Key messages

- "The lower the better" is the predominant view in treating high blood pressure, but some studies have indicated that lowering diastolic pressure below a certain level may promote myocardial infarction
- In our 10 year follow up of 484 elderly men the risk of an ischaemic cardiac event was higher in men who were taking antihypertensive drugs than in those who were not
- Among men with diastolic blood pressure >90 mm Hg, the risk was increased twofold but disappeared when adjustments were made for other cardiovascular risk factors
- Among those with diastolic blood pressure ≤90 mm Hg, the risk associated with taking antihypertensive drugs was four times higher and remained after adjustment for other cardiovascular risk factors
- These findings support the concept of a J shaped curve for risk of myocardial infarction in relation to treated diastolic blood pressure

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Prevalence and determinants of antibodies to hepatitis C virus and markers for hepatitis B virus infection in patients with HIV infection in Aquitaine

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Abstract

***Objective*—To evaluate the prevalence of antibodies to hepatitis C virus and serological markers for hepatitis B virus infection in patients with HIV.**

***Design*-Cross sectional survey.**

***Setting*—Aquitaine, southwestern France, 1991-94.**

***Subjects*—1935 HIV positive patients seen at least once since June 1991.**

***Main outcome measures*—Presence of antibodies to hepatitis C virus were detected by second or third generation enzyme linked immunosorbent assay (ELISA) and recombinant immunoblot assay (RIBA) and markers for hepatitis B virus detected by ELISA.**

***Results*—The prevalence was 42.5% (823) for antibodies to hepatitis C virus, 56.4 (507) for antibodies to hepatitis B core antigen, 6.9% (133) for hepatitis B surface antigen, 30.2% (584) for antibodies to hepatitis B core and surface antigen with no detectable surface antigen, 26.2% (507) for antibodies to core antigen only, and 4.8% (92) for antibodies to surface antigen only. The prevalence of antibodies to hepatitis C virus was 86.1% (726/843) in subjects who had bloodborne HIV infection and 7.3% (66/899) in those with sexually acquired infection. The prevalence of markers for hepatitis B was higher among homosexuals than in the other groups of patients, except for antibodies to surface antigen alone. The relation between markers for hepatitis B and hepatitis C virus was negative among men but positive among women.**

***Conclusions*—The results favour the hypothesis that hepatitis C virus is sexually transmitted much less commonly than either HIV or hepatitis B virus.**

Introduction

Hepatitis C virus was quickly identified as the agent causing most cases of non-A non-B hepatitis after transfusion.[1] Second and third generation enzyme linked immunosorbent assays (ELISA) and recombinant immunoblot assays (RIBA) detecting hepatitis C virus antibody are reliable tools for diagnosing infection.[2] We provide an estimate of the prevalence of antibodies to hepatitis C virus and serological markers of hepatitis B virus infection in a representative sample of patients with HIV infection in Aquitaine, southwestern France.

Patients and methods

In 1987 the Groupe d'Epidémiologie Clinique du SIDA en Aquitaine started a surveillance system of HIV infection which allowed the creation of the Aquitaine cohort described elsewhere.[3] Our study population comprised 2957 HIV positive patients from Aquitaine who were recruited or followed up at least once between June 1991 and September 1994. The start date was chosen because second generation tests for hepatitis C virus were introduced at this time.

We routinely used two ELISAs for detecting hepatitis C virus antibodies (Ortho Diagnostic and Diagnostics Pasteur). If the two results did not agree a RIBA test (Chiron RIBA HCV) was used. The criterion for posi-

Table 1—*Number (%) of patients with antibodies to hepatitis C virus and of serological markers of hepatitis B virus according to route or transmission of HIV*

