ORIGINAL ARTICLE

The relationship between lymphopenia and development of late complications in severe acute pancreatitis

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ABSTRACT

BACKGROUND: In this study we aimed to predict patients who would develop late stage acute pancreatitis related complications. So we would be able to ease the decision making process about the timing of cholecycstectomy. On the other hand we also suggest a possible insight into the mechanisms which lead development of lyphopenia in severe acute pancreatitis and its possible effects on prognosis.

METHODS: In this study, 163 severe acute pancreatitis case who has been treated as inpatient between January 2013 and January 2018 has been involved. Patients charts and all documented data has been analysed retrospectively. According to the existence or absence of late complications of severe acute pancreatitis, patients have been divided into two groups; Group 1 had no late complication, Group 2 had either pseudocyst or WON (Walled of Necrosis) at 1st month CT.

RESULTS: The difference between two groups in terms of 48th hour lymphocyte percentage was significant (p=0.000; p<0.05). Group 2 had remarkably longer duration of hospital stay (p=0.000; p<0.05). 48th hour CRP level of group 2 was significantly higher than of group 1 (p<0.000).

CONCLUSION: There is a statistically significant relation between the presence of lymphopenia, at 48th hour of presentation in severe biliary pancreatitis patients and development of delayed complications. We can strongly say that there would be no late term pancreatitis related complications if there was no lymphopenia at 48 hour and an early cholecystectomi can be performed in such cases. Lymphopenia seen around 48. hr of admission is highly related to development of late complications in severe acute pancreatitis.

Keywords: Inflammation; lymphopenia; severe acute pancreatitis.

INTRODUCTION

Acute pancreatitis is a common disease which could lead to local or systemic complications and which could run a severe course. Differential characteristic of disease is minimal or lack of fibrosis within the gland. It is among the most common gastrointestinal diseases which require hospital admission. Worldwide incidence is 5–80/100.000[1] and an increase in its incidence has been observed worldwide[2,3] likely due to prolongation of mean life expectancy and increase in incidence of obesity. Differences in incidence among races and countries have been reported.[4] General mortality rate is 1/100.000; however, it can increase up to 10–30% in severe cases.[5]

Pancreatic inflammation induced by obstructing biliary calculi or sludge is known as biliary pancreatitis. To make the diagnoses other etiologies such as alcohol, enfection, hypertriglisseridemia, toxins, and drugs, leading to development of acute pancreatitis must be eliminated. The main etiology of biliary pancreatitis is obstruction or edema caused by passage of

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biliary calculi. Clinical course has wide range from mild self-restricted disease to infected pancreatic necrosis, multi-organ failure, and high mortality disease.[6]

Diagnosis

Two of the three below criteria should be met to make the diagnose of acute pancreatitis; pain, elevated amylase, and lipase levels, imaging.[7,8]

Pathophysiology

Premature intraasiner activation of digestive enzymes has been demonstrated in several animal models of acute pancreatitis. This results in auto-digestion of pancreatic gland. [9] Trypsinogen activation is a critical step, minimal panceratic damage has been observed in trypsinogen -/- mice.[10]

The exact mechanism of intraaciner activation of zymogens is not yet well understood. Activated zymogens are co-localized with Kathepsin B within zymogen granules and Kathepsin B activates trypsin.[11] Trypsin activation is not occur and pancreatic damage is minimal in Kathepsin -/- mice.[12] In sytosole Kathepsin B activates aciner cell apoptosis.[13]

Following aciner cell injury neutrophils are the first cells to arrive the area. Superoxides (oxidative burst) and enzymes (katepsin, elastase, and collegenase) secreted from neutrophills increase tissue damage. TNF α, IL6, and IL8 from macrophages induce local and systemic inflammation. As a result, pancreas vascular permeability increases leading edema and hemorrhage. Microthrombuses develops within vascular structures which causes hypoperfusion and necrosis of pancreatic tissue.

