WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

Relation between Liver Iron Levels and HBV-Related HCC Tumours: An Extensive Investigation

Ms. Anuradha[1], Dr.Jaya Jain[2]

[1]Research scholar, Malwanchal University, Indore
[2]Research Supervisor, Malwanchal University, Indore

Introduction

Iron is necessary for life and is involved in a number of different metabolic processes, including the formation of new cells and their division. The presence of excessive iron in the human body, on the other hand, raises the risk of fostering the growth of neoplastic cells. This is due to the function that iron plays in redox cycling and the generation of free radicals. The concentration of iron in the blood serum is a frequent indication used in the laboratory to assess liver biochemistry. A number of liver illnesses have been linked to an excess of iron in the liver, which has been the subject of investigation in the past. The association between serum iron levels and the prognosis of HBV-related HCC is still not well understood, despite the fact that there is a known relationship between hepatic iron excess and the development of HCC. There is a paucity of evidence about the ways in which blood iron levels impact the progression of chronic HBV infection and the way in which they influence the prognosis of HCC. In light of the information presented above, a research was carried out to illustrate the relationship between blood iron levels and the prognosis of HCC and the progression of chronic HBV infection.

Methodology

Patients hospitalised at Index Medical College with a diagnosis of CHB, HBV-related liver cirrhosis (HBV-related LC), or HBV-related head and neck cancer between January 2018 and August 2019 were eligible to participate. Two hundred volunteers with normal kidney and liver function, no history of liver sickness in their families, no alcohol use, no history of cardiovascular or haematological disease in their families, and no other acute or chronic illnesses were included as controls. Patient medical and pharmaceutical histories, as well as smoking and drinking habits, liver biochemistry, routine blood work, alanine aminotransferase (AFP), and hepatitis B virus (HBV) viral load, were documented upon admission. The patient's medical history, medication history, and smoking and drinking habits were all covered. In every step of the analysis, the SPSS software, version 21.0, was utilised. Categorical data were shown as a percentage, whereas continuous data were shown as a mean + standard deviation.

Results

Researchers looked at the relationships between serum iron levels and other laboratory markers to determine the clinical significance of reducing blood iron levels in HBV-related HCC patients. Figures 1A and 1B showed that there was an inverse relationship between serum iron levels and white blood cell (WBC) counts (r = 0.094, P = 0.032) and platelet (PLT) counts (r = 0.168, P 0.001). Conversely, C and D showed that serum iron levels positively correlated with haemoglobin (Hb; r = 0.170, P 0.001) and lysozyme (LY; r = 0.289, P 0.001). Analogously, E and F showed that serum RBP (r = 0.183, P 0.001) and TBA (r = 0.091, P = 0.055) were positively correlated with blood iron levels. We also looked at the possibility of a correlation between the two metrics (HBV DNA levels and serum iron levels). However, serum iron levels were not correlated with HBV DNA concentrations (r = 0.043, P = 0.462). Serum iron levels of 16.2 mol/l were chosen as the appropriate cut-off value according to the ROC curve. The area under the curve (AUC) for serum iron levels was 0.722 [95% CI: 0.734-0.853, P 0.001]. Using the ROC curve, we divided the patients into two categories depending on their serum iron levels. Patients with iron levels below 16.1 mol/l were placed in one group, while those with iron levels over 16.1 mol/l were placed in another group. The platelet count was observed to be lower in individuals whose serum iron levels were more than 15.1 mol/l (118.3 vs. 130.4, P 0.001) compared to those whose serum iron levels were less than 16.1 mol/l. The findings demonstrated this. Serum iron levels more than 16.1 mol/l were associated with a higher incidence of Child-Pugh A, BCLC stages A and B, HBV-related HCC of 4 cm and HBV-related HCC of 4-6 cm, and antiviral medication prior to the diagnosis of HBV-related HCC (all of them, P 0.05). It was the same regardless of the iron content in the serum. Other laboratory parameters, including white blood cell count, lymphocyte count, red blood cell count, haemoglobin content, alanine aminotransferase activity, tyrosine phosphorylase activity, rheumatoid factor activity, alpha fetoprotein concentration, and hepatitis B virus load, showed no significant differences between the two groups. In addition, neither group showed any significant differences from the other in terms of patients' ages, sex

distributions, or cirrhosis severity.

