The development of placenta increta following pelvic transcatheter artery embolization for postpartum hemorrhage

K. Kitao, *M.D., Ph.D.*; **N. Makihara,** *M.D.*; **H. Morita,** *M.D., Ph.D.*; **M. Yamasaki,** *M.D., Ph.D.*; **S. Matsuoka,** *M.D., Ph.D.*; **N. Ohara,** *M.D., Ph.D.*; **T. Maruo,** *M.D., Ph.D.*

Department of Obstetrics and Gynecology, Kobe University Graduate School of Medicine, Kusunoki-Cho, Chuo-Ku, Kobe (Japan)

Summary

Objective: Pelvic transcatheter artery embolization (TAE) has been widely used for the management of postpartum hemorrhage (PPH). However, the adverse effects of TAE on the subsequent pregnancy remain poorly understood. *Case:* A 30-year-old woman, gravida 2, para 1, developed PPH due to atonic bleeding and underwent TAE. Thereafter, her menstrual cycle became irregular with less blood volume. Three years later, she became pregnant despite a thin endometrial thickness of 6 mm during the ovulatory period. She delivered a healthy baby at 39 weeks of gestation. No signs of placental separation were obtained, and an attempt at manual extraction of the placenta failed, followed by massive PPH. She underwent emergent TAE. The placenta was not spontaneously delivered even on day 8 postpartum. A supracervical hysterectomy was performed due to a worsening intrauterine infection. Pathological examination revealed findings compatible with placenta increta. *Conclusion:* A TAE-associated thin endometrium may be attributable to the development of placenta increta. Pregnant women undergoing TAE should be managed carefully because the information about pregnancy outcomes after TAE remains scanty.

Key words: Placenta increta; Transcatheter artery embolization; Postpartum hemorrhage.

Introduction

There is little information regarding the severe adverse effects in pregnant women who undergo pelvic transcatheter artery embolization (TAE), while a pregnancy subsequent to TAE for postpartum hemorrhage (PPH) has been reported to have a risk of recurrence of PPH [1]. We report a case of the development of placenta increta in a woman who underwent TAE for PPH in a previous pregnancy.

Case Report

The patient was 30-year-old woman, gravida 2, para 1. At the age of 27, she had undergone bilateral pelvic transcatheter artery embolization (TAE) of the internal iliac arteries due to atonic bleeding. Thereafter, her menstrual cycle had become irregular with less blood volume. Three years later, she became pregnant despite a thin endometrial thickness of 6 mm during the ovulatory period. The course of pregnancy was uneventful until 20 weeks of gestation when an episode of uterine bleeding was noted. The episode repeated several times in the latter half of the second trimester. Ultrasonography (US) demonstrated many anechoic areas with convoluted margins around the placental and maternal interface (Figure 1). These findings of placental lacunae were suggestive of placenta accreta.

The patient gave birth to a healthy male infant weighing 3,248 g at 39 weeks, but considerable bleeding began 10 min after delivery without any signs of placental expulsion. An attempt at manual removal of the placenta and treatment to cease massive hemorrhage failed to succeed. Total blood loss amounted to 3,490 g, and her hemoglobin level dropped to 3.5 g/dl. She underwent an emergent bilateral TAE of the internal iliac arteries, and the refractory bleeding was successfully controlled.

Gadolinium-enhanced magnetic resonance imaging (MRI) displayed a thin or absent retroplacental myometrial zone in the anterior uterine wall where placental fragments were attached, suggesting placenta accreta. Conservative treatment with antibiotics and uterotonic agents was given because of the patient's strong desire to preserve the uterus. On day 3 postpartum, severe intrauterine infection manifested. Five days later, the patient's body temperature rose to greater than 39°C with a white blood cell count of 19,200/μl and C-reactive protein of 10.1 mg/dl. She underwent a supracervical hysterectomy due to the worsening uterine infection. At laparotomy, the placenta was visible through the thin uterine wall, suggesting placenta increta (Figure 2). A foul-smelling specimen of the placenta was tightly adhered to the uterine wall. Total blood loss amounted to 1,075 g and six units of red blood cell were transfused. The pathological diagnosis was consistent with placenta increta. The postoperative course was uneventful, and the patient was discharged home on day 15 postoperation.

