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Case Report: Intensive Care Management of Preeclampsia and HELLP Syndrome

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Abstract

Introduction: Preeclampsia, eclampsia, and HELLP syndrome are life-threatening conditions in 2-8% of pregnant women and result in 70,000 maternal deaths and 50,000 infant deaths worldwide. Preeclampsia, eclampsia, and HELLP syndrome with organ failure are indications for intensive care in pregnant women. The most important goal of management of patients with preeclampsia is to prevent eclampsia and reduce maternal blood pressure. **Case:** A 35 year old woman with G3P2A0 gravida 29-30 weeks with impending eclampsia who underwent caesarean section. The history revealed complaints of severe headache, blurry vision, heartburn, and a history of high blood pressure during this pregnancy. On the examination of vital signs, the blood pressure was 160/100 mmHg. In laboratory examination, the results of proteinuria (+3) and other results were within normal limits. Preoperative management of intravenous magnesium sulfate, with the oral antihypertensive Methyldopa. Intraoperative general anesthesia was performed, the operation lasted 1 hour, the total bleeding was 250 cc. Postoperatively the patient was transferred to the semi-intensive room (HCU), the patient experienced worsening due to uterine atony. After being resuscitated and intubated, the patient was performed relaparotomy and hysterectomy under general anesthesia. The operation lasts for 2 hours. The patient is then transferred to the intensive care unit (ICU) for close observation. **Conclusion:** Determination of the basic diagnosis and appropriate initial management and prevention of complications in preeclampsia, eclampsia, and HELLP syndrome can reduce the incidence of morbidity and mortality.

Keywords: Anesthesia Management, Sectio Caesarea, Preeclampsia, Eclampsia

INTRODUCTION

Preeclampsia, eclampsia, and HELLP syndrome are life-threatening conditions for expectant mothers and also unborn baby during pregnancy period. Preeclampsia is referred to as pregnancy-related illness and appears in 2-8% of the expectant mother. Worldwide, its mortality rate is about 70.000 and 50.000 for expectant mothers and unborn babies, respectively. Preeclampsia, eclampsia, and HELLP syndrome, when present, usually are indications for intensive care unit admission, especially if there is more than two organs failure presents and if the patient needs mechanical ventilation support.

There are some diagnostic criteria for preeclampsia as follows: (1) systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg occur two times consecutively in between 4 hours of period measurement, (2) proteinuria ≥ 300 mg/day in ≥ 20 weeks of gestation with unknown history of previous hypertension. In some cases, during the course of preeclampsia, proteinuria may not be found in laboratory examination to establish the diagnosis of preeclampsia. However, preeclampsia still can be diagnosed as hypertension accompanied by thrombocytopenia, liver function disturbance, renal insufficiency, pulmonary edema, and central nervous system dysfunction along with new-onset visual disorder. (Lam & Dierking, 2017 ; Jeyabalan, 2014 ; Robert, et al., 2013)

Eclampsia, defined as episodic convulsive symptoms or decrease of consciousness of patient with preeclampsia where all other possible causes of seizure have been excluded. HELLP syndrome is a life-threatening condition which is accompanied by several symptoms as following: the presence of hemolysis, elevated liver enzyme and decreased of platelet count. (Lam & Dierking, 2017)

The purpose of diagnosis establishment and early rapid management of an expectant mother with preeclampsia, eclampsia, and HELLP syndrome is to reduce morbidity and mortality rate. Therefore, the management requires multivarious disciplines, including obstetricians and intensivists. (Lam & Dierking, 2017) The primary goal for patients with preeclampsia is to lower the blood pressure of expectant mothers below 160/110 mmHg. Magnesium sulfate is a drug of choice to prevent eclampsia episode. However, the mechanism of action is unknown, but it is assumed that Magnesium sulfate has an antagonist effect towards calcium ion therefore depressing neurotransmitter release in the neurons. (Kelsey, 2015)

CASE REPORT

Anamnesis

A 35-year-old housewife referred from maternity clinic came to the emergency department of RSHS on July 17th, 2019, with a chief complaint of severe headache from 3 hours prior to hospital arrival. The patient also complained of heartburn symptoms and blurred vision. This expectant mother was on her third pregnancy of 29-30 gestational weeks. She denied having abdominal contraction or vaginal discharges. The fetal movement was within normal.

She has had history of hypertension during pregnancy (160/100 mmHg) and did not take any regular medication. The history of hypertension from previous pregnancy, DMT2, heart problems, and asthmatic episodes were denied.

The last menstruation period was on December 26th, 2018.

Delivery history:

1. Giving birth assisted by midwife, sponatenous delivery of a fully termed male baby, birth weight was 1500 grams, alive
2. Giving birth assisted by Midwife, spontaneous delivery with a fully termed male baby, birth weight was 2500 grams, deceased when he was 7-year-old
3. G3P2A0

Physical examination

Consciousnes	: Compos mentis
Blood Pressure	: 160/100 mmHg
Heart rate	: 90 x/min
Respiratory	: 20x/min
Temperature	: 36.0° C
Fetal heart beat	: 140-144 x/min
Height	: 152 cm
Weight	: 70 kg
Body Mass Index	: 30,3

Head : pink palpebra conjunctiva, anicteric sclera
 Neck : JVP 5 ±2 cmH₂O, unpalpable lymph nodes
 Chest : fully symmetrical chest expansion,
 Vesicular breathing sounds, no ronchi and no rales
 Abdomen : Rounded contour, fundal height corresponding to gestational weeks.
 Ekstremitas : warm extremities, capillary refill time < 2 seconds

Laboratorium examinations

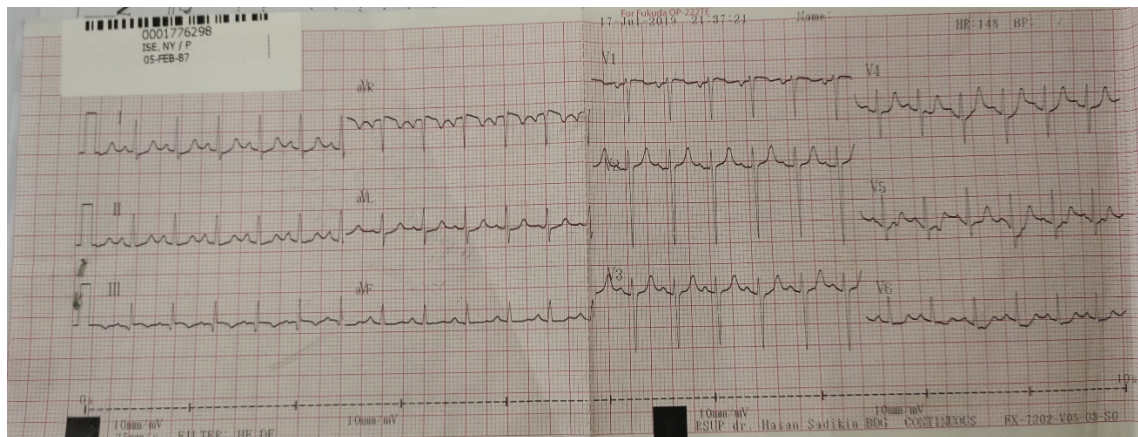
Abdominal Ultrasound (on July 17th, 2019):

Detection of single alive fetal-head with estimation of 29-30 weeks of gestational age, amniotic fluid presents appropriately, SOP 3.3 cm, placenta in the posterior corpus, determined birth weight was 1300 grams.

Laboratory Results

On July 17 th , 2019 at 08.01 am							
Hb	Ht	L	Tr	PT	INR	APTT	Protein Urine
14	41,4	8,86	111.000	11,0	0,81	25,3	+3

EKG



Diagnosis

G3P2A0 Gravida 29-30 weeks gestational age, with impending eclampsia

Early management in the Emergency department

- A thorough observation of general condition, vital signs, and fetal heartbeat regularly
- O₂ supplementation with nasal cannula 3 liters/min
- Pregnancy termination (Sectio Cesarea) as indicated due to impending eclampsia
- MgSO₄ 20% 4 grams in 100ml of Ringer Lactate solution dripped intravenously within 10-15 minutes
- MgSO₄ 20% 10 grams as maintenance dosage in 500ml of Ringer Lactate solution, dripped intravenously 20-30 drops per minutes
- Methyldopa 3x500 mg per-oral
- Informed consent to patient and families

Observation sheet in Emergency department

Time	Contraction	Fetal heartbeat	BP	HR	R	Information
07.10-07.40	-	140-144	160/100	90	20	
07.40-08.10	-	144-148	160/100	90	20	
08.10-09.10	-	148-152	170/100	100	20	
At 9.10 am, the patient was brought to emergency operating theatre						

Morning Shift Emergency Team

Section cesarea procedure was delivered with general anesthesia. The surgery went uneventful about an hour, and bleeding volume was about 250ml totally. Postoperative management was in an intermediate observational room.

