Chinese Guidelines for the Diagnosis and Treatment of AIDS (2018 Edition) Post-print

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Abstract

AIDS represents a significant public health challenge in China. In 2005, the AIDS Group of the Infectious Diseases Branch of the Chinese Medical Association established the first edition of the "Guidelines for the Diagnosis and Treatment of AIDS," with subsequent updates in 2011 and 2015. The 2018 version represents an update to the third edition, incorporating domestic clinical practice and the latest research findings on AIDS in China. The revisions primarily reflect advances in the diagnosis and treatment of opportunistic infections and human immunodeficiency virus (HIV)-related tumors, antiretroviral therapy, HIV post-exposure prophylaxis, and prevention of mother-to-child transmission. The 2018 guidelines introduced HIV post-exposure prophylaxis for the first time, proposed the novel concept of whole-course management of HIV infection, and provided detailed recommendations on its implementation. These guidelines will be regularly updated based on the latest clinical research evidence.

Full Text

Preamble

Chinese Guidelines for Diagnosis and Treatment of HIV/AIDS (2018 Edition)

AIDS and Hepatitis C Professional Group, Society of Infectious Diseases, Chinese Medical Association; Chinese Center for Disease Control and Prevention

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Abstract

Acquired immunodeficiency syndrome (AIDS) represents a significant public health challenge in China. The first edition of the Chinese Guidelines for Diag-

nosis and Treatment of AIDS was formulated in 2005 by the AIDS Professional Group of the Society of Infectious Diseases, Chinese Medical Association, with subsequent updates in 2011 and 2015. The 2018 fourth edition represents a revision based on the third edition, incorporating the latest domestic clinical practices and research findings. Key updates include new research progress in opportunistic infections, HIV-associated malignancies, antiretroviral therapy, post-exposure prophylaxis, and prevention of mother-to-child transmission. Notably, the 2018 edition introduces pre-exposure prophylaxis (PrEP) for the first time and proposes the concept of whole-course management of HIV infection, providing detailed guidance on its implementation. These guidelines will be regularly updated according to the latest clinical evidence.

Keywords: acquired immunodeficiency syndrome; human immunodeficiency virus; diagnosis; treatment; guideline

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1. Epidemiology

1.1 Current Epidemiological Status

HIV/AIDS has become a major public health threat in China. According to UNAIDS estimates, China has reported [number] living HIV cases and [number] deaths in [year]. In [year], newly identified HIV/AIDS cases were reported, with over 90% attributed to sexual transmission. The "90-90-90" targets aim to control annual new infections below [threshold] through comprehensive, intensive interventions.

1.2 Sources of Infection

HIV primarily exists in the blood, semen, vaginal secretions, breast milk, and other bodily fluids of infected individuals and AIDS patients.

1.3 Routes of Transmission and Infection

Transmission occurs through: (1) sexual contact (including unsafe homosexual, heterosexual, and bisexual contact); (2) blood and blood products (including intravenous drug use with needle sharing, unsafe medical procedures, transfusion of contaminated blood); and (3) mother-to-child transmission (including intrauterine infection, during delivery, and through breastfeeding).

1.4 High-Risk Populations

High-risk groups include men who have sex with men (MSM), individuals with multiple sexual partners, commercial sex workers, intravenous drug users, and sexual partners of HIV/AIDS patients.

1.5 Disease Reporting

According to the Law of the People' s Republic of China on Prevention and Treatment of Infectious Diseases, HIV/AIDS patients must report their condition to local disease prevention and control centers promptly. Healthcare providers should offer voluntary counseling and testing (VCT) and provider-initiated testing and counseling (PITC) while maintaining confidentiality.

1.6 Preventive Measures

Prevention strategies include: consistent condom use, safe sexual practices, avoiding needle sharing, strengthening hospital infection control, promoting voluntary blood donation, screening blood donors, strict sterilization protocols, and preventing occupational exposure.

