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RESEARCH ARTICLE

Study of Serum Interleukin-33 and Soluble ST2 Levels, and HLA-B27 Typing in Iraqi Patients of Ankylosing Spondylitis

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ABSTRACT

Ankylosing spondylitis (AS) is a type of arthritis that causes inflammation in the joints and ligaments of the spine. The current study aimed to determine the level of serum interleukin-33 (IL-33), soluble stromelysin-2 (sST2) interleukin, and HLA-B27 typing among Iraqi AS patients. The whole blood samples of the patients were collected from medical hospitals in Baghdad city. The ELISA technique was used to measure the serum levels of IL-33 and sST2, and the HLA-B27 typing was performed by sequence-specific primer PCR (SSP-PCR). The patients were divided into patients newly diagnosed with the disease and patients receiving biological treatment (etanercept) in addition to the healthy control individuals. The mean serum IL-33 levels for newly diagnosed patients, patients treated with etanercept, and healthy control individuals were (82.84 ± 3.59), (55.91 ± 2.11), (37.32 ± 1.69) respectively. The mean serum sST2 levels for newly diagnosed patients, patients treated with etanercept, and healthy control individuals were (165.62 ± 8.92), (78.82 ± 5.84), (48.01 ± 2.11) respectively. In addition, the positive results of HLA-B27 for newly diagnosed patients, patients treated with etanercept, and healthy control were n = 18 (60.0%), n = 22 (73.3%), n = 0 (0.00%), respectively. In conclusion the present research confirmed the significance of interleukin-33 (IL-33), sST2, and HLA-B27 allele as a rapid marker for diagnosis of AS also in Iraqi As patients.

Keywords: Ankylosing spondylitis, HLA-B27, IL-33, Iraqi patients, sST2

Introduction

Ankylosing spondylitis (AS) is a chronic inflammatory disease characterised by a gradual fusion of the axial joints,[1] considered a type of spondyloarthritis (SpA).[2] Numerous disciplines, including genetics, environment, gut microbiota, and hormones, have previously been studied in relation to the etiology of AS.[3] It is believed that the prevalence rates of AS range from 0.1 percent to 0.3 percent around the world. The disease affects approximately 1.3 million to 1.6 million individuals in Europe and 4.6 million to 5.0 million patients in Asia.[4] The first anatomical description is based on a patient's skeleton with advanced AS. However, it was first published in 1695 by Bernard Connor.[5] The inflammation frequently results in calcification and bone growth, as well as damaging bone lesions, which ultimately lead to spinal fusion, a loss of flexibility, and persistent back pain. For the past ten years, there has been a substantial amount of progress made in both the understanding of the mechanism behind this autoimmune disease and the development of a treatment for inflammation.[2]

The development of inflammatory arthritis is dependent upon the presence of an unbalanced cytokine network.[6,7] Th2 cells are responsible for the production of a cytokine known as interleukin (IL)-33, which functions as a chemoattractant for human Th2 cells. The production of IL-33 by mast cells occurs after they

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have been stimulated by immunoglobulin IgE.[8] The AS disease is significantly affected by IL-33, which is involved in rheumatoid arthritis. In addition to this, there has been little research done on the implications of alterations in IL-33 levels on AS.[9]

Human stromelysin-2 (ST2) is also known as IL-1 receptor-like 1 (IL1RL-1).[10] IL1RL-1 functions as a decoy receptor and sequesters free IL-33. The activation of the nuclear factor kappa B (NF-κB) signaling pathway is responsible for the enhancement of mast cell, Th2 cell, regulatory T cell (Treg), and innate lymphoid cell type 2 activities.[11]

One of the most important aspects of the World Health Organization (WHO) diagnostic criteria for axial SpA (axSpA) is the human leukocyte antigen B27 (HLA-B27), which is classified as an MHC class I marker.[12] Peptide antigens are presented to CD8 T lymphocytes by HLA-B27, which is a member of the HLA Class I family of MHC genes. Its assignment is to perform this function.[13] Across the many continents and racial and cultural groupings that make up the world, there is a vast variety of HLA B27 prevalence.[14] The HLA-B27 was first described in 1973, and the scientific community continues to confront a major challenge in determining how it contributes to SpA and other conditions associated with SpA. There is a growing interest in HLA-B27–based effects, particularly in HLA-B27(+) patients who have AS, although the diagnostic and prognostic functions of HLA-B27 in AS are still being contested.[15] Additionally, patients with HLA-B27 positivity have a considerably younger age at onset, a greater number of cases of uveitis, and a higher frequency of involvement of peripheral joints and hips compared to patients with HLA-B27 negative.[16] The present study aimed to identify the serum IL-33 and soluble ST2 (sST2) levels correlated with HLA-B27 in AS.

