International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Hassan MF et al. Int J Reprod Contracept Obstet Gynecol. 2022 Apr;11(4):1048-1053
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20220882

Original Research Article

Labor outcome among obese postdate women undergoing labor induction

Mahmoud F. Hassan[1]*, Nancy M. A. Rund[2]

[1]Department of Obstetrics and Gynecology, Faculty of Medicine, Ain Shams University, Cairo, Egypt
[2]Department of Obstetrics and Gynecology at Bugshan Hospital, Jeddah, Saudi Arabia

Received: 06 February 2022
Accepted: 02 March 2022

***Correspondence:**
Dr. Mahmoud F. Hassan,
E-mail: mahmoudfathy74 @yahoo.com

ABSTRACT

Background: Maternal obesity and postdate pregnancy are common findings among pregnant women worldwide. We aimed to evaluate the influence of maternal obesity on the outcome of labor induction for postdate pregnant women.
Methods: We conducted a prospective observational study to compare 118 obese women (≥30 kg/m^2) with 118 non-obese women (<30 kg/m^2) undergoing labor induction for postdate pregnancy (≥41 weeks). We induced all participants by a uniform protocol according to the Bishop score. The primary outcome measures were the cesarean delivery (CD) rate and the rate of failed induction. Secondary outcomes included prolonged induction- delivery time, prolonged first and second stage of labor, and rate of instrumental delivery. We performed a multivariate regression model to assess for the relation between obesity and the study outcomes of interest.
Results: Cesarean delivery was significantly higher in obese women when compared with non-obese women (25.4% vs. 12.7%, p=0.02). Likewise, failed induction rate was significantly lower among non-obese women (5.1% vs. 14.4%. p=0.026). Obese women had increased odds for CD (adjusted odds ratio: 2.24; 95% confidence-interval: 1.13-4.33), failed induction rate (adjusted OR 2.96; 95% CI: 1.15-8.17), prolonged induction-delivery time (adjusted OR 4.57; 95% CI: 1.42-14.74), prolonged first stage of labor (adjusted OR 3.32; 95% CI: 1.07-9.89), prolonged second stage of labor (adjusted OR 4.21; 95% CI: 1.27-13.62), and rate of instrumental delivery (adjusted OR 2.97; 95% CI: 1.16-8.23).
Conclusions: Obesity adds more risk to postdate women undergoing induction of labor. Obesity increases the incidence of CD and failed induction among induced postdate women. Therefore, obstetricians should encourage obese women to reduce weight before getting pregnant, and to comply with the optimal weight gain during pregnancy in attempt to reduce the rates of postdating, CD and failed induction.

Keywords: Cesarean section, Delivery, Labor induction, Obesity, Postdate

INTRODUCTION

Obesity is a global growing epidemic disease. Several impacts arise at the maternal and fetal levels among obese pregnant women that necessitate multidisciplinary care in the obstetric practice.[1] Obesity is commonly presents with comorbid medical illnesses as diabetes mellitus and hypertensive disorders. Additionally, many researches declared that obese women are at increased risk of developing intrapartum and postpartum complications including labor dysfunction, shoulder dystocia, and caesarean delivery (CD).[2,3] Conspicuously, labor induction is also a common procedure among obese pregnant women.[4] It was estimated that nearly 35% of obese women might require labor induction.[5]

On the other hand, prolonged pregnancy is another risky obstetric identity that raises the probability of postpartum

hemorrhage and CD rates.[6,7] Furthermore, concerns arising from postdate pregnancies are common among obese women.[8] Several studies demonstrated that obesity was associated with a reduced likelihood of spontaneous labor at term and an increased risk of postdate pregnancy.[9,10] The combination of prolonged pregnancy and obesity increases the obstetric challenge, and requires an efficient and coordinated management between obstetricians, midwives, neonatologists, anesthesiologists, and other medical professions according to the possible pre-existing medical co-morbidities.[2,11] Noticeably, data regarding the impact of obesity on labor induction particularly in postdate women are not fully elucidated.[12] Therefore, the present study was designed to evaluate the relation between maternal obesity and the success rates of labor induction among women with postdate pregnancy.

