Serum albumin and platelet count but not portal pressure are predictive of death in patients with Child-Pugh A hepatitis C virus-related cirrhosis

Pierre NAHON (1), Nathalie GANNE-CARRIE (1), Françoise DEGOS (2), Karine NAHON (1), Jacques PARIES (1), Véronique GRANDO (1), Cendrine CHAFFAUT (3), Corinne NJAOPUM (2), Christos CHRISTIDIS (1), Jean-Claude TRINCHET (1), Sylvie CHEVRET (3), Michel BEAUGRAND (1)


SUMMARY

Aim — The presence of esophageal varices has been reported to be a prognostic factor in patients with compensated hepatitis viral C induced cirrhosis. We studied the prognostic value of hepatic venous pressure gradient in addition to epidemiological and clinical parameters in these patients.

Methods — Among patients with Child-Pugh A hepatitis C induced cirrhosis, prospectively followed in two Parisian centers, 100 had measurement of occluded and free hepatic venous pressures. We evaluated hepatic venous pressures as a predictive factor of death by Cox models (survival) and Fine and Gray models (liver-deaths).

Results — Median hepatic occluded pressure and gradient were 21.5 (15-24) and 13 mm Hg (9-15), respectively. The median duration of follow-up was 85 months (range: 70-112); 38 deaths or liver transplantation were registered. Hepatic venous pressure gradient was not significantly related to survival in the studied population but as a continuous variable was predictive of death from liver disease. On multivariable analysis serum albumin < 40 g/L and platelet count < 90 000 /mm³ were the only selected prognostic factors.

Conclusion — Hepatic venous pressure gradient has a limited value for assessing the prognosis of patients with Child-Pugh A hepatitis C virus induced cirrhosis; prognosis is accurately predicted by serum albumin and platelet count.

La pression portale n’est pas prédicte de la mort au cours de la cirrhose virale C Child-Pugh A

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Objectifs — La présence de varices oesophagiennes est prédicte de la mort chez les malades ayant une cirrhose virale C compensée. Nous avons étudié la valeur pronostique du gradient de pression veineuse hépatique associée à des paramètres cliniques et épidémiologiques chez ces malades.

Méthodes — Parmi les malades ayant une cirrhose virale C Child-Pugh A suivis prospectivement dans deux centres d’hépatologie, 100 ont eu une mesure des pressions veineuses hépatiques libres et occluses. Nous avons évalué la valeur pronostique sur la survie des pressions veineuses hépatiques en utilisant les modèles de Cox (survie) et Fine et Gray (mortalité spécifique).

Résultats — Les valeurs médianes de pression hépatique occluse et gradient de pression étaient respectivement de 21.5 (15-24) et 13 mm Hg (9-15). La durée de survie médiane était de 85 mois (70-112) ; 38 décès ou transplantations ont été recensés. Le gradient de pression n’était pas significativement corrélé à la survie mais était prédicte du décès en tant que variable continue chez les malades dont la mort était due à la maladie hépatique. En analyse multivariée, seules l’hypoalbuminémie < 40 g/L et la thrombopénie < 90 000/mm³ étaient prédictees du décès.

Conclusions — La mesure du gradient de pression veineuse hépatique est peu utile pour déterminer le pronostic des malades ayant une cirrhose virale C compensée. L’hypoalbuminémie et la thrombopénie sont de bons indices prédicteurs de décès.

Although often asymptomatic, patients with well compensated Child-Pugh A hepatitis C (HCV)-related cirrhosis have a high rate of severe complications including death. Natural history of compensated HCV-related cirrhosis has been clarified by prospective studies [1-4]. In these patients, death generally occurs due to complications of cirrhosis, the most frequent being hepatocellular carcinoma (HCC). Serfaty and al. [3] reported a higher rate of complications at 4 years (death in 16% of cases, HCC in 11.5% of cases) however, in this study a small percentage of patients belonged to Child Pugh B group and had advanced, even if, compensated cirrhosis. In contrast, Fattovich and al. [2] reported Child Pugh A patients with a reasonable outcome (incidence of HCC and death at 4 years were 5.6% and 7%, respectively). Unfortunately, the conditions of recruitment might have biased the results: patients were not recruited consecutively and some centers had included as little as ten patients. Moreover, it was not clear that all participating centers performed routine liver biopsy in all patients suspected of cirrhosis. The exclusion of patients with severe thrombopenia, a well known prognostic factor, in relation to absence of liver biopsy could have modified results. Degos et al. [1] tried to avoid these pitfalls: all patients with Child-Pugh A scores were included consecutively and the two involved centers performed routine hepatic biopsy in all anti-HCV positive viraeic patients using the transjugular route in patients with low portal pressure.

