# CURRENT VIEW ON HIV-2 – EPIDEMIOLOGY, DIAGNOSIS AND TREATMENT

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**SUMMARY**

Human immunodeficiency virus 2 (HIV-2) is a retrovirus related to HIV-1. It is believed to have evolved from the simian immunodeficiency virus (SIV). The disease caused by HIV-2 is manifested by a slower progression compared to HIV-1. Data from West Africa show that 37% to 50% of HIV-2 infected people have undetected or very low viremia and if left untreated, have a much more gradual decline in CD4 T lymphocytes, thus longer survival. However, treatment should not be postponed even in this case, because the course of immune reconstitution inflammatory syndrome (IRIS) in people with HIV-2 is more severe than in HIV-1, and also the year-on-year increase in the number of CD4 T lymphocytes is approximately half that of HIV-1. Approximately 6% of people living with HIV-2 (PWH2) can be defined as non-progressors and 9% as elite controllers. However, most untreated PWH2 develop AIDS.

*Key words:* antiretroviral therapy, human immunodeficiency virus 2, people living with HIV-1, people living with HIV-2

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## INTRODUCTION

Human immunodeficiency virus type 2 (HIV-2) is a less common and less virulent relative of HIV-1. While both viruses share similarities in their structure and replication mechanisms, HIV-2 exhibits distinct virologic properties that influence its transmission, pathogenicity, and epidemiology (1–3). With regard to the possible risk of HIV-2 import to the countries of Central and Eastern Europe, the authors have tried to prepare an up-to-date overview of information for the needs of physicians of various specialties. The database PubMed with keywords HIV-2, epidemiology, diagnosis, clinical picture, and treatment was used for selection of cited literature.

## Virologic Properties of HIV-2

HIV-2 is an enveloped retrovirus belonging to the Lentivirus genus. Its genome is composed of single-stranded RNA, similar to HIV-1, but shares only about 55% genetic similarity with HIV-1. HIV-2 contains all major structural (gag, pol, env) and regulatory/accessory genes (tat, rev, vif, vpr, vpx, nef), but it uniquely includes the vpx gene, which is absent in HIV-1 (3–5). Eight genotypes (A–H) of HIV-2 and two recombinant forms have currently been described (6).

HIV-2 has a lower replication capacity compared to HIV-1, which contributes to its reduced viral load (VL) in infected individuals (1, 3). It exhibits a preference for infecting macrophages and CD4 T cells, with lower affinity for CD4 receptors and co-receptors such as CCR5 and CXCR4 (3). HIV-2 is less efficient at utilizing CXCR4, which may partially explain its slower progression to AIDS compared to HIV-1. In vitro experiments showed CCRL2 to function as a newly identified coreceptor for primary HIV-2 isolates conveniently (7). Understanding the virologic properties of HIV-2 is critical for designing effective diagnostic, therapeutic and preventive strategies (3).

## Epidemiology

It is estimated that between one and two million people in the world are infected with HIV-2 (8). The highest prevalence is in the countries of West Africa, where the virus was first detected in 1985 and later described by Clavel et al. (9). In Europe, it is more common in Portugal, France, Great Britain, and Spain, countries with historical ties to West Africa (10) (Table 1). Its prevalence is decreasing over time due to lower transmission rates and competition with HIV-1 (11). It is also thought that HIV-2 has lower rates of sexual, vertical, and parenteral transmission compared to HIV-1 (12) (Table 2).

***Table 1.*** *Distributions of HIV-2*

| | Estimated prevalence (%) | Countries |
|---|---|---|
| HIV-2 infection | 3 | Cabo Verde, Burkina Faso, Cote d'Ivoire, Guinea-Bissau, Mali, Gabon, Cameroon, France |
| HIV-/HIV2 coinfection | 10 | Guinea-Bissau, Burkina Faso, Cote d'Ivoire, Mali, Gabon, South Africa |

Source: Williams et al. (8)

