




Potential Immunomodulatory Role of IL-37⁺CD4⁺ Helper T Cells in Autoimmune Hepatitis

 Seyda Tasci,¹  Lala Imata,¹  Narges Bagheri,¹  Gamze Yesilay,²
 Sevinc Keser,³  Selma Sengiz Erhan,⁴  Kubra Sevgin,⁵
 Gulam Hekimoglu⁵

¹International School of Medicine,
University of Health Sciences,
Istanbul, Türkiye

²Department of Molecular Biology
and Genetics, University of Health
Sciences, Hamidiye Institute of
Health Science, Istanbul, Türkiye

³Department of Pathology,
University of Health Sciences,
Kartal Dr. Lutfi Kirdar City Hospital,
Istanbul, Türkiye

⁴Department of Pathology,
University of Health Sciences, Prof.
Dr. Cemil Tascioglu City Hospital,
Istanbul, Türkiye

⁵Department of Histology and
Embryology, University of Health
Sciences, International School of
Medicine, Istanbul, Türkiye

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Correspondence: Gulam
Hekimoglu,
Department of Histology and
Embryology, University of Health
Sciences, International School of
Medicine, Istanbul, Türkiye

E-mail: gulam.hekimoglu@sbu.
edu.tr



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ABSTRACT

Objective: Autoimmune hepatitis (AIH) is a chronic liver disease characterized by inflammation of unknown origin. It is marked by high levels of immunoglobulin G (IgG) and autoantibodies in the blood. Pathologically, AIH shows infiltration of inflammatory cells, particularly plasma cells, and signs of interface hepatitis. The role of a specific type of CD4⁺ helper T cells, which produce interleukin (IL)-37, has been established in autoimmune diseases. However, their role in AIH is still not well understood. To shed light on this, our study aimed to investigate the gene and protein expression of IL-37 in liver biopsies of AIH patients and determine the involvement of tissue-resident IL-37-producing helper T cells in the development of AIH.

Methods: We conducted a comparative study involving 20 patients diagnosed with AIH and 17 healthy individuals as controls. IL-37 gene expression was measured using quantitative real-time polymerase chain reaction (qRT-PCR), and immunofluorescence double staining was performed to identify IL-37⁺CD4⁺ helper T cells in the liver tissue. The ratio of IL-37⁺CD4⁺ helper T cells among total inflammatory cells was calculated to assess their abundance in AIH patients.

Results: Our findings revealed a significant increase in the ratio of IL-37⁺CD4⁺ helper T cells in patients with AIH ($p < 0.05$). Additionally, IL-37 gene expression was upregulated in AIH patients ($p < 0.01$). Importantly, the ratio of IL-37⁺CD4⁺ Th cells showed a negative correlation with the severity of portal inflammation and Ishak scoring.

Conclusion: Our results highlight IL-37-producing helper T cells may be potential therapeutic targets for autoimmune hepatitis. Further studies are warranted to elucidate their precise mechanisms and therapeutic implications.

INTRODUCTION

Autoimmune hepatitis (AIH) is a chronic liver disorder mediated by immunity that may develop into liver fibrosis.^[1] The pathophysiology of AIH is influenced by CD8⁺ T cell

cytotoxicity, autoantibodies made by B cells,^[2] unique genetic characteristics,^[3] and compromised immunological systems such as CD4⁺ T cells^[4] and Treg cells.^[5] Damage to the liver results from the disturbance of the equilibrium between Treg cells and effector cells, which leads to the

development of the autoinflammatory response.^[6] Treg cells offer immunotolerance by limiting the proliferation, cytokine production, and cytotoxicity of effector cells.^[7] A progressive necro-inflammatory, fibrotic process results from the T cell-mediated immune system attacking liver antigens when this immunological tolerance is weakened.^[8] AIH is diagnosed histologically using interphase hepatitis, increased serum immunoglobulin G (IgG) levels, and the presence of autoantibodies. Additionally, the efficacy of therapy and post-treatment remission are tracked using these markers.^[4] Currently, azathioprine and prednisone are used to treat AIH. These combination therapies are used to lessen the side effects of prednisone or steroids taken alone, and they are effective in 80–90% of patients.^[9] Since there is no full cure for pharmacological therapy, the major objectives of treatment are to manage liver inflammation, produce biochemical remission, alleviate and/or reduce symptoms, halt the disease's development, assist in fibrosis regression, and minimize drug-related adverse effects.^[3]

