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Research Article

Alcohol: is it detrimental to HIV infected individuals on antiretroviral therapy?

Kavita S. Joshi*, Gajanan Chavan

Department of Medicine, Seth G S Medical College, Parel, Mumbai, India

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***Correspondence:**
Dr. Kavita S. Joshi,
E-mail: kavitajoshi@kem.edu

ABSTRACT

Background: Chronic Alcohol consumption is immunosuppressant and HIV causes immunodeficiency. Both in same patient can have detrimental effect. HIV can progress rapidly in an alcoholic patient. This study is looking for adverse effects of alcoholism on CD4 response, adherence and adverse drug reactions on patients who are on highly active antiretroviral therapy.
Methods: A prospective observational study done over period of one year after local institutional ethics committee approval was obtained. All consecutive HIV infected male patients presenting for first time and indicated to start on HAART above age of 18 years who were willing to give consent were recruited for this study. Patients with Hepatitis B and Hepatitis C were excluded from the study. Results were analyzed using ANOVA and paired T test.
Results: The CD4 counts remained lower in habitual alcoholics compared to social alcoholic and nonalcoholic study groups, P value being 0.0385 (habitual alcoholic); 0.0291 (social alcoholic) and 0.0251 (non-alcoholic) respectively. It was found (13%) alcoholic patients; 12 (8%) social alcoholic and 4 (2.7%) Non-alcoholic patients were not maintaining pill counts. 36 (24.5%) of total Patients were not maintaining pill counts. Patients consuming alcohol even did not follow up nor followed instructions, P value being 0.000.
Conclusions: Alcoholic patients were not following regularly for treatment. Hence we can conclude that alcohol has negative effect on adherence to HAART. Therefore in our part of country alcoholics should be considered as high risk groups for HIV care.

Keywords: Alcohol, HIV, HAART

INTRODUCTION

Alcohol consumption has gone up in urban India due to increased social acceptance. Alcohol per capita in 15+ years has increased from 3.6 in 2005 to 4.3 (in litres of pure alcohol) during the year 2010. The prevalence of heavy episodic drinking is 11 % in drinkers and 1.7 % in population as per WHO data.

Alcohol use in an individual causes mitochondrial damage. The increase in reactive oxygen species (ROS) production associated with ethanol metabolism damages mitochondrial components and also affects hepatocyte viability. Both protein oxidation and increases in mtDNA fragmentation can be considered consequences of increased ROS production associated with ethanol consumption. As the age advances the damage due to the alcohol increases, due to increased susceptibility of the mitochondrial genome to oxidative damage in older individuals. Indeed, in the alcoholic, large deletions of mtDNA similar to those seen in very old individuals are observed, suggesting that alcohol abuse leads to

premature aging of the mitochondrial genome.[1] Alcohol is immunosuppressant acting directly through T cell apoptosis, mitochondrial damage, and inhibition of T cell responses, natural killer (NK) cell activity, and macrophage phagocytic activity. Indirectly alcohol also suppresses the immune system through alcohol-induced malnutrition and liver disease.

Globally 37 million people are living with HIV (PLHIV) AIDS, 54% of the HIV positive individuals are aware of their illness. 17 million of them are receiving highly active antiretroviral therapy (HAART) in developing countries. The total number of people living with HIV/AIDS in India was estimated at around 20.9 lakh in 2011, 86% of whom were in 15-49 years age-group. The estimated number of PLHIV in India has maintained a steady declining trend from 23.2 lakh in 2006 to 20.9 lakh in 2011.[2] Nationally, the prevalence rate for adult HIV prevalence among the general population in 2015 is 0.36 %

Females are 0.29 % while for males it is 0.43 per cent. This means that for every 100 people living with HIV and AIDS (PLHIV), 61 are men and 39 women. Prevalence is also high in the 15-49 age group (88.7% of all infections), indicating that AIDS still threatens the cream of society, those in the prime of their working life. Because of its effects on immunity, there has been concern that alcohol consumption among HIV individuals may increase susceptibility to opportunistic infections and accelerate disease progression.

Alcohol abusers may be at increased risk for infection due to risky sex practices compared with non-abusers, either before or at the time of exposure, increases susceptibility to infection and hastens the progression from asymptomatic HIV infection to full-blown AIDS. This study is aimed at studying effect of alcohol on HIV disease progression and treatment outcome.

