Measuring the effects of stress on the cardiovascular system during a disaster: the effective use of self-measured blood pressure monitoring

Kazuomi Kario

Journal of Hypertension 2010, 28:657–659

There is growing evidence that psychological stress contributes to hypertension and cardiovascular disease [1–4]. Previous studies [5–7] have clearly demonstrated that major earthquakes and other natural disasters can trigger cardiovascular events through psychological and physical stress in the victims. Moreover, the health-related effects of such disasters are not only confined to the victims but also extend to the participants who care for them. In this issue, Azuma et al. [8] report that the pressor effects associated with the recent Mid-Niigata Earthquake in Japan often continued after the event, particularly in exhausted social workers (governmental staff). Their study indicates that both the direct psychological effects of such disasters and an overload of chronic work stress can cause persistent blood pressure (BP) increases and corresponding elevation of cardiovascular risk.

In this editorial comment, I will characterize the pressor effect of the disaster and potential mechanism to recommend the practical management of BP, using self-measured BP during disaster situations.

Cardiovascular diseases such as coronary artery disease, stroke, cardiac sudden death, Takotsubo cardiomyopathy, and fatal ventricular arrhythmia, as well as pulmonary thromboembolism and deep vein thrombosis, have been reported during and after various disasters, including earthquakes, the Iraqi missile war, and the World Trade Center attack [4–7,9–12].

I was working at the Awaji-Hokudan public clinic during the Hanshin-Awaji earthquake of 1995, which measured 7.2 on the Richter scale and had its epicenter in the town of Hanshin-Awaji. The earthquake caused 5488 deaths and tens of thousands of other casualties [9]. I worked hard to care for the victims immediately after the event, and retrospectively studied the effects of the earthquake on cardiovascular disease and related risk factors. Cardiovascular death (stroke, coronary artery disease, and sudden death) indeed occurred more frequently in the few months just after the earthquake when compared with the same period of the previous years, and the number of such deaths increased in the high-risk older population in proportion to the damage of the disaster [6,7]. Increases in such deaths typically occurred during night-time, suggesting that poor sleep quality might have affected disaster-induced cardiovascular disease [6,7].

Various cardiovascular risk factors, including BP, are known to be potentiated by disasters through sympathetic nervous activation (Fig. 1) [13]. In the earlier period after a disaster, fear may be the major acute stressor activating the sympathetic nervous system. Several studies have shown that BP and heart rate (HR) are increased at the time of a disaster. In our study of hypertensive patients who lived near the earthquake epicenter, increases of approximately 18 mmHg in SBP and 8 mmHg in DBP were found during the first 2 weeks after the earthquake when compared with the BP levels before the earthquake [14]. An additional BP increase might have occurred in felt aftershocks and triggered cardiovascular events. Parati et al. [15] previously reported a patient in whom ambulatory BP monitoring (ABPM) was performed on the day of an earthquake, and found a transient increase in BP (130/85–150/122 mmHg) and HR (83–150 bpm) at the time of the shock. A similar acute pressure effect on ambulatory BP was found in hypertensive patients at the Wenchuan Earthquake recently occurring in China [16].

In most patients, this earthquake BP increase is transient, and returns to the pre-earthquake baseline levels within 4 weeks. A similar time course of BP changes was also observed by Saito et al. [17] and by Minami et al. [18], using home BP monitoring. This characteristic of disaster-induced BP increase is important because persistent intense antihypertensive treatment for patients with high BP at the time of a disaster could result in huge reductions of BP after the stress has disappeared.

The effect of acute stress may be prolonged [19]. Chronic stress caused by disaster-induced environmental changes can prolong the initial increase in BP. The loss of finances and health experienced by the victims and their families, and the long hours put in by social workers can lead to
chronic stress, depression, and sleep disturbances that in turn can activate the neuroendocrine and immune systems (sympathetic nervous system, hypothalamus–pituitary–adrenal axis, and cytokines) and confer related cardiovascular risks (Fig. 1).

In addition, increases in salt intake and body weight, both of which are caused by behavioral changes after a disaster, may contribute to an increase in BP. Stress induces sodium retention particularly in obese patients [20]. In addition, even in the younger normotensive individuals, obesity and smoking augment stress-induced increases in BP and augmentation index, indicating that the central aortic pressure may be further increased in obese patients than estimated by brachial pressure [21]. Thus, to prevent a BP increase over the weeks and months following a disaster, restriction of sodium intake and body weight control is essential.

The presence of microalbuminuria may be one of the determinants of persistence of the disaster-induced BP increase. In hypertensive patients with microalbuminuria, recovery from a transient BP increase is impaired and BP elevation persists for several months or more [14]. We observed some patients with white-coat hypertension who shifted to a pattern of sustained hypertension (both clinic and ambulatory high BP) after the earthquake. In these patients, the BP increase was still present 2 months after the earthquake, and antihypertensive medication was then needed to control BP [23]. All these patients had microalbuminuria. After 1 year of treatment, the 24-h ambulatory BP decreased to the level before the earthquake. However, the clinic BP remained high, indicating that the white-coat effect can persist in treated patients [24].

Because the effects of stress on cardiovascular risk and its persistence differ substantially among individual victims and social workers, an individual approach would be the most effective for preventing cardiovascular events in a disaster situation. Self-measured BP monitoring is useful to determine the effects of stress on the cardiovascular system. Self-measured BP, particularly in the morning, may partly reflect poor sleep quality caused by disaster-related psychological stress and depression.

In a disaster situation, the BP readings taken by doctors and medical volunteers unfamiliar to the patient may be increased by an exaggerated white-coat effect. Thus, self-measured BP-guided BP management is recommended for the BP control in hypertensive patients in a disaster situation. In medicated hypertensive patients, even those whose clinic BP is well controlled, the morning BP level before taking medicine frequently remains high [25]. Thus, BP measurement in the morning before taking pills is recommended to determine the effect of disaster-related stress and BP control status. Controlling the morning BP level to below 135/85 mmHg is important for the effective prevention of cardiovascular disease, as recommended in the international guidelines [26–28].

There is no clear evidence advocating for a specific class of antihypertensive drugs during a disaster. Two studies [14,29] have demonstrated that hypertensive patients taking alpha or beta-adrenergic blockers are likely to exhibit a smaller earthquake-induced BP increase than those taking other classes of antihypertensive drugs, but these are retrospective studies with small numbers of patients. Antihypertensive medication should be evaluated repeatedly every 2 weeks because a disaster-induced BP increase may return to the predisaster level after the stress has disappeared.

References

Stress and blood pressure in disasters

Kario 659


