

A "broken heart" on a cruise ship

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ABSTRACT

An elderly female cruise ship passenger developed chest pain shortly after an emotionally charged incident involving shipboard authorities. The electrocardiogram and cardiac enzyme profile were indicative of myocardial infarction but the final diagnosis, established after aeromedical evacuation, was stress-related cardiomyopathy. This case is an example of a relatively unknown clinical entity that is easily mistaken for acute myocardial infarction and which may be disproportionately prevalent in the cruise ship passenger population.

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Key words: acute chest pain, ischaemic heart disease, cardiac enzymes, electrocardiogram, takotsubo cardiomyopathy, maritime medicine

INTRODUCTION

Acute chest pain is a medical challenge, especially at sea. What is the cause, can it be treated aboard, and if not, what can be done until safe evacuation to definitive care is possible? A cruise ship patient with chest pain and electrochemical findings suggestive of myocardial infarction (MI) following emotional stress is presented.

CASE REPORT

Around midnight on the first evening of a 5-day sea crossing a 72-year-old lady sought emergency consultation for acute chest pain. She was experiencing an ache in the left side of the anterior chest, radiating to between the shoulder blades and into the left side of the neck. The pain was of gradual onset, having begun whilst reading the menu at the dining table several hours earlier. Throughout the meal and the subsequent after-dinner entertainment the pain slowly increased to the point where at bedtime she decided to seek medical attention.

The lady was a non-smoker with no discernible risk factors for MI. Her only regular medications were prednisolone, taken for fibromyalgia, and thyroxine.

She had been experiencing no prior chest symptoms nor had there been any physical trauma. However, a few hours prior to the onset of her pain she had experienced considerable emotional trauma as a result of playing a central role in an investigation into missing necklaces from the ship's jewellery shop.

Initial assessment showed a slim patient, looking remarkably well and seemingly in minimal discomfort. Clinical examination of heart, lungs and peripheral circulation were normal; blood pressures in both arms were equal (139/73 mm Hg) and the only abnormal finding was a sinus tachycardia of 115 bpm.

However, the electrocardiogram (ECG) was far from normal, with deep Q waves in leads V_2 and V_3 (Fig. 1), associated with borderline ST elevation.

Initial cardiac enzymes were also abnormal: troponin I and creatinine kinase myocardial band isoenzyme (CK-MB) were elevated; myoglobin was within normal limits (Table 1). These investigations were thought to represent a recent MI in the time frame of 12–24 h earlier, although the patient denied having any prior symptoms.

A working diagnosis of non-ST elevation MI (NSTEMI) was established and treatment commenced accordingly. The patient's pain quickly resolved with sub-lingual nitrate spray and a small dose of intravenous opiate. Anti-platelet medication and low molecular weight heparin were initiated after a chest radiograph had demonstrated no mediastinal widening.

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Figure 1. Comparison of the patient's initial (**A**) electrocardiogram, taken at time of presentation, and her final (**B**) electrocardiogram, taken 32 h later. The development of T wave inversion in leads I, II, $V_2 - V_6$ in the final trace is seen, along with increased T inversion in aVL and flattening of T waves in III and aVF. Q waves are seen in V_2 and V_3 of the initial (**A**) trace with the development of R waves in V_2 and V_3 in the final (**B**) trace

Table 1. Biochemical analyses performed on board ship

Time from pain onset [h]	5	8	12	18	36
Time from presentation [h]	1	4	8	14	32
Myoglobin (NR: 0-107 ng/mL)	49	40	31	38	60
CK-MB (NR: 0-4.3 ng/mL)	7.0	6.4	4.6	4.2	2.7
Troponin I (NR: 0-0.4 ng/mL)	0.95	0.65	0.32	0.26	0.16
B-type natriuretic peptide (NR: 0-100 pg/mL)	66	-	466	807	461

NR - normal range; CK-MB - creatinine kinase myocardial band isoenzyme; Onboard analysis using Biosite Triage meter with Triage Profiler SOB cartridges.

The ship was several days from the next port of call so the patient remained in the ship's medical facility for monitoring. During the course of her stay she was asymptomatic, although, on day two, left ventricular failure became clinically apparent and was associated with elevation of B-type natriuretic peptide to 807 pg/mL (Table 1). This responded well to a nitrate infusion and oral diuretics.

