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COMMENT

Time course of disaster-related cardiovascular disease and blood pressure elevation

Keisuke Narita[1] · Satoshi Hoshide[1] · Kazuomi Kario[1]

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Comment regarding "Five-year blood pressure trajectories of survivors of the tsunami following the Great East Japan Earthquake in Iwate," by Takahashi et al. *Hypertens Res* 2021

Immediately after a natural or man-made disaster, the sympathetic nervous system of individuals affected by the disaster is abnormally activated; in addition, their blood pressure (BP) may rise due to this sympathetic hyperactivity and the mental and physiological stress, insomnia, lack of exercise, increased salt intake, and changes in the living environment that occur when evacuees are moved from their homes to shelters [1–3]. The rate of cardiovascular events has also been reported to increase immediately after disasters [1–3].

Few studies have reported the relationship between disasters and changes in BP values. Takahashi et al. prospectively observed BP trajectories from 2010 to 2015 in Rikuzentakata City, Iwate Prefecture, which is one of the areas that was affected by the 2011 Great East Japan Earthquake and tsunami, and they focused on areas that were heavily damaged by the tsunami that followed the earthquake [4]. The study's BP data were drawn from the annual health checkups of the participants, who were divided into two groups: the participants whose lives were severely disrupted by the tsunami and who were forced to move away from their homes and the participants whose lives were relatively mildly disrupted by the tsunami and who did not leave their homes. The results of the data analysis demonstrated that in the entire population, the BP values tended to decrease both before and after the earthquake [4]. Table 1 summarizes the previous and current evidence regarding BP changes during disasters (i.e., earthquakes, a hurricane, and the September 11, 2001 attack). Earlier studies reported that the subjects' BP values were elevated when measured 1–2 weeks after the disaster and that 3–6 weeks later, the BP values peaked and gradually returned to baseline (i.e., the values recorded before the disaster). Other studies that followed subjects for several years before and after a disaster have also reported that their BP values were not elevated at time points >1 year after the disaster [1, 3]. These results are similar to those of the recent Takahashi et al. study. Moreover, in the Takahashi et al. investigation, the tendency for a reduction in BP was more pronounced in the participants who obliged to move to temporary housing after the tsunami than in the participants who did not move. Takahashi et al. speculated that this finding was affected by the enormous investment of medical resources in Iwate Prefecture, especially in the areas in which the tsunami damage was severe after the earthquake.

Disaster hypertension, which specifically refers to the condition of elevated BP levels (>140/90 mmHg) after a disaster, occurs immediately after a disaster and continues until both the residents' living environment and their lifestyle habits have improved and stabilized [2, 3]. Systolic blood pressure has been reported to increase by an average of 5–25 mmHg in the 2–4 weeks after an earthquake [2, 3]. In addition, disaster hypertension is likely to persist for a long period in elderly patients; in patients with increased salt sensitivity, such as those with metabolic syndrome; and in individuals with chronic kidney disease, microalbuminuria, or obesity [1, 3, 5]. The mechanism underlying the onset of disaster hypertension includes physical and mental stress due to the disaster and changes in the living environment. Disruptions of circadian rhythms due to decreased daytime activity and sleep disorders each promote sympathetic hyperactivity and increase the levels of stress-induced hormones, such as glucocorticoids [3]. It has been reported that even some children who were exposed to psychiatric trauma showed prolonged elevations of BP during a period of several years after a disaster [6].

✉ Kazuomi Kario
kkario@jichi.ac.jp

[1] Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical University School of Medicine, Tochigi, Japan