Patients and methods

Study design

The case-control study was carried out from December 2022 to April 2023 at the main medical hospitals in Baghdad in the rheumatology department of the Baghdad Teaching Hospital. A total of 243 samples were taken from individuals between the ages of 17 and 66. They were divided into three groups: newly diagnosed patients infected with AS without treatment, AS patients treated with etanercept, and healthy control individuals. Clinical evidence is consistent with an AS diagnosis, as determined by the Assessment of SpA International Society (ASAS).[17] The AS patients included in the study were selected as treated with biological DMARDs: etanercept and new diagnosis, some of these patients have other diseases like hypertension and diabetes mellitus. At the same time, the history of cancer, the presence of other severe acute or chronic medical conditions, pregnancy and breastfeeding, alcohol consumption, hepatotropic viral infections, human immunodeficiency virus infections, other autoimmune disorders, and an unwillingness or inability to cooperate were among the categories that were excluded from the study.

Ethical approval

The study secured ethical approval from the Iraqi Ministry of Health-Department of Medical Teaching City (Reference No. 53131, dated December 15, 2022). The treating rheumatologist referred eligible patients with the patient's consent.

Sample collection

Venous blood specimens (10 ml) were collected as follows: 8 ml for immunological and serological analysis in gel and EDTA tubes and 2 ml for genetic tests in EDTA tubes. The gel tubes were centrifuged for 10 min at 2683.2 xg. The serum sample was carried to add into eppendorf tubes and immediately frozen at –20 °C.

Immunological investigation

The estimation of IL-33 and sST2 in human sera was performed by sandwich enzyme-linked immunesorbent assay technology using kits of (Sunlong company, China).

Examination of inflammatory markers

The inflammatory characteristics of the disease, like blood erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were performed by Westergren method and (LTA C reactive protein company, Italy), respectively.

Genetic detection

DNA extraction and quantification

Genomic DNA was extracted according to the protocol quality of the (gSYNC™ DNA extraction, China) Kit. The purified DNA is quantified with analysis by gel electrophoresis and ultraviolet light. DNA purity and concentration were measured using the NanoDrop method. To confirm the extraction of genomic DNA, a gel electrophoresis procedure was used.[18]

Detection of HLA-B27 gene

For diagnosis of the gene of HLA-B27, we used the (Olerup SSP® HLA-B*27, Sweden). The content of the HLA-B*27 SSP typing was the primer set containing two vials of 5'- and 3'-primers for identifying the HLA-B27 specificity, B*27:01 to B*27:256. The primer solutions consist of specific primer mixes, i.e., group-specific primers, as well as a control primer pair matching non-allelic sequences Per sample, at room temperature in a 0.5 ml tube the following were added: $(2 \times 2)\ \mu l = 4\ \mu l$ DNA, $(2 \times 3)\ \mu l = 6\ \mu l$ PCR Master Mix complete with Taq – mixed well and $(2 \times 5)\ \mu l = 10\ \mu l$ dH_2O. The mixture was mixed well, then 10 μl of the DNA-PCR master mix was dispensed and blended into each of the 2 wells of an HLA-B27 typing. The reaction conditions were 1 cycle of 94 ˚C for 2 min, followed by 10 cycles of 94 ˚C for 10 s, 65 ˚C for 60 s; after that, 20 cycles of 94 ˚C for 10 s, 61 ˚C for 50 s and 72 ˚C for 30 s.

Gel electrophoresis

By melting 1.5 g of agarose in 100 ml of previously prepared TBE buffer, a 1.5% agarose gel was made. After being brought to a boil, agarose was allowed to cool at 45–50 ˚C. Following the preparation of the agarose support plate and the fixing of the comb to create holes for the samples, the gel was poured into the pour plate. After carefully pouring the gel to prevent air bubbles, it was allowed to cool down for a half-hour. From the solid agarose, the comb has been carefully removed. In the horizontal electrophoresis unit, which is represented by the electrophoresis tank, the plate has been fixed to its support as it has been secured. To cover the gel surface, TBE buffer has been added to the tank. To verify the genomic DNA extraction of all of the isolates that had clear bands, a gel electrophoresis procedure was carried out.

