Report on takotsubo cardiomyopathy

Abstract

This is a report on Takotsubo cardiomyopathy. It includes details of the left ventricle, proposed mechanisms of the disease, reasons as to why certain regions are affected and some aren’t, the exact problem in detail, symptoms and clinical features including tests and ECG readings, treatment options used and prognosis, incidence and recurrence, some case reports to show the condition in its clinical setting and a conclusion summing up the important points and what direction needs to be taken with regards to the disease.

Introduction

What do I mean by a broken heart?

A broken heart can mean many things, both physiologically and philosophically. Physiologically a broken heart can be a myocardial infarction, arrhythmia, angina, the list is endless. However, philosophically speaking a broken heart is generally perceived as the loss of a loved one or a relationship breakup but in this context can also mean other things which would also cause severe emotional stress such as legal issues, sudden poor financial situation, intense arguing, finding out you have a chronic terminal illness or a loved one does, surgery and car accidents and even the stoppage of use of addictive drugs.

We know profound stress can cause certain cardiovascular issues such as high blood pressure, high cholesterol and coronary heart disease. A relatively recent cardiomyopathy has been discovered, and is called broken heart

syndrome. This is because it is highly associated with having a profound amount of emotional stress.

Heres some anatomy of the left ventricle and myocardium muscle.

The left ventricle will be concentrated on as that is the general area this cardiomyopathy occurs. The left ventricular free wall is thickest at the base and thinnest at the apex (usually around 1-2mm). The left ventricular free wall in general is 3 times thicker than the right ventricular free wall.

The role of noradrenaline

Noradrenaline is a hormone and a neurotransmitter produced in the human body. It is released upon excitement, threat etc and directly increases the heart rate. It is released in the fight or flight response.

So what is broken heart syndrome?

Broken heart syndrome was first recorded by Dote et al in 1991. They discovered that this form of cardiomyopathy involved the left ventricle and particularly the apex. They found that instead of contracting normally to pump blood around the body, the ventricle ballooned and expanded. This is due to akinesia of the distal anterior wall and apical wall and hypercontraction of the basal wall. Obviously, this would have a severe effect on the blood flow to vital organs so could be quite problematic. It was named Takotsubo cardiomyopathy because the appearance of the left ventricle in an angiogram resembles an octopus pot, which translates as Takotsubo in Japanese. It is also referred to as apical ballooning syndrome, due to the ballooning of the apex.

A shows what happens in Takotsubo cardiomyopathy in contraction, as can be seen the volume of the ventricle increases. B shows what a normal contraction should look like.

What are the physiological mechanisms behind Takotsubo cardiomyopathy?

There have been a few proposed theories, but no one is quite sure.

Multivessel coronary vasospasm

One theory is that many of the coronary arteries spasm and constrict at the same time so blood flow is greatly reduced to the myocardium muscle and other areas of the heart. However in some studies a vasospasm has been induce by acetylcholine and it has not been conclusive. Also the duration of the abnormal wall motion is longer than it should be.

Release of catecholamines

In almost all cases of Takotsubo cardiomyopathy, increased levels of catecholamines (adrenaline, noradrenaline, dopamine) have been found in the body. There are many things which can induce a release of catecholamines and emotional excitement or stress are some of them. The catecholamines would be released to the heart to make it beat harder and faster. This is the most widely accepted mechanism for Takotsubo but as mentioned earlier, there is no clear answer.

Microvascular spasm

It has been found that there is impaired microcirculation in this syndrome, but again there are many challenges to this theory. The “ slow-flow” phenomenon is not observed. Impaired microcirculation may be the result of the primary myocardial injury not necessarily the cause of it.

So why is the apical wall of the left ventricle and the mid-ventricle the place that is most affected by a surge of these catecholamines?

Well, there are a few anatomical and physiological causes for this.

There is a markedly higher concentration of adrenergic receptors in the apex of the left ventricle than other parts of the heart and it is these receptors that noradrenaline and adrenaline will bind to. Also due to increased responsiveness of the apical myocardium to stimulation of these receptors.

The apical wall is especially vulnerable structurally as when the rest of the heart has a three layered myocardial composition, the apex does not and is therefore a lot thinner.

The area of the apex tends to lose its elasticity a lot more easily after many extreme expansions and does not correctly go back to its original state.

The apex is a lot more likely to become ischemic as the blood flow is not large in that area so at any time when blood flow is reduced the apex loses out quickly.

The Electro Cardio Gram of a person with Takotsubo cardiomyopathy usually shows ST elevation in the acute stage, T wave inversions and a prolonged QT interval in the sub acute stage and the inverted T wave can persist for weeks in the recovery stage.

Clinical presentation

The clinical presentation and diagnostic tools are very similar to that of acute coronary syndrome and acute myocardial ischemia. A diagnosis of Takotsubo

cardiomyopathy should not be made until ACS, coronary artery disease, acute myocardial infarction, myocarditis, pericarditis etc have been ruled out. Along with the ECG there are other symptoms which might lead to a diagnosis. Among these are chest pain and shortness of breath. Temporary loss of consciousness and shock have been reported. A slight elevation of cardiac damage bio markers troponin and creatine kinase can be detected. Surprisingly, it generally occurs in patients without significant blockage of coronary arteries or any acute plaque rupture.

Treatments

Long term therapy should include:

Beta blockers, which work by blocking the transmission of certain nervous impulses to the heart and reduces the heart rate and force of the beat.

Angiotensin converting enzyme (ACE) inhibitors which stop the conversion of angiotensin 1 to angiotensin 2, which reduces arteriole resistance and increases the venous capacity.

