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Original Research Article

Study of Factors Influencing the Onset of Lactogenesis II

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Abstract

Background: The benefits of breastfeeding have been established in infancy and beyond. Lactogenesis II which is defined as onset of copious milk production after birth typically occurs within 72 hours postpartum and is a critical factor in initiating and establishment of successful breastfeeding. If this stage occurs beyond 72 hours after childbirth it is termed as delayed onset of lactation (DOL). DOL is found to be associated with reduced milk supply, shortened breastfeeding duration, problematic weight loss in newborns, early cessation of breastfeeding and thereby negatively influencing the rate of exclusive breastfeeding. A thorough understanding of prevalence of DOL in the mother and the factors influencing it helps in anticipating difficulties in advance and to provide enhanced support to breastfeeding mothers in this critical period.

Objectives:

- To study the prevalence of delayed onset of lactogenesis II.
- To study the factors influencing the onset of lactogenesis II and the risk factors for delayed onset of lactation.

Methods: A cross-sectional study involving 107 mother-infant dyads was conducted to assess onset of Lactogenesis II in relation to maternal age, education, BMI, parity, mode of delivery, and breastfeeding practices, among other factors. Statistical significance was evaluated using p-values (significance set at $p<0.05$).

Results: The overall prevalence of delayed onset of Lactogenesis II was 26.2%. No statistically significant associations were found between Lactogenesis II and maternal age, education, socioeconomic status, infant gender, comorbidities, gestational age, birth weight, or most breastfeeding practices. However, significant associations were observed for maternal BMI ($p=0.001$), parity ($p=0.045$), and mode of delivery ($p=0.021$). Vaginal delivery and multiparity were positively correlated with the early onset of Lactogenesis II, whereas higher BMI had increased incidence of DOL. Lesser number of feeds in the first 2 days of postpartum was associated with significant delay in onset of lactogenesis II ($p=0.014$ on day 1 and $p=0.008$ on day 2) Newborns of mothers with DOL had significant weight loss on day 4 of life ($p=0.001$).

Conclusion: Our findings demonstrated that, while most demographic and clinical variables showed no significant effect, maternal BMI, parity, and mode of delivery were significant predictors of Lactogenesis II. These findings highlight the importance of considering maternal physiology and obstetric history in postnatal lactation support strategies.

Keywords: Lactogenesis II; Exclusive breastfeeding; DOL; Maternal BMI; Parity; Mode of delivery; Breastfeeding initiation; Infant weight loss; Cesarean section; Breastfeeding patterns; Neonatal outcomes.

Introduction

Breast milk serves as the natural and ideal first food for infants, supplying all the essential energy and nutrients required during the initial months of life. Lactation refers to the biological process through which milk is produced and secreted from the mammary glands following childbirth—a

practice commonly known as breastfeeding or nursing [1,2].

Breastfeeding is universally acknowledged for its exceptional health benefits for both mothers and infants. For mothers, it significantly reduces the risk of developing breast cancer, ovarian cancer, and type 2 diabetes. For infants, breastfeeding is crucial in supporting immune system regulation and tissue development. It offers a comprehensive supply of essential nutrients required for optimal growth, while also delivering biologically active components such as immune cells, probiotics, triglycerides, oligosaccharides, and other immune-enhancing factors [3,4].

Lactogenesis I typically begins around the 16th week of gestation. During this phase, the epithelial cells within the mammary acini undergo differentiation into secretory cells that begin to produce colostrum—a nutrient-rich, immunoglobulin-dense fluid. Following childbirth and the complete expulsion of the placenta, progesterone levels rapidly decline, lifting the inhibition on prolactin and initiating lactogenesis II [5,6]. This second phase of lactation, marked by a substantial increase in milk production, generally occurs within 48 to 72 hours postpartum and is commonly experienced by mothers as breast fullness or engorgement [7,8]. The timely onset of lactogenesis II fosters confidence in breastfeeding and reduces the likelihood of early formula supplementation. However, the transition relies heavily on physiological changes in the mammary epithelial cells, which can be delayed or disrupted by factors such as preterm birth or cesarean delivery. When lactogenesis II begins 72 hours or more after birth, it is considered to be delayed [7,9]. A notable delay in the onset of lactogenesis II can negatively affect successful lactation outcomes. Several risk factors have been associated with delayed or failed lactogenesis II, including being a first-time mother (primiparity), maternal obesity, and certain medical conditions such as gestational diabetes mellitus, pregnancy-induced hypertension, and hypothyroidism. Additional contributing factors may include a stressful labor and delivery experience, unplanned cesarean sections, a delay in initiating the first breastfeeding session, low frequency of breastfeeding during the immediate postnatal period, and a history of breast surgery or trauma [2,10,11]. Thus, this study was planned to investigate the factors influencing the onset of Lactogenesis II after childbirth.