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platelet count and all patients had long-term follow-up with repeated ultrasonographic examinations. In this study the overall incidence of HCC and death at five years were 13.4% and 15.25%, respectively.

An individual prognosis is still difficult to assess in these patients even if some factors influencing the risk of HCC and death have been identified. These include the presence of large esophageal varices and severe thrombopenia which is supposed to be at least in part related to portal hypertension. Better knowledge of individual prognostic factors could obviously help in making important therapeutic decisions. As two important manifestations of portal hypertension seem to have a prognostic value in patients with Child-Pugh A cirrhosis, we speculated that the measurement of portal pressure by hepatic venous pressure gradient could yield additional prognostic information. This has been exemplified by the post operative outcome of patients treated surgically for HCC, most of them with HCV-related cirrhosis [5]. These authors demonstrated that hepatic venous pressure gradient (HVPG) was by itself predictive of the post-operative outcome as the mean HVPG was 13.9 mm Hg in patients with post operative decompensation versus 7.4 mm Hg in patients without. These authors introduced also the concept of “significant portal hypertension” mixing patients with esophageal varices and those with elevated portal pressure [5].

The aim of this study was to evaluate the influence of baseline HVPG and “significant portal hypertension” on survival of patients with Child-Pugh A well compensated HCV-related cirrhosis.

Materials and methods

Patients

Between January 1987 and December 1997, 555 new consecutive patients with HCV-related cirrhosis were referred to either Beaujon hospital or Jean Verdier hospitals (APHP) and fulfilled the following criteria: 1) presence of serum HCV antibodies (enzyme-linked immunosorbent assay or radio immunoblot assay); 2) absence of alcohol intake; 3) liver biopsy diagnostic of cirrhosis; 4) compensated liver disease (A status in Child-Pugh classification); 5) no evidence of HCC defined as homogenous liver on ultrasonographic liver examination and serum α-fetoprotein level under 50 ng/ml; 6) absence of co-infection by human immunodeficiency virus, hepatitis B virus, and of other chronic liver disease; 7) residence in France; 8) acceptance of a regular follow-up. Among them, initial liver biopsy was obtained via a transjugular route in 100 cases due to contraindication, refusal or failure of transparietal liver biopsy and measurement of occluded hepatic venous pressure (OHVP) and free hepatic venous pressure (FHVP), leading to calculation of HVPG, was performed. This group of 100 patients comprised the studied population.

Methods

CLINICAL AND PARACLINICAL PARAMETERS

The following initial data were retrospectively collected: gender, age, presence of esophageal varices (none, moderate = grade 1, large = grade 2 or 3) or gastric varices, serum bilirubin and albumin levels, prothrombin time, serum alanine aminotransferase activity, platelet count, OHVP, FHVP and HVPG (tables I and II).

HEMODYNOmic STUDY

After an overnight fast, patients were placed in the supine position for at least 2 hours. Arterial pressure was monitored with an external sphygmomanometer (Dinamap, Critikon, Tampa, FL) and heart rate was monitored by continuous electrocardiographic tracing. The OHVP and FHVP were measured with a balloon catheter as previously described. [6]. A balloon catheter was introduced into an hepatic vein via the jugular vein. With the catheter 2 cm within an hepatic vein, FHVP was measured using a strain-gauge transducer previously calibrated with a mercury manometer. The zero reference point was arbitrarily set 5 cm below the sternum. With continuous monitoring of pressure, the balloon was expanded with 2 ml of saline containing hypeaque to obstruct the vein and the OHVP was recorded. Confirmation of the occluded position was obtained by gently injecting a small amount (2-3 ml) of contrast agent through the catheter to demonstrate retention of the dye in the occluded portion of the hepatic vein. Deflation and redistention of the balloon permitted checking OHVP and FHVP as many times as desirable. Three measurements of OHVP and FHVP were taken for each patient and mean value was recorded.