A member of the IL-1 cytokine family, human interleukin-37 (IL-37) was discovered lately. Despite being identified *in silico* in 2000, IL-37's anti-inflammatory effects were just recently identified.^[10] Nold et al.^[11] claim that IL-37 is special in that it lowers inflammation by reducing the synthesis of cytokines that promote inflammation. IL-37 is abundantly expressed in humans. Through its ability to block the production of many inflammatory cytokines, IL-37 plays a significant role in both innate and adaptive immune responses. IL-37 controls the expression of cytokines, cell division, metabolism, and transcription of genes.^[11] Outside the cell, it suppresses the synthesis of IFN and lessens signal transmission through toll-like receptors, acting as an immunosuppressive drug.^[12] Also, the autoimmune disorders of systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and inflammatory bowel disease (IBD) have been linked to IL-37.^[13] Hepatocytes, cholangiocytes, and invading immune cells are the primary cell types that express IL-37. Kupffer cells, hepatic stellate cells, and Treg cells were all found to be positive for IL-37.^[14] Hepatic fibrosis, the quantity of invading immune cells, is positively correlated with IL-37 production. In a study performed on mice, IL-37 has been shown to have effects on prolonging survival, reducing liver damage, expression of early fibrosis markers, and liver fibrosis.^[15]

Additionally, T-cell subsets, such as IL-37⁺CD4⁺ helper T cells, have been linked to the control of immunological responses and the preservation of immune tolerance in autoimmune disorders.^[16] The etiology of autoimmune disease has been linked to the deregulation of various immune cell types. For instance, the suppressive lymphocytes known as regulatory T cells are essential for immunoregulation and immunological homeostasis maintenance.^[17] However, it is yet unknown how specifically IL-37⁺CD4⁺ helper T cells contribute to the regulation of the development and severity of AIH. Understanding their probable function may offer insightful information about the immunopathogenesis of AIH and suggest new treatment targets.

MATERIALS AND METHODS

This study was approved by the Ethical Committee of Kartal Dr. Lutfi Kırdar City Hospital, School of Medicine, University of Health Sciences-Türkiye (Date: 19.07.2023; No: 2023/514/254/25), and Informed Consent was obtained from all participants after the Helsinki Declaration.

Patients

All subjects gave their informed permission in accordance with the Helsinki Declaration. Paraffin blocks were chosen randomly to obtain a total of 20 patients, and 17 healthy volunteers were chosen for this study. The following criteria had to be fulfilled by study participants. Individuals with an autoimmune hepatitis diagnosis must be at least eighteen years old and free of other liver conditions. Liver tissues that had been partially removed for other purposes were sampled for the control group.

Clinical Evaluation

Our patients' liver function and autoimmunity test results were systematically scored. The AST/ALT ratio was scored as follows: Negative for a ratio less than 0.6, 1 for a ratio greater than 0.6, 2 for a ratio greater than 0.8, and 3 for a ratio greater than 1.0. GGT levels were scored as negative for values less than 85 U/L, 1 greater than 1 time 85 U/L, 2 for values greater than 2 times, and 3 for values greater than 3 times. For ALP, the scoring was negative for values less than 147 IU/L, 1 for values greater than one time 147, 2 for values greater than 2 times, and 3 for values greater than 3 times. Total Bilirubin, Albumin, and IgG levels were each scored according to their multiples of 1.2 mg/dl, 5.4 g/dl, and 16 g/l, respectively. Autoantibodies such as ANA, LKM, ASMA, and AMA were scored based on the number of positive results: 1 For one positive, 2 for two, and 3 for three. The Ishak index was scored according to the severity: Negative for none, 1 for mild, 2 for moderate, and 3 for marked. The evaluation results are presented in Figure 1.

