Objective
Preeclampsia originates in the placenta with a usually progressive clinical course and is only cured by delivery of the placenta. It affects 3–5% of pregnancies and is a major cause of maternal and perinatal mortality. Although the aetiology and pathogenesis remain to be elucidated, the placenta undoubtedly is involved, as termination of pregnancy eradicates the disease and delivery of the placenta is the only known cure.

Methods
We present two cases of twin pregnancies without resolution of preeclamptic symptoms after intrauterine death of one twin.

Results
Case 1: A 37 year old nulliparous woman was referred at 26 weeks of gestation because of arterial hypertension, oedema and growth restriction in one twin. On admission her blood pressure was 160/100 mmHg. In our centre, ultrasound examination confirmed growth restriction (weight estimation: 389 g) and reversed umbilical flow with cerebral redistribution in one twin, with a normal co-twin (estimated weight: 855 g). This was a dichorionic, diamniotic twin pregnancy. The quantitative analysis of proteinuria in a 24-hour urine sample taken after admission showed 2.708 g/day. Expectant management was decided, in order to avoid prematurity. The patient received alpha-methyldopa in a dosage of 1000 mg daily and magnesium sulphate in a dosage of 1500 mg daily for blood pressure control. Two days later because of intolerable nausea and vomiting we had to stop magnesium sulphate but we kept going on alpha-methyldopa. During the following three weeks, beside blood pressure was under control partially, ultrasound examinations did not show evidence of any growth of the restricted twin and confirmed severe Doppler abnormalities, while the co-twin’s assessment was reassuring. Some days later restricted twin died but doppler profiles of the surviving twin were normal. So we decided to prolong the pregnancy under close observation. At 31 weeks of gestation, a significant proteinuria reappeared accompanied by an increase in blood pressure. During the following three weeks, ultrasound examinations showed a reduced growth velocity of the surviving fetus and reversed umbilical flow. At the end of the 34th week of gestation, cesarean section has been performed and a healthy female infant weighing 1670 g was delivered, followed by a macerated female fetus of approximately 200 g. The mother recovered quickly and her blood pressure was normal on the third day postpartum. Case 2: A 33 year old nulliparous woman was referred at 27 weeks of gestation because of arterial hypertension and growth restriction in one twin. On admission her blood pressure was 170/105 mmHg. In our centre, ultrasound examination confirmed growth restriction (weight estimation: 698 g) and reversed umbilical flow in one twin, with a normal co-twin (estimated weight: 1060 g). This examination also showed us a dichorionic, diamniotic twin pregnancy. The quantitative analysis of proteinuria in a 24-hour urine sample taken after admission showed 4.850 g/day. Expectant management was decided, in order to avoid prematurity. The patient received alpha-methyldopa in a dosage of 1000 mg daily and magnesium sulphate in a dosage of 1500 mg daily for blood pressure control. During the following two weeks, beside blood pressure was under control partially, ultrasound examinations did not show evidence of any growth of the restricted twin and confirmed severe Doppler abnormalities, while the co-twin’s assessment was reassuring. At 31 weeks of gestation, restricted twin died and a significant proteinuria (6.650 g/day) reappeared but doppler profiles of the surviving twin were normal. During the following two weeks, ultrasound examinations showed a reduced growth velocity of the surviving fetus and reversed umbilical flow. At the end of the 33th week of gestation, cesarean section has been performed and a healthy female infant weighing 1370 g was delivered, followed by a macerated female fetus of approximately 800 g. We discharged the mother from our hospital after her blood pressure was normal on the fourth day of postpartum.

Conclusion
It has been known for nearly 100 years that preeclampsia is a placentical condition. But the central role of the placenta in the pathogenesis of preeclampsia is undisputed. The importance of poor placentaent as a feature of the disorder is well documented. But we still don’t know the reasons of poor placentaliation. In these cases there are two separated placentas. One of them are preeclamptic (up to pathologic report, they had low weights with fibrins and thrombosis inside the vessels) and the others were normal. We still don’t know why one of the twin placentas has abnormal developing, when both of them the are in the same maternal factors like immunity or blood pressure. One explanation of this question is local factors, like fetus or placentical localization, which could possibly effect placental growth up. Another interesting point of these cases was the secondary effects on the co-twin’s. During the time after intrauterine deaths of one twin, the surviving fetuses started to show a reduced growth velocity and reversed umbilical flow and mothers had increased blood pressure and proteinuria again. We think that all these are the proofs of late on set systemic maternal effects ( like systemic maternal endothelial activation and/or systemic maternal inflammatory response) depends on preeclampsia. In general, preeclampsia takes a progressive course without any major improvement until delivery. Few cases of resolution of preeclampsia after spontaneous intrauterine death of one twin or selective termination of a dichorionic pregnancy have been reported. However according to these cases even if the fetus die, preeclampsia could not be curable disease anytime unless placental separation come true. Because preeclampsia is a progressive clinical course which fully depends on the placenta. To our knowledge, these are the first cases describing a later reappearance of the preeclamptic symptoms in twins after the intrauterine death of one twin in the literature. Further studies are needed for a better understanding of preeclampsia and identification of the placentical factors responsible for persistence of the disease.