Content available at: https://www.ipinnovative.com/open-access-journals

IP International Journal of Comprehensive and Advanced Pharmacology

Journal homepage: https://www.ijcap.in/

Case Report

Raised liver enzyme in a patient receiving *Helicobacter pylori* triple regimen: A case report

Ajay Kumar[1], Dinesh Kansal[1,*], Swatantra Gupta[2], Atal Sood[1], Suman Bodh[1]

[1]*Dept. of Pharmacology, Dr. Rajendra Prasad Govt. Medical College & Hospital, Kangra, Himachal Pradesh, India*
[2]*Dept. of Gastroenterology & Hepatology, Dr. Rajendra Prasad Govt. Medical College & Hospital, Kangra, Himachal Pradesh, India*

ARTICLE INFO

Article history:
Received 21-04-2022
Accepted 22-04-2022
Available online 16-05-2022

Keywords:
H. pylori
Liver function
Liver enzymes

ABSTRACT

Helicobacter pylori infection remains a worldwide spread disease with a definitemorbidity and mortality.Indeed, this infection is the main cause of non-ulcer dyspepsia, peptic ulcers and gastric tumors, including bothlow-grade MALT-lymphoma and adenocarcinoma. A 65-year-old female patient visited outpatient department of Gastroenterology clinic for a routine follow-up after completion of 14-day triple drug therapy for *H. pylori*. After a few days of empirical treatment for raised liver enzymes, her liver enzymes normalized. Clinicians should be aware about the rare adverse event during *H. pylori* eradication treatment.

This is an Open Access (OA) journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprint@ipinnovative.com

1. Introduction

Helicobacter pylori infection remains a worldwide spread disease with a definite morbidity and mortality.[1] Indeed, this infection is the main cause of non-ulcer dyspepsia, peptic ulcers and gastric tumors, including both low-grade MALT-lymphoma and adenocarcinoma.[2–4]

The current standard triple therapies include a proton pump inhibitor (PPI), clarithromycin (500 mg) plus amoxicillin (1 g) or metronidazole/tinidazole (500 mg); all given twice daily for 7–14 days, as suggested in the international guidelines.[5,6] Fourteen day regimen is recommended where bacterial resistance rate is higher in population.

2. Case Report

A 65-year-old female patient visited outpatient department of Gastroenterology clinic for a routine follow-up after completion of 14 day triple drug therapy for *H. pylori*. Liver function test at the time of follow-up revealed raised liver enzymes.

She earlier had complaints of burning sensation epigastrium, nausea, and upper abdominal pain for 3-4 months; for which she underwent upper gastrointestinal endoscopic examination and biopsy was taken. She was confirmed with *H. pylori* infection by histopathological examination.

She was receiving tablet pantoprazole 40 mg twice daily oral, cap. amoxicillin 1000 mg twice daily per oral, and tab. clarithromycin 500 mg twice daily for the last 14 days for treatment of *H. pylori* infection.

Her baseline AST, ALT and alkaline phosphatase (ALP) were 56 IU/ml, 32 IU/ml, and 56 IU/ml respectively. After 14 days, her AST, ALT and ALP raised to 71.9 IU/ml, 133 IU/ml, and 86 IU/ml respectively (ALT >4 times after 14 days).

After a few days of empirical treatment for raised liver enzymes, her liver enzymes normalized.

* *Corresponding author*.
E-mail address: dinesh.kansal56@gmail.com (D. Kansal).

https://doi.org/10.18231/j.ijcaap.2022.020
2581-5555/© 2022 Innovative Publication, All rights reserved.

3. Discussion

Although, it has been reported that adverse effects due to the *H. pylori* triple regimen are mild and does not lead to discontinuation of therapy.[7] Recently, Hafeez et al reported that 76.2% of their patients experienced adverse events due to proton pump inhibitor, amoxicillin, and clarithromycin therapy.[8] The most common included alteration in taste followed by abdominal pain/diarrhea, and diarrhea.

Regarding our patient's drug history; it was found that liver enzymes were found to be raised after completion of *H. pylori* treatment. Since pantoprazole was not very likely the culprit, clarithromycin and amoxicillin were the drugs in suspicion. It has been known for many years that several antibiotics can cause severe hepatic injury.

