Volvulus of the sigmoid colon is associated with hypotrophy of the left lateral segment of the liver and the absence of sigmoid diverticulum

M. Barral\textsuperscript{a,b,c,*}, L. Lassalle\textsuperscript{d}, R. Dautry\textsuperscript{a}, C. Eveno\textsuperscript{b,c,e}, J. De Laveaucoupet\textsuperscript{d}, S. Maitre\textsuperscript{d}, A. Dohan\textsuperscript{a,b,c}, M. Pocard\textsuperscript{b,c,e}, P. Soyer\textsuperscript{a,b,c}

\textsuperscript{a} Department of Radiology, hôpital Cochin, 27, rue du Faubourg-Saint-Jacques, 75014 Paris, France
\textsuperscript{b} Université Paris Descartes Paris 5, Sorbonne Paris Cité, 2, rue de l’Odéon, 75006 Paris, France
\textsuperscript{c} UMR Inserm 965, 2, rue Ambroise-Paré, 75010 Paris, France
\textsuperscript{d} Department of Radiology, hôpital Antoine-Beclère, 157, rue de la Porte-de-Trivaux, 92140 Clamart, France
\textsuperscript{e} Department of Visceral Surgery, hôpital Lariboisière, Assistance publique–Hôpitaux de Paris, 2, rue Ambroise-Paré, 75475 Paris, France

**KEYWORDS**
Sigmoid diverticula;
Sigmoid volvulus;
Left lateral segment of the liver hypotrophy;
Liver volume

**Abstract**

**Purpose:** To investigate the possible relationships between sigmoid diverticula, the volume of the left lateral segment of the liver and sigmoid colon volvulus.

**Material and methods:** The presence of sigmoid diverticula was analyzed in 36 patients (24 men, 12 women; mean age, 70.77 ± 19.86 [SD] years) with sigmoid volvulus (group 1). The volumes of left lateral segment of the liver (i.e., segments 2 and 3 and further referred to as liver 1), liver 2 (i.e., segments 1, 4, 5, 6, 7 and 8), total liver volume and liver volume ratio (LVR) (i.e., [liver 1/liver 2] × 100) were calculated from abdominal CT performed distantly from the acute episode of sigmoid volvulus. Results of volumetric measurements in group 1 were compared with those of two groups of age and gender-matched control patients without hepatopathy: one patient group with sigmoid diverticula (group 2) and one group without sigmoid diverticula (group 3).
Colonic diverticulosis is most frequently observed in developed countries, where studies have confirmed the presence of diverticula in about 5 to 10% of the population before 50 years old, in 30% of those aged over 50 years, in 50% of those over 70 years and in 66% of people over 85 years of age [1]. The prevalence of sigmoid diverticula is considered as very high [2] but the actual prevalence is difficult to determine because most affected people are asymptomatic [3]. A comparison of the earliest and most recent autopsy and barium enema studies indicate that the actual prevalence is increasing over time [1]. Two main hypotheses have been suggested to explain colon diverticula formation. They include a high-pressure gradient and a weakness in a colonic wall promoted by a low fiber diet [4–6].

Sigmoid volvulus results from abnormal twisting or rotation of a portion of the sigmoid colon around its mesentry. It is generally admitted that the pathogenesis of sigmoid volvulus relates to an anatomic abnormality [7–12]. There is a long loop of redundant sigmoid colon with a narrow base of attachment of a dolichomesosigmoid [9–12]. However, the reasons why sigmoid volvulus may occur remain speculative to date. As for diverticula formation, anatomic abnormalities noted in sigmoid volvulus can be related to high-pressure gradient in the colonic lumen due to fecal overload, promoted by a low fiber diet [9–12].

Hypotrophy of the left lateral segment (LLS) of the liver is a variation that can be associated with a number of conditions, including alcoholic cirrhosis or post-necrotic cirrhosis [13–15]. However, marked hypotrophy of the LLS of the liver can be associated with apparently healthy, non-cirrhotic livers. It has been postulated that slightly decreased liver volumes can be the result of increased pressure from surrounding organs, congenital abnormality or underdevelopment, chronic segmental biliary obstruction and obstruction of the common hepatic duct [14–16].

We hypothesized that the behavior of the colonic wall in high-pressure gradient condition is different in patients with sigmoid diverticula and those with sigmoid volvulus, and that both diseases may exclude each other. We further hypothesized that hypotrophy of LLS of the liver is associated to sigmoid volvulus either because of iterative sub occlusion or as a predisposing factor.

