Differences in clinical features of HIV/HCV co-infected individuals compared with HCV mono-infected individuals in Southern Thailand

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Abstract

Objectives HIV/HCV co-infection is associated with more aggressive liver than HCV mono-infection. However, there are limited data in Thailand. This study was done to compare demographic data, basic laboratory results and hepatic fibrosis between HIV/HCV co-infected patients and HCV mono-infected patients in a major tertiary care center in Southern Thailand. Methods This was a cross-sectional single center 2-year retrospective study of HCV-treatment-naïve HIV/HCV co-infected patients and HCV mono-infected patients in Songklanagarind Hospital in Southern Thailand. The records of demographic data, basic laboratory results and hepatic fibrosis information were noted from all eligible patients. Results Data from 151 treatment-naïve HCV infected patients during the years 2018-2019 were collected. 51(34%) patients had HIV/HCV co-infections. Genotype 3a was predominant in HCV mono-infected patients(51%), while genotype 1a was predominant in HIV/HCV co-infected patients(35%). The median BMI in HCV mono-infected patients was higher than in HIV-HCV co-infected patients(23.1, IQR 20.3-24.8 vs 20.7, IQR 18.9-22.1, p<0.001). In multivariate analysis, HIV/HCV co-infected patients had higher alcohol consumption(AOR 24.9, p< 0.001), higher rates of unsafe sex(AOR 18.6, p=0.004) and MSM (AOR 8.48, p=0.006), and higher percents of genotype 1a(AOR 8.48, p=0.006) and HCV-RNA(AOR 2.3, p=0.035), while HIV/HCV co-infected individuals were less likely to be married (AOR 0.02, p<0.001), had higher median BMI(AOR 0.82, p=0.02), a higher rate of hepatic steatosis as assessed by controlled attenuation parameter (CAP)(AOR 0.98, p=0.02) and hepatic fibrosis as assessed by FibroScan 502(AOR 0.91, p<0.001) than HCV mono-infected patients. In HIV/HCV co-infected individuals, hepatic fibrosis as evaluated by liver stiffness measurement was not correlated with level of CD4 in our study. Conclusions HIV/HCV co-infected individuals were associated with genotype 1a, unsafe sex, single status and low BMI, while HCV mono-infected individuals were associated with high CAP and high hepatic fibrosis. Large-scale cohort studies are needed to confirm these results, especially hepatic fibrosis in this era of high-fat food consumption.

Introduction

Hepatitis C virus (HCV) infection is a global public health concern, with approximately 70 million infected patients worldwide in 2019(1). Infected individuals will eventually develop chronic hepatitis, cirrhosis and hepatocellular carcinoma resulting in liver-related mortality(2). The prevalence of HCV infection and its particular genotype varies between different countries and regions. In Thailand, recent surveys in 7 of Thailand's 76 provinces found approximately 759,000 anti-HCV + individuals and 357,000 who had viremic HCV infection (3, 4), with genotype 3a predominant (36.4%). For genotypic distribution in Thailand, genotype 3 was predominant in southern Thailand, while, genotype 6 was more common in the North than the other regions(5).

Human immunodeficiency virus is also a major global public health problem. Thirty-five million people are estimated to be infected worldwide with nearly 520,000 in Thailand(6). HIV and HCV share a common route of transmission and thus co-infection is not uncommon, and among the 35 million HIV-infected individuals worldwide, 4–5 million (11–14%) are co-infected with HCV. The prevalence of HIV/HCV co-

infection depends on the mode of acquiring HIV infection, with approximately 50% of patients acquiring their HIV infection through intravenous drug use (IVDU) and 5–20% through men who have sex with men (MSM)(7, 8). An HIV/HCV co-infected patient is mainly characterized by a faster progression to liver cirrhosis that may lead to hepatic decompensation or the development of hepatocellular carcinoma (HCC) at a younger age(9). In addition, HIV/HCV co-infection results in higher mortality rates, especially in patients with low CD4 counts and high HIV viral loads(10–12).

