orphanaesthesia

Anaesthesia recommendations for patients suffering from

Stress cardiomyopathy

Disease name: Stress cardiomyopathy

ICD 10: I42

Synonyms: Ballooning cardiomyopathy, broken heart syndrome

Stress cardiomyopathy (SCM) is a transient clinical condition, which mimics myocardial infarction in patients with no coronary heart disease.

Typical SCM affects the apex of the left ventricle and causes ballooning of apex. This is popularly known as “Takotsubo cardiomyopathy” or “Broken heart syndrome”. SCM wherein there is no involvement of the apical segment of the heart is called as atypical or variant SCM.

The occurrence of SCM in the perioperative period is no longer rare. It is being reported regularly even immediately prior to surgery in patients with excessive anxiety. The prevalence of SCM is estimated to be 1.2-2.2%, and the atypical SCM constitutes 40% of these cases.

Even though the aetiology of SCM is considered to be exposure to excessive levels of catecholamines in genetically susceptible individuals, the exact mechanism is not yet proven. The pathophysiology could be explained by (1)enhanced sympathetic activity originating from the central nervous system (2) catecholamine induced microvascular endothelial dysfunction or (3) coronary vasospasm.

Medicine in progress

Perhaps new knowledge

Every patient is unique

Perhaps the diagnostic is wrong

Find more information on the disease, its centres of reference and patient organisations on Orphanet: www.orpha.net

Disease summary

Atypical SCM constitutes four types depending on the patterns of LV involvement: (1) inverted/reverse (2) mid-ventricular (3) focal and (4) global type. Recently a fifth type'' Reverse mid-ventricular' has been described, where the apex and base are akinetic/hypokinetic and the mid-left ventricle is hyper dynamic.

There are case reports of involvement of both ventricles or the right ventricle alone. The difference in the distribution, density and sensibility of adrenergic receptors over the myocardium determines the area of hypokinesis while the cause remains the same. The area with a highest density of adrenergic receptors is typically the most affected region. The most common of these atypical forms is the inverted type with hyperdynamic apex and akinetic base of the LV.

The density of adrenergic receptors is relatively high at the apex in the elderly population compared to younger patients. Hence, elderly patients, especially postmenopausal women, are more prone for classical SCM where as younger patients are more prone for the atypical variety. Atypical variety presents with a lower prevalence of T-wave inversion. Release of troponin is higher, which is the consequence of the larger muscle region involved. Basic natriuretic peptides (BNP) are more elevated in apical and mid-ventricular patterns, which is evident by more severe symptoms. The rise in cardiac markers and ECG changes may be similar to acute coronary syndrome but a rise in BNP is much more in SCM.

The most common complications described are cardiogenic shock (10% of patients), ventricular arrhythmias, formation of apical thrombus, stroke, obstruction of the left ventricular (LV) outflow tract, LV rupture and sudden cardiac death. Some might even develop pleural effusion and mitral valve regurgitation (resulting from chordal tethering as well as systolic anterior motion of the mitral valve apparatus).

Diagnosis is made by coronary arteriography, left ventriculography, and echocardiography. Differential diagnosis includes acute coronary syndrome and other types of transient ventricular dysfunction such as seen in sepsis or myocarditis.

The condition is transient and the main stay of treatment is supportive. β-adrenergic blockers, angiotensin converting enzyme inhibitors, statins, diuretics and heparin with or without clopidogrel or aspirin are the mainstay of treatment.

Patients who develop hypotension must be evaluated for a dynamic intra-ventricular pressure gradient in the left ventricular cavity and left ventricular outflow tract. Echocardiography or left heart catheterisation can be used for this evaluation.

Dynamic intra-ventricular obstruction in patients with the syndrome is managed by administration of beta-blockers (to increase diastolic ventricular filling time and left ventricular end-diastolic volume), administration of phenylephrine (to increase afterload with subsequent reduction of the intra-ventricular gradient) and administration of fluid resuscitation if pulmonary congestion is not present. However, beta blockers and phenylephrine would not be recommended for the treatment of dynamic intra-ventricular obstruction in patients with documented epicardial coronary vasospasm. In this situation, a nondihydropyridine calcium-channel blocker, such as diltiazem or verapamil, could be considered.

