Clinical Obstetrics, Gynecology and Reproductive Medicine

Case Study

Warning signs of uterine rupture after previous caesarean section: a case review

Susanne Hesselman[1,2*], Ulf Högberg[1], Eva-Britta Råssjö[2] and Maria Jonsson[1]

[1]Department of Women's and Children's Health Uppsala University, 751 85 Uppsala Sweden

[2]Center for Clinical Research Dalarna, Falun, Sweden

Abstract

Background: To describe clinical signs and outcomes of uterine rupture by stage of labour in women with one previous caesarean section and assess management of labour in these cases.

Methods: Cases with uterine rupture (n=103) were identified in a cohort of women having a trial of labour after one caesarean section (n=7683). Detailed review of medical records and fetal heart tracing was performed for cases with uterine rupture. Clinical signs of uterine rupture and maternal and neonatal outcomes were assessed in relation to stage of labour, in which rupture occurred.

Results: Fetal distress (65%), abdominal pain (56.3%) and protracted labour (54.4%) were the most frequent clinical signs preceding rupture, with no differences by stage of labour. Diagnosis of uterine rupture during the second, compared to the first stage of labour, carried a higher risk of adverse neonatal outcome, whereas diagnosis postpartum was associated with an increased risk of maternal complications ($p<0.01$). Uterine rupture was associated with induction of labour, epidural analgesia and failed operative vaginal delivery.

Conclusion: Warning signs of rupture are fetal distress, abdominal pain and protraction disorders. Diagnosis at a later stage of labour is associated with adverse neonatal and maternal outcomes.

Abbreviations: AS: Apgar score; BS: Bishop score; CTG: cardiotochography; HIE: hypoxic ischemic encephalopathy; IOL: induction of labour; PPH: postpartum haemorrhage; TOLAC: trial of labour after caesarean

Introduction

Uterine rupture is an obstetric emergency associated with maternal and neonatal morbidity and mortality [1-3]. The main risk factor is a previous caesarean section [1] and rupture occurs principally after onset of labour. Although a trial of labour after caesarean (TOLAC) is considered optional and relatively safe [4], recommendations differ between countries and the rate of TOLAC was 15-25% during the years 2001-2009 in the US [5] compared to 62% in a Swedish cohort [6].

Several risk factors for uterine rupture are known but prediction models for rupture have shown poor reliability for clinical use [7,8]. Clinical guidelines advise restrictive use of medical induction and careful assessment of the progress of labour, with cautious use of oxytocin and the availability of resources for immediate laparotomy [9-11] in case of suspected rupture. Therefore, vigilant monitoring of symptoms in a trial of labour after previous caesarean delivery is essential. However, recognition of clinical signs of uterine rupture is challenging due to the lack of specific symptoms. For example, uterine rupture is associated with abnormalities of fetal heart rate patterns [12] and protraction disorders [13-15] but these features can also be present in labours with or without previous caesarean section in which no rupture occurs.

Efforts to improve safety of vaginal delivery after caesarean section have focused on the identification of risk factors for rupture and few studies have examined the actual course of labour or aspects of intrapartum management in cases of rupture [14,15]. Such information may aid in the process of perceiving and responding to early signs, and in identifying issues of care that may improve practice and prevent adverse maternal and neonatal outcomes.

The inconsistency of diagnostic criteria for uterine rupture has made it difficult to evaluate the clinical signs and outcome of uterine rupture [3]. There is also a lack of information about the prevalence of symptoms and outcomes in relation to when in labour uterine rupture occurs.

The aim of this study was to describe clinical signs preceding uterine rupture after previous caesarean delivery, and to describe maternal as well as neonatal outcomes in relation to the stage of labour at which rupture was diagnosed. Further, we aimed to explore management of protracted labour, particularly the use of oxytocin.

Materials and methods

Cases with uterine rupture were identified by the diagnosis codes O71.0 and O71.1, according to the 10th revision of the International Classification of Disease [16], in a cohort of women having a trial of labour after one caesarean section (n=7683) 2001-2009 at 42 maternity

Correspondence to: Susanne Hesselman, CKF. Nissers väg 3, 791 82 Falun, Sweden, Tel: +46 23 492000 (operator), **E-mail:** susanne.hesselman@ltdalarna.se

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units in Sweden. For identified cases with uterine rupture, labour records, surgical reports, partograms and cardiotocographic (CTG) tracings were obtained from the maternity departments. The Regional Ethical Review Board at Uppsala approved the study. Medical records, including fetal heart rate tracings, were reviewed for cases with uterine rupture in a standardized manner by the authors (S.H., M.J., E-B.R.). Uterine rupture was defined as a full disruption of the myometrium or a covered rupture associated with symptoms, such as vaginal bleeding, fetal heart rate abnormalities, abdominal pain, or palpable fetal part in the abdomen.