Transmission category	No of patients (men/women)	Antibodies to hepatitis C virus	Hepatitis B surface antigen	Antibodies to hepatitis B core and surface antigen, no surface antigen	Antibodies to hepatitis B core antigen only	Antibodies to hepatitis B surface antigen only	No marker for hepatitis B
Homosexuals	629 (629/0)	24 (3.8)	69 (11.0)	271 (43.1)	256 (40.7)	24 (3.8)	9 (1.4)
Intravenous drug users	645 (445/200)	584 (90.5)	41(6.4)	172 (26.7)	152 (23.6)	24 (3.7)	256 (39.7)
Homosexuals and intravenous drug users	38 (38/0)	29 (76.3)	1 (2.6)	10 (26.3)	11 (28.9)	6 (15.8)	10 (26.3)
Heterosexuals	270 (87/183)	42 (15.6)	10 (3.7)	39 (14.4)	31 (11.5)	18 (6.7)	172 (63.7)
Blood recipients and haemophilic patients	160 (109/51)	113 (70.6)	4 (2.5)	60 (37.5)	26 (16.3)	12 (7.5)	58 (36.2)
Other / unknown	193 (128/65	31 (16.1)	8 (4.1)	32 (16.6)	31 (16.1)	8 (4.1)	114 (59.1)
Total	1935 (1436/499)	823 (42.5)	133 (6.9)	584 (30.2)	507 (26.2)	92 (4.8)	619 (32.0)

tivity for the RIBA was the presence of at least two antigenic reactive bands.

Serological markers of hepatitis B infection were detected by ELISA (Behring), and patients were divided into five mutually exclusive categories:

1) Patients with hepatitis B surface antigen—that is, chronic carriers of hepatitis B virus regardless of their antibody profile.

2) Patients with antibodies to hepatitis B surface antigen and core antigen who did not have detectable hepatitis B surface antigen, reflecting former contact with hepatitis B virus and cure.

3) Patients who carried antibodies to hepatitis B core antigen only, suggesting previous contact with the virus but no confirmation of cure.

4) Patients who carried antibodies to hepatitis B surface antigen only, suggesting immunisation before any contact with the virus or incomplete laboratory investigation.

5) Patients with no marker of hepatitis B virus infection.

Alanine aminotransferase concentrations were considered abnormal when they exceeded 40 IU/l. The χ^2 test was used to compare percentages and Student's *t* test to compare means. Prevalence estimates are presented with their 95% confidence interval.

We used univariate analysis and logistic regression to study the determinants of carriage of hepatitis C virus antibodies, with route of HIV transmission and presence or absence of hepatitis B serological marker as explanatory variables and sex as a stratifying variable. The same analyses were performed for serological markers for hepatitis B, taking route of HIV transmission and presence or absence of antibodies to hepatitis C virus as determinants and sex as a stratifying variable. In the multivariate analyses HIV transmission categories were coded with an indicator (dummy) variable. Among men, homosexuals were the reference category, and men who reported both homosexual contacts and intravenous drug use were classified as intravenous drug users. Among women, heterosexuals served as the reference category.

Results

The serological status for both hepatitis B and C viruses was known for 1935 of the 2957 (65%) patients. Of the remaining 1022, 74 had been tested for only hepatitis C, 596 for only hepatitis B, and 352 for neither; these patients were excluded from the analyses. There was no difference between the two groups with regard to sex, age, and route of HIV transmission.

At the time of testing for antibodies to hepatitis C virus, 934 (48.3%) of the 1935 patients had normal alanine aminotransferase concentrations. Among the 670 patients who were tested for only hepatitis B or hepatitis C virus, 333 (49.7%) had normal alanine aminotransferase concentrations. The maximum alanine aminotransferase concentration recorded during the study period was normal in 162 (46.1%) of the 352 patients not tested for hepatitis B or C virus. No significant difference was found between the groups in the proportion of patients with normal alanine aminotransferase concentration (P = 0.5).

