Original Article

Clinical Parameters and Association of CCHCR1 Single-Nucleotide Polymorphisms in Psoriasis Arthritis Patients

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Abstract

Background: Previous information states that autoimmune and chronic inflammatory diseases, such as psoriasis and a psoriasis arthritis (PsA), mostly identified by multiple idiopathic cause. **Objective:** The current study resulted in new positive and significance changes in serum biochemical indicators for the patient and healthy participant groups. **Materials and Methods:** The present study is a case-control study represented by two groups (the first group 50 patients, while 40 healthy persons where been and the second group). Clinical biochemical parameters were symbolized by Vitamin K_2 (estimated by the ELISA technique) and the reduced glutathione (GSH)/oxidized glutathione (GSSG) ratio was evaluated using high-performance liquid chromatography. **Results:** The outcomes of the genetic parameter study of (CCHCR1 Gene) revealed a clear polymorphism for the (rs3130453) single nucleotide polymorphism in the first group. Polymerase chain reaction (REFLIP) type and gel electrophoresis were used as techniques for genetic examination. Levels of serum Vitamin K_2 and GSH parameters were decreased in the PsA group, where's as present, in normal level of healthy group. Meantime, the study explained an increase in the level of serum GSSG for PsA group, while, still normal in the second group. **Conclusion:** The actual study shows that an elevation of oxidative stress, which in final clarify and concludes a low GSH/GSSG ratio with the genetic polymorphism studied group.

Keywords: CCHCR1 gene, GSH, GSSG, psoriasis arthritis, Vitamin K2

Introduction

Thick, scaly areas are abundant causes of infected skin. While there cannot be a cure forever for this disorder, psoriasis treatment can help manage the symptoms of disease severity. Doctors or dermatology specialists may prescribe special creams or ointments for psoriasis.[1,2]

Psoriasis is an incapacitating, painful, deformed, and chronic noncommunicable conditions that have an enormous impact on a patient's quality of life with no known cure. It can also happen at any age, though the median age group of 50–69 is where it is most frequently documented.[3] The most reported prevalence of psoriasis in many countries ranges between 0.09%[4] and 11.4%.[5,6]

Prevalence of Iraqi psoriasis patients has been approximately 1.5%–2% of population. Psoriasis also has a significant impact on patients quality of life and in surveys, patients feel that the current treatments, although often effective, do not provide a satisfactory long-term solution.[7]

Polygenic psoriasis is a dermatological disorder characterized by being immune-mediated in nature. Likewise, a range of environmental factors such as trauma, infections, and drugs can exacerbate the disease in individuals who already possess vulnerable to it. The typical lesion is a well-defined reddened plaque

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with micaceous scale, which can be found locally or extensively.[7]

The chronic inflammation and apparently redness scales around are fairly common. Typical psoriatic scales that are mostly seen are whitish-silver and may develop in thick patches for many patients with red patches. The darker skin and a tone can also appear more purplish, such dark brown with gray scales. Sometimes, these patches will crack and bleed.[8]

One type of enlargement and tenderness is arthritis. Joint stiffness and pain, usually in one or more joints, are the primary symptoms of arthritis and usually get worse with age. The two most prevalent varieties of arthritis are rheumatoid arthritis and osteoarthritis.[9]

Psoriasis arthritis is one of the forms of arthritis disorders that affect some of patient's peoples who suffer from psoriasis, a skin condition that results in red skin areas covered in silvery scales. Before being diagnosed with psoriatic arthritis (PsA), the majority of patients first develop psoriasis. However, for some people, joint issues start concurrently with or before skin patches occur.[10]

PsA generally distinguished by joint pain, stiffness, and swelling. Symptoms can impact any part of the human body, especially the fingertips and spine, and range in severity from mild to severe. Both psoriasis and PsA can have periods of remission accompanied by episodes.[10]

Over activity of the immune system may be speeds up skin cell growth. Normally, the skin cells were in a completely grow and shed in a month. Psoriasis affected with skin cells like the keratinocytes and the dendric cells this is only in 3 or 4 days. Plaques and scales may appear on any part of the body, although they are commonly found on the elbows, knees, and scalp.[7]

Patients who have psoriasis may develop PsA, a chronic inflammatory arthritis. Some view it as a component of the diverse range of illnesses that are collectively referred to as spondyloarthritis (SpA). At least a few subtypes fall under the SpA category, including the axial and oligoarticular subtypes. There is little knowledge about the etiology and pathophysiology.[11]