An important component of AP pathophysiology is systemic inflammation which could lead to multiple organ failure. NFκB pathway is responsable from systemic events. Tyripsin and NFκB activation develops simultaneously; however, they are independent from each other. In tyripsin -/- mice NFκB activation still occurs leading to AP. Intracellular calcium is required for activation of both. After NFκB activation profound amount of cytokines and chemokines are secreted and released to systemic circulation.[14]

Immune System

Immune system is responsible for defense and protection against microorganisms and diseases. Immune response is a collective and coordinated work of immune cells and molecules. Immune response is capable of causing tissue damage and diseases in some cases. Therefore, a more accurate definition of immune response would be a chain of reactions against foreign or damaged molecules whatever the consequences are.[15]

Immune Tolerance

Tolerance is the activation of immune system against foreign antigens, while the organism remains unresponsive to its own antigens. Tolerance protects the organism against autoimmune tissue damage. Immune tolerance is transferred to the future generations hereditarily or immune tolerance may be induced.

Tissue-Specific Antigens (Tissue Restricted Antigens)

Presentation of organism's own antigens to T cells is realized in thymus epithelial cells. T cells, which reacts against these self-antigens, are devastated. Antigen-presenting cell located in thymus carry the tissue-specific antigens on their surfaces ectopically as a result of AIRE gene (Autoimmune Regulator gen) expression, and present them to T cells.[16] Genetically, multi-endocrine deficiency syndromes develop in subjects, which AIRE is deleted. Today, some of the known pancreas-specific antigens are insulin, islet cells, glutamic acid decarboxylase, pancreas-associated protein-1, and CD1d.

MATERIALS AND METHODS

163 patients with severe acute pancreatitis who have applied and admitted to our hospital between January 2013 and January 2018 were included in this study. Patients, whose Ranson criteria were 3 and more, 48 h CRP is 15 and more, or which organ failure was determined that did not recovered within 48 h, were considered as severe acute pancreatitis.[17] 48th h Ranson values were not included; hence, we used the Ranson values only as one of the criteria of severe pancreatitis at admission. The Modified Marshall scoring system was used to define organ failure.[18] Patients, who had any unreached data, did not have follow-up tomography, cases progressing with mortality, the recurrent pancreatitis, and non-biliary pancreatitis were excluded from the study.

Lymphocyte percentages of all cases during the application to the emergency service and at 48th h were recorded. Lymphopenia cutoff value was less than the 2/3 of the reference range threshold, The reason why we emphasize <2/3 of normal lymphocyte percentages is because this cutoff value was the most relevant and strong indicator of prospective possible complications.

The patients, who had pseudocyst or walled of necrosis diagnosis in their 1st-month control tomography, were defined as Group 2 which had delayed AP complications and those who did not develop any complications are defined as Group 1. Group 1 included 63 male, 71 female and total of 134 patients. Group 2 included 13 male, 16 female and total of 29 patients.

When evaluating the findings obtained in this study, IBM SPSS Statistics 22 (IBM SPSS, Turkey) program was used for statistical analysis. When evaluating the data of the study, descriptive statistical methods (Median, Standard deviation, frequency) were used, as well as the Continuity (Yates) Correction used in comparing the qualitative data. Significance was evaluated at $p<0.05$ level.

RESULTS

The arrival and 48th-h lymphocyte values between the groups, and the changes in the lymphocyte values within the groups were compared. Demographical data of the groups were investigated.

As shown in Table 1, 48th-h lymphocyte values of Group 2 were found to be significantly lower than Group 1 (p=0.000; p<0.05). There was no significant statistical difference between the groups regarding the arrival lymphocyte values (p>0.05).

Again, in Table 1, changes in the lymphocyte percentages of the two groups were evaluated separately within the groups. Accordingly, in Group 1, the changes seen in 48th h lymphocyte values are not statistically significant according to the arrival lymphocyte values (p>0.05). In Group 2, the decrease seen in 48th-h lymphocyte values is statistically significant, according to the arrival lymphocyte values (p=0.049; p<0.05).

In Table 2, the 48th-h CRP value and lymphocyte percentages were evaluated per groups. It was found that the 48th-h CRP values of Group 2 were significantly higher than Group 1 (p=0.000; p<0.05).

In Table 3, groups were compared regarding the arrival Ranson values. It was seen that there were not any statistically significant difference between the groups regarding the arrival Ranson values (p>0.05). 48th h Ranson values were not included hence we used the arrival Ranson values only as one of the admission criteria of severe pancreatitis.

In Table 4, ages and hospitalization periods of the groups were compared. There are not any statistically significant differences between the groups regarding age average (p>0.05). It was found that the hospitalization period values of Group 2 were statistically significantly higher than Group 1 (p=0.000; p<0.05). There are not any statistically significant differences between the groups regarding gender distribution rates (p>0.05).

