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Original Article

Elevated oxidative stress markers in patients with ankylosing spondylitis: A case–control study

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ABSTRACT

Background: Ankylosing spondylitis (AS) is a chronic inflammatory musculoskeletal disease with an unclear pathogenesis. Oxidative stress (OS) is currently becoming an area of interest in AS pathogenesis and progression.
Objective: The aim of this study was to evaluate serum oxidative and anti-oxidative status in patients with AS and to assess the relationships between OS markers and disease indices.
Patients and methods: Forty-four AS patients (mean age: 44.9 ± 14.12 years, sex-ratio M/F: 2.14) and 46 healthy controls (mean age: 44.62 ± 10.74 years, sex-ratio M/F: 2.28) with no known diseases were included in the study. Oxidant markers – advanced oxidative protein products (AOPP) and malondialdehyde (MDA) – and anti-oxidant molecules – catalase (CAT), glutathione peroxidase (GPx) and reduced gluthatione (GSH) – were measured. The Bath ankylosing spondylitis disease activity index (BASDAI) and the Bath ankylosing spondylitis functional index (BASFI) were calculated.
Results: In AS patients, mean disease duration was 21.11 months [range: 2–54] and the mean BASDAI and BASFI scores were 4.52 ± 1.45 and 4.64 ± 2.52, respectively. No differences in term of age, sex, body mass index, blood pressure, glucose, lipid profile, uric acid, total bilirubin and homocystein level were observed. The levels of oxidant markers (AOPP and MDA) were significantly higher ($p < 10^{-6}$) and the levels of anti-oxidant markers (CAT, GPx and GSH) were significantly lower ($p < 10^{-6}$) in AS patients than those in healthy control subjects. Correlation analysis showed a significant positive correlation between AOPP levels and BASDAI score ($p = 0.001$) and BASFI score ($p = 0.006$) and a significant negative correlation between GPx activity and BASDAI score ($p = 0.001$) and BASFI score ($p = 0.01$).
Conclusion: This study demonstrates an oxidative stress condition characterized by increased oxidant markers and decreased antioxidant capacity in AS patients compared to controls, suggesting that OS may play a crucial role in the pathogenesis and progression of the disease.

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Marcadores elevados de estrés oxidativo en pacientes con espondilitis anquilosante: un estudio caso-control

Palabras clave:
Espondilitis anquilosante
Estrés oxidativo
Oxidación de proteínas
Malondialdehído
Antioxidantes

RESUMEN

Introducción: La espondilitis anquilosante (EA) es una enfermedad musculoesquelética inflamatoria crónica cuya patogenia no está clara. El estrés oxidativo (EO) se está convirtiendo actualmente en un área de interés en la patogénesis de la EA y la progresión

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Objetivo: El objetivo de este estudio fue evaluar el estado oxidativo sérico y el estado antioxidante en los pacientes con EA, y evaluar las relaciones entre los marcadores de EO y los índices de la enfermedad.
Pacientes y métodos: Cuarenta y cuatro pacientes con EA (edad: 44,9 ± 14,12 años, ratio sexo M/F: 2,14) y 46 controles sanos (edad: 44,62 ± 10,74 años, ratio sexo M/F: 2,28) fueron incluidos en el estudio. Se midieron los marcadores oxidantes —productos proteicos oxidativos avanzados (AOPP) y malondialdehído (MDA)— y las moléculas antioxidantes —catalasa (CAT), glutatión peroxidasa (GPx) y glutatión reducida (GSH)—. Se calcularon el índice de actividad de la enfermedad de la espondilitis anquilosante de Bath (BASDAI) y el índice funcional de la espondilitis anquilosante de Bath (BASFI).
Resultados: En los pacientes con EA, la duración media de la enfermedad fue de 21,11 meses (intervalo: 2-54) y las puntuaciones medias BASDAI y BASFI fueron de 4,52 ± 1,45 y 4,64 ± 2,52, respectivamente. No se observaron diferencias en cuanto a edad, sexo, índice de masa corporal, tensión arterial, glucosa, perfil lipídico, ácido úrico, bilirrubina total y nivel de homocisteína. Los niveles de marcadores oxidantes (AOPP y MDA) fueron significativamente superiores ($p < 10^{-6}$) y los niveles de marcadores antioxidantes (CAT, GPx y GSH) fueron significativamente inferiores ($p < 10^{-6}$) en los pacientes con EA, que en los sujetos sanos de control. El análisis de correlación mostró una correlación positiva significativa entre los niveles de AOPP y la puntuación BASDAI (p = 0,001) y puntuación BASFI (p = 0,006), y una correlación negativa significativa entre actividad GPx y puntuación BASDAI (p = 0,001) y puntuación BASFI (p = 0,01).
Conclusiones: Este estudio demuestra un estado de estrés oxidativo caracterizado por un aumento de los marcadores oxidantes, y una disminución de la capacidad antioxidante en los pacientes con EA en comparación con los controles, lo que sugiere que el EO puede desempeñar un papel crucial en la patogenia y progresión de la enfermedad.
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Introduction

