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ORIGINAL RESEARCH ARTICLE

Change in prevalence of major levator ani muscle defects from 6 weeks to 1 year postpartum, and maternal and obstetric risk factors: A longitudinal ultrasound study

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Abstract

Introduction: The present study aimed first to investigate the change in prevalence of major levator ani muscle (LAM) defects, also called avulsions, from 6 weeks to 1 year postpartum, and second to assess maternal and obstetric risk factors for having persistent major LAM defects/avulsions at 1 year postpartum.

Material and methods: This is a secondary analysis of data from a prospective cohort study including 300 nulliparous women at 17-19 weeks of gestation. Major LAM defects were diagnosed at 6 weeks and 1 year postpartum using transperineal ultrasonography. We defined persistent major LAM defects as a defect diagnosed both at 6 weeks and 1 year postpartum. Maternal and obstetric data were obtained from the hospital's electronic birth records. Pelvic floor muscle function was measured vaginally by manometer at 21 weeks of gestation. The main outcome measurement was change in prevalence of major LAM defects. Maternal and obstetric risk factors for having persistent major LAM defect were also assessed.

Results: Prevalence of major LAM defects was 19.4% at 6 weeks and 10.4% at 1 year postpartum. No new major LAM defects were diagnosed at 1 year postpartum. Persisting major LAM defects were associated with longer second stage of labor (median 74.5 minutes vs median 48.0 minutes, $P = .012$) and higher neonatal birthweight (mean difference of 232.3 g, 95% confidence interval [CI] 21.5-443.1). Vacuum delivery was independently associated with persistent major LAM defects, adjusted OR 3.0 (95% CI 1.0-9.0).

Conclusions: There was a 50% reduction of sonographically diagnosed major LAM defects from 6 weeks to 1 year postpartum. This finding suggests that assessment of the major LAM 6 weeks postpartum may be too early to diagnose defects/avulsions. Long second stage of labor, high neonatal birthweight and vacuum delivery were associated with persistent major LAM defects/avulsions.

Abbreviations: aOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; cOR, crude odds ratio; LAM, levator ani muscle; PFM, pelvic floor muscle; RCT, randomized controlled trial.

KEYWORDS
levator ani muscle avulsion, levator ani muscle defect, maternal and obstetric risk factors, pelvic floor disorder, transperineal ultrasound, vacuum extraction delivery

1 | INTRODUCTION

During vaginal delivery the most medial portion of the levator ani muscle (LAM) has to stretch up to three times its resting length,[1] which may lead to tearing of its muscle fibers from its bony insertion. Such injuries, known as major LAM defects or avulsions, have been found to be a risk factor for pelvic organ prolapse.[2,3] The prevalence of major LAM defects diagnosed by transperineal ultrasonography range between 13% and 36% in primiparous women within the first 6 months.[4-6] The highest prevalence is reported shortly after delivery.[4,6] As the prevalence seems to decrease during the first postpartum year, the process of natural healing is discussed.[7-10] Furthermore, a possible false-positive diagnosis of major defects, particularly when the diagnosis takes place shortly after delivery, may impact the prevalence numbers.[7,8,11] To our knowledge, only a few studies have investigated the change in prevalence of major LAM defects up to 1 year after delivery.[7,12] These studies have limitations in terms of high losses to follow up.

Several maternal and obstetric risk factors for LAM defects have been described.[13-17] Besides maternal age and pre-pregnancy body mass index (BMI), the woman's pelvic floor anatomy and antenatal pelvic floor muscle (PFM) function may influence the risk for major LAM defects.[16,18-20] Prolonged second stage of labor, forceps delivery and neonatal birthweight appears to be the foremost intrapartum risk factors for acquiring major LAM defects.[5,15,21] The literature concerning the impact of vacuum delivery on major LAM defects is inconsistent.[21] The present study aimed first to investigate the change in prevalence of major LAM defects from 6 weeks to 1 year postpartum, and second to assess maternal and obstetric risk factors for having persistent major LAM defects at 1 year postpartum.

Key message

Long second stage of labor and high birthweight were associated with persistent major LAM defects/avulsions. Vacuum delivery was independently associated with persistent major LAM defects/avulsions.

