

Effects of childhood psychological trauma on rheumatic diseases

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Abstract

Objective: The etiology of rheumatic diseases is unclear, but it is thought that environmental factors added to immunogenetic mechanisms in chronic inflammatory diseases play a role. Many inflammatory disorders, autoimmune diseases, and painful conditions have been shown to be associated with the psychological trauma of childhood. The aim of the present study was to investigate childhood psychological trauma that is considered to be one of the environmental factors that initiate inflammation on patients with rheumatic diseases.

Methods: In our study, a total of 440 patients (220 patients who have rheumatic diseases as the case group and 220 patients who have no rheumatic disease as the control group) were examined. The Childhood Trauma Questionnaire-28 (CTQ-28) was administered and was completed by the patients. This was a cross-sectional study design.

Results: No statistically significant differences were found between the case and control groups with respect to age, gender, marital status, and educational level. The CTQ-28 scale was found to be significantly higher in patients with rheumatic diseases (ankylosing spondylitis, rheumatoid arthritis, and connective tissue disease) in our study.

Conclusion: We think that childhood psychiatric traumas are effective in the etiopathogenesis of rheumatic diseases. To make this relationship more understandable, multidisciplinary research and long-term follow-up studies are needed to examine neuroendocrine, genetic, and epidemiological factors.

Keywords: Rheumatic diseases, childhood trauma, hypothalamic–pituitary–adrenocortical axis

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Introduction

The etiology of rheumatic disorders has not been fully elucidated; however, it is thought that environmental factors, together with immunogenetic mechanisms, play a role in the development of chronic inflammatory disorders (1). It has been shown that many inflammatory disorders, autoimmune diseases, and painful conditions are associated with psychological trauma in childhood (2). Negative experiences during childhood appear to be associated with psychiatric disorders, as well as mortality causes, in adults (3). The assumption that adverse experiences in early life can result in immunological sequelae has been investigated in short- and long-term animal studies (4). The neuroendocrine immunological abnormalities occurring in unfavorable childhood can lead to the development of proinflammatory phenotype in adult life (5).

The inflammatory response associated with childhood psychological trauma involves the increased production of proinflammatory cytokines, such as interleukin-6 and tumor necrosis factor-alpha, and C-reactive protein (CRP) (6). It is shown that there is a relationship between negative childhood experiences and CRP elevation (7). Hypothalamic–pituitary–adrenal (HPA) axis is an important regulator of inflammatory activity. Negative experiences in childhood disrupt the regulatory role of HPA, and resultant inflammatory activation plays important roles on physical and mental health (8). Prolonged dysregulation in HPA axis causes decreased glucocorticoid secretion in response to stress; in turn, a decreased glucocorticoid level affects the inflammatory process (7, 9). Confounding factors, such as age, gender, the origin of violence, time from experience of violence, cultural characteristics, and experience of several types of violence in combination, make investigations more complex in this field (10).

The aim of the present study was to compare the effect of childhood trauma on rheumatic disorders occurring on the grounds of inflammatory and autoimmune processes with healthy controls.

Methods

Our case-control study protocol was approved by the Cumhuriyet University Ethics Committee (2013-07/11) in accordance with the ethical principles of the Declaration of Helsinki and with the guidelines for good clinical practice. Written informed consent was obtained from all participants before participation in the study.

A total of 220 patients who were diagnosed with rheumatic disease in the Rheumatology Clinic of Physical Medicine and Rehabilitation, University Hospital were included in the study. The study included 93 male and 127 female patients. The mean age of the patients was 48.97 ± 10.12 (20–65) years.

Inclusion criteria were as follows: age >20 years (the scale used in the present study assesses age <20 years (11)); mental capacity sufficient to read, understand, and answer tests used in the study; no cognitive disorder and/or language problem (ability to involve self-reporting process); and acceptance of the study protocol.

Exclusion criteria were as follows: no serious neurological disease (e.g., cerebrovascular event) or developmental disorder (e.g., mental retardation) that may affect the completion of tests used in the study and presence of a psychiatric disorder that may affect the individual's insight or judgment.

In all subjects, sociodemographic characteristics, such as age, marital status, or educational level, were recorded. All subjects were asked to complete the Childhood Trauma Questionnaire-28 (CTQ-28). The subjects were informed about the duration of the test that lasted for 10–15 min.

