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ORIGINAL ARTICLE

Endometrium-free uterine closure technique and abnormal placental implantation in subsequent pregnancies

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ABSTRACT

Background: Abnormal placentation can result in massive hemorrhage, which is the leading cause of severe maternal morbidities and mortality in its management. Over the past 50 years, the incidence of placenta previa (PP), abnormal implantation of the placenta, and cesarean scar pregnancy have continued to rise. This coincides with the well-documented parallel rise in the rate of cesarean deliveries, the performance of multiple repeat cesarean deliveries and the adoption of newer uterine closure techniques. However, no studies have examined the role of uterine closure techniques in abnormal placentation in women with a history of a prior cesarean delivery.

Objective: To assess the practicality of one specific uterine closure technique at cesarean delivery and to evaluate the relationship between previous cesarean delivery and subsequent development of abnormal implantation of the placenta, as well as neonatal and other perioperative outcomes after receiving an endometrium-free uterine closure technique.

Methods: This retrospective observational study considered cesarean deliveries ($n = 727$) and subsequent vaginal births after cesarean delivery ($n = 109$) among total deliveries ($n = 4496$) performed in private practice at NYU Langone Health from 1985 to 2015. All cesarean deliveries were performed using the endometrium-free uterine closure technique. The primary outcome was the incidence of abnormal implantation of the placenta in subsequent pregnancies. The secondary outcomes were neonatal and maternal complications, specifically postoperative hemoglobin and hematocrit concentration losses. The association between independent variables and outcomes were evaluated using mixed-effect regression models.

Results: In contrast to published data, independent of the number of repeat cesarean deliveries, the presence of 26 (3.1%) PPs and of 366 (43.8%) anterior placentas, there were no patients with abnormal implantation of the placenta in a cesarean scar, neither prenatally nor at delivery. Maternal hemorrhage, postoperative and neonatal complications did not reach clinical significance. The statistical analysis revealed that, when compared with women who had fewer repeat cesarean deliveries using endometrium-free uterine closure technique, those with the most had a lesser risk of forming PP and less blood loss, as measured by both hematocrit and hemoglobin evaluation.

Conclusion: In this retrospective cohort study, the exclusion of the endometrium during the endometrium-free uterine closure technique was associated with fewer placental abnormalities in subsequent pregnancies and reduced life-threatening maternal morbidity for future cesarean deliveries.

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Abnormal placentation; cesarean technique; placenta accreta; placenta previa; uterine scar

Introduction

The rise in cesarean delivery rates and the ability to perform multiple repeat cesarean deliveries has coincided with a rise in abnormal implantation of the placenta (aka abnormal placentation) [1–6]. Abnormal placentation, which is associated with anterior placental location [7], includes cesarean scar pregnancy, the spectrum of abnormal placental adherence and invasion into and through the myometrium [8,9]. The rate of cesarean deliveries rose from 4.5% in 1965 to 33% today, with a concurrent rise in the incidence of placenta accreta from 1:2510 pregnancies in the 1980s to 1:333 pregnancies in the past decade [10,11].

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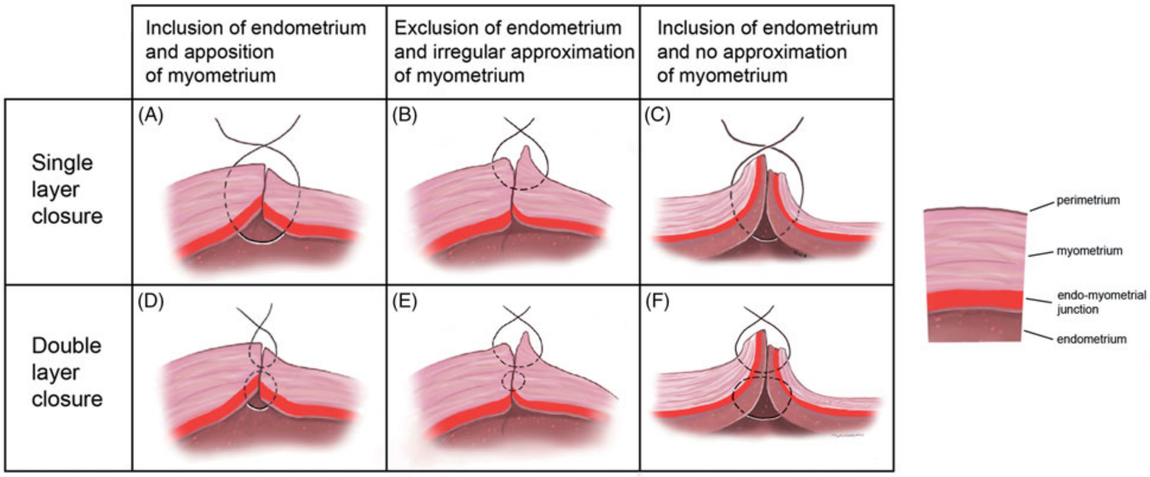

Figure 1. Illustrations of currently practiced uterine closure techniques.

