ABO blood group and the risk of CHB patients with decompensated cirrhosis: a case-control study

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Short Report

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Abstract

Objective: ABO blood group, a genetic marker of blood, has been shown to be associated with risk of cardiovascular disease and several malignancies. The study aimed to assess whether there was associated with hepatitis B decompensated cirrhosis.

Results: After adjusting for age, hepatitis B virus (HBV)-DNA nucleic acid (HBV-DNA), blood group A was an independent risk factor for decompensated cirrhosis compared to patients with type O. Age after multivariate stratified analysis showed that the risk of decompensated cirrhosis was also significantly higher in patients with aged 55 years or older with blood group A compared to blood group O. No significant differences were found in the severity of laboratory indicators in patients with decompensated cirrhosis among different ABO blood groups. Additionally, there was no significant difference in the survival rate of decompensated cirrhosis during the 12-month observation period among different ABO blood groups. Taken together, these findings indicated that the independent risk factor of developing decompensated cirrhosis was associated with A blood group, age and HBV-DNA levels in CHB patients.

1. Introduction

The World Health Organization reports that an estimated 240 million people with hepatitis B virus (HBV) infection worldwide are living with chronic hepatitis B (CHB). Approximately 2–4% of patients each year can develop compensated cirrhosis without effective treatment, and 1.5–4% of patients with cirrhosis each year can further develop decompensated cirrhosis (symptoms such as ascites, hepatic encephalopathy, and bleeding from gastrointestinal varices), resulting in repeated hospitalizations, severe loss of quality of life, and even death. As a result of cirrhosis, hepatocellular carcinoma (HCC) occurs in about 3–6% of the patients\textsuperscript{[1, 2]}. In addition, patients with decompensated cirrhosis have a higher rate of liver transplantation, mortality and HCC, and a poorer prognosis\textsuperscript{[3, 4]}. The population of China has a high prevalence of HBV infection, and nearly 80% of HCC cases are related to chronic HBV infection, of which about 60%-90% occur in patients with cirrhosis\textsuperscript{[5]}. The ABO blood group system is the first genetic polymorphism discovered in humans, and is divided into A, B, AB and O blood groups. They are located on the surface of red blood cells and play an important role in the physiology and pathology of the cells\textsuperscript{[6]}. The ABO blood group system is of great importance in the study of different diseases. Dependence on the function of blood group structures can link blood groups to disease and health\textsuperscript{[6]}. Today, ABO blood type has been found to be associated with the risk of cardiovascular disease and a variety of malignancies, including stomach cancer, pancreatic cancer, epithelial ovarian cancer and skin cancer\textsuperscript{[7–10]}. Two recent genome-wide association studies (GWAS) suggest that ABO blood group antigens may influence systemic inflammatory status, single nucleotide polymorphisms (SNPs) in the ABO locus are associated with two serum markers of inflammation: tumor necrosis factor alpha (TNF-\(\alpha\)) and soluble intercellular adhesion molecule 1 (sICAM-1)\textsuperscript{[11–13]}. Enhanced expression of TNF-\(\alpha\) is associated with liver inflammation and hepatocarcinogenesis\textsuperscript{[14]}. Plasma sICAM-1
levels are associated with liver disease activity\textsuperscript{[15]}. All these adhesion molecules are important mediators of the systemic inflammatory response and are essential for immune cell recruitment. Compensated cirrhosis is an asymptomatic disease state with histological features based on the extent and pattern of fibrous scar tissue in the liver. Cirrhosis is associated with a multifaceted state of immune dysfunction, cirrhosis-associated immune dysfunction (CAID), which involves immunodeficiency and persistent activation of pro-inflammatory immune cells. CAID affects innate and adaptive as well as cellular and non-cellular mechanisms of the immune system\textsuperscript{[16]}. Systemic inflammation is a consequence of this immune stimulation and leads to hemodynamic disturbances and portal hypertension, accelerating the development of decompensation and organ failure\textsuperscript{[17–18]}. Therefore, there may be an association between ABO blood group and the development of decompensated cirrhosis. In addition, the relationship of ABO blood groups and hepatitis B-associated hepatocellular carcinoma had been reported in previous studies\textsuperscript{[19]}. However, there is little literature on whether there is an association with decompensated cirrhosis. Therefore, the aim of this study was to assess the association between ABO blood group and the development of decompensated cirrhosis and its short-term overall survival (OS) in patients with CHB.