Stages of labour were categorized into: first stage, which also included patients in the latency phase; second stage, including both the passive and active phase; and the immediate postpartum period. Information on cervical dilation was retrieved from examinations recorded in the partograms or in the text of medical charts.

The mode of Induction of Labour (IOL) was defined by the primary method used and divided into prostaglandins (all types and modes of administration), cervical catheter, oxytocin or amniotomy. Bishop score (BS) was recorded in all cases prior to IOL.

Protracted labour was defined as a cervical dilation ≤1 cm/hour in the active phase of the first stage, or no descent of the presenting part within 2 hours, during the second stage of labour. A second stage lasting ≥ 3 hours was defined as prolonged [16].

Use of oxytocin comprised both induction and labour augmentation. Progress of the active phase of labour within 2 hours of treatment was considered as adequate response to therapy [17]. Oxytocin treatment >2 hours without progress or resulting in uterine hyperactivity (≥6 contractions/10 minutes) were considered as deviations from recommendations regarding oxytocin treatment in women with a uterine scar [9,11], and was in this study defined as misuse. Repeat epidural dosing, mode of delivery, use of fundal pressure, blood and fetal part in the abdomen were recorded.

CTGs were assessed unblinded by the authors (S.H., M.J., E-B. R.) and classified according to the recommendation of the Swedish Society of Obstetrics and Gynaecology (SFOG). If CTGs were missing, notes regarding CTG interpretations in the medical charts were utilised. Fetal distress was defined if the CTG was classified as pathological within the last hour prior to delivery. Pathological changes included bradycardia (<100 beats/minute ≥3 minutes duration), complicated variable decelerations, late decelerations and tachycardia (>170 beats/minute). A combination of these changes was classified according to the pattern recorded immediately before delivery.

Maternal symptoms prior to diagnosis of uterine rupture were retrieved from medical records and included: pain (abdominal or referred to thorax or shoulder); loss of station of fetal presenting part; vaginal bleeding; haematuria; and nausea. Abnormalities noticed during physical examination of the uterus such as a palpable scar defect or abnormal uterine shape and uterine tone were recorded. Each patient could present with more than one symptom. As there was a substantial lack of blood pressure measurements, hypovolemic shock and/or hypotension could not be included in the analysis.

Postpartum haemorrhage (PPH) was categorized into <2000 ml and ≥2000 ml. Hysterectomy, PPH ≥2000 ml, reoperation or bladder injury were defined as major maternal complication. There was one case of intrauterine fetal death prior to labour and onset of symptoms that was excluded in the analysis of fetal outcomes. An Apgar score (AS) <7 or <4 at 5 minutes was dichotomized. Umbilical blood gas analysis was routinely performed, and arterial or venous cord pH<7.00 defined fetal acidosis. AS <4 at 5 minutes and/or a diagnosis of hypoxic ischemic encephalopathy (HIE) grade 1-3 were classified as adverse neonatal outcomes.

The Swedish medical birth register provided data about induction of labour, use of epidural anaesthesia, mode of delivery, failed operative vaginal delivery for the cohort. The data record system Obstetrix® provided information about duration of labour (hours), calculated from 3 cm of cervical dilation until delivery, which was available for 45 cases with uterine rupture and 853 cases without rupture delivered by caesarean section.

SPSS version 20.0 0 (IBM SPSS statistics, Armank, NY) for MAC software package was used to perform descriptive statistics and bivariate analysis. Statistical significance was calculated using Student t-test, Chi square test and Fishers exact test when appropriate. A two sided p-value < 0.05 was considered statistically significant.

Results

There were 103 cases of uterine rupture (1.3%) in women having a trial of labour, 70 complete ruptures and 33 incomplete. Medical records were available for all cases, partograms for 65 patients and review of CTG tracings was made in 51 cases. Postpartum the diagnosis was confirmed by laparotomy and, in one case, by abdominal ultrasound. The majority of uterine ruptures were diagnosed during the first stage (n=67), one third (n=29) in the second stage and seven was diagnosed postpartum. The primary method for IOL (n=27) was by prostaglandins (n=16), cervical catheter (n=5), oxytocin (n=4) and amniotomy (n=2). 18/27 had a BS ≤3 prior IOL.

