Postpartum-Cardiomyopathy With Severe Hypocalcemia And Tetany

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Citation

S Mishra, R Bhat, Anita, N Mahesh, P Kundra, B Hemavathy, A Badhe. *Postpartum-Cardiomyopathy With Severe Hypocalcemia And Tetany*. The Internet Journal of Anesthesiology. 2006 Volume 12 Number 1.

Abstract

We present a case of postpartum cardiomyopathy who presented with carpopedal spasm 72 hours after undergoing a caesarian section under spinal anaesthesia. A 25 year old primigravida with mild PIH was posted for emergency LSCS for CPD in labor. LSCS was done under spinal anaesthesia, later converted to GA for internal iliac artery ligation due to atonic uterus. Intraoperative vital parameters were stable, end operatively patient was shifted to ICU and extubated after 2 hours of ventilatory support.
3 days later, she had repeated episodes of carpopedalspasm, not responding to IV calcium infusion and progressed to laryngospasm requiring intubation. The patient had tachypnoea with respiratory alkalosis during this period. Serial X-rays showed progressively increasing bilateral infiltrates suggesting cardiac failure. Echocardiography was done which revealed severe LV dysfunction and a diagnosis of Postpartum Cardiomyopathy was made.

INTRODUCTION

Peripartum Cardiomyopathy is a relatively rare but life-threatening disease. A wide variation in incidence rates ranging from 1 per 1500 to 1 per 15,000 live births has been reported although the currently accepted incidence is approximately 1 per 3000 to 1 per 4000 live births. Peripartum Cardiomyopathy is defined by the presence of four criteria. These include: ($_1$) Development of cardiac failure in the last month of pregnancy or within five months of delivery; ($_2$) Absence of an identifiable cause for cardiac failure; ($_3$) Absence of recognizable heart disease prior to the last month of pregnancy; and ($_4$) Left ventricular systolic dysfunction demonstrated by echocardiography criteria such as decreased ejection fraction.

Stricter echocardiography criteria have been recommended (a left ventricular ejection fraction of less than 45 percent, fractional shortening of less than 30 percent on an M-mode echocardiography scan, or both, and a left ventricular end-diastolic dimension of more than 2.7 cm per square meter of body-surface area).

We describe here a case of postpartum Cardiomyopathy with severe hypocalcaemia.

CASE REPORT

A 25 years old primigravida with mild PIH posted for emergency LSCS for large baby with CPD in labor. She was febrile-37.8°C. There was no pallor or edema, and she had a heart rate of 90/minute, blood pressure 130/80mmhg, and respiratory system -LRI with minimal crepitation in the left infra-scapular area. Cardiovascular system –normal heart sounds, no adventitious sounds. Cesarean section was done under spinal anaesthesia, later converted to GA due to atonic uterus .Blood loss was around 2 Liters. Hysterectomy and bilateral internal iliac artery ligation was done. Total duration of surgery was three hours. She received 1000 ml of Colloids, 600 ml of Packed Cells and four units of FFP. Blood pressure was stable, urine output 200ml, SPO2 98-100 % with FIO2 of 33%. Because of massive blood loss and hypothermia (35.5 ° C) she was transferred to the ICU. The patients vitals were stable, temperature improved to 37 °C and she was extubated within 2 hr in ICU. Postextubation ABG were within normal limit.

On postoperative day-2, the patient was febrile, 38-39°C and had a heart rate of 120/min and respiratory rate of 25 to 30/min. Room air SPO_2 was 92% and with 40% FiO_2 98%. Decrease air entry and crepitation was noted on the left lower lung.

CXR showing infiltrates over both lower lung fields. Spo2 improved with intermittent NIV. SpO2 -98% in room air and started on antibiotics. Laboratory examination- Hb 8 gm%,

completes haemogram within normal limits. Blood urea, creatinine, electrolytes and sugar were within normal limits.

On postoperative day 3 and 4, the patient had two more episode of tetany, treated with IV calcium gluconate. Serum total calcium was less than 7 meq/dl. Potassium was 2.8meq/dl. Infusion IV calcium-gluconte (elementary calcium up to 2mg/hr)

(1 Ampules of 10% calcium gluconate containand 93.4 mg of elementary calcium.) IV Potassium Chloride 5meq/hr were started.

Calculation: An estimate to correct for hypoalbuminemia is to subtract 0.8 mg/dL from the total serum calcium for each 1.0-g/dL decrease in albumin below 4.0 g/dL.

On postoperative day 5 and 6 the patient had repeated episodes of carpopedalspasm, associated with tachypnea, disorientation, restlessness, confusion and blurring of vision, treated with IV calcium gluconate SPO_2 was 98% with 40% FIO_2. Heart rate: 120/minutes, Blood Pressure: 110/70mmHg with CVP of 12mmHg. She was treated with IV calcium gluconate, and Infusion IV calcium-gluconte,

Laboratory examination: total Calcium <7gm%, Magnesium 1.8 mg/dl, sodium-138, potassium-2.4meq/dl, Albumin2.6gm/dL, Total protein-4.6, 24hr Urinary calcium, phosphate and Uric acid within normal limits.

ECG: Qtc prolonged.

ABG: PaO2-182, PaCo2-37, PH-7.49 HCO3--28, SPO2-99% A-aDO2-56.