Immunofluorescent double staining

Paraffin block biopsy samples were prepared by cutting 4 µm-thick pieces for immunofluorescent staining. After deparaffinization, tissue samples were blocked with superblock and 3% peroxidase block solutions before being cooked under pressure in a 10% citrate-buffered solution to expose antigens. At 4 °C, samples were incubated with anti-IL-37b (ab153889, rabbit polyclonal antibody, 1:200; Abcam) and anti-CD4 (ab25804, mouse monoclonal antibody, 1:200; Abcam) antibodies. Following primary antibody incubation, samples were biotinylated before secondary antibody incubations were carried out. The streptavidin conjugate Qdot R 525 (Q10141MP, Life Technologies, Eugene, OR, USA) was used to measure green fluorescence. Strep avidin R phycoerythrin conjugate (S-3402, Sigma-Aldrich, St. Louis, MO, USA) was incubated for an hour at a dilution of 1:200 to produce red fluorescence. Hoechst 33342 (Thermo Scientific, 62249) was in-

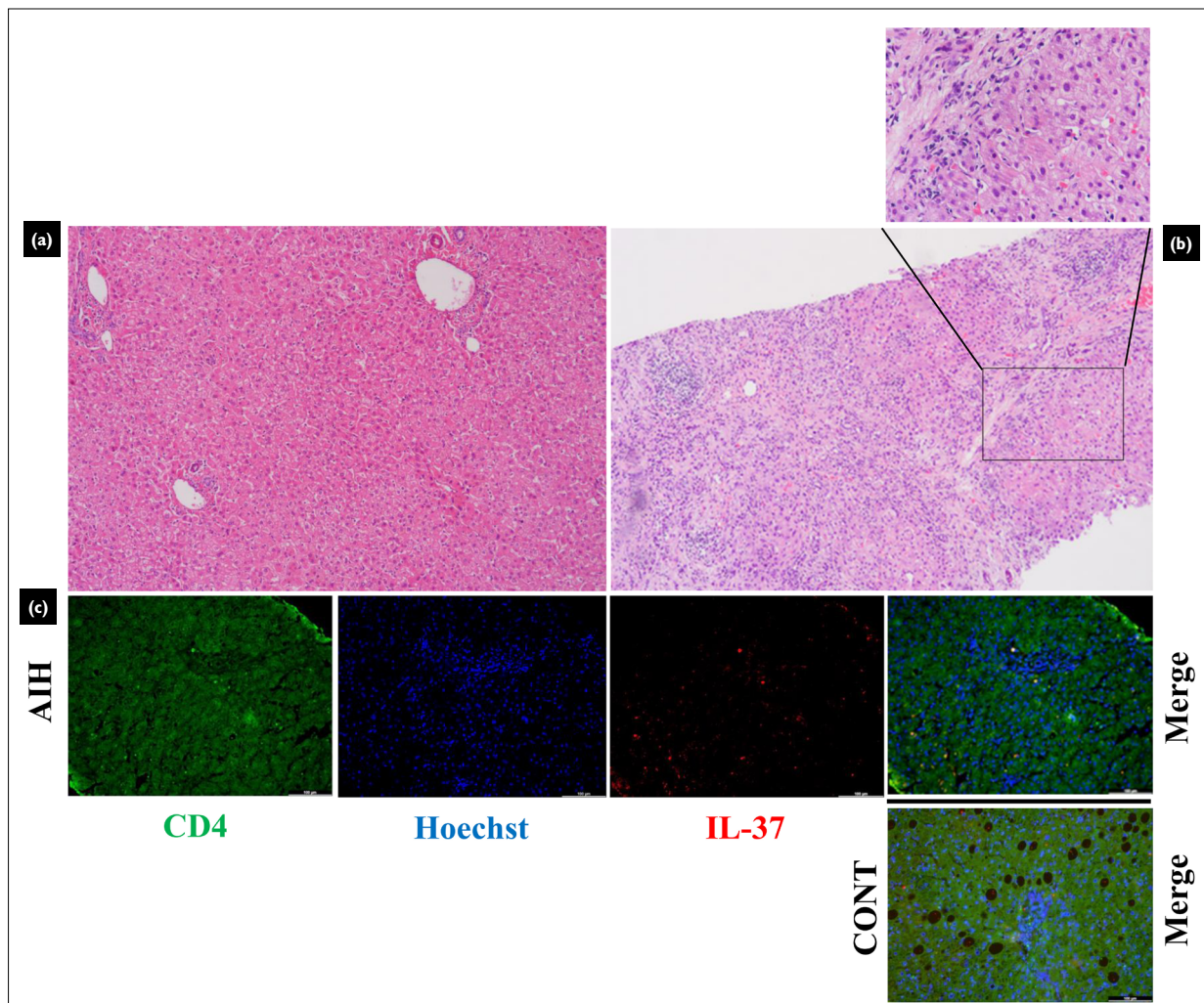


Figure 2. Histopathology of normal liver and AIH and immunofluorescent double staining of IL-37-producing helper T cells. **(a)** Normal liver (H&E; original magnification 10x). **(b)** Mononuclear inflammatory cells consisting of plasma cells, interphase hepatitis, and focal inflammation in the parenchyma were visible in the portal areas (H&E; original magnification 10x). Plasma cells in the portal area, periportal hepatocyte rosette formation, and emperipolesis were observed in the parenchyma. (Right upper corner image) (H&E; original magnification 40x). **(c)** Immunofluorescence double staining of IL-37/CD4 in AIH and CONT (Original magnification was 20x). AIH: CD4 (green); IL-37 (red); Hoechst (blue); Merge (yellow) (IL-37⁺CD4⁺ helper T cells signed in yellow arrow). Control: Merge (yellow). AIH for autoimmune hepatitis, CONT for control.

