CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.3270770

Available online at: http://www.iajps.com *Research Article*

ANALYSIS OF LOW SERUM FERRITIN LEVEL IN PATIENTS OF DECOMPENSATED CHRONIC LIVER DISEASE

Dr Hooria Shahzad[1], Dr Anoosh Fatima Gilani[2], Dr Haris Asif[3]

[1]Medical officer at RHC Dullanwala, [2]Shalamar Medical and Dental College, [3]Baqai Medical College.

Article Received: May 2019 **Accepted:** June 2019 **Published:** July 2019

Abstract:

Introduction: *Liver fibrosis and its end-stage cirrhosis resulting from chronic liver injury are major causes of morbidity and mortality worldwide.*

Objectives of the study: *To determine the frequency of low serum ferritin level in patients of decompensated chronic liver disease.*

Methodology of the study: *This cross sectional study was conducted in Shalamar medical and dental college during November 2018 to March 2019. All adult and middle age 20-60 years patients of both genders, diagnosed case of chronic liver disease (previously or recently diagnosed having features of decompensated disease) were included in the study. The data was collected from 100 patients. History was taken in detail and thorough examination was performed.*

Results: *The data was collected from 100 patients. Descriptive statistics of age (years) of patient was also calculated in terms of mean and standard deviation. Mean age (years) in the study was 53.74+8.66 with ranges from 20 to 60 years. Effect modifier like age was stratified and compared with frequency low serum ferritin level in patients of decompensated chronic live disease secondary to hepatitis C virus infection.*

Conclusion: *It is concluded that elevated serum ferritin worsened liver injury. Hence it represents early marker for the severity of chronic liver disease, related both to the degree of liver fibrosis and to the necro-inflammatory activity.*

Corresponding author:

Dr. Hooria Shahzad,

Medical officer at RHC, Dullanwala.

QR code

Please cite this article in press Hooria Shahzad et al., ***Analysis of Low Serum Ferritin Level in Patients of Decompensated Chronic Liver Disease.,*** *Indo Am. J. P. Sci, 2019; 06(07).*

INTRODUCTION:

Liver fibrosis and its end-stage cirrhosis resulting from chronic liver injury are major causes of morbidity and mortality worldwide. Among the etiologies of hepatic cirrhosis, viral infection is most common (e.g. hepatitis B and C), and currently affects 1–2% of the US population, with cirrhosis projected to reach 45% of those infected with hepatitis C virus (HCV) in 2030. In Pakistan more people die of liver disease due to chronic hepatitis every day than terrorism in a year [1].

Hepatic fibrosis was historically thought to be a passive and irreversible process due to the collapse of the hepatic parenchyma and its substitution with a collagen-rich tissue. Currently, it is considered a model of the wound-healing response to chronic liver injury. Early clinical reports in the 1970s suggested that advanced liver fibrosis is potentially reversible [2]. However, liver fibrosis received little attention until the 1980s, when hepatic stellate cells (HSCs), formerly known as lipocytes, Ito cells, or perisinusoidal cells, were identified as the main collagen-producing cells in the liver [3]. This cell type, first described by von Kupffer in 1876, undergoes a dramatic phenotypic activation in chronic liver diseases with the acquisition of fibrogenic properties [4]. Methods to obtain HSCs from both rodent and human livers were rapidly standardized in the 1980s, and prolonged culture of HSCs on plastic was widely accepted as a model for the study of activated HSCs. Key signals that modulate HSCs' fibrogenic actions were delineated. Experimental models for studying liver fibrogenesis in rats and in transgenic mice were developed, which corroborated the cell culture studies and led to the identification of key fibrogenic mediators [5]. Besides HSCs, portal myofibroblasts and cells of bone marrow origin have been recently shown to exhibit fibrogenic potential. At the clinical level, the natural history of liver fibrosis, from early changes to liver cirrhosis, was delineated in patients with chronic HCV infection. Rapid and slower fibrosers were identified, and genetic and environmental factors influencing fibrosis progression were partially uncovered. Since the demonstration, in the 1990s, that even advanced liver fibrosis is reversible, researchers have been stimulated to identify antifibrotic therapies. Biotechnology and pharmaceutical companies are increasingly interested in developing antifibrotic programs, and clinical trials are currently underway [6]. However, the most effective therapy for treating hepatic fibrosis to date is still to remove the causative agent. A number of drugs are able to reduce the accumulation of scar tissue in experimental models of chronic liver injury [7].

Objectives of the study:

To determine the frequency of low serum ferritin level in patients of decompensated chronic liver disease.

Methodology of the study:

This cross sectional study was conducted in Shalamar medical and dental college during November 2018 to March 2019. All adult and middle age 20-60 years patients of both genders, diagnosed case of chronic liver disease (previously or recently diagnosed having features of decompensated disease) were included in the study.

The data was collected from 100 patients. History was taken in detail and thorough examination was performed. After receiving patient in Gastroenterology department, all patients were assessed clinically. After clinical examination, the diagnosis of chronic liver disease was made. After taking all aseptic measures blood was taken and stored in a serum bottle. The serum bottle was send to the hospital laboratory in order to check serum iron, ferritin, TIBC, and LFT, s.