OVERALL PREVALENCE

Table 1 shows that the overall prevalence of antibodies to hepatitis C virus was 42.5% (95% confidence interval 40.3% to 44.7%). The prevalence of serological markers for hepatitis B infection was 6.9% (5.7% to 8.0%) for surface antigen, 30.2% (28.1% to 32.2%) for antibodies to core antigen and surface antigen, 26.2% (24.2% to 28.2%) for antibodies to core antigen only, and 4.8% (3.8% to 5.7%) for antibodies to surface antigen only. The overall prevalence of antibodies to core antigen, reflecting previous contact with hepatitis B virus, was 56.4% (54.2% to 58.6%). The overall prevalence of all markers for hepatitis B virus was 68% (65.9% to 70.1%).

DETERMINANTS OF PRESENCE OF ANTIBODIES TO HEPATITIS C VIRUS

In the univariate analysis the prevalence of antibodies to hepatitis C virus varied according to the route of HIV transmission (table 1). The prevalence was 89.7% among intravenous drug users (homosexual and heterosexual), 70.6% among blood recipients and haemophilic patients, 15.6% among heterosexuals, and 3.8% among homosexuals (P<0.001). The prevalence of antibodies to hepatitis C virus was 11.8 times lower among those with sexually acquired HIV infection (66/899, 7.3%) than those with bloodborne HIV infection (726/843, 86.1%).

The crude prevalence of antibodies to hepatitis C virus was lower among men (567, 39.5%) than women (256, 51.3%). Antibodies to hepatitis C virus were less common in patients with markers for hepatitis B virus (501/1316, 38.1%) than in those without (322/619, 52.0%).

Among men, the prevalence of antibodies to hepatitis C virus differed according to HIV transmission category and presence of antibodies to hepatitis B virus (table 2). Compared with homosexuals, intravenous drug users had the highest risk of carrying antibodies to hepatitis C virus (odds ratio = 203, 95% confidence interval 123 to 335), followed by haemophilic patients and blood recipients (66.3, 36.5 to 120), and heterosexuals (6.9, 3.5 to 13.7). An independent and inverse relation was observed between antibodies to hepatitis C virus and presence of markers for hepatitis B virus (0.5, 0.4 to 0.7). Table 2

For women the prevalence of antibodies to hepatitis C virus was also associated with bloodborne HIV trans-

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Table 2—*Determinants of carriage of antibodies to hepatitis C virus according to sex. Logistic regression analysis*

	Prevalence of antibodies (%)	Odds ratio (95% confidence interval)	P value
Men (n = 1436)			
HIV transmission category:			
Homosexuals	3.8	Reference	
Intravenous drug users*	89	203.0 (123.0 to 335.0)	0.001
Heterosexuals	19.5	6.9 (3.5 to 13.7)	0.001
Blood recipients and haemophilic patients	67.9	66.3 (36.5 to 120.0)	0.0001
Other/unknown	17.2	2.4 (3.42 to 12.0)	0.001
Serological markers for hepatitis B:			
None	86.5	Reference	
At least one	44.4	0.5 (0.4 to 0.7)	0.002
Women (n = 499)			
HIV transmission category:			
Heterosexuals	13.7	Reference	
Intravenous drug users	91.5	52.4 (26.9 to 102.0)	0.001
Blood recipients and haemophilic patients	76.5	23.0 (10.4 to 50.9)	0.001
Other/unknown	13.8	1.1 (0.5 to 2.6)	0.79
Serological markers for hepatitis B:			
None	34.3	Reference	
At least one	73.3	2.3 (1.3 to 4.1)	0.006
Overall	51.3	—	—

*Including drug users who were also homosexuals.

