Aftershock Triggers Augmented Pressor Effects in Survivors: Follow-Up of the Great East Japan Earthquake

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BACKGROUND
Previous reports focused on the significant acute effects immediately after an earthquake on an increase in blood pressure (BP) assessed by ambulatory BP monitoring. However, there have been no data on the impact of environmental triggers on the long-term response to earthquake.

METHODS
We analyzed the ambulatory BP monitoring data of 8 patients who lived in the disaster area of the 11 March 2011 Great East Japan Earthquake on the day of the largest aftershock with a first tsunami warming (sirens) on 7 December 2012.

RESULTS
There was no significant difference in the BP in either the period 1 hour before (median (range): 126.5 (121.5–138.0) vs. 137.8 (129.5–177.0) mm Hg, P = 0.07) or that 1 hour after (139.3 (113.0–143.5) vs. 137.5 mm Hg) the aftershock between those living at home and those who had been living in temporary housing. After the time of aftershock, the systolic BP levels at that night (124.9 (113.2–137.9) vs. 107.0 (101.9–110.1) mm Hg, P = 0.021) and systolic BP levels at the following morning (149.3 (131.0–196.2) vs. 129.5 (128.8–131.0) mm Hg, P = 0.029) were also significantly higher in those living in temporary housing at the time compared to those living in their own homes.

CONCLUSION
Our data suggest that the stress of a change in living conditions following the disaster might have contributed an increased risk of cardiovascular events.

Keywords: aftershock; blood pressure; earthquake; hypertension; temporary housing.

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The Great East Japan Earthquake that occurred in Tohoku in the northeast region of Japan's main island, Honshu, on 11 March 2011 had a magnitude of 9.0, triggering a powerful tsunami wave. This major disaster forced thousands of people in a very widespread area to evacuate their homes and resort to temporary housing. Previous studies demonstrated that natural disasters and the subsequent changes in the affected population's lifestyle can cause “disaster hypertension” and increase the incidence of cardiovascular disease.1,2

On 7 December 2012, at 5:18 p.m., the largest palpable aftershock of the Great East Japan Earthquake occurred, reaching a magnitude of 7.2 on the Richter scale. At that time, the tsunami warning (sirens) and the evacuation order to a nearby hill were issued first in the Minamisanriku area (one of the areas most severely damaged by the 2011 tsunami). An earthquake can trigger the blood pressure (BP) pressor effect, but most of the relevant extant studies focused on BP changes on the day of an earthquake1−5 or BP changes during a relatively short period after an earthquake.1−3,6,7 There are no reports on the acute pressor effect of a second earthquake or aftershock (i.e., the most intensely felt aftershock) that occurred long after a primary major earthquake, to our knowledge.

A significant number of people continue to live in temporary housing since the Great East Japan Earthquake occurred in 2011. Marked heterogeneity exists in the individual cardiovascular reactivity to the same event. The environmental changes under post-disaster temporary housing conditions can impair the mental and physical conditions of the residents,8 which might result in consequent abnormal circadian rhythms even after a long-term disaster. Recurring emotional reactions, such as the reactions to an earthquake aftershock or the sound of sirens, even long after the disaster, can trigger upsetting memories of the traumatic experience. These triggers may be accompanied by anxiety that the stressful event will be repeated.

We had the opportunity to analyze the ambulatory BP monitoring data of individuals living in temporary housing in the town of Minamisanriku on the day of the largest...
palpable aftershock. In the present report, we describe how the effects on BP persisted in relation to the living condition even several years after the earthquake among the individuals in temporary/emergency housing, and we indicate how important it is to improve the environment and lifestyle of disaster-affected persons as soon as possible.

METHODS AND PATIENTS

Minamisanriku is a town located in the northeast of Miyagi Prefecture in the Tohoku district of Japan (Supplementary Figure S1A). On 11 March 2011, the 9.0-magnitude Great East Japan Earthquake caused a massive tsunami (more than 16 m) that hit Minamisanriku (Supplementary Figure S1B). Approximately 800 people died or disappeared, and approximately 60% of the town’s residential area was also demolished by the tsunami (Supplementary Figure S1C). The number of Minamisanriku’s residents who had to move to temporary housing because their homes were destroyed (Supplementary Figure S1D) was 6,000 people (40% of the total population) in March 2011. The living area provided to each family in temporary housing was insufficient (29.7 m² area).

As a substudy of the Japan Ambulatory Blood Pressure Prospective (JAMP) study, we had started the JAMP-Recovery from EarthQuake (REQ) study 20 months after the earthquake in Minamisanriku to study the effects of long-term psychological stress and environmental changes on BP and cardiovascular consequences. The JAMP study is a nationwide prospective observational study of Japanese patients with one or more cardiovascular risk factors that aims to evaluate regional differences in BP control status and its effect on cardiovascular events in different areas all around Japan. We were able to analyze the present cases during the registry period of the JAMP-REQ study.