Follow-up

In the setting of HCC screening, patients were prospectively evaluated every 6 months by routine evaluation including physical examination, liver ultrasonography and determination of serum α-fetoprotein level. If HCC was suspected on increasing levels of α-fetoprotein or ultrasound findings, liver computed tomodensitometry and/or magnetic resonance imaging and/or guided biopsy were performed to confirm diagnosis according to the Barcelona conference criteria [7].

STATISTICAL ANALYSIS

The end-point was overall survival whatever the cause of death, liver transplantation and cause-specific-mortality differentiating liver-related deaths (due to liver failure, esophageal variceal bleeding, or HCC) and non-liver-related deaths.

The follow-up ended at the date of death or liver transplantation or was censored at the last recorded visit before January 2001. The prognostic value of initial clinical, biochemical and hemodynamic parameters for overall survival was estimated by using the Kaplan-Meier method [8] and Cox model [9]. Prognostic analyses for cause-specific mortality were based on competing risk time-failure methods (non-liver-related mortality was considered as a competing risk event for liver-related death). Estimation of cumulative cause-specific incidence curves was used and curves were compared through the use of Gray test [10] while multivariable analyses were based on the Fine and Gray model [11]. All tests were two-sided, with a p-value of 0.05 or less denoting statistical significance. All statistical analyses were performed on the SAS 8.2 (SAS, Inc, Cary, NC) and Splus 2000 (Math Soft, Inc, Seattle, WA) software packages.

Results

Initial characteristics of patients

The study population was composed of 100 patients with HCV-related Child A cirrhosis who had hepatic venous pressure measurements at time of initial liver biopsy. Indication of transjugular route of liver biopsy was hemostatic abnormalities in 78 patients (thrombopenia < 80 000/mm³ in 66 cases, increased bleeding time > 10 minutes by Ivy method in 5 cases, decreased prothrombin activity < 60% in 3 cases, increased TCA > 1.5 the upper limit of control in 4 cases), and anti-coagulant or anti-agregant treatment in 8 cases. In other cases, hepatic vein catheterization was decided because of peri-hepatic ascites detected only by ultrasonography (N = 2), refusal or failure of transparietal route (N = 12).

Among these patients, 54 were men. Initial median values (25-75th percentiles) for age, albumin, platelet count, prothrombin activity, serum alanine aminotransferase activity were 58 years (48.0-64.5), 40 g/L (36-43), 90 000/mm³

ABBREVIATIONS

EV : Esophageal varices
FHVP : Free hepatic venous pressure
GV : Gastric varices
HCC : Hepato-cellular carcinoma
HCV : Hepatitis C virus
HVPG : Hepatic venous pressure gradient
OHVP : Occluded hepatic venous pressure
Portal pressure and survival in HCV cirrhosis

Initial median values (25-75th percentiles) of OHVP and HVPG were 21.5 (15–24) and 13 (9–15) mm Hg, respectively. Among eighty-one patients who underwent upper gastro-intestinal endoscopy at enrollment, forty-six (57%) had esophageal varices and 5 (6%) had gastric varices. Initial characteristics distribution is shown in tables I and II. Significant correlation was found between esophageal varices and HVPG value at time of inclusion (median HVPG were 11 and 14 mm Hg respectively in patients without and with varices, P = 0.0066, Wilcoxon rank sum test) and between platelet count and HVPG (Spearman correlation coefficient = -0.28, P = 0.0048, figure 1).

During follow-up, prophylactic treatment to prevent digestive hemorrhage due to portal hypertension was performed in 34 patients: non cardioselective β-blockers (N = 32), and/or sclerotherapy or band ligation (N = 7). Six patients had portal hypertension-related hemorrhage, leading to death in 3 cases. Forty-four patients were treated during the study with recombinant alpha-2a or alpha-2b interferon (mean dose 427 millions units). Seven were responders at the end of treatment and only 3 of them had sustained virological response.

Outcome and prognostic factors

Median time of follow-up was 85 (70-112) months. On January 1st 2001, 38 were recorded as dead (including 6 who underwent liver transplantation for hepatic failure) and 21 developed HCC. Median time of survival was 3 200 days, survival rate at 3 and 5 years were respectively 85.7 and 70.6%. Death was due to liver disease in 60% of cases (HCC N = 12, hepatic failure N = 8, digestive hemorrhage N = 3), and due to extra-hepatic causes (N = 10) or unknown causes (N = 5, which were included as 3 liver-related deaths and 2 extra-hepatic deaths according to the results of cause-specific mortality in our population).