Objective of the Study

The study was conducted to study the prevalence of delayed onset of lactogenesis II, to study the factors influencing the onset of lactogenesis II and to understand the risk factors for delayed lactogenesis.

Methodology

We conducted a cross sectional study for a period of 3 months from march 2025 to may 2025, where we enrolled healthy mothers and term newborns breastfeeding in postnatal ward at Oxford Medical College and Hospital.

Informed consent of participating individuals was obtained. Our study included 107 mother-infant dyads and was conducted to assess Lactogenesis II in relation to maternal age, education, BMI, parity, mode of delivery, and breastfeeding practices, among other factors.

The exclusion criteria for the study included newborns requiring NICU admission, mothers with a history of breast surgery, mothers on medications known to affect lactation, those experiencing post-delivery complications, multiple births and preterm deliveries

The data was collected and compiled in MS Excel. Descriptive statistics has been used to present the data. To analyse the data SPSS (Version 26.0) was used. Significance level was fixed as 5% (α = 0.05). Qualitative variables are expressed as frequency and percentages and Quantitative variables are expressed as Mean and Standard Deviation. To compare the proportion between variables, chi-square test was used.

Results

Table 1:

Variables	Lactogenesis II, n (%)		p-value
	Normal N=79 (73.8%)	**Delayed N=28 (26.2%)**	
I Antenatal Factors			
A) Age group of Mother			
<20 years	5 (83.3%)	1 (16.7%)	0.924
20–30 years	64 (73.6%)	23 (26.4%)	
>30 years	10 (71.4%)	4 (28.6%)	
B) Educational status of mother			
Illiterate	2 (66.7%)	1 (33.3%)	0.763
Primary	5 (83.3%)	1 (16.7%)	
Middle School	14 (87.5%)	2 (12.5%)	

High School	37 (67.3%)	18 (32.7%)	
Diploma	6 (75.0%)	2 (25.0%)	
Graduate	14 (77.8%)	4 (22.2%)	
Postgraduate	1 (100.0%)	0 (0.0%)	
C) Socioeconomic Status			
Upper	1 (50.0%)	1 (50.0%)	0.890
Upper Middle	9 (75.0%)	3 (25.0%)	
Lower Middle	36 (72.0%)	14 (28.0%)	
Upper Lower	26 (74.3%)	9 (25.7%)	
Lower	7 (87.5%)	1 (12.5%)	
D) Parity			
Primipara	34 (70.8%)	14 (29.2%)	**0.045**
Multipara	45 (76.3%)	14 (23.7%)	
E) BMI			
Underweight	19(95%)	1 (5%)	**0.001**
Normal	44(83%)	9(16.9%)	
Overweight	11(55%)	9(45%)	
Obesity	5(35.7 %)	9(64.2%)	
F) Breastfeeding Counselling during antenatal period			
Yes	3 (60.0%)	2 (40.0%)	0.604
No	76 (74.5%)	26 (25.5%)	
G) Comorbidities			
Yes	12 (75.0%)	4 (25.0%)	0.908
No	67 (73.6%)	24 (26.4%)	
H) Gestational Weight Gain			
GWG	12.09 ± 3.46	12.36 ± 3.89	0.733
II. Natal Factors			
A) Gestational Age At Birth			
37–42 weeks	78 (73.6%)	28 (26.4%)	1.000
>42 weeks	1 (100.0%)	0 (0.0%)	
B) Mode of Delivery			
Normal Vaginal Delivery	56 (90.2%)	6 (9.6%)	**0.021**
Assisted Vaginal Delivery	4 (80.0%)	1 (20.0%)	
Elective LSCS	12 (48.0%)	13 (52.0%)	
Emergency LSCS	7 (46.6%)	8 (53.3%)	
C) Gender of baby			
Male	47 (78.3%)	13 (21.7%)	0.459
Female	32 (68.1%)	15 (31.9%)	
D) Birth Weight			
<2.5 kg	10 (71.4%)	4 (28.6%)	0.826
2.5–4.0 kg	69 (74.2%)	24 (25.8%)	
E) Weight for Gestational Age Classification			
SGA	23 (74.2%)	8 (25.8%)	0.666
AGA	54 (75.0%)	18 (25.0%)	
LGA	2 (50.0%)	2 (50.0%)	
F) Initiation of Breastfeeding			
<30 minutes	54 (79.4%)	14 (20.6%)	0.235
30 minutes–1 hour	19 (63.3%)	11 (36.7%)	
1–2 hours	6 (66.7%)	3 (33.3%)	
G) Feeding of Colostrum			
Yes	75 (73.5%)	27 (26.5%)	0.834
No	4 (75.0%)	1 (25.0%)	
III. Postnatal Factors			
A) Pattern of Breastfeeding			
On Demand	27 (77.1%)	8 (22.9%)	
Timed Feeds	52 (72.2%)	20 (27.8%)	0.646

