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ACCURACY AND SIGNIFICANCE OF COMPUTED TOMOGRAPHIC SCAN ASSESSMENT OF HEPATIC VOLUME IN PATIENTS UNDERGOING LIVER TRANSPLANTATION

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Abstract

Background. A smallliver volume is considered to be a poor prognostic factor in cirrhosis, often indicative of advanced liver disease. Radiologic assessment of liver volume before liver transplant is routinely performed in many transplant centers. We sought to assess the accuracy and significance of computed tomographic (CT) scanning in hepatic volumetric analysis by correlating CT-derived estimation of liver volume with that of corresponding liver explants.

Methods. A chart review of all patients aged 17 years or older undergoing liver transplant at Mount Sinai Medical Center between 1989 and 1995 was performed. Each patient underwent conventional CT scanning with measurement of liver volume (CTLV). Recipient liver volume (RLV) was defined as weight of liver explant after all attached ligaments, portal structures, and gallbladder were dissected free. Expected liver volume was calculated pretransplant based on age, gender, height, and weight. Patients were categorized into three groups based on etiology of liver disease: (1) hepatocellular (e.g., viral hepatitis, alcohol-related), (2) cholestatic (e.g., primary biliary cirrhosis), and (3) cryptogenic. The ratio of CTLV to RLV was used as a measure of the accuracy of CT volumetric analysis.

Results. A total of 579 patients was studied (group 1=376, group 2=139, group 3=64). All three groups were statistically similar with regard to age, prothrombin time and total bilirubin. Median CT liver volume was 1308 ml (range: 338-3847), 1651 ml (range: 641-3861), and 1210 ml (range: 348-2575) in groups 1–3, respectively; median ratio of CTLV to RLV was 1.02 (range: 0.50–2.31), 1.05 (range: 0.52–2.22), and 1.05 (range: 0.50–1.56) for groups 1–3, respectively. When RLV was small, it tended to be overestimated by CTLV. In contrast, when RLV was large, it was often underestimated. Clinical features such as history of esophageal variceal bleed, encephalopathy or ascites, and laboratory data did not influence accuracy of CT volumetric analysis.

Conclusions. CT-derived estimation of liver volume appears to correlate closely with actual weight of liver explant regardless of the etiology of chronic liver disease. With extremes in CT volumetric analysis, actual liver volume tends to be under- or overestimated. For patients with end-stage liver disease, both CT-derived and actual liver volume are greater in cholestatic than in hepatocellular disorders.

Total liver volume bears a relatively constant relationship to the body weight or surface area (2–2.7% of body weight) in normal subjects, but this does not hold true in the presence of intrinsic liver disease (1, 2). A small liver volume has proven to be a poor prognostic factor in cirrhosis, often gradually decreasing in the presence of progressive liver disease (3, 4). Previous studies demonstrated that liver volume, as assessed by serial ultrasonography, significantly decreased in a cohort of cirrhotic patients who died over a 55-month follow-up period (4). Liver volume has also been demonstrated to be significantly greater in survivors of fulminant hepatic failure than in non-survivors (5). Although bedside physical examination fails to provide accurate data pertaining to the overall size and volume of the liver (2), ultrasonography (4, 6), computed tomographic (CT) scanning (7–9) and, more recently, magnetic resonance imaging (10) have all been shown to provide adequate noninvasive measurements of liver volume in very small groups of patients. Several studies in which a direct comparison was made between ultrasound and CT scan have failed to conclusively show that one imaging modality is more accurate than another in estimating liver volume (4–6).

Accurate preoperative measurement of liver volume has become important in estimating segmental liver volume of donors for living related liver transplantation (11), predicting the extent of hepatectomy in patients with liver cancer (12, 13) and in assessing hepatic metastatic tumor burden (14). At many centers, preoperative estimation of liver volume has become a standard element of the evaluation of all potential candidates for orthotopic liver transplantation (OLT), although small liver volume is never considered to be an indication for OLT without appropriate clinical and biochemical determinants (15, 16). In this study, we sought to assess the accuracy of CT scanning in hepatic volumetric analysis by correlating CT-derived pre-OLT estimation of liver volume with the actual volume of corresponding liver explants, and to determine its significance in various categories of end-stage liver disease.