To date there are limited data on HIV/HCV co-infections in Thailand, which is a problem because knowing genotype distributions and the rate of hepatic fibrosis in HCV-infected individuals with or without an HIV co-infection are essential for guiding optimal treatment regimens. To help fill this knowledge gap, in this study we aimed to evaluate demographic data, basic laboratory results and hepatic fibrosis among HIV/HCV co-infected individuals compared to HCV mono-infected individuals in Southern Thailand.

Material And Methods

Study procedure

Demographic and laboratory data of HIV/HCV co-infected patients and HCV mono-infected patients who visited Songklanagarind Hospital, Hatyai, Thailand during 2018–2019 were collected retrospectively from medical records. Age, sex, ethnicity, marital status, and physician-reported HCV risk factors were collected. Liver biopsies are not routinely done at our hospital, so noninvasive fibrosis measurements by both a scoring system and liver stiffness measurements from a FibroScan 502 were used for evaluation of hepatic fibrosis. Hepatic steatosis was measured by controlled attenuation parameter (CAP). Aspartate aminotransferase (AST), alanine aminotransferase (ALT), and platelet count levels were obtained from the same year as the initial HCV genotype testing and used to calculate APRI and FIB-4 fibrosis scores. The equations for APRI and FIB-4 were as follows: APRI = (AST/upper limit of normal)/platelet count (expressed as platelets × 109/L) × 100 (13, 14), FIB-4 = age [years] × AST [IU/L]/platelet count [expressed as platelets × 109/L] × (ALT1/2[IU/L]) (15). APRI and FIB-4 cutoff values were used to categorize hepatic fibrosis status as “no significant fibrosis” (Stage 1: APRI ≤ 0.5; FIB-4 ≤ 1.45), “intermediate stage” (Stage 2: APRI 0.51–1.5; FIB-4 1.46–3.25), or “significant fibrosis” (Stage 3: APRI > 1.5; FIB-4 > 3.25). HCV RNA quantification was determined by the Cobas AmpliP-rep/Cobas TaqMan HCV assay (CAM/CTM) (Roche Molecular Systems, Inc., Pleasanton, CA, USA), with a lower detection limit of 15 IU/mL.

Statistical analysis

We determined correlations of hepatic fibrosis by both liver stiffness measurement and fibrosis scores among the HIV/HCV co-infected study patients. Demographic and laboratory differences between HCV mono-infected and HIV/HCV co-infected individuals were examined using the Pearson Chi squared test for categoric variables and the Student’s t-test or Wilcoxon rank sum test in cases of non-normal distribution for continuous variables. Stepwise logistic univariate regression analysis was used to investigate correlates of HIV/HCV co-infection among HCV-infected individuals. Variables with a p-value < 0.05 were included in the model. Univariate and multivariate linear regression models with an increased

fibrosis score as a continuous variable were also evaluated. The Akaike Information Criterion (AIC) was used as a measure of relative-goodness-of-fit to discriminate among various estimated models. Our final set of models was based on a combination of AIC goodness-of-fit as well as inclusion of clinically relevant variables, for which we used adjusted odds ratios (AOR). All analyses were performed using SPSS for Windows (Statistical Package for the Social Sciences, version 21.0, Armonk, New York, NY, USA), and GraphPad Prism, version 5.

Results

There were 151 HCV-infected individuals enrolled in the study, including 51 (33.7%) HIV/HCV co-infected individuals. The baseline characteristics for all study participants are provided in Table 1. The mean age of the entire cohort was 51.8 years and 69% were male. The mean BMI was 22 kg/m2. The physician-reported HCV risk factors were IVDU (31%), illicit drug use (28.5%), blood transfusion (40.4%), unsafe sex (9.9%), multiple partners (5.3%), tattooing (10.6%), MSM (7.3%) and unknown risk (11.3%). The recorded comorbidities were DM (6%) and DLP (5.3%). The HCV genotypes were 1a (21.2%), 1b (16.6%), 2 (0.7%), 3 (49%), 4 (0.7%) and 6 (11.9%).

