Original Research Article

A study of prevalence of HCV/HIV Co-Infection in a Tertiary Health Care Centre in Punjab

Sanjay Goyal[1], R.P.S. Sibia[2], Vikas Sharma[3*], Anjali Chawla[4], Ajay Pal[5], Mallika Jain[6], Aarzoo Chawla[7]

[1]Assistant Professor,[2] Professor and Head,[3] Assistant Professor,
[4&5] Senior Resident,[6] Junior Resident,[7] MBBS student
Department of Medicine, Government Medical College, Patiala, Punjab, India

Corresponding Author:
Dr. Vikas Sharma, Assistant Professor
Department of Medicine, Government Medical College, Patiala, Punjab, India

Article History:
Received: 2nd November 2019
Received in revised form: 13th November 2019
Accepted: 16th November 2019

Abstract:

Background: Coinfection with HIV and HCV is common since both infections share similar routes of transmission. Co-infection with hepatitis C virus and human immunodeficiency virus is common in certain populations. Among HCV (+) persons, 10% are also HIV (+), and among HIV (+) persons, 25% are also HCV (+). Many studies have shown that in intravenous drug users, co-infection prevalence can be as high as 90%-95%. There is increasing evidence supporting the concept that people infected with HIV have a much more rapid course of their hepatitis C infection. Treatment of co-infection is often challenging because highly active anti-retroviral therapy (HAART) is frequently hepatotoxic, especially in the presence of HCV. The purpose of this review is to describe the effects that HIV has on hepatitis C and the treatment options in this challenging population. This article shows the prevalence of Hepatitis C in HIV patients reported to Government Medical College/ Rajindra Hospital, Patiala.

Methods: 4924 HIV positive patients registered under ART centre of GMC / Rajindra Hospital Patiala from 1st October, 2018 to 30th September, 2019 were included in this study. HCV positive cases were detected by Elisa IgM and HCV RNA detection.

Results: Out of 4924 HIV patients screened for HCV, 673 (13.66%) patients were positive for HCV. Out of which 473 (70.28%) were males and 200 (29.7%) were females. Age distribution was done among these reactive patients and it was found that maximum number of HCV and HIV co-infected patients (383) were among the age group of 20 -40 years, which was 56.9% of total HCV and HIV co-reactive patients.

Conclusion: HCV infection is common in HIV reactive patients. This coinfection is important due to common modes of infection in these two populations and prognostically due to early and rapid progression of liver disease in these patients.

Key Words: Coinfection, prevalence, hepatitis.

Introduction:

India is the second largest populated country in the world with more than 1.1 billion people [1]. Globally, a total of 39.5 million were living with HIV in 2006, of whom approximately 5.7 million (3.4-9.4 million) were in India [2]. Acquired immunodeficiency syndrome has grown more rapidly than the scientific progress of understanding how to control the main causative agent. Globally, hepatitis C virus (HCV) has infected more than 170 million people [3] and thus represents a viral pandemic seven times more widespread than infection with the HIV. Hepatitis C virus (HCV) is a globally prevalent pathogen and a leading cause of death and morbidity .The most recent estimates of disease burden show an increase in sero prevalence over the last 15 years to 2.8%, equating to >185 million infections worldwide.2 Persistent HCV infection is associated with the development of liver cirrhosis, hepatocellular cancer, liver failure, and death, and HCV is now the most common cause of death in HIV-positive patients on highly active antiretroviral therapy(4) In a meta-analysis of over 780 studies evaluating HIV-infected populations

worldwide, the overall prevalence of HCV coinfection was estimated to be approximately 6%. Globally, an estimated 80% of HIV-infected individuals with a history of injection drug use have evidence of HCV coinfection [5]. In the United States and in European countries, it is estimated that approximately the prevalence of HIV/ HCV coinfection in the HIV population ranges from 30 to 50%. [6] Vertical transmission of HCV appears to be facilitated by HIV coinfection. A meta-analysis of 10 studies demonstrated that maternal coinfection increases the odds of vertical HCV transmission by approximately 90 percent compared with maternal HCV infection alone [7]. HIV/ HCV-coinfected patients are less likely to clear viral infection, have more rapid rates of fibrosis, and have a higher risk of hepatic decompensation compared with HCV mono-infected patients[8]. The introduction of potent ART has been associated with a decline in liver-related mortality and slower rates of fibrosis progression [9]. ART may slow down disease progression due to immune reconstitution[10] HIV and HCV show some common biological features like both are RNA viruses and both show a large heterogenicity of their viral genomes producing various genotypes. These viruses also have some differences, like HCV belongs to the Flaviviridae family and HIV to the Retroviridae family. Falviviruses have a single RNA strand whereas retroviruses have double RNA strands. The HIV-RNA, transcripted to DNA by the reverse transcriptase (RT), integrates in the infected cell's genome, constituting the integrated provirus; this integration is the cause of the irreversibility of HIV infection. In contrast, the HCV genome does not integrate into the cell's genome and the replication of the virus takes place in the liver cell's cytoplasm. This non-integration makes it easier to eradicate HCV and hence to cure the infection. These viruses share similar routes of transmission like through blood and blood products, sharing of needles to inject drugs and sexual route.

