International Journal on Infectious Disease and Epidemiology

# Hepatitis B and C Virus Infection Among HIV Patients at Coxs Bazar ART Center, Bangladesh

*Research Article*

Volume 3 Issue 4- 2022

Author Details

*Yasir Arafat M[1]*, Bobby Z[2], Naz B[1], Nazir MS[1], Adnan ABM[1], Hussain MF[1] and Barua A[1]*

[1]*Coxs Bazar Medical College and Hospital, Bangladesh*
[2]*Coxs Bazar Medical College, Bangladesh*

*Corresponding author

Yasir Arafat M, Coxs Bazar Medical College and Hospital, Bangladesh

Article History

Received: September 19, 2022 Accepted: September 23, 2022 Published: September 26, 2022

## Abstract

**Background:** Coinfections of HIV patients with hepatitis B virus (HBV) and hepatitis C virus (HCV) are major public health problems, resulting in increased hepatic morbidity and mortality. Our study aimed to analyze rates of HBV and HCV infection among BD (Bangladeshi) and FDMN (Forcibly Displace Myanmar Nationals) living with HIV (Human Immunodeficiency Virus) attending ART (Anti-Retroviral Therapy) center in Coxs Bazar.

**Methods:** In this cross-sectional study, blood samples were tested for hepatitis B surface antigen (HBsAg) and anti-HCV antibodies from January 2018 to January 2021. Demographic and laboratory data were obtained from the patients attending at ART center.

**Results:** In this study, out of 304 patients, 69 (23%) patients were from BD and 235 (77%) from FDMN. Most (149,49%) of the patients were females with FDMN, whereas only 9.5% were female patients from BD (p=.005 with 95% CI). HBV-HIV coinfection rate was 1.44% and 2.55% among BD and FDMN. HCV-HIV coinfection rate was 1.6% and 44.7% respectively among BD and FDMN. The prevalence of HIV-HCV was significantly high (p=.000, 95% CI) among FDMN but similar among BD in comparison to multiple other studies. Most (30.14%) of the HCV-HIV co-infection among FDMN was high in the 31 to 40 years of age. Females have a higher likelihood of HCV-HIV infection than males among FDMN (OR=1.142, 95%CI 0.66-1.9). Significant differences (p=.000. 95% CI) were observed by mode of transmission in chronic hepatitis C infection in FDMN patients. Most of the cases of CHC transmission in PLHIV patients occurred by using unsafe injection (47%) followed by sexual transmission (28.67%) among FDMN people in our study. In our study, FDMN people who acquire HCV-HIV infection by unsafe injections and sexual relationships have 1.4 and 1.8 times more chance of likely having coinfection than those who acquire infection by an unknown cause. HBV-HCV-HIV co-infection among FDMN people was less than 1%.

**Conclusion:** HIV patients are at higher risk of HBV and HCV infection. This study will help to find out the prevalence of coinfection in the community. So, all HIV patients should be screened for HBV and HCV infection for proper selection of an Anti-retroviral regimen.

**Keywords:** HBV; HCV and HIV at Coxs bazar; HBV-HCV-HIV coinfection among BD and FDMN

## Introduction

With less than 0.1 percent of the population estimated to be HIV-positive, Bangladesh is a low HIV prevalence country [1]. UNAIDS estimates the number of adults and children with HIV in Bangladesh was around 14,000 (12,000-16,000) [2]. 240,000 people were living with HIV in Myanmar in 2018. After Thailand, Myanmar has the second-highest prevalence in Southeast Asia at 0.8% [3]. It is estimated that the HCV-HIV co-infection rate is 2-15% worldwide (and up to 90% among the People Who Inject Drugs (PWID) and the HBV-HIV co-infection rate is 5-20%. The global estimate of the burden of HIV-HCV co-infection is 2.75 million of whom 1.3 million are PWID and for HBV-HCV coinfection 2.6 million. The burden of these co-infections is highest in the African and Southeast Asia Regions [4]. The rate of coinfection is geographically heterogeneous and relies on various factors, for example, distribution of risk groups, execution of HBV vaccination programs, and ranges of endemicity in the general population [5-7].

