Invasion with *Toxoplasma gondii* can promote pneumocystis pneumonia in individuals with HIV/AIDS

Ihor H. Hryzhak

Ivano-Frankivsk National Medical University, Ivano-Frankivsk, Ukraine and Ivano-Frankivsk Regional Center for AIDS Prevention and Fight, Ivano-Frankivsk, Ukraine

Abstract: Pulmonary pathology is common in HIV-infected individuals, but the possible role of the parasitic protist *Toxoplasma gondii* (Nicolle et Manceaux, 1908) is not completely known. The present account reports result of a retrospective cohort study. Medical cards of 907 HIV-positive people, which included 120 deceased patients, were analysed. During a three-year follow-up, the pulmonary pathology was diagnosed in 306 patients (33.7 ± 1.6%); pneumocystis pneumonia in 124 (13.7 ± 1.1%), primary pulmonary tuberculosis in 113 (12.5 ± 1.1%), bacterial pneumonia in 58 (6.4 ± 0.8%) toxoplasmosis pneumonia in two (0.2 ± 0.2%), and others. All patients were divided into two cohorts: 531 individuals seropositive for *T. gondii* and 376 seronegative ones. It has been found out that general lung pathology is more common in patients with seropositivity to *T. gondii* than in seronegative ones (43.3 ± 2.2% vs. 20.1 ± 2.0%, p < 0.001). The diagnosis of pneumocystis pneumonia was made ten times more often in the cohort of seropositive patients than in the cohort of seronegative ones (21.9 ± 1.8% vs. 2.1 ± 0.7%, respectively, p < 0.001) and in deceased patients of these cohorts it was 5.5 times more (45.1 ± 5.9% vs. 8.2 ± 3.9, respectively, p < 0.001). In patients with fatal outcome and seropositivity to *T. gondii*, the incidences of pneumocystis pneumonia increased by 23.2% (p < 0.001) and bacterial pneumonia by 12.4% (p < 0.05), whereas in seronegative individuals only pulmonary tuberculosis increased by 13.1% (p < 0.05) compared with corresponding whole cohorts. Pearson’s contingency coefficient showed the mean strength association between infection with *T. gondii* and incidence of pneumocystis pneumonia both in whole cohort (C = 0.272) and in patients with fatal outcomes (C = 0.368). In conclusion, significantly increasing rate of pneumocystis pneumonia in patients with HIV/AIDS and *T. gondii* infection can be caused by certain synergism between *T. gondii* and *Pneumocystis jiroveci* and in some cases overdiagnosis pneumocystis pneumonia due to undiagnosed toxoplasmosis pneumonia.

Keywords: toxoplasmosis, pulmonary diseases, immunodeficiency

The parasitic protist *Toxoplasma gondii* (Nicolle et Manceaux, 1908) (Apicomplexa) is an important opportunistic pathogen. In severely ill immunocompromised patients, the acute hypoxemic respiratory failure (ARF) due to pulmonary infection can be caused by invasive fungal infections (species of *Aspergillus* and *Mucorales*, and *Pneumocystis jiroveci*) and *T. gondii* infection (Azoulay et al. 2020). The prevalence of *T. gondii* is considerable among population and increased in immunocompromised patients. According to global meta-analysis, the pooled prevalence of *T. gondii* infection in HIV/AIDS patients was 42.1%, compared with 32.0% in the control (Wang et al. 2017). In University Hospital of South Manchester 100% of the patients with lung cancer were infected with *T. gondii* and only 10% in the controls (Bajnok et al. 2019). Cases of lung lesion caused by *T. gondii* were described in patients with chronic inflammatory arthritis and methotrexate and corticosteroid therapy (Abdulkareem et al. 2017), with leukemia (Scerra et al. 2013), lung cancer (Lu et al. 2015), and also in positive anti-toxoplasma serology bone marrow recipients, who got immunosuppressive therapy for “graft-versus-host” disease after allogenic transplantation, especially in highly endemic for toxoplasma areas (de Medeiros et al. 2001).

