Current Problems in Diagnostic Radiology 48 (2019) 224–228

Current Problems in Diagnostic Radiology

journal homepage: www.cpdrjournal.com

Hepatic Heterogeneity and Attenuation on Contrast-Enhanced CT in Patients With the Hypovolemic Shock Complex: Objective Classification Using a Contemporary Cohort

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Objective: When objectively measured on computed tomography (CT), does hepatic heterogeneity or overall liver attenuation predict the presence of shock?

Methods: This retrospective study included 73 patients (mean age 33 years) with the hypoperfusion shock complex (HSC) on CT (cases) and 100 patients (mean age 43 years) with negative trauma CT scans (controls). Liver heterogeneity was calculated by using consistently sized regions of interest (ROIs) to measure the 2 highest and the 2 lowest areas of hepatic density (in Hounsfield units [HU]). The difference between the means of the 2 highest and 2 lowest ROIs was considered the heterogeneity. Attenuation was calculated using the mean of 3 randomly placed ROIs. Both heterogeneity and attenuation were then compared between cases and controls.

Results: Median hepatic heterogeneity was 16.8 HU (IQR: 10.7-23.4) for the HSC group and 9.0 HU (IQR: 7.0-10.4) for the controls ($P < 0.001$). The area under the curve was 0.79, and a threshold of 30 HU yielded a specificity of 100%. Median hepatic attenuation was not significantly different between the HSC and the control groups, with an area under the curve of 0.56.

Conclusions: Increased hepatic heterogeneity may represent an objective marker of the HSC that performs in a similar manner to other established signs. By comparison, overall hepatic hypoattenuation is a poor indicator of the HSC.

Introduction

The hypovolemic shock complex (HSC) represents a collection of signs on computed tomography (CT) that correlate with clinical hypovolemic shock.[1-8] Although the primary aim of CT in patients with shock is to locate a potential causative factor, such as hemorrhage or infection or both, recognition of the findings of the HSC can have prognostic and therapeutic implications.[7] Since Taylor's original documentation of the HSC in 1987,[8] several CT signs have been described including findings related to the vasculature, solid viscera, and hollow viscera, among others.[9-11] Although numerous findings have emerged (some as recently as 2005[12]), evidence supporting these signs is generally based on small numbers of patients. Moreover, some signs have clear objective criteria, whereas others are infrequently mentioned and may be subjective.

One organ affected by hypovolemia that remains incompletely classified is the liver. Hepatic assessment in the setting of the HSC is sometimes subjective,[13] and nomenclature may differ between studies. Some sources refer to hepatic hypoenhancement as a sign of the HSC,[10,14] while others comment on heterogeneity as a feature.[13] Sometimes the 2 terms appear to be used interchangeably.[11] Although it appears that abnormal liver enhancement likely falls somewhere along the HSC spectrum, more objective classification is needed.

The purpose of our study was to categorize abnormal liver enhancement objectively in the setting of the HSC, in regard to both heterogeneous enhancement of the liver and hepatic attenuation. We also aimed to assess the relative frequency of abnormal liver findings compared to other common features of the HSC and to determine any association with mortality.

Materials and Methods

This Health Insurance Portability and Accountability Act-compliant study was approved by our Institutional Review Board, which granted a waiver of consent due to the study's retrospective nature.

Patient Selection

A retrospective review of our institution's picture archiving and communications system was performed from January 2004 to July 2014. Search terms including "hypoperfusion," "hypovolemic shock complex," "shock bowel," and "shock" (among others) were used to identify potential CT scans of the abdomen and pelvis that

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https://doi.org/10.1067/j.cpradiol.2018.02.004
0363-0188/

might feature the HSC. This search yielded 422 CT scans, which were then narrowed after radiology report and imaging review to identify CT scans that actually had 2 or more features of the HSC (see Appendix for definitions). After reviewing medical records, patients who did not meet clinical criteria for shock[1] were excluded ($n = 10$).[15,16] The end of the search yielded 73 patients. For the control population, we identified an additional 100 consecutive CT scans with the indication of trauma that had no evidence of injury (ie, negative CT scans). We reviewed the medical records to ensure that these patients did not meet clinical criteria for shock.

