Received: 21 October 2020 | Revised: 5 January 2021 | Accepted: 6 January 2021

DOI: 10.1111/jch.14192

REVIEW PAPER

WILEY

Disaster hypertension and cardiovascular events in disaster and COVID-19 pandemic

Keisuke Narita MD[1,2] | Satoshi Hoshide MD, PhD[1] | Kelvin Tsoi BSc, PhD[3] | Saulat Siddique MBBS, MRCP, (UK), FRCP, (Lon)[4] | Jinho Shin MD[5] | Yook-Chin Chia MBBS, FRCP[6,7] | Jam Chin Tay MBBS, FAMS[8] | Boon Wee Teo MB, BCh[9] | Yuda Turana MD, PhD[10] | Chen-Huan Chen MD[11,12,13] | Hao-Min Cheng MD, PhD[11,12,13,14] | Guru Prasad Sogunuru MD, DM[15,16] | Tzung-Dau Wang MD, PhD[17,18] | Ji-Guang Wang MD, PhD[19] | Kazuomi Kario MD, PhD[1]

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

[2]Department of Cardiovascular Medicine, Karatsu Red Cross Hospital, Saga, Japan

[3]JC School of Public Health and Primary Care, The Chinese University of Hong Kong, Shatin, Hong Kong

[4]Punjab Medical Center, Lahore, Pakistan

[5]Faculty of Cardiology Service, Hanyang University Medical Center, Seoul, Korea

[6]Department of Medical Sciences, School of Healthcare and Medical Sciences, Sunway University, Bandar Sunway, Malaysia

[7]Department of Primary Care Medicine, Faculty of Medicine, University of Malaya Kuala, Lumpur, Malaysia

[8]Department of General Medicine, Tan Tock Seng Hospital, Singapore, Singapore

[9]Division of Nephrology Department of Medicine, Yong Loo Lin School of Medicine, Singapore, Singapore

[10]School of Medicine and Health Sciences, Atma Jaya Catholic University of Indonesia, Jakarta, Indonesia

[11]Institute of Public Health and Community Medicine Research Center, National Yang-Ming University School of Medicine, Taipei, Taiwan

[12]Division of Cardiology, Department of Medicine, Taipei Veterans General Hospital, Taipei, Taiwan

[13]Faculty of Medicine, National Yang-Ming University School of Medicine, Taipei, Taiwan

[14]Department of Medical Education, Center for Evidence-based Medicine, Taipei Veterans General Hospital, Taipei, Taiwan

[15]MIOT International Hospital, Chennai, India

[16]College of Medical Sciences, Kathmandu University, Bharatpur, Nepal

[17]Department of Internal Medicine, Cardiovascular Center and Division of Cardiology, National Taiwan University Hospital, Taipei City, Taiwan

[18]Department of Internal Medicine, Division of Hospital Medicine, National Taiwan University Hospital, Taipei City, Taiwan

[19]Department of Hypertension, Centre for Epidemiological Studies and Clinical Trials, Shanghai Institute of Hypertension, Shanghai Key Laboratory of Hypertension, Ruijin Hospital, Shanghai Jiaotong University School of Medicine, Shanghai, China

Correspondence
Kazuomi Kario, Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical University School of Medicine, 3311-1 Yakushiji, Shimotsuke 329-0498, Japan.
Email: kkario@jichi.ac.jp

Abstract

The incidence of large disasters has been increasing worldwide. This has led to a growing interest in disaster medicine. In this review, we report current evidence related to disasters and coronavirus disease-2019 (COVID-19) pandemic, such as cardiovascular diseases during disasters, management of disaster hypertension, and cardiovascular diseases associated with COVID-19. This review summarizes the time course and mechanisms of disaster-related diseases. It also discusses the use of information and communication technology (ICT) as a cardiovascular risk management strategy

to prevent cardiovascular events. During the 2011 Great East Japan Earthquake, we used the "Disaster Cardiovascular Prevention" system that was employed for blood pressure (BP) monitoring and risk management using ICT. We introduced an ICT-based BP monitoring device at evacuation centers and shared patients' BP values in the database to support BP management by remote monitoring, which led to improved BP control. Effective use of telemedicine using ICT is important for risk management of cardiovascular diseases during disasters and pandemics in the future.

1 | INTRODUCTION

Many large disasters such as the Great Hanshin-Awaji Earthquake (1995), the Great East Japan Earthquake (2011), and the Kumamoto Earthquake (2016) have occurred in Japan. As of August 2020, the coronavirus disease-2019 (COVID-19) pandemic is still a tense situation because the momentum of infection has not ended and countermeasures are taken worldwide. Anyone may encounter a disaster or pandemic crisis anytime and anywhere. Thus, medical practitioners should be aware of disasters and pandemic-related diseases. Moreover, the incidence of disaster-related cardiovascular diseases (CVDs) changes concurrently with time immediately after the occurrence of a disaster.

2 | TIME COURSE AND MECHANISMS OF DISASTER-RELATED DISEASE

Characteristics of disaster-related diseases are defined based on the chronological sequence of disease that occurs during and after a disaster.[1,2] Activated sympathetic nervous system-induced diseases such as Takotsubo cardiomyopathy and pulmonary embolism (PE) occur during the first few weeks after a disaster. The risks of sudden death and hypertensive-related diseases such as stroke, coronary artery disease, and heart failure are approximately doubled after a disaster (Figure 1).[1] The major disaster-related diseases are listed in Table 1.