In the presence of a uterine scar, an important risk factor for placenta accreta is PP [1,4]. The entity of placenta previa (PP)-accreta progressively increases with the number of previous cesarean deliveries, which may reach up to 10 per individual woman [10,12,13]. A large multicenter study reported that the risk of placenta accreta in women presenting with PP increased from 3% at first cesarean delivery to 67% in women having five or more cesarean deliveries [1]. As the rate of placenta accreta and PP has increased, peripartum hysterectomies, neonatal complications, maternal hemorrhage, and maternal mortality have also risen [10,14–16]. The maternal mortality in the USA rose from 7.2 deaths per 100,000 live births in 1987 to 17.2 deaths per 100,000 live births in 2015. Hemorrhage was one of the leading causes [17]. It is estimated that if the cesarean delivery rate continues to rise, by 2020, there will be 4504 placenta accreta cases annually resulting in an additional 130 maternal deaths [18].

Only recently have clinicians and pathologists started to understand the pathogenesis of abnormal placentation [3,19]. Specifically, the etiology of placenta accreta has been traced back to an early first-trimester invasion of the trophoblasts into defective myometrium devoid of the Nitabuch fibrinoid layer, which is also called cesarean scar pregnancy. Identical histology and concurrent increasing incidence of placenta accreta and cesarean scar pregnancy have been documented [20,21]. A recent review of pathological and clinical findings of accreta cases detailed the role of prenatal ultrasound imaging in screening women for abnormal implantation of the placenta [22]. These findings emphasized the observation that a damaged decidual-myometrial zone in the uterine scar favors trophoblastic invasion.

Uterine scarring after cesarean delivery leads to a diversity of anatomical and clinical consequences [7,23–25]. Full-thickness suturing including the endometrial layer and correct approximation of the hysterotomy margins, that is decidua-to-decidua and myometrium-to-myometrium, have been shown to improve the uterine scar strength [26]. As currently practiced, uterine closure techniques are physician-dependent, offering little-to-no guarantee of layer approximation, depending on the technique used at the prior uterine closure and immediacy for hemostasis (Figure 1) [27–29]. Many obstetricians use a closure technique based on the work by Kerr, which employs a lower uterine segment incision and closure in two layers [30]. Newer uterine closure techniques simplify Kerr's work by employing blunt tissue dissection, one layer uterine closure, and limited layer approximation. These approaches provide short-term benefits related to blood loss, operating time, and cost [31–33]. However, no published studies that examine the long-term effects of these closure techniques on the development of invasive placentation were found in the literature research [34–36].

This retrospective observational study considers a surgical technique, termed endometrium-free uterine closure technique (EFCT), that avoids suturing the endometrium into the myometrial reapproximation. The main objectives of the study are (1) to assess the practicality of the EFCT in cesarean deliveries, especially in higher-order repeat cesarean deliveries, and (2) to evaluate the association between maternal risk factors, clinical characteristics, neonatal and perioperative outcomes, and development of abnormal implantation of the placenta in subsequent pregnancies compared to patients with first and only cesarean delivery without subsequent pregnancies and within patients with successive cesarean deliveries, VBACs or subsequent abortions.

Materials and methods

Subjects

A retrospective study of cesarean deliveries ($n = 727$), subsequent VBACs ($n = 109$), and abortions ($n = 47$) among total deliveries ($n = 4496$) performed in private practice was conducted reviewing records at NYU Langone Health from January 1985 to September 2015. IRB approval (s15-01161) was granted. The cohort was divided into five groups according to the number of prior cesarean deliveries 0 to $\geq$4 (Table 1).

Inclusion criteria

Cesarean deliveries were performed using EFCT. For women who left the practice, their records were requested and reviewed for reproductive history to include abortions and deliveries by VBAC or by repeat cesarean.

Exclusion criteria

Women diagnosed with invasive placentation from a previous uterine surgery performed at another practice and/or who underwent a hysterectomy for a complication unrelated to abnormal placentation were excluded. Pathology reports were requested. Women who left the practice were excluded if follow-up history was unavailable.

Endometrium-free uterine closure technique

After delivery, the uterus was exteriorized for EFCT. The endometrium, endometrial-myometrial junction, and myometrium were identified. The angle sutures were placed and the myometrial closure was achieved in two layers using a continuous interlocking chromic suture. For the first layer, the needle was placed in the lower myometrium to exit at the endometrial-myometrial junction and entered the corresponding junction of the superior margin to incorporate an equal thickness of the myometrium. This was repeated throughout the length of the uterine incision, avoiding the endometrium. The second layer approximated the remaining myometrium, focusing on opposing opposite perimetral margins and accomplishing a full-thickness closure (Video 1).

Outcome measures

The primary outcomes were PP and abnormal implantation of the placenta following the EFCT. The secondary outcomes were related to perioperative courses and neonatal outcomes. The changes in pre- and post-operative hemoglobin and hematocrit concentrations were recorded.