Table 1 Evidence of blood pressure regarding disasters

Time course after the disaster	Yr of publ'n	First author	Disaster	BP msrmt.	Participants	Summary of findings
Several hours						
During the first 24 h post- disaster	2001	Parati [14]	Central Italy earthquake	ABPM	A 34-yr-old normotensive woman	SBP/DBP rose to 150/122 mmHg from 130/85 mmHg at the time of the strongest tremor. Pre-earthquake BP levels were restored only 1 h later, but BP remained characterized by a pronounced variability throughout the following 6 h.
	2009	Chen [15]	Wenchuan earthquake	ABPM	11 hypertensive patients	Mean BP increase 125.8 ± 17.3 to 150.5 ± 20.3 mmHg (average time of the first measurements was 14 min after the first tremor), and BP remained high until 6 h after the earthquake.
	2015	Nishizawa [16]	Great East Japan earthquake	ABPM	8 patients living in temporary housing or own home	Ambulatory BP measurements on the day of the largest aftershock with a first tsunami warming (sirens). After the time of the aftershock, nighttime and morning SBP was significantly higher in those living in temporary housing than those living in their own home (nighttime, median [quartile] 124.9 [113.2–137.9] vs. 107.0 [101.9–110.1] mmHg; morning, 149.3 [131.0–196.2] vs. 129.5 [128.8–131.0] mmHg).
Several weeks						
1–2 wks	1992	Trevisan [17]	Southern Italy earthquake	Office BP	607 male employees	BP levels did not change between before and after the disaster. Increases in pulse rate, serum cholesterol.
1–2 wks	1997	Kario [18]	Hanshin Awaji earthquake	Office BP	42 elderly hypertensive patients	BP significantly increased between before and after a disaster. SBP, 152 (142–164) to 170 (161–178) mmHg; DBP, 83 (79–88) to 91 (84–96) mmHg
1–5 wks	2001	Kario [19]	Hanshin Awaji earthquake	Office BP	124 elderly hypertensive patients (mean age 69 yrs)	During the 1–2 weeks after the earthquake, SBP increase (14 ± 16 mmHg), but these values returned to the baseline by 3–5 wks after the earthquake. The earthquake-induced BP increase correlated with the "white coat" effect (office and ambulatory BP).
1–4 wks	1997	Minami [20]	Hanshin Awaji earthquake	Home BP	36 hypertensive patients	Home BP significantly increases (+11/+6 mmHg) the first week after the earthquake. Within 4 wks, home BP gradually returned to the baseline.
1–6 wks	1997	Saito [21]	Hanshin Awaji earthquake	Office BP	221 hypertensive patients (mean age 59 years)	During the 4 weeks after the earthquake, BP significantly increased in both 105 patients living in the severely damaged area (+4.2 ± 1.0 mmHg) and 116 patients living in the surrounding area (+2.4 ± 0.7 mmHg). In the former, the BP increase peaked in the first week (+6.7 ± 1.6 mmHg), declined thereafter, and returned to the baseline within 6 weeks after the disaster.
2–8 wks	1986	Trevisan [22]	Southern Italy earthquake	Office BP	1400 male employees (mean 42 yrs)	In 2–8 weeks after the disaster, 277 individuals who were exposed to the disaster underwent screening for BP and blood tests. In this screening, BP levels did not show a significant difference compared with 1133 individuals who were not exposed to the disaster.

Table 1 (continued)

Time course after the disaster	Yr of publ'n	First author	Disaster	BP msrmt.	Participants	Summary of findings
8 wks	2005	Gerin [23]	September 11, 2001 attack	Home BP	427 hypertensive patients	Telemonitoring of mean SBP significantly increases during the 8 wks after 9/11, across the four sites, compared with that assessed during the previous 8 wks (range of observed differences, 1.7–3.8 mmHg).
9 wks	1995	Kario [24]	Hanshin Awaji earthquake	ABPM	3 patients with white-coat HT	White-coat HT was shifted to a pattern of sustained HT (elevated office and ambulatory BP) 9 wks after the disaster.
Several months to 1 year						
3–6 mos.	2006	Kamoi [25]	Mid-Niigata earthquake	Home BP	222 diabetic patients	Mean morning home SBP and UACR increased within 3 mos. but returned to the pre-earthquake level at 6 mos.
6–16 mos.	2009	Fonseca [26]	Hurricane Katrina	Office BP	1795 diabetic patients (mean 62 yrs, African Americans)	In a comparison between 6 mos. before and 6–16 mos. post-disaster, SBP increased from 130.7 ± 17.0 to 141.2 ± 22.0 mmHg. HbA1c and LDL-C levels also increased.
1-yr follow-up	2017	Konno [27]	Great East Japan earthquake	Office BP	225 public employees and 1232 general population	In the yr of the disaster (2011), the public employees showed significantly higher BP (129.8 ± 14.0/78.0 ± 11.7 vs. 117.0 ± 14.4/71.6 ± 11.4 mmHg). At 1 yr post-disaster (2012), the SBP of the public employees remained higher than that of the general population (125.3 ± 16.0 vs. 119.9 ± 15.5 mm Hg). Prolonged BP elevation among the public employees was observed for >1 yr after the disaster.
1-yr follow-up	2018	Nagai [28]	Great East Japan earthquake	Office BP	Approx. 10,000 men and 12,000 women aged 40–74 yrs	The age-standardized prevalence of HT peaked in the 1 yr after the disaster (2012) at 48.8% in men and 39.0% in women. By the 3 yrs after the disaster (2014), the treatment and control of HT had increased to 66.3% and 67.1% in men, and 70.6% and 68.1% in women. The prevalence of HT peaked 1 yr post-disaster, while the treatment and control of HT increased thereafter.
Several years						
2-yr follow-up	2016	Ohira [29]	Great East Japan earthquake	Office BP	Fukushima participants without use antihypertensives, $n = 11,037$	At 2-yr follow-up, office SBP and DBP increase in both evacuees and non-evacuees. SBP (mmHg), in men, non-evacuees, 121.5 to 126.1; evacuees, 121.5 to 127.3, all p-values<0.001. In women, non-evacuees, 119.8 to 123.9; evacuees, 119.6 to 124.0, all p-values<0.001. In male participants, the incidence of HT increased in evacuees compared to non-evacuees (adjusted HR, 1.20).
4-yr follow-up	2000	Bland [30]	Southern Italy earthquake	Office BP	693 male employees	There was no relationship between BP and earthquake experience at the 4-yr follow-up (Olivetti Longitudinal Study).
4-yr follow-up	2019	Watanabe [6]	Great East Japan earthquake	Office BP	227 children (mean age 6.6 yrs)	Total 227 children were followed up for 4 yr and the association between BP and experiences of trauma was assessed. Children reporting disaster trauma and witnessing a fire were significantly related to higher DBP. Disaster-related trauma was associated with higher DBP among young children 4 yr after the disaster.