After a disaster, people are forced to live in shelters. Physical and mental stresses resulting from changes in the living environment lead to an increase in sympathetic nervous activity, which induces elevation of blood pressure (BP) and a tendency to develop thrombosis. This causes plaque rupture and thrombus formation, resulting in CVD development.[1-8] Additionally, sleep disturbances develop over a long period after the disaster.[9] Figure 2 illustrates the mechanism of CVD incidence during a disaster.[1-9]

Takotsubo cardiomyopathy caused by physical and mental stress develops immediately after a disaster. An obvious increase in the incidence of Takotsubo cardiomyopathy was reported after the 2004 Niigata Chuetsu Earthquake compared with its incidence in the previous 3 years. A total of 25 cases were reported within 3 weeks of the disaster, while prior to the disaster, only one case was reported in 2002, 0 in 2003, and one in 4 weeks before the 2004 earthquake. Furthermore, 11 (44%) of the reported cases occurred in older individuals within 6 h after the earthquake, and 24 (96%) of the reported cases occurred in women.[3]

Fatal arrhythmia and sudden cardiac death increase immediately after a disaster.[4] Zhang and colleagues reported that the incidence of fatal ventricular tachyarrhythmia increased in China after the 2008 Wenchuan Earthquake (67 events/10 000 person-days vs. 7 and 14 events/10 000 person-days [2 control periods], all p values < .001).[10] Hao and colleagues reported that the incidence of sudden cardiac death increased immediately after the 2011 Great East Japan Earthquake in individuals and areas at risk, which included women, older individuals (>75 years old), and coastal areas severely damaged by tsunami.[11]

The incidence of PE increases from the second to the third day after a disaster.[2] Long-term rest in a car or shelter, inadequate water intake, and post-disaster stress promote thrombus formation, resulting in deep-vein thrombosis (DVT) and PE. Risk factors for DVT include female sex, age >40 years, living in a car, trauma, and toilet environment.[2] Sato and colleagues reported that 178 (10.6%) of 1673 individuals who were screened for DVT using a portable echo 1 month after the 2016 Kumamoto Earthquake had DVT. They indicated the following the risk factors for DVT: age >70 years, daily use of sleeping medications, lower leg edema, and varices in the lower extremities.[12] Moreover, D-dimer measurement and portable echocardiography are suggested useful for DVT screening.

Hypertension-related diseases develop immediately after a disaster, and their risk continues until the living environment improves. The elevation of BP immediately after a disaster has been reported.[7,13] Disaster hypertension causes various CVDs such as stroke, coronary artery diseases (CADs), and heart failure.[1,2] Aoki and colleagues reported that the number of stroke, including cerebral infarction and cerebral hemorrhage, increased immediately after the 2011 Great East Japan Earthquake.[5] Similarly, an increase in the incidence of CADs and aortic dissection was observed.[5,14,15] The prevalence of CADs was higher 5 years after the 2008 Sichuan Earthquake in China than in other years.[14] An increase in disaster hypertension and arrhythmias such as atrial fibrillation, which is caused by acute stress due to the disaster, may lead to arterial thrombus and plaque rupture in patients at the risk of arteriosclerosis and CVDs.[1]

Subsequently, an increase in the incidence of heart failure during disasters was first reported during the Great East Japan Earthquake.[5] The causes were sympathetic hyperactivity, elevated BP, arrhythmias during the disaster, stagnation of drug procurement, excessive salt intake due to stored food consumption, exposure to

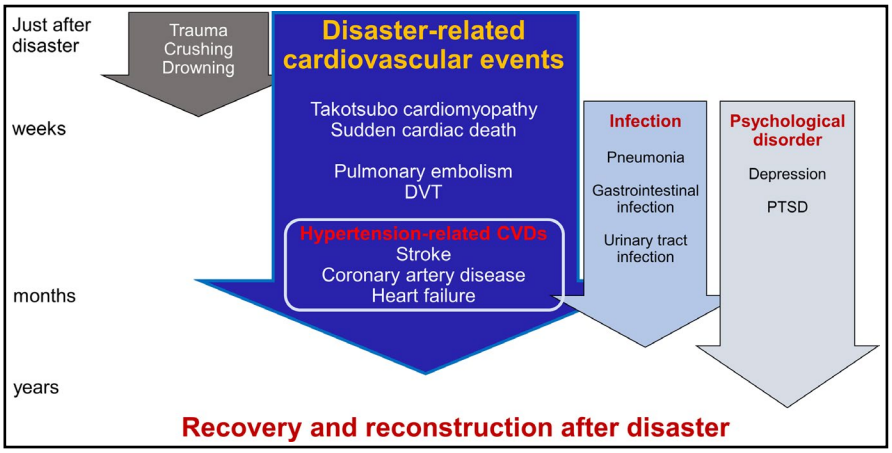

FIGURE 1 Time course of the onset of disaster-related disease. DVT, deep-vein thrombosis; PTSD, post-traumatic stress disorder. Source: Kario and colleagues[1]

cold due to difficulty in controlling room temperature, and pneumonia and other infectious diseases.[1,2]

3 | DISASTER HYPERTENSION: TIME COURSE AND MECHANISM

Disaster hypertension is defined as elevated BP levels (>140/90 mmHg) after a disaster. In Asian patients, BP level is strongly associated with CVD outcomes, and salt sensitivity is strong.[16,17] Hence, intensive BP control during a disaster is important for the prevention of CVDs in Asians.[1]

Disaster hypertension occurs immediately after a disaster and continues until both the living environment and lifestyle habits are improved and stabilized. Systolic BP (SBP) has been reported to increase by an average of 5–25 mmHg for 2–4 weeks after an earthquake.[1,2,18] Large individual differences in the elevation of BP and duration of elevated BP exist. Disaster hypertension is likely to persist for a long period in older patients, in patients with increased salt sensitivity such as those with metabolic syndrome, and in patients with chronic kidney disease, microalbuminuria, and obesity.[1,2,6–8,19]

The mechanism of disaster hypertension onset includes physical and mental stress due to disasters and changes in the living environment. Disruption of circadian rhythms due to decreased daytime activity and sleep disorders promote sympathetic hyperactivity and increase stress-induced hormones such as glucocorticoids.[1,2,9,10] Moreover, our research group previously demonstrated that excess salt intake is one of the causes of disaster hypertension in evacuees.[8] We conducted a health survey to monitor BP control in the victims of the 2011 Great East Japan Earthquake who lived in evacuation shelters after the earthquake. We investigated the relationship between salt intake, as estimated by the spot urine method, and the presence of disaster hypertension in 272 evacuees. In this study, disaster hypertension, defined as an SBP of ≥140 mmHg or a diastolic BP of ≥90 mmHg as measured using an automatic BP monitoring device at an evacuation center, was found in 58% of the participants, and its prevalence increased by 16% for each 1-g increase in estimated salt intake. The odds ratio (95% confidence interval) for developing disaster hypertension per 1-g increase in estimated salt intake in participants without a diagnosis of hypertension before the earthquake and with a high risk of salt sensitivity (age ≥65 years and presence of obesity, chronic kidney disease, and diabetes) was 1.46 (1.19–1.79). These findings suggest that increased salt intake and high salt sensitivity may be important mechanisms for the development of disaster hypertension.[8] Although there is still a lack of robust evidence regarding the mechanism of disaster hypertension, preventive measures may be important to maintain the circadian rhythm by improving the living environment and sleep quality and maintaining physical activity during a disaster as well as to reduce salt intake in high-risk patients.[1,2,6,7,8,19]

