Journal of Clinical & Experimental Cardiology

Jackson, J Clin Exp Cardiolog 2016, 7:3
DOI: 10.4172/2155-9880.1000426

Case Report Open Access

Bushfires, not to be Taken Light Heartedly

Damon Jackson[1], **Doris Bartl**[1], **David Tong**[2], **Jamie Layland**[3*]

[1]*Peninsula Health, Hastings Road, Frankston, Victoria, Australia*

[2]*Peninsula Health Hastings Road, Frankston, Victoria and University of Melbourne, Australia*

[3]*Peninsula Health Hastings Road, Frankston, Victoria, and Monash University, Australia*

[*]**Corresponding author:** Jamie Layland, Peninsula Health, Hastings Road, Frankston, Victoria and Monash University, Australia, Tel: +61 3 9411 7111; E-mail: jamielayland@hotmail.com

Received date: February 16, 2016; **Accepted date:** March 29, 2016; **Published date:** March 31, 2016

Case Report

We present the case of a 71 year-old woman admitted to hospital with Takotsubo Cardiomyopathy (TTC) in the context of a nearby bushfire. Cardiovascular complications may be an under-recognized contributor to bushfire related morbidity and mortality. We review the current literature pertaining to TTC and discuss the effects of Bushfires and other natural disasters on the cardiovascular system.

Keywords: Apical ballooning syndrome; Bushfire; Cardiomyopathy; Natural disaster

Clinical Case

Ambulance paramedics were called to a 71 year-old woman complaining of severe central chest pressure soon after witnessing a fire burning at the periphery of her property. On arrival she appeared anxious and stated that although she had been concerned by the local fires, it wasn't until she witnessed first-hand a nearby bushfire that she started experiencing the pain. The Early Warning Network issued an Emergency Warning: "There is a fast moving, out of control bushfire travelling in a southerly direction [1]. "Leaving now is the safest option, before conditions become too dangerous. Emergency Services may not be able to help if you decide to stay" [1].

At presentation to emergency a 12-lead ECG showed global T-wave inversion (Figure 1), troponin I was 0.85 μg/L (< 0.03 μg/L) and Creatine kinase 126 units/L (0-170), her standard pathology and biochemistry were otherwise unremarkable. Chest radiograph showed borderline heart size with no evidence of pulmonary edema. Her past medical history was significant for hypertension.

She was brought forward for coronary angiography demonstrating essentially normal coronary arteries. A left ventriculogram demonstrated regional wall motion abnormalities consistent with the classical apical variant of TTC (Figure 2). TTE the following day reported moderate LV systolic dysfunction with apical hypokinesis in keeping with the diagnosis of TTC.

The patient was commenced on medical therapy (beta-blocker, angiotensin-converting enzyme inhibitor (ACEI)) and discharged the following day. Repeat TTE several months later demonstrated normalization of her LV function.

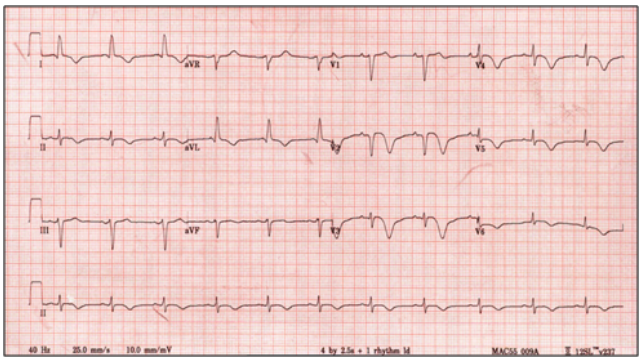

Figure 1: At time of presentation with chest pressure the ECG showing global T-wave inversion.

Discussion

The impact of bushfires on the cardiovascular system is an under-recognized entity. In Australia there have been over 700 bushfire fatalities since the first recorded death in 1850 [2]. Between 1967 and 1999 the estimated average annual cost of major bushfires was $77 million with bushfires being the fourth most frequent disaster type [2]. Little attention is given to potential life threatening complications of bushfire including TTC. Recently published data by Haikerwal et al. [3] suggest that exposure to smoke from bushfires in the southeast region of Australia in December 2006 – January 2007 was associated with an increase in out of hospital cardiac arrests in men and ischaemic heart disease events in women. Fine particulate matter produced by bushfires has also been linked to cardiovascular admissions [4]. Our case highlights another potential health related consequence of bushfire not previously documented. Given that the effects of climate change may lead to an increase in the likelihood of destructive bushfires [2], we may see an increase in the number of bushfire related cardiovascular presentations in the future, including TTC.