METHODS

We conducted the present prospective observational study at the department of obstetrics and gynecology of a private hospital, Jeddah, Saudi Arabia, from February 2010 till March 2017. This trial was performed in compliance with the declaration of Helsinki. The study included postdate women (≥ 41 weeks gestation) with a single viable fetus in cephalic presentation, a reactive non-stress test, a Bishop score of ≤7, and with no spontaneous uterine contractions (< 3-4 uterine contractions within 20 minutes). Gestational age was calculated based on the last menstrual period or an early first-trimester sonography. We excluded women with body mass index (BMI) <18.5 kg/m^2, multiple pregnancy, prior uterine surgery, active labor, ruptured membranes, chorioamnionitis, antepartum hemorrhage, contra-indication to prostaglandins use, diabetes, hypertension, and major fetal anomalies or demise. All participants signed an informed consent before enrolment in the study.

Our study incorporated 236 women with postdate pregnancy planned for induction of labor (IOL). Baseline data and maternal BMI were obtained from all participating women immediately before IOL. We stratified the participating women into two groups: 118 obese women with a BMI ≥30 kg/m^2 and 118 non-obese women with a BMI <30 kg/m^2. Labor induction was conducted according to our hospital protocol. A sterile vaginal examination was done to find out the Bishop score. Accordingly, patients with Bishop score ≤ 7 received 3 mg dinoprostone vaginal tablet into the posterior vaginal fornix for a maximum of three doses with 6 hourly intervals. Before application of each prostaglandin dose, we performed a vaginal examination to ascertain the Bishop score and external cardiotocography (CTG) to assess fetal well-being and frequency of uterine contractions. Whenever any patient deemed a Bishop score more than 7 or passed into active labor, she was started the active management of labor 6 hours after placement the last dinoprostone dose. Active labor was defined as at least three firm, rhythmic uterine contractions with duration ≥ 40 seconds occurring within a 10-minute period, or achievement of 4 cm dilatation. Active management of labor included amniotomy followed by intravenous oxytocin after two hours if no efficient uterine contractions. We started oxytocin infusion with initial dose of two milliunits per minute. Then, we increased the infusion rate by two milliunits per minute at 30-minute intervals to a maximum dose of 32 milliunits per minute or till 3 - 4 uterine contractions per 10 minutes were achieved. We commenced a continuous electronic fetal monitoring, once the oxytocin infusion was started.

The primary outcome measures were the caesarean delivery (CD) rate and rate of failed labor induction (defined in our institution as a failure to attain active labor within 12 hours after the third dinoprostone vaginal tablet). Secondary outcome measures were prolonged induction to delivery time (defined as more than 95th percentile in this cohort, >26.5 hours), prolonged first stage (defined as more than 95th percentile in this cohort >10.5 hours), prolonged second stage (defined as more than 95th percentile in this cohort >1.5 hours), instrumental delivery, third-or fourth-degree laceration, postpartum hemorrhage (defined as the loss of more than 500 ml of blood following vaginal delivery or more than 1,000 ml following CD), puerperal infection (defined as the occurrence of any of the following: endometritis, wound cellulitis requiring antibiotics, wound reopened for fluid collection or infection, or wound dehiscence), APGAR score at 5 minutes, and neonatal intensive care unit (NICU) admission.

Statistical analysis

The required sample size was calculated using the PS - power and sample size calculation, version 3.0.43 (department of biostatistics, Vanderbilt University, Nashville, TN, USA). Sample size calculation was based on CD rate in obese versus non-obese groups as the primary outcome. According to a previous study, there was 14.5% CD rate in non-obese pregnant women undergoing labor induction.[13] We set the power at 80%, alpha error at 0.05, and ratio of the two study groups at 1:1. Accordingly, 118 cases were needed in each group to detect 15% difference in CD rate. The data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) version 23. We used the Kolmogorov-Smirnov test to assess for the normality of distribution of the continuous variables. The quantitative data were presented as mean and standard deviations when parametric and median with inter-quartile range (IQR) when non-parametric. In addition, qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done by the Chi-square test or Fisher exact test when the expected count in any cell found less than 5. The comparison between two independent groups with quantitative data and parametric distribution was

done by Independent t-test. Meanwhile, non-parametric data were compared using Mann-Whitney test. Logistic regression analysis was done between obese and non-obese regarding CD rate, failed induction rate, prolonged induction to delivery time, prolonged first and second stage of labor, and instrumental delivery rate. Then, multivariate regression analysis was done to adjust for maternal age, parity, smoking, gestational age, Bishop score, fetal weight, and amniotic fluid index. The Hosmer-Lemeshow goodness-of-fit test was used to evaluate the final models.

