REVIEW ARTICLE

Acute HIV Infection

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SUMMARY

Acute HIV infection occurs in 85% of people recently infected with HIV-1 after an incubation period of 2–6 weeks. Diagnosing the acute HIV infection is difficult because the symptoms are non-characteristic and include fever, pharyngitis, and skin rash. Early recognition of HIV infection is, of course, most desirable in order to prevent further transmission of the infection and because early treatment can slow down the later progression of the disease.
Key words: acute HIV infection, early diagnosis, antiretroviral therapy.

Čas. Lék. čes., 2004, 143, pp. 584–587.

Many lay people and physicians believe that severe manifestations of the HIV infection do not occur until late stages of the disease, when significant immunodeficiency is present. However, striking clinical features do occur - although only transiently - at the very onset of the disease, shortly after exposure. These symptoms often induce the infected individual to present to a physician, or even to undergo hospitalization; however, the diagnosis is mostly incorrect and the disease dismissed as a common viral infection. Consequently the diagnosis of HIV infection is often delayed for many years, with resulting adverse consequences both in terms of the health status of the patient himself/herself, and from the epidemiological point of view (1).

About 85% of newly infected individuals develop so-called acute HIV infection (AHI) within 2-6 weeks of exposure, shortly before seroconversion (AHI). This is a feverish condition usually lasting 2-3 weeks, with spontaneous remission without therapy, with HIV infection then progressively turning into an asymptomatic stage (2).

AHI is consistent with primary viremia, whereby invasion of HIV into the body is followed by massive viral replication, not yet mitigated by any defense mechanisms. It is not until after a period of several weeks, usually shortly before seroconversion (production of anti-HIV antibody), that the massive viral replication starts to be opposed by an intense response by cytotoxic CD8+ T lymphocytes (3). Viral replication slows down, blood viral count decreases, in many cases, to undetectable levels, clinical features of acute disease resolve, and the asymptomatic period (clinical category A) starts – whereby the patient experiences no HIV-infection-related health problems, yet the virus is present in the body and can be transmitted to other individuals via blood or genital tract secretions.

CLINICAL COURSE

Incubation time

The interval from infection to the first clinical symptoms is usually 2 to 6 weeks, with seroconversion occurring 1 to 3 weeks later; however, the longest period to development of antibody is generally believed to be 12 weeks or, in exceptional cases, 6 to 12 months (4).

Clinical features

Overt AHI usually occurs from full health and mostly takes the form of an acute flu-like disease (5). The typical features include high fever, nighttime sweating, generalized lymphadenopathy, hepatosplenomegaly, morbiliform exanthema (6), mucocutaneous lesions of the mouth and genitals (7), impaired nervous system function, myalgia, diarrhea and weight loss, with other symptoms such as rhabdomyolysis occurring exceptionally (8) – a list of symptoms is shown in Table 1.

Tab. 1. List of clinical features of AHI

Symptom	Occurrence rate in %
fever	96
lymphadenopathy	74
pharyngitis	70
exanthema	70
myalgia, arthralgia	54
mucocutaneous lesions (oral cavity, genitals)	35
diarrhea	32
headache	32
nausea, vomiting	27
nighttime sweating	22
hepatosplenomegaly	14
weight loss	13
soor (oral cavity, vulva)	12
neurological symptoms	10

Adapted from the US Dept. of Health and Human Services, Guidelines for the Use of Antiretrovirals in Adults and Adolescents, 2001.

While neurological symptoms occur in only about 10% of AHI cases, they are the most common cause of hospitalization of patients with this disease and may include headache, aseptic meningoencephalitis, isolated paresis of the n. facialis, polyradiculitis and even comatose states (9). Neuropsychological symptoms, from mood swings to depression, memory lapses, increased fatigueability and hypersomnia, are also frequent. These problems may persist for weeks.

Rarely, patients with AHI may develop, as a result of a temporary major fall in CD4+ T lymphocytes, opportune infection typical of AIDS – mostly esophageal candidosis, but also cytomegalovirus (CMV), esophagitis, toxoplasmosis or pneumocystosis (6, 10–12).

