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Case Report: Maternal Mortality Care/3 Overdue 4 Overly

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INTRODUCTION

Preeclampsia is a hypertensive condition that occurs in pregnant women whose gestational age has reached 20 weeks or after delivery who previously had normal blood pressure and increased to 140/90 mmHg or more[1]. Preeclampsia condition is a vasospastic disease that involves many systems and is characterized by hemoconcentration, hypertension, and proteinuria >300 mg/24 hours[2]. These symptoms can be seen during pregnancy, childbirth and in the puerperium which can be accompanied by convulsions and coma[3]. Preeclampsia is one of the causes of maternal and perinatal mortality, and can be detected at gestational age >34 weeks with blood pressure < 140 /110 mmHg, then the patient may have preeclampsia[4].

Currently, the prevalence of the Maternal Mortality Rate (MMR) in Indonesia is still high, which is around 220 out of 100,000 live births. The high mortality rate in pregnant women can be caused by many factors, one of which is due to preeclampsia in pregnant women with an incidence prevalence of 5%-15% of all pregnancies[5]. The etiology of preeclampsia is unknown but is believed to be influenced by several factors, such as age (less than 16 years or over 45 years), a history of maternal hypertension before pregnancy, obesity during pregnancy, multiple pregnancies (two fetuses), and a family history of preeclampsia[6].

Severity of pre-eclampsia symptoms is characterized by blood pleasure systolic > 160 mmHg or diastolic > 110 mmHg, protein product above 300 mg per 24 hour with proteinuria 1+, eclampsia, hemolysis, abnormal fetal growth, obstruction, pulmonary edema, and oliguria. Proteinuria and hypertension are the most dominant clinical features seen in cases of preeclampsia because the kidneys fail to function, while seizures in patients with severe preeclampsia can increase the MMR and cause fetal death due to circulatory collapse[7].

CASE REPORT

The patient, Mrs. Y, 30 years old, came to the ER at 8:00 a.m. with a complaint of amniotic fluid coming out 1 hour before being admitted to the hospital. The patient was referred from the *Puskesmas* with a certificate of severe preeclampsia with blood pressure of 160/130 mmHg and had been given nifedipine 10 mg sublingually. The patient admitted that she was 9 months pregnant. Antenatal care (ANC) with the midwife was done regularly every month. Ultrasound examination was done at the hospital with live intrauterine single head presentation, biometry DBP 9.1 cm, FL 6.6 cm, LP 32.5 cm, TBJ 3000 grams with ICA 15.9, with placenta implanted in the back fundus. The pregnancy assessment was 36-37 weeks and there were no major congenital abnormalities. During ANC there was no high blood pressure during pregnancy. History of asthma, heart disease, hypertension before pregnancy, as well as diabetes in the patient and family were denied. This was Mrs. N first pregnancy.

About 4 hours earlier the patient complained of pain and nausea in her stomach. There was no nausea, vomiting, heartburn, blurred vision, headaches or complaints of shortness of breath. General status examination was obtained, consciousness was compos mentis, blood pressure 140/100 mmHg, pulse rate 108 times/minute, respiratory rate 24 times/minute, and temperature 37 C. Conjunctiva was not pale, and sclera was not icteric. Cardiac and pulmonary examinations were within normal limits. There was no edema in the extremities.

On examination of obstetrical status, the uterine fundal height was 33 cm, and the estimated fetal weight was 3300 grams. Fetal heart sound was 144 beats/minute. On internal inspection: the vulva and urethra were calm, while closer inspection found that some portions were smooth, and clear amniotic fluid was seeping. Other results showed the Leukocyte Esterase / LEA (-), in vaginal touché/ VT examination, portio spongy, axial, 1 cm thick and there was

no cervical dilatation, with head of fetus on H I-II (pelvic score: 2).