2 | MATERIAL AND METHODS

This is a secondary analysis of data from a prospective cohort study conducted at Akershus University Hospital, Lørenskog, Norway, from December 2009 to 2012. A total of 300 nulliparous women were recruited from the antenatal care clinic at 17-19 weeks of gestation. The women were followed longitudinally until 1 year postpartum, with the overall aim to study anatomical and functional changes of the pelvic floor during this period.[8] Inclusion criteria were nulliparity, having a singleton pregnancy, and the ability of the women to be able to speak and understand a Scandinavian language. Exclusion criteria were previous pregnancy of more than 16 weeks of gestation and serious maternal pathology which could interfere with the ability to cohere to the comprehensive follow-up regimen during pregnancy and within the first postpartum year, or fetal pathology. Ongoing exclusion criteria were stillbirth, missing delivery data and premature delivery <37 weeks of gestation in the ongoing pregnancy. For the purpose of the current study, we omitted women undergoing cesarean section. Figure 1 is a flowchart of the study participants with an overview of excluded women and women lost to follow up. Of the 300 women recruited to the cohort study, 139 participated in a randomized controlled trial (RCT).[22] This RCT aimed to explore the effect of PFM training between 6 weeks and 6 months postpartum on pelvic floor anatomy and function.

At 6 weeks and 1 year postpartum, study participants underwent a three- and four-dimensional transperineal ultrasound examination using the GE Kretz Voluson E8 (GE Medical Systems) with 4-8 MHz curved array 3D/4D ultrasound transducer (RAB4-8l/obstetric). The examination was performed by two investigators using a previously described methodology.[19]

Diagnosis of major LAM defects was based on offline assessment of sonographic volumes at maximum pelvic floor contraction using tomographic ultrasound imaging in the axial plane. According to Dietz et al,[23] the minimal criteria for diagnosing a major LAM defect is the presence of abnormal muscle insertion at the level of minimal hiatal dimensions and at 2.5 and 5.0 mm cranial to this reference plane. The two trained investigators analyzed ultrasound volumes in accordance with the elaborated methodology mentioned above. Inter-rater agreement between them was good to excellent ($\kappa > 0.63$).[24] Three women were not able to contract the PFM correctly at 6 weeks postpartum, and one woman was still not able to contract correctly at 1 year postpartum. In case of inability to contract, ultrasound volumes at rest were analyzed instead.

We defined three groups based on sonographic findings:

- a "no defect group" having no observed major LAM defect at 6 weeks or 1 year postpartum,
- a "non-persistent defect group" having a major LAM defect diagnosed at 6 weeks, but no fulfilment of the diagnostic criteria 1 year postpartum,
- a "persistent defect group", having a major LAM defect diagnosed both 6 weeks and 1 year postpartum (Figure 2).

