

Introduction

Cardiomyopathy during pregnancy presents significant challenges for the multidisciplinary team (MDT) in maternal medicine centres. We describe a case of dilated cardiomyopathy with severe left ventricular (LV) dysfunction diagnosed following COVID-19 infection.

Antepartum

- 41 year old gravida 4 para 3 – previous uneventful pregnancies with spontaneous vaginal deliveries.
- Past medical history – nil of note.
- Presented at 24 weeks gestation with COVID-19 pneumonitis complicated by severe LV dysfunction.
- She was transferred to a cardiac centre for further investigation and treatment.
- Transthoracic echo (TTE) revealed a severely dilated LV with severely impaired function (EF 20-25%) with moderate mitral regurgitation.
- COVID myocarditis was considered, however diagnostic criteria were not met, and MRI did not support this diagnosis.
- She was discharged on medical treatment and anticoagulation, for follow up by the maternal cardiac MDT.
- At 30 weeks, TTE showed severe biventricular dysfunction. A delivery plan was made by the maternal cardiac MDT for both caesarean section and spontaneous vaginal delivery.

Intrapartum

- Delivery was by elective caesarean section at 36 weeks gestation.
- She was admitted one day prior to her caesarean section for pre-optimisation.
- Peripheral and central venous access were obtained.
- Arterial line was inserted for invasive blood pressure and cardiac output monitoring
- Combined spinal-epidural was performed.
 - Spinal component** – 5mg heavy bupivacaine plus diamorphine.
 - Epidural top up** – 20ml ‘fast mix’ in slow increments.
- Bloop pressure was maintained with noradrenaline up to a dose of 0.1mcg/kg/minute.
- Oxytocin was given via the cardiac oxytocin regime.
- The caesarean section was uncomplicated, with an estimated blood loss of 530ml.

Postpartum

- After delivery she was admitted to intensive care unit for weaning of inotropic support, close observation, and fluid balance management. Noradrenaline was weaned and an even to negative 500ml fluid balance was maintained.
- TTE prior to discharge showed severely impaired LV function (EF 20-25%) with severe mitral regurgitation and right ventricular impairment.
- She was discharged day seven post-delivery with a plan for follow up in the Obstetric Cardiac clinic in two weeks.

- Day 13 post-delivery she was admitted with an acute decompensation, hypotension and acute kidney injury secondary to E. coli sepsis. She was admitted to a cardiac intensive care unit and was stabilised with antibiotics and inotropic support.
- She is now followed up by the heart failure team and her most recent TTE showed some improvement in LV function on standard heart failure therapy (LVEF 42%).
- She has been referred for a cardiomyopathy genetic screen.

References

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Dilated cardiomyopathy perioperative SOP¹