Human Immunodeficiency Virus (HIV), Hepatitis B Virus (HBV), and Hepatitis C Virus (HCV) co-infection has emerged as a leading cause of morbidity due to liver disease throughout the world in the last two decades [7,8]. Among HIV-infected patients, HBV and HCV co-infections are more prevalent due to overlapping transmission routes [9]. Consequently, the presence of co-morbidities such as chronic liver disease due to HBV and HCV infection is being identified as critical problems.

In co-infection, the presence of one virus influences the natural course of the other virus. HIV accelerates the natural history of HBV and HCV infection and facilitates the faster progression of liver disease to cirrhosis and hepatocellular carcinoma. Disease progression to cirrhosis in HIV-positive patients is almost three times faster compared to HIV-negative patients [10-12]. Most of the studies [12-14] in HIV-HBV and HIV-HCV co-infected patients have been conducted among western patient populations. Understanding HBV-HCV co-infection with HIV is particularly necessary for Asian countries due to the high background prevalence of HBV and HCV [15].

Since HIV and HBV share the same transmission routes (mainly sexual), coinfection is frequent, mainly within key populations including men who have sex with men (MSM) [16]. The influence of HBV on the course of HIV infection and the success of Antiretroviral Therapy (ART) is controversial. Some studies suggest a slower HIV response, [17] while others report no impact on the progression to AIDS or response to ART [8,18,19].

The principal risk factor for HCV is Injecting Drug Use (IDU), while heterosexual intercourse is not a significant transmission route. MSM and women infected with HIV or other Sexually Transmitted Diseases (STDs) appear to have an increased chance of sexual HCV transmission [20,21]. The role of HCV as a co-factor in HIV disease progression remains controversial, but some studies have found lower absolute CD4+ cell counts as well as diminished immunological and virological responses following ART initiation [9,22,23].

As stated by the ECDC (European Center for Disease prevention and Control) prevention, care and treatment of HBV, HCV and HIV should be an integrated strategy to reduce disease-related mortality. The basis of such a strategy is the realization of the local epidemiology of these chronic infections [24]. Moreover, the presence of HBV and HCV infection with HIV also needs a specific regimen. The HIV-HBV co-infection should be treated with Tenofovir (TDF) and Lamivudine (3TC) along with antiretroviral therapy. Similarly, all HIV patients should be screened for hepatitis C and should be treated with combinations of sofosbuvir and velpatasvir along with antiretroviral therapy. The preferred first-line regimen for all adults/adolescents, pregnant or breast-feeding women is a three-drug regimen including tenofovir, lamivudine and dolutegravir [25]. The presented study analyzed the rates of HBV and HCV infection among adult PLHIV attending ART centers in Coxs Bazar, Bangladesh which is near the Myanmar border.

## Materials and Methods

In this cross-sectional (analytic) observational study, after obtaining informed written consent, serological tests had been performed to determine HIV using Rapid Antigen kits (Determine, first response, and Uni-gold). All confirmed HIV individuals were tested for HBsAg and Anti-HCV by using an immunochromatographic test or ELISA (Enzyme-Linked Immunosorbent Assay). The liver function test was assessed by measuring the SGPT level. All patients who agree and had available serology reports were included in this study. Age, sex, mode of transmission, HBV and HCV status, and SGPT were noted from January 2018 to January 2021. Outcome variables include the relationship of HBV and HCV with nationality, age, sex, mode of transmission, and SGPT level. The protocol of the study was approved by the Ethics Committee of Cox's Bazar medical college and hospital. Data were analyzed using SPSS (Statistical Package for the Social Sciences) 25. Categorical variables were summarized with absolute values & proportions at their 95% Confidence Interval (CI) and continuous variables were summarized as median with IQR (Interquartile range). Categorical variables of two groups were compared by Chi-square test and Fisher exact test and continuous variable by independent sample t-test. P-value was considered significant when it was less than .05 with 95% CI. For univariable logistic regression analyses, Odds Ratios (OR) and 95% Confidence Interval (CI) were calculated. Multivariable logistic regression was used to determine independent risk factors associated with co-infections, and Adjusted Odds Ratios (AOR) were calculated at a 95% Confidence Interval (CI).

