ORIGINAL ARTICLE
Turk J Surg 2023; 39 (1): 76-82

Etiology and severity features of acute pancreatitis in HIV-positive patients with different immune status

Dmitriy Plotkin[1,2], Umedzhon Gafarov[1], Mikhail Reshetnikov[1], Sergey Sterlikov[3], Elena Bogorodskaya[1]

[1] Department of Surgery, Moscow Research and Clinical Center for Tuberculosis Control, Moscow, Russia
[2] Department of General Surgery, Pirogov Russian National Research Medical University, Moscow, Russia
[3] Federal Monitoring Center for Prevention of Tuberculosis Transmission, Russian Federation Russian Research Institute of Health, Moscow, Russia

ABSTRACT

Objective: Acute pancreatitis is common in HIV-infected patients; however, the causes and severity of pancreatitis in HIV-positive patients have a number of significant features that affect both the severity of destruction of the pancreas and the methods of diagnosis and treatment.

Material and Methods: Anamnestic data, results of diagnosis and treatment of two groups of patients with acute pancreatitis were analyzed. The first group included 79 patients with acute pancreatitis combined with HIV infection who were admitted to the clinic for the period from 2017 to 2021. In people living with HIV, drugs and infectious agents caused acute pancreatitis in 11.4% and 24.1% of the cases, respectively. As our study showed, in patients with normal immune status, the drug etiology of pancreatitis prevailed in the structure of the causes of AP, in patients with immunodeficiency, infectious causes of pancreatitis were dominant.

Results: According to the results of data analysis, it is clear that HIV infection is a factor that makes the course of pancreatitis about two times worse regardless of the presence of immunosuppression. The etiological structure of HIV-associated acute pancreatitis directly depends on the patient's immune status and differs in many ways from that of HIV-negative patients or patients receiving ART.

Conclusion: The severity of the disease and the risk of death remain high in acute pancreatitis caused by infectious agents against the background of immunosuppression.

Keywords: Acute pancreatitis, HIV-infection, antiretroviral therapy, immune status, SAPS II

INTRODUCTION

The HIV pandemic has been a global threat to human health for the past 30 years. As of 2021, there are up to 38.4 million people living with HIV worldwide (1,2), and among them 1.562.570 patients live in the Russian Federation (3). People living with HIV suffer from a large number of various diseases which are not so different from the main population in their nosological composition (but not in occurrence rate). Various forms of acute pancreatitis (AP) are no exception, the incidence of which in Russia in 2021 was 37.8 per 100.000 population. At the same time, attention has been drawn to the extremely high mortality rate in certain clinical forms of acute pancreatitis (18-25%) (4).

The number of researchers claim that the main mechanisms of the pancreas damage in HIV-induced disease include the direct impact of the virus on organ tissues, inflammatory and destructive changes on the background of opportunistic infections and/or the toxic effect of ART drugs leading to the death of gland cells (5-9). The risk of acute pancreatitis has until recently been associated with the use of nucleoside reverse transcriptase inhibitors such as zidovudine, didanosine and stavudine (8). Modern ART drugs rarely lead to the development of acute pancreatitis but can cause transitory hyperamylasemia (9). As for opportunistic infections that affect the pancreatic tissue with a low immune status, the most common references in the literature are cytomegalovirus infection (CMV), tuberculosis (TB) and non-tuberculosis mycobacteria (NTMB), toxoplasma and pneumocyst (9-11).

The incidence and prevalence of AP vary depending on the characteristics of the studied population and the method of data collection, but the risk clearly increas-

Cite this article as: Plotkin D, Gafarov U, Reshetnikov M, Sterlikov S, Bogorodskaya E. Etiology and severity features of acute pancreatitis in HIV-positive patients with different immune status. Turk J Surg 2023; 39 (1): 76-82.

Corresponding Author
Mikhail Reshetnikov
E-mail: taxol@bk.ru

Received: 01.11.2022
Accepted: 30.11.2022
Available Online Date: 03.03.2023

DOI: 10.47717/turkjsurg.2023.5939

es with the progression of the disease and the stage of HIV infection. Therefore, given the high frequency of HIV infection in the population and taking into account the increased risk of acute pancreatitis in people living with HIV the problem of HIV/pancreatitis comorbidity is topical for clinical practice (12-14).

MATERIAL and METHODS

This is a retrospective study based on a retrospectively collected database from two centers, Moscow Research and Clinical Center for TB Control & Rozanov Moscow Regional Hospital.

Approval for the study was obtained from the ethics committee of Moscow Research and Clinical Center for TB Control (Protocol No: 325, Date: 13.05.2022). Informed written consent was obtained from all patients included in the study.