DISCUSSION

There are many studies showing that the development of lymphopenia at the beginning of severe acute pancreatitis during its course may cause serious local/systemic complications, organ failure, pancreatic sepsis, and mortality. However, there are no studies emphasizing the relation between the severe lymphopenia seen at the beginning of AP and delayed AP complications developing after 4 weeks.

It is observed in some experimental and clinic studies that there is a significant decrease in number and function of CD4+ Th1 in severe acute pancreatitis, when compared with CD4+ Th2. As a result of this, number of Th2, which secretes anti-inflammatory cytokines, becomes dominant, and possibility of infective complications/sepsis increases.[18,19]

TNF, which is secreted from CD4+Th1 cells and macrophages, which has arrived into inflammation area previously, activates NFκB. When NFκB is activated, a large quantity of cytokines are synthesized, and secreted into the systemic circulation. Thus, the systemic inflammation and organ failure table, which are seen in severe AP, is developed.[20] Activation of trypsin and NFκB activation, which initiates intrapancreatic

Table 1. Assessment of lymphocyte parameters of arrival - 48th hour between and within the groups

	Group 1 (n=134)	Group 2 (n=29)	Total (n=163)	p[1]
	(Min-Max)–(Av.±SD)	(Min-Max)–(Av.±SD)	(Min-Max)–(Av.±SD)	
Arrival lymphocyte %	(1.9–41)–(13.16±8.45)	(2.4–45.7)–(12.52±9.95)	(1.9–45.7)–(13.05±8.7)	0.723
48th hour lymphocyte %	(2.3–36)–(14.86±7.72)	(3.9–27)–(9.3±4.66)	(2.3–36)–(13.87±7.57)	0.000
Arrival-48th hour p[2]	0.059	0.049*		

[1]Student t-test. [2]Paired sample's t test. *P<0.05. Av.: Min: Minimum; Max: Maximum; Average; SD: Standard deviation.

Table 2. Assessment of normality parameters of 48th hour CRP and lymphocyte value between groups

	Group 1	Group 2	Total	p
	(Min-Max)–(Av.±SD)	(Min-Max)–(Av.±SD)	(Min-Max)–(Av.±SD)	
48. hour CRP	(0.7–45)–(17.68±9.11)	(15.2–36)–(25.24±6.04)	(0.7–45)–(19.03±9.1)	[1]0.000*
Lymphocyte value normality, n (%)				
Normal	97 (72.4)	11 (37.9)	108 (66.3)	[2]0.001*
Abnormal	37 (27.6)	18 (62.1)	55 (33.7)	

[1]Student t-test. [2]Continuity (yates) correction. *P<0.05. CRP: C-reactive protein; Av.: Min: Minimum; Max: Maximum; Average; SD: Standard deviation.

Table 3. Assessment of arrival Ranson values between the groups

	Arrival Ranson
	(Min-Max)-(Av.±SD [median])
Group 1	(0–4)–(2.11±1.21 [2])
Group 2	(0–5)–(2.07±1.16 [2])
Total	(0–5)–(2.1±1.19 [2])
p	0.552

Mann-Whitney U test. Av.: Min: Minimum; Max: Maximum; Average; SD: Standard deviation.

incidents, is simultaneous, but independent from each other. An increase occurs in intracellular calcium in AP, and increased calcium plays a role both in trypsinogen activation (by activating the cathepsin B enzyme, and located in lysosomes) and in NFκB activation.[21]

Again, TNF triggers activation of more lymphocytes. While there is an increase in number of function of all T lymphocytes as a result of being stimulated with TNF in healthy persons, B lymphocytes are not affected from TNF. There is a statistically significant decrease regarding the number and function of entire T lymphocyte sub-groups in severe AP after being stimulated with TNF, instead of an increase. Besides the decrease in the number of T lymphocytes responding TNF stimulation, these cells also give a significantly weak response against bacterial stimulation, and these patients become susceptible to infection.[22]

In another study, a significant decrease has been seen in the number of both CD4+ and CD8+ T lymphocytes and B lymphocytes; however, it was recorded that the ratios have changed in different phases of AP (1st day, 10th day, and 30th day). In the same study, an increase was observed in the quantity of the molecules showing apoptosis in lymphocytes.[23] Similarly, it was shown that the numbers of both T and B lymphocytes decrease in circulation and numbers in pancreas tissue in severe AP significantly, but decrease in B lymphocytes is more significant. Again, in this study lymphocyte apoptosis indicators found high.[24]