Table 3—*Determinants of serological markers for hepatitis B virus according to sex. Logistic regression analysis*

	Prevalence of markers (%)	Odds ratio (95% confidence interval)	P value
Men (n = 1436)			
HIV transmission category:			
Homosexuals	98.6	Reference	
Intravenous drug users*	63.6	0.6 (0.4 to 1.1)	0.07
Heterosexuals	59.6	0.4 (0.2 to 0.7)	0.01
Blood recipients and haemophilic patients	82.6	1.0 (0.7 to 2.3)	0.10
Other/unknown	53.1	0.3 (0.2 to 0.6)	0.01
Serological markers for hepatitis B:			
None	81.5	Reference	
At least one	66.6	0.5 (0.3 to 0.7)	0.001
Women (n = 499)			
HIV transmission category:			
Heterosexuals	25.1	Reference	
Intravenous drug users	43.0	3.9 (2.1 to 7.3)	0.001
Blood recipients and haemophilic patients	23.5	0.5 (0.2 to 1.1)	0.10
Other/unknown	16.9	0.6 (0.3 to 1.1)	0.10
Antibodies for hepatitis C:			
None	23.8	Reference	
Present	62.1	2.3 (1.3 to 4.2)	0.01
Overall	31.1	—	—

*Including drug users who were also homosexuals.

mission (table 2). Compared with heterosexuals, female intravenous drug users had a strong increase in risk of carrying antibodies to hepatitis C virus (52.4, 26.9 to 102), as did those who had had blood transfusions (23, 10.4 to 50.9). An independent but positive relation was observed between antibodies to hepatitis C virus and presence of markers for hepatitis B virus (2.3, 1.3 to 4.1). Because of the interaction between sex and markers for hepatitis B (P = 0.01) we have not reported an overall analysis for the study of determinants of carrying antibodies to hepatitis C.

DETERMINANTS OF THE PREVALENCE OF MARKERS FOR HEPATITIS B VIRUS

In the univariate analysis the prevalence of serological markers for hepatitis B varied with HIV transmission category, except for carriers of antibodies to surface antigen only (table 1). The other markers were more common among homosexuals than in any other transmission category. Thus, the prevalence of hepatitis B surface antigen was 11.0% among homosexuals and 5.5%(46/843) when combining the three categories of bloodborne HIV infection.

The crude prevalence of all hepatitis B markers was higher among men than women: 8.6% (123) *v* 2.0%(10) for surface antigen and 79.5% (141) *v* 31.1% (155) for all markers combined. The crude prevalence of all markers for hepatitis B was lower in patients with antibodies to hepatitis C virus than in those without (60.9% (501) *v* 73.3% (815)).

Table 3 shows the multivariate analysis stratified by sex. Among men, heterosexuals had a lower risk of carrying markers for hepatitis B than homosexuals (0.4, 0.2 to 0.7). The difference from homosexuals was not significant for intravenous drug users or haemophilic patients and blood recipients. In addition, presence of antibodies to hepatitis C virus in men was negatively associated with the presence of markers for hepatitis B (0.5, 0.3 to 0.7). Among women, intravenous drug users had a high risk of carrying markers for hepatitis B than heterosexuals (3.9, 2.1 to 7.3). An independent but positive relation was observed in women between markers for hepatitis B and antibodies to hepatitis C virus (2.3, 1.3 to 4.2). Because of the interaction between sex and antibodies to hepatitis C virus (P = 0.01) we have not reported an overall analysis for the determinants of markers for hepatitis B.

Discussion

Our study of a large sample of patients infected with HIV allowed us to estimate precisely the prevalence of the different serological markers of hepatitis C and B infections. Indeed, during 1991-93, 84% of the 1095 new cases of HIV infection diagnosed in Aquitaine were managed by the clinicians participating in the surveillance system.[4] Determination of antibodies to hepatitis C and B viruses was not part of the systematic follow up procedures adopted in the surveillance system[3] but was left to the decision of the clinicians. Results were spontaneously reported and included in the database. In addition, we systematically searched for serological results in the medical records before this study. We did not identify any important differences with regard to sex, age, and HIV transmission categories between the study sample and the rest of the Aquitaine cohort. Furthermore, our sample did not have a higher proportion of symptomatic patients with raised alanine aminotransferase concentrations. These factors suggest that our sample was representative of the population of patients infected with HIV.

