

# HATiP

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# Viral hepatitis in low and middle-income settings

## Co-management of HIV and Hepatitis B or C

By Theo Smart

First, a cautionary tale.

"Putting someone with hepatitis C on antiretroviral therapy (ART) is like passing a death sentence on him," an Indian patient advocate was quoted saying in an article in the Indian press last year. (In the hope that he was misquoted, we won't reprint his name or organisation.) The news story went on to declare that "hepatitis rules out the use of [antiretroviral drugs] - which are toxic to the liver - to arrest the effects of the AIDS-causing virus."

Such misinformation and confusion about hepatitis and ART is quite common in a number of resource limited settings. According to Chris Green, a treatment advocate working in Indonesia, "Even doctors are confused - and scared - by liver problems, tending to stop ART when liver enzymes rise [to twice the upper limit of normal]."

In general, clinical data show that people with both HIV disease and hepatitis fare *much better* on ART. Nevertheless, liver toxicity can occur on ART, and HIV management can be more complicated in the context of liver disease. In addition, chronic hepatitis itself needs ongoing care.

In this issue of HATIP, we review what is and isn't known about HIV and hepatitis co-management. We'll focus primarily on hepatitis B and C viruses (HBV and HCV) as the most important causes of chronic hepatitis, while keeping in mind that other common factors, such as alcohol and other pharmaceutical and traditional medications, can contribute to liver problems.

## HBV vs. HCV: transmission and progression

Though both HBV and HCV affect the liver, there are important differences in how the viruses are transmitted, the populations most at risk, and how the infection evolves. Understanding the pattern and distribution of each liver infection is important in gauging the potential for coinfection in settings — particularly as screening for either virus is not common before initiating ART.

## Hepatitis B (HBV)

In much of the developing world, HBV is endemic among the general population. Exposure to HBV is common; about a third of the planet's population have at one time had the infection.

HBV is transmitted in more or less the same way as HIV: through exposure to infected blood and other bodily fluids — but it is far more infectious.

As a result, most infections occur early in life — perinatally (from mother-to-child) and between children in household situations. Unsafe injections and transfusions account for some exposure. Sexual transmission is less common in the developing world though it remains a possibility.

The timing of HBV transmission influences both the likelihood of a chronic infection and the severity of its outcome — the earlier in life someone is infected with HBV, the more likely the infection is to become chronic and to later progress to cirrhosis or liver cancer. 90% of infants infected in their first year, and 30 to 50% of children infected between one to four years of age, develop chronic infection. The risk of death from HBV-related liver cancer or cirrhosis is

approximately 25% for persons who become chronically infected in childhood. People who contract HBV as adults, however, have less than a 5% chance of developing chronic disease.

In 1999 and 2000, the World Health Organisation published estimates on the global prevalence of chronic HBV (see graphic 1). According to their calculations, around 10% of the general population of sub-Saharan Africa and much of Asia have chronic HBV. Globally, about 387 million people are chronically HBV infected.

However, there are differences in frequency of chronic HBV from country to country. For example, a recent analysis of HBV and HIV coinfection in Africa reported that as many as 20% of the general population in the Democratic Republic of Congo carry HBV surface antigen (Burnett). There is also considerable variation between rural and urban settings within countries: in South Africa, for example, the predominantly rural Eastern Cape reports a prevalence of 15.5%, compared with 1.3% in the township of Soweto (see <http://www.aidsmap.com/en/news/9E241956-2B80-420D-BFA3-8361511B46FC.asp>).

## Prevalence of chronic HBV carriers

350 million HBV carriers worldwide

3-5 million HIV co-infected\*

## Clusters of HCV infection

HCV, on the other hand, is transmitted via blood-blood exposure (most commonly from reuse of inadequately sterilised medical equipment, or needle-sharing among drug-users or from blood transfusions), and is only rarely transmitted perinatally or through sexual contact. In contrast to HBV, HCV is much more likely to be found clustered in risk groups than among the general population, especially injecting drug users.

