DOI: 10.1111/j.1471-0528.2009.02136.x
www.bjog.org

Uterine rupture in the Netherlands: a nationwide population-based cohort study

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Accepted 3 November 2008.

Objective To assess incidence of uterine rupture in scarred and unscarred uteri and its maternal and fetal complications in a nationwide design.

Design Population-based cohort study.

Setting All 98 maternity units in the Netherlands.

Population All women delivering in the Netherlands between August 2004 and August 2006 (n = 371 021).

Methods Women with uterine rupture were prospectively collected using a web-based notification system. Data from all pregnant women in the Netherlands during the study period were obtained from Dutch population-based registers. Results were stratified by uterine scar.

Main outcome measures Population-based incidences, severe maternal and neonatal morbidity and mortality, relative and absolute risk estimates.

Results There were 210 cases of uterine rupture (5.9 per 10 000 pregnancies). Of these women, 183 (87.1%) had a uterine scar, incidences being 5.1 and 0.8 per 10 000 in women with and without uterine scar. No maternal deaths and 18 cases of perinatal death (8.7%) occurred. The overall absolute risk of uterine rupture was 1 in 1709. In univariate analysis, women with a prior caesarean, epidural anaesthesia, induction of labour (irrespective of agents used), pre- or post-term pregnancy, overweight, non-Western ethnic background and advanced age had an elevated risk of uterine rupture. The overall relative risk of induction of labour was 3.6 (95% confidence interval 2.7–4.8).

Conclusion The population-based incidence of uterine rupture in the Netherlands is comparable with other Western countries. Although much attention is paid to scar rupture associated with uterotonic agents, 13% of ruptures occurred in unscarred uteri and 72% occurred during spontaneous labour.

Keywords Incidence, population based, unscarred uterus, uterine rupture, VBAC.

Please cite this paper as: Zwart J, Richters J, Öry F, de Vries J, Bloemenkamp K, van Roosmalen J. Uterine rupture in the Netherlands: a nationwide population-based cohort study. BJOG 2009;116:1069–1080.

Introduction

Uterine rupture is a rare complication of pregnancy potentially leading to severe maternal and fetal morbidity and mortality. Several risk factors have been identified, the most important being a uterine scar (mostly from previous caesarean) and the use of uterotonic agents for induction of labour.[1–5] The Netherlands has a caesarean delivery rate which is among the lowest in the world, although it is increasing. The same is true for countries worldwide, as a result of which the incidence of uterine rupture is likely to increase. The sheer quantity of recent reports on the safety of vaginal birth after caesarean (VBAC) demonstrates the increased awareness of this issue.

In a WHO systematic review of uterine rupture worldwide, the median incidence was 5.3 per 10 000 births.[6] If only population-based studies in high-income countries are taken into consideration, the mean incidence was around 3 per 10 000 deliveries. This figure, however, was based on only five of 83 included studies, the great majority being from low-income countries, facility-based, or only concerning women with a previous caesarean. A clear distinction is

made between uterine scar rupture and rupture of an unscarred uterus. Scar rupture often presents less dramatic but the incidence is rising in Western countries. Rupture of the unscarred uterus is much more frequent in low-income countries due to obstructed labour and leads to more severe feto-maternal complications, being even an important cause of direct maternal death in these countries. It is, however, a rare event in Western countries with an estimated incidence of 0.6 per 10 000, based on only ten cases.[1] Our aim was to assess the population-based incidence of uterine rupture in the Netherlands, as well as the case fatality rate, the most specific symptoms and signs at diagnosis and possible risk factors.

Methods

This study was part of a larger nationwide enquiry into severe maternal morbidity in the Netherlands, called LEMMoN. Details on design of the LEMMoN study have been published elsewhere.[7] The study was centrally approved by the medical ethics committee of Leiden University Medical Centre. It enrolled cases from 1st August 2004 until 1st August 2006. In the Netherlands, there are ten tertiary care centres, 33 nonacademic teaching hospitals and 55 general hospitals. In 2005, the number of deliveries per hospital ranged from 93 to 2655 and 41% of deliveries were under guidance of a midwife or family physician, either at home (30%) or in the hospital (11%). Pregnancies in women with a uterine scar from a previous caesarean are considered high risk pregnancies. Although these women have to deliver in hospital under guidance of an obstetrician, they are allowed to have antenatal care with the midwife or family physician until 36 weeks of gestational age. The latest published caesarean delivery rate in the Netherlands is 14% in 2002.[8]

Uterine rupture was defined as the occurrence of clinical symptoms (abdominal pain, abnormal fetal heart rate pattern, acute loss of contractions, vaginal blood loss) leading to an emergency caesarean delivery, at which the presumed diagnosis of uterine rupture was confirmed; or peripartum hysterectomy or laparotomy for uterine rupture after vaginal birth. Cases of scar dehiscence found during elective caesarean section without preceding clinical symptoms were not included. Women without a known uterine rupture or perforation were considered having an unscarred uterus, also after previous D&C or hysteroscopy, as these women will clinically be considered as having an unscarred uterus.

Requests for notifications of cases of uterine rupture were sent to all 98 local coordinators on a monthly basis. Cases were communicated to the National Surveillance Centre for Obstetrics and Gynaecology (NSCOG) in a web-based design. Absence of cases was also reported. Reminders were sent to nonresponders every month until they had returned the monthly notification card.

After notification, a case record form was sent to us, accompanied by anonymous photocopies of all relevant parts of the hospital case notes and correspondence. A detailed review of cases was completed by one of the researchers (JJZ) and all cases were centrally entered into an Access database.

We recorded maternal characteristics (age, body mass index, parity, ethnicity, socio-economic status, single household, language skills, smoking), obstetric history (including type of caesarean, type of incision and interpregnancy interval), all essential data on pregnancy and delivery, and neonatal outcome. We also recorded data on the specific complication, such as diagnosis-to-delivery interval, epidural analgesia, dilatation of the cervix at diagnosis, symptoms and signs at diagnosis, medicaments administered, and whether the fetus was (partially) extruded into the abdomen. A total of 108 items were entered into the database for each case. Characteristics of each hospital were also recorded (university or teaching hospital, annual number of deliveries).