Data collection

Maternal data and neonatal data collected are described in Table 1. Additional information was recorded for uterine rupture, placenta appearance, hysterectomy, and organ injury. The uterine closure timing began in 2013. An indicator variable was created to represent the number of repeat cesarean deliveries and VBACs of each patient as described. Patients in Group 1 were included to establish a baseline prevalence of PP, invasive placentation, perioperative courses and neonatal outcomes in the absence of prior uterine surgery. In all other instances, the condition of the placenta of the last delivery was assessed in relation to the prior uterine closure. The diagnosis of PP was established antenatally by transvaginal third-trimester ultrasound. In 2003, the prenatal diagnostic unit began to use a distance of less than 2 cm from the internal cervical opening to confirm the diagnosis. Blood transfusions were given for perioperative hemoglobin of 7 g/dl.

Statistical analysis

The characteristics of subjects were summarized using descriptive statistics in mean ± standard deviations (SD) for continuous variables, or % (counts) for categorical variables. A mixed-effect logistic regression (MLR) model that accounted for the correlation among repeated outcomes was used to evaluate the association between independent variables and primary outcome variables. MLR was conducted in a univariate manner. Variables were evaluated in one multivariate model to control for confounding effects. All variables with a p-value $<.1$ for the bivariate association with subsequent development of PP, abnormal implantation of the placenta and secondary outcomes were included in the multivariate model. Those with p-value $<.05$ were retained in the final model. Similarly, MLR models were used for the continuous secondary outcomes of the changes in hemoglobin and hematocrit concentration. Results were reported as odds ratios (OR) with 95% confidence intervals (CI) for the primary outcomes and coefficients with standard deviations

Table 1. Clinical characteristics of the cohort of 727 cesarean deliveries and 109 vaginal deliveries after cesarean delivery.

Variables	Group 1	Group 2	Group 3	Group 4	Group 5	Value
Delivery types						
1 CD (Group1)						221 (26.4%)
2 CDs: 1 CD + 1 VBAC (Group2)						224 + 75 (35.8%)
3CDs: 2CDs + 1VBAC (Group3)						138 + 26 (19.6%)
4CDs: 3CDs + 1VBAC (Group4)						71 + 4 (9%)
≥5 CDs: ≥4 CDs + 1VBAC (Group5)						73 + 4 (9.2%)
Clinical characteristics averages per group						
Maternal age (Years)	32.57	32.99	31.83	32.24	34.61	32.74 ± 6.85
Gravidity (pregnancies)						
1	108	0	1	0	0	109 (13%)
2	39	115	0	0	0	154 (18.4%)
3	23	71	66	0	0	160 (19.1%)
4	20	35	42	29	1	127 (15.2%)
5+	31	78	55	46	76	286 (34.3%)
Parity (live births)						
1	164	4	0	0	0	168 (20.1%)
2	23	197	5	0	0	225 (26.9%)
3	11	35	104	0	0	150 (17.9%)
4	9	19	15	52	1	96 (11.5%)
5+	14	44	40	23	76	197 (23.6%)
Gestational age at birth (weeks)	37.37	38.30	39.18	40.01	37.84	38.38 ± 2.22
Birth weight (g)	3167.70	3340.68	3601.85	3223.21	3238.68	3327 ± 753.3
1-min APGAR score						
<8	40	35	16	13	5	109 (13.1%)
8	84	89	45	20	24	262 (31.8%)
9	96	171	99	41	47	454 (55%)
5-min APGAR score						
<9	20	28	8	10	3	69 (8.3%)
9 or 10	200	267	152	64	73	756 (91.7%)
Clinical/surgical outcomes intraoperative and postoperative						
Hospital stay (d)	4.34	3.92	3.89	3.92	3.91	4.02 ± 1.36
Operation time (min)	97.98	108.75	110.00	129.41	121.72	109.1 ± 33.42
Blood loss (ml)	812.75	743.04	731.56	712.28	758.29	758.8 ± 301.4
Preoperative Hct (g/dl)	36.40	36.10	35.37	35.57	35.17	35.87 ± 4.01
Postoperative Hct (g/dl)	29.95	31.07	31.05	31.58	31.27	30.88 ± 4.78
Hct level change (g/dl) from baseline to follow-up	6.51	4.98	4.36	3.99	3.90	5.00 ± 3.76
Preoperative Hgb (g/dl)	12.36	12.26	11.95	12.10	11.95	12.17 ± 1.497
Postoperative Hgb (g/dl)	10.12	10.50	10.45	10.63	10.58	10.43 ± 1.64
Hgb level change (g/dl) from baseline to follow-up	2.28	1.74	1.52	1.47	1.40	1.76 ± 1.33
Estimated blood loss (EBL) Hct	6.61	4.69	4.36	3.99	3.90	3.95 ± 0.40
EBL Hgb	2.27	1.60	1.52	1.47	1.40	1.70 ± 1.42
Accreta no cases						0 (0%)
Blood transfusion (RBC units)	0.08	0.03	0.03	0.01	0.00	0.04 ± 0.07
Placenta previa						
	18	4	1	1	2	26 (3.1%)
Placental location						
Anterior	90 (40%)	129 (43%)	84 (51%)	33 (44%)	30 (38%)	366 (43.8%)
Posterior	94 (42%)	117 (39%)	52 (31%)	29 (38%)	36 (46%)	328 (39.2%)
Fundal	10 (4%)	16 (5%)	11 (6%)	8 (10%)	5 (6.4%)	50 (6%)
C-shaped	1 (0.4%)	9 (3%)	0 (0%)	1 (1.3%)	1 (1.2%)	12 (1.4%)
Lateral	3 (1%)	1 (0.3%)	3 (1.8%)	0 (0%)	2 (2.5%)	9 (1.1%)
Unknown	19 (8.5%)	27 (9%)	14 (8%)	3 (4%)	3 (3%)	66 (7.9%)