Supplementing with Vitamin K2 reduced the total inflammatory results on blood tests in these investigations. Studies particularly examining the effects of Vitamin K on psoriasis are still lacking. The best way to combine Vitamin K2 with Vitamin D is still up for debate.[12,13]

Overall, despite the paucity of research, Vitamin K may be helpful in the management of PsA. To properly comprehend its function in the illness and to ascertain the best type and dosage of Vitamin K for the therapy of PsA, more research is necessary. Before taking Vitamin K for PsA or any other ailment, like with any supplement, it's crucial to speak with a doctor.[14]

Psoriasis has been shown to be linked both locally by the elbow and knees joint sites and systemically to increased oxidative stress and decreased glutathione (GSH) levels. It has been demonstrated that obese psoriasis patients had higher levels of oxidized GSH, which are linked to increased oxidative stress and systemic inflammation.[15]

Both GSH and oxidized glutathione (GSSG) possess numerous titration sites (GSSG has six, while GSH has four), and pH invariably affects their conformational space. Give a thorough morphological analysis of GSH and GSSG across a pH range.[16]

Accurate detection of GSSG and GSH/GSSG ratios is dependent on a variety of factors, including the low concentration of GSSG (high GSH/GSSG ratio) in tissues and the necessity of preventing GSH oxidation during the preparation of the sample. The administration of N ethylmaleimide (NEM) to react with GSH and create a stable complex was initially described by Guntherberg and Rost. The technique eliminated GSH before GSSG was quantified in tissues. Regretfully, NEM suppresses GR. Griffith used 2 vinyl pyridine (2-VP) to derivatize GSH to get around this issue.[17]

In contrast to the variation in clinical manifestations of PsA, the psoriatic phenotype remains heterogeneous. Research continues to demonstrate that there may be a substantial genetic link to PsA and that the illness etiologic involves a complicated interaction between environmental, immunological, and genetic variables.[18]

Psoriatic arthritis or PsA is a cutaneous and joint autoimmune situation that often lasts a long time. It is identified by lesions in the skin and joint pain, swelling, and inflammation. Numerous genes, including the CCHCR1 gene, have been linked to the complicated genetic foundation that underlies PsA.[19]

The CCHCR1 gene generates a structural protein containing multiple domains, which include zinc finger and coiled-coil domains. It has been suggested that these regions have significance for deoxyribonucleic acid (DNA) binding and protein-protein interactions. The CCHCR1 protein comprises multiple categories, including zinc finger and coiled-coil domains, which are assumed to be involved in binding to DNA and protein–protein interactions. Although the precise function of the CCHCR1 protein is undetermined, it has been suggested to be involved in immune response regulation as well as physiological transmission processes.[20]

Materials and Methods

Subjects and inclusion criteria

A (50) of PsA patients with inclusion criteria who were included in this study, as their history and past diagnosed psoriasis arthritis. Selections were made only for chronic patients who recurrently visit the dermatology and

rheumatology clinics of the hospital. The subjects were divided into the first (50 patients) group (patients) and (40 participants) the second healthy control group. Hence, this work was conducted using a case–control study design. A period of the present study period started in November 2022 to October 2023. A practical work of the current study was carried out at the branch of clinical biochemistry in laboratory of Medical Teaching City of Merjan, Hilla, Iraq.

Participant's exclusion criteria

Exclusion criteria included participants who suffering from any other chronic diseases such as chronic heart disease and diabetes with a matching in age, to excluded participants who were pregnant, smokers, taking any medication other than psoriasis treatment, and patients who had received treatment during the previous 2 weeks to 1 month. The 50 patients PsA, there where been 30 males and 20 females. But the sex did not affect the severity between patients' gender. Our study enrolled patients who had stopped the treatments intake.

The PsA severity detection score (PSAJAI)

To establish electronic programmed that could most accurately separate active medication from placebo. The models were mainly derived from clinical information and statistical considerations. Regression models based on the individual components of these response criteria were investigated, and data and the current PsA response criteria were evaluated. The psoriatic arthritis joint activity index (PsAJAI) served as a model for the American College of Rheumatology's 30% response threshold. PsAJAI was calculated by the electronic PsA calculator.[21,22]