The laboratory results were median AST = 50 IU/L, median ALT = 52 IU/L and median platelets = 177,000. Advanced fibrosis was demonstrated by both fibrosis score as APRI stage 3 (23.8%), FIB-4 stage 3 (31.1%) and by liver siffness measurement as Fibroscan 502 in which the median = 12 kPa. Liver steatosis was also evaluated by the same instrument in which the mean CAP = 207.1 dB/m.

The study found significant demographic differences between HCV-infected individuals and HIV/HCV co-infected individuals in age (54.2 vs 47.1, $p < 0.001$), male gender (63% vs 80.4%, $p < 0.046$), BMI (23.1 vs 20.7, $p < 0.001$), and married marital status (91% vs 33.3, $p < 0.001$), respectively. HCV genotype 1a infection was more common in HIV/HCV co-infected patients (35.3% vs 14% in HCV mono-infected patients, $p = 0.005$), while genotype 3 was predominant in HCV mono-infected patients (54%). The route of HCV transmission showed significant differences between HCV-mono infection and HIV/HCV co-infection patients [IVDU 23% vs 47.1%, $p = 0.005$, blood transfusion 53% vs 15.7%, $p < 0.001$, tattooing 5% vs 11%, $p = 0.004$, and MSM 1% vs 19.6, $p < 0.001$, respectively. Biochemically, no differences were found in transaminase enzymes (AST and ALT). HCV mono-infected individuals had a significantly higher hepatic fibrosis stage than HIV/HCV co-infected patients, with APRI score median 0.8 vs 0.6, $p = 0.046$, FIB-4 score median 2,4 vs 1.7, $p = 0.008$, and FibroScan median 13.1 vs 10.7, $p = 0.05$.

Univariate and multivariate analyses showed that HIV/HCV co-infected individuals were more likely to report a history of alcohol consumption (OR = 8.25, $p < 0.001$, AOR = 53.22, $p < 0.001$), unsafe sex (OR = 2.47, $p = 0.007$, AOR = 18.6, $p = 0.004$), or to have HCV genotype 1a (OR = 3.22, $p = 0.014$,AOR = 8.48, $p = 0.006$) compared to HCV mono-infected individuals, while HIV/HCV co-infected individuals were less likely to have high BMI (OR = 0.85, $p = 0.03$, AOR = 0.82, $p = 0.02$), liver fibrosis measured by FibroScan 502 (OR = 0.97, $p = 0.04$, AOR = 0.91, $p < 0.001$), or a high CAP result (OR = 0.98, $p = 0.03$, AOR = 0.98, p =

0.02) (Table 2). In HIV/HCV co-infected individuals, the level of CD4 count did not correlate strongly with level of fibrosis (Table 3).

Discussion

Our study found that the main risk factors for HIV/HCV co-infection were intravenous drug use and MSM, which is consistent with studies from Western countries on HIV/HCV co-infection(16, 17).

In this study, we also evaluated fibrosis severity in both HCV mono-infection and HIV/HCV co-infection patients. Surprisingly, we found more severe hepatic fibrosis by both fibrosis scores (APRI and FIB-4) and liver fibrosis measured by FibroScan in HCV mono-infected individuals than in HIV/HCV co-infection individuals, findings which are in contrast to most studies (18, 19). As this was a cross-sectional study, however, we could not examine hepatic fibrosis progression, and a higher fibrosis stage in HCV mono-infection patients could not be directly associated with more aggressive fibrosis progression. Therefore, long term follow-up studies are required for determining hepatic fibrosis progression rates. A possible explanation for this finding may be that a high percentage of mono-infected individuals in this study were infected with HCV genotype 3a (51%) which is the strain most associated with rapid fibrosis progression(20–22). Our study also found higher BMIs and CAP scores in mono-infected individuals than in co-infected individuals, which may reflect a higher degree of liver steatosis leading to aggressive liver fibrosis progression(23).

