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Original Research Article

# Prevalence of Various Patterns of Anemia in Liver Cirrhosis

**Sathisha B.[1], Divya S.T.[2]**

[1]Consultant Physician, District Government Hospital (CMCRI) Chitradurga, Karnataka, India
[2]Senior Resident, Department of General Medicine, Chitradurga Medical College and Research Institute (CMCRI) Chitradurga, Karnataka, India

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**Corresponding Author: Dr. Divya S.T.**
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**Abstract**

**Objective:** To examine the prevalence of various patterns of anemia in liver cirrhosis.

**Methods:** This prospective observational study was conducted on patients admitted to the wards and ICU of SDM College of Medical Sciences and Hospital, Sattur, Dharwad. Ethical clearance was taken from the institution's Ethics Committee.

**Result:** The mean age was 48.85 ± 9.97 years with male predominance (90.00%). Alcohol was the predominant etiology (78.33%). The mean hemoglobin level was 9.07 ± 1.80 g/dL, with normocytic anemia being most common (65.00%), followed by macrocytic (20.00%) and microcytic anemia (15.00%). Macrocytic anemia was associated with higher MELD scores (20.17 ± 5.29) compared to microcytic anemia (14.22 ± 5.04, p=0.015).

**Conclusion:** The predominance of normocytic anemia (65.00%) in our cohort, consistent with anemia of chronic disease, points to the complex pathophysiology of anemia in liver cirrhosis, involving multiple mechanisms including impaired erythropoietin production, inflammatory cytokine-mediated suppression of erythropoiesis, hypersplenism, and reduced red cell survival.

**Keywords:** Anemia; Liver cirrhosis; MELD score; Normocytic anemia; Macrocytic anemia; Microcytic anemia.

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## Introduction

Liver cirrhosis represents the end stage of chronic liver disease where the liver undergoes irreversible scarring, leading to significant hepatic dysfunction and portal hypertension. One of the critical sequelae of this pathology is anemia, which can arise from multiple mechanisms including blood loss, hemolysis, nutritional deficiencies, and bone marrow suppression due to hypersplenism or the liver's diminished capacity to produce erythropoietin (EPO) [1].

The pathophysiology linking anemia to the severity of cirrhosis involves several pathways. Chronic liver disease can lead to a state of chronic inflammation, which can impair erythropoiesis through mechanisms like increased hepcidin levels, leading to iron sequestration and decreased iron availability for hemoglobin synthesis [2]. Additionally, portal hypertension often results in splenomegaly, which in turn causes sequestration of red blood cells, further contributing to anemia [3].

This thesis will delve into these associations by analyzing a cohort of cirrhotic patients at various stages of disease severity, as determined by the MELD score, and their corresponding anemia profiles. The study aims to provide a clearer picture of how anemia impacts the clinical course of liver cirrhosis, potentially offering insights into better management strategies and treatment approaches for this patient population.

## Material and Methods

This prospective observational study was conducted on patients admitted to the wards and ICU of SDM College of Medical Sciences and Hospital, Sattur, Dharwad. Ethical clearance was taken from the institution's Ethics Committee.

### Inclusion Criteria

- Liver cirrhosis patients aged over 18 years, irrespective of etiology.
- Patients with hemoglobin levels <13 g/dL in males and <12 g/dL in females.

### Exclusion Criteria

- Patients who had received a blood transfusion within the past three months. Patients diagnosed with chronic kidney disease.
- Patients with any other cause of anemia not

directly or indirectly related to liver cirrhosis.

**Sampling:** Sampling Population

Patients diagnosed with cirrhosis of the liver who were admitted to SDM College of Medical Sciences were included.

**Sample Size Calculation**

The sample size was calculated using the formula: Sample size

Sample size calculation-$Zx^2PQ/e^2$

This calculation resulted in a sample size of 60.

**Sampling Technique:** Out of these, 60 patients were selected using simple random sampling. Those fulfilling the inclusion criteria were enrolled.

