Journal of Diabetes and Metabolism
ISSN: 2155-6156

Chen et al., J Diabetes Metab 2012, 3:3
DOI: 10.4172/2155-6156.1000185

Case Report Open Access

Acute Fatty Liver of Pregnancy in a Woman with Type 2 Diabetes

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Abstract

We report a 35 year-old woman with type 2 diabetes who developed acute fatty liver of pregnancy in her 35th week of pregnancy. She presented with nausea, vomiting, elevated transaminases, elevated bilirubin, renal impairment, and coagulopathy. Her condition improved dramatically after an emergency cesarean section.

Keywords: Acute fatty liver of pregnancy; Diabetes mellitus

Introduction

Acute Fatty Liver of Pregnancy (AFLP) is a rare life-threatening complication of pregnancy. The estimated incidence of AFLP was 5 cases per 100,000 pregnancies and case fatality rate was 1.8% in a prospective study [1]. Reported maternal mortality has ranged from none to 11% [2-4]. Non-alcoholic Fatty Liver (NAFL) is very common in type 2 diabetes [5]. However, the occurrence of AFLP in type 2 diabetes has not been previously reported. AFLP occurring in women with diabetes could be very dangerous. We report a rare case of AFLP in a woman with type 2 diabetes and raise an issue on the linkage between type 2 diabetes and AFLP with a brief literature review.

Case Report

A 35-year-old woman in her 35th week of pregnancy was hospitalized via the emergency department with nausea, vomiting, and malaise for 1 week in February 2011. She had been diagnosed as type 2 diabetes in January 2010 with fasting sugar 245 mg/dl, Hb A1c 9.9% (HPLC 4.3 - 6.5%), and C-peptide 1.38 nmol/l (normal 0.9-4.0 nmol/l) . Her body height was 160 cm, weight 58 kg, Body Mass Index (BMI) 22.6 kg/m^2 and blood pressure 122/87 mmHg. Her mother has type 2 diabetes under oral hypoglycemic agent treatment. Her younger brother and sister are well without other autoimmune disease. Although Latent Autoimmune Diabetes in Adult (LADA) is possible, we cannot prove it without islet autoantibodies. Since then, she had been treated with premixed insulin in preparing for a planned pregnancy which was diagnosed in June 2010, her last menstrual period having occurred in May. A1C levels were 7.4% and 7.3% in July and September. She was doing well until 1 week before visiting the emergency department due to persistent symptoms despite prior visits to the obstetrical and medical outpatient clinics. On physical examination, she was alert, appeared ill, and not icteric. Laboratory data were aspartate aminotransferase (AST) 550 U/l (normal 0-35 U/l), alanine aminotranferease (ALT) 651 U/l (normal 0 - 32 U/l), alkaline phosphatase (ALP) 1106 U/l (normal 125 - 250 U/l), LDH 218 (normal 98 - 192 U/l) total bilirubin (T Bil) 1.4 mg/dl (normal 0.2 - 1.2 mg/dl) , creatinine (Cr) 1.39 mg/dl (normal 0.8 - 1.3 mg/dl), activated partial thrombin time (APTT) 39.4 s (normal 28.2 s) and prothrombin time (PT) 13.1 s (normal 10 s),. Viral hepatitis A, B, and C were excluded by negative results for anti-HAV IgM, HBsAg, and anti-HCV Ab. Gallbladder stones were shown with abdominal ultrasound.

After hospitalization, diabetes insipidus was also diagnosed and treated with DDAVP for a short period. Her condition progressively deteriorated with elevated levels of Cr to 1.53 mg/dl and 1.93 mg/dl, ALP to 1168 U/l, GPT to 704 U/l, and T Bil to 2.2 mg/dl and 3.9 mg/dl, as shown in Figure 1. Elevated plasma uric acid 9.9 mg/dl (normal 2.4 - 6.0 mg/dl), lower albumin level 2.7 g/dl (normal 3.5 - 5.5 g/dl), and calcium 7.1 mg/dl (normal 8.4 - 10.4 mg/dl) were also found. The following days of hospitalization, PT was slightly increased to 13.8 s. Because of this, an emergency cesarean section was performed on the 5th day of hospitalization and a healthy 2665 gm male baby was delivered. Her condition improved dramatically with lowering of AST to 34 U/l, ALT to 62 U/l, ALP to 747 U/l, and T bil to 1.8 mg/dl (Figure 1). Direct bilirubin (D bil) was 0.7 mg/dl (normal 0 - 0.4 mg/dl). Her postpartum condition was good in March 2011 with A1C 6.5%. Fatty acid oxidation screening was normal in the baby. Her C-peptide level was 1.54 nmol/l in August 2011.