Pulmonary toxoplasmosis is a serious pulmonary disease, which has symptoms such as cough, fever, and shortness of breath, rales, myalgias, arthralgias and lymphadenopathy. It can imitate many common lung conditions in the immunocompromised patient, including atypical pneumonia, pneumocystis pneumonia and interstitial lung diseases. Chest X-ray usually reveals bilateral interstitial infiltrates. Lymphadenopathy and hepatosplenomegaly have been reported more frequently in immunocompetent patients than in immunocompromised ones. Serological evidence of active toxoplasmosis arises in immunocompetent but not in immunosuppressed patients. Mortality rate among patients with toxoplasma pneumonia was 55% (Pomeroy and Filice 1992). In a case report about pulmonary toxoplasmosis...
in a patient with methotrexate and corticosteroid therapy
a chest computed tomography showed extensive bilateral
ground-glass opacities and subcentimetre bilateral hilar
lymphadenopathy (Abdulkareem et al. 2017).

In HIV-infected patients, the lung is a major site of *T. gondii*
infected after the central nervous system. HIV/
AIDS patients with low CD4 counts (< 200 cells/mm³)
have high epidemiological risk as well as immunological
risk of toxoplasmosis (Yenilmez and Cetinkaya 2018).
Pulmonary toxoplasmosis should be considered in HIV-infected
patients with late stage of HIV, CD4 count less than
100 cells/µl and a poor adherence to Highly Active Anti-
tiretroviral Therapy (Velasquez et al. 2016). Disseminated
toxoplasmosis with severe necrotising pneumonia with nu-
umerous tachyzoites in lung tissue occur in patients with ad-
vanced AIDS and toxoplasmosis encephalitis (Pastorello et
al. 2018). Also, there is one case report of an AIDS patient
with both pulmonary and cerebral toxoplasmosis associ-
ated with pneumocystis pneumonia (Rey et al. 2017).

The pulmonary toxoplasmosis rarely occurs in immu-
nocompetent patients. Clinical and image-based findings
of toxoplasmosis pneumonia overlap with other kinds of
atypical pneumonia. Toxoplasmosis should be considered
and immunoglobulin M-specific antibodies should be de-
tected (Leal et al. 2007) in an individual with the specific
ic imaging manifestations such as patchy and flocculent
high-density shadows or the ground-glass opacity of the
lung. Sometimes immunocompetent individuals acquire
toxoplasma pneumonia through inhalation high toxicity
species of *T. gondii* (Shen et al. 2015).

MATERIALS AND METHODS

Design of the study

The three-year retrospective cohort research was conduct-
ed at the Department of Infectious Diseases and Epidemi-
ology of Ivano-Frankivsk National Medical University and at
Ivano-Frankivsk Regional Centre for AIDS Prevention and
Fight (Ukraine). It is a fragment of complex research project
of Department of Infectious Diseases and Epidemiology en-
titled “The course of infectious diseases on the background
of concomitant pathology, combined chronic infections and
invasions, correction of treatment”, Ukrainian state regis-
tration number: 0112U005012 (2019–2023). The medical data
of 907 HIV-infected patients (men 58.3%) with mean age of
35.2 ± 0.3 years (18–65 years) within 2013–2015 were stud-
ied. According to the test results for IgM, IgG to *Toxoplasma
gondii*, the patients were divided into two groups: 531 se-
ropositive individuals and 376 seronegative ones. Inclusion
criteria of the patients to the seropositive cohort were the
presence of anti-*T. gondii*-IgG or IgM, or IgG, or both IgG
and IgM in the blood at the first detection. Some previous
seronegative individuals were transferred to the seropositive
group after the appearance of seroconversion (the appear-
ance of specific IgM or IgG, or both IgG and IgM simulta-
neously). People who did not have antibodies *T. gondii* at
the first detection and without seroconversion during the next 3
years were included in the seronegative cohort.

The diagnosis of pneumocystis pneumonia was based
on signs of interstitial pneumonia (X-ray symptom of
ground-glass opacity of lung), increased levels of lactate
dehydrogenase (LDH) and microscopic identification of
*Pneumocystis jiroveci* in sputum. The PCR test did not use
to detect DNA of *P. jiroveci* or *T. gondii* in a sputum sample.

