e-ISSN (online): 2745-9497

Journal of Anesthesiology and Clinical Research (JACR)

Journal website: https://hmpublisher.com/index.php/jacr

The Use of Inferior Vena Cava Ultrasonography to Assess Fluid Overload in Acute Lung Oedema in Severe Preeclampsia Patient

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ARTICLE INFO

Keywords:
Fluid overload
Inferior vena cava
Intensive care unit
Pulmonary oedema
Preeclampsia

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/jacr.v5i1.410

A B S T R A C T

I**ntroduction:** Accurate evaluation of volume status is essential for appropriate therapy because inadequate assessment of volume status can result in unnecessary administration of therapy, which can increase mortality. This study aimed to describe the use of inferior vena cava ultrasound in assessing fluid overload in severe preeclampsia patients. **Case presentation:** We report a 23-year-old female patient with a diagnosis of acute pulmonary oedema, and severe preeclampsia at G1P0A0 40 weeks gestation with complaints of shortness of breath. On physical examination, the patient appeared short of breath with RR 32 x/minute, fine wet crackles in both lung fields, SpO_2 92% with NRM 15 L/m, HR 160 x/minute, lifting strength, CRT < 2 seconds, blood pressure 160/120 mmHg. This patient underwent emergency termination of pregnancy by C-section under general anesthesia rapid sequence induction followed by intensive care in the ICU. Management in the ICU this patient was given mechanical ventilation, midazolam sedation 0.05 mg/kg, analgesic morphine 10 mcg/kg, fluid restriction with a fluid balance target of (-)1000 ml/24 hours and given furosemide 10 mg/hour to reduce fluid overload so that fluid in the lungs can be reduced. Evaluation of fluid overload by IVC ultrasound. **Conclusion:** Acute pulmonary edema requires proper management to get a good outcome. Measurement of the diameter of the inferior vena cava (IVC) can also be used to assess fluid volume status. Lack of volume is assessed with an IVC diameter of <1.5 cm, while an IVC diameter of >2.5 cm indicates volume overload.

1. Introduction

Pulmonary edema is a major health problem accounting for approximately 10% of intensive care unit (ICU) admissions and is associated with an estimated acute hospital mortality of between 10% and 25% and one-year mortality exceeding 40%.[1] The pulmonary interstitium and, ultimately, the alveoli are the sites of intravascular fluid leakage into the lungs, which results in pulmonary edema. Acute pulmonary edema can be caused by increased capillary pressure (hydrostatic or cardiogenic pulmonary edema) or by increased capillary permeability.

Pulmonary edema usually presents as bilateral, symmetric perihilar opacities on chest radiographs. This "butterfly" appearance is more commonly seen with increased capillary pressure than with increased capillary permeability. An air bronchogram shows increased permeability for pulmonary edema.[2-4] Accurate evaluation of volume status is essential for appropriate therapy because inadequate assessment of volume status can result in unnecessary administration of therapy, which can increase mortality. There are several methods for evaluating fluid status. Following are several inspection methods for evaluating volume status: physical examination,

chest X-ray, BNP, chest ultrasound, and inferior vena cava (IVC) ultrasound.[5,6] This study aimed to describe the use of inferior vena cava ultrasound in assessing fluid overload in severe preeclampsia patients.

2. Case presentation

A 23-year-old woman with G1P0A0 at 40 weeks of gestation came with complaints of shortness of breath 4 days before admission to the hospital, and it was getting worse. On physical examination, we found the patient was short of breath with a respiratory rate of 32 times per minute, smooth ronchi (+/+), SpO_2 92% with non-breathing oxygen face mask 15 L per minute, heart rate 160 times per minute, lifting strength, CRT < 2 seconds, blood pressure 160/120 mmHg and GCS 15. Neurologic deficit and seizure were negative. Laboratory evaluation is presented in Table 1. The patient was diagnosed with acute pulmonary oedema in pregnancy with severe preeclampsia with ASA IVE. Then, the patient underwent emergency pregnancy termination by c-section under general rapid anesthesia with sequence induction (GARSI) followed by intensive treatment in the ICU.

Table 1. Laboratory evaluation of the patient before surgery.