The CTQ-28 is a self-reported, Likert-type scale that assesses mistreatment in childhood (age <20 years) in five domains, including emotional, physical, and sexual abuse and physical and emotional neglect (11, 12). It also involves three additional questions that assess minimization or denial. The latter questions were included to achieve more accurate assessment of results. The questions are rated by a 5-point Likert scale as follows: 1, never true; 2, rarely true; 3, sometimes true; 4, often true; and 5, very often true. While scoring, scores from positive statements are inverted (items 2, 5, 7, 13, 19, 26, and 28). The individual items are summed to obtain subscales from 5 to 25 points. The total score ranges from 25 to 125 points. The scores of items 10, 16, and 22 (which are related to minimization) are not

inverted since these items assess denial and have no influence on total score. The cut-off values are as follows: ≥ 13 points for emotional abuse, ≥ 10 points for physical abuse, ≥ 8 points for sexual abuse, ≥ 15 points for emotional neglect, and ≥ 10 points for physical neglect. It allows the estimation of separate scores for traumatic experience subscales and total score (11–13). Turkish validation and reliability studies were performed (11).

Statistical Analysis

All data were analyzed using Statistical Packages of Social Sciences version 23.0 (IBM Corp.; Armonk, NY, USA). Data obtained from the groups are expressed as mean \pm standard deviation. A p value <0.05 was considered as statistically significant. Chi-square test was used to compare sociodemographic characteristics within the groups, whereas Student's t-test was used to compare the groups. In addition, Pearson's or Spearman's correlation test was used to assess some parameters within the groups. One-way ANOVA and post hoc Tukey's HSD tests were used to perform further analyses according to disease subtypes in patients with rheumatic diseases. A p value <0.05 after Bonferroni correction was considered as statistically significant.

Results

There was no significant difference in age, gender, marital status, and educational level between the patient and control groups (Table 1). In the patient group, there were 87 (39.5%)

patients with ankylosing spondylitis (AS), 82 (37.3%) patients with rheumatoid arthritis (RA), 51 (24.2%) patients with collagen tissue disorder (21 cases with systemic lupus erythematosus, 17 cases with scleroderma, 7 cases with Behçet's disease, and 6 cases with Sjögren syndrome).

In the CTQ-28, the total score was 41.28 ± 12.51 points in the patient group, whereas it was 33.60 ± 8.64 points in the control group, indicating a significant difference between the groups ($p=0.001$; $t=7.493$). All subscale scores were found to be significantly higher in the patient group than in the control group ($p=0.001$) (Table 2).

The CTQ-28 subscales were assessed in disease subtypes in the patient group. No significant difference was found among patients with AS, RA, or collagen tissue disorder (Table 3).

Discussion

The present study was based on the hypothesis that childhood psychological trauma could be more common in patients with rheumatic disorder than in healthy controls. Our results favor that exposure to traumatic experiences in childhood increases the risk for the development of rheumatic disorders in adult life.

In many studies, it was shown that long-term psychological trauma causes the dysregulation of HPA axis and decreases glucocorticoid lev-

Table 1. Comparison of sociodemographic characteristics of the patient and control groups

	Patient group (n=220)	Control group (n=220)		p
Age (mean \pm SD), years	48.97 \pm 10.12	50.75 \pm 12.48	t=1.63	0.103
Gender (%)				
Female	127 (57.7%)	116 (57.7%)	$\chi^2=1.11$	0.29
Male	93 (42.3%)	104 (47.3%)		
Marital status (%)				
Married	167 (75.9%)	178 (80.9%)	$\chi^2=1.82$	0.40
Single	33 (15.0%)	28 (12.7%)		
Divorced	20 (29.1%)	14 (6.4%)		
Educational level, n(%)				
Low (primary–middle school)	129 (58.6%)	126 (57.3%)	$\chi^2=0.49$	0.97
Medium (high school)	55 (25.0%)	59 (26.8%)		
High (university or higher education)	36 (16.4%)	35 (15.9%)		

Table 2. Comparison of the CTQ-28 between the patient and control groups

CTQ-28	Patient group (n=220)	Control group (n=220)	t	p
	Mean±SD	Mean±SD		
Physical abuse	6.24±2.67	5.41±1.80	3.829	0.001
Emotional abuse	7.39±3.08	5.87±1.85	6.240	0.001
Sexual abuse	5.40±1.76	5.02±0.18	3.209	0.001
Physical neglect	9.35±3.63	7.32±2.62	6.712	0.001
Emotional neglect	12.89±5.14	9.96±4.43	6.398	0.001
CTQ-28 total	41.28±12.51	33.60±8.64	7.493	0.001

CTQ: Childhood Trauma Questionnaire; SD: standard deviation; t: Student's t-test was used
 Bold data indicate p<0.001