## Results

A total of 304 PLHIV patients were included in this study, of which 235 (77%) were FDMN and 69 (23%) were BD (Table 1). The median age of the BD patients was 33 with an IQR of 26 to 41 and FDMN patients were 30 with an IQR of 22 to 45. The maximum number of patients were 21 to 30 years of age group, 19.7% from FDMN and 7.6% from BD (Figure 2). Most of the study patients were FDMN females (49%,149) whereas only (9.5%,29) were BD females. 28.3% were FDMN male and 13.2% were BD male. Age, gender, and sex distribution are shown in (Table 1). The number of HCV-HIV coinfection was much higher (57.87%) among FDMN than in BD patients (7.24%) (Table 2). HBV-HIV coinfection was found in 1.44% and 7.24% cases among BD and FDMN respectively (Table 3). HIV-HBV-HCV coinfection was found among FDMN people in 0.85% of cases (Figure 1). SGPT was elevated in 1.4% and 3% of cases of BD and FDMN patients who had HCV-HIV. In the case of HCV-HIV, sexual exposure (5.8%) was the highest mode of transmission among BD in comparison to unsafe injections (27.2%) among FDMN.

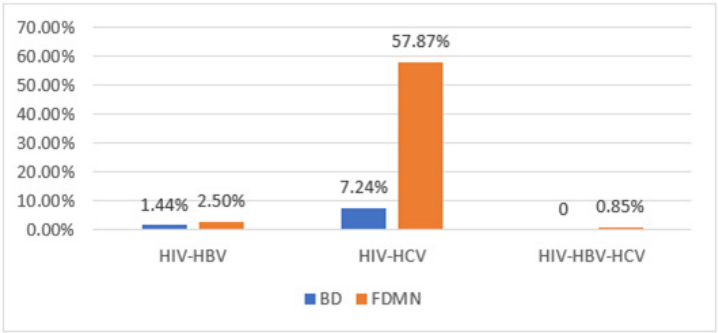


**Figure 1:** Rates of coinfection.

**Table 1:** Demographic Profile of HIV patients (n=304).

| | BD | FDMN | P-value |
|---|---|---|---|
| Total number (% of Total) | 69 (23%) | 235 (77%) | 0.006 |
| Age (median with IQR) | 33 (26 to 41) | 3 (22 to 45) | |
| **Age group** | | | |
| Less than 20 | 7 (2.3%) | 58 (19.1%) | 0.08 |
| 21 to 30 | 23 (7.6%) | 60 (19.7%) | |
| 31 to 40 | 22 (7.2%) | 53 (17.4%) | |
| 41 to 50 | 13 (4.3%) | 41 (13.5%) | |
| 51 to 60 | 3 (1%) | 20 (6.6%) | |
| 61 or more | 1 (0.3%) | 3 (1%) | |
| **Gender** | | | |
| Male | 40 (13.2%) | 86(28.30%) | 0.002 |
| Female | 29 (9.5%) | 149(49.0%) | |

**Table 2:** Characteristics of HCV-HIV coinfection.

| | BD (69) | FDMN (235) | P-value |
|---|---|---|---|
| Total affected | 5 (7.24%) | 136 (57.87%) | 0 |
| **Sex** | | | |
| Male | 5 (7.24%) | 48 (20.42%) | 0.003 |
| Female | 0 | 88 (37.44%) | |
| **Age Group** | | | |
| Less than 20 | 1 (1.44%) | 11 (8%) | 0.905 |
| 21 to 30 | 1 (1.44%) | 39 (28.67%) | |
| 31 to 40 | 1 (1.44%) | 41 (30.14%) | |
| 41 to 50 | 1 (1.44%) | 30 (22%) | |
| 51 to 60 | 1 (1.44%) | 14 (10.29%) | |
| 60 or more | 0 | 1 (0.73%) | |
| **Mode of Transmission** | | | |
| BT | 0 | 6 (4.4%) | 0.07 |
| IDU | 0 | 1 (0.7%) | |
| MTCT | 1 (1.44%) | 3 (2.2%) | |
| Sex | 4 (5.79%) | 45 (33%) | |
| Spouse | 0 | 1 (0.7%) | |
| Syringe | 0 | 77 (56.6%) | |
| Unknown | 0 | 3 (2.2%) | |
| **SGPT** | | | |
| Elevated | 1 (1.44%) | 15 (6.38%) | 0.001 |

