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Short Communication

Paraoxonase levels in acute mushroom poisoning cases treated in emergency department

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The present study was undertaken to investigate the activities of paraoxonase in acute mushroom poisoning cases. This investigation was made between June 2009 to June 2010 with 64 mushroom poisoning patients who admitted to our emergency department in which we analyzed the 6 and 48 h paraoxonase 1 (PON 1) levels. Mean value of PON1 A(6 h) was 74.83 ± 41.68 U/ml and PON1 B(48 h) 91.90 ± 51.08 U/ml. The difference was statistically significant (p = 0.001) Mushroom poisoning cases, which are a public health problem, are frequently reported in Europe, United States, and in the Far East. Paraoxonase levels can be used as prognostic markers for acute mushroom poisoning.

Key words: Mushroom poisoning, paraoxonase, prognostic markers, emergency services, oxidative stress.

INTRODUCTION

Of more than 2000 identified mushroom species throughout the world, only about 50 are toxic for humans (Mas, 2005; Broussard et al., 2001; Diaz, 2005). Mushroom poisoning cases, which are a public health problem, are frequently reported in Europe, in the United States, and in the Far East (Mas, 2005; Broussard et al., 2001; Diaz, 2005; Yilmaz et al., 2006).

There have been frequent cases of mushroom poisoning also in Turkey in recent years (Yilmaz et al., 2006; Unluoglu and Tayfur, 2003).

Biomarkers, such as paraoxonase, occur as a result of hereditary or environmental influences. They are sensitive indicators in individuals to a special xenobiotic or xenobiotic groups with similar characteristics (Akay, 2004). The oxygen analogs are hydrolyzed by the serum A-esterase, paraoxonase (PON1), which appears to play a central role in their detoxification and in their toxicity (Costa et al., 1999; Akgür et al., 2003). PON1 exhibits a substrate dependent polymorphism. This polymorphism is related with two polymorphic sites at amino acid positions 55 and 192 (La Du et al., 1999; Furlong et al., 2000).

The gene frequency for PON1R192 allele is 0.31 in Caucasian population, 0.41 in Hispanic populations, 0.66 in a Japanese population (Furlong et al.,2000) and 0.31 in Turkish population (Aynacioglu et al., 1999). Following the first description of serum A-esterase by Aldrige in 1953, interest to paraoxonase became popular in the field of toxicology (Mackness et al., 1996).

The present study was undertaken to investigate the activities of paraoxonase, in acute mushroom poisoning cases.

MATERIALS AND METHODS

This is a cross-section study made with 64 mushroom poisoning patients who admitted between June 2009 - June 2010 to our emergency department. We analyzed the 6th and 48th h paraoxanase 1 (PON 1) levels.

The patients with mushroom poisonings were included in the study. Patients with chronic liver disease, type 2 DM, coronary heart disease, elevated liver functions without any diagnosis and hiperlipidemia were excluded.

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Table 1. Mean value of PON1 A and PON1 B.

	Mean ± SD	p
PON1 A	74.83 ± 41.68	0.001
PON1 B	91.90 ± 51.08	

Blood samples

Blood specimens were obtained at the 6th (PON1A) and 48th h (P PON 1B) from acute mushroom poisoning cases who admitted to Emergency Service. The blood samples were analyzed within the same day or they were collected at -20°C for further analysis, usually done within 5 week, after centrifugation.

Enzyme activity assays

PON1 enzyme activity was performed with a spectrophotometer by measuring p-nitrophenol absorbance increase in a minute, at 25°C ambient temperature and 412 nm. P-nitrophenol was obtained by paraoxan hydrolysis. Serum PON1 activity determination was performed by modification of Eckerson, Furlang, Juretic, and Mackness methods (Juretic et al., 2001).

Statistical analysis

Statistical Package for the Social Sciences 16.0 was used for statistical analysis. The PON1 groups means differences were tested with paired-sample T test. Demographic features were demonstrated with arithmetical mean ± SD and $p<0.05$ values were accepted as significiant.

Ethical approval (02-06-2009) was taken from the ethics committee.

RESULTS

Sixty four patients, 37 female and 27 male, with acute mushroom poisoning admitted to the emergency department. The mean age for the female and males is 39 ± 20.83 and , 36 ± 16.68 years, respectively. All of the patients were discharged within 72 h and none of the patient died.