Imaging Technique

Most CT examinations ($n = 67$) were performed on a GE Lightspeed VCT MDCT scanner (General Electric Healthcare, Inc., Waukesha, WI). A small number of examinations were performed on a GE Lightspeed 16 ($n = 3$), a GE Lightspeed QX/i ($n = 1$), a Siemens Somatom Definition Flash ($n = 1$) (Siemens Medical Solutions USA, Inc., Malvern, PA), or a Siemens Definition ($n = 1$). All images were acquired during the portal venous phase of contrast material enhancement. Patients over the age of 12 ($n = 70$) received 150 mL of iopadmidol 300 mg/mL (Isovue-300, Bracco Diagnostics, Inc., Princeton, NJ) at an injection rate of 3-4 mL/sec. All patients were imaged using 120 kVp with a section thickness and intersection gap of 5 mm. Patients under the age of 12 ($n = 3$) received a weight-based dosage of iopamidol 300 mg/mL (1-2 mL/kg) and were imaged using 140 kVp. In the pediatric population, the section thickness and intersection gap ranged from 2.5-5 cm.

Quantitative Measurement of Hepatic Heterogeneity

To measure liver heterogeneity, images were analyzed using the TeraRecon image viewer (TeraRecon Inc., Foster City, CA). Four circular regions-of-interest (ROIs) (2.0 cm^2 in size, 784 pixels) were manually positioned in liver parenchyma at the level of the main portal vein (axial image). The 2 ROIs with the highest obtainable Hounsfield units (HU) value were averaged, as were the 2 ROIs with the lowest obtainable HU (Fig 1). The 4 ROIs could not overlap, nor could they include hepatic vessels, regions of hepatic injury, areas of any appreciable streak or beam-hardening artifact, or any hypoenhancing or hyperenhancing hepatic lesion. Furthermore, no measurements were obtained in common locations of focal fatty sparing or focal fatty deposition (ie, perilligamentous, perihilar, and pericholecystic regions).

Quantitative Measurement of Hepatic Attenuation

Overall hepatic attenuation was determined by placing three circular ROIs (2.0 cm^2 in size, 784 pixels) in liver parenchyma at the level of the main portal vein (axial image); 2 ROIs were randomly placed in the right hepatic lobe and 1 in the left hepatic lobe. These 3 ROIs were then averaged. The same restrictions regarding ROI placement (no overlap, no vessels, no lesions, etc.) were again used. The average attenuation was divided by the attenuation of the left psoas muscle (at any level where the ROI would fit inside the muscle), which served as an internal reference. Left psoas muscle attenuation was measured using a circular ROI (2.0 cm^2, 784 pixels).

[1] Shock defined by the presence of any one of the following:
1. Overt sign(s) of tissue hypoperfusion such as mottled skin.
2. Systolic blood pressure < 90 mm Hg or mean arterial pressure < 65 mm Hg.
3. Lactate level ≥ 4.0 mmol/L, pH ≤ 7.1, or base deficit of ≤−5 mEq/L.

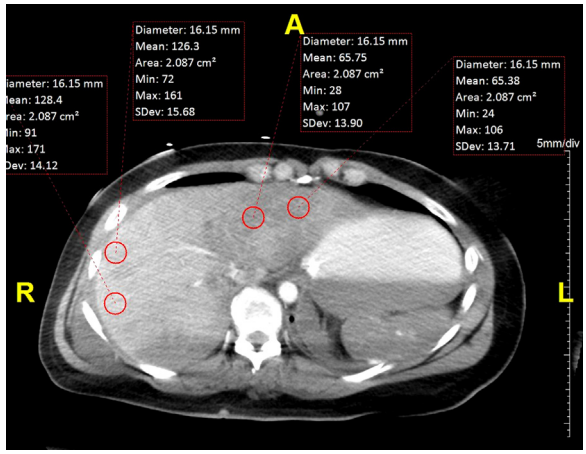

FIG 1. Quantification of hepatic heterogeneity using 4 ROIs. As part of the calculation, 2 ROIs with the highest obtainable HU and 2 ROIs with the lowest obtainable HU were obtained. (Color version of figure is available online.).

Quantitative Assessment of Other Signs of HSC

Each of the CTs was also assessed for 10 additional signs of the HSC according to established criteria: (1) small aorta, (2) small IVC ("flat cava" sign), (3) IVC halo, (4) gallbladder wall hyperenhancement, (5) pancreatic parenchymal hyperenhancement, (6) peripancreatic fluid, (7) adrenal gland hyperenhancement, (8) renal cortical hyperenhancement, (9) small bowel mucosal hyperenhancement, and (10) splenic parenchymal hypoenhancement (see Appendix for definitions and references used for each sign). A single reader (a fourth-year radiology resident) compiled the measurements for determinations of these signs.