Patient's condition at Intermediate Observational Recovery Room

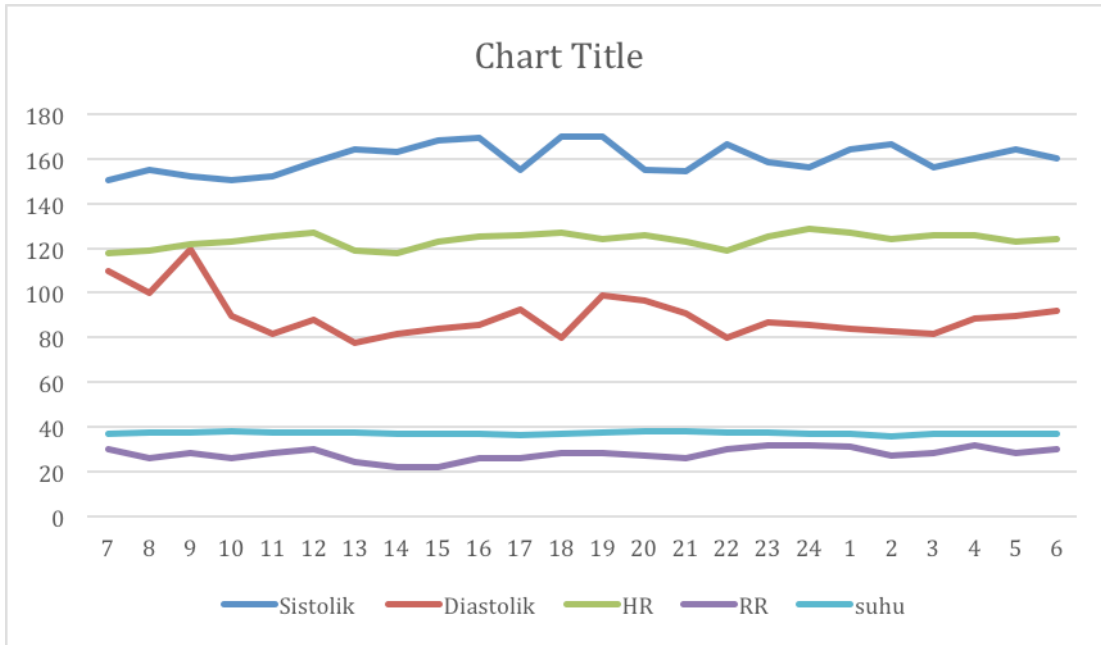
The patient was admitted to an intermediate observational recovery room for about 3 hours. Her condition deteriorated due to atonia uteri with massive vaginal bleeding. She was resuscitated and intubated. She was stabilized and brought immediately to operating theatre for emergency re-laparotomy.

Evening Emergency Shift Team

The patient arrived at the operating theatre at 11.30 pm, then emergency re-laparotomy and hysterectomy were done under general anesthesia. The operation went for about 2 hours and then she was transferred to the intensive care unit postoperatively.

On July 17th, 2019 at 10.36 pm					
Hb	Ht	L	Tr	Amylase	Lipase
5,9	16,9	15.080	85.000	734	4982
SGOT	SGPT	Bil Total	Bil direk	Bil Indir	Albumin
2262	1502	7.871	5.121	2.750	1.10
Ureum	Cr	GDS			
55,3	2,71	127			

Patient monitoring at ICU**Day I (July, 18th 2019)**



OBJECTIVE	ASSESSMENT	PLANNING
<p>CNS Consciousness: under the influence of drugs</p> <p>CVS BP : 132 / 68 mmHg HR : 140 x/min Temp: 36,8 C</p> <p>Respiration RR: 28 x/min CMV/RR 14/TV 400/PEEP 5/FiO2 80% SpO2: 99 %</p> <p>GIT soft non distended, bowel sound (+)</p> <p>GUT UO : 5-20-0 cc/ hour Balance : + 4640 cc/24jam</p>	<p>Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + hypoalbuminemia</p> <p>Lab result 18/7/19 Hb 5,6 Ht 16,4 L 6.050 Tr 39.000 PT 13,1 INR 1,18 APTT 36,1 Hb 10,2 Ht 30,3 L 11.570 Tr 113 PT 22,9 INR 2,0 APTT 45,7 SGOT 2040 SGPT 1651 Bil Total 10,391 Bil direct 8,354 Bil indirect 2,037 Albumin 1,92 Ur 64,8 Cr 3,1 Na 139 K 5,1 Cl 107 Ca 4,8 Mg 2,4 pH 7,226 pCO2 46,7 pO2 110,5 HCO3 19,6 BE -6,5 Sat 93,6</p> <p>Blood culture 18/7/19 Result: not released yet</p> <p>Echohemodynamic : CO 4,8 L/mnt CI 2,82 L/mnt/m2 SV 39 ml/beat IVC max 1,9 IVC min 1,3 IVC Distensibility 46% VTi max 16,25 VTi min 15,4 SVR 1650 Dyne Result: stable hemodynamic with fluid responsive</p>	<p>F: Fasting → initiate feeding test A: Fentanyl 30 mcg/hour, Paracetamol 1gr/6 hours S: Midazolam 3mg/hour T : (-) H: Head up 30° U: Omeprazole 2x40 mg iv G : (-)</p> <p>Th/ Noradrenaline 0,1 mcg/kgBW/min Rocuronium 20 mg/jam Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (1) Furosemid drip 30 mg/hour Nebulization with NaCl 0,9% every 6 hours Transfusion of 4 bags of PRC Transfusion of 4 bags of FFP Ca Gluconas 2 gram iv post-transfusion</p>

On July, 18 th 2019 at 02.37 am						
Hb	Ht	L	Tr	PT	INR	APTT
5,6	16,4	9.320	31.000	16,1	1,48	49.7

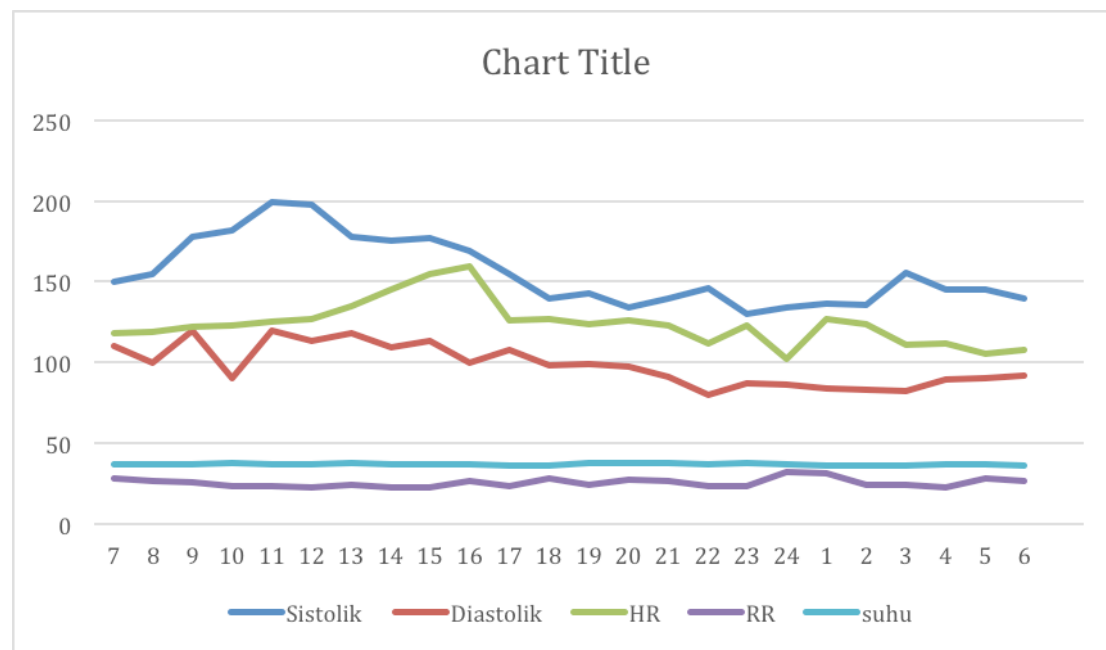
GDS	SGOT	SGPT	Bil Total	Bil Direct	Bil Indirect	Albumin
108	1716	970	6.906	5.532	1.374	1.40
Ur	Cr	Na	K	Cl	Ca	Mg
54	2,77	131	7,7	104	3,75	2,6
Fibrinogen	D dimer					
104,4	3,32					

On July, 18 th 2019 at 10.08 am						
Hb	Ht	L	Tr	PT	INR	APTT
10,2	30,3	11.570	113.000	22,9	2,00	45.7
SGOT	SGPT	Bil Total	Bil Direct	Bil Indirect	Albumin	
2040	1651	10.391	8.354	2.037	1.92	
Ur	Cr	Na	K	Cl	Ca	Mg
64,8	3,10	139	5,1	107	4,80	2,4
pH	pCO2	pO2	HCO3	BE	SpO2	
104,4	3,32	61,3	21,7	-7,8	78,6	

On July, 18 th 2019 at 12.21 pm					
pH	pCO2	pO2	HCO3	BE	SpO2
7,226	46,7	110,5	19,6	-6,5	93,6

On July, 18 th 2019 at 10.49 pm						
Hb	Ht	L	Tr	PT	APTT	INR
5,8	16,4	6.050	39.000	13,1	1,18	36,1

Day 2 (July 19th, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
CNS Level of consciousness: E2M3VT CVS BP: 161/96 mmHg Hr : 115 x/min Temp: 38 C Respiration RR: 20 times/minutes PSIMV/RR 12/PC 16/PS 15/PEEP 5/FiO2 60% (TV 400-550) SpO2: 99 % GIT distention (-), bowel sounds (+) GUT UO: 0-0-8 cc/ hour Balance : - 2092 cc/24hour	Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + electrolyte imbalance 19/7/19 Hb 10,2 Ht 29,5 L 8,33 Tr 56.000 pH 7,279 pCO2 44,0 pO2 54,8 HCO3 20,1 BE -4,8 Sat 74,5 Fibrinogen 232,2 D-dimer 2,53 Laktat 2,3 pH 7,134 pCO2 54,2 pO2 140,3 HCO3 18,4 BE -9,9 Sat 97,6 Blood culture 18/7/19 No results yet	F: Test feeding → gradual liquid diet A: Fentanyl 30 mcg/hour, Paracetamol 1g/6 hour S : - T : - H : Head Up 30 U : Omeprazole 2x40 mg iv G : Th/ Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (2) Furosemid drip 10 mg/hour Nebu NaCl 0,9% per 6 hour Ca gluconate 2 gram iv The first hemodialysis (UF)

July 19 th , 2019 Time: 06.21						
Hb	Ht	L	Tr	PT	INR	APTT
10,2	29,5	8.330	56.000	17,8	1,47	37.5
GDS	Fibrinogen	D dimer				
77	270	3,89				
Ur	Cr	Na	K	Cl	Ca	Mg
117	5,10	135	6,8	107	3,89	2,1
pH	pCO2	pO2	HCO3	BE	SpO2	
7,134	54,2	140,3	18,4	-9,9	97,6	