Data are expressed as mean ± SD/median or number (%). CDs: cesarean deliveries; VBAC: vaginal birth after cesarean.

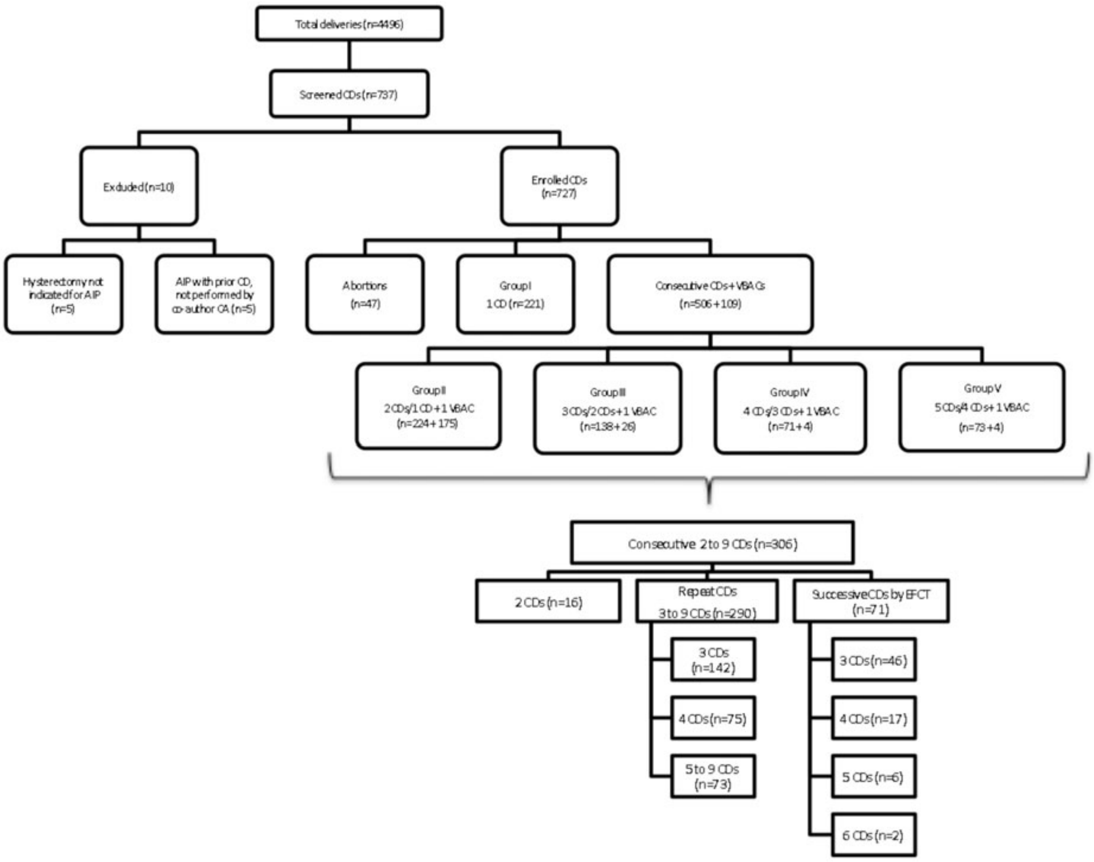

Figure 2. Flowchart demonstrating number of pregnancy outcomes.

(SDs) for secondary outcomes, respectively. OR greater than 1 and coefficients greater than 0 indicated positive association with outcome values. Two-sided *p*-values $<.05$ were considered to be statistically significant.

Results

Study population

Over three decades, a total of 4496 deliveries were performed, including cesarean deliveries ($n = 727$) and VBACs ($n = 109$). The records of women who left the practice ($n = 47$) were reviewed for subsequent reproductive history. Women ($n = 10$) who met exclusion criteria were omitted. The pathology reports of the removed uteri confirmed five normal placentations and five invasive ones. The endometrial-myometrial junction was identified in all cesareans ($n = 727$) and EFCT was confirmed in the operative reports to be reliable and feasible.

