Earthquakes: another cause of heart failure?

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This editorial refers to ‘The Great East Japan Earthquake disaster and cardiovascular diseases’†, by T. Aoki et al., on page 2796

The role of acute stress in triggering cardiac events, especially in vulnerable individuals, is no longer disputed. The consequences of an acute and unexpected stressor have been examined during earthquakes, providing a population-based sampling methodology, the known exact timing of the stressor, and tabulation of acute cardiac events before and after the event.

The first study by Leor et al. was conducted in the wake of the Northridge earthquake which occurred at 04:31 h on 17 January 1994, one of the strongest earthquakes ever recorded. The authors reviewed the daily mortality and determined the underlying causes of death for the 7-day period before the earthquake, the day of the quake, and 6 days thereafter. Data were compared with the corresponding period in 1991, 1992, and 1993.

On the day of the earthquake, there was a sharp increase in sudden cardiac deaths immediately after the earthquake compared with the week before and after the earthquake, and with the corresponding control periods. Deaths typically occurred among people with advanced coronary atherosclerosis. Leor et al. conclude that this finding, along with the unusually low incidence of such deaths in the week after the earthquake, suggests that emotional stress may trigger cardiac emergencies in people who are predisposed to such events.

Suzuki and colleagues analysed the occurrence of acute myocardial infarction (AMI) during the Hanshin-Awaji earthquake in the Kobe region on 17 January 1995. The authors investigated the weekly frequency of MI and defined a 7-week period: 2 weeks before the earthquake, the week in which the earthquake took place, and 4 weeks after the quake. Data were compared with the corresponding period in 1992–1994. An increase in the number of patients admitted for AMI was reported in the week the earthquake took place (17–23 January 1995) and returned to normal 4 weeks after the quake.

Nakagawa and colleagues have studied the long-term effect of the Niigata-Chuetsu earthquake which took place on 23 October 2004 on AMI mortality. The authors examined death certificate data for all those who died in the area between 1 October 1999 and 30 September 2007. In the disaster area, the mortality rate of men and women increased significantly for 3 years after the earthquake compared with 5 years before the quake. The authors conclude that clinicians and policymakers should be aware of the need to provide long-term prevention of AMI among those living in earthquake areas.

Tsuchida et al. addressed the impact of the Noto Peninsula earthquake in Japan on 25 March 2007 on the occurrence of acute coronary syndrome (ACS) and stroke. The authors investigated patients who were admitted to hospital from 25 March to 29 April 2007. Data were compared with the same period in the previous 3 years. The first case of ACS occurred ~15 min after the earthquake, and the first case of stroke 72 h after the event. During the 35 days after the earthquake, among 49 patients who were attended by the local ambulance, five patients with ACS (10.2%) and eight with stroke (16.3%) were documented. The total number of cases was significantly increased compared with the control period.

In a population-based study in the greater Munich area, we could demonstrate that not only a natural catastrophe but also intense strain and emotional pressure watching the national team playing in an important football match may trigger acute cardiac emergencies.

The World Cup was held in Germany from 9 June to 9 July 2006, the prospectively assessed study period. The periods of 1 May to 8 June and 10 July to 31 July in 2005 and 2003, as well as 1 May to 8 June and 10 July to 31 July 2006, constituted the control periods.

Patients who had contacted emergency services, had been evaluated by an emergency medicine physician, and had been given the diagnosis ACS or symptomatic cardiac arrhythmias up to cardiac arrest were studied. We found a significant increase in the incidence of ACS and symptomatic cardiac arrhythmias within 2 h of matches in which the German team played compared with matches without German participation and compared with a control period, especially in men with known coronary heart disease. This major increase is exemplified during a very dramatic game which included a penalty shoot-out (Germany beat
Argentine), the number of events being increased fourfold, and the same increase during a match when Germany lost against Italy. Apparently, it is not the outcome of a game but rather the intense strain during the viewing of a dramatic match which is of prime importance for triggering a cardiac event.6

There has been considerable interest in identifying the consequences of daily life stress such as death of a family member, divorce, unemployment, social isolation, bullying at work, or burn-out on cardiovascular morbidity and mortality, but further studies are needed. In addition, disclosing the underlying pathophysiology and vulnerability to the potentially pathogenic effect of stress may improve risk prediction and prevention.