Thorax PA Rongent (July 18th, 2019)

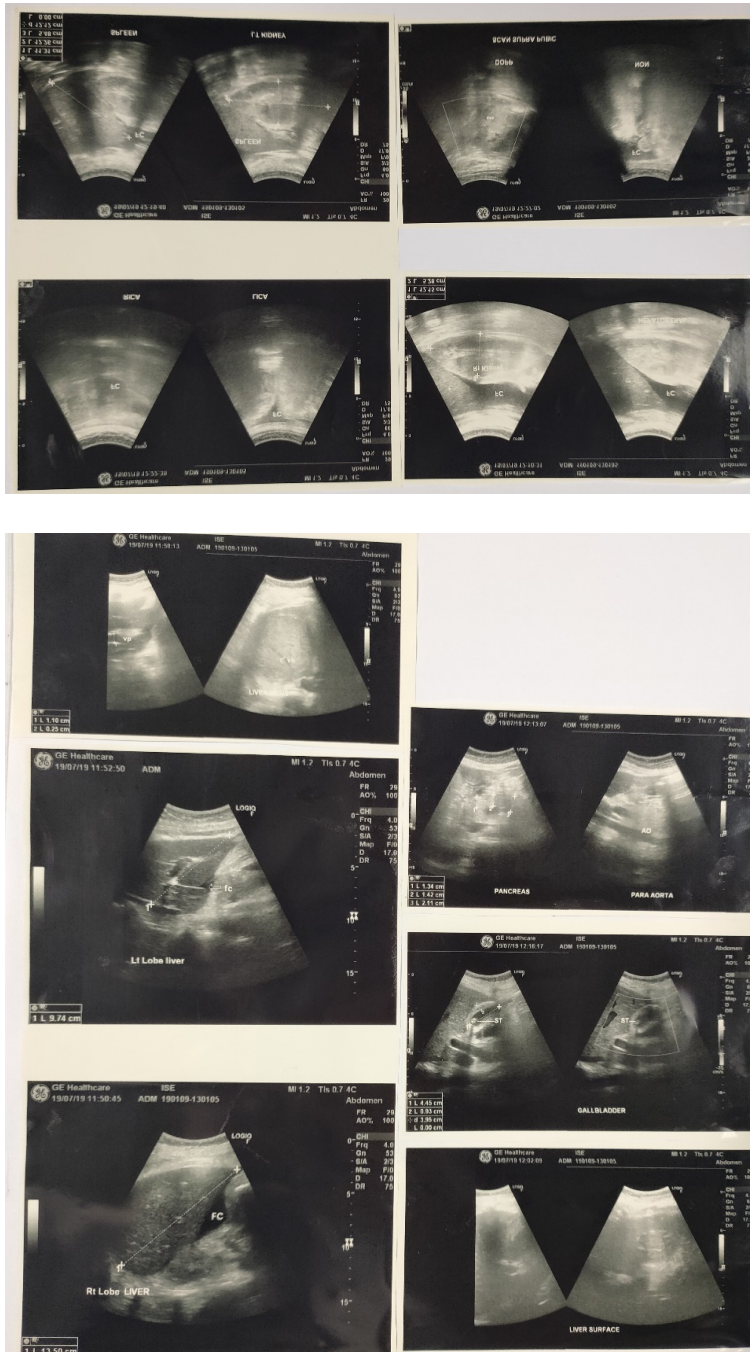
Cardiomegaly without lung congestion, no pneumonia or pulmonary edema



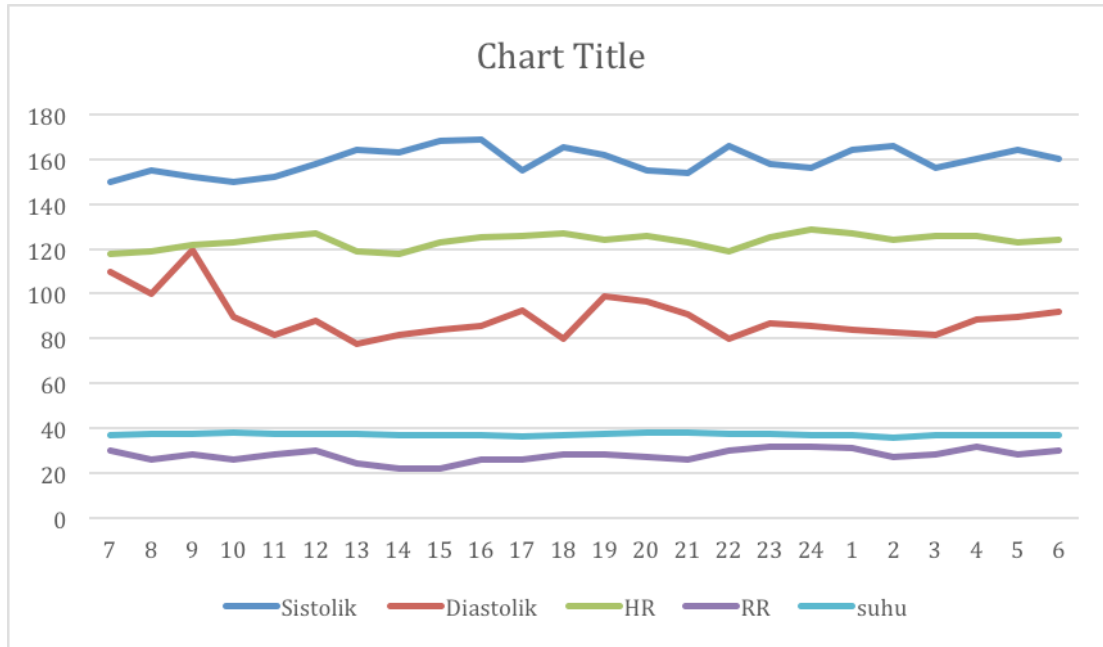
Abdomen Ultrasonography

Impression:

- Isoechoic lesion with indistinctive border, regular in the suprapubic region → suggestive blood clot
- Collection of fluid in hepatorenal, splenorenal and suprapubic areas → ascites
- Multiple cholelithiases, splenomegaly
- No apparent enlargement of the paraaortic / parailiac lymph nodes
- Ultrasonography of the liver, kidneys, and pancreas does not show any abnormality



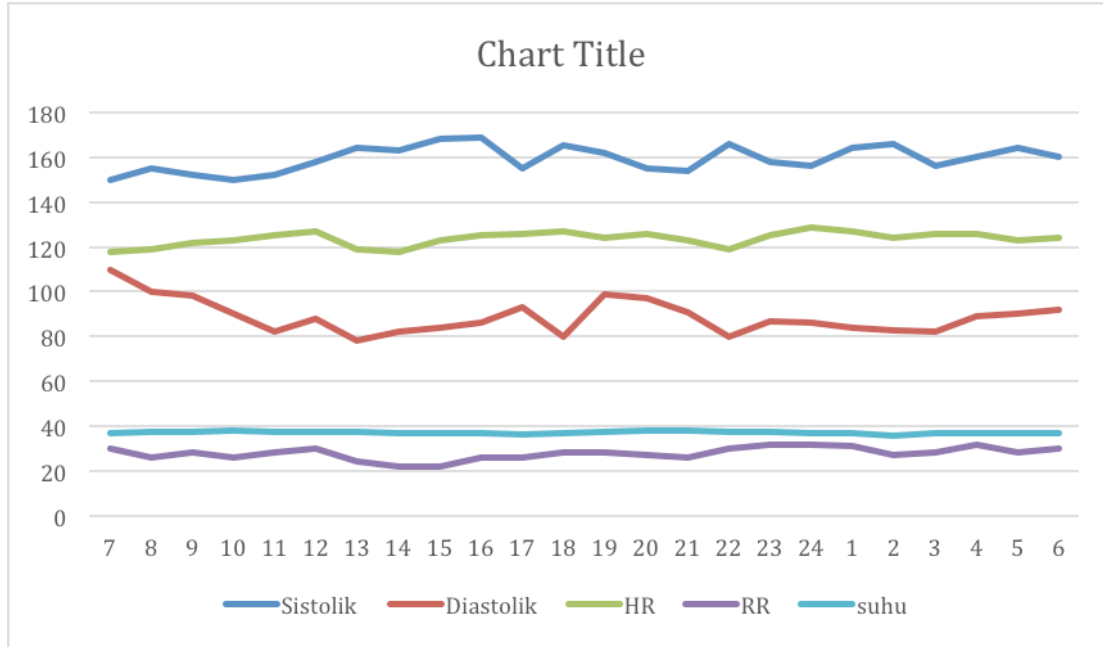
Day 3 (July 20th, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
CNS E2M2Vt CVS BP : 157 / 91 mmHg HR : 94 x/min Temp: 37 C Respiration RR: 20 x/minute PSIMV/RR 12/PC 15/PEEP 6/FiO2 65% (TV 400-500) SpO2: 99 % GIT distension (-) bowel sound (+) GUT UO : 0-10-10 cc/ hour Balance : +419,8 cc/24hour	Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + hypoalbuminemia 20/7/19 Hb 8,1 Ht 22 L 7.910 Tr 39.000 PT 10,1 INR 0,9 APTT 28,3 Laktat 1,6 Ur 64 Cr 2,83 Na 134 K 2,83 Cl 100 Ca 4,91 Mg 1,6 pH 7,545 pCO2 25,5 pO2 248,0 HCO3 22,3 BE 0,2 Sat 99,5 Blood culture 18/7/19 No result yet	F: gradual liquid diet A: Fentanyl 30 mcg/hour, Paracetamol 1g/6hour S: - T: - H: Head-Up 30 degree U: Omeprazole 2x40 mg G : Th/ Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (3) Furosemid drip 30 mg/hour KCl 35 meq/4 hour Nebu NaCl 0,9% per 6 hour