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2. Pathogen Characteristics

HIV belongs to the Lentivirus genus of the Retroviridae family. The virus particle is spherical, 100-120 nm in diameter, consisting of a core and envelope. The core contains capsid protein (p24), two identical single-stranded RNA genomes, and essential enzymes (reverse transcriptase p51/p66, integrase p32, and protease p10). The envelope, derived from host cell membrane, contains embedded glycoproteins gp120 and gp41. Beneath the envelope lies matrix protein p17.

The HIV genome is approximately 9.7 kb, containing three structural genes (*gag*, *pol*, *env*), two regulatory genes (*tat*, *rev*), and four accessory genes (*vif*, *vpr*, *vpu*, *nef*). HIV exhibits high genetic variability, particularly in the *env* gene, due to: (1) error-prone reverse transcriptase without proofreading function, (2) high-frequency replication, (3) host immune pressure, (4) inter-subtype recombination, and (5) drug selection pressure.

HIV-1 is the predominant circulating strain in China, with subtypes including B, B' , C, D, F, G, H, J, and various circulating recombinant forms (CRFs). The main subtype is CRF01_AE. HIV-2 has been identified in some regions.

HIV enters cells via CD4 receptors and co-receptors (CCR5 or CXCR4). Early-stage viruses typically use CCR5, while late-stage viruses often utilize CXCR4. The viral replication cycle involves: (1) attachment and membrane fusion, (2) reverse transcription, (3) nuclear entry and integration, (4) transcription and translation, and (5) assembly and budding.

HIV has weak viability outside the body and is sensitive to heat (56°C for 30 minutes reduces infectivity; 100°C for 20 minutes inactivates the virus). Standard disinfectants effective against hepatitis B virus (70% alcohol, sodium hypochlorite) also inactivate HIV.

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3. Laboratory Testing

Laboratory testing for HIV/AIDS includes: HIV-1/2 antibody detection, CD4+ T lymphocyte count, HIV nucleic acid (qualitative and quantitative) testing, and drug resistance testing. These tests are essential for diagnosis, disease staging, treatment efficacy monitoring, and prognosis assessment.

3.1 HIV-1/2 Antibody Testing

Antibody testing comprises screening and confirmatory tests. Screening methods include enzyme-linked immunosorbent assay (ELISA), chemiluminescence immunoassay, and rapid tests. Confirmatory tests include Western blot and nucleic acid testing. A negative screening test indicates no HIV infection (except during window period). Positive screens require repeat testing with two different assays. Discordant or dual-positive results require confirmatory testing. Confirmatory tests reporting "HIV-1/2 antibody positive" confirm infection; "indeterminate" results require nucleic acid testing or follow-up.

3.2 CD4+ T Lymphocyte Testing

CD4+ T cells are the primary target of HIV infection. Progressive CD4 depletion indicates disease progression and immune dysfunction. Flow cytometry is the standard method. CD4 count guides treatment initiation, efficacy assessment, and opportunistic infection risk stratification. Treatment-naive patients should have CD4 testing every 3-6 months; patients on stable HAART can be tested every 6-12 months.

3.3 HIV Nucleic Acid Testing

Plasma HIV RNA quantification (viral load) predicts disease progression and monitors treatment response. Methods include reverse transcription PCR (RT-PCR), nucleic acid sequence-based amplification (NASBA), and real-time fluorescence quantitative PCR. Treatment-naive patients should have baseline viral load; after HAART initiation, testing at 4-8 weeks, then every 3-4 months until suppression, then every 6-12 months for stable patients.

3.4 HIV Genotypic Resistance Testing

Resistance testing guides regimen selection. Testing is recommended: (1) before HAART initiation, (2) when viral load fails to suppress or rebounds, (3) for pregnant women, and (4) for recently infected individuals. Testing should be performed while on antiretrovirals; if stopped, within 4 weeks. Results indicating resistance suggest possible drug resistance, but must be interpreted with clinical context, adherence, and pharmacokinetics.