Statistical analysis

The data were examined for normality, homogeneity, and normal distribution, mean ± SE of mean, by using the IBM SPSS version 28.0. Tests were used to calculate the probability by ANOVA (Duncan test) for parametric data. For non-parametric data, Data were presented in simple measures of frequency percentage. The relationship between the variables study was determined using a Pearson's correlation. Furthermore, it represented a strong correlation between the studied groups under the biological treatment and Pain relievers of the three studied groups to parameters of the AS.

Table 1. Demographic characteristics of Iraqi AS patients.

Characteristics	AS Patients
Sex	
Males	158 (81.9%)
Female	35 (18.1%)
Age	
(17–26)	29 (15%)
(27–36)	67 (34.7%)
(37–46)	57 (29.5%)
(47–56)	36 (18.7%)
(57–66)	4 (2.1%)
Smoking	
Yes	89 (46.1%)
No	104 (53.9%)
BMI (Kg/m2)	
Underweight (Mild thinness)	3 (1.6%)
Normal range	48 (24.9%)
Overweight (Pre-obese)	68 (35.2%)
Obese (Class I)	53 (27.5%)
Obese (Class II)	12 (6.2%)
Obese (Class III)	9 (4.7%)
Medical history	
Non	180 (93.3%)
Reactive arthritis	7 (3.6%)
Psoriasis	5 (2.6%)
Crohn's disease	1 (0.5%)
Family history	
Non	124 (64.2%)
AS	46 (23.8%)
Reactive arthritis	15 (7.8%)
Psoriasis	7 (3.6%)
SLE	1 (0.5%)
COVID-19	
Not Infected	120 (62.2%)
Past infected	73 (37.8%)
COVID-19 Vaccine	
Non	92 (47.7%)
Pfizer	71 (36.8%)
Sinopharm	24 (12.4%)
AstraZeneca	6 (3.1%)
NSAIDs	
Yes	81 (42%)
No	112 (58%)
Analgesic	
Yes	30 (15.5%)
No	163 (84.5%)

Results and discussion

Inflammation of the axial spine, peripheral joints, and enthuses are the hallmarks of AS, a persistent form of immune-mediated arthritis. The AS disease is a significant problem in terms of both healthcare and the economy, as it is believed that one out of every two hundred persons is impacted by it.[19] Demographic and clinical features of 193 patients of AS were collected at the rheumatology unit of Baghdad Teaching Hospital were listed in Table 1. 158 (81.9%) of the study's participants were men, a significant proportion when compared to the number

of female participants during the data collection period. In comparison, the percentage of women was 35 (18.1%), and this agrees a previous study conducted in Baghdad by Salloom.[20] These outcomes are similar to those of Turkey's national registers,[21] where males constituted 76% of the study population. While the study was conducted on Chinese AS patients, the results did not agree with the current study, as the male infection rate was (58.7%).[22] The ethnic origin of the patients with AS that we observed in our study of the Iraqi population; this could account for the variation in disease severity across the sexes in other studies. AS occurs frequently in young and middle-aged adults, and our study shows the largest cohort of Iraqi AS patients (27–36) years, which constituted 34% younger than what was reported in previous studies (>25 years).[23] This might be because this study consists of a relatively high percentage of patients with an older age at onset (>27 years).

On the other hand smoking cigarettes is linked to a lot of different diseases, and healthcare professionals all over the world are very interested in this issue. It also affects both innate and adaptive defense. It can be dysregulating immunity in two ways: it can either make pathogen-fighting immune response hyperactivation or make protective immunity weaker.[24] Some studies show that smoking makes the disease more severe,[25] but the mechanism is still not clear.[26] The results for the patients were approximate between smokers and non-smokers (46.1 and 53.9, respectively). Low back pain and degenerative diseases of the spine are more common in people who smoke, which may be one of the bad effects of smoking on AS patients.[27] The study showed that smoking is not related to the disease, as the results showed that there is no relationship between smokers and non-smokers for people with AS compared to healthy people.[28] The widely held opinion is that halting smoking is a necessary first step in reducing the activity of the disease and that additional research is required.

