MANAGEMENT OF PERIOPERATIVE PULMONARY EDEMA IN PATIENT WITH PRE-ECLAMPSIA/ECLAMPSIA UNDERGOING C-SECTION

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ABSTRACT

Retrospective analysis of the medical record in the year 2003, found twelve cases of perioperative pulmonary edema in patients with pre-eclampsia/eclampsia undergoing C-section. The patient was admitted with respiratory distress, and was treated with oxygen administration, diuretics, digitalis glycosides, fluid restriction and intermittent positive pressure ventilation. Six patients were given artificial ventilation for twenty four to forty eight hours. Eight patients were managed under negative fluid balance on the first day. Two patients died in the second week after C section. Both were multigravidae (fourth and ninth) and aged thirty-five and forty year old.

***Keywords**: anesthesia, pre-eclampsia, pulmonary edema, Cesarean Section*

INTRODUCTION

Preeclampsia is associated with reduction in intravascular volume, haemoconcentration and hypoproteinenia. However, pulmonary edema occurs in some cases and therefore raises controversy on the correct fluid therapy. Pulmonary edema is significant cause of maternal and perinatal morbidity and mortality in one large series. Seventy percent of the cases occurred post partum (Sibai BM et al, 1987). The incidence of pulmonary edema was also higher in older multigravida and in those with chronic hypertension. Pulmonary edema occurs as a result of (a) a low COP in association with increased intravascular hydrostatic pressure, and (b) increased capillary permeability (Sibai BM et al, 1987).

Pulmonary edema may be due to left ventricular dysfunction secondary to high systemic vascular resistance, iatrogenic volume overload in the face of contracted intravascular space, decreased plasma colloid oncotic pressure (occurs in normal pregnancy and is exaggerated in preeclampsia), or pulmonary capillary membrane injury. Colloid oncotic pressure may decrease further following intravenous fluid replacement with crystalloid and as a result of rapid intravascular mobilization of edema fluid after delivery. Management consists of diuretics and oxygen, with digitalis glycosides reserved for rare patient with evidence of left ventricular dysfunction.

MATERIALS AND METHODS

A retrospective study was done on perioperative pulmonary edema patients with pre-eclampsia/eclampsia undergoing C-section in year 2003, at Dr Soetomo Hospital Surabaya. Data reviewed included all treatment modalities from preoperative extending to post operative intensive care till discharge.

RESULT

Patient Characteristics

Clinical demographic characteristics of the patients were listed on Table 1.

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Table 1. Clinical demography

			Mother condition				Fetal
Patient	Age	Gravidae	Diagnosis	BP	HR	RR	Condition
1	35	V	Severe pre-eclampsia	200/125	145	38	Distress
2	25	I	Eclampsia	190/130	150	36	Distress
3	19	I	Eclampsia	145/74	120		Distress
4	40	IX	Severe pre-eclampsia	176/113	139		Distress
5	39	VI	Severe pre-eclampsia	154/95	138	36	Distress
6	18	I	Eclampsia	170/115	140	36	Distress
7	18	I	Eclampsia	160/100	109	32	
8	16	I	Eclampsia	146/94	160	30	
9	25	I	Eclampsia	139/50	150	30	Distress
10	21	I	Severe pre-eclampsia	240/120	153	40	Distress
11	35	IV	Severe pre-eclampsia	144/100	113	40	
12	35	IV	Severe pre-eclampsia	175/110	140	36	

Preoperative problems

All twelve patients were admitted at the resuscitation unit of emergency department Dr Soetomo Hospital with respiratory distress and altered consciousness but with Glasgow Coma Score > 10. Eight patients were given artificial ventilation using 100% oxygen, diuretics and digitalis. Four patients were given 100% oxygen by mask, diuretics and digitalis. The diagnosis of six patients was eclampsia while the other six were severe pre eclampsia. Patient number 1, 2, 4, 10 and 12, had blood pressure over 170 mmHg and were given anti hypertensive agent. In all cases were not unconscious, the Glasgow Coma Scale were over than 10.