In the study, performed on observing a decrease in spleen density in abdominal-contrasted tomography of severe AP patients, severe pancreatitis was induced in mice and spleen was examined pathologically. It was seen that T lymphocytes in spleen remain normal in number, but B lymphocytes decreased, spleen hemoperfusion decreased, and B lymphocytes are damaged. The peptide serum level, which is secreted from spleen specifically, named as "Tuftsin," have immune regulator effects, and used in measuring immune functions of spleen, was measured as significantly low.[25]

Cause of the decrease in blood levels of lymphocytes is not known clearly. One of the causes may be the dense migration of the lymphocytes from blood to inflammation area and other affected organ areas such as lung and kidney. Another cause may be the increase measured in apoptosis indicators and induction of apoptosis in lymphocytes, as shown in various studies. It was shown that severe AP patients have tendency to apoptosis rather than activation and proliferation, in another in vitro study.[26]

The factor which leads to the induction of apoptosis in lymphocytes may be the deterioration of pro- and anti-inflammatory mechanisms, as well as there is possibility of molecule or molecules secreted from pancreas or other localization, which we have not defined yet. The suggestion that dense migration into inflammation area causes the decrease in blood levels, is weak, because in recent studies it was shown that lymphocytes decrease in also pancreas tissue. Therefore, it shall be more accurate to direct the studies whether there is a molecule having apoptosis inducing characteristic or not. This molecule is probably a self-antigen which pancreas tissue is limited, and emerges with acinar cell damage. Central tolerance and peripheral tolerance (Treg cell proteins; tissue TGFβ, IL10, IL35, and CD25) induce apoptosis in lymphocytes by their nature, and decrease is seen in their numbers. The possibility must be considered that the lymphopenia, which is developed as a result of apoptosis in severe AP, has protective effect on

Table 4. Assessment of age, hospitalization period, and gender between the groups

	Group 1	Group 2	Total	p
	(Min-Max)–(Av.±SD)	(Min-Max)–(Av.±SD)	(Min-Max)–(Av.±SD)	
Age	(19–93)–(66.05±17.02)	(35–88)–(61.72±13.33)	(19–93)–(65.28±16.47)	[1]0.201
Hospitalization (days) (median)	(2–28)–(7.7±4.71 [6])	(2–68)–(16.61±14.95 [11.5])	(2–68)–(9.24±8.2 [7])	[2]0.000*
Gender, n (%)				
Men	63 (47)	13 (44.8)	76 (46.6)	[3]0.993
Women	71 (53)	16 (55.2)	87 (53.4)	

[1]Student t-test. [2]Mann-Whitney U test. [3]Continuity (yates) correction. *P<0.05. Av.: Min: Minimum; Max: Maximum; Average; SD: Standard deviation.

organism survival, in expense of severe local destruction of pancreatic tissue itself.

There are several limitations of this study. One of them is the relatively low number of patients in group 2. There would be a better understanding of justifiability of the results if the number of patients in group two was larger. Another limitation is that this was a retrospective study. Further prospective study might aid assessing prognosis of early cholecystectomy in suitable patients with severe acute pancreatitis. Thereby we would be able to argue whether early cholecystectomy would cause any detrimental effect in such cases or provoke development of any late complications. Finally, 48th h Ranson criteria were not included in the results. This might seemingly be interpreted as incompleteness. However we used the Ranson values only as one of the criteria of severe pancreatitis at admission. Ranson values at 48th h were irrelevant to the aim of our study.

Conclusion

There is a statistically significant relation between the presence of lymphopenia at 48th h of presentation in severe biliary pancreatitis patients and development of delayed complications. We can strongly say that there would be no late term pancreatitis related complications if there was no lymphopenia at 48 h and an early cholecystectomy can be performed in such cases.

Informed Consent: Written informed consent was obtained from the patients for the publication of the case report and the accompanying images.

Peer-review: Internally peer-reviewed.