The patient had one more episode of carpopedalspasm with laryngospasm associated with desaturation and bradycardia, requiring intubation and ventilation on SIMV mode with respiratory rate of 12/minutes, PSV 12 and 40 % FIO_2 , SpO_2 was 98% . Repeat CXR showed cardiomegaly and ECG was normal.

Figure 1

Serial ABG

	DAY-1	DAY-2	DAY-3 -4	DAY 5-6	DAY-7	Day-9
PH	7.467	7.482	7.499	7.487	7.417	7.445
Pao2 40%FIo_2	104	70.5	110	102	134	94
Paco2	37	37	38.9	37.5	35	41
Hco3	22.2	27	28.2	26	22.2	27
BE	-1	5.5	5.6	3	-1	4
SpO2	98.7	95.3	98.5	99	98.7	97.4

Echocardiography: Severe LV dysfunction with EF 35%, global hypokinesia, mild MR and mild pericardial effusion. Ophthalmology: Fundus examination was normal.

Serial CXR: Progressive increase in bilateral basal infiltrate.

Echo heart diagnosed as postpartum Cardiomyopathy presenting as hypocalcemia.

Tablet Enalapril 2.5 mg twice daily, and tablet spiranolactone 12.5 mg once daily was started. The patient was put on fluid restriction, and maintained CVP of 10 to12 mmHg.

The patient gradually improved after 3 days of treatment. Vital parameters were normal. Spo2 was 99% with 40% FIO2. ABG was within normal limits. Serum total calcium improved to 8.5mg/dl. She was weaned and extubated on post-operative day-10. Post-extubation vital parameters were within normal limits. Repeat Echo heart after 2 weeks showed mild LV dysfunction (ejection fraction-50%) with minimal pericardial effusion, mild PAH, and no thrombus.

DISCUSSION

Peripartum cardiomyopathy is a rare but serious complication of pregnancy, presenting more commonly in the early postpartum period. Medical treatment of peripartum cardiomyopathy is similar to that for other dilated cardiomyopathies.

Management goals include preload optimization, afterload reduction and increase contractility. Anticoagulation is also considered in many patients because of the significant risk of thromboembolism before delivery

When the patient develops cardiac failure before delivery, some treatment modifications are required. Angiotensin-converting enzyme inhibitors are routinely used for afterload reduction in congestive heart failure. However, these drugs

are contraindicated during pregnancy because of adverse fetal effects. Alternative treatments for afterload reduction during pregnancy include amlodipine or a combination of hydralazine and nitroglycerin.

The main stay of treatment is ACE inhibitors ,diuretics and fluid restriction. This case was complicated by hypocalcemia precipitated by alkalosis due to hyperventilation.

References

1. Lampert MB, Lang RM. Peripartum cardiomyopathy. Am Heart J. 1995;180:860-70.
2. Cunningham FG, Pritchard JA, Hankins GD et al. Peripartum heart failure: idiopathic cardiomyopathy or compounding cardiovascular events? Obstet Gynecol. 1986;67:157-68.
3. Pearson GD, Veille JC, Rahimtoola S et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. JAMA. 2000;283:1183-8.
4. Bell EA. Why mothers die in the southeastern United States. Anesthesiology. 2003;98 Supp 1:A15.
5. Heider Al, Kuller JA, Strauss RA et al. Peripartum cardiomyopathy: a review of the literature. Obstet Gynecol Surv. 1999;54:526-31.
6. Midei MG, DeMent SH, Feldman AM et al. Peripartum myocarditis and cardiomyopathy. Circulation. 1990;81:922-8.
7. Nizeq MN, Rickenbocker PR, Fowler MB et al. Incidence of myocarditis in peripartum cardiomyopathy. Am J Cardiol. 1994;74:74-7.
8. O'Connell JB, Costanzo-Nordin MR, Subramanian R et al. Peripartum cardiomyopathy: clinical, hemodynamic, histologic, and prognostic characteristics. J Am Coll Cardiol. 1986;8:52-6.
9. Lee W. Clinical management of gravid women with peripartum cardiomyopathy. Obstet Gynecol Clin North Am. 1991;18:257-71.
10. Demakis JG, Rahimtoola AI, Sutton GC et al. Natural course of peripartum cardiomyopathy. Circulation. 1971;44:1053-61.
11. Witlin AG, Mabie WC, Sibai BM. Peripartum cardiomyopathy: an ominous diagnosis. Am J Obstet Gynecol. 1997;176:182-8.
12. Sutton MSJ, Cole P, Plappert M et al. Effects of subsequent pregnancy on left ventricular function in peripartum cardiomyopathy. Am Heart J. 1991;121:1776-8.
13. Lampert MB, Weiner L, Hibbard J et al. Contractile reserve in patients with peripartum cardiomyopathy and recovered left ventricular function. Am J Obstetr Gynecol. 1997;176:189-95.
14. George LM, Gatt SP, Lowe S. Peripartum cardiomyopathy: four case histories and a commentary on anaesthetic management. Anaesth Intens Care. 1997;25:292-6.
15. Beus E, van Mook WN, Ramsay G, Stappers JL, van der Putten HW. Peripartum cardiomyopathy: a condition intensivists should be aware of. Intensive Care Med. 2003 Feb;29(2):167-74.

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