2. Materials and Methods

2.1 Individuals data

Based on admission diagnosis and discharge diagnosis\textsuperscript{[20]}, 326 eligible patients with chronic hepatitis B virus (HBV) infection were enrolled from the First Affiliated Hospital, Zhejiang University School of Medicine between January 2017 and December 2017. Of these, 125 patients were HBV-associated decompensated cirrhosis, and 201 patients were chronic hepatitis B (CHB) infection without cirrhosis. The clinical and laboratory data including gender, age, medical history, ABO blood group and other relevant laboratory indices (bilirubin, albumin and prothrombin time etc.) before treatment after admission were collected from the enrolled patients. The populations were divided into case group (125 cases) and control group (201 cases) according to whether decompensated cirrhosis occurred or not.

2.2 Patient follow-up

Patient follow-up information was obtained through follow-up hospital records, or by contacting the patient's family. The observation period was one year after admission to the hospital. Overall survival (OS) was defined as the time interval between the date of diagnosis and the last follow-up visit or death due to decompensated cirrhosis. This study was approved by the Medical Ethical Committee of the First Affiliated Hospital, Zhejiang University School of Medicine in the study.

2.3 Statistical analysis

The relationship between blood group and decompensated cirrhosis was analyzed using a multivariate logistic regression model, and adjusted advantage ratios (AOR) and 95% confidence intervals (CI) were calculated by adjusting for possible confounding effects between variables. One-way ANOVA was used for comparison of relevant laboratory indicators among ABO blood groups in patients with CHB.
decompensated cirrhosis. Survival analysis was performed using the Kaplan-Meier method, and differences between survival curves were performed using the log-rank test. All data were statistically analyzed using SPSS 26.0 software, and all tests were two-tailed tests, with \( P \) values < 0.05 considered as a significance, and were plotted using GraphPad Prism 8.0.1 software.

3. Results

3.1 Basic data of patients with CHB-associated decompensated cirrhosis

As expected, the majority of CHB patients were male (76.1%), while the proportion of men with decompensated cirrhosis (75.2%) was also much higher than the proportion of women (24.8%). Patients with decompensated cirrhosis were older than controls, with a mean age ± standard deviation of 53.344 ± 11.902 years for patients with decompensated cirrhosis and 42.328 ± 9.871 years for controls (\( P < 0.001 \)). The proportion of HBV-DNA > 105 copies/mL was lower in cases than in controls (Table 1).

### Table 1
Basic information of CHB patients with decompensated cirrhosis

<table>
<thead>
<tr>
<th>factors</th>
<th>Cases</th>
<th>Controls</th>
<th>single-factor regression</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( N = 125 )(%)</td>
<td>( N = 201 )(%)</td>
<td>OR(95%CI)</td>
<td></td>
</tr>
<tr>
<td>sex</td>
<td></td>
<td></td>
<td></td>
<td>0.851</td>
</tr>
<tr>
<td>male</td>
<td>94(75.2)</td>
<td>153(76.1)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>female</td>
<td>31(24.8)</td>
<td>48(23.9)</td>
<td>1.051(0.625–1.767)</td>
<td></td>
</tr>
<tr>
<td>age(year)</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&lt;55</td>
<td>68(54.4)</td>
<td>127(86.6)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>( \geq 55 )</td>
<td>57(45.6)</td>
<td>27(13.4)</td>
<td>5.402(3.158–9.241)</td>
<td></td>
</tr>
<tr>
<td>HBV-DNA(IU/ml)</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&lt;10^5</td>
<td>95(79.2)</td>
<td>68(37.4)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>( \geq 10^5 )</td>
<td>25(20.8)</td>
<td>114(62.6)</td>
<td>0.157(0.092-0.268)</td>
<td></td>
</tr>
</tbody>
</table>

OR, advantageous ratio; CI, confidence interval; HBV-DNA, Hepatitis B virus nucleic acid.