**Table 3:** Characteristics of HBV-HIV infection.

| | BD (69) | FDMN (235) | P-value |
|---|---|---|---|
| Total affected | 1(1.44%) | 6 (2.55%) | |
| **Gender** | | | |
| Male | 1(1.44%) | 1 (0.42%) | 0.04 |
| Female | 0 | 5 (2.13%) | |
| **Age Group** | | | |
| Less than 20 | 0 | 2 (0.9%) | 0.07 |
| 21 to 30 | 0 | 2 (0.9%) | |
| 31 to 40 | 0 | 1 (0.4%) | |
| 41 to 50 | 1(1.44%) | 1 (0.4%) | |

| | | | |
|---|---|---|---|
| 51 to 60 | 0 | 0 | |
| 60 or more | 0 | 0 | |
| **Mode of Transmission** | | | |
| BT | 0 | 1 (0.4%) | 0.094 |
| IDU | 0 | 0 | |
| MTCT | 0 | 1 (0.4%) | |
| Sex | 1 (1.4%) | 1 (0.4%) | |
| Spouse | 0 | 0 | |
| Syringe | 0 | 3 (1.3%) | |
| Unknown | 0 | 0 | |
| SGPT (Elevated) | 0 | 2 (0.9%) | |

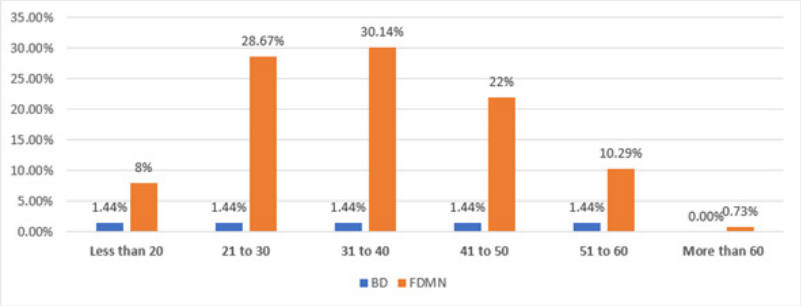


**Figure 2:** Distribution of HIV-HCV infection according to age group.

## Discussion

Most of the patients were FDMN (77%) whereas 23% were BD in this study. The median age of BD was higher (33) than FDMN, but the Interquartile ranges were wide among FDMN people (22 to 45), p=.006 with 95% CI. Most (49%) of the patients were females from FDMN, whereas only 9.5% were female patients from BD. (p=.005 with 95% CI) HBsAg status was found positive only in 1.44% cases of BD and 2.55 % cases of FDMN, both of which are low than the study done by Supanat et al. [26] among HIV patients in Bangkok, Thailand which was 6% [26]. Globally, the estimated prevalence rate of chronic hepatitis B with PLHIV is 7.4%, higher than our study [27]. Lonita et al. [28] found the prevalence of HBV-HCV co-infection among PLHIV in Nepal was 4.4% and 19% respectively [28]. However, the epidemiological data on HIV-HBV, HIV-HCV, and HIV-HBV-HCV co-infections among HIV patients are scattered and variable even within the different centers of the nation and abroad. The prevalence of HIV-HBV, HIV-HCV, and HIV-HBV-HCV co-infections was 3.62%, 2.93%, and 0.34%, respectively, in a study by Bhattarai et al. [29]. In North India, the prevalence of HBV and HCV co-infection among PLWH was 5.32% and 2.43%, respectively [30]. Similarly, in Hunan Province, China, the prevalence of HIV-HBV, HIV-HCV, and HIV-HBV-HCV co-infections among HIV patients was 9.27%, 9.98%, and 2.72%, respectively [31].