TTC was first described in a case series of five Japanese patients in 1991 [5]. Although multiple case reports have since been published, our case represents a novel precipitant stressor in the form of a bushfire. Considered mechanisms for TTC include multi-vessel coronary spasm [6] and microvascular dysfunction [7], however, catecholamine excess seems to be the most likely significant contributor. Abraham et al. [8] described 9 cases of TTC precipitated

by epinephrine or dobutamine infusion. In their series, all morphological variants of TTC (apical, mid and basal) were demonstrated, suggesting a link with catecholamine excess. Wittstein et al. [9] demonstrated that catecholamine levels were approximately two to three times higher in TTC than in cases of acute myocardial infarction resulting in clinically significant LV dysfunction. They also found pathological findings consistent with catecholamine-induced myocardial injury in the TTC cases. Despite the fact that a multitude of emotional and physical catecholamine mediated triggers for TTC have been reported, a stressor is not identified in up to one-third of patients [10].

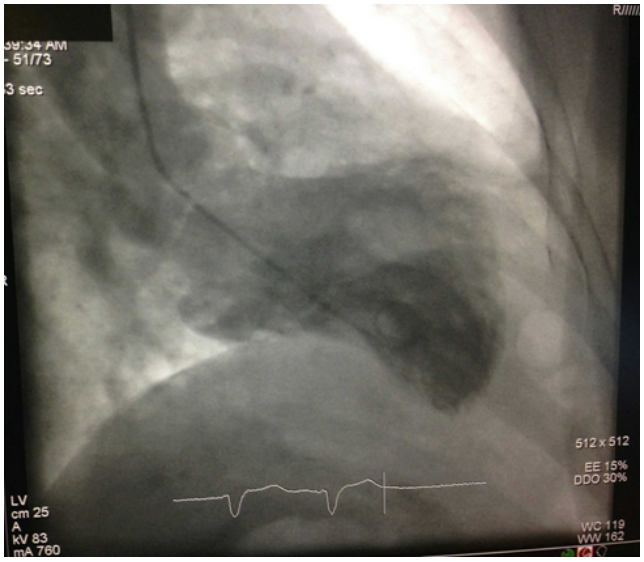

Figure 2: Left ventriculogram at end-systole.

Natural disasters, potential adrenergic stressors, have been associated with adverse cardiac outcomes including TTC. For example, recent earthquakes led to an increase in TTC presentations [11]. The incidence of TTC one month after an earthquake in Japan in 2004 was approximately 24-fold higher near the epicenter than that before the earthquake [12]. There have also been two documented cases of TTC reported in the context of severe floods [13].

As in our case, the most common presenting symptom relating to TTC is chest pain, seen in 70-90% of cases and occurs most frequently in post-menopausal women [14]. In the first large Japanese series describing TTC 76 patients were women, 12 were men and the median age was 67 +/- 13 years [15]. The diagnosis of TTC is one of exclusion. Current suggested criteria require the development of new ECG abnormalities or modest elevation in cardiac troponin with associated transient LV wall motion abnormalities in the absence of obstructive coronary disease or plaque rupture [16].

The patient in our report presented with global T wave inversion on ECG, which is within the wide spectrum of ECG changes previously demonstrated in TTC. ST segment elevation is present in approximately one third of patients, with the anterior leads most commonly involved [14]. Common changes observed with serial ECGs include prolongation of the QT interval and deep symmetrical T wave inversion [14]. Troponin is almost always elevated at presentation but the levels are markedly lower than would be anticipated by the extent of wall motion abnormality and ECG changes demonstrated [14].

There is no randomized data regarding the management of TTC. The initial management strategy should be directed towards the treatment of ischemia until emergent coronary angiography excludes obstructive coronary disease or atherosclerotic plaque rupture [17]. Following exclusion, LV wall motion is assessed by left ventriculography. Initial beta-blocker therapy is considered reasonable given excess catecholamines may be involved in the pathophysiology of the condition. They may also reduce complications such as cardiogenic shock because of LV outflow obstruction, ventricular arrhythmias or ventricular rupture [18]. An ACEI should also be commenced, at least until LV function recovers [18]. Therapeutic anticoagulation may be considered until LV recovery as systemic thromboembolism has been reported in TTC [19].