In order to achieve this goal, the diagnosis and treatment results of two groups of patients with acute pancreatitis were analyzed. The first group included 79 patients with an established diagnosis of AP in combination with HIV infection who were admitted to the clinic in the period from 2017 to 2021. The second group consisted of 558 patients with an established diagnosis of acute pancreatitis without HIV infection who were on inpatient treatment for the same period of time. In the first group of patients, males dominated (59; 74.7%) and aged from 24 to 63 years, the age of women (20; 25.3%) ranged from 34 to 57 years. The cohort of HIV-negative patients was also predominantly male (376; 67.4%) aged 19 to 69 years. The diagnosis of acute pancreatitis was established on the basis of clinical, laboratory, and instrumental criteria (15).

Additionally, the following laboratory tests were used in the group of patients with HIV infection: detection of the causative agent of tuberculosis and NTMB in sputum and other biological fluids by polymerase chain reaction (PCR) and using cultural and bacterioscopic techniques, quantitative determination of cytomegalovirus DNA (CMV DNA) in the blood, as well as determination of genetic material of toxoplasma and pneumocyst in biological material using PCR diagnostics. In cases of fatal outcomes, histological studies obtained during autopsy were used to confirm the diagnosis.

In the group of HIV-positive patients, the immunogram of each patient was studied which reflected the state of the immune system at that time. If the number of helper T-lymphocytes in 1 mL of blood (CD^{4+}) was more than 200, it was considered an acceptable immune indicator, in which the risk of developing opportunistic infections is minimal, on the contrary, when the number of CD^{4+} cells was less than 200 in mL, it was defined as immunosuppression with a high risk of secondary diseases (16).

The severity of patient's condition with acute pancreatitis was assessed on the SAPS II score (Original Simplified Acute Physiology Score). This is an original simplified scale for assessing physiological disorders which uses 15 readily determined biological and clinical indicators (17), the resulting sum of points allowed predicting mortality in each specific case of acute pancreatitis. Patients' treatment was carried out according to the protocols proposed in the clinical guidelines for acute pancreatitis and, in some cases, was supplemented by draining endoscopic interventions aimed at decompression of the Wirsung's duct and bile ducts as well as punctures of acute fluid accumulations (if necessary).

Descriptive statistics of the research results are presented in the tables. In the course of statistical data processing, extensive indicators were calculated and their 95% confidence intervals (95% CI) by the Wilson method determined the probability of a statistical error of the first kind (p). When analyzing cross-tabulation tables with a dimension of more than 2 x 2 Fisher's exact test was used. To test the hypothesis about the effect of HIV status on the severity of patients' condition on the SAPS II scale, we compared groups of HIV-negative and HIV-positive patients with different immunogram characteristics (CD^{4+}< 200 and CD^{4+}> 200 cells in MCL). Since using point indicators needs operating with nonparametric data, we applied quartile test to compare them and the Mann-Whitney U test to calculate the statistical significance of the differences.

RESULTS

The sex and age composition of the studied groups is presented in Table 1. As can be seen from Table 1, sex differences in the study groups were statistically insignificant (p= 0.2). Age differences were also statistically insignificant (p= 0.4). Thus, the main and control groups were balanced by sex and age.

All patients included in the study had acute pancreatitis, the interstitial form of the disease being established in 62 HIV-positive patients (78.5%; 95% CI 68.2-86.1), and necrotizing pancreatitis,

Table 1. Sex and age composition of the study groups

Group	Sex	Total	Up to 20 years old	Up to 21-30 years old	31-40 years old	41-50 years old	Over 50 years old
HIV+	m	59	0	8	26	16	9
79	w	20	0	0	9	9	2
HIV-	m	376	2	69	165	102	38
558	w	182	1	13	49	57	62

respectively, was detected in 17 cases (21.5%; 95% CI 13.9-31.8). In HIV-negative patients, interstitial pancreatitis was diagnosed in 429 cases (76.9%; 95% CI 73.2-80.2), and necrotizing one-in 129 patients (23.1%; 95% CI 19.8-26.8). Differences in the frequency of necrotizing pancreatitis in the main and control groups were statistically insignificant (p= 0.7). Mortality was seen in six patients in the group with HIV infection (7.6%; 95% CI 3.5-15.6), and in 18 cases (3.2%; 95% CI 2.1-5.0) in the group with HIV-negative patients (p= 0.1). In the group with HIV-negative patients, the main factor of the occurrence of the pathological process in the pancreas could be considered the use of alcohol and fatty foods (291; 52.2%); however, it should be noted that when using radiological diagnostic methods in this group of patients, there was no indication of the presence of concretions in the gallbladder and ducts as well as no mention of the use of medicines and prior infectious diseases. The presence of gallstones and the dilatation of Wirsung's duct (more than 2 mm), common bile duct (more than 8 mm) and intrahepatic bile ducts (more than 2 mm) were considered by us as the second main cause of AP in this group of patients (201; 36.0%).