B) Problems Associated with Breast Feeding			
Breastfeeding Issues present	17 (80.9%)	4 (19.04%)	0.248
No Problems	62 (72.1%)	24 (27.9%)	
C) Stool Color on Day 4			
Transitional	63 (72.4%)	24 (27.6%)	0.582
Normal	16 (80.0%)	4 (20.0%)	
D) Avg. Number of Feeds on Day 1			
	9.2 ± 2.1	6.5 ± 2.5	**0.014**
E) Avg. Number of Feeds on Day 2			
	12.1± 1.7	9.8 ± 1.9	**0.008**
F) Urine Voids on Day 4			
	5.37 ± 1.69	4.68 ± 1.63	0.064
G) Stool Voids on Day 4			
	3.37 ± 2.19	2.68 ± 1.16	0.115
H) Weight Loss on Day 4 (WL)			
	4.67 ± 3.33	7.47 ± 3.63	**0.001**

A total of 107 healthy mothers and term newborns were studied. Majority of the mothers were in the age group between 20 - 30 years. Among the mothers studied, 26.2% of mothers experienced delayed onset of lactogenesis. Maternal age (p=0.924), educational (p= 0.763) and socioeconomic status (p=0.890) did not have an impact on onset of lactation. Among antenatal factors, higher BMI (p= 0.001) and primiparity (p=0.045) were associated with delayed onset of lactogenesis. Antenatal counselling regarding breastfeeding (p=0.604), presence of maternal co morbidities (p=0.908) did not influence the time of onset of lactation.

Mothers who delivered by LSCS (p= 0.021) and mothers of babies who received fewer feeds on day 1 (p = 0.014) and day 2 (p = 0.008) of life also experienced delayed onset of lactation. Babies of mothers who had delayed onset of lactation had excess weight loss on D4 of life (p = 0.001) compared to mothers of babies with early onset of lactation

Discussion

In our study, among 107 participants, most of the women (73.8%) experienced lactogenesis II within 72 hours of birth, whereas 26.2% had a delayed or absent lactogenesis II, similarly, Mohammed et al. [12] reported a incidence at 30.5% and in the Salahudeen et al. [11] study, 25% of mothers experienced delayed lactogenesis II, with a mean onset time of 66.95 hours. In a meta - analysis study done by Peng et al [13], overall incidence of delayed onset of lactogenesis was 30%. Among mothers of male infants, 78.3% showed early signs of Lactogenesis II, compared to 68.1% of female infants. However, with a p-value of 0.459, this difference was not statistically significant in our study.

In the present study, we examined the relationship between maternal age and the onset of Lactogenesis II. Mothers younger than 20 years had the highest prevalence of early lactogenesis (83.3%), followed by those aged 20–30 years (73.6%) and over 30 years (71.4%). Despite these differences, the p-value of 0.924 indicated no significant association. Similarly, Peng et al [13] and Salahudeen et al. [11] reported no statistically significant relationship between maternal age and delayed lactogenesis.

In our study, the educational level of the mother did not significantly affect the occurrence of Lactogenesis II (p = 0.763) similar to meta-analysis done by Peng et al.

