Research Article

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Myotonic dystrophy type 1 and oxidative imbalance: evaluation of ischemia-modified albumin and oxidant stress

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Abstract

Objectives: Oxidative stress occurs when free radicals accumulate at high levels in the body, affecting biological molecules. Some studies have focused on the multisystemic involvement, signs of premature aging, and the potential role of oxidative stress in the pathogenesis of myotonic dystrophy type 1 (DM1). We aimed to evaluate the levels of oxidative stress markers in patients diagnosed with DM1.

Methods: In a cross-sectional study, 27 clinically and genetically confirmed DM1 patients and 34 healthy individuals matched for age and gender were included. Oxidative stress markers, including ischemia-modified albumin (IMA), total antioxidant status (TAS), total oxidant status (TOS) levels, ischemia-modified albumin to albumin ratio (IMAR), and oxidative stress index (OSI) were examined.

Results: TOS ($p=0.001$), IMA levels ($p=0.001$), IMAR ($p=0.004$), and OSI ($p<0.001$) in the patient group were significantly higher than in the healthy group, while TAS ($p<0.001$) was lower in the patient group.

Conclusions: The elevation of oxidative stress markers suggests the potential effect on the pathogenesis of DM1. Thus, antioxidant therapy approaches could be crucial to reducing oxidative stress in patients with DM1.

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Introduction

Myotonic disorders encompass a group of rare heterogeneous syndromes characterized by myotonia. Myotonia refers to difficulty and slowness in muscle relaxation following activation. It is characterized by painful or painless muscle stiffness. Myotonias are classified as dystrophic and non-dystrophic. Myotonic dystrophies are further categorized into types 1 and 2 [1].

Myotonic dystrophy type 1 (DM1) is a severe phenotype autosomal dominant disease. The incidence of myotonic dystrophy type 1 is 1–15 per 100,000, and it is a rare disease. DM1 arises from mutations in the protein kinase gene located on chromosome 19q13.3. The mutation is typically characterized by a triplet repeat of cytosine-thymine-guanine within the gene. DM1 protein kinase is part of a mitochondrial complex with antioxidants and pro-survival properties. Diagnosis is made through clinical examination and genetic testing. Effective treatments targeting the pathogenesis of DM1 are not yet available. Treatment for DM1 is symptomatic [1].

Recent studies focus on the relationship between premature aging and oxidative stress in DM1 patients. [1, 2]. Oxidative stress is an imbalanced increase of free radicals in the body, leading to cellular damage. Oxygen radicals, metabolic by-products of oxidative stress, attack cellular proteins, lipids, and DNA bases, initiating a chain of oxidation reaction by removing a hydrogen ion. Consequently, intracellular lipids, proteins, and DNA are damaged, leading to cellular injury and death [3, 4].

Oxidative stress arises from the imbalance between the total oxidant status (TOS) and total antioxidant status (TAS). The TOS measures the levels of free radicals and other oxidant substances to indicate oxidative stress. Free radicals, heavy metals, hydrogen peroxide, and toxic chemicals contribute to TOS. The TAS assesses the presence and quantity of antioxidant compounds, indicating how effectively cells are protected against free radicals [5]. The body's oxidative stress balance is evaluated by the oxidative stress index (OSI) [5, 6]. This index indicates oxidative stress by considering the levels of free radicals and antioxidants.

Albumin has an antioxidant function that removes oxidative stress products from the environment. In oxidative stress conditions, the capacity of albumin to bind and remove heavy metal ions at the N-terminal end decreases, leading to ischemia-modified albumin (IMA) formation. IMA is considered a biomarker for oxidative stress and an indicator of tissue damage [7]. Elevated levels of IMA have been observed in conditions such as pulmonary embolism, ischemic diseases, skeletal muscle ischemia after exercise, inflammatory diseases, diabetes mellitus, certain cancers, infections, and peripheral vascular diseases [8].