When collecting anamnesis in three cases, it was found out that one patient developed AP against the background of epidemic parotitis and two more patients with the manifestation of adenovirus infection (without mentioning the alimentary factor and in the absence of concretions and biliary hypertension). Thus, these facts allowed us to believe that the cause of AP in these observations was a viral infection (0.5%) (18). Other six (1.1%) cases of AP we regarded as a lesion of the pancreatic parenchyma due to the use of medications: in four patients, there was a link between the initiation of treatment of hypertension with angiotensin converting enzyme inhibitors (lisinopril), and in two more cases-with the use of atorvastatin (19,20). In 57 (10.2%) patients, it was not possible to establish the etiology of acute pancreatitis.

The severity of acute pancreatitis on the SAPS II scale in the group of HIV-negative patients was determined by the number of points: up to 14 points in 225 patients (40.3%), from 15 to 34 points in 288 cases (51.6%), which means that the overwhelming number of patients (91.9%) had pancreatitis in mild and moderate form in which the risk of death does not exceed 15.3%. Only in 45 cases (8.1%) AP was severe with the sum of points from 38 to 70 according SAPS II and the risk of death was determined in the range from 21.3% to 83.8%. It should be noted that all 18 fatal cases were related to this group of patients.

Analyzing the causes of acute pancreatitis in HIV-positive patients revealed that alimentary factors caused pancreatitis in 25 patients (31.7%), biliary concretions and hypertension in 13 patients (16.5%), but we were unable to establish initiating factors of the disease in 13 cases (16.5%). People living with HIV had acute pancreatitis due to drugs and infectious agents in 11.4% (9) and 24.1% of cases, respectively (19). Establishing causal relationships, we proceeded from the following considerations: the onset of acute pancreatitis was associated with the ART administration in five patients (lopinavir + ritonavir, darunavir, kemeruvir), at this there were no mention of errors in diet and alcohol consumption, biliary concretions and signs of ductal hypertension were not visualized by ultrasound, and the phenomena of AP were stopped with the withdrawal of medicines (7,9). In four more patients, acute pancreatitis was probably associated with the use of the combined drug trimethoprim + sulfamethoxazole. The symptoms of acute interstitial pancreatitis also regressed after the end of the course of treatment (21).

The most interesting group was a cohort of 19 HIV-positive patients in whom we considered a viral or bacterial infection to be the cause of acute pancreatitis. In 13 patients, acute pancreatitis was associated with cytomegalovirus infection (CMV), and in two cases, the process was destructive and resulted in the death of patients. In all patients, PCR diagnostics showed CMV DNA in blood leukocytes exceeded 2-3.5 Lg per 100 thousand cells (22). Indirect signs of cytomegalovirus as an etiological factor of AP were: CT-symptoms of viral pneumonia in nine patients and CMV-retinitis in five cases as well as positive clinical course in response to ganciclovir therapy in 10 cases (Figures 1 and 2). As noted above, we were able to verify the diagnosis in two patients using autopsy examination of pancreatic tissues (Figure 3).

In one HIV-positive patient with generalized tuberculosis, damaged lungs and mesenteric lymph nodes, it was possible to identify the tuberculosis etiology of necrotizing pancreatitis according to postmortem examination: the presence of caseous necrosis in the gland tissue and a positive PCR test for DNA of tuberculous mycobacteria (Figure 4).

In the other five observations, we linked the etiology of AP directly to the human immunodeficiency virus. A prerequisite for

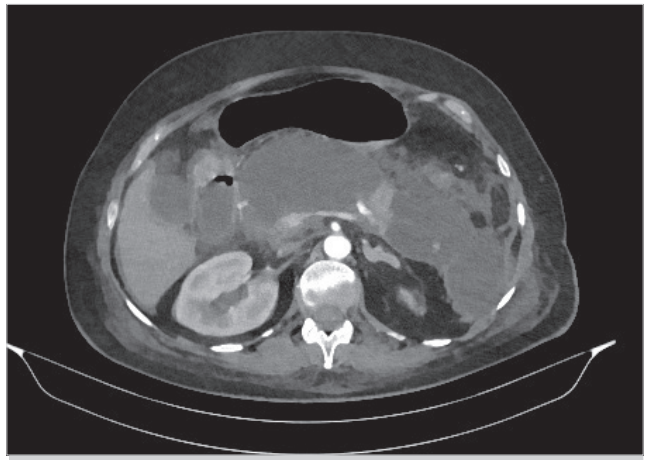

Figure 1. CT scan. Acute necrotizing pancreatitis associated with CMV.