In our study, higher maternal BMI was associated with delayed onset of lactogenesis with obese mothers showing the highest rate followed by overweight mothers. Similar to our study, Salahudeen et al. [11] study showed a statistically significant association (p = 0.09), where delayed onset was slightly more common in those with BMI >24 with 7% obese in their study. Peng et al [13] also revealed pre pregnancy BMI > 25kg/m2 is also a risk factor for delayed onset of lactogenesis. Higher BMI and obesity before pregnancy are strongly associated with delayed lactogenesis. Obesity induces hormonal dysregulation (elevated leptin, slower estrogen clearance, reduced prolactin response), chronic inflammation of mammary tissue, and circadian disruptions that impair timely milk production [15,16,17]. Research has indicated that women who are overweight or obese before pregnancy exhibit a reduced response to prolactin stimulated by sucking [15]. Animal studies have suggested that obesity might hinder lactation performance by causing prolactin resistance [16]. Obesity is a significant risk factor for insulin resistance and impaired insulin secretion; insulin is now believed to have a direct role in lactation,

including secretory differentiation, secretory activation, and mature milk production [17].

In the present study, parity showed a significant association with Lactogenesis II ($p = 0.045$). Multiparous mothers had a higher incidence of early lactogenesis (76.3%) compared to primiparous mothers (70.8%), whereas delayed lactogenesis were observed in 29.2% of primiparas vs. 23.7% in multiparas in our study and similarly, Mohammed et al. [12] reported delayed onset in 37.7% of primiparas vs. 19.3% in multiparas ($p < 0.01$), suggesting that previous childbirth experience may favor earlier lactogenesis. However, in Salahudeen et al. [11] study, although primiparas had a higher prevalence of delay (16%) compared to multiparas (9%), this was not statistically significant. First-time mothers (primiparous) may experience DOL more frequently than multiparous women, possibly due to less efficient mammary tissue priming or less effective suckling stimulation [18].

In our study, receiving breastfeeding counselling did not significantly impact the onset of Lactogenesis II ($p = 0.604$). Interestingly, 74.5% of mothers who did not receive counselling exhibited early Lactogenesis II, compared to 60% of those who did. However, due to the very small counselling group ($n = 5$), the result lacks statistical power. Professional guidance and maternal education on effective breastfeeding techniques are consistently linked to earlier onset and sustaining of lactogenesis [19]. According to Peng et al [13], receiving breastfeeding guidance was a protective factor against DOL.

In the current study, maternal comorbidities were not significantly associated with Lactogenesis II ($p = 0.908$) probably due to less sample size. The prevalence was nearly the same between mothers with comorbidities (75%) and those without (73.6%). At the same time, in Salahudeen et al. (11) study, hemoglobin <12 g/dL was significantly associated with delayed lactogenesis ($p = 0.01$), pregnancy induced hypertension (PIH), gestational diabetes mellitus (GDM), and hypothyroidism were all significantly associated with delay (all $p < 0.01$). Gestational diabetes mellitus [20] and hypertension [21] are metabolic disorders which are linked with DOL, implicating underlying hormonal and vascular changes that may impair mammary gland function. Poorly regulated thyroid function during pregnancy showed an association with delayed lactogenesis II, highlighting the importance of overall endocrine health [22].

Study by Peng et al [13] showed gestational diabetes mellitus (GDM), hypertensive disorders of pregnancy (HDP), and thyroid diseases during pregnancy are linked to delayed onset of lactation (DOL). This agrees with DeBortoli et al.'s systematic review [25], which also highlighted GDM as a risk factor for DOL. The possible reason could be due to insulin resistance and/or issues with beta cell insulin release that lead to GDM, with insulin resistance being the main cause [26]. This resistance might also affect the process of lactation [27]. The balance between insulin and glucose, along with levels of a hormone called adiponectin, might play a role in initiation of lactation [28] [48] [49] [50].