Thirty-two hours after admission the patient's ECG had evolved quite markedly, with T wave inversion now present throughout the precordial leads. Leads V_2 and V_3 , which had initially shown deep Q waves, developed R waves (Fig. 1).

The serial cardiac enzyme profile was atypical for MI. Troponin I and CK-MB were elevated at presentation but had normalised by 8 h and 14 h, respectively. Myoglobin remained within normal limits throughout (Table 1).

During her admission arrangements were made for a ship diversion due to a concurrent case of appendicitis and the opportunity was taken to arrange aeromedical transfer of this patient from the island's airstrip at the same time.

Ultimately she was successfully transferred to hospital on the mainland, where angiography showed patent coronary arteries. A few days later she was released with a diagnosis of transient, stress-related cardiomyopathy.

DISCUSSION

The Love Boat was a very popular United States sitcom series set on a cruise ship. It ran from 1977 until 1987 [1] and contributed greatly to the increasing success of the cruise industry. A combination of comedy, romance and high drama, the series shaped to a certain degree the public's concept of cruising. The ship's doctor, present in all 250 episodes [2], played a prominent part, often having to try to mend emotionally and medically 'broken hearts'. The present case of a 72-year-old lady with chest pain after a stressful confrontation on board reads like a typical Love Boat storyline.

In 1990, 3 years after The Love Boat series had been taken off air, cardiologists in Japan first published reports of patients with a reversible cardiomyopathy involving distension of the muscle at the ventricular apex [3]. It was subsequently shown that, although presenting with symptoms suggestive of cardiac ischaemia and having supportive electrocardiographic changes, these patients had patent coronary vessels on angiography [4].

Ventriculogram showed ballooning of the cardiac apex during systole, associated with tight contraction of the ventricular base. These appearances were thought reminiscent of a narrow-necked bulbous fishing pot used in local waters to catch octopus. The pot is called a takotsubo and the condition became known as takotsubo cardiomyopathy (TCM).

TCM patients typically present with acute chest pain resembling MI and associated with electrocardiographic changes of ST elevation, usually in the precordial leads. Over the first few days, transient anteroseptal Q waves are seen in up to one third of patients, along with T wave inversion and prolongation of the QT interval [5, 6]; all of these changes were seen in the case reported here.

No electrocardiographic criteria have been identified that can reliably differentiate TCM from STEMI [5] and, as there is often an associated elevation of CK-MB and troponins, the mis-diagnosis and treatment of these patients as STEMI rather than TCM is a distinct possibility.

It is thought that TCM is responsible for 1-2% of all cases of suspected MI [7] and up to 6% of all females presenting with acute coronary syndrome may in fact be cases of TCM [5].

The precise pathogenesis remains ill understood. High concentrations of circulating catacholamines may play a primary role as levels in TCM have been shown to be statistically higher than those found during MI [8].

The majority of TCM cases arise in post-menopausal females, typically between the ages of 60 and 75, and most patients have recently experienced a profound, emotionally disturbing event prior to symptom onset, such as: emotional

confrontations and arguments, the unexpected death of a close relative or spouse, receipt of a catastrophic medical diagnosis, devastating financial and gambling losses and fear of medical procedures [6].

The mortality rate of TCM is 1-3%, with death arising from dysrhythmia, heart failure or systemic embolisation of mural thrombus [5]. Most patients, however, will survive the acute phase, requiring only supportive treatment before making a full recovery in the first few weeks [5, 8].

Following the first reports of TCM in the early 1990s it was around 2005 that reports started to appear in Western medical literature showing that this condition was not limited to Japanese ladies but was occurring in elderly Caucasian females also; in fact, exactly the sort of ladies who traditionally populate cruise ships. It's a matter of conjecture just how many elderly ladies over the years have made miraculous full recoveries from their apparent MI on a cruise ship, having in fact been unrecognised cases of TCM all along.

The term TCM, although evocative for Japanese cardiologists, turned out to be less user-friendly for the English-speaking medical community and several alternative names for this condition have been adopted: 'apical ballooning syndrome', 'stress induced cardiomyopathy' and, because the unexpected death of a loved one is a frequent precipitant [8], 'broken heart syndrome'.

CONCLUSIONS

Consequently, the patient in this case report can be considered to represent an example of broken heart syndrome on a Love Boat!

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