Clinical pelvimetry examination found: promontory not palpable, belly circumferential measurement was -½, side wall straight, sacrum concave, spine not protruding, with Interspenosum Diameter (ID) > 9.5 cm, Angle Pubic (AP) > 90, moderate normal pelvic, and Feto Pelvic draw in good condition. On examination, stick protein was found (++). Ultrasound examination (USG) results were: live single head presentation fetus, while the fetal biometry showed Biparietal Diameter (BPD) 9.3 cm; Head Circumferential (HC): 30.6 cm; Abdominal Circumferential (AC): 34.8cm; and Femur Length (FL) 6.6 cm. Estimated fetal weight (EFW) was 3,450 grams, while the amniotic fluid Index (AFI) : 8. Placenta implants indicated the fundus during pregnancy at term, with cephalic presentation fetus. In this patient, G1 problems were found at 39 weeks pregnant with a live single head presentation, and ruptured membranes one hour before delivery (pelvic score : 2) with severe preeclampsia.

The patient underwent supporting examinations, including complete peripheral blood, urinalysis, temporary blood sugar, SGOT, SGPT, urea, creatinine, uric acid and LDH. As well as CTG, EKG, cardiology and eye consultations.

The patient was scheduled for vaginal delivery and observed for signs of intrapartum infection, cord compression and worsening of severe preeclampsia. Cervical ripening was performed with 25 ug misoprostol repeated 6 hours if the cervix was immature. The patient received severe eclampsia treatment, with $MgSO_4$ 4 gram bolus, followed by $MgSO_4$ 1 gram/hour, with nifedipine 10 mg titrated until the target of 20% MAP was reached, and N-acetylcysteine 3 x 600 mg and vitamin C 2 x 400 mg. Antibiotic ampicillin sulbactam 1.5 grams was given intravenously then orally 2 x 375 mg.

The patient was given an explanation that this time she had preeclampsia with ruptured membranes, so the delivery had to be in the hospital under close supervision. The patient was started with a maintenance doses of $MgSO_4$ infusion of 6 grams in 500cc RL at 8:00 a.m. Laboratory results showed proteinuria (+2) and hypoalbuminemia (3.2 g/dl) and there was no deterioration from other laboratories (see Table 1).

Table 1. Results of laboratory tests

Hb/Ht/ Leukocyte/Thrombocyte	13.6/ 40/ 9100/ 222,000 per ml
Urinalysis	Protein (+2)
Ureum/Creatinine	17/0,9
SGOT/SGPT	29/15
Blood sugar	79
Gout	5,0
Albumin	3,2
LDH	500

Results of consultation with the eye department showed in this patient, there were no signs of ODS hypertensive retinopathy. The results of consultations with the cardiology department recommended to give ISDN 5 mg sublingual antihypertensive every 5 minutes until the target blood pressure 140/90 mmHg to be given a maximum of 3 times, followed by maintenance with 3 x 250 mg methyldopa, titrated up to 3 x 500 mg.

Misoprostol I (25 ug) was installed at 10:00 a.m. and re-assessment was planned at 4:00 p.m. Observations in the first 6 hours obtained systolic blood pressure ranging from 140-160 mmHg and diastolic blood pressure of 100 mmHg. The pulse rate ranged from 98 to 106 beats per minute. 6 hours after administration of misoprostol I, her blood pressure at that time was 140/90 mmHg, respiratory rate 24 times/minute, and pulse rate 100 beats/minute. His is not present, while fetal heart rate was 140 bpm, and re-examination obtained pelvic score 3. The patient continued receiving the first $MgSO_4$ and cervical ripening with misoprostol II (25ug) and she was reassessed at 10:00 p.m. The patient received an injection of antibiotics at 6:00 p.m. Observation for the next 6 hours obtained blood pressure was still140/90 mmHg. The pulse rate is 98-100 beats per minute. The patient finished $MgSO_4$ I and continued with $MgSO_4$ II.

At 10:00 p.m., the pelvic score was reassessed 4, and she received misoprostol III with an increased dose of 50 g. Planned re-assessment was at 4:00 a.m. At 4:00 a.m., the pelvic score was 4, followed by IV misoprostol at a dose of 50 g and a re-assessment was planned at 10:00 a.m. At 10:00 a.m. $MgSO_4$ II was finished. She complained of heartburn often, and obtained his 2 times in 10 minutes, moderate strength, good relaxation, with a fetal heart rate of 142 bpm. Internal examination revealed a mature cervix with an opening of 5 cm. This was followed by induction titration of oxytocin 5 IU in 500 cc RL starting at 8 drops per minute, titrating every 30 minutes until an adequate hist was achieved. The $MgSO_4$ infusion was discontinued.