According to medical charts and review of partograms, protracted labour was assessed in 54%; a prolonged second stage was recorded in 43% of women reaching full dilation (n=35). Oxytocin was administered in 72% and misuse of oxytocin was considered in 1/3 (n=26). In 29 /68 repeat epidural dosing was used. All women with uterine rupture had operative deliveries (94% CS) and six women underwent laparotomy postpartum. Operative vaginal delivery was attempted in 16 and the majority (n=10) failed, in four cases uterine fundal pressure was used. One third had blood or fetal part in the abdomen.

Bradycardia prior to delivery (49%) was the most common CTG pattern associated with rupture.

Clinical signs associated with uterine rupture, in relation to the stage of labour in which the rupture was diagnosed, are presented in Table 1. Fetal distress (65%), abdominal pain (56%) and protracted labour (54%) were the most frequent clinical signs of rupture regardless of stage of labour when rupture was diagnosed. Vaginal bleeding (11%), palpable scar defect (11%) or abnormal shape of the uterus (6%) was less reported clinical findings. Loss of station of fetal presenting part and hypotone uterus were clinical findings associated with diagnosis at second stage of labour, indicating overt rupture.

There was one intrauterine fetal death diagnosed prior to uterine rupture but no neonatal or maternal deaths. Maximum and median PPH were 8000 and 650 mL respectively. Table 2 presents maternal and neonatal outcomes after uterine rupture. Thirteen women suffered from a major complication, which was associated with diagnosis at later stages of labour. AS <7 at 5 minutes was recorded in 15% diagnosed first stage compared with 43% second stage (p<0.01). Cord pH values were available in 84% and infants delivered at first stage of labour had a mean of 7.14 (SD 0.17) compared with 7.04 (SD 0.23) when delivered

Table 1. Clinical signs prior to diagnosis of uterine rupture (n=103) classified by stage of labour.

	All n=103		1st stage n=67		2nd stage n=29		Postpartum n=7		*p-value* *
	n =	%	n =	%	n =	%	n =	%	
Fetal distress	67	65.0	41	61.2	20	67.0	6	85.7	0.44
Abdominal pain	58	56.3	38	56.7	17	58.6	3	42.9	0.81
Referred pain	6	5.8	3	4.5	1	3.6	2	28.6	0.07
Protracted labour	56	54.4	36	53.7	16	55.2	4	57.1	1.00
Hypertone uterus	15	14.6	12	17.9	1	3.4	2	28.6	0.08
Vaginal bleeding	11	10.7	9	13.4	2	6.9	-	-	0.57
Palpable scar defect	11	10.7	8	11.9	3	10.3	-	-	1.00
Loss of station of presenting part	11	10.7	3	4.5	8	27.6	-	-	0.01
Nausea	9	8.7	4	6.0	5	17.2	-	-	0.22
Abnormal uterine shape	6	5.8	5	7.5	1	3.4	-	-	0.78
Hypotone uterus	5	4.9	-[a]	-	5[b]	17.2	-	-	<0.01
Haematuria	3	2.9	2	3.0	-	-	1	14.3	0.21

*Fisher´s Exact test (2-sided). Each superscript letter denotes a stage category that differs significantly from each other.

Table 2. Maternal (n=103) and neonatal outcome (n=102) associated with diagnosis of uterine rupture at different stages of labour.

	All		1st stage		2nd stage		Postpartum		*p*- value§
	n=103	%	n=67	%	n=29	%	n=7	%	
PPH >2000 ml (n=99)	8	8.1	2[a]	3.1	3[a]	10.7	3[b]	42.9	<0.01
Hysterectomy	2	1.9	-[a]	-	1	3.4	1[b]	14.3	0.04
Bladder injury	7	6.8	2	3.0	4	13.8	1	14.3	0.06
Maternal complication* (n=99)	13	13.1	3[a]	4.7	6[b]	21.4	4[b]	57.1	<0.01
Fetal acidosis †(n=86)	23	26.7	14	23.0	7	35.0	2	40.0	0.44
Apgar Score < 7 at 5 min	23	22.5	10[a]	14.9	12[b]	42.9	1	14.3	0.02
Apgar Score < 4 at 5 min	9	8.8	1[a]	1.5	7[b]	25.0	1[b]	14.3	<0.01
HIE grade 1-3	7	6.9	3	4.5	4	14.3	-	-	0.24
Adverse neonatal outcome ‡	11	10.9	3[a]	4.5	8[b]	28.6	1	14.3	<0.01

PPH: post partumhaemorrhage, HIE: hypoxic ischemic encephalopathy
* PPH, Hysterectomy, bladder injury or reoperation
† Umbilical artery pH < 7.00
‡ HIE or AS<4 at 5 min
§ Fisher´s Exact test (2-sided). Each superscript letter denotes a stage category that differs significantly from each other.

second stage (p 0.03). A diagnosis of rupture during the second stage, compared with the first stage, of labour carried a higher risk of adverse neonatal outcome (p <0.01). Adverse neonatal outcome was related to failed operative vaginal delivery (p 0.02) but not to a prolonged second stage or oxytocin administration (results not shown).