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4. Pathogenesis

HIV primarily attacks the immune system, targeting CD4+ T lymphocytes, monocytes/macrophages, and dendritic cells. Progressive CD4 depletion leads to cellular immune deficiency, opportunistic infections, and malignancies.

After exposure, HIV reaches regional lymph nodes within 24-48 hours, with detectable viremia in peripheral blood. Acute infection causes transient CD4 decline, which typically recovers without treatment. However, incomplete viral clearance leads to chronic infection (asymptomatic and symptomatic phases). CD4 counts decline gradually during chronic infection due to: (1) direct viral cytotoxicity, (2) apoptosis/pyrosis, (3) immune-mediated destruction, and (4) thymic damage.

Host immune responses include innate immunity and adaptive immunity (neutralizing antibodies, non-neutralizing inhibitory antibodies, CD8+ cytotoxic T lymphocytes). HAART can restore immune function to near-normal levels.

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5. Clinical Manifestations and Staging

HIV infection progresses through three stages: acute phase, asymptomatic phase, and AIDS phase. Clinical progression varies based on viral load, host immunity, genetics, nutrition, and lifestyle.

5.1 Acute Phase

Occurs 2-4 weeks after infection, characterized by viremia and acute immune injury. Symptoms are usually mild and self-limiting, with fever being most common, accompanied by sore throat, rash, arthralgia, lymphadenopathy, and neurological symptoms. HIV RNA and p24 antigen are detectable; CD4 count transiently decreases with CD4/CD8 ratio inversion.

5.2 Asymptomatic Phase

May follow acute phase or occur directly after infection, lasting 6-8 years on average. Patients generally have no symptoms except possible persistent lymphadenopathy. HIV continuously replicates with gradual CD4 decline.

5.3 AIDS Phase

The final stage marked by CD4 <200 cells/ L and high viral load. Clinical manifestations include: (1) persistent fever >1 month, (2) chronic diarrhea >1 month, (3) weight loss >10%, (4) neurological symptoms, (5) persistent generalized lymphadenopathy, and (6) opportunistic infections or malignancies.

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6. Diagnostic Criteria

Diagnosis requires comprehensive analysis of epidemiological history, clinical manifestations, and laboratory findings.

6.1 Acute Phase Diagnosis

Epidemiological history within 6 months plus laboratory diagnosis criteria, or positive HIV antibody screening with confirmatory test.

6.2 Asymptomatic Phase Diagnosis

No clear epidemiological history but meets laboratory diagnostic criteria.

6.3 AIDS Phase Diagnosis

For adolescents and adults: HIV-positive with CD4 <200 cells/ L, or HIV-positive with any AIDS-defining condition (opportunistic infections, malignancies, wasting syndrome, etc.).

For children: Different CD4 percentage thresholds apply by age group (<25% for <12 months, <20% for 12-36 months, <15% for 37-60 months, <200 cells/ L for 5 years) plus HIV-positive status.

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7. Opportunistic Infections

7.1 Pneumocystis Pneumonia (PCP)

7.1.1 Clinical Features: Subacute onset with progressive dyspnea, fever, dry cough, and hypoxemia. Physical signs are minimal relative to symptom severity. Chest X-ray shows diffuse interstitial infiltrates; CT reveals ground-glass opacities. Diagnosis requires identification of Pneumocystis organisms in sputum, BAL fluid, or lung tissue.

7.1.2 Treatment: First-line therapy is trimethoprim-sulfamethoxazole (TMP-SMX) for 21 days. For mild-moderate disease: TMP 15-20 mg/kg/day + SMX 75-100 mg/kg/day orally in 3-4 divided doses. For severe disease: IV administration at same doses, possibly extended. Alternatives include primaquine + clindamycin. Adjunctive corticosteroids are recommended for PaO <70 mmHg.

7.1.3 Primary Prophylaxis: Indicated for CD4 <200 cells/ L. TMP-SMX is preferred; alternatives include dapsone or atovaquone. Prophylaxis can be discontinued after CD4 >200 cells/ L for 3 months on HAART.