Items	Results	Items	Results
Hemoglobin	8.1 g/dl	LDH	931 u/l
Hematocrit	23%	Ureum	17 mg/dl
Leucocyte	17.600/mm	Creatinine	0.8 mg/dl
Thrombocyte	290.000/μL	Prothrombin time	12.5 second
Erythrocyte	$4x10^6$ /μL	APTT	28.4 second
Blood glucose	86 g/dl	INR	0.96
SGOT	34 u/L	Natrium	130 mmol/L
SGPT	12 u/L	Potassium	3.8 mmol/L
Urine protein	+3	Chloride	107 mmol/L

In intensive care unit, patient was installed with endothracheal tube (ETT) 6.5 level 21 cm with synchronized intermittent mandatory ventilation (SIMV) mode VC f-14 Vt 320 PEEP 5.0 FiO_2 50% with outcome Vt 300-330, ftot 22, MV 6 SpO_2 98-99%, chest expansion right = left, vesicular breath sound (+/+), smooth rhonchi (+/+), HR 100x/minute, blood pressure 121/61 mmHg, urine output > 1 ml/kg/hour with fluid balance (-)760 ml/24 hours. Laboratory evaluation revealed hemoglobin level 7,8 g/dL, Ht 26%, leucocyte 39.200/mm, thrombocyte 219.000/μL, erythrocyte $3,55x10^6$/μL, blood glucose 83 g/dL, albumin 3.3, natrium 132 mmol/L, potassium 5.0 mmol /L, and calcium 0.97 mmol/L. Echocardiography examination showed fluid overload, left ventricle systolic failure and right ventricle. On chest X-ray examination showed perihilar haziness and infiltrates in the lungs accompanied by air bronchograms in both lung fields, enlarged cast with cardio-thorax ratio (CTR) 68% and pulmonary oedema. The patient was treated with IVFD ringer-lactate 10 mL/hour, head up 30^o, enteral diet per sonde 6x200 mL, morphine injection 10 mcg/kg/hour, midazolam 4 mg/hour, furosemide 10 mg/hour, packed red cell (PRC) transfusion 1 colf, plasmin 25% 100 mL, ranitidine intravenous 50 mg/12 hr, ampicillin sulbactam intravenous 1.5 gr/8 hour, metronidazole intravenous 500 mg/8 hours, paracetamol injection 1 gr/8 hours, $MgSO_4$ 1 gr/hour for 24 hours, methyldopa 500 mg/8 hours, nifedipine 10 mg/8 hours, vitamin C 50 mg/12 hours. Figure 1 show ultrasound examination on day 1 of ICU treatment.

The second day of treatment, the patient's condition was getting better with the airway still in ETT with mechanical ventilation SIMV mode f-14, Vt 320, PEEP 5.0, FiO_2 50%, with outcome Vt 300-330, ftot 22, MV 6 SpO_2 98 - 99%, chest expansion right = left, vesicular breath sound (+/+), smooth rhonchi (-/-), HR 92 x/minute, blood pressure 111/64 mmHg, urine output > 1 ml/kg/hour with fluid balance (-)2370 ml/24 hours, $E_4M_6V_{ETT}$ consciousness. On ultrasound examination, IVC also showed improvement with a diameter of about 2 cm and collapsibility > 50% (Figure 2). Chest X-ray revealed

cardiomegaly with pulmonary edema. Compared with the previous photo, the impression infiltrate is reduced (Figure 3). Morphine, midazolam, and $MgSO_4$ therapy were discontinued.

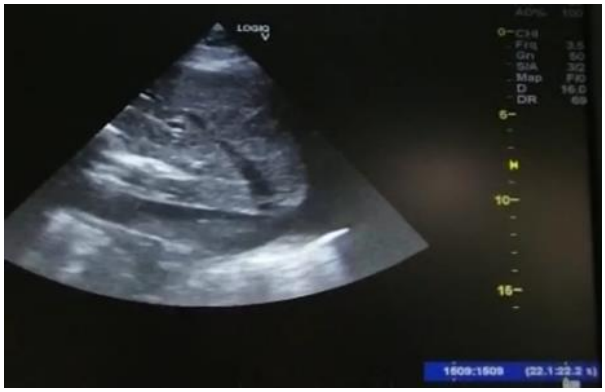

Figure 1. First-day IVC ultrasound. Maximum diameter 2,5 cm, minimum diameter 2,4 cm.

Figure 2. Second-day IVC ultrasound, diameter + 2 cm, collapsibility > 50%.

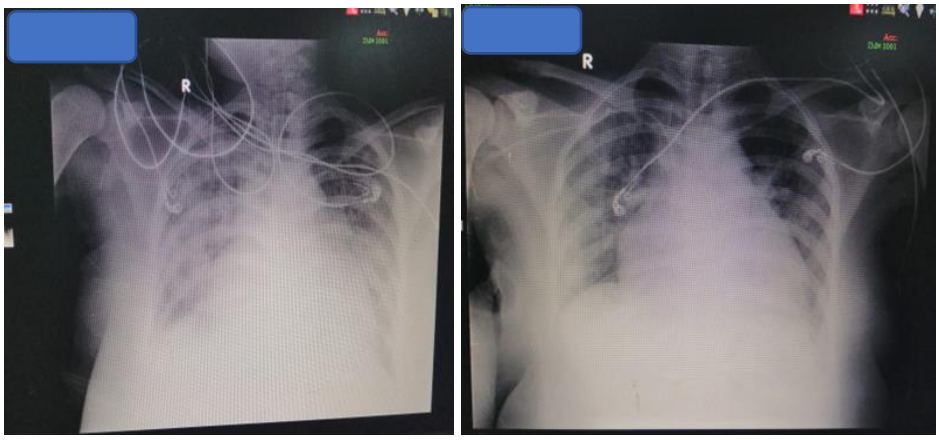

Figure 3. Comparison of chest radiographs day-1(left) and day-2 (right).