In Ghana, the prevalence of HIV-HBV, HIV-HCV, and HIV-HBV-HCV co-infections among HIV patients was 12.5%, 5.5%, and 18.0% respectively in a study by Boateng et al. [32] and was 6.1%, 0.5%, and 0.0% respectively in a study by Pappoe et al. [33] In the present study, HCV-HIV coinfection was 1.6% and 44.7% respectively among BD and FDMN. The prevalence of HIV-HCV was significantly high (p=.000, 95% CI) among FDMN but similar among BD in comparison to multiple other studies. One possible reason for high HCV-HIV coinfection among FDMN could be that the use of unsafe injectables in a rural area could be playing a significant role in the transmission of HCV compared to another route, which is also evident from our study. HBV-HCV-HIV infection among FDMN people was less than 1%, which was lower than the findings in the study conducted in Abuja city, the capital of Nigeria at 1.5% [34].

Most (30.14%) of the HCV-HIV co-infection among FDMN was high in the 31 to 40 years of age, but other study shows its highest in 51 to 60 years of age [35]. It may be due to multiple sexual partners and using sharing injections for treatment. But the exact reason needs to be elucidated from other prospective cohort studies. Females have a higher likelihood of HCV-HIV infection than males among FDMN (OR=1.142, 95%CI 0.66-1.9). But Lonita et al. [28] found the opposite where the males with HIV were 5.7 times more likely to have HIV-HCV co-infection [36].

Significant differences (p=.000. 95% CI) were observed by mode of transmission in chronic hepatitis C infection in FDMN patients. Most of the cases of CHC transmission in PLHIV patients occurred by using unsafe injection (47%) followed by sexual transmission (28.67%) among FDMN people in our study. But other study shows both CHB and CHC infection mostly occurred through IVDU among PLHIV [37]. In our study, FDMN people who acquire HCV-HIV infection by unsafe injections and sexual relationships have 1.4 and 1.8 times more chance of likely having coinfection than those who acquire infection by an unknown cause.

In most of the cases, HIV/HBV+ was higher than HIV/HCV+ [34,38]. But in our study HCV-HIV is higher than HIV-HBV among BD and FDMN. It means that, although the three viruses have similar routes of transmission; they are not transmitted at the same rate. HIV infection with a history of intravenous use or multiple transfusions has been consistently found to be the most important risk factor for HCV coinfection, while sexual transmission of HCV is relatively inefficient [39]. Prevalence of HIV/HBV is commoner among men who have sex

with men, heterosexual persons with multiple sex partners, or in contact with sex workers among others.

## Conclusion

HCV-HIV coinfection among the FDMN people was significantly higher in the present study. The physicians involved in the care of HIV/AIDS should be alert to screen for these infections frequently as well as select the appropriate treatment regimens. To find out the co-infection rate at the national level further study will be needed in other centers of the country.

## Limitation of the Study

There were a few limitations in our study. This study was conducted at one site, many of them are FDMN who are uneducated and have limited knowledge about HIV. Thus, our study sample might not represent the overall population of Bangladesh. Nucleic acid testing for HBV DNA and HCV RNA would have been ideal to confirm positive serological HBsAg and anti-HCV test results respectively. But this technique is expensive and not readily available in our center. We did not perform HCV RNA in anti-HCV-positive clients; therefore, the observed anti-HCV seroprevalence may be higher than the actual infection rate.

## Conflict of Interest

The author declares no conflict of interest.

## Acknowledgement

This work was made possible by the help of ART corner staffs, RMO Dr. Shaheen Abdur Rahman Chy and lab staffs of hospital. Also, special thanks to the Director of the hospital.