Overweight patients made up 68 (35.2%) of the patient groups with the highest BMI percentage, conversely, the study's lowest percentage belonged to the underweight and obese Class III group and these outcomes accept with research done on Chinese patientsWhile the highest percentage of BMI for patient groups was overweight, which constituted 68 (35.2%), in contrast, underweight and obese Class III was the type with the lowest percentage in the study, and these results agree with a study conducted on Chinese patients,[29] this suggests that weight management, to maintain it at a normal level, should be one of the disease management strategies in patients with AS.

The medical history of AS patients constituted the largest percentage of patients without a history of immunological disease (93.3%), which indicates that there is no relationship between AS and other immune diseases. A positive family history was defined as having a first or second-degree relative with AS. The study showed that there is a significant statistical relationship between patients in terms of family history of AS. Another study reported a positive family history in 53% of cases.[30] Blood relationships may play a role in these results. Consanguineous marriage is a common feature of family systems in the Middle East, Central, South, and West Asia.[31]

The percentage of patients with AS who had previously had COVID-19 was 73 (37.8%), according to the cross-sectional study conducted at Baghdad Teaching Hospital in the Rheumatology Unit on Iraqi patients. The study proved that there is no relationship between disease AS and infection with disease COVID-19. As for the COVID-19 vaccine, the result for patients who did not receive the COVID-19 vaccine was 92 (47.7%). The study also proved that there is no relationship between the COVID-19 vaccine and AS. In another study, only one case of axSpA caused by SARS-CoV-2 infection has been stated.[32] Several cases of acute reactive arthritis have been reported in the recent literature, though, after the symptoms of COVID-19 disappeared.[33] Virus-induced arthritis is thought to be caused by several different processes that start when immune complexes enter and form in the joint areas. That excludes arthralgia and arthritis as possible causes.[34] The impact of COVID-19 on AS patients may come after some time, as well as the long period of receiving cortisone treatments. The impact of the coronavirus (COVID-19) on AS patients is being proved by further research and a larger number of patients than the current study.

Several laboratory markers, including CRP, were used to evaluate the severity of the disease were listed in Table 2. The comparison of the means of CRP among the three study groups revealed that the newly diagnosed group had a mean of 21.17 ± 5.03, the patients treated with the etanercept group had a mean of 7.60 ± 0.69, and the newly diagnosed group had a mean of 21.17 ± 5.03 compared to the control group, which had a mean of 4.60 ± 0.50. These findings were highly significant because the P-value was smaller than 0.01. The results of the current study appeared similar to Beyazal et al.[35] This increase in markers in AS patients suggests their potential use in monitoring AS. On the other hand, a comparison of the mean ESR values across the three groups revealed that the newly diagnosed group had a significantly higher ESR 25.83 ± 3.28 compared to the patients treated with the etanercept group 13.73 ± 1.83. The newly

Table 2. Estimation of the mean values of inflammatory markers in three study groups.

Parameter	New-diagnosed group	Etanercept group	Control group	P. Value
CRP mean ± SE (mg/dl)	21.17 ± 5.03	7.60 ± 0.69	4.60 ± 0.50	H.S
ESR mean ± SE (mm/hr)	25.83 ± 3.28	13.73 ± 1.83	4.60 ± 0.05	H.S

CRP: C-reactive protein, ESR: erythrocyte sedimentation. The quantity data are given as mean ± SE. P-value < 0.01: highly significant (H.S).

Table 3. Estimation of the mean values of parameters in individuals newly diagnosed and treated with etanercept for AS patients and the control group.

Parameter	New-diagnosed group	Etanercept group	Control group	P. Value
IL-33 mean ± SE (pg/ml)	82.84 ± 3.59	55.91 ± 2.11	37.32 ± 1.69	H.S
sST2 mean ± SE (pg/ml)	165.62 ± 8.92	78.82 ± 5.84	48.01 ± 2.11	H.S

IL-33: interleukin-33, sST2: soluble human stromelysin-2. The quantity data are given as mean ± SE. P-value < 0.01: highly significant (H.S).

diagnosed group had a significantly higher ESR 4.60 ± 0.05 compared to the control group, with highly significant results. These statistics agreed with the results conducted by Al Hafidh in a previous study for AS patients,[36] which proves that a high level of ESR is related to the severity of AS. Therefore, ESR and CRP are nowadays of important value for monitoring this disease. However, these are not satisfactory due to their low sensitivity and specificity.[37]