Ankylosing spondylitis (AS) is a chronic inflammatory musculoskeletal disease that mainly affects the spine and sacroiliac joints, but may also affect peripheral joints and entheses. This disease generally appears in the third decade of life with a higher prevalence in men. The pathogenesis of AS remains unclear. Interactions between genetic factors, innate immunity, lifestyle, and environmental factors are thought to be responsible.[1,2] In addition to these factors, oxidative stress (OS) has become an area of interest in AS pathogenesis and progression.

OS is a condition caused by an imbalance between increased production and accumulation of reactive oxygen species (ROS) and the ability of the antioxidant defense to detoxify these ROS. The presence of excessive and uncontrolled levels of ROS leads to cell and tissue damage (oxidative stress).[3,4] Increased OS is implicated in the pathogenesis of some diseases such as cardiovascular and neurodegenerative diseases, but also in the pathogenesis of some inflammatory diseases such as rheumatoid arthritis and inflammatory bowel disease.[5–7]

In the literature, there have been few studies concerning oxidative metabolism in patients with AS and the relation between OS and the characteristics of this disease. Authors suggest that increased OS may be implicated in the pathogenesis of AS, as well as in the progression and exacerbation of this disease.[7,8] Further studies on this subject are needed.

The primary aim of our study was to investigate serum oxidative and anti-oxidative status in patients with AS and to compare them with that of age- and sex-matched healthy control. Additionally, relationships between the OS markers and disease indices were assessed.

Patients and methods

Study population

This case–control study was carried out on Tunisian patients with AS followed up in the Rheumatology department of the Hedi Chaker Hospital of Sfax (Tunisia), over a 12-month period (January 2023 to December 2023). AS diagnosis was confirmed by a rheumatologist according to Assessment of Spondyloarthritis International Society (ASAS) criteria of 2009.[9] The exclusion criteria were hypertension, diabetes mellitus, hyperlipidemia, cardiovascular diseases and neoplastic diseases. Patients with chronic inflammatory diseases (rheumatoid arthritis, Reiter's syndrome, psoriasis, inflammatory bowel disease, systemic lupus erythematosus) were also excluded. Sex-, age- and body mass index (BMI)-matched healthy controls with the same exclusion criteria as the patients were enrolled. Informed consent was obtained from all the subjects (patients and controls) of this study.

For all the patients, we collected the demographic characteristics, the clinical features of the disease (duration, age of onset of symptoms, axial manifestations, peripheral articular and enthesitic manifestations, extra-articular manifestations). We evaluated the severity of the disease using the Bath ankylosing spondylitis disease activity index (BASDAI) (range: 0–10) and the Bath ankylosing spondylitis functional index (BASFI) (range: 0–10). The BASDAI is a validated score to assess the activity and the severity of AS.[10] The BASFI is a validated functional score used to assess the degree of functional limitation in patients with SA.[11]

Laboratory tests

Blood samples were collected for tests after overnight fasting. The erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), fasting blood glucose, serum lipids (total cholesterol, high density lipoprotein (HDL) cholesterol, LDL cholesterol, and triglycerides), uric acid (UA) and total bilirubin (TB) were measured using standard methods. Homocysteine (Hcy) level was determined using a fluorescence polarization immunoassay. HLA B-27 antigen was also determined in the patients group. For the measurement of OS markers, blood samples were collected after overnight fasting into anti-coagulant-free tubes and immediately placed onto ice until processing. Blood samples were centrifuged at 1000 g at room temperature for 10 min to separate plasma and stored at −80 °C until analysis.

