A case of Ballantyne syndrome
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Objective
To present a case study of the Ballantyne syndrome (the Mirror Syndrome) in the 31-multigravida, associated with rapidly progressing non-immune fetal hydrops in 22/24 week of pregnancy, mirroring with severe maternal oedema and anaemia of unknown origin, resulting in intrauterine fetal demise in 24wk.

Methods
31-year-old multigravida (history of 1 uncomplicated pregnancy, spontaneous, term delivery, healthy newborn, normal development) was referred to the Department of Obstetrics and Maternal Diseases in 16wk due to the signs of non-immune fetal hydrops. The ultrasound examination revealed the single fetus with generalised subcutaneous tissue oedema (thickness up to 11mm), ascites (up to 3.4mm), placental oedema (up to 31mm), normal amniotic fluid index (AFI) and normal maxium vertical pocket (MVP). No major fetal congenital malformation was detected on ultrasound examination. Further studies did not revealed the etiology of the fetal hydrops - amniotic fluid and serum polymerase chain reaction (PCR) for parvovirus, cytomegalovirus and toxoplasma were negative. The amniocentesis confirmed normal female 46, XX karyotype.

Results
Maternal examination was consistent with mirror syndrome - face and limbs oedema, oliguria and anemia. Blood pressure was normal, occasionally at upper normal range not requiring pharmacological treatment. There were also the severe alterations in the maternal biochemical status and clinical status - increasing lower and upper limbs oedema, face and abdominal oedema and dyspnea, fluid retention (ingested p. o. /administered i. v. fluid more than excreted), weight gain of 7kg between 17-22wk, deteriorating anaemia (low HGB, HCT) with normal ferrum level and red-cells parameters. The level of transaminases (AIAT, AspAT at normal range), there was a slight decrease in PLT but the final level was normal. There was a normal serum concentration od creatinin and urea. 24-hour urine collection revealed not significant proteuria, whereas the total serum protein, albumin and glomerual filtration rate (GFR) were decreased. There was also an increasing concentration od C-reactive protein (CRP) and D-dimer. Fetal counselling was conducted by the obstetrical and paediatric/neonatal staff and the prognosis was estimated to be very poor due to massive early fetal hydrops and threatened heart failure. After the discussion with the parents – the expectant management was introduced. In 24wk, due to regular uterine contractions and severe fetal bradycardia, after repeated counselling, the cesarean section was performed – for maternal benfit only (indication – fetal bradycardia, severe fetal hydrops). The perinatal outcome was as follows: female newborn-stillbirth in 24wk, weight 3090g, placenta was grossly edematous (weight 700g), Apgar score at 1st min – 0 pts, at 5th min. – 0 pts, umbilical artery pH 6, 82, umbilical vein pH 6, 93.

Conclusion
According to our results, we can conclude, that the mirror syndrome may develop in pregnancy complicated by NIHF of the unknown origin.