dl; AST, 287.1–318.6 U/l; ALT, 402.5–493.3 U/l; alkaline phosphatase (ALP), 152.1–99.4 U/l; gamma-glutamyl transferase (GGT), 123.9–95.6 U/l; and albumin, 21.84–19.68 g/dl. The grading of necroinflammatory activity was mild (8/20, 40%), moderate (9/20, 45%), and severe (3/20, 15%) according to the results of the Ishak grading and staging method. F0 (2/20, 10%), F1 (9/20, 45%), F2 (5/20, 25%), F3 (0/20, 00.0%), F4 (0/20, 00.0%), and F5 (4/20, 20%) were the stages of fibrosis. In control, the male/female ratio was 7/10, and the mean age at diagnosis was 55.6 years.

Histopathological findings

In the normal control liver, no evident inflammation was found (Fig. 2A). In AIH patients, portal inflammation was observed to be mostly constituted of lymphocytes with

varying amounts of plasma cells (Fig. 2B). Plasma cell clusters were seen. The lobular alteration, which varied from patchy to confluent necrosis, was mostly caused by necrotic-inflammatory damage. Confluent necrosis and inflammation were visible in the perivenular region. It was connected to the typical peri-portal inflammation of the portal area. In several instances, bridging necrosis was also seen, which suggests that interphase hepatitis deeply penetrated the lobules.

The number of IL-37⁺CD4⁺ helper T cells increased in the AIH

In samples from people with AIH and healthy controls, IL-37⁺CD4⁺ helper T cells were stained with anti-CD4 and anti-IL-37 antibodies (Fig. 2C). In all AIH biopsies, the

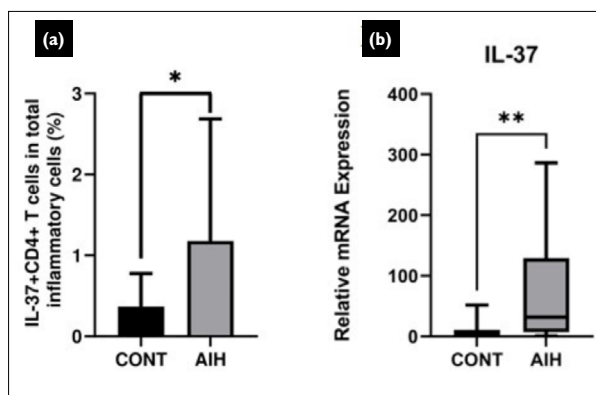


Figure 3. Proportion of IL-37-producing tissue-derived helper T cells in AIH versus relative mRNA expression of IL-37 in AIH. **(a)** Comparison of AIH and CONT IL-37⁺CD4⁺ helper T cells. When compared to the CONT group, the AIH group showed a larger ratio of double-positive IL-37⁺CD4⁺ helper T cells (AIH n=20 vs. CONT n=17, *p<0.05). **(b)** AIH group's liver biopsy showed a significantly greater relative expression of IL-37 mRNA than the CONT group. (AIH n=20 vs. CONT n=15, **p<0.01). AIH stands for autoimmune hepatitis; CONT stands for control.

ratio of IL-37⁺CD4⁺ helper T cells in total inflammatory cells was calculated. Contrary to the healthy control, IL-37⁺CD4⁺ helper T cells were considerably higher in the AIH (Median 0.725, range 0.474-1.884) vs. Control (Median 0.410, range 0.156-0.578), p=0.033) (Fig. 3A).

