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Original Research Article

Uterine atony risk factor after vaginal delivery in a tertiary hospital in Antananarivo, Madagascar

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ABSTRACT

Background: Postpartum hemorrhage is the leading cause of maternal death in developing countries. Uterine atony is the cause in 80% of cases. Through this study, we want to determine risk factors for uterine atony after vaginal delivery route with oxytocin-mediated delivery.
Methods: This is a retrospective case-control study ranging from January 1st 2017 to June 31st 2018 at the Befelatanana University Hospital Centre of Gynecology-Obstetrics. The cases consisted of patients who had spontaneous vaginal delivery in the centre and had uterine atony. Authors studied maternal, obstetrical, neonatal parameters. Authors used the R software for the statistical analysis of the results.
Results: We found 40 cases of uterine atony out of 5421 deliveries with a prevalence of 0,73%. The average age was 27.73 years old±6.46 years old (p=0.113). The average parity was 2.67±1.62 (p=0.22). The total duration of labor was 6.88±2.95 hours (p=0.0187). The average duration of rupture of the membrane was 5.80±11.90 hours (0.003376). We found as risk factor of uterine atony the increase in oxytocin infusion rate during labor (OR=18.67, 95% CI 2.21-157.57), the artificial rupture of membranes (OR=5, 27, 95% CI 2.11-13.19), artificial induction of labor (OR=7.08, 95% CI 2.06-24.28) and labor over six hours (OR=2.53, 95% CI) % 1.18-5.47). In univariate analysis, premature delivery and a hypotrophic fetus were a factor risk of uterine atony (OR=3.07, 95% CI 1.27-7.44 and OR=3.43 95% CI 1.48-8.09 respectively) but this risk is not statistically significant in multivariate analysis with logistic regression (OR=1.27, 95% CI 0.40-3.84 and OR=2.19 95% CI 0.77-6.22). The main treatment was uterotonic drug use (72.5%). Authors identified seven cases of haemostasis hysterectomy and two cases of maternal death.
Conclusions: Present study confirms risk factors for uterine atony already known as prolonged labor and increased oxytocic infusion rate. Unrecognized factors have been identified as a risk factor for uterine atony such as the duration of rupture of the membranes and artificial rupture of the membranes. A minimal inflammation hypothesis that reduces susceptibility to oxytocin may explain this association. Knowing these factors would reduce the occurrence of uterine atony to reduce maternal mortality.

Keywords: Emergency peripartum hysterectomy, Oxytocic therapy, Postpartum hemorrhage, Risk factor, Uterine atony

INTRODUCTION

Postpartum hemorrhage is the leading cause of maternal death in the world. This is a major problem in public health. Despite the evolution of healthcare during obstetric labor, its frequency remains high. It affects 10% of deliveries and would be responsible for 13,200 annual maternal deaths worldwide.[1,2] Uterine atony is the main

etiology. In fact, 80% of postpartum hemorrhages are secondary to uterine atony.[3,4]

Uterine atony is defined as the generalized hypocontractility of the myometrium after delivery.[5] In Madagascar, almost half of emergency peripartum hysterectomie are secondary to uterine atony.[6]

The occurrence of postpartum hemorrhage by uterine atony is a significant source of maternal morbidity and mortality. The causes are numerous.

Risk factors such as overdistension of uterine cavity (twin pregnancy, polyhydramnios, macrosomia), prolonged use of oxytocic, infection, prolonged labor are recognized.[5] Several ways have been proposed to reduce the risk of uterine atony.

The use of uterotonic agents, early clamping of the umbilical cord and controlled traction of the umbilical cord are the three keys components of active management of the third phase of labor.

Routine administration of oxytocic after anterior shoulder delivery significantly reduces the risk of bleeding.[7] From our Centre, the administration of oxytocic drug during delivery is systematic. Despite this practice, uterine atony is a significant morbidity and mortality factor in this Centre.