Induction of labour increased the risk of uterine rupture (26.2% *vs.* 16.0% p<0.01) and epidural analgesia was used in 66% of women with rupture compared with 50% in women with no rupture (p<0.01). Failed operative vaginal delivery occurred in 9.7% compared with 0.7% in women with no uterine rupture. There was no difference in diagnosis of dystocia in women with or without uterine rupture (30.1% *vs.* 25.2%). Mean duration of labour was 5.7 (SD 3.7) hours in case of uterine rupture compared with 8.0 (SD 4.9) hours in women without rupture delivered by CS (p<0.01), (Figure 1).

Discussion

Fetal distress, abdominal pain and protracted labour are important clinical signs prior to diagnosis of uterine rupture in trial of labour after previous caesarean delivery. The high rate of misuse of oxytocin in our material is of concern and indicates non-adherence to guidelines or that guidelines are missing. Uterine rupture diagnosed in the second stage of labour has a stronger association with adverse neonatal outcome than when the diagnosis is during the first stage. Major maternal complications are related to diagnosis in the immediate postpartum period.

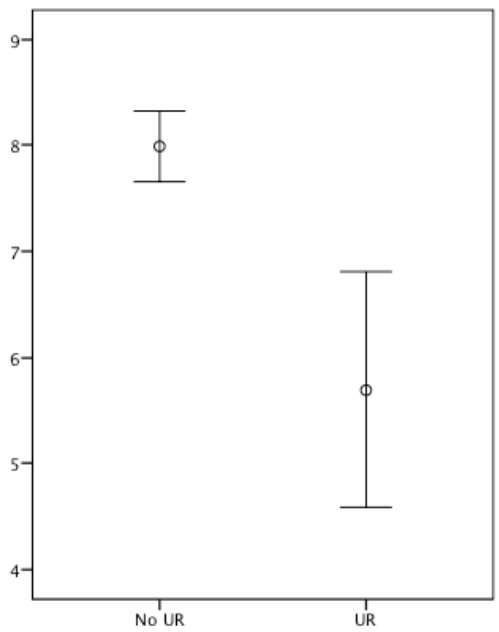

Figure 1. Duration of labour (hours) in case of no uterine rupture (n=853) and uterine rupture (n=45).
UR= uterine rupture

Strength of this study is the population-based design, with a large number of cases with verified uterine ruptures from different maternity wards in Sweden. The study included women with one prior caesarean delivery, who in their second delivery attempted vaginal birth. Thus, no woman had a previous vaginal birth that could influence the interpretation of the results. A further strength of the study is the high frequency of cord sampling that enabled an objective estimation of the rate of acidosis at birth.

The retrospective design of our study limits the reliability of reported subjective maternal symptoms and clinical findings, with a risk of both over- and underestimation. Pre-operative documentation could actually have been made when the diagnosis was confirmed. Partograms were available for only two thirds. CTGs were missing in half of patients and the interpretations of CTGs were not blinded. Further, the study design does not permit conclusion about risk management since there was limited information about labour management in the control group.

The incidences of uterine rupture in high-income countries are estimated to 0.4-0.7% [2,3] for women with TOLAC, whereas we found an incidence of 1.3%. The difference could be explained by inconsistency of diagnostic criteria of uterine rupture [3] or could reflect differences in selection of women for TOLAC and intrapartum management.

Previous studies have related induction of labour after a previous caesarean with an increased risk of uterine rupture, both with the use of oxytocin and prostaglandins [18,19]. We found that more than half of inductions were with prostaglandins in case of rupture. Information about method of induction was not available for the whole cohort.