Because obesity is associated with insulin resistance, GDM, obesity, and insulin resistance might all be connected to DOL in some way [29]. Hypertensive disorders of pregnancy can affect both when and how long breastfeeding lasts [30], and treatments like diuretics might lower milk production [31]. HDP could also cause problems with the placenta and reduce the amount of prolactin, a hormone that helps with lactation [32]. Pre-eclampsia might lead to issues with blood vessel function, causing low levels of albumin in the blood [33], and low albumin levels could indicate poor nutrition, which may contribute to DOL[34]. Animal studies show that hypothyroidism can stop the breast from making and releasing milk properly, leading to lactation problems in women with thyroid issues during pregnancy [35]. On the other hand, hyperthyroidism might interfere with the release of oxytocin, which is needed for milk let-down, leading to milk buildup, damage to milk-producing cells, and lactation issues [36].

In the present study, mode of delivery significantly influenced Lactogenesis II ($p = 0.021$). Mothers who had normal vaginal deliveries had the highest prevalence (90.2%), followed by assisted vaginal deliveries (80%), elective cesarean sections (48%) and emergency cesarean sections (46.6%). Similar to our study, in the Mohammed et al. [12] study, 48.7% of cesarean deliveries had delayed lactogenesis vs. 28.3% in vaginal deliveries ($p < 0.01$) and in Salahudeen et al. [11] study, normal delivery had lower delay (6.5%) compared to cesarean (18.5%) ($p = 0.002$). Vaginal delivery is generally associated with a more rapid onset of lactogenesis compared to cesarean section. This difference is attributed to several factors, including hormonal changes, maternal stress, and the timing of the first breastfeeding session. Women who undergo cesarean sections may experience a delay in the onset of lactogenesis due to factors such as postoperative pain, reduced mobility, and delayed skin-to-skin contact with the newborn. Additionally, the stress response associated with surgical delivery can interfere with the hormonal cascade necessary for milk production. However, it is important to note that with proper support and early initiation of breastfeeding, women who have cesarean deliveries can still successfully establish

lactation [23,24]. Peng et al [13] showed that caesarean delivery and unscheduled caesarean delivery were also risk factors for DOL.

In our study, birth weight showed no significant association with Lactogenesis II (p = 0.826). Infants weighing between 2.5–4.0 kg had a slightly higher incidence of early lactogenesis (74.2%) compared to those under 2.5 kg (71.4%), whereas Salahudeen et al. [11] study observed that lower birth weight (<2.5 kg) was significantly associated with delayed lactogenesis (13 out of 27 LBW infants experienced delay). In the current study, no significant difference was observed in the onset of Lactogenesis II based on weight-for-gestational-age classification (p = 0.666). Both SGA (74.2%) and AGA (75%) infants showed similar rates, while LGA infants had a lower prevalence (50%). According to Peng et al [13] birth weight < 2.5KG and > 4 kg were also risk factors for DOL.

In the present study, timing of initiation of breastfeeding was not significantly associated with Lactogenesis II (p = 0.235). However, Mohammed et al. [12] found a direct, statistically significant correlation (p < 0.001) between delayed initiation and delayed lactogenesis. In our study, feeding colostrum did not significantly influence Lactogenesis II (p = 0.834). Mothers who fed colostrum showed a prevalence of 73.5%, nearly identical to the overall rate. In the current study, the pattern of breastfeeding (on-demand vs timed) did not significantly impact Lactogenesis II (p = 0.646). Slightly more mothers practicing on-demand feeding (77.1%) had Lactogenesis II compared to those using timed feeds (72.2%). Early initiation of breastfeeding within the first hour after birth stimulates hormone production and promotes milk production. Frequent feeding every 2-3 hours helps establish milk supply and triggers the let-down reflex.

In our study, breastfeeding problems were not significantly associated with onset of Lactogenesis II (p = 0.248). However, all mothers with issues such as wrong technique or other breast-related concerns exhibited early Lactogenesis II by proper and timely counselling, constant and adequate support, while those with sore or retracted nipples showed lower rates on onset. Most mothers with no breastfeeding issues had a prevalence of 72.1%. In concordance to our study findings, in the study by Mohammed et al. [12], several risk factors were identified as contributors to delayed Lactogenesis II. The most prevalent among these was incorrect breastfeeding technique, observed in 32.7% of the delayed cases, followed closely by cesarean delivery at 29.5%. Other contributing factors included the use of pacifiers (9.8%), inverted nipples (8.1%), supplemental feeding (8.1%), and prelacteal feeds (6.5%).