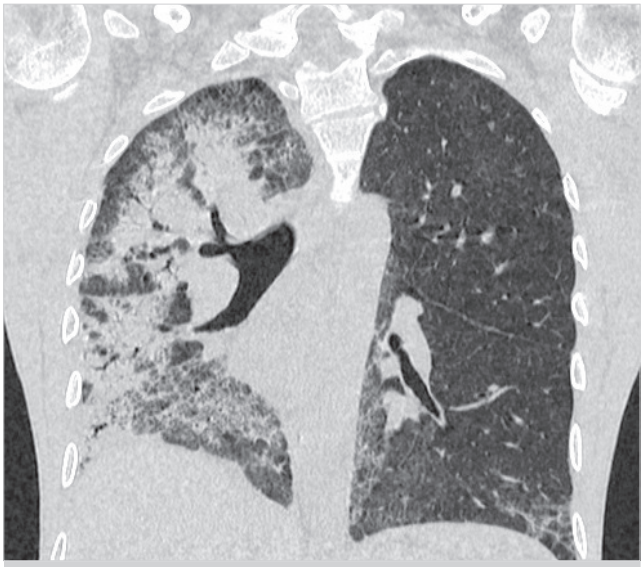

Figure 2. CT scan. Right-sided viral pneumonia associated with CMV.

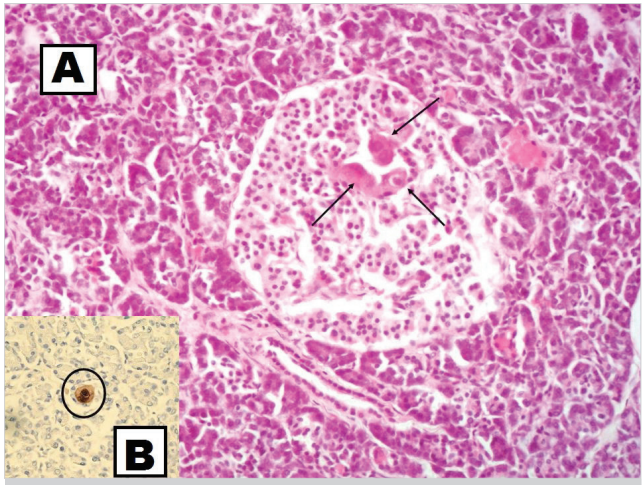

Figure 3. A. Cytomegalovirus pancreatitis. Cytomegalic transformation of Langerhans islet cells (indicated by arrows). Stained with hematoxylin and eosin. X400. **B.** "Owl's eye cell" in the pancreas during immunohistochemical examination with antibodies to cytomegalovirus. X400.

this conclusion was the Marques LM et al. study (2015) in which there was a direct correlation between sudden hyperglycemia and hypotrophic changes in the pancreas against the background of HIV infection. According to a number of researchers, the virus causes apoptosis of acinar and islet cells which leads to exocrine and endocrine organ failure (23,24). The combination of newly diagnosed hyperglycemia, signs of pancreatic hypotrophy (reduction in size with CT and ultrasound), the presence of HIV infection as well as hyperamylasemia and a typical clinical picture of the disease, we interpreted as pancreatitis directly associated with the human immunodeficiency virus. In our observations it was not possible to prove the link of non-tuberculosis

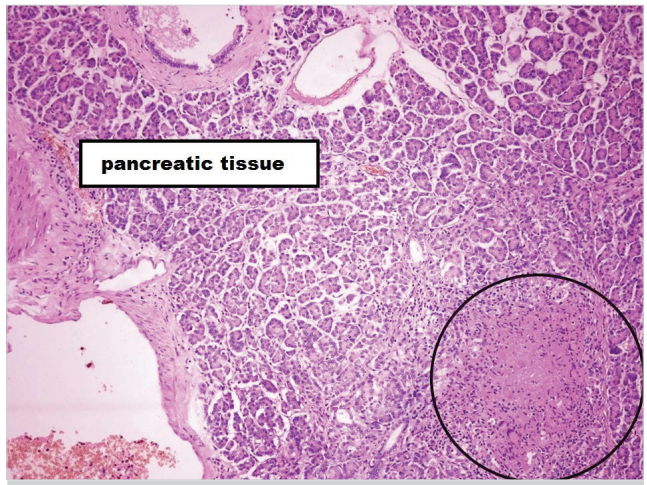

Figure 4. Tuberculosis of the pancreas. There is a miliary lesion in the pancreas with a necrosis site in the center. Stained with hematoxylin and eosin. X200.

mycobacteria, toxoplasmas and pneumocysts with the development of acute pancreatitis in people with HIV infection.