Anesthesia problems

High risk patients needed intensive preparation before the induction of anesthesia intended to save the mother and the baby. The preparation of three patients took less than one hour, in seven patients between one and two hours and in two patients more than two hours. Eight patients were intubated at the resuscitation unit using thiopenthal, muscle relaxant and narcotic. The other four patients were intubated during induction of anesthesia. Two patients were given intravenous lidocaine 2% and fentanyl before induction because the blood pressures were over than 170 mmHg. Anesthesia were maintained with 100% oxygen and 1.0-1.5% isoflurane. Morphine and additional muscle relaxant were given after the baby was born. Oxytocin infusion was started immediately after delivery.

Problem of the neonates

First minute evaluation showed that one was vigorous while ten babies were in severe asphyxia and five in mild asphyxia consequently. After resuscitation, evaluation at five minutes showed improvements. Five babies had become vigorous, while three others were still in asphyxia. Four cases were twins. Thirteen babies weighed less than 2500gm.

Postoperative problems

Artificial ventilation were continued post operatively in the intensive care unit. One patient required less than twenty four hours, six patients (50%) betwen required between 24 and 48 hours and two patients (16.67%) were ventilated for 96 hours. Fluid balance monitoring were continued untill the signs of pulmonary edema subsided. Negative balance on the first day occured in nine patients, on the second day in six patients, on the third day in five patients, on the fourth day in one patient and on the fifth day in one patient. Patients number 5 and 12 died in the second week due to ARDS and sepsis. They were multigravidae (fourth and ninth) and aged thirty-five and forty year old. Ten patients recovered well and were discharged within two to three weeks.

DISCUSSION

Pulmonary edema may occur as a result of left ventricular dysfunction secondary to high systemic vascular resistance, iatrogenic volume overload in the face of contracted intravascular space, decreased plasma colloid oncotic pressure (occurs in normal pregnancy and is exaggerated in preeclampsia), or pulmonary capillary membrane injury (Sibai BM et al, 1987; Beall M, 2002). Pulmonary edema normally occurs at a PCWP of 20 to 25 mm Hg, but if the patient also has a low COP, clinical symptoms may manifest earlier. Pre-eclampsia lowers COP, but correction of COP with antepartum albumin produces much higher filling pressures after delivery. These women then often need diuretic therapy (Sibai BM et al, 1987). In severe preeclampsia plasma albumin is reduced as a result of loss in urine and across the leaky capillaries; consequently, colloid osmotic pressure (COP) is reduced. After delivery COP normally decreases as a result of fluid shift; typically in normotensive patients at 16-18 hours post delivery COP may be in the order of 16.2 mm Hg while in pre eclampstic patient at this time, COP can be as low as 13.8 mmHg (Mushambi MC et al, 1996; Hawkiens JL, 1993).

Pulmonary edema is not uncommon in preeclampsia. Sibai found incidence of 2.9% in preeclampsia and eclampsia; 70% of the cases developed pulmonary edema 71 h after delivery (Mushambi MC et al, 1996). It occurred more commonly in association with multiple organ dysfunctions than as an isolated complication. Increased hydrostatic pressure in pulmonary capillaries may occur as a result of left heart dysfunction, iatrogenic fluid overload and post partum mobilization of extra vascular fluid. The most common cause of pulmonary edema was alteration in hydrostatic-oncotic forces that occurred within 15 hours postpartum and no patient had left ventricular dysfunction (Hawkiens JL, 1993). The use of colloids without adequate monitoring may increase the risk of pulmonary edema. The pathology underlying increased capillary permeability is not clear but it is probably due to endothelial damage. In this situation pulmonary edema may be precipitated by acute fluid administration, amniotic fluid emboli or sepsis (Mushambi MC et al, 1996). and may lead to adult respiratory distress syndrome (ARDS).

The crystalloid-colloid controversy is still debated in the management of preeclamptic patients because most cases had mixed conditions of low COP and leaky capillaries which tended to develope non-cardiogenic pulmonary edema (Mushambi MC et al, 1996). Infusion of crystalloid alone decreases oncotic pressure further, while the use of colloid such as albumin may result in high CVP and PAWP values. If pulmonary edema develops, its treatment includes oxygen, diuretics, fluid restriction to achieve reduction of preload and after load, and intermittent positive pressure ventilation (Mushambi MC et al, 1996; Hawkiens JL, 1993). After C-section all patients had fluid restriction. Negative fluid balance were achieved in this series (Figure 1); on the first day occured in nine patients, on the second day in six patients, on the third day in five patients, on the fourth day in one patient and on the fifth day in one patient.