HCV may be more difficult to contract than HBV, but the consequences are more likely to be serious regardless of how the infection occurs. Approximately 40% of those infected with HCV are likely to go on to develop some form of liver damage, with liver cancer and liver failure the most serious consequences of HCV infection. The risk of developing liver damage increases with the duration of infection, **and is seriously exacerbated by alcohol consumption**. The average time from infection to development of cirrhosis of the liver is between 30 and 40 years in HIV-negative people, which is why the public health consequences of HCV infection are still poorly recognised in most countries.

In 1999, WHO estimated that 170 million people were living with HCV worldwide. The highest prevalence is in Asia and Africa, where WHO estimated between four and five per cent of the population are infected.

HIV and HCV are co-epidemics in countries where injecting drug use is the main driver of the HIV epidemic, just as HIV and TB are co-epidemics in Africa and much of Asia.

In one recent study of IDUs in Russia, the anti-HCV prevalence was 87%, and 56% were HIV-positive, of whom 93% were coinfecting with HIV. (Rhodes).

A cross-sectional study of HIV-positive patients at Ramathibodi hospital in Bangkok, Thailand found that 8.7% had hepatitis B infection and 7.8% had HCV infection (Sunkanuparph 2004). However a recent vaccine preparedness study found that 50% of men who were HIV-positive also had HCV (Paris 2003).

HIV and HCV will be inextricably linked as long as an absence of harm reduction measures assists the spread of both viruses. The

absence of opiate substitution programmes throughout Asia and Eastern Europe, the extreme criminalisation of drug use and the lack of needle exchange programmes all create conditions that can only complicate the treatment of HIV infection where it occurs.

## Hepatitis C: A Global Health Problem

170-200 million carriers worldwide  
10 million HIV co-infected\*

# HIV and hepatitis in non-injecting drug users

Wherever HCV is found in a significant proportion of the general population, it is often the result of a mass inoculation programme where vaccination devices were used repetitively without proper sterilisation.

For example, Egypt has the highest rate of HCV in the world (around a third of the general population) because HCV was spread by mass intravenous anti-schistosomiasis inoculations in the 1960s-70s (Deuffic-Burban). Studies suggest that small-pox inoculations had a similar impact in Pakistan (Aslam).

But even in these situations, HCV infections tend to be clustered by age group (depending upon when the unsafe vaccination programmes took place). For example, a study of one rural village in the Nile Delta reported that by far the highest HCV seroprevalence (of more than 40%) was detected in males aged 40-54 years (Arafa).

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HCV is frequently restricted to isolated risk groups so there are also dramatic differences in the HCV prevalence among different cohorts within the same country. Madhava et al reviewed data from 160 different cohorts in sub-Saharan Africa, and the HCV rates ranged from 0-40%. The highest rates and widest ranges were reported in Cameroon and Burundi, but even once these outliers were excluded, the prevalence ranged between 0-17%. It's generally agreed that injection drug use has not made major inroads into Africa, although transfusions may contribute to the spread of HCV there (few Africa countries consistently screen the blood supply for HCV). Some HCV transmission may come from unexpected sources such as tattooing, scarification, piercing, circumcision or other ceremonial, traditional medical or cultural practices involving blood-blood exposure.

## Scale of co-infection

In countries where HBV is not endemic, studies have demonstrated a very high prevalence of HBV in people with HIV. This is likely in those settings because both viruses are transmitted by "high risk" behaviour (most frequently unprotected sex).

But in countries where HBV is endemic, the association between HIV and HBV infection is not as great — though it may still exist. Several studies in Africa suggest that active HBV may roughly twice as common in people with HIV as in the general population (Burnett).

In the case of HCV, the burden of coinfection depends on which behaviours are driving the HIV epidemic locally. If HIV is spread primarily by sexual or perinatal transmission, HCV should generally be found in relatively the same frequency of people with HIV as in the general population (Madhava); but where HIV is contracted mostly from exposure to infected blood (due to injection drug use or transfusions), there is a very good chance of encountering HCV in the same patients.

For example, in the Russian study mentioned above, 93% (214/230) of HIV-positive IDUs were found to be coinfecting with HCV. Similarly, in a study of the 236 HIV-infected individuals in China (63% of whom were blood/transfusion recipients or IDUs), 57% were coinfecting with HCV (Zhang).

