Earthquake-Induced Cardiovascular Disease and Related Risk Factors in Focusing on the Great Hanshin-Awaji Earthquake

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The major earthquake increases cardiovascular disease during the period from night-time to morning, especially in the elderly patients living around the epicenter. Earthquake-induced stress increased BP and blood viscosity determinants, and enhanced fibrin turnover with endothelial cell stimulation in a group of hypertensive elderly subjects. Earthquake might trigger the cardiovascular events through the potentiation of these acute risk factors. Reduction of stress and related acute risk factors in this period may suppress cardiovascular deaths following a major earthquake. Further investigation of the mechanisms triggering cardiovascular events after the a major event such as a major earthquake is a necessary part of the strategy for the prevention of cardiovascular disease. J Epidemiol, 1998; 8 : 131-139.

Hanshin-Awaji earthquake, cardiovascular disease, coronary artery disease, stroke, acute risk factor

On 17 January 1995, the south part of Hyogo Prefecture in Japan was struck by a typical major earthquake (Hanshin-Awaji earthquake), measuring 7.2 on the Richter scale, that hit directly above the focus 1,2. There have been a few reports of increases of the number of coronary heart disease (CHD) deaths and of changes in cardiovascular risk factors after a major earthquake 3-6. Immediate and appropriate practice based on disaster medicine is required. However, the available information as to the characteristics of earthquake-related CHD is scanty.

Since the Hanshin-Awaji earthquake occurred, several studies have been undergoing, and some of these have been published 9-17. We have reviewed these studies to clarify the characteristics of earthquake-induced cardiovascular disease.

EARTHQUAKE-INDUCED CARDIOVASCULAR DISEASES

There have been a few reports of short-term increases of the number of coronary heart disease (CHD) deaths after a major earthquake 3,5-7, and but these studies were either death certificate-based 3 or had target population limited to subjects younger than 70 years old 8.

In the Awaji Island where includes the epicenter of this major earthquake, most of medical facilities were able to continue services for the earthquake victims without interruption by this disaster situation, and Jichi Medical School Cohort Study has been going in this area (Hokudan) 18,19. Thus, we were able to study in the detail characteristics of earthquake-related CHD mortality on the basis of direct access to physicians' records 16. The Tsuna region is the north part of the Awaji Island, including the epicenter, and is one of the areas where the degree of the damage was the heaviest (Figure 1). The CHD deaths after the earthquake were increased for a few months, and the total number after the earthquake during January 17 to April 30 (1995) was 45, which was significantly (1.5-times) higher than the 31 deaths during the same period of the previous year (1994). The CHD deaths after the earthquake all occurred in elderly individuals above 60 years or more, and had a positive correlation with earthquake-induced
damages (of percentage refugees and percentage completely destroyed house, Figure 2). The actual number of CHD deaths in each district were 23 and 10 in Hokudan, 8 and 5 in Ichinomiya, 3 and 2 in Goshiki, 3 and 4 in Awaji, 5 and 6 in Tsuna, 3 ad 4 in Higashiura in 1995, and in 1996, respectively.

As well as CHD mortality, Suzuki et al. also demonstrated the increased incidence of myocardial infarction after the earthquake compared with previous years on the Awaji Island. 10.

**DURATION OF EFFECTS OF EARTHQUAKE**

The previous studies of the increased coronary accidents and deaths were confined to within the first few days after the major earthquake 3, 5, 6. As more direct evidence, myocardial ischemia and cardiac rhythm disturbance measured by Holter monitoring during a major earthquake have also been found 20, 21. In our results, CHD death mortality was shown to be increased not only in the first 2-week period, but also in the following few months during which the major felt aftershocks were markedly reduced 14. This discrepancy of the duration of study of increased CHD mortality between our and the previous reports might be related to the degree of magnitude of the disaster. The earthquake-induced damage in the Hanshin-Awaji earthquake is among the greatest of any

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**Figure 1.** Map of the epicenter of the Hanshin Awaji earthquake (1995) and the Tsuna region on the Awaji Island.