When analyzing the physiological state within the HIV-positive group according to the SAPS II scale, the severity of the disease was slightly different in patients with low immune status (CD^{4+} cells less than 200) and with CD^{4+} lymphocytes more than 200 in 1 mL. Thus, we observed the sum of points from 0 to 14 only in six patients without immunodeficiency (7.6%), from 15 to 34 points in 32 cases (40.5%) with normal immunogram and in six cases with immunosuppression (7.6%), that is, more than half of the patients (55.7%) had pancreatitis in mild and moderate-severe form in which the risk of death did not exceed 15.3%. Only in four patients with more than 200 CD^{4+} cells and in five patients with immunodeficiency (11.4%), AP was severe, along with this total score on the SAPS II score ranging from 38 to 70, the risk of death was determined in the range from 21.3% to 83.8%. At the same time, 26 patients with a CD^{4+} cell less than 200 whose the total score was 38 (21.3% risk of death) demonstrated a moderate-severe course of AP-no deaths were recorded among them.

DISCUSSION

In the last decade, due to the introduction of new drugs for the treatment of HIV that are less toxic to the pancreas, a wider coverage of ART has also changed the structure of etiological factors for the occurrence of acute pancreatitis in HIV-positive patients (7,9). In the conducted study, we made an attempt to compare the severity of the course and the totality of the causes of AP in HIV-infected and HIV-negative patients (Figure 5).

The differences in the structure of the etiological causes of pancreatitis between the groups were statistically significant ($p< 0.001$). This made it possible to reject the null hypothesis

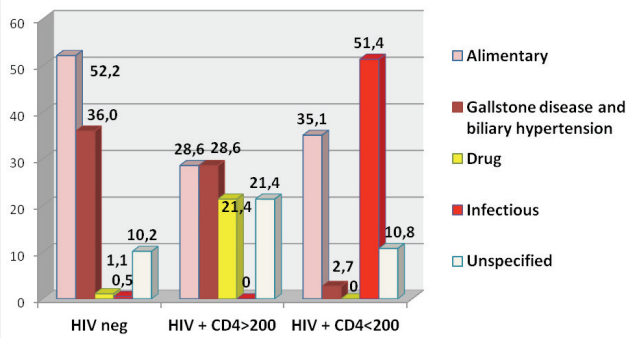

Figure 5. Structure of causes of acute pancreatitis in HIV-negative patients and HIV-positive patients with different immune status.

and confirm the previously described in the literature provisions about the possible predominance of the medicinal etiology of pancreatitis as a consequence of taking medicals, including antiretroviral, in a group of HIV-infected patients and infectious etiology in the same group due to immunodeficiency (5,8,11,22).

The comparison of the results of our study and the data published by Anderson et al. (2017), based on the analysis of the causes of AP in 2001-2010, clearly demonstrates the changes in the etiological structure of AP (25). Thus, according to the authors, gallstones and biliary hypertension have caused acute pancreatitis in 23.6% of HIV-negative patients and 17.9% of HIV-infected (1: 1.3) which is fundamentally different from the data we obtained where this ratio was 36.0% and 16.5%, respectively (2.2; 1). Probably, this can be explained by hereditary factors that in most cases determine the formation frequency of gallstones in different ethnic groups and residents of different continents as well as the nature of nutrition and lifestyle. In our study, the vast majority of the individuals were Europeans, while in the analysis conducted by Anderson et al. (25), they were immigrants from the African continent. Alcohol and fatty food, as causes of AP, still occupy the leading positions in both groups of patients. Thus, more than 10 years ago, alimentary factors triggered pancreatitis in 24.5% of HIV-positive patients and 68.3% of HIV-negative patients (1; 2.8). According to the results of our research this ratio is approximately 52.2% and 31.7% (1; 1.6).

Over the past decade, in the group of patients living with HIV, a significant decrease in cases of acute pancreatitis associated with ART could be noted, so in the past, medications caused 35.8% of the cases of AP, but according to the data we received, their share may be about 11.4%. This can probably be explained by the widespread introduction into clinical practice of less toxic to the pancreas integrase inhibitors and non-nucleoside reverse transcriptase inhibitors. Infectious agents are still factors in the development of acute pancreatitis in HIV-infected patients in about one in five cases, 18.9% according to Anderson et al. (25) and 24.1% according to the results of their own research (p= 0.3).