AHI usually lasts 1 to 3 weeks; the course is usually benign with spontaneous remission of clinical and laboratory features. However, a protracted course - which may last, in extreme cases, 10 to 12 weeks - will usually signal more rapid progression of HIV infection into the later stages of the disease (6, 13).

About 15% of AHI patients are usually hospitalized (6).

Laboratory findings

Results of most laboratory studies are consistent with an acute viral infection-erythrocyte sedimentation rate, and C-reactive protein levels are usually not increased. Blood count at 1 and 2 weeks will usually show leukopenia with lymphopenia due to a decrease in helper CD4+ T lymphocytes, often below 300/μL; thrombocytopenia also occurs frequently. At week 3 to 4, lymphopenia is typically replaced by lymphocytosis as a result of a reactive increase in both HIV-specific CD4+ T lymphocytes and cytotoxic CD8+ T lymphocytes. A mild elevation in liver transaminases is also often seen (6).

In everyday practice, HIV infection is identified by antibody screening using ELISA; however, the anti-HIV antibody is usually not detectable during the course of AHI and especially so in its first half. As a rule, seroconversion does not occur until 2 to 3 weeks after the onset of disease, mostly after the clinical features have been resolved (14).

Shortly after the acquisition of HIV infection there is a rapid rise in primary temporary viremia, and the presence of HIV in the body can usually be confirmed by the second half of the incubation period by detecting viral nucleic acid using polymerase chain reaction (PCR) or by evidence of viral antigen p24 in plasma.

A cerebrospinal fluid finding in the presence of meningitis suggests serous inflammation with monocyte pleiocytosis, a mild elevation of protein levels and normal glycorrhachia (15).

Diagnosis and differential diagnosis

At present, AHI is diagnosed very rarely, with most cases identified in retrospect (16). Acute HIV infection should always be considered when examining any individual with known (or suspected) at-risk behavior who has developed a feverish disease (17). Unfortunately, patients are reluctant to provide information about their behavior associated with a risk, so their medical history data cannot be regarded as entirely reliable.

Special attention is to be given to feverish disease with generalized lymphadenopathy, exanthema, neurological symptoms, diarrhea and lymphopenia (18). The most differential diagnostic challenge may be posed by infectious mononucleosis (19), particularly infectious mononucleosis caused by the Epstein-Barr virus (EBV) (Table 2); further, a distinction is to be made between the acute nodal form of toxoplasmosis, severe post-drug reaction, rubeolla, prodromal period of viral hepatitis, secondary states of syphilis, and a host of other diseases occurring less frequently in these geographic locations, primarily of viral etiology such as dengue (20). During AHI there may also be temporarily sensitive Paul-Bunnell's or Ericson's reaction with heterophillic antibody (19).

As stated above, the commonly employed screening serological tests are not yet positive during AHI, so the presence of HIV infection in this stage can only be documented using special methods such as providing evidence of HIV ribonucleic acid (RNA) using PCR or antigen p24 detection (21). To date, these investigations in the Czech Republic have only been performed in a single center, the National AIDS Reference Laboratory at the State Health Institute (Státní zdravotní ústav, SZÚ) in Prague. However, at present so-called dual methods detecting both anti-HIV antibody and antigen p24 during a single test are being introduced into routine diagnosis, making it possible to detect the infection a couple of days earlier (22, 23). In a situation whereby it is critical to determine whether an individual is anti-HIV positive, one of the available rapid bedside methods can be used, making it possible to determine the presence of the antibody with a relatively high degree of accuracy, in blood, plasma or serum within a period of several minutes (24). A limitation of these methods is that they cannot be used until seroconversion has occurred, and their result must always be confirmed by a proper investigation in an accredited laboratory (25). Blood collection for an HIV test should always be performed with the patient's informed consent.

In case of doubt, it is appropriate to consult a specialist in the respective regional AIDS Center or at the National Reference AIDS Laboratory of the State Health Institute.