Table 1 details the pregnancy outcomes. Group 1 consists of one cesarean delivery ($n = 221$) on patients without subsequent pregnancies. The remaining cesarean deliveries ($n = 506$) consist of patients with two-to-six successive pregnancies. Group 2 consists of secondary cesarean deliveries ($n = 224$) in addition to subsequent VBACs after one cesarean delivery ($n = 75$); Group 3, tertiary cesarean deliveries ($n = 138$) and subsequent VBACs after two cesareans ($n = 26$); Group 4, quaternary cesarean deliveries ($n = 71$) and subsequent VBACs after three cesareans ($n = 4$); Group 5, quinary to nonary cesarean deliveries ($n = 73$). Group 5 includes quinary ($n = 43$), senary ($n = 20$), septenary ($n = 9$), octonary ($n = 4$), nonary cesarean deliveries ($n = 1$), and subsequent VBACs after four cesareans ($n = 4$), (Figure 2). After one or two previous cesarean deliveries, VBACs were routinely allowed. The VBACs after three or four cesareans were unplanned.

Maternal and neonatal characteristics

Table 1 shows the characteristics of the study cohort. The women varied in age from 20 to 55 years old. The average operating time was 109 min, with an average of 38.8 min for EFCT. The average quantified blood loss was 759 ml. The effective change of hemoglobin and hematocrit from admission to discharge averaged 1.7 and 3.9,

Table 2. Mixed effect logistic regression results showing odds ratios [95% CI] for risk factor for placenta previa.

Variables	Unadjusted OR [95% CI]	Adjusted OR [95% CI]
Number of delivery types		
1 CD (Group1)	Ref	Ref
2 CDs: 1 CD + 1 VBAC (Group2)	0.15 [0.05, 0.46]**	0.17 [0.05, 0.57]**
3CDs: 2CDs + 1VBAC (Group3)	0.07 [0.01, 0.53]*	0.11 [0.01, 0.83]*
4CDs: 3CDs + 1VBAC (Group4)	0.15 [0.02, 1.18]	0.17 [0.02, 1.46]
≥5 CDs: ≥4 CDs + 1VBAC (Group5)	0.30 [0.07, 1.33]	0.30 [0.06, 1.56]
Maternal age (years)	1.06 [0.99, 1.13]	1.04 [0.97, 1.12]
Gravidity		
1	Ref	
2	0.70 [0.17, 2.86]	
3	1.39 [0.41, 4.74]	
4	0.86 [0.21, 3.53]	
5+	0.57 [0.16, 2.05]	
Parity		
1	Ref	
2	0.74 [0.27, 2.02]	
3	0.41 [0.11, 1.59]	
4	0.21 [0.03, 1.73]	
5+	0.63 [0.21, 1.85]	
Gestational age at birth (weeks)	0.68 [0.68, 0.68]***	0.79 [0.69, 0.91]**
Birth weight (1000 g)	0.62 [0.30, 1.27]	
1-min APGAR score		
<8	Ref	Ref
8	1.04 [0.39, 2.76]	3.54 [0.85, 14.78]
9	0.19 [0.06, 0.64]**	0.84 [0.17, 4.16]
5-min APGAR score		
<9	Ref	
9 or 10	0.89 [0.59, 1.36]	
Hospital stay (d)	1.14 [1.01, 1.28]*	1.06 [0.92, 1.22]
Operation time (min)	0.97 [0.93, 1.01]	
Hct level change (g/dl)	1.06 [0.95, 1.18]	
Hgb level change (g/dl)	1.10 [0.91, 1.34]	
Blood transfusion (RBC units)	2.47 [1.55, 3.95]***	2.35 [1.35, 4.10]**

Ref: reference category, has an odds ratio of 1.0. *$p < .05$; **$p \leq .01$; ***$p \leq .001$.

respectively. Thirteen cases (1.5%) required blood transfusion. There were no hysterectomies at cesarean delivery, organ injury, or uterine rupture in any of the patients. Antenatal ultrasonography revealed an anterior placental location in 366 patients (43.8%) and no invasive placentation. There were a total of 26 PPs (3.1%), all confirmed at surgery. All placentas were removed immediately after delivery without difficulty and appeared intact. The gestational ages vary from 37 to 40 weeks and the birth weights 3167–3601 g. At 1 min, 86.8% of Apgar scores were >8, while at 5 min, 91.7% were 9 or 10.

Statistical findings

The association between clinical profiles and PP was evaluated and summarized in Table 2. The univariate analysis revealed that the risk of getting PP in Groups 2 and 3 significantly decreased by 85% (OR 0.15; 95% CI 0.05–0.46; $p = .001$) and by 93% (OR 0.07; 95% CI 0.01–0.53; $p = .01$) respectively, in comparison to Group 1. The trend persisted when Groups 4 and 5 were compared to Group 1; however, this was not statistically significant ($p = .072$ and .113). The relationship persisted after multivariate adjustment for factors such as number of previous cesarean deliveries, gestational age at birth, 1-min Apgar scores, or the need for a blood transfusion. The risk of PP, abnormal implantation of the placenta and increasing blood loss were not statistically significant among Groups 2–5, when Group 1 was excluded from the analysis (OR 0.981; 95% CI 0.839–1.146).