Ethnicity was defined by country of origin (geographical ethnic origin) and grouped according to the most common population groups in the Netherlands (Western Europe, Morocco, Surinam/Dutch Antilles, Turkey, Sub-Saharan Africa and Middle and Far East). We used the definitions of Statistics Netherlands.[9] Women born in the Netherlands with at least one parent born abroad were considered to be from the same origin as their parent(s) from abroad. Women from other Western European countries, and women from North America, Japan and Indonesia are considered Western immigrants according to Statistics Netherlands. All other immigrant women are considered non-Western. Major obstetric haemorrhage was defined as blood loss necessitating 4 or more units of red blood cells. Weekdays from 8 a.m. to 6 p.m. were considered office hours (which equates to 30% of all hours during a week).

Denominator data for number of births in the Netherlands during the exact study period were obtained from Statistics Netherlands.[9] Births are registered based on birth certificates, which are mandatory by law beyond 24 weeks of gestational age in the Netherlands. Reference values for possible risk factors for uterine rupture were obtained from Statistics Netherlands (exact study period) and The Netherlands Perinatal Registry (LVR-2; 2005).[8] LVR-2 is the Dutch national perinatal database that covers nearly 100% of births under guidance of an obstetrician, in which parity, gestational age at delivery, mode of delivery, and place of antenatal care (midwife or obstetrician) are reliably registered. Each case is entered into the database by the attending clinician directly after birth. Data that were compared between cases and noncases were collected using the

same fact-sheet from LVR-2. Case fatality rate was calculated by dividing the number of deaths by the total number of cases.

To control for underreporting, we cross-matched our database with the LVR-2 database. During a 5-month period, cases of uterine rupture reported to this database but not to us, were identified and local coordinators were sought to re-analyse these cases and report when appropriate.

Relative risks and confidence intervals were calculated in univariable analysis. Differences between groups were identified using chi-squared or Student's *t* tests. Significance was defined as $P < 0.05$. Statistical analysis was performed using the Statistical Package for the Social Sciences 14.0 (SPSS Inc., Chicago, IL, USA).

Results

During the study period, 371 021 deliveries occurred in the Netherlands. From all 2352 (98 hospitals, 24 months) monthly notification cards, 97% were returned. Therefore, the study represents 358 874 deliveries in the Netherlands.

A total of 218 cases of uterine rupture were reported, the incidence of uterine rupture being 5.9 per 10 000 deliveries. We received detailed information of all cases (100%). Eight cases were excluded because asymptomatic dehiscence of the uterine scar was found at elective caesarean, leaving 210 confirmed cases. No maternal deaths due to uterine rupture occurred during the study period. Other severe maternal and neonatal complications are listed in Table 1. Incidence varied largely by hospital, ranging from 0 to 45.2 per 10 000. The mean 'hospital-incidence', concerning only deliveries under secondary or tertiary care, was 9.3 per 10 000; 15.4 for tertiary care centres and 8.6 for general hospitals ($P = 0.03$). Incidence figures did not differ by volume of maternity unit (data not shown). There was a trend towards more liberal use of prostaglandins for induction of labour in low-volume hospitals as compared to middle- and high-volume hospitals (24.4% versus 13.0% of cases, $P = 0.29$). Characteristics of women are shown in Table 2. Most ruptures occurred intrapartum ($n = 188$; 89.5%). In 20 cases (9.5%), rupture occurred before the onset of labour, and in two cases (1.0%) as a complication of second trimester instrumental abortion. In 16 of the intrapartum cases (8.5%), rupture was only suspected after childbirth. Ten of these were spontaneous deliveries, five were ventouse deliveries and one rupture of the posterior uterine wall was diagnosed at re-laparotomy after caesarean delivery.

Clinical symptoms that led to the diagnosis of uterine rupture included abdominal pain (69%), abnormal fetal heart rate pattern (67%), vaginal bleeding (27%), hypertonia (20%) and acute absence of contractions (14%). Among 162 women with complete reporting of all five mentioned symptoms, 91 women (56%) presented with a combination of symptoms, the most frequently encountered combination being abdominal pain and abnormal fetal heart rate pattern (Table 3).

Of all 171 cases with emergency intrapartum caesarean, 31 ruptures (18.1%) occurred during the second stage of labour. In four women, dilatation at diagnosis was not mentioned, 15 women (8.8%) had no dilatation, and in the

Table 1. Maternal and neonatal morbidity due to uterine rupture by type of induction and mode of delivery

	MOH	Hysterectomy	ICU admission	Perinatal death*	Asphyxia**	NICU admission***
Onset of delivery						
Spontaneous ($n = 130$)	19 (14.6)	4 (3.1)	11 (8.5)	9 (6.9)	21 (16.2)	12 (9.4)
Induction cervical prostaglandins ($n = 28$)	8 (28.6)	5 (17.9)	5 (17.9)	3 (10.7)	7 (25.0)	2 (9.0)
Induction oxytocin ($n = 22$)	6 (27.3)	2 (9.1)	4 (18.2)	2 (9.1)	6 (27.3)	2 (10.5)
Induction sulproston ($n = 4$)	2 (50.0)	1 (25.0)	3 (75.0)	0	0	0
Induction mechanical dilatation ($n = 4$)	0	0	0	0	0	0
Caesarean without labour ($n = 20$)	8 (40.0)	5 (25.0)	3 (15.0)	4 (20)	1 (5.0)	8 (42.1)
Mode of delivery						
Spontaneous ($n = 12$)	9 (75)	4 (33.3)	8 (66.7)	0	0	0
Ventouse ($n = 8$)	4 (50)	0	1 (12.5)	1 (12.5)	0	1 (12.5)
Caesarean ($n = 188$)	30 (16.0)	13 (6.9)	17 (9.0)	17 (9.0)	35 (18.6)	23 (12.9)
Overall ($n = 208$**)**	43 (20.7)	17 (8.2)	26 (12.5)	18 (8.7)	35 (16.8)	24 (12.1)

Values in brackets are percentages.
MOH, major obstetric haemorrhage; (N)ICU, (neonatal) intensive care unit.
*Excluding death due to congenital malformations.
**Defined as pH directly postpartum <7.00.
***Percentage among 198 neonates from 25 weeks of gestational age onwards.
****Excluding two cases of uterine rupture after instrumental abortion.