Statistical analysis

In both cohorts, we set the percentage of different
pulmonary pathologies and their average error (P ± m).
Pearson’s Chi-square test (χ²) was used to determine the
significance of differences rates of lung disease in this
cohorts. Pearson’s contingency coefficient (C) was used
to estimate the relation between anti-*T. gondii* seropositive
status and each pulmonary disease. The significance
of difference between frequency of each pulmonary pathology
in group of died patients and in the entire corresponding
cohort was assessed by the Student’s t-test. Our research
was approved by Ethics Commission of Ivano-Frankivsk
National Medical University (Expert decision No. 96/17

RESULTS

A total of 531 individuals (61.4% men and 38.6% wom-
en) were included in the cohort of patients with seropositivity
to *Toxoplasma gondii* and 376 individuals (53.85% men
and 46.15% women) were included to the seronegative
cohort. In seropositive cohort 91 (17.1 ± 1.6%) patients had
stage I of HIV infection, 101 (19.0 ± 1.7%) had stage II,
80 (15.1 ± 1.6%) had stage III, and 259 (48.8 ± 2.2%) had
stage IV. In seronegative cohort 48 (12.8 ± 1.7%, p < 0.05)
patients had stage I of HIV-infection, 78 (20.7 ± 2.1%, p
> 0.05) had stage II, 76 (20.2 ± 2.1%, p < 0.05) had stage
III, and 174 (46.3 ± 2.6%, p < 0.05) had stage IV. There-
fore, the distribution of patients regarding to the stage of
HIV-infection was practically the same in both cohorts.

Antiretroviral therapy (ART) was received by 352
patients in the seropositive cohort (66.3 ± 2.1 %), which
is less than in the cohort of seronegative individuals, i.e.,
287 (76.3 ± 2.2%, p = 0.002). In the seropositive cohort,
134 patients (25.2 ± 1.9%) and 88 ones in the seronegative
cohort (23.4 ± 2.3 %, p > 0.05) received sulfamethoxazole/
trimethoprim (SMX/TMP) 400/80 mg twice daily for the
prevention of pneumocystis pneumonia. In the seropositive
cohort 50 persons (9.4 ± 1.3%) and in the seronegative
cohort 34 patients (9.0 ± 1.5 %, p > 0.05) received azith-
romycin (1,200 mg once a week) for the prevention of
the infection with the *Mycobacterium avium* complex.
Therefore, SMX/TMP and azithromycin, which can pre-
vent active toxoplasmosis, were administrated to the same
proportion of patients in these two cohorts.

During a three-year follow-up of 907 patients
opportunist pulmonary pathology was diagnosed in 306
patients (33.7 ± 1.6%): pneumocystis pneumonia in 124
(13.7 ± 1.1%), primary pulmonary tuberculosis in 113
(12.5 ± 1.1%), bacterial pneumonia in 58 (6.4 ± 0.8%),
and others were in 10 patients (1.1 ± 0.4%). Among other
diseases there were: toxoplasma pneumonia, pulmonary
aspergiloma, Kaposi’s sarcoma – two cases of each (0.2 ±
Table 1. The distribution of pulmonary pathologies in HIV-infected individuals with and without antibody to *Toxoplasma gondii* (Nicolle et Manecaux, 1908).