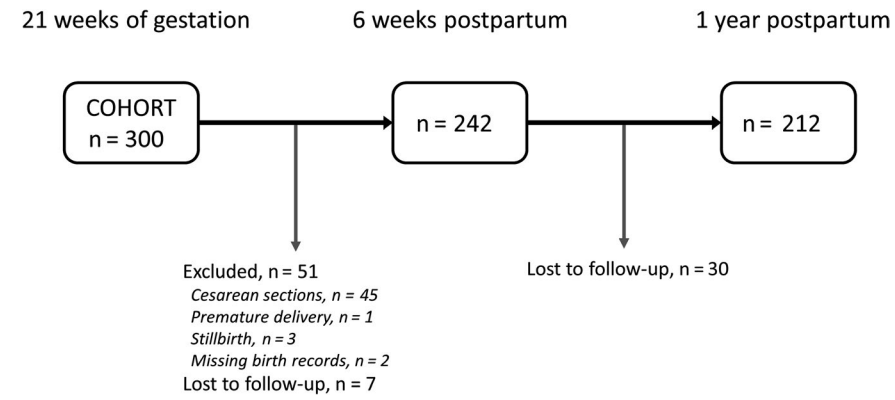

FIGURE 1 Flowchart of study participants

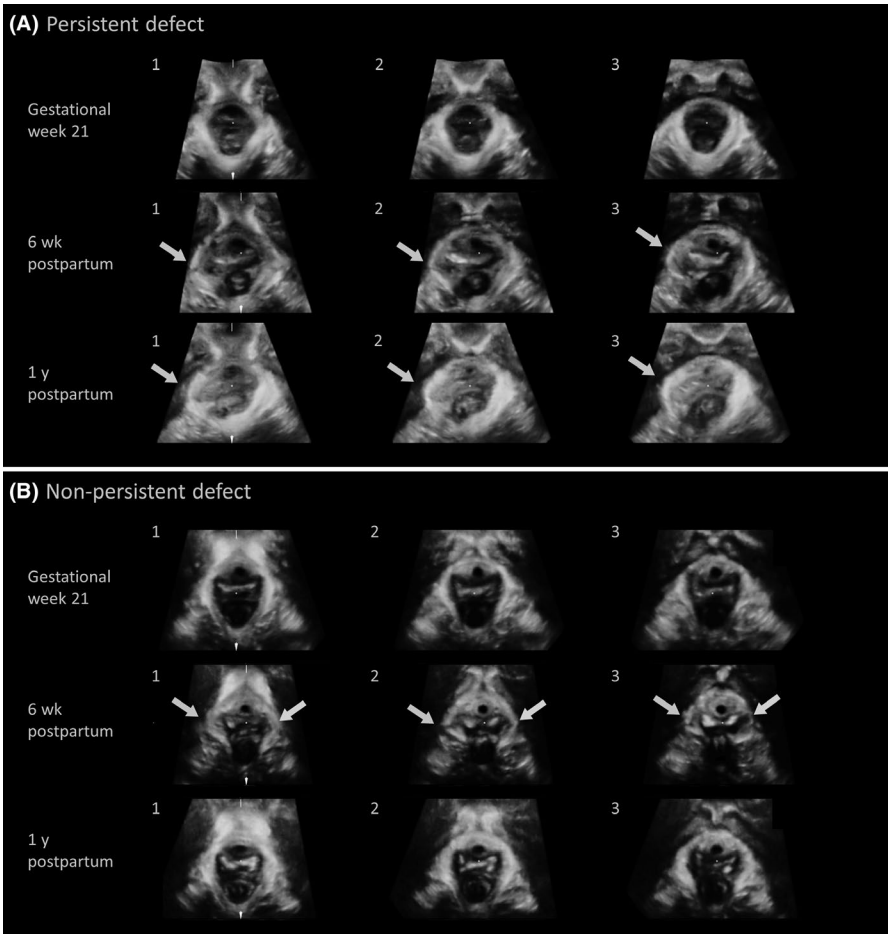

FIGURE 2 Tomographic ultrasound images of the three central slices (1-3) taken at the level of minimal hiatus dimensions. (A) Images from one woman with a persistent major levator ani muscle (LAM) defect: no major LAM defect at gestational week 22 (top row), white arrows showing a right-sided major LAM defect at both 6 weeks postpartum (middle row) and 1 year postpartum (bottom row). (B) Images from one woman with a non-persistent major LAM defect: no major LAM defect at gestational week 21 (top row), white arrows showing bilateral major LAM defect at 6 weeks postpartum (middle row). No major LAM defect at 1 year postpartum (bottom row)

PFM function measurements were obtained at 21 weeks of gestation. The measurements included vaginal resting pressure, PFM strength and endurance. Measurements were undertaken with an air-filled vaginal balloon connected to a high precision pressure transducer (Camtech AS, Sandvika, Norway).[25] The procedure for measuring PFM function has been thoroughly described in a previous paper from our study group.[22]

Maternal and obstetric data were obtained from the hospital's electronic birth records.

Maternal variables included age (years) and pre-pregnancy BMI (kg/m^2). Obstetric variables included were epidural anesthesia, oxytocin augmentation, episiotomy (lateral incision to the left side), the duration of the second stage of labor defined as the time interval (in minutes) between complete cervical dilation and delivery of the child, neonatal birthweight (in grams), neonatal head circumference (in centimeters) and delivery mode.

Vaginal delivery mode such as non-instrumental, vacuum or forceps delivery was registered. Vacuum and forceps delivery were attempted and performed in consensus with current obstetric guidelines.[26] The investigators performing the ultrasonography were blinded to previous ultrasound assessments and obstetric history.