Table 1 (continued)

Time course after the disaster	Yr of publ'n	First author	Disaster	BP msrmt.	Participants	Summary of findings
5-yr follow-up	2021	Takahashi [4]	Great East Japan earthquake	Office BP	3914 patients	At the 5-yr follow-up (annual follow-up), BP levels gradually decreased. This tendency was stronger in the severely damaged area where people needed to move compared to the relatively mildly damaged area.

ABPM ambulatory blood pressure monitoring, *BP* blood pressure, *BP msrmt.* BP measurement, *DBP* diastolic BP, *HT* hypertension, *HR* hazard ratio, *LDL-C* low-density lipoprotein cholesterol, *mos.* months, *SBP* systolic BP, *UACR* urine albumin creatinine ratio, *wks* weeks, *Yr of publ'n* year of publication.

In the management of disaster hypertension, although no clear evidence is available regarding the target BP level during a disaster, we recommend that the primary target BP level should be an office BP level <140/90 mmHg in the acute phase of the disaster because the diagnostic criteria for disaster hypertension is a BP level ≥140/90 mmHg [2, 3]. After the improvement of an individual's living environment after the disaster, the target BP level should be an office BP level <130/80 mmHg, based on most international guidelines.

Although medical interventions for controlling BP in the medium to long term after a disaster can be important, it is also known that the incidence of disaster-related cardiovascular diseases (CVDs) increases immediately and from several hours to several months after a disaster [1–3]. Figure 1 shows the time course of BP variation and the incidence of disaster-related CVDs after the occurrence of disasters. The incidences of sudden cardiac death, fatal arrhythmia, and Takotsubo cardiomyopathy have been reported to increase immediately after a disaster [2]. It was also demonstrated that the incidences of sudden cardiac death and fatal arrhythmia increased immediately after the 2011 Great East Japan Earthquake; in addition, pulmonary embolism (PE) was observed to be likely to occur several days after that disaster [7, 8].

Long-term residence in a car or shelter, inadequate water intake, and postdisaster stress promote thrombus formation, potentially resulting in deep vein thrombosis (DVT) or PE. The risk factors for DVT include female sex, age >40 years, living in a car, trauma, and poor toileting conditions [1, 2, 8]. Sato et al. reported that 178 (10.6%) of 1673 individuals who were screened for DVT with a portable echo device 1 month after the 2016 Kumamoto earthquake had experienced DVT [8]. The measurement of the D-dimer level and portable echo measurements are suggested to be useful for DVT screening.