July 20 th , 2019 Time: 06.21						
Hb	Ht	L	Tr	PT	INR	APTT
8,1	22	7.910	39.000	10,2	0,90	28,3
Ur	Cr	Na	K	Cl	Ca	Mg
64	2,83	134	4,1	100	4,91	1,6
pH	pCO2	pO2	HCO3	BE	SpO2	Laktat
7,545	25,5	248,0	22,3	0,2	99,5	1,6

Day 4 (July 21st, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
CNS E2M3VT CVS BP : 145 / 84 mmHg HR : 111 x/min Temp: 37 C Respiration RR: 20 x/ minute PSIMV/RR 12/PC 14/PEEP 8/FiO2 60% (TV 400-500) SpO2: 99 % GIT distension (-) bowel sound (+) GUT UO : 30-10-10 cc/ hour Balance : - 2530 cc/24hour	Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + electrolyte imbalance + hypoalbuminemia 21/7/19 Hb 7,5 Ht 22,2 L 16.410 Tr 46.000 PT 10,6 INR 0,94 APTT 119,3 GDS 165 Alb 1,7 Lactat 1,6 Ur 131 Cr 5,67 Na 135 K 6,3 Cl 100 Ca 4,29 pH 7,349 pCO2 48,9 pO2 159,8 HCO3 27,2 BE 2,4 Sat 98,4 Blood culture 18/7/19 No result yet Echohemodynamics: CO 6.8 L / min CI 4.16 L / min / m2 SV 52 ml / beat SVI 32 ml / Beat / m2 SVR 871 dyne.sec.atm IVC max 1.66 MIVC Min 1.18 Distesibility index 34% Structural: Normal echo chamber, LVEF eyeballing> 50% Normokinetic Impression: fluid responsive, normal cardiac function	F : :Liquid diet A : Fentanyl 30 mcg/hour, Paracetamol 1g/6hour S : - T : - H : Head Up 30 U : Omeprazole 2x40 mg G : Th/ Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (4) Furosemid drip 30 mg/hour Nebu NaCl 0,9% per 6 hour Ca gluconate 2 gram Platelet (thrombocyte) transfusion : 7 bags Second haemodialysis Re-evaluate: PT, INR, aPTT

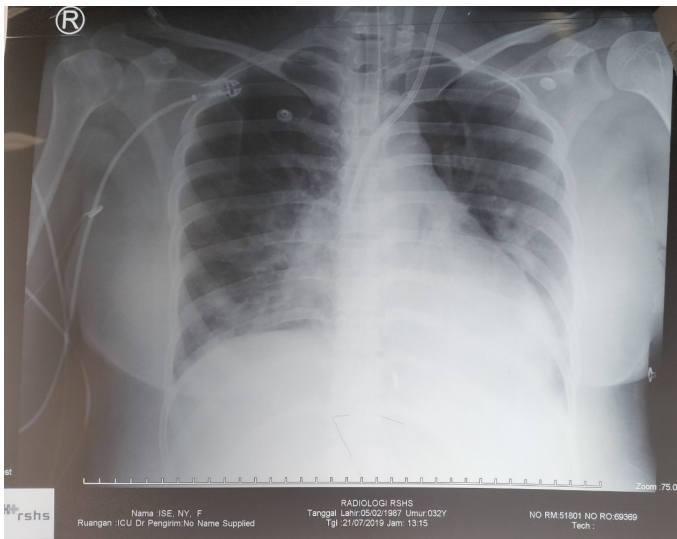
July 21 st , 2019 Time: 05.15 am						
Hb	Ht	L	Tr	PT	INR	APTT
7,5	22,2	16.410	46.000	10,0	0,94	119,3
Ur	Cr	Na	K	Cl	Ca	Mg
131	5,67	135	6,3	100	4,29	2,0
pH	pCO2	pO2	HCO3	BE	SpO2	Laktat

7,349	48,9	159,8	27,2	2,3	98,4	1,6
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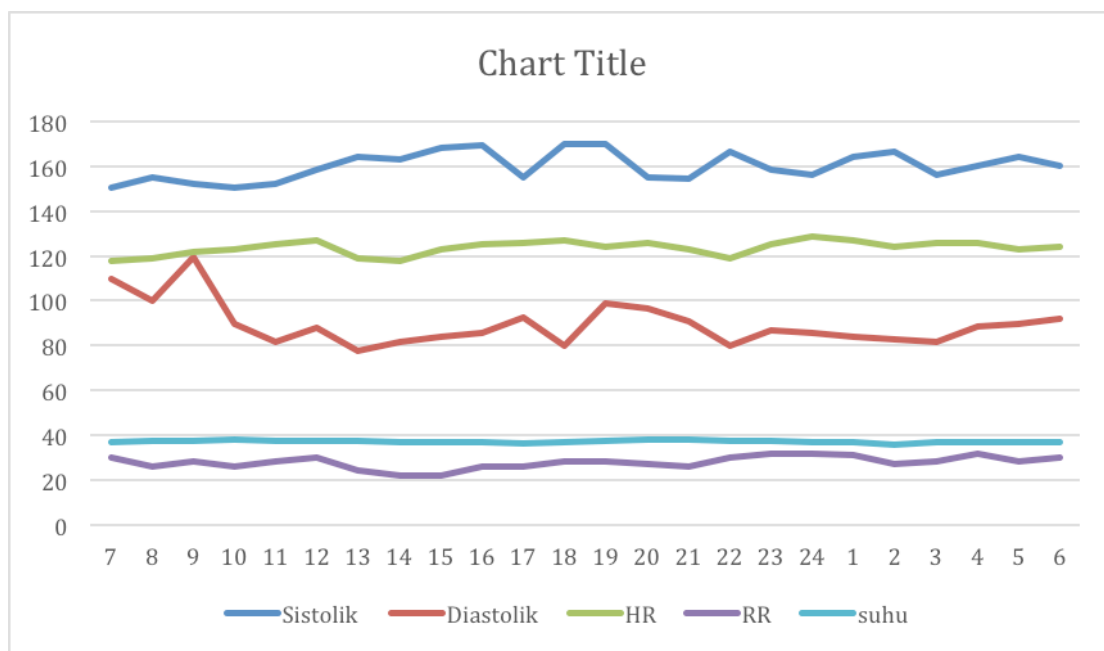
July 21 st , 2019 Time: 01. 34 pm						
Hb	Ht	L	Tr	PT	INR	APTT
10,6	21,1	18.430	40.000	9,8	0,87	23,9
Ur	Cr	Na	K	Cl	Ca	Mg
113,9	4,55	145	5,3	99	4,54	2,2
Albumin						
2,2						

Thorax PA X-ray (July 21st, 2019)

Bronchopneumonia, there is no sign of cardiomegaly



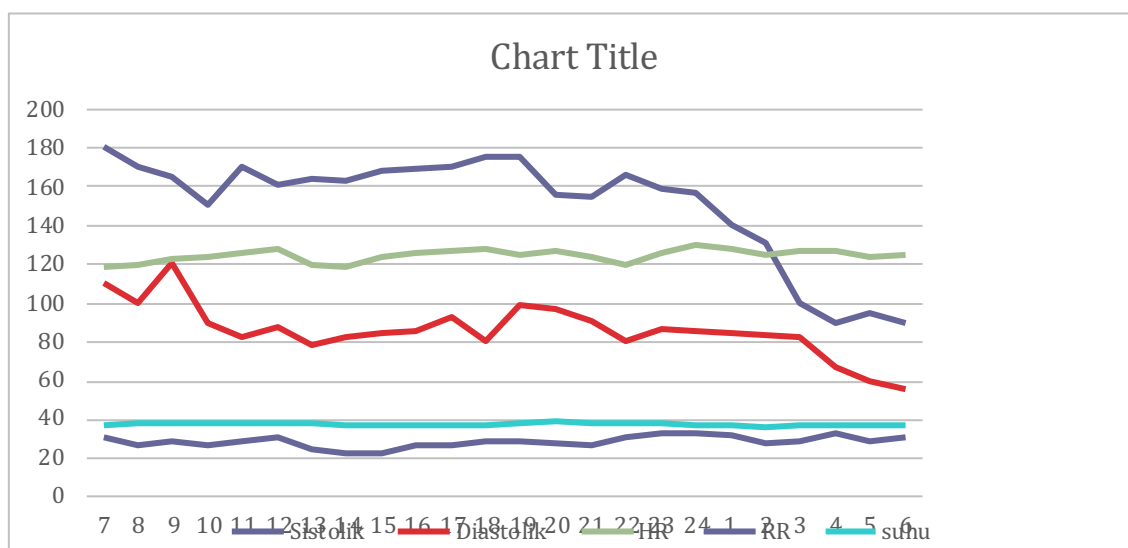
Day 5 (July 22nd, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
<p>CNS E2M3VT</p> <p>CVS BP : 161 / 96 mmHg HR : 115 x/ menit Temp: 38 C</p> <p>Respiration RR : 24 x/ minutes Spontan/PS 15/PEEP 8/FiO2 60% (TV 400-500) SpO2: 99 %</p> <p>GIT distension (-) bowel sound (+)</p> <p>GUT UO : 0-14-0 cc/ hour Balance : - 24 cc/24hour</p>	<p>Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + electrolyte imbalance + hypoalbuminemia</p> <p>22/7/19 Hb 8,8, Ht 26,3 L 14.600 Tr 77.000 Ur 143,3 Cr 5,97 Na 134 K 5,8 Cl 98 Ca 4,05 Mg 2,3 pH 7,381 pCO2 48,9 pO2 172,6 HCO3 29,1 BE 4,3 Sat 99,4</p> <p>Blood culture 18/7/19 No result yet</p>	<p>F: Liquid diet A: Fentanyl 30 mcg/hour, Paracetamol 1g/6hour S: - T: - H: Head-Up 30 degree U: Omeprazole 2x40 mg G :</p> <p>Th/ Tranexamic Acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (5) Furosemid drip 30 mg/hour Nebu NaCl 0,9% per 6 hour</p>