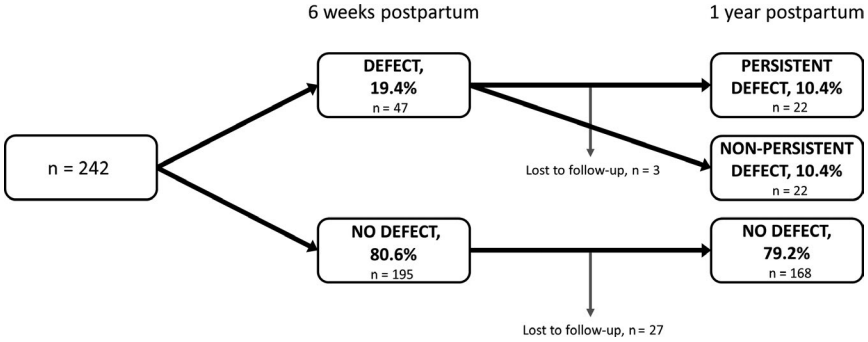

FIGURE 3 Prevalence of major levator ani muscle (LAM) defects at 6 weeks and 1 year postpartum in 242 women with vaginal delivery. A non-persistent defect was a major LAM defect diagnosed at 6 weeks postpartum, but was no longer observed at 1 year postpartum. A persistent defect was a major LAM defect diagnosed at both 6 weeks and 1 year postpartum

TABLE 1 Demographic and obstetric data of the study population. The three study groups were defined based on sonographic findings at 6 weeks and 1 year postpartum. (1) "No defect group" comprising women having no observed major levator ani muscle (LAM) defect at 6 weeks or 1 year postpartum. (2) "Non-persistent defect group" comprising women having major LAM defect diagnosis at 6 weeks, but no observed major LAM defect at 1 year postpartum. (3) "Persistent defect group" comprising women having major LAM defect diagnosed both 6 weeks and 1 year postpartum

	No defect group, n = 168	Non-persistent defect group, n = 22	Persistent defect group, n = 22	*P*
Maternal age (years)	29.5 (SD 4.1)	29.8 (SD 4.0)	29.6 (SD 4.0)	NS
Pre-pregnancy BMI (kg/m^2)	23.9 (SD 4.0)	24.5 (SD 3.1)	22.4 (SD 4.1)	NS
Length of second stage (min) (n = 162)	Median 48.0 (9-234)	Median 88.0 (15-309)	Median 74.5 (16-180)	.010[a] .012[b]
Neonatal birthweight (g)	3478.6 (SD 457.0)	3545.7 (SD 420.5)	3710.9 (SD 573.1)	.031[b]
Neonatal head circumference (cm) (n = 167)	35.0 (SD 1.6)	35.4 (SD 1.7)	35.1 (SD 1.6)	NS
Total gestational length (days) (n = 164)	280.8 (SD 10.9)	282.0 (SD 9.2)	283.4 (SD 9.5)	NS
PFM strength, week 21 (cmH_2O)	36.7 (SD 19.7)	34.8 (SD 16.1)	32.9 (SD 14.8)	NS
Vaginal resting pressure, week 21[c] (cmH_2O)	42.6 (SD 9.1)	43.9 (SD 9.0)	42.7 (SD 12.8)	NS
PFM endurance, week 21 (cmH_2O/sec)	253.7 (SD 141.3)	249.5 (SD 142.9)	225.7 (SD 113.3)	NS
Epidural (yes)	65 (38.7%)	9 (40.9%)	6 (27.3%)	NS
Oxytocin augmentation (yes)	20 (11.9%)	2 (9.1%)	4 (18.2%)	NS
Episiotomy (yes)	56 (33.3%)	3 (13.6%)	8 (36.4%)	NS
Mode of delivery[d]				
Non-instrumental, vaginal (NVD)	143 (85.1%)	13 (59.1%)	12 (54.5%)	N/A
Vacuum (VD)	23 (13.7%)	9 (40.9%)	8 (36.4%)	.002[a] .003[b]
Vacuum + forceps or forceps only (FD)	2 (1.2%)	0 (0.0%)	2 (9.1%)	N/A

Notes: Values are given as means with standard deviations (SD), and as medians with ranges for not normally distributed data or as frequencies and percentages (%). $P < .05$.