Table 2. Characteristics of women with uterine rupture

Age (mean 33.0), years	
<25	2 (1.0)
25–35	134 (63.8)
35–40	63 (30.0)
≥40	11 (5.2)
Socio-economic status	
Low	54 (28.4)
Middle	75 (39.5)
High	61 (32.1)
Unknown	20
Smoking during pregnancy	
Yes	18 (15.0)
No	108 (85.0)
Unknown	84
Body mass index	
<18.5	3 (2.1)
18.5–24.9	62 (44.3)
25.0–29.9 (overweight)	47 (33.6)
30.0–34.9 (obese)	16 (11.4)
≥35.0 (morbidly obese)	12 (8.6)
Unknown	70
Geographical ethnic origin	
The Netherlands	158 (75.2)
Morocco	9 (4.3)
Turkey	10 (4.8)
Surinam/Dutch Antilles	7 (3.3)
Sub-Saharan Africa	9 (4.3)
Other non-Western	13 (6.2)
Other Western	4 (1.9)

Values are given as *n* (%).

remaining 121 women, rupture occurred at 1–9 cm dilatation, with the highest incidence at 4–5 cm dilatation ($n = 41$).

Possible risk factors are shown in Table 4. Of all women, 182 (86.7%) had at least one previous caesarean. Seven women (3.3%) were nulliparous, four of whom were primigravid. Non-Western immigrant women did have a significantly increased risk of experiencing uterine rupture as compared to Western women [relative risk (RR) 1.4; 95% confidence interval (CI) 1.0–1.9]. Sub-Saharan African women had the highest risk (RR 3.9; 95% CI 2.0–7.7). Fifty-nine percent of uterine ruptures occurred outside office hours. Median interval between diagnosis and childbirth was 30 minutes (range 7–172) for ruptures occurring during office hours, and 40 minutes (range 9–240) outside office hours ($P = 0.09$).

The two cases of uterine rupture during instrumental abortion were complications of second trimester termination of pregnancy at 21 and 22 weeks of gestation in unscarred uteri. Reasons for termination were unwanted pregnancy and bilateral facial cleft. Both women were referred from a primary care abortion clinic. One of these women had a hysterectomy performed because of major obstetric haemorrhage. These two cases will further be disregarded as they concern complications of instrumental abortion and characteristics of delivery do not apply.

Scar rupture

Uterine rupture occurred in 183 women with a scarred uterus, population-based incidence being 5.1 per 10 000 deliveries. In two of these women, the localisation of rupture was not the uterine scar itself. All but one woman had a singleton pregnancy. Median gestational age was 40.2 weeks (range 17.2–42.7). One woman had a scar from previous myomectomy; the remaining 182 women had a scar from previous caesarean. All but six of these women (96.7%) had one previous caesarean, four had two and two had three previous caesareans. Previous caesarean was performed without labour in 72 women (39.6%) and during labour in 106 (58.2%). Three women had both types of caesarean in their obstetric history and in one the type of previous caesarean was unknown. In 18 women (9.9%), the

Table 3. Symptoms and signs at the moment of diagnosis

	Presence of symptom	Combinations of two symptoms			
		Abnormal CTG	Vaginal bleeding	Hypertonia	Acute absence contractions
Abdominal pain	133/194 (68.6%)	90/189 (47.6%)	34/181 (18.8%)	34/181 (18.8%)	16/174 (19.2%)
Abnormal CTG	134/201 (66.7%)		29/186 (15.6%)	31/185 (16.8%)	19/182 (10.4%)
Vaginal bleeding	52/190 (27.4%)			12/179 (6.7%)	5/176 (2.8%)
Hypertonia	38/188 (20.2%)				7/176 (4.0%)
Acute absence of contractions	25/184 (13.6%)				

Values in brackets are percentages.

Table 4. Possible risk factors for uterine rupture (n = 210)

	LEMMoN (%)	The Netherlands (%)	RR (95% CI)	Absolute risk (overall 1 in 1709)
Patient				
Age ≥ 35	35.2	24.7*	1.7 (1.3–2.2)	1 in 1195
Low income	28.4	n/a		
Single household	3.3	n/a		
BMI ≥ 25 (overweight)	53.6	31.7*	2.5 (1.8–3.5)	1 in 1011
BMI ≥ 30 (obese)	20.0	9.8*	2.3 (1.5–3.5)	1 in 837
BMI ≥ 35 (morbidly obese)	8.6	n/a		
Non-Western immigrants	21.0	16.8*	1.4 (1.0–1.9)	1 in 1315
Pregnancy				
Prior caesarean delivery	86.7	10.1[4]	65.1 (42.9–98.7)	1 in 198
Short interpregnancy interval (≤12 months)	13.9	n/a		
VBAC in obstetric history	10.5	n/a		
Nulliparity	3.8	45.2*	0.05 (0.02–0.10)	1 in 20 259
Primiparity	78.1	18.9**	15.3 (11.1–21.3)	1 in 413
Parity ≥ 3	5.8	5.0*	1.2 (0.6–2.1)	1 in 1493
Multiple pregnancy	1.0	1.7*	0.5 (0.1–2.2)	1 in 3116
Artificial reproduction techniques: IVF/ICSI	1.9	1.9[10]	1.0 (0.4–2.6)	1 in 1740
Delivery				
Induction of labour	33.3	12.3**	3.6 (2.7–4.8)	1 in 629
Induction of labour, prostaglandin	15.5	n/a		
Induction of labour, oxytocin	13.0	n/a		
Augmentation, oxytocin	24.2	18.9**	1.4 (1.0–1.9)	1 in 1336
Epidural anaesthesia	40.1	5.9**	10.7 (8.1–14.1)	1 in 251
Preterm birth (<37 weeks)	13.0	5.8**	2.4 (1.6–3.7)	1 in 760
Post-term birth (≥42 weeks)	9.2	4.3**	2.2 (1.4–3.6)	1 in 801