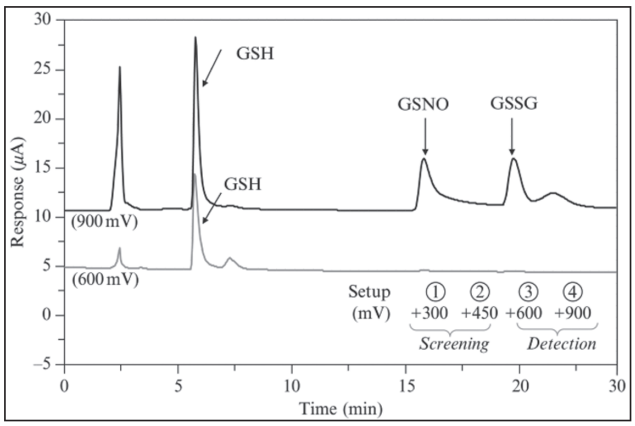

Figure 1: High performance liquid chromatography of reduced glutathione (GSH) and oxidized glutathione (GSSG) standards peaks

Serum determination of Vitamin (K_2)

The evaluation of serum vit. K2 levels for PsA participants represented by the PsA and the control groups that were done by use ELISA kit and according to the factory original manual.[23]

Serum determination of reduced and oxidized glutathione (GSH and GSSG)

The evaluation of serum GSH and GSSG was done by high performance liquid chromatography after derivatization with ortho-phthalaldehyde using Shimadzu LC-10AV model equipped with a binary delivery pump model LC-10AV with fluorescence detector. Hence, GSH appeared at 5 min and GSSG was appearing at min 20 of the eluting profile shown in Figure 1.[24]

The molecular and determination of (CCHCR1) gene

The polymerase chain reaction (PCR) technique is known as the based on the enzyme-mediated replication of DNA. In PCR, primer-mediated enzymes are utilized to amplify a short DNA segment. Additional strands of DNA that are identical to the DNA template are produced by DNA polymerase.[25]

When extracting DNA, the final step is to use a nano-drop to measure the purity and concentration of the extracted material. The measurement of absorbance is read at 260 nm.[26]

The genotyping determination of CCHCR1 gene

The single nucleotide polymorphism (SNP) is represented by the transversion in exon 4, with 445 bp. This was

Table 1: Physical and biochemical parameters results means and SD of PSAJAI score Vitamin K2, reduced glutathione (GSH)/ oxidized glutathione (GSSG) concentrations for psoriasis arthritis (PsA) patients and the control groups in the present study, with reference ranges and accepted *P* values

Parameter	Subject	Results	Std. dev.	Std. error	CI. lower	CI. upper	*P* values	Reference value
PSAJAI score	Patients	152	±25.412	0.76551	123	185	Less 0.05	0
	Control	0	0	0	0	0		
Vitamin K_2	Patients	1.350	±0.18710	0.167880	1.01310	1.68728	Less 0.05	1.5–3.2 ng/mL
	Control	2.132	±0.55029	0.087011	1.95625	2.30850		
GSH	Patients	0.2949	±0.18025	0.02549	0.2437	0.3461	Less 0.05	0.5–0.99 µM
	Control	0.8382	±0.33007	0.05219	0.7327	0.9438		
GSSG	Patients	1.410	±0.31992	0.04570	1.3185	1.5023	Less 0.05	0.1–0.33 µM
	Control	0.2133	±0.19430	0.03072	0.1511	0.2754		

amplified from DNA as 1.1-kb PCR product using primers.[27]

Results

Psoriasis arthritis is among the vast majority of difficulties having recent medical emphasis, and research is being done to identify potential treatments and cures that can be applied quickly or permanently. These studies also aim to identify the primary causes, associated hazards, and treatment options. The current study discusses the genetic level and pathogenesis of a PsA disease disorder.

The present study results with a physical parameter like (PSAJAI score) for detecting PsA severity cases, biochemical evaluations are shown in Table 1. Genetic polymorphism results also be discussed in Tables 2, 3 and 4 as well as demonstrated in Figures 2 and 3. Tables show the means and the SD±, STD. Error, CI. Lower, Cl. Upper, *P* values and the reference values for the biochemical indicators. In addition to the genotyping percentages, alleles frequencies and odds ratios for the genetic polymorphism results.