***Table 2.*** *Basic differences between HIV-1 and HIV-2*

| | HIV-1 | HIV-2 |
|---|---|---|
| Occurrence | Worldwide | West Africa, South and West Europe |
| Genetic nearness | | 55% |
| Different ENV/GAG gene products | gp120, gp41, p24 | gp105/125, gp36/41, p26 |
| Different POL gene products | p66, p31 | p68, p31/34 |
| Transmission: heterosexual | | 3-to 6-fold lower |
| Transmission: perinatal | 1%–20% | 0%–5% |
| Time to AIDS | 5–10 years | 10–25 years |
| Viral load in untreated PWH | High | Low |
| Specific antibody levels | Detectable | Low |
| Treatment | All ART classes | NNRTIs, enfuvirtide and fostemsavir are excluded |

## Clinical Picture

HIV-2 infection has clinical similarities to HIV-1 but generally follows a slower disease progression and is less transmissible (1, 13).

- Primary infection is often asymptomatic or associated with mild, nonspecific symptoms such as fever, rash, lymphadenopathy, or flu-like illness.
- Asymptomatic phase has prolonged latency period compared to HIV-1. Patients may remain asymptomatic for many years without antiretroviral therapy (ART).
- Early symptomatic phase with immune system decline is characterized by slower CD4 T-cell decline compared to HIV-1 and lower viral loads, but eventual progression to immune deficiency.
- Fully developed disease – AIDS, with symptoms of immunosuppression and opportunistic infections (OIs) similar to those seen in HIV-1 but occurring later in the disease course.

Tuberculosis (TB) is more frequent in HIV-2, particularly in endemic regions. Bacterial, viral, fungal, and parasitic infections are similar to HIV-1. Wasting syndrome with fever, severe weight loss and chronic diarrhoea occurs in advanced stages. Neurological manifestations, called HIV-associated neurocognitive disorders (HAND) are less common but may still occur in late stages. Also, prevalence of HIV-associated tumours both Kaposi sarcomas and non-Hodgkin's lymphomas is less common than in HIV-1 (14, 15).

## Prognosis

Without treatment, HIV-2 can progress to AIDS, but the timeline is often delayed compared to HIV-1. With effective ART, the prognosis can be favourable, but access to appropriate therapy remains a challenge in resource-limited settings (2).

## Diagnostics

HIV-2 infection is more often found in people from West African countries and in people with high-risk behaviour and sexual contacts with people with a high incidence of HIV-2. Dual HIV-1 and HIV-2 infections, which are very rare in Europe, are no exception in these people (16). Initial testing consists of a standard examination of antibodies and antigen with dual fourth-generation tests, detecting anti-HIV-1, anti-HIV-2 and p24 antigen (17). Reactive samples are always tested using a higher specificity method (18, 19). The confirmatory Western blot test for our patients could be performed at the National Reference Laboratory for HIV/AIDS (NRL) at the National Institute of Public Health in Prague. Therefore, in people who respond to screening tests and at the same time have undetectable HIV-1 RNA, it is necessary to consider also HIV-2 testing. Qualitative detection of HIV-2 RNA by a commercial test has been available since mid-2024 in NRL. A commercial test for the quantitative determination of HIV-2 RNA (viral load) will also be available in the near future in the Czech Republic (20).

Also, properly treated people living with HIV-1 (PWH1), with undetectable HIV-1 RNA, showing a sustained decrease in CD4 T lymphocytes, should be tested for HIV-2 antibodies. The confirmation by commercial PCR tests detecting HIV-2 RNA is generally available in many countries, but a negative result does not completely rule out infection, as viremia is often very low in HIV-2 infection even without ART (21). In this case, it is recommended to test for proviral DNA in peripheral blood mononuclear cells (22). Detection of mutations associated with the development of HIV-2 resistance (RAMs) is possible by RNA sequencing (23).

## Other Examinations

Laboratory tests used in PWH1 are the same in people with HIV-2. Regular check-ups every three, later every six months are necessary. CD4 T lymphocyte counts, HIV-2 RNA levels (repeated more often when RNA increases), standard haematological, biochemical and microbiological examinations are monitored. Genotype testing for resistance to nucleoside reverse transcriptase inhibitors (NRTIs), to protease inhibitors (PIs) and to viral integrase inhibitors (InSTIs) is recommended before starting treatment. At low RNA levels, proviral DNA can be amplified (22). Primary resistance in the French cohort was set at 5%. It is formed much faster with suboptimal ART dosing (24, 25).