Table 3. Evaluation of the relationship with CTQ subscales between subgroups of rheumatic diseases

CTQ-28	RA (n=82)	AS (n=87)	Collagen tissue disorder (n=42)	p
	mean±SD	mean±SD		
Physical abuse	6.68±3.38	5.92±1.99	6.07±2.47	0.168
Emotional abuse	7.74±3.32	7.05±2.61	7.29±3.45	0.334
Sexual abuse	5.59±1.91	5.23±0.80	5.07±0.26	0.073
Physical neglect	9.89±4.05	8.85±3.24	9.38±3.55	0.180
Emotional neglect	13.61±4.98	12.05±5.04	12.88±5.39	0.138
CTQ-28 total	43.51±13.63	39.09±10.92	40.69±12.29	0.066

CTQ: Childhood Trauma Questionnaire; RA: rheumatoid arthritis; AS: ankylosing spondylitis

els released in response to stress, altering the neurohumoral response against stress (14, 15). In a prospective study from Canada, it is suggested that the risk for arthritis is increased in individuals exposed to childhood trauma (16). Our results are in agreement with the literature.

In a retrospective cohort from the USA, it is suggested that childhood trauma increases the likelihood for admission due to autoimmune disorder during adult life (17). In another study comparing patients with RA and healthy controls, it was reported that the risk for RA could be increased by exposure to trauma in childhood in individuals with genetic vulnerability, emphasizing the need for further studies in this field (18). In our study, the total score in the CTQ-28 was found to be significantly higher in patients with rheumatic disease than in controls; however, no significant difference was found among disease subtypes (AS, RA, and collagen tissue disorder) within the patient group.

Based on our results, it could be suggested that childhood trauma may trigger or at least increase the risk for autoimmune inflammatory disorders; however, no conclusion could be

drawn about which disease type has a stronger relationship with such disorders.

In a Swedish study, patients with RA with or without psychological problems at the time of diagnosis were followed up for 2 years. It was observed that the disease had a poorer prognosis in patients with RA with psychological problems at the time of diagnosis (19). Similar to our study, this finding also supports that negative experiences in early life may trigger or at least worsen inflammatory diseases.

In a retrospective study of patients with psoriatic arthritis, it was suggested that negative experiences in childhood could be linked to the disease (20).

All studies, including case reports, original investigations, and epidemiological research, conducted on this field so far favor the role of environmental factors in the development of rheumatic disorders. However, our understanding of the linkage between environmental risk factors and rheumatic disorders is insufficient due to several reasons, including heterogeneity in phenotype and pathogenesis of rheumatic

disorders, quantitative assessment of environmental exposure, and poorer capacity of assessment for the etiology of these complex disorders (21). In our study, factors with the potential to influence on disease could not be excluded due to the chronic nature of the diseases.

In the literature, there are studies investigating childhood trauma in patients with fibromyalgia syndrome, RA, or psoriatic arthritis; however, childhood trauma has not been studied in patients with AS so far. In addition, childhood trauma was investigated in a disease group alone or by comparing to one disease or control or to two diseases without control in previous studies. To the best of our knowledge, this is the first study that compared patients with several chronic inflammatory diseases, including AS, RA, and collagen tissue disorder, with healthy controls.

Our study has some limitations. First, there may be the likelihood of bias and/or inaccurate responses due to the use of self-reported tools in the study as subjects might not want to remember some experiences. In addition, subjects might not be objective enough due to failure to recall or discounting. Since we assessed subjects in one occasion, factors with the potential to have a negative impact on disease could not be assessed.

In our study, the CTQ-28 score was found to be higher in patients with rheumatic disease. We think that childhood trauma plays a role in the etiopathogenesis of rheumatic disorders. There is a need for multidisciplinary and long-term investigations on neuroendocrine, genetic, and epidemiological factors for better understanding of this relationship.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Cumhuriyet University School of Medicine (No: 2013-07/11).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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References

