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COMMENT

Psychological distress—pathophysiology of newly developed hypertension after the Great East Japan Earthquake

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At 14:46 on March 11, 2011, there was a major earthquake with a moment magnitude of 9.0 on the Pacific coast of the Tohoku region. It was later named the Great East Japan Earthquake. After the earthquake, a large tsunami and a large-scale nuclear accident occurred at the Fukushima Daiichi Nuclear Power Station, releasing radioactive elements into the environment. Many inhabitants have died, and survivors suffer from sequelae [1]. In earlier studies conducted after a major earthquake, 24% of survivors were diagnosed with posttraumatic stress disorder (PTSD), and 5.8–54% of adult survivors had a major depressive disorder. Twenty percent of survivors experienced significant anxiety symptoms [2].

In the paper by Kobari et al. in this issue of the *Journal*, a 7-year follow-up study of the Fukushima Healthcare Survey investigated the impact of lifestyle or psychosocial factors on the development of hypertension after the Great East Japan Earthquake [1]. Drinking, obesity, and evacuation were significant predictors of newly developed hypertension during the long-term follow-up period [1]. A previous study observed an increase in systolic blood pressure (BP) of 18 mmHg and diastolic BP of 8 mmHg in hypertensive patients living near the epicenter of the Hanshin-Awaji earthquake 1–2 weeks after the earthquake [3].

A meta-analysis of recent studies showed an acute biphasic effect of ethanol on BP, which decreased up to 12 h after ingestion and then increased. This effect is mediated by vagal suppression and sympathetic activation. Evidence from various studies has converged on the risk of chronic alcohol consumption for the incidence of hypertension [4]. In another analysis of the Fukushima Healthcare Survey, sleep disorders and more severe traumatic symptoms were significantly associated with non-drinkers becoming drinkers [5].

The neurobiological explanation that is believed to account for loss of control and compulsive urges to drink primarily involves behavioral and brain circuits involved in the handling of rewards and stress associated with emotional and motivational information [6]. In alcohol use disorders, brain regions, including structures such as the striatum, medial prefrontal cortex, anterior cingulate cortex, amygdala, and insular cortex (Ic), have been proposed as addiction circuits [6]. In an arterial spin labeling magnetic resonance imaging (MRI) study, cerebral blood flow (CBF) in the Ic was negatively associated with heavy drinking, and the severity of alcohol use was associated with reduced CBF in key nodes of the salience network composed of the anterior Ic and anterior cingulate cortex [7]. In the population presented in the manuscript by Kobari et al. [1], it might be difficult to correct drinking habits after the earthquake due to shifting brain circuits to a mode of alcoholism.

In the Fukushima Health Management Survey reported by Kobari et al. [1], only among females was a significant relationship observed between obesity and new-onset hypertension in the fully adjusted model. In addition to obesity at baseline, overeating as a response to stress may be involved in the development of hypertension specifically in women. In the 2010 Canterbury earthquake, women who scored high on emotional eating reported an increase in post-earthquake binge eating [8]. The Nurses' Health Study and the Health Professionals Follow-up Study have shown that the degree of overweight predicts the incidence of hypertension [9].

Obesity is associated with the autonomic imbalance and is thought to be involved in the development of hypertension through an increase in the sympathetic nervous system.

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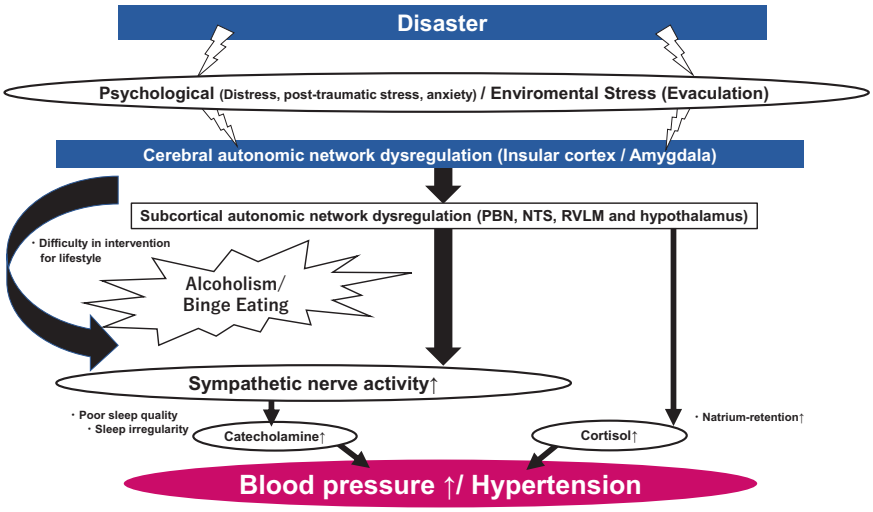