We aimed to evaluate the relationship between the oxidative stress indicators (TOS, TAS, IMA) and DM1 to understand disease pathogenesis and develop new treatment options.

Materials and methods

Twenty-seven patients with DM1, diagnosed clinically, phenotypically, and genetically and admitted to the physical therapy and rehabilitation clinic, were included in the study [1]. Thirty-four healthy individuals matched for age, gender, and similar characteristics were formed. Individuals with a history of acute or chronic infection, coronary artery disease, type 2 diabetes mellitus, liver disease, hypertension, kidney failure, malignancy, or who had received antioxidant therapy in the previous two months, or who were pregnant, were excluded from both the control and patient groups.

Patients diagnosed with DM1 were classified as follows: 1) no muscle weakness, 2) minimal findings, 3) distal weakness, 4) mild to moderate proximal weakness, and 5) severe proximal muscle weakness in addition to distal weakness [2].

The ethics approval was obtained from the local committee on 20.03.2024, with decision no. 1522. Informed consent was obtained from the participants involved in the study. The patients' demographic data (age, gender, occupation, treatments received, family history, etc.), clinical complaints such as pain, cramps, fatigue, myotonic dystrophy staging, and functional ambulation level were recorded.

Blood collection

Blood samples were collected within tubes containing a clot activator and gel separator (BD Vacutainer® SST-II Advance, Plymouth, UK) after 8–12 h of fasting of patients. After centrifugation at 1,500×*g* for 10 min, the separated serum samples were aliquoted and stored at −80 °C until analysis. The samples were thawed at room temperature and mixed thoroughly before analysis.

Oxidative stress markers

Serum IMA, TOS, and TAS were analyzed using Rel Assay kits (Lot number: KM24017I, MT23151O, and MT23137A, respectively) (Rel Assay Diagnostics, Mega Tip, Gaziantep, Turkey) on Roche Cobas E801 autoanalyzer (Roche Diagnostics, Mannheim, Germany). The intra-assay and inter-assay coefficients of variation for TOS and TAS are below 3 %, and for IMA, they are below 9 % [9].

Serum IMA, a modified form of albumin, was measured spectrophotometrically using the albumin cobalt binding (ACB) test developed by Bar-Or et al. The N-terminal of normal albumin combines with Co(II) when cobalt chloride solution is added, and the concentration of free Co(II) in the solution decreases. When the serum of patients with myocardial ischemia contains IMA, the ability of albumin to bind to Co(II) decreases, so free Co(II) concentration increases. High-free Co(II) concentration forms colored products when adding dithiothreitol (DTT). The change of absorbance at 470 nm is proportional to the concentration of free Co(II). Serum IMA was reported as absorbance units [10].

Ischemia-modified albumin to albumin ratio (IMAR) is used to correct IMA values for serum albumin concentrations. IMAR was calculated as follows: [(serum albumin level in individual/median albumin level in the population) × IMA] and reported as absorbance units [11].

Total antioxidant capacity was measured using a colorimetric method developed by Erel et al. According to this method, the most potent free radical, hydroxyl radical, is generated by mixing ferrous ions with hydrogen peroxide. Then, the specimen's antioxidant effect against the oxidative reactions started by the produced hydroxyl radical is measured. In the test, antioxidants in the sample reduce dark blue-green colored 2,2′-azino-bis (3-etilbenzotiyazolin-6-sülfonik asit) (ABTS) radical to colorless reduced ABTS form. The change of absorbance at 660 nm is related to the total antioxidant level of the sample. The assay is calibrated using a stable antioxidant standard solution, traditionally called Trolox Equivalent, a vitamin E analog. Serum TAS level was reported as mmol Trolox equivalent/L [12].

Serum TOS level was measured using a colorimetric method developed by Erel et al. [7]. This method is based on the oxidation of ferrous ions to ferric ions by oxidants in the sample in a medium containing abundant glycerol molecules that cause the oxidative reaction. In the acidic medium, these ferric ions form a colored complex with xylenol orange. The color intensity, which can be measured at 560 nm, is related to the total amount of oxidant molecules in the sample. The assay is calibrated with hydrogen peroxide, and the results are expressed as µmol H_2O_2 equivalent/L.