Results: No patients with sigmoid volvulus had diverticulum. Liver 1 volume was lower in group 1 (193.8 cm³) than in group 2 (273.75 cm³) ($P = 0.0003$). Mean LVR was greater in group 2 (24.18%) than in group 1 (14.46%) ($P = 1 \times 10^{-7}$) and group 3 (18.36%) ($P = 0.003$). Mean LVR was greater in group 3 than in group 1 ($P = 0.01$). No significant differences in liver 2 volume and total liver volumes were found between the 3 groups.

Conclusion: Elasticity of colon wall associated with relative hypotrophy of left lateral segment of the liver are significantly associated with sigmoid volvulus. Further studies are needed to elucidate the pathophysiological mechanisms behind this association.

The purpose of this study was to investigate the possible relationships between sigmoid diverticula, the volume of the LLS of the liver and sigmoid volvulus.

Materials and methods

Patients

From January 2009 through December 2016, 76 patients were referred to two institutions for suspected or confirmed sigmoid volvulus. Of these, 62 patients had computed tomography (CT) of the abdomen. Thirty-six patients (24 men, 12 women) with a mean age of 70.77 ± 19.86 (standard deviation [SD]) years (range: 17–93 years) who had CT of the abdomen distant to the acute episode of sigmoid volvulus were identified and constituted the group 1 of this study. The need for informed consent was waived due to the retrospective nature of the data analysis.

Two other groups were further constituted. They both consisted of 36 patients who were randomly selected and matched for age and gender. Group 2 consisted of 36 patients (24 men, 12 women) with a mean age of 69.64 ± 20.38 (SD) years (range: 17–93 years), who had sigmoid diverticula and no sigmoid volvulus. Group 3 consisted of 36 patients (24 men, 12 women) with a mean age of 71.25 ± 19.87 (SD) years (range: 17–93 years) that had no diverticula of the sigmoid colon as evidenced on CT examination and no sigmoid volvulus. Patients in these two groups were selected when they had CT of the abdomen for conditions unrelated to the hepatobiliary system and no visible hepatic disease on CT images. Patients with prior history of hepatic disease, such as cirrhosis, fibrosis, hemochromatosis, Wilson disease or steatosis were not included in groups 2 and 3. Patients with conditions potentially affecting the liver or the biliary tree or associated with diffuse liver disease were also excluded as well as those with a known right cardiac insufficiency. Patients were included in groups 2 and 3 when they had normal hepatic blood tests, including aspartate aminotransferase, alanine transaminase, alkaline phosphatase, gamma-glutamyl transpeptidase and total bilirubin.
Computed tomography

All patients had abdominopelvic CT using a Somatom Sensation 64® (Siemens Healthcare, Forchheim, Germany) in center 1 and GE LightSpeed VCT® (GE Medical Systems, Milwaukie, WI, USA) in center 2. All CT examinations were routinely performed in the supine position with the same protocol. Patients were placed headfirst. The following scanner parameters were used: 279–450 mm field of view, 38.4 mm beam collimation (64 × 0.6 mm collimator setting), 120 peak kVp tube potential, 0.5–0.8 s gantry revolution time and 46 mm per gantry rotation table speed resulting in a beam pitch of 1.2 and 310–500 mm field of view, and 55 mm per gantry rotation table speed resulting in a beam pitch of 1.38. Online, real time, anatomy-adapted, attenuation-based tube current modulation techniques (Care Dose 4D®, Siemens Medical Solution and Smart mA, GE Healthcare) were used with a tube current set to 120–170 effective mAs. At the start of the procedure, 120 mL of non-ionic iodinated contrast material (Iomepron, Iomeron®, Bracco Imaging SpA, Milan, Italy or iopamidol, Iopamiron®, Guerbet, Roissy-Charles de Gaulle, France) were injected intravenously through a 20-Gauge catheter into an antecubital vein, at the rate of 3 mL/s by using an automated power injector. One single pass imaging set was obtained 70 s after the start of the contrast material administration. All CT examinations were performed from the hepatic dome to the lower margin of the symphysis pubis, using a cephalocaudal direction after breath hold instruction was given.

After acquisition, CT data were reconstructed at 0.6 mm thickness at 0.5 mm intervals for transverse and multiplanar reconstructions and 3D imaging. All data were stored on internal picture archiving and communication system (PACS, Directview, V12.1.5.1156, Carestream Health Inc., Rochester, NY, USA).