7.2 Tuberculosis (TB)

7.2.1 Diagnosis: HIV-TB coinfection presents uniquely. Diagnosis requires clinical, pathological, and imaging correlation. CD4 count influences presen-

tation: higher counts resemble typical TB; lower counts show disseminated or extrapulmonary disease. Acid-fast smear and culture remain diagnostic gold standards.

7.2.2 Treatment: Principles mirror non-HIV TB treatment but require attention to drug interactions with antiretrovirals. Standard regimen: 2-month intensive phase (isoniazid, rifampin, pyrazinamide, ethambutol) followed by 4-month continuation phase (isoniazid + rifampin). For drug-susceptible TB, total duration is 6 months; for CNS or bone/joint TB, extend to 9-12 months. All HIV-TB patients should receive HAART regardless of CD4 count.

7.2.3 Timing of HAART: For CD4 <50 cells/ L, initiate HAART within 2 weeks of starting TB treatment; for CD4 50 cells/ L, initiate within 8 weeks. For pregnant women or those with CNS TB, earlier HAART initiation is recommended.

7.2.4 IRIS Management: TB-IRIS is common but rarely fatal. Continue HAART and TB therapy; short-course corticosteroids may be used for severe cases.

7.2.5 Preventive Therapy: For latent TB infection (positive TST/IGRA), isoniazid 300 mg daily for 6-9 months is recommended, with pyridoxine to reduce neuropathy.

7.3 Nontuberculous Mycobacterial (NTM) Infection

7.3.1 Clinical Features: Most common is Mycobacterium avium complex (MAC), presenting with disseminated disease, fever, weight loss, anemia, hepatosplenomegaly, and lymphadenopathy. Diagnosis requires culture or molecular identification from blood, bone marrow, or sterile sites.

7.3.2 Treatment: Preferred regimen: clarithromycin 500 mg twice daily + ethambutol 15 mg/kg/day ± rifabutin 300 mg daily. For severe immunosuppression (CD4 <50 cells/ L), add amikacin or fluoroquinolone. Continue until CD4 >100 cells/ L for 6 months on HAART.

7.3.3 Primary Prophylaxis: For CD4 <50 cells/ L and MAC-positive, azithromycin 1200 mg weekly or clarithromycin 500 mg twice daily is recommended. Discontinue when CD4 >100 cells/ L for 3 months.

7.4 Cytomegalovirus (CMV) Infection

7.4.1 CMV Retinitis: Presents with floaters, visual field defects, or rapid vision loss. Fundoscopy shows yellow-white retinal lesions with hemorrhage. Treatment: ganciclovir 5 mg/kg IV every 12 hours for 14-21 days, then maintenance therapy. Oral valganciclovir is an alternative.

7.4.2 CMV Pneumonia/Enteritis: Diagnosis is challenging, requiring clinical, imaging, and histopathological evidence. Treatment: ganciclovir ± foscarnet for 3-4 weeks or until symptom resolution.

7.4.3 HAART: Initiate HAART within 2-4 weeks of anti-CMV therapy.

7.4.4 Secondary Prophylaxis: Oral valganciclovir 900 mg daily until CD4 >100 cells/ L for 6 months.

7.5 Herpes Simplex and Varicella-Zoster Virus Infections

7.5.1 Clinical Features: HSV causes oral/genital ulcers; VZV causes dermatomal or disseminated rash.

7.5.2 Treatment: Acyclovir 400 mg three times daily for 5-14 days depending on severity. For acyclovir-resistant HSV, use foscarnet.

7.6 Toxoplasmic Encephalitis

7.6.1 Clinical Features: Presents with fever, headache, focal neurological deficits, seizures. CT/MRI shows multiple ring-enhancing lesions. Diagnosis requires clinical, serological (positive toxoplasma IgG), and radiological correlation; definitive diagnosis requires brain biopsy.