The high frequency of infectious and drug etiology of pancreatitis in the group of HIV-positive patients should logically correlate with the immune status of the patient. As our study showed, in patients with normal immune status who are highly likely to regularly take ART the drug etiology of pancreatitis prevailed in the structure of the causes of AP and in patients with immunodeficiency infectious causes of pancreatitis prevailed respectively. The results of testing this hypothesis are presented in Table 2.

Differences in the frequency of the causes of AP in the groups are statistically significant (p< 0.001). This allows to reject the null hypothesis and accept an alternative hypothesis about the role of the immune status in the genesis of pancreatitis in HIV-infected patients. Although the group of patients with HIV-positive status and the number of CD^{4+} cells over 200 in MCL differs in structure from patients without HIV infection but is closer to it compared to patients with low immune status (Figure 5).

When comparing the score assessment of the severity of the patients' condition using the SAPS II scale, the differences between the groups were statistically significant (p< 0.001). According to the results of data analysis, it is clearly visible that HIV infection is a factor that, regardless of the presence of immunosuppression, aggravates the course of pancreatitis by about two times (Table 3).

Nevertheless, if we compare the severity of pancreatitis in groups with normal immune status and with immunosuppression it turns out that in patients with CD^{4+} lymphocytes more than 200 AP is more severe and the number of points on the SAPS II scale is on average 38, versus 24. In our opinion, this is

Table 2. Causes of pancreatitis in HIV-positive patients with different immune status

	Etiology of pancreatitis									
	Alimentary		**Gallstone disease and biliary hypertension**		**Drug**		**Infectious**		**Unspecified**	
Number of CD^{4+} cells in MCL.	**abs.**	**%**	**abs.**	**%**	**abs.**	**%**	**abs.**	**%**	**abs.**	**%**
>200	12	**28.6**	12	**28.6**	9	**21.4**	0	**0.0**	9	**21.4**
<200	13	**35.1**	1	**2.7**	0	**0.0**	19	**51.3**	4	**10.8**

Table 3. Comparison of indicators of the severity of the condition of patients with HIV-negative and HIV- positive status, according to the SAPS II scale

	Quartils			
Group	0%	25%	50%	75%
HIV-	9	14	17	24
CD^4> 200	14	17	24	34
CD^4< 200	24	38	38	38
CD^4< 200-17	7	21	21	21
HIV+	14	24	34	38
HIV+ (CD4< 200-17)	7	17	21	29

due to incorrect scoring in the group of HIV-positive patients with normal immune status (17). As practice shows, 17 points are automatically mistakenly added to the total score of these patients indicating that the patient has AIDS. If we consider the severity of the condition of this subgroup of patients with HIV infection and take into account the adjustment of the sum of points by 17, it turns out that the severity of the course of pancreatitis in them differs little from the severity of the course of AP in the group of HIV-negative patients (21 and 17 respectively). Pancreatitis is most severe in people with low immune status, which is probably due to frequent infectious causes: pneumonia, meningoencephalitis and generalized infections complicating the course of the disease.

CONCLUSION

Currently, there is a clear tendency to change the environmental background in acute pancreatitis in HIV-infected patients. Modern medicals used for ART are much less likely to cause acute pancreatitis and the causes of pancreatic damage in this group do not differ much from the triggers of acute pancreatitis in HIV-negative patients. The same trend concerns the severity of the course of the disease and the risk of death in patients receiving modern antiretroviral therapy, with a CD^{4+} lymphocyte count of more than 200 cells per μL. On the contrary, the severity of the disease and the risk of death remain high in acute pancreatitis caused by infectious agents against the background of immunosuppression. Timely determination of the etiological factor of acute pancreatitis in HIV-infected patients is a key moment for adequate etiotropic therapy and reducing the risk of death.

Ethics Committee Approval: This study was approved by the Ethics Committee of Moscow Research and Clinical Center for TB Control (Protocol No: 325, Date: 13.05.2022).