OSI indicates the imbalance between lipid peroxidation products and total antioxidant capacity. This index receives a high value when oxidative stress increases. Before calculation, TAS is converted from mmol Trolox equivalent/L to µmol Trolox equivalent/L. OSI was calculated as follows: [(TOS, µmol H_2O_2 equivalent/L)/(TAS, µmol Trolox equivalent/L) × 100], and reported in arbitrary unit [13].

Statistical analysis

Data analysis was performed using the SPSS version 25.0 statistical package program. Data were presented as mean, standard deviation, frequency, and percentages. The normality of data was analyzed using the Shapiro–Wilk test. Parametric tests were used for variables showing normal distribution, while non-parametric tests were used for those not. Student's *t*-test or Mann–Whitney *U* test was used to compare parameters between patient and control groups. The comparison of non-numerical data was analyzed using the chi-square test. Receiver operating characteristic (ROC) analysis was performed to differentiate the level of oxidative stress markers in the patient and control groups. A p-value of <0.05 was considered statistically significant.

Results

The average age of patients was 43.7 ± 9.3 years, while that of healthy controls was 45.8 ± 8.6 years, with no significant difference observed (p=0.319). The female/male ratios of patient and healthy groups were 28/6 and 16/11, respectively, and were found to be similar (p=0.087). The average duration of illness for DM1 patients was 11.2±4.1 years. The clinic characteristics of the DM1 patient group are shown in Table 1. The majority of DM1 patients were in Stages 1 and 2. A total of 88.8 % had functional ambulation scales 3 and 4.

Table 1: Demographic and clinical characteristics of myotonic dystrophy type 1 patients.

Total number, n	27
Age, years	43.7 ± 9.3
Gender, n (%)	
Female	16 (59.3)
Male	11 (40.7)
Disease stage, n (%)	
Stage 1	2 (7.5)
Stage 2	11 (40.7)
Stage 3	11 (40.7)
Stage 4	3 (11.1)
Functional ambulation scale (FAS), n (%)	
FAS 1	0 (0)
FAS 2	2 (7.4)
FAS 3	11 (40.7)
FAS 4	13 (48.1)
FAS 5	1 (3.8)
Medical treatment, n (%)	
Absent	4 (14.8)
Present	23 (85.2)
Cramps, n (%)	
Absent	1 (3.7)
Present	26 (96.3)
Pain, n (%)	
Absent	7 (25.9)
Present	20 (74.1)
Fatigue, n (%)	
Absent	4 (14.8)
Present	23 (85.2)

The age is given as mean ± standard deviation.

Table 2 shows the biochemical data of DM1 patients and the control group. Statistically significant differences were found between the DM1 patients and the control group in TAS, TOS, OSI, IMA, and IMAR levels. In the patient group, the levels of TOS, OSI, IMA, and IMAR, indicators of oxidative stress, were high, while the levels of TAS were low.

The cut-off values, sensitivity, specificity, predictive values, and area under the curve (AUC) of markers used to distinguish between DM1 patients and the control group are shown in Table 3 and Figure 1. OSI, TAS, TOS, IMA, and IMAR AUC values were ordered from highest to lowest.

Table 2: Biochemical data of myotonic dystrophy type 1 patients and control group.

Parameters	Patient group	Control group	p-Values
TAS, mmol Trolox equivalent/L	1.41 ± 0.26	1.66 ± 0.23	<0.001[a]
TOS, µmol H_2O_2 equivalent/L	5.13 ± 2.07	4.34 ± 3.21	0.001[a]
OSI, arbitrary unit	0.36 ± 0.12	0.26 ± 0.18	<0.001[a]
IMA	0.51 ± 0.12	0.41 ± 0.11	0.001[a]
IMAR	0.49 ± 0.10	0.42 ± 0.10	0.004[a]

TAS, total antioxidant status; TOS, total oxidant status; OSI, oxidative stress index; IMA, ischemia-modified albumin; IMAR, ischemia-modified albumin to albumin ratio. The parameters are given as means ± standard deviations. [a]p<0.05 was considered statistically significant.