July 22 nd , 2019 Time. 05.41 am						
Hb	Ht	L	Tr	Ur	Cr	
8,8	26,3	14.660	77.000	143,3	5,97	
Na	K	Cl	Ca	Mg		
134	5,8	98	4,05	2,3		
pH	pCO2	pO2	HCO3	BE	SpO2	
7,381	48,9	172,6	29,1	4,3	99,4	
Ur	Cr	Na	K	Cl	Ca	Mg
168	6,52	136	5,1	95	4;21	2,2

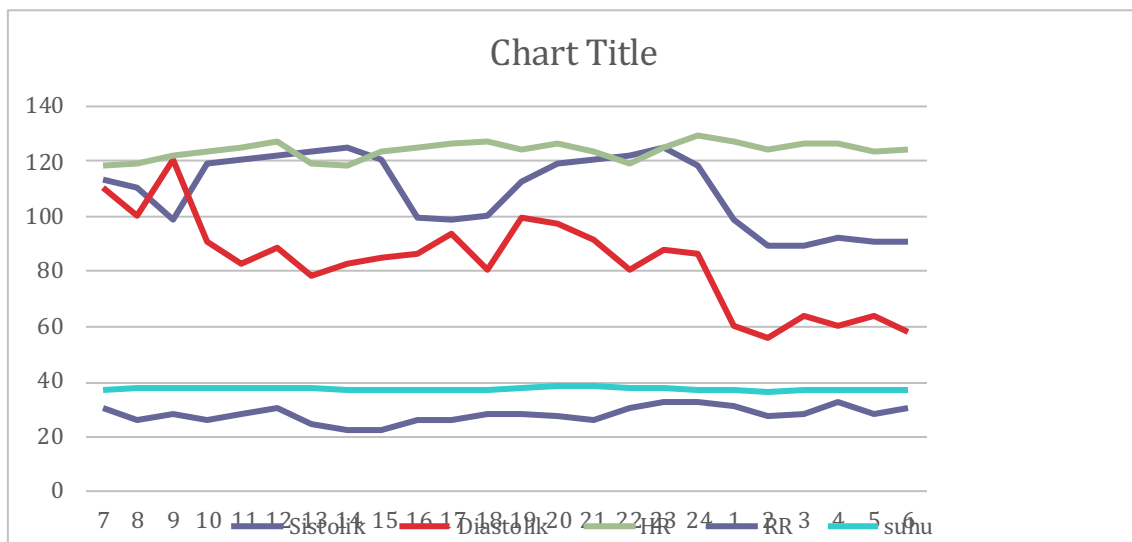
Day 6 (July 23rd, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
<p>CNS E1M2V1</p> <p>CVS TD: 180/103 mmHg N:110 x/minute SpO2: 96%</p> <p>Respiration RR : 12 x/minute Spontan/PS 15/PEEP 8/FiO2 60% (TV 400-500) SpO2: 99 %</p> <p>GIT distension (-) bowel sound (+)</p> <p>GUT UO : 0-14-0 cc/ hour Balance : - 24 cc/24hour</p>	<p>Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + electrolyte imbalance + hypoalbuminemia</p> <p>22/7/19 Hb 9,2 Ht 26,3 L 23.000 Tr 48.500 Ur 209,1 Cr 7,75 Na 134 K 5,3 Cl 98 Ca 4,03 Mg 2,2 SGOT 193 SGPT 525 Alb 1.80 pH 7.403 pCO2 33,8 pO2 158,9 HCO3 21,3 BE -2,2 Sat 02 98,4</p> <p>Kultur darah 18/7/19 Tidak terdapat pertumbungan mikroorganisme</p> <p>Culture 7/23/19 1: Escherichia Coli 2: Klebsiella pneumonia Sensitive: Amikacin, Cefepime, Tigecycline, Meropenem</p>	<p>F: Liquid diet A: Fentanyl 30 mcg/hour, Paracetamol 1g/6hour S: Midazolam 5mg/hour T: - H: Head-Up 30 degree U: Omeprazole 2x40 mg G :</p> <p>Th/ Tranexamic Acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (5) Nebu NaCl 0,9% per 6 hour Dextrose 40% extra 10 units of insulin in D40% 2 extra cycles Ca gluconate in 2 grams of NaCl 0.9% 100 cc drip in 1 hour Midazolam 15mg 5mg / hour NaCL dust 0.9% Perdipin 1mcg / kg / min Furosemide 20mg/hour Hemodialysis</p>

July 23 rd , 2019 Time: 05.41						
Hb	Ht	L	Tr	Ur	Cr	
9,2	26,3	23.000	48.500	209,1	7,75	
Na	K	Cl	Ca	Mg		
134	5,3	98	4,03	2,2		
PT	aPTT	INR	SGOT	SGPT	Alb 1,80	
10,6	23,9	0,94	193	525		
pH	pCO2	pO2	HCO3	BE	SpO2	
7,403	33,8	158,9	21,3	-2,2	98,4	

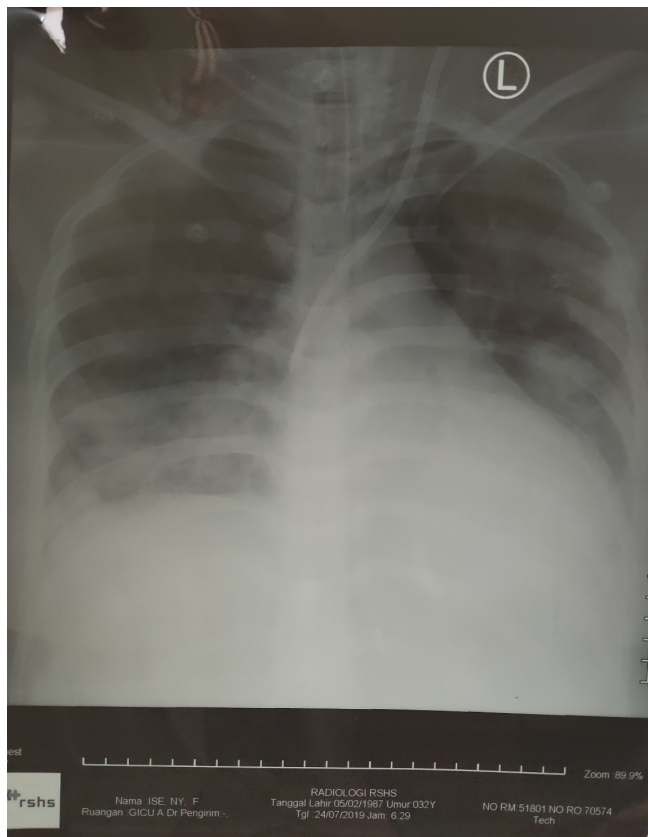
Day 7 (July 24th, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
<p>CNS E₁M₂V₁</p> <p>CVS BP: 128/78 mmHg HR: 114 x/minute SpO₂: 96%</p> <p>Respiration RR : 25 x/minute Spontan/PS 15/PEEP 8/FiO₂ 60% (TV 400-500) SpO₂: 99 %</p> <p>GIT distention (-) bowel sound (+)</p> <p>GUT UO : 15-34-44 cc/ hours Balance : +594,8 cc/24 hours</p>	<p>Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + electrolyte imbalance + hypoalbuminemia</p> <p>22/7/19 Hb 10,8 Ht 30,7 L 22.930 Tr 88.000 Ur 116,0 Cr 5,02 Na 137 K 4,3 Cl 98 Ca 4,65 Mg 2,3 Alb 1,8 pH 7,260 pCO₂ 62,2 pO₂ 76,1 HCO₃ 28,2 BE 1,1 Sat 90,6 Total bilirubin 8,789 Bilirubin 7,238 Indirect Bilirubin 1.551</p>	<p>F : liquid diet A : Morfin 10mcg/kgbb/hour S : Midazolam 5mg/hour T : - H : Head Up 30 U : Omeprazole 2x40 mg G :</p> <p>Th/ Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (5) Drip furosemid 30 mg/hours NaCl 0,9% nebulation/ 6 hours Levofloxacin 3x200mg</p>

July 24th, 2019 at 11.13 am

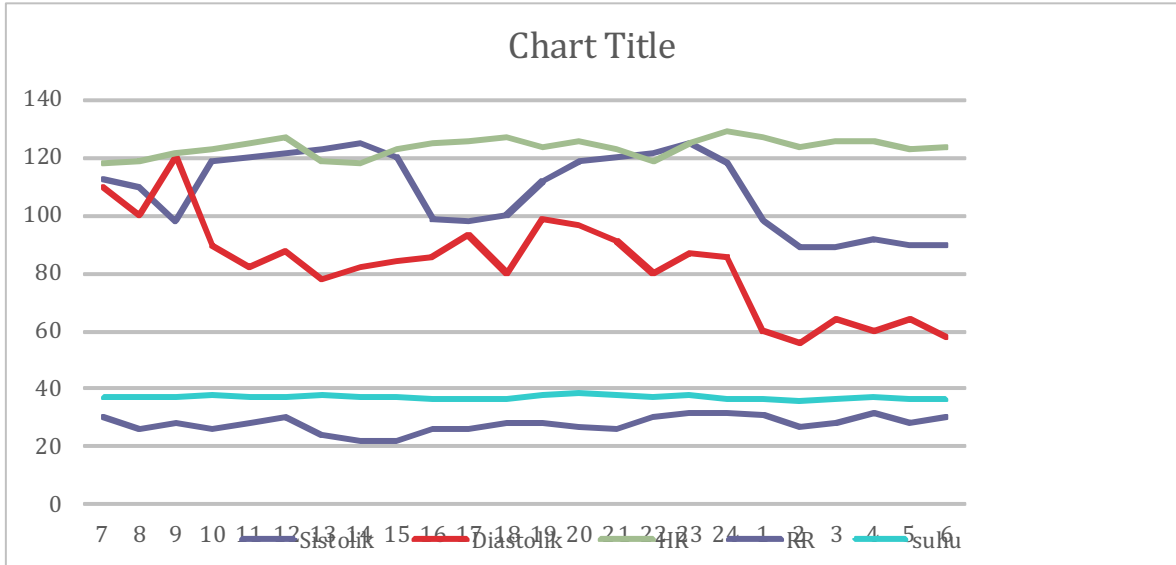
Hb	Ht	L	Tr	Ur	Cr	
10,8	30,7	22.930	89.000	116.0	5,02	
Na	K	Cl	Ca	Mg	Alb	
137	4,3	98	4,65	1,8	1,65	
pH	pCO ₂	pO ₂	HCO ₃	BE	SpO ₂	
7,260	62,2	76,1	28,2	1,1	90,6	