One study's examination of survival data found that iron in the blood was a reliable predictor of future health. We found that a hazard ratio (HR) of 2.477 (P 0.001) indicated a statistically significant decrease in overall survival when blood iron levels were lower than 16.1 uml/l. In this post, we looked at several additional predictors of outcome. These included BCLC stage A (hazard ratio = 0.384, P 0.001), antiviral therapy (ART) prior to the diagnosis of HBV-related HCC (hazard ratio = 0.443, P 0.001), and tumour size of 4 cm (hazard ratio = 0.172, P 0.001), 4-5 cm (hazard ratio = 0.244, P 0.001), 4-10 cm (hazard ratio = 0.691, P = 0.003).

Conclusion

In light of these findings, which demonstrated an inverse relationship between blood iron levels and the growth of HBV-related HCC tumours, one may deduce that there is a connection between serum iron levels and the progression of chronic HBV infection. A subgroup investigation of patients with HBV-related HCC found that those with tumours bigger than 10 centimetres had the lowest levels of blood iron. The iron levels in the tissue were compared to the iron levels in the serum since the former needed a laboratory test that was less invasive than the latter. As a result, the most noteworthy parts of our analysis were the novel results that provide HBV-associated HCC patients a reliable, repeatable, and easily detectable prognostic indicator. In addition, a threshold serum iron level was suggested, which would assist medical professionals in making the most effective use of serum iron as a predictive marker in their everyday practise. Since this experiment did not reveal any information on the link between serum iron and iron metabolism, it is not possible to draw any conclusions from it. As a result, we intend to continue our prior study on the association between serum iron and iron metabolism and look for new findings.

Reference

1) Brown RS Jr, Lake JR. The survival impact of liver transplantation in the MELD era, and the future for organ allocation and distribution. *Am J Transplant* 2005; 5: 203- 204.
2) Habib S, Berk B, Chang CC, Demetris AJ, Fontes P, Dvorchik I, et al. MELD and prediction of post-liver transplantation survival. *Liver Transpl* 2006; 12: 440- 447.
3) Kamath PS, Wiesner RH, Malinchoc M, Kremers W, Therneau TM, Kosberg CL, et al. A model to predict survival in patients with end-stage liver disease. *Hepatology* 2001; 33: 464- 470.
4) Silberhumer GR, Hetz H, Rasoul-Rockenschaub S, Peck-Radosavljevic M, Soliman T, Steininger R, et al. Is MELD score sufficient to predict not only death on waiting list, but also post-transplant survival? *Transpl Int* 2006; 19: 275- 281.
5) Kim WR, Biggins SW, Kremers WK, Wiesner RH, Kamath PS, Benson JT, et al. Hyponatremia and mortality among patients on the liver-transplant waiting list. *N Engl J Med* 2008; 359: 1018- 1026.
6) Luca A, Angermayr B, Bertolini G, Koenig F, Vizzini G, Ploner M, et al. An integrated MELD model including serum sodium and age improves the prediction of early mortality in patients with cirrhosis. *Liver Transpl* 2007; 13: 1174- 1180.
7) Ruf AE, Kremers WK, Chavez LL, Descalzi VI, Podesta LG, Villamil FG, et al. Addition of serum sodium into the MELD score predicts waiting list mortality better than MELD alone. *Liver Transpl* 2005; 11: 336- 343.
8) Weismüller TJ, Kirchner GI, Scherer MN, Negm AA, Schnitzbauer AA, Lehner F, et al. Serum ferritin concentration and transferrin saturation before liver transplantation predict decreased long-term recipient survival. *Hepatology* 2011; 54: 2114- 2124.
9) Walker NM, Stuart KA, Ryan RJ, Desai S, Saab S, Nicol JA, et al. Serum ferritin concentration predicts mortality in patients awaiting liver transplantation. *Hepatology* 2010; 51: 1683- 1691.
10) Al-Freah MA, Kriese S, Foxton MR, Quaglia A, Bomford A, Heaton ND, et al. The association of pretransplant ferritin level with waiting list and post-transplant survival. does ferritin actually predict outcome? *Transpl Int* 2013; 26: 1070- 1079.
11) Weismüller TJ, Manns MP, Strassburg CP. Ferritin and liver allocation? impact on mortality not only on the waiting list but also after orthotopic liver transplantation should be considered. *Hepatology* 2010; 52: 392- 393.
12) Bruns T, Nuraldeen R, Mai M, Stengel S, Zimmermann HW, Yagmur E, et al. Low serum transferrin correlates with acute-on-chronic organ failure and indicates short-term mortality in decompensated cirrhosis. *Liver Int* 2017; 37: 232- 241.
13) Maras JS, Maiwall R, Harsha HC, Das S, Hussain MS, Kumar C, et al. Dysregulated iron homeostasis is strongly associated with multiorgan failure and early mortality in acute-on-chronic liver failure. *Hepatology* 2015; 61: 1306- 1320.