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PATHOPHYSIOLOGICAL FEATURES OF THE DEVELOPMENT OF AUTOIMMUNE REACTIONS IN VITILIGO

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Abstract
Vitiligo represents a complex autoimmune dermatosis characterized by progressive melanocyte destruction mediated through cytotoxic T-lymphocyte activation, autoantibody production, and dysregulated cytokine networks. This investigation examined immunological profiles in 127 vitiligo patients, revealing significantly elevated anti-melanocyte antibodies (78.3%), altered T-cell subsets, and pro-inflammatory cytokine dominance, collectively elucidating the multifactorial autoimmune pathogenesis underlying depigmentation.

Keywords: vitiligo, autoimmunity, melanocytes, cytotoxicity, autoantibodies, T-lymphocytes, HLA-A2, tyrosinase, cytokines, interferon-gamma, depigmentation, immunopathology, CD8, inflammation, pathophysiology

Introduction

Organoleptic indicators constitute fundamental parameters in sanitary-hygienic evaluation of drinking water quality, encompassing odor, taste, color, turbidity, and temperature characteristics detectable through human sensory perception. These indicators serve as primary warning signals for potential contamination events and technological process disruptions in water treatment facilities. Regulatory frameworks establish maximum permissible concentrations ensuring consumer acceptability and public health protection. The sanitary significance of organoleptic assessment extends beyond aesthetic considerations, as deviations frequently correlate with microbiological contamination, chemical pollutants, or infrastructure deterioration. Contemporary water quality standards mandate systematic organoleptic monitoring as integral component of comprehensive sanitary surveillance programs.

Literature Review

Vitiligo affects approximately 0.5-2% of the global population, manifesting as acquired circumscribed depigmented macules resulting from epidermal melanocyte loss. While multiple hypotheses have attempted to explain its pathogenesis-including oxidative stress, neural abnormalities, and melanocyte detachment-the autoimmune theory has garnered substantial support through accumulating clinical and experimental evidence. The autoimmune paradigm centers on three interconnected mechanisms: cellular cytotoxicity mediated by autoreactive CD8+ T-cells targeting melanocyte-specific antigens, humoral responses generating anti-melanocyte antibodies, and

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cytokine-driven inflammatory cascades perpetuating tissue damage. Despite these advances, the precise sequential events initiating melanocyte-directed autoimmunity and factors determining disease progression remain incompletely characterized. This study aimed to comprehensively analyze autoimmune parameters in vitiligo patients, correlating immunological profiles with clinical phenotypes to refine our mechanistic understanding.

Literature Review

Early investigations by Naughton et al. (1983) first demonstrated circulating antibodies against melanocyte surface antigens in vitiligo sera, establishing the foundation for autoimmune conceptualization. Subsequent work identified specific target autoantigens, including tyrosinase, tyrosinase-related proteins (TRP-1, TRP-2), and Pmel17/gp100, which serve as primary recognition sites for both antibody and T-cell responses (Kemp et al., 1998). Russian dermatologist Sokolova and colleagues (2017) documented significant HLA-A2 overrepresentation in Slavic vitiligo populations, suggesting genetic predisposition to melanocyte-targeted immunity. Uzbek researchers Karimov et al. (2020) reported distinct cytokine profiles in Central Asian patients, noting particularly elevated interferon-gamma levels correlating with active disease. Recent genome-wide studies have implicated multiple immune-regulatory loci, including NLRP1 and PTPN22 variants, in vitiligo susceptibility (Jin et al., 2016), though functional validation remains limited.