Table 3: Performance features of oxidative stress markers to distinguish between myotonic dystrophy type 1 patients and control group.

Parameters	Cut-off values	Sens, %	Spec, %	PPV, %	NPV, %	p-Values	AUC (95 % CI)
TAS, mmol Trolox equivalent/L	<1.64	64.7	85.2	84.6	65.7	<0.001[a]	0.772 (0.655–0.889)
TOS, µmol H_2O_2 equivalent/L	>4.53	63.0	85.3	77.3	74.4	0.001[a]	0.754 (0.625–0.883)
OSI, arbitrary unit	>0.25	96.3	70.6	72.2	96.0	<0.001[a]	0.854 (0.747–0.961)
IMA	>0.51	55.6	85.3	75.0	70.7	0.001[a]	0.741 (0.617–0.865)
IMAR	>0.43	77.8	61.8	61.8	77.8	0.004[a]	0.714 (0.581–0.846)

Sens, sensitivity; Spec, specificity; PPV, positive predictive value; NPV, negative predictive value; 95 % CI, 95 % confidence interval; TAS, total antioxidant status; TOS, total oxidant status; OSI, oxidative stress index; IMA, ischemia-modified albumin; IMAR, ischemia-modified albumin to albumin ratio. The parameters are given as means ± standard deviations. [a]$p<0.05$ was considered statistically significant.