Statistics

Descriptive statistics were compiled for liver heterogeneity, liver attenuation, and other signs of HSC. A Mann-Whitney test was used to compare liver heterogeneity and liver attenuation values between the HSC group and the control group. The study population was then randomly divided into 2 halves, each with a mixture of cases and controls. The first half was considered a training set, and receiver-operating-characteristic (ROC) curves were generated for hepatic heterogeneity and attenuation, and area under the curve (AUC) was calculated for each sign. The Youden index was then calculated for heterogeneity and for attenuation. Using each Youden index, sensitivity and specificity for heterogeneity and for attenuation were then calculated using the second half of the patients, the test set.

Next, to test whether a given sign of HSC was observed more often when hepatic heterogeneity was abnormal, Chi-square tests were calculated between each sign of HSC and hepatic heterogeneity. This was conducted using the entire dataset. Lastly, a logistic regression was performed to determine whether having a heterogeneous liver was associated with increased odds of mortality. Statistical analysis was completed using Microsoft Excel (Microsoft Corp., Redmond, WA) and R (theR-project.org). P values of less than 0.05 were considered significant.

TABLE 1
Demographics of HSC and control patients

	HSC group, $N = 73$	Control group, $N = 100$
Age (years)	33 ± 16	43 ± 18
Sex	53 M, 20 F	68 M, 32 F
Avg. CT signs of the HSC	5.1	0

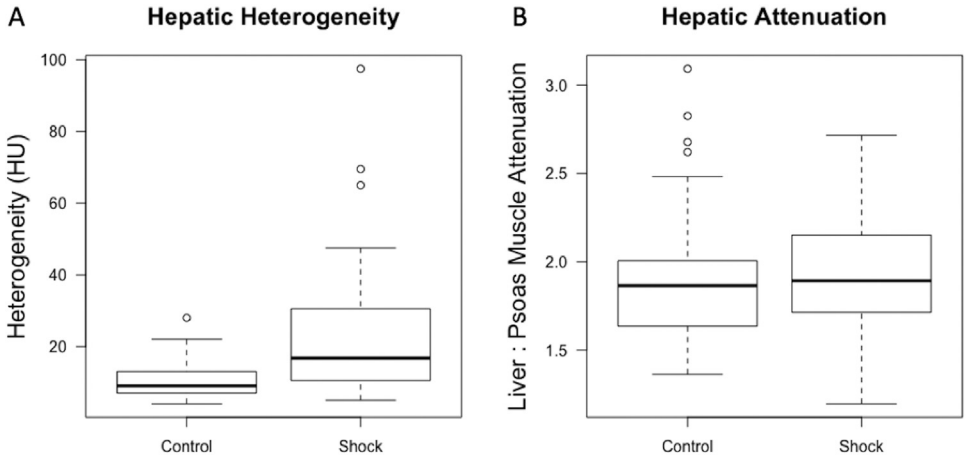

FIG 2. Box-and-whisker plots for (A) hepatic heterogeneity and (B) hepatic attenuation for control vs HSC patients. Median heterogeneity in the HSC group was higher than controls, with individual values greater than any patient in the control group. For hepatic attenuation, no significant differences were seen between groups.

Results

A total of 73 patients with a CT of the abdomen and pelvis for trauma had CT scans featuring at least 2 signs of the HSC. All of these patients met the clinical criteria for shock. The mean age was 33 years (range: 0-83), and there were 53 men and 20 women (Table 1). Control demographics can also be found in Table 1.

In the HSC group (of the training set of cases), median hepatic heterogeneity was 16.8 HU (interquartile range [IQR]: 10.7-23.4), while median heterogeneity in the control group was significantly lower at 9.0 HU (IQR: 7.0-10.4) ($P < 0.001$). The highest heterogeneity value in the control group was 28.0 HU (range: 4.0-28.0); by comparison, 29% of HSC cases had values higher than 28.0 HU (range: 5.0-97.5) (Fig 2A). The ROC curve for hepatic heterogeneity can be found in Fig 3A, with an AUC of 0.79. The Youden index for heterogeneity as an indicator of the HSC was 16.3 HU, which when applied to the test set yielded a sensitivity of 50% and a specificity of 82%. Using a threshold of 30 HU yielded a sensitivity of 20% and a specificity of 100%.