No studies have ever been conducted on risk factors for uterine atony after vaginal delivery route in Madagascar. This context led us to carry out this study, which aims to determine risk factors for uterine atony after vaginal delivery route with oxytocin-mediated delivery.

METHODS

This is a case-control study conducted from January 1st 2017 to June 31st 2018 at the Befelatanana University Hospital of Gynecology-Obstetrics (CHUGOB). It is a reference Hospital in the capital and its surroundings in the field of Gynecology and Obstetrics.

Inclusion criteria

- All cases of uterine atony in parturient who had vaginal delivery and who received 10 IU oxytocin before delivery to UHCGOB after 22 weeks of amenorrhea (WA).

Exclusion criteria

- Cases of uterine atony referred to the Centre after delivery outside from the Centre,
- Cases of multifactorial postpartum hemorrhage (uterine rupture, cervicovaginal lesions) associated with uterine atony.

The cases were patients with uterine atony who had been diagnosed within 24 hours of delivery. The diagnosis is made by a senior obstetrician. The data was collected by consulting medical records, birth and admission records.

The controls consisted of parturient who delivered after 22 WA at the same Centre during the study period but did not show uterine atony. For each case we recruited two controls.

Authors studied:

- Maternal parameters: age, gestational age, parity, occupation, maternal body mass index, level of maternal education, obstetrical history, smoking, alcoholism
- Obstetric parameters: number of prenatal consultations, birth interval, number of previous miscarriages, gestational age, uterine height, duration of labor, attempt at vaginal delivery route, occurrence of fever during labor, induction of labor, color of amniotic fluid, duration of rupture of the amniotic membrane, artificial rupture of membranes, occurrence of hypertension during pregnancy, history of cesarean section, fetal death in utero.
- Neonatal parameters: Apgar score at birth (first minute, fifth minute), birth weight, gender of the newborn
- Treatment of uterine atony

Authors compared one case for two controls. Categorical variables were compared using Khi-square or Fisher's exact tests where appropriate. Significance for all analyses was defined as a P value < 0.05. Parametric variables were expressed as mean±standard deviation. Student's T test was used for comparison of parametric variables. We used the R software for the statistical analysis of the results.

RESULTS

During the study period, we had 5 421 births at CHUGOB. Among these, 40 patients had uterine atony after delivery, which represented 0.74 % of deliveries. We recruited 89 controls.

The average age of cases was 27.73±6.46 years old. The average birth weight of the cases was 2691.35g. We found only one case of fetal macrosomy during the study period.

There is no significant difference in the two groups. The number of miscarriages is significantly associated with the occurrence of uterine atony ($p=0.01$). Labor time in the case group was higher than in the control group ($p=0.02$).

The duration of the rupture of the membrane was significantly associated with the occurrence of uterine atony ($p=0.003$). The amount of bleeding and length of Hospital stay were higher in the case group (Table 1 and 2).

Table 1: Demographics, obstetric, and neonatal data.

Variables	Case (Mean±SD)	Control (Mean±SD)	P-value
Birth weight	2691.35±648.79	2867.42±546.04	0.113
Neonatal size (cm)	45.44±4.23	47.54±4.28	0.02073 *
Age	27.73±6.46	28.35±6.95	0.6311
Gestity	2.67±1.62	2.82±1.89	0.674
Parity	1.32 ±1.29	1.72±1.85	0.2254
Number of antenatal care	3.70±1.70	4.18±1.32	0.08374
Inter-delivery interval (years)	2.75±2.87	2.30±2.38	0.3577
Maternal body mass index	22.02±3.54	21.71±2.78	0.5914
Number of previous miscarriage	0.35±0.77	0.09±0.36	0.009695*
Gestational age (weeks of amenorrhea)	36.67±3.74	37.44±3.15	0.2322
Uterine size (cm)	28.90±2.92	29.40±2.53	0.32
Duration of labor	6.88±2.95	5.89±1.81	0.0187 *
Duration of rupture of the amniotic membrane	5.80±11.90	1.97±1.70	0.003376*
Apgar score 1st mn	8.20±3.41	9.13±2.18	0.06273
Apgar score 5th mn	9.18±2.45	9.51±1.71	0.3783
Blood loss volume	824±285.26	160.28±54.90	2.10-16*
Duration of hospitalization	3.58±2.71	2.82±1.15	0.03132*

*statistically significant

Table 2: Maternal parameters.