4 | MANAGEMENT OF DISASTER HYPERTENSION AND DISASTER-RELATED CVDS

The flow chart of disaster hypertension management is presented in Figure 3.[1,2] First, as regards BP evaluation, an increase in the white-coat effect is observed during a disaster.[6] Therefore, we recommend the evaluation of self-BP measurements (out-of-office BP) along with that of BP values measured by the relief team and medical institutions (office BP). An automatic BP monitoring device should be installed in evacuation centers for self-BP measurements.

Second, no clear evidence is available regarding the target BP level during a disaster. However, we recommend that the primary target BP level should be an office BP of less than 140/90 mmHg in the acute phase of the disaster because the diagnostic criteria for disaster hypertension are a BP of ≥140/90 mmHg.[1,2] Once the living environment improves, the target BP level should be an office BP of less than 130/80 mmHg based on most international guidelines.[20,21] With regard to the use of antihypertensive medications during disasters, pre-disaster medications should be continued if possible. In addition, we recommend the use of long-acting calcium channel blockers (CCBs) at the beginning of the treatment for disaster hypertension or when BP control is poor during a disaster. The BP-lowering effect of long-acting CCBs depends on the BP level upon administration. The higher the BP, the lower the BP; however, if the BP is relatively low, it may not be lowered further. Alternatively, long-acting CCBs are considered suitable

TABLE 1 Characteristics of disaster-related CVDs

Disaster-related cardiovascular diseases	Time of onset	Characteristics
Takotsubo cardiomyopathy	Immediately after the disaster to several weeks	Causes: physical and mental stress, and sympathetic abnormality Risk factors: old age and female sex Comments: benign prognosis if appropriate primary treatment is provided
Dysrhythmia Fatal dysrhythmia Atrial fibrillation Sudden cardiac death		Causes: physical and mental stress, sympathetic abnormality, insomnia Risk factors: presence of arteriosclerosis and high-risk factors of CVD (old age, smoking habit, HT, diabetes, dyslipidemia, and CKD)
PE* DVT	1–3 days to several weeks or several months after the disaster *Especially in people living in a shelter, the onset of PE is most often after 1–2 weeks of the disaster	Risk factors: age more than 40 years, female sex, living in a car, comorbidity of trauma, dehydration, poor toilet environment, and living in areas with severe environmental damage
Coronary heart disease Myocardial infarction Unstable angina Stroke Cerebral hemorrhage Cerebral infarct	Several days to several months or several years after the disaster	Causes: physical and mental stress, sympathetic abnormality, insomnia Risk factors: presence of arteriosclerosis and high risk of CVD (old age, smoking habit, HT, diabetes, dyslipidemia, and CKD), elevated BP (disaster hypertension), dehydration, and lack of usual medications
Heart failure		Causes: physical and mental stress, sympathetic abnormality, insomnia, excess salt intake due to consumption of stored food, and infection (pneumonia) Risk factors: presence of arteriosclerosis and high risk of CVD (old age, smoking habit, HT, diabetes, dyslipidemia, and CKD), elevated BP (disaster hypertension), and irregular intake of usual medications
Disaster hypertension		Causes: physical and mental stress, sympathetic abnormality, insomnia, excess salt intake due to consumption of stored food, and infection (pneumonia)
Respiratory infection	Several days to several months after the disaster	Respiratory infection (pneumonia) may induce CVDs such as heart failure Risk factors: old age and group life in shelters

Abbreviations: BP, blood pressure; CKD, chronic kidney disease; CVD, cardiovascular disease; DVT, deep-vein thrombosis; HT, hypertension; PE, pulmonary embolism.

for disaster hypertension because of the reliable reduction in BP levels and BP variability.[1]

The authors developed an assessment and prevention score for disaster-related CVDs (Disaster Cardiovascular Prevention [DCAP] risk/prevention score) based on the characteristics of the disaster-related disease and risk factors. The DCAP score consists of a risk score (AFHCHDC7) and a prevention score (SEDWITMP8) (Figure 4). We asked the medical volunteer teams to use this score to prevent disaster-related CVDs during the Great East Japan Earthquake.[22,23] Individuals with a risk score of ≥4 points were categorized into the high-risk group. Individuals in the high-risk group should attempt to improve their living environment and lifestyle to achieve a prevention score of ≥6 points.[1,2,22,23]

During the Great East Japan Earthquake, we used the DCAP system that was used for BP monitoring and risk management using information and communication technology (ICT). In cooperation with the practitioners in Minamisanriku Town, where the damage due to the earthquake was serious, we introduced an ICT-based BP monitoring device at evacuation centers and shared patients' BP values in the database to support BP management by remote monitoring (Figure 5).[22–24] Consequently, we succeeded in improving BP control and suppressing seasonal variation in BP (ie, an increase in BP from summer to winter), which has been reported over the years.[23,25] Thus, we believe that ICT might be useful for anticipating needed interventions against BP elevation after a disaster and may contribute to the suppression of disaster-related CVDs. Moreover, patients living in the disaster area may have difficulty visiting medical facilities. Recently, these ICTs have made it possible for home BP monitoring and medical consultation in telemedicine. Thus, by using ICT to manage high-risk patients closely, we can reduce the burden on medical institutions in disaster areas and support efficient risk management.[24,25] In the future, ICT is expected to be a cooperation system that connects temporary housing and regional medical institutions in disaster areas.