n/a, not available.
National reference values from *Statistics Netherlands (exact study period) and **The Netherlands Perinatal Registry (LVR-2; 2005).

previous caesarean was expedited before 36 weeks of gestation. In 53 women (29.1%), the previous caesarean was electively performed because of breech presentation. Incision had been low transverse in 177 cases, classical in one case, and in four cases, the type of incision was unknown.

Three women had a uterine rupture in their obstetric history. In the first one, caesarean delivery was planned because of a previous classical incision, but she experienced uterine rupture at 30 weeks. The second woman had a caesarean without labour performed at 35 weeks of gestation because of placenta praevia and thrombocytopenia. Peripartum hysterectomy was performed because of major obstetric haemorrhage due to uterine rupture and placenta praevia. The third woman experienced hypovolemic shock at 29 weeks of gestation. A fundal uterine rupture was found at emergency caesarean, along with 3 l of intra-abdominal blood and intrauterine fetal death. Peripartum hysterectomy was performed. In another woman, obstetric history revealed a scar dehiscence.

Trial of labour was attempted in 167 women (91.3%), four of whom had the previous caesarean performed before 34 weeks of gestation. The other 16 women (8.7%) had an emergency caesarean performed, most important indications being spontaneous onset of labour before planned elective caesarean, placenta praevia/percreta and suspicion of placental abruption. Relative risks of different uterotonic agents during trial of labour are shown in Table 5. In 22 of 183 cases (12.0%), prostaglandins were used for induction of labour. Reasons for induction with prostaglandins included (nearly) post-term pregnancy (n = 10), intra uterine fetal death/multiple congenital abnormalities (n = 5), elective (n = 3), pregnancy induced hypertension (n = 2), intra uterine growth restriction (n = 1) and prelabour rupture of membranes (n = 1). Prostaglandin analogues used included different variants of dinoproston (n = 16), sulproston (n = 2) and misoprostol (n = 1). In three cases, two different prostaglandin analogues were administered successively. Individual assessment of regimens of administration in these 23 cases revealed no new insights. Dosages ranged from 0.5 to 2.0 mg with a minimal interval of 4 h in between.

Mean interpregnancy interval, defined as the time between immediate previous caesarean and conception was

Table 5. Risk of uterotonic agents in trial of labour, as compared to spontaneous labour

Onset of labour	LEMMoN (*n* = 167)	The Netherlands* (*n* = 3274)	RR (95% CI)
Spontaneous labour	77	2056	1.0
Augmentation after spontaneous onset	43	536	2.1 (1.5–3.1)
Induction of labour	47	682	1.8 (1.3–2.7)
Oxytocin	20	308	1.7 (1.0–2.9)
Prostaglandin	16	203	2.1 (1.2–3.7)
Prostaglandin + oxytocin	6	94	1.7 (0.7–4.0)
Mechanical dilation ± oxytocin	5	77	1.7 (0.7–4.4)

*Reference values from a large representative sample from the Netherlands.[4]

33 months (range 3–135). Only four women had an interpregnancy interval of <6 months. Twenty-two women (12.2%) had one to three VBACs in their history. Previous VBAC tended to be protective to the fetus, but the risk of severe maternal morbidity tended to be elevated (Table 6). Complete or partial extrusion of the fetus was reported in 21 and 29 cases (11.4 and 15.9%, respectively). In nine women (4.9%) uterine rupture was complicated by rupture of the bladder.

Rupture of the unscarred uterus

Besides the two ruptures complicating second trimester instrumental abortion, 25 women experienced rupture of an unscarred uterus, incidence being 0.7 per 10 000 deliveries. Median gestational age was 38.7 weeks (range 20.7–42.8). Factors possibly associated with the rupture were history of instrumental abortion or postpartum curettage ($n = 10$), history of hysteroscopy ($n = 2$), history of ectopic pregnancy ($n = 2$), history of other pelvic surgery ($n = 1$), endometriosis ($n = 2$), uterine fibroids ($n = 1$), and twin pregnancy ($n = 1$). In 13 women (52%), we could not identify any risk factor. Severe maternal and neonatal morbidity and mortality were clearly more often observed among women with an unscarred uterine rupture as compared to uterine scar rupture (Table 7). In 11 women (44%), labour was induced, in all but one with prostaglandins. Four ruptures occurred before spontaneous onset of labour, three were discovered postpartum. In 18 women (72%), rupture occurred outside office hours. Localisation of rupture included posterior wall ($n = 5$), anterior wall ($n = 5$), lateral ($n = 3$), fundal ($n = 4$), low uterine segment ($n = 2$) and other ($n = 5$). Cervix and bladder were

Table 6. Uterine rupture after previous vaginal birth after caesarean (VBAC)

Severe morbidity/mortality	VBAC, *n* (%)	No VBAC, *n* (%)	RR (95% CI)
Maternal			
ICU admission	4 (18.2)	11 (7.0)	2.6 (0.9–7.5)
Major obstetric haemorrhage (≥4 units)	6 (27.3)	20 (12.7)	2.2 (1.0–4.8)
Major obstetric haemorrhage (≥10 units)	2 (9.1)	5 (3.2)	2.9 (0.6–13.9)
Hysterectomy	3 (13.6)	7 (4.4)	3.1 (0.9–11.0)
Fetal			
Perinatal death	1 (4.5)	12 (7.6)	0.6 (0.1–4.4)
Asphyxia	3 (13.6)	30 (19.0)	0.7 (0.2–2.2)

ICU, intensive care unit.