Several limitations of this study should be noted. First, our sample size was small compared to the scale of HIV/HCV co-infection in Thailand. Second, all participants came from a single hospital in Songkhla province and thus there may be generalization issues. Third, some demographic data such as educational status could not be obtained and may have lead to some bias. Regardless of these potential limitations, we believe that our finding of strong associations between HIV/HCV co-infection with IVDU and MSM is valid, given similar findings in previously published epidemiological research(24, 25). In addition, this was a cross-sectional retrospective study which limited our ability to examine disease duration, which may be important as our mono-infection group was significantly older than the co-infection group which could indicate a longer disease duration leading to more severe hepatic fibrosis in mono-infected individuals.

Conclusion

HIV/HCV co-infected individuals were associated with genotype 1a, unsafe sex, single status and low BMI, while HCV mono-infected individuals were associated with high CAP and high levels of hepatic fibrosis. Large-scale cohort studies with follow up hepatic fibrosis assessment is required to better understand the rate of hepatic fibrosis progression.

Abbreviations

HIV human immunodeficiency virus, HCV hepatitis C virus, CAP liver steatosis unit, MSM men who have sex with men, IVDU intravenous drug use, BMI body mass index, OR odds ratio, AOR adjusted odds ratio, CI confidence interval.

Declarations

Acknowledgement

The authors are greateful to all electronic medical record provided by Faculty of Medicine, Prince of Songkla University, Thailand.

Ethics approval

The study protocol was approved by the Ethics Committee at Faculty of Medicine, Prince of Songkla University.

Potential conflicts of interest

None.

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Tables

Table 1 Demographic and clinical baseline characteristics of study participants

Demographic / clinical data	Total (n=151)	HCV mono-infection (n=100)	HIV/HCV co-infection (n=51)	p-value
Age (y) mean (SD)	51.8 (11.1)	54.2 (11.6)	47.1 (8.4)	**<0.001**
Gender, n (%)				**0.046**
Male	104 (68.9)	63 (63)	41 (80.4)	
BMI median (IQR)	22 (19.9,24.2)	23.1 (20.3,24.8)	20.7 (18.9,22,.1)	**<0.001**
Marital status, n (%)				**<0.001**
Married	108 (71.5)	91 (91)	17 (33.3)	
HCV risk factors, n (%)				
IVDU	47(31.1)	23(23)	24(47.1)	**0.005**
Illicit drug use	43 (28.5)	23 (23)	20 (39.2)	0.058
Blood transfusion	61 (40.4)	53 (53)	8 (15.7)	**<0.001**
Unsafe sex	15 (9.9)	7 (7)	8 (15.7)	0.161
Multiple partners	8 (5.3)	1 (1)	7 (13.7)	0.002
Tattooing	16 (10.6)	5 (5)	11 (21.6)	**0.004**
MSM	11 (7.3)	1 (1)	10 (19.6)	**<0.001**
Unknown	17 (11.3)	11 (11)	6 (11.8)	1
Comorbidities, n (%)				
DM	9 (6)	8(8)	1 (2)	0.274
DLP	8 (5.3)	6 (6)	2 (3.9)	0.718
HCV genotype, n (%)				**0.016**
1a	32 (21.2)	14 (14)	18 (35.3)	**0.005**
1b	25 (16.6)	19 (19)	6 (11.8)	0.368
2a	1 (0.7)	0 (0)	1 (2)	0.338
3a	69 (45.7)	51 (51)	18 (35.3)	0.097
3b	5 (3.3)	3(3)	2 (3.9)	1
4	1 (0.7)	0 (0)	1 (2)	0.338
6	18 (11.9)	13 (13)	5 (9.8)	0.758
AST, median(IQR)	50(37,74.5)	49.5 (37,78)	50 (36,69)	0.382
ALT, median(IQR)	52 (35.5,74.5)	51.5 (36.8,74.2)	52 (34.5,75)	0.992
Platelet count ($x10^3$) median (IQR)	177(128,229.5)	166.5(117,220)	202(141,261.5)	**0.011**
APRI score median (IQR)	0.7 (0.4,1.5)	0.8 (0.4,1.8)	0.6 (0.4,1)	**0.046**
APRI score , n (%)				0.11
Stage 1	54 (35.8)	34 (34)	20 (39.2)	
Stage 2	61 (40.4)	37 (37)	24 (47.1)	
Stage 3	36 (23.8)	29 (29)	7 (13.7)	
FIB-4 score median (IQR)	2 (1.2,3.5)	2.4 (1.2,4.4)	1.7(1,2.7)	**0.008**
FIB-4 score, n (%)				0.063
Stage 1	53 (35.1)	30 (30)	23 (45.1)	
Stage 2	51 (33.8)	33 (33)	18 (35.3)	
Stage 3	47 (31.1)	37 (37)	10 (19.6)	
Liver fibrosis measured by FibroScan 502 median(IQR)	12 (7.7,22.5)	13.1 (8.2,26.7)	10.7 (6.1,17.4)	**0.05**
CAP mean (SD)	207.1 (36.4)	214.2 (34.1)	193.2 (37.2)	**<0.001**
Log_{10} HCV-RNA mean (SD)	5.9 (0.8)	5.8 (0.8)	6.1 (0.8)	**0.015**