## Treatment

According to European guidelines (26), treatment is initiated based on clinical status, CD4 T lymphocyte count and HIV-2 RNA. It is always indicated in people with HIV-specific symptoms, i.e., clinical stages B and C. In asymptomatic PWH2, it is initiated at CD4 T lymphocytes < 500/ul, or when CD4 drops more

than 30/ul per year, or when HIV-2 RNA is repeatedly detected, and in comorbidities (e.g., chronic VHB). In non-progressors with CD4 T lymphocytes counts above 500/ul and undetected RNA, the initiation of treatment may be delayed. However, the DHHS recommendation (USA) is the same as for HIV-1 (27). Initiating treatment as soon as possible after detecting the infection also works as prevention.

## First-line Treatment

The choice of drugs is limited in HIV-2. Non-nucleoside reverse transcriptase inhibitors (NNRTIs), which cannot be used at the active site of HIV-2 reverse transcriptase (RT), fusion inhibitors (enfuvirtide), and the attachment inhibitor fostemsavir are completely excluded (28, 29). Older protease inhibitors (PIs) are also less effective against HIV-2. All nucleoside and nucleotide reverse transcriptase inhibitors (NRTIs, NtRTIs), potentiated PIs (darunavir, lopinavir, saquinavir), all integrase inhibitors (InSTIs) (28, 30), and CCR5 inhibitors (maravirok) in the case of the CCR5-tropic variant of HIV-2 are the choices. In PWH2 with proven multi-resistance of HIV-2, ARTs from new drug groups can be used (31).

As with HIV-1, it is advisable to start treatment with 2NRTI or (1NRTI+1NtRTI) + (InSTI or potentiated PI) (Table 3). In the absence of resistance-associated mutations (RAMs), potentiated darunavir (DRV) at the same dosage as for HIV-1 is appropriate. When RAMs are detected, the InSTI option is selected (30). Bictegravir (BIC) is recommended for the treatment of HIV-2 infection, its effect is comparable to dolutegravir (DTG) (29, 30). Some ART-resistance have already been described (31). Similar combinations are also chosen for dual HIV-1/2 infections.

Lenacapavir (LEN) also exhibited activity against HIV-2, but it was 11- to 14-fold less potent against HIV-2 in comparison to HIV-1. Mutations in HIV-2 that confer resistance to other antiretrovirals did not confer cross-resistance to lenacapavir. Although lenacapavir-containing regimens might be considered appropriate for patients with HIV-2, more frequent viral load and/or CD4 testing may be needed to assess clinical response (32).

HIV-2 exhibits greater resistance to neutralizing antibodies, which may also complicate vaccine development (33).

## Treatment Control

After initiation of treatment, regular monitoring is required for one, three and six months after initiation of treatment (VL and especially CD4 T lymphocytes is sometimes the only parameter indicating a worsening of the condition). In CD4 < 200/ul are checked every three months, in CD4 200–500/ul every three to six months, and in CD4 levels greater than 500/µl PWH2 are tested as appropriate, depending on adherence, clinical status and comorbidities. Resistance is tested when virologic failure is suspected. If there are signs of clinical progression, CD4 and VL are tested immediately (25). Some individuals with HIV-2 can remain asymptomatic for extended periods, often referred to as "long-term non-progressors" (19).

***Table 3.*** *Recommended treatment for people living with HIV-2*

| | | | |
|---|---|---|---|
| (A) | 2 NRTIs or 1 NRTI + 1 NtRTI | (B) | PI/r/c or InSTI |
| | TAF/TDF + FTC/3TC | | 1 PI (DRV/r, DRV/c, LPV/r) |
| | ABC + 3TC | | 1 InSTI (RAL, DTG, EVG, BIC) |
| | AZT + 3TC | | 1 EI (MRV*, LEN) |

