

Acute Severe Pancreatitis in Pregnancy Masquerading as Partial Hemolysis Elevated Liver enzymes Low Platelet (HELLP) Syndrome

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Received: March 31, 2015; Accepted: April 14, 2015

Introduction: Acute severe pancreatitis may result in biochemical abnormalities resembling those seen in Hemolysis Elevated Liver enzymes Low Platelet (HELLP) Syndrome.

Case Presentation: A 17-year-old female with 27 weeks of gestation presented mild acute pancreatitis. Based on the criteria of persistent Systemic Inflammatory Response Syndrome (SIRS) she subsequently developed severe pancreatitis. Bilirubin 2.2 mg/dL, lactate dehydrogenase 2171 IU/L and platelet of 53000 mm³ after 48 hours of the onset of pain, also indicated the possibility of partial HELLP syndrome. However, the results of the differential diagnosis ruled out the presence of Disseminated Intravascular Coagulation (DIC), Thrombotic Thrombocytopenic Purpura (TTP), Systemic Lupus Erythematosus (SLE) and Anti phospholipids syndrome. We terminated her pregnancy due to the above-mentioned diagnoses and postponed the cholecystectomy.

Conclusions: Termination of pregnancy was performed as it would save the patient's life in either deteriorated acute severe pancreatitis or HELLP.

Keywords: Pancreatitis; Syndrome; Pregnancy

1. Introduction

Acute pancreatitis is a clinical condition, which is classified by mild, moderate and severe types. In its severe type the resultant biochemical abnormalities may resemble those seen in Hemolysis Elevated Liver enzymes Low Platelet (HELLP). The reported incidence of acute pancreatitis in pregnancy varies in different studies as a result of differences in age, genetic and habits; incidence in Mid-western USA was 1 in 3021 pregnancies (1, 2). Incidence is higher during the third Trimester of pregnancy (3). In recent studies the reported mortality of mothers has decreased to less than 1%, and prenatal mortality is from 0 to 18% (2). The main cause of prenatal mortality is preterm delivery in acute pancreatitis and its decline is due to improved care in specialized neonatal intensive care units. The reduction in maternal mortality is due to earlier diagnosis (2, 4). The common causes of acute pancreatitis in pregnancy include gallstones (65%), alcohol abuse (10%), idiopathic complications (15%) and hyperlipidemia (5%) (4). Gallstones can result in transient or permanent obstruction of the pancreatic duct thereby causing acute pancreatitis (1, 4). To establish the diagnosis of acute pancreatitis, two of the following criteria must be present: (I) abdominal pain consistent with the disease, such as continuous pain in the upper abdomen (II) serum amylase

and / or lipase greater than three times the upper limit of normal, and (III) characteristic findings from abdominal imaging for those who fail to improve clinically within the first 48 - 72 hours (1). The aim of this report was to establish the point that in case of deteriorated Acute Severe Pancreatitis in Pregnancy masquerading as Partial Hemolysis Elevated Liver enzymes Low Platelet (HELLP) Syndrome, termination of pregnancy would still save the life of the patient.

2. Case Presentation

A 17-year-old female patient at the 27th week of her first pregnancy presented severe epigastric pain accompanied with nausea and vomiting for 11 hours. The pain used to be aggravated by supine and relieved with bending position. Clinical findings were Blood Pressure (BP): 130/80 mmHg, Pulse Rate (PR): 86/minute, Respiratory Rate (RR): 22/minute, and Oral Temperature (OT): 37.3°C. Upon abdominal examination there was tenderness in epigastrium with Fundal Height (FH) 28 week and Fetal Heart Rate (FHR): 146/minute. She was admitted to the labor ward and investigations were requested. On urgent abdominal ultrasonography, her gallbladder had a normal size and wall thickness containing several stones with normal

biliary ducts, yet evaluation of pancreas was not possible and active fetus with gestational age of 27 weeks and four days, with regular fetal heart rate, adequate amniotic fluid and posterior placenta was found. Acute pancreatitis was diagnosed as per her laboratory findings (Table 1). During the next day, the patient developed oliguria of about 20 cc/hour and had BP: 120/70 mmHg, PR: 115/min and axillary temperature: 37.50°C, yet after treatment 100 cc/hour urinary output was achieved therefore possibility of Systemic Inflammatory Response Syndrome (SIRS) was considered. Magnetic Resonance Cholangiopancreatography (MRCP) on the same day showed acute pancreatitis; findings included a swollen pancreas with fat stranding with gallstones and normal biliary ducts with accompanying ascites and left pleural effusion. On the same day, with respect to her laboratory data, indicating deteriorated severe acute pancreatitis masquerading as partial HELLP, the patient was sulfated and underwent caesarian section with midline incision and about 100cc of the ascites were released. A boy with an Apgar score of 5/8 with 1100g of weight and presentation of footling breech was born. The pancreas was severely inflamed and no evidence of cholecystitis was found. During the surgery she received 1 IU of packed RBC, 7 IU of platelet and 1