Variables		Case n=40(%)	Control n=89 (%)	P
Age	<18 years	4 (10)	9 (10.11)	0.9964
	19-25 years	12 (30)	25 (28.09)	
	25-35 years	19 (47.5)	44 (49.44)	
	>35 years	5 (12.5)	11 (12.36)	
Profession	Housewife	20 (50)	45 (50.56)	0,9768
	Primary sector	9 (22.50)	22 (24.72)	
	Secondary sector	2 (15)	11 (12.36)	
	Tertiary sector	5 (12.50)	11 (12.36)	
Parity	Nulliparous	14 (35)	27 (30.34)	0.6392
	Primiparous	9 (22.5)	23 (25.84)	
	Pauciparous	17 (42.5)	36 (40.45)	
	Multiparous	0 (0)	3 (3.37)	
Study level	University	5 (12.5)	12 (13.48)	0,6159
	Illiterate	17 (42.5)	32 (35.96)	
	Primary school	7 (17.5)	25 (28.09)	
	Secondary school	5(12.5)	6 (6.74)	
	High school	6 (15)	14 (15.73)	
Smoking		1 (2,5)	0	0.6802
Alcohol		1 (2,5)	0	0.6802
Maternal death		2 (5)	0	0.1752

Maternal age and parity are not a risk factor for uterine atony (Table 3). The occurrence of hypertensive disorder was identical between the two groups. Neither the use of antispasmodics nor the use of magnesium sulphate was associated with the occurrence of uterine atony. 40% of uterine atony occurred after artificial rupture of membranes. The induction of labor was significantly associated with uterine atony (Table 4). The increase in oxytocic infusion rate increased the risk of uterine atony by 18 times (OR 18.67, 2.21-157.57). Artificial rupture of membrane was five times more likely to be associated with uterine atony (OR 5.27 (2.11-13.19).) Artificial labor induction was seven times higher in the case group (OR 7.08). % 2.06-24.28) Prolonged labor for more than six hours increased the risk of uterine atony by 2.5 times (p=0.0191), but the occurrence of in utero fetal death, preeclampsia, Prolonged and prolonged pregnancy, transfer for failed attempted vaginal delivery route from another Centre, fever during labor, and meconium amniotic fluid were not associated with uterine atony (Table 5). The occurrence of uterine atony is not

correlated with the occurrence of neonatal asphyxia (Table 6 and 7). In univariate analysis, premature delivery and a hypotrophic fetus were a risk factor for uterine atony (OR=3.07, 95% CI 1.27-7.44 and OR=3.43 95% CI 1.48-8.09 respectively) (Table 7) but this risk is not statistically significant in multivariate logistic regression analysis (aOR=1.27, 95% CI 0.40-3.84 and a OR=2.19 95% CI 0.77- 6.22) (Table 8).

The main treatment was uterotonic drug use (72.5%). Authors identified seven cases of emergency peripartum hysterectomy (Table 9). Authors identified two cases of maternal death.

Table 3: Association between maternal parameters and uterine atony.

Variables		OR	95%	P
Age (years)	<19	Referent	-	
	19-25	1.08	0.29-4.63	0.912
	25-35	0.97	0.28-3.94	0.965
	>35	1.02	0.21-5.24	0.978
Parity	Nulliparous	1.10	0.46-2.61	0.8323
	Primiparous	0.83	0.3-2.14	0.7019
	Pauciparous	Referent	-	-
	Multiparous	0	0	-

Table 4: Obstetrical parameters.

