Early Development of HELLP Syndrome: A Case Report

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Key Words
HELLP
Preeclampsia, treatment of

Abstract
The HELLP syndrome is a severe complication of pregnancy. It usually occurs in the middle of the third trimester. In our case, however, a very early development is reported.

Introduction
The HELLP syndrome (H for hemolysis, EL for elevated liver enzyme values and LP for low platelet counts) is a considerable complication of pregnancy [1], which occurs usually in the third trimester. We report, however, a very early and acute development of HELLP as a contribution to the now accepted picture of the entity.

Case Report
A.V.A. (N 8439/16th May. 1989) a 28-year-old primigravida in the 20th week of gestation was admitted because of elevated arterial tension (170/110 mm Hg), headache and severe edemas. She denied having prior renal or hypertensive diseases; family history was also negative. Somatic investigation revealed that the uterus corresponded to the term of gestation. Cardiovascular, pulmonary and abdominal examination produced no particular abnormalities. The course of the most relevant laboratory data is presented in table 1. As shown liver enzyme values increased rapidly after admission; platelet counts decreased strongly in the last 12 h.
There were also significant oliguria – 375 ml high concentration urine for the first 24 h – and proteinuria of 2+ on admission but never more than 3 g/l by the next examinations. Serum creatinine (144.7 µmol/l) and urea (9.7 µmol/l) were slightly elevated and did not increase further. Serum uric acid was significantly high on admission (470 µmol/l) and remained above normal range. Peripheral blood smear displayed burr cells and schistocytes; fibrinogen split products were more than 40 µg/ml by semiquantitative measurement (table 1). Ultrasonic scanning of the liver presented decreased sonodentity. There was one live fetus with a slight growth retardation, biparietal diameter 42 mm and thorax diameter 36 mm in the uterus. On Table 1. Laboratory data Parameters SGOT (ASAT) (0–40), IU/l SGPT (ALAT) (0–40), IU/l Platelets (300–150), × 109/l Total bilirubin (3.4–21.0), µmol/l Conjugated bilirubin (0.8–8.5), µmol/l Hemoglobin (110–150), g/l Hematocrit (35–45), % Total serum protein (58–80), g/l Time after admission, h

Discussion and Conclusion
References

Clinical observations of the HELLP syndrome have a relatively short history. Pritchard et al. [2] reported in 1954 3 cases with hemolysis and thrombopenia. Twenty years later Killam et al. [3] published their observations of 5 hypertensive pregnant women with signs of liver lesions and development of DIC. Goodlin [4] presented in 1976 16 cases of pregnancy, complicated by hypertension, thrombopenia and abnormal liver enzyme values, falsely interpreted as unrelated to gestation disorders. However, it was not until 1981 that Weinstein [1] systematized all published data and his own experience and postulated a new entity, the HELLP syndrome, establishing its clinical and laboratory characteristics. Various manifestations of this syndrome are described in the literature we reviewed, but all the reported cases developed in the middle and late second half of the pregnancy. Recently Schaldach et al. [5] presented a ‘late’ HELLP syndrome after delivery. In our case the typical signs were found unusually early, i.e. in the 20th gestational week and they developed extremely rapidly. Pregnancy aggravated by HELLP syndrome is related to very high maternal and fetal (neonatal) morbidity and mortality. Opinions on treatment of such patients are, however, extremely contradictory. While some authors suggest a prompt delivery [1,3, 6], others prefer a short period of waiting in order to achieve better fetal maturity [7–9]. Early development of the HELLP syndrome is possible and should not surprise obstetricians. In such cases prompt delivery has to be performed, because long-lasting conservative management could only deteriorate the mother’s condition.