EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 11
Issue: 5
351-353
Year: 2024

THE INFLUENCE OF OMEPRAZOLE ON LIPID PROFILE OF PATIENTS WITH ACID PEPTIC DISEASE

Neveen Ahmad Al-Alian Ph*, Nadia Ahmad Al Omari Ph*, Mohammad Bassam Jalayleh Ph*, Lujain Emad Nsour Ph*, Ghofran Nedal Al Alaween Ph*, Dr. Bilal Smadi and Laith Safadi*****

*Pharmacist, Royal Medical Services, Jordan.
**Consultant Gastroenterology, Royal Medical Services, Jordan.
***Medical Student, Jordan University of Science and Technology.

***Corresponding Author: Neveen Ahmad Al-Alian Ph**
Pharmacist, Royal Medical Services, Jordan.

Article Received on 22/03/2024 **Article Revised on 11/04/2024** **Article Accepted on 03/05/2024**

ABSTRACT

Objective: To evaluate the effect of omeprazole on the lipoprotein fractions of patients with acid peptic disease. **Patients and Methods:** Eighty five patients suffering from acid peptic disease who treated with omeprazole were enrolled in this prospective study. Prior to enrollment, all patients had an investigation of lipoprotein fractions (serum cholesterol, triglycerides, HDL and LDL), and only those with normal results were included in the research. A fasting blood test was obtained from all patients to evaluate lipid profile at zero, four, eight, twelve and sixteen weeks following therapy. **Results:** The total number of patients included in this study was eighty five; only sixty patients came for regular follow and were tested for lipid profile. There were forty three males and seventeen females. Age of patients ranged between 19-46 years (mean 32.1 years). Regarding the body weight of patients, it was ranging between 50-93 kg (mean 57.2 kg). None of the included patients had history of alcohol consumption. There was no objective or subjective side effects among the patients. Serum cholesterol, Triglycerides, LDL and HDL were within normal range after four, eight, twelve and sixteen weeks after treatment. **Conclusion:** Omeprazole had no significant impact on lipid profile of individuals with acid peptic illness when administered for a duration of twelve weeks. However additional trials with big number of patients are required.

KEYWORDS: Omeprazole, lipid profile, acid peptic disease.

INTRODUCTION

Omeprazole is a substituted benzimidazole derivative that acts as a gastric proton pump inhibitor, regulating intragastric acidity independently of stimuli.[1] It effectively inhibits both basal and stimulated gastric acid secretion, making it a potent antisecretory drug. This medication is highly effective in promoting the rapid healing of peptic ulcers and erosive esophagitis, as well as in reducing gastric acid hypersecretion in patients with Zollinger-Ellison syndrome.[2,3,4]

Omeprazole is a type of proton pump inhibitor that works by reducing the secretion of gastric acid through inhibiting the H+/K+-ATPase in the gastric parietal cells. By inhibiting this enzyme, the medication prevents the formation of gastric acid.[3,5,6]

The documented occurrence rate of adverse drug reactions linked to this medication among individuals residing in the Western world is around 1.1%.[2] Limited information exists regarding the adverse effects of this medication on lipoprotein fractions; nevertheless, patients with heart disease who are concurrently taking these medications may benefit from this effect.

This study was conducted to evaluate the effect of omeprazole on the lipoprotein fractions of patients with acid peptic disease.

MATERIAL AND METHODS

Eighty five patients suffering from acid peptic disease who treated with omeprazole were enrolled in this prospective study.

The patients were diagnosed with the following conditions: duodenal ulcer (33.2%), gastritis (29.8%), oesophagitis (22.7%), duodenitis (8.1%) and gastric ulcer (6.2%).

The diagnosis was established by obtaining a pertinent medical history, doing a comprehensive physical examination, and then confirming it with upper GI endoscopy. Alcohol use history was collected from all

patients. Patients who first used alcohol were instructed to abstain from alcohol as part of the treatment.

Prior to enrollment, all patients had an investigation of lipoprotein fractions (serum cholesterol, triglycerides, HDL and LDL), and only those with normal results were included in the research.

Patients with any other coexisting condition or undergoing concurrent medication were not included in the research.

A fasting blood test was obtained from all patients to evaluate lipid profile at zero, four, eight, twelve and sixteen weeks following therapy.

RESULTS

The total number of patients included in this study was eighty five; only sixty patients came for regular follow and were tested for lipid profile. There were forty three males and seventeen females. Age of patients ranged between 19-46 years (mean 32.1 years).

Regarding the body weight of patients, it was ranging between 50-93 kg (mean 57.2 kg).

None of the included patients had history of alcohol consumption.

There was no objective or subjective side effects among the patients.

Serum cholesterol, Triglycerides, LDL and HDL were within normal range after four, eight and twelve weeks after therapy. Table I

Table I: Lipid Profile After Four, Eight, Twelve and Sixteen Weeks of Therapy.