Although the natural history of TTC suggests that LV function will recover, in hospital mortality among patients with TTC is 4.5%, of which 38% are directly related to TTC complications. Male gender and advanced age are associated with an increased mortality rate [20]. TTC may recur in 5-10% of patients and appear as early as three weeks or as late as four years after the initial event [10].

Bushfire related cardiovascular complications including TTC are a major public health issue with a definite mortality association. We have demonstrated a clear association between bushfire and TTC. Improved public awareness of bushfire associated cardiac issues is important and may decrease bushfire related morbidity and mortality.

Acknowledgements

No financial assistance was provided for the preparation of this case report.

References

1. (2015) Country Fire Authority. VIC Bushfire Emergency Warning: Hastings, Bittern, Crib Point. Kingscliff, NSW: Early Warning Network.
2. Middleman MH (2007) Natural hazards in Australia: identifying risk analysis requirements. Canberra: Geoscience Australia.
3. Haikerwal A, Akram M, Del Monaco A, Smith K, Sim MR, et al. (2015) Impact of fine particulate matter (PM2. 5) Exposure during wildfires on cardiovascular health outcomes. Journal of the American Heart Association 4:e001653.
4. Crabbe H (2012) Risk of respiratory and cardiovascular hospitalisation with exposure to bushfire particulates: new evidence from Darwin, Australia. Environmental geochemistry and health 34: 697-709.
5. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M (1991) [Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases]. J Cardiol 21: 203-214.
6. Sansen V, Holvoet G (2007) Takotsubo cardiomyopathy presenting as multivessel coronary spasm syndrome: case report and review of the literature. Acta Cardiol 62: 507-511.
7. Elesber A, Lerman A, Bybee KA, Murphy JG, Barsness G, et al. (2006) Myocardial perfusion in apical ballooning syndrome: correlate of myocardial injury. American Heart Journal 152:469. 9-13.
8. Abraham J, Mudd JO, Kapur N, Klein K, Champion HC, et al. (2009) Stress cardiomyopathy after intravenous administration of catecholamines and beta-receptor agonists. Journal of the American College of Cardiology 53: 1320-1325.
9. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, et al. (2005) Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 352: 539-548.

10. Elesber AA, Prasad A, Lennon RJ, Wright RS, Lerman A, et al. (2007) Four-year recurrence rate and prognosis of the apical ballooning syndrome. J Am Coll Cardiol 50: 448-452.
11. Chan C, Elliott J, Troughton R, Frampton C, Smyth D, et al. (2013) Acute myocardial infarction and stress cardiomyopathy following the Christchurch earthquakes. PLoS One 8: e68504.
12. Sato M, Fujita S, Saito A, Ikeda Y, Kitazawa H, et al. (2006) Increased incidence of transient left ventricular apical ballooning (so-called 'Takotsubo' cardiomyopathy) after the mid-Niigata Prefecture earthquake. Circ J 70: 947-953.
13. Butterly SJ, Indrajith M, Garrahy P, Ng AC, Gould PA, et al. (2013) Stress-induced takotsubo cardiomyopathy in survivors of the 2011 Queensland floods. Med J Aust 198: 109-110.
14. Hurst RT, Prasad A, Askew JW, Sengupta PP, Tajik AJ (2010) Takotsubo cardiomyopathy: a unique cardiomyopathy with variable ventricular morphology. JACC: Cardiovascular Imaging 3:641-649.
15. Tsuchihashi K, Ueshima K, Uchida T, Oh-mura N, Kimura K, et al. (2001) Angina pectoris-myocardial infarction investigations in Japan. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Journal of American College of Cardiology 38: 11-8.
16. Scantlebury DC, Prasad A (2014) Diagnosis of Takotsubo cardiomyopathy. Circ J 78: 2129-2139.
17. Prasad A, Lerman A, Rihal CS (2008) Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. Am Heart J 155: 408-417.
18. Kurisu S, Kihara Y (2012) Tako-tsubo cardiomyopathy: clinical presentation and underlying mechanism. J Cardiol 60: 429-437.
19. Andò G, Saporito F, Trio O, Cerrito M, Oreto G, et al. (2009) Systemic embolism in takotsubo syndrome. Int J Cardiol 134: 42-43.
20. Singh K, Carson K, Shah R, Sawhney G, Singh B, et al. (2014) Meta-analysis of clinical correlates of acute mortality in takotsubo cardiomyopathy. Am J Cardiol 113: 1420-1428.