But given regional variations for both HBV and HCV, it pays to know the local prevalence of both chronic HBV and HCV and to be aware of local cultural practices that might be associated with a higher HCV prevalence. This may require improved surveillance for both HBV and HCV in local populations.

## Impact of coinfection on disease progression

The majority of cohort studies show that coinfection with HIV and HCV accelerates liver damage caused by HCV and individuals with both HCV and HBV in addition to HIV have a higher risk of death than HIV-positive people coinfecting with only one hepatitis virus (Bonacini, Salmon-Ceron).

People with HIV who become HBV infected are more likely to develop chronic hepatitis, and rates of HBV and HCV viral replication are higher in people with AIDS.

Also, this ongoing HBV/HCV replication may not immediately be associated with detectable symptoms, because AIDS partially suppresses the inflammation that mediates liver damage.

This is important, because liver function tests may not identify a person with AIDS and HBV or HCV infection that is not detectable by standard tests (occult infection) who has yet to start ART. In the absence of a liver biopsy, liver disease may only become evident following a liver enzyme elevation once ART is started.

## Impact of coinfection since ART

Since the introduction of ART, liver disease has become a more common cause of death in people with HIV (Mocroft) (see <http://www.aidsmap.com/en/news/45AB11EA-F29C-48E1-9B50-FBC6D9C20110.asp>). One researcher found that since ART, HIV / HCV coinfecting individuals were approximately twice as likely to be hospitalised and three times more likely to die compared with HCV-negative individuals with HIV alone (Klein). Another recent study found that HCV coinfection increased the risk of death amongst HIV-positive US veterans by between 30% and 80% (Backus). A meta-analysis presented at a United States National Institutes of Health consensus conference on hepatitis C in June 2002 showed that HIV / HCV coinfecting people had a two-fold greater risk of cirrhosis and a six-fold greater risk of end-stage liver disease than those with HCV alone (National Institutes of Health 2002).

Again, the increase in morbidity and mortality related to liver disease among HIV-positive people since the advent of ART is due in part to the fact that individuals receiving effective anti-HIV

treatment are much less likely to die from other causes. In addition, as coinfecting individuals live longer, there is more time for progressive liver damage due to chronic hepatitis B or C to develop.

Furthermore, “flare-ups” of hepatitis in people on ART can be due to immune reconstitution inflammatory syndrome – a potentially deadly reaction to an HBV or HCV infection (which was previously occult). This is most common in the people with advanced HIV, with very low CD4 cell counts or CD4 cell percentages (Sherman and Ratnam).

Finally, a proportion of liver-related adverse events and deaths in people with HIV may be due to hepatotoxicity associated with antiretroviral drugs or with other hepatotoxic drugs such as isoniazid. This is most common when chronic HBV or HCV has not been identified before starting ART (and when liver function has not been monitored closely enough in such patients).

In the various studies that have been performed, it is difficult to ferret out whether drug toxicity, IRIS, or simply the relentless progression of liver disease due to chronic HBV or HCV is the cause of more hepatitis in people on ART.

Several other factors also confound interpretation of the studies:

- Heavy alcohol use by a person with chronic hepatitis greatly increases the risk of cirrhosis and liver decompensation and is common among some HIV-infected populations.
- There could be dangers associated with mega-doses of vitamins or other supplements (<http://www.aidsmap.com/en/news/DB3DF39C-B2ED-48B5-9F53-CD698E0F6337.asp>)
- People on ART are frequently also taking other medicines that are toxic to the liver.

For example, in one recent study in JAIDS, researchers from Chennai, India reported hepatitis in 33 out of 1184 people put on a nevirapine-based regimen which led to a treatment switch (Kumarasamy) (see <http://www.aidsmap.com/en/news/DFE0AD54-F746-47B9-A490-B08190959082.asp>). 27(82%) of these cases were also concurrently on rifampicin-based anti-TB medications. This is curious, because rifampicin significantly lowers blood levels of nevirapine – and thus one should be less likely to observe nevirapine-induced toxicity. Furthermore, these individuals had fairly low CD4 cell counts (~65 at baseline)– and nevirapine-induced hepatitis typically occurs in people with higher CD4 cell counts (over 250). So it seems quite possible that the toxicity may have been due to the TB regimen, to IRIS (no information was given on whether HBV or HCV were screened for) or possibly to an as of yet unidentified synergistic toxicity between nevirapine and rifampicin (which are not commonly prescribed together because of the effect on nevirapine drug levels).

