Despite major advances in treatment over recent decades, the human immunodeficiency virus (HIV) remains a significant and complex global health issue. Nurses have a vital and diverse role in meeting the challenges posed by HIV, particularly due to the complex holistic factors that determine the overall health status of people living with the virus. This article discusses HIV prevalence, pathophysiology and modes of transmission.

**Definition and epidemiology**

HIV targets and destroys essential components of the human immune system. It belongs to a genus of viruses called lentiviruses, which cause chronic disease in humans and other mammals. Left untreated, HIV infection causes progressive and critical damage to the immune system, rendering the host susceptible to potentially fatal opportunistic infections and cancers.

Use of the term AIDS (acquired immune deficiency syndrome) is now rare, but it was previously used to describe the advanced stage of HIV infection, in which the immune system has declined to such a level that infections and cancers can emerge and proliferate. This phase of the infection is now more commonly called advanced or late-stage HIV infection.

HIV is one of the most devastating infectious diseases to have emerged in recent history, affecting millions of people worldwide. The virus is believed to have originated in West Africa in the early 20th century and it is widely accepted that it crossed from chimpanzee subspecies to humans in a process called zoonosis (Sharpa and Hahn, 2011) – most likely via bushmeat hunting and consumption.

The first major clusters of HIV infection appeared in the US in the early 1980s, when infections and cancers that were normally prevalent in immunosuppressed populations began to emerge in fit and healthy homosexual men. A New York Times story by Lawrence Altman (1981) announced a:

“Rare cancer seen in 41 homosexuals: outbreak occurs among men in New York and California – eight died inside two years”.

This unexplained acquired immunodeficiency was initially described by the press as gay-related immune disease because it disproportionately affected homosexuals, although it was also seen in people with haemophilia and those who injected drugs intravenously. By 1986, the virus had been isolated and identified, but had already
infected thousands of people across 85 countries worldwide (Bureau of Hygiene and Tropical Diseases, 1986). In 1989, the global spread of HIV was coined an epidemic, with over 400,000 cases reported (World Health Organization, 1989). In 2018, it was estimated that there were 37.9 million people living with HIV worldwide; 770,000 related deaths were recorded that year (WHO, 2018).

The virus has the highest prevalence in sub-Saharan Africa; it is estimated that 68% of people living with HIV are in this region (Avert, 2020). The number of global deaths from HIV has fallen by >50% in the past 15 years due to improved treatment (WHO, 2018), but still remains a challenge in resource-poor nations. In the UK, estimates suggest there are 103,600 people living with HIV – men who have sex with men and Black African populations are disproportionately affected (Public Health England, 2019a). Across the UK, new HIV diagnoses declined between 2013 and 2018, driven by a reduction in the number of diagnoses in gay and bisexual men (PHE, 2019b).

Pathophysiology

Viral structure

Viruses cannot survive or replicate without a host, and can only do so inside a living cell. Viral structure is simple. Viruses lack the complex components present in bacterial or human cells, so HIV is solely dedicated to identifying and infecting its target.

One commonality of viral, bacterial and human cells is that they possess genetic material that contains all the information needed to build and maintain an organism. In humans and bacteria, this is called deoxyribonucleic acid (DNA), which comprises two strands of genetic information twisted into a helix. In viruses such as HIV, genetic data is organised into single strands; this is called ribonucleic acid (RNA).

The RNA present in a virus is protected by a protein coat called a capsid. Outside the capsid are enzymes the virus uses to infect its host and replicate. These structures are surrounded by an envelope comprising glycoproteins, which help the virus identify and bind to its target (Fig 1).

Viral targets

The human immune system has many vital cells that fight infection and destroy abnormal cells; this includes lymphocytes called T-cells, which determine the immune system’s response to foreign antigens. HIV targets and infects a particular type of T-cell called CD4 ‘helper’ cells. These are so called because they do not kill or neutralise foreign antigens but, instead, signal to and recruit other immune cells to do so.

After entering a host’s body, HIV rapidly seeks out the CD4 cells and infects them. The virus commandeers the function of the CD4 cells, turning them into factories that produce multiple new copies of the virus; between 10 million and 10 billion new virus cells can be produced daily. Once infected, CD4 cells develop a much shorter lifespan and are eventually destroyed; their progressively declining number in the host causes immunological failure and susceptibility to infection.

Box 1 lists the stages of HIV infection; these are all targets for HIV medications that interrupt the lifecycle of the virus and inhibit infection and replication. Use of these medications is known as anti-retroviral therapy.

Stages of HIV infection

The course of HIV infection is often divided into different phases (Table 1) but, in practice, it is not easy to precisely demarcate patients into separate, distinct stages. Several factors determine individual progression of HIV, such as genetics and comorbidities. As HIV is very genetically diverse and often mutates, viral factors – such as deletions in certain viral genes, viral subtype and coreceptor usage – can also determine the rate of HIV progression. As such, the phases are used as a guide.

Fig 2 shows a typical representation of HIV infection, from initial infection to death if the virus is not treated. It illustrates how the CD4 count declines over time and outlines the trajectory of the viral load.

