, treatment with cardiopulmonary resuscitation, and prior splenectomy. The subjects were divided into three groups based on their pathological findings: normal spleen, splenic infarct, and splenic tumor infiltration. All cadavers were carefully transported on a stretcher and were kept in the supine position at room temperature from the time of death until postmortem CT.

### Antemortem CT imaging

All antemortem upper abdominal CT scans were obtained without contrast medium using a 64-detector-row helical CT scanner (Aquilion 64, Toshiba Medical Systems Corporation; Discovery CT750 HD and LightSpeed VCT, GE Healthcare) or a 320-detector-row helical CT scanner (Aquilion ONE, Toshiba Medical Systems Corporation), in the craniocaudal direction with the subject in the supine position with both arms raised. The scan parameters were as follows: slice thickness, 5 mm; slice interval, 5 mm; rotation time, 0.5 s; and tube voltage, 120 kVp. Automatic tube current modulation was performed using Volume EC (Toshiba) and AutomA (GE). Images were reconstructed at 0.5 mm intervals with a 350-mm field of view and a 512 × 512 image matrix.

### Postmortem CT imaging

Whole-body postmortem CT studies were obtained without contrast medium using a 4-detector-row CT scanner (Robusto, Hitachi Medical Corporation) in helical mode in the craniocaudal direction. For all scans, the cadaver was laid in the supine position with arms placed on either side of the body. The scan parameters were as follows: slice thickness, 2.5 mm; slice interval, 1.25 mm; rotation time, 0.5 s; tube voltage, 120 kVp; and tube current, 250 mA. Images were reconstructed at 1.25 mm intervals with a 350-mm field of view and a 512 × 512 image matrix.

### Image interpretation

All images were interpreted by a board-certified radiologist (6 years of experience) who was not provided with clinical information. The postmortem and most recent antemortem CT images were compared. CT images were reviewed on a two-dimensional workstation (OsiriX, Pixmeo Inc.). We measured splenic volume (in cm<sup>3</sup>) and attenuation (in Hounsfield units, HU) by manually contouring the spleen as the region of interest (ROI) on each slice the spleen was observed. Accessory spleens were not measured. We then calculated the total volume and the mean splenic attenuation using the OsiriX three-dimensional ROI tool.

### Pathological analysis

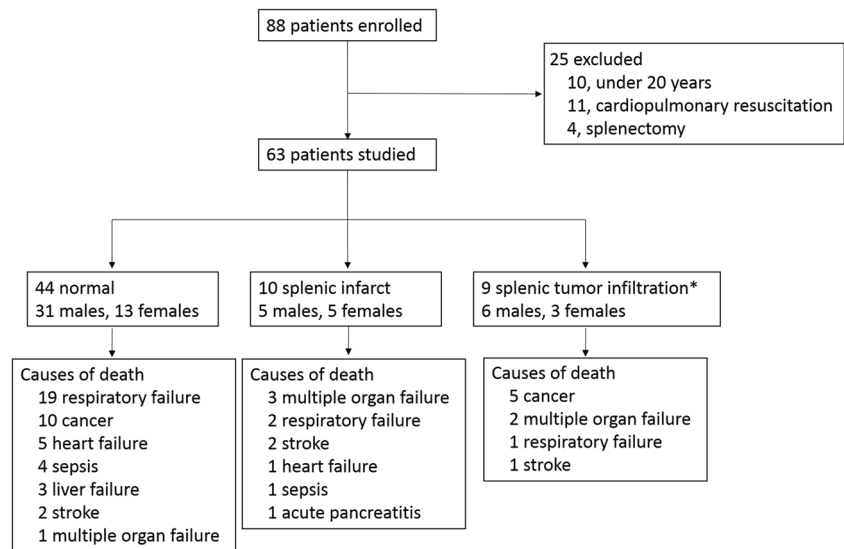
The subjects were divided into three groups based on the following pathological findings: normal spleen, splenic infarct, or splenic tumor infiltration (including tuberculoid granuloma). Normal spleen was defined as the absence of gross pathological findings and space-consuming lesions. Tiny benign lesions, such as cysts and calcified granulomas, were classified as normal. The pathological diagnosis of splenic infarct or splenic tumor infiltration was confirmed microscopically if it was uncertain on macroscopic autopsy or postmortem CT. The cause of death was determined based on the subject's antemortem clinical course and the postmortem pathological findings.