With regard to mean hepatic attenuation, there was no significant difference when comparing the HSC group to the control group. The median ratio of liver-to-psoas muscle attenuation in the HSC group (among the training cases) was 1.9 (IQR: 1.7-2.0), compared to 1.9 (IQR: 1.6-2.0) for the controls ($P = 0.34$) (Fig 2B). The ROC curve for hepatic attenuation in Figure 3B shows an AUC of 0.56. Using the Youden index of 1.7 to calculate performance in the test set yielded a sensitivity of 27% and a specificity of 80%.

Many additional signs of the HSC were seen throughout the HSC group, with an average of 5.1 signs per case. The most frequently occurring sign was a small IVC, present in 75.3% of cases (Table 2). The sign seen least often was the IVC halo sign, present in 23.6% of cases. By using 30 HU as a threshold, hepatic heterogeneity was identified in 24.6% of cases. When present, liver heterogeneity was correlated with the presence of small bowel hyperenhancement ($P = 0.01$) but not with any other signs (Table 2). When controlling for age and sex, increased hepatic heterogeneity (above 30 HU) was not correlated with an increased likelihood of death ($P = 0.28$). Similarly, the total number of signs was not correlated with death ($P = 0.69$).

Discussion

Our study objectively quantified hepatic heterogeneity and attenuation on CT in trauma patients with 2 or more signs of the HSC. There was a significant difference in heterogeneity when comparing patients with the HSC to a control group, suggesting that hepatic heterogeneity continue to stand as a marker of HSC (Fig 4). By comparison, hepatic attenuation (as defined by a ratio of liver-to-psoas muscle attenuation) was not different between HSC

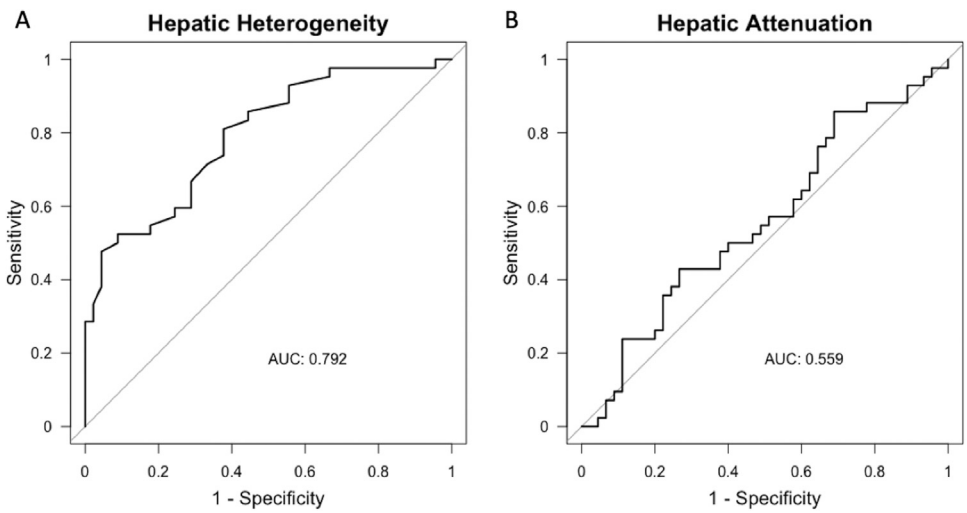

FIG 3. Receiver-operating-characteristic (ROC) curves for (A) hepatic heterogeneity and (B) hepatic attenuation. Comparing the area under the curves highlights the superiority of heterogeneity as a marker of the HSC.

TABLE 2
Frequency of CT signs among patients with HSC and correlation with heterogeneous liver sign

HSC patients ($N = 73$)	Percent of HSC cases	Chi-square p-value	Percent in Ames et al [13]	Percent in Ryan et al [14]
Heterogeneous liver	24.6	n/a	n/a	n/a
Small aorta	43.8	0.45	15	50
Small IVC	75.3	0.19	61	100
Halo sign	23.2	0.84	n/a	77.8
Gallbladder hyperenhancement	47.9	0.94	n/a	33.3
Pancreatic hyperenhancement	63.0	0.93	n/a	n/a
Peripancreatic fluid	43.8	1	29	29.6
Adrenal hyperenhancement	26.0	0.61	n/a	51.9
Renal hyperenhancement	64.3	0.96	n/a	n/a
Small bowel hyperenhancement	65.7	0.01	n/a	70
Splenic hypoenhancement	34.2	1	12	29

patients and controls and does not appear to represent a consistent marker for the HSC.

