

Placenta accreta (PA) can be diagnosed antenatally with ultrasound or magnetic resonance imaging (MRI), clinically at delivery and by histopathological examination of hysterectomy or partial myomectomy specimens. In their population-based cohort study in the Nordic countries, Thurn et al (BJOG, 123:1348-55) define abnormally invasive placentation (AIP) as women with delivery by caesarean section (CS) assessed by the obstetrician to have AIP or a vaginal delivery assessed to be AIP requiring blood transfusion and laparotomy. To avoid including cases of common placental retention in their population, they exclude vaginal birth with difficult manual delivery if a laparotomy was not undertaken. Their data set does not include prenatal diagnosis data nor histopathological confirmation of PA.

PA is a spectrum disorder including abnormally adherent (creta or adherenta) and abnormally invasive (incretta and percreta) placentation. Placenta increta and percreta are both associated with major changes of the myometrial vasculature (extensive neovascularity) and in the case of percreta, villi penetrate the serosa and can invade into organs adjacent to the uterus. Therefore both these conditions can be reliably diagnosed clinically at laparotomy by inspecting the uterus (Jauniaux and Burton, BJOG. 2012;33:244-51). By contrast the adherent or non-invasive form of PA requires either histopathological confirmation or specific clinical diagnosis using predefined criteria such as those used by the European Working Group on AIP (published in Collins et al 2015). Common placental retention may also result in massive post-partum haemorrhage (PPH) and cannot be differentiated from superficial PA by inspection of the uterus at laparotomy. Conversely, several of the cases which did not proceed to laparotomy may have had areas of abnormal adherence. The lack of consistency in clinical definitions may explain the wide range (2-90 per 10 000 births) in the incidence of PA reported in recent epidemiological studies.

The historical series of Irving and Hertig (Surgery, Gynecol Obstet 1937;64:178-200) included 20 cases of PA, of these only one woman had had a previous CS. Similarly, in their review of 86 cases up to 1935, only one was found after a CS. Predisposing factors at that time were a previous manual delivery and/or “vigorous” uterine curettage. Until the 1950s, surgery and in particular, hysterectomy was almost the only therapeutic approach for PA and major PPH. Thurn et al found that prior PPH is a novel risk factor associated with PA. This is not surprising since conservative management including blood transfusion enables an increasing number of women with PA or PPH, with or without placental retention, to preserve their fertility.

PA remains undiagnosed before delivery in around 50% of the cases in the UK (Fitzpatrick K et al BJOG. 2014;121:62-71) and in 70% of Thurn et al cohort study. The authors present a theoretical calculation of the identification

of PA antenatally with ultrasound using an 80% sensitivity and 90% specificity. A recent systematic review and meta-analysis of 23 ultrasound studies of pregnancies at risk of PA found that the overall performance of ultrasound is excellent (sensitivity 91%; specificity 97%) (D'Antonio F et al Ultrasound Obstet Gynecol. 2013;42:509-17). These studies may overestimate accuracy because they were conducted in center specialized in prenatal diagnostic and the number of PA were small. Prenatal diagnosis of PA in general and the differential diagnosis between adherent and invasive PA is essential to improve the maternal and neonatal outcome of abnormal placentation and more accurate diagnosis will only be achieved by the use of standardised protocols including detailed prenatal findings correlated with accurate clinical findings and histopathological examination.