Measurement of protein oxidation products

The concentration of protein oxidation products was calculated as the advanced oxidation protein products (AOPP) concentrations, using a spectrophotometric method according to Witko-Sarsat et al.[12,13] The concentration of AOPP was expressed in micromoles of chloramine-T equivalents per litre of plasma (μmol/l).

Measurement of lipid peroxidation products

The concentration of lipid peroxidation products was calculated as the serum malondialdehyde (MDA) concentrations, using a spectrophotometric method described by Guidara et al.[13] This method is based on the reaction between MDA and thiobarbutiric acid (TBA). The concentration of MDA was expressed in micromoles per milligram of proteins (μmol/mg).

Measurement of activity of antioxidant enzymes

Catalase (CAT) activity in erythrocytes was measured by Aebi et al. spectrophotometric method,[14] based on the decomposition of H_2O_2. CAT activity was expressed as millimolar of H_2O_2 degraded per minute per milligram of protein (mM/min/mg).

Glutathione peroxidase (GPx) activity was measured according to the method of Rotruck et al. with slight modifications by Guidara et al.[13,15] The assay is based on the GPx-catalyzed reduction of H_2O_2 to water, with concurrent oxidation of reduced glutathione (GSH) to glutathione disulfide (GSSG). GPx activity was expressed as millimolar of GSH consumed per minute per milligram of protein (mM/min/mg).

Reduced glutathione (GSH) was determined using the method of Jollow et al.[16] In this method, 5,5′-dithiobis-2-nitrobenzoic acid (DTNB) acts as a disulfide chromogen that reacts with sulfhydryl groups to form a yellow-colored compound. The concentration of GSH was expressed in nanomoles per milligram of proteins (nmol/mg).

Statistical analysis

Statistical analysis was performed using IBM SPSS statistics (version 20) software. After normality was verified using the Shapiro–Wilk test, continuous variables were described in terms of means and standard deviation and compared between the two groups using an independent sample Student's *t*-test. Qualitative variables were described by their frequencies and compared between the two groups using the Chi-square test (χ^2). Correlations between OS markers and disease indices were performed using Pearson's correlation coefficient. Enter methods was used for linear regression analysis. A two-tailed *p*-value <0.05 was considered as statistically significant.

Results

This study included 44 patients with AS (mean age: 44.9 ± 14.12 years, sex-ratio M/F: 2.14) and 46 healthy controls (mean age: 44.62 ± 10.74 years, sex-ratio M/F: 2.28). In patients with AS, the mean disease duration was 21.11 months [range: 2–54] and the mean age at symptom onset was 36.8 ± 9.2 years. The AS was axial in 63.6% ($n = 28$), axial and peripheral in 34.1% ($n = 15$) and only peripheral in 2.3% ($n = 1$). Hip joint involvement was observed in 20.4% of cases ($n = 9$), and thoracic wall involvement in 25% ($n = 11$). Extra-articular manifestations were reported in 50% of patients ($n = 22$), including: anterior uveitis (22.7%, $n = 10$), restrictive respiratory syndrome (18.2%, $n = 8$), cardiac conduction disorders (9.1%, $n = 4$), aortic insufficiency (4.5%, $n = 2$), and IgA mesangial nephropathy (2.3%, $n = 1$). HLA-B27 antigen was identified in 61.4% of patients ($n = 27$).

The mean BASDAI score was 4.52 ± 1.45. The disease was active (BASDAI ≥4) in 68.2% of patients ($n = 30$) and inactive (BASDAI <4) in 31.8% ($n = 14$). The mean BASFI score was 4.64 ± 2.52.

The demographic, clinical and laboratory data of the subjects (patients and controls) are summarized in Table 1. No significant differences were found between the two groups regarding age, sex, BMI, blood pressure, smoking, alcohol, glucose, lipid levels, uric acid, total bilirubin and homocystein level. Inflammatory biomarkers (ESR and CRP) were significantly higher in patients than in healthy controls ($p < 0.001$).

