

## MN-SOD POLYMORPHISM IN CHRONIC PELVIC PAIN SYNDROME PATIENTS

*Elif D Buyuktuncer\*, Serdar Arisan, Murat Can Kiremitci, Hatice Tigli, Narcin Palavan Unsal, Turhan Caskurlu, Erbil Ergenekon, Istanbul, Turkey*

**INTRODUCTION AND OBJECTIVE:** Although chronic pelvic pain syndrome (CPPS) is a common illness, its etiology is still investigated. We evaluated the relationship of an alanine or valine polymorphism at amino acid sequence 16 [Val(16)Ala] of manganese superoxide dismutase (MnSOD) CPPS with patients. In some studies showed that second exon of Mn-SOD which targets mitochondrial transcription regions cause mitochondrial damage and dysfunction of some signal protein when Ala-9-Val (GCT/GTT) polymorphism is occurred. We evaluated the relationship of an alanine or valine polymorphism at amino acid sequence 16 [Val(16)Ala] of manganese superoxide dismutase (MnSOD) with CPPS and healthy donors. Also we investigated their Mn-SOD activity in blood and sperm samples.

**METHODS:** Val(16)Ala genotyping of Mn-SOD was done by polymerase chain reaction-restriction fragment length polymorphism with a restriction enzyme (Bsa<sup>1</sup>) in 72 Turkish CPPS patients and 29 healthy subjects. The genotype distribution of CPPS and healthy subjects was then compared, and the association of genotype with NIH classification was evaluated in the CPPS patients. All DNA samples were extracted from blood (Qiagen, DNA mini blood kit). Enzyme activity was determined in blood and sperm samples (RANSOD, SOD activity kit). Sperm motility and characteristics, Endtz test were investigated for each donor.

**RESULTS:** The VV type showed a significantly higher frequency in the CPPS with 3a NIH classification than did the AA or VA type. The frequency of -9Ala allele was 0,17 (10/53) and -9Val allele frequency was 0,83 (43/53) in all patients. In control group, -9Ala frequency was 0,29 (5/17) and -9Val allele frequency was 0,71 (12/17). Ala/Val genomic distribution was 0,40 (7/17) and Val/Val genomic distribution was 0,60 (10/17) for control group. None of the group members have Ala/Ala genomic distribution. Ala/Val genomic distribution frequency for patients was 0,27 (14/53) and Val/Val frequency was 0,73 (39/53). Furthermore, logistic regression analysis showed that this polymorphism is associated with CPPS (odds ratio = 0.461925, P = 0.03). Enzyme activity was lower in CPPS patients than control subjects (P= 0.456). There was no difference between type 3a and type 3b patients enzyme profile.

**CONCLUSIONS:** Accordingly, the Val(16)Ala polymorphism of Mn-SOD may be responsible one of the mechanism etiology of type 3a and type 3b CPPS. Oxidative stress effect on CPPS should be investigated more detail.

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