Mean value of PON1 A was 74.83 ± 41.68 U/ml and PON1 B 91.90 ± 51.08 U/ml. The difference was statistically significant (p=0.001) Table 1.

DİSCUSSİON

Following the description of serum A-esterase by Aldrige in 1953, interest to paraoxonase became popular in the field of toxicology (Mackness et al., 1996).

It is shown that red wine and polyphenols (quercetin, catechin) increase PON1 activity whereas smoking inhibits PON1 activity in a dose- and time-dependent manner in mice with Apo B deficiency (Laplaud et al., 1998).

The effects of diets rich in lipid oxidation products on serum PON1 activity and low density lipoprotein (LDL) oxidation susceptibility in humans is not completely clarified. Sutherland et al. (Sutherland et al., 1999) compared the effect of a meal rich in oxidized lipids in the form of fat that had been used for deep-frying in a fast food restaurant and a control meal rich in the corresponding unused fat on postprandial serum paraoxonase (arylesterase) activity. Four hours into the postprandial period, serum paraoxonase activity had decreased significantly after the used fat meal (-17%, p=0.005) and had increased significantly after the meal rich in unused fat (14%, p=0.005).

In healthy middle-aged men with low risk coronary artery disease it has been shown that low to moderate alcohol intake increase PON1 activity, this increase is associated with modest increases of high dansity lipoprotein (HDL) levels (Van der Gag et al., 1999). Blatter et al., (Blatter et al., 1997) detected that the lipid content of the diet regulated PON specific activity by external influences on lipoprotein particles. PON1's most well-known protective function in humans is the ability to hydrolyze organophosphate nerve agents, aromatic carboxylic acid esters and insecticides. Paraoxon, potent inhibitor of acetylcholine esterase, leads to accumulation of acetylcholine in the synaptic junction by sequential stimulation of neurons (Mackness et al., 1996). The detoxification reaction takes place in the liver while glutathione-S transferase, mono-oxygenase and paraoxonase enzymes are more capable. The effects of hydrolytic products occured by PON1 comprising paraoxons itself is relatively harmless (Mackness et al., 1996; Laplaud et al., 1998).

In fact there are many studies about PON1 effect in organophosphate poisonings, whereas PON1 effect in mushroom poisoning cases is not examinated in the literature (Akgür et al.,2003; Akgür et al., 1999; Stevens et al., 2008; Li et al.,1993).

While organophosphates are synthetic compounds, which are normally not found in nature the detoxification by paraoxonase may be a coincidence (Azarsız and Sözmen, 2000).

In some studies it was reported that PON1 activity decreased in serum among patients with diabetic retinopathy and hypertension due to increased lipid peroxidation susceptibility (Mackness et al., 2000; Sözmen et al., 1999). PON1 activity increased for a week after Pyloric stenosis surgery in infants(Mackness et al., 1996). While the relationship between Alzheimer's disease

and atherosclerosis is known, PON polymorphism has been examined, but the statistical results were not significant (Sodcyama et al., 1999). Coronary heart disease was observed in elderly among south asian origin neonates with low birth weight and PON2 homozygous A148/AI48 enzyme (Busch et al., 1999).

Serum PON1 levels are influenced by diet, acute phase proteins, pregnancy and events that affect the level of apo AI (Azarsız and Sözmen, 2000).

PON1 is an HDL-associated enzyme with antioxidant and antiatherogenic functions, protecting LDL lipo-proteins against oxidative modification. (Pannbacker, 2003; Mackness et al., 1997). Also it is reported that PON1 decreases in chronic renal disease (Paragh et al., 1999).

Studies examining PON1 activity in cases among the patients with acute mushroom poisoning are not included in the literature. In our study PON1 B activities were higher than PON1 A. We think that the lower enzyme level in the first stage (PON1 A) can be explained by it's use for detoxification, is replaced by the liver after the acute phase of mushroom poisoning (PON1 B). If the PON1 level shows an increasing trend during the follow up period this may indicate that the patient is in the recovery period. Also this can be a predictor for prognosis, and can be used for discharge; if the patient has nor clinical symptoms neither pathological laboratuary results for mushroom poisoning.

While the study was made with limited number of patients and none of them had severe intoxication signs, only gastrointestinal symptoms were seen, the results and clinical value of PON1 can be investigated with a larger patient trial who has severe mushroom intoxications signs. But still, this type of small-scale studies may guide for future works to be done.

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