### Statistical analyses

We compared the differences in volume and splenic attenuation between antemortem CT and postmortem CT using paired *t* tests. The postmortem changes in volume and splenic attenuation were calculated as the ratio of volume and splenic attenuation on postmortem CT to that on antemortem CT. Simple linear regression was used to analyze the correlation between the autopsy weight and the postmortem CT volume of the spleen. Significant differences among the three groups (normal spleen, splenic infarct, and splenic tumor infiltration

**Fig. 1** Subject disposition.

\*Of the nine patients with splenic tumor infiltration, the pathology reports stated that the spleen was infiltrated by lymphoid tumor in five subjects, by metastases of the original tumor in three subjects (one each of hepatocellular carcinoma, pancreatic cancer, and renal pelvic cancer), and by tuberculoid granuloma in one subject



groups) were determined using the Kruskal–Wallis test. We used unpaired *t* tests to analyze the postmortem changes in splenic volume and attenuation between sexes and linear least squares regression to examine the effects of age and the time elapsed since death on splenic volume and attenuation. The Kruskal–Wallis test was used to determine the association between the cause of death and the postmortem changes in splenic volume and attenuation in each of the three groups (normal spleen, splenic infarct, and splenic tumor infiltration groups). The level of statistical significance was set at 0.05. All statistical analyses were performed using R version 3.0 (The R Foundation for Statistical Computing).

## Results

A total of 88 consecutive subjects were initially enrolled in this study. After applying the exclusion criteria, 63 adults (42 males, 21 females) were included in this study (Fig. 1). The mean age at death was 67 years (range, 39–90 years; median, 68 years). Antemortem CT was performed at a median of 23 days before death (range, 1–246 days). Postmortem CT

was performed at a median of 4.8 h after death (range, 1.5–28 h) and was immediately followed by autopsy.

The subjects were divided into three groups based on their pathological findings: normal spleen ( $n=44$ ; 31 males, 13 females), splenic infarct ( $n=10$ ; 5 males, 5 females), and splenic tumor infiltration ( $n=9$ ; 6 males, 3 females). Of the nine subjects with splenic tumor infiltration, the pathology reports stated that the spleen was infiltrated by lymphoid tumor in five subjects, by metastases of the original tumor in three subjects (one each of hepatocellular carcinoma, pancreatic cancer, and renal pelvic cancer), and by tuberculoid granuloma in one subject. The causes of death were respiratory failure ( $n=19$ ), cancer ( $n=10$ ), heart failure ( $n=5$ ), sepsis ( $n=4$ ), liver failure ( $n=3$ ), stroke ( $n=2$ ), and multiple organ failure ( $n=1$ ) in the normal spleen group; multiple organ failure ( $n=3$ ), respiratory failure ( $n=2$ ), stroke ( $n=2$ ), heart failure ( $n=1$ ), sepsis ( $n=1$ ), and acute pancreatitis ( $n=1$ ) in the splenic infarct group; and cancer ( $n=5$ ), multiple organ failure ( $n=2$ ), respiratory failure ( $n=1$ ), and stroke ( $n=1$ ) in the splenic tumor infiltration group. There were no cases of sudden death or hemorrhagic shock. No apparent gas was found in the spleen on antemortem CT, postmortem CT, or at autopsy.