Abbreviations: N/A, not applicable; NS, not significant.

[a]Statistically significant difference between the "no defect group" and the "non-persisting defect group".

[b]Statistically significant difference between the "no defect group" and the "persisting defect group".

[c]Equal variances not assumed.

[d]Comparing non-instrumental delivery and vacuum delivery.

2.1 | Statistical analyses

Statistical analysis was performed using IBM SPSS Statistics 25.0. To assess differences between study groups, the independent sample Student's *t* test and Chi-square test were performed. The Mann-Whitney *U* test was performed when continuous data were not normally distributed. Univariate and multivariate logistic regression analysis was performed. In cases of significant results in the

TABLE 2 Univariate and multivariate logistic regression to calculate crude odds ratio (cOR) and adjusted odds ratio (aOR) with 95% confidence interval for having a persistent or non-persistent major levator ani muscle (LAM) defect. Adjusted odds ratios are adjusted for vacuum delivery, length of second stage of labor (in minutes) and neonatal birthweight (in grams). Three study groups were defined based on sonographic findings at 6 weeks and 1 year postpartum. (1) "No defect group" comprising women having no observed major LAM defect at 6 weeks or 1 year postpartum. (2) "Non-persistent defect group" comprising women having major LAM defect diagnosis at 6 weeks, but no observed major LAM defect at 1 year postpartum. (3) "Persistent defect group" comprising women having major LAM defect diagnosed both 6 weeks and 1 year postpartum

	No defect group vs persistent defect group				No defect group vs non-persistent defect group			
	cOR (95% CI)	*P*	aOR (95% CI)	*P*	cOR (95% CI)	*P*	aOR (95% CI)	*P*
Vacuum	4.145 (1.529-11.234)	.005	3.035 (1.019-9.036)	.046	4.304 (1.653-11.210)	.003	3.160 (1.097-9.104)	.033
Length of second stage (min)	1.009 (1.000-1.018)	.058	1.004 (0.993-1.014)	.477	1.011 (1.003-1.019)	.007	1.008 (.999-1.018)	.075
Neonatal birthweight (g)	1.001 (1.000-1.002)	.017	1.001 (1.000-1.002)	.082	1.000 (0.999-1.001)	.499	1.000 (.999-1.001)	.749

univariate analysis, a multivariate logistic regression analysis was performed to control the findings for possible obstetric confounding factors. Crude (cOR) and adjusted odd ratio (aOR) was reported with 95% confidence intervals (CI). *P* values < .05 were considered significant. A power calculation was not performed.

2.2 | Ethical approval

Ethical approval for the cohort study was granted by the Regional Medical Ethics Committee, Norway (REC South East D 2009/170) and the Norwegian Social Science Data Service (17-055). Informed written consent was obtained from all participating women.

3 | RESULTS

The study sample consisted of 212 primiparous women (Figure 1). Mean maternal age at delivery of the 212 study participants was 29.5 years (SD 4.1) and mean pre-pregnancy BMI was 23.8 kg/m^2 (SD 3.9). Mean gestational age at delivery was 281.2 days (SD 10.6). Mean follow-up time at the clinical assessments postpartum was 6.1 weeks (SD 1.0) and 51.6 weeks (SD 3.1). Of 212 women 168 (79.2%) had a non-instrumental vaginal delivery. The majority of instrumental vaginal deliveries were performed with vacuum (18.9%, 40/212). Two women underwent forceps delivery and two women underwent forceps delivery after a vacuum delivery attempt. The background population (n = 2547) of pregnant nulliparous women scheduled for delivery at Akershus University Hospital during the inclusion period did not differ in maternal age or pre-pregnancy BMI when compared with the study population (n = 300). Our vacuum delivery rate is comparable to the background population of our hospital (22.3%) and to the Norwegian population (22.7%) of primiparous women.[27] However, neonatal birthweight were significantly higher in the study population than in the background population (mean difference of 74.4 g, 95% CI 4.9-143.8). Women lost to follow up (n = 37) were younger (mean difference 1.7, 95% CI 0.3-3.2) and had weaker PFM strength measurements (mean difference 6.7, 95% CI 2.5-11.0) and PFM endurance measurements (mean difference 51.4, 95% CI 17.6-85.3) at gestational week 21 compared with the remaining study participants (n = 212).