The role of CT in identifying and grading hypovolemic shock is well-documented, and while numerous signs are described in the literature, not every feature is completely understood; hepatic enhancement represents one such feature. Specifically, many authors do not specify whether they are measuring heterogeneity or hypoenhancement, which can lead to confusion. For example, Tarrant et al[10] assert that "hepatic enhancement is typically heterogeneous in HSC," but then say "a reduction in hepatic enhancement (25 HU less than the spleen) is thought to be significant." Similarly, Wang et al[11] describe both a heterogeneous liver and hepatic hypoenhancement, without clarity. In a study of the CT halo sign, Ryan et al[14] document decreased hepatic enhancement in a series of HSC patients, but do not define their criteria. In their description of 41 patients with shock bowel, Ames et al[13] outline a subjective assessment of liver hypoenhancement in one section but then document that 10% of the patients had a heterogeneously enhancing liver. Compared to other vascular and visceral signs, the liver in the HSC remains poorly documented.

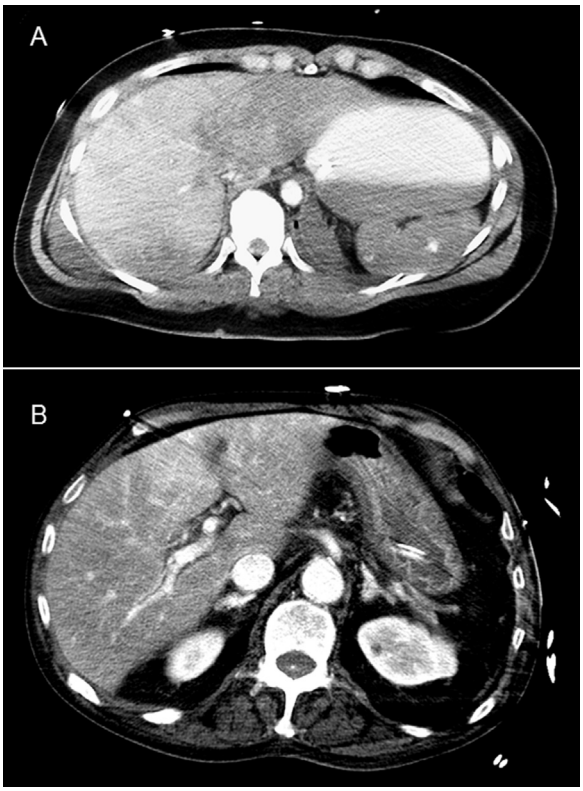

FIG 4. Examples of increased hepatic heterogeneity in 2 different patients (A and B) with the HSC.

To our knowledge, no widely accepted, objective criteria have been established for defining either hepatic heterogeneity or hypoenhancement. Compared to other studies, our series of 73 patients with the HSC constitutes a relatively large dataset that can serve to establish such criteria. Based on our results, we propose a threshold of 30 HU when defining hepatic heterogeneity in the HSC. Although the ROC analysis points to a lower threshold (16 HU), 30 HU may be more appropriate for 2 reasons. First, when using a sign to diagnose shock, maintaining the highest possible specificity helps readers be more certain of their diagnosis. Second, our authors felt that heterogeneity values above 30 HU could be subjectively and easily identified visually while interpreting a study. Using 30 HU as a threshold for hepatic heterogeneity resulted in a frequency of 24.6% in our cohort, in line with the prevalence of other signs of HSC.

Compared to heterogeneity, differences in hepatic attenuation between HSC patients and controls could not be established. Using the psoas muscle (a relatively hypoenhancing anatomical structure) as an internal reference standard, hepatic attenuation was not significantly different between the 2 groups, and ROC analysis showed that the test performed poorly. This makes sense, as it has been postulated that autoregulation and dual blood supply make global hepatic hypoattenuation a rare sign seen only in cases of severe shock.[14] By comparison, an organ with a single blood supply, such as the spleen, has been shown to hypoenhance in multiple studies.[13,14]

In general, 73 patients with the HSC demonstrated CT signs with frequencies relatively similar to previously published values. Comparing each individual sign, its prevalence, and its merits is beyond the scope of this investigation, but in establishing a baseline for liver heterogeneity, it is important to demonstrate that our cohort was relatively similar to previous reports of HSC patients. For the vasculature, for example, our incidences of small aorta (43.8%) and small IVC (75.3%) fell between previously published values of 15%-50% for the aorta and 61-100% for the IVC.[13,14] The frequency of shock bowel (65.7%) and splenic hypoenhancement (34.2%) was also similar to previously published values of 70% and 12%-29%, respectively.[14]