3.2 ABO blood type and risk of developing decompensated cirrhosis

Blood type A was more distributed among cases of decompensated cirrhosis than that of among CHB controls (43.9% vs 28.4%), unlike the distribution of O, AB, and B blood types among cases and controls.
The unadjusted preponderance ratio for blood type A associated with risk of decompensated cirrhosis was 1.895 (95% CI, 1.101–3.259) compared with patients with blood type O. In multivariate logistic regression, the adjusted superiority ratio was 2.384 (95% CI, 1.227–4.629) after adjustment for age and HBV-DNA (Table 2). We further assessed whether the association between ABO blood group and risk of decompensated cirrhosis differed by stratification of other known risk factors, including age, and HBV-DNA level. A higher risk was observed in patients with aged ≥ 55 years for blood type A compared with those for O blood type (Table 3).

<table>
<thead>
<tr>
<th>Table 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk factors of ABO blood group and CHB patients with decompensated cirrhosis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ABO Blood group</th>
<th>Cases</th>
<th>Controls</th>
<th>Single-factor regression analysis</th>
<th>Multi-factor regression analysis*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 125(%)</td>
<td>N = 201(%)</td>
<td>OR(95%CI)</td>
<td>P</td>
</tr>
<tr>
<td>O</td>
<td>37(29.6)</td>
<td>74(36.8)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>54(43.2)</td>
<td>57(28.4)</td>
<td>1.895(1.101–3.259)</td>
<td>0.021</td>
</tr>
<tr>
<td>B</td>
<td>25(20.0)</td>
<td>56(27.9)</td>
<td>0.893(0.483–1.651)</td>
<td>0.718</td>
</tr>
<tr>
<td>AB</td>
<td>9(7.2)</td>
<td>14(7.0)</td>
<td>1.286(0.509–3.245)</td>
<td>0.595</td>
</tr>
</tbody>
</table>

AOR, adjusted advantage over; CI, confidence interval; Cases: patients with decompensated cirrhosis; Controls: patients with CHB. *Logistic regression analysis adjusted for age and HBV-DNA.
Table 3
ABO blood group and risk of cirrhotic decompensation in patients with chronic hepatitis B

<table>
<thead>
<tr>
<th>factors</th>
<th>ABO Blood group</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>O</td>
<td>A</td>
<td>B</td>
<td>AB</td>
</tr>
<tr>
<td>age(year) &lt;55</td>
<td>19/59</td>
<td>27/52</td>
<td>16/50</td>
<td>6/14</td>
</tr>
<tr>
<td>AOR(95%CI)</td>
<td>1</td>
<td>1.693</td>
<td>0.790</td>
<td>1.259</td>
</tr>
<tr>
<td>P</td>
<td>0.201</td>
<td>0.593</td>
<td>0.717</td>
<td></td>
</tr>
<tr>
<td>≥ 55</td>
<td>18/16</td>
<td>27/5</td>
<td>9/6</td>
<td>3/0</td>
</tr>
<tr>
<td>AOR(95%CI)</td>
<td>1</td>
<td>4.670</td>
<td>1.237</td>
<td>/</td>
</tr>
<tr>
<td>P</td>
<td>0.019</td>
<td>0.755</td>
<td>/</td>
<td></td>
</tr>
<tr>
<td>HBV DNA(IU/ml) &lt;10^5</td>
<td>29/25</td>
<td>38/17</td>
<td>19/23</td>
<td>9/3</td>
</tr>
<tr>
<td>AOR(95%CI)</td>
<td>1</td>
<td>2.086</td>
<td>0.834</td>
<td>3.047</td>
</tr>
<tr>
<td>P</td>
<td>0.073</td>
<td>0.67</td>
<td>0.129</td>
<td></td>
</tr>
<tr>
<td>≥ 10^5</td>
<td>7/43</td>
<td>13/33</td>
<td>5/27</td>
<td>0/11</td>
</tr>
<tr>
<td>AOR(95%CI)</td>
<td>1</td>
<td>2.920</td>
<td>1.450</td>
<td>/</td>
</tr>
<tr>
<td>P</td>
<td>0.088</td>
<td>0.617</td>
<td>/</td>
<td></td>
</tr>
</tbody>
</table>