The oxytocin infusion was set at 10:15 a.m. starting at 8 drops per minute. At 11:45 a.m., the adequate his was achieved with 12 drops per minute. Planned reassessment started at 2.15 p.m. and the contractions increased frequently, blood pressure 140/90 mmHg, pulse rate 84 beats/minute, respiratory rate 20 beats/minute, with temperature afebrile. She reached his 3 times in 10 minutes and 40 seconds, was well relaxed with a fetal heart rate of 148 bpm. Check results showed progress in appropriate PK I active with an opening of 7 cm, head on HI-II. Plan for reassessment of labor progress was set at 5:15 p.m.

At 5:15 p.m. the patient had seizures, delirium

consciousness, blood pressure 200/120 mmHg, pulse rate 108 times/minute, respiratory rate 28 times/minute, and the internal examination showed progress to PK II; head at HIII-IV, and UUK front. Analysis indicated signs of eclampsia gravidarum in stage II parturition. A bolus of 4 grams of $MgSO_4$ was administered slowly intravenously and the second stage was accelerated with forceps extraction. At 5:25 p.m. with forceps extraction, a baby girl was born weighing 2,950 grams, US 6/7, with a small amount of greenish amniotic fluid. At that time, the blood pressure measurement was 160/100 mmHg. The mother was injected with oxytocin 10 IU intramuscularly. The placenta was delivered five minutes later complete. Postpartum bleeding was about 500 cc, blood pressure was 100/60 mmHg, pulse rate 120 beats per minute, while contractions were not good (hypotony), and uterine fundus exam obtained 2 fingers above the center. Exploration of the birth canal found an intact portion, with no uterine rupture, and grade II perineal rupture. Analysis indicated signs of hypovolemic shock ec., HPP ec., and uterine hypotonia. Uterine fundus massage was performed, and simultaneously administered uterotonic oxytocin 10 IU intramuscularly and methergine 0.2 mg intramuscularly, with drip oxytocin 20 IU and misoprostol 1,000 ug rectally. At 5:40 p.m. uterine contractions improved. At that time blood pressure was 140/90 mmHg, with pulse rate of 96 beats per minute. Perineorrhaphy was performed.

At 6:00 p.m., HPP happened, with somnolent-delirium consciousness. There were pale conjunctiva, unmeasured blood pressure, and unmeasured pulse rate. Respiratory rate was 18 times per minute and heart rate was 132 beats per minute with cold feet and hands. On obstetric examination the fundus exam of the upper indicated 4 fingers of the center, atony, with vaginal bleeding of about 500 cc. Diuresis was 150 cc concentrated. The perineal wound was sutured. Analysis again indicated signs of irreversible hypovolemic shock ec., HPP ec., with uterine atony. At that time, a repeat DPL examination was done, while 1,000 cc of whole blood and 1,000 cc of PRC were made available, and anesthesia consulted for cito hysterectomy, with ICU/HCU consulted for post-op care. Bimanual compression was performed while preparing to wait for OK. Fluid resuscitation was performed with 1,000 cc crystalloid and 1,000 cc colloid.

After being notified the attending consultants were agreeing to cyto laparotomy in view of irreversible shock ec., with uterine atony, then informed consent was given by the husband. At 6:30 p.m. the patient was brought to the OK with supportive awareness, unmeasured blood pressure and no palpable pulse. She was bradypneic with a respiratory rate of 12 breaths per minute while waiting for preparation until OK (bimanual compression and fluid resuscitation were performed). Laboratory results obtained Hb 5.1 g/dl; Ht 16g%; leukocytes 15,600u/L; platelets 44,000 u/L. and the Susp analysis. DIC.