At univariable analyses, HVPG was not significantly related to survival (figure 2) among the studied population. When predictive value of HVPG as a continuous variable was analyzed among patients who died of liver-related complications (according to Cox model), we found a significant relation between HVPG and liver-related death (hazard ratio = 1.161, 95% CI = 1.009-1.337, P = 0.037). Portal hypertension defined as evidence of grade 2 or 3 esophageal varices or gastric varices and/or HVPG ≥ 13 mm Hg was associated either to overall mortality (hazard ratio 2.38, 95% CI: 1.09-5.20, P = 0.02) (figure 3) and to liver-specific mortality (P = 0.02). In addition, liver-specific mortality was related to albuminemia < 40 g/L (P = 0.005) and platelet count below 90 000/mm3 (P = 0.02) (tables I and II). At multivariate analysis, only hypoalbuminemia < 40 g/L (P = 0.040) and platelet count (P = 0.048) had independent prognostic value. Figure 4 shows the estimated cumulative incidence of death due to liver disease according to serum albumin level and platelet count. Portal hypertension had no prognostic value for the occurrence of HCC (data not shown).

### Table I. – Distribution and pronostic value of baseline clinical and biological data in 100 patients with Child-Pugh A hepatitis C-related cirrhosis.

<table>
<thead>
<tr>
<th>Patients (N = 100)</th>
<th>Total deaths (N = 38)</th>
<th>HR* (95% CI)</th>
<th>P</th>
<th>Liver-deaths (N = 28)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Male</td>
<td>54</td>
<td>21</td>
<td>1.00</td>
<td>0.96</td>
<td>1.00</td>
</tr>
<tr>
<td>Female</td>
<td>46</td>
<td>17</td>
<td>0.98 (0.51-1.88)</td>
<td>0.37</td>
<td>0.99</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
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<tr>
<td>&lt; 58</td>
<td>49</td>
<td>17</td>
<td>1.00</td>
<td>0.75</td>
<td>0.02</td>
</tr>
<tr>
<td>≥ 58</td>
<td>51</td>
<td>21</td>
<td>1.34 (0.70-2.53)</td>
<td>1.00</td>
<td>0.98</td>
</tr>
<tr>
<td>Platelets (10^3/mm³)</td>
<td></td>
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</tr>
<tr>
<td>&lt; 90</td>
<td>44</td>
<td>17</td>
<td>1.00</td>
<td>0.01</td>
<td>0.05</td>
</tr>
<tr>
<td>≥ 90</td>
<td>53</td>
<td>20</td>
<td>0.90 (0.47-1.72)</td>
<td>0.89</td>
<td>0.76</td>
</tr>
<tr>
<td>Prothrombin index (%)</td>
<td></td>
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<td></td>
<td>---</td>
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<tr>
<td>&lt; 76</td>
<td>47</td>
<td>18</td>
<td>1.00</td>
<td>0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>≥ 76</td>
<td>53</td>
<td>20</td>
<td>0.95 (0.50-1.81)</td>
<td>0.01</td>
<td>0.28</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td></td>
<td></td>
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<td></td>
<td>---</td>
</tr>
<tr>
<td>&lt; 40</td>
<td>41</td>
<td>22</td>
<td>1.00</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>≥ 40</td>
<td>48</td>
<td>12</td>
<td>0.39 (0.19-0.80)</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>ALT (× N)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>&lt; 2.5</td>
<td>45</td>
<td>22</td>
<td>0.42 (0.22-0.82)</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>≥ 2.5</td>
<td>54</td>
<td>15</td>
<td>1.00</td>
<td>0.04</td>
<td>0.04</td>
</tr>
</tbody>
</table>

**Abbreviations**

* hazard ratio of death, ALT: Alanine aminotransferase, N: upper normal limit

(71000-126000), 76% (19–100), and 2.5 times the upper limit of normal value (1.9-4.1), respectively. Initial median values (25-75th percentiles) of OHVP and HVPG were 21.5 (15–24) and 13 (9–15) mm Hg, respectively. Among eighty-one patients who underwent upper gastro-intestinal endoscopy at enrollment, forty-six (57%) had esophageal varices and 5 (6%) had gastric varices. Initial characteristics distribution is shown in tables I and II. Significant correlation was found between esophageal varices and HVPG value at time of inclusion (median HVPG were 11 and 14 mm Hg respectively in patients without and with varices, P = 0.0066, Wilcoxon rank sum test) and between platelet count and HVPG (Spearman correlation coefficient = -0.28, P = 0.0048, figure 1).