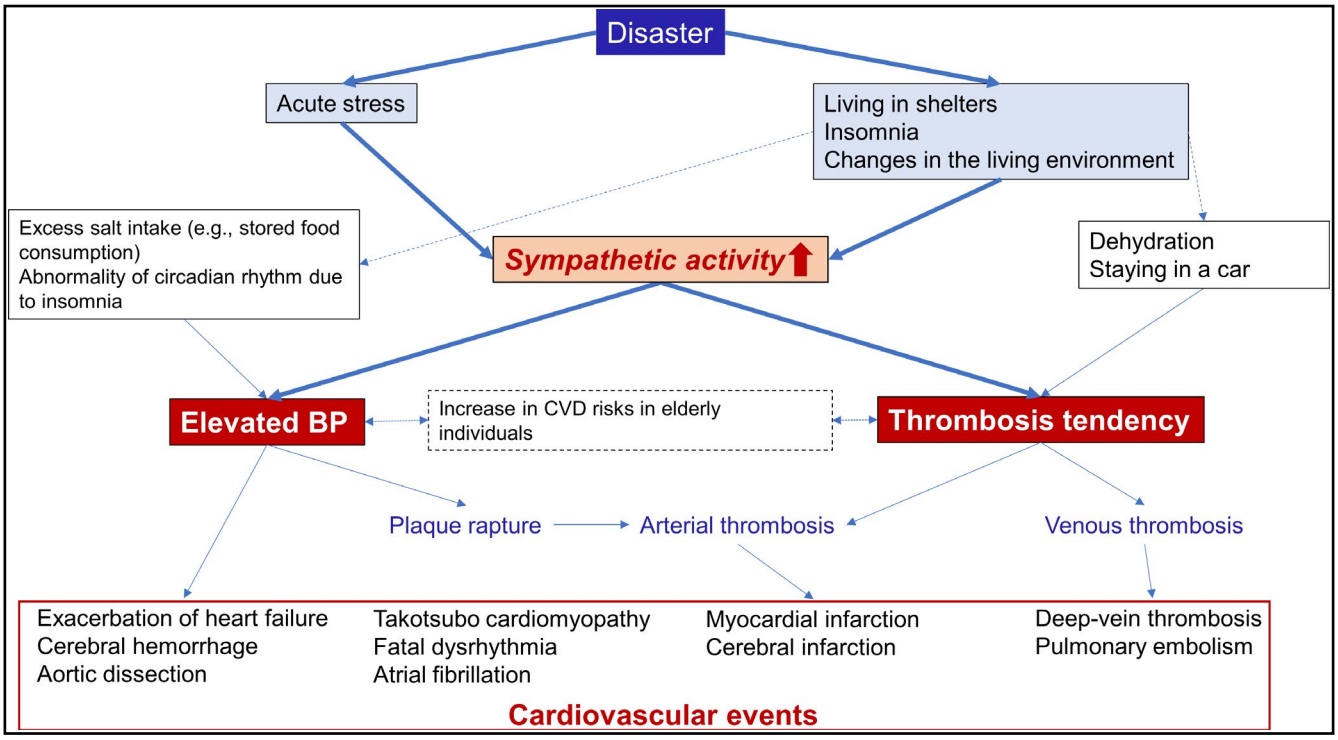

FIGURE 2 Potential mechanisms of the disaster-related cardiovascular risk factors. A disaster induces acute stress and changes in the living environment, which leads to hyperactivity of the sympathetic nervous system. Furthermore, excess salt intake and disruption of the circadian rhythm (eg, sleep disturbances) induce BP elevation or disaster hypertension. In addition to sympathetic hyperactivity, changes in the living environment and lifestyles, such as long stasis and dehydration, cause a tendency for thrombosis. Sympathetic hyperactivity, elevation of BP, and thrombotic tendencies are considered the causes of cardiovascular events. BP, blood pressure; CVD, cardiovascular disease