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REFERENCES

1. Fisher WE. Schwartz's Principles of Surgery. 10th ed., Ch. 33. Australia: Sayfa; 2015. p. 1341–6.
2. Petrov MS, Shanbhag S, Chakraborty M, Phillips AR, Windsor JA. Organ failure and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. Gastroenterology 2010;139:813–20. [CrossRef]
3. Banks PA. Epidemiology, natural history, and predictors of disease outcome in acute and chronic pancreatitis. Gastrointest Endosc 2002;56:S226–30. [CrossRef]
4. Yadav D, Lowenfels AB. Trends in the epidemiology of the first attack of acute pancreatitis: A systematic review. Pancreas 2006;33:323–30.
5. Spanier BW, Dijkgraaf MG, Bruno MJ. Epidemiology, aetiology and outcome of acute and chronic pancreatitis: An update. Best Pract Res Clin Gastroenterol 2008;22:45–63. [CrossRef]
6. Harrison EM, Parks RW. Blumgart's Surgery of the Liver Biliary Tract and Pancreas. 5th ed., Ch. 51. Australia: Sayfa; 2012. p. 817–21. [CrossRef]
7. Aziret M, Ercan M, Toka B, Parlak E, Karaman K. Risk factors for morbidity in walled-off pancreatic necrosis and performance of continuous postoperative lavage: A single-center experience. Ulus Travma Acil Cerrahi Derg 2018;24:488–96. [CrossRef]
8. Akhtar AJ, Shaheen M. Extrapancreatic manifestations of acute pancreatitis in African-American and Hispanic patients. Pancreas 2004;29:291–7. [CrossRef]
9. Kaya E, Dervisoglu A, Polat C. Evaluation of diagnostic findings and scoring systems in outcome prediction in acute pancreatitis. World J Gastroenterol 2007;13:3090–4. [CrossRef]
10. Güngör B, Cağlayan K, Polat C, Seren D, Erzurumlu K, Malazgirt Z. The predictivity of serum biochemical markers in acute biliary pancreatitis. ISRN Gastroenterol 2011;2011:279607. [CrossRef]
11. American Gastroenterological Association (AGA) Institute on "Management of Acute Pancreatits" Clinical Practice and Economics Committee. AGA Institute Governing Board. AGA Institute medical position statement on acute pancreatitis. Gastroenterology 2007;132:2019–21.
12. Lee JK, Enns R. Review of idiopathic pancreatitis. World J Gastroenterol 2007;13:6296–313. [CrossRef]
13. Saluja A, Steer M. Pathophysiology of pancreatitis. Role of cytokines and other mediators of inflammation. Digestion 1999;60:27–33. [CrossRef]
14. Dawra R, Sah RP, Dudeja V, Rishi L, Talukdar R, Garg P, et al. Intra-acinar trypsinogen activation mediates early stages of pancreatic injury but not inflammation in mice with acute pancreatitis. Gastroenterol 2011;141:2210–17.e2212. [CrossRef]
15. Buchmann K. Evolution of innate immunity: Clues from invertebrates via fish to mammals. Front Immunol 2014;5:459. [CrossRef]
16. Bluestone JA, Bour-Jordan H, Cheng M, Anderson M. T cells in the control of organ-specific autoimmunity. J Clin Invest 2015;125:2250–60.
17. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis-2012: Revision of the Atlanta classification and definitions by international consensus. Gut 2013;62:102–11. [CrossRef]
18. Marshall JC, Cook DJ, Christou NV, Bernard GR, Sprung CL, Sibbald WJ. Multiple organ dysfunction score: A reliable descriptor of a complex clinical outcome. Crit Care Med 1995;23:1638–52. [CrossRef]
19. Ueda T, Takeyama Y, Yasuda T, Takase K, Nishikawa J, Kuroda Y. Functional alterations of splenocytes in severe acute pancreatitis. J Surg Res 2002;102:161–8. [CrossRef]
20. Baud V, Karin M. Signal transduction by tumor necrosis factor and its relatives. Trends Cell Biol 2001;11:372–7. [CrossRef]
21. Hietaranta AJ, Singh VP, Bhagat L, van Acker GJ, Song AM, Mykoniatis A, et al. Water immersion stress prevents caerulein-induced pancreatic acinar cell nf-kappa b activation by attenuating caerulein-induced intracellular Ca2+ changes. J Biol Chem 2001;276:18742–7. [CrossRef]
22. Oiva J, Mustonen H, Repo H, Kyhälä L, Kuuliala K, Kemppainen E, et al. Acute pancreatitis with organ dysfunction associates with abnormal blood lymphocyte signaling: Controlled laboratory study. Crit Care 2010;14:R207. [CrossRef]
23. Căluianu EI, Alexandru DO, Târtea EA, Georgescu M, Mercu ȚD, Traşcă ET, et al. Assessment of T- and B-lymphocytes and VEGF-A in acute pancreatitis. Rom J Morphol Embryol 2017;58:481–6.
24. Ozdemir H, Akut Pankreatitli Hastalarda Sitokeratin 18 Düzeyinin Önemi Ve Apopitozisin Bir Göstergesi Olan Sitokeratin 18 Düzeyi İle Akut Pankreatitin Klinik Seyri Arasındaki Korelasyon TC. Gaziantep Üniversitesi Tıp Fakültesi, Gastroenteroloji Anabilim Dalı, 2010, Uzmanlık Tezi.
25. Shao G, Zhou Y, Song Z, Jiang M, Wang X, Jin X, et al. The diffuse reduction in spleen density: An indicator of severe acute pancreatitis? Biosci Rep 2017;37:BSR20160418. [CrossRef]
26. Takeyama Y, Takas K, Ueda T, Hori Y, Goshima M, Kuroda Y. Peripheral lymphocyte reduction in severe acute pancreatitis is caused by apoptotic cell death. J Gastrointest Surg 2000;4:379–87. [CrossRef]