**Data Collection Method:** Participants were screened for eligibility. Those eligible were briefed about the study, and written informed consent was obtained.

Demographic data including age, sex, and history of comorbidities (e.g., hypertension, diabetes mellitus), along with personal habits like alcohol consumption and smoking, were recorded.

A thorough physical examination included checking vitals and performing a systemic examination.

Diagnosis of liver cirrhosis was confirmed with ultrasound findings, blood investigations, and other relevant tests, which were documented on a pre-designed, pre-tested proforma.

**Investigations:** Complete haemogram, blood group, random blood sugar, blood urea, serum creatinine, serum electrolytes, liver function tests, PT-INR and viral markers were evaluated for each patient.

Additional procedures like ascitic fluid analysis, upper GI endoscopy, ultrasonography, colonoscopy, and CT abdomen were performed where clinically indicated and feasible.

**MELD Score:** The MELD score was calculated for each patient to assess the severity of liver disease.

**Statistical Analysis:** Data analysis was performed using SPSS version 20. Data were collected on a periodic basis throughout the inpatient stay, with all entries made into an MS Excel spreadsheet Descriptive statistics were used for demographic data. Pearson's correlation was employed to explore the relationship between anemia and the severity of liver cirrhosis. The Chi-square test was used for comparing categorical variables. A p-value less than 0.05 was considered statistically significant.

**Results**

The study included 60 patients with a mean age of 48.85 ± 9.97 years, ranging from 32 to 72 years. This distribution indicates that middle-aged individuals (40-59 years) represented the majority (63.33%) of the study population.

Male predominance is consistent with the epidemiological pattern of liver cirrhosis, particularly that associated with alcoholic etiology

Abdominal distension was the most common clinical presentation, occurring in 80.00% (n=48) of the patients, followed by pedal edema in 51.67% (n=31), jaundice in 36.67% (n=22), and bleeding manifestations in 30.00% (n=18).

Alcohol intake was the predominant risk factor, present in 78.33% (n=47) of the patients. Viral hepatitis was less common, with HBsAg positivity in 11.67% (n=7) and HCV positivity in 3.33% (n=2). The high prevalence of alcohol as a risk factor is consistent with the demographic profile of the study population, which had a male predominance.

The mean hemoglobin level was 9.07 ± 1.80 g/dL, with a wide range from 3.7 to 12.8 g/dL, indicating varying degrees of anemia. The mean MCV was 91.63 ± 12.98 fL, ranging from 32.4 to 118.2 fL, suggesting diverse patterns of anemia in the study population.

**Table 1: Classification of Anemia Based on MCV**

| Anemia Type | Number (%) |
|---|---|
| Microcytic (MCV < 80 fL) | 9 (15.00%) |
| Normocytic (MCV 80-100 fL) | 39 (65.00%) |
| Macrocytic (MCV > 100 fL) | 12 (20.00%) |

Based on MCV values, normocytic anemia was the most prevalent type, affecting 65.00% (n=39) of the patients. Macrocytic anemia was found in 20.00% (n=12), while microcytic anemia was present in 15.00% (n=9). The predominance of normocytic anemia suggests that multiple mechanisms, including chronic disease and hemolysis, may contribute to anemia in liver cirrhosis.

**Table 2: Association Between Anemia Types and Clinical Features**

| Clinical Feature | Microcytic Anemia (n=9) | Normocytic Anemia (n=39) | Macrocytic Anemia (n=12) | p-value |
|---|---|---|---|---|
| Jaundice | 2 (22.22%) | 14 (35.90%) | 6 (50.00%) | 0.387 |
| Abdominal Distension | 7 (77.78%) | 32 (82.05%) | 9 (75.00%) | 0.852 |
| Pedal Edema | 5 (55.56%) | 19 (48.72%) | 7 (58.33%) | 0.822 |
| Bleeding Manifestation | 3 (33.33%) | 12 (30.77%) | 3 (25.00%) | 0.903 |
| Altered Sensorium | 0 (0.00%) | 2 (5.13%) | 1 (8.33%) | 0.689 |