RESULTS

In total, 267 women were screened in this study. Thirty-one women declined from the study as they were not meeting the criteria for inclusion (N=22) or refusing induction of labor (N=9). Two hundred Thirty-six women were stratified into an obese women group (N=118) and a non-obese women group (N=118). The baseline characteristics in both groups (obese and non-obese) were similar as depicted in (Table 1).

Table 1: Baseline characteristics of the study population (n=118).

Variables	Obese, N (%)	Non-obese N (%)	P value
Age (years) (mean±SD)	26.8±4.23	27.5±5.32	0.264
Maternal age above 35 years	10 (8.5)	8 (6.8)	0.807
Parity	1 (0-2)	1 (0-2)	0.553
Multiparity	67 (56.8)	61 (51.7)	0.514
Nulliparity	51 (43.2)	57 (48.3)	
Gestational age (weeks) (mean±SD)	40.3±2.4	39.8±3.2	0.175
BMI (kg/m^2) (mean±SD)	33.5±4.15	26.4±3.25	<0.001
Smoking	5 (5.9)	4 (4.7)	1.0
Neonatal birth weight (g) (mean±SD)	3205.5±117.6	3182.7±205.5	0.297
Amniotic fluid index (cm) (mean±SD)	10.3±3.2	9.8±2.8	0.203
Bishop score	4 (2-5)	3 (1-5)	0.650
Total dinoprostone doses needed for induction	2 (1-3)	2 (1-2)	0.731

Data presented as mean±standard deviation, median (interquartile range), or number (%), p value <0.05 was considered as significant.

Table 2: Clinical outcome measures of labor induction in the study population (n=118).

Variables	Obese, N (%)	Non-obese N (%)	P value
Cesarean section	30 (25.4)	15 (12.7)	0.02
Multipara	18/67 (26.9)	4/61 (6.6)	0.002
Primipara	12/51 (23.5)	11/57 (19.3)	0.764
Failure of induction	17/118 (14.4)	6/118 (5.1)	0.026
Indication of cesarean delivery			
Failure of induction	17/30 (56.7)	6/15 (40)	0.469
Fetal distress	5/30 (16.6)	5/15 (33.3)	
Failure of progress	8/30 (26.7)	4/15 (26.7)	
Duration till vaginal delivery (hours) (mean±SD)	22.8±5.8	15.1±4.3	0.017
Prolonged induction- delivery time	14/88 (15.9)	4/103 (3.9)	<0.001
Prolonged first stage	12/88 (13.6)	5/103 (4.9)	<0.001
Prolonged second stage	13 (14.8)	4 (3.9)	<0.001
Instrumental delivery	14 (15.9)	6 (5.8)	<0.001
3rd or 4th degree vaginal laceration	2 (2.3)	1 (0.8)	0.623
Postpartum hemorrhage	4 (3.4)	6 (5.1)	0.749
Puerperal infection	4 (3.4)	2 (1.7)	0.683
APGAR score at 5 minutes	7 (5-9)	8 (6-10)	0.067
NICU admission	3 (2.5)	5 (4.2)	0.722

Data presented as mean±standard deviation, median (interquartile range), or number (%), p value <0.05 was considered as significant.

No statistical difference was found in maternal age, parity, gestational age, smoking habit, neonatal birth weight, amniotic fluid index, Bishop scoring, and total dinoprostone doses needed for labor induction. The outcome measures of labor induction is depicted in (Table 2). The total cesarean section rate was significantly higher in the obese women compared to non-obese women (25.4% vs. 12.7%, respectively; p=0.02). Cesarean delivery in multiparous obese women was also higher than non-obese group (p=0.002). Meanwhile, primiparous women had similar cesarean section rate in both groups (p=0.764). The induction

failure rate was significantly higher in the obese group compared to the non-obese group (14.4% vs. 5.1%, respectively; p=0.026). Third- or fourth-degree laceration in vaginal delivery, postpartum hemorrhage, puerperal infection, APGAR score at 5 minutes, and NICU admission did not differ between the two groups. However, duration till vaginal delivery was significantly longer in the obese group than the non-obese women (22.8±5.8 vs. 15.1±4.3, respectively; p=0.017). As well, prolonged induction to delivery time, prolonged first and second stage of labor, and instrumental delivery rate were significantly increased in the obese group when compared to the non-obese group (p<0.001). The association between obesity and clinical outcomes of interest is shown in (Table 3).