METHODS

A prospective observational study was conducted in Seth G S Medical College, KEM Hospital, Parel, Mumbai over period of one year. Local Institutional ethics committee approval was obtained. All consecutive HIV infected male patients presenting for first time and indicated to start on HAART above age of 18 years who were willing to give consent were recruited for this study. Patients with hepatitis B and hepatitis C were excluded from the study. We included only male patients in our study in view of our social & cultural setting where prevalence of alcoholism in females is extremely low.

Study procedure

All the subjects were divided in to three groups;

Group HA-habitual alcoholic (HA): Frequent Alcohol use (>60 gm/day)-2 or more drinks/day.

Group SA-social alcoholic (SA): Moderate Alcohol user (<60 gm/day)-1 or less drinks /day.

Group NA-non-alcoholics (NA): Abstainers. Upon enrolment in study, each subject was assigned a unique number i.e. Personal Identification Number (PIN). For example: HA 2O-11, there by meaning 20th subject of group HA 11 means year of study. A proper folder was maintained for each Individual subject, bearing subjects identification number (PIN).

The end point of study was completion of study duration or death of patient. Detailed history demographic parameters were recorded at the time of recruitment. Through clinical examination was done and patients were followed up for 6 months. At the end of six months all data was recorded again.

Statistical analysis

Statistical analysis was done using ANOVA, paired T-test.

RESULTS

Total number of patients presented for HAART during this period was 593. Out of these there were 431 male patients and 162 female patients. Study included male patients exclusively.

Patients were divided in three groups demographic data was matched. Number of patients in each group is as follows: HA=47, SA=48 and NA=50. 50 consecutive non-alcoholic (NA) patients were selected to make comparison precise. Age group 30 to 40 & 40 to 50 years comprised major portion of patients. There were 79.31 % patients in age group 30 to 50 years. There were 41.37% patients in age group 30 to 40 years & 37.93% patients were in age group 40 to 50 years. We recruited 145 HIV-seropositive patients and followed each patient for 6 months. Median CD4 cell count was 168.2553 (habitual alcoholic) 238.06 (social alcoholic) 226.62 (non-alcoholic) & 211.49 cells/µl of all patients. In the study patients the 29 % (28 out of total of 95 patients) of patients consumed beer.

At presentation (Table 1 & 2) the patients were having CD4 counts 200 & above at presentation value of CD4 count distribution was0.045 and 0.036 respectively. Figure 1, at presentation oral candidiasis was present in 5 habitual alcoholic subjects (10.64%), 5 social alcoholic subjects (10 %) and 2 non-alcoholic subjects (4.17%) (p value=0.456). Herpes zoster was present in 3 habitual alcoholic (6.38 %), 3 social alcoholic subjects (6 %) and 2 non-alcoholic subjects (4.17%) (p value=0.816). 2 social alcoholic subjects (4%) had toxoplasmosis at presentation (p value=0.212) Cryptococcal meningitis was present in 2 habitual alcoholic subjects (4.26%) (p value=0.104). Only 1 habitual alcoholic subject (2.13 %) had *Pneumocystis jirovecii* pneumonia, (p value=0.324).

Herpes simplex was present in 1 social alcoholic subject (2%) at presentation, (p value=0.325). Since p values of all opportunistic infections are >0.05 it indicates, occurrence of different opportunistic infections in different study group were similar. At follow-up after 6 months 6 habitual alcoholic subjects (12.77%), 1 social alcoholic subject (5%), had oral candidiasis, (p value=0.03) 1 habitual alcoholic subject (2.13 %) had herpes zoster at follow-up after 6 month (p value =0.324). Toxoplasmosis was present in 1 habitual alcoholic subject 2.13% (P value=0.324). When compared statistically for candidiasis p value is 0.003(<0.05) which shows significant statistical association with habitual alcoholics. Occurrence of herpes zoster & toxoplasmosis was similar. Table 3, describing the signs and symptoms, also the adverse drug reaction (ADR) after 6 months of therapy. The adherence was accessed by monthly follow up, pill count and weather patient is following given instructions (Table 4).

Table 1: CD4 count.