AOR, Adjusted advantage over; CI, confidence interval

Logistic regression analysis adjusted for age and HBV DNA

3.3 ABO blood group and severity of decompensated cirrhosis

Univariate analysis showed no significant difference in laboratory indices such as aspartate aminotransferase (AST), alanine transaminase (ALT), albumin (Alb), total bilirubin (TB), international normalized ratio (INR), model of end-stage liver disease score (MELD) in patients with decompensated cirrhosis with different ABO blood groups (P > 0.005) (Table 4).
Table 4
ABO blood group and severity of decompensated cirrhosis

<table>
<thead>
<tr>
<th>factors</th>
<th>ABO blood group</th>
<th>O(n = 37)</th>
<th>A(n = 54)</th>
<th>B(n = 25)</th>
<th>AB(n = 9)</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST(U/L)</td>
<td></td>
<td>63.649 ±</td>
<td>87.611 ±</td>
<td>52.640 ±</td>
<td>46.667 ±</td>
<td>2.078</td>
<td>0.107</td>
</tr>
<tr>
<td></td>
<td></td>
<td>54.077</td>
<td>89.965</td>
<td>49.075</td>
<td>18.193</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALT(U/L)</td>
<td></td>
<td>61.892 ±</td>
<td>98.352 ±</td>
<td>68.360 ±</td>
<td>40.111 ±</td>
<td>0.998</td>
<td>0.396</td>
</tr>
<tr>
<td></td>
<td></td>
<td>93.868</td>
<td>155.396</td>
<td>112.527</td>
<td>26.088</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alb(g/L)</td>
<td></td>
<td>31.276 ±</td>
<td>32.407 ±</td>
<td>34.170 ±</td>
<td>32.434 ±</td>
<td>1.089</td>
<td>0.356</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6.55</td>
<td>5.705</td>
<td>6.843</td>
<td>6.206</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TB(µmol/L)</td>
<td></td>
<td>99.487 ±</td>
<td>113.556 ±</td>
<td>54.800 ±</td>
<td>33.000 ±</td>
<td>1.894</td>
<td>0.134</td>
</tr>
<tr>
<td></td>
<td></td>
<td>139.876</td>
<td>148.225</td>
<td>66.588</td>
<td>29.669</td>
<td></td>
<td></td>
</tr>
<tr>
<td>INR</td>
<td></td>
<td>1.460 ±</td>
<td>1.573 ±</td>
<td>1.457 ±</td>
<td>1.453 ±</td>
<td>1.033</td>
<td>0.381</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.289</td>
<td>0.447</td>
<td>0.293</td>
<td>0.205</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MELD</td>
<td></td>
<td>11.734 ±</td>
<td>13.596 ±</td>
<td>11.456 ±</td>
<td>9.780 ±</td>
<td>1.28</td>
<td>0.284</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.979</td>
<td>8.060</td>
<td>5.032</td>
<td>4.96</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AST, glutamic aminotransferase; ALT, glutamic alanine aminotransferase; Alb, albumin; TB, total bilirubin; INR, international normalized ratio; MELD, model for end-stage liver disease score

3.4 ABO blood groups and survival differences in decompensated cirrhosis

During the 12-month observation period, survival time data were collected from 125 patients with decompensated cirrhosis. At the termination of the observation, 108 patients survived, with a mortality rate of 13.6%, and there was no significant difference between any of the different ABO blood groups ($P = 0.683$) (Fig. 1).