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Outcome and prognostic factors

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Discussion

After many controversies [12-15] it has been shown that OHVP accurately reflects portal pressure in HCV-related cirrhosis as in other causes of micronodular cirrhosis, especially alcoholic cirrhosis [16, 17]. The main goal of the present study was to define the overall prognostic value of HVPG at time of diagnosis of Child-Pugh A HCV-related cirrhosis as a marker of severity of liver disease. The overall prognosis of our population may have been improved and the prognostic value of HVPG lowered by the management of liver disease including esophageal variceal bleeding prevention by β-blocker or band ligation. However, this pitfall is common with all similar studies [18]. Moreover, as our patients were included in the early era of antiviral treatments, only three sustained virological response were observed and the impact of anti-viral therapy on portal pressure and the outcome is therefore necessarily reduced.

Our results show that portal pressure has a low statistically non-significant prognostic value and that “significant portal hypertension” mixing the presence of varices and HVPG ≥ 13 mm Hg gives a more accurate prognostic indication but does not appear to be superior to hypoalbuminemia or thrombopenia which bear independent prognostic value. Furthermore, we show that platelet count was only weakly related to portal pressure, a fact that could give rise to different hypotheses concerning the mechanisms of its prognostic significance.

In patients with alcoholic cirrhosis, several studies assessed the prognostic value of HVPG. Some found that the prediction of survival based on the Child-Pugh scoring system was significantly improved by adding hemodynamic data obtained from

### Table II. – Distribution and pronostic value of baseline hemodynamic data in 100 patients with Child-Pugh A hepatitis C-related cirrhosis.

 Répartition et valeur pronostique des paramètres hémodynamiques initiaux chez 100 patients ayant une cirrhose virale C Child-Pugh A.

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Total deaths</th>
<th>HR* (95% CI)</th>
<th>P</th>
<th>Liver-deaths</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 10</td>
<td>N = 38</td>
<td></td>
<td></td>
<td>N = 28</td>
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<tr>
<td>Esophageal varices (EV)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>No or grade 1</td>
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<td>20</td>
<td>1.00</td>
<td>16</td>
<td></td>
<td></td>
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<tr>
<td>Grade 2 or 3</td>
<td>34</td>
<td>10</td>
<td>1.75 (0.81-3.76)</td>
<td>7</td>
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<td></td>
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<tr>
<td>Gastric varices (GV)</td>
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<tr>
<td>Absent</td>
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<td>29</td>
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<td></td>
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</tr>
<tr>
<td>Present</td>
<td>5</td>
<td>1</td>
<td>0.55 (0.07-4.04)</td>
<td>1</td>
<td></td>
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<tr>
<td>OHVP (mm Hg)</td>
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<td></td>
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<tr>
<td>&lt; 21.5</td>
<td>50</td>
<td>16</td>
<td>1.00</td>
<td>11</td>
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<td></td>
</tr>
<tr>
<td>≥ 21.5</td>
<td>50</td>
<td>22</td>
<td>1.63 (0.85-3.10)</td>
<td>17</td>
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<tr>
<td>HVPG (mm Hg)</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 10</td>
<td>27</td>
<td>9</td>
<td>1.00</td>
<td>5</td>
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<tr>
<td>≥ 10</td>
<td>73</td>
<td>29</td>
<td>1.30 (0.62-2.75)</td>
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<tr>
<td>HVPG, mm Hg</td>
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<tr>
<td>&lt; 12</td>
<td>39</td>
<td>13</td>
<td>1.00</td>
<td>9</td>
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<tr>
<td>≥ 12</td>
<td>61</td>
<td>25</td>
<td>1.42 (0.73-2.77)</td>
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<tr>
<td>HVPG (mmHg) Continuous variable</td>
<td>1.161 (1.009-1.337)</td>
<td>28</td>
<td>0.037</td>
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<tr>
<td>(Grade 2 or 3 EV) × (GV) × (HVPG ≥ 13)</td>
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<td></td>
</tr>
<tr>
<td>Absent</td>
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<td>8</td>
<td>1.00</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>67</td>
<td>30</td>
<td>2.38 (1.09-5.20)</td>
<td>23</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations**