At 7:30 p.m. a subtotal hysterectomy was performed (blood pressure was not measured, pulse was not palpable; heart rate was 155 bpm. Operation report: after opening the peritoneum, the uterus was atonic, and pale. The blood vessels supplying the uterus were clamped (a. bilateral ovaries and a. bilateral uterine) followed by a subtotal hysterectomy. At 7:45 p.m. the patient went into cardiac and pulmonary arrest, and cardiopulmonary resuscitation was performed by anesthesia. At 7:50 p.m. the patient was declared dead. Analysis of the cause of death in this case was irreversible shock ec. HPP ec., with uterine atony.

DISCUSSION

The patient, Mrs. Y, aged 30 years, underwent a history, complete physical examination, examination of fetal status and supporting examinations. The patient was previously diagnosed with severe preeclampsia with a gestational age of 36-37 weeks and this was the first pregnancy.

This patient was treated with nifedipine 10 mg sublingually to lower her blood pressure at that time and planned vaginal delivery and observed worsening of severe preeclampsia and cervical ripening with 25 ug misoprostol every 6 hours. The patient was given antibiotics IV and then oral 2 x 375 mg. The patient was given an infusion of 6 grams of $MgSO_4$ in 500cc and the antihypertensive ISDN 5 mg sublingually every 5 minutes until the target blood pressure was 140/90 mmHg.

Cervical ripening was continued with misoprostol I (25 ug) and observed for 6 hours so that her blood pressure was 140/90 mmHg. Misoprostol II (25 ug) was placed and observed until misoprostol IV (50 ug) was obtained twice in 10 minutes. Internal examination revealed a mature cervix with an opening of 5 cm. Continued induction titration of oxytocin 5 IU 500 cc RL 8 drops per minute, titrated every 30 minutes until an adequate his was achieved.

After opening 7 cm, the patient had a seizure with blood pressure 200/120 mmHg, pulse rate 108 times/minute, respiratory rate 28 times/minute, internal examination obtained PK II; head in HIII-IV, front UUK and given MgSO4 4 grams IV. Then the second stage was accelerated with forceps extraction and a baby girl with a weight of 2,950 grams was born.

However, the patient's blood pressure was still >160/100 mmHg, then 10 IU IM was given oxytocin then the postpartum hemorrhage was about 500 cc, blood pressure was 100/60 mmHg, and pulse rate was 120 beats per minute. With somnolent-delirium consciousness, pale conjunctiva, unmeasured blood pressure, no palpable pulse rate, and while waiting for OK preparation, 1,000 cc of crystalloid and 1,000 cc of colloid were given and she resuscitated.

Then the patient underwent a subtotal hysterectomy laparotomy (blood pressure had not been measured, pulse was not palpable; and heart rate was 155 bpm). The blood vessels supplying the uterus were clamped. She underwent cardiopulmonary resuscitation by anesthesia after cardiac and pulmonary arrest occurred but the patient was declared dead. Analysis of the cause of death in this case was irreversible shock ec., HPP ec., with uterine atony.

In this case, the cause of the mother's death was due to a history of severe preeclampsia and shock. Severe

preeclampsia is preeclampsia with systolic blood pressure 160 mmHg and diastolic blood pressure 110 mmHg accompanied by proteinuria more than 5gr/24 hours[8]. Management in this case is actively handled, including fluid management by monitoring fluid input from the patient to the hospital with blood pressure >140/120 mmHg which is then observed so that normal delivery can be reached in the patient.

Administration of $MgSO_4$ was done as an anticonvulsant to prevent eclampsia (seizures). Magnesium sulfate is the first choice of anticonvulsant in preeclampsia or eclampsia. Magnesium sulfate will work by inhibiting or decreasing levels of acetylcholine on nerve fiber excitability by inhibiting neuromuscular transmission that requires calcium at the synapse. In the administration of magnesium sulfate, magnesium will shift calcium, so that the flow of stimulation does not occur[9].

Antihypertensives can be given if the systolic/diastolic blood pressure is > 160/110 mmHg and MAP > 125 mmHg and if there is no decrease, it can be given again in 5 minutes until the blood pressure is normal. While in the operating room, the patient's identity is rechecked and a letter of approval for action is re-examined. The patient's vital signs were re-evaluated[10, 11].