If there is no dynamic intra-ventricular obstruction, inotropes of choice are levosimendan or Milrinone. If cardiogenic shock is persisting, intra-aortic balloon pump (IABP) or ventricular assist devices (VAD) can also be used. Use of extracorporeal membrane oxygenation has also been reported.

Typical surgery

All elective cases with SCM should be postponed.

Any surgical procedure which has to be done on an emergency basis in patients who are exposed to severe mental or physical stress, for example, emergency craniotomy because of intracranial bleed, post poly trauma surgeries, postpartum bleed and explorations, might have to be accepted with due risk of perioperative cardiac failure, ventricular arrhythmias, left ventricular rupture and sudden cardiac death.

Type of anaesthesia

Peripheral nerve blocks can be used wherever possible like upper limb vascular injuries, orthopaedic open fracture fixations or wound debridements.

Use of a laryngeal mask airway can be useful if the procedure is short and patient is not having severe hypotension, pulmonary oedema or pleural effusions. This avoids the stress response to intubation.

General anaesthesia (GA) with endotracheal intubation is the choice if the patient is haemodynamically unstable, if there is pulmonary oedema or pleural effusion, surgical risk is higher or there are other indications for GA.

A decrease in systemic vascular resistance secondary to spinal or epidural anaesthesia might not be well tolerated. In addition, the patient may be on anticoagulants, which could prevent the use of neuraxial anaesthesia.

Necessary additional diagnostic procedures (preoperative)

ECG, Trans thoracic or transoesophageal echocardiogram.

Cardiac enzyme markers, coronary angiogram to ruleout acute coronary syndrome. Cardiac enzymes help not only in diagnosing the disease, their increasing trend indicates ongoing ischemic damage and poor myocardial function.

Chest X-ray to rule-out pulmonary congestion.

Serum electrolytes, liver and renal function tests and coagulation profile.

Particular preparation for airway management

Care to prevent stress response to laryngoscopy and intubation.

Particular preparation for transfusion or administration of blood products

Intravenous fluid administration should be done with caution and preferably guided by transesophageal echocardiogram (TEE). Use of CVP, Pulmonary artery catheter is not much useful in the presence of LV failure. Systolic volume variation (SVV) can be another useful guide for fluid therapy. Blood product administration should follow general guidelines of maintaining adequate blood volume and haemostats without causing volume overload.

Particular preparation for anticoagulation

Patient may be on anticoagulants. Due care should be taken.

Particular precautions for positioning, transport or mobilisation

Invasive monitoring should be instituted preferably in the intensive care unit itself. Patient should be continuously monitored while shifting. Rapid changes in the body position should be avoided as the heart might not be able to compensate. Sudden head low or head up might not be well tolerated as the heart can't cope up for sudden increase or decrease of preload with these positions respectively.

Probable interaction between anaesthetic agents and patient's long-term medication

Patient might have been started on β-adrenergic blockers, angiotensin converting enzyme inhibitors (ACEI), diuretics and heparin.

Continue β blockers and if possible stop diuretics and ACEI's atleast one day prior to surgery. If not possible to stop, we should be aware that inhibition to Renin Angiotensin Aldosterone System (RAAS) by ACEI's can increase the risk of intraoperative hypotension. This can be treated with phenylephrine or vasopressin.

As these patients usually come for emergency procedure, cardiologist can delay starting of clopidogrel until surgery is over. However heparin can be started if there is time to stop it 6 hours prior to surgery, especially if the surgery involves brain, spine or intraocular structures. Reversing heparin effect with protamine might cause LV thrombus. Pros and cons should be considered.

Anaesthesiologic procedure

Management strategies are similar to managing a case of acute cardiac failure patient coming for emergency surgeries.

Intra-operatively:

Anaesthetic goals would be:

A. Maintain sinus rhythm

B. Avoid sudden increases in preload like rapid fluid boluses or sudden head low.

C. Maintain normal after load: High or low will not be tolerated

D. Monitor and maintain optimum contractility of the heart.

Before induction of anaesthesia, along with standard non invasive monitors, arterial line has to be inserted under local anaesthesia if it is not there already. Good peripheral vascular access should be obtained if there is no central line. Attach defibrillator pads to the chest and apply pneumatic compression stockings to the lower limbs.