No significant associations were found between anemia types and clinical features. Jaundice was present in 22.22% (n=2/9) of microcytic, 35.90% (n=14/39) of normocytic, and 50.00% (n=6/12) of macrocytic anemia patients (p=0.387). Abdominal distension was present in 77.78% (n=7/9) of microcytic, 82.05% (n=32/39) of normocytic, and 75.00% (n=9/12) of macrocytic anemia patients (p=0.852). Pedal edema was present in 55.56% (n=5/9) of microcytic, 48.72% (n=19/39) of normocytic, and 58.33% (n=7/12) of macrocytic anemia patients (p=0.822). Bleeding manifestation was present in 33.33% (n=3/9) of microcytic, 30.77% (n=12/39) of normocytic, and 25.00% (n=3/12) of macrocytic anemia patients (p=0.903). Altered sensorium was present in 0.00% (n=0/9) of microcytic, 5.13% (n=2/39) of normocytic, and 8.33% (n=1/12) of macrocytic anemia patients (p=0.689). These findings suggest that the type of anemia did not influence the clinical presentation of liver cirrhosis.

**Table 3: Association Between Anemia Severity and Portal Hypertension Features**

| Feature | Mild Anemia (n=11) | Moderate Anemia (n=33) | Severe Anemia (n=16) | p-value |
|---|---|---|---|---|
| Ascites | 9 (81.82%) | 29 (87.88%) | 15 (93.75%) | 0.592 |
| Varices | 5 (45.45%) | 19 (57.58%) | 11 (68.75%) | 0.462 |
| PHG | 3 (27.27%) | 16 (48.48%) | 10 (62.50%) | 0.179 |

No significant associations were found between anemia severity and portal hypertension features. Ascites was present in 81.82% (n=9/11) of mild, 87.88% (n=29/33) of moderate, and 93.75% (n=15/16) of severe anemia patients (p=0.592). Varices were present in 45.45% (n=5/11) of mild, 57.58% (n=19/33) of moderate, and 68.75% (n=11/16) of severe anemia patients (p=0.462). Portal hypertensive gastropathy (PHG) was present in 27.27% (n=3/11) of mild, 48.48% (n=16/33) of moderate, and 62.50% (n=10/16) of severe anemia patients (p=0.179). Although there was a trend towards higher prevalence of portal hypertension features with increasing anemia severity, the differences did not reach statistical significance.

**Table 4: Comparison of Hematological Parameters Between Males and Females**

| Parameter | Males (n=54) | Females (n=6) | p-value |
|---|---|---|---|
| Hemoglobin (g/dL) | 9.08 ± 1.85 | 8.98 ± 1.38 | 0.892 |
| MCV (fL) | 92.76 ± 12.34 | 80.35 ± 14.49 | 0.022* |
| MCH (pg) | 30.15 ± 5.12 | 24.23 ± 3.96 | 0.008* |
| MCHC (g/dL) | 32.88 ± 1.85 | 30.47 ± 0.98 | 0.003* |

*Statistically significant ($p<0.05$) by independent t-test

Hemoglobin levels were similar between males (9.08 ± 1.85 g/dL) and females (8.98 ± 1.38 g/dL) (p=0.892). However, significant differences were found in other red cell indices. Males had significantly higher MCV (92.76 ± 12.34 fL vs. 80.35 ± 14.49 fL, p=0.022), MCH (30.15± 5.12 pg vs. 24.23 ± 3.96 pg, p=0.008), and MCHC (32.88 ± 1.85 g/dL vs. 30.47 ± 0.98g/dL, p=0.003) compared to females. These findings suggest that while the degree of anemia was similar between genders, the pattern differed, with males tending towards normocytic or macrocytic anemia and females towards microcytic anemia.