The mean serum IL-33 level in the AS patients with a new diagnosis of AS patients (82.84 ± 3.59) compared to those taking the etanercept drug (55.91 ± 2.11) were highly significantly different (P < 0.01). In addition, the mean IL-33 level in AS patients among newly diagnosed (82.84 ± 3.59) compared with the control group (37.32 ± 1.69) were highly significantly different were listed in Table 3. As for the difference between patients receiving the biological treatment (etanercept) and the control group, it was noted that the difference between the concentrations was slight, and this shows that the biological treatment has a positive effect on AS patients. The IL-33 levels are increased in the sera of AS patients when compared with healthy controls and positively correlate with disease activity.[9] The study reported by Li et al.[38] agrees with our result and shows a highly significant difference in IL-33 in patients with AS compared to the control group. While the study conducted by Ulusoy et al.[39] shows that there is no relationship in serum IL-33 levels between patients with AS and healthy controls, which disagrees with the results of the study we conducted. The reason for the difference between the results may be due to the nature of the geographical and genetic distribution between the studies. Variation in serum IL-33 levels can partly reflect disease activity and suggest that IL-33 may play an important role in the pathogenesis of AS. In our study, serum IL-33 levels were different between AS patients and healthy groups; that may be due to the small sample size in this study. Another possibility is that about half of our AS patients were receiving anti-TNF-α therapy (etanercept), so serum IL-33 levels may have decreased to near-normal levels. This assumption can be supported by published data indicating decreased serum IL-33 levels after anti-TNF treatment in psoriasis and ulcerative colitis, as well as in patients with rheumatoid arthritis.[40]

On another side, suppression of tumorigenicity 2 (ST2) is a member of the IL-1 receptor family, also known as IL1RL1 was an orphan receptor that was studied in the context of inflammatory and autoimmune disease.[41] Our result found that the mean serum sST2 level results were highly significant with newly diagnosed patients (165.62 ± 8.92) compared to patients treated with etanercept drug (78.82 ± 5.84) results were highly significantly different (P < 0.01). Also, the mean sST2 level in AS patients among newly diagnosed (165.62 ± 8.92) compared with the control group (48.01 ± 2.11) was highly significant. As for the difference between patients receiving the biological treatment (etanercept) and the control group, it was noted that the difference between the concentrations was slight, and this indicates that the etanercept drug has a positive effect on AS patients, as shown in Table 3. Our results found that the concentration of sST2 was measured in the three study groups, and it was observed that the concentration of sST2 was significantly higher in newly diagnosed patients compared to patients receiving biological therapy (etanercept) and also in the control group. As for the difference between patients receiving biological treatment (etanercept) and the control group, it was noted that the difference between the concentrations was slight, and this indicates that the etanercept drug has a positive effect on AS patients. The sST2 significantly increased in AS patients compared to the control group, similar