Regarding the oxidative profile in our patients, significantly higher concentrations of protein oxidative products (AOPP) and lipid peroxidation products (MDA) were noted in AS patients in comparison to the control group (57.62 ± 11.72 vs. 42.64 ± 9.18 μmol/l, $p < 10^{-6}$; and 55.69 ± 16.37 vs. 28.71 ± 10.85 μmol/mg, $p < 10^{-6}$, respectively) (Table 2).

On the other hand, catalase (CAT) enzyme and glutathione peroxidase (GPx) activities and reduced glutathione (GSH) level were significantly lower in AS patients than in those of healthy controls (3.47 ± 1.82 vs. 4.75 ± 3.17 mM/min/mg; 1.94 ± 0.85 vs. 2.75 ± 0.91 mM/min/mg; and 0.48 ± 0.23 vs. 1.11 ± 0.41 nmol/mg, respectively; $p < 10^{-6}$) (Table 2).

In AS patients, AOPP levels were positively correlated with disease activity (BASDAI, $p = 0.001$) and functional impairment (BASFI, $p = 0.006$) (Fig. 1). No significant correlation was found between MDA concentrations and BASDAI and BASFI scores. Among antioxidant markers, GPx activity correlated negatively with BASDAI ($p = 0.001$) and BASFI ($p = 0.01$) (Fig. 2). No significant correlations were observed between CAT activity or GSH levels and BASDAI or BASFI scores.

No statistically significant correlations were found between oxidant/antioxidant parameters (AOPP, MDA, CAT, GPx, and GSH) and inflammatory biomarkers (ESR, CRP).

Discussion

In the current study, the plasma levels of oxidant markers (AOPP and MDA) in AS patients were significantly higher than those of the healthy control subjects. Additionally, the levels of anti-oxidant molecules (GPx and CAT activities, GSH) in AS patients were significantly lower than those of the healthy control subjects. This indicates an imbalance between oxidant and anti-oxidant molecules in the plasma of patients with AS.

Although many studies have examined oxidative metabolism in inflammatory diseases such as rheumatoid arthritis and inflammatory bowel diseases,[5,6,17,18] there are few studies in the available literature concerning the oxidative status in patients with AS.

In our study, in agreement with the studies previously published, increased markers of oxidative stress, especially AOPP and MDA, were reported in patients with AS. Similarly, several studies have documented higher levels of AOPP,[19,20] MDA,[21–23] and total oxidant status (TOS)[7,8,24] in AS patients compared with healthy controls.

Regarding the anti-oxidant status in AS, data from previous studies are controversial and inconsistent. Some studies, consistent with our findings, reported a decrease in antioxidant defenses in AS patients compared with healthy controls.[8,19,25,26] Conversely, other studies documented increased activities of antioxidant enzymes, such as superoxide dismutase (SOD), catalase (CAT), and GPx.[21,23,24] These increases were attributed to a compensatory antioxidative response to excessive oxidative stress.

The exact mechanisms involved in the increased OS observed in AS remain unknown. In general, OS is defined as a condition resulting from an imbalance between the production of reactive oxygen species (ROS) – primarily generated from oxygen within

Table 1
Comparison of ankylosing spondylitis (AS) patients and healthy controls in terms of demographic, clinical and laboratory parameters.

Parameters	Patients with ankylosing spondylitis ($N = 44$)	Healthy controls ($N = 46$)	p-Value
Sex-ratio, H/F	30/14	32/14	0.92
Age (years), mean (SD)	44.9 (14.12)	44.62 (10.74)	0.63
BMI[a], kg/m^2, mean (SD)	26.26 (5.98)	26.72 (3.25)	0.88
Systolic blood pressure (mmHg), median (range)	120 (90-135)	110 (90-130)	0.16
Diastolic blood pressure (mmHg), median (range)	75 (55-90)	72 (60-85)	0.18
Smoking, *n* (%)	13 (29.5%)	10 (21.7%)	0.24
Alcohol, *n* (%)	6 (13.6%)	6 (13.1%)	0.44
ESR[b], mm/h, mean (SD)	52 (12.25)	10.2 (4.82)	<0.001*
CRP[c], mg/l, mean (SD)	17.85 (6.25)	2.23 (1.91)	<0.001*
Glucose, mmol/l, mean (SD)	5.87 (4.38)	5.65 (2.66)	0.75
Total cholesterol, mmol/l, mean (SD)	4.47 (1.15)	4.74 (1.04)	0.28
HDL-cholesterol, mmol/l, mean (SD)	1.09 (0.23)	1.21 (0.33)	0.62
Triglyceride, mmol/l, mean (SD)	1.31 (0.64)	1.28 (0.59)	0.69
Uric acid, µmol/l, mean (SD)	280.49 (100.28)	280.75 (73.76)	0.29
Total bilirubin, mg/l, mean (SD)	8.60 (4.24)	9.84 (5.76)	0.23
Homocysteine, µmol/l, mean (SD)	9.85 (3.55)	10.10 (5.22)	0.82