7.6.2 Treatment: Pyrimethamine 100 mg loading dose, then 50-75 mg/day + sulfadiazine 1-1.5 g four times daily + leucovorin for 6 weeks, then maintenance until CD4 >200 cells/ L for 6 months. Alternatives include TMP-SMX or clindamycin + pyrimethamine.

7.6.3 Primary Prophylaxis: For CD4 <100 cells/ L and toxoplasma IgG-positive, TMP-SMX is recommended.

7.7 Fungal Infections

7.7.1 Candidiasis: Oral candidiasis is treated with nystatin suspension or fluconazole 100-200 mg daily for 7-14 days. Esophageal candidiasis requires fluconazole 200-400 mg daily for 14-21 days.

7.7.2 Cryptococcal Meningitis: Induction therapy with amphotericin B (0.5-0.7 mg/kg/day) + flucytosine (100 mg/kg/day) for 2 weeks until CSF culture negative. Consolidation with fluconazole 400-800 mg daily for 8 weeks, then maintenance with fluconazole 200 mg daily until CD4 >100 cells/ L for 6 months on HAART. Manage increased intracranial pressure aggressively.

7.7.3 Talaromycosis (Penicilliosis): Common in southern China. Treatment: amphotericin B induction, then itraconazole 200 mg twice daily for 10 weeks, then maintenance until CD4 >100 cells/ L for 6 months.

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8. Antiretroviral Therapy (ART)

8.1 Treatment Goals

Maximally suppress viral replication, restore immune function, reduce morbidity and mortality, improve quality of life, and prevent transmission.

8.2 Available Antiretroviral Drugs

Six drug classes are available internationally: NRTIs, NNRTIs, PIs, INSTIs, FIs, and CCR5 antagonists. In China, available drugs include:

- **NRTIs:** AZT, 3TC, TDF, FTC, ABC, TAF
- **NNRTIs:** EFV, NVP, RPV
- **PIs:** LPV/r, DRV/c
- **INSTIs:** RAL, DTG, EVG/c

provides detailed drug information including dosing, adverse effects, and interactions.

8.3 Timing and Regimens for Adults and Adolescents

8.3.1 Treatment Initiation: Immediate ART is recommended for all HIV-positive individuals regardless of CD4 count, after addressing opportunistic infections and ensuring patient readiness.

8.3.2 Initial Regimens: Recommended backbone: two NRTIs (TDF/FTC or ABC/3TC) plus a third agent: - NNRTI: EFV or RPV - PI: LPV/r or DRV/c - INSTI: DTG or RAL

Single-tablet regimens are preferred when available.

8.4 Special Populations

8.4.1 Children: All HIV-infected children should initiate ART immediately. Preferred regimens vary by age and weight. For infants <3 years: AZT+3TC+LPV/r. For older children: ABC+3TC+EFV or TDF+3TC+EFV. Monitor growth, development, and adherence closely.

8.4.2 Pregnant Women: All pregnant women should receive lifelong ART. Preferred regimen: TDF/FTC (or ABC/3TC) + LPV/r or RAL. EFV can be used in all trimesters. Avoid NVP if CD4 >250 cells/ L. RAL is preferred for high viral loads.

8.4.3 Breastfeeding: Breastfeeding is not recommended due to transmission risk. If chosen, exclusive breastfeeding for 6 months while mother and infant receive ART.

8.4.4 TB Coinfection: Standard regimen: TDF/FTC + EFV (or RPV). Rifampin reduces PI and INSTI levels; consider rifabutin instead. Initiate HAART within 2-8 weeks of TB treatment based on CD4 count.

8.4.5 HBV Coinfection: Use TDF/FTC (or TAF/FTC) backbone to treat both viruses. Avoid 3TC or FTC alone. Monitor HBV DNA and liver function.

8.4.6 HCV Coinfection: Treat both infections. Use INSTI-based HAART with minimal hepatotoxicity. DAA regimens for HCV require assessment of drug interactions.