In our study, analysis of continuous variables showed significant differences. Babies of Mothers with delayed Lactogenesis II had fewer average feeds on Day 1 (6.5 vs. 9.2, p = 0.014) and Day 2 (9.8 vs. 12.1, p = 0.008), and significantly more infant weight loss on Day 4 (4.67% vs. 7.47%, p = 0.001).

Similar to our study, in the study conducted by Mohammed et al. [12], neonatal weight loss and output parameters emerged as significant clinical indicators of delayed Lactogenesis II. By Day 4, infants in the delayed lactogenesis group experienced a mean weight loss of 9.3%, significantly higher than the 5.4% observed in the early lactation group (p = 0.001). Additionally, the mean number of urine voids was markedly lower in the delayed group (2.6) compared to the early group (5.7), though there was no statistical significance. Similarly, the average number of stools passed by Day 4 was 2.6 in the delayed group versus 4.4 in the early group. In another study by Salahudeen et al. [11], weight loss (day 4) were observed in 9.3% (delayed) vs. 5.4% (early) with a statistical significance (p < 0.001). These findings underscore the value of monitoring infant weight and elimination patterns as accessible, non-invasive indicators of adequate milk transfer and timely onset of lactogenesis.

The World Health Organization recommends that all pregnant women and those with newborns get guidance on breastfeeding to improve their skills, confidence, and ability to breastfeed [42]. A review by McFadden et al. [43] also showed that breastfeeding support helps improve breastfeeding outcomes. From our study we concluded that getting support with breastfeeding can help protect against DOL. Our research shows that how often a mother breastfeeds is important for preventing DOL. When a baby suckles, it helps the pituitary gland release oxytocin, which helps the uterus return to its normal size [44]. Also, breastfeeding often and making sure the breasts are emptied well can help increase milk supply [45,46]. These findings suggest that when a mother is separated from her baby and the baby can't suck, it's important to start expressing breast milk manually or using a pump. This can help stimulate the nipple and promote oxytocin release [46], reducing the chance of DOL.

Breastfeeding counseling plays a crucial role in supporting mothers and promoting the health of both infants and mothers. Counseling can educate mothers on the advantages, such as enhanced immunity for the baby, reduced risks of certain diseases, and emotional bonding. Many mothers face difficulties when starting to breastfeed, such as pain, engorgement, or concerns about milk supply. Counseling helps address these challenges, offering practical solutions and reassurance. It also provides

evidence-based information, allowing mothers to make informed choices about breastfeeding.

This includes understanding how breastfeeding can be integrated into their lifestyles. The emotional and psychological aspects of breastfeeding can be significant. Counseling offers support and encouragement, helping mothers navigate their feelings and anxieties associated with breastfeeding. Many new mothers feel uncertain about their breastfeeding abilities. Personalized counseling can boost their confidence, helping them trust their bodies and instincts. Effective breastfeeding counseling takes into account cultural beliefs and practices. It ensures that the advice given aligns with the values and traditions of the mother's community. Establishing a successful breastfeeding relationship can encourage a positive outlook on infant nutrition and health that lasts beyond the breastfeeding period, promoting overall well-being.

Conclusion

Our study highlighted various maternal, neonatal, and perinatal factors associated with the onset of Lactogenesis II. The overall prevalence of Lactogenesis II was found to be 73.8%. Statistically significant associations were observed with maternal BMI ($p = 0.001$), parity ($p = 0.045$), mode of delivery ($p = 0.021$), and infant weight loss on day 4 ($p = 0.001$), indicating that lower BMI, multiparity, vaginal delivery, increased frequency of feeds are favorable for the timely onset of Lactogenesis II. Additionally, a lower average number of feeds on Days 1 and 2 and higher BMI was significantly associated with the presence of delayed onset of Lactogenesis II. Although other variables such as gender of the newborn, maternal age, education, socioeconomic status, gestational age, breastfeeding counseling, birth weight, breastfeeding initiation, and feeding practices showed no significant associations, descriptive trends suggested potential clinical relevance worth exploring further. These findings emphasize the multifactorial nature of Lactogenesis II and highlight the importance of optimizing maternal health and delivery practices to support early lactation. Breastfeeding counseling is vital in helping mothers overcome obstacles, understand the benefits of breastfeeding, and feel supported in their journey. This comprehensive support ultimately leads to healthier families and communities.

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