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – DP, UG, EB; Design - UG, SS, MR; Supervision - DP, SS, MR, EB; Funding – UG, DP, EB; Materials - UG, SS; Data Collection and/or Processing - DP, MR; Analysis and/or Interpretation - DP, UG, SS; Literature Review - DP, MR, EB; Writer- UG; Critical Review - EB, SS, DP.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. *UNAIDS. Fact sheet - Latest global and regional statistics on the status of the AIDS epidemic. Available from: www.unaids.org/sites/default/files/media_asset/UNAIDS_FactSheet_en.pdf (Accessed date: 13.11.2022).*
2. *Sharp PM, Hahn BH. Origins of HIV and the AIDS pandemic. Cold Spring Harb Perspect Med 2011; 1(1): a006841. https://doi.org/10.1101/cshperspect.a006841*
3. *Reference of the FBIS «Central Research Institute for Epidemiology» of the Federal Service for Surveillance. - 2021; 4p. http://www.hivrussia.info/wp-content/uploads/2022/03/Spravka-VICH-v-Rossii-na-31.12.2021-g.pdf*
4. *Walkowska J, Zielinska N, Tubbs RS, Podgórski M, Dłubek-Ruxer J, Olewnik Ł. Diagnosis and treatment of acute pancreatitis. Diagnostics (Basel) 2022; 12(8): 1974. https://doi.org/10.3390/diagnostics12081974*
5. *Petri SI, Darvin VV. Acute pancreatitis in HIV-infected patients: The current developments. Int Res J 2021; 6(108): 137-42. https://doi.org/10.23670/IRJ.2021.108.6.060*
6. *Riedel DJ, Gebo KA, Moore RD, Lucas GM. A ten-year analysis of the incidence and risk factors for acute pancreatitis requiring hospitalization in an urban HIV clinical cohort. AIDS Patient Care STDS 2008; 22(2): 113-21. https://doi.org/10.1089/apc.2007.0034*
7. *Oliveira NM, Ferreira FA, Yonamine RY, Chehter EZ. Antiretroviral drugs and acute pancreatitis in HIV/AIDS patients: Is there any association? A literature review. Einstein (Sao Paulo) 2014; 12(1): 112-9. https://doi.org/10.1590/S1679-45082014RW2561*
8. *Dragovic G. Acute pancreatitis in HIV/AIDS patients: An issue of concern. Asian Pac J Trop Biomed 2013; 3(6): 422-5. https://doi.org/10.1016/S2221-1691(13)60091-X*
9. *Szőke D, Ridolfo A, Valente C, Galli M, Panteghini M. Frequency of pancreatic hyperamylasemia in human immunodeficiency virus-positive patients in the highly active antiretroviral therapy era. Am J Clin Pathol 2016; 145(1): 128-33. https://doi.org/10.1093/ajcp/aqv020*
10. *Dutta SK, Ting CD, Lai LL. Study of prevalence, severity, and etiological factors associated with acute pancreatitis in patients infected with human immunodeficiency virus. Am J Gastroenterol 1997; 92(11): 2044-8.*

11. *Chehter EZ, Longo MA, Laudanna AA, Duarte MI. Involvement of the pancreas in AIDS: A prospective study of 109 post-mortems. AIDS 2000; 14(13): 1879-86. https://doi.org/10.1097/00002030-200009080-00001*
12. *Blanchard JN, Wohlfeiler M, Canas A, King K, Lonergan JT. Pancreatitis with didanosine and tenofovir disoproxil fumarate [corrected]. Clin Infect Dis 2003; 37(5): 57-62. https://doi.org/10.1086/376991*
13. *Plotkin DV, Rodoman GV, Reshetnikov MN, Belentceva OV, Gafarov UO, Stepanov EA, et al. CMV infection as the cause of acute surgical pathology. Surgeon (in RUS) 2019; 7: 55-69.*
14. *Manfredi R, Calza L. HIV infection and the pancreas: Risk factors and potential management guidelines. Int J STD AIDS 2008; 19(2): 99-105. https://doi.org/10.1258/ijsa.2007.007076*
15. *Kiriyama S, Gabata T, Takada T, Hirata K, Yoshida M, Mayumi T, et. al. New diagnostic criteria of acute pancreatitis. J Hepatobiliary Pancreat Sci 2010; 17(1): 24-36. https://doi.org/10.1007/s00534-009-0214-3*
16. *Wingfield T, Wilkins E. Opportunistic infections in HIV disease. Br J Nurs 2010; 19(10): 621-7. https://doi.org/10.12968/bjon.2010.19.10.93543*
17. *Le Gall JR, Lemeshow S, Saulnier F. A new Simplified Acute Physiology Score (SAPS II) based on a European/North American multicenter study. JAMA 1993; 270(24): 2957-63. https://doi.org/10.1001/jama.1993.03510240069035*
18. *Simons-Linares CR, Imam Z, Chahal P. Viral-attributed acute pancreatitis: A systematic review. Dig Dis Sci 2021; 66(7): 2162-72. https://doi.org/10.1007/s10620-020-06531-9*
19. *Kanbay M, Sekuk H, Yilmaz U, Gur G, Boyacioglu S. Acute pancreatitis associated with combined lisinopril and atorvastatin therapy. Dig Dis 2005; 23(1): 92-4. https://doi.org/10.1159/000084729*
20. *Madsen JS, Jacobsen IA. Angiotensin converting enzyme inhibitor therapy and acute pancreatitis. Blood Press 1995; 4(6): 369-71. https://doi.org/10.3109/08037059509077623*
21. *Dickey SE, Mabry WA, Hamilton LA. Possible sulfamethoxazole/trimethoprim-induced pancreatitis in a complicated adolescent patient posttraumatic injury. J Pharm Pract 2015; 28(4): 419-24. https://doi.org/10.1177/0897190015585750*
22. *Osiro S, Muhammad R, Ruiz J, Sladen A, Mathenge N, Tiwari KJ. Acute necrotizing pancreatitis in the setting of CMV viremia and AIDS: A case report and review of literature from 1980 to 2012. JOP 2012; 13(6): 705-11.*
23. *New-Aaron M, Ganesan M, Dagur RS, Kharbanda KK, Poluektova LY, Osna NA. Pancreatogenic diabetes: Triggering effects of alcohol and HIV. Biology (Basel) 2021; 10(2): 108. https://doi.org/10.3390/biology10020108*
24. *Marques LM, Hurtado R, Chehter EZ. HIV and Pancreas in the HAART ERA: Endocrinological Patterns. JOP. J Pancreas (Online) 2015; 16(6): 540-6.*
25. *Anderson F, Thomson SR. Pancreatitis in a high HIV prevalence environment. S Afr Med J 2017; 107(8): 706-9.https://doi.org/10.7196/SAMJ.2017.v107i8.10296*