Table 3: Association between maternal obesity and clinical outcomes after labor induction (n=118).

Variable	Obese	Non-obese	OR (CI)	P value	Adjusted* OR (CI)	P value
Failure of induction	17 (14.4)	6 (5.1)	3.14 (1.19-8.28)	0.021	2.96 (1.15-8.17)	0.031
Cesarean section	30 (25.4)	15 (12.7)	2.34 (1.18-4.63)	0.014	2.24 (1.13-4.33)	0.022
Prolonged Induction- delivery time †	14/88 (15.9)	4/103 (3.9)	4.68 (1.48-14.8)	0.009	4.57 (1.42-14.74)	0.018
Prolonged first stage †	12/88 (13.6)	5/103 (4.9)	3.4 (1.16-9.95)	0.026	3.32 (1.07-9.89)	0.032
Prolonged second stage †	13/88 (14.8)	4/103 (3.9)	4.29 (1.35-13.69)	0.014	4.21 (1.27-13.62)	0.021
Instrumental delivery †	14/88 (15.9)	6/103 (5.8)	3.06 (1.22-8.34)	0.029	2.97 (1.16-8.23)	0.037

*Odds ratio was adjusted for maternal age, parity, smoking, gestational age, Bishop score, fetal weight, and amniotic fluid index.
† The denominator varies from the total sample size because it was limited to women with vaginal deliveries, p value <0.05 was considered as significant.

Obesity was associated with a more than twofold increase in the odds of caesarean delivery and a more than threefold increase in the odds of failed induction of labor. Obesity also associated with greater odds of prolonged induction to delivery time, prolonged first and second stage of labor, and instrumental delivery (Table 3). In multivariable analysis, obesity remained significantly associated with caesarean delivery (adjusted OR 2.24, 95% CI: 1.13-4.33), failed induction (adjusted OR 2.96, 95% CI: 1.15-8.17), prolonged induction to delivery time (adjusted OR 4.57, 95% CI: 1.42-14.74), prolonged first stage (adjusted OR 3.32, 95% CI: 1.07-9.89), prolonged second stage (adjusted OR 4.21, 95% CI: 1.27-13.62), and instrumental delivery (adjusted OR 2.97, 95% CI: 1.16-8.23) (Table 3).

DISCUSSION

Obesity is now a widespread chronic illness. Consequently, more obese women of reproductive age are incessantly increasing.[14] Conspicuously, obese pregnant women are more liable to progress to postdate, and to endure an induction of labor.[16] However, data regarding the impact of obesity on labor induction of postdate pregnant women are deficient in literature. In this prospective observational study, we evaluated the impact of obesity on the labor induction outcomes in postdate women. We found that obesity increased the incidence of failed induction and caesarean delivery among induced postdate pregnant women. Moreover, obesity prolongs the duration of first and second stage of labor, and induction to delivery time. These differences in labor initiation and labor progress may be caused by plentiful inflammatory and endocrine changes present in obese women.[2] Many in vitro studies displayed a reduced frequency and force of uterine contractions as maternal BMI increased.[17,18] The inhibitory effects of elevated maternal leptin or cholesterol levels and dwindled calcium flux pay take part in uterine quiescence or a suppression of myometrial activity in obese women.[19,20] Furthermore, maternal obesity is associated with lower corticotrophin releasing hormone and cortisol levels, or potentially elevated estrogen levels which might impact length of the pregnancy and delay parturition.[21,22]

