Journal of Drug Design and Discovery Research
JDDDR, 3(2): 116-117
www.scitcentral.com

Case Report: Open Access

Favipravir Induced Liver Injury

Subodh Kumar Mahto[1*], Kritika Gupta[2], Ankita Sheoran[2] and Nitasha Pascharicha[2]

*[*1]Anandi Prakash Hospital, Hospital Road, Sitamarhi, Bihar-843302, India*

[2]Atal Bihari Vajpayee Institute of Medical Sciences & DR.RML Hospital, New Delhi, India.

Received May 13, 2022; Revised June 26, 2022; Accepted June 29, 2022

ABSTRACT

The treatment for COVID-19 pneumonia is a challenge to evidence-based medicine. Based on various in vitro studies done in short course of time, multiple drugs were approved for the treatment with restricted emergency indication. Favipiravir being with one of the approved drugs in severe covid pneumonia. Here, we report a rare side effect of the drug Favipiravir: Hepatotoxicity.

Keywords: Drug induced liver injury, Favipiravir, COVID-19

CASE REPORT

A 32-year-old lady, with no known previous comorbidity, presented to our hospital with complains of SARI like illness for 3 days. On examination, she was conscious oriented, with temperature 99.8° F, pulse 90/min, BP 110/70 mm of Hg, RR 26/min and saturation of 88% on room air. Rest of the systemic examination was normal. As per the hospital protocol, patient was tested for COVID-19 that turned out to be positive and was clinically classified as Severe COVID-19 and shifted to ICU for management. Patient routine investigation and inflammatory markers were sent which were grossly normal except CRP of 26.8 mg/dl. She was started on guidelines directed management for COVID-19, with oxygen support, Inj. Enoxaparin 40 mg od, and Tab Favipiravir (600 mg on Day 1, followed by 400 mg BD from day 2-14). Patient was monitored with serial inflammatory markers and LFT, KFT every third day and a good clinical response was observed, with patient maintaining saturation of 94% off oxygen. However, on day 12 of illness, patient reported increasing yellowish discoloration of urine and dull aching pain in right upper quadrant. On evaluation LFT was deranged with AST/ALT 436/466 U/dl, ALP 246U/dl (R factor 6.2), Total Bilirubin 4.5 mg/dl, S. albumin 3.8 g/dl, which worsened over 48 h to a value of AST/ALT 956/779 U/dl and T. Bil: 15.3 mg/dl. Serum ammonia 126 µmol/L, INR 0.9. A panel of investigations were sent for evaluation of etiology and Tab Favipiravir was stopped and oral steroids were started. Patient tested negative for serology for EBV, CMV, HAV, HEV, HbsAg, Anti-Hbc, HIV, Plasmodium falciparum and vivax, rickettsia and leptospira. Blood cultures and S. procalcitonin was also negative. Ultrasound and doppler study of hepatobiliary system was also normal. Workup for Wilson was also negative. Autoimmune workup with ANA, Anti-LKM, AMA, ASMA antibodies were also negative. Patient gave negative consent for liver biopsy. Patient was managed conservatively with supportive therapy and was given Ursodeoxycholic acid as she complained of pruritus. On follow up tests, LFT started to normalize and complete recovery was seen at the end of 4 weeks. A possibility of DILI was kept secondary to use of Tab Favipiravir.

DISCUSSION

Liver injury can be multifactorial and many a times an overlapping etiology could be attributed to deranged liver function. A diagnosis of DILI is considered when some pharmacological cause is suspected and other etiologies have been ruled out, which could be dose dependent DILI or idiosyncratic and unpredictable [1]. Favipiravir, an antiviral, with uncertain clinical efficacy for COVID-19, was approved as restricted emergency drug in patients with severe COVID-19. This oral prodrug is a purine-based nucleoside analogue that inhibits the enzyme RNA dependent RNA polymerase of SARS-CoV-2 virus thereby

Corresponding author: Subodh Kumar Mahto, Anandi Prakash Hospital, Hospital Road, Sitamarhi, Bihar-843302, India, Tel: +918527083233; E-mail: drsubodhkr05@gmail.com

Citation: Mahto SK, Gupta K, Sheoran A & Pascharicha N. (2022) Favipravir Induced Liver Injury. J Drug Design Discov Res, 3(2): 116-117.

restricting the viral replication. Favipiravir is associated with mild self-limiting transaminitis [2]. A mild derangement of liver enzymes has also been observed in COVID-19, however its rarely progresses to state of acute hepatitis. In our patient, the baseline liver enzymes were normal and presented with deranged liver function test after the resolution of symptoms of COVID-19. Also, the symptoms completely resolved on withdrawing the drug, similar to case reported by Kumar [3]. So, we conclude that the possible cause of Hepatocellular pattern of liver injury in our case was favipiravir induced. Further studies are needed for identification of risk factors and mechanism of liver injury by favipiravir.

REFERENCES

1. Kuna L, Bozic I, Kizivat T, Bojanic K, Mrso M, et al. (2018) Models of Drug Induced Liver Injury (DILI) - Current Issues and Future Perspectives. Curr Drug Metab 19: 830-838.
2. Pilkington V, Pepperrell T, Hill A (2020) A review of the safety of favipiravir - a potential treatment in the COVID-19 pandemic? J Virus Erad 6: 45-51.
3. Kumar P, Kulkarni A, Sharma M, Rao PN, Reddy DN (2021) Favipiravir-induced Liver Injury in Patients with Coronavirus Disease 2019. J Clin Transl Hepatol 9: 276-278.