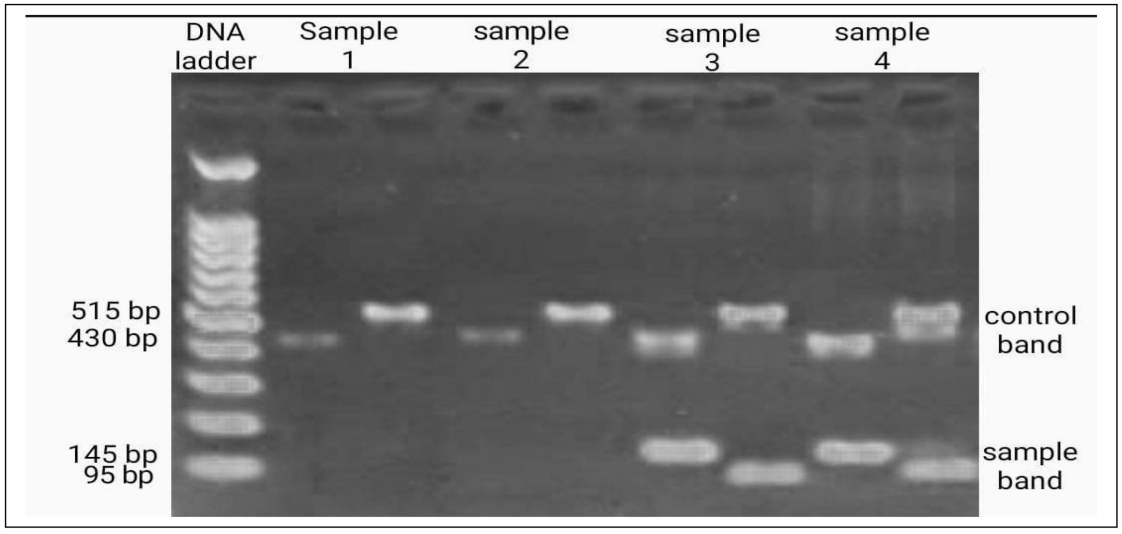

Fig. 1. Gel electrophoresis showing HLAB-27 specific band with a molecular ladder, positive control band 515bp and 430bp (all samples); negative sample (samples 1 and 2); positive sample (samples 3 and 4).

to the study conducted in 2013.[38] In contrast, there was another study conducted in Korea on patients with another immune disease, which is Behçet's disease, where the level of sST2 in the patients was higher than that of healthy control.[42] These studies showed that serum sST2 levels can partially reflect disease activity and may play an important role in the pathogenesis of AS. These findings indicate that IL-33 and sST2 play an important role in the pathogenesis of AS. Previous research found that levels of ST2 were much higher in people with AS compared to healthy people. These levels were also strongly linked to the Bath AS disease activity index. The sST2 values were linked to C-reactive protein, erythrocyte sedimentation rate, and HLAB-27. Higher amounts of sST2 were found in people with peripheral arthritis, and sST2 levels were higher in people with hip involvement.[38]

HLA-B27 allele showed an association with AS, and spondyloarthropathy plays a major role in disease pathogenesis and has an association with HLAB27 antigen in 90–95% of patients with SpA.[43] The presence of both 149bp and 95bp among newly diagnosed and specific bands, as well as 515bp and 430bp control bands on the 1.5% agarose gel, was indicative of the presence of a particular allele positive for human leukocyte antigen-B27 as in Fig. 1. The HLA-B27 was positive in 18 (60%) of newly diagnosed AS patients while negative in 12 (40%) compared to a control group as shown in Table 4. Current results do not agree with the Iranian study of an HLA-B27 prevalence of (73%),[44] which may be due to a more comprehensive sample size containing a wide ethnic background and geographic variation.

Indicative of the presence of human leukocyte antigen-B27 specific allele positive was the presence of both 145bp and 95bp among patients treated with etanercept, specific band, as well as 515bp and 430bp control band on the 1.5% agarose gel as in Fig. 2. The HLA-B27 was positive in 22 (73.3%) etanercept treatment with etanercept AS patients while negative in 8 (26.7%) compared to a control group as shown in Table 4. The finding is also similar to other various studies.[45,46]

A negative human leukocyte antigen-B27 specific allele was suggested by the absence of both 145bp and 95bp within the apparent control group, which consisted of 515bp and 430bp control bands on the 1.5% agarose gel as in Fig. 3. The HLA-B27 was positive in 0 (0.0%) while negative in 30 (100%) compared to the control group as shown in Table 4. In this study, the healthy subjects were found negative for the HLA-B27 allele in Iraqi patients. The current result does not agree with the Nessa et al.[47]

Table 4. The HLA-B27 number and percentage of newly diagnosed, treated with etanercept AS patients and healthy control groups.

HLA-B27	Newly diagnosis group	etanercept drug group	Healthy control group
Positive	18 (60.0%)	22 (73.3%)	0 (0.00%)
Negative	12 (40.0%)	8 (26.7%)	30 (100.0%)