When a decision of trial of labour is made, clinical guidelines advocate careful assessment of progress of labour, irrespective of oxytocin augmentation, in women with previous caesarean [9,11]. This guidance is based on studies suggesting that protracted labour in these women is associated with uterine rupture. In particular, labour dystocia at advanced dilations (>7 cm) may be a sign of impending uterine rupture [14]. One study also implies that caesarean section could prevent 42% of ruptures in labours with slow progress i.e. dilation less than the 10th percentile, and arrested for ≥2 hours (15). In line with previous studies, protracted labour was diagnosed >50% of our cases and a prolonged second stage (> 3 hours) were found in 43% of women reaching full dilation. Women with prolonged or arrested labour are likely to be exposed to oxytocin and the risk of uterine rupture has been found to increase in situations of prolonged labour with augmentation [15,20]. In a population-based cohort study, Dekker *et al.* [19] found that the risk of uterine rupture increased by a factor of 14 in spontaneous labour augmented by oxytocin, compared with no oxytocin, among women in TOLAC. Duration of oxytocin administration, as well as the dose given, could be of importance [21]. For augmentation, oxytocin doses > 20 mU/min have been associated with a four-fold increase in risk of uterine rupture [22]. We assessed misuse of oxytocin in 35% of treated cases and 20% had received doses > 20 mU/min.

In prolonged labour there is a more frequent use of epidural analgesia [23]. We found that epidural was associated with uterine rupture and tried to investigate the relationship further. We could not find a difference in reported abdominal or referred pain between women who used epidural analgesia and those who did not. Cahill *et al.* [24] suggested that repeat epidural dosing could implicate impending rupture, and we noted requirements of epidural dosing in 29/68 patients, which could reflect prolonged labour or presence of abdominal pain.

The clinical presentation of uterine rupture commonly comprises sudden fetal heart rate abnormalities and abdominal pain. In addition, altered uterine tone, cessation of contractions, vaginal bleeding, and signs of hypovolemia are associated symptoms [25-27]. In accordance with these studies, we found that abdominal pain was a common symptom, irrespective of labour stage in which the rupture was diagnosed, whereas referred pain was more common in the postpartum period. In our study, vaginal bleeding and alteration in the shape of uterus were infrequent findings. Altered uterine shape and excessive vaginal bleeding seems more common with uterine rupture of an unscarred uterus [25,28]. There was no reliable information about haemodynamic status of the women with rupture in our study, but one third of patients presented with haemoperitoneum, suggesting that internal bleeding is more frequent than external.

Consistent with previous studies, the most common CTG pattern associated with uterine rupture was bradycardia [12, 27]. Information on acidosis at birth in case of uterine rupture has been infrequently reported. In the study by Landon *et al.* [2], 33% had pH ≤ 7.00 among 114 cases with no information about sampling rate. Our sampling rate was 83.5% and 27% had pH ≤ 7.00.

In general, studies have not considered symptoms in relation to the stage of labour at which rupture is diagnosed. Our results indicate that major maternal complications are associated with a diagnosis of rupture at later stages of labour, the main contributor being a blood loss of 2000 ml or more. Zwart *et al.* [29] also report that major obstetric haemorrhage is an important symptom of rupture diagnosed in the postpartum period.

The risk of adverse neonatal outcome was significantly higher with a diagnosis of rupture in the second stage, compared to in the first stage. Possible reasons are that CTG patterns are more difficult to monitor and assess and that decelerations appear in virtually all deliveries during the second stage. Further, the high failure rate of operative deliveries (63%) reflects dysfunctional uterine contractions in case of uterine rupture. In women without uterine rupture the corresponding failure rate was 4.7%. Further, use of fundal pressure (n=4), a non-evidence based practice, has been related to uterine rupture [30].

To explore the impact of protracted labour as a risk factor or clinical sign of impeding uterine rupture we analysed a diagnosis of dystocia in the cohort. There was no difference in diagnosis of dystocia in women with or without uterine rupture, but there was a substantial discrepancy among rates based on diagnosis in registers (30%) and according to review of medical records (54%) among cases with uterine rupture. Labour was shorter among cases with uterine rupture than controls with failed TOLAC, reflecting an immediate delivery in case of rupture.

In conclusion, warning signs of uterine rupture are fetal distress, abdominal pain and protraction disorders. Neonatal and maternal outcomes depend on at which stage of labour uterine rupture is diagnosed; a diagnosis at a later stage is associated with poorer outcome.

Authors' contributions study conception and design

All authors contributed to study conception, design, interpretation and manuscript revision. S.H, M.J, E-B.R reviewed the medical records and fetal heart rate tracings. S.H performed the statistical analysis. All authors read and approved the final manuscript.

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Competing interests

The authors declare that they have no competing interests.

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