At Presentation CD4	N	Mean(cells/mm$^{3)}$	Std. Deviation	Std. Error	Minimum	Maximum
Habitual alcoholic	47	168	102.91374	15.01151	21.00	460.00
Social alcoholic	48	238	166.23828	23.99443	14.00	845.00
Non alcoholic	50	226	3.26789	25.35231	3.00	943.00
Total	**145**	**211**	**155.76768**	**12.93580**	**3.00**	**943.00**
After 6 months CD4	N	Mean	Std. Deviation	Std. Error	Minimum	Maximum
Habitual alcoholic	47	271	144.83809	21.12681	23.00	672.00
Social alcoholic	48	376	194.10741	28.01699	37.00	816.00
Non alcoholic	50	365	204.67890	28.94597	64.00	1026.00
Total	**145**	**338**	**188.21883**	**15.63072**	**23.00**	**1026.00**

Table 2: Distribution of patients according to CD4 counts groups.

CD4 count cells/mm^3	No habitual alcoholic (n)	Social Alcoholic(n)	Non-alcoholic (n)	Total	P value
At presentation					
0-100	17 (11.7%)	6 (4.13%)	8 (5.51%)	31(21.37%)	0.045
101-200	13 (8.96%)	16 (11.03%)	19 (13.10%)	48 (33.10%)	
>200	17 (11.76%)	26 (17.93%)	23 (15.86%)	66 (45.52%)	
After 6 months of HAART					
0-100	4 (2.76%)	2 (1.38%)	3 (2.07%)	9(6.21%)	0.039
101-200	13 (8.96%)	4 (2.76%)	5 (3.45%)	22 (15.17%)	
>200	30 (8.97%)	42 (28.97%)	42 (28.97%)	114 (78.62%)	
Total	47	48	50	145	

Table 3: Signs and ADRs.

	Habitual alcoholic		Social alcoholic		Non Alcoholic	
	Baseline	**On treatment**	**Baseline**	**On treatment**	**Baseline**	**On treatment**
Nausea	34.04	10.64	27.08	25	16	0
Vomiting	27.66	10.64	22.92	25	16	0
Anaemia	23.4	19.15	10.42	8.33	16	16
Peri.neuropathy	19.15	2.13	6.25	2.08	4	2
Nephrotoxicity	0	0	0	0	2	0
Hepatotoxicity	0	8.51	2.08	8.33	0	0
Lipodystrophy	4.26	2.13	0	6.25	2	0
Hyperglycemia	0	0	2.08	2.08	4	2
Skin rash	0	0	2.08	0	0	2

Table 4: Adherence.

	Habitual alcoholic	Social alcoholic	Non alcoholic	P Value
Following monthly	31 (66%)	31 (66%)	46 (92%)	0.001
Pill count >85%	27 (57%)	36 (75%)	46 (92%)	0000
Following Instructions	31 (66%)	28 (58%)	45 (90%)	0.001
Total	47	48	50	

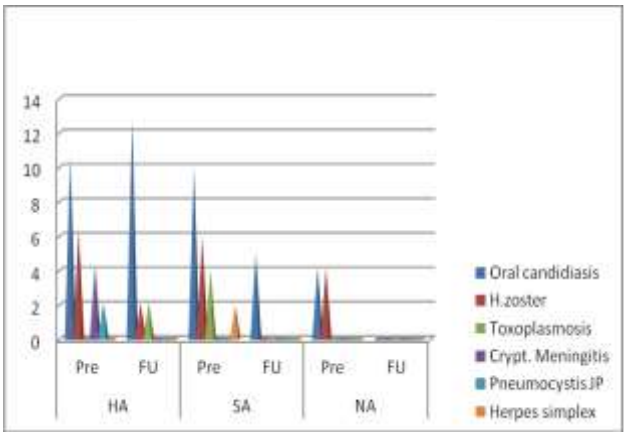

Figure 1: Opportunistic infections at presentation.

DISCUSSION

The majority of patients (89%) in our study who were habitual alcohol drinkers at baseline reported alcohol intake in more than half of their follow-up visits. Of these, 67% reported daily or more than daily drinking and 26%reported continued alcohol intake. We found that habitual alcohol consumption is a predictor of CD4 cell decline, as evidenced by a significantly greater difference of CD4 cell counts in patients who were on ART. Thus, habitual alcoholic patients had less mean presenting CD4 count compared to the SA and NA. In our study, habitual alcoholics were not significantly different from social alcoholics or non-alcoholics in factors such as stage of HIV disease. 67% of the patients in study were in stage 3 of disease. Therefore is seems the healthcare facilities available were similar for all patients. We found; though CD4 counts of all three groups showed increasing trend average response in CD4 were more in SA and NA groups. Increase in CD4 counts of habitual alcoholics can be attributed to effects of ART.[3,4] A recent study found that CD4 cell counts in heavy alcohol users were lower compared to non-alcoholic patients. They were not on ART. It is supporting evidence for an effect of alcohol on acceleration of HIV disease progression. This finding is consistent with our results of a more chances of CD4 counts remaining lower in habitual alcoholics compared to social alcoholic and nonalcoholic study groups independent of ART. P value was 0.0385 (habitual alcoholic); 0.0291 (social alcoholic) and 0.0251(non-alcoholic) respectively.