The association between clinical profiles and blood loss was also evaluated. Univariate analysis showed that blood loss was inversely associated with the number of subsequent cesarean deliveries. Compared to one cesarean delivery, patients with two or more had significantly less hematocrit and hemoglobin concentration loss. There was no relationship between blood loss and a woman's age, birth weight, 1-min Apgar score, 5-min Apgar score or operation time. The statistical significance of the number of previous cesarean deliveries and blood loss was sustained in multivariate analyses for both hematocrit and hemoglobin changes.

Discussion

Previous abnormal placentation research has been directed towards the understanding of the increasing trend, pathogenesis, risk factors, diagnosis and optimal treatment [3,10,12,22,31–33,37–46]. Prior research has examined uterine scar strength and healing at 40–42 d postoperatively [26]. This study instead focuses on uterine closure and the prevention of future invasive placentation. It is one of the first studies to examine the possibility of an association between a specific uterine closure technique, namely EFCT, and the development of abnormal placentation. Also, among the first to evaluate the long-term impact of a specific closure technique on successive pregnancies, specifically on higher-order cesarean deliveries.

Using multivariate analysis, the number of repeat cesarean deliveries and the presence of PP or anterior placenta were independent risk factors for abnormal placentation in pregnancies preceded by a prior cesarean delivery. The lack of associated abnormal implantation of the placenta and PP supports the published hypothesis that the two entities represent a sequence of villous implantation in the cesarean scar, downward expansion of the placenta and a limited ability to migrate [35]. This study revealed no abnormal placentation following a cesarean delivery in which the EFCT was used. In addition, the EFCT was inversely associated with blood loss, even with increasing number of cesarean births.

The EFCT relies on the identification of the endomyometrial junction and the evaluation of the myometrium for needle placement before closure and provides distinct anatomical landmarks for reproducibility. The EFCT longer procedure time reflects the above steps, as well as the two-layer closure and bladder flap reapproximation. Furthermore, additional time is required to teach assistant surgeons this newer technique. The EFCT minimizes scarring of the endometrial-myometrial interface at the incision site, allowing the cut margins to unite and heal as separate layers, thereby protecting the integrity of both the endometrium and myometrium rendering abnormal placentation unlikely. The decreased blood loss with the increasing number of cesarean deliveries represent the combined results of the EFCT and increasing devascularization of the lower uterine segment from repeated scarring [47].

Currently, the practice standards recommended by the American College of Obstetricians and Gynecologists and other governing guideline-issuing bodies do not endorse any particular uterine closure technique [40,48]. Dodd et al. endorsed this autonomy of obstetricians and argued that the choices of their technique are equivalent for short-term maternal outcomes. However, these authors admit that there is an absence of data regarding the use of different closure techniques on long-term complications for future pregnancies [49]. The EFCT introduces a model for the standardization of uterine closure technique, which is essential for evaluating invasive placentation in large numbers of patients who underwent cesarean deliveries by a diversity of obstetricians.

The strengths of this retrospective cohort study include (1) data collected over three decades, (2) one specific uterine closure technique used in successive cesarean deliveries, (3) the absence of serious neonatal and maternal complications, (4) the potential for successful reproducibility of EFCT by other surgeons. A continuous interlocking chromic suture was used in all cases. The possible effect of abnormal placentation as a result of pressure-induced ischemia in the uterine wall was not observed.

While this study has encouraging results, its limitations include: (1) a lack of a matched control group, (2) an absence of intersurgeon variability, (3) a limitation to one suture material, (4) an omission of other materials and methods used during cesarean deliveries, (5) a larger sample size, given the low incidence of abnormal placentation. While the relationship between uterine closure and scar strength has been considered, these studies have not analyzed the long-term relationships between closure technique and abnormal placentation in subsequent pregnancies [26].

On the basis of these findings, we believe that surgical technique at cesarean delivery is a contributing factor in the development of abnormal placentation in subsequent pregnancies. The inverse association of PP and blood loss with increasing number of cesarean deliveries, the presence of anterior placentation, the absence of abnormal placentation and serious maternal morbidities are attributed to the EFCT. The prevention of abnormal placentation began with layer approximation of the uterine incision at closure during the cesarean delivery, which prioritizes exclusion of the endometrium into the myometrial closure and maintains the integrity of both layers distinctly. Prospective clinical trials with larger patient populations and a multicenter/multisurgeon design are essential next steps to evaluate the impact of the EFCT on future placentation in pregnancies after a prior cesarean delivery.