On 7 December 2012, 8 patients who were being seen for hypertension at the Shizugawa Public Hospital in Minamisanriku were in the process of performing ambulatory BP monitoring when the largest aftershock (7.2 magnitude) of The Great East Japan Earthquake occurred. Their ambulatory BP monitoring recordings (TM 2431, A&D, Saitama, Japan) were started from 9:00 to 11:00 on 7 December 2012 and were taken every 30 minutes throughout the 24-hour period. On 7 December, an aftershock occurred at 17:18, and all the 8 patients noticed that their homes were damaged. All of the patients heard the tsunami siren twice at 17:22 and 19:20.

Statistical analysis

Data are expressed as the median (range). A nonparametric Mann–Whitney test was used for comparisons between 2 groups. Wilcoxon signed rank tests were used to assess BP during the hour before and the hour after the aftershock. All the statistical analyses were performed with SPSS version 18.0 (SPSS, Chicago, IL).

RESULTS

The characteristics and BP parameters of each the 8 patients are shown in Table 1. Three were male and 5 were female, and the average age was 76 (68–82) years. Median of 24-hour systolic BP, diastolic BP, and pulse rate were 125.7 (119.4–136.0) mm Hg, 72.4 (68.4–78.6) mm Hg, and 68.2 (57.0–72.8) bpm, respectively. There was no significant difference in age (73.5 (73–79) vs. 78 (68–82) years, P = 0.69), systolic BP (130.4 (120.2–136.0) vs. 124.5 (119.4–125.8), P = 0.20) or diastolic BP (71.9 (71.1–78.6) vs. 72.7 (68.4–74.1) mm Hg, P = 0.89) between the 4 patients living in temporary housing (4 females) and the 3 males and 1 female patients living in their own homes. All of the patients noticed that their systolic BP during the hour immediately after the aftershock (2 BP readings) was significantly elevated compared with that during the hour just before the event (133.0 (113.0–143.5) vs. 137.5 (125.0–192.0) mm Hg, P = 0.025). When subjects were categorized by housing conditions, there was no significant difference in the systolic BPs between the hour before and the hour after the aftershock in either those living at home (126.5 (121.5–138.0) vs. 137.8 (129.5–177.0) mm Hg, P = 0.07) or those who had been living in temporary housing (139.3 (113.0–143.5) vs. 137.5 (125.0–192.0) mm Hg, P = 0.27).

Next, we investigated the Bp difference in the sleep BP at that night and the following morning BP after the aftershock between those living in temporary housing and those living at home. The systolic BP at 22:00–2:00 (124.9 (113.2–137.9) vs. 107.0 (101.9–110.1) mm Hg, P = 0.021) and 7:30–9:00 (149.3 (131.0–196.2) vs. 129.5 (128.8–131.0) mm Hg, P = 0.029) in the patients living in temporary housing was significantly higher than that in those living in their own homes. Finally, we investigated the difference in the following pulse rate after the aftershock between those living at home and those living in temporary housing. The pulse rate throughout the period after the aftershock in patients living in temporary housing was significantly higher than that in those living in their own homes (70.1 (68.8–70.3) vs. 62.5 (53.9–64.7), P = 0.021).

DISCUSSION

The results of the present analyses showed that the people who lost their homes and still live in temporary housing exhibited less dipping of nocturnal BP and more exaggerated morning BP surges at the time of a palpable aftershock, even at 19 months after the Great East Japan Earthquake, compared to the people who living in their own homes.

Previous studies have focused on increases in BP and pulse rate in response to the acute stress of experiencing an earthquake. The impact of an acutely stressful event such as an earthquake on the elevation of BP is believed to involve a sympathetic nervous system response. Our present analyses showed that the patients’ BP values were significantly higher 1 hour just after a major aftershock compared to 1 hour just before supported the findings in these previous studies. The new finding obtained in the present study is that there was no significant difference in the BPs either in first hour before or in the first hour after the aftershock between those living at home and those living in temporary housing. We speculated that this was because a BP change would be a physiological response. In contrast, the findings that those in temporary housing had higher the following sleep and morning BP levels after the aftershock than those in their own homes may have been due to pathological responses. The patients living in temporary
housing had lost their homes to the tsunami, and such a loss itself is stressful; protracted living in temporary housing, as well as separation from their communities after the disaster, took a heavy emotional toll on the victims and their families. These patients might be likely to feel more fear or anxiety over a tsunami than those whose homes were not significantly damaged by the tsunami. As a result, a synergistic effect of emotional stress, i.e., the palpable aftershock and tsunami warning and the environmental stress of living in temporary housing for so long might have augmented the disrupted circadian BP variation even 19 months after the disaster.