8.4.7 Renal Insufficiency: Adjust TDF dosing if eGFR <60 mL/min; avoid if <30 mL/min. TAF has less renal toxicity.

8.5 Treatment Monitoring

8.5.1 Virologic Monitoring: Viral load should decrease by 1 log within 4-8 weeks and become undetectable (<50 copies/mL) by 3-6 months. Test every 3-4 months until suppressed, then every 6-12 months.

8.5.2 Immunologic Monitoring: CD4 count increases by 50-150 cells/ L in the first year indicates good response. Test every 3-6 months initially, then every 6-12 months when stable.

8.5.3 Clinical Monitoring: Assess weight gain, opportunistic infections, drug toxicities, and adherence at each visit.

8.5.4 Drug Concentration Monitoring: Therapeutic drug monitoring (TDM) is recommended for: patients with drug interactions, hepatic/renal impairment, pregnancy, suspected treatment failure despite good adherence, and selected drugs with narrow therapeutic windows.

8.6 Regimen Switching and Treatment Failure

Virologic Failure: Defined as viral load >200 copies/mL after previous suppression, or failure to achieve suppression by 6 months.

Management: Assess adherence first. Perform resistance testing. Switch to at least two fully active drugs, preferably a new class. Options include INSTI-based or PI-based regimens.

Regimen Simplification: May be considered for virologically suppressed patients to reduce pill burden, improve tolerability, or avoid interactions. Supported two-drug simplification options include: - DTG + RPV - DTG + 3TC - DRV/r + RAL - PI + 3TC

8.7 Drug Interactions

ART drugs interact with many medications through CYP450 metabolism. Key interactions include: - Rifampin significantly reduces NNRTI, PI, and INSTI levels - Acid-reducing agents reduce ATV and RPV absorption - Statins: avoid simvastatin/lovastatin with PIs; use pravastatin or rosuvastatin - Methadone: monitor for withdrawal with EFV or NVP - Antifungals: azoles increase PI levels

9. Immune Reconstitution Inflammatory Syndrome (IRIS)

9.1 Diagnosis

IRIS is a clinical syndrome occurring after HAART initiation, characterized by paradoxical worsening of pre-existing infections or emergence of subclinical infections. Diagnostic criteria: 1. Temporal association with HAART initiation (typically within 3 months) 2. Clinical deterioration despite virologic suppression and CD4 improvement 3. Exclusion of treatment failure, drug toxicity, or new infection

Common manifestations include TB-IRIS, cryptococcal-IRIS, CMV-IRIS, and Kaposi' s sarcoma-IRIS.

9.2 Management

Continue HAART and treat the underlying infection. Mild IRIS is self-limiting. Moderate-severe cases may require short-course corticosteroids (except for Kaposi' s sarcoma). NSAIDs can be used for symptom control.

9.3 Risk Factors

Low baseline CD4 count (<50 cells/ L) and high baseline viral load are major risk factors. Pre-emptive treatment of opportunistic infections before HAART initiation reduces IRIS risk.

10. HIV-Related Tumors

Major HIV-associated malignancies include non-Hodgkin lymphoma and Kaposi' s sarcoma. Diagnosis requires histopathology. Management involves individualized comprehensive therapy (chemotherapy, surgery, radiation) with concurrent HAART. Multidisciplinary care is essential. All patients with HIV-related tumors should start HAART early, considering drug interactions and myelosuppression.

11. Prevention of Mother-to-Child Transmission (PMTCT)

11.1 Principles

PMTCT requires comprehensive measures: (1) ART for all pregnant women regardless of CD4 count, (2) safe delivery practices, (3) infant prophylaxis, and (4) appropriate feeding guidance.

11.2 Safe Delivery

Infected pregnant women should deliver in healthcare facilities. Avoid invasive procedures (fetal scalp monitoring, episiotomy, forceps) when possible. Elective cesarean section is not routinely recommended with viral suppression.

11.3 Postpartum Feeding

Artificial feeding is recommended. If breastfeeding is chosen due to socioeconomic constraints: (1) exclusive breastfeeding for 6 months, (2) mother and infant must receive ART, (3) stop breastfeeding abruptly at 6 months, (4) no mixed feeding.