Poor ventricular function can cause severe hypotension on induction with propofol. Etomidate in titrated doses or opioid based induction is well tolerated. Avoid any stress responses during intubation. Positive pressure ventilation and positive end expiratory pressures (Peak pressure < 30 cm of H2O) are beneficial to improve oxygenation.

Cardiac output monitoring with pulmonary artery catheter or Pulse index Continuous Cardiac Output (PiCCO) monitor can be used, but is not routinely required. Pulmonary capillary wedge pressure might not be useful for intravenous fluid management due to poor ventricular compliance. Hence trans oesophageal echocardiogram (TEE) is recommended in such cases. In addition TEE can also give information regarding ventricular wall motion, presence or absence of dynamic ventricular outflow obstruction and valvular functions.

If inotropic support is needed, a calcium sensitiser such as levosimendan may be a good choice if it is available. It is an inodilator and improves myocardial contractility without increasing intracellular levels of calcium and hence there is no increase in myocardial oxygen demand. Milrinone is reported to be of benefit in one case where patient had SCM following head injury. Adding a vasopressor like phenylephrine to counter vasodilatation caused by these drugs can be helpful. Excessive inotropy may worsen LV outflow obstruction and hence the inotropes should be carefully titrated.

In case catecholamines has to be used, avoid using them in high doses. But there is always chances of developing tachyarrhythmia.

Close watch on urine output, core body temperature, airway pressures and acid base status will help in better management.

Dosage of Levosemendan: 18-36ug/kg bolus followed by 0.05 to 0.2 µg/kg/min as a continuous infusion. Totally avoiding or using reduced bolus dose of 6-24 µg/kg can

be considered as bolus dose can cause precipitous drop in blood pressures. Levosimendan should not be administered to children or adolescents under 18 years of age. It is not advised for more than 24hours.

Dosage of Milrinone: 50 mcg/kg loading dose by IV push over 10 minutes, then 0.375-0.5 mcg/kg/min IV. Bolus dose can be avoided.

Particular or additional monitoring

Five lead ECG, Cardiac output (PiCCO), transesophageal echocardiogram and SVV

Keep defibrillator, pacemaker, IABP or VAD's as standby.

Possible complications

Cardiogenic shock, ventricular arrhythmias, formation of apical thrombus, stroke, obstruction of the LV outflow tract, LV rupture and sudden cardiac death.

Postoperative care

Postoperative extubation depends upon severity of heart failure and type of surgery.

Continued monitoring in intensive cardiac care unit.

Adequate anxiolytics and good pain relief.

Continue all the preoperative medications.

Information about emergency-like situations / Differential diagnostics

Hypotension and arrhythmias are the main complications expected. TEE will help in early diagnosis and appropriate management. Arrhythmias can be managed as per standard protocols and life threatening hypotension needs IABP or VAD's

Ambulatory anaesthesia

Not advised.

Obstetrical anaesthesia

General anaesthesia with all the safety and precautions is advisable. Invasive arterial line inserted prior to induction is must. Modified rapid sequence induction with Etomidate, Fentanyl and Succinyl choline can be acceptable. Insertion of central line

is better done prior to induction. If surgery is emergency atleast external jugular vein should be accessed. Inotropes should be loaded and kept ready.

Use of TEE or PA catheter should be decided on case to case basis. Neonatologists should be well informed about the possibility of respiratory depression in newborn. They should keep intubation trolly and naloxone ready. Fluid management should be done very cautiously as parturients have very delicate fluid requirements.

Hypotension on induction can be managed with phenylephrine. Oxytocin should be administered carefully as it causes tachycardia and reduces systemic vascular resistance. Post delivery there is possibility of pulmonary oedema because of abrupt inflow of large volumes of blood into the systemic circulation as a consequence of uterine contraction. Small dose of frusemide after the delivery of the baby can be given. Sometimes patient might need postoperative mechanical ventilation.

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Last date of modification: March 2017

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