Diuretics get rid if the water built up in lungs from heart failure. Aspirin is used to reduce the risk of another heart attack and prevent more cardiac muscle death. Calcium channel blockers are generally used to lower blood pressure. The treatment for this disease is generally to try and take the stress away that caused it in the first place and is of a supportive nature. Most people suffering from broken heart syndrome recover within about 2 weeks to 2 months.

Incidence

Various different studies have shown that Takotsubo cardiomyopathy is a generally a female condition as between 70% and 100% of all cases are in women, most of whom are post menopausal. The reason for this is unclear, however some explanations have been proposed. Sex hormones may definitely influence the sympathetic neurohormonal axis and coronary vasoreactivity. Females may also be more susceptible to myocardial stunning that is mediated by the sympathetic nervous system. Endothelial function is altered due to changes in oestrogen levels and this could be another reason for the much more common occurrence in post menopausal women.

A Danish study suggested that 234 out of 100, 000 people get acute coronary syndrome and an American study reported that 1. 7% to 2. 2% of patients thought to have acute coronary syndrome actually have Takotsubo cardiomyopathy. So as can be seen it is a very rare illness.

Around 99% of patients in hospital with Takotsubo survive the disease and fully recover.

Some reports suggest that up to 10% of patients who recover will get it again. This makes it difficult from a treatment point of view as to how long to treat for.

Some case reports

Case 1

67 year old woman presenting with chest pain that has lasted a day has ST elevations on ECG. Chest pain is substernal and is linked with dyspnoea. Pain

radiates to left arm. When resting pain and dyspnoea goes after an hour but comes back throughout the day. ECG is concerning. She is sent to the emergency department from a clinic. Her blood pressure is 140/86 mmHg, pulse is 86 beats per minute, breathing is 14 breaths per minute, O2 saturation is 100%, she is cannulised and on 2 litres per minute nasally. Her temperature is 37. 1°C. She smokes 20 packs of cigarettes a year.

She is asked about a history of heart problems in her family and she begins to cry and describes her sister's death from a heart attack just 2 days before.

ECG shows small Q waves, T wave inversions, ST elevation and poor R wave progression.

All other tests are normal, a left ventriculogram reveals akinesia of parts of the apex.

7 days later she has normal left ventricular function and apex wall motion is normal, though the base appears to be contracting harder than apex.

Case 2

86 year old woman is in emergency department presenting with chest pain that has lasted 10 hours. It is substernal and does not radiate. Her blood pressure is 185/88 mmHg, pulse is 71 beats per minute, breathing rate is 20 breaths per minute O2 saturation is 98% and she is breathing normal air. Her temperature is 35. 7°C. Medical history is hypertension, gout a hysterectomy and hypothyroidism. She has a family history of coronary disease.

ECG shows ST elevation and T wave inversion. All other tests are normal.

The left ventriculogram reveals akinesia of the apex and mid anterior wall. The base has normal function. Mid septum shows hypertrophy and apex shows hypotrophy. Left ventricular function is at just 34% ejection fraction.

It is found out that her son was killed accidentally just 2 weeks prior to onset of symptoms.

Conclusion

Yes, by all means it is possible to die of a “ broken heart”. Both cases showed that a profound amount of severe emotional stress led to the Takotsubo “ broken heart” syndrome Although it is a very rare disease and almost all patients survive and recover fully, if the condition goes unnoticed or untreated it can prove fatal. As can be seen from the cases, the symptoms are very general with tests not showing many abnormalities. It seems to have no predisposition in people with heart problems. Its symptoms and clinical features are very similar to that of acute coronary syndrome. The most defining tests seem to be the ECG which shows similar results each time, and a left ventriculography, which can show up exactly which parts of the ventricle wall have akinesia.

The relatively recent discovery of the disease means that although it is fully recognised by medical institutions it is not fully integrated into clinical practise. Although incidence is rare, this does need to change.

More research must be done in the field to fully understand the mechanism behind the illness, as all the proposed mechanisms are unproved and conflicting. By finding the correct mechanism the treatment options can also be broadened to suit more specifically the cause and not just management.

References

- Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review
- Gianni M, Dentali F, Grandi AM, Sumner G, Hiralal R, Lonn E. European Heart Journal 2006.
- Takotsubo Cardiomyopathy, or Broken-Heart Syndrome
- Virani SS, Khan AN, Mendoza CE, Ferreira AC, de Marchena E.
- Texas Heart Institute Journal 2007 v. 34
- Recognition of the Apical Ballooning Syndrome in the United States G. William Dec, MD Circulation 2005 American Heart Association
- Stress, emotion and the heart: tako-tsubo cardiomyopathy
- Iqbal MB, Moon JC, Guttmann OP, Shanahan P, Goadsby PJ, Holdright DR
- Postgrad Med J 2006; 82: e29
- www. takotsubo. com
- www. wrongdiagnosis. com
- Hurst’s The Heart
- A case of Takotsubo cardiomyopathy mimicking ACS Metzl et al 2006 Nat Clin Pract Cardiovasc Med
- Apical and Midventricular Transient Left
- Ventricular Dysfunction Syndrome (Tako-Tsubo Cardiomyopathy): Frequency, Mechanisms and Prognosis Kurowski et al 2007 CHEST
- Systematic Review: Transient Left Ventricular Apical Ballooning:
- A Syndrome That Mimics ST-Segment Elevation Myocardial Infarction
- Bybee et al December 2004 Annals
- Takotsubo cardiomyopathy a case series and review of the literature

- West j emergency medicine
- Acute coronary syndrome: incidence and prognosis, Nielsen KM,

Danish Medical Bulletin – No. 1. February 2006. Vol. 53 Page 95