<table>
<thead>
<tr>
<th></th>
<th><em>T. gondii</em>-positive (n = 531)</th>
<th><em>T. gondii</em>-negative (n = 376)</th>
<th>Chi-square test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>P = m ± m (%)</td>
<td>Number</td>
</tr>
<tr>
<td><strong>Pneumocystic pneumonia</strong></td>
<td>116*</td>
<td>21.9 ± 1.8</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.1 ± 0.7</td>
<td>72.515</td>
</tr>
<tr>
<td><strong>Bacterial pneumonia</strong></td>
<td>39</td>
<td>7.3 ± 1.1</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.1 ± 1.1</td>
<td>1.931</td>
</tr>
<tr>
<td><strong>Pulmonary tuberculosis first detected</strong></td>
<td>70</td>
<td>13.2 ± 1.5</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11.4 ± 1.6</td>
<td>0.616</td>
</tr>
<tr>
<td><strong>Pulmonary aspergiloma</strong></td>
<td>1</td>
<td>1.2 ± 0.2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.3 ± 0.3</td>
<td>0.060</td>
</tr>
<tr>
<td><strong>Cytomegaloviral pneumonia</strong></td>
<td>1</td>
<td>1.2 ± 0.2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.3 ± 0.3</td>
<td>0.079</td>
</tr>
<tr>
<td><strong>Bacterial pleurisy</strong></td>
<td>0</td>
<td>0.7 ± 0.3</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.414</td>
<td>0.235</td>
</tr>
<tr>
<td><strong>Toxoplasmic pneumonia</strong></td>
<td>2</td>
<td>0.4 ± 0.3</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.3 ± 0.3</td>
<td>1.412</td>
</tr>
<tr>
<td><strong>Bronchopulmonary candidiasis</strong></td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.3 ± 0.3</td>
<td>1.414</td>
</tr>
<tr>
<td><strong>Kaposi’s sarcoma with lung disseminations</strong></td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.5 ± 0.4</td>
<td>2.831</td>
</tr>
<tr>
<td><strong>Non-Hodgkin’s lymphoma of lung</strong></td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.3 ± 0.3</td>
<td>1.414</td>
</tr>
<tr>
<td><strong>Total number of patients with pulmonary pathologies</strong></td>
<td>230*</td>
<td>43.3 ± 2.2</td>
<td>76</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20.1 ± 2.0</td>
<td>52.551</td>
</tr>
</tbody>
</table>

* – the difference between the indexes in cohorts of patients with and without antibody to *T. gondii* is statistically significant; χ² – Pearson’s chi-squared test; C – Pearson’s contingency coefficient; P – percentage; m – standard error of percentage; p – statistical significance.

Table 2. The distribution of pulmonary pathologies in deceased HIV-infected individuals with and without antibody to *Toxoplasma gondii* (Nicolle et Manecaux, 1908).

<table>
<thead>
<tr>
<th></th>
<th><em>T. gondii</em>-positive (n = 71)</th>
<th><em>T. gondii</em>-negative (n = 49)</th>
<th>Chi-square test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>P = m ± m %</td>
<td>Number</td>
</tr>
<tr>
<td><strong>Pneumocystic pneumonia</strong></td>
<td>32*</td>
<td>45.1 ± 5.9</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8.2 ± 3.9</td>
<td>18.805</td>
</tr>
<tr>
<td><strong>Bacterial pneumonia</strong></td>
<td>14</td>
<td>19.7 ± 4.7</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>14.3 ± 5.0</td>
<td>0.593</td>
</tr>
<tr>
<td><strong>Pulmonary tuberculosis first detected</strong></td>
<td>15</td>
<td>21.1 ± 4.8</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td></td>
<td>24.5 ± 6.1</td>
<td>0.188</td>
</tr>
<tr>
<td><strong>Kaposi’s sarcoma with lung disseminations</strong></td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4.1 ± 2.8</td>
<td>2.947</td>
</tr>
<tr>
<td><strong>Non-Hodgkin’s lymphoma of lung</strong></td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.0 ± 2.0</td>
<td>1.461</td>
</tr>
<tr>
<td><strong>Total number of patients with pulmonary pathology</strong></td>
<td>61*</td>
<td>85.4 ± 4.1</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td></td>
<td>53.9 ± 7.1</td>
<td>15.696</td>
</tr>
</tbody>
</table>

* – the difference between the indexes in cohorts of patients with and without antibody to *T. gondii* is statistically significant; χ² – Pearson’s chi-squared test; C – Pearson’s contingency coefficient; P – percentage; m – standard error of percentage; p – statistical significance.