Statistical analysis: Student's *t*-test was used for continuous variables and the Chi-square test for categorical variables.
[a] Body mass index.
[b] Erythrocyte sedimentation rate.
[c] C-reactive protein.
* $p < 0.001$ (highly significant).

Table 2
Comparison of oxidative stress biomarkers in patients with ankylosing spondylitis (AS) patients and healthy controls.

Parameters	Patients with ankylosing spondylitis($N = 44$)	Healthy controls($N = 46$)	p-Value
AOPP[a], mmol/l, mean (SD)	57.62 (11.72)	42.64 (9.18)	$<10^{-6}$*
MDA[b], µmol/mg, mean (SD)	55.69 (16.37)	28.71 (10.85)	$<10^{-6}$*
CAT[c], mM/min/mg, mean (SD)	3.47 (1.82)	4.75 (3.17)	$<10^{-6}$*
GPX[d], mM/min/mg, mean (SD)	1.94 (0.85)	2.75 (0.91)	$<10^{-6}$*
GSH[e], nmol/mg, mean (SD)	0.48 (0.23)	1.11 (0.41)	$<10^{-6}$*

[a] Advanced oxidation protein products.
[b] Malondialdehyde.
[c] Catalase activity.
[d] Glutathione peroxidase activity.
[e] Reduced glutathione.
* $p < 0.001$ (highly significant).