The data was analyzed at two intervals i.e.at presentation and at follow up at 6 months. It was found that the significant difference in CD4 counts of our study groups indicating CD4 counts had increased in all three subject groups but the increase was lower in alcoholic patients. Since the data was collected on two time instances for the same patients; it is analyzed using paired t-test. The paired t –test is carried out for each group (p value 0.011) this finding is comparable to study conducted by Samet JH, Horton NJ, Meli SJH et al.[3] In our study we found CD4 counts were increasing in all study groups but it was found to be at lower rate in alcoholic patients. We can consider here that habitual alcohol intake was predictive of a faster decline of CD4 cells progressively in active alcohol users though more of them were compliant to treatment. This is consistent with findings from a recent report of data from the HIV alcohol longitudinal cohort (ALC) and the HIV Longitudinal Interrelationships of Viruses and Ethanol study (LIVE). It can be considered that habitual alcohol use is a risk factor for accelerating HIV disease progression. It is supported by a faster decline of CD4 cell count. However as our study was conducted over short duration; actual decline could not be proved statistically. Even moderate adverse effects of heavy alcohol intake on CD4 cell count are masked by beneficial effects of ART on CD4 cell count. Positive changes in CD4 counts has occurred more in patients who had CD4 counts in range of 100 to 200 (p value 0.036).

A cross-sectional study found lower CD4 cell counts & significantly higher HIV viral load in those who were on ART and drank heavily after controlling for ART adherence.[5] Our findings of an effect of habitual alcohol intake on CD4 cells in patients with ART are similar to this cross-sectional study. We compared CD4 counts of habitual alcoholic with social and non-alcoholics; CD4 cell counts of social alcoholic patients were not significantly different from those of non-alcoholic patients. This is similar to finding of study that did not find a significantly lower CD4 cell count in "light" drinkers, defined as once weekly or less.[4] In our study we have defined social alcoholics as those taking one or less drink per day (<60 gm/day); findings of our study can be compared with findings of Samet et al study who also concluded that there is no association of moderate alcohol intake with CD4 cell count.[8] The earlier cross-sectional study done by Samet JH, Horton NJ, Meli S et al, showed significantly lower CD4 cell count and higher HIV viral load in both moderate and at-risk drinkers, compared to abstainers, even after controlling for ART adherence.[3] The discrepancies in the research findings with moderate alcohol use require further investigation. These results suggest that the effect of alcohol on CD4 cells is through metabolic pathways and is independent of the deleterious effect that alcohol might have on adherence to antiretroviral. We attempted to find effect of duration of alcoholism on HIV disease progression with respect to CD4 cell counts; there was statistical difference in average CD4 counts of different study patients (p value