Tab. 2. Comparison of some clinical features of AHI and infectious mononucleosis of EBV etiology

Acute HIV infection	Infectious mononucleosis (EBV)
sudden onset	progressive onset of symptoms
no or mild tonsillar hypertrophy	marked tonsillar hypertrophy
enanthema involving the palate	enanthema involving both the palate and velum
exudative pharyngitis rare	exudative pharyngitis common
mucocutaneous ulceration common	no mucocutaneous ulceration
skin rash common	skin rash rare (except for states after aminopenicillin therapy)
neurological symptoms frequent	neurological symptoms rare
diarrhea frequent	diarrhea usually not present

Therapy

Treatment of most manifestations of AHI is symptomatic and does not essentially differ from the treatment of similar conditions in the presence of other acute viral infection.

The mainstay of treatment is physical rest and administration of analgesics-antipyretics and, alternatively, antihistamines. Caution is to be exercised when treating and examining the patient: body fluids, primarily reproductive tract secretions, blood and cerebrospinal fluid are highly infectious, more infectious than during the ensuing asymptomatic period (26)!

Views regarding the initiation of antiretroviral therapy on establishing the diagnosis of AHI are not unanimous (27–29); however, it seems that this therapy may – at least in some patients – appreciably slow down later progression of the disease (30). AHI is a key component of the natural course of the disease, whereby the therapy-induced radical suppression of viral replication and a decrease in the viral burden in the plasma will slow down HIV dissemination within the body and will maintain the reactivity of HIV-specific CD4+ T lymphocytes (31). Adequate functional capacity of CD4+ T lymphocytes is a *conditio sine qua non* of long-term natural HIV suppression, hence slowing down the progression of HIV infection (32). As a rule, antiretroviral agents should be administered to patients with a protracted AHI course (13). If a decision is made to treat the patient, therapy should be initiated as soon as possible once the diagnosis has been confirmed, and within six months after the acute infection at the latest. Combination AHI therapy is performed following the same principles as those applicable to HAART (highly active antiretroviral therapy)

of chronic HIV infection, i.e., usually involving a combination of one HIV protease inhibitor or a non-nucleoside HIV reverse transcriptase inhibitor (most often, ritonavir or efavirenz-boosted lopinavir) with two nucleoside HIV reverse transcriptase inhibitors (usually zidovudine + lamivudine). Initial AHI treatment should last 6 months to one year, after which time an attempt can be made during the asymptomatic stage – in the presence of very low viral burden (HIV RNA copy count <50/mL plasma) and a sufficient CD4+ T lymphocyte count (CD4+ T lymphocytes >500/μL) – to discontinue antiretroviral therapy. Many patients can fare well without this therapy for years while on permanent follow-up of their clinical status and laboratory levels; however, in other patients, withdrawal of initial therapy is followed by rapid deterioration of both their laboratory findings and clinical status, requiring resumption of antiretroviral therapy (33). Antiretroviral therapy is also of epidemiological importance as it reduces the infection capacity of treated individuals (2).

Decisions about the indication for and administration of antiretroviral therapy, as well as comprehensive care of HIV-positive patients, are essentially to be made by a specialist; this therapy can only be provided by a specialist AIDS Center at the Regional Department of Infectious Disease (Bulovka University Hospital in Prague, and University Hospital at České Budějovice, Plzeň, Ústí nad Labem, Hradec Králové, Ostrava, Brno).

CASE REPORTS

Case Report 1

A 16-year-old girl, after her first unprotected intercourse with an HIV-positive male, developed fever up to 39.5 °C and acute pharyngitis with marked cervical lymphadenitis. Her general practitioner provided the patient with cefuroxime, but the patient's condition did not improve and the patient was hospitalized a week later.

Laboratory findings: CRP 6.8 mg/L, leukocytes $1.9x10^9$/L, thrombocytes $74x10^9$/L, ALT 0.98 μkat/L, AST 1.42 μkat/L. Azithromycin was given, reducing the patient's temperature after 5 days, while the marked cervical lymphadenopathy persisted. However, fever recurred after a week and the patient complained of headache and marked meningeal irritation. Cerebrospinal fluid examination revealed lymphocyte pleiocytosis 666/3 and hyperproteinorrhachia 900 mg/L. In the differential diagnosis, consideration was given to acute HIV, with blood tests providing the following results: ELISA anti-HIV antibody positive, PCR HIV RNA 215409 copies/mL, antigen p24 positive. Immunological investigations revealed a decrease in CD4+ T lymphocyte count to 574/μL, the immunoregulatory index was 0.46. Symptomatic therapy of aseptic meningitis was instituted, with the patient's clinical status and laboratory findings normalizing after a week (6 weeks after the onset of disease). The patient was discharged from hospital with the diagnosis of AHI and meningoencephalitis; antiretroviral therapy with a combination of nelfinavir with zidovudine and lamivudine was started. In retrospect, it was found the source of infection was long being treated for HIV infection.