This guidance provides general advice for the management of women with dilated cardiomyopathy in pregnancy, not every aspect will be applicable to an individual patient, particularly if they have mixed disease. All patients with moderate and severe cardiac conditions will be seen by the Maternity Cardiac MDT and have a personalised delivery plan , which should be followed.		Regional or general anaesthesia in severe DCM in pregnancy	Haemodynamic targets: Rhythm: Maintain sinus rhythm to optimize LV filling, AF poorly tolerated Rate: Maintain normal heart rate and avoid bradycardia Afterload: Maintain by avoiding hypertension or hypotension and keeping blood pressure within the range normal for her. Systemic vascular resistance (SVR): Maintain at all times. Peripheral vasoconstrictor infusion from induction –an abrupt drop in SVR with anaesthesia acutely reduces coronary perfusion and may initiate an ischaemic spiral of decompensation Contractility: Inotropic support <i>only if evidence of acute decompensation</i> . Calcium chloride or dilute adrenaline boluses (10mcg/ml). If good response considers low dose inotrope adrenaline (0.01-0.04) +/- milrinone (0.1–0.2) infusions.		
Physiological impact of pregnancy, labour and left ventricular dysfunction	Physiological changes in pregnancy: The physiological increases in heart rate and plasma volume with pregnancy can result in heart failure. Greater risk of pulmonary oedema due to the reduction in oncotic pressure during pregnancy. Pregnancy carries an excessive maternal risk in patients with severe dilated cardiomyopathy (ejection fraction of <30% or NYHA class III – IV), women with dilated cardiomyopathy (DCM) are at risk of heart failure particularly during the 3 rd trimester and postpartum. Broad aims of high-risk management = rate control, maintaining afterload, contractility and normovolaemia, +/- diuresis, serial ECHO to assess LV. Labour is poorly tolerated in patients with severe dilated cardiomyopathy: 1 st stage – CO increases by 30% 2 nd stage – Valsalva causes a reduction in the preload and increase in the afterload during the manoeuvre and cardiac output surge after the manoeuvre. 3 rd stage – Risk of acute pulmonary oedema from autotransfusion.				
Optimising Cardiac Function	The first two medications are used to optimise cardiac function and are often started antenatally. They should be continued during and after delivery until review by the cardiologist. Beta-blockers – to reduce tachycardia of pregnancy and AF prevention as fast AF risks an acute decompensation Diuretics – to maintain fluid balance even to negative or as negative as tolerated haemodynamically. Ensure that the blood pressure is always maintained within the range normal for the patient. Maintaining contractility – Ephedrine boluses as a first line medication, if low cardiac output persist consider milrinone with the addition of phenylephrine/adrenaline to maintain blood pressure.				
		Anaesthetic plan	Cautious regional anaesthesia is usually the anaesthetic of preference unless specific indication for GA. For regional or GA: - Minimum 2 IV cannulae - Maintain patient’s baseline HR & BP (use phenylephrine infusion for tight BP control) - Aim even fluid balance (replace like with like) - Acute AF likely to be very poorly tolerated, aim to keep in sinus (e.g., have 2.5 – 5 mg bolus doses of metoprolol available) Discussion with, or attendance by, cardiac anaesthetist (oncall at Barts) if requires TOE to guide further management or manage cardiac instability <table><tr><td>For regional: - may need Low-dose sequential CSE, e.g. 1.5 ml 0.5% heavy bupivacaine + diamorphine 300 mcg, plus slow epidural top-ups 5 ml every 5 min as required (if urgency allows) or just epidural with slow top–ups if cardiovascularly not stable. </td><td>For GA: - Arterial line before induction, baseline ABG useful - Use high dose opiate technique to minimise cardiovascular instability - Keep pO2 & pCO2 in the normal range, avoid high ventilation pressures - Aim for smooth (non coughing) extubating </td></tr></table>	For regional: - may need Low-dose sequential CSE, e.g. 1.5 ml 0.5% heavy bupivacaine + diamorphine 300 mcg, plus slow epidural top-ups 5 ml every 5 min as required (if urgency allows) or just epidural with slow top–ups if cardiovascularly not stable.	For GA: - Arterial line before induction, baseline ABG useful - Use high dose opiate technique to minimise cardiovascular instability - Keep pO2 & pCO2 in the normal range, avoid high ventilation pressures - Aim for smooth (non coughing) extubating
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Fluid Balance	- Even fluid balance is reasonable in the context of acute bleeding and ongoing uncertainty regarding haemostasis. - Once bleeding is under control, and certainly in an otherwise normal delivery even to negative fluid balance, or “as negative as tolerated” for patients with severe dilated cardiomyopathy is preferable. - Measure fluid input and output meticulously, keeping 24 hour fluid balance between EVEN to negative 500ml, or as negative as tolerated haemodynamically.
Physiological aims during delivery	Minimise cardiovascular stress Avoid: - Bradycardia - Hypervolaemia (risk of pulmonary oedema) - Myocardial depression - Profound/sudden vasodilation (Afterload and SVR must be maintained) - Hypoxaemia/Hypercarbia (increased pulmonary vascular resistance → reduced left ventricular filling)
Monitoring	Input-output chart, ECG, SpO2, BP (Invasive or NIBP) throughout admission High risk cases may require antenatal HDU admission for monitoring and optimisation Invasive arterial monitoring should be used in severe DCM Cardiac output monitoring intraoperatively and in intensive care setting particularly in women with severe disease Central venous access may be required in severe pathology
Delivery options	Delivery option dependent on timing and symptoms. Mild cases of DCM with NYHA I/II (Appendix 1) can usually plan for a vaginal delivery and be managed on the labour ward. Caesarean sections (with invasive monitoring) should be considered in the following cases: - NYHA class III/IV - Deteriorating LV function - Heart Failure
Management of Uterotonics and PPH	Cardiac oxytocin regimen - Draw up the usual 40 units of oxytocin in 500 ml 0.9% saline - Take 25 ml (2 units) from this mixture and run this in a syringe driver over 15 min (=100 ml/hr) as the ‘bolus’ - Then start the usual volumetric infusion with the remaining 38 units in 475 ml at 5 units/hr over 5 hours (= 63 ml/hr with total volume to be infused 250 ml) AVOID ERGOMETRINE – causes pulmonary vasoconstriction Misoprostol has less vasoactive effects and is preferred to haemabate. Mechanical methods such as bimanual compression, Bakri balloon and B-lynch suture are preferred in patients with moderate-severe MS.
Decompensation / Low CO state*	Clinical: B: Increasing RR, increasing oxygen requirements, pulmonary oedema C: Cool peripheries, SYS BP <90, HR >120 or <50, ScVO2 <60%, raised JVP with peripheral oedema (suggestive of right heart failure) D: Drowsy, agitated, confused E: Oliguric <0.5 ml/kg/hr Biochemical: Lactate >2 mmol/L, BNP >128 Imaging: LVEF <30%, RV impairment, lung US with characteristic B lines *If concerns regarding decompensation refer to Barts ECMO team Management: Urgent medical review plus ECHO if signs of cardiac decompensation (see above) Discuss with cardiology, obstetrics and ITU re potential expedited delivery. - Arterial line essential - Furosemide bolus +/- GTN, and expedited delivery plan - Optiflow or CPAP (in Critical Care setting) - If evidence of low cardiac output state, site CVC and start milrinone / adrenaline infusion The Labour ward might not be the safest place for admission, discuss with ITU, cardiology and obstetric teams the need for 4th floor ITU admission

Discussion

The overall mortality for pregnant women with cardiomyopathy is 2.4%.² If a patient has moderate–severe LV dysfunction, the risk of a cardiac event during or up to 6 months after pregnancy increases to 64%.³ COVID-19 during pregnancy is associated with a variety of cardiovascular complications. Whilst it was not the direct cause of ventricular dysfunction in this case, it may have affected her presentation. Mortality data of women with cardiac disease shows that only 14% of women die during the pregnancy, with the majority dying in the postpartum period.⁴ This highlights the importance of dedicated MDT follow up of these patients in the weeks and months after delivery.

Conclusion

Cardiac disease is still one of the leading indirect causes of death in pregnant women. Those with cardiomyopathy have worse outcomes when compared to other cardiac diseases.² Having considered many differentials, this woman was diagnosed with pre-existing DCM unveiled by the haemodynamic challenges of pregnancy. The peripartum management of patients with severe LV dysfunction requires robust MDT planning for preoptimisation, intrapartum, and postpartum care. Most deaths occur in the postpartum period, and the deterioration in this case is a reminder of how brittle these patients are and that close postpartum follow-up is essential.