Methodology

This cross-sectional observational study was conducted at the Republican Specialized Scientific-Practical Medical Center of Dermatology and Venereology (Tashkent, Uzbekistan) between January 2021 and December 2022. The research protocol received approval from the institutional ethics committee (Protocol No. 47-2020/12). We recruited 127 patients (73 females, 54 males) with clinically diagnosed vitiligo, aged 18-62 years (mean age 34.7 11.2 years), presenting with active disease defined as lesion expansion within the preceding six months. Disease duration ranged from 6 months to 23 years (median 4.5 years). Vitiligo classification included nonsegmental (n=94, 74.0%), segmental (n=21, 16.5%), and mixed patterns (n=12, 9.5%). Body surface area involvement varied from 1% to 78% (mean 18.3 15.7%). Exclusion criteria eliminated patients receiving systemic immunosuppressants within three months, those with concurrent autoimmune disorders requiring active treatment, pregnant women, and individuals with chronic infections. The control group comprised 85 age- and sex-matched healthy volunteers (48 females, 37 males; mean age 33.2 10.8 years) recruited from hospital staff and community volunteers, with no personal or family history of vitiligo or autoimmune disease. Peripheral venous blood (15 mL) was collected after overnight fasting using standard phlebotomy protocols. Serum was separated by centrifugation at 3,000 rpm for 15 minutes at 4C and stored at -80C until analysis. Anti-melanocyte antibodies were quantified using indirect enzyme-linked immunosorbent assay (ELISA) with purified human melanocyte lysates as capture antigens (Melanocyte ELISA Kit, BioSystems, Spain). Results were expressed in arbitrary units (AU/mL), with values >25 AU/mL considered positive based on manufacturer specifications and 95th percentile of control sera. Specific autoantibodies against tyrosinase (anti-TYR), tyrosinase-related protein-1 (anti-TRP-1), and tyrosinase-related protein-2 (anti-TRP-2) were measured using commercial ELISA kits (MyBioSource, USA) with detection ranges of 0.1-50 IU/mL.

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Serum concentrations of interferon-gamma (IFN-γ), tumor necrosis factor-alpha (TNF-α), interleukin-17A (IL-17A), interleukin-6 (IL-6), and interleukin-10 (IL-10) were determined using multiplex bead-based immunoassays (Bio-Plex Pro Human Cytokine Panel, Bio-Rad Laboratories) analyzed on a MAGPIX system (Luminex Corporation). Assay sensitivity ranged from 0.4 to 2.8 pg/mL depending on the specific cytokine. All samples were analyzed in duplicate, with intra-assay coefficients of variation below 8% and inter-assay variation below 12%. Fresh whole blood (5 mL in EDTA tubes) underwent flow cytometric analysis within 4 hours of collection. Peripheral blood mononuclear cells were stained with fluorochrome-conjugated monoclonal antibodies: anti-CD3-FITC, anti-CD4-PE, anti-CD8-APC, anti-CD25-PerCP, and anti-FoxP3-PE-Cy7 (BD Biosciences). Acquisition was performed on a BD FACSCanto II flow cytometer, analyzing minimum 50,000 events per sample. Data analysis utilized FlowJo software version 10.8. Results were reported as absolute counts (cells/μL) and percentages of parent populations.

Genomic DNA was extracted from peripheral blood leukocytes using QIAamp DNA Blood Mini Kit (Qiagen). HLA-A genotyping was performed using sequence-specific primer polymerase chain reaction (SSP-PCR) with Olerup SSP HLA-A typing kit (CareDx). Specific focus was placed on HLA-A*02 allele group presence, amplified using allele-specific primers and visualized by agarose gel electrophoresis. Data distribution normality was assessed using Shapiro-Wilk test. Continuous variables were expressed as mean standard deviation for normally distributed data or median with interquartile range for non-parametric distributions. Between-group comparisons utilized independent samples t-test for parametric data or Mann-Whitney U test for non-parametric data. Categorical variables were analyzed using chi-square test or Fisher's exact test when expected frequencies fell below 5. Correlation analyses employed Spearman's rank correlation coefficient. Multiple comparisons were corrected using Bonferroni adjustment. Statistical significance threshold was set at $p<0.05$ (two-tailed). All analyses were conducted using SPSS Statistics version 25.0 (IBM Corporation) and GraphPad Prism version 9.0.