PATIENTS AND METHODS

A retrospective chart review was performed in all patients aged 17 years or older who underwent OLT at Mount Sinai Medical Center between 1989 and 1995. At the center, an abdominal CT scan with oral and intravenous contrast performed on site is a prerequisite for all potential OLT candidates. CT scans were often repeated every one or two years up until the time of OLT. The following data were obtained from the retrospective chart review: patient demographics, etiology of liver disease, presence or absence of intrahepatic tumor, patient's height and weight, body mass index (BMI), CT scan measurement of liver volume (CTLV), prothrombin time, total bilirubin and albumin, and the presence of ascites, portosystemic encephalopathy, and history of esophageal variceal bleed. A formula to estimate the expected liver volume (ELV) of each patient was developed using the data from 500 previous normal donor livers that were used for transplantation. Variables examined included age, sex, height, and weight. Multiple regression analysis using SPSS statistical software (SPSS Inc., Chicago, IL) was employed. The formula is as follows:*liver volume* (*in cubic cm*) =6 ×*weight* (*in pounds*) + 4 ×*age* (*in years*) + 350. This formula was found to be valid for livers from donors greater than age 11. Sex and height were not found to contribute further to the predictive model.

All patients underwent conventional CT scans performed on a 9800 CT scanner (General Electric, Milwaukee, WI). Serial transaxial scans were obtained from the diaphragm to the iliac crests by 10-mm collimation sections. Scans were obtained during suspended respiration after administration of oral contrast (E-Z-CAT; E-Z-M, Inc., Westbury, NY) as well as 150 ml of intravenous contrast (Omnipaque 240; Sterling Pharmaceutical, Inc., Barceloneta, Puerto Rico). The borders of the liver were manually outlined using a trackball while excluding the gallbladder. The areas outlined were integrated with computer assistance. The areas of all slices were summed up and then multiplied by 1 cm, which is the slice thickness, giving the CT-derived total liver volume in cubic cm. The approximate time for conventional image acquisition was 2 sec/image, and computer image construction required approximately 9 sec/image. Typical complete image acquisition and construction time for conventional CT is approximately 5 min, 40 sec for 20 cm of tissue.

After OLT, the native liver was weighed to the nearest gram after the attached ligaments, gallbladder, portal structures, and extraneous tissue had been dissected free. In this way no artifact was introduced as a result of tissue shrinkage after formalin fixation. The weight of the liver explant was defined as recipient liver volume (RLV). Because the liver in vivo contains a substantial quantity of blood, no attempt was made to remove the blood from the explant. The weight determination was performed without the knowledge of the prior estimated volume calculated from the CT scan images. Volumetric measurement via water displacement was not performed as the absolute value of liver weight is considered to be equivalent to actual liver volume, as the density of the liver is nearly the same as that of water (1 g of liver = l ml of fluid) (6, 17). Thus, in this study, CTLV/RLV was used as a measure of the accuracy of CT volumetric analysis.

Patients.

A total of 579 OLT patients met the criteria for inclusion in this study. They were separated into three groups, based on the etiology of their liver disease (see Table 1). Subjects with primarily hepatocellular liver disease (e.g., viral hepatitis or alcohol-related) comprised group 1, and patients with a predominantly cholestatic form of liver disease comprised group 2. Patients with cryptogenic cirrhosis (group 3) had denied any history of appreciable ethanol ingestion and had no definable etiology for chronic liver disease after completion of a history and physical examination, and analysis of standard hepatitis viral serologies, antinuclear antibodies, anti-smooth muscle antibody, serum iron, transferrin saturation, ferritin, ceruloplasmin, and [alpha]1-antitrypsin level.