During the period from several days to several months after disasters, the incidences of myocardial infarction, stroke, and heart failure reportedly increase [1, 2, 9]. An increase in the incidence of heart failure during disasters was first reported during the Great East Japan Earthquake [9]. The causes were considered to be sympathetic hyperactivity, elevated BP, arrhythmias (including atrial fibrillation) during the disaster, medication procurement delays, excessive salt intake due to stored food consumption, exposure to cold due to difficulty in controlling room temperatures, and pneumonia, and other infectious diseases [1–3]. In light of these findings, disaster-related CVDs may be most common immediately or several hours to several weeks or months after disasters. Moreover, myocardial infarction, stroke, and heart failure, which are likely to be triggered by an acute elevation of BP, are thought to coincide with the timing of the elevation of BP levels after

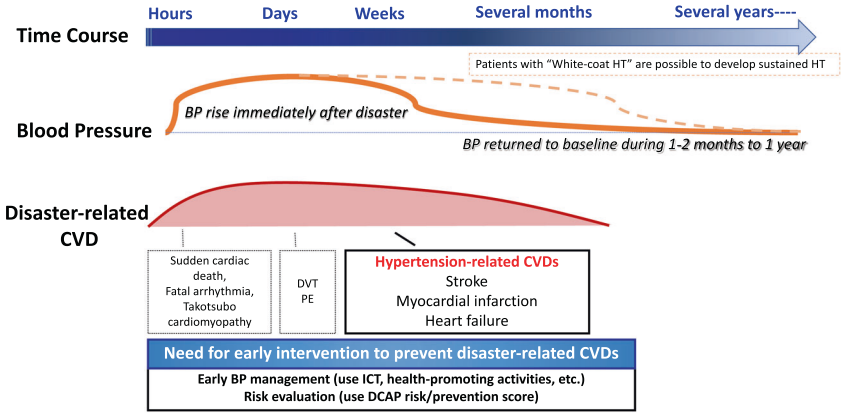

Fig. 1 Time course of BP changes and disaster-related CVDs during disasters. BP blood pressure, CVD cardiovascular disease, DCAP disaster cardiovascular prevention, DVT deep venous thrombosis, ICT information communication technology, PE pulmonary embolism

disasters [1, 3]. To achieve the goal of preventing disaster-related CVDs, it is thus important to intervene as quickly as possible after a disaster.

Immediately after the 2011 Great East Japan Earthquake, our group distributed the "Disaster Cardiovascular Prevention (DCAP) risk/prevention score" on a website to contribute to the prevention of disaster-related CVDs. Individuals with a risk score of more than four points were categorized into the high-risk group, and such high-risk patients were advised to attempt to improve their living environment and lifestyle to achieve a prevention score of greater than six points [3, 10, 11]. We recommend that medical teams use the DCAP score in evacuation facilities during a disaster [10, 11].

We also used information and communication technology (ICT) to perform BP monitoring and risk management. In cooperation with healthcare practitioners in Minamisanriku Town, which was severely damaged by the earthquake and tsunami, we introduced an ICT-based BP monitoring device at evacuation centers and shared patients' BP values in the database to support BP management with remote monitoring [10, 11]. Consequently, we succeeded in improving the evacuees' BP control and suppressing the seasonal variation in their BP (i.e., an increase in BP from summer to winter) during the acute to chronic phase after the disaster [11, 12]. We thus propose that ICT can be useful for anticipating the interventions needed due to BP elevation after a disaster and can contribute to the prevention of disaster-related CVDs. ICT-based devices are now available for home BP monitoring, and these devices could be used to evaluate home BP levels during disasters. By using ICT to closely manage high-risk patients, we can reduce the burden on medical institutions in disaster areas and support efficient risk management [13].

The results of the Takahashi et al. study of BP trajectories from 2010 to 2015 (which covers a period before and after the 2011 Great East Japan Earthquake) suggested that mid- to long-term medical interventions after multiple disasters in 2011 (earthquake, tsunami, and nuclear power plant disaster) may have contributed to the improvement of BP control. In addition to these mid- to long-term interventions in a disaster-affected area, early interventions that consider the timing of the onset of disaster-related CVDs could be useful to suppress the incidence of disaster-related CVDs.

Compliance with ethical standards

Conflict of interest KK has received research funding from Teijin Pharma Limited, Omron Healthcare Co., Fukuda Denshi, Bayer Yakuhin Ltd., A&D Co., Daiichi Sankyo Co., Mochida Pharmaceutical Co., EA Pharma, Boehringer Ingelheim Japan Inc., Tanabe Mitsubishi Pharma Corp., Shionogi & Co., MSD K.K., Sanwa Kagaku Kenkyusho Co., and Bristol-Myers Squibb KK, and honoraria from Takeda Pharmaceutical Co. and Omron Healthcare Co.

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