1. Düzgün N. Romatizmal Hastalıkların Tanımı ve Sınıflandırılması. *Türkiye Klinikleri J Immunol Rheumatol* 2002; 2: 3-5.
2. Bayram K, Erol A. Childhood Traumatic Experiences, Anxiety, and Depression Levels in Fibromyalgia and Rheumatoid Arthritis. *Noro Psikiyatr Ars* 2014; 51: 344-9. [\[CrossRef\]](#)
3. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, et al. The relationship of adult health status to childhood abuse and household dysfunction. *Am J Prev Med* 1998; 14: 245-58. [\[CrossRef\]](#)
4. Zeugmann S, Buehrsch N, Bajbouj M, Heuser I, Anghelescu I, Quante A. Childhood maltreatment and adult proinflammatory status in patients with major depression. *Psychiatr Danub* 2013; 25: 227-35.
5. Chida Y, Sudo N, Sonoda J, Hiramoto T, Kubo C. Early-life psychological stress exacerbates adult mouse asthma via the hypothalamus-pituitary-adrenal axis. *Am J Respir Crit Care Med* 2007; 175: 316-22. [\[CrossRef\]](#)
6. Coelho R, Viola TW, Walss-Bass C, Brietzke E, Grassi-Oliveira R. Childhood maltreatment and inflammatory markers: a systematic review. *Acta Psychiatr Scand* 2014; 129: 180-92. [\[CrossRef\]](#)
7. Danese A, Pariante CM, Caspi A, Taylor A, Poulton R. Childhood maltreatment predicts adult inflammation in a life-course study. *Proc Natl Acad Sci U S A* 2007; 104: 1319-24. [\[CrossRef\]](#)
8. Baumeister D, Akhtar R, Ciufolini S, Pariante CM, Mondelli V. Childhood trauma and adulthood inflammation: a meta-analysis of peripheral C-reactive protein, interleukin-6 and tumour necrosis factor- α . *Mol Psychiatry* 2016; 21: 642-9. [\[CrossRef\]](#)
9. Jacobson L, Sapolsky R. The Role of the Hippocampus in Feedback Regulation of the Hypothalamic-Pituitary-Adrenocortical Axis. *Endocr Rev* 1991; 12: 118-34. [\[CrossRef\]](#)
10. Keeshin BR, Cronholm PF, Strawn JR. Physiologic changes associated with violence and abuse exposure: an examination of related medical conditions. *Trauma Violence Abuse* 2012; 13: 41-56. [\[CrossRef\]](#)
11. Şar V, Öztürk E, İkikardeş E. Validity and Reliability of the Turkish Version of Childhood Trauma Questionnaire. *Türkiye Klinikleri J Med Sci* 2012; 32: 1054-63. [\[CrossRef\]](#)
12. Grilo CM, Masheb RM. Childhood Psychological, Physical, and Sexual Maltreatment in Outpatients with Binge Eating Disorder: Frequency and Associations with Gender, Obesity, and Eating-Related Psychopathology. *Obes Res* 2001; 9: 320-5. [\[CrossRef\]](#)
13. Bernstein DP, Fink L. *Childhood Trauma Questionnaire: A retrospective self-report manual* San Antonio, TX: The Psychological Corporation; 1998.
14. Dorn LD, Burgess ES, Susman EJ, Eye A von, De Bellis MD, Gold PW, et al. Response to oCRH in depressed and nondepressed adolescents: Does gender make a difference? *J Am Acad Child Adolesc Psychiatry* 1996; 35: 764-73. [\[CrossRef\]](#)
15. Luecken L.J. Childhood attachment and loss experiences affect adult cardiovascular and cortisol function. *Psychosom Med* 1998; 60: 765-72. [\[CrossRef\]](#)
16. Kopec JA, Sayre EC. Traumatic experiences in childhood and the risk of arthritis: a prospective cohort study. *Can J Public Health* 2004; 95: 361-5.
17. Dube SR, Fairweather D, Pearson WS, Felitti VJ, Anda RF, Croft JB. Cumulative Childhood Stress and Autoimmune Diseases in Adults. *Psychosom Med* 2009; 71: 243-50. [\[CrossRef\]](#)
18. Spitzera C, Wegertb S, Wollenhauptb J, Wingenfeldc K, Barnowd S, Grabee HJ. Gender-specific association between childhood trauma and rheumatoid arthritis: A case-control study. *J Psychosom Res* 2013; 74: 296-300. [\[CrossRef\]](#)
19. Gåfväls C, Hägerström M, Nordmark B, Wändell P. What predicts negative effects of rheumatoid arthritis? A follow-up two years after diagnosis. *SpringerPlus* 2014; 3: 118. [\[CrossRef\]](#)
20. Simonić E, Peternel S, Stojnić-Soša L, Rončević-Gržeta I, Prpić-Massari L, Massari D, et al. Negative and positive life experiences in patients with psoriatic arthritis. *Rheumatol Int* 2013; 33: 1587-93. [\[CrossRef\]](#)
21. Gourley M, Miller FW. Mechanisms of disease: Environmental factors in the pathogenesis of rheumatic disease. *Nat Clin Pract Rheumatol* 2007; 3: 172-80. [\[CrossRef\]](#)