TABLE 1. Categorization of patients based on etiology of liver disease

Group 1 (hepatocellular disease)	(n=376)
Viral hepatitis (B or C)	174 (46.3%)
Alcohol-related	85 (22.6%)
Viral hepatitis/alcohol	57 (15.2%)
Autoimmune hepatitis	21 (5.6%)
Hepatocellular carcinoma[a]	16 (4.2%)
Miscellaneous[b]	14 (3.7%)
Other hepatic malignancy[c]	9 (2.4%)
Group 2 (cholestatic disease)	(n=139)
Primary biliary cirrhosis	81 (58.3%)
Primary sclerosing cholangitis	47 (33.8%)
Other[d]	11 (7.9%)
Group 3 (Cryptogenic)	(n=64)

[a] In the presence of alcohol-related liver disease or viral hepatitis.
[b] Hemochromatosis, Wilson's disease, Budd-Chiari, hyperoxaluria, familial amyloidotic polyneuropathy, or α1-antitrypsin deficiency.
[c] Metastatic carcinoid tumor, metastatic leiomyosarcoma, or epithelioid hemangioendothelioma.
[d] Sarcoidosis, biliary atresia, secondary biliary cirrhosis, cystic fibrosis, Alagille's syndrome, or Caroli's disease.

Table 1. Categorization of patients based on etiology of liver disease[a] In the presence of alcohol-related liver disease or viral hepatitis.[b] Hemochromatosis, Wilson's disease, Budd-Chiari, hyperoxaluria, familial amyloidotic polyneuropathy, or [alpha]1-antitrypsin deficiency.[c] Metastatic carcinoid tumor, metastatic leiomyosarcoma, or epithelioid hemangioendothelioma.[d] Sarcoidosis, biliary atresia, secondary biliary cirrhosis, cystic fibrosis, Alagille's syndrome, or Caroli's disease.

Patients were excluded from the study if they had not had a CT scan performed at our center. In patients with significant renal dysfunction, magnetic resonance imaging scans were performed instead of CT scans to avoid potential contrast-induced nephropathy. Patients with fulminant and subfulminant liver failure were also excluded, as many did not have CT scans performed before OLT. Any patient requiring repeat transplantation was also excluded from the statistical analysis, as were all patients undergoing split and adult living related OLT. When serial CT scans were performed, only the CTLV performed closest to the time of OLT was considered. Patient characteristics are shown in Table 2. All demographic and laboratory data were obtained from the time of performance of the CT scan.

TABLE 2. Patient demographics and median values of liver volumes[a]

Patient demographic	Median value of liver volume		
	Group 1	Group 2	Group 3
Gender ($P<0.001$)			
Female	128 (35%)	89 (64%)	34 (53%)
Male	248 (65%)	50 (36%)	30 (47%)
Ascites ($P<0.001$)			
No	70 (19%)	72 (52%)	15 (24%)
Yes	297 (81%)	66 (48%)	48 (76%)
Variceal bleed ($P=0.500$)			
No	240 (67%)	92 (69%)	37 (61%)
Yes	118 (33%)	41 (31%)	24 (39%)
Encephalopathy ($P<0.001$)			
No	192 (54%)	113 (85%)	38 (59%)
Yes	166 (46%)	20 (15%)	26 (41%)
Age ($P=0.06$)	51.5 (18–75)	54.3 (17–71)	55.5 (27–70)
BMI ($P<0.0001$)	26.4 (15.0–47.6)	23.4 (15.4–40.8)	26.8 (17.8–49.4)
CTLV ($P<0.0001$)	1308 (338–3847)	1651 (641–3861)	1210 (348–2575)
ELV ($P<0.0001$)	1542 (1038–2186)	1398 (875–2024)	1592 (1220–2162)
RLV ($P<0.0001$)	1227 (300–4440)	1560 (630–3090)	1148 (350–2329)
CTLV/RLV ($P=0.505$)	1.02 (0.50–2.31)	1.05 (0.52–2.22)	1.05 (0.50–1.56)
CTLV/ELV ($P<0.0001$)	0.82 (0.23–3.15)	1.16 (0.41–2.39)	0.77 (0.25–1.52)
Prothrombin time (sec) ($P<0.0001$)	14.7 (11.0–25.1)	13.3 (11.1–24.3)	14.8 (11.4–22.5)
Bilirubin (mg/dl) ($P<0.0001$)	2.5 (0.2–36.5)	6.3 (0.5–46.5)	2.5 (0.4–23.0)

[a] Values are median (range); volumes are measured in milliliters.
P-value from Kruskal-Wallis test for measurement data and chi-square test for categorical variables.