Ronzoni and colleagues retrospectively examined the association between BMI and CD rate among a cohort of 7,543 women with singleton term pregnancies undergoing labor induction and reported a higher CD rate in obese women compared with non-obese women (37% vs. 25%, respectively; p<0.001).[23] Similarly, a recent retrospective study for 329 participants analyzed the effect of BMI on the outcome of labor induction and concluded that women with higher BMIs had an increased risk of CD (p<0.0006) and a longer induction to delivery time (p<0.01).[24] In collaboration with our data, Wolfe and co-workers performed a population based cohort study to compare the rate of failed IOL between obese and normal-weight women and declared that the induction failure rates were allied with increasing obesity class from 13% in normal weight women to 29% in class III obese women (body mass index ≥40 kg/m^2). On top, obesity had increased odds for failed IOL by more than two folds. We also demonstrated prolonged first and

second stage of labor in obese women in comparison to non-obese women. Comparable results were stated by Kominiarek et al. who evaluated 118,978 women with singleton term cephalic pregnancies. Among this cohort, nulliparous women with a BMI of ≥40 took 1.2 hours longer to reach 10 cm than women with a BMI <25. For multiparous women, it took women with a BMI of ≥40 significantly longer time to reach 6 cm compared with women with a BMI of <25 (3.4 vs. 2.4h). In addition, Kominiarek et al declared that the incidence of operative vaginal delivery increased in consort with the rise of BMI in both the nulliparous women ($p<0.001$) and the multiparous women ($p<0.05$).[25]

Our findings, however, differ from that of Roloff and co-workers who found that obesity (BMI>30) did not influence the risk of CD ($p=0.475$), active labor duration ($p=0.383$), and operative delivery ($p=0.530$) among 133 induced pregnant women.[26] Possible explanations might be that Roloff used a smaller sample size. Besides, in their study, obese women required a larger cumulative oxytocin dose to achieve vaginal birth during labor induction. Another study by Beckwith et al stated no difference in the caesarean delivery rate among obese and non-obese women with mechanical cervical ripening (31% versus 29%, $p<0.69$). Furthermore, there was a comparable rate of failure to achieve active labor in both obese and non-obese women undergoing mechanical cervical ripening (19 versus 15%, $p=0.55$).[27] It is plausible that mechanical cervical ripening had a different nature in comparison with prostaglandins induction method. As well, mechanical induction might be more independent of maternal weight than prostaglandins. Current study had many strength points. It was a prospective and powered study. Besides, we studied all patients in a single center managed by a single practice group according to a uniform labor induction protocol. Nevertheless, our study has some limitations. First, the study was conducted in a single center that may limit the generalizability of our conclusions. Second, we didn't study the effect of various methods of labor induction as mechanical cervical ripening. Third, we used the BMI at the labor admission which might be biased because it takes into account the gestational weight gain. Therefore, future multi-centeric studies considering a gestational weight gain along with BMI at labor admission might provide better evaluation for the impact of obesity on labor induction.

CONCLUSION

In conclusion, obesity added more risk to postdate pregnant women who required induction of labor. Obese postdate women undergoing labor induction had a higher incidence for caesarean delivery, failed induction, prolonged induction to delivery time, and prolonged active stage of labor when compared to non-obese women. Hence, obstetricians should make every attempt to encourage obese women for weight reduction prior to get pregnant and to advise them for optimal weight gain during pregnancy so as to avoid the bad consequences of obesity during pregnancy and at labor induction.

