

SVV Guided Fluid Therapy in a Case of Heart Failure with Hypotension

Case Study

INTRODUCTION

Fluid resuscitation in severely critical patients can be challenging, particularly when the patient is in cardiogenic shock with pulmonary edema and hypoxia needing mechanical ventilation. Traditional hemodynamic monitoring parameters obtained via a pulmonary artery catheter do not address the issue of fluid responsiveness in patients on mechanical ventilation. In this case study, we describe the use of stroke volume variation (SVV) monitoring, which guided our decision to add fluids in order to improve cardiac output (CO). SVV guided fluid optimization led to a successful outcome in this seriously ill patient.

Clinical Events

Patient details: 32-year-old male
Medical history: Diagnoses: type 2 diabetes; idiopathic dilated cardiomyopathy with left ventricular ejection fraction (LVEF) of 20%. Receiving optimal anti-heart failure therapy of torsemide, ramipril, carvedilol, digoxin, plus insulin

CASE NOTES

The patient was admitted with chief complaints of progressive dyspnea even at rest (NYHA Class IV heart failure) and swelling of feet. He was in sinus rhythm, his heart rate was 90 beats/minute, blood pressure was 108/60 mmHg, (mean arterial pressure 78 mmHg), respiratory rate was 22/minute. He was treated with intravenous diuretics, nitroglycerin, dobutamine and mask oxygen. The patient was evaluated and referred for cardiac resynchronization therapy (CRT).

Investigations before CRT:

- Hemogram: Hb – 11.9 gm%, total leucocyte count (TLC) – 11,000/cmm, platelets – 252,000/cmm
- Blood urea levels (BUL) – 24 mg%, serum creatinine – 1.1 mg%, serum sodium – 132 mEq/L, serum potassium – 3.5 mEq/L, serum chloride – 97 mEq/L
- Blood sugar levels were well controlled on insulin
- ECG showed LBBB pattern with QRS duration of 146 msec
- Chest x-ray showed cardiomegaly with bilateral lower zone haziness suggestive of pulmonary edema

- Pre-procedure echocardiographic evaluation showed dilated cardiomyopathy with global hypokinesia, LVEF 20%, Grade I mitral regurgitation with mild pulmonary hypertension with pulmonary arterial systolic pressure (PH) of 38 mm

CRT procedure:

The patient underwent CRT in the form of bi-ventricular pacing under general anesthesia. Drugs used during the procedure were fentanyl 100 mcg, midazolam 4 mg, and ketamine 50 mg for induction and propofol 1-1.2 mg/min for maintenance. Total procedure time was 5 hours. Patient required dopamine 5-8 mcg/kg/min throughout the procedure to maintain blood pressure. Central venous pressure (CVP) and intra-arterial blood pressure monitoring was performed throughout the procedure.

Advanced hemodynamic monitoring (such as PA catheter, arterial pulse based cardiac output, transpulmonary thermodilution methods or transesophageal echo-Doppler) was not used during the procedure.

Ventilatory requirements during the procedure were high due to cardiogenic pulmonary edema. Patient received 60 mg of furosemide during the procedure. Patient was ventilated with pressure-controlled ventilation, peak inspiratory pressure of 35 cm, PEEP of 10 cm, and FiO₂ of 1. Intra-operative ABG values were: pH – 7.19, PO₂ – 62 mm, PCO₂ – 74 mm, HCO₃ – 28.3 mmol/L. Intra-operative blood loss was 300 ml and urine output was 1200 ml. The patient received 500 ml of Ringer’s lactate during the procedure.

Post-CRT:

After the CRT procedure, the patient was moved to the Intensive Care Unit (ICU). Hemodynamic and ventilatory issues identified in the ICU were:

- HR 130/min, sinus rhythm, BP was 84/50 mmHg on dopamine 14 mcg/kg/min, MAP was 61 mmHg
- Severe hypoxia. Volume-controlled ventilation, tidal volume - 500 ml, respiratory rate – 18/min, needing PEEP of 12 cm with FiO₂ of 0.7, peak inspiratory pressure reaching 41 cm with plateau pressure of 32 cm
- CVP was persistently in the 18 to 20 mmHg range
- 1000 ml negative fluid balance while in operating room

At this point, our therapeutic dilemmas were:

1. If we give fluids to correct negative fluid balance, there was risk of aggravation of pulmonary edema, as the patient was already extremely hypoxic. Secondly what would be our goal or target for fluid therapy?
2. If we escalate dopamine to achieve higher perfusion pressure, there is risk of worsening of tachycardia and further deterioration of cardiac function, plus the risk of high-dose dopamine in terms of renal hypoperfusion and subsequent renal impairment.
3. To achieve afterload reduction and improvement in cardiogenic pulmonary edema, nitroglycerin or nitroprusside could not be given, as the BP was very low (same concern about dobutamine).
4. At such low MAP, would diuresis have an effect?

We decided to monitor cardiac output, cardiac index, systemic vascular resistance index (SVRI) and stroke volume variation (SVV) for better hemodynamic monitoring to get answers to these questions:

1. Is cardiac output adequate?
2. What is peripheral vascular resistance?
3. Is this shock fluid-responsive?

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A radial arterial line was connected to the FloTrac system and hemodynamic variables displayed on the Vigileo monitor (see table). There were no arrhythmias. The patient was under deep sedation and paralysis on controlled mechanical ventilation. We obtained repeated SVV values by making Vt 8 ml/kg. SVV was between 20% and 22%.

Hemodynamic Monitoring Values:	
CO	4.7 l/min
CI	2.6 l/min/m ²
SVV	20%
SVRI	1261

We felt that this information offered significant and fairly reliable evidence of preload dependency of this hemodynamic insufficiency. Therefore, we administered the first fluid bolus of 250 ml of 0.9% isotonic NaCl in 30 minutes, which resulted in marginal improvement in CO to 5 l/min, and MAP increased to 66 mm. Fluid boluses were continued until SVV dropped to < 15% on controlled ventilation. A total of 2450 ml fluids were given in the 14 hours post-operatively. MAP improved with fluid boluses and dopamine was rapidly tapered to 5 mcg/kg/min at the end of 6 hours in the ICU. HR settled to 100/min. Dobutamine was started at 5 mcg/kg/min to improve cardiac output and nitroglycerin was administered. Once hemodynamic stability was achieved, 40 mg of furosemide was given. The patient’s ventilatory requirements reduced rapidly and he was extubated 24 hours after ICU admission. Dobutamine was continued for the next 24 hours and tapered gradually. Ramipril and digoxin were restarted. BSLs were controlled with insulin infusion. Five days post-procedure, cardiac evaluation via 2D echocardiography and Doppler showed improved LV function to 25%, and PH was marginally reduced. The patient was discharged from the hospital on day 7.

DISCUSSION

We feel that CO, CI and SVV monitoring helped us immensely in improving this patient’s critical hemodynamic condition. If we had monitored only MAP, CVP or PCWP in this kind of acute cardiogenic pulmonary edema plus cardiogenic shock, fluid resuscitation would not have been attempted, and the outcome would likely have been far less successful. Instead, with the indication that the patient would respond positively, we could proceed confidently with fluid replacement. Further, the SVV parameter guided us regarding when to stop administering fluids.

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