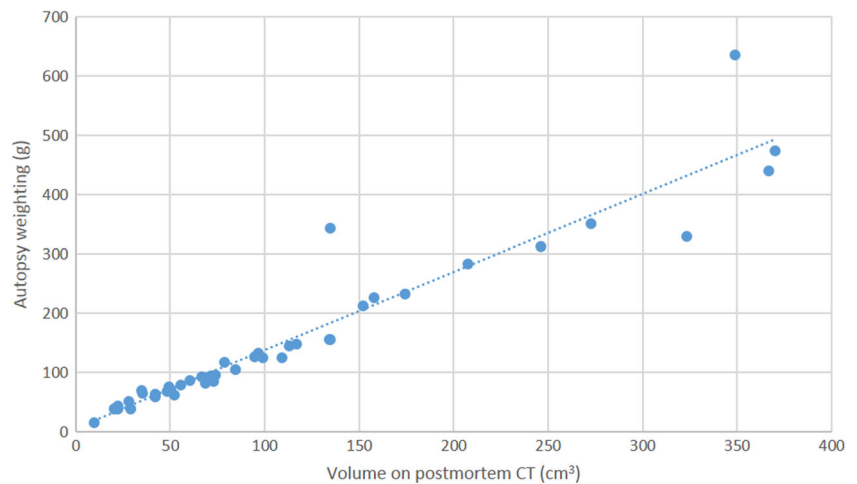
**Table 1** Comparison of splenic volume between antemortem and postmortem CT scans

	Normal group	Splenic infarct group	Splenic tumor infiltration group
Antemortem volume (cm <sup>3</sup> )	161.8±175.8	179.4±213.4	234.4±186.8
Postmortem volume (cm <sup>3</sup> )	110.9±96.9	138.8±170.3	166.5±171.7
<i>P</i> value <sup>a</sup>	<0.01	0.20	0.03
Postmortem/antemortem ratio	0.80±0.31	0.95±0.38	0.70±0.24

Values are presented as the mean ± standard deviation.

<sup>a</sup> Splenic volume was compared between the antemortem and postmortem CT scans using paired *t* tests.

**Fig. 2** Volume on postmortem CT versus autopsy weight. A simple linear regression analysis revealed a significant correlation between the measured volume on postmortem CT and the autopsy weight ( $P < 0.001$ ,  $R = 0.95$ ). The autopsy weight of the spleen ( $W$ ) can be calculated from the volume on postmortem CT ( $V$ ) as follows:  $W = (1.3 \times V) + 6.1$



The splenic volumes ( $\text{cm}^3$ ) on antemortem and postmortem CT are summarized in Table 1 according to the pathological group. Splenic volume was significantly less on postmortem CT than on antemortem CT in the normal ( $P < 0.01$ ) and splenic tumor infiltration ( $P = 0.03$ ) groups. In the splenic infarct group, the spleen tended to be smaller on postmortem CT than on antemortem CT, although the difference was not statistically significant. There were no significant differences among the three groups in terms of splenic volume between antemortem and postmortem CT ( $P = 0.20$ ).

The splenic volumes on postmortem CT and the autopsy weights of the spleens are shown in Fig. 2. The mean  $\pm$  standard deviation weight of the autopsy specimens was  $154.1 \pm 134.8$  g. A simple linear regression analysis revealed a significant correlation between the measured volume on postmortem CT and the autopsy weight ( $P < 0.001$ ,  $r = 0.95$ ). The autopsy weight of the spleen ( $W$ ) could be calculated from the volume determined on postmortem CT ( $V$ ) as follows:  $W = (1.3 \times V) + 6.1$ .

The CT attenuation values (in HU) of the spleen on antemortem and postmortem CT are listed in Table 2. The spleen showed significantly higher attenuation on postmortem CT than on antemortem CT in the normal ( $P < 0.001$ ) and splenic

tumor infiltration ( $P = 0.03$ ) groups. In the splenic infarct group, splenic attenuation tended to be higher on postmortem CT than on antemortem CT, although the difference was not statistically significant. There were no significant differences among the three groups in terms of CT attenuation values between antemortem and postmortem CT ( $P = 0.59$ ).

There was no statistically significant association between sex and postmortem change in splenic volume or splenic attenuation in any group (Table 3).