0.045). CD4 counts of habitual alcoholic patients were lower compared to CD4 counts of social alcoholic & non-alcoholic patients though they had similar duration of illness. This is similar to observations by Samet JH et al who stated that HIV positive patients who were not on ART had lower CD4 counts than ART taking alcoholic patients.[3] All our patients were ART naïve before enrolment in our study and alcoholic. Patients had lower CD4 counts at presentation. On comparing CD4 counts of alcoholic patients with CD4 counts of nonalcoholic patients who were HIV positive for similar duration; it was found that Alcoholic patients had lower CD4 counts; implying that alcoholism had Negative effect on CD4 counts and is risk factor for rapid HIV disease progression.[6] We found pulmonary tuberculosis to be more common opportunistic infection. 30 (20.68%) patients in our study patients (n=145) had tuberculosis. 9 (6%) patients among all patients reported to have experienced pulmonary Tuberculosis in past. 21 (14.5%) patients had pulmonary tuberculosis at presentation (n=145). 11 (52%) out of these were habitual alcoholic. Understanding HIV-TB co-infection is of great importance because of increasing prevalence of co-infection, severity of clinical presentation of TB in HIV-positive. Prevalence of HIV among patients with radiologic or bacteriologic confirmation of TB in India ranges from 2.8 to 9.4 per cent.9 In India, the most common opportunistic infection among people with HIV Infection is pulmonary tuberculosis.10,11 Our study shows follow-up incidence of pulmonary tuberculosis had decreased; only 10 (6%) out of 145 patients developed pulmonary tuberculosis after Starting ART. Kumarasamy et al reported that 35% patients had pulmonary tuberculosis.[7] This difference can be attributed to their larger study size (6815). We again tried to find relation of alcoholism with incidence of Pulmonary tuberculosis; here we found 21 (70%) out of 30 patients who had Pulmonary tuberculosis were alcoholic (habitual & social). The risk of developing TB after an infectious contact is 5-10 per cent per year among HIV Infected individuals compared to 5-10 per cent during the lifetime of HIV negative individuals.[12] As alcohol is also found to have deleterious effect on immunity we can say that alcoholism had affected immunity negatively and resulted in higher incidence of tuberculosis in patients consuming alcohol.[13] Unlike cryptococcal meningitis or toxoplasmosis, which occur at very low CD4 counts, TB is unique in that it can occur over a wide range of CD4 counts, Although it is more frequent at CD4 counts <300 cells/µl. The typical presentation of cough, sputum, dyspnea, fever, and weight loss with apical lobe infiltrates or cavitatory lesions on chest radiograph might only be seen in patients with high CD4 counts whose immune systems are more comparable to HIV-uninfected individuals. TB in HIV patients with CD4 counts less than 300 cells/µl can present in an atypical pattern. Chest radiographs can also be normal in immune compromised patients despite presence of mycobacterium tuberculosis in sputum. Extra-pulmonary tubercular manifestations occur in 46 to 79 per cent of patients. Pulmonary TB and HIV and is more frequent in severely immune compromised patients.[14,15] In our study also there were 34 (23.44%) patients who reported extra Pulmonary manifestation of tuberculosis (n=145). Among patients with extra pulmonary tuberculosis 28 (82.35%) had reported to be consuming alcohol; indicating significant role of alcoholism in worsening outcome of HIV infection.

One study in India, extra pulmonary TB was the cause of 69 per cent of previously unexplained prolonged fever in 100 HIV positive patients.[16] In another study, severe weight loss in HIV patients, defined as loss of greater than 10 per cent of body weight in one month, significantly correlated with diagnoses of pulmonary and extra Pulmonary tuberculosis Just as HIV infection can contribute to the severity of TB.[5] There is increasing evidence that TB can affect HIV disease progression. We should also be aware of immune reconstitution syndrome (IRS), which is described as a paradoxical worsening of clinical status after initiation of HAART in a patient with an active opportunistic infection such as TB. In our study only one subject reported *Pneumocystis jirovecii* pneumonia. Reasons for this could be the predominance of other pulmonary diseases like TB and due to under diagnosis of incident cases. PCP occurs in patients with CD4 counts <200 cells/µl. Oral candidiasis occurs frequently in individuals with HIV infection; it has been reported as the most common HIV-associated condition, experienced in up to70 per cent of cases.[7] In our study we had only 12 (6%) patients with oral candidiasis at presentation and 7 (4.8%) patients reported to have oral candidiasis; amounting to 13% of all. All patients having oral candidiasis were consuming alcohol. There was only one subject with herpes simplex. Herpes simplex lesions are the third. 3 patients were identified with toxoplasmosis and there were 9(6.21%) Patients having herpes zoster with increasing affordability and accessibility of HAART, as use of ART has increased it has come out with broad spectrum of toxicities resulting from HIV therapy. While the safety, tolerability and efficacy of HAART regimens have been established, there are associated toxicities that can treatment outcome In HIV positive patients. In our study we observed significant ADRS associated with the use of HAART. Nausea, vomiting, anemia &hepatitis were most prevalent reported ADR in our study. Most ADRS were moderate and needed symptomatic treatment in some patients. Adverse effects in form of skin rash were reported more in patients on nevirapine-containing HAART regimen. Skin rash developed in 2(1.38%) out of 145 patients both of them were on nevirapine containing regimen. 16.55% (24/145) were having hematological abnormalities in form of anemia. All of these patients were on zidovudine containing ART regimen. Peripheral neuropathy was observed in 9.6% (14/145) patients. They were on stavudine based containing regimen. 11.7% (17/145) patients reported nausea, vomiting also experienced by 11.7% patients (17/145). Majority of the patients experienced vomiting after ingestion of zidovudine based

