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Postpartum HELLP Syndrome Complicated by a Rare Acute Acalculous Pancreatitis

Muhammad Hairie Hakimi bin Harun[1]*, Mohd Fairudzi Afzanizam bin Mohd Salleh[2], Muhammad Firdaus bin Zaidi[3], Nor Diana binti Borian[4]

[1]Department of Obstetrics and Gynaecology, Hospital Pengajar Universiti Sultan Zainal Abidin, Terengganu, Malaysia
[2]Department of Obstetrics and Gynaecology, Hospital Sultanah Nur Zahirah, Terengganu, Malaysia
[3]Department of Family Medicine, Hospital Universiti Sains Malaysia, Kelantan, Malaysia
[4]Department of Radiology, Hospital Sultanah Nur Zahirah, Terengganu, Malaysia

*Corresponding author: hairieharun@unisza.edu.my

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Abstract
HELLP syndrome is one of the worst complications associated with severe pre-eclampsia (PE), characterized by hemolysis, elevated liver enzymes or transaminitis and low platelet count or thrombocytopenia which accounts for almost 1% of all pregnancies. Interestingly, HELLP syndrome can be complicated with acute pancreatitis. Here we are demonstrating a rare and yet interesting case of 29 years old, gravida 3 para 2 which presented to us with acute acalculous pancreatitis as a complication of postpartum HELLP syndrome. Early recognition and treatment would significantly improve maternal outcomes.

Keywords:
Postpartum, HELLP Syndrome, Acalculous, Acute Pancreatitis

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Introduction

Acute pancreatitis (AP) is an uncommon complication in HELLP syndrome during pregnancy, occurring in fewer than 0.5% of cases. Multiple factors such as gallbladder stones, metabolic disorders, and biliary tract diseases may be to blame. Conversely, HELLP syndrome may predispose to acute acalculous pancreatitis and make the disease worse. The microvascular alterations that occur during severe pre-eclampsia are the mechanism; these will be covered in further detail in the discussion below. In most situations, primary surgical treatment is recommended; however, in some unfavorable conditions, a more conservative approach may be necessary, and each case must be evaluated individually. Early detection is essential to avoid further complications.

Case report

29 years old, gravida 3 para 2 at 35 weeks period of amenorrhea with no significant antenatal issue except for maternal obesity with a body mass index of 36 kg/m2 was initially referred to a tertiary hospital in view of facial puffiness and new onset of erythematous rashes over the right nasolabial for one week duration. She also had on and off fever for a few days. She otherwise denied any other significant history suggestive of infection such as upper respiratory tract infection, urinary infection, premature rupture of membrane and is asymptomatic of labor. She has no significant of excessive weight gain in a short period of time.

Her vital signs were stable except she had a fever of 38.5. Her blood investigation revealed hemoglobin (Hb) level 11 g/L, total white cell (TWC) $6.4x10^9$/L, platelet $207x10^9$/L, and C-reactive protein (CRP) level 50 mg/L. She was treated as an occult infection and was investigated for autoimmune disease. Blood aerobic culture and sensitivity was sent and no growth documented. She was then started with IV rocephin and intravenous fluid of 4 pints over 24 hours with proper I/O charting and monitoring. Autoimmune screening was done and manifested as seropositive systemic lupus erythematosus (SLE) with The European Alliance of Associations for Rheumatology (EULAR) score of 17. The echocardiogram done was normal.

A transabdominal scan was done and revealed fetal growth restriction (FGR) with normal umbilical artery doppler (UAD).

Multi-disciplinary discussion among the respective teams involving obstetricians, neonatologist, rheumatologist and anesthesiologist agreed to treat as active systemic lupus erythematosus (SLE), and was started with a low dose of oral prednisolone. Thus, she was decided for early delivery via induction of labor (IOL). However, she had spontaneous preterm vaginal delivery and delivered a good Apgar score baby with weight of 1.8kg.

Unfortunately, on day 2 postpartum, she began to complain of sudden onset epigastric pain that required regular analgesics. She had episodes of hypertensive crisis ranging 163–180/95-110mmHg with significant proteinuria. Preceding that, her blood pressure was normotensive ranging 120-135/80-85 mmHg and no episode of proteinuria was noticed. She otherwise denied any other symptoms of impending eclampsia. Otherwise, she had no increase per vaginal bleeding and normal lochia seen.