**Table 5: Correlation Matrix Between Hematological and Liver Function Parameters**

| Parameters | Hemoglobin | MCV | Total Bilirubin | INR | MELD Score |
|---|---|---|---|---|---|
| Hemoglobin | 1.000 | 0.176 | -0.315* | -0.362* | -0.481* |
| MCV | 0.176 | 1.000 | 0.245 | 0.128 | 0.203 |
| Total Bilirubin | -0.315* | 0.245 | 1.000 | 0.196 | 0.683* |
| INR | -0.362* | 0.128 | 0.196 | 1.000 | 0.722* |
| MELD Score | -0.481* | 0.203 | 0.683* | 0.722* | 1.000 |

*Statistically significant correlation ($p<0.05$)

Hemoglobin showed significant negative correlations with total bilirubin ($r=-0.315$, $p<0.05$), INR ($r=-0.362$, $p<0.05$), and MELD score ($r=-0.481$, $p<0.05$), indicating that anemia worsened with increasing liver dysfunction. MELD score showed strong positive correlations with total bilirubin ($r=0.683$, $p<0.05$) and INR ($r=0.722$, $p<0.05$), consistent with its calculation formula. No significant correlations were found between MCV and other

parameters, suggesting that the pattern of anemia (microcytic, normocytic, or macrocytic) was not directly related to the degree of liver dysfunction.

**Table 6: Multivariate Analysis of Factors Associated with Severe Anemia**

| Factor | Odds Ratio | 95% CI | p-value |
|---|---|---|---|
| MELD Score >19 | 3.86 | 1.18-12.64 | 0.025* |
| Alcohol Intake | 2.31 | 0.45-11.78 | 0.314 |
| Bleeding Manifestation | 1.76 | 0.52-5.94 | 0.367 |
| Total Bilirubin >3 mg/dL | 2.08 | 0.63-6.83 | 0.227 |
| Varices | 1.93 | 0.58-6.41 | 0.285 |

*Statistically significant (p<0.05)

In multivariate analysis, only MELD score >19 was significantly associated with severe anemia (OR=3.86, 95% CI 1.18-12.64, p=0.025), indicating that patients with high MELD scores were nearly four times more likely to have severe anemia compared to those with lower scores. Other factors, including alcohol intake (OR=2.31, 95% CI 0.45-11.78, p=0.314), bleeding manifestation (OR=1.76, 95% CI 0.52-5.94, p=0.367), total bilirubin >3 mg/dL (OR=2.08, 95% CI 0.63-6.83, p=0.227), and varices (OR=1.93, 95% CI 0.58-6.41, p=0.285), were not independently associated with severe anemia. This finding underscores the importance of liver dysfunction severity as a determinant of anemia severity. No significant differences were found in anemia patterns between alcoholic and non-alcoholic cirrhosis. In alcoholic cirrhosis (n=47), microcytic anemia was present in 12.77% (n=6), normocytic in 63.83% (n=30), and macrocytic in 23.40% (n=11). In non-alcoholic cirrhosis (n=13), microcytic anemia was present in 23.08% (n=3), normocytic in 69.23% (n=9), and macrocytic in 7.69% (n=1). The p-values for comparisons were 0.352 for microcytic, 0.715 for normocytic, and 0.201 for macrocytic anemia, indicating that the etiology of cirrhosis did not significantly influence the pattern of anemia.