In the case of the penicillins, the combination amoxicillin-clavulanate and the penicillinase-resistant penicillins oxacillin, (di-)cloxacillin, and flucloxacillin can cause (mainly cholestatic) hepatitis. There are case reports indicating liver failure may be caused by amoxicillin alone.[9]

Regarding clarithromycin, fulminant liver failure has been described in the literature.[10] As we know that clarithromycin is primarily metabolized in the liver, the patient information leaflet warns about administration of this drug in patients with advanced liver dysfunction. In patients with mild liver dysfunction, frequent monitoring of AST, ALT, GGT, alkaline phosphatase, and bilirubin is recommended. Since clarithromycin inhibits liver enzyme CYP3A4, clinicians have to be aware that plasma levels of drugs that are metabolized by this enzyme may increase. However, in our case, we could not study any drug interactions.

Weidmann et al reported that a case report about raised liver enzymes of *H. pylori* eradication in a patient with moderate chronic and moderate active pangastritis.[11] The liver dysfunction was self-limited and normalized with no treatment.

According to Naranjo adverse drug reaction probability assessment scale, clarithromycin was the probable drug responsible for causing hepatitis. Not many cases of amoxycillin induced hepatitis have been reported in literature

4. Conclusion

In summary, clinicians should be aware about the rare adverse event during *H. pylori* eradication treatment. Although only mild liver injury was detected in our patient, physicians should be prepared for rapid management of acute liver failure

5. Conflict of Interest

The authors declare no relevant conflicts of interest.

6. Source of Funding

None.

References

1. Sonnenberg A, Lash RH, Genta RM. National study of Helicobactor pylori infectionin gastric biopsy specimens. *Gastroenterology*. 2010;139(6):1894–901. doi:10.1053/j.gastro.2010.08.018.
2. Alakkari A, Zullo A, Hj O. Helicobacter pylori and nonmalignant diseases. *Helicobacter*. 2011;16:33–40.
3. Zullo A, Hassan C, Cristofari F, Perri F, Morini S. Gastric low-grade mucosal-associatedlymphoid tissue lymphoma: Helicobacter pylori and beyond. *World J Gastrointest Oncol*. 2010;2(4):181–6.
4. Fuccio L, Eusebi LH, Bazzoli F. Gastric cancer, Helicobacter pylori infection andother risk factors. *World J Gastrointest Oncol*. 2010;2(9):342–7. doi:10.4251/wjgo.v2.i9.342.
5. Malfertheiner P, Megraud F, O'Morain CA, Atherton J, Axon AT, Bazzoli F, et al. Management of Helicobacter pylori infection-the Maastricht IV/Florence Consensus Report. *Gut*. 2012;61(5):646–64. doi:10.1136/gutjnl-2012-302084.
6. Fock KM, Katelaris P, Sugano K, Ang TL, Hunt R, Talley NJ, et al. Second Asia-PacificConsensus Guidelines for Helicobacter pylori infection. *J Gastroenterol Hepatol*. 2009;24(10):1587–600. doi:10.1111/j.1440-1746.2009.05982.x.
7. Fischbach LA, Van Zanten S, Dickason J. Meta-analysis: the efficacy, adverse events, and adherence related to first-line anti-Helicobacter pylori quadruple therapies. *Aliment Pharmacol Ther*. 2004;20(10):1071–82.
8. Hafeez M, Qureshi ZA, Khattak AL, Saeed F, Asghar A, Azam K, et al. Helicobacter Pylori Eradication Therapy: Still a Challenge. *Cureus*. 2021;13(5):e14872. doi:10.7759/cureus.14872.
9. Maggini M, Raschetti R, Agostinis L, Cattaruzzi C, Troncon MG, Simon G, et al. Use of amoxicillin and amoxicillin-clavulanic acid and hospitalization for acute liver injury. *Ann Ist Super Sanita*. 1999;35(3):429–33.
10. Tietz A, Heim MH, Eriksson U, Marsch S, Terracciano L, Krähenbühl S, et al. Fulminant liver failure associated with clarithromycin. *Ann Pharmacother*. 2003;37(1):57–60. doi:10.1345/aph.1C171.
11. Wiedmann M, Müller C, Lobeck H. Sudden elevation of liver enzymes in a 64-year-old patient: a case report. *Cases J*. 2009;2:205. doi:10.1186/1757-1626-2-205.

Author biography

Ajay Kumar, Junior Resident

Dinesh Kansal, Professor and HOD

Swatantra Gupta, Assistant Professor

Atal Sood, Associate Professor

Suman Bodh, Principal Nursing Officer

Cite this article: Kumar A, Kansal D, Gupta S, Sood A, Bodh S. Raised liver enzyme in a patient receiving *Helicobacter pylori* triple regimen: A case report. *IP Int J Comprehensive Adv Pharmacol* 2022;7(2):106-107.