Table 7. Delivery and outcome in scar versus nonscar uterine rupture

Item	Nonscar (*n* = 25), %	Scar (*n* = 183), %	RR (95% CI)
Delivery			
Induction with prostaglandins	40.0	12.1	4.9 (1.7–11.2)
Before 32 weeks of gestational age	24.0	4.9	6.1 (1.5–16.7)
Prelabour emergency caesarean	16.0	8.8	2.0 (0.6–6.9)
Outcome			
ICU admission	36.0	8.8	5.5 (2.2–15.4)
≥4 units of blood transfused	56.0	15.4	6.8 (2.6–15.4)
≥10 units of blood transfused	16.0	6.0	3.7 (1.1–13.7)
Hysterectomy	24.0	6.0	4.9 (1.7–15.8)
Peripartum fetal death	24.0	7.7	3.8 (1.4–11.8)
Asphyxia*	33.3	31.4	1.1 (0.2–6.3)
Fetus completely extruded	28.0	11.0	3.0 (1.2–8.6)

ICU, intensive care unit.
*Percentages among 111 cases with a known umbilical cord pH directly after birth.

involved in six and seven cases, respectively. Complete or partial extrusion of the fetus into the abdomen was reported in nine cases (36.0%). In one case, in which the woman presented with anhydramnios and diminished fetal movements at 32 weeks of gestation, uterine rupture was diagnosed antepartum by an intra abdominal leg on MRI.[11]

Discussion

Thirteen percent of all uterine ruptures occurred in the unscarred uterus, the proportion being higher than reported before.[12] The overall incidence of uterine rupture of 5.9 per 10 000 is well within the range of incidences reported in Western countries.[6] The overall incidence reported in a WHO systemic review of uterine rupture was 5.3 per 10 000 for population-based studies, and 31 per 10 000 for facility-based studies.[6] Kwee *et al.*[13] conducted a 1-year prospective study of uterine rupture in the Netherlands, from which we could calculate a similar incidence of 5.8 per 10 000. They, however, reported only three ruptures in unscarred uteri on a total of 98.

Although no cases of maternal death due to uterine rupture occurred in our study, each of the last four triennial reports of the Confidential Enquiry into Maternal and Child Health in the United Kingdom contained at least one case of maternal death due to uterine rupture, and the most recent report described two cases.[14]

This study includes the largest prospective report of uterine rupture in women without a previous caesarean in a Western country. The only other study mentioned in the WHO systematic review reported a comparable incidence of 0.6 per 10 000,[6,15] attesting to the rarity of uterine rupture in the absence of a previous caesarean in Western countries. However, unlike previously reported,[16] we demonstrate that severe maternal and neonatal morbidity and mortality are clearly higher in these cases as compared to uterine scar rupture. Therefore, uterine rupture should always be suspected in case of clinical signs, particularly—but not exclusively—in the presence of risk factors such as previous caesarean section, primiparity, induction of labour, epidural anaesthesia, overweight or advanced age.

The majority of scar ruptures occur in the absence of macroscopic or clinical signs of blood loss. Contrarily, major haemorrhage, ICU admission and hysterectomy occur more frequent with rupture of the unscarred uterus. This is probably caused by a much lower index of suspicion in an unscarred uterus which may add to a delay in diagnosing uterine rupture. There may also be reduced blood loss in rupture of scar tissue compared to unscarred uterine rupture. Major obstetric haemorrhage is also an important presenting symptom of uterine rupture diagnosed after childbirth, which represents 8.6% of all ruptures. Therefore, differential diagnosis of major obstetric haemorrhage after previous caesarean should always include uterine rupture.

Controversy remains regarding the additional risk of uterine surgical procedures in general history like D&C or myomectomy. Even though perforations are known to go unrecognized, evidence of a causal relationship remains only circumstantial.[17] However, we report 13 cases of uterine rupture in unscarred uteri in the absence of any known risk factor.

A major strength of this study is that we prospectively collected all cases of uterine rupture instead of relying on ICD-10 codes. Therefore, the definition of uterine rupture was uniform and could be explicitly confirmed. Other large studies had to rely on ICD-codes for case ascertainment,[3,5], which have been shown to be only about 40% accurate.[18] Another key strength of the study is its nationwide and population-based design, giving a precise and generalisable estimation of the incidence for a Western country. However, the nationwide design confers also the major limitation of the study, since specific reference values of the pregnant population, such as previous method of caesarean delivery or uterotonic agents used, are missing in the national registries. This was met by using reference data from a recent representative cohort of Dutch women attempting trial of labour collected by Kwee *et al.*[4] Unfortunately, we could not adjust relative risks for possible confounding variables, since only aggregated instead of individual data were available for the nationwide reference cohort of women without uterine rupture. Furthermore, data on previous scar closure was not available, but single layer closure is common practice in the Netherlands.

We found a 3.6-fold increased risk of uterine rupture after induction of labour as compared to the general pregnant population, irrespective of agents used. Controversy remains with respect to earlier stated additional risk of induction of labour with prostaglandins. Several studies report that induction with prostaglandins confers the highest risk of uterine rupture (relative risk up to 15), but two large studies could not confirm this.[4,19,20] Case ascertainment was suboptimal using ICD-9 codes, and bias by indication may also have played a role. For the Dutch setting, Kwee *et al.*[4] reported odds ratios among 3274 trials of labour of 2.2, 3.8 and 6.8 for augmentation, induction and induction with prostaglandins, respectively. Using the same reference cohort, we could not confirm these high relative risks few years later although reported incidences of uterine rupture were similar. It is possible that the incidence has stabilised as a result of the rising prevalence of previous caesarean delivery on one hand, and the more restrictive use of uterotonic agents in women with a uterine scar on the other hand. When comparing our cohort of women experiencing uterine rupture during trial of labour to the

cohort of Kwee *et al.* (2002–03), we observed significantly less induction of labour overall ($P = 0.04$) and with prostaglandins ($P = 0.005$).