ORİJİNAL ÇALIŞMA-ÖZET
Turk J Surg 2023; 39 (1): 76-82

Farklı bağışıklık durumlarına sahip HIV pozitif hastalarda akut pankreatitin etiyolojisi ve şiddet özellikleri

Dmitriy Plotkin[1], Umedzhon Gafarov[1], Mikhail Reshetnikov[1], Sergey Sterlikov[3], Elena Bogorodskaya[1], Dmitriy Plotkin[2]

[1] Moskova Tüberküloz Kontrol Araştırma ve Klinik Merkezi, Cerrahi Kliniği, Moskova, Rusya
[2] Pirogov Rus Ulusal Araştırma Tıp Üniversitesi, Genel Cerrahi Anabilim Dalı, Moskova, Rusya
[3] Rusya Federasyonu'nda Tüberküloz Bulaşmasını Önleme Federal İzleme Merkezi Rus Sağlık Araştırma Enstitüsü, Moskova, Rusya

ÖZET

Giriş ve Amaç: Akut pankreatit, HIV ile enfekte hastalarda sık görülür, ancak HIV pozitif hastalarda pankreatit nedenleri ve şiddeti, hem pankreasın yıkımının şiddetini hem de tanı ve tedavi yöntemlerini etkileyen bir takım önemli özelliklere sahiptir.

Gereç ve Yöntem: İki grup akut pankreatitli hastanın anamnestik verileri, tanı ve tedavi sonuçları incelendi. Birinci grup, 2017-2021 yılları arasında kliniğe başvuran HIV enfeksiyonu ile kombine akut pankreatitli 79 hastayı içermektedir. HIV ile yaşayan insanlarda ilaçlar ve enfeksiyöz ajanlar sırasıyla olguların %11,4'ü ve %24,1'inde akut pankreatite neden olmuştur. Çalışmamızın da gösterdiği gibi, AP'nin nedenlerinin yapısında immün durumu normal olan hastalarda pankreatitin ilaç etiyolojisi, immün yetersizliği olan hastalarda pankreatitin enfeksiyöz nedenleri baskındı.

Bulgular: Veri analizi sonuçlarına göre, HIV enfeksiyonunun, immünsupresyon varlığından bağımsız olarak, pankreatit seyrini yaklaşık iki kat kötüleştiren bir faktör olduğu açıktır. HIV ile ilişkili akut pankreatitin etiyolojik yapısı doğrudan hastanın bağışıklık durumuna bağlıdır ve birçok yönden HIV negatif hastalardan veya ART alan hastalardan farklıdır.

Sonuç: İmmünsupresyon zemininde enfeksiyöz ajanların neden olduğu akut pankreatitte hastalığın şiddeti ve ölüm riski yüksektir.

Anahtar Kelimeler: Akut pankreatit, HIV enfeksiyonu, antiretroviral tedavi, bağışıklık durumu, SAPS II

DOİ: 10.47717/turkjsurg.2023.5939