DOI: 10.7860/JCDR/2019/41848.13218

Case Report

Pharmacology Section

Omeprazole Induced Increase in Liver Markers-A Case Report

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ABSTRACT

Proton pump inhibitors are one of the most widely used antisecretory agents used in the treatment of hyperacidity. These proton inhibitors have direct propulsive effect on gastrointestinal bacteria towards liver in absence of gastric acid. The report is about a patient suffering from Gastro-oesophageal Reflux Disease (GERD) receiving ranitidine and omeprazole. On measuring the biomarkers of liver function, the serum levels of Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Gamma Glutamyl Transferase (GGT), Albumin to Globulin Ratio (A/G ratio), cholesterol and creatinine were found to be increased. After cessation of the treatment with PPIs, diet regulation and usage of herbal products, the biomarker levels decreased and after another one month ALT and AST returned to normal.

Keywords: Gastroesophageal reflux, Gastric bacteria, Proton pump inhibitors

CASE REPORT

A 53-year-old man with gasterosophageal reflux complained of heartburn and hyperacidity manifestations especially after meals. Patient was treated with ranitidine 150 mg (twice daily) for 3 months but without any improvement. Later the physician added omeprazole (20 mg) once daily with ranitidine, for 6 months. Liver markers were done before starting omeprazole which found no abnormality; hepatitis markers were also negative.

Later during routine examination, assessment of liver function was ordered. The results were; Alanine aminotransferase (AST 95 U/L), Aspartate aminotransferase (AST 114 U/L), Gamma Glutamyl Transferase (GGT 100 U/L), increased albumin to globulin ratio (A/G ratio), cholesterol (5.15 mmol/L) and creatinine (116 umol/L).

The patient regulated his diet and used herbal products containing myrrh, as prescribed by the physician and both ranitidine and omeprazole were stopped. The herbal product was prescribed twice a day. On follow-up and re-analysis after two months, the liver enzyme levels were found to be decreased. The values were: ALT (48 U/L), AST (43 U/L) and total bilirubin (0.91 mg/dL). HCV (HCV Ab), HBV (HBs Ag) were negative.

DISCUSSION

GERD is one of the common gastrointestinal tract disorders. Gastric antisecretory agents such as H_2-receptor blockers and proton pump inhibitors are commonly used to block stomach acid secretions and relieve symptoms of frequent heartburn, acid reflux and GERD. These are also used in patients suffering from chronic liver diseases [1]. There is an earlier report that provides evidence that suppression of acid secretion in the stomach alters specific bacteria in gastrointestinal tract that may lead to the liver injury that may progress to chronic liver disease [2].

There are concerns that the suppression of gastric acid will alter the bacterial flora of upper gastrointestinal tract and lead to complications such as cancer, infections of the intestine or other organs and mal-absorption. However, Williams C and McColl KE reported that PPIs do alter the bacterial population [3].

Approximately, 10% of causes of drug induced hepatocellular injury manifested by jaundice result in fatality, so that rare instances of PPI-induced acute liver injury (which is typically hepatocellular) are likely to result in death. Acute liver failure with a short latency period (1 to 4 weeks) has also been reported with esomeprazole therapy, which is a PPI similar to omeprazole [4].

The use of PPIs in patients suffering from cirrhosis has been linked to the development of hepatic encephalopathy in three large, multi-centre, randomised trials [5]. PPI affect hepatic P-450 cytochrome through suppression of mono-oxygenases activities. They also induce cytochrome P-450 enzymes which potentiate the hepatotoxic effect of some drugs [6].

It has been proven that gut microbiome and the communities of bacteria and other microbes can influence liver disease risk. Further, suppression of acid secretion promotes growth of Enterococcus in the intestines which get translocated to the liver leading to exacerbation of inflammation and chronic liver disease. Hence, such reports recommended that the initial data should at least get people thinking about reducing their use of PPIs in cases where it can be avoided [1].

There are many cheap, available and reliable antacid which can alternate PPI like (Pepto-Bismol, Tums or H2-blockers like Zantac and Tagamet) suppress gastric acid to a lesser degree [7]. Other classes of antisecretory agents were also studied. Llorente C et al., reported that agents that suppress gastric acid effectively alter gut bacteria and thus augments progression of chronic liver disease [2]. H_2 blockers (e.g., Rantidine) are tolerated but it was found that there was a link between its usage and clinically apparent liver injury [8].

The non-pharmacological methods approached for management of heartburn may be an option in some patients; that include losing weight and reducing intake of pro-ulcerogenic factors such as alcohol, caffeine, and fatty and spicy foods [9]. Myrrh, the resin from Africa and Peninsula can regulate body weight, blood ammonia and other major liver and blood physiological parameters through production of several antioxidants and detoxifying proteins in liver, kidney and cerebrum [10,11]. This case report confirms Llorente's team results in mice about hepatic damage due to acid suppression.

CONCLUSION

PPIs may induce hepatic damage. Hence, people should reduce their use of PPI in cases where they aren't a necessity. Using alternative medicines like Myrrh can be considered along with non-pharmacological methods for managing heartburn like lowering body weight, reducing intake of caffeine and fatty and spicy foods.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: May 02, 2019
- Manual Googling: Jun 17, 2019
- iThenticate Software: Sep 18, 2019 (12%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:

- Financial or Other Competing Interests: No
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Apr 28, 2019**
Date of Peer Review: **May 20, 2019**
Date of Acceptance: **Jul 09, 2019**
Date of Publishing: **Oct 01, 2019**