Mechanical dilation of the cervix with Dilapam or balloon catheter seems to be a good alternative on theoretical grounds,[21] although we also encountered one case of uterine rupture after induction by mechanical dilatation alone.

The majority of all uterine ruptures (80.5%) occurred during trial of labour. Assuming an estimated trial of labour percentage after caesarean in the Netherlands of 71.7%, and a percentage of women with a previous caesarean of 10.1% as reported by Kwee *et al.*,[4] 25 989 trials of labour were attempted in the Netherlands during the study period. The risk of uterine rupture would then be 0.64%, which is considerably lower than reported by Kwee (1.47%; $P < 0.001$) and well within the range of reported incidences in large reviews and retrospective studies of 0.22–0.74%.[22]

A previous VBAC is generally considered to be a protective factor for the occurrence of uterine rupture and its complications during trial of labour. However, in our study this seems to only apply to the fetus, if at all. Risk of severe maternal morbidity seemed to be rather elevated after a previous VBAC. This is an important observation that needs to be addressed by future research.

With 29% of all previous caesareans being performed for breech presentation, we clearly show the negative side effects and long-term adverse consequences of routinely performing elective caesarean for breech delivery.[23–27]

Conclusion

While much attention has been paid to the risk of induction of labour, almost half of all scar ruptures occurred during spontaneous labour. As the number of caesareans needed to prevent one uterine rupture is very high, the only means of reducing the incidence of uterine rupture is to minimise the number of inductions of labour and to closely monitor women with a uterine scar. Symptoms and signs of uterine rupture, in particular abnormal fetal heart rate pattern and abdominal pain, should be taken very seriously even in women with an unscarred uterus. Caesarean delivery should be promptly expedited in case of suspicion of uterine rupture. Between 2003 and 2006, the rate of uterine rupture associated with induction for trial of labour decreased significantly in the Netherlands. Ultimately, the best prevention is primary prevention, i.e. reducing the primary caesarean delivery rate. The obstetrician who decides to perform a caesarean has a joint responsibility for the late consequences of that decision, including uterine rupture.

Disclosure of interests

All authors declare that they have no competing interests.

Contribution to authorship

J.J.Z. conducted the project, helped supervise enrolment, analysed and interpreted the data, and wrote the manuscript. J.M.R. and F.Ö. conceived the project, provided background knowledge to the data analysis and interpretation, and provided feedback on earlier drafts of the manuscript. J.I.P.d.V. and K.W.M.B. were involved in the design of the study as members of the expert panel, and provided feedback on earlier drafts of the manuscript. J.v.R. conceived the project, was project leader, provided background knowledge to the data analysis and interpretation, and provided feedback on earlier drafts of the manuscript. J.v.R. will act as study guarantor.

Details of ethics approval

The study was centrally approved by the medical ethics committee of Leiden University Medical Centre (P04-020; 8 March 2004).

Funding

The study was supported by the Dutch Organisation for Health Research (ZonMw; grant 3610.0024) and the Matty Brand Foundation. The funding sources had no role in study design, data collection, data analysis, data interpretation, or writing of the report. JZ and JvR had full access to all study data and had final responsibility for the decision to submit for publication.

Acknowledgements

We would like to thank the members of the LEMMoN expert panel for their contribution: K.W.M. Bloemenkamp (Leiden University Medical Centre), H.W. Bruinse and A. Kwee (University Medical Centre Utrecht), E.A.P. Steegers and W. Visser (Erasmus Medical Centre), G.D. Mantel (Isala Klinieken), M.G. van Pampus (University Medical Centre Groningen), J.I.P. de Vries (VU University Medical Centre). We thank R. Rodrigues Pereira for his cooperation in establishing the national surveillance system (NSCOG).

We also greatly thank all local coordinators who kindly participated: Alkmaar: C. Akkerman (Medisch Centrum Alkmaar), Almelo: P.J.A. van der Lans (Ziekenhuis Groep Twente), Almere: G. Kleiverda (Flevoziekenhuis), Amersfoort: E. Lenters (Meander Medisch Centrum), Amstelveen: C.L. van der Wijden (Ziekenhuis Amstelveen), Amsterdam: M.H.B. Heres (Sint Lucas Andreas Ziekenhuis), J.I.P. de Vries, J.H.K. van Brummelen-Joosten (VU University Medical Centre), J.M.M. van Lith (Onze Lieve Vrouwe Gasthuis), M. Pel (Academic Medical Centre), B.M.I. Doekhie (BovenIJ Ziekenhuis), E.D. van Oudgaarden (Slotervaartziekenhuis), Apeldoorn: W.A. Spaans (Gelre Ziekenhuizen), Arnhem: K. de Boer (Ziekenhuis Rijnstate), Assen: M.W. Glas (Wilhelmina Ziekenhuis),