**Figure 2.** Relationship between CHD mortality and the degree of earthquake-induced damage. Refugees (%) are calculated as the ratio of the maximum number of refugees in each district to total residents of each district. Completely destroyed houses (%) are calculated as the ratio of the completely destroyed houses in each district to the total houses in each district.
recorded in which coronary accidents were studied, and it was moreover a typical earthquake that hit directly above the focus. Furthermore, our studied region was one of the most severely damaged areas and included the epicenter. In this regard it is interesting that the study results revealed that CHD deaths had a significant positive correlation with the degree of the earthquake-induced damage. The fact that the quake occurred in winter might also have contributed to the increase of earthquake-induced CHD death and helped to prolong this increase. Compared with previous studies, the characteristic demography of our highly-aged community, Tsuna region studied is a community with large elderly population with 31% of the total of 64,000 residents are 60 years old or more. All those who dead from CHD after the earthquake were elderly subjects aged 60 years or more. Elderly individuals might be prone to earthquake-induced CHD death, which condition might be prolonged after a major earthquake.

**CIRCADIAN VARIATION**

There are no report on the onset time of earthquake-induced cardiovascular events. In our study, when we divided the 24-hr period into four 6-hr periods, CHD deaths occurred 1.8 times more often (p<.05) in the night-time (PM 11 - AM 5) and 1.4 times as often during the morning (AM 5 - AM 11), while their occurrence did not vary during 12-hr period from AM 11 to PM 11 (Figure 3). The fatal CHD occurred 1.8 times more frequently in the night-time and 1.4 times more frequently in the morning than in the afternoon or evening, while there was no differences between the numbers of deaths in the afternoon and the evening. Recently coronary accidents have been reported to form a cluster in the morning from the time of awakening. In the Tsuna region, the incidence of CHD led to death in this period was about 2-fold higher than that in any of the other 3 periods in the 1994. At this time of day, various coronary risk factors are known to change; for example, increase of blood pressure, blood viscosity, and platelet aggregation and marked decrease of fibrinolytic activity. These coronary risk factors, commonly multiple in the elderly, are reported to be exacerbated under stressful condition. Thus, the additional earthquake-induced potentiation of these risk factors might lead to clinically overt coronary accidents in elderly persons.

The most striking increase in fatal cases of CHD led to death was found in the night-time period (PM 11-AM 5), when before the earthquake most people were usually asleep. After the earthquake, sleep disturbance and emotional stress might have been brought about due to the difference in the place of sleeping or due to the recollection of the awful earthquake or the financial loss due to the earthquake. This psychosocial effect might be lesser in the day-time when they are otherwise occupied. Thus, for the prevention of earthquake-induced coronary accidents, stress reduction during the period from nigh to morning is probably the most important objective in the preventive medical efforts directed at earthquake victims among the elderly.

**STRESS AND CARDIOVASCULAR EVENTS**

The causative factors for the increased incidence of cardiovascular events after a major earthquake remain unclear. Extreme stress like a major earthquake would influence the sympathetic nervous system and various cardiovascular risk factors. The acute stress caused by the earthquake and the sub-

Figure 3. The time of onset of CHD death. CHD deaths occurred 1.8 times more often (p<.05) in the night-time (PM 11 - AM 5) and 1.4 times as often during the morning (AM 5 - AM 11), while their occurrence did not vary during 12-hr period from AM 11 to PM 11.
sequent chronic stress of living in the shelter and so forth are both graded at the highest degree (catastrophe) in the classification of DSM-III-R [26]. The prolongation of the period of susceptibility to earthquake-induced CHD death might also be due to the persistent chronic stress caused by emotional upset due to loss of financial security by the comprehensive and profound changes in their lifestyles. The potentiation of coronary risk factors caused by this sustained stress or by changes in their lifestyles including eating habits and physical activity might also affect the persistent increase in earthquake-induced CHD deaths. Elderly subjects might be more prone to being affected by these factors than younger adults.

**EARTHQUAKE-INDUCED BP ELEVATION**

There are a few reports on the changes in cardiovascular risk factors after a major disaster, describing the transient increases in blood pressure (BP), heart rate and the levels of total cholesterol and triglycerides, but there are some conflicting results in these studies [4,8,10,12,15,27]. In previous reports on the changes in cardiovascular risk factors after a major disaster, transient increase in BP level or an increase in heart rate was found [29]. In our study period (7 to 14 days after the earthquake), the BP elevation was not accompanied by an increase in heart rate [28]. Factors other than sympathetic arousal might have contributed to the BP elevation. Increased white-coat effect might be candidate for this phenomenon. In our preliminary study, in the majority of the well-controlled hypertensive patients, office BP was temporarily increased after the earthquake and was then decreased to the pre-earthquake level within 4 weeks after the major felt-aftershocks ceased [12]. This transient earthquake-induced BP increase during limited period (4 weeks) has been also confirmed by the following recent other studies. Saito K et al. also found the earthquake-induced BP elevation within 4 weeks in the treated hypertensive patients, which is smaller in those treated with β-blockers compared with those treated with other drugs [16]. Minami et al. found the home BP elevation in the treated hypertensive patients [17].