The patient had AHI with a protracted course complicated by neurological manifestations. The diagnosis was correctly considered and, as blood samples were not obtained until after several weeks, anti-HIV antibody was detectable in addition to PCR and antigen p24 positivity. Given the protracted course of the disease, antiretroviral therapy was instituted in the patient as appropriate.

Case Report 2

A 20-year-old woman had repeated unprotected sexual intercourse with an HIV-positive male; within 4 weeks, she developed fever of up to 39.5 °C, headache, myalgia, arthralgia, and maculopapulous exanthema involving her entire body. Her general practitioner provided her with roxithromycin, but the fever persisted, and the patient started to vomit and experience photophobia and meningeal irritation. The patient was sent to a hospital. On admission, she was found to have, in addition to skin rash and meningeal symptoms, pharyngitis, generalized lymphadenopathy (nodes as big as a prune in size) and mild hepatomegaly. Cerebrospinal fluid examination revealed lymphocyte pleiocytosis 192/3 and hyperproteinorrhachia 1130 mg/L. Erythrocyte sedimentation rate was 30/50, leukocyte count $2.9x10^9$/L, fibrinogen levels 3.4 g/L, AST 0.53 μkat/L, ALT 0.62 μkat/L. Serologic examination for Lyme borreliosis, toxoplasma, tick meningoencephalitis, brucelosis, listeriosis, syphilis and herpes viruses was negative. After 3 days on symptomatic therapy, her temperature decreased, her clinical status and laboratory finding normalized and, after 3 weeks, the patient was discharged from hospital with the diagnosis of acute meningoencephalitis. On follow-up one month after discharge, the patient had no complaints at all and her sickness leave was terminated.

Two years later, a venereological examination of the patient showed HIV positivity. During history taking, the patient noted she had been hospitalized at a department of infectious disease with the diagnosis of acute meningoencephalitis. Once her medical history had been extended to include multiple unprotected sexual intercourse with a man being treated for HIV infection over a number of years, it became clear that patient had AHI associated with meningoencephalitis. Given the circumstances and the interval between contact with the man and outbreak of the disease with typical features, it can be reasonably assumed that AHI was involved, although the diagnosis could not be verified retrospectively by serology.

At the time of acute disease, the correct diagnosis was not considered, the patient was discharged from hospital, and it is quite possible that she had infected other individuals over the period of 2 years, during which time she had a number of sexual partners.

CONCLUSION

AHI is a feverish disease with a variety of non-characteristic symptoms commonly shared by a number of common viral infections. However, the disease has some features which may be helpful in establishing a correct diagnosis.

While early identification of HIV infection is essential for the future development of the health status of the patient; another major consideration is the epidemiological aspect, since early detection of HIV infection may protect other individuals, primarily sexual partners of the patient, against the risk of contracting the disease. Each patient with documented HIV infection should be referred to the catchment AIDS Center to receive professional care.

The prevalence of HIV infection in the Czech Republic is 60 new cases reported each year over the last decade. Assuming up to 10 undetected cases for each newly-diagnosed case of HIV infection, the number of newly infected individuals in the Czech Republic can be estimated at 600 individuals a year. Of this number, an approximate 500 individuals develop AHI – and in the overwhelming majority of cases this is not recognised.

Abbreviations

AHI	–	acute HIV infection
AIDS	–	acquired immunodeficiency syndrome
CMV	–	cytomegalovirus
EBV	–	Epstein-Barr virus
HAART	–	highly active antiretroviral therapy
HIV	–	human immunodeficiency virus
PCR	–	polymerase chain reaction
RNA	–	ribonucleic acid
SZÚ	–	Státní zdravotní ústav (State Health Institute)

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This study was supported by IGA grant No. 5673/3 and University Medical School Research Project No. 111300003.