Table 2. Patient demographics and median values of liver volumes[aa] Values are median (range); volumes are measured in milliliters.*P*-value from Kruskal-Wallis test for measurement data and chi-square test for categorical variables.

Statistical analysis.

Data were summarized and compared using SAS statistical software. Chi-square tests were used to test for differences between groups in categorical variables, and Wilcoxon or Kruskal-Wallis tests were used for measurement data. Differences between CT and recipient liver volumes are plotted by the methods recommended by Bland and Altman (17). The ratio of CTLV to RLV was used to compare CT scan accuracy between different groups of patients with end-stage liver disease. The ratio of CTLV to ELV was used to assess the association between liver volume and clinical condition in different categories of chronic liver disease.

RESULTS

The majority of patients in group 1 had viral hepatitis and/or alcohol-related chronic liver disease (84%). Approximately 6.6% of all patients had neoplasms. Very few patients in group 1 had infiltrative-type liver disease that might expect to increase total liver size (Table 1). The majority of patients in Group 1 were male, and had ascites but did not have history of variceal bleed or encephalopathy at initial transplant evaluation, as shown in Table 2. The majority of patients in Group 2 were female (as expected with the large number of patients with primary biliary cirrhosis) without ascites, history of variceal bleed, or encephalopathy. The three groups were statistically similar with regard to age, BMI, median prothrombin time, and serum bilirubin.

The median CTLV was greater for the cholestatic group when compared to groups 1 and 3. There was a large range of liver volumes for each of the three groups. Patients with alcoholic liver disease and tumors in group 1 appeared to have large CTLV. Extremely small livers were found in groups 1 and 3, whereas the smallest CTLV found in group 2 was 641 ml. As expected, there was much greater variability with regard to CTLV and RLV than with ELV. Both CTLV and RLV were overestimated in relation to ELV in groups 1 and 3 before OLT, whereas they were underestimated in the cholestatic group 2.

The ratio of CTLV/RLV was used as a measure of the accuracy of CT scan volumetric analysis. As shown in Figure 1, there was a linear relationship between RLV and CTLV. Median values of CTLV/RLV were similar in all groups: 1.02 (range: 0.50–2.31) in group 1, 1.05 (range: 0.52–2.22) in group 2, and 1.05 (range: 0.50–1.56) in group 3, as shown in Table 2. Thus, overall, CTLV appeared to correlate quite closely to RLV regardless of underlying liver disease. As a whole, CTLV tended to slightly overestimate RLV in all three groups. When RLV was small (i.e., less than 1000 ml), it tended to be overestimated by CTLV and when RLV was large (i.e., greater than 2500 ml), it tended to be underestimated (Fig. 2).

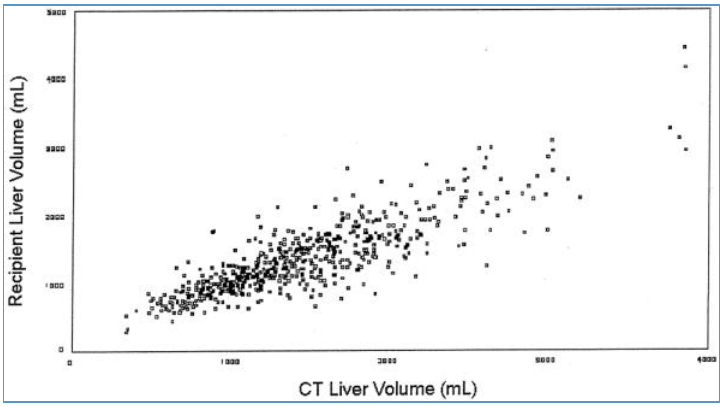

Figure 1. Relationship between pretransplant CTLV and explanted RLV. There exists a close correlation between CTLV and RLV.