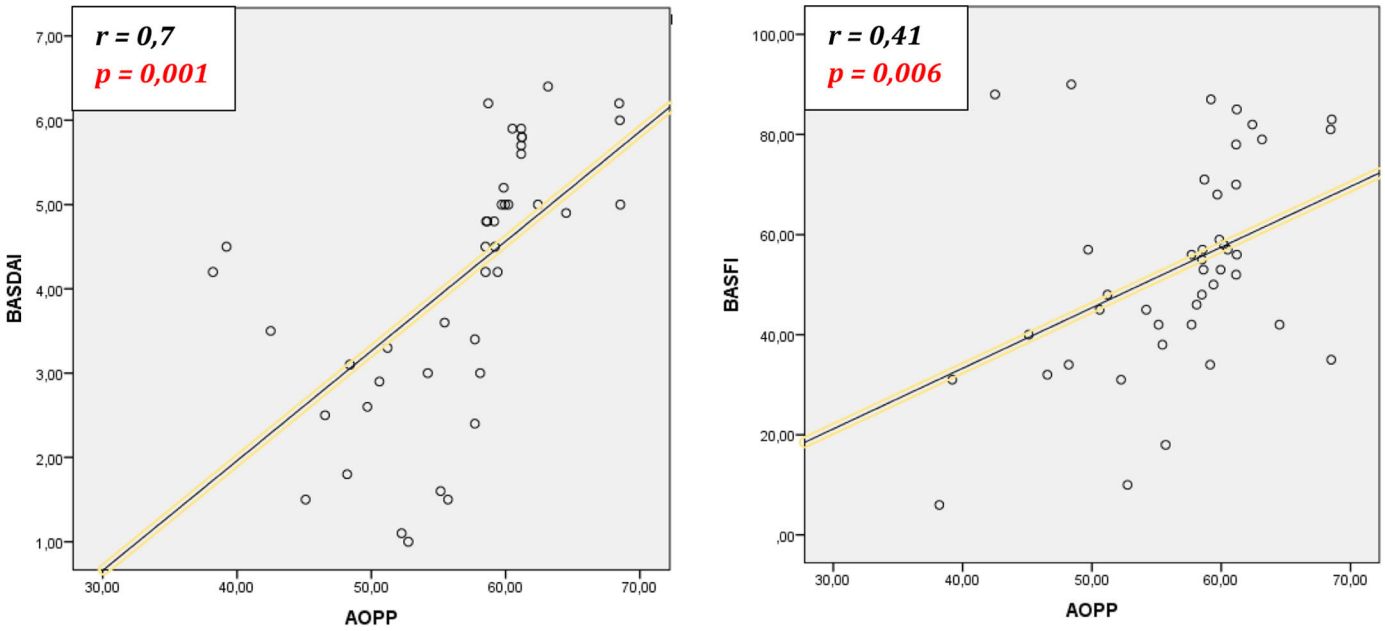

Fig. 1. Positive correlation of AOPP levels with BASDAI score and BASFI score.

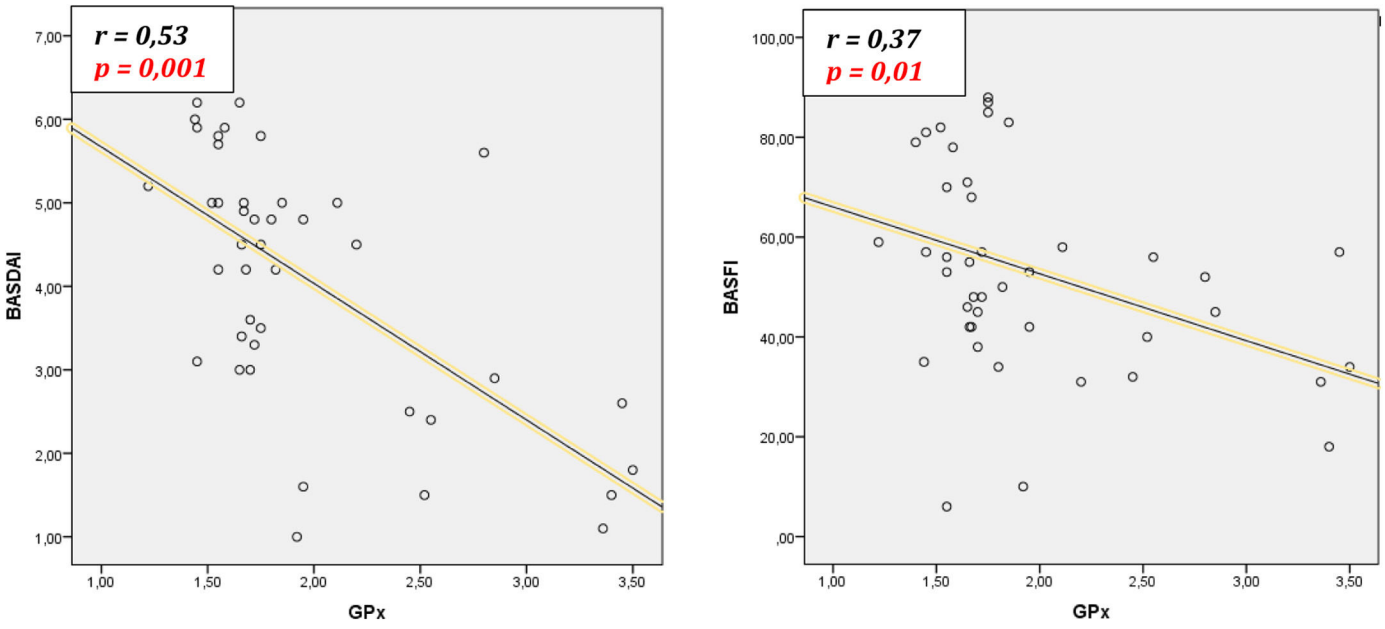

Fig. 2. Negative correlation of GPx activity with BASDAI score and BASFI score.

human cells, particularly in the mitochondria – and the availability of antioxidants in the body. As a consequence, the body is no longer able to effectively control the excessive and prolonged presence of toxic ROS, which are potentially harmful and responsible for cellular damage and impaired cellular function.[27] Various endogenous and exogenous factors can stimulate and induce excessive ROS production, leading to OS. Exogenous sources include smoking, alcohol consumption, excessive sun exposure (ultraviolet radiation), intense physical exercise, stress, an unbalanced diet (rich in fats), pollution, and exposure to chemicals and toxic substances. Endogenous sources include obesity, hyperglycemia, dyslipidemia, hyperhomocysteinemia, and iron overload.[3,27] In our study, most of these risk factors were assessed and compared between the two groups, and no significant differences were found between patients and controls regarding age, BMI, smoking and alcohol consumption, blood glucose levels, lipid profile, and homocysteine levels. No significant difference between the two groups was found regarding uric acid and total bilirubin levels, both of which have antioxidant properties. However, certain risk factors for OS were not assessed in our study, such as physical and sports activity, sun exposure, diet, geographical origin, and exposure to toxins and pollution.