Bergen op Zoom: J. Ramondt (Ziekenhuis Lievensberg), Beverwijk: J. Roest (Rode Kruis Ziekenhuis), Blaricum: J.J. Dieles (Tergooiziekenhuizen), Boxmeer: A.G. Minkhorst (Maasziekenhuis), Breda: D.N.M. Papatsonis (Amphia Ziekenhuis), Capelle a/d IJssel: W.F. Lam (IJsselland Ziekenhuis), Delft: A. Dijkman, H.A. Bremer (Reinier de Graaf Groep), Delfzijl: A. van Zanten (Delfzicht Ziekenhuis), Den Bosch: H.P. Oosterbaan, M.H.H.M. Kerkhof (Jeroen Bosch Ziekenhuis), Den Haag: F.T.H. Lim, J. van Dillen (HagaZiekenhuis), C.A.G. Holleboom (Ziekenhuis Bronovo), C.B. Vredevoogd (Medisch Centrum Haaglanden), Den Helder: J. Friederich (Gemini Ziekenhuis), Deventer: J.M. Schierbeek (Deventer Ziekenhuis), Dirksland: N.P.J. Vreuls (Van Weel-Bethesda Ziekenhuis), Doetinchem: F.J.L. Reijnders (Slingeland Ziekenhuis), Dokkum: M.M. Henselmans (Ziekenhuis Talma-Sionsberg), Dordrecht: B.M.C. Akerboom (Albert Schweitzer Ziekenhuis), Drachten: P.J. van den Hurk (Ziekenhuis Nij Smellinghe), Ede: H.J. Kwikkel (Ziekenhuis Gelderse Vallei), Eindhoven: S.M.I. Kuppens (Catharina Ziekenhuis), Emmen: J.M. Burggraaff (Scheperziekenhuis), Enschede: P.R. Poeschmann (Medisch Spectrum Twente), Geldrop: A. Neijmeijer-Leloux (Sint Annaziekenhuis), Goes: M. Baaij (Oosterscheldeziekenhuizen), Gorinchem: R. Euser (Rivas Medizorg), Gouda: J.C.M. van Huisseling (Groene Hart Ziekenhuis), Groningen: G.G. Zeeman (University Medical Centre Groningen), A.J. van Loon (Martini Ziekenhuis), Haarlem: J. Clements (Kennemer Gasthuis), Hardenberg: P.J.M. Baudoin (Röpcke Zweers Ziekenhuis), Harderwijk: R.L. van de Pavert (Ziekenhuis Sint Jansdal), Heerenveen: G.H. Weenink (Ziekenhuis Tjongerschans), Heerlen: F.J.M.E. Roumen (Atrium Medisch Centrum), Helmond: J.H.J.M. van der Avoort (Elkerliek Ziekenhuis), Hengelo: P. Paaymans (Streekziekenhuis Midden-Twente), Hilversum: M. van Hoven (Tergooiziekenhuizen), Hoofddorp: A. Lub, M. de Lange (Spaarne Ziekenhuis), Hoogeveen: M. Koppe (Ziekenhuis Bethesda), Hoorn: T.W.A. Huisman (Westfries Gasthuis), Leeuwarden: J.G. Santema (Medisch Centrum Leeuwarden), Leiden: J.C.M. Spiekerman (Diaconessenhuis), K.W.M. Bloemenkamp (Leiden University Medical Centre), Leiderdorp: O.J.A. Mattheussens (Rijnland Ziekenhuis), Lelystad: C.N. de Boer (IJsselmeerziekenhuizen), Maastricht: C. Willekes (University Hospital Maastricht), Meppel: G.M. Vermeulen (Diaconessenhuis), Nieuwegein: E. van Beek (Sint Antonius Ziekenhuis), Nijmegen: D.H. Schippers (Canisius-Wilhelmina Ziekenhuis), Nijmegen: J.W.T. Creemers (Radboud University Nijmegen Medical Centre), Oss: M.A.L. Verwij-Didden (Ziekenhuis Bernhoven), Purmerend: H. Prins (Waterlandziekenhuis), Roermond: L.C.G. Wetzels (Laurentius Ziekenhuis), Roosendaal: R. Pal (Franciscus Ziekenhuis), Rotterdam: G.C.H. Metz (Ikazia Ziekenhuis), P.E. van der Moer (Medisch Centrum Rijnmond Zuid), N. van Gemund (Sint Franciscus Gasthuis), J.J. Duvekot (Erasmus Medical Centre), Sittard: J. Alleman (Maaslandziekenhuis), Sneek: E.A. van Eyk (Antonius Ziekenhuis), Spijkenisse: F.W. Worst (Ruwaard van Putten Ziekenhuis), Stadskanaal: G.W. Oostendorp (Refaja Ziekenhuis), Terneuzen: J.W.E. Voitus van Hamme (Ziekenhuis Zeeuws-Vlaanderen), Tiel: R. Hardeman (Ziekenhuis Rivierenland), Tilburg: C.M. van Oirschot (Sint Elisabeth Ziekenhuis), A.E.M. Roosen (TweeSteden Ziekenhuis), Utrecht: J. Lange (Mesos Medisch Centrum), H.W. Bruinse (University Medical Centre Utrecht), N.W.E. Schuitemaker (Diakonessenhuis), Veghel: Y.H.C.M. van Zwam (Ziekenhuis Bernhoven), Veldhoven: B.W.J. Mol (Máxima Medisch Centrum), Venlo: C.G.M. de Rooy (Vie Curi Medisch Centrum Noord Limburg), Vlaardingen: R.J.H. Oostendorp (Vlietland Ziekenhuis), Vlissingen: S. de Boer (Ziekenhuis Walcheren), Weert: W.E. Nolting (Sint Jansgasthuis), Winschoten: P.H. van Drooge (Sint Lucas Ziekenhuis), Winterswijk: D.M.R. van der Borden (Streekziekenhuis Koningin Beatrix), Woerden: I.H. Goedhuis (Hofpoort Ziekenhuis), Zaandam: K. Brouwer (De Heel Zaans Medisch Centrum), Zevenaar: R.J.C. Mouw (Ziekenhuis Zevenaar), Zoetermeer: J.M.T. Roelofsen (Lange Land Ziekenhuis), Zutphen: Y.A.J.M. Dabekausen (Gelre Ziekenhuizen), Zwolle: J. van Eyck (Isala Klinieken). ■

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Mini commentary on 'Uterine rupture in the Netherlands: a nationwide population-based cohort study'

This population-based prospective cohort study generates questions that need to be considered in counselling and managing women with previous caesarean section (CS) scar. The incidence of uterine rupture was 5.9 per 10 000 deliveries; the denominator was deliveries and not those who had a previous CS scar. This low incidence may be because of the strict criteria used to define rupture and because of the lower incidence of women with previous CS in Netherlands (CS rate was 14% in 2002). Eighty-seven percent of ruptures were in women who were known to have a scar. Although induction of labour was associated with a relative risk of 3.6, 72% of rupture occurred during spontaneous labour and rupture was witnessed at various cervical dilatations. The morbidity and mortality for the infant was higher with a rupture and the most common symptom and sign were abdominal pain and an abnormal fetal heart rate (FHR) pattern and were witnessed in two-third of cases. These observations are likely to bring about reservations to the leniency expressed in management of women having labour remote from hospital and not having continuous fetal monitoring if they were not on oxytocics or not well advanced in labour.