Table 4 shows the results of correlation analysis for postmortem change in splenic volume and splenic attenuation with each of age and the time elapsed since death. Only the splenic tumor infiltration group showed a statistically significant positive correlation between splenic volume ratio and age ( $P < 0.01$ ,  $r = 0.81$ ). Splenic volume was not correlated with age in either the normal or splenic infarct groups. Furthermore, the splenic volume ratio was not significantly correlated with the time elapsed since death in any group, and splenic attenuation was not correlated with age or time elapsed since death in any group. The cause of death was not significantly associated with the postmortem changes in splenic volume or splenic attenuation in any group (Table 5).

**Table 2** Comparison of splenic attenuation between antemortem and postmortem CT scans

	Normal group	Splenic infarct group	Splenic tumor infiltration group
Antemortem attenuation (HU)	$46.8 \pm 9.2$	$48.0 \pm 9.4$	$44.5 \pm 5.9$
Postmortem attenuation (HU)	$54.3 \pm 12.4$	$50.7 \pm 9.9$	$52.9 \pm 7.0$
$P$ value <sup>a</sup>	$<0.001$	0.46	0.03
Postmortem/antemortem ratio	$1.17 \pm 0.25$	$1.08 \pm 0.25$	$1.17 \pm 0.25$

Values are presented as the mean  $\pm$  standard deviation

HU Hounsfield units

<sup>a</sup> Splenic attenuation was compared between the antemortem and postmortem CT scans using paired  $t$  tests

**Table 3** Associations of splenic volume and attenuation with sex

	Splenic volume			Splenic attenuation		
	Male	Female	<i>P</i> value <sup>a</sup>	Male	Female	<i>P</i> value <sup>a</sup>
Normal group	0.78±0.30	0.86±0.33	0.46	1.17±0.24	1.19±0.28	0.79
Splenic infarct group	0.78±0.37	1.13±0.34	0.16	0.97±0.23	1.18±0.24	0.20
Splenic tumor infiltration group	0.67±0.23	0.76±0.31	0.69	1.23±0.19	1.15±0.35	0.72

Values are presented as the mean ± standard deviation

<sup>a</sup> Unpaired *t* tests were used to compare splenic volume and splenic attenuation between males and females

## Discussion

The normal splenic weight in the adult is approximately 100–150 g based on operative specimens and autopsy material [18, 19], and the normal splenic volume is approximately 200–220 cm<sup>3</sup> [20–22]. However, the weight or volume of an excised spleen differs from that in vivo, owing to several factors, such as the amount of entrapped blood, circulatory factors during surgery, the duration of vessel clamping, and uncontrolled blood loss from the specimen before it is weighed [23]. We do not need to worry about circulatory factors during surgery or the duration of vessel clamping in cadavers, but the amount of entrapped blood and uncontrolled blood loss from the specimen before it is weighed could affect the splenic weight recorded at autopsy. In this study, we compared the splenic volume on postmortem CT with the splenic weight at autopsy in the same subjects, where autopsy was promptly performed after postmortem CT. The strong positive correlation between the splenic volume on postmortem CT, and the splenic weight measured at autopsy indicates that the weight of the spleen can be estimated from the splenic volume measured on postmortem CT. In the present study, some outliers can be seen in Fig. 2, and these are likely due to an excessive amount of entrapped blood or uncontrolled blood loss from the specimen.

We found a reduction in splenic volume on postmortem CT compared with antemortem CT in all three groups. The splenic

volume decreases in hypovolemic or distributive shock [24, 25]. Because death is the ultimate state of hypovolemic or distributive shock, with the cessation of blood flow and a drop in circulatory pressure to approximately 7 mmHg [26, 27], it is reasonable to expect a postmortem reduction in splenic volume, as was observed in our study.

The present results show that splenic attenuation was higher on postmortem CT than on antemortem CT in all of the three groups. Postmortem clotting and sedimentation of blood components may explain the higher attenuation of the spleen on the postmortem images. However, these phenomena usually form layered structures or horizontal planes, owing to the effects of gravity [28], but no clear layers or obvious differences in attenuation between ventral and dorsal sites in the spleen were observed in our subjects. It is possible that the serous component leaked from the spleen and the concentrated blood left in the spleen caused higher attenuation on postmortem CT. However, the attenuation of blood on postmortem CT is similar to that on antemortem CT [29]. Thus, postmortem changes of the blood itself are unlikely to explain the higher splenic attenuation on postmortem CT.