Prevalence of major LAM defects in the study group was 19.4% (47/242) at 6 weeks postpartum and 10.4% (22/212) at 1 year postpartum (Figure 3). At 1 year postpartum, 10.4% (22/212) of the women diagnosed with a major LAM defect at 6 weeks postpartum no longer fulfilled the diagnostic criteria for a major LAM defect. No new major LAM defects were diagnosed at 1 year postpartum.

Table 1 shows the distribution of maternal and obstetric factors in the three study groups. We found that the "persistent defect group" had a significantly longer second stage of labor (median 74.5 vs 48.0 minutes, *P* = .012) and a higher neonatal birthweight (mean difference of 232.3 g, 95% CI 21.5-443.1) when compared with the "no defect group". The same was true for the "non-persistent defect group" in relation to length of second stage of delivery (median 88.0 vs 48.0 minutes, *P* = .010) when compared with the "no defect group".

Table 2 shows the results from our univariate and multivariate logistic regression analysis. The cOR for having a persistent major LAM defect after vacuum delivery was cOR 4.1 (95% CI 1.5-11.2) and the cOR for having a non-persistent defect after vacuum delivery was cOR 4.3 (95% CI (1.7-11.2). Vacuum delivery remained an independent risk factor for a persistent major LAM defect when adjusting for neonatal birthweight and length of second stage (aOR 3.0, 95% CI 1.0-9.0). The same is true for the "non-persistent defect group", where vacuum delivery remained a significant risk factor when adjusting for the same confounding factors (aOR 3.2, 95% CI 1.1-9.1). In relation to maternal age, pre-pregnancy BMI, total days of gestational length, epidural analgesia, oxytocin augmentation or episiotomy, there was no observed statistical significant difference between any of the study groups.

We found the highest PFM function measurements (strength and endurance) at 21 weeks of gestation in the "no defect group", and

the lowest measurements in the "persistent defect group". However, differences between study groups were not statistically significant.

4 | DISCUSSION

In the present study, we found that half of the women showing a major LAM defect at 6 weeks postpartum did not fulfil the diagnostic criteria for a major LAM defect 1 year postpartum. No new defects were diagnosed at 1 year postpartum. Previously described obstetric risk factors for a major LAM defect diagnosed within 6 months postpartum, such as a long second stage of labor and high neonatal birthweight, were confirmed to be associated with persistent major LAM defect 1 year postpartum. Vacuum delivery was an independent risk factor for having persistent major LAM defects.

A strength of the present study is the longitudinal, prospective follow up from 6 weeks to 1 year postpartum. The study includes a relatively large sample size and only a few participants were lost to follow up. Another strength of our study is the minimal variation in time of the ultrasound examinations. Diagnosis of major LAM defects was assessed according to a reliable and valid sonographic method.[23] Two independent investigators diagnosed major LAM defects with a high interrater reliability.[28] Throughout the study period they were both blinded to each other's findings and conclusions, and also blinded to previous ultrasound assessments and obstetric history when performing the analysis. Furthermore, trained physiotherapists obtained measurements of PFM function with validated methods of assessments.[29]

A study limitation is the lack of a priori power calculation, which was not performed for the research questions of the present study. This is a single-center study. There are differences in obstetrical practice between both countries and individual hospitals reflected in different rates of instrumental vaginal deliveries and cesarean sections reported. This may have influenced the results of our study. One inclusion criterion was the ability to speak and understand a Scandinavian language; this may have introduced a selection bias in our study. Our study participants mainly consisted of caucasian women and therefore generalization to other ethnic groups may be limited. Some of the women participated in an RCT with PFM training as intervention,[22] and being exposed to training may interfere with the prevalence rate, as training is believed to speed up tissue healing.[30] However, the RCT showed no statistically significant effect of PFM training on major LAM defects.[31]