Discussion

Highly complex and heterogeneous interest in PsA illness is a significant and difficult task in clinical research investigations and the practice of medicine. In a reasonably large case–control study, the clinical diagnosis, genetic levels, and clinical biochemical data on well-characterized

Table 2: Molecular genetic results of CCHCR1 gene polymorphism single nucleotide polymorphism (rs3130453) for the psoriasis arthritis patients and the control groups, with genotyping percentages

Groups	*N*	Genotyping		
		XbaI (−/−)	XbaI (+/−)	XbaI (+/+)
Patients	50	10 (20%)	15 (30%)	25 (50%)
Healthy	40	28 (70%)	8 (20%)	4 (10%)

Table 3: Molecular genetic results of CCHCR1 gene polymorphism single nucleotide polymorphism (rs3130453) for the psoriasis arthritis patients and the control groups, with alleles frequencies

Groups	*N*	Allele frequency	
		XbaI (−) (%)	XbaI (+) (%)
Patients	50	39	61
Healthy	40	80	20

Table 4: The odd ration and CI (95%) for CCHCR1 Gene polymorphism characterization in patient and control groups

Genotype	Patients group	Control group	Odds ratio	CI (95%)	*P* value
XbaI (−/−)	2	28		Reference	
XbaI (+/−)	10	8	17.5	3.1668–96.70	0.001
XbaI (+/+)	38	4	133.0	22.7–777.8	0.001

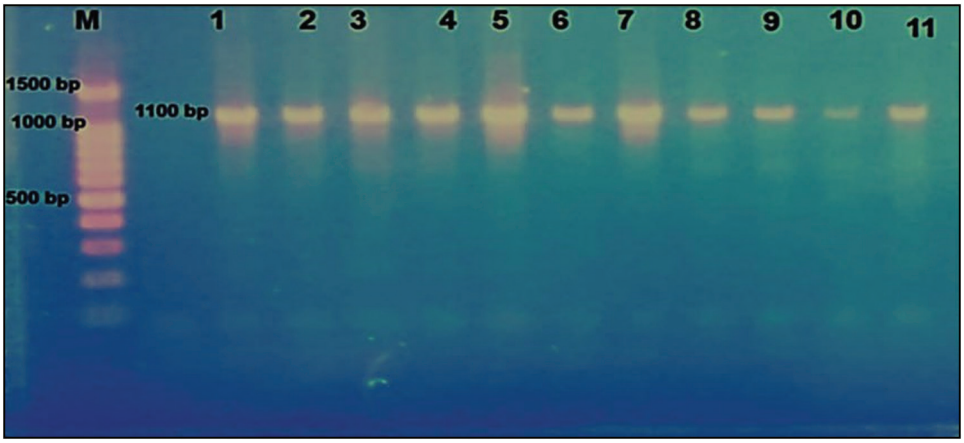

Figure 2: Polymerase chain reaction gel electrophoresis of a CCHCR1 gene (rs3130453) polymorphism site of the (G-T) mutated nucleotied

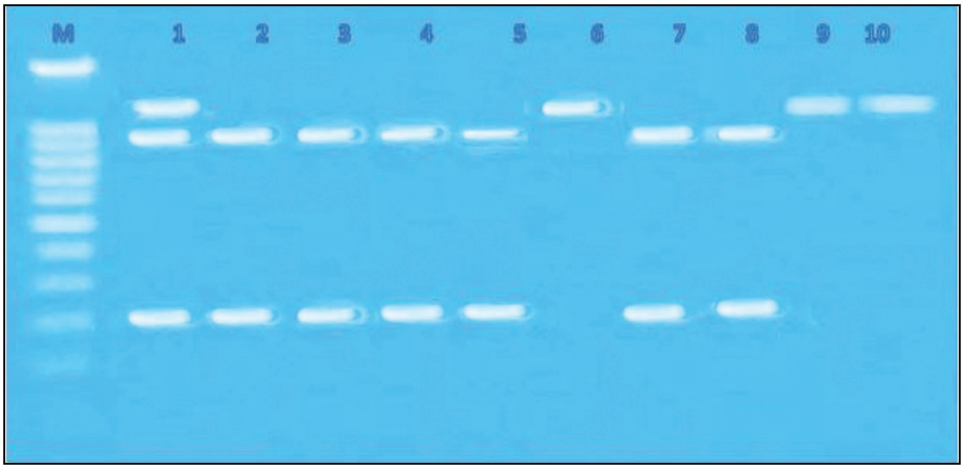

Figure 3: Restriction fragment length polymorphism pattern results of the (CCHCR1) gene polymorphism site. Lane M: deoxyribonucleic acid ladder (marker). Lane 6 XbaI (−/+) Genotype. Lane 9, 10:XbaI (−/−) genotype. Lane 1, 2, 3, 4, 5, 7, and 8 XbaI genotype (+/+)