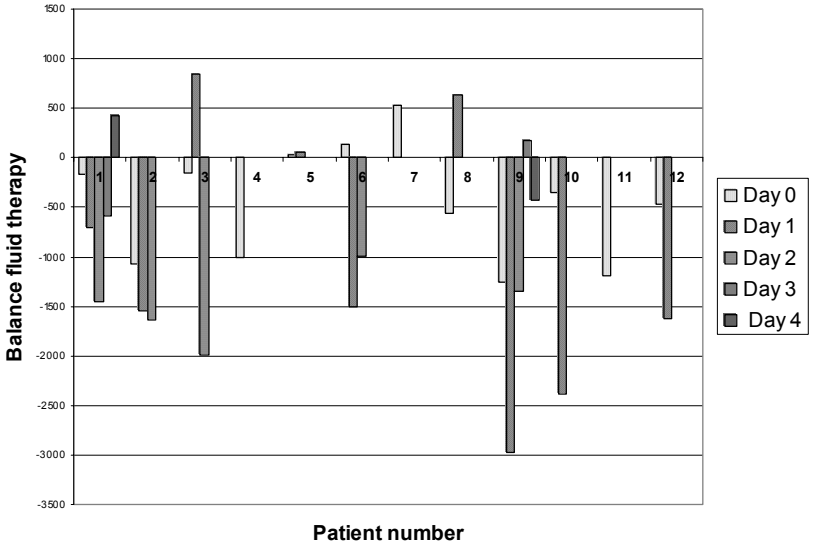


Figure 1. Balance fluid therapy after C section patient with pulmonary edema and pre-eclampsia/eclampsia

Eight patients were treated with artificial ventilation using 100% oxygen, diuretics and digitalis. Four patients were given by 100% oxygen mask. After C-section, artificial ventilation was continued post operatively in intensive care unit. One patient required less than twenty four hours, 6 patients betwen 24 and 48 hours and five for up to 96 hours (Figure 2).

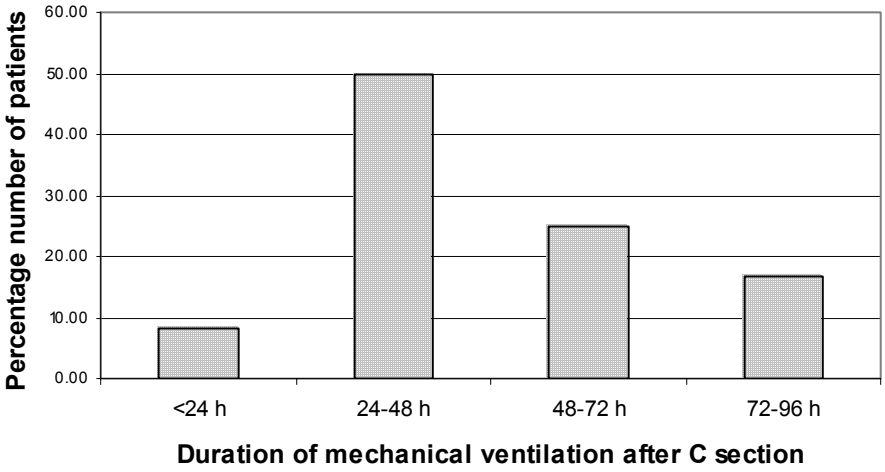


Figure 2. Duration of mechanical ventilation after C-section patient with pulmonary edema and pre-eclampsia/eclampsia

As maternal oxygen reserve is decreased in pregnancy, significant arterial desaturation will occur if the patient becomes apneic for even a short time. Such episode increases the hypoxic risk to the fetus as well. Mechanical ventilator should be adjusted to maintain the PCO_2 in the range of 30 to 32 torr. Available data on permissive hypercapnic ventilation in pregnant patients is limited, although maternal PCO_2 up to 60 torr has not been shown to be detrimental to the fetus.

CONCLUSION

A multi modal treatments which ensure an optimal oxygenation and stable hemodynamics with the lowest amount of fluid seemed to be justifiable. In the absence of complete understanding of the pathogenesis of the pulmonary edema, to reduce maternal and fetal complications, management is based mainly on treatment of symptoms and signs of the secondary effects of pre eclampsia. The management of these patients should ideally be multidisciplinary and the anesthetist should be involved in the care of patients from the early stage.

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