4. Discussion

In this study, the CHB patients with type A had a significantly higher risk of developing decompensated cirrhosis than that of the CHB patients with type O, especially in the patients with older than 55 years. To our knowledge, this is the first study to demonstrate an association between ABO blood group and the risk of developing decompensated cirrhosis in patients with CHB. However, several previous studies have shown an association between ABO blood group and liver diseases$^{[21,22]}$, so the finding of an association between ABO blood group and decompensated cirrhosis is not surprising. In addition no effect of ABO blood group has been found on the clinical severity and short-term survival time of patients with decompensated cirrhosis.

ABO blood groups were associated with the levels of several important cytokines that were known to be associated with the development of decompensated cirrhosis, including TNF-α, sICAM-1, E-selectin, and P-selectin$^{[11-13,23,24]}$. TNF is a pleiotropic cytokine involved not only in apoptosis but also in
inflammation, hepatocyte protection, and proliferation. Polymorphisms of TNF-α is associated with risk of advanced liver fibrosis\[^{25}\]. Although the relationship between ABO blood group and TNF-α level remains unclear, ABO blood group is associated with E-selectin that is positively induced by TNF-α\[^{26,27}\]. Another GWAS reported ABO as the major locus of serum soluble E-selectin levels\[^{24}\]. ICAM-1 belongs to the immunoglobulin superfamily and plays an important role in the regulation of immune responses, especially in antigen presentation mechanisms. It has been reported that sICAM-1 levels are associated with liver disease activity and the development of cirrhosis\[^{28}\]. There is growing evidence that ABO blood groups are highly and significantly associated with variation of many biomarkers. A recent large-scale genomic study has shown that P-selectin levels are also associated with ABO gene variation and that this association is caused by the A1 allele of the ABO blood group\[^{13}\]. P-selectin has an important role in inflammatory processes, tumor formation and metastasis, which is a member of the selectin family of adhesion molecules and is mainly expressed on the surface of platelets and endothelial cells. These suggest a possible association of ABO blood group with platelets. Platelets are a key factor in inflammation and essential for liver regeneration\[^{29}\]. Platelet count has been considered as a screening tool for decompensated cirrhosis\[^{30}\]. Thus, platelets may mediate another possible inflammatory pathway linking ABO blood type to risk of decompensated cirrhosis.

The ABO blood group motif is not associated with genes encoding ICAM, E-selectin, and P-selectin. However, ABO gene product-associated glycosylation may affect the shedding/cleavage of these biomarkers from the endothelium, possibly through glycosylation of P-selectin, E-selectin, and ICAM-1. Glycosylation can also affect the clearance of P-selectin, E-selectin, and sICAM-1 from the blood\[^{31}\]. Reduced cleavage of adhesion molecules by endothelial cells associated with the A allele means more adhesion molecules on endothelial cells and increased adhesion and inflammation\[^{11}\]. In conclusion, many studies suggest that ABO blood group A may increase the risk of decompensated cirrhosis by associating with several inflammatory pathways.

Non-O blood group reported to be an independent risk factor for progression of liver fibrosis in HCV infection\[^{32}\]. These suggest an association between ABO blood type and progression of liver inflammation and fibrosis in patients with CHB. In addition in the general population, the effect of ABO blood type on venous thromboembolism (VTE) is well established, for example, in a study evaluating 1.5 million blood donors, patients with non-O blood type had an 80% increased risk of VTE\[^{33}\]. Patients with cirrhosis are at significantly increased risk of developing VTE. Cirrhosis may even be considered a pre-thrombotic disease, and elevated levels of vascular hemophilia factor (VWF) and coagulation factor VIII (FVIII) may contribute to the risk of thrombosis\[^{34}\]. It has been shown that VWF levels depend on ABO blood type, with non-O individuals having approximately 25% higher VWF and FVIII levels compared to O individuals. In addition, aging and ABO blood type have an effect on VWF/FVIII levels, with non-O elderly individuals exhibiting higher levels of VWF and FVIII\[^{35–36}\]. This is why in patients ≥ 55 aged years, blood type A has a higher risk of developing decompensated cirrhosis than blood type O.
ABO blood group was related to several aspects of blood biochemistry: ALT, AST, creatinine, triglycerides, direct bilirubin, and total bilirubin\[^{37}\]. However, in this study, there was no difference in the above serum markers in patients with decompensated cirrhosis, which may be due to the greater influence of disease factors on these markers.