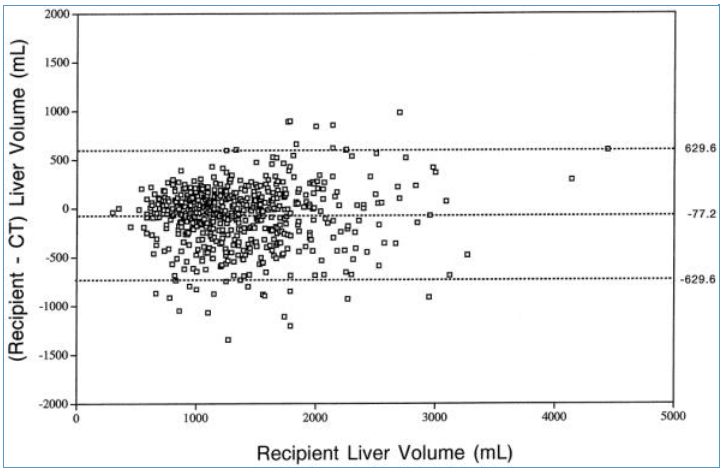

Figure 2. Relationship between difference of RLV and CTLV liver volumes and RLV. When RLV was smaller than 1000 ml, it tended to be overestimated by CTLV. In contrast, when RLV was greater than 2500 ml, it tended to be underestimated by CTLV. The dotted lines represent the mean difference + 2 standard deviations.

DISCUSSION

Radiologic determination of liver volume has become important in the preoperative evaluation of patients involved in living-related and other segmental OLT (11), and as a guide in decision making in resectional surgery for liver tumors

(12, 13). Determination of liver volume before OLT, usually via CT scanning, is performed in many centers (15, 16). Although liver volume is never the sole indication for proceeding with OLT (without considering biochemical and clinical variables), a decreasing liver volume on ultrasonography over time or an extremely small CTLV have been shown to be indicative of more advanced liver disease (3, 4). In one study, CTLV was significantly larger among survivors of fulminant hepatic failure when serial CT scans were performed before and after hepatic coma (5).

To date, the studies assessing the accuracy of CT analysis of liver volume have either included small numbers of patients or compared CTLV with liver volumes determined at the time of autopsy (4–7). In these studies, patients dying from cardiogenic shock or other conditions leading to hepatic congestion were included and may have had falsely elevated liver volumes (1). In addition, CTLV may have been overestimated as a result of the inclusion of juxtahepatic structures having contrast-enhanced CT attenuation similar to that of the liver (7, 8, 15, 16). Volumes of large livers are more likely to be overestimated because they are adjacent to a greater number of organs of similar density, which could potentially be included within the CT analysis (6, 8, 15).

The largest series of patients studied has been by Van Thiel and colleagues (6) at the University of Pittsburgh, who imaged 99 patients, distributed almost equally between patients with hepatocellular and cholestatic liver diseases. They found that both CT and ultrasonographic volumetric estimation approximated that of the actual liver volume determined by water displacement. Only 33 and 42 patients were imaged independently with CT scan and ultrasound, respectively; 27 patients were imaged with both modalities. The authors found ultrasound to be more accurate than CT, although both modalities showed a linear relationship between radiologically determined and actual liver volumes. Many of the CT-derived liver volumes were approximately 10% greater or less than the volumes determined directly by water displacement. CTLV appeared less accurate in livers of large volumes. Patients were not separated into separate groups of primarily hepatocellular and cholestatic etiologies; about 15% of patients had liver cancer, which might be expected to interfere with calculation of CTLV (6, 14).