Previously, blood transfusion was a major mode of HCV transmission but now that donor blood is thoroughly screened for the virus, majority of the cases are injectable drug users. HCV is also transmitted perinatally, by improperly sterilized dialysis equipment (68% [11] of the cases) and by unprotected sex with an infected partner. Cohort studies report that men who have sex with men (MSM) and those with other sexually transmitted infections are at a greater risk of contracting HCV from unprotected sex. An estimated 20% of people with chronic HCV infection will progress to cirrhosis over a 20-50-year interval. [12] A greater proportion of HIV/ HCV coinfected people may progress to cirrhosis and liver disease than those with HCV alone. [13] HIV-infected individuals have a high probability of getting coinfected with HCV. HIV disease progression and enhanced immunosuppression has a direct bearing on the natural history and pathogenesis of these infections. There have been some reports of the prevalence of HCV infection in HIV infected patients in various populations in India, but a lot more has to be done in this field.

Aims and Objectives:

The objective in this study, therefore, was to determine the prevalence of HCV infection in HIV-infected Indian population. This is aimed at providing the baseline data on HIV/ HCV coinfection. In order to gain a better understanding of the public health issues in these countries, we evaluated the anti-HCV antibody and HCV RNA in 4972 confirmed HIV-positive individuals in ART center, GMC/ Rajindra Hospital, Patiala.

Methods:

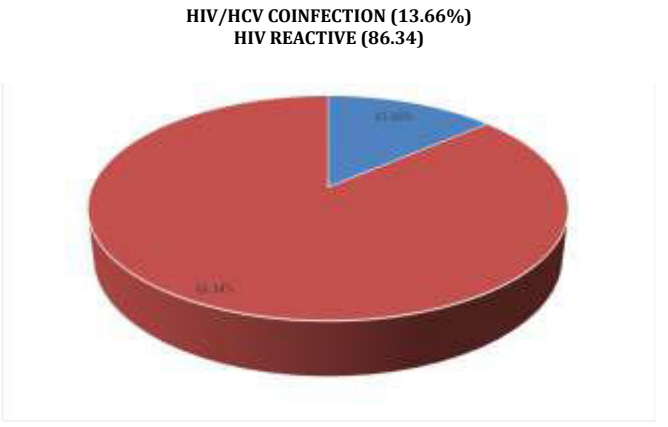
This Study was conducted at GMC/ Rajindra Hospital, Patiala. The study was conducted during 1st October 2018, to 30th September 2019 among 4972 HIV reactive patients who were registered under ART center of GMC/ Rajindra Hospital Patiala. Serologic specimens of patients were obtained and tested for anti-HCV antibody, HCV Ribonucleic acid (RNA) on anti-HCV positive samples.

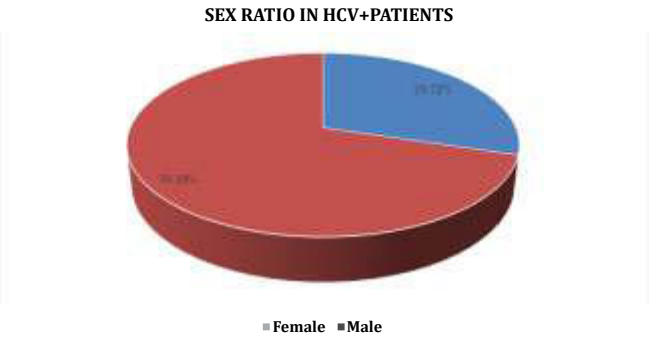
RESULTS:

We report the prevalence of HIV-HCV coinfection in patients who reported to Rajindra Hospital, GMC/ Patiala from 1st October 2018-30th September 2019.

4924 HIV patients registered under ART centre of GMC/ Rajindra Hospital, Patiala were tested for Anti HCV antibodies and HCV viral load, Out of these 673(13.66%) came out to be HCV reactive. 473(70.28%) weremales and 200(29.72%) were females. Age distribution was also done in these patients and maximum cases (56.9%) were found in age group 20 to 40 year.