Description :

- Bilateral pneumonia
- cardiomegaly dd/ position

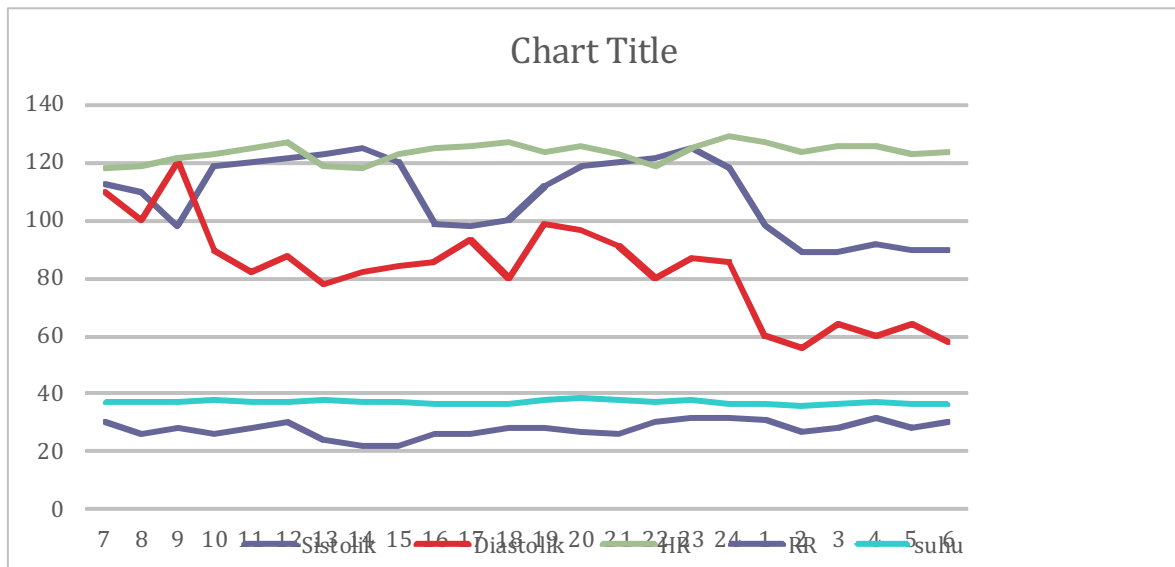
Day 8 (July 25th, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
CNS E1M2Vt CVS BP: 109/60 mmHg HR:98 x/minute SpO2: 94% Respiration RR : 16 x/minute Spontan/PS 15/PEEP 8/FiO2 60% (TV 400-500) SpO2: 99 % GIT distention (-) bowel sound (+) GUT UO : 0-14-0 cc/ hour Balance : - 24 cc/24 hours	Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + electrolyte imbalance + hypoalbuminemia 25/7/2019: Hb/ht/L/T: 8,4/25,0/24.230/67.000 Urem:215,1/Creatinin:7,37/albumin:1,77 Na/K/Cl/Ca/Mg:136/5,0/97/4,33/2,2 pH 7,213 pCO2 56,6 pO2 116,4 HCO3 23,0 BE -4,5 Sat 96,2	F : Liquid diet A : Morfin 10mcg/kg/hour S : Midazolam 5mg/hour T : - H : Head Up 30 U : Omeprazole 2x40 mg G : Th/ Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (5) Furosemid drip 30 mg/hour NaCl 0,9% nebulation/ 6 hours Cefepime 3x1gr Levofloxacin 3x200mg Norepinephrine 0,5mcg/kg/minute Hemodialysis

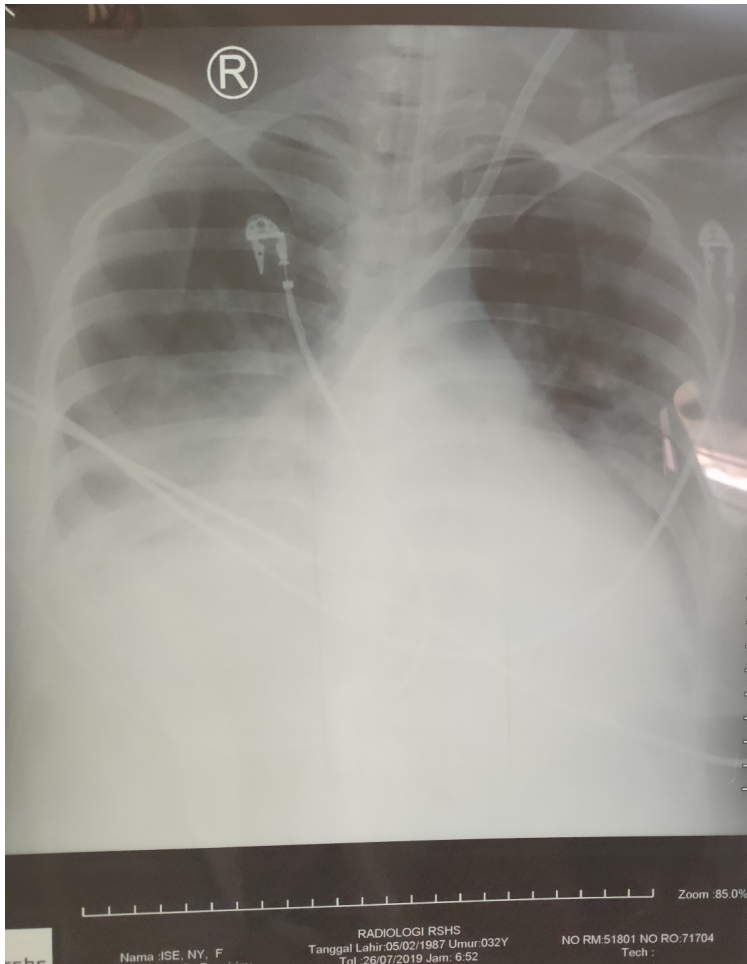
July 25th, 2019 05.41						
Hb	Ht	L	Tr	Ur	Cr	
8,4	25,0	24,230	67.000	215,1	7,37	
Na	K	Cl	Ca	Mg	Alb	
136	5,0	97	4,33	2,2	1,77	
pH	pCO2	pO2	HCO3	BE	SpO2	
7,213	56,6	116,4	23,0	-4,5	96,2	

Day 9 (July 26th, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
CNS E _i M _i V _t	Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + electrolyte imbalance + hypoalbuminemia	F: liquid diet A: paracetamol 4x1gr S: - T: - H: Head-Up 30 degree U: Omeprazole 2x40 mg G :
CVS TD: 97/51 mmHg N:134 x/minute SpO ₂ : 100%	26/7/2019: Hb/ht/L/T: 8,5/26,0/35.700/70.000 PT:10,40 APTT:32,60 INR:0,92 Ureum:77,0 creatinin:3,57 Na/K/Cl/Ca/Mg:145/4,3/104/5,37/1,7	Th/ Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (5) Furosemid drip 30 mg/hour NaCl 0,9% nebulation /6 hours Cefepime 3x1gr Levofloxacin 3x200mg Norepinephrine 0,5mcg/kg/minute Dobutamin 5mcg/kg/minute Vasopressin 0,02 unit/hour
Respiration RR : 15 x/minute Spontan/PS 15/PEEP 8/FiO ₂ 60% (TV 400-500) SpO ₂ : 99 %		
GIT distention (-) bowel sound (+)		
GUT UO : 0-14-0 cc/ hour Balance : - 24 cc/24 hours		

July 26th, 2019 05.41						
Hb	Ht	L	Tr	Ur	Cr	
8,5	26,0	35.700	70.00	77.0	3,75	
Na	K	Cl	Ca	Mg		
145	4,3	104	5,3	1,7		
PT	aPTT	INR				
10,4	32,6	0,92				
pH	pCO ₂	pO ₂	HCO ₃	BE	SpO ₂	lactat
7,196	56,8	100,8	22,2	5,4	94,7	5.0

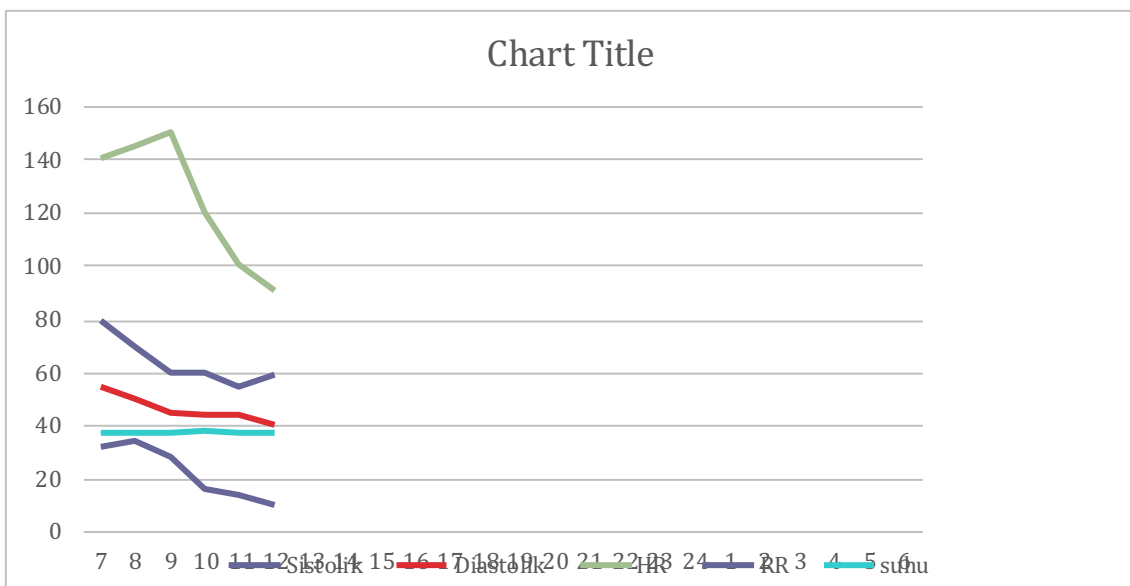


26/7/19

description :