Variables		Case n=40 (%)	Controls n=89 (%)	P
HTA (n=39)	Without HTA	30 (75)	80 (89.89)	0.2213
	Pregnancy hypertension	5 (12.5)	5 (5.62)	
	Preeclampsia	3(7.5)	2 (2.25)	
	Eclampsia	1 (2.5)	2 (2.25)	
Increase in oxytocic infusion rate		7(17.5)	1 (1.12)	0.001512
Antispasmodic drug		1 (2.5)	0 (0)	0.6802
Magnesium sulfate injection		1 (2.5)	0(0)	
Intrauterine fetal death		2 (5)	2 (2.25)	0.7755
Artificial rupture of membrane		16 (40)	10(11.24)	0.0004164
Gestational age (week of gestation)	< 37	14 (35)	15 (16.85)	0.01652
	37-41	21 (52.5)	69 (77.53)	
	> 41	5 (12.5)	5 (5.62)	
Preeclampsia		4 (10)	4 (4.49)	0.3993
Prolonged labour		21 (52.50)	27 (30.34)	0.02698
Scared uterus		0	2 (2.24)	0.8531
Anterior postpartum hemorrhage		1 (2.5)	0	0.6802
Neonatal asphyxia		4 (10)	4 (4.49)	0.421
Referred for failure of vaginal birth attempt		2 (5)	4 (4.49)	0.8997
Fever during labour		3(7.50)	2 (2.25)	0.349
Labor induction		10 (25)	4 (4.49)	0.001593
Amniotic fluid N=85	Meconial	9 (22.5)	10 (11.24)	0.3322
	Clear	31 (77.5)	75 (84.27)	

Table 5: Association between obstetrical parameters and occurrence of uterine atony.

Variables		OR	95% CI	P
Increase in oxytocic infusion rate		18.67	2.21-157.57	0.0011
Antispasmodic drug		-	-	0.3101
Intrauterine fetal death		2.29	0.31-16.86	0.5874
Artificial rupture of membrane		5.27	2.11-13.19	0.0003
Gestational age (week of amenorrhea)	< 37	3.07	1.27-7.44	0.0123
	37-41	1	-	-
	> 41	3.29	0.84-12.9	0.0801
Preeclampsia		2.43	0.58-10.26	0.2455
Prolonged labour		2.53	1.18-5.47	0.0191
Referred for failure of vaginal birth attempt		1.12	0.09-8.18	0.8997
Absence of hyperthermia during labour		0.28	0.04-1.76	0.1725
Labor induction		7.08	2.06-2428	0.0012
Amniotic fluid N=85	Meconial	1.81	0.61-5.23	0.3031
	Clear	1	-	-

Table 6. Neonatal parameters.

Variables		Case, N=40(%)	Control, N=89 (%)	P
Birth weight	Eutrophic	23 (57.5)	74 (83.15)	0.004536
	Hypotrophy	16 (40)	15 (16.85)	
	Macrosomia	1 (2.5)	0 (0)	
Gestational age (week of gestation)	< 37	14 (35)	15 (16.85)	0.01652
	37-41	21 (52.5)	69 (77.53)	
	> 41	5 (12.5)	5 (5.62)	
Neonatal asphyxia		4 (10)	4 (4.49)	0.421
Gender	Male	30 (75)	59 (66.29)	0.485
	Female	10 (25)	29 (33.71)	

Table 7: Association between neonatal parameters and occurrence of uterine atony.

Variables		Or	95%	P
Birth weight	Eutrophic	1	-	-
	Hypotrophy	3.43	1.48-8.09	0.00426**
	Macrosomia	-	-	0.99083
Prematurity (< 37 SA)		3.07	1.27-7.44	0.0123*
Gender	Male	1	-	0.4129
	Female	1.47	0.63-3.42	

Table 8: Factor associated with uterine atony, multiple logistic regressions.