ACKNOWLEDGMENTS

Authors would like to thank the obstetrics and gynecology department at Bugshan hospital for the great assistance during the data collection.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Ng M, Fleming T, Robinson M. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2014;384(9945):766-81.
2. Carlson NS, Hernandez TL, Hurt KJ. Parturition dysfunction in obesity: time to target the pathobiology. Reprod Biol Endocrinol. 2015;13:135.
3. Kobayashi N, Lim BH. Induction of labour and intrapartum care in obese women. Best Pract Res Clin Obstet Gynaecol. 2015; 29(3):394-405.
4. Goldstein RF, Abell SK, Ranasinha S. Association of gestational weight gain with maternal and infant outcomes: a systematic review and meta-analysis. JAMA. 2017;317(21):2207-25.
5. Arrowsmith S, Wray S, Quenby S. Maternal obesity and labour complications following induction of labour in prolonged pregnancy. BJOG. 2011;118(5):578-88.
6. Lee VR, Darney BG, Snowden JM. Term elective induction of labour and perinatal outcomes in obese women: retrospective cohort study. BJOG. 2016; 123(2):271-8.
7. Tanaka K, Matsushima M, Izawa T. Influence of maternal obesity on fetal growth at different periods of pregnancies with normal glucose tolerance. J Obstet Gynaecol Res. 2018;44(4):691-6.
8. Caughey AB, Stotland NE, Washington AE. Who is at risk for prolonged and postterm pregnancy? Am J Obstet Gynecol. 2009;200(6):683.e1-683-5.
9. Denison FC, Price J, Graham C. Maternal obesity, length of gestation, risk of postdates pregnancy and spontaneous onset of labour at term. BJOG. 2008;115(6):720-5.
10. Dinatale A, Ermito S, Fonti I. Obesity and fetal-maternal outcomes. J Prenat Med. 2010;4(1):5-8.
11. Brusati V, Brembilla G, Cirillo F, et al. Efficacy of sublingual misoprostol for induction of labor at term and post term according to parity and membrane integrity: a prospective observational study. J Matern Fetal Neonatal Med. 2017;30(5):508-13.
12. Maged AM, El-Semary AM, Marie HM, et al. Effect of maternal obesity on labor induction in postdate pregnancy. Arch Gynecol Obstet. 2018;298(1):45-50.

13. Rasmussen OB, Rasmussen S. Cesarean section after induction of labor compared with expectant management: no added risk from gestational week 39. Acta Obstet Gynecol Scand. 2011;90(8):857-62.
14. Arrowsmith S, Kendrick A, Hanley JA. Myometrial physiology--time to translate? Exp Physiol. 2014; 99(3):495-502.
15. Carpenter JR. Intrapartum Management of the Obese Gravida. Clin Obstet Gynecol. 2016;59(1):172-9.
16. Wolfe KB, Rossi RA, Warshak CR. The effect of maternal obesity on the rate of failed induction of labor. Am J Obstet Gynecol. 2011;205(2):128.e1-7.
17. Cedergren MI. Non-elective caesarean delivery due to ineffective uterine contractility or due to obstructed labour in relation to maternal body mass index. Eur J Obstet Gynecol Reprod Biol. 2009;145(2):163-6.
18. Fyfe EM, Rivers KS, Thompson JM. Elevated maternal lipids in early pregnancy are not associated with risk of intrapartum caesarean in overweight and obese nulliparous women. BMC Pregnancy Childbirth. 2013;13:143.
19. Moynihan AT, Hehir MP, Glavey SV. Inhibitory effect of leptin on human uterine contractility in vitro. Am J Obstet Gynecol. 2006;195(2):504-9.
20. Zhang J, Bricker L, Wray S. Poor uterine contractility in obese women. BJOG. 2007;114(3):343-8.
21. Mercer BM, Macpherson CA, Goldenberg RL. Are women with recurrent spontaneous preterm births different from those without such history? Am J Obstet Gynecol. 2006;194(4):1176-85.
22. Mission JF, Marshall NE, Caughey AB. Obesity in pregnancy: a big problem and getting bigger. Obstet Gynecol Surv. 2013;68(5): 389-99.
23. Ronzoni S, Rosen H, Melamed N. Maternal obesity class as a predictor of induction failure: a practical risk assessment tool. Am J Perinatol. 2015; 32(14):1298-304.
24. Lassiter JR, Holliday N, Lewis DF. Induction of labor with an unfavorable cervix: how does BMI affect success? J Matern Fetal Neonatal Med. 2016;29(18): 3000-2.
25. Kominiarek MA, Zhang J, Vanveldhuisen P, et al. Contemporary labor patterns: the impact of maternal body mass index. Am J Obstet Gynecol. 2011;205(3): 244.e1-8.
26. Roloff K, Peng S, Sanchez-Ramos L. Cumulative oxytocin dose during induction of labor according tomaternal bodymass index. Int J Gynaecol Obstet. 2015;131(1):54-8.
27. Beckwith L, Magner K, Kritzer S. Prostaglandin versus mechanical dilation and the effect of maternal obesity on failure to achieve active labor: a cohort study. J Matern Fetal Neonatal Med. 2017;30(13):1621-6.

Cite this article as: Hassan MF, Rund NMA. Labor outcome among obese postdate women undergoing labor induction. Int J Reprod Contracept Obstet Gynecol 2022;11:1048-53.