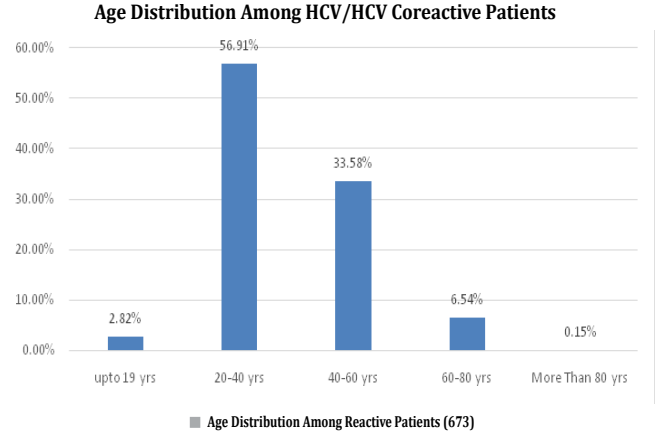
HIV/HCV Coinfection





Age Distribution among HIV and HCV Co Reactive Patients (673)

Age Group in Years	Reactive Patients	Percentage
Up to19	19	2.82
20-40	383	56.91
40-60	226	33.58
60-80	44	6.54
More Than 80	1	0.15



Discussion:

All HIV-infected persons should be screened for HCV infection using enzyme immunoassays; those with antibody to HCV should have quantitative HCV RNA testing, which confirms the presence of viremia and serves as abaseline prior to therapy [14]. HIV-associated factors that have been associated with fibrosis progression include CD4cell count <200 cells/ microl, HIV viremia, and lack of ART (15,16).

HCV antibodies usually develop 6 weeks to 6 months after infection. All positive HCV antibody results should be confirmed by testing for HCV RNA. Anti-HCV test is not adequate to diagnose chronic HCV infection, however, as some patients spontaneously clear the virus without treatment but remain antibody positive, HCV viral load (HCV RNA) test is necessary to confirm or rule out chronic HCV infection. Studies have reported spontaneous viral clearance rates from 15 to 45% in HIV-negative persons.[17] Although spontaneous viral clearance is less likely to occur among people who are HIV positive, some, particularly those with higher CD4 cell counts, do spontaneously clear HCV infection.[18] In Indian studies, there is a paucity of information of HIV/ HCV coinfection. The global coinfection studies reported so far have been variable, depending on the geographical area, type of exposure and the risk behaviour groups. The present investigation was a prevalence study, not an incidence study. In our study, prevalence of coinfection with HCV among known HIV patients was 13.66%.

In a study of 122 HIV/ HCV-coinfected and 122HCV-monoinfected patients, matched for age, sex, alcohol use, age at HCV infection, and duration of HCV infection, the prevalence of extensive liver fibrosis onbiopsy was greater among coinfected patients (54 versus 30percent in mono infected patients) [19].HCC is an emerging complication in the HIV-infected population. Proportions of liver-related deaths among HIV-infected patients that were attributable to HCCincreased from15 to 25percent in France from 2000to 2005 [20]. Similarly, in a study from Spain, the incidence of HCC among HIV-infected patients increased from 0.1 to 1.1 cases per 1000person years between1999 and 200915. Almost all of the cases were associated with HCV coinfection. In a prospective, observational cohort in the United States, the incidence of liver cancer was eight fold higher in HIV-infected patients compared with the

general population[21]. One study also demonstrated that the rate of CD4 cell gains was lower among patients who had chronic HCV infection compared with those who had spontaneously cleared their viremia [22]. Those with HCV/ HIV coinfection may experience greater rates of nonhepatic complications compared with HIV-infected patients who are HCV uninfected. Such complications include osteoporosis or bone fractures and chronic kidney disease[18-19]. Medical management in coinfection will be improved by enhancing HCV detection, with annual serologic testing, screening with HCV RNA to detect acute infection, and HIV testing of HCV-infected individuals.

Conclusion:

This study highlights the importance of mandatory HCV testing in all HIV-infected individuals. Where there is paucity of information on HIV/ HCV coinfection prevalence, particularly in developing countries like India, our study provides a useful insight to researchers working on HIV/ HCV coinfection. Finally, further studies of HIV/ HCV coinfection are needed to explore in more detail for prevention strategies and therapeutic management of this condition. This study may also create awareness of HIV/ HCV coinfection. This study further helps in early diagnosis, management of this condition and also helps in achieving W.H.O. goals of elimination of hepatitis C by 2030.

