

The elevation of oxidative stress after the great East Japan earthquake

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To the Editor

At 1446 JST on 11 March 2011, Eastern Japan, including the catchment area of our hospital (Jikei University Kasai Hospital), was struck by the great East Japan earthquake—the most destructive known earthquake in this country, and one of the five most powerful earthquakes in the world overall since 1900. Residents of our catchment area suffered from severe physical and psychological stress because of daily life damages such as blackouts, cutoffs of the water supply, paralysis of public transportation and liquefaction of the ground.

During physical and physiological stress, the underlying mechanisms involved are the activation of the hypothalamic–pituitary–adrenal axis and sympatho-adrenal-medullary systems, causing the release of corticosterone together with the release of catecholamines. Furthermore, the elevation in catecholamine levels generates free radicals, which may accelerate oxidative stress [1]. It is therefore supposed that earthquake stress could have induced the elevation of oxidative stress; however, oxidative stress after severe earthquakes has not been evaluated as of yet. Thus, we investigated the level of oxidative stress in CKD patients before and after this disaster.

We used the “redox state of human serum albumin (HSA)” as a marker of oxidative stress. Chemically, HSA can be divided into at least three forms according to the redox state of the cysteine residue at portion 34 from the N-terminus: not oxidized (human mercaptoalbumin, or HMA), reversibly oxidized (human nonmercaptoalbumin-1, or HNA-1) and strongly oxidized (human nonmercaptoalbumin-2, or HNA-2). These three forms can be distinguished by high-performance liquid chromatography (HPLC) using a Shodex-Asahipak ES-502 N 7C column (Showa Denko, Tokyo, Japan) [2].

The subjects were 17 CKD patients (15 males and 2 females, mean age of 59 ± 11 years) on peritoneal dialysis treatment. All of these patients had the same PD procedure performed before and after the disaster without missing regular changes of the dialysate. Plasma samples for measurement of HSA redox were obtained 1 week before and after the disaster. Table 1 shows the values of urea nitrogen and HSA redox before and after the disaster. The *f* (HNA-1) value was significantly increased (48.2 ± 6.4 and 54.9 ± 7.0 %, $P < 0.0001$), whereas the *f* (HMA) value was significantly decreased (47.6 ± 6.7 and 40.9 ± 7.2 %, $P < 0.0001$). Thus, oxidative stress was significantly increased after the disaster.

This result suggests that an extreme stress like a great earthquake can enhance oxidative stress, the causative factor of cardiovascular disease (CVD) [2]. In fact, several reports suggested that severe earthquake stress could induce CVD [3, 4]. Moreover, a recent animal experiment suggested the protective effect of anti-oxidant treatment against physical and physiological stress [5]. Therefore, after a severe earthquake, including antioxidant strategies as part of the treatment of residents—at least residents with apparent CVD risk such as advanced CKD—should perhaps be encouraged.

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Table 1 The values of urea nitrogen and HSA redox before and after the great East Japan earthquake

	Pre-earthquake	Post-earthquake	<i>P</i> value
UN (mg/dL)	56.5 ± 9.7	60.3 ± 13.6	Ns
HSA-redox (%)			
HMA	47.6 ± 6.7	40.9 ± 7.2	<0.0001
HNA-1	48.2 ± 6.4	54.9 ± 7.0	<0.0001
HNA-2	4.2 ± 0.8	4.3 ± 0.6	Ns

Conflict of interest We declare that we have no conflicts of interest.

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