Transmission

HIV can only be acquired through specific activities. It is vital for health professionals to have a good understanding of the modes of transmission, not only to protect staff and patients, but also to help reduce myths and inaccuracies that drive the social stigma faced by people living with HIV.

Only certain bodily fluids can transmit the virus: semen, pre-semenal fluid, blood, rectal fluid, vaginal fluid and breast milk. To transmit HIV, one of these fluids must come into contact with a mucous membrane (such as the mouth, vagina or rectum) or damaged tissue, or be directly injected into the bloodstream. HIV is not transmitted through saliva, sweat or tears, and cannot be spread by ticks or mosquitoes, social contact, touch or sharing food with somebody who has HIV.

Possible modes of HIV transmission are:

● Having anal or vaginal intercourse with someone who has HIV without using a condom or taking medicines that treat or prevent HIV;

● Receiving a transfusion of infected blood or blood products – however this

Box 1. Cycle of HIV infection of CD4 cells

Binding and entry – the virus seeks out the CD4 cell and attaches itself to receptors on the cell’s outer membrane; it then fuses itself to the cell and releases viral RNA and enzymes into it

Reverse transcription – the virus converts its single-stranded viral RNA into double-stranded DNA using an enzyme called reverse transcriptase

Integration – the virus integrates its newly created viral DNA into the CD4 cell’s nucleus using an enzyme called integrase; by integrating its genetic instructions, it commandeers the CD4 cell

Replication – the CD4 cell starts to build new copies of the virus; this process can sometimes be clumsy, causing mutations and variations in the new virions

Budding and maturation – the new HIV virions migrate towards the outer membrane of the CD4 cell. An enzyme called protease helps convert the immature virions into mature, infectious virions. They then push themselves out of the cell, which is called budding, and seek out other CD4 cells to repeat the process

DNA = deoxyribonucleic acid; RNA = ribonucleic acid.
is now very rare because all donations are screened for bloodborne viruses and donors are risk stratified in most countries:

- **Mother-to-child transmission** through pregnancy, birth or breastfeeding;
- **Sharing needles or syringes** with someone who has HIV;
- Through **oral sex** – this is theoretically possible if ejaculate from a male who is HIV positive penetrates the oral membrane of a person who is HIV negative but this is extremely rare;
- Receiving an **injury from a sharp**, such as a needle, is a risk for health professionals, but it is very uncommon that this leads to HIV acquisition.

**Undetectable equals untransmittable**

People receiving HIV treatment can have a viral load test; this measures the amount of virus per millilitre of blood to determine whether the treatment used is adequately suppressing viral replication. The measurement is recorded as copies per millilitre. The standard viral load tests used in clinics can generally measure viral load to as low as 20 or 40 copies per millilitre of blood. Anything lower than this is considered undetectable and means the person has very little virus to the point that there is not enough for it to be transmitted to someone else.

Following several prominent studies (such as those by Rodger et al, 2019; 2016), more than 770 organisations from 95 countries have endorsed the “undetectable equals untransmittable” (U=U) statement. This means people living with HIV cannot sexually transmit the virus to a partner if they are consistently and correctly taking anti-retroviral therapy and have achieved an undetectable viral load in their blood for at least six months. This has been endorsed by the British HIV Association (2018) and is advocated in UK practice.

**Table 1. Phases of HIV infection**

<table>
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<tr>
<th>Phase</th>
<th>Description</th>
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<tr>
<td>Acute phase</td>
<td>The acute phase refers to the immediate weeks after initial infection with HIV. During this time, the virus rapidly multiplies and spreads, attacking CD4 cells at a significant pace. The level of HIV in the blood is very high, which greatly increases the risk of transmission. There is an initial drop in the number of CD4 cells, associated with the high level of circulating virus. Antibodies to HIV are produced during the early weeks of infection (seroconversion), which typically creates flu-like symptoms</td>
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<tr>
<td>Chronic latent phase</td>
<td>After initial infection, the body develops antibodies to the HIV virus; this slows the viral replication. The virus continues to multiply, but at a reduced pace. Patients are usually asymptomatic during this phase but may experience minor symptoms as their number of CD4 cells continues to decline. People on effective HIV treatment will often stay in this stage because the virus is suppressed by medication. Damage to CD4 cells is, therefore, significantly reduced and their total number can recover</td>
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<tr>
<td>Advanced HIV infection (previously called AIDS)</td>
<td>Without treatment, chronic infection usually progresses to an advanced stage in an average of 8–10 years (HIV i-Base, 2018), although this can vary depending on virus and host factors. In the most advanced stage of infection, the immune system is severely damaged and unable to fight infections that an HIV-negative immunocompetent person would be able to fight (called opportunistic infections). People who have a CD4 cell count of &lt;200 cells/mm³ or develop certain opportunistic infections or cancers are diagnosed with advanced HIV infection</td>
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AIDS = acquired immune deficiency syndrome. HIV = human immunodeficiency virus.

**References**

HIV i-Base (2016) How CD4 and Viral Load are Related. HIV i-Base.  
WHO (2018) Number of Deaths Due to HIV/AIDS. WHO.  