On the third day of treatment in the ICU, the patient was extubated. The patient's condition was good, fully conscious with GCS $E_4M_6V_5$, spontaneous breathing, RR 18 x/minute, SpO_2 99% with O_2 nasal cannula 2 L/minute, chest expansion right = left, vesicular breath sound (+ /+), smooth rhonchi (-/-), HR 86 x/minute, blood pressure 132/74 mmHg, urine output > 1 ml/kg/hour with fluid balance (-)1307 mL/24 hours. Laboratory evaluation on day 3 was Hb 8.4 g/dL, Ht 27 %, leucocyte 19.600/mm, thrombocyte 182.000/µL, erythrocyte $3,53x10^6$ million/µL, blood glucose 83 g/dL, natrium 138 mmol/L, potassium 3.1 mmol/L, chloride 97 mmol/L, calcium 0.97 mmol/L. On IVC ultrasound

examination, IVC diameter less than 1,5 cm and collapsibility more than 50%. The patient was given IVFD ringer lactate 10 cc/hour, head up 30o, soft diet 1500 kcal, ranitidine intravenous 50 mg/12 hours, ampicillin sulbactam intravenous 1.5 gr/8 hours, metronidazole 500 mg/8 hours, paracetamol injection 1 gr/8 hours, furosemide 20 mg/12 hours, methyldopa 500 mg/8 hours, nifedipine 10 mg/8 hours, vitamin C 50 mg/12 hours, KSR 1 tablet/8 hours, PRC transfusion 1 colf and the patient was transferred to a regular ward.

Figure 4. Ultrasound examination on the third day. IVC diameter (+)1.5 cm, collapsibility > 50%.

3. Discussion

Pulmonary edema (PE) is caused by leakage of intravascular fluid into the pulmonary interstitium and eventually into the alveoli. Acute pulmonary edema can be caused by increased capillary pressure (hydrostatic or cardiogenic pulmonary edema) or by increased capillary permeability. Cardiogenic pulmonary edema is characterized by dyspnea, tachypnea, and signs of sympathetic nervous system activation (hypertension, tachycardia, diaphoresis) that are often more pronounced than that seen in patients with increased permeability (noncardiogenic) pulmonary edema. In this patient, the presence of dyspnea, tachypnea, hypertension, and tachycardia supports the presence of cardiogenic pulmonary edema. Pulmonary edema is one of the signs of severe pre-eclampsia. After surgery, the patient was admitted to the ICU for further management of acute pulmonary edema and other possible complications. Mechanical ventilation with intubation has become the standard of care for severe respiratory failure. Mechanical ventilation was adjusted according to the protocol for protective pulmonary ventilation.[7-10]

The usefulness of the inferior vena cava collapsibility index (IVC-CI) as a substitute measure of volume status is still a topic of discussion in the literature. The inferior vena cava (IVC) is very flexible, so its size and movement change depending on the central venous pressure (CVP). When there is no blockage in the vena cava, CVP is the same as right atrial pressure (RAP). When you breathe normally, the pressure in your chest drops. This lets more blood flow into your right atrium, which then makes the inferior vena cava smaller. When the central venous pressure (CVP) rises, like when you breathe out or when your body has a positive fluid balance, the flow of blood in the vena cava slows down, and the diameter of the inferior vena cava (IVC) gets bigger. An invasive monitoring method that involves putting in a central venous catheter is generally thought to be the best way to test RAP. However, ultrasound (US) provides a non-invasive and more readily available alternative. Several studies have shown a strong association between the collapsibility of the inferior vena cava (IVC) and right atrial pressure (RAP). The diagnostic precision of IVC-CI is enhanced by employing predetermined threshold values that identify RAP as either high or low. Nevertheless, it is still uncertain whether IVC-CI can accurately forecast fluid response in hypovolemic patients.[11-16] Further studies in a large population should be investigated for the accuracy of these diagnostic methods.

4. Conclusion

Acute pulmonary edema requires proper management to get a good outcome. Ultrasound measurement of IVC can be used to assess fluid volume status in pulmonary oedema. Measurement of the diameter of the inferior vena cava (IVC) can also be used to assess fluid volume status.

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