- bilateral pleuropneumonia

Day 10 (July 27th, 2019)



OBJECTIVE	ASSESSMENT	PLANNING
CNS E ₁ M ₁ V _t CVS BP: 77/53 mmHg HR: 130 x/minute SpO ₂ : 90% Respiration RR : 21 x/minute Spontan/PS 15/PEEP 8/FiO ₂ 60% (TV 400-500) SpO ₂ : 99 % GIT distention (-) bowel sound (+) GUT UO : 0-14-0 cc/ hour Balance : - 24 cc/24 hours	Post hemorrhagic shock + Post supravaginal hysterectomy ec atonia uteri in P3A0 premature parturition with cesarean section due to bishop score <6 on impending eclampsia + HELLP syndrome + DIC + acute kidney injury + anemia + electrolyte imbalance + hypoalbuminemia	F: Nefrisol+Boostpoimun A: paracetamol 4x1gr S: - T: - H: Head-Up 30 degree U: Omeprazole 2x40 mg G : Th/ Tranexamic acid 3x500 mg Vitamin K 3x10 mg Ceftriaxone 1x2 gr iv (5) Furosemide drip 30 mg/hour NaCl 0,9% Nebulation/ 6 hour Cefepime 3x1gr Levofloxacin 3x200mg Norepinephrine 0,5mcg/kg/minute Dobutamin 5mcg/kg/minute
12.34 Asystole		the patient was declared dead

DISCUSSION

Patients are G3P2A0 with 29-30 weeks gestation, during pregnancy known to have a history of uncontrolled hypertension. On physical examination, found blood pressure of 160/100 mmHg and proteinuria 3+. The results of investigations found a decrease in platelet count (Tr 85,000), increased serum transaminase levels (SGOT 2262, SGPT 1502), and increased serum creatinine levels (Cr 2.71). The diagnosis of preeclampsia consists of several criteria, which can be seen in Table 1.

Table 1 Preeclampsia Criteria (Robert, et al., 2013)

Blood Pressure	<ul style="list-style-type: none"> Increased systolic blood pressure \geq 140 mmHg or diastolic blood pressure \geq 90 mmHg at two measurements with a range of at least 4 hours after 20 weeks' gestation in pregnant women not previously known to have hypertension Increased systolic blood pressure \geq 160 mmHg or diastolic blood pressure \geq 110 mmHg found in short (minute) intervals that immediately require the administration of antihypertensive drugs
Dan	
Proteinuria	<ul style="list-style-type: none"> \geq 300 mg on urine sample measurements for 24 hours or Protein / creatinine ratio \geq 0.3 Protein content in dip paper 1+
Or if no proteinuria is found	
Thrombocytopenia	<ul style="list-style-type: none"> Platelet count $<$100,000 / μL
Renal insufficiency	<ul style="list-style-type: none"> serum creatinine levels $>$ 1.1 mg / dL or doubling of serum creatinine levels without any other cause of impaired kidney function
Liver disfunction	<ul style="list-style-type: none"> Increased serum transaminase levels more than doubled to normal
Pulmonary edema	

Impaired brain
function or vision
with new-onset

The patient was later diagnosed with impending eclampsia because, in addition to the signs and symptoms of preeclampsia, the patient complained of severe headaches accompanied by blurred vision and heartburn. In impending eclampsia, there are several clinical symptoms found, such as persistent occipital or frontal headache, blurred vision, photophobia, pain in the epigastric region or the right upper quadrant or both, and changes in consciousness without accompanying seizures. If impending eclampsia is not treated quickly, it can cause seizures or eclampsia. (Robert, et al., 2013)

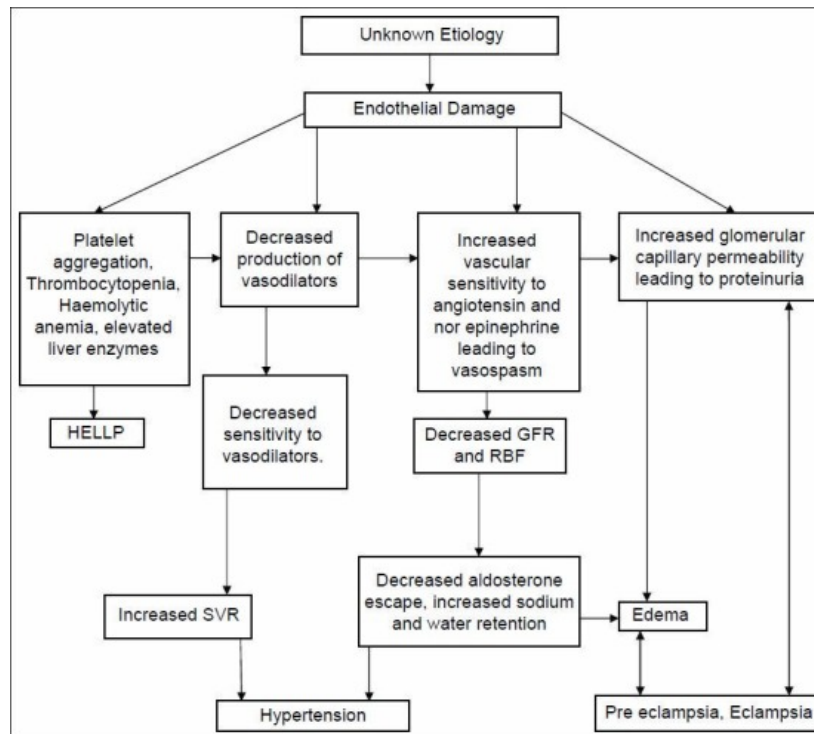
The etiology of preeclampsia is unknown, but there are several risk factors for preeclampsia including primipara, history of preeclampsia in a previous pregnancy, family history of hypertension, multiple or multiple pregnancies, history of thrombophilia, maternal age over 40 years, in vitro fertilization, diabetes, obesity, systemic lupus erythematosus, and a history of chronic hypertension.⁷ The risk factors for preeclampsia present in these patients are obesity (body mass index 30.3). Obesity increases the risk of preeclampsia by 2-3 times. Some pathophysiology linking obesity with preeclampsia include insulin resistance, inflammatory reaction due to the release of proinflammatory mediators from adipose tissue (CRP, Interleukin-6, TNF- α), reactions due to oxidative stress, adipokine imbalance (leptin and adiponectin), and imbalance angiogenic factors (placental growth factor, vascular endothelial growth factor). (Jeyabalan, 2014)

The pathophysiology underlying preeclampsia is abnormal placental development and an imbalance between angiogenic factors. The pathophysiology of preeclampsia begins with the disturbance of trophoblast invasion of the spiral arteries between 8 and 16 weeks' gestation triggered by immunological disorders. Abnormal invasion results in failure of the process of remodeling of the arteries that function to nourish the placenta, resulting in uteroplacental blood flow fail to meet needs, then the placenta becomes ischemic which triggers the release of proinflammatory mediators. (Lambert, 2014)

Placental growth factor (PIGF) and vascular endothelial growth factor (VEGF) are potent angiogenic factors that function to strengthen the vasodilation effects of prostaglandins (PG) and nitrous oxide (NO) and enhance endothelial function. In preeclampsia occurs the formation of anti-angiogenic mediators such as tyrosine-kinase (sFlt-1), which interferes with vasodilation and results in endothelial dysfunction. An imbalance between pro-angiogenic and anti-angiogenic factors results in extensive endothelial dysfunction, microangiopathy, and vascular vasospasm. This results in impaired perfusion in various organs such as the liver and kidneys. (Lambert, 2014)

The released proinflammatory mediator also triggers the release of pro-coagulation factors so that activation of the coagulation system can then result in disseminated intravascular coagulation. The pathogenesis of disseminated intravascular coagulation is a complex mechanism by which intravascular fibrin deposition occurs and the use of coagulation and platelet factors. This mechanism then causes the formation of thrombus in the microvascular which inhibits perfusion to the tissue resulting in organ failure, in addition to the consumption of many coagulation factors resulting in a deficiency of the coagulation and platelet factors leading in bleeding. (Sahin, 2014)

Figure1 Pathophysiology of preeclampsia



Cited from: Parthasarathy (Parthasarathy, 2013)

The main goal of the management of preeclampsia is to prevent seizures and reduce maternal blood pressure below 160/110 mmHg.(Kelsey, 2015) Management of preeclampsia, eclampsia, and HELLP syndrome consists of:

1. Monitoring the condition of the mother and fetus closely.
2. Giving corticosteroid therapy at 24-34 weeks' gestation to help the fetal lung maturation. The fetus can be born if corticosteroid therapy has been given for 48 hours.
3. Labor is the only definitive therapy in patients with preeclampsia, eclampsia, and HELLP syndrome. At a gestational age of fewer than 24 weeks, pregnancy termination is recommended. In patients with HELLP syndrome and eclampsia who have a gestational age <33-34 weeks, delivery by cesarean section is the first choice.
4. Provision of magnesium sulfate therapy as seizure prophylaxis. The initial dose of magnesium sulfate is given 6 grams intravenously for 20 minutes, followed by a maintenance dose of 2 grams/hour intravenously up to 24 hours postpartum. In the event of a seizure, a bolus of magnesium sulfate can be given 2 grams intravenously for 3-5 minutes. In administering magnesium sulfate periodically, it is necessary to monitor magnesium toxicity by examining serum magnesium levels with a target therapeutic value of magnesium of 5-8 mg / dL.
5. The provision of antihypertensive drugs is recommended if blood pressure \geq 160/110 mmHg. The purpose of antihypertensive medication is to prevent an increase in intracranial pressure resulting in brain edema and intracranial hemorrhage. Antihypertensive drugs that can be given include hydralazine 5-10 mg intravenously for 2 minutes or labetalol 20-80 mg for 2 minutes or nifedipine 10-20 mg orally with a target blood pressure range of 140-150 / 90-100 mmHg. Other alternative antihypertensive medications include labetalol or nicardipine drip.
6. Platelet transfusion can be given if platelet count <50,000 / μ L in patients undergoing cesarean section or platelets \leq 20,000-50,000 / μ L in vaginal delivery or platelets <20,000 / μ L accompanied by active bleeding.(Lam & Dierking, 2017; Lambert, et al., 2017; Parthasarathy, 2013)