In this study, involving 579 patients, we have shown that, by comparing CTLV with the weight of explanted livers, CT scan can closely approximate actual liver volume. Overall, CT scan tended to overestimate RLV by a total of less than 10%. Thus, this is in agreement with the results of other studies involving much smaller numbers of patients. Although CT scan sometimes grossly under- or overestimates RLV, there is often a fairly close correlation between the two, as shown in Figure 1. At extreme values in CTLV, RLV tended to be underestimated (CTLV < 1000 ml) or overestimated (CTLV > 2500 ml). This correlation is not dependent on whether the etiology of liver disease is cholestatic or hepatocellular in nature. Clinical factors such as the presence of hepatic encephalopathy, esophageal variceal bleed or ascites, as well as laboratory parameters, such as serum albumin and bilirubin, did not appear to influence the accuracy of CTLV. Thus, severity of underlying liver disease did not appear to effect the accuracy of CTLV in the estimation of a patient's actual liver volume. This is in agreement with previous studies, which showed that Child's classification (18) was unimportant in the calculation of CTLV (3). Furthermore, BMI also did not influence CTLV in this study.

In all analyses of liver volumes, group 3 was similar to group 1. Although occult cholestatic disease might have existed in some patients with cryptogenic cirrhosis, appreciable cholestasis present during pretransplant evaluation would have led to a more extensive work-up, including cholangiography, to exclude this. Histological review of the liver explant may have revealed occult [alpha]1-antitrypsin deficiency, hemochromatosis, or burnt-out autoimmune hepatitis. The majority of patients in group 3 would be expected to and were indeed found to have liver volume analyses similar to those for patients in group 1 with hepatocellular diseases.

There are several variables that potentially could have affected the calculation of CTLV and its correlation with RLV (Tables 3 and 4). In this study, the most recent CTLV of each patient was utilized, but the time varied from several weeks up to nearly 1 year before the time of OLT and the weighing of the explanted liver. Thus, there may have been further significant loss of RLV with clinical deterioration of liver disease leading to CTLV overestimation. However, serial CT scans in patients on the pre-OLT waiting list did not dramatically change within a 12-month follow-up (data not shown). In addition, there appears to be up to a 6–10% day-to-day variability and 4–8% inter-observer variability in calculating CTLV (6, 16). Many of the CT scans were of the nonhelical type; starting in 1994, scans were performed using helical CT scanners. Helical CT scanning might be expected to have an advantage over conventional CT scanning, as consistent levels of inspiration are essential in the latter to acquire accurate contiguous axial images of the liver. Respiratory variation, often a problem in patients with significant ascites, and patient motion can cause omission of several anatomic imaging levels and thus lead to diagnostic inaccuracy. However, several recent studies have shown that there is no significant difference between helical and conventional CT scan measurements of liver volume (11, 15, 16). Finally, assessment of the volume of water displaced by the liver explant is probably the method of choice for obtaining actual liver volume. Although we did not use this method, it appears that the absolute value of liver weight is equivalent to actual liver volume because the density of liver is nearly the same as that of water (6, 19). Thus, weighing the actual explanted volume after all attached ligaments portal structures were dissected free should not affect the correlation between CTLV and RLV.

TABLE 3. Effect of patient variables on RLV/ELV[a]

	Group 1	Group 2	Group 3
Prothrombin time (sec)	P=0.0001	P=0.03	P=0.0003
>14.0	0.73	0.98	0.66
<14.0	0.89	1.20	0.94
Albumin (g/dl)	P=0.0001	P=0.016	P=0.004
≤3.0	0.73	0.99	0.67
>3.0	0.87	1.20	0.91
Bilirubin (mg/dl)	P=0.002	P=0.10	P=0.07
>3.0	0.72	1.14	0.67
<3.0	0.83	1.03	0.77
Esophageal variceal bleed	P=0.16	P=0.10	P=0.49
Yes	0.82	1.03	0.77
No	0.76	1.12	0.75
Encephalopathy	P=0.07	P=0.15	P=0.55
Yes	0.76	0.98	0.75
No	0.80	1.11	0.76
Ascites	P=0.79	P=0.0001	P=0.29
Yes	0.78	0.95	0.74
No	0.79	1.24	0.80

[a] Wilcoxon P-value is shown; median values are listed.