Clinically, this characteristic of earthquake-induced BP elevation should be taken into account in the BP management, because the antihypertensive therapy could lead to cardiovascular accident due to excessive BP reduction. We presented a case of earthquake-induced BP elevation, in which inadequate long-term administration of antihypertensive agents apparently resulted in syncope and orthostatic fall [28]. In this patient, after marked earthquake-induced BP elevation was found and antihypertensive therapy had been begun, the casual BP measured by medical rescue team physicians had been reduced rapidly to below 140/90 mmHg within 3 weeks after the quake. The reduction of home BP, which was less than 120/80 mmHg, showed even more markedly reduction than did the casual BP. Fainting, probably due to marked reduction of BP, developed at about 3 weeks after the quake. Thus, the prolonged antihypertensive therapy might lead to cardiovascular accidents due to excessive reduction of BP, especially in elderly subjects with cardiovascular risk factors.

On the other hand, some patients with white-coat hypertension developed sustained hypertension which lasted for several months after the earthquake [13]. These sustained-hypertensive patients should receive antihypertensive therapy to prevent cardiovascular accidents. However, this sustained hypertension was not so established that it persisted as of about one year after the earthquake, when the daily lives of the patients had returned to normal [13].

**MANAGEMENT STRATEGY FOR EARTHQUAKE-INDUCED HIGH CASUAL BP**

Considering the above specific characteristics of earthquake-induced BP elevation, we proposed the management strategy for newly-detected high casual BP in disaster situations as shown in Figure 4 [29]. Transient increase of casual BP without symptoms related to high BP (such as headache, palpitation, insomnia) should not be treated with antihypertensive agent, especially when hypertension had not been detected before the disaster. In these cases, BP monitoring should be performed by the patients themselves or by trusted familiar faces such as public health nurses in non-medical settings. Antihypertensive agent could be started, in disaster victims with high casual BP and symptoms related to high BP or positive past history of hypertension. As the first choice of antihypertensive agent, β-blockers would be recommended, because diuretics may accelerate the hemoconcentration that usually occurs in or soon after a disaster [16]. When antihypertensive therapy is continued in disaster victims with high casual BP for 3-4 weeks after the event, an effort should be made to obtain the BP in a non-medical setting to help avert cardiovascular accidents due to excessive reduction of BP.

**EARTHQUAKE-INDUCED CHANGE IN SERUM LIPID PROFILE**

We could not find any change in serum lipid levels after the earthquake [15] whereas the levels of total cholesterol and triglycerides were increased in another reported study [4]. This discrepancy might be due to the time of the study after the earthquake and the nutritional state of the people. Our study was conducted from 7 to 14 days after the earthquake, when the food supply was limited. On the other hand, the previous study documenting an increase in total cholesterol and triglycerides was conducted from 2 to 6 weeks after the earthquake [4], when the chronic stress might have prompted overeating of lipid-rich foods sent from neighboring areas.
EARTHQUAKE-INDUCED POTENTIATION OF ACUTE RISK FACTORS

Recently, hemostatic abnormalities and endothelial cell dysfunction have been recognized as cardiovascular risk factors. Hemostatic abnormalities might be an acute risk factor triggering cardiovascular events after the major earthquake as well as hemodynamic and vasoconstrictive forces. But there are no reports on the effects of earthquake on the hemostatic system or endothelial cell state.

Due to our role of providing continuous longitudinal medical care for sick people at Awaji-Hokudan Public Clinic, we were able to study the earthquake-induced potentiation of cardiovascular risk factors. Hemostatic abnormalities might be an acute risk factor triggering cardiovascular events after the major earthquake as well as hemodynamic and vasoconstrictive forces. But there are no reports on the effects of earthquake on the hemostatic system or endothelial cell state.

