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Does Fluoride Toxicity Cause Hypertension in Patients with Endemic Fluorosis?

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We have read the article published by Amini et al. with a great interest [1]. They have examined the relationship of fluoride (F) in groundwater resources of Iran with the blood pressure of Iranian population in an ecologic study. They have found statistically significant positive correlations between the mean concentrations of F in the groundwater resources and the hypertension prevalence of males, females, and overall. They have also found statistically significant positive correlations between the mean concentrations of F in the groundwater resources and the mean SBP of males and a borderline correlation with females. We congratulate the authors for this clinical study. There was no previous study evaluating the relationship between F toxicity and blood pressure in clinical setting. There is only one experimental study that has been done in laboratory animals. In this animal study, in which Wistar rats were used, there was a significant correlation between the blood pressure of male rats and perinatal exposure to sodium F. Moreover, perinatal exposure to sodium F in Wistar rats resulted in a dose-dependent, long-lasting functional impairment in hemodynamic control [2].

The main source of fluoride for humans is the intake of groundwater contaminated by geological sources (maximum concentrations reaching 30–50 mg/l). The level of fluoride contamination is dependent on the nature of the rocks and the occurrence of fluoride-bearing minerals in groundwater. Fluorosis is a worldwide health problem and is endemic in some areas where fluoride content is high in drinking water. In our city, Isparta, which is located in the southwest part of Turkey, fluoride content of natural drinking water is high in some parts of the city. As a result, fluorosis is still endemic in some parts of our city [3]. According to Isparta Health Organization data, the mean fluoride level in drinking water was 2.74±064 ppm in the endemic fluorosis region and 0.53±0.06 mg/dl in the non-endemic region [3].

The effect of chronic F exposure on the cardiovascular system in human being is a missing issue. In our previous two studies, we have examined the effect of chronic F exposure on the cardiovascular system of endemic fluorosis patients living in endemic fluorosis area in our province. We have shown that aortic elasticity and left ventricular diastolic and global functions were impaired in patients with endemic fluorosis [4, 5]. The assessment of aortic stiffness which is calculated from pulsatile changes in ascending aorta by echocardiography is an important determinant of vascular changes and left ventricular function. Aortic stiffness is a marker of cardiovascular disease including hypertension and/or an independent risk factor for cardiovascular mortality All patients were selected according to Wang criteria, as described by Wang et al.: (1) living in the endemic fluorosis region since birth, (2) having mottled tooth enamel, indicating dental fluorosis, (3) consuming water with fluoride levels above 1.2 mg/l (normal 1 mg/l), and (4) a urine fluoride level greater than 1.5 mg/l. (normal <1.5 mg/l) [6]. The mean ages were 33.9±8.6 and 32.7± 8.8 years old, respectively, in our two studies. We have also looked at systolic and diastolic blood pressures of patients in these two studies. There were no significant differences between fluorosis patients and the controls with respect to the systolic and diastolic blood pressures. Perhaps low-dose chronic F toxicity does not reach to significant level to affect blood pressure in endemic fluorosis patients. All systolic and diastolic blood pressures values were in normal range as in study by Amini et al. Although F induces an increase in systolic and diastolic blood pressure on laboratory animals in experimental level, there is no clear evidence that this entity is not absolutely true in patients with endemic fluorosis. Hypertension is a complex disease. Perhaps low-dose

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chronic F toxicity does not reach to significant level to affect blood pressure in endemic fluorosis patients.

Oxidative stress is a recognized mode of action of fluoride exposure that has been observed in vitro in several types of cells and also in vivo in different organ systems in animals and in people living in areas of endemic fluorosis [7]. Oxidative stress has been implicated in chronic diseases including hypertension [8]. Although oxidative stress may not be the sole cause of hypertension, it amplifies blood pressure elevation in the presence of other prohypertensive factors (salt, renin-angiotensin system, and sympathetic hyperactivity). It is clear that oxidative stress is important in the molecular mechanisms associated with cardiovascular and renal injury in hypertension. As a result, oxidative stress that is present in F toxicity can also cause tendency in increasing blood pressure. On the other hand, we can speculate that chronic low-dose fluoride toxicity may not be the sole cause of hypertension in patients with endemic fluorosis. Drinking water F concentrations might be an important determinant factor in the occurrence of hypertension.

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