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Disclosure statement

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References

[1] Silver RM, Landon MB, Rouse DJ, et al. Maternal morbidity associated with multiple repeat cesarean deliveries. Obstet Gynecol. 2006;107(6):1226–1232.
[2] Higgins MF, Monteith C, Foley M, et al. Real increasing incidence of hysterectomy for placenta accreta following previous caesarean section. Eur J Obstet Gynecol Reprod Biol. 2013;171(1):54–56.
[3] Jauniaux E, Jurkovic D. Placenta accreta: pathogenesis of a 20th century iatrogenic uterine disease. Placenta. 2012;33(4):244–251.
[4] Wu S, Kocherginsky M, Hibbard JU. Abnormal placentation: twenty-year analysis. Am J Obstet Gynecol. 2005;192(5):1458–1461.
[5] Marshall NE, Fu R, Guise JM. Impact of multiple cesarean deliveries on maternal morbidity: a systematic review. Am J Obstet Gynecol. 2011;205(3):262.e1–262.e8.
[6] Creanga AA, Bateman BT, Butwick AJ, et al. Morbidity associated with cesarean delivery in the United States: is placenta accreta an increasingly important contributor? Am J Obstet Gynecol. 2015;213(3):384.e1–384.e11.
[7] Gielchinsky Y, Rojansky N, Fasouliotis SJ, et al. Placenta accreta – summary of 10 years: a survey of 310 cases. Placenta. 2002;23(2–3):210–214.
[8] Luke RK, Sharpe JW, Greene RR. Placenta accreta: the adherent or invasive placenta. Am J Obstet Gynecol. 1966;95(5):660–668.
[9] Clark EAS, Silver RM. Long-term maternal morbidity associated with repeat cesarean delivery. Am J Obstet Gynecol. 2011;205(6):S2–S10.
[10] Miller DA, Chollet JA, Goodwin TM. Clinical risk factors for placenta previa-placenta accreta. Am J Obstet Gynecol. 1997;177(1):210–214.
[11] Publications Committee. Society for Maternal-Fetal Medicine, Belfort MA. Placenta accreta. Am J Obstet Gynecol. 2010;203(5):430–439.
[12] Usta IM, Hobeika EM, Musa AA, et al. Placenta previa-accreta: risk factors and complications. Am J Obstet Gynecol. 2005;193(3):1045–1049.
[13] Juntunen K, Mäkäräinen L, Kirkinen P. Outcome after a high number (4–10) of repeated caesarean sections. BJOG Int J Obstet Gynaecol. 2004;111(6):561–563.
[14] Shellhaas CS, Gilbert S, Landon MB, et al. The frequency and complication rates of hysterectomy accompanying cesarean delivery. Obstet Gynecol. 2009;114(2):224–229.
[15] Wright JD, Pri-Paz S, Herzog TJ, et al. Predictors of massive blood loss in women with placenta accreta. Am J Obstet Gynecol. 2011;205(1):38.e1–38.e6.
[16] Mehrabadi A, Hutcheon JA, Liu S, et al. Contribution of placenta accreta to the incidence of postpartum hemorrhage and severe postpartum hemorrhage. Obstet Gynecol. 2015;125(4):814–821.
[17] Centers for Disease Control and Prevention [Internet]. Atlanta (GA): CDC. Pregnancy mortality surveillance system; 2019 [cited Jun 4]. Available from: https://www.cdc.gov/reproductivehealth/maternalinfanthealth/pregnancy-mortality-surveillance-system.htm.
[18] Solheim KN, Esakoff TF, Little SE, et al. The effect of cesarean delivery rates on the future incidence of placenta previa, placenta accreta, and maternal mortality. J Matern Fetal Neonatal Med. 2011;24(11):1341–1346.
[19] Tantbirojn P, Crum CP, Parast MM. Pathophysiology of placenta creta: the role of decidua and extravillous trophoblast. Placenta. 2008;29(7):639–645.
[20] Sinha P, Mishra M. Caesarean scar pregnancy: a precursor of placenta percreta/accreta. J Obstet Gynaecol. 2012;32(7):621–623.
[21] Timor-Tritsch IE, Monteagudo A, Cali G, et al. Cesarean scar pregnancy and early placenta accreta share common histology. Ultrasound Obstet Gynecol. 2014;43(4):383–395.
[22] Jauniaux E, Collins S, Burton GJ. Placenta accreta spectrum: pathophysiology and evidence-based anatomy for prenatal ultrasound imaging. Am J Obstet Gynecol. 2018;218(1):75–87.
[23] Thurmond AS, Harvey WJ, Smith SA. Cesarean section scar as a cause of abnormal vaginal bleeding: diagnosis by sonohysterography. J Ultrasound Med. 1999;18(1):13–6.
[24] Silver RM. Implications of the first cesarean: perinatal and future reproductive health and subsequent cesareans, placentation issues, uterine rupture risk, morbidity, and mortality. Semin Perinatol. 2012;36(5):315–323.
[25] Roberge S, Demers S, Girard M, et al. Impact of uterine closure on residual myometrial thickness after cesarean: a randomized controlled trial. Am J Obstet Gynecol. 2016;214(4):507.e1–507.e6.
[26] Yazicioglu F, Gökdogan A, Kelekci S, et al. Incomplete healing of the uterine incision after caesarean section: is it preventable? Eur J Obstet Gynecol Reprod Biol. 2006;124(1):32–36.