What can we learn from the new report on the Great East Japan Earthquake of 11 March 2011?7 The catastrophe consisted of (i) the second largest earthquake ever recorded in Japan, the epicentre being located ~150 km east of the Pacific coast under the Pacific ocean; (ii) a huge tsunami; (iii) several aftershocks; and (iv) a nuclear power accident. The studied area is the Miyagi prefecture (population 2.3 million).

While the effects of the nuclear power accident in the neighbouring prefecture Fukushima are judged to be minimal, >95% of the victims and damage in the Miyagi prefecture occurred in the coast area adjacent to the Pacific ocean due to the tsunami.

Based on the diagnoses of medical doctors in the emergency room of 57 hospitals, collected by 12 fire departments of the prefecture which had transferred emergency patients, the following results were obtained. As compared with the years 2008–2010, and 4 weeks before the earthquake: (i) the daily occurrence of cardiopulmonary arrest due to all causes and cardiopulmonary causes increased on days 1, 3, 5, and 6, heart failure on days 5–7, stroke on days 5–7, and pneumonia on days 3 and 5–7 of the first week; (ii) the weekly occurrence of cardiopulmonary arrest due to all causes and cardiopulmonary causes increased in the first and second week, ACS in the second week, stroke in the first 3 weeks, heart failure in the first 4 weeks, and pneumonia in the first 5 weeks; (iii) after the largest aftershock, a secondary peak was noted for 1 week in cardiopulmonary arrest of all causes and cardiopulmonary causes, as well as stroke—a minor increase was also noted for heart failure and pneumonia; and (iv) increased cardiovascular diseases were not associated with age, gender, and area of residence; however, more cases of pneumonia were seen in the sea coast area vs. the inland area, most probably due to aspiration, cooling, drowning, etc., due to the tsunami.

The authors claim to be the first to have demonstrated a marked and prolonged increase in heart failure following an earthquake. To perform a population-based study in the midst of such a disaster poses conspicuous obstacles, and the authors should be commended for their work.

However, several shortcomings should be mentioned. (i) The diagnoses of heart failure and pneumonia given by doctors in the emergency room were not defined; as to heart failure, symptoms were not reported, and it is not known which findings documented the presence of structural heart disease. (ii) Regarding cardiopulmonary arrest, there are no data to differentiate between cardiac and pulmonary causes. This is unfortunate in view of the fact that the tsunami caused an increase of pneumonia and was responsible for most of the deaths, and many cases of cardiopulmonary arrest might have been due to aspiration, cooling, drowning, etc. (iii) No information is available about disease history and drug treatment of the affected patients.

Thus, we are left with considerable uncertainty regarding the diagnoses of heart failure and pneumonia, and the causes of cardiopulmonary arrest. Hopefully, the authors can reveal this uncertainty in a follow-up examination based on evaluation of these patients in hospital.

Finally, what could be the mechanism of the prolonged increase in cardiovascular disease, especially of heart failure by 4 weeks, diagnosed when patients were attended by emergency ambulances? Explanations given by the authors are purely speculative.

Stress-induced rupture of atherosclerotic plaques leading to myocardial infarction and sudden cardiac death closely related in time with the earthquake2,3 is not documented here: ACS is unaffected within the first week, and an increase of cardiac causes exclusively of cardiopulmonary arrest are not demonstrated.7

Therefore, either the stressor had persisted for weeks, for example because of frequent aftershocks, or the increase of cardiovascular disease, and especially heart failure, is caused—more likely in our opinion—in a complex way by the consequences of the humanitarian catastrophe: the trauma, breakdown of lifelines (water, food, electricity, traffic, the emergency system), evacuation, and freezing temperature, plus aspiration pneumonia and drowning due to the tsunami.

Whatever the mechanism, however, the study by Aoki et al.7 clearly points out that a catastrophe such as this is a challenge to any healthcare system forced to deal with increased cardiovascular disease not only for days, but for weeks and months.

Conflict of interest: none declared.

References