IU of Fresh Frozen Plasma (FFP) and after the surgery in the ICU another 1 IU of packed RBC, one unit of whole blood, 3 IU of platelet and 4 IU of FFP were administered, and on the morning of the next day she also received one unit of whole blood. At this point she developed densely colored urine with an output of 200 cc/hour while creatinine was 1.1 mg/dL and urinalysis revealed protein 3+, blood 3+, RBC >100. After two hours, the patient developed respiratory distress and Central Venous Pressure (CVP) line was obtained. Abdominal ultrasonography revealed 3 - 4 cc of ascites and about 25 cc of left pleural effusion, of which 10 cc was aspirated and found to be exudative with amylase of 1556 mg/dL. At the same time, the patient had a respiratory rate of 22/minute and mild bilateral decreased breath sounds with no crackles and with nasal O₂ of 4 L/minute, and O₂ saturation of 95%. In Arterial Blood Gas (ABG) she had PH: 7.39, PCO₂ 2.28 mmHg, PO₂: 79 mmHg, HCO₃: 16.5 mmol/L, and O₂ saturation: 92%; i.e. metabolic acidosis with respiratory compensation and mild degree of Acute Respiratory Distress Syndrome (ARDS) because of P/QO₂/P/IO₂ of 200. Abdominal Computerized Tomography (CT) with oral and Intravenous (IV) contrast was performed and the patient was advised to attend the surgical outpatient department.

Table 1. Laboratory Parameters^a

| | 9.9.2013 (Admission) | 10.09.2013 (9 AM) | 10.09.2013 (6 PM) | 11.09.2013 | 12.09.2013 | 18.09.2013 |
|------------------------------|----------------------|-------------------|-------------------|------------|------------|------------|
| WBC, PMN % | 14100 (73) | 20000 (87) | 22400 (-) | 15900 (93) | 17000 (88) | 16500 |
| Hb, g/dL | 12.3 | 10.6 | 9.5 | 8.5 | 8.8 | 7.8 |
| Plt, /mm ³ | 282000 | 75000 | 53000 | 46000 | 41000 | 385000 |
| Schistocyte, % | - | 1.5 | 3.5 | slight | - | - |
| AST, IU/L | 110 | 67 | - | 42 | - | - |
| ALT, IU/L | 62 | 37 | - | 21 | - | - |
| LDH, IU/L | 484 | 1723 | 2171 | 2050 | 1818 | 496 |
| Total Bilirubin, mg/dL | 1.5 | 2.2 | - | 2.2 | 1.7 | - |
| Indirect Bilirubin, mg/dL | 0.7 | 1.6 | - | 1.6 | 0.9 | - |
| Amylase, IU/L | 690 | 636 | - | - | - | 88 |
| Lipase, IU/L | 625 | - | - | - | - | - |
| Calcium, mg/dL | 9.3 | 8.5 | 8.2 | 9 | 8.6 | - |
| BUN, mg/dL | 8 | 20 | 24 | 35 | 48 | 7 |
| Creatinine, mg/dL | 0.6 | 1.4 | 1.3 | 1.6 | 1.6 | 0.9 |
| Triglyceride, mg/dL | - | - | - | - | 217 | - |
| C-reactive protein | - | 115 | 146 | 140 | 76 | - |
| Urine protein | 1+ | - | - | 3+ | 2+ | - |
| Urine blood | - | 2+ | - | 3+ | 3+ | - |
| urinary protein, gr/24 hours | - | - | - | - | 1.3 | - |
| Urinary dysmorphic RBC | - | - | - | - | negative | - |
| PT, Sec. | 14 | 15 | - | 14 | 14 | - |
| PTT, Sec. | 28 | 40 | - | 35 | 28 | - |

^a Abbreviations: ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; BUN, Blood urea nitrogen; Hb, Hemoglobin; LDH, Lactate dehydrogenase; Plt, Platelet; PMN, polymorphonuclear; PT, Prothrombin time; PTT, Partial thromboplastin time; RBC, Red blood cells; WBC, white blood cells.

