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ABSTRACT
Placental abruption (PA, abruptio placentae) is defined as premature separation of the normally sited placenta. This obstetric emergency complicates 0.2?1.3 per cent of all deliveries and it accounts for up to one third of all cases of perinatal mortality. Moreover, PA brings about fetal hypoxia and prematurity and thus short- and long-term morbidity. Placental abruption also remains a significant cause of maternal morbidity via increased rates of Caesarean section, hypovolaemia and disseminated intravascular coagulopathy. Since the aetiology and pathogenesis of placental abruption are largely enigmatic, this adverse obstetric outcome is still unpredictable and thus unpreventable.
This study was designed to elucidate the pathophysiology, epidemiology, risk factors, outcomes, and genetic background of PA. The study population consisted of 170 women with PA and 22,905 healthy control women who gave birth at Kuopio University Hospital between 1989?2002. The overall incidence of the placental abruption was 0.74%. We found that cigarette smoking, preeclampsia, grand multiparity, velamentous umbilical cord insertion, prior fetal demise, advanced maternal age (> 35 years) and previous miscarriage were independent reproductive risk factors of PA. We also showed that the outcome of PA is still poor, as the perinatal mortality rate was 10% and 59.4% of the newborns were premature. The recurrence rate of PA was as high as 11.9%. The obstetric prognosis after PA was comparable to that of the general obstetric population if there was no recurrence of PA. We also assessed the risk of PA in first- degree relatives of index patients, and cases of placental abruption appeared to cluster only in families with recurrent placental abruption.
In studies of the polymorphism in two genes involved in placental haemodynamics and in detoxification processes, we found that the low activity haplotype of the microsomal epoxide hydrolase (EPHX) gene was protective against placental abruption.
We found no association between Glu298Asp polymorphism in the gene for endothelial nitric oxide synthase (eNOS) and placental abruption in this Finnish population.
CONCLUSIONS: Despite investigation of risk factors, much remains

unclear about the underlying disease mechanisms of PA and the condition is still unpredictable.
The genetic component appears to be modest. Most cases of placental abruption seem to be sporadic, with no family history and no recurrence.
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