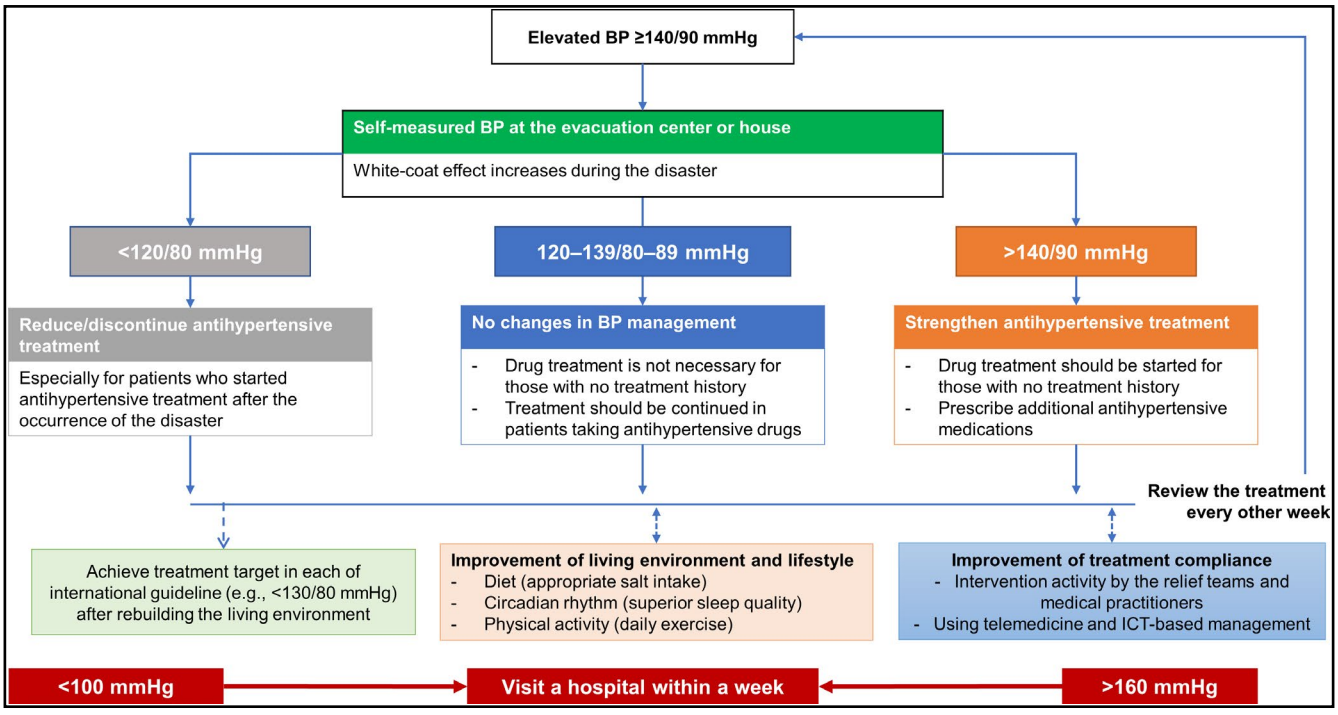

FIGURE 3 Flow chart of disaster hypertension management. BP, blood pressure

DCAP-AFHCHDC7 Risk Score ✓

1 · Age (A)	> 75 yrs	☐
2 · Family (F)	death or hospitalization (partner, parents, or children)	☐
3 · Housing (H)	completely destroyed	☐
4 · Community (C)	completely destroyed	☐
5 · Hypertension (H)	positive (under medication, or SBP > 160 mmHg)	☐
6. Diabetes (D)	positive	☐
7 · Cardiovascular disease (C)	positive (coronary artery disease, stroke, heart failure)	☐

Average total score* ____

DCAP-SEDWITMP8 Prevention Score ✓

1. Sleep (S)	Sleep duration > 6 h, arousal < 3 times during sleep	☐
2. Physical activity (E)	Walking > 20 min/day	☐
3. Diet (D)	Reduce salt intake with high potassium intake (three serves of green vegetable, fruit, or seaweed/day)	☐
4. Body weight (W)	Change < ± 2 kg	☐
5. Infection prevention (I)	Regular face mask use and washing hands	☐
6. Thrombosis (T)	Sufficient water intake >1000 ml per day	☐
7. Medication (M)	Continuous use of antihypertensive medication and antiplatelet agents and/or anticoagulation	☐
8. Blood pressure control (P)	< 140 mmHg SBP (clinic, shelter, or self-measured)	☐

Average total score# ____

FIGURE 4 Disaster cardiovascular prevention risk score (AFHCHDC7) and prevention score (SEDWITMP8). *Total number of risk factors as an individual's risk score (0–7 points). #Total number of prevention factors as an individual's prevention score (0–8 points). The target prevention score was ≥6 particularly in high-risk patients. DCAP, disaster cardiovascular prevention; SBP, systolic blood pressure. Source: Kario and colleagues[1]

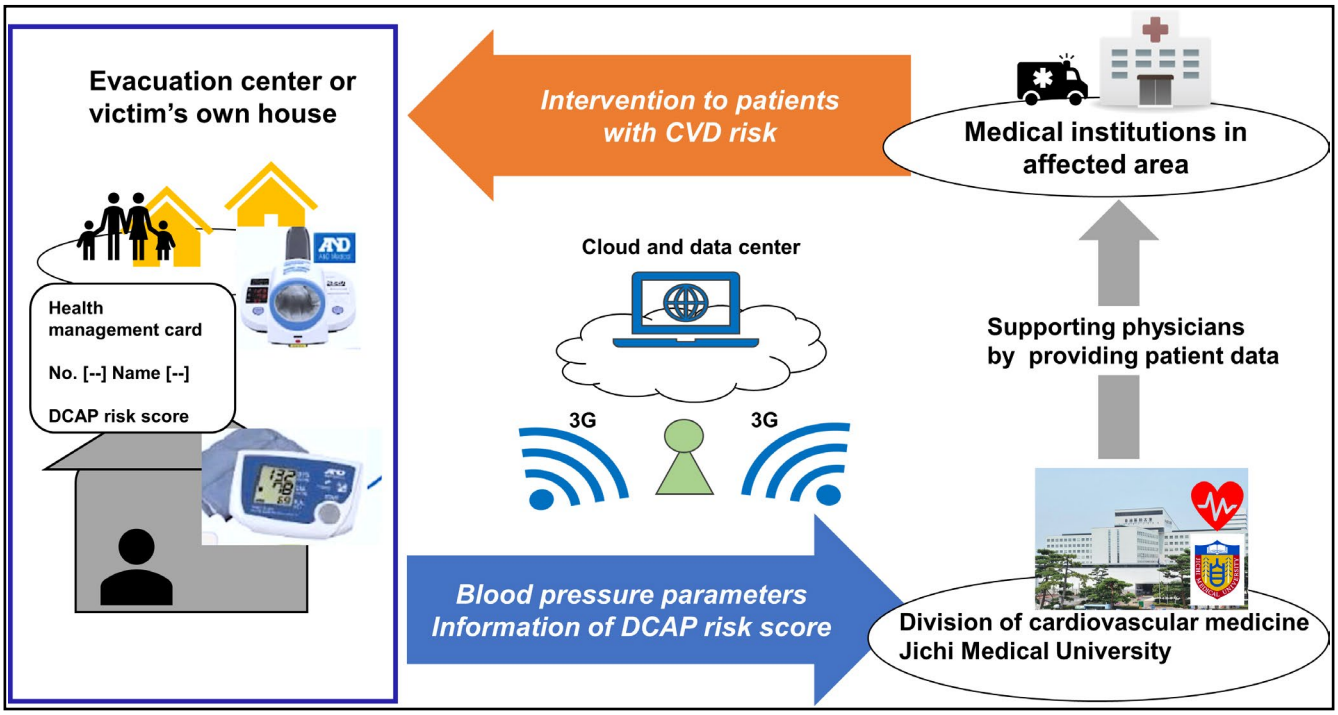

FIGURE 5 Disaster cardiovascular prevention network. The DCAP network system uses a blood pressure (BP) monitoring device equipped with a card reader and information and communication technology to measure BP and sends the measured BP values to a cloud data center. This BP monitoring device can be used for home BP monitoring. The DCAP network system can be useful for the prevention of cardiovascular events in high-risk patients living in evacuation centers or their houses during a disaster. DCAP, disaster cardiovascular prevention. Source: JCS, JSH, and JCC Joint Working Group,[2] Kario and colleagues[22]

