

TAKOTSUBO CARDIOMYOPATHY IN A CASE OF LAPAROSCOPIC CHOLECYSTECTOMY PATIENT- A CASE REPORT

Mahak Kalra¹, Pratik Bhagat²

¹Consultant, Department of Anaesthesia, Bhagat Hospital, Delhi, India.

²Consultant, Department of Anaesthesia, Bhagat Hospital, Delhi, India.

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PRESENTATION OF THE CASE

A 53-year-old male presented to a secondary care hospital with pain abdomen for the past 2 months. He was diagnosed to have biliary colic and was posted for elective laparoscopic cholecystectomy. He was a known case of diabetes mellitus, controlled on oral hypoglycaemics. He had an exercise tolerance equivalent to more than 4 METS (Measurement of Exercise Tolerance) before the surgery.

On examination, the patient was average built, and the general condition was fair. Pulse rate (PR) was 74 beats per minute (bpm), blood pressure (BP) – 132/84 mmHg, SpO₂ – 99% on room air. All other systemic examinations were within normal limits. Investigations like electrocardiography (ECG) and chest radiograph were normal. Ultrasound abdomen showed multiple gall bladder calculi with grade 1 fatty liver, minimal ascitic fluid. Pre-operatively, all routine blood investigations were within normal limits.

Patient was prepared and shifted to the operation theatre. His pre-induction patient vitals were within normal limits (pulse rate 98 beats per min, BP – 131/88 mmHg, SpO₂ – 99% on room air). He was pre-medicated with fentanyl and midazolam and induced with propofol and vecuronium.

Anaesthesia was maintained with O₂, N₂O (50%), and isoflurane (0.6%-1.2%) using an endotracheal tube and intermittent doses of vecuronium. Analgesia was maintained with acetaminophen and fentanyl bolus.

EtCO₂ was preserved between 30 and 40 mmHg throughout the procedure, while PR and BP were maintained within 20% of baseline. At the end of surgery, neuromuscular blockade was reversed with injection neostigmine and glycopyrrolate.

After reversal and extubation, Non-Invasive Blood Pressure (NIBP) suddenly rose to 220/120 mmHg while heart rate had risen to more than 110 bpm and oxygen saturation fell below 94%. So, trachea was re-intubated with a cuffed endotracheal tube and mechanical ventilation with 100% oxygen provided. Injection furosemide and fentanyl were given.

Monitor ECG tracing showed ST segment elevation in the chest leads. 12 lead ECG was done, which was suggestive of an Acute Coronary Syndrome (ST changes in chest leads) with an anterior wall myocardial infarction (MI). Ryle's tube was inserted and tablet clopidogrel with tablet ecospirin were given. Injection Metoprolol was started that gradually decreased patient's PR to around 90 bpm stabilizing his BP around 150/90 mmHg.

Post-operatively, he was monitored in the intensive care unit (ICU) and put on controlled ventilation. Injection morphine boluses were given in gradual doses and put on fentanyl-midazolam infusion. Immediate post-op 2D echo was done that showed an enlarged left ventricle (apical) with a hypokinetic left anterior descending (LAD) territory. There was reduced left ventricular ejection fraction (LVEF) of 30-35% with grade II diastolic dysfunction, along with a trace mitral regurgitation (MR) and tricuspid regurgitation (TR).

So, the patient was given injection heparin as IV bolus followed by maintenance via intravenous infusion. Cardiac enzymes were sent and found to be within normal limits.

Within few hours, patient's vitals had settled down to PR- 78/min, BP- 100/60 mmHg. He was conscious and oriented, so was weaned to continuous positive airway pressure (CPAP) mode. After about 6 hours of the initial event, he was extubated, while ECG showed slight resolution of the ST changes with inverted T-waves.

A 2D echocardiography on the next day revealed an increased LVEF of 50%, decreased hypokinesis of LAD territory, a grade 1 LV diastolic dysfunction and mild systolic dysfunction.

Following this, patient was shifted to Low Molecular Weight Heparin (LMWH - Enoxaparin), ACE-Receptor inhibitor (Ramipril) and a beta-blocker (Metoprolol) therapy. Coronary Angiography revealed a non-critical coronary artery disease (30% obstruction in LAD).

CLINICAL DIAGNOSIS

Given the sudden decompression of vascular parameters along with acute ST segment elevation, a clinical diagnosis of acute coronary syndrome was made, which was later confirmed on 2D echo to be Takotsubo cardiomyopathy.