Abbreviations: IVDU intravenous drug use, CAP liver steatosis unit, MSM men who have sex with men

APRI categories: stage 1 (<0.5), stage 2 (0.51-1.5), stage 3 (>1.5)

FIB-4 categories: stage 1 (<1.45), stage 2 (1.46-3.25), stage 3 (>3.25)

Table 2 Correlates of HIV/HCV co-infection using univariate (OR) and multivariate logistic regression (AOR)

Characteristic	OR (95% CI)	p-value	AOR (95% CI)	p-value
Older age	0.94 (0.91,0.98)	0.743		
Male	2.41 (1.08,5.37)	0.242		
Married	0.05 (0.02,0.13)	**<0.001**	0.02 (0.01,0.09)	**<0.001**
High BMI	0.85 (0.76,0.95)	**0.03**	0.82 (0.69,0.98)	**0.02**
Alcohol consumption	8.25 (3.65,18.66)	**<0.001**	53.22 (8.95,316.44)	**<0.001**
IVDU	2.98 (1.45,6.12)	0.207		
Blood transfusion	0.16 (0.07,0.4)	0.715		
Tattooing	5.22 (1.71,16.01)	0.647		
Unsafe sex	2.47 (0.84,7.26)	**0.007**	18.6 (2.2,157.12)	**0.004**
Multiple partners	15.75 (1.88,131.89)	0.369		
MSM	24.15 (2.99,194.67)	0.192		
Genotype 1a	3.22 (1.41,7.37)	**0.014**	8.48 (1.64,43.97)	**0.006**
Genotype 3a	0.55 (0.27,1.13)	0.189		
Tuberculosis co-infection	21.21 (2.61,172.76)	0.485		
ALP	1.02 (1.01,1.03)	**0.01**	1.02 (1.01,1.04)	**<0.001**
Liver stiffness measurement by FibroScan Touch 502	0.97 (0.94,1)	**0.043**	0.91 (0.85,0.97)	**<0.001**
High CAP	0.98 (0.97,0.99)	**0.036**	0.98 (0.96,0.99)	**0.022**
Log_{10} HCV-RNA	1.75 (1.1,2.79)	**0.039**	2.3 (1.03,5.14)	**0.035**
Platelet count	1.004 (1.0005-1.0088)	0.425		

Abbreviations: OR odds ratio, AOR adjusted odds ratio, CI confidence interval, IVDU intravenous drug use, BMI body mass index, CAP liver steatosis unit, MSM men who have sex with men

Table 3 Associations with liver stiffness by FibroScan 502 among HIV/HCV co-infected individuals (n=51)

Correlate	ß Coefficient (95% CI)	p-value
CD4 </=350 (Reference)		
CD4 351-500	4.57(-4.41,13.55)	0.311
CD4 >500	-5.35 (-13.09,2.39)	0.17