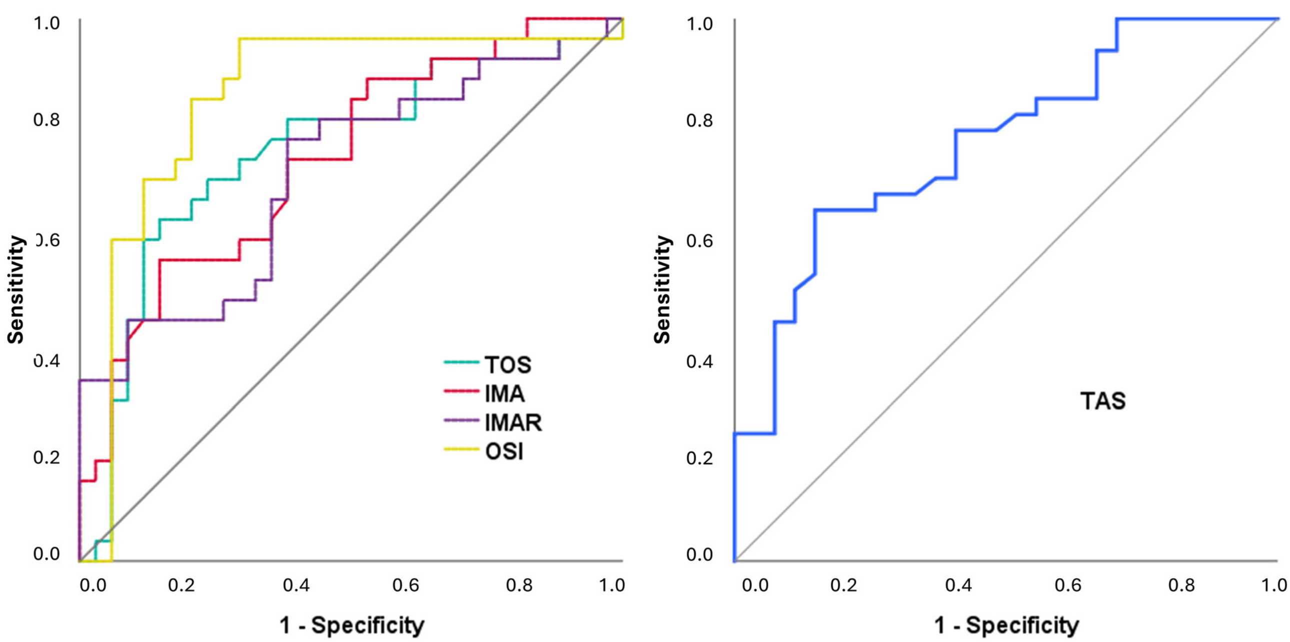

Figure 1: ROC curve of oxidative stress markers to distinguish between DM1 patients and the control group. TAS, total antioxidant status; TOS, total oxidant status; OSI, oxidative stress index; IMA, ischemia-modified albumin; IMAR, ischemia-modified albumin to albumin ratio; ROC, receiver operating characteristic curve; DM1, myotonic dystrophy type 1.

Discussion

In our DM1 patients, which is a very rare disease, we evaluated IMA and IMAR for the first time. The main finding is that the levels of oxidative stress indicators are elevated in DM1 patients.

Oxidative stress has a role in the pathogenesis of many diseases, such as cancer, diabetes, heart diseases, neurodegenerative diseases, and aging [8, 14–17]. Increases in free radical levels and/or decreases in antioxidant activities have been demonstrated in studies of these diseases and the aging process [14]. In DM1, multisystemic muscle weakness, cognitive impairment, cataracts, hearing loss, infertility, fatigue, and premature hair loss resemble a multisystemic disorder associated with early aging, and it is stated that oxidative stress plays a role in the pathogenesis of the disease [18, 19].

Ihara et al. reported increased levels of free radicals and lipid peroxides and decreased antioxidant levels in DM1 patients compared to controls [20]. Usuki and Ishiura suggested that the mutation in myotonic dystrophy (DM) has a role in oxidative stress. They stated that this mutant RNA has a toxic effect on skeletal muscle fibers and leads to clinical symptoms [21]. Similarly, oxidative stress is reported to contribute to the progression of neurodegenerative diseases [15, 16].

In contrast to these studies, Rodriguez and Tarnopolsky evaluated oxidative stress in myotonic dystrophy by measuring the 24-h urinary 8-hydroxy-2′-deoxyguanosine (8-OHdG) excretion. They included patients with muscular dystrophy (MD) (Duchenne MD + Becker MD), with myotonic dystrophy, and healthy controls. A higher ratio of 8-OHdG/creatinine in MD patients was found compared to healthy controls, but this high ratio in myotonic dystrophy patients was not detected. They noted that the dystrophin protein in MD is much more sensitive to free radicals, leading to higher oxidative damage [22].

As far as is known, our study is the first to differentiate oxidative stress status in DM1 patients from the control group. The discriminatory power of oxidative markers was OSI, TAS, TOS, IMA, and IMAR, respectively. If OSI>0.25, it is thought that oxidative stress status in patients may play a role in pathogenesis, and treatment targets can be determined accordingly. Studies have also been conducted in different disease groups. In Fabry disease, which is a metabolic disease and can affect muscles, it was found that the AUC values were 0.6714 in TAS ($p<0.05$), 0.5016 ($p>0.05$) for TOS, and 0.5699 ($p>0.05$) for OSI [23]. These values were lower than those in our study. The cut-off of IMA for acute coronary syndrome was approximately 0.4–0.5, similar to our study [24]. It has been stated that when the IMA level exceeds this threshold, it may indicate the presence of ischemia. However, the literature has no widely accepted cut-off value for DM. In our study, when comparing patient and control groups, the IMA and IMAR levels were found to be significantly higher in the patient group. However, their distinctive features were lower than those of OSI, TAS, and TOS.

