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Pulmonary embolism in the setting of HELLP syndrome



M. Prin, A. Gaffney, S.W. Mankowitz

Department of Anesthesiology, Columbia University College of Physicians & Surgeons, New York, NY, USA

ABSTRACT

HELLP syndrome (hemolysis, elevated liver enzymes and low platelets) complicates 0.5–0.9% of pregnancies and is frequently associated with multiorgan dysfunction. Treatment relies on prompt diagnosis, delivery and supportive care. The clinical presentation may make the concurrent diagnosis and management of other disease entities challenging. This case report describes a patient with postpartum HELLP syndrome complicated by severe multiorgan dysfunction and pulmonary embolism.

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Introduction

The first case of HELLP syndrome was described by Pritchard et al. in 1954,¹ although it was not recognized as a distinct entity until 1982.² It affects 0.5–0.9% of all

pregnancies and 10–20% of parturients diagnosed with severe preeclampsia.³ Although the diagnosis of HELLP syndrome requires evidence of hemolysis, hepatic dysfunction and thrombocytopenia, specific diagnostic criteria and clinical presentations vary, making the precise diagnosis challenging.^{3–6} Furthermore, HELLP syndrome may be complicated by multiorgan dysfunction, which makes it difficult to differentiate from disseminated intravascular coagulation (DIC), pulmonary embolism (PE), amniotic fluid embolism (AFE), acute

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Correspondence to: Meghan Prin, MD, Columbia University College of Physicians & Surgeons, 622 West 168th Street, PH5, New York, NY 10032, USA.

E-mail address: mep9042@nyp.org

fatty liver of pregnancy (AFLP), and thrombotic thrombocytopenic purpura (TTP). This case report describes a patient with postpartum HELLP syndrome complicated by severe multiorgan dysfunction and pulmonary embolism.

Case report

A 29-year-old nulliparous woman was admitted from the outpatient clinic at 26 weeks and three days of gestation for anhydramnios and fetal intrauterine growth restriction (IUGR). She denied vaginal bleeding, amniotic fluid leakage or contractions and described normal fetal movements. Oligohydramnios and symmetric IUGR were diagnosed 10 days previously. Her past medical history was significant for a hemoglobin A variant. Her current pregnancy was complicated by hyperemesis gravidarum, gestational hypertension and complete breech position of the fetus.

On admission, she was hypertensive (blood pressure 160/90 mmHg) with a heart rate of 64 beats/min. Blood tests were within the normal range for pregnancy (Table 1). Intermittent variable fetal heart rate decelerations were noted on external monitoring. The obstetric plan was for pregnancy to continue for as long as maternal and fetal conditions allowed. The obstetric anesthesia team was consulted on the day of admission to prepare for possible emergent cesarean delivery. The patient weighed 76 kg; she had a Mallampati class 3 airway and normal spinal anatomy. It was decided that the urgency of delivery would determine anesthetic man-

agement: general anesthesia to expedite a category 1 delivery and, barring development of coagulopathy, spinal anesthesia for a category 2–4 delivery.⁷

Magnesium sulfate was prescribed on hospital day 1 for fetal neuroprotection, but discontinued due to nausea. A 24-h urine collection contained protein 577 mg, which in combination with the patient's elevated blood pressure indicated a diagnosis of preeclampsia. Blood pressure remained moderately elevated over the next few days ranging from 117/60 mmHg to 157/80 mmHg, but no treatment was initiated, as she did not develop severe hypertension (>160/110 mmHg). She remained on continuous external fetal heart rate monitoring, and variable fetal heart rate decelerations persisted intermittently with increasing duration. Although initially magnesium sulfate was discontinued, it was restarted and administered as tolerated through to hospital day 7 when the patient vomited and complained of abdominal discomfort. At this time, external fetal monitoring showed fetal bradycardia with minimal variability and it was decided to proceed with a category 1 cesarean delivery.

The patient was transferred to the operating suite where standard American Society of Anesthesiologists monitoring was commenced. General anesthesia was induced with intravenous propofol 150 mg. Rapid-sequence intubation was facilitated with succinylcholine 150 mg and anesthesia maintained with sevoflurane 1.5% in oxygen. A low-transverse cesarean delivery was performed. The fetus was delivered within 2 min of incision, but died shortly thereafter (Apgar scores 1

Table 1 Laboratory investigations during hospital course

Hospital day	1		6		7			8			9		10	16
Time	18:00	06:00	03:00	07:30	10:00	16:00	20:00	00:00	04:00	08:00	20:00	10:00	06:00	06:00
Event location	Labor ward		CD	Recovery Area		ICU		OR1			ICU	OR2	ICU	Ward
Hemoglobin (g/dL)	11.2	11.6	11.7	4.2	6.0	7.4	8.8	8.2	6.8	7.9	9.2	6.9	8.3	7.9
Hematocrit (%)	33.1	32.7	32.8	12.1	17.1	20.7	25.4	23.1	19.8	22.2	25.4	20.1	24.2	22.9
White cells ($\times 10^9/L$)	7.9	8.1	7.7	3.6	8.8	17.8	13.6	16.6	18.3	16.8	12.3	9.1	10.9	13.4
Platelets ($\times 10^9/L$)	207	204	175	20	30	41	78	83	67	102	86	63	45	202
Glucose (mmol/L)	4.0	5.0		3.3	7.6	3.3	3.8	4.4	14.6	8.4	6.0	6.8	6.9	4.9
Creatinine (mg/dL)	0.76	0.70		0.39	1.22	2.10	2.00	2.38	2.78	2.73	2.78	2.51	1.82	0.73
Lactate (mmol/L)						8.5		9.5	8.7	5.8		3.2	1.5	
AST (U/L)	20	31				706	648	1053	1572	1969	1903	1135	674	51
ALT (U/L)	10	25				533	629	1264	1714	1736	1306	1229	1082	203
Bilirubin _{total} (mg/dL)	0.2	0.2				2.3	2.3	2.5	2.0	2.6	5.3	3.5	1.9	1.0
LDH (U/L)										2242		890	526	
ALP (U/L)	90	94				105	91	98	74	87	113	129	111	96
PT (s)				30.5	26.3	23.0	20.2	22.0	25.4	24.6	20.1	19.3	16.4	15.0
PTT (s)				61.7	47.8	44.1	42.6	40.6	42.1	44.4	37.9	34.3	29.5	26.4
INR				3.1	2.5	2.1	1.8	2.0	2.4	2.3	1.8	1.7	1.4	1.2
Fibrinogen (g/L)				0.6	0.9	1.0	1.4	1.6	1.7	1.7	3.0	3.6	5.2	

CD: cesarean delivery; OR1: decompression laparotomy, followed by return to ICU; OR2: washout and closure, followed by return to ICU; AST: aspartate transaminase; ALT: alanine transaminase; LDH: lactate dehydrogenase; ALP: alkaline phosphatase; PT: prothrombin time; PTT: activated partial thromboplastin time; INR: international normalized ratio.

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