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A case of abnormal placentation and severe late-onset preeclampsia

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Objective

To describe the case of a patient presenting rare combination of abnormally invasive placenta (AIP) and severe late-onset preeclampsia (PE) and to conduct a review of the literature.

Methods

Case report and search for publications reporting the association of AIP and PE using the PubMed database.

Results

We report a case of a 30-year-old woman with a previous term caesarean section for breech presentation and an early miscarriage treated by curettage. At 28 weeks of gestation, she was referred for a suspicion of abnormally invasive placenta. The ultrasound examination confirmed an anterolateral left placenta praevia with loss of the hypoechoic plane in the myometrium underneath the placental bed and extreme thinning of the myometrium overlying the placenta. Those results were confirmed by a magnetic resonance imaging. Fetal growth, uterine and umbilical dopplers were normal. An elective caesarean-hysterectomy was planned at 37 weeks. However, the patient was urgently admitted the day before surgery and severe late-onset PE was diagnosed (hypertension of 200/120 mmHg, massive proteinuria and lower-extremity edema). The biochemistry results was normal, except for elevated uric acid and the soluble fms-like tyrosine kinase-1/ placental growth factor (sFlt-1/PlGF) ratio was increased up to 179. She received intravenous antihypertensive drugs and magnesium sulfate. As expected, the caesarean section was complicated by haemorrhage requiring blood transfusion. The planned hysterectomy could be realized. A vesical breech occurred intraoperatively and was treated by double-layer sutures and a Foley-catheter for 10 days. The newborn was healthy with a birthweight of 2230 grams. The patient stayed for 2 days in the intensive care unit and left the hospital after 1 week with a combination of 2 oral antihypertensive drugs. Six weeks later, she suffered from an acute subarachnoid haemorrhage due to aneurysm rupture. We found 4 case reports in the PubMed database describing the combination of AIP and PE. The pathophysiology of this association is interesting since PE develops in case of inadequate trophoblast invasion, whereas AIP is related to excessive trophoblast invasion. A decreased incidence of PE is described in case of placenta praevia, but the association between AIP and PE is not well defined in the literature.

Conclusion

The combination of AIP and severe PE is a rare condition. PE develops in case of an abnormal invasion of the spiral arteries by trophoblast cells. However, late-onset PE is caused by an imbalance between angiogenic and anti-angiogenic factors and can thus also develop in case of excessive trophoblast invasion. Further and deeper analysis of such clinical cases could help us to understand the pathophysiology of AIP and PE.