[27] Babu K, Magon N. Uterine closure in cesarean delivery: a new technique. North Am J Med Sci. 2012;4(8):358–361.

[28] Vachon-Marceau C, Demers S, Bujold E, et al. Single versus double-layer uterine closure at cesarean: impact on lower uterine segment thickness at next pregnancy. Am J Obstet Gynecol. 2017;217(1):65.e1–65.e5.

[29] CAESAR study collaborative group. Caesarean section surgical techniques: a randomised factorial trial (Caesar). BJOG. 2010;117(11):1366–1376.

[30] Kerr JMM. The technique of cesarean section, with special reference to the lower uterine segment incision. Am J Obstet Gynecol. 1928;1928:729–734.

[31] Saad AF, Rahman M, Costantine MM, et al. Blunt versus sharp uterine incision expansion during low transverse cesarean delivery: a metaanalysis. Am J Obstet Gynecol. 2014;211(6):684.e1–684.11.

[32] Moroz L, DiNapoli M, D'Alton M, et al. Surgical speed and risk for maternal operative morbidity in emergent repeat cesarean delivery. Am J Obstet Gynecol. 2015;213(4):584.e1–584.e6.

[33] Peleg D, Ahmad RS, Warsof SL, et al. A randomized clinical trial of knotless barbed suture vs conventional suture for closure of the uterine incision at cesarean delivery. Am J Obstet Gynecol. 2018;218(3):343.e1–343.e7.

[34] Sholapurkar SL. Surgical techniques at caesarean might modify placenta accreta risk. BJOG Int J Obstet Gynaecol. 2015;122(1):143–143.

[35] Sumigama S, Sugiyama C, Kotani T, et al. Uterine sutures at prior caesarean section and placenta accreta in subsequent pregnancy: a case-control study. BJOG Int J Obstet Gynaecol. 2014;121(7):866–874.

[36] Sholapurkar SL. Increased incidence of placenta praevia and accreta with previous caesareans – a hypothesis for causation. J Obstet Gynaecol. 2013;33(8):806–809.

[37] Silver RM, Fox KA, Barton JR, et al. Center of excellence for placenta accreta. Am J Obstet Gynecol. 2015;212(5):561–568.

[38] Shamshirsaz AA, Fox KA, Erfani H, et al. Multidisciplinary team learning in the management of the morbidly adherent placenta: outcome improvements over time. Am J Obstet Gynecol. 2017;216(6):612.e1–612.e5.

[39] Jauniaux E, Bhide A. Prenatal ultrasound diagnosis and outcome of placenta previa accreta after cesarean delivery: a systematic review and meta-analysis. Am J Obstet Gynecol. 2017;217(1):27–36.

[40] ACOG. Committee opinion no. 529: placenta accreta. Obstet Gynecol. 2012;120(1):207–211.

[41] Brennan DJ, Schulze B, Chetty N, et al. Surgical management of abnormally invasive placenta: a retrospective cohort study demonstrating the benefits of a standardized operative approach. Acta Obstet Gynecol Scand. 2015;94(12):1380–1386.

[42] Jauniaux E, Collins SL, Jurkovic D, et al. Accreta placentation: a systematic review of prenatal ultrasound imaging and grading of villous invasiveness. Am J Obstet Gynecol. 2016;215(6):712–721.

[43] Jurkovic D, Hillaby K, Woelfer B, et al. First-trimester diagnosis and management of pregnancies implanted into the lower uterine segment cesarean section scar. Ultrasound Obstet Gynecol. 2003;21(3):220–227.

[44] Timor-Tritsch IE, Monteagudo A, Bennett TA, et al. A new minimally invasive treatment for cesarean scar pregnancy and cervical pregnancy. Am J Obstet Gynecol. 2016;215(3):351.e1–351.e8.

[45] Timor-Tritsch IE, Monteagudo A, Santos R, et al. The diagnosis, treatment, and follow-up of cesarean scar pregnancy. Am J Obstet Gynecol. 2012;207(1):44.e1–44.e13.

[46] Zhuang Y, Huang L. Uterine artery embolization compared with methotrexate for the management of pregnancy implanted within a cesarean scar. Am J Obstet Gynecol. 2009;201(2):152.e1–152.e3.

[47] Flo K, Widnes C, Vårtun Å, et al. Blood flow to the scarred gravid uterus at 22–24 weeks of gestation. BJOG Int J Obstet Gynaecol. 2014;121(2):210–215.

[48] Temmerman M. Caesarean section surgical techniques: all equally safe. Lancet. 2016;388(10039):8–9.

[49] Dodd JM, Anderson ER, Gates S, et al. Surgical techniques for uterine incision and uterine closure at the time of caesarean section. Cochrane Database Syst Rev. 2014;(7):CD004732.