Image analysis

For all patients, transverse CT images were analyzed along with multiplanar and 3D images using the PACS workstation. Liver volume calculation was performed by two abdominopelvic radiologists using a combination of contour drawing, thresholding and region growing. Interpolation between the marked slices was performed semi-automatically using a linear algorithm [17,18]. The total liver volume was divided into liver 1 and liver 2. Liver 1 was defined as the LLS of the liver (i.e., segments 2 and 3). Liver 2 consisted of the right liver (i.e., segments 5, 6, 7 and 8), the left median segment (i.e., segment 4) and the caudate lobe (i.e. segment 1). The limit between liver 1 and liver 2 was defined as an imaginary boundary from the falciform ligament to the confluence of the left and middle hepatic veins at the inferior vena cava (Fig. 1) [19–22]. The inferior vena cava, the extra hepatic portion of the portal vein and the gallbladder were excluded from liver segmentation. The total liver volume was the sum of liver 1 volume and liver 2 volume. In addition, a liver volume ratio (LVR = [liver 1/liver 2] × 100) was calculated. A subset of 18 patients was randomly selected for independent assessment of reproducibility of volume measurements by the two readers.

Figure 1. Volume measurement of the left lateral segment of the liver (i.e., liver 1) in a 56-year-old woman with history of sigmoid volvulus. Abdominal CT image in the transverse plane obtained during the portal phase after intravenous administration of iodinated contrast material shows segmentation of the left lateral segment of the liver (i.e., liver 1) using a semi-automated technique. The limit between liver 1 and liver 2 was defined as an imaginary boundary from the falciform ligament to the confluence of the left and middle hepatic veins at the inferior vena cava. The left lateral segment of the liver is in grey.

Statistical analysis

Statistical analyses were performed using SPSS version 20.0, SAS version 9.2 (SAS Institute, Cary, NC, USA) and StatView (StatView 5.0, Abacus Concepts Inc, Palo Alto, CA, USA) software. Quantitative variables were presented using their mean value and standard deviation and qualitative variables were expressed as percentages. Inter-observer reproducibility of volume measurements (liver 1, liver 2, and LVR) was assessed using intraclass correlation coefficient (ICC) and 95% confidence interval (95% CI). Normality of continuous variables (liver 1 and liver 2, LVR, and age) was tested with the Shapiro-Wilk normality test. Differences between the 3 groups for total liver volume, liver 1, liver 2, LVR, and age were tested by one-way ANOVA and pairwise t-tests using the Bonferroni correction to adjust α-error level. Adjusted P values were displayed for pairwise comparisons.

Results

Inter-observer reproducibility was excellent for liver 1 volume (ICC = 0.93; 95% CI: 0.82–0.97), liver 2 volume (ICC = 0.94; 95% CI: 0.75–0.98) and LVR (ICC = 0.87; 95% CI: 0.71–0.96).

The results of the volumetric comparisons between the three groups are reported in Table 1. No patient with sigmoid volvulus (group 1) had sigmoid diverticulum. The mean liver 1 volume in group 1 (193.8 ± 86.27 [SD] cm³; range: 36.80–399.74 cm³) was lower than that in group 2 (273.75 ± 94.25 [SD] cm³; range: 75.11–526.94 cm³) (P = 0.001) (Figs. 2–4). No differences in liver 1 volume were found between group 3 (232.98 ± 81.79 [SD] cm³) and...
**Table 1** Results of colonic wall analysis and liver volume measurement.

<table>
<thead>
<tr>
<th>Patients characteristics</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>70.77±19.86</td>
<td>69.64±20.38</td>
<td>71.25±19.8</td>
<td></td>
</tr>
<tr>
<td>Presence of sigmoid diverticulum</td>
<td>0 (0/36; 0%)</td>
<td>36 (36/36; 100%)</td>
<td>0 (0/36; 0%)</td>
<td></td>
</tr>
<tr>
<td>Liver 1 volume (cm³)</td>
<td>193.8±86.27</td>
<td>273.75±94.25</td>
<td>232.98±81.79</td>
<td>0.001</td>
</tr>
<tr>
<td>Liver 2 volume (cm³)</td>
<td>1169.62±336.27</td>
<td>1164.25±285.9</td>
<td>1086.72±322.21</td>
<td>0.46</td>
</tr>
<tr>
<td>Total liver volume (cm³)</td>
<td>1363.42±369.19</td>
<td>1438.00±332.09</td>
<td>1319.70±359.39</td>
<td>0.36</td>
</tr>
<tr>
<td>Mea liver volume ratio (%)</td>
<td>14.46±0.36</td>
<td>24.18±0.46</td>
<td>18.36±0.36</td>
<td>&lt;10⁻⁶</td>
</tr>
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Group 1: patient with sigmoid volvulus; group 2: patients with sigmoid diverticula and no sigmoid volvulus; group 3: patients with no sigmoid diverticula and no sigmoid volvulus. Quantitative variables are expressed as mean±standard deviation (SD). Numbers in brackets are ranges.