Her abdomen was soft but tender over the right hypochondriac region with negative Murphy sign. She was not jaundiced and no stigmata of chronic liver disease were seen.

A formal ultrasound of the hepatobiliary system (HBS) was performed; there is focal fatty infiltration at the left lobe of the liver suggesting possible early abscess formation and no sonographic evidence of gallbladder empyema.

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Computer tomography (CT) was done; the pancreas was not bulky, there was no focal pancreatic lesion, the pancreatic duct was not dilated, and there was no peripancreatic fat stranding or collection of free fluid seen. Her renal profile and venous blood gas was normal. Other blood investigations were listed in Table 1 below.

She was treated as postpartum HELLP syndrome complicated with acute acalculous pancreatitis.

Intravenous (IV) magnesium sulfate was commenced and optimization of blood pressure was achieved with infusion hydralazine. IV Human Albumin was initiated for 3 days with adequate hydration. Meanwhile, the antibiotic was escalated to IV tazocin to complete for one week.

She was discharged home after 10 days of admission and scheduled for regular follow-up under rheumatology and gastroenterology teams. She was doing well and her daily blood pressure was controlled and remained normotensive on regular oral labetalol 200mg trice a day.

Table 1: There is elevation of the liver enzymes (transaminitis), elevation of serum LDH and thrombocytopenia to make a diagnosis of HELLP syndrome. Significant elevation of serum amylase gives a clue of acute pancreatitis.

Blood Parameters	Prior delivery		Day 2 postpartum	Day 4 postpartum	Day 6 postpartum
Serum Amylase, units/L	[redacted]		**1282**	769	207
Lactate Dehydrogenase (LDH), units/L	577	779	**854**	562	511
Alanine Transaminase (ALT), units/L	22	100	**220**	115	80
Aspartate Transaminase (AST), units/L	63	109	**172**	79	19
Alkaline Phosphatase (ALP), units/L	203	176	**496**	261	212
C-Reactive Protein (CRP), mg/L	50	49.9	**43.7**	38.3	4.5
Albumin, g/L	24	25	**22**	24	27
Full blood count (FBC)	Hb 11 TWC 6.4 Plt 207		**Hb 9.2** **TWC 13.6** **Plt 122**	Hb 9.4 TWC 11.6 Plt 159	Hb 9.8 TWC 10.5 Plt 212
Urine Protein Dipstick	3+ (0.5g/24H)				

Discussion

Acute pancreatitis (AP) is the inflammation of the pancreas. Its clinical manifestation can be varied from mild to severe form that may require hospitalization. AP in pregnancy itself is an uncommon condition complicating 1 in 1000 to 1 in 5000 deliveries or approximately 0.1-0.5%.

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Even so, the incidence of the AP has become more frequent since the past few decades[1]. AP is usually associated with cholelithiasis, alcohol consumption, biliary tract disease and hyperlipidemia[1,2,3]. In this present case, none of the aforementioned aetiologies were found. However, even though AP that is complicated by severe pre-eclampsia (PE) or HELLP syndrome is uncommon, but the trend is increasing in number among pregnant women. The presence of the AP in HELLP syndrome patients will complicate and challenge the management of PE. Cuiqin Sang et al. observed that 5 out of 407 patients with severe PE complicated with AP, or around 1.2% of the total, in a cohort of 21,466 births. This would explain that severe PE could be a probable risk factor for development of AP.

Severe PE is correlated with microvascular changes in splanchnic circulation resulting in arterioles vasoconstriction, micro thrombosis, vasculitis and disseminated intravascular coagulopathy which compromising the functions of multiple systems and organs including the pancreatic gland that further susceptible to splanchnic ischemia[2,3]. On the other hand, the endothelial injury, disposition of the fibrin in the lumen of the vessels with the activation of platelet and vascular epithelium in HELLP syndrome[6]. will result in more production of the thromboxane A2 and serotonin level. This will lead to platelet aggregation and will damage the endothelium and impaired the production of prostacyclin thus causing imbalance of the thromboxane A2/prostacyclin ratio as seen in severe PE and HELLP syndrome patients[7]. The other possible mechanism is by the inflammatory mediators activation that triggers the inflammation of the pancreatic tissue which subsequently provokes development of AP[2,8,9,10].