DIFFERENTIAL DIAGNOSIS

The above derangement of vascular parameters can be explained by either Acute Coronary Syndrome (ACS) or Stress-Induced Cardiomyopathy (Takotsubo Cardiomyopathy- TTC). While, ACS is a non-reversible condition, stress-induced cardiomyopathy is known to have left ventricular dysfunction reversal with removal of the

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Corresponding Author:
Dr. Mahak Kalra,
#G-83, Second Floor,
Phase – 1, Ashok Vihar,
Delhi- 110052, India.
E-mail: mahakkalra@gmail.com
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stimulus. In our case, the patient showed improvement over a couple of days, pointing towards a reversible cardiomyopathy. Takotsubo typically affects anterior wall to the apex and is not associated with any vascular stenosis or blockade while ACS can affect any wall and is generally associated with vascular supply abnormality. Also, cardiac enzymes, which are a diagnostic marker for ACS, is only mildly elevated in TTC.

PATHOLOGICAL DISCUSSION

Takotsubo Cardiomyopathy (TTC) is a type of non-ischemic cardiomyopathy, in which, there is a sudden temporary dysfunction of the myocardium.^{1,2}

In the early 1990s, Japanese physicians first recognized a syndrome characterized by chest pain, electrocardiographic changes, transient left ventricular apical aneurysm, and normal coronary arteries.

They reported five cases which presented with symptoms and ECG changes suggestive of acute MI. Echocardiography showed apical akinesia which was transient and resolved over 7 days. Further evaluation by cardiac catheterization disclosed no coronary artery stenosis in any of the patients.³

Subsequently, cases were reported by several physicians with similar findings. Patients presented with a wide range of severity of symptoms. By bee et al proposed criteria for diagnosis of this condition (mayo criteria).⁴ Because this dysfunction can be triggered by emotional stress, anxiety, death of a loved one or a break up this condition is also called as "broken heart syndrome".

It was termed the 'Takotsubo syndrome' as the left ventriculogram resembles an 'octopus trap' with a balloon like bottom and a narrow neck. Various other terms have been coined for the condition, such as stress - induced cardiomyopathy, apical ballooning syndrome or broken - heart syndrome.²

It has been described following some stressful events such as lightning strikes, hypoglycaemia, hyperthyroidism, earthquakes, and alcohol withdrawal, following surgery and following emotional stress.

Presentation of Takotsubo Cardiomyopathy mimics acute coronary syndrome. Patient can present with typical angina and dyspnoea. ECG changes seen in these patients include ST changes and T-wave inversion. ST elevation in aVR is more common in Takotsubo Cardiomyopathy than in MI. Q waves are often absent. Chest radiograph might show pulmonary congestion.

Typically, patients with TTS manifest modest increases in creatinine kinase-MB and cardiac troponin concentrations compared with patients with STEMI. Of interest, in TTS, there is a disparity between the degree of biomarker elevation and the extent of myocardial dysfunction observed at left ventriculography.

Echocardiography will show wall motion abnormalities may occur beyond the distribution of any single coronary artery. The type of TTC depends on the location of the wall motion abnormality, as detected by ventriculography or echocardiography. In the classic variety, the apical segment

(with or without the mid - ventricular segment) is akinetic or dyskinetic with hyper contractile basal segments.²

Diagnostic coronary angiography shows normal coronary arteries or non-obstructive coronary artery disease in the vast majority of patients. Cardiac catheterization - LV apical ballooning with normal coronaries.⁵

DISCUSSION OF MANAGEMENT

Treatment is similar to any acute MI. Long term treatment depends on whether LV function persists.

In our case, the patient was immediately started on antiplatelet drugs, diuretics and beta-blocker to manage the acute coronary event. He was given sedatives and painkillers to reduce the stressful event. He was electively ventilated for a few hours, maintaining alongside on anticoagulants and cardiac protective agents like ACE inhibitors and beta-blockers.

Prognosis is good, unless there is a serious complication (like MR or ventricular rupture). Recovery is expected in most cases after a period of weeks to months.⁶ Rarely, repeated episodes have been observed.⁷

FINAL DIAGNOSIS

Takotsubo Syndrome/Stress-Induced Cardiomyopathy

It occurs following surgical stress during reversal of anaesthesia. Re-intubation, with treatment to control vascular parameters along with heparinisation of blood seemed to help relieve the symptoms and improve cardiac parameters, which were monitored and followed up with echocardiography. Prognosis is mostly good, and recovery takes few days to weeks. Active, rapid therapy with knowledge of the subject can help with early recovery and provide a better prognosis.

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