## Benefits of ART in coinfecting patients

Such events only serve to reinforce misconceptions about the safety of ART in patients with impaired liver function and hepatitis. Reports of an increase in adverse events and liver disease in people with HIV since the advent of ART are the likely cause for misinformation such as that quoted at the beginning of this article. But a number of recent studies confirm that, on the whole, people coinfecting with HIV and one of the liver viruses are better off taking ART.

For example, a study in the January 2nd 2006 issue of AIDS reported that mortality fell in HIV/HCV coinfecting people in the years after the introduction of effective HIV treatment (Lumbreras) (see <http://www.aidsmap.com/en/news/9ECCC721-86FB-4341-BEB4-0>

[AABB84A1A61.asp](http://www.aidsmap.com/en/news/9ECCC721-86FB-4341-BEB4-0)); while a study in the January 15 issue of Clinical Infectious Diseases found that progressive liver damage from chronic hepatitis C is much less common in coinfecting people when they are taking effective ART (Verma) (see <http://www.aidsmap.com/en/news/4658A9C0-D5DC-4E08-BCCB-05D9F466C249.asp>). Similar data has been reported for people with coinfecting with HIV and HBV (Konopnicki) (see <http://www.aidsmap.com/en/news/9434925B-C63E-49A4-AF95-01B5CA7F4AF7.asp>).

## Considerations before using ART in people coinfecting with HBV or HCV

While ART should not be deferred simply because a person with HIV also has HBV or HCV, there may be a number of ways to optimise his or her response to therapy.

These are:

- Monitoring liver function to identify individuals with poor liver function
- Screen for HBV and HCV
- Treat chronic HBV with an antiviral drug or include a drug active against HBV in the anti-HIV combination
- Treat the hepatitis C infection where resources permit
- Choose antiretrovirals to minimise the risk of liver toxicity

## Monitoring liver function

Liver function tests (LFTs) are good indicators of liver disease in some patients, though not all (particularly if they have occult HBV or HCV).

Traditionally, a liver profile would evaluate alanine amino transferase (ALT/SGPT) and aspartate aminotransferase (AST/SGOT). Both these tests look for enzymes which are usually present in liver cells, but may leak out into the bloodstream when liver cells become damaged. ALT is thought to be a more accurate measure of liver damage, as AST can be produced by other organs such as the heart.

The normal range for both ALT and AST is 5-40 IU/L (International Units per Litre). Minor elevations in ALT and AST (up to three times the upper limit of normal level) are common in people with HIV, particularly those with advanced disease. The reasons are numerous but could include drug therapy, alcohol or malnutrition. But elevations greater than 3 times the upper limit of normal suggest hepatitis.

The safety of a treatment may depend on how closely it is monitored and there is a range of opinion as to how close laboratory monitoring of antiretroviral therapy needs to be in resource-constrained settings.

In some settings, LFTs are only checked at baseline before putting people on ART and then not again until six months later. In the study in Chennai, the median time to development to hepatotoxicity was 34 days, which led its authors to conclude “in light of our data, however, earlier laboratory monitoring for toxicity is advisable.”

## Screen patients for HBV or HCV

Where resources permit, baseline lab tests for HBV and HCV could help identify which patients might benefit from closer monitoring on ART.



A negative hepatitis B surface antigen (HBsAg) and core IgG antibody test results can also identify a patient who is at risk of acute HBV (and may be an indication for anti-HBV vaccination).

Where resources do not permit screening for HBV or HCV, detailed history taking, clinical screening and an assessment of the local populations risk for either virus could help identify patients likely to have chronic HBV or HCV and to guide the frequency of liver function tests.

### Consider treatment for chronic HBV

The anti-HIV drugs lamivudine (and the similar emtricitabine (FTC)) and tenofovir are active against hepatitis B and could be considered as part of an ART regimen. As a treatment for hepatitis B, lamivudine results in viral clearance in about 20-30% of people after one year. It may be possible that therapy longer than one or two years is required.