In individuals with AIH, the relative mRNA expression of IL-37 increased

By using qRT-PCR, we assessed the levels of IL-37 gene expression in the AIH biopsies of patients who had been diagnosed with the disease, as well as in control biopsy samples to look into the potential function of IL-37 in AIH. Relative IL-37 mRNA expression in AIH was (Median 31.98, range 0.8138-286.58) (n=20), while it was (Median 4.573, range 0.0249-51.91) (n=15) in controls. When compared to the healthy control, AIH patients had considerably higher levels of IL-37 expression compared to the healthy control (p=0.0068) (Fig. 3B).

Negative relationship between IL-37⁺CD4⁺ helper T cell count and severity of portal inflammation and Ishak scoring of AIH

In the Spearman rank coefficient analysis, IL-37⁺CD4⁺ helper T cell count was considerably negatively connected with the grading of inflammatory activity (r=-0.66, p=0.002) and negatively correlated with Ishak scoring (r=-0.51, p=0.026).

DISCUSSION

The investigation's findings provide insight into the pathophysiology of AIH and the function of Th cells that release IL-37. AIH patients had a much-increased ratio of IL-37⁺CD4⁺ helper T cells, which suggests that these cells

may play a role in the disease-related immune response. This result was in line with previous studies that proposed the genesis and regulation of certain autoimmune diseases were mediated by immune cells that produced IL-37.^[11] The idea that IL-37 could play a crucial role in regulating the inflammatory response in AIH was further reinforced by the fact that AIH patients had higher levels of IL-37 expression. The intriguing finding was that IL-37⁺CD4⁺ helper T cells were inversely connected to the degree of the disease. It suggested that in addition to its development, IL-37 might also play a reversal role in the onset, course, and severity of AIH.

IL-37 has been shown to have anti-inflammatory effects by blocking the action of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF- α), interleukin-6 (IL-6), and interferon-gamma (IFN- γ).^[10] IL-37 suppresses the sustained hepatic IFN- γ /TNF- α production and T cell-dependent liver injury.^[19] Immune cell infiltration and fibrosis in pediatric autoimmune liver diseases are correlated with IL-37 expression.^[14] IL-37 likely acts as a negative immune response regulator, inhibiting the inflammatory cascade and promoting immunological tolerance in AIH. IL-37⁺CD4⁺ helper T cells are significantly present in the liver tissue of AIH patients, indicating that these cells actively trigger a localized immune response within the hepatic milieu. Tissue-derived helper T cells have been implicated in the pathogenesis and development of tissue-specific inflammation in autoimmune diseases.^[5] The rise of IL-37⁺CD4⁺ helper T cells in the liver tissue suggested that the immune system was trying to balance the inflammatory milieu and return to immunological homeostasis. To precisely identify the mechanisms by which IL-37⁺CD4⁺ helper T cells are drawn to and activated in the liver tissue of AIH patients, additional investigation is necessary.

Through the regulation of the inflammatory response and the restoration of immunological tolerance, strategies targeted at augmenting IL-37 expression or activity may have therapeutic implications for AIH. In preclinical models of various autoimmune disorders, therapeutic strategies that target IL-37 have demonstrated promise.^[5,12] Developing targeted medicines or interventions to increase IL-37 production or strengthen its anti-inflammatory effects is required to study the therapeutic potential of IL-37 modulation in AIH.

Limitations

This study had several limitations. First, due to its retrospective design, patients could not be followed or monitored in a standardized timeframe. Although all cases met the diagnostic criteria for AIH, histopathological findings varied in severity, ranging from mild to severe disease. The lack of synchrony in the timing of biochemical marker assessments further complicated data interpretation. Additionally, the sample size was smaller than anticipated. As this was a student-led project with limited funding, we were unable to perform certain immunofluorescent stains that could have provided further insights, such as

the Foxp3 regulatory T cells marker. In the future, we aim to conduct prospective studies with a larger cohort and more comprehensive data to better elucidate the pathogenesis of AIH.