Even AP is rare but it mostly occurs during the third trimester or early postpartum[4,5] as seen in our patient whereby she was complaining of sudden onset of the right hypochondriac region post-delivery necessitating regular analgesics postpartum. Kirk D Ramin in the literature mentioned that women who are diagnosed with AP will be exhibited with a new onset of upper abdominal tenderness and pain in association with nausea and vomiting, and biochemically elevation of serum amylase level more than 3 times of the upper normal limit[1]. Our patient similarly had the same presentation with marked increases in the level of serum amylase.

Managing patients with AP in the presence of severe PE and HELLP syndrome is very challenging. Because of the bleeding tendencies seen in the patients with HELLP syndrome, primary surgical treatment is not recommended as it will be significantly problematic to control hemorrhage intraoperatively and may end up with maternal mortality complications. [3] Thus, conservative management with proper hydration, optimum analgesics and antibiotic administration is more favorable in this situation to avoid such complications[11,12].

In conclusion, the possible complications of HELLP syndrome should be explained to all the patients intrapartum, postpartum and in the future because the recurrence rate can be as high as 50%. Early recognition of AP in HELLP syndrome would significantly reduce the fatal outcomes.

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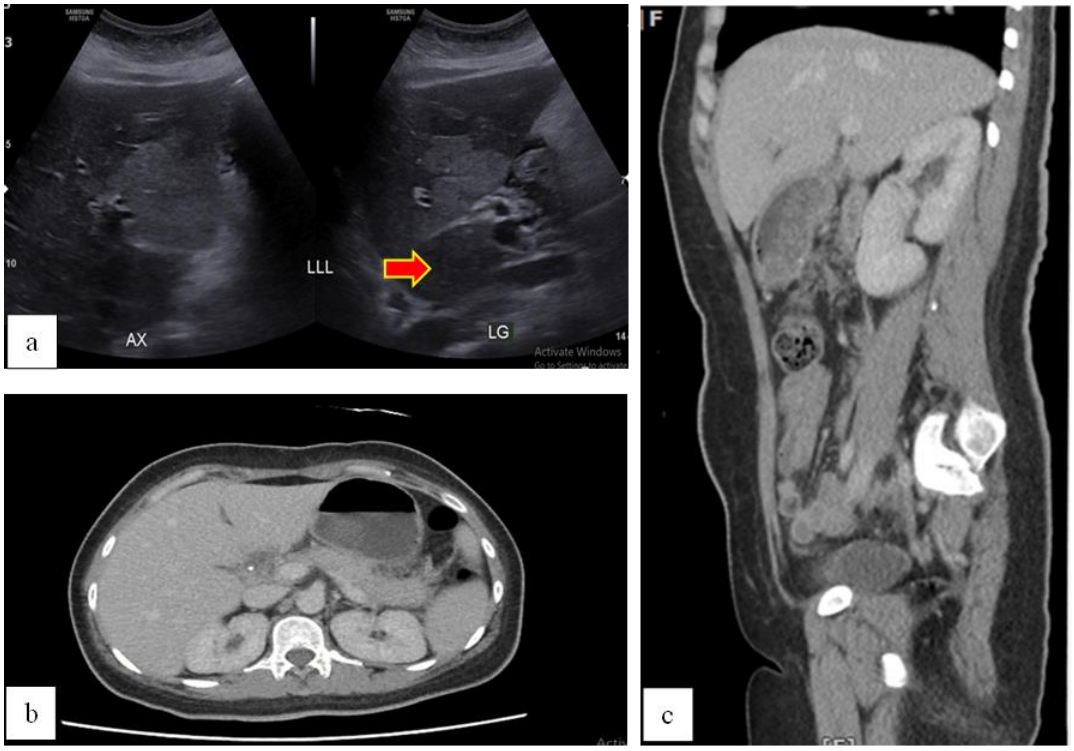

a) Liver has normal parenchymal echogenicity, homogenous echotexture and smooth surface. The intrahepatic and extrahepatic biliary trees are not dilated. Common duct is not dilated measuring 0.4cm in calibre. Portal vein is not dilated measuring 0.9cm in diameter. Gallbladder is well distended with clear bile. Gallbladder wall not thickened. No gallbladder calculi or peri-cholecystic collection (see the red arrow). No sonographic evidence of gallbladder empyema. Unable to visualize pancreas due to being obscured by bowel gasses.

b) and c) Pancreatic is not bulky, no focal pancreatic lesion. Pancreatic duct is not dilated. No peri-pancreatic fat stranding, collection or free fluid seen.

Conflict of interest

The authors declared no conflicts of interest

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