*P* < 0.05 between group 1 and group 2.

**P** < 0.05 between group 2 and group 1, group 3 and group 1, and between group 2 and group 3.

Figure 2. Boxplots show comparison of the volume of the left lateral segment of the liver (liver 1) between 3 groups of patients. Group 1 corresponds to patients with sigmoid volvulus; group 2 corresponds to patients with sigmoid diverticula; and group 3 corresponds to patients without sigmoid diverticula or sigmoid volvulus. Central rectangle spans the first quartile to the third quartile. Segment inside the rectangle shows the median and "whiskers" above and below the box indicates minimum and maximum values.

865.00–2255.57 cm³ and group 3 (1319.70±359.39 [SD] cm³; range: 572.48–2242.96 cm³) (P=0.36).

**Discussion**

Our results show that sigmoid diverticula and sigmoid volvulus are not associated. Hence, we found no sigmoid diverticulum in a group of patients with sigmoid volvulus. It could thus be postulated that such colon adaptation to high intraluminal pressure depends on the patient’s colonic wall elasticity. Although these two conditions result from the same *primum movens* (i.e., an increased pressure gradient within the colonic lumen), our results suggest that the same *primum movens* has a different result. Whereas
Sigmoid diverticula thus dient lus; some

Figure 1/liver between quartile.

Thus, in patients

Figure 2

In quartile.

1, 2 and 3

(range: 572.48–2255.57 cm$^3$), in the range of previous studies [28–32]. These results are consistent with those found in the literature. Soyer et al. found a mean hepatic volume of 1.588 cm$^3$ ± 330 (SD) cm$^3$ (range: 938–2.559 cm$^3$) in a series of 100 patients without liver disease [28]. Similarly other studies reported a mean total volume of the liver of 1313.3 cm$^3$ [32], 1.493 ± 230 (SD) cm$^3$ [29], 1518 ± 353 (SD) cm$^3$ [30], and 1531 cm$^3$ in 292 patients without hepatobiliary diseases [31]. We found that liver volumes were 193.8 ± 86.27 (SD) cm$^3$, 273.75 ± 94.25 (SD) cm$^3$ and 232.98 ± 81.79 (SD) cm$^3$ (range: 36.80–526.94 cm$^3$) and liver 2 volumes were 1169.62 ± 336.27 (SD) cm$^3$, 1164.25 ± 285.9 (SD) cm$^3$ and 1086.72 ± 322.21 (SD) cm$^3$ (range: 288.85–1987.03 cm$^3$) respectively in groups 1, 2 and 3 with a LVR ranging from 14.46 to 24.18%. Abdallah et al. found an average volume of 997 ± 279 (SD) cm$^3$ for liver 2 and 242 ± 79 (SD) cm$^3$ (range: 101–490 cm$^3$) for liver 1 that on average contributed to 16% of the total liver volume, with a range of 5% to 27% [30].

In our study, we found an excellent inter-observer variability in liver volume measurement. Several studies have proven that reliable results can be obtained using different observers, equipment and software with estimated accuracy variation of ±5% [29,31,33,34]. In addition, another study found that the mean difference in total liver volume measurement between two different observers was less than 2% [35], suggesting that hepatic volume measurement is highly reproducible.
In the present study, we found that the volume of the LLS of the liver (i.e., liver 1) in patients with sigmoid volvulus was smaller than that of patients with sigmoid diverticula and no history of sigmoid volvulus. Furthermore, we found that LVR was greater in patients with sigmoid diverticula than in those without sigmoid diverticula with or without a history of sigmoid volvulus, and that patients with sigmoid volvulus had a smaller ratio than those without sigmoid diverticula and sigmoid volvulus. These observations support the hypothesis that, first, a level of hypotrophy of the LLS of the liver is necessary to allow the sigmoid colon to twist, and second, that both colonic wall elasticity and LLS of the liver hypotrophy are favoring or participating factors to sigmoid volvulus. However, it remains unclear whether LLS of the liver hypotrophy is an anatomical prerequisite or a consequence of iterative sigmoid volvulus.

In conclusion, sigmoid volvulus and sigmoid diverticula are both colonic conditions that could exclude each other, reflecting an interindividual variation of the colonic wall response to high-pressure gradient. The volume of the LLS of the liver is smaller in patients with sigmoid volvulus and this condition could facilitate the occurrence of sigmoid volvulus. Those two prerequisites could be participating factors that could explain the multifactor pathophysiology of sigmoid volvulus. Further studies are needed to elucidate the pathophysiological mechanisms behind this association.

Disclosure of interest

The authors declare that they have no competing interest.

References