Our estimate of the overall prevalence of antibodies to hepatitis C virus (42.5%) is higher than has been previously reported. Quan *et al* reported a prevalence of 3.5% (n = 226) in Canada,[5] Sherman *et al* 5.6% (n = 90)[6] Wright *et al* 17%,[7] and Nubling *et al* 20.8% (n = 383) with second generation tests.[8] Two main reasons may explain these differences. Firstly, most of these studies used, at least partly, first generation ELISAs with low sensitivity.[2] In a previous study using first generation assays we found a prevalence of 31.0% (95% confidence interval 24.6% to 37.4%) in the Aquitaine cohort.[9] Secondly, the distribution of HIV transmission categories varies among the studies. Quan *et al* used a group with 10% of intravenous drug users[5] whereas they accounted for a third of our sample. It is now recognised that hepatitis C virus is primarily transmitted by blood contacts, as indicated by the high prevalences of antibodies to hepatitis C virus among intravenous drug users,[10 11] haemophilic patients,[10] and those who have received blood transfusions.[10 12] We also found that the prevalence of antibodies to hepatitis C virus varied with route of HIV transmission.

Key messages

- Little is known about the spread of hepatitis C virus in patients with HIV infection
- The prevalence of antibodies to hepatitis C virus among HIV positive patients in Aquitaine was 42.5% and almost two thirds had a marker for hepatitis B infection
- The prevalence among subjects with sexually acquired infection was 11.8 times lower than in the those with bloodborne infection
- The relation between markers for hepatitis B and hepatitis C infection was significantly negative among men but significantly positive among women
- Hepatitis C virus seems to be much less commonly sexually transmitted than either HIV or hepatitis B virus

ROUTE OF TRANSMISSION

In our sample, 15.6% of those infected with HIV by heterosexual contact and 3.8% of those infected by homosexual contact were also infected with hepatitis C virus. The prevalence in homosexuals is at least three times higher than the prevalence in the French general population, which is reported at 0.5-1.5%.[13 14] This finding suggests that sexual transmission of hepatitis C virus does occur. However, some of the homosexuals may have chosen not to report intravenous drug use, and half of the patients who acquired HIV infection by heterosexual contact and who carried antibodies to hepatitis C virus reported a sexual partner who was an intravenous drug user.

We estimated that around 7% of HIV infected patients carried hepatitis B surface antigen and 56% antibodies to hepatitis B core antigen, which agrees with the literature.[15 16] Sexual transmission of hepatitis B virus is particularly efficient by homosexual contact, and this population is known to be at high risk of infection.[17] We found the prevalence among heterosexual men was double that in heterosexual women. This can be explained in two ways. Firstly, some men classified in other categories may not have reported homosexual contacts and thus artificially increased the prevalence in other groups. Secondly, sexual contacts with prostitutes, a group at high risk of hepatitis B infection, is likely to have been an important source of contamination for heterosexual men. This is also likely to explain the highest figures among men in the other or unknown transmission category of HIV infection, but cannot account for the difference observed with sex among blood recipients. This difference disappeared when the 42 haemophilic men were excluded from the analysis (data not shown).

The presence of any marker for hepatitis B virus was negatively associated with antibodies to hepatitis C virus among men but positively associated among women. Since a high proportion of men acquired hepatitis B infection by sexual contact this supports the theory of low sexual transmissibility of hepatitis C virus.[18 19]

In conclusion, we found a high prevalence of antibodies to hepatitis C virus in our sample of HIV positive patients in southwestern France. It is now well established that presence of antibodies strongly correlates with active hepatitis C virus infection.[20] Similarly, almost two thirds of our patients also had at least one serological marker of hepatitis B infection. Further study of the interaction between HIV and hepatitis B and C viruses is needed to improve surveillance and case management. Finally, the uneven distribution of hepatitis B markers in relation to antibodies to hepatitis C virus, transmission category of HIV, and sex, and the low prevalence of antibodies to hepatitis C virus in patients with sexually acquired HIV infection favour the hypothesis that hepatitis C is much less commonly sexually transmitted than either HIV or hepatitis B virus.[19 21]

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