Poor sleep quality causes increasing sympathetic nervous activity, which in turn results in disrupted circadian BP variation, non-dipping in nocturnal BP, and/or morning hypertension. In the present analysis, environmental changes including living in temporary housing after a disaster (which might lead to different sleeping habits from before the disaster) may induce sympathetic nervous activation, which might lead to high sleep and morning BP elevation. Morning hypertension and disrupted circadian BP variability such as non-dipper/riser pattern and exaggerated morning BP surge have been reported to be associated with organ damage and adverse cardiovascular events. The issue of whether a long-term increased rate of cardiovascular events and other chronic diseases such as hypertension occurs after a natural disaster is controversial. However, our findings suggest that a long-term risk persisting after a natural disaster might contribute to an increase in cardiovascular events when the environmental conditions and lifestyle are not improved. In the future, it will be important to observe how long abnormal BP persists after an aftershock. We were not able to examine this issue in depth in the present study due to the relatively small number of subjects.

The effect of an earthquake on cardiovascular reactivity can persist for at least a few years. Here, even long after the Great East Japan Earthquake, the major aftershock and tsunami warning readily triggered an augmented pressor response in disaster survivors. The environmental changes after an earthquake may augment disrupted circadian BP variability, which is associated with a risk of cardiovascular disease. An individualized approach to the improvement of living conditions is required for individuals who are obliged to live in temporary housing over the long-term.

SUPPLEMENTARY MATERIALS

Supplementary materials are available at American Journal of Hypertension (http://ajh.oxfordjournals.org).

Table 1. Patient characteristics

<table>
<thead>
<tr>
<th>Patient characteristics</th>
<th>Living in their own homes</th>
<th>Living in temporary housing</th>
<th>P value</th>
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<tbody>
<tr>
<td></td>
<td>n = 4</td>
<td>n = 4</td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td>78 (68–82)</td>
<td>73.5 (73–79)</td>
<td>0.69</td>
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<td>Male, n</td>
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<td>Complication, n</td>
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<td>Hyperlipidemia</td>
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<td>4</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
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<td>3</td>
<td></td>
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<tr>
<td>Chronic kidney disease</td>
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<td>2</td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
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<td>0</td>
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<td>Antihypertensive drug, n</td>
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</tr>
<tr>
<td>Calcium antagonist</td>
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<td>Angiotensin II receptor blocker</td>
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<td>3</td>
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<tr>
<td>Diuretics</td>
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<tr>
<td>Beta blocker</td>
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<td></td>
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<tr>
<td>Ambulatory blood pressure monitoring</td>
<td></td>
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<tr>
<td>24-Hour SBP, mm Hg</td>
<td>124.5 (119.4–125.8)</td>
<td>130.4 (120.2–136.0)</td>
<td>0.20</td>
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<tr>
<td>24-Hour DBP, mm Hg</td>
<td>72.7 (68.4–74.1)</td>
<td>71.9 (71.1–78.6)</td>
<td>0.89</td>
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<tr>
<td>24-Hour PR, bpm</td>
<td>63.4 (57.0–67.2)</td>
<td>72.2 (69.3–72.8)</td>
<td>0.021</td>
</tr>
<tr>
<td>SBP in the 1 hour before the aftershock, mm Hg</td>
<td>126.5 (121.5–138.0)</td>
<td>139.3 (113.0–143.5)</td>
<td>0.49</td>
</tr>
<tr>
<td>SBP in the 1 hour after the aftershock, mm Hg</td>
<td>137.8 (129.5–177.0)</td>
<td>137.5 (125.0–192.0)</td>
<td>1.00</td>
</tr>
<tr>
<td>PR after the aftershock, bpm</td>
<td>62.5 (53.9–64.7)</td>
<td>70.1 (68.8–70.3)</td>
<td>0.021</td>
</tr>
<tr>
<td>SBP at that night (22:00–2:00) after the aftershock, mm Hg</td>
<td>107.0 (101.9–110.1)</td>
<td>124.9 (113.2–137.9)</td>
<td>0.021</td>
</tr>
<tr>
<td>Morning SBP at the following morning (7:30–9:00) after the aftershock, mm Hg</td>
<td>129.5 (128.8–131.0)</td>
<td>149.3 (131.0–196.3)</td>
<td>0.029</td>
</tr>
</tbody>
</table>

Data are shown as the number or median (range). Abbreviations: DBP, diastolic blood pressure; PR, pulse rate; SBP, systolic blood pressure.
ACKNOWLEDGMENTS

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DISCLOSURE

The authors declared no conflict of interest.

REFERENCES