In patients with AS, the increase of OS is attributed mainly to the excessive production of reactive oxygen species (ROS) by inflammatory cells as activated neutrophils during the inflammatory process.[28] Moreover, the inflammatory cells infiltrating the joints in AS release pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α). TNF-α has in recent years been shown to activate many pathways involved in OS via nuclear translocation of NF-κB.[29] A number of studies have reported a decreased in the oxidant markers in patients with a TNF-α targeting agent than in those with conventional drugs.[8,30]

These notions explain the significant correlation between the levels of oxidative markers levels and the disease activity score (BASDAI) observed in many previous studies[7,20,21] and in our study i.e. SO is more pronounced in patients with active disease compared to patients in remission. In the current study, a significant positive correlation between AOPP levels and BASDAI score ($p = 0.001$) and a significant negative correlation between GPx activity and BASDAI score ($p = 0.001$) were found.

Finally, as in the current study, many researchers have hypothesized that OS may contribute to the exacerbation and the progression of the disease.[7,25] Indeed, in our study, markers of OS (AOPP and GPx) were significantly correlated with the BASFI score i.e. with the importance of the functional disability caused by the disease, suggesting the association of the increased OS with a more severe and a more impairing disease in AS patients. Our study is not the only one to report such findings; other studies, such as those by Solmaz et al.[7] and Nazıroğlu et al.,[25] have also demonstrated a significant association between OS biomarkers and disease severity scores, particularly the functional disability score (BASFI) and the spinal mobility assessment score (BASMI).

Beyond its impact on quality of life and functional ability, increased OS in AS has been shown to potentially contribute to a higher cardiovascular risk and may itself represent an additional cardiovascular risk factor in these patients. Indeed, OS – mainly through the oxidation of low-density lipoproteins (LDL) and the deposition of oxidized LDL in the arterial wall – is responsible for endothelial dysfunction, which plays a major role in the pathogenesis of cardiovascular diseases and the formation of atherosclerotic plaques.[31,32]

The main limitation of our study and all other published studies is the small population size and the cross-sectional nature. Further studies with a greater number of patients are required to clarify the role of OS in the etiopathogenesis and the evolution of AS.

Based on these findings, it can be suggested that novel therapies targeting OS pathways and enhancing the body's antioxidant defense systems could be beneficial in the management of AS. Such approaches might improve the prognosis of this disease and contribute to better management of the increased cardiovascular risk. Future studies are needed to further document and validate these concepts. In this context, it is important for AS patients to maintain optimal antioxidant defenses. Moderate and regular physical activity, weight reduction when necessary, and a healthy diet low in fats and rich in fruits and vegetables – particularly those high in antioxidants such as nuts, whole grains and seeds, spinach, carrots, tomatoes, and citrus fruits – should always be encouraged in patients with AS.

Conclusion

Our results demonstrated the imbalance between oxidant and anti-oxidant molecules in the plasma of patients with AS, with significantly increased levels of oxidant markers and significantly decreased levels of anti-oxidant molecules than those in the healthy control subjects, suggesting that OS might play a crucial role in the onset and the development of the disease. These findings require confirmation through studies with larger populations and controlled trials. In this context, new therapies targeting OS pathways could be valuable in treating patients with AS.

CRediT authorship contribution statement

ZG: Conception, writing, data collection; HF: Supervision; MM and AE: Analysis and interpretation; AF: Data collection; MK, SB and KC: Revision; FA: Supervision and validation.

Ethical approval

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. All participants were fully informed about the study's purpose, and procedures. Written informed consent was obtained from each participant. Participation was voluntary, and participants were assured that they could withdraw from the study at any time.

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Conflicts of interest

None declared.

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