**Table 7: Relationship Between Bleeding Manifestations and Anemia Severity**

| Anemia Severity | Bleeding Present (n=18) | Bleeding Absent (n=42) | p-value |
|---|---|---|---|
| Mild | 2 (11.11%) | 9 (21.43%) | 0.347 |
| Moderate | 9 (50.00%) | 24 (57.14%) | 0.606 |
| Severe | 7 (38.89%) | 9 (21.43%) | 0.149 |

No significant associations were found between bleeding manifestations and anemia severity. Among patients with bleeding manifestations (n=18), mild anemia was present in 11.11% (n=2), moderate in 50.00% (n=9), and severe in 38.89% (n=7). Among those without bleeding manifestations (n=42), mild anemia was present in 21.43% (n=9), moderate in 57.14% (n=24), and severe in 21.43% (n=9). The p-values for comparisons were 0.347 for mild, 0.606 for moderate, and 0.149 for severe anemia. Although there was a trend towards higher prevalence of severe anemia in patients with bleeding manifestations (38.89% vs.21.43%), the difference did not reach statistical significance, suggesting that factors other than overt bleeding contributed to anemia severity.

**Table 8: Correlation Between MCV Values and Severity of Liver Disease**

| MCV Category | Mean MELD Score | p-value |
|---|---|---|
| Microcytic (<80 fL) (n=9) | 14.22 ± 5.04 | Reference |
| Normocytic (80-100 fL) (n=39) | 17.05 ± 5.33 | 0.147 |
| Macrocytic (>100 fL) (n=12) | 20.17 ± 5.29 | 0.015* |

*Statistically significant (p<0.05) compared to microcytic category by ANOVA with post-hoc analysis

Mean MELD scores increased progressively with increasing MCV values: 14.22 ± 5.04 in microcytic anemia, 17.05 ± 5.33 in normocytic anemia (p=0.147 compared to microcytic), and 20.17 ± 5.29 in macrocytic anemia (p=0.015 compared to microcytic). The significant difference between macrocytic and microcytic anemia suggests an association between macrocytosis and more severe liver disease, possibly reflecting the impact of alcohol, which was the predominant etiology in this cohort.

No significant differences were found in clinical features across anemia severity groups. Jaundice was present in 27.27% (n=3/11) of mild, 33.33% (n=11/33) of moderate, and 50.00% (n=8/16) of severe anemia patients (p=0.379). Abdominal distension was present in 63.64% (n=7/11) of mild, 84.85% (n=28/33) of moderate, and 81.25% (n=13/16) of severeanemia patients (p=0.284). Pedal edema was present in 45.45% (n=5/11) of mild, 51.52% (n=17/33) of moderate, and 56.25% (n=9/16) of severe anemia patients (p=0.853). Bleeding manifestation was present in 18.18% (n=2/11) of mild, 27.27% (n=9/33) of moderate, and 43.75% (n=7/16) of severe anemia patients (p=0.295). Altered sensorium was present in 0.00% (n=0/11) of mild, 3.03% (n=1/33) of moderate, and 12.50% (n=2/16) of severe anemia patients (p=0.222). Although there were trends towards

higher prevalence of certain features (e.g., jaundice, bleeding, altered sensorium) with increasing anemia severity, the differences did not reach statistical significance.

## Discussion

In our cohort of 60 patients with liver cirrhosis, we observed a mean hemoglobin level of 9.07 ± 1.80 g/dL, indicating moderate anemia on average. The prevalence of anemia severity varied, with moderate anemia being the most common (55.00%, n=33), followed by severe (26.67%, n=16) and mild anemia (18.33%, n=11). These findings are consistent with previous studies reporting high anemia prevalence in cirrhotic patients. Scheiner et al. examined 200 patients with advanced chronic liver disease and found a 65% prevalence of anemia, with more severe anemia associated with advanced liver disease [4]. Similarly, Qamar and colleagues reported anemia in 58% of patients with compensated cirrhosis [5], while a study by Maan et al. noted anemia in up to 75% of cirrhotic patients depending on the stage of liver disease [6].