In patients seropositive to *T. gondii* the general lung pathology was twice as frequent as in seronegative ones (43.3 ± 2.2% vs. 20.1 ± 2.0%, p < 0.001; see Table 1). First of all, the difference concerns pneumocystis pneumonia frequency which is pronounced by 15.1% (21.9 ± 1.8% vs. 2.1 ± 0.7%, p < 0.001). In this case, the Pearson’s contingency coefficient C = 0.272 reflected the mean strength association between *T. gondii* infection and pneumocystis pneumonia. It is noteworthy that Kaposi’s sarcoma with lung dissemination was observed only in patients with seronegative status to *T. gondii* – two cases (0.5 ± 0.4%). In seropositive individuals Kaposi’s sarcoma occurred only as cutaneous manifestations – 2 (0.4 ± 0.3%).

Within three years, 120 patients died: 71 patients (13.4 ± 1.5%) of the seropositive cohort and 49 patients (13.0 ± 1.7%) of the seronegative one. The mortality rate did not differ in the two cohorts (p > 0.05). Among persons with fatal outcome, in seropositive individuals the total number of patients with general lung pathology was higher than in seronegatives ones (85.9 ± 4.1% vs. 53.9 ± 7.1%, p < 0.001; see Table 2). In seropositive deceased persons, the pneumocystis pneumonia frequency also was higher 5.5 times than in seronegatives ones (45.1 ± 5.9% vs. 8.2 ± 3.9%, p < 0.001), and it had a mean strength association with infection with *T. gondii* (C = 0.368; see Table 2).

In deceased seropositive to toxoplasmosis individuals, the frequency of general lung pathology was significantly higher by 42.6% compared with this entire cohort (85.9 ± 4.1% vs. 43.3 ± 2.2%, respectively, t = 9.15, p < 0.001), including increased incidences of pneumocystis pneumonia by 23.2% (45.1 ± 5.9% vs. 21.9 ± 1.8%, respectively, t = 3.76, p < 0.001) and bacterial pneumonia by 12.4% (19.7 ± 4.7% vs. 7.3 ± 1.1%, respectively, t = 2.55, p < 0.05; Fig. 1). In the group of deceased persons out of the seronegative cohort, the frequency of general lung pathology also increased by 33.7% compared with this entire cohort (53.9 ± 7.1% vs. 20.2 ± 2.1%, t = 4.56, p < 0.001), mainly due to increasing incidence of pulmonary tuberculosis by 13.1% (24.5 ± 6.1% vs. 11.4 ± 1.6%, t = 2.05, p < 0.05; Fig. 1). Thus, difference was observed between deceased persons compared with their entire cohorts: the incidence of pneumocystis and bacterial pneumonia increased in seropositive individuals but pulmonary tuberculosis increased only in seronegative ones.

Not all of these pulmonary diseases were the main causes of death in patients, but they were observed during the follow-up, and reflected a gradual worsening of patients’ health condition. In deceased seropositive patients, pulmonary pathology as a cause of death was

Folia Parasitologica 2021, 68: 018
presented as the following pathologies: tuberculosis – 12 patients (16.9 ± 4.5%), pneumocystis pneumonia – 1 (1.4 ± 1.4%), bacterial destruction – 1 (1.4 ± 1.4%); in the group of seronegative deceased persons: tuberculosis – in 11 patients (2.5 ± 6.0%), Kaposi’s sarcoma – 2 (4.1 ± 2.8%), non-Hodgkin’s lymphoma – 1 (2.0 ± 2.0%), pneumocystis pneumonia – 1 (2.0 ± 2.0%).

**DISCUSSION**

It has been found out that general pulmonary pathology occurs more often in individuals seropositive to *Toxoplasma gondii* than in seronegatives ones, which may be explained by some influence of *T. gondii* on the development of pulmonary pathology. In particular, it was found that the frequency of pneumocystis pneumonia in the cohort of seropositive individuals is ten times higher than in seronegative one. In many cases, *Pneumocystis jirovecii* was not detected in sputum, so doctors made a diagnosis based on clinical symptoms that are common to both pneumocystis pneumonia and toxoplasmosis pneumonia. Moreover, treatment with SMX/TMP is effective against both pathogens. Therefore, toxoplasmosis pneumonia in some cases could be hidden under the mask of overdiagnosed pneumocystis pneumonia.