Hemorraghic Post Partum (HPP) is bleeding of 500 mL or more from the birth canal in spontaneous vaginal delivery after the third stage (after the placenta is born) or 1,000 mL in caesarean section delivery. However, because of the difficulty of calculating the amount of bleeding, all cases with the amount of bleeding that have the potential to cause hemodynamic disturbances can be referred to as postpartum hemorrhage which can cause death with previous triggers such as preeclampsia[12].

The direct causes of death of pregnant women in Indonesia are often caused by postpartum hemorrhage, hypertension/ eclampsia, and infection. However, the indirect cause of maternal death is that there are still many cases of 3 late and 4 too late which are not realized and known by the public so that there are still many cases of death in pregnant women.

Case 3 is late, including:
1. Delay in recognizing the danger signs of labor and making decisions.
2. Late referral to health facilities.
3. Delayed treatment by health workers in health care facilities.

Case 4 too, includes:
1. Too old to be pregnant (> 35 years)
2. Too young to get pregnant (< 20 years)
3. Too many (number of children > 4)
4. Too close time between births (< 2 years)[13].

The role of family physicians in case reports of maternal deaths with preeclampsia:
1. Provide motivation, counseling and education to patients in an effort to prevent the occurrence of preeclampsia as one of the severe complications of pregnancy.
2. Provide counseling about preeclampsia and provide routine antenatal care for pregnant women to detect preeclampsia early, such as being vulnerable at the age of 31-35 years, heavy work, parity in primigravida, and obesity with body mass index > 30.00.
3. Immediately carry out appropriate handling procedures to prevent bleeding and complications, if there are complications, give first aid and stabilize the patient before being referred, while following up the referral process to ensure effective services in the hospital.

Recommendations for patients with preeclampsia:
1. Measurement of blood pressure in pregnant women should use a mercury sphygmomanometer, and sitting position of the patient with the cuff according to the level of the heart.
2. Determination of proteinuria, which can be enforced urine production of more than 300 mg per 24 hours, and can use a urine dipstick > 1+.
3. Primary prevention of preeclampsia, by screening the risk of developing PE for pregnant women from the beginning of pregnancy.
4. Prevention of secondary preeclampsia: use of low-dose aspirin (75 mg/day).
5. Supplementation of calcium at least 1 g / day, especially at low calcium intake.
6. Use of low-dose aspirin (75 mg/day) and calcium supplements (1g/day), in women at high risk of preeclampsia.
7. For expectative care in severe preeclampsia, where the pregnancy is less than 34 weeks, administration of corticosteroids for fetal lung maturation, and hospitalization during expectant care.
8. For expectative care for preeclampsia without severe symptoms, where gestational age is below 37 weeks, blood pressure evaluation twice a week, platelet evaluation weekly, ultrasound evaluation twice a week, if there is PJT evaluation using Doppler velocimetry.
9. Administration of MgSO4 in severe PE, and administration of antihypertensives in severe hypertension, systolic blood pressure>160mmHG, or diastolic>110mmHg. First choice is short acting oral nifedipine, hydralazine, parenteral labetalol, nitroglycerine, methyldopa.

Prevention for preeclampsia in future pregnancy:
1. Exercise counseling, and be aware of possibility of preeclampsia and eclampsia if there are predisposing factors, such as regular antenatal care check, at least for 4 visits, every trimester (I, II and III) for early screening.
2. Calcium supplementation can decrease the possibility of hypertension in pregnancy with low calcium intake, 1 gram/day minimal recommended for prevention of preeclampsia in woman at high risk of preeclampsia.
3. Mental health counseling are needed because mental health in pregnancy affects how the mother's emotion may impact on hypertension if she having stress so they need counseling.
4. Visit a specialist if necessary because sometime an expert is needed to handle the case.

CONCLUSIONS

Preeclampsia can lead to complications that can lead to death, so pregnant women are recommended to have

regular check-ups with ANC services to detect early if there are complications and get appropriate treatment.

Proper education is given to pregnant women to be able to reduce the incidence of preeclampsia and midwives can prevent it by reducing risk factors by detecting risk factors early, providing counseling to mothers to regulate reproductive age (20-35 years), regulating maternal weight, and doing ANC visits at least 4 times.

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