CONCLUSION

Our research demonstrated that patients with autoimmune hepatitis have significantly higher levels of IL-37 gene expression and IL-37⁺CD4⁺ helper T cell proportions than healthy controls. Significantly, histological scoring and the degree of portal inflammation were adversely connected with the higher number of IL-37⁺CD4⁺ T cells, indicating a possible protective or regulatory function in the course of the disease. These results point to helper T cells that produce IL-37 as possible treatment targets for autoimmune hepatitis. To clarify their exact mechanism and therapeutic implications, more research is necessary.

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Ethics Committee Approval

The study was approved by the Ethical Committee of Kartal Dr. Lutfi Kırdar City Hospital, School of Medicine, University of Health Sciences (Date: 19.07.2023, Decision No: 2023/514/254/25).

Informed Consent

Retrospective study.

Peer-review

Externally peer-reviewed.

Data availability statement

The datasets used or analyzed during the current study are available from the corresponding author upon reasonable request.

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Authorship Contributions

Concept: S.T., L.I., N.B., G.Y., S.K., S.E., K.S., G.H.; Design: S.T., L.I., N.B., G.Y., S.K., S.E., K.S., G.H.; Supervision: S.E., S.K., G.H.; Materials: S.E., S.K., G.H.; Data collection &/or processing: S.E., S.K., G.H.; Analysis and/or interpretation: S.E., S.K., G.H., G.Y.; Literature search: S.T., L.I., N.B., G.Y., S.K., S.E., K.S., G.H.; Writing: G.H.; Critical review: S.T., L.I., N.B., G.Y., S.K., S.E., K.S., G.H.

Conflict of Interest

None declared.

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Otoimmün Hepatitte IL-37⁺CD4⁺ Yardımcı T Hücrelerinin Potansiyel İmmünomodülatör Rolü

Amaç: Otoimmün hepatit (AIH), kökeni bilinmeyen inflamasyonla kendini gösteren kronik bir karaciğer hastalığıdır. Kanda yüksek immüno-globulin G (IgG) ve otoantikör seviyeleri ile karakterizedir. Patolojik olarak AIH, özellikle plazma hücreleri olmak üzere inflamatuvar hücrelerin infiltrasyonu ve arayüz hepatiti belirtileri gösterir. İnterlökin (IL)-37 üreten belirli bir CD4⁺ yardımcı T hücresi tipinin otoimmün hastalıklardaki rolü belirlenmiştir. Ancak, AIH'deki rolleri henüz tam olarak anlaşılammıştır. Bu konuya ışık tutmak için çalışmamız, AIH hastalarının karaciğer biyopsilerinde IL-37 gen ve protein ekspresyonunu araştırmayı ve dokuda yerleşik IL-37 üreten yardımcı T hücrelerinin AIH gelişimindeki rolünü belirlemeyi amaçlamıştır.

Gereç ve Yöntem: AIH tanısı almış 20 hasta ve kontrol grubu olarak 17 sağlıklı bireyi içeren karşılaştırmalı bir çalışma yürüttük. IL-37 gen ekspresyonu, kantitatif gerçek zamanlı polimeraz zincir reaksiyonu (qRT-PCR) kullanılarak ölçüldü ve karaciğer dokusunda IL-37⁺CD4⁺ yardımcı T hücrelerini tanımlamak için immünofloresan çift boyama yapıldı. AIH hastalarında IL-37⁺CD4⁺ yardımcı T hücrelerinin toplam inflamatuvar hücreler arasındaki oranı hesaplanarak sayımları yapıldı.

Bulgular: Bulgularımız, AIH hastalarında IL-37⁺CD4⁺ yardımcı T hücrelerinin oranında anlamlı bir artış olduğunu ortaya koydu ($p<0.05$). Ek olarak, IL-37 gen ekspresyonu AIH hastalarında artmıştır ($p<0.01$). Daha da önemlisi, IL-37+CD4+ yardımcı T hücrelerinin oranı, portal inflamasyonun şiddeti ve Ishak skorlaması ile negatif korelasyon göstermiştir.

Sonuç: IL-37 üreten yardımcı T hücrelerinin otoimmün hepatit için potansiyel tedavi hedefleri olabileceğini vurgulamaktadır. Kesin mekanizmalarını ve tedavi edici etkilerini aydınlatmak için daha fazla çalışmaya ihtiyaç vardır.

Anahtar Sözcükler: Otoimmün hepatit; IL-37; yardımcı T hücresi; Ishak skorlaması.