In patients with fatal HIV-infection, the incidence of general pulmonary pathology increased significantly in both groups during the follow-up, reflecting deterioration in general health condition and decreased immunity. In deceased seropositive persons, the most common lung pathology was pneumocystis pneumonia. Instead, pulmonary tuberculosis was the most common in the seronegative persons. This may be due to pneumocystis pneumonia as first manifestation in seropositive patients and tuberculosis in seronegative ones. When there is manifestation of pneumocystis pneumonia, all patients receive isoniazid for the tuberculosis prevention, and when pulmonary tuberculosis manifests, patients receive SMX/TMP for the pneumocystis pneumonia prevention. Also, Pearson’s contingency coefficient (C = 0.368) shows a medium strength relationship between toxoplasmosis infection and pneumocystis pneumonia and suggests some association between pneumocystis and toxoplasmosis. These data indicate a possible synergism between *T. gondii* and *P. jirovecii*.

Therefore, under conditions of immunodeficiency, the body protection against pathogenic fungi depends on innate immunity, namely on the function of alveolar macrophages. Normally, IFNy reprogrammed macrophages to a permissive M1-biased phenotype. M1 cells exhibited significantly increased production of iNOS, NO, TNF-α, IL-1β, and IL-6 (Bhagwat et al. 2018). However, ToxoROP16 induced macrophage bias to M2 cells (in vitro), showing decreased production of NO and iNOS and increased expression of Arg-1, IL-10 and TGF-β1 (Denkers 2003). In turn, IL-10 is a potent antagonist of IFN-gamma, which is a critical cytokine involved in mediating *T. gondii*-induced immunosuppression in the infected host (Khan et al. 1995). Also, intracellular *T. gondii* infection causes a blockade in the NFKappaB macrophage signaling pathway and reduced IL-12 and TNF-alpha production. The parasite also prevents STAT1 activity, resulting in decreased levels of IFN-gamma-stimulated MHC surface antigen expression. Extracellular pathways of suppression involve soluble mediators such as IL-10 and lipoxins that have potent IL-12 down-regulatory effects (Meira et al. 2014). For this reason, *T. gondii* can prevent the eradication of infections, thereby allowing the parasite to persist in the host.
tion of pneumocystis and bacterial pathogens from the lungs and help pneumonia development.

Some lung oncology problems such as pulmonary dissemination of Kaposi’s sarcoma and non-Hodgkin’s lymphoma were detected only in seronegative deceased persons, which remained without satisfactory explanation. It can be explained by profound immunodeficiency in some patients with seronegative status who did not take ART for a long time. These findings are important for the physician as they indicate the necessity of the diagnosis and treatment of active T. gondii infection in HIV-positive people to prevent not only toxoplasmosis lung lesions but could play certain role in prevention pneumocystis pneumonia.

The results of the study may be affected by different number of patients in the two groups who used ART. Thus, the number of seropositive individuals to T. gondii who used ART was by 10% lower than seronegative ones. It has not been known how many people had the compliance to ART and how many of them violated the treatment regimen. In the mentioned period of the study the ART was prescribed for patients with advanced stages of AIDS, so satisfactory reconstitution of immunity in many patients was not observed. This could lead to a worsening of immunodeficiency and development of various opportunistic diseases including pneumocystis pneumonia. This pathological process does not depend on the presence of T. gondii.

In conclusion, significantly increasing rate of pneumocystis pneumonia in patients with HIV/AIDS and T. gondii infection can be caused by certain synergism between T. gondii and P. jirovecii and some cases of overdiagnosis pneumocystis pneumonia due to undiagnosed toxoplasmosis pneumonia.

Acknowledgements. We would like to thank the doctors, nurses and other medical staff of the Ivano-Frankivsk Regional Centre for AIDS Prevention and Fight for their professional assistance through-out the study.

REFERENCES