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COMMENTARY

Comment on Paper by L. Machala and R. Černý on Acute HIV Infection

The paper draws attention to the most important issue of early detection and treatment of acute HIV infection. Although the clinical course of HIV infection was recognized more than a decade ago, and described in detail as closely related to the pathogenesis of HIV infection, awareness among health care workers of the clinical features of acute HIV infection is still very low, and this diagnosis – particularly as part of the differential diagnosis of feverish states with lymphadenitis – is seldom considered.

As highlighted by the authors, the clinical features of AHI very often resemble a variety of other infections or unspecific syndromes. The dominant clinical feature may occasionally be inexplicable extreme fatigue or even exhaustion of the patient. The symptom is present in up to 90% of all episodes of acute HIV infection. Among laboratory findings consistent with viral infection, AHI could be signaled by significant thrombocytopenia, present in up to 45% of patients. Given the polyvalence of clinical and laboratory symptoms of AHI, it is most likely that the patient may present not only to his/her general practitioner but also to specialists, or may be hospitalized in a department of internal medicine, neurology, dermatovenereology, and so on. There is no doubt that the diagnosis of AHI is occasionally very difficult to establish, especially when there is lack of awareness of the epidemiological context.

As the symptoms of AHI are non-specific, it is difficult to determine the incidence of symptomatic disease in newly infected individuals. It is estimated that symptoms consistent with AHI can be seen in 40–90% of newly infected individuals. However, studies have not included control groups, so the reported symptoms could naturally have been due to other concomitant diseases. A cohort trial conducted at the University of Washington followed individuals at high risk for HIV infection on a regular 6-monthly basis. Of those found later to have been infected, 87% had symptoms of AHI and 95% sought medical care. Acute HIV infection was diagnosed on the basis of symptoms in only 25% of patients seeking medical care.

Still, it should be noted that early recognition of acute HIV infection is not only of prognostic relevance for the HIV-infected patient but also has epidemiological ramifications. Early detection of infection will help prevent HIV spread in the population at large, and the potential for nosocomial infection is not to be disregarded, either. Needless to say, every health care center has to abide by hygienic-epidemiological standards (as defined by a methodological manual) so as to avoid HIV infection of their staff or other patients. Regrettably, these standards are not adhered to by all centers. One can still see staff collecting blood without gloves on, aspiring secretions without glasses on, and often not displaying proper care when handling biological material obtained from patients. It is precisely the acute phase of HIV infection which poses a high risk for viral transmission either via blood or other biological material, which is - as pointed out in the paper - highly infectious in this stage of disease. In the event of injury (a prick from a needle, contamination of skin or mucosa by biological material, etc.), and should the clinical course of the disease or epidemiological considerations (a patient with a risk-associated behavior) suggest HIV positivity, the patient should be asked to have acute blood testing at the National AIDS Reference Laboratory at the Prague-based State Health Institute. Under current law, testing for HIV antibody cannot be performed unless the patient has given his/her consent. Rapid blood tests at the onset of infection need not be positive, but they can also be used for guidance. The injured person must wash the wound properly, disinfect it and, in the case of exposure to blood or biological material from an HIV-infected patient, the former must report as soon as possible (preferably within 24 hours) to the appropriate AIDS Center to have so-called zero blood withdrawn (for forensic purposes). Then, depending on the nature of injury, the patient will be offered post-exposure antiretroviral prophylaxis for 4-6 weeks, and a report on injury will be made. The injured person will continue to be taken care of by the AIDS Center where specific blood collections will be made. It is not until a year after the injury of a health-care worker that the report is archived.

Individuals with acute HIV infection carry a high risk. They are a major source of infection for those with whom they are in contact. Those benefiting mainly from early recognition of a newly infected individual will be the patients themselves. Although there has not been unanimity regarding early therapy initiation, preliminary data emerging from controlled trials indicate that initiation of antiretroviral therapy in the period of AHI may help prevent serious immunological damage, significantly reduce viral replication, and delay clinical progression of HIV infection. Therapeutic intervention in the period of AHI currently seems to be a major factor that has become an integral part of the fight against the HIV epidemic.

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