Statistical analysis:

The data was analyzed by SPSS software version 16. Descriptive statistics was calculated for all variables like age, gender, low serum ferritin. Post stratification chi-square test was applied and p value was less than 0.05 and was significant.

RESULTS:

The data was collected from 100 patients. Descriptive statistics of age (years) of patient was also calculated in terms of mean and standard deviation. Mean age (years) in the study was 53.74+8.66 with ranges from 20 to 60 years. Effect modifier like age was stratified and compared with frequency low serum ferritin level in patients of decompensated chronic live disease secondary to hepatitis C virus infection. There were 03 (100.0) patients who age 50 – 60 years with low serum ferritin level in patients of decompensated chronic live disease secondary to hepatitis C virus infection which was statistically not significant (p-value 0.242).

Table 01: Descriptive statistics of Age (years) of patients

	n	Minimum	Maximum	Mean	Std. Deviation
Age (years)	165	20	60	53.74	8.66

Table 02: Effect modifier like Age stratification with low Serum Ferritin (< 20 ng/ml)

			Low Serum Ferritin level (< 20 ng/ml)		P-value
			yes	no	
Age group	20 - 50 years		0	51	0.242
			0.0%	31.5%	
	50 - 60 years		3	111	
			100.0%	68.5%	
Total			3	162	
			100.0%	100.0%	

DISCUSSION:

Chronic hepatitis C (CHepC) is frequently associated with hepatic iron overload. Elevation of serum iron indices or stainable hepatic iron has been shown in 40 to 70% of patients with CHepC. From these observations, iron-induced oxidative stress has been considered to be an underlying mechanism of liver injury and of development of hepatocellular carcinoma [8].

The mechanisms of hepatic iron overload in CHepC have not yet been elucidated. However, hepcidin has attracted much attention as an important factor in the disease process. Hepcidin is exclusively produced in the liver and regulates body iron stores [9]. Hepcidin causes internalization and degradation of iron-transporter ferroportin on duodenal enterocytes and macrophages, thereby blocking iron absorption and iron recycling, respectively [10]. In hereditary hemochromatosis (HH), defective hepcidin synthesis results in a subsequent increase in body iron stores. In CHepC, hepatic iron overload has been attributed to the mutation of the hemochromatosis protein (HFE) gene, since several reports have found an association between HFE genotypes and iron overload in patients with CHepC [11]. Another possible mechanism is the direct effect of the hepatitis C virus (HCV) on hepcidin synthesis. Transgenic mice expressing HCV polyprotein have been shown to have decreased hepatic expression of hepcidin due to HCV-induced oxidative stress [11].

CONCLUSION:

It is concluded that elevated serum ferritin worsened liver injury. Hence it represents early marker for the severity of chronic liver disease, related both to the degree of liver fibrosis and to the necro-inflammatory activity.

REFERENCES:

1. Cohen-Naftaly M, Friedman SL. Current status of novel antifibrotic therapies in patients with chronic liver disease. J Therap Adv Gastroenterol. 2011;4:391–417.
2. Memon MS, Zaki M. Burden of chronic liver disease and liver transplantation in Sindh. J Liaquat Uni Med Health Sci.2013;12:(1)1-2.
3. Kumar EH, Radhakrishnan A. Prevalence of anemia in decompensated chronic liver disease. World J Med Sci. 2014;10(1):56-60.
4. Fauci AS, Braunwald E, Kasper DL. Iron deficiency anemia. Harrison's principles of internal medicine. 18thed. Hauser SL, Longo DL, Jamson JL. New York: McGraw-Hill Medicine. 2015;4:749-62.
5. Mitsuyoshi H, Yasui K, Yamaguchi K, Minami M, Okanoue T, Itoh Y et al. Pathogenic Role of Iron Deposition in Reticuloendothelial Cells

during the Development of Chronic Hepatitis C. Int J Hepatol.2013;2013:686420.
6. Codruta V, Camelia S, Simona R. Serum iron markers in patients with chronic hepatitis C infection. Hepat Mon Int J. 2013;13(10): e13136.
7. Friedman SL. Liver fibrosis - from bench to bedside. J. Hepatol. 2003;38(Suppl. 1):S38–S53.
8. Friedman SL. Hepatic stellate cells: protean, multifunctional, and enigmatic cells of the liver. Physiol Rev. 2008;88:125–172
9. Li Z, Dranoff JA, Chan EP, Uemura M, Sévigny J, Wells RG.. Transforming growth factor-beta and substrate stiffness regulate portal fibroblast activation in culture. Hepatology. 2007;46:1246–1256
10. Wells RG. The role of matrix stiffness in regulating cell behavior. Hepatology. 2008;47:1394–1400.
11. Bird TG, Lorenzini S, Forbes SJ. Activation of stem cells in hepatic diseases. Cell Tissue Res. 2008;331:283–300.