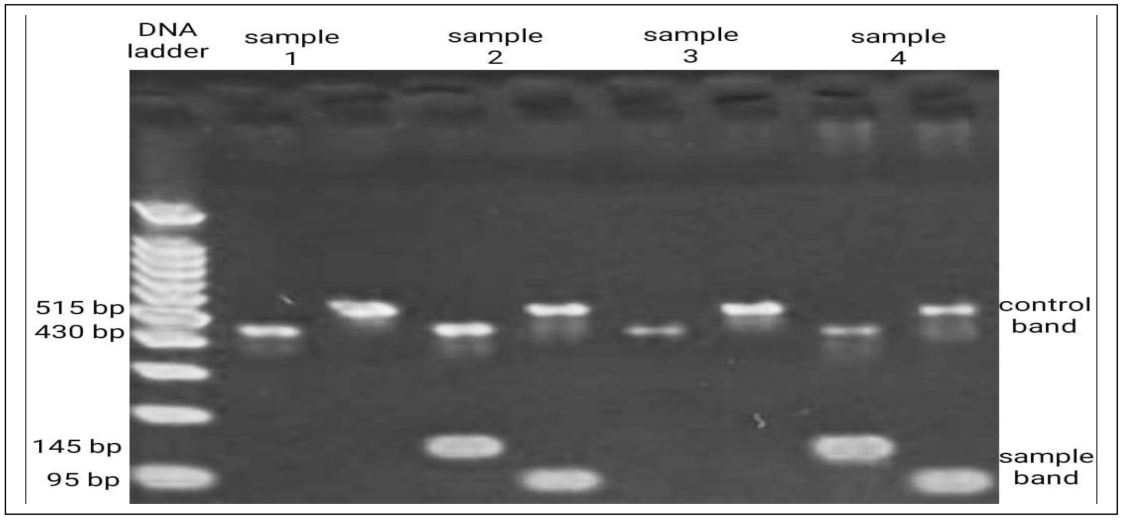

Fig. 2. Gel electrophoresis showing HLAB-27 specific band with a molecular ladder, positive control band 515bp and 430bp (all samples); negative sample (samples 1 and 3); positive sample (samples 2 and 4).

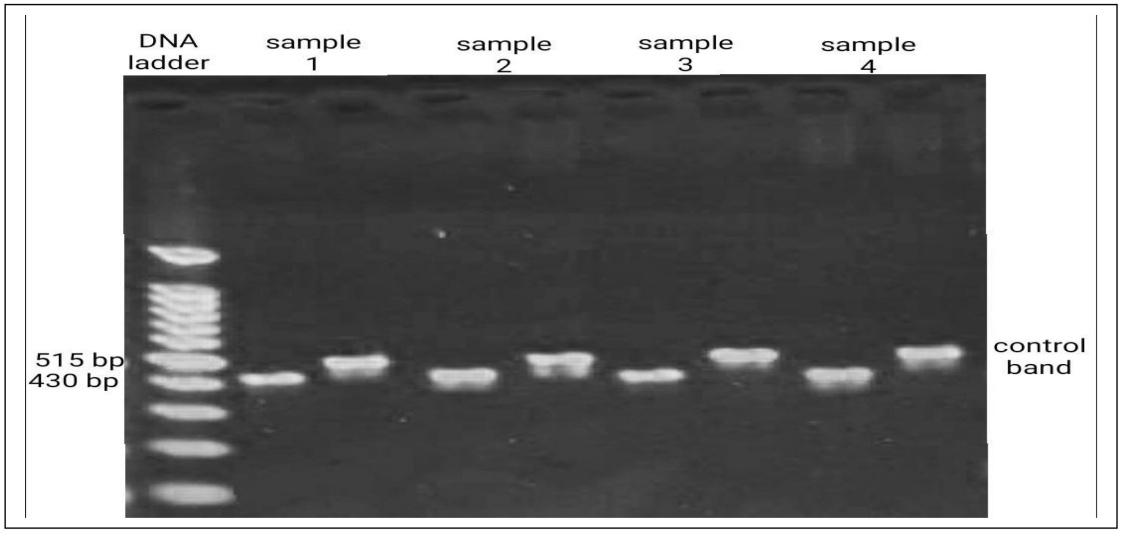

Fig. 3. Gel electrophoresis showing HLAB-27 specific band with molecular ladder, positive control band 515bp and 430bp (all samples); negative sample (all samples).

study observed that 4% (4/25) of the healthy subjects were found positive for the HLA-B27 allele. Also, the prevalence of HLA-B27 among healthy individuals was found to be 18–50% in American Indians, 2–6% in Southern Europe, 6–8% in Pakistanis, 2–6% in Indians, 1% in Japanese, and 1% in Africans, 10.7% in Bangladeshians. [48]