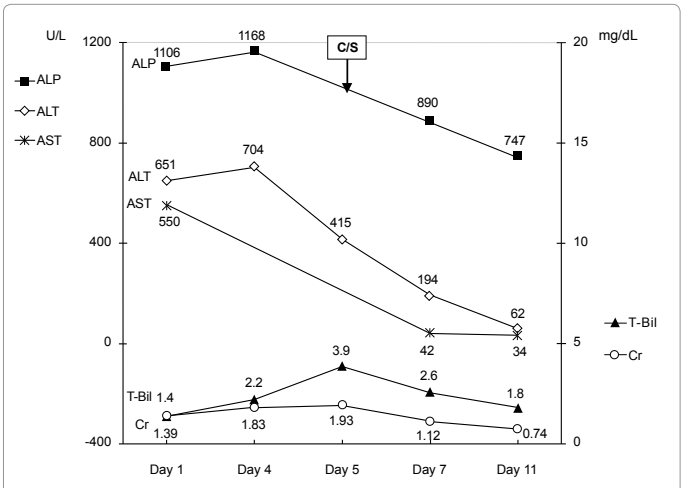

Figure 1:

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Received February 21, 2012; **Accepted** April 22, 2012; **Published** April 26, 2012

Citation: Chen KW, Yang CC, Li YM, Lee YS (2012) Acute Fatty Liver of Pregnancy in a Woman with Type 2 Diabetes. J Diabetes Metab 3:185. doi:10.4172/2155-6156.1000185

Discussion

This is a rare case of AFLP coexisting with type 2 diabetes and the first such case reported in Taiwan. There have been 18 patients with AFLP reported in a tertiary medical center in Taiwan over a 22-year period [4]. The diagnosis of AFLP in our case was made by typical history, symptoms and laboratory findings. There was no pathological confirmation by liver biopsy due to safety considerations and urgency for emergency delivery. We made the diagnosis of AFLP based upon the presence of at least 9 (vomiting, abdominal pain, elevated transaminases, elevated bilirubin, elevated urate, leucocytosis, polydipsia/polyuria, renal impairment, and coagulopathy) of the 14 criteria used in a previous report and absence of another explanation [6]. Diabetes insipidus occurring in AFLP has been reported in other reports [7,8]. However, how they are associated has not been well studied. The prognosis of AFLP is influenced by the interval between occurrence of AFLP and delivery [3]. Lower mortality was also found in mothers and babies following cesarean section as compared with vaginal delivery [3].

The mechanism of AFLP has not been fully elucidated. Natarajan et al. [9] reported that increased oxidative stress and accumulation of toxic mediators such as arachidonic acid were found in the placental mitochondria and peroxisomes of women with AFLP. Women with acute liver disease during pregnancy may have a genetic mutation in long-chain hydroxyacyl-CoA dehydrogenase (LCHAD). However, heterozygosity in the mother cannot alone account for the adverse effects and homozygous or compound heterozygous genetic defects in the fetus is the best known cause [10]. Nineteen percent of the offspring of women with AFLP might have LCHAD deficiency [11].

Screening of fatty acid oxidation including LCHAD with tandem mass spectrometry was performed and this was normal in our patient's baby. However, diverse etiological factors have been suggested in the Chinese population after genetic studies of LCHAD in proband and relatives [12]. Pathogenesis other than LCHAD still awaits further elucidation. Acute fatty liver can recur in subsequent pregnancies, even if LCHAD mutations are absent.

Although the association of non-alcoholic fatty liver (NAFL) with in type 2 diabetes has been reported [5], the association between AFLP and type 2 diabetes has not been previously reported as far as we know. We describe this case to call more attention to the association of AFLP and type 2 diabetes.

Acknowledgement

We are grateful to Professor Wilfred Y. Fuji motor, University of Washington, for his review of this manuscript.

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