Lipid profile	0 week	4 week	8 week	12 week	16 week
Cholesterol (Normal 150-250 mg%)	210.1 ± 8.6	209.7 ± 8.88	211.3 ± 6.50	207.9 ± 5.4	215.2 ± 2.3
Triglycerides (Normal 60-I 60 mg%)	122.3 ± 12.8	121.1 ± 13.53	114.2 ± 8.92	118.3 ± 8.3	119.5 ± 9.4
HDL (Normal 25-75 mg%)	42.4 ± 0.3	42.4 ± 0.61	41.6 ± 0.64	42.1 ± 0.35	42.4 ± 0.2
LDL (Normal <150 mg%)	142.5 ± 6.5	142.3 ± 5.43	141.1 ± 5.56	144.5 ± 4.90	142.1 ± 5.2

DISCUSSION

Omeprazole decreases stomach acid output by a highly specific method of action. It generates specific dosage dependent suppression of the enzyme H+/K+-ATPase (the proton pump) at the secretory surface of the parietal cell. This action stops the last step of stomach acid production, and so suppresses both basal stimulated acid discharges regardless of the stimulus.[3,5,6]

Omeprazole has a rapid onset of action and relieves symptoms quickly and profoundly within two days in most of patients. It has bactericidal effect on helicobacter pylori and it can suppress the bacterium; the organism is implicated in gastritis and classified as ulcerogenic. The eradication of H. pylori has been linked with a long-term resolution of the condition known as peptic ulcers in virtually all cases.[7,8,9,10]

In the current study we did not detect any occurrence of subjective adverse medication reactions or any alterations in lipid profile with omeprazole. The overall incidence of adverse medication reactions owing to this medicine has been estimated in Western populations to be approximately 1.1%.[2]

However; in order to determine this incidence more participants are necessary to be included in the research.

Conversely no significant statistical variation was detected in the lipid profile with the usage of omeprazole.

The findings demonstrate that ranitidine, omeprazole had no significant influence on lipid profile when administered for a duration of twelve weeks. This is in agreement with literature.[2,9]

CONCLUSION

Omeprazole had no significant impact on lipid profile of individuals with acid peptic illness when administered for a duration of twelve weeks. However additional trials with big number of patients are required.

REFERENCES

1. Walan A. Omeprazole. Clin Gastroenterol, 1988; 2: 629-40.
2. McTavish D. Buckley MMT, Heel RC. Omeprazole. An updated review of its pharmacology and therapeutic use in acid related disorders. Drugs, 1991; 42: 138-70.
3. Mirshahi F, Fowler G, Patel A, Shaw G. Omeprazole may exert both a bacteriostatic and a bacteriocidal effect on the growth of Helicobacter pylori (NCTC 11637) in vitro by inhibiting bacterial urease activity. J Clin Pathol, 1998; 51: 220–4.
4. McGowan CC, Cover TL, Blaser MJ. The proton pump inhibitor omeprazole inhibits acid survival of

Helicobacter pylori by a urease-independent mechanism [corrected and republished article originally printed in Gastroenterology 1994; 107(3): 738–43]. Gastroenterology, 1994; 107: 1573–8.
5. Mauch F, Bode G, Malfertheiner P. Identification and characterization of an ATPase system of Helicobacter pylori and the effect of proton pump inhibitors [Letter]. Am J Gastroenterol, 1993; 88: 1801±1802.
6. Holtmann G, Layer P, Goebell H. Proton-pump inhibitors or H2-receptor antagonists for Helicobacter pylori eradication —a meta-analysis [letter]. Lancet, 1996; 347: 763.
7. Classen M, Damman HG, Domschke W, et al. Omeprazole heals duodenal but not gastric ulcers more rapidly than ranitidine. Hepatogastroenterology, 1985; 32: 243-5.
8. Walan A, Bader J-P, Classen M, et al. Effects of omeprazole and ranitidine on ulcer healing and relapse rates in patients with benign gastric ulcer. N EngIJf Med, 1989; 320: 69-75.
9. Castot A, Bidault I, Dahan R, Efthymiou ML. Evaluation of unexpected and toxic effects of omeprazole reported to the regional centres of pharmacovigilance during the first 22 post marketing months. Therapie, 1993; 48: 469-74.
10. Malfertheiner P**,** Bayerdorffer E, Diete U, Gil J, Lind T, Misiuna P, O'Morain C, Sipponen P, Spiller RC, Stasiewicz J, Treichel H, Ujszaszy L, Unge P, Zanten SJ, Zeijlon L. The GUMACH study: the effect of 1-wk omeprazole triple therapy on Helicobacter pylori infection in patients with gastric ulcer. Aliment Pharmacol Ther, 1999; 13: 703-712.