



TAKOTSUBO CARDIOMYOPATHY IN A YOUNG MALE PATIENT WITH ALCOHOL INDUCED ACUTE GASTRITIS

Cardiology

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ABSTRACT

A 49 year male presented with severe chest pain and vomiting followed by alcohol intake. Electrocardiogram showed ST elevation and T- wave inversion with echocardiogram showed low ejection fraction with wall motion abnormality typically apical wall akinetic and compensatory basal wall hyper contractibility. Coronary angiogram was normal. Upper GI endoscopy was showing hiatus hernia with erosive esophagitis and duodenitis. He recovered with proton pump inhibitor. This is a rare case of takotsubo cardiomyopathy (TCM) due to alcohol induced acute gastritis.

KEYWORDS

acute gastritis, takotsubo cardiomyopathy

CASE REPORT

Mr. SK, a 49 year old male came to Emergency room with complaint of acute chest pain, burning epigastrium associated with vomiting since 24 hours. All events occurred followed by intake of 200 ml of alcohol. Patient was a chronic alcoholic.

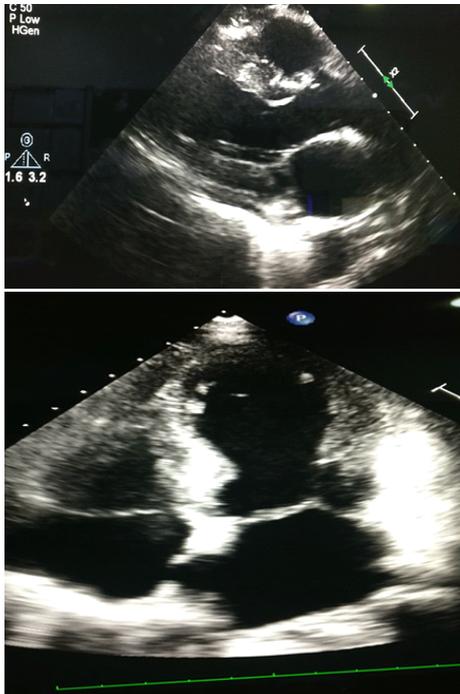


Figure 1. Echocardiography



Figure 2. Coronary Angiogram – normal coronary artery



Figure 3. Endoscopy showing duodenal



ECG was showed ST elevation in anterior leads and fresh ECG revealed deep T wave inversion in V1-V4. He was afebrile with blood pressure 150/90 mm of hg and heart rate of 110/min. Blood test results showed elevated total leucocyte count 14100/l, Troponin I was 15.8 ng/ml , NT pro BNP was 5949 pg/ml, CKMB Mass 97.09 ng/ml with normal serum Amylase(30.7 U/L) and lipase level (27 U/L) and very high catecholamine level. A transthoracic echocardiogram showed an ejection fraction of 25% with apical akinesis with compensatory basal hyperkinesis, resulting in the characteristic systolic apical ballooning (figure 2). Coronary angiogram showed normal coronaries (figure 1). Myocardial SPECT showed normal perfusion in anterior, septal,

lateral and inferior wall with subnormal resting LV systolic function with hypokinesia of the apical and anteroapical walls. Upper GI endoscopy was performed and showed Hiatus hernia with erosive esophagitis and erosive duodenitis (figure 3). Gastritis induced takotsubo cardiomyopathy was diagnosed. Patient was managed for gastritis and carvedilol, Ramipril and Torsemide with Spironolactone was given at discharge. Six weeks later, the patient was completely asymptomatic and echocardiogram revealed no left ventricular dilatation, resolved wall motion abnormality with improved left ventricular function.

DISCUSSION

Takotsubo cardiomyopathy also known as stress induced cardiomyopathy or apical ballooning syndrome is very difficult to distinguish from acute coronary syndrome in the early clinical stages. Characteristic clinical features includes acute chest/epigastric pain in majority and dyspnea⁴ in few patients. Dynamic ECG changes usually ST elevation and eventually T wave inversion is characteristic ECG finding.⁴ Apical ballooning with compensatory hyperkinetic basal area on echocardiography and absence of obstructive coronary artery disease on coronary angiogram is typical finding in TCM. More than 2/3¹⁴ of takotsubo cardiomyopathy(TCM) occur in older postmenopausal females, whereas the incidence of TCM in males has been described to be even rare.¹²⁰ Several studies reported the mean age of TCM has ranged from 62 to 76 years.¹⁶ Overall 90% (range 65%-100%) of the patients with TTC are women¹⁶. The number of men affected with takotsubo syndrome is higher in prospective studies from Asia, ranging from 13% to 35%.^{17,18,19} The mean age is similar in male and female patients with TTC. In this case the patient is a mid- aged male which is not very common.