## References

1. WHO, HIV/AIDS, Published on 25 July 2019.
2. (2020) HIV/AIDS in Bangladesh.
3. (2019) UNAIDS data.
4. https://www.who.int/hiv/topics/hepatitis /hepatitis info/en/
5. Alter MJ (2006) Epidemiology of viral hepatitis and HIV co-infection. J Hepatol 44(1 Suppl): 86-89.
6. Singh KP, Crane M, Audsley J, Avihingsanon A, Sasadeusz J, et al. (2017) HIV-hepatitis B virus coinfection: epidemiology, pathogenesis, and treatment. AIDS 31(15): 2035-2052.
7. Martins S, Livramento A, Andrigueti M, KretzerI F, Machado M J, et al. (2014) The prevalence of hepatitis B virus infection markers and socio-demographic risk factors in HIV-infected patients in Southern Brazil. Rev Soc Bras Med Trop 47(5): 552-558.
8. Jones R, Duming J, Nelson M (2005) HIV and hepatitis with co-infection. Int J Clin Pract 59: 1082-1092.
9. Rockstroh JK (2006) Influence of viral hepatitis on HIV infection. J Hepatol 44(1Suppl): S25-S27.
10. Alter MJ (2006) Epidemiology of viral hepatitis and co-infection. J Hepatol 44(Suppl 1): S6-S9.
11. Mocroft A, Ledergerber B, Katlama C, Kirk O, Reiss P, et al. (2003) Decline in AIDS and death rates in EuroSIDA study; an observational study. Lancet 362(9377): 22-29.
12. Soto B, Sanchez Quijano A, Rodrigo L, del Olmo JA, García-Bengoechea M, et al. (1997) Human immunodeficiency virus infection modifies the natural history of chronic parentally acquired hepatitis C with an unusually rapid progression to cirrhosis. J Hepatol 26(1): 1-5.
13. Vallet-Pichard A, Pol S (2006) Natural history and predictors of severity of chronic hepatitis C virus (HCV) and human immunodeficiency virus (HIV) co-infection. J Hepatol 44(1 Suppl) : 528-534.
14. Konopnicky D, Mocroft A, de Wit S, Antunes F, Ledergerber B, et al. (2005) Hepatitis B and HIV: Prevalence, AIDS progression, response to highly active antiretroviral therapy and increased mortality in the EuroSIDA cohort. AIDS 19(6): 593-601.
15. Amin J, Kaye M, Skidmore S, Pillay D, Cooper DA, et al. (2004) HIV & hepatitis C co-infection within the CAESAR study. HIV Med 5(3): 174-179.
16. Hoffmann CJ, Thio CL (2007) Clinical implications of HIV and hepatitis B coinfection in Asia and Africa. Lancet Infect Dis 7(6): 402-409.
17. Idoko J, Meloni S, Muazu M, NimzingL, Badung B, et al. (2009) Impact of hepatitis B virus infection on human immune deficiency virus response to anti-retroviral therapy in Nigeria. Clin Infect Dis 49(8): 1268-1273.
18. Konopnicki D, Mocroft A, deWit S, Antunes F, Ledergerber B, et al. (2005) Hepatitis B and HIV: prevalence, AIDS progression, response to highly active antiretroviral therapy and increased mortality in the Euro SIDA cohort. AIDS 19(6): 593-601.
19. Omland LH, Weis N, Skinhoj P, Laursen A, Christensen PB, et al. (2008) Impact of hepatitis B virus co-infection on response to highly active antiretroviral treatment and outcome in HIV-infected individuals: a nationwide cohort study. HIV Med 9(5): 300-306.
20. Tohme R A, Holmberg S D (2010) Is sexual contact a major mode of hepatitis C virus transmission? Hepatol-ogy 52(4): 1497-1505.
21. McFaul K, Maghlaoui A, Nzuruba M, Farnworth S, Foxton M, et al. (2015) Acute hepatitis C infection in HIV-negative men who have sex with men. J Viral Hepat 22(6): 535-538.
22. Portocarrero Nunez J A, Gonzalez-Garcia J, Berenguer J, Gallego M J V, Loyarte J A I, et al. (2018) Impact of co-infection by hepatitis C virus on immunological and virological response to antiretroviral therapy in HIV-positive patients. Medicine 97(38): e12238.
23. Weis N, Lindhardt BO, Kronborg G, Hansen AB, Laursen AL, et al. (2006) Impact of hepatitis C virus coinfection on response to highly active antiretroviral therapy and outcome in HIV-infected individuals: a nationwide cohort study. Clin Infect Dis 42(10): 1481-1487.
24. European Centre for Disease Prevention and Control (2018) Public health guidance on HIV, hepatitis B and C testing in the EU/EEA.
25. National Centre for AIDS and STD Control (NCASC) (2021) HIV epidemic update of Nepal, and/or National HIV Testing and treatment guidelines of Nepal.
26. Thitipatarakorn S, Chinbunchorn T, Peelay J, Seekaew P, Amatavete S, et al. (2022) Prevalence and the associated factors of hepatitis B and hepatitis C viral infections among HIV-positive individuals in same-day antiretroviral therapy initiation program in Bangkok, Thailand. BMC Public Health 22(1): 144.
27. World Health Organization (2021) Global hepatitis report 2017. Geneva: World Health Organization; 2017.
28. Lonita G, Malviya A, Rajbhandari R, Schluter WW, Sharma G, et al. (2017) Seroprevalence of hepatitis B virus and hepatitis C virus co-infection among people living with HIV/AIDS visiting antiretroviral therapy centres in Nepal: a first nationally representative study. International Journal of Infectious Diseases 60: 64-69.
29. Bhattarai M, Baniya JB, Aryal N, Shrestha B, Rauniyar R, et al. (2018) Epidemiological Profile and Risk Factors for Acquiring HBV and/or HCV in HIV-Infected Population Groups in Nepal. BioMed Research International 2018: 9241679.
30. Gupta S, Singh S (2006) Hepatitis B and C virus co-infections in human immunodeficiency virus positive North Indian patients. World J Gastroenterol 12(42): 6879-6883.
31. Su S, Fairley CK, Sasadeusz J, He J, Wei X, et al. (2018) HBV, HCV, and HBV/HCV co-infection among HIV-positive patients in Hunan province, China: Regimen selection, hepatotoxicity, and antiretroviral therapy outcome. Journal of medical virology 90(3): 518-525.
32. Boateng R, Mutocheluh M, Dompreh A, Obiri-Yeboah D, Anto EO,