Early detection and management of preeclampsia, eclampsia, and HELLP syndrome are key ingredients in helping to prevent severe complications. In preeclampsia, complications can arise both short and long term for both mother and baby. Maternal complications include pulmonary edema, myocardial infarction, stroke, acute respiratory distress syndrome, coagulopathy, bleeding, disseminated intravascular coagulation, and injury to the retina. This is related to organ dysfunction caused by the pathophysiological process of preeclampsia. (Robert, et al., 2013)

This patient experienced complications of postpartum hemorrhage due to uterine atony, resulting in hemorrhagic shock (Hb 5,6). The patient is then intubated, fluid resuscitation is performed, then a hysterectomy re-laparotomy is performed. The patient was transferred to the intensive care unit after the operation was completed. In patients with preeclampsia, the incidence of postpartum hemorrhage increases 1.53-fold. The WHO definition of postpartum hemorrhage is a blood loss of ≥ 500 ml within 24 hours. The main causes of postpartum hemorrhage include uterine atony, residual placenta, and coagulopathy. (Altenstadt, 2013)

Predisposing factors for postpartum hemorrhage in these patients are preeclampsia, coagulopathy (INR 1.48, Fibrinogen 104.4, d-dimer 3.22), and thrombocytopenia (Tr 31,000). The patient has a score of The International Society of Thrombosis and Hemostasis (ISTH) with a total of 5 points, which means the patient has disseminated intravascular coagulation. Patients were subsequently given a transfusion of 4 pumpkin PRC, three pints FFP, and seven pints of Thrombocyte concentrate.

The main goal of the management of disseminated intravascular coagulation is to improve the underlying obstetric causes. Besides that, supportive therapy is given to correct coagulation abnormalities. Platelet transfusion can be given if the platelet count is less than 50,000, accompanied by massive bleeding. If there is an extension of the PT, aPTT, and INR values, then a fresh frozen plasma (FFP) of 10-20 ml/kg BW can be given. Non-activated prothrombin complex concentrate (PCC) can be given 25-30 U / kgBB to substitute FFP administration in patients who have experienced excess fluid. If there is only a blood fibrinogen deficiency <1 gram / L, cryoprecipitate transfusion can be given. The administration of a 4 gram cryoprecipitate transfusion can increase serum fibrinogen levels to 1 gram / L. The expected laboratory results are PT, aPTT <1.5 from normal, platelets $> 50,000 / \mu\text{L}$, and fibrinogen > 1 gram / L. (Sahin, et al., 2014)

On the first day of treatment in the intensive care unit, the patient's hemodynamic status profile was still assisted with noradrenaline administration. Noradrenaline was stopped because the patient's hemodynamic condition stabilized after the administration of the PRC and FFP transfusions aimed at replacing blood loss when the patient was in hemorrhagic shock. During observation in the intensive care unit, the patient was known to have reduced urine production (5-20-0 cc/hour) accompanied by an increase in serum creatinine levels (Cr 3.1). The patient is then given an echo-hemodynamic examination to ensure adequate fluid. Patients are given diuretic therapy (furosemide and hemodialysis are performed to support kidney function because the patient has an acute kidney injury. Acute kidney injury in these patients can be caused by preeclampsia, which is also aggravated by the condition of hemorrhagic shock resulting in decreased perfusion of renal blood flow.

Acute kidney injury occurs in 1-5% of patients with preeclampsia. Preeclampsia results in a decrease in renal blood flow, a reduction in glomerular filtration rate of 30-40%, and renal vasoconstriction, causing kidney damage. Acute kidney injury aggravates conditions arising from complications of preeclampsia for example in placental abruption, disseminated intravascular coagulation, sepsis, postpartum hemorrhage, and intrauterine fetal death. (Prakash & Ganiger, 2017)

Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease (RIFLE) criteria were introduced in 2014 to help determine the requirements and severity of acute kidney injury. RIFLE criteria can be seen in table 2.

Table 2 RIFLE Criteria (Lopes & Jorge, 2013)

Class	Glomerular Filtration Rate (GFR)	Urine Production
<i>Risk</i>	Increased SCreat x 1.5 or GFR decrease $> 25\%$	UO < 0.5 ml/jg/h x 6 h
<i>Injury</i>	Increased SCreat x 2 or GFR decrease $> 50\%$	UO < 0.5 ml/kg/h x 12 h
<i>Failure</i>	Increased SCreat x 3 GFR decrease 75% or SCreat ≥ 4 mg/dL Acute rise ≥ 0.5 mg/dL	UO < 0.3 ml/kgH x 24 or anuria x 12 h
<i>Loss of kidney function</i>	Persistent ARF = complete loss of kidney function > 4 weeks	

<i>End-stage kidney disease</i>	End stage kidney disease (>3 months)	
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Management of acute kidney injury includes the provision of supportive therapy, dialysis, and treatment of underlying disease. Renal supportive therapy aims to maintain kidney function from further damage, such as avoiding the use of nephrotoxic drugs, overcoming source of infection, maintaining adequate fluid through intravenous infusion, and maintaining sufficient perfusion of renal blood flow. These general steps are also followed by administration of pharmacological therapy to overcome complications due to acute kidney injury such as hypertension, hyperkalemia, metabolic acidosis, and anemia.(Prakash & Ganiger, 2017)

The indications for hemodialysis in acute kidney injury conditions include uremic symptoms (encephalopathy, pericarditis, or neuropathy), fluid overload, hyperkalemia, and metabolic acidosis that have no response to treatment. Hemodialysis is recommended if the glomerular filtration rate drops below 20 ml/min / 1.73 m².(Prakash & Ganiger, 2017)

CONCLUSION

Preeclampsia, eclampsia, and HELLP syndrome are life-threatening conditions for both mother and fetus during pregnancy that require treatment in Intensive Care Unit. Determining the basis for proper diagnosis and initial management and prevention of complications in preeclampsia, eclampsia, and HELLP syndrome can reduce the incidence of morbidity and mortality.

References

- Altenstadt, J.F.V.S.A., Hukkelhoven, C.W.P.M., Roosmalen, J.V., & Bloemenkamp, K.W.M. (2013). Preeclampsia Increases the Risk of Postpartum Hemorrhage: A Nationwide Cohort Study in The Netherlands. *PLoS One*, 8(12), e81959. <https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0081959>
- Jeyabalan, A., (2014). Epidemiology of Preeclampsia: Impact of Obesity. *Nutr Rev*, 71(01), 1111. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3871181/>
- Kelsey, J. J., (2015). Obstetric Emergencies in The ICU. *The Journal of the American College of Clinical Pharmacy*, 7, 7-18. <https://www.accp.com/docs/bookstore/psap/p7b02sample01.pdf>
- Lam, M. T. C., & Dierking, E. (2017). Intensive Care Unit Issues in Eclampsia and HELLP Syndrome. *Int J Crit Illn Inj Sci*, 7(3), 136-41. <https://pubmed.ncbi.nlm.nih.gov/28971026/>
- Lambert, G., Brichant, J.F., Hartstein, G., Bonhomme, V., & Dewandre, P. Y. (2014). Preeclampsia: An Update. *Acta Anaesth. Belg.*, 65, 137-49. https://www.sarb.be/site/assets/files/1142/01-lambert_et_al.pdf
- Lopes, J.A., & Jorge, S. (2013). The RIFLE and AKIN Classifications for Acute Kidney Injury: a Critical and Comprehensive Review. *Clinical Kidney Journal*, 6(1), 8-14.
- Parthasarathy, S., Kumar, V.R.H., Sripiya, R., & Ravishankar, M. (2013). Anesthetic Management of a Patient Presenting with Eclampsia. *Anesth Essays Res*, 7(3), 307-12. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4173542/>
- Prakash, J., & Ganiger, V, C. (2017). Acute Kidney Injury in Pregnancy-specific Disorders. *Indian J Nephrol*, 27(4), 258-70. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5514821/>
- Roberts, J. M., August, P. A., Bakris, G., Barton, J.R. (2013). Classification of Hypertensive Disorders in Hypertension in Pregnancy. *American College of Obstetricians and Gynecologists*, vol 1, 13-6.
- Roberts, J. M., August, P. A., Bakris, G., Barton, J.R. (2013). Establishing the Diagnosis of Preeclampsia and Eclampsia in Hypertension in Pregnancy. *American College of Obstetricians and Gynecologists*, vol 1, 17-20.
- Roberts, J. M., August, P. A., Bakris, G., Barton, J. R. (2013). Hypertension in Pregnancy. *American College of Obstetricians and Gynecologists*, vol 1, 21-5.
- Roberts, J. M., August, P. A., Bakris, G., Barton, J. R. (2013). Management of Preeclampsia and HELLP Syndrome in Hypertension in Pregnancy. *American College of Obstetricians and Gynecologists*, vol 1, 31-46.

Sahin, S., Eroglu, M., Tetik, S., & Guzin, K. (2014). Disseminated Intravascular Coagulation in Obstetrics: Etiopathogenesis and Up to Date Management Strategies. *J Turk Soc Obstet Gynecol*, 11(1), 42-51.
<https://pdfs.semanticscholar.org/a4b7/d3e24dd23eb779f46e8517cbb8d5b81594b6.pdf>