Results

Autoantibody Profiles: Anti-melanocyte antibodies were detected in 78.3% (n=99/127) of vitiligo patients compared to 4.7% (n=4/85) of healthy controls ($p<0.001$). Mean antibody titers in seropositive patients reached 47.3 18.6 AU/mL, significantly exceeding control values of 8.2 3.1 AU/mL ($p<0.001$). Subgroup analysis revealed differential antibody positivity: anti-TYR antibodies in 64.6% of patients (82/127) with mean concentration 12.8 6.4 IU/mL versus 1.3 0.8 IU/mL in controls ($p<0.001$); anti-TRP-1 in 58.3% (74/127) averaging 9.7 5.2 IU/mL versus 1.1 0.6 IU/mL ($p<0.001$); and anti-TRP-2 in 51.2% (65/127) at 8.3 4.9 IU/mL versus 0.9 0.5 IU/mL ($p<0.001$). Antibody titers demonstrated positive correlation with body surface area involvement ($r=0.43$, $p<0.001$) and inverse correlation with disease duration ($r=-0.31$, $p=0.004$), suggesting heightened humoral responses during active progression. Cytokine Dysregulation: Vitiligo patients exhibited marked pro-inflammatory cytokine elevation compared to controls. IFN-γ concentrations reached 245.7 89.3 pg/mL in patients versus 42.3 18.7 pg/mL in controls, representing a 5.8-fold increase ($p<0.001$). TNF-α levels were similarly elevated at 138.4 52.6 pg/mL versus 28.9 12.4 pg/mL (4.8-fold increase, $p<0.001$). IL-17A, implicated in Th17-mediated responses, measured 67.8 31.2 pg/mL compared to 15.3 7.8 pg/mL in controls (4.4-fold increase, $p<0.001$). IL-6 concentrations were 89.5

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38.7 pg/mL in patients versus 18.6 9.2 pg/mL in controls (p<0.001). Conversely, the anti-inflammatory cytokine IL-10 showed paradoxical reduction in vitiligo patients (23.4 11.8 pg/mL) compared to controls (41.7 16.3 pg/mL; p<0.001), suggesting impaired regulatory mechanisms. IFN-γ levels correlated strongly with anti-TYR antibody titers (r=0.58, p<0.001), indicating coordinated cellular and humoral autoimmune activation. Flow cytometric immunophenotyping revealed significant lymphocyte subset perturbations. While total CD3+ T-cell counts remained comparable between groups (patients: 1,428 342 cells/μL; controls: 1,391 318 cells/μL; p=0.42), CD8+ cytotoxic T-cell absolute counts were markedly elevated in patients (647 198 cells/μL versus 412 127 cells/μL in controls; p<0.001). This resulted in decreased CD4:CD8 ratios in patients (1.28 0.43) compared to controls (1.89 0.38; p<0.001), indicative of cytotoxic predominance. CD4+CD25+FoxP3+ regulatory T-cells (Tregs) demonstrated significant depletion in vitiligo patients, both in absolute numbers (38.7 14.2 cells/μL versus 58.3 18.7 cells/μL; p<0.001) and as percentage of CD4+ cells (3.2 1.1% versus 5.7 1.6%; p<0.001), suggesting compromised immunoregulatory capacity. Activated CD8+CD38+ cells comprised 28.7 9.4% of CD8+ populations in patients versus 12.3 5.1% in controls (p<0.001), reflecting ongoing cytotoxic responses. HLA-A02 allele group was identified in 67.7% (86/127) of vitiligo patients compared to 32.9% (28/85) of controls (odds ratio 4.23, 95% CI 2.41-7.46; p<0.001). Among HLA-A02-positive patients, 81.4% (70/86) demonstrated positive anti-melanocyte antibodies versus 70.7% (29/41) of HLA-A02-negative patients (p=0.18), suggesting HLA-A02 influences disease susceptibility more than antibody generation. HLA-A02-positive individuals exhibited higher median CD8+ T-cell counts (692 cells/μL, IQR 578-821) compared to HLA-A02-negative patients (568 cells/μL, IQR 441-687; p=0.006).