Table 3. Effect of patient variables on RLV/ELV[aa] Wilcoxon *P*-value is shown; median values are listed.

TABLE 4. Effect of patient variables on CTLV/ELV[a]

	Group 1 (n=376)	Group 2 (n=130)	Group 3 (n=64)
Prothrombin time (sec)	P=0.0002	P=0.130	P=0.005
>14	0.77	1.09	0.69
≤14	0.92	1.19	0.88
Albumin (g/dl)	P=0.0001	P=0.002	P=0.020
≤3.0	0.76	0.95	0.71
>3.0	0.94	1.26	0.82
Bilirubin (mg/dl)	P=0.007	P=0.070	P=0.330
>3.0	0.77	1.19	0.76
≤3.0	0.87	1.09	0.78
Esophageal variceal bleed	P=0.610	P=0.030	P=0.960
Yes	0.84	1.08	0.77
No	0.82	1.20	0.76
Encephalopathy	P=0.006	P=0.120	P=0.290
Yes	0.75	1.04	0.74
No	0.86	1.19	0.78
Ascites	P=0.04	P=0.006	P=0.27
Yes	0.81	1.03	0.76
No	0.90	1.31	0.87

[a] Wilcoxon P-value is shown; median values are given.

Table 4. Effect of patient variables on CTLV/ELV[aa] Wilcoxon *P*-value is shown; median values are given.

Overall, patients with cholestatic liver disease were found to have larger RLV than patients with viral hepatitis and alcohol-related cirrhosis. This is in agreement with clinical studies examining the natural history of patients with cholestatic liver disease. Hepatomegaly is a frequent clinical finding in patients with cholestatic liver diseases such as primary biliary cirrhosis and sclerosing cholangitis (20). However, hepatomegaly is also often seen in hemochromatosis, Wilson's disease, and some patients with alcoholic liver disease, especially at an early stage of cirrhosis. Patients with cholestatic end-stage liver disease in this study were found to have greater CTLV (1651 ml vs. 1308 ml) and RLV (1560 ml vs.1227 ml) than those with hepatocellular diseases. Although CTLV overestimated RLV in all groups, RLV was significantly greater than ELV in the cholestatic group, but lower in groups 1 and 3. As ELV was calculated based on weight, it is not surprising that the median ELV for group 2 is low. A majority of patients in group 2 were women with primary biliary cirrhosis; frequently, there is hepatomegaly in the presence of a small physical build or body habitus (20).

Knowledge of CTLV by itself *is* somewhat limited as a single prognostic factor that can be helpful in assessing the need for OLT. In this study, no comparison between CTLV and RLV was made in the same patients over a period of time or between patients of different Child's classifications. Thus, no conclusion can be drawn regarding whether one or serial CTLV could be helpful in predicting progression of liver disease. One generalization that can be made is that, when the ratio CTLV/ELV is >1.0 in a patient with cryptogenic cirrhosis, it may suggest an occult cholestatic condition such as anti-mitochondrial antibody-negative primary biliary cirrhosis or small duct sclerosing cholangitis in the

appropriate clinical setting.

In summary, this study has shown that CT scan estimation of liver volume appears to closely correlate with the actual weight of the liver in patients undergoing OLT regardless of their underlying liver disease. Both CTLV and actual liver volumes are greater in patients with end-stage liver disease as a result of cholestatic rather than hepatocellular disorders. Thus, although CTLV itself as a single prognostic factor may not be helpful, it has a role in assessing the severity of liver disease in patients being evaluated for OLT, when used in conjunction with other clinical and biochemical data.