Myotonic dystrophy type 1, symptoms that negatively affect daily life, may be observed. Cramps were present in 96 %, fatigue in 85 %, and pain in 74 % of our patients. Our results were similar to the studies of Solbakken et al. [25]. Baldanzi et al. evaluated oxidative stress, fatigue, and daytime sleepiness in DM1 and suggested that oxidative stress contributes to fatigue associated with DM1 [26]. Vitamin E, thiamine (B1), B6, and B12 are non-enzymatic antioxidants. Constantini et al. reported positive developments in muscle strength and daily life activities in DM1 patients after long-term high-dose thiamine therapy. They emphasized that thiamine effectively reduces oxidative stress by activating energy metabolism [27]. It has been demonstrated that patients with DM1 are prone to developing metabolic irregularities and frequently present with conditions of overweight and obesity. This may be attributed to reduced energy expenditure at rest and an impaired oxidative metabolism within muscles [28].

Ileri et al. found higher IMA and thiol-disulfide values in elderly patients with osteosarcopenia compared to healthy controls. They suggested that IMA and thiol-disulfide could be indicators of increased oxidative stress with aging [29]. Therefore, IMA may be an indicator of premature aging in DM1 patients.

Kumar et al. evaluated the oxidative stress markers, including malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPX), glutathione-S-transferase (GST), reduced glutathione (GSH), TAS levels, and clinical symptoms and phenotypes in DM1 patients. They reported significant positive correlations between oxidative stress markers and diabetes mellitus, cardiac creatine kinase MB and MM levels, and negative correlation with facial weakness [30].

OSI has been used to assess the level of oxidative stress, particularly in conditions such as myotonic dystrophy [31]. We also found high levels of OSI in DM1 patients. Indices like this may have a significant role in the pathogenesis of neurodegenerative diseases such as myotonic dystrophy.

It was thought that our study had some limitations. The sample size was limited to 27 DM1 patients and 34 healthy individuals. The small sample size limits the generalization of the results. Studies with a larger sample size would allow for more robust and reliable results. Although the present study concentrated on oxidative stress markers, it did not comprehensively analyze the relationship between clinical symptoms (e.g., muscle strength, fatigue, or activities of daily living) and oxidative stress markers in patients with DM1. A detailed investigation of the relationship between oxidative stress markers and clinical characteristics can be recommended.

The lack of standardization and traceability in the ACB test and differences in the IMA unit limit the comparability of IMA measurements in clinical studies [32]. Without considering these technical problems, positive results were obtained in meta-analysis studies that evaluated IMA measurement only from a clinical perspective [33]. Therefore, standardization in IMA measurement as a promising marker was recommended [32].

According to our study, the high levels of TOS, OSI, IMA, and IMAR and low levels of TAS in DM1 patients may be responsible for the negative effects of free radicals on patient clinical outcomes. Medical research for DM1 patients is ongoing, especially focusing on antioxidant therapy. The increase in oxidative stress can disrupt intracellular mitochondrial functions, slowing energy metabolism and causing structural damage to muscle cells. Additionally, the damage caused by reactive oxygen species can heighten inflammation in muscle tissue, potentially worsening the progression of the disease. This inflammatory response contributes to muscle degeneration and may accelerate the clinical course of disorders like DM1. Moreover, oxidative stress can overwhelm the antioxidant defense systems, impairing mitochondrial function and contributing to muscle weakness and damage [34]. Therefore, antioxidant therapies aimed at reducing oxidative stress in DM1 patients have received increasing attention in recent years. Although animal studies have been conducted on antioxidants such as vitamin C, vitamin E, coenzyme Q, carnitine, N-acetylcysteine, metformin, and rapamycin, clinical research is needed to evaluate the effectiveness of these therapeutic approaches in humans [31, 34, 35].

Our study is the first to evaluate IMA and IMAR levels as an oxidative stress marker in DM1. Comprehensive studies involving more patients and evaluating the relationship of oxidative stress markers with clinical parameters are believed to shed light on the pathophysiology and treatment of the disease.

Research ethics: Informed consent was taken from patients.
Informed consent: Informed consent was taken from patients.
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