5 | COVID-19 PANDEMIC AND CVD

COVID-19 is currently a pandemic because of its explosive infectivity. Patients with CVD comorbidities have a higher risk for severe COVID-19 infection. Furthermore, COVID-19 infection may cause abnormal coagulation and myocardial injury due to acute inflammation and may be associated with CVDs.[26,27]

Patients with hypertension may have a poor prognosis after COVID-19 infection. An epidemiological study in Italy reported that the mean age of the 3200 patients who died of COVID-19 was 78.5 years, of which 98.7% had one or more comorbidities, 74% had hypertension, and 52% used renin–angiotensin system (RAS) inhibitors.[28] However, the results of these observational studies could not be confirmed by direct evidence because of many confounding

TABLE 2 COVID-19 and comorbidities of CVD: assessment and management

Patients with hypertension, especially older individuals and those with other known risk factors, are at an increased risk of developing severe symptoms during COVID-19 infection
High-risk patients with CVDs, such as those with hypertension, are more likely to develop cardiac injury during COVID-19 infection. Particularly, in these high-risk patients, several biomarkers such as troponin and D-dimer should be assessed along with respiratory symptoms to evaluate organ injury including myocardial injury
In hospitalized COVID-19 patients, cardiac injury, which was evaluated by high-sensitivity troponin and creatinine kinase-myocardial band, was associated with a higher risk of in-hospital mortality
Anticoagulation therapy may be effective for improving the prognosis in COVID-19 patients who require treatment with as well as without respiratory ventilation
An observational study reported that anticoagulation therapy was associated with improved prognosis during hospitalization[33]
In patients with diabetes, myocardial injury and arteriovenous thrombosis should be carefully evaluated
Oxygen saturation should be determined at presentation; if oxygen saturation is <94%, then COVID-19 should be considered severe
Antihypertensive therapy with ACE inhibitors or ARBs in COVID-19 patients should be continued with careful monitoring of hypertension and kidney injury
Use of ACE inhibitors or ARBs does not increase the incidence of hospitalization or mortality compared with other classes of antihypertensive medications
Unmedicated older COVID-19 patients whose only comorbidity is hypertension can be treated with calcium channel blockers
Medical practitioners should be aware of physical manifestations of stress (eg, cardiovascular events), even in individuals without COVID-19 (especially those with pre-existing hypertension)
Even after recovery from COVID-19, the existence of myocardial injury should be considered
In hospitalized COVID-19 patients, cardiac injury, which was evaluated by high-sensitivity troponin and creatinine kinase-myocardial band, was associated with a higher risk of in-hospital mortality. An observational study using cardiac MRI revealed that patients who had recovered from COVID-19 infection had abnormal findings[36]
Medical practitioners working on treatment of COVID-19 should be checked for their mental state and psychological distress. Moreover, general people, that is, non-medical health care workers, also need to be checked for psychological distress in the era of COVID-19[37]

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; COVID, coronavirus disease; CVD, cardiovascular disease; MRI, magnetic resonance imaging.

factors such as age and because hypertension is a risk factor for poor prognoses such as severe illness and death. It is reasonable to conclude that older COVID-19 patients and general risk factors of CVD are at risk for severe morbidity and mortality.[26,27]

As a mechanism to enter and infect cells, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) binds to the transmembrane of angiotensin-converting enzyme 2 (ACE2), indicating that high ACE2 expression is susceptible to infection with SARS-CoV-2. Additionally, pathways related to ACE on the cell membrane affect inflammatory cytokines, which represent potential mechanisms for the cytokine storm seen in patients with COVID-19. Therefore, there is a concern that RAS inhibitors may increase the incidence and mortality of COVID-19 because RAS inhibitors have been shown to increase the expression of ACE2 on cell membranes.[26] However, observational studies from several countries have reported that the use of RAS inhibitors is not related to the incidence and severity of COVID-19 infection.[29–31] Additionally, a recent observational study reported that RAS inhibitors could be beneficial for the prevention of disturbance of consciousness.[32] At the time of writing this paper, no robust evidence confirmed that RAS inhibitors increase the risk of infection and mortality of COVID-19. Many societies and experts in Japan and other countries have announced that RAS inhibitors should not be discontinued in patients with hypertension or heart failure due to fear of COVID-19 infection.[27,30]

In severe COVID-19 infection, in addition to the severe respiratory syndrome, thrombotic tendency, and myocardial injuries develop, which may lead to arterial or venous thrombosis and heart failure. Further, an observational study reported that anticoagulation therapy was associated with improved outcomes during hospitalization.[33] Moreover, some observational studies have reported that the levels of some biomarkers related to myocardial injury and thrombosis, such as troponin and D-dimer, are elevated in COVID-19 patients.[34–36] In severe cases, evaluation of biomarkers related to myocardial injury and of lung lesions is necessary to stratify the risk of CVD complications and organ damage.[26] Table 2 summarizes the points of evaluation and management of CVDs related to COVID-19, which are described in this section.[26–37] Besides, the COVID-19 pandemic has brought great changes in people's lifestyles. The number of outpatients who directly visit a medical institution decreases due to restrictions on going outdoors. To solve this problem, telemedicine using ICT would be useful in outpatient practice.[38]

6 | DISRUPTION OF HEALTH CARE SYSTEMS

In the event of a disaster, health care systems are disrupted.[39] Even routine care preventing new CVD or control of pre-existing CVD may be affected. Many countries and regions differ in health care resilience due to available resources and the degree of preparedness. Medical provider groups will need to adjust their usual practice and mobilize resources to manage a disaster situation quickly.[40] This may mean that elective clinical work, research, and academic duties are deferred, and resources are pushed toward direct medical care in the affected disaster area.