The observed reduction of 50.0% of major LAM defects within the first postpartum year lies within findings of other study groups. Van Delft et al[7] found that 62% of major LAM defects were no longer evident at 1 year postpartum. Chan et al[12] found a defect reduction of 20.8% from 8 weeks to 1 year postpartum in Chinese primiparous women.[12] Valsky et al[9] found 19% reduction of major LAM defect in a cohort of primiparous women examined 72 hours postpartum and 3-21 months postpartum. The decrease in defect prevalence shown by others and by the present authors could very likely be explained by diagnosing false-positives early postpartum.[4-6] Our major LAM defect prevalence is in line with the existing literature. Interpreting ultrasound images within a short time interval from birth may be difficult due to challenging tissue discrimination and/or poor image quality. Another explanation could be muscle remodeling, where spontaneous recovery is one aspect of this process. Van Delft et al[7] proposed a spontaneous "healing" as an explanation for their observed reduction of LAM avulsion. However, it is hard to imagine how a muscle which is completely torn from its insertion could reattach spontaneously on to the pubic bone.[10] In some cases, the LAM muscle may not be completely detached from its insertion. In those cases, scar tissue between the muscle and the pubic bone might appear as an anatomical improvement visualized sonographically.[10] It is furthermore unclear whether a seemingly recovered LAM predisposes to pelvic floor dysfunction later in life. In accordance with Van Delft et al,[7] we found no new major LAM defects diagnosed after the first postpartum examination and therefore we surmise that it may be safe to exclude a major LAM defect diagnosis early postpartum.

We found that a long second stage of labor and high neonatal birthweight were associated with persistent major LAM defects. There seems to be a successive reduction in neonatal birthweight from the "persistent defect group" (highest mean birthweight), through the "non-persistent defect group" and to the "no defect group" (lowest mean birthweight). Several authors found an association between high neonatal birthweight and large neonatal head circumference with major LAM defects.[15,32] The same is true in our study population for high neonatal birthweight, but we did not find any association in relation to neonatal head circumference.

In our study population, almost all women with an instrumental vaginal delivery underwent a vacuum delivery. In this study, vacuum delivery was an independent statistically significant risk factor for a persistent major LAM defect even after controlling for neonatal birthweight and length of second stage of labor. However, the result is borderline significant and has to be interpreted with caution owing to the small sample size. The association between vacuum delivery and the risk of LAM defects is discussed in the literature. In a recently published meta-analysis by Friedman et al,[21] with over 5000 deliveries, the association between vacuum delivery and the risk of LAM avulsion did not reach statistical significance. However, those authors observed a trend towards vacuum delivery as a risk factor. Interestingly, we found the highest median length of second stage of labor in the "non-persistent defect group". Furthermore, vacuum delivery also remained a risk factor in the "non-persistent defect group" even when controlling for significant univariate variables. We interpret the finding of non-persistent defects as a sign of unspecific tissue aberration recognized by ultrasonography in this subgroup of women. For the time being, the clinical relevance of this ultrasonographic finding is questionable.

Although none of the differences in PFM function measurements between the study groups reached statistical significance, we observe a tendency for women in the "no defect group" to have higher PFM strength and endurance measurements compared with women in the other defect groups. The successive reduction

in PFM strength is consistent with a previous finding of our group where less shortening of the LAM during contraction assessed by ultrasonography at 37 weeks of gestation was independently associated with major LAM defects at 6 weeks postpartum.[19] Van Delft et al[33] found that PFM strength measured with digital palpation at 36 weeks of gestation was significantly lower in women with major LAM avulsions diagnosed at 3 months postpartum compared with women without avulsion. These findings suggest that antenatal PFM training might improve the ability of LAM to facilitate delivery and to resist major LAM defects.

5 | CONCLUSIONS

There was a 50% reduction of sonographically diagnosed major LAM defects from 6 weeks to 1 year postpartum. This finding suggests that assessment of the major LAM 6 weeks postpartum may be too early to diagnose defects/avulsions. Long second stage of labor, high neonatal birthweight and vacuum delivery were associated with persistent major LAM defects/avulsions.

CONFLICT OF INTEREST

The authors have stated explicitly that there are no conflicts of interest in connection with this article.

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