Cardiac biomarkers are found to be modestly elevated in TCM. Pawlak et al. found that the median troponin I values in TCM patients are 2.99 ±5.36 ng/mL compared to a median value of 42.70± 64.79 ng/mL in the STEMI patients.¹⁵ Mild elevation in levels of troponin is seen in most patients with TCM, with absence of the typical increasing trend in serial measurements as seen with acute coronary syndrome (ACS)⁵. In our case a mid-aged male patient presented with chest pain and typical ST elevation with T wave inversion. Peak troponin was 15.8 ng/ml, which was higher than the mean troponin level seen in literature of TCM. The Mayo Clinic Criteria describe a “modest” rise of cardiac biomarkers.⁶ Definitive feature of TCM on Echocardiography classically reveal extensive apical and/or midventricular akinesia or hypokinesia with basal segment function preserved or hyperkinetic.⁴ Apart from the apical involvement, new variants affecting mid-ventricular and basal segments of left ventricle have also been described. Repeat assessment on echo 6–8 weeks later showed complete recovery of left ventricular function in terms of wall motion and LVEF. Criteria proposed by the Mayo Clinic Group are most often used for diagnosis.⁶ In the study done by Pawlak et al., it was found that both patients suffering from a STEMI and TCM had a lower LVEF at initial presentation; however, follow-up echocardiograms demonstrated a significantly higher LVEF in the TCM patients than those who had STEMI.¹⁵ In our case echocardiography was showing typical changes of TCM with LVEF-25% and repeat echocardiogram showed completely recovered LVEF after six week, consistent with the transient nature of TCM.

The incidence of TCM is around 2.2% of all the admission presumed to have Acute.⁴ The etiology of TTC is related to stress and catecholamine-induced myocardial injury with elevated norepinephrine levels noted in many patients⁷. About 44% of the stressful events are emotional and 36% are physiological stressor.³ Other proposed etiology includes genetic predisposition¹⁴ or protective role of estrogen¹⁵. The pathophysiology of TCM is correlated to transient catecholamine surges due to any stress , which directly causes either multivessel epicardial spasm or microvascular coronary spasm and leads to possible Myocyte injury.⁷⁻⁹ Wittstein et al¹⁰ compared plasma catecholamine level in patients with TCM and acute MI with killip class III on presentation in emergency and found that catecholamine level were three times higher in TCM group than acute MI group. In our case coronary angiogram had ruled out any possibility of Myocardial infarction. Catecholamine level was elevated as per literature. So in this case our belief is that TCM has occurred due to elevated catecholamine caused by stress and pain due to gastritis. A review literature had shown acute gastritis as very rare cause of TCM.

There are no randomized treatment trials for TCM and since initial presentation mimics with MI so initial diagnosis and management is on

the line of acute MI with antiplatelet, and anticoagulants. Urgent coronary angiography is must to make the diagnosis of TCM. After the diagnosis of TCM has been established, therapy is mainly supportive with early start of Beta-Blockers and ACE inhibitors and continued until recovery of cardiac function.¹¹ Beta-blockers should be given as indefinite therapy in the absence of contraindications to help prevent future attacks in stressful situations.¹²

CONCLUSION

Takotsubo cardiomyopathy is a distinct reversible cardiomyopathy resulting from the effects of stress. It is important to identify the presence of emotional or physical stressors, including conditions that can cause severe pain such as acute gastritis in the case above described. This diagnosis can only be made after proof of recovery has been obtained on regular follow up. Our case is one of the rarest causes of Takotsubo cardiomyopathy described in literature.

Although various gastrointestinal conditions can trigger stress-induced cardiomyopathy, ours is the few gastritis-induced Takotsubo cardiomyopathy reported case. Clinicians should be aware of Takotsubo cardiomyopathy as a possible complication of acute gastritis and should consider this possibility in patients with chest pain, ECG changes, or left ventricular dysfunction with typical echocardiographic appearance in patients with acute gastritis.

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