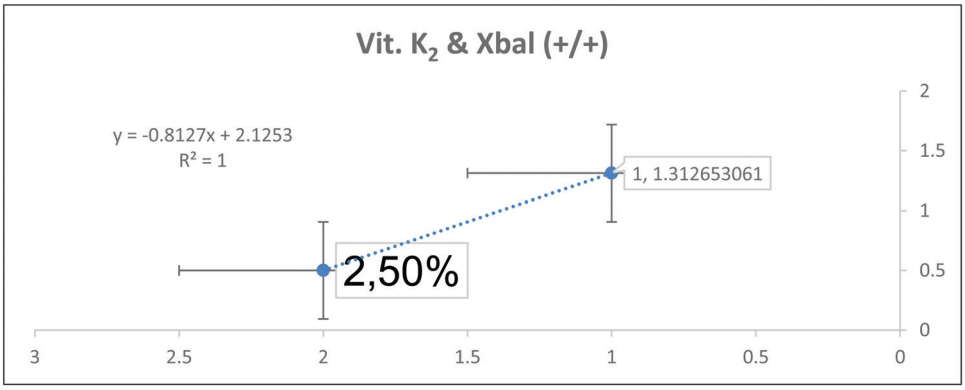

Figure 4: Correlation between the vitamin K_2 and the XbaI (+/+) genotyping for psoriasis arthritis group with acceptable $R^2 = 1$

PsA patients are connected in this current update study. The previous work pointed to various aspects of PsA markers that were examined, and intriguing findings were produced with new generations.

The PSAJAI score for PsA patients obtained results that revealed that all of PsA cases selected and enrolled in this study were calculated the scoring unit of disease activity by a (PSAJAI score) level. This will provide a clearer idea of the disease indication and severity. Results of the current study found that there was an increase in PASJAI score levels in almost all patients, with a mean and SD (152 ± 25), as well as being normal with a zero level in a healthy group, these results were in agreement with.[28]

The Vitamin K_2 was detected after estimation in the present study in patients' group in a decreased level, so the Vitamin K_2 concentration for PsA group in mean and SD (1.350 ± 0.18 ng/mL) and for the second group being (2.132 ± 0.55 ng/mL) as shown in Table 1, some of PsA patients enrolled by this study have a normal vit. K_2 because supplement intake; they had a negative or normal plasma CRP and were excluded from this parameter statistical analysis. These results were in agreement with the study by Elizabeth[29] that mentioned further research is needed on Vitamin K to determine the potential benefit of supplementation in psoriasis patients, and with the study by Ebina *et al.*[13] As well as many past studies were harmonized with our obtained results. Vitamin K_2 was very important for the diseases suppressants as reported by several studies in China[30] and with the American dietary study [Figure 4].[31]

Glutathione of PsA serums evaluations for the patients group levels or its activity in patients with psoriasis arthritis were found significantly lower than in general population (healthy control group). Table 1 shows the mean and SD ± results for PsA group (.2949 ± .18025 µg/mL) and for the healthy group (.8382 ± .33007 µg/mL). Very clear gap between the two groups with an acceptable *P* value less than 0.05 due to the severe oxidative stress state found in PsA group, so harvest data were compatible with several authors worldwide [Figure 5].[32,33]

The GSSG represents the abbreviation of oxidized form of the glutathione enzyme, harvest the outcomes of the serum GSSG level of the present study for the PsA were elevated compared to the normal healthy group by a mean

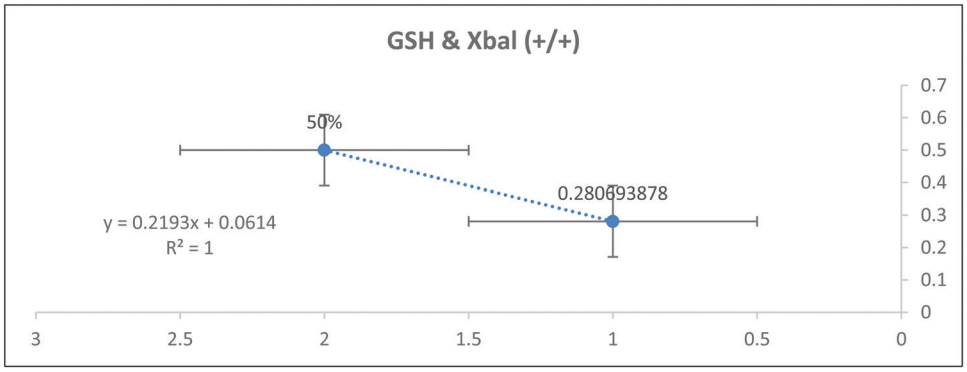

Figure 5: Correlation between the serum GSH and the Xbal (+/+) genotyping for psoriasis arthritis group with acceptable $R^2 = 1$