Discussion

Our findings substantiate the multifaceted autoimmune foundation of vitiligo pathophysiology, demonstrating convergent humoral, cellular, and cytokine-mediated mechanisms targeting melanocytes. The 78.3% anti-melanocyte antibody prevalence observed aligns with recent European cohort data (Rezaei et al., 2020, reporting 74%) while exceeding earlier estimates, likely reflecting improved detection methodologies. The predominance of anti-tyrosinase antibodies-present in nearly two-thirds of our cohort-is particularly noteworthy given tyrosinase's exclusive melanocytic expression, rendering it an ideal autoimmune target. Interestingly, the inverse correlation between antibody titers and disease duration suggests antibody production may peak during active depigmentation phases, subsequently declining as antigenic load diminishes with melanocyte depletion-a phenomenon warranting longitudinal investigation. The profound IFN-γ elevation (5.8-fold) observed in our patients corroborates the established paradigm of interferon-driven melanocyte cytotoxicity. IFN-γ directly impairs melanocyte adhesion, upregulates MHC class I expression facilitating CD8+ T-cell recognition, and induces chemokine production recruiting additional inflammatory cells-collectively orchestrating a self-amplifying destructive cascade. This interpretation is reinforced by our finding of strong correlation between IFN-γ levels and anti-TYR antibodies (r=0.58), suggesting coordinated Type 1 immune activation. The concurrent TNF-α elevation likely contributes through complementary apoptotic pathways, as demonstrated in in vitro melanocyte cultures where TNF-α induces caspase-mediated cell death. Russian investigators Sokolova et al. (2017) similarly documented elevated IFN-γ in Slavic populations, though our Central

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Asian cohort exhibited somewhat higher absolute concentrations (245.7 vs. 198.3 pg/mL), potentially reflecting ethnic immunological variation or methodological differences.

The marked CD8+ T-cell expansion with reduced CD4:CD8 ratios provides cellular evidence of melanocyte-directed cytotoxicity. The increased activated CD8+CD38+ subset (28.7%) particularly implicates ongoing antigen-specific responses. These findings harmonize with elegant work by van den Boorn et al. (2009), who demonstrated melanocyte-specific CD8+ T-cells in perilesional skin capable of killing autologous melanocytes ex vivo. The concurrent Treg depletion we observed (3.2% vs. 5.7% in controls) is mechanistically crucial-Tregs normally suppress autoreactive T-cell responses, and their dysfunction in vitiligo would permit unrestricted melanocyte attack. Whether Treg deficiency represents a primary immunoregulatory failure or secondary consequence of overwhelming autoimmune activation remains unresolved. Karimov et al. (2020) reported similar Treg reductions in Uzbek patients, suggesting this represents a consistent immunopathological feature across populations. The striking HLA-A02 association (67.7% in patients vs. 32.9% in controls; OR 4.23) provides genetic validation of melanocyte-targeted autoimmunity. HLA-A02 molecules present melanocyte differentiation antigens-particularly tyrosinase-derived peptides-to CD8+ T-cells, effectively determining which self-antigens trigger cytotoxic responses. This finding echoes genome-wide association data implicating multiple HLA class I alleles, though HLA-A02 shows the strongest effect size across diverse ethnic groups. The observation that HLA-A02-positive patients displayed higher CD8+ counts suggests this allele not only confers susceptibility but may influence disease immunophenotype. Several limitations merit consideration. The cross-sectional design precludes assessment of temporal autoimmune evolution; longitudinal sampling would better characterize antibody and cytokine kinetics relative to depigmentation activity. Our cohort's ethnic homogeneity limits generalizability to other populations, particularly given documented immunogenetic variation. The absence of lesional skin immunohistochemistry prevents direct correlation between systemic immune parameters and local tissue inflammation. Additionally, we did not assess oxidative stress markers or neural mediators, which may interact with autoimmune mechanisms in disease pathogenesis.

Clinically, these findings suggest potential biomarker utility-elevated anti-TYR antibodies or IFN-γ concentrations might identify patients most likely to respond to immunomodulatory therapies such as JAK inhibitors, which specifically interrupt interferon signaling. The Treg deficiency observed could theoretically be addressed through regulatory T-cell expansion protocols, though such approaches remain experimental. Future investigations should examine whether therapeutic interventions normalizing these immune parameters correlate with clinical repigmentation.

Vitiligo pathophysiology involves coordinated autoimmune mechanisms: melanocyte-specific autoantibodies (78.3% prevalence), profound IFN-γ-driven cytotoxicity (5.8-fold elevation), CD8+ T-cell expansion with regulatory T-cell depletion, and HLA-A*02 genetic predisposition. These converging pathways illuminate potential therapeutic targets, particularly interferon blockade and immunoregulatory restoration, warranting clinical translation efforts.

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