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BILDGALERIE

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TABLE 1. Categorization of patients based on etiology of liver disease

Group 1 (hepatocellular disease)	(n=376)
Viral hepatitis (B or C)	174 (46.3%)
Alcohol-related	85 (22.6%)
Viral hepatitis/alcohol	57 (15.2%)
Autoimmune hepatitis	21 (5.6%)
Hepatocellular carcinoma[a]	16 (4.2%)
Miscellaneous[b]	14 (3.7%)
Other hepatic malignancy[c]	9 (2.4%)
Group 2 (cholestatic disease)	(n=139)
Primary biliary cirrhosis	81 (58.3%)
Primary sclerosing cholangitis	47 (33.8%)
Other[d]	11 (7.9%)
Group 3 (Cryptogenic)	(n=64)

[a] In the presence of alcohol-related liver disease or viral hepatitis.
[b] Hemochromatosis, Wilson's disease, Budd-Chiari, hypercoagulopathia, familial amyloidotic polyneuropathy, or α1-antitrypsin deficiency.
[c] Metastatic carcinoid tumor, metastatic leiomyosarcoma, or epithelioid hemangioendothelioma.
[d] Sarcoidosis, biliary atresia, secondary biliary cirrhosis, cystic fibrosis, Alagille's syndrome, or Caroli's disease.

Table 1

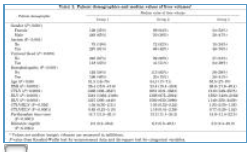

Table 2

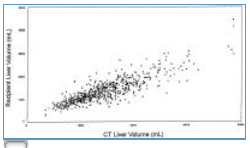

Figure 1

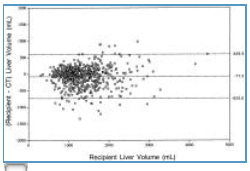

Figure 2

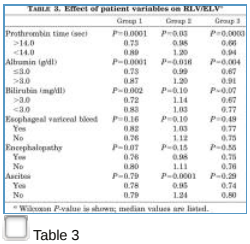

TABLE 3. Effect of patient variables on RLV/ELV[a]

	Group 1	Group 2	Group 3
Prothrombin time (sec)	P=0.0001	P=0.03	P=0.0003
>14.0	0.73	0.98	0.66
≤14.0	0.89	1.20	0.94
Albumin (g/dl)	P=0.0001	P=0.016	P=0.004
≤3.0	0.73	0.99	0.67
>3.0	0.87	1.20	0.91
Bilirubin (mg/dl)	P=0.002	P=0.10	P=0.07
>3.0	0.72	1.14	0.67
≤3.0	0.83	1.03	0.77
Esophageal variceal bleed	P=0.16	P=0.10	P=0.49
Yes	0.82	1.03	0.77
No	0.76	1.12	0.75
Encephalopathy	P=0.07	P=0.15	P=0.55
Yes	0.76	0.98	0.75
No	0.80	1.11	0.76
Ascites	P=0.79	P=0.0001	P=0.29
Yes	0.78	0.95	0.74
No	0.79	1.24	0.80

[a] Wilcoxon P-value is shown; median values are listed.

Table 3

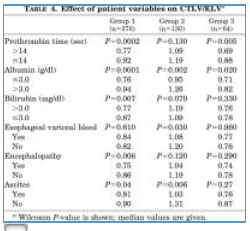

TABLE 4. Effect of patient variables on CTLV/ELV[a]

	Group 1 (n=376)	Group 2 (n=139)	Group 3 (n=64)
Prothrombin time (sec)	P=0.0002	P=0.130	P=0.005
>14	0.77	1.09	0.69
≤14	0.92	1.19	0.88
Albumin (g/dl)	P=0.0001	P=0.002	P=0.020
≤3.0	0.76	0.95	0.71
>3.0	0.94	1.26	0.82
Bilirubin (mg/dl)	P=0.007	P=0.070	P=0.330
>3.0	0.77	1.19	0.76
≤3.0	0.87	1.09	0.78
Esophageal variceal bleed	P=0.610	P=0.030	P=0.960
Yes	0.84	1.08	0.77
No	0.82	1.20	0.76
Encephalopathy	P=0.006	P=0.120	P=0.290
Yes	0.75	1.04	0.74
No	0.86	1.19	0.78
Ascites	P=0.04	P=0.006	P=0.27
Yes	0.81	1.03	0.76
No	0.90	1.31	0.87

[a] Wilcoxon P-value is shown; median values are given.

Table 4

Zum Seitenanfang