3TC – lamivudine; ABC – abacavir; AZT – azidothymidine; BIC – bictegravir; c – cobicistat; DRV/y – darunavir potentiated with r or c; DTG – dolutegravir; EI – entry inhibitors; EVG/c – potentiated elvitegravir; FTC – emtricitabine; HIV-2 – human immunodeficiency virus–2; InSTI – HIV integrase inhibitor; LEN – lenacapavir; LPV/r – potentiated lopinavir; MRV – maravirok (*only in CCR5-tropic variant of HIV-2); NRTI – nucleoside reverse transcriptase inhibitor; PI – proteinase inhibitor; r – ritonavir; RAL – raltegravir; TAF – tenofovir alafenamide; TDF – tenofovir disoproxyl fumarate

## Resistance and Virologic Failure

Proving virologic failure is much more difficult due to low levels of HIV-2 RNA and slow adjustment of CD4 T lymphocyte counts. HIV-2 resistance is defined by the detection of HIV-2 RNA in the following two blood samples, after previous undetectable VL, a decrease in CD4 T lymphocytes, or persistence or new HIV-specific symptoms. Second-line drugs are selected based on RAMs testing, drug tolerance and adherence. For example, AZT+ MRV (in R5-tropic HIV-2) + SQV/r can be considered. Determination of resistance by genotyping and their evaluation with the help of European databases is carried out by the National Reference Laboratory for HIV at the National Institute of Public Health in Prague. The genetic barriers for the development of resistance to NRTIs and PIs are lower in HIV-2 (23–25).

## HIV-2 and Pregnancy

The risk of vertical transmission is lower for HIV-2 than for HIV-1. For women of reproductive age, effective, sparing ART regimens are recommended with regards to the possibility of pregnancy. In pregnant women with HIV-2 infection, the procedure is the same as in HIV-1 infection. ART is usually initiated from the 12th week of pregnancy, but according to DHHS recommendations, it can be offered to all pregnant women with HIV-2 infection, even in the case of undetected RNA levels and the absence of comorbidities. CD4 and VL checks are done every 1–3 months. If the woman is already treated with ART and the treatment is not toxic to the foetus, the same scheme is continued. The change is approached individually depending on clinical status of the mother, HIV-2 genotype, adherence and tolerance of ART. The safety information in the SPC must be observed. DTG and BIC are now considered as safe and generally recommended treatment in pregnancy. It is better to avoid regimens containing, cobicistat and TAF. EVG/c is not recommended during pregnancy because of insufficient levels in the 2nd and 3rd trimesters. On the other hand, DRV/r twice a day is considered suitable in the third trimester. If there are detectable levels of viral RNA shortly before birth, it is advisable to add RAL and consider the need for a caesarean section. In case of complications in childbirth/perinatal exposure with a risk of vertical infection, the newborn is given AZT i.v./p.o. for one month. Complete three-component post-exposure prophylaxis is given to the newborn if the mother's VL is unknown for a longer period of time or if its value in the prenatal period was higher than the detection limit (26).

## Postexposure Prophylaxis of HIV-2 infection

Administration of TAF/TDF + FTC/3TC together with RAL or DRV/r is recommended in case of high-risk contact with both

untreated and treated PWH, infected with HIV-2 with detected or unknown VLs. In previously treated patients, it is advisable to select the components of ART with regard to previous identified resistances/treatment failures (26).

## Preexposure Prophylaxis of HIV-2 Infection

Preexposure prophylaxis (PrEP) is also being discussed as a possible prevention of the spread of HIV-2, although the risk of infection during sexual contact is about five times lower than with HIV-1. Improving access to PrEP could increase PrEP use among all eligible MSM but should include public health strategies to target socioeconomic and demographic disparities in the knowledge and use of PrEP (34).

## CONCLUSIONS

Compared to HIV-1, there is still limited data available about HIV-2 in all of the above-mentioned areas. Diagnostics using modern tests to quantify HIV-2 can be performed by national reference laboratories. The spectrum of suitable antiretroviral drugs is limited. Existing national guidelines are based on clinical experience, in vitro tests and information derived from HIV-1 studies. Information on resistance and tropism of HIV-2 in particular countries are very limited. Further cooperation between clinicians and researchers, especially from countries with a higher incidence of HIV-2, is needed.

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**Conflicts of Interest**

None declared

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