We studied the changes in cardiovascular risk factors any 42 elderly outpatients with well-controlled hypertension living near the epicenter (Awaji-Hokudan districts) 7 to 14 days after earthquake when the major felt-aftershocks persisted. After the earthquake, hematocrit, fibrinogen, a marker of fibrin turnover [D-dimer], fibrinolytic factors [plasmin-α 2-plasmin inhibitor complex (PIC) and an endothelial cell-derived marker [von Willebrand factor (vWF)]] were increased. When the patients were divided into the high-stress and moderate-stress groups according to the degrees of damages to their house and injury of family members, the levels of fibrinogen, vWF, (PIC) and tissue-type plasminogen activator (tPA) antigen were increased only in the former group, while BP, hematocrit, and D-dimer levels were increased in both groups. The above abnormalities of acute risk factors except vWF were transient and decreased to the pre-quake level 4-6 months after the quake. The earthquake-induced stress seems to induce transient increases in BP, blood viscosity determinants, and fibrin turnover, and prolonged endothelial cell stimulation. These abnormalities of risk factors might induce cardiovascular accidents in high-risk subjects just after a major earthquake.

The plasma D-dimer levels were clearly elevated just after the earthquake (Figure 5). Plasma D-dimer is released from cross-linked fibrin, and its level reflects the state of fibrin formation and its degradation (fibrin turnover) in blood vessels. In a previous study, the increase of plasma D-dimer levels showed sensitivity to various mild physiological and pathological prethrombotic conditions (normal aging, atherosclerotic disease, bed-ridden state, malignancy, etc.) as well as to overt thrombosis (disseminated intravascular coagulation, deep vein thrombosis, and pulmonary thromboembolism). In our high-stress group, the PIC, an activation marker of fibrinolysis, and tPA antigen levels after the earthquake were also increased (Figure 6). Thus, our results indicate that the major earthquake induced the rapid fibrin turnover with hyperfibrinolysis in the elderly patients studied. This enhanced fibrin turnover would probably be a factor predisposing to clinical cardiovascular events in elderly patients just after earthquake, but it would probably also be transient, given that the D-dimer level in our outpatients had returned to normal 4-6 months after the earthquake (Figure 5).

In increase in vWF, a glycoprotein stored in endothelial cell and secreted into the circulation by various types of endothelial
Figure 5. Change of plasma D-dimer levels after the Hanshin-Awaji earthquake  
A: Moderate-stress group; B: High-stress group.

STRESS AND POTENTIATION OF ACUTE RISK FACTORS

The precise mechanism of the earthquake-induced potentiation of these acute risk factors remains unknown. The extreme stress of a major earthquake would be a causative factor. Similarly, during the Iraqi missile war, also a highly stressful condition, the incidence of acute myocardial infarction and sudden death among Israeli civilians was increased, and emotional stress due to fear of missile attack probably would have been a causative factor. In the period of blood collection of this study, major aftershocks occurred 5-20 times or more...
per day in our Awaji-Hokudan district, and the subjects studied were continuously frightened during the period. Stress is reported to cause increased levels of hematocrit and fibrinogen (major determinants of blood viscosity) and to induce rheological change in blood flow and elevate BP. Emotional stress in atherosclerotic monkeys does lead to changes in endothelial cell, and it exacerbates coronary artery atherosclerosis. Adrenaline and noradrenaline released during stress may have several effects, including strong potentiation of platelet aggregation in response to aggregating agents, vessel wall damage, a release of tPA and vWF from endothelial cell, and coronary artery spasm. Thus, the increased catecholamine levels associated with stress may enhance spasms, vessel injury, the formation of platelet-rich thrombi, and subsequent fibrin high turnover.

**Figure 6.** Change of plasma levels of fibrinogen, vWF, PIC and tPA after the Hanshin-Awaji earthquake in the high-stress group. vWF = von Willebrand factor, PIC = plasmin-α2-plasmin inhibitor complex, tPA = tissue-type plasminogen activator

**EFFECTS OF ENVIRONMENTAL CHANGES**

The transiently increased blood viscosity and hemostatic abnormalities found in our patients might partly be attributed to the drastic changes in their life styles due to the catastrophic events, since many had to abandon their homes and live in shelters and the composition of their diet would also have been drastically changed. Hemoconcentration could result from diminished water intake secondary to the prolonged psychological stress and profound changes in sociological status. As some of them were under physical stress from the effort to put their residences in order during the study period, the physical exertion after earthquake might also have induced fibrinolytic activation and subsequent elevation of tPA, D-dimer, and PIC. Cigarette smoker may
have increased their consumption in response to the stress, but we have found the no significant differences of the earthquake-induced potentiation of acute risk factors between smokers and nonsmokers.

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