Funding: Nil

Conflict of Interest: None

References:-

1. Provisional Population Totals: India. Census of India 2001. Series 1, Paper 1 of 2001.
2. UNAIDS/ WHO 2006 Report on the global AIDS epidemic.
3. WHO, Global surveillance and control of hepatitis C. Report of a WHO consultanion organized in collaboration with the viral hepatitis prevention board, Antwerp, Belgium. J Viral Hepat 1999; 6:35-47
4. Goyal S, Sachdeva S, Singh G, Aggarwal R, Kaur S. A survey determining the different genotypes of Hepatitis C amongst subjects reporting to a tertiary care centre in Punjab. Int J Med Research Prof 2018 Jan; 4(1);283-85.
5. PlattL, Easterbrook P, Gower E, etal. Prevalence and burden of HCV co-infection in people living with HIV:aglobal systematic review and meta-analysis. Lancet Infect Dis 2016;16:797.
6. Mauss S, Rockstroh JK. HCV/ HIV-coinfection - is there a state of the art after APRICOT and RIBAVIC? J Antimicrob Chemother 2005;56:615-8.
7. Polis CB, Shah SN, Johnson KE, Gupta A. Impact of maternal HIV coinfection on the vertical transmission of hepatitis Cvirus: ameta-analysis. ClinInfect Dis 2007; 44:1123.
8. KimAY, Chung RT. Coinfection with HIV-1 and HCV--aone-two punch. Gastroenterology 2009;137:795.
9. Bräu N, Salvatore M, Ríos-Bedoya CF, etal. Slower fibrosis progression in HIV/ HCV-coinfected patients with successful HIV suppression using antiretroviral therapy. J Hepatol 2006;44:47.
10. Anderson KB, GuestJL, Rimland D.Hepatitis C virus coinfection increases mortality in HIV-infected patients in the highly active antiretroviral therapy era: data from the HIV Atlanta VA Cohort Study. ClinInfect Dis2004;39:1507.
11. Alter MJ. Prevention of spread of hepatitis C. Hepatology 2002; 36:S93-
12. Lauer GM, Walker BD. Hepatitis C virus infection. N Engl J Med 2001; 345:41-52.
13. Bonacini M, Puoti M. Hepatitis C in patients with human immunodeficiency virus infection: Diagnosis, natural history, meta-analysis of sexual and vertical transmission and therapeutic issues. Arch Intern Med 2000;160:3365-73.
14. StraderDB,WrightT, Thomas DL, etal. Diagnosis, management, and treatment of hepatitis C. Hepatology 2004;39:1147.
15. Kim HN, Nance R, Van Rompaey S, etal. Poorly Controlled HIV Infection: An Independent Risk Factor for Liver Fibrosis. J AcquirImmune Defic Syndr2016;72:437.
16. Newsum AM, Kooij KW, Boyd A, etal. Progressiono fliver fibrosis following acute hepatitis C virus infectionin HIV-positive MSM. AIDS 2019;33:833.
17. Augenbraun M, Goedert JJ, Thomas D, Feldman J, Seaberg EC, French AL, *et al* . Incident hepatitis C virus in women with human immunodeficiency virus infection. Clin Infect Dis 2003; 37:1357-64.

18. Salmon-Ceron D, Rosenthal E, Lewden C, etal. Emerging role of hepato cellular carcinoma among liver-related causes of deaths in HIV-infected patients: The Frenchnational Mortalité 2005 study. JHepatol2009;50:736.

19. Merchante N, Merino E, López-Aldeguer J, etal. Increasing incidence of hepato cellular carcinoma in HIV-infected patients in Spain. Clin Infect Dis 2013;56:143.

20. Patel P, Hanson DL, Sullivan PS, etal. Incidence of types of cancer among HIV-infected persons compared with the general population in the United States, 1992-2003. AnnIntern Med 2008;148:728.

21. Potter M, Odueyungbo A, Yang H, etal. Impact of hepatitis C viral replication on CD4+T-lymphocyte progression in HIV-HCV coinfection before and after antiretro viral therapy. AIDS 2010;24:1857.

22. Dong HV, Cortés YI, Shiau S, Yin MT. Osteoporosis and fractures in HIV/hepatitis C virus coinfection: a system aticre view and meta-analysis. AIDS2014;28:2119.

23. O'Neill TJ, Rivera L, Struchkov V, etal. The effect of HIV-hepatitis C co-infection on bone mineral density and fracture: ameta-analysis. PLoSOne 2014;9:e101493.