In univariate analysis, advanced age, pre- and post-term pregnancy, over weight, non-Western ethnic background and use of epidural had an elevated risk of uterine rupture. It would be difficult to change these factors in a given labour except the epidural analgesia. There is no mechanism to explain why epidural should increase the risk of uterine rupture. Anecdotal cases suggest that it may be because of an increase in intra-abdominal and thereby the intra-uterine pressure causing stress on the scar over a period of time when the woman is flexed to administer the epidural. This may be greater in an over weight woman. One may not be able to avoid the rupture, but diagnose it early if continuous electronic fetal monitoring (EFM) is undertaken during this time.

An acute bradycardia, rapidly worsening cardio toco-graphy (CTG) pattern (increase in depth and duration of decelerations within a 10 to 20 minutes window) or CTG changes with abdominal pain are likely to be associated

with a rupture or impending rupture. But FHR changes of a 'lesser degree' can be because of other causes other than scar rupture. There is no mechanism to determine that the FHR changes are because of a mechanism of cord compression similar to what may occur in a woman without a scar and not because of imminent scar rupture. This philosophy will tempt one to postpone operative intervention by doing a fetal pH by scalp blood sampling (FBS). In such situations, review of the whole clinical picture, that is, parity, current cervical dilatation, rate of dilatation, whether the woman is on oxytocin (Alliance Pharmaceuticals, Chippenham, UK) infusion and other risk factors may help in making an appropriate decision to intervene and deliver or to do FBS. Certainly, one needs to be hesitant to allow progress with repeated FBSs.

We should aim to reduce primary CS rates, scrutinise indications and avoid if possible induction in women with previous CS to reduce the incidence of uterine rupture. We should provide information to women and the levels of risk for them to consider regarding the place of labour, use of EFM, epidural and FBS to provide satisfaction, but minimise risks associated with uterine rupture.

Disclosure of interest

None. ■

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Editor-in-Chief's Commentary

Continuous fetal heart rate monitoring is a sensitive screening test for fetal hypoxia. However, because hypoxia can be produced and fetal heart rate (FHR) patterns affected by temporary factors that do not lead on to acidosis, such as intermittent cord compression and maternal postural hypotension, it has a high false positive rate for asphyxia. Indeed, only about half of fetuses with even the most abnormal FHR patterns will have a significant acidosis (Beard *et al.*, *J Obstet Gynaecol Br Commonw* 1971;78:865–81). The most direct way to assess fetal acidosis is to take a fetal blood sample and measure its pH (FBS), and this has been promoted as a way to avoid an unnecessarily high caesarean section (CS) rate for 'fetal distress' (Parer, *Clin Obstet Gynecol* 1980;23(2):565–82). However, there is surprisingly little objective evidence to support the usefulness of FBS. For example, the latest guidelines of the National Institute for Health and Clinical Excellence (NICE) in the UK (updated June 2008, http://www.nice.org.uk/nicemedia/pdf/CG55FullGuideline.pdf), state that 'There is limited evidence from randomised trials that FBS with continuous fetal monitoring may reduce instrumental birth and CS. The research evidence does not support the use of FBS because of the lack of direct comparison, but clinical experience and evidence from indirect comparisons suggests that FBS avoids some instrumental births and CS'. Thus, the use of FBS is based on clinical impression rather than on direct evidence and in many parts of the world (for example the USA), it is seldom used. The NICE guidelines go on to state that 'Where assisted birth is contemplated because of an abnormal FHR pattern, in cases of suspected fetal acidosis FBS should be undertaken in the absence of technical difficulties or any contraindications. Where there is clear evidence of acute fetal compromise (for example, prolonged deceleration >3 minutes), FBS should not be undertaken and urgent preparations to expedite birth should be made'. One contraindication is suspected chorio-amnionitis. pH is not usually abnormal, except in the terminal stages, in fetuses affected by infection (Maberry *et al.*, *Obstet Gynecol* 1990;76:351–4) and thus, FBS is inappropriate if it is thought that fetal compromise is secondary to this cause. In my view, another contraindication is a uterine scar. Fetal oxygenation is unlikely to be sufficiently impaired to cause acidosis until a scar rupture is substantial, by which time the decision to deliver is arguably already too late. In the current study by Zwart *et al.*, an abnormal fetal heart rate pattern was one of the signs that led to the diagnosis of uterine rupture in two-thirds (67%) of cases. As uterine rupture can progress very rapidly from the initial signs through to a catastrophic collapse of both maternal and fetal circulations secondary to haemorrhage, I would argue that the finding of a normal fetal pH despite an abnormal FHR pattern cannot be regarded as sufficient reassurance that the labour should be allowed to continue. I agree with the authors' conclusion that 'Caesarean delivery should be immediately performed in case of suspicion of uterine rupture, without prior assessment of fetal condition by fetal scalp blood sampling', because delaying delivery may prove fatal.

Supporting information

The following supplementary materials are available for this article:

Appendix S1. Declaration of Interests.

Additional Supporting Information may be found in the online version of this article.

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting information supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author. ■

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