3. Discussion

Our patient initially presented mild acute pancreatitis during pregnancy. Her pancreatitis was probably due to biliary causes although her aminotransferases, i.e. Aspartate Aminotransferase (AST) and Alanine Aminotransferase (ALT), were less than 150 while they should have been more than 150 (with 95% positive predictive value) to fulfill the criteria for being biliary (5). On the second day of admission although the increase in her oliguria and creatinine level could have been partially due to prerenal causes (as the former improved with further hydration and furosemide), yet the deterioration of her pancreatitis towards the severe form was still possible as her initial creatinine of 0.6 mg/dL increased gradually to 1.6 on the third day and fell to 0.9 on the eighth day as an evidence of organ failure lasting for more than 48 hours; these findings fulfill the criteria of severe pancreatitis (6). Abdominal CT with Intra Venous (IV) and oral contrast on the sixth day showed an inflamed pancreas with inflammation of its surrounding fat with ascites and left pleural effusion with normal parenchyma, which indicated non-complicated pancreatitis. More than 48 hours from the onset of pain (after 38 hours of admission) with fall of platelet to 53000 and increment of Lactate Dehydrogenase (LDH) to 2171 mg/d, schistocyte of 3.5% and polychromasia of 2% with hemoglobin of 9.5 g/dL (12.3 on admission), and bilirubin of 2.2 mg/dL, the possibility of masquerading partial HELLP was considered. If out of the four criteria for the diagnosis of complete HELLP, namely presence of hemolysis, elevated level of lactate dehydrogenase, total bilirubin > 1.2 mg/dL, elevated alanine aminotransferase greater than two folds, two criteria are met partial help is diagnosed (7). Association of HELLP with pancreatitis is very rarely reported, although our patient had masquerading partial HELLP and as there is no consensus for the diagnosis of HELLP, termination of pregnancy was performed. This was a sound decision because even if the complication was solely due to deteriorated severe pancreatitis, the same procedure could have helped due to the persistent SIRS that lasted more than 48 hours (Table 2) (3, 8, 9). However, based on another study, pregnancy could have been continued to term (10). Her pancreatitis should have been managed conservatively (3). Antiphospholipid syndrome and systemic lupus erythematosus were also ruled out as other differential diagnoses of HELLP syndrome by the following laboratory data AbIgG < 3, lupus anticoagulant 39.3, Antinuclear Antibodies (ANA) 0.1, Anti ds-DNA 0.1, C3 110 mg/dL, C4 23 mg/dL (11). Based on the recommendations advising postponement of cholecystectomy for at least three weeks following the

presentation of acute severe pancreatitis due to the risk of increased infection, her cholecystectomy was postponed (3). Our success was to have both the mother and the child alive.

Table 2. Systemic Inflammatory Response Syndrome Criteria ^a

| | Criteria | Patient's |
|--|----------|-----------|
| Heart Rate, /min | > 90 | 115 |
| WBC, /mm ³ | > 12000 | 20000 |
| Plt, /mm ³ | < 100000 | 75000 |
| Creatinine increment, mg/dL | > 0.5 | 0.8 |
| Urine Output, mL/kg/hour for two hours | < 0.5 | 0.33 |

^a Abbreviations: Plt, Platelet; WBC, White Blood Cells.

Acknowledgements

The authors are grateful for the valuable coordination and dedicated cooperation of the Clinical Research Development Unit of Velayat hospital.

References

1. Tenner S, Baillie J, DeWitt J, Vege SS, American College of G. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol.* 2013;**108**(9):1400-15.
2. Tang SJ, Rodriguez-Frias E, Singh S, Mayo MJ, Jazrawi SF, Sreenarasimhaiah J, et al. Acute pancreatitis during pregnancy. *Clin Gastroenterol Hepatol.* 2010;**8**(1):85-90.
3. Sun Y, Fan C, Wang S. Clinical analysis of 16 patients with acute pancreatitis in the third trimester of pregnancy. *Int J Clin Exp Pathol.* 2013;**6**(8):1696-701.
4. Ducarme G, Maire F, Chatel P, Luton D, Hammel P. Acute pancreatitis during pregnancy: a review. *J Perinatol.* 2014;**34**(2):87-94.
5. Coffey MJ, Nightingale S, Ooi CY. Predicting a biliary aetiology in paediatric acute pancreatitis. *Arch Dis Child.* 2013;**98**(12):965-9.
6. Stevens T, Parsi MA, Walsh RM. Acute pancreatitis: problems in adherence to guidelines. *Cleve Clin J Med.* 2009;**76**(12):697-704.
7. Asherson RA, Spargo C, Gomez-Puerta JA. Partial HELLP syndrome in pregnancy complicated by recurrent deep vein thromboses and palmar skin lesions in a patient with prothrombin gene 20210a mutation and antiphospholipid antibodies: an unusual case. *Clin Rheumatol.* 2008;**27**(2):245-8.
8. Qihui C, Xiping Z, Xianfeng D. Clinical study on acute pancreatitis in pregnancy in 26 cases. *Gastroenterol Res Pract.* 2012;**2012**:271925.
9. Mofidi R, Duff MD, Wigmore SJ, Madhavan KK, Garden OJ, Parks RW. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. *Br J Surg.* 2006;**93**(6):738-44.
10. Juneja SK, Gupta S, Virk SS, Tandon P, Bindal V. Acute pancreatitis in pregnancy: A treatment paradigm based on our hospital experience. *Int J Appl Basic Med Res.* 2013;**3**(2):122-5.
11. Haram K, Svendsen E, Abildgaard U. The HELLP syndrome: clinical issues and management. A Review. *BMC Pregnancy Childbirth.* 2009;**9**:8.