Although we aimed to provide an accurate and objective measurement for hepatic heterogeneity and hypoenhancement, our study has limitations. First, false positives could limit specificity for hepatic heterogeneity, namely, a liver with marked heterogeneous pattern of fat deposition, over-hydration, hepatitis, or congestive hepatopathy. Ideally, the latter two could be differentiated on clinical grounds, but this is nonetheless a limit of this sign. Second, our population included patients with 2 or more signs of the HSC. This excludes those patients with only 1 sign and who might demonstrate more subtle findings of the HSC. Third,

there are practical limits of using this sign; heterogeneity may only be appreciated at values of 30 HU or above, which limits sensitivity in the clinical setting. In addition, it is cumbersome to draw multiple ROIs, and finding exact values may not be practical in a daily clinical workflow. Despite these limitations, these data help clarify and separate the terms heterogeneity vs attenuation/enhancement regarding the liver in the setting of shock. Although the thresholds may not be derived from a large enough population to make them completely generalizable, they are likely sufficient to support future investigation of hepatic heterogeneity rather than attenuation.

In conclusion, hepatic heterogeneity may represent an objective marker of the HSC in the appropriate setting. By comparison, a hypoattenuating liver is not a reliable sign of the HSC.

Disclosure

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflicts Of Interest and Source of Funding

— Dr. Wildman-Tobriner has no disclosures.
— Dr. Enslow has no disclosures.
— Dr. Nelson has the following disclosures:
  — Consultant, General Electric Company
  — Consultant, Nemoto Kyorindo Co, Ltd
  — Consultant and Research Support, VoxelMetrix, LLC
  — Consultant, Bracco Diagnostics
  — Research Support, Wolters Kluwer

Appendix. : Definitions for CT Signs of HSC

1. *Slit-like IVC*: Defined as extrahepatic IVC measuring $<$ 9 mm in AP diameter on at least 3 consecutive slices.[1,7,10,11,13,14,17-19]
2. *IVC halo sign* (*ring of edema around a collapsed intrahepatic IVC*): Defined as the short axis diameter of the opacified intrahepatic IVC measuring less than half the short axis diameter of the intrahepatic IVC bed at the same level.[11,14]
3. *Small aorta*: Defined in patients above the age of 12, as having an AP diameter $<$ 1.3 cm measured 2 cm above and 2 cm below the renal arteries.[6,7,9-11,13,14] For patients 12 years old and younger, a small aorta was defined as an AP diameter $<$ 6 mm, measured 1 cm below the origin of the SMA.[17,19]
4. *Shock bowel*: Defined as small bowel wall thickening (submucosal edema $>$ 3 mm) and mucosal enhancement greater than the psoas muscle.[7,8,10,11,13,14,18,20]
5. *Gallbladder hyperenhancement*: Defined as gallbladder mucosal hyperenhancement. It is a described member of the HSC but has been determined subjectively in previous literature.[10,11,14] We define gallbladder hyperenhancement as gallbladder mucosal CT attenuation values at least 50 HU greater than the psoas muscle. Gallbladder mucosal attenuation values were measured using a line histogram profile plotting distance on the X-axis and HU on the Y-axis. Psoas muscle enhancement was measured on the left with a standard circular ROI 2.0 cm^2 in size (784 pixels). If a left psoas hematoma was present, the right psoas muscle was used.
6. *Pancreatic enhancement*: Defined as parenchymal enhancement measuring at least 20 HU greater than the liver or spleen; or parenchymal enhancement measuring at least 20 HU less than the liver.[10,11,17] Three circular ROIs (2.0 cm^2 in size, 784 pixels) were placed at the pancreatic head, body, and tail, and averaged.
7. *Peripancreatic fluid*: Defined as circumferential peripancreatic fluid measuring less than 20 HU.[10,11,13,14] Two circular ROIs (0.5 cm^2 in size, 49 pixels) were placed on the peripancreatic fluid, if present, and the attenuation values were averaged.
8. *Adrenal hyperenhancement*: Bilateral adrenal attenuation values greater than the IVC.[7,10,11,14,17,19,21,22] If there was unilateral hyperenhancement, this sign was recorded as not being present.
9. *Renal cortical hyperenhancement*: Defined as renal cortical enhancement greater than the aorta.[7-11,14] If there was unilateral hyperenhancement, this sign was recorded as not being present, except in the case of prior nephrectomy.
10. *Splenic hypoenhancement*: Defined as splenic enhancement at least 20 HU less than the liver.[7,10,11,13,14,17,23,24] The methods for determining splenic and hepatic attenuation values are the same as used for determining pancreatic enhancement (described above).

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