HBV-associated decompensated cirrhosis can easily develop into HCC. HCC is a very rapidly progressing malignant tumor\[^{38}\]. In a recent study, the mean OS was 9 months. In the same study; HCC patients showed 1- and 2-year survival rates equal to 49.3% and 35.3\%\[^{39}\]. There are no enough studies showing the relationship between HCC survival and blood groups. In this study, 1-year survival was 86.4\% in patients with decompensated cirrhosis, and there was no statistically significant difference in the influence of ABO blood group on OS. This may be due to the relatively small number of patients.

5. Conclusion

These results suggest that ABO blood group is associated with the risk of developing decompensated cirrhosis in patients with CHB. Blood group increases the risk of decompensated cirrhosis in patients with CHB, and this association is age-related. ABO blood group does not differ in the severity of disease or short-term survival time in patients with hepatitis B decompensated cirrhosis. Further studies are needed to confirm and elucidate the mechanisms by which ABO antigens may influence the risk of decompensated cirrhosis. In the future, ABO blood group has the potential to be included in predictive models for hepatitis B decompensated cirrhosis along with other human genetic, hepatitis B infection-related risk and environmental factors.

6. Limitations

Alcoholic cirrhosis, mixed cirrhosis, and other causes of decompensated cirrhosis were excluded from this study, and the hospitalized CHB controls mainly represented the population with cases of hepatitis B decompensated cirrhosis. However, there were some limitations in this study, firstly the study population consisted mainly of Chinese Han CHB patients, and it was a single-center retrospective study with limited cases included and a small study sample, which limited the generalization of the results to some extent. Secondly, other blood group systems, such as the RH blood group system, were not considered.

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>HBV</td>
<td>Hepatitis B virus</td>
</tr>
<tr>
<td>CHB</td>
<td>Chronic hepatitis B</td>
</tr>
<tr>
<td>HCC</td>
<td>Hepatocellular carcinoma</td>
</tr>
<tr>
<td>TNF-α</td>
<td>Tumor necrosis factor alpha</td>
</tr>
</tbody>
</table>
sICAM-1  Soluble intercellular adhesion molecule 1
CAID  Cirrhosis-associated immune dysfunction
OS  Overall survival
AST  Aspartate aminotransferase
ALT  Alanine transaminase
MELD  Model of end-stage liver disease score
VTE  Venous thromboembolism
VWF  Vascular hemophilia factor

**Declarations**

**Ethics approval and consent to participate**

This study was performed in accordance with the Declaration of Helsinki for all human or animal experimental investigations. This study was approved by the Medical Ethical Committee of the First Affiliated Hospital, Zhejiang University School of Medicine in the study and informed consent obtained from participants.

**Consent for publication**

Not applicable.

**Authors' contributions**

JF, DC designed the project. HY interpreted and wrote the manuscript. DY, YH, RY, GG, XC review the manuscript. All authors read and approved the final manuscript.

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**Availability of data and materials**

The data used to support the findings of this study are included within the article.
Competing interest

The authors declare that they have no competing interests.

References

1. in Guidelines for the Prevention, Care and Treatment of Persons with Chronic Hepatitis B Infection WHO Guidelines Approved by the Guidelines Review Committee (2015).


**Figures**
Figure 1

Survival curves of patients with ABO groups in the decompensated phase of liver cirrhosis. No significant difference was found among four ABO blood groups in this study. OS: Overall Survival.