11.4 Infant Follow-Up

HIV-exposed infants should receive: - NVP prophylaxis for 6 weeks - HIV DNA/RNA testing at birth, 6 weeks, and 3 months - Antibody testing at 12 and 18 months - Routine pediatric care and nutritional support

11.5 Discordant Couples and Reproductive Options

Male-negative, female-positive: Can conceive naturally if female achieves viral suppression on HAART. Sperm donation eliminates transmission risk.

Male-positive, female-negative: Natural conception is possible when male achieves sustained viral suppression (>6 months, undetectable viral load). If viral suppression is not achieved, consider sperm washing with IVF, or PrEP for female partner (TDF/FTC) starting 1 month before conception attempts.

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12. HIV Exposure Management

12.1 Occupational Exposure

Definition: Percutaneous, mucous membrane, or non-intact skin contact with HIV-infected blood or body fluids during healthcare or public safety work.

Risk Assessment: Transmission risk is approximately 0.3% for percutaneous and 0.09% for mucous membrane exposure. Risk increases with deep injury, visible blood, hollow-bore needle, and high viral load source.

Post-Exposure Prophylaxis (PEP): - Initiate within 2 hours, ideally within 24 hours (may be considered up to 72 hours) - Regimen: TDF/FTC + INSTI (RAL or DTG) for 28 days - Monitor: Baseline and follow-up HIV testing (6 weeks, 12 weeks, 6 months), CBC, LFTs

Prevention: Standard precautions, use of safety devices, proper sharps disposal.

12.2 Non-Occupational Exposure (nPEP)

Assessment and management follow similar principles as occupational PEP. Initiation within 72 hours is recommended for high-risk exposures (unprotected sex with HIV-positive partner, sexual assault, needle sharing).

12.3 HBV Coinfection in Exposed Persons

HBV vaccination is recommended for all susceptible individuals. For those with isolated anti-HBc, consider HBV DNA testing. Vaccine response may be blunted in advanced HIV; consider double-dose vaccination or revaccination after immune reconstitution.

12.4 Pre-Exposure Prophylaxis (PrEP)

Definition: Daily medication for HIV-negative individuals at high risk to prevent infection.

Indications: MSM with inconsistent condom use, serodiscordant couples, PWID.

Regimen: TDF/FTC daily. Alternative: on-demand dosing (2-1-1) for MSM.

Monitoring: HIV testing every 3 months, renal function every 6 months.

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13. Comprehensive HIV Management

HAART has transformed HIV into a manageable chronic disease requiring long-term, multidisciplinary care. The whole-course management model encompasses:

13.1 Prevention and Early Diagnosis

- Risk reduction counseling, condom promotion
- PrEP and PEP services
- Regular screening with sensitive assays (including nucleic acid testing)
- Partner notification and testing

13.2 Opportunistic Infection Management

- Primary prophylaxis based on CD4 count
- Prompt diagnosis and treatment
- IRIS prevention and management
- Vaccination (HBV, pneumococcal, influenza)

13.3 Individualized ART

- Adherence counseling before initiation
- Regimen selection based on: comorbidities, drug interactions, pregnancy status, renal/hepatic function, resistance profile
- Simplified regimens when appropriate (two-drug maintenance)
- TDM for selected patients
- Regular monitoring for toxicity and resistance

13.4 Non-AIDS Defining Conditions

Screen and manage comorbidities including: - Cardiovascular disease, diabetes, dyslipidemia - Chronic kidney and liver disease - Osteoporosis and fractures - Non-AIDS malignancies - Neurocognitive impairment - Mental health disorders

13.5 Psychosocial Support

- Mental health screening and treatment
- Substance use disorder management
- Social support services
- Palliative and end-of-life care when needed
- Geriatric assessment for aging patients

This comprehensive, patient-centered approach improves quality of life, reduces mortality, and addresses the evolving needs of people living with HIV throughout their lifespan.

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Note: Figure translations are in progress. See original paper for figures.