regimen. Finding study by Khalili H, dashtikhavidaki hepatotoxicity was reported by 8 patients (5% n=145).[17] Hepatotoxicity was defined as total bilirubin greater than 1.5. As Nevirapine also have adverse effects on hepatocytes. Alcoholism can be considered to be responsible for increased risk of developing hepatotoxicity. Though p value was >0.05; development of hepatotoxicity more in alcoholic patients implies association of alcoholism with hepatotoxicity. In our study; 3 (2.06%) patients developed lipo-dystrophy, 3 (2.06%) patients reported hyperglycemia and only 1 (0.69%) subject had nephrotoxicity. We could not correlate these finding with any specific study. Statistical analysis did not reveal any significant difference in occurrence of ADRS (p value >0.05 for all ADRS) among different subject groups. Alcohol consumption is not found to have any influence over ADRS to ART in our study.

We also attempted to find effect of alcoholism on adherence to ART. For this purpose we asked patients to maintain diary of events e.g. Day of taking alcohol; time of taking tablets daily; maintaining pill count; day of follow up for ART. We found 20 (13%) alcoholic patients; 12 (8%) social alcoholic and 4 (2.7%) Non-alcoholic patients were not maintaining pill counts. 36 (24.5%) of total Patients were not maintaining pill counts. Patients consuming alcohol even did not maintain regular diary. P value being 0.000; signifies alcoholic patients were more non-adherent to treatment. 24.5% (36/145) patients were not maintaining pill count. Out of these 88.88% (32/36) patients were alcoholic. 25.52% (37/145) patients were not following regularly. 89.18% (33/37) patients not following regularly were alcoholic. 28.2% (41/145) patients were not taking tablets as per prescribed schedule. Most of the patients (87.81%; 36/41) not taking tablets as per prescribed schedule were alcoholic. Alcohol consumption has been associated with non-adherence in a wide variety of settings and patient groups, with estimates of effect ranging from odds ratios 1 of 1.7 to 4.7.18. In other words, alcohol consuming patients were 1.7 to 4.7 times more likely to exhibit non-adherence than were abstainers.[19] We also studied if patients were particular in terms of taking tablets on prescribed time; we found 16 (11%) habitual alcoholic patients 20 (13.7%) social alcoholic 5(3%)non-alcoholic patients had missed doses on scheduled time of day (p value=0.001). On further observation it was evident that more of the doses were missed either on day or following day of consuming alcohol. Our observations are similar Braithwaite and colleagues study who described that regardless of HIV status, consumption of alcohol on a particular day was associated with decreased adherence to medications on that day and on the following day (i.e., the post drinking day).[20] We Can conclude that alcohol consumption has causal association in making patients non-adherent to treatment. Regarding follow up for ART we found 16 (11%) habitual alcoholic 17 (11.7%)social alcoholic & 4 (25%) non-alcoholic patients were not following regularly to ART OPD (p value=0.001). Out of 37 patients who were irregular in follow up 33 (89%) were from alcohol consuming group. This implies alcohol consumption has decreased compliance. Decreased compliance leads decreased response to ART; elaborated by decrease in CD4 counts. Decrease in CD4 counts leads to worsening of symptoms and progression of disease to terminal stage. CD4 counts of different study groups (habitual alcoholic, social alcoholic and non-alcoholic) were different statistically implying alcoholism has negative impact on CD4 counts of HIV positive male alcoholic patients.

CD4 counts being surrogate marker of disease it would have been more effective if we could consider viral load for this purpose. Larger sample size and longer duration of follow up can give statistically significant results.

CONCLUSIONS

CD4 counts of different study groups (habitual alcoholic, social alcoholic and non-alcoholic) were different statistically implying alcoholism has negative impact on CD4 counts of HIV positive male alcoholic patients. Opportunistic infections were more common in alcoholic patients in our study. Tuberculosis was found to be more common opportunistic infection in our study population. There was no statistical difference in adverse drug reactions among and occurrence of OIs different study groups when analyzed statistically. We cannot conclude whether alcoholism has negative impact on adverse drug reactions to HAAT Alcoholic patients were non adherent to HAART compared to non-alcoholic patients. They were more irregular in taking tablets as per prescribed schedule. Alcoholic patients were not following regularly for treatment. Hence we can conclude that alcohol has negative effect on adherence to HAART. Therefore in our part of country alcoholics should be considered as high risk groups. Special attention can be given for de-addiction of these patients. Life style modification and de-addiction will be useful in better compliance in turn for better disease control.

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