

Endometriosis and major obstetrical complications

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Received: 1 December 2009 / Accepted: 1 December 2009 / Published online: 9 December 2009
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Keywords Endometriosis · Pregnancy · Decidualization · Hemoperitoneum · Preterm birth

While endometriosis is a lesion defined by the presence of endometrium-like tissue outside the uterus, it also shows a variable response to ovarian steroids by proliferation, decidualization, desquamation, and bleeding and at the end of reproductive life by atrophy. In the non-treated control arms of six randomized trials, endometriosis was progressive in one third of patients and stable in another one third, whereas it had disappeared at the time of second-look laparoscopy in the remaining one third [1]. During treatment with progestogens, lesions may become decidualized and small lesions, even invisible. Pregnancy has been suggested as the optimal prophylactic treatment for endometriosis as symptoms and signs regress during gestation and for varying periods thereafter. The regression is probably due to a combination of anovulation and amenorrhea as well as decidualization of functional endometriotic tissue resulting in apoptosis and loss of cells. For this reason, Kistner [2] advocated in 1975 pseudopregnancy for 6 months as the treatment of choice. In addition, he recommended short periods of pseudopregnancy after conservative surgery if not all areas of endometriosis could be excised, expecting that 40–50% of these patients became pregnant within 24 months.

Today, the relationship between endometriosis and pregnancy appears to be more complex. First, spontaneous hemoperitoneum in pregnancy (SHiP) has been recognized as a relatively rare complication occurring during the second half of pregnancy and resulting in a high fetal mortality rate of over 30%. In recent reports, SHiP is increasingly linked with bleeding of an endometriotic implant [3–5]. The complication may occur in pregnancy after in vitro fertilization as well as excisional surgery [6, 7]. Although endometriosis may appear invasive and destructive [8], SHiP is, in most cases, caused by bleeding of superficial lesions on the parametrium or the uterus [9]. When the diagnosis of SHiP is delayed, fetal mortality remains high. On the other hand, early diagnosis and coagulation of the hemorrhagic site may not only save the pregnancy but also, in preterm cases, allow continuation of pregnancy till term [9].

Secondly, recent publications indicate that endometriosis increases the risk of late miscarriage and preterm birth [10–12]. Placental bed biopsy studies by Kim et al. [13, 14] have shown that preterm birth with or without rupture of the membranes is associated with incomplete transformation of the uteroplacental arteries. The findings suggest that women with endometriosis are at risk for defective deep placentation and decidual ischemia [15]. Retrospective studies have shown conflicting data on the association between endometriosis and the risk of preeclampsia [16, 17]. Therefore, prospective clinical and pathological studies are needed to determine whether women with endometriosis are at increased risk of major obstetrical complications including preterm birth, preeclampsia, and small-for-gestation age. In view of the complexity of endometriosis, the therapeutic approach in endometriosis should be “problem-oriented” and not “lesion-oriented” [18].

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