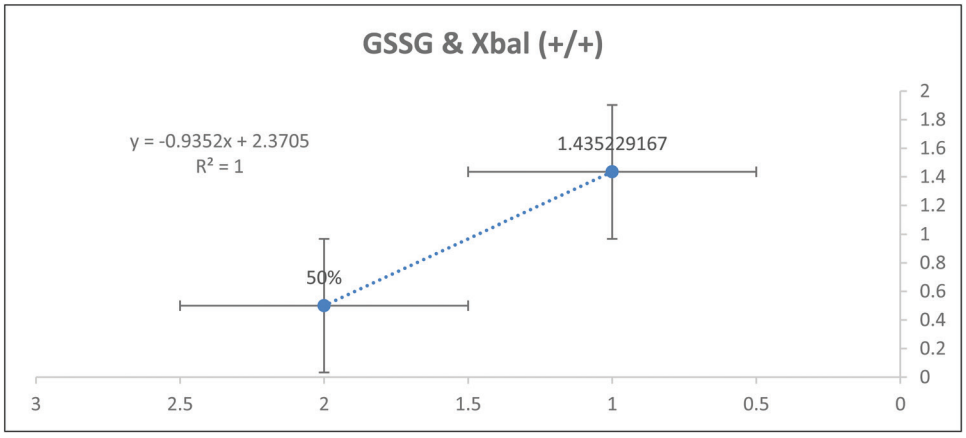

Figure 6: Correlation between the serum GSSG and the Xbal (+/+) genotyping for psoriasis arthritis group with acceptable $R^2 = 1$

and SD (1.410 ± .31992 µg/mL), while the second control group was (.2133 ± .19430 µg/mL). There has been no previous study estimating this parameter for psoriasis arthritis patients, as well as its elevation in PsA patients due to the large capacity of oxidative stress state and free radical hyperactivity [Figure 6].

Currents study results in Table 2 reveal that about 60% of all subjects of all the bulk of a (50 PsA persons) patients from the all (90 persons) that there enrolled in our present study. All study participants were divided into three subgroups due to the genetic results analysis that revealed. Hence, the first sub group represented a 20%[10] patients from the PsA big group they presence without any polymorphism, which abbreviated XbaI (−/−). As well as a 30%[15] patients of PsA represented the second subgroup, they found XbaI (−/+), which depends a disorder carrier without a triggered gene. The third group represented the big bulk of a PsA patients that detected with a clear polymorphism (+/+), that they composed a 50%[25] of all patient's number that enrolled at our present study. Subjects of healthy control group had opposite results, as indicated in the mentioned table.[34]

Group of a healthy control, through this work, was also divided into three sub groups, but with an opposite reveled result that also shown in Table 2. The big sub group or a bulk with a negative mutation polymorphism XbaI (−/−), when make a correlation between the two big group we will find a significance relationship, due to the significance different between them.

Table 3 shows the results of the alleles frequency in our study results. For the PsA patient and control groups, these results revealed that the XbaI (+) was been for (61%) of PsA patients and (20%) for the control groups. As well as the XbaI (−) was been (39%) for a patient and (80%) for a control group.

The presented results are revealed in Table 4 that the odds ratios for (28 subjects) from the control group and (2 subjects) from the PsA patients' group served as a reference for all participants because of the clear negative results with genotyping (−/−). Furthermore, the results showed a (+/−) genotyping type with an odds ratio (17.5) for (10 persons) from the PsA patients' group, (8 persons) with the control group.

As well as the most prevalent or a large bulk of all subjects enrolled had been a (+/+) genotyping for the patient group with 38 persons, and very rare positives for the control group with only 4 persons from 40 original subjects, which was abbreviated with an odd ratio (133.0). The present results were in good accordance with other studies conducted in China[35] and Iraq.[10,36]

Conclusion

According to the mentioned odds ratio results, which indicated that PsA was triggered by a genetic polymorphism mostly and by more than one SNP. There was a positive and significant correlation between the biochemical serological parameters and the PsA severity, as well as with genetic factors. Studies pointing to oxidative stress and free radical have grait effects on the patient status.

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Nil.

Conflicts of interest

There are no conflicts of interest

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