Conclusion

TAE has been thought to contribute to the pathogenesis of abnormal placentation such as placenta accreta. Pron *et al.* [2] reported two cases of placenta accreta among 18 pregnancies undergoing uterine artery embolization (UAE) for leiomyomas. El-Miligy *et al.* [3] also reported a case of placenta accreta following bilateral UAE for leiomyomas. Although it is uncertain whether an abnormal placentation in the present case was directly related with TAE, it is speculated that the uterus damaged by TAE might have caused endometrial atrophy.

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Fig. 1

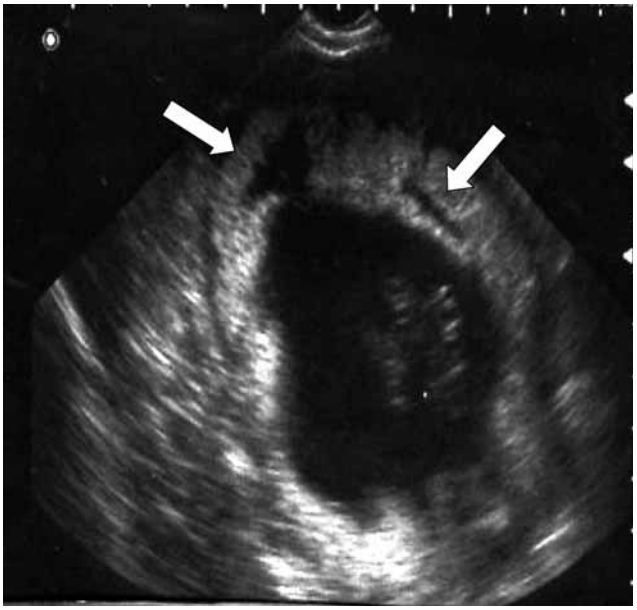

Fig. 2

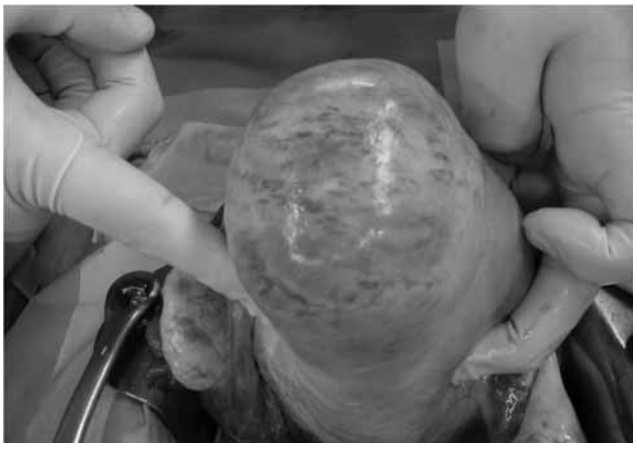

Figure 1. — Placental lacunae around the placental and maternal interface early in the second trimester. The white arrows show anechoic areas with convoluted margins.
Figure 2. — The uterine surface of placenta increta. The placenta is visible through the thin uterine wall.

The thinness of the endometrium may in turn lead to the defect of an appropriate development of the decidua with subsequent abnormal placental attachment. The mechanism underlying endometrial atrophy associated with TAE remains unknown. Nonetheless, it is tempting to speculate that a reduced endometrial blood supply by TAE may cause endometrial atrophy. A relevant study has been reported by Tropeano *et al.* [4], who described a case of permanent amenorrhea associated with endometrial atrophy which occurred following UAE for uterine fibroids. In the present case, we observed placental lacunae around the placental and maternal interface early in the second trimester. A recent report has indicated that visualization of placental lacunae can predict a high possibility of placenta accreta as early as 15 to 20 weeks of gestation [5].

In conclusion, it is suggested that the thin endometrium following pelvic TAE may be involved, at least in part, in the development of placenta increta. The present case warns that a subsequent pregnancy following TAE should be carefully monitored in the second and third trimesters of pregnancy.

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Address reprint requests to:
K. KITAO, M.D., Ph D.
Department of Obstetrics and Gynecology,
Hyogo Prefectural Tsukaguchi Hospital
6-8-17 Minamitsukaguchi-Cho,
Amagasaki, 661-0012 (Japan)
e-mail: tajirou2@yahoo.co.jp