Variables		OR	95%	P
Duration of rupture of the amniotic membrane		1.17	1.03-1.39	0.0363
Birth weight	Eutrophic	Referent	-	-
	Hypotrophy	2.19	0.78-6.22	0.13
	Macrosomia	-	-	
Gestational age (weeks of gestation)	37-41	Referent	-	-
	< 37	1.28	0.39-3.84	0.67
	> 41	2.86	0.70-11.7	0.14

Table 9: Uterine atony treatment.

Variables	Case N=40(%)	Control N=89 (%)
Expectative	0 (0)	89 (100)
Uterotonic drug (oxytocin, prostaglandin)	29 (72.5)	0
Uterine packing	3 (7.5)	0
B-lynch	1 (2.5)	0
Hysterectomy	7 (17.5)	0

DISCUSSION

The average age of our patients was 27.7 years old; almost half of them were between 25 and 35 years old. This is similar to the literature data.[8-11] Age is not associated with the occurrence of uterine atony in our study. Butwick, Feerasta and Rueda found the same result.[9-13] The average parity was the same between the two groups. There is no significant difference regarding parity. Wetta and Grotegut found that nulliparous parturients were 1.4 times more likely to have uterine atony (OR 1.4 IC 1.0-2.1).[8,11] In the literature, multiparity is the main risk of uterine atony.[5,7] However, in this study, we did not identify multipara in the case group. A large-scale study with a larger population would be needed to assess the role of parity in the occurrence of uterine atony after vaginal delivery route. In our study, the number of previous miscarriages exposed the risk of uterine atony. Our study is the first to demonstrate this association. In-depth studies would provide explanations on this association. The likelihood of placenta accreta may explain this association and increase the risk of uterine atony.

The duration of labor was associated with the occurrence of uterine atony. This risk factor was already identified by several authors.[7,13] The origin cause was the muscular exhaustion at the origin of a uterine inertia. The duration of the rupture of the membrane is also associated with the occurrence of uterine atony. To our knowledge, this is the first study that highlights this association. In a multivariate analysis, prolonged rupture of the membranes poses a risk of uterine atony. According to Zackler et al, the occurrence of chorioamnionitis would affect the uterine contraction by decreasing its contractility.[14] However, a prolonged rupture of the membrane exposes the occurrence of chorioamnionitis. Inflammation in the uterus induces IL-1β release, which acts as a down regulation of the oxytocin receptor on the surface of myometrial cells.[15] Although we did not demonstrate evidence of chorioamnionitis in this study, this local mechanism of inflammation cannot be ruled out. Thus, an increase in oxytocic infusion rate would be necessary to avoid uterine atony. Moreover, in our study, artificial rupture of the membranes increases the risk of uterine atony by five times compared with spontaneous rupture. For Wetta and Feerasta, this association is not significant.[8,10] In a univariate analysis, deliveries of a fetus <2500 g were most at risk of uterine atony. In a multivariate analysis associating the birth weight, the term, the duration of rupture of the amniotic membrane, this risk becomes statistically insignificant. In the literature, it is the deliveries of a macrosome which are the factors of a uterine atony by mechanism of overdistension.[5,7] This is a classic risk factor for postpartum hemorrhage.[1] Wetta et al did not find a

statistically significant association between the occurrence of uterine atony and fetal weight. In this study, we did not find fetal macrosomia. A large-scale study with a larger population would study this association.