* hazard ratio of death, OHVP: occluded hepatic pressure, HVPG: hepatic venous pressure gradient.
Portal pressure and survival in HCV cirrhosis

hepatic venous catheterization. Conversely, a recent study showed that measurement of HVPG is not helpful in the prognostic assessment in a selected group of patients with severe alcoholic cirrhosis [18]. Despite the fact that the predictive value of portal pressure on survival remains controversial [19-21], the lowering of HVPG by pharmacological treatments improves survival without variceal hemorrhage [19-24] and a high HVPG during hemorrhage has been shown to be predictive of re-bleeding and death [24-26]. Due to the low number of hemorrhagic events in our population, we were unable to study the prognostic value of HVPG on variceal hemorrhage.

The lack of prognostic value of HVPG measurement in our patients suggests different explanations. Firstly, portal hypertension was not by itself an important cause of death in our patients as only three died from variceal bleeding. This low rate is probably due to the fact that an active prevention of variceal bleeding was carried out. Most of our patients with varices were treated either by band ligation, sclerotherapy, or \( \beta \)-blockers as primary prevention of bleeding, the choice of the method depending on the size of the varices and individual preference of the clinician in charge. The overall policy proved to be successful as previously suggested [27]. Secondly, a lowering of portal pressure due to significant porto-systemic shunting could occur more frequently in HCV-related cirrhosis than in alcoholic cirrhosis. The chronic and slow progression in HCV chronic hepatitis allows progressive development of porto-systemic anastomosis and lowering of portal pressure, HVPG being as a whole lower than in alcoholic cirrhosis. This could also explain why some patients had esophageal varices despite a moderately elevated portal pressure. Finally, mortality in patients with HCV cirrhosis is

![Fig. 1](image1.png)

**Fig. 1** – Correlation between platelet count and hepatic venous pressure gradient at enrollment in 100 patients with Child-Pugh A hepatitis C-related cirrhosis.

**Corrélation entre le taux de plaquettes et le gradient de pression hépatique veineuse chez 100 patients ayant une cirrhose virale C Child-Pugh A.**

![Fig. 2](image2.png)

**Fig. 2** – Survival according to the median value of hepatic venous pressure gradient (threshold = 13 mm Hg) at enrollment in 100 patients with Child-Pugh A hepatitis C-related cirrhosis (Kaplan-Meier).

**Survie selon la médiane de la pression hépatique veineuse (seuil = 13 mmHg) à l’inclusion de 100 patients ayant une cirrhose virale C Child-Pugh A (méthode de Kaplan-Meier).**

![Fig. 3](image3.png)

**Fig. 3** – Survival according to the presence of portal hypertension assessed either by increased hepatic venous pressure gradient \( \geq 13 \) mm Hg, and/or by the presence of grade II or III esophageal varices and/or gastric varices at enrollment in 100 patients with Child-Pugh A hepatitis C-related cirrhosis (Kaplan-Meier).

**Survie selon la présence d’hypertension portale confirmée soit par une augmentation du gradient de la pression hépatique (13 mmHg) et/ou par la présence de varices œsophagiennes de stade II ou III et/ou de varices gastriques à l’inclusion de 100 patients ayant une cirrhose virale C Child-Pugh A (méthode de Kaplan-Meier).**

![Fig. 4](image4.png)

**Fig. 4** – Estimated cumulative incidence of death due to liver disease according to serum albumin level and platelet count in 100 patients with Child-Pugh A hepatitis C-related cirrhosis (non-parametric estimate of cumulative incidence function).

**Incidence cumulée de l’incidence de décès par maladie hépatique selon le taux d’albumine et le taux de plaquettes chez 100 patients ayant une cirrhose virale C Child-Pugh A (estimation non paramétrique).**
mainly related to HCC a condition which is not directly influenced by portal hypertension.

We conclude therefore that the measurement of HVPG is of little help for establishing a prognosis in patients with Child-Pugh A HCV-related cirrhosis. The presence of varices adds more information about the risk of hemorrhage or even overall prognosis. In this study more useful information with respect to prognosis was given for two simple blood parameters, platelet count and serum albumin level [28]. This result is consistent with recent observations showing that the severity of liver disease and not the manifestations of decomposition predicts survival (MELD studies) [29].

REFERENCES