Around 20% of people experience a hepatitis flare-up when they stop taking lamivudine. This may lead to a rebound in hepatitis B DNA in around 20% of patients, and in a small minority (2-4%), to increased ALT and bilirubin levels.

Prolonged use of lamivudine (3TC) in individuals with hepatitis B as the single agent that is active against HBV leads to resistance in about one third of individuals. However, there is evidence that 3TC-resistant hepatitis B remains sensitive to adefovir and tenofovir. Studies have also shown that 3TC plus tenofovir reduces HBV viral load more effectively than 3TC alone.

A study last year reported such flare-ups of hepatitis despite the inclusion of 3TC in two patients who showed no evidence of 3TC-resistant HBV (Drake). The authors emphasised the need to "maximally suppress HBV replication from the outset."

The authors recommend that combination therapy with lamivudine plus tenofovir "should be employed at least for cirrhotic patients at risk for hepatic decompensation and, perhaps, even for all coinfecting patients with HBV replication." (see <http://www.aidsmap.com/en/news/75BDBF2F-8218-4D60-A983-2A2F11AC12D6.asp>).

However, at this point, tenofovir's role (or importance) in the treatment of HIV in resource limited settings is uncertain – and thus it may be too soon to follow this recommendation without further study.

### And for HCV?

While middle income countries may be able to treat patients with intravenous alpha interferon combined with ribavirin (which is available generically), this form of treatment is both too expensive and impractical to employ in the many resource-constrained settings. In such places, ART and palliative care represents the only and best way to care for chronic HCV in HIV coinfecting patients for the foreseeable future.

### Choose antiretrovirals to minimise the risk of liver toxicity?

Although cohort studies in the developed world repeatedly show that coinfection with HBV or HCV is the most important risk factor for liver toxicity, the association between particular antiretrovirals and liver toxicity in people with hepatitis coinfection is hard to pin down.

For example, nevirapine is associated with liver toxicity in individuals with higher CD4 cell counts, but that toxicity seems to be a classic liver toxicity rather than one driven by hepatitis or decreased liver function. Nevirapine's manufacturer has reported

no greater frequency of liver toxicity in people who receive nevirapine in clinical trials when compared to the control group, and an Italian cohort study published last year found that high baseline liver enzyme levels were the only predictor of liver toxicity (<http://www.biomedcentral.com/1471-2334/5/58>).

More stringent monitoring rather than denial of treatment or avoidance of particular drugs seems to be the most appropriate course to take where hepatitis coinfection is known or suspected, most experts agree.

### Further information on hepatitis B and C

Hepatitis B overview

<http://www.aidsmap.com/en/docs/5C509E1C-7894-4D63-99DA-8A12BD47D1E4.asp>

Hepatitis C overview

<http://www.aidsmap.com/en/docs/BE313493-C188-4F68-BE63-D5F180CCD6FE.asp>

Hepatitis news stories

<http://www.aidsmap.com/en/news/C8B61025-2693-435B-9CEA-648878CD1800.asp>

Patient information

- Hepatitis B factsheet

<http://www.aidsmap.com/en/docs/DEA4B7A1-5CD0-49B9-88F7-D4701B916962.asp>

- Hepatitis C factsheet

<http://www.aidsmap.com/en/docs/EA9B8E8B-F16C-41A4-A5DD-43830784BAE7.asp> (also in Spanish and Portuguese)

- HIV & Hepatitis information booklet

<http://www.aidsmap.com/en/docs/FA6438D2-C366-4552-87BA-14FF35B617A5.asp>

- HIV and hepatitis booklet in French

<http://www.aidsmap.com/fr/docs/F8ED0AE4-A9EF-4300-ABB1-81430DB5BBC1.asp>

#### Future Issue:

HATIP poses the question of how best to manage chronic hepatitis in HIV infected patients to our advisory panel shortly for a further exploration of this subject.

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## about HATiP

A regular electronic newsletter for health care workers and community-based organisations on HIV treatment in resource-limited settings.

The newsletter is edited by Theo Smart (Cape Town) and Keith Alcorn, NAM's Senior Editor (London).

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