In a univariate analysis, premature labor is a risk of uterine atony in this study. This risk is four times higher compared to a term pregnancy (OR 3.87, 95% CI (1.03-14.78).) In a multivariate analysis, this risk does not exist. The etiology of premature deliveries were dominated by premature rupture of the membranes, because these premature deliveries had a longer average duration of amniotic membrane rupture compared to a term of pregnancy (6.93hours vs. 1.93 hours). In a multivariable logistic regression models, only the duration of membrane rupture was significant, which confirms our hypothesis of the existence of local inflammation after rupture of membranes decreasing uterine sensitivity to oxytocin. Wetta et al. did not find a significant association between premature delivery and the occurrence of uterine atony.[8]

The increase in oxytocic infusion rate during labor increases the risk of uterine atony by eighteen times. Long-term exposure to oxytocic drug and increased plasma levels have been shown to promote the development of uterine atony. This is explained by the fact that oxytocin receptors (OXTR) undergo desensitization after activation. Such a situation would be responsible for lack of response after delivery resulting in uterine atony.[16-19] this explains the occurrence of uterine atony despite the use of oxytocin 10 IU intravenous injection before anterior shoulder delivery in our study. This risk of postpartum hemorrhage is much higher if the increase in oxytocin infusion rate is short-lived (<20 min).[20] The role of certain medications in the occurrence of uterine atony has not been demonstrated in our study. In the literature, the use of magnesium sulfate increases the risk of postpartum hemorrhage by uterine atony.[7,8] For Wetta et al, the use of $MgSO_4$ increases the risk of uterine atony by twice. This medication is a calcium-like inhibitor and acts as a utero-relaxant. Antispasmodics such as phloroglucinol increase the risk of uterine atony four times (OR 4.67, CI 1.31-16.68, p=0.018).[21] The size of our sample is small and very low. Very few patients had received these medications.

Hypertensive disorders during pregnancy and preeclampsia are not a risk factor for uterine atony in this study. Butwick et al, Feerasta et al found a similar result.[10,12] In contrast, Wetta and Grotetegut found an increased risk of uterine atony in preeclamptic patients (OR=2.8 and 4.90 respectively).[8,19] In our context, it is the retroplacental hematoma that is complication of preeclampsia which is the leading cause of uterine atony. But in practice, we do not accept vaginal delivery route unless for a very few patients. This makes the occurrence of uterine atony after vaginal delivery route very rare in preeclamptic patients in this study. Indeed, when all uterine atony were combined during the study period in the Centre, regardless of the route of delivery, preeclampsia increased the risk of uterine atony four times (OR=4.35, CI95% 1.66 to 11.39).

Our study confirms data from the literature concerning the role of labor induction in the occurrence of uterine atony (OR=5.27 95% CI 2.11-13.19). Wetta et al found a risk twice as high.[7,8,12,13,22] In three-quarters of the cases, treatment was medicated by uterotonic administration. This is the first-line treatment for uterine atony. Second-line treatment includes uterine packing, Bakri balloon, external compression with uterine sutures and uterine devascularization techniques. Hysterectomy is the treatment of last resort.[23] In our experience, in case of hemodynamic instability from the outset, hysterectomy hemostasis is indicated. Previous studies in the Centre had shown that uterine atony was responsible for 48.39% of hemostasis hysterectomy. The absence of uterine embolization explains the use of this technique.[6] The limitation of this study is the small sample size, the monocentric nature of the study. Our sample size may not be sufficient to evaluate potential risk factors that are less frequent. Thus, several classic risk factors such as multiple pregnancies, macrosomia, polyhydramnios, multiparity, manual delivery and obesity were not identified in this study. This makes it difficult to extrapolate to the general population.

CONCLUSION

To conclude, present study confirms risk factors for uterine atony already known as prolonged labor and increased oxytocic flow. Unrecognized factors have been identified as a risk factor for uterine atony such as rupture time of membranes and artificial rupture of membranes. A minimal inflammation hypothesis that reduces susceptibility to oxytocin may explain this association. A large-scale multicenter study would provide confirmation of these variables. These risk situations must be identified as soon as the patient is admitted for close supervision at the time of delivery. The use of uterotonics should be systematic and the learning of conservative techniques in uterine atony should be developed to reduce maternal morbidity and mortality secondary to uterine atony.

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