Because attenuation is determined by the physical density of an object [30], higher attenuation on postmortem CT indicates that the splenic components have a higher density after death than during life. The spleen has a rich blood flow, so we can assume that it loses blood and becomes more compact at death, leading to the higher attenuation on postmortem CT.

Autolysis could have contributed to the postmortem changes observed in the spleen in our study. Although autolysis is known to occur during agonal stage [31–33], CT evidence of

**Table 4** Correlation of splenic volume and attenuation with age and time elapsed since death

	Splenic volume		Splenic attenuation	
	Age	Time elapsed since death	Age	Time elapsed since death
Normal group	0.45	0.37	0.42	0.72
Splenic infarct group	0.20	0.97	0.36	0.78
Splenic tumor infiltration group	<0.01	0.57	0.40	0.96

*P* values are shown and were determined by linear least squares regression

**Table 5** Associations of splenic volume and attenuation with the cause of death

	Volume	Attenuation
Normal group	0.15	0.21
Splenic infarct group	0.17	0.13
Splenic tumor infiltration group	0.12	0.23

*P* values are shown and were determined using the Kruskal–Wallis test

autolysis generally appears later than 24–48 h after death [28]. In the present study, postmortem CT was performed within 28 h after death, which is outside the timing of visualization of autolysis. Accordingly, the present results show no significant associations between the postmortem changes in splenic volume or attenuation and the time elapsed since death at 1.5–28 h after death.

We also investigated possible confounding factors of postmortem changes such as sex, age, and the cause of death. Although a positive association between splenic volume and age was observed in the splenic tumor infiltration group, the reason for this is uncertain. Otherwise, there were no statistically significant associations between the confounding factors of sex, age, or cause of death with the postmortem changes in splenic volume or splenic attenuation. Overall, these results indicate that volume reduction and splenic hyperattenuation are general postmortem findings regardless of sex, age, cause of death, or the presence of splenic diseases such as infarct and tumor infiltration.

As mentioned above, it is common to observe a small spleen under the condition of hypovolemic or distributive shock in life. However, we cannot conclude that a subject died from hypovolemic or distributive shock based on the postmortem CT finding of a reduced splenic volume because the healthy spleen loses volume postmortem. We also need to bear in mind that we may underestimate or even do not notice splenomegaly, especially in cases of splenic tumor infiltration in which the splenic volume decreases more than normal after death.

Splenic calcification, haemochromatosis [34], and Thorotrast accumulation [35, 36] are known to cause splenic hyperattenuation in life, and this finding is not specific to postmortem changes. However, if no comparable antemortem CT images are available in the same subject, then we should consider that splenic hyperattenuation on postmortem CT is a normal postmortem change rather than evidence of an underlying disease.

Our study has some limitations. First, interscanner variability of the CT attenuation values should be considered [37, 38] because we used several different CT scanners. However, recent technological innovations have greatly decreased the effect of scanner variability [39], and we standardized the tube voltage at 120 kVp for this reason. Second, it is possible that underlying diseases or cadaver storage might contribute to the postmortem differences [40, 41]. However, these factors are unlikely to affect our conclusions because we separated subjects with splenic diseases from those with a normal spleen based on the pathological findings, and all of the cadavers were preserved at our hospital in the same place, at the same temperature, and in the same position.

In conclusion, our study showed a reduction in splenic volume and splenic hyperattenuation on postmortem CT images compared with antemortem CT images in subjects

divided into normal, splenic infarct, and splenic tumor infiltration groups. These results suggest that, when interpreting postmortem CT, especially in subjects with splenic tumor infiltration, it is important to avoid underestimating splenomegaly, misinterpreting reduced splenic volume as a condition of hypovolemic or distributive shock while alive, and mistaking normal findings for those of a disease process depicted by splenic hyperattenuation.

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