ORİJİNAL ÇALIŞMA - *ÖZ*

Ağır biliyer pankreatit olgularında lenfopeni varlığının geç dönem komplikasyon gelişimi ile ilişkisi

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AMAÇ: Bu çalışmada ağır akut pankreatit (AP) olgularında hangi hastaların geç dönem komplikasyon geliştireceğini ön görebilmek hedeflendi. Böylece bu hastalarda tartışmalı olan kolesistektomi zamanlaması ile ilgili karar alma sürecini kolaylaştırabiliriz. Diğer taraftan ağır AP hastalarında gelişen lenfopeninin mekanizması ve bunun prognoza etkisi aydınlatılmaya çalışıldı.

GEREÇ VE YÖNTEM: Ocak 2013 ve Ocak 2018 tarihleri arasında hastanemizde yatarak tedavi edilen ağır bilier pankreatit tanılı 163 hastanın kayıtları geriye dönük olarak incelendi. Hastaların demografik verileri, geliş ve 48. saat lenfosit yüzdeleri kayıt edildi. Yatış tarihinden itibaren 7–10. günler arası çekilen İV-Oral kontrastlı bilgisayarlı batın tomografileri ile birinci ay ve takip tomografileri incelendi. Bu verilerden herhangi biri eksik olan hastalar çalışma dışı bırakıldı. Hastalar komplikasyon gelişen ve gelişmeyen olarak 2 gruba ayrıldı; Group 1 herhangi bir geç dönem komplikasyon bulgusu olmayan hastalar, grup 2 ise 1. ay BT'de WON (Walled of Necrosis) veya psödokist geliştiren hastalar olarak tanımlandı. Grup 1'de 134, grup 2'de 29 hasta yer aldı. Çalışmada yer alan hastaların 89'u kadın, 78'i erkekti.

BULGULAR: Gruplar arasında cinsiyet ve yaş açısından fark saptanmadı ($p>0.05$). Grup 2'nin hastane yatış sürelerinin grup 1'den anlamlı ölçüde uzun olduğu saptandı. Gruplar arasında geliş lenfosit değerleri açısından istatistiksel olarak anlamlı bir farklılık bulunmadı ($p>0.05$). Grup 2'in 48. saat lenfosit değerleri, Grup 1'den istatistiksel olarak anlamlı düzeyde düşük bulundu ($p=0.000$; $p<0.05$). 48. saatte belirgenleşen lenfopeni geç dönem komplikasyon geliştirme riski ile ilişkili olarak değerlendirildi.

TARTIŞMA: Ağır biliyer pankreatitte ilk 48 saatte görülen lenfopeni ile geç dönem gelişen komplikasyonlar arasında anlamlı bir ilişki vardır. 48. saatte lenfopeni görülmüyor ise geç dönem komplikasyon gelişmeyeceğini ve bu hasta gurubunda da erken kolesistektominin güvenle yapılabileceğini söyleyebiliriz. Ağır pankreatitte 48. saatte görülen lenfopeni ile geç dönem komplikasyon gelişimi arasında anlamlı bir ilişki vardır.

Anahtar sözcükler: Akut pankreatit; enflamasyon; lenfopeni.

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