7 | CONCLUSIONS

Recently, interest in medical care during disasters has been increasing owing to the occurrence of many large natural disasters

worldwide. New findings on disaster-related CVDs and disaster hypertension are being reported, and their preventive and coping methods are being developed. Moreover, in the future, the effective use of telemedicine using ICT would be important for risk management of CVDs during disasters and pandemics.[23–25,38]

ACKNOWLEDGMENTS

We would like to express our gratitude to Ayako Okura, the academic editorial coordinator of the Department of Cardiology, Jichi Medical University.

CONFLICT OF INTEREST

J Shin received lecture honoraria from Pfizer Inc, Hanmi Pharm. Co. Ltd., Yuhan Co. Ltd., Boryung Pharmaceutical Co. Ltd., and consulting fees from Hanmi Pharm. Co. Ltd. Handok Kalos Medical Inc, and research grants from Sanofi Pharm. and Hanmi Pharm. Co. Ltd. Siddique has received honoraria from Bayer, Novartis, Pfizer, ICI, and Servier, and travel, accommodation, and conference registration support from Hilton Pharma, Atco Pharmaceutical, Highnoon Laboratories, Horizon Pharma, and ICI. YC Chia has received speaker honoraria and sponsorship to attend conferences and seminars from Boeringher-Ingelheim, Pfizer, Omron, Servier, and Xepa-Sol and investigator-initiated research grants from Pfizer and Omron. CH Chen reports personal fees from Novartis, Sanofi, Daiichi Sankyo, SERVIER, Bayer, and Boehringer Ingelheim Pharmaceuticals, Inc HM Cheng received speaker honoraria and sponsorship to attend conferences and CME seminars from Eli Lilly and AstraZeneca; Pfizer Inc; Bayer AG; Boehringer Ingelheim Pharmaceuticals, Inc; Daiichi Sankyo, Novartis Pharmaceuticals, Inc; SERVIER; Co., Pharmaceuticals Corporation; Sanofi; TAKEDA Pharmaceuticals International; Menarini Co., Ltd., and served as an advisor or consultant for ApoDx Technology, Inc JG Wang has received research grants from Chendu Di-Ao and Omron, and lecture and consulting fees from AstraZeneca, Novartis, Omron, Servier, and Takeda. K Kario reports receiving research grants from A&D Co. and Omron Healthcare. Other authors report no potential conflicts of interest related to this review.

AUTHORS' CONTRIBUTION

Kario K takes primary responsibility for this paper. Narita K wrote the manuscript. Narita K, Hoshide S, Tsoi K, Siddique S, Shin J, Chia YC, Tay JC, Teo BW, Turana Y, Chen CH, Cheng HM, Sogunuru GP, Wang TD, Wang JG, and Kario K reviewed/edited the manuscript.

ORCID

Keisuke Narita https://orcid.org/0000-0002-2020-898X
Satoshi Hoshide https://orcid.org/0000-0001-7541-5751
Kelvin Tsoi https://orcid.org/0000-0001-5580-7686
Saulat Siddique https://orcid.org/0000-0003-1294-0430
Jinho Shin https://orcid.org/0000-0001-6706-6504
Yook-Chin Chia https://orcid.org/0000-0003-1995-0359
Jam Chin Tay https://orcid.org/0000-0001-7657-4383
Boon Wee Teo https://orcid.org/0000-0002-4911-8507
Yuda Turana https://orcid.org/0000-0003-4527-0285
Chen-Huan Chen https://orcid.org/0000-0002-9262-0287
Hao-Min Cheng https://orcid.org/0000-0002-3885-6600
Guru Prasad Sogunuru https://orcid.org/0000-0002-1410-9328
Tzung-Dau Wang https://orcid.org/0000-0002-7180-3607
Kazuomi Kario https://orcid.org/0000-0002-8251-4480

REFERENCES

1. Kario K. Disaster hypertension-its characteristics, mechanisms, and management. *Circ J*. 2012;76(3):553-562.
2. JCS, JSH, and JCC Joint Working Group. Guidelines for disaster medicine for patients with cardiovascular diseases (JCS 2014/JSH 2014/JCC 2014)-Digest version. *Circ J*. 2016;80(1):261-284.
3. Watanabe H, Kodama M, Okura Y, et al. Impact of earthquake on Takotsubo cardiomyopathy. *JAMA*. 2005;294(3):305-307.
4. Leor J, Poole WK, Kloner RA. Sudden cardiac death triggered by an earthquake. *N Engl J Med*. 1996;334(7):413-419.
5. Aoki T, Fukumoto Y, Yasuda S, et al. The Great East Japan Earthquake disaster and cardiovascular diseases. *Eur Heart J*. 2012;33(22):2796-2803.
6. Kario K, Matsuo T, Ishida T, Shimada K. "White coat" hypertension and the Hanshin-Awajji earthquake. *Lancet*. 1995;345(8961):1365.
7. Kario K, Matsuo T, Kobayashi H, Yamamoto K, Shimada K. Earthquake-induced potentiation of acute risk factors in hypertensive elderly patients: possible triggering of cardiovascular events after a major earthquake. *J Am Coll Cardiol*. 1997;29(5):926-933.
8. Hoshide S, Nishizawa M, Okawara Y, et al. Salt intake and risk of disaster hypertension among evacuees in a shelter after the Great East Japan Earthquake. *Hypertension*. 2019;74(3):564-571.
9. Geng F, Fan F, Lei MO, Simandi I, Liu X. Sleep problems among adolescent survivors following the 2008 Wenchuan earthquake in China: a cohort study. *J Clin Psychiatry*. 2013;74(1):67-74.
10. Zhang XQ, Chen M, Yang Q, Yan SD, Huang DJ. Effect of the Wenchuan earthquake in China on hemodynamically unstable ventricular tachyarrhythmia in hospitalized patients. *Am J Cardiol*. 2009;103(7):994-997.
11. Hao K, Takahashi J, Aoki T, et al. Factors influencing the occurrence of cardiopulmonary arrest in the Great East Japan Earthquake disaster. *Int J Cardiol*. 2014;177(2):569-572.
12. Sato K, Sakamoto K, Hashimoto Y, et al. Risk factors and prevalence of deep vein thrombosis after the 2016 Kumamoto Earthquakes. *Circ J*. 2019;83:1342-1348.
13. Satoh M, Kikuya M, Ohkubo T, Imai Y. Acute and subacute effects of the Great East Japan Earthquake on home blood pressure values. *Hypertension*. 2011;58:e193-e194.
14. Huang K, Huang D, He D, et al. Changes in hospitalization for ischemic heart disease after the 2008 Sichuan earthquake: 10 years of data in a population of 300,000. *Disaster Med Public Health Prep*. 2016;10(2):203-210.
15. Komorita T, Fujisue K, Sueta D, et al. Clinical features of patients with acute aortic dissection after an earthquake: experience from the Kumamoto Earthquake 2016. *Am J Hypertens*. 2020;33(3):261-268.
16. Kario K, Chia YC, Sukonthasarn A, et al. Diversity of and initiatives for hypertension management in Asia – Why we need the HOPE Asia Network. *J Clin Hypertens*. 2020;22(3):331-343.
17. Chia YC, Kario K, Turana Y, et al. Target blood pressure and control status in Asia. *J Clin Hypertens*. 2020;22(3):344-350.
18. Chen Y, Li J, Xian H, et al. Acute cardiovascular effects of the Wenchuan earthquake: ambulatory blood pressure monitoring of hypertensive patients. *Hypertens Res*. 2009;32(9):797-800.
19. Nishizawa M, Fujiwara T, Hoshide S, et al. Winter morning surge in blood pressure after the Great East Japan Earthquake. *J Clin Hypertens*. 2019;21(2):208-216.

20. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2018;71(6):e13-e115.
21. Umemura S, Arima H, Arima S, et al. The Japanese Society of Hypertension Guidelines for the Management of Hypertension (JSH2019). *Hypertens Res*. 2019;42(9):1235-1481.
22. Kario K, Nishizawa M, Hoshide S, et al. Development of a disaster cardiovascular prevention network. *Lancet*. 2011;378(9797):1125-1127.
23. Nishizawa M, Hoshide S, Okawara Y, Matsuo T, Kario K. Strict blood pressure control achieved using an ICT-based home blood pressure monitoring system in a catastrophically damaged area after a disaster. *J Clin Hypertens*. 2017;19(1):26-29.
24. Omboni S, McManus RJ, Bosworth HB, et al. Evidence and recommendations on the use of telemedicine for the management of arterial hypertension: An International expert position paper. *Hypertension*. 2020;76(5):1368-1383.
25. Park S, Kario K, Chia YC, et al. The influence of the ambient temperature on blood pressure and how it will affect the epidemiology of hypertension in Asia. *J Clin Hypertens*. 2020;22(3):438-444.
26. Kario K, Morisawa Y, Sukonthasarn A, et al. COVID-19 and hypertension-evidence and practical management: guidance from the HOPE Asia Network. *J Clin Hypertens*. 2020;22(7):1109-1119.
27. Shibata S, Arima H, Asayama K, et al. Hypertension and related disease in the era of COVID-19: a report from the Japanese society of hypertension task force on COVID-19. *Hypertens Res*. 2020;43(10):1028-1046.
28. Instituto Superiore Di Santia. Characteristics of COVID-19 patients dying in Italy. Report based on data available on March 20th, 2020. Available from: https://www.epicentro.iss.it/coronavirus/bollettino/Report-COVID-2019_20_marzo_eng.pdf. Accessed 12 May 2020.
29. Li J, Wang X, Chen J, Zhang H, Deng A. Association of renin-angiotensin system inhibitors with severity or risk of death in patients with hypertension hospitalized for coronavirus disease 2019 (COVID-19) infection in Wuhan, China. *JAMA Cardiol*. 2020;5:825-830.
30. Park S, Lee HY, Cho EJ, et al. and on behalf of the Korean Society of Hypertension. Is the use of RAS inhibitors safe in the current era of COVID-19 pandemic?. *Clin Hypertens*. 2020;26:11.
31. Son M, Seo J, Yang S. Association between renin-angiotensin-aldosterone system inhibitors and COVID-19 infection in south Korea. *Hypertension*. 2020;76(3):742-749.
32. Matsuzawa Y, Ogawa H, Kimura K, et al. Renin-angiotensin system inhibitors and the severity of coronavirus disease 2019 in Kanagawa, Japan: a retrospective cohort study. *Hypertens Res*. 2020;43(11):1257-1266.
33. Paranjpe I, Fuster V, Lala A, et al. Association of treatment dose anticoagulation with in-hospital survival among hospitalized patients with COVID-19. *J Am Coll Cardiol*. 2020;76(1):122-124.
34. Zhai Z, Li C, Chen Y, et al. Prevention Treatment of VTE Associated with COVID-19 Infection Consensus Statement Group. Prevention and treatment of venous thromboembolism associated with coronavirus disease 2019 infection: a consensus statement before guidelines. *Thromb Haemost*. 2020;120(6):937-948.
35. Shi S, Qin M, Shen B, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. *JAMA Cardiol*. 2020;5(7):802-810.
36. Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from Coronavirus disease 2019 (COVID-19). *JAMA Cardiol*. 2020;5(11):1265-1273.
37. Tan BYQ, Chew NWS, Lee GKH, et al. Psychological impact of the COVID-19 pandemic on health care workers in Singapore. *Ann Intern Med*. 2020;173(4):317-320.
38. Wang LYT, Low TT, Yeo TJ. Telehealth in COVID-19 and cardiovascular disease-ensuring equitable care. *Ann Acad Med Singap*. 2020;49:902-904.
39. Carballo M, Daita S, Hernandez M. Impact of the Tsunami on healthcare systems. *J R Soc Med*. 2005;98(9):390-395.
40. Lee MBH, Chua HR, Wong WK, et al. Going to war on COVID-19: mobilizing an academic nephrology group practice. *Nephrology*. 2020;25(11):822-838.

How to cite this article: Narita K, Hoshide S, Tsoi K, et al. Disaster hypertension and cardiovascular events in disaster and COVID-19 pandemic. *J Clin Hypertens*. 2021;23:575–583. https://doi.org/10.1111/jch.14192