The most common pattern of anemia in our study was normocytic (65.00%, n=39), followed by macrocytic (20.00%, n=12) and microcytic anemia (15.00%, n=9). This distribution aligns with findings from Gonzalez-Casas et al., who reported a predominance of normocytic anemia in cirrhotic patients, reflecting the multifactorial nature of anemia in liver disease [7]. Our observation that macrocytic anemia was associated with higher MELD scores (20.17 ± 5.29 vs. 14.22 ± 5.04 in microcytic anemia, p=0.015) and significantly elevated total bilirubin levels (8.57 ± 6.54 mg/dL vs. 2.36 ± 2.49 mg/dL, p=0.007) is noteworthy and consistent with Yang et al.'s retrospective study of 144 patients with HBV-related decompensated cirrhosis, which found macrocytosis to be associated with more severe liver impairment [8].

The higher prevalence of normocytic anemia in our study is likely due to the predominance of anemia of chronic disease in cirrhotic patients. This form of anemia is characterized by reduced erythropoietin production, impaired iron utilization, and shortened red cell survival, all of which are common in chronic liver disease [9]. Weiss and Goodnough have described this as a multifactorial anemia involving iron-restricted erythropoiesis despite adequate iron stores, driven primarily by inflammatory cytokines [10]. In cirrhotic patients, elevated hepcidin levels due to systemic inflammation can block iron release from reticuloendothelial cells, contributing to functional iron deficiency despite normal or elevated iron stores [11].

Our analysis revealed interesting patterns in the distribution of anemia types across different MELD categories and their association with clinical parameters. Macrocytic anemia, present in 20.00% (n=12) of our patients, was significantly associated with higher MELD scores and elevated total bilirubin levels compared to microcytic anemia. This is consistent with findings from Yang et al., who reported that macrocytosis was associated with more severe liver impairment in HBV-related cirrhosis, with mean Child-Pugh scores of 9.62 ± 2.32 in patients with macrocytic anemia compared to 8.31 ± 2.24 in those with non-macrocytic anemia ($p<0.001$) [8].

The predominance of macrocytosis in alcoholic liver disease has been well-documented. Wu et al. observed macrocytosis in 96% of alcoholic patients with associated folate deficiency [12]. In our study, although the prevalence of macrocytic anemia was higher in alcoholic cirrhosis (23.40%, n=11/47) compared to non-alcoholic cirrhosis (7.69%, n=1/13), the difference did not reach statistical significance (p=0.201). This may be due to our relatively small sample size or the multifactorial nature of macrocytosis in liver disease, which can result from folate deficiency, vitamin B12 deficiency, or direct alcohol toxicity on erythropoiesis [13].

Microcytic anemia, present in 15.00% (n=9) of our patients, was associated with lower MELD scores and total bilirubin levels. This pattern most commonly results from iron deficiency, which can occur in cirrhotic patients due to reduced dietary intake, malabsorption, or occult gastrointestinal bleeding [14]. Gkamprela et al. reported that up to 32% of cirrhotic patients have iron deficiency anemia, often due to portal hypertensive gastropathy or esophageal varices [15]. In our cohort, the relatively lower prevalence of microcytic anemia might be explained by the competing effects of iron deficiency and the anemia of chronic disease, which can mask the microcytic pattern. The predominance of normocytic anemia (65.00%, n=39) in our study reflects the complex pathophysiology of anemia in liver cirrhosis. This pattern is typically associated with anemia of chronic disease, which involves multiple mechanisms including impaired erythropoietin production, inflammatory cytokine-mediated suppression of erythropoiesis, and reduced red cell survival [16]. Gonzalez-Casas et al. noted that normocytic anemia is the most common pattern in cirrhotic patients, particularly in those with advanced disease [7].

## Conclusion

The predominance of normocytic anemia (65.00%) in our cohort, consistent with anemia of chronic disease, points to the complex pathophysiology of anemia in liver cirrhosis, involving multiple mechanisms including impaired erythropoietin production, inflammatory cytokine-mediated suppression of erythropoiesis, hypersplenism, and

reduced red cell survival. The significant association between macrocytic anemia and higher MELD scores (20.17 ± 5.29 vs. 14.22 ± 5.04 in microcytic anemia, p=0.015) suggests that MCV could potentially serve as a simple additional marker for disease severity in cirrhotic patients.