Fig. 1 A possible pathway for the relationship between disaster and hypertension in the Great East Japan Earthquake. PBN parabrachial nucleus, NTS nucleus tractus solitarius, RVLM rostral ventrolateral medulla

Diet- and exercise-based weight loss appears to increase parasympathetic nerves and reduce sympathetic nervous system activity, with the opposite effect observed with weight gain [10].

The central role of the hypothalamus in the generation and/or processing of feeding-related stimuli is regulated by the activity of functional areas of the brain involved in feeding behavior. In an early study of positron emission tomography, hunger was associated with significantly increased regional CBF (rCBF) in the hypothalamus and Ic. Changes in plasma insulin levels with diet were negatively correlated with changes in rCBF in the Ic [11]. In a meta-analysis of functional MRI studies, in response to food images, obese subjects had reduced activation of the left dorsolateral prefrontal area and left Ic compared to healthy-weight subjects [12]. Leptin is an adipocyte hormone that regulates energy balance and hippocampal plasticity via the basal hypothalamus and exhibits high receptor expression in anterior Ic pyramidal neurons [13, 14]. Decreased excitability of leptin-sensitive anterior Ic pyramidal neurons was observed in a rat model of compulsive food demand [14].

Evacuees who rebuilt their permanent homes after the Great East Japan Earthquake have been shown to continue to suffer from psychological distress, posttraumatic stress, and radiation-induced health anxiety, even if their living environment improves after evacuation [15]. In the Fukushima Health Management Survey reported by Kobari et al. [1], only among males was a significant relationship observed between evacuation experience and new-onset hypertension in a fully adjusted model. Males are more likely to feel stress during evacuation than females, which may affect long-term psychological distress.

It has been speculated that a central autonomic network (CAN) comprising the periaqueductal gray matter, parabrachial nucleus, nucleus tractus solitarius, ventrolateral medulla, hypothalamus, amygdala, and the Ic regulates the human cardiovascular system and that the network of these cortical regions is necessary to regulate the CAN in response to emotional and environmental stress [16]. The CAN functions as an integral component of an internal regulation system with which the brain controls the visceromotor and neuroendocrine systems [16]. Earlier neuroimaging studies revealed that activation of the Ic is also involved in the processing of negative emotions such as fear and anxiety [17]. Previous neuroimaging data revealed that Ic is involved in a variety of neuropsychiatric disorders, such as depression, panic disorder, PTSD, and eating disorders [17].

In spontaneously hypertensive rats, increased sympathetic nervous system activity was shown to be attributed to alterations in the neurovascular unit in the Ic, leading to changes in neurogenic BP elevation [18]. In the Jichi Medical School ABPM Study Wave 2 Core, right Ic atrophy had a significant correlation with the level of noradrenaline [16]. In addition, Ic atrophy was associated with disrupted diurnal ambulatory BP rhythm [19]. Disaster distress is associated with CAN dysfunction, leading to behavioral changes such as drinking, overeating, and increasing sympathetic nervous system activity (Fig. 1). In the Fukushima Health Management Survey, disruption of CAN, including Ic, may cause the predominant sympathovagal balance to shift toward increased sympathetic nervous system activity and increase the risk of new developments in hypertension, as reported by Kobari et al. [1].

To date, few long-term follow-up studies have evaluated the impact of lifestyle or psychosocial factors on the development of hypertension after a natural disaster. It may be important to minimize psychological distress and anxiety to prevent further disaster-related cardiovascular events. Therefore, the data presented in the study by Kobari et al. provide important implications for the prevention of possible future disaster hypertension.

Compliance with ethical standards

Conflict of interest The authors declare no competing interests.

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