It was previously thought that HLA-B*27 had a predisposition toward familial association, a pathogenic involvement in the development of AS, and a role in antigen presentation. [49] There are very few investigations on the relationship between HLA-B*27 and AS in the Indian population, and previous studies found a wide range of frequencies, from 18% to 94%. [50]

Conclusion

The AS disease can be better understood and treated in Iraqi patients by using PCR-SSP techniques as a quick diagnostic marker. Identifying the HLAB 27 type at the start of the disease is essential for initiating the appropriate medication. In addition, serum IL-33 and sST2 levels were significantly higher in newly diagnosed AS patients and those receiving biologic treatment compared to the healthy control group.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been included with the necessary permission for republication, which is attached to the manuscript.
- Authors sign on ethical consideration's approval.
- No animal studies are present in the manuscript.
- Ethical Clearance: The project was approved by the local ethical committee at the College of Health & Medical Technology - Baghdad, Middle Technical University.

Authors' contribution statement

F. A. K.r wrote the manuscript, corrected errors, collected the data that are used in this work, The supervisor, M. N. I., worked on following up the research regarding the immunological part, as well as all the details of the research, and the supervisor, H. N. A., also played her role in the molecular biology part of the research and followed up on all the other details.

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دراسة مستويات مصل إنترلوكين -33 و ST2 القابلة للذوبان والنمط الجيني لمُسْتَضِدَّات الكُرَيَّاتِ البيضِ البَشَرِيَّة-ب27 لدى المرضى العراقيين المصابين بالتهاب الفقار المقسط

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المستخلص

التهاب الفقار المقسط هو نوع من التهاب المفاصل الذي يسبب التهابًا في المفاصل والأربطة في العمود الفقري. هدفت الدراسة الحالية إلى تحديد مستوى الإنترلوكين 33 و إنترلوكين sST2 القابل للذوبان في مصل الدم، بالاضافة الى دراسة جين مُسْتَضِدَّات الكُرَيَّاتِ الدم البيضِاء البَشَرِيَّة-ب27 بين مرضى التهاب الفقار المقسط . تم جمع عينات الدم الكاملة للمرضى من المستشفيات الطبية في مدينة بغداد. تم إجراء تقنية ELISA لقياس الإنترلوكين 33 و sST2 القابلة للذوبان، وكذلك تم تحديد جين مُسْتَضِدَّات كُرَيَّاتِ الدم البيضِاء البَشَرِيَّة-ب27 بواسطة تسلسل خاص من الجين (SSP-PCR). تم تقسيم المرضى إلى مرضى تم تشخيص إصابتهم حديثاً بالمرض ومرضى يتلقون العلاج البيولوجي (Etanercept) بالإضافة إلى مجموعة السيطرة الصحية. كان متوسط مستويات الإنترلوكين 33 في مصل الدم للمرضى الذين تم تشخيصهم حديثاً، والمرضى الذين عولجوا بعقار Etanercept، و مجموعة السيطرة الصحية (82.84 ± 3.59)، (55.91 ± 2.11)، (37.32 ± 1.69)، على التوالي. كان متوسط مستويات sST2 القابلة للذوبان في المصل للمرضى الذين تم تشخيصهم حديثًا والمرضى الذين عولجوا بعقار Etanercept ومجموعة السيطرة الصحية (165.62 ± 8.92)، (78.82 ± 5.84)، (48.01 ± 2.11)، على التوالي. بالإضافة إلى ذلك، كانت النتائج الإيجابية لجين مُسْتَضِدَّات كُرَيَّاتِ الدم البيضِاء البَشَرِيَّة-ب27 للمرضى الذين تم تشخيصهم حديثًا والمرضى الذين عولجوا بعقار Etanercept ومجموعة السيطرة الصحية (60.0٪)، (73.3٪)، (0.00٪)، على التوالي. والخلاصة أن البحث الحالي أكد على أهمية الانترلوكين 33 و sST2 القابل للذوبان والأليل لجين مُسْتَضِدَّات كُرَيَّاتِ الدم البيضِاء البَشَرِيَّة-ب27 كعلامة سريعة لتشخيص التهاب الفقار المقسط لدى المرضى العراقيين.

الكلمات المفتاحية: التهاب الفقار المقسط، المرضى العراقيين، جين مُسْتَضِدَّات كُرَيَّاتِ الدم البيضِاء البَشَرِيَّة-ب27، الإنترلوكين 33، sST2 القابل للذوبان.