The absence of significant associations between anemia and clinical manifestations or portal hypertension features underscores that the relationship between anemia and liver cirrhosis severity is primarily biochemical rather than clinical. Similarly, the lack of significant associations between alcohol intake and anemia patterns or severity suggests that liver dysfunction severity, rather than etiology, is the primary determinant of anemia in cirrhosis.

**References**


1. Guralnik JM, Eisenstaedt RS, Ferrucci L, Klein HG, Woodman RC. Prevalence of anemia in persons 65 years and older in the United States: evidence for a high rate of unexplained anemia. Blood. 2004;104(8):2263-8.
2. Nemeth E, Tuttle MS, Powelson J, Vaughn MB, Donovan A, Ward DM, et al.Hepcidin regulates cellular iron efflux by binding to ferroportin and inducing its internalization. Science. 2004;306(5704):2090-3.
3. Giannini EG, Botta F, Testa E, Romagnoli P, Polegato S, Malfatti F, et al. Splenomegaly is an independent predictor of anemia in cirrhosis. Hepatol Res. 2005;32(4):236-40.
4. Scheiner B, Semmler G, Maurer F, Schwabl P, Bucsics TA, Paternostro R, et al. Prevalence of and risk factors for anaemia in patients with advanced chronic liver disease. Liver Int. 2020;40(1):194-204.
5. Qamar AA, Grace ND, Groszmann RJ, Garcia-Tsao G, Bosch J, Burroughs AK, et al. Incidence, prevalence, and clinical significance of abnormal hematologic indices in compensated cirrhosis. Clin Gastroenterol Hepatol. 2009;7(6):689-95.
6. Maan R, van Tilburg M, van der Meer AJ, Groothuismink ZMA, Feld JJ, Hansen BE, et al. Anemia in chronic liver disease: a neglected complication of a common disease. World J Hepatol. 2016;8(3):144-51.
7. Gonzalez-Casas R, Jones EA, Moreno-Otero R. Spectrum of anemiaassociated with chronic liver disease. World J Gastroenterol. 2009;15(37):4653-8.
8. Yang J, Yan B, Yang L, Li H, Fan Y, Zhu F, et al. Macrocytic anemia is associated with the severity of liver impairment in patients with hepatitis B virus-related decompensated cirrhosis: a retrospective cross-sectional study. BMC Gastroenterol. 2018;18:161.
9. Shah A, Kowdley KV. Anemia in chronic liver disease: a neglected complication of a common disease. World J Hepatol. 2011;3(4):109-15.
10. Weiss G, Goodnough LT. Anemia of chronic disease. N Engl J Med.2005;352(10):1011-23.
11. Vela D. Low hepcidin in liver fibrosis and cirrhosis; a tale of progressive disorder and a case for a new biochemical marker. Mol Med. 2018; 24:5.
12. Wu A, Chanarin I, Slavin G, Levi AJ. Folate deficiency in the alcoholic—its relationship to clinical and haematological abnormalities, liver disease and folate stores. Br J Haematol. 1975;29(3):469-78.
13. Ballard HS. The hematological complications of alcoholism. Alcohol Health Res World. 1997;21(1):42-52.
14. Gkamprela E, Deutsch M, Pectasides D. Iron deficiency anemia in chronic liver disease: etiopathogenesis, diagnosis and treatment. Ann Gastroenterol. 2017;30(4):405-13.
15. Gkamprela E, Deutsch M, Pectasides D. Iron deficiency anemia in chronic liver disease: etiopathogenesis, diagnosis and treatment. Ann Gastroenterol.2017;30(4):405-13.
16. Weiss G, Goodnough LT. Anemia of chronic disease. N Engl J Med.2005;352(10):1011-23.