et al. (2019) Sero-prevalence of Hepatitis B and C viral co-infections among HIV-1 infected ART-naïve individuals in Kumasi, Ghana. PLoS One 14(4): e0215377.

33. Pappoe F, Hagan CKO, Obiri-Yeboah D, Nsiah P (2019) Sero-prevalence of hepatitis B and C viral infections in Ghanaian HIV positive cohort: a consideration for their health care. BMC Infectious Diseases 19(1): 380.

34. Adewole OO, Anteyi E, Ajuwon Z, Wada I, Elegba F, et al. (2009) Hepatitis B and C virus co-infection in Nigerian patients with HIV infection. J Infect Dev Ctries 3(5): 369-375.

35. Seyed Alinaghi SA, Jam Sara, Mehrkhani F, Fattahi F, Sabzvari D, et al. (2011) Hepatitis-C and Hepatitis-B Co-Infections in Patients with Human Immunodeficiency Virus in Tehran, Iran. Acta medica Iranica 49(4): 252-257.

36. (2021) National Immunization Program (NIP) of Nepal.

37. Shrestha LB, Yadav GK, Pradhan S, Sharma A, Pandit T, et al. (2022) Co-infection of Hepatitis B and Hepatitis C among HIV-infected patients: A cross-sectional study from tertiary care hospital of eastern Nepal. PLoS One 17(3): e0264791.

38. Diwe CK, Okwara EC, Enwere OO, Azike JE, Nwaimo NC, et al. (2013) Sero-prevalence of hepatitis B virus and hepatitis C virus among HIV patients in a suburban university teaching hospital in south-east Nigeria. PAMJ 16: 7.

39. Tedaldi EM, Hullsiek KH, Malvestutto CD, Arduino RC, Fisher EJ, et al. (2003) Prevalence and characteristics of hepatitis C virus coinfection in human immunodeficiency virus clinical trials group: the Terry Beirn community programs for clinical research on AIDS. Clin Infect Dis 36(10):1313-1317.