**The natural history of levator ani muscle avulsion four years following childbirth**

Short title: Natural history of LAM avulsion

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**Abstract**

*Objectives:* The primary aim was to prospectively study the natural history of levator ani muscle (LAM) avulsion four years following first delivery and correlate to signs and symptoms of pelvic floor dysfunction (PFD). The secondary aim was to investigate the effect of a second vaginal delivery on the incidence of LAM avulsion and PFD.

*Methods:* Nulliparae at 36 weeks of gestation, three months, one year and four years postpartum were assessedat all visits for signs and symptoms of PFD. Transperineal ultrasound was performed to assess LAM integrity and hiatal biometry. Differences in signs and symptoms of PFD over time were evaluated using a linear mixed model in women with one and ≥2 deliveries.

*Results:* Of 269 nulliparae, 147 (55%) were examined 3.8 (0.4 SD) years after their first delivery. 74 (50%) had a subsequent delivery. Prevalence of LAM avulsion four years after first vaginal delivery was 13% with no difference between one or more vaginal deliveries. Women with intact LAM showed no change in signs and symptoms of PFD. In women with previous LAM avulsion, four years after one delivery 42% was no longer evident, however significant worsening in pelvic floor muscle strength, prolapse on clinical examination and hiatal area on ultrasound were found. After subsequent vaginal delivery LAM avulsion became more extensive in 44% and hiatal area increased.

*Conclusions:* The first vaginal delivery is at greatest risk for LAM avulsion to occur, with impact on PFD four years later. A second vaginal delivery could result in deterioration of LAM avulsion, but no new avulsions were found.

**Contribution:**

**What are the novel findings of this work?**

The first vaginal delivery is at greatest risk for LAM avulsion to occur, with an impact on PFD four years later. A second vaginal delivery could result in deterioration of LAM avulsion, but no new avulsions were found.

**What are the clinical implications of this work?**

Larger longer term prospective studies of primiparous and multiparous women are needed to establish whether a caesarean section would benefit women who have sustained a previous LAM avulsion. Attention needs to be focused on preventative strategies to minimise the risk of sustaining LAM avulsion.

**Introduction**

The levator ani muscle (LAM) is the supportive muscle of all pelvic organs. During a vaginal delivery this muscle has to stretch tremendously and can tear from its insertion to the anterior pubic rami. It has been shown that LAM avulsion reduces pelvic organ support, leading to pelvic organ prolapse (POP) and hiatal ballooning on ultrasound1-3. Furthermore, LAM avulsion could cause pelvic floor dysfunction (PFD) as shown in 36-50% of women attending a tertiary urogynaecology unit1,2. Women with LAM avulsion presenting with symptomatic POP requiring a surgical intervention are usually younger than women without LAM avulsion4, and they are at increased risk of POP recurrence after surgical repair5,6.

The incidence of LAM avulsion diagnosed on three dimensional transperineal ultrasound (TPUS) a few months after a first vaginal delivery is approximately 19-21%7,8. The association between LAM avulsion and signs and symptoms of PFD using validated clinical assessment tools and questionnaires has been assessed prospectively at three months and one year postpartum9,10. However, longer-term follow-up is required to establish the clinical relevance, including the impact of minor LAM avulsion, as it has been shown that 50-62% of LAM avulsions were no longer evident at one year follow-up10,11.

Cumulative parity has been shown to be a significant risk factor for pelvic organ prolapse in a prospective longitudinal study 12 years following childbirth12. The impact of a second vaginal delivery on LAM avulsion and PFD has been studied, however retrospectively or in a specific ethnic group13-17, and when prospectively with a maximum follow-up of 2.7 years after first delivery18. Moreover, difference in symptoms and signs of PFD before and after second delivery accounting for LAM avulsion has not been assessed robustly.

The primary aim was to prospectively study the natural history of LAM avulsion four years following first delivery and correlate to signs and symptoms of PFD. The secondary aim was to investigate the effect of a second vaginal delivery on the incidence of LAM avulsion and PFD, with the objective to assess whether women with pre-existing LAM avulsion are at increased risk of PFD following a second vaginal delivery.

**Methods**

In this prospective longitudinal study, 269 nulliparous women were recruited antenatally between January 2011 and May 2012 at Croydon University Hospital, Croydon, United Kingdom. This study is the four-year follow-up of the primary study with the aim to establish the prevalence of LAM avulsion during childbirth and to correlate these with pelvic floor symptoms and pelvic floor muscle strength7,9. Inclusion criteria were a singleton pregnancy, no previous pregnancies beyond 20 weeks of gestation, age ≥18 years, and being able to read and understand English. Women were examined at 36 weeks of pregnancy, three months, one year and four years postpartum. The study was approved by the National Research Ethics Service South West London Committee (REC10/H0806/87). All participants gave written informed consent.

Patient demographics and delivery details were recorded prospectively. The investigators were blinded to the obstetric exposure of the participants, but not to the time of assessment. All assessments were carried out according to the same protocol. Investigator training and observation by the principal investigator (RT) was conducted prior to the start of the study to minimise measurement and technique variability.

Validated questionnaires were used to evaluate urinary, bowel, sexual function and symptoms of pelvic organ prolapse (POP). Urinary incontinence (UI) was assessed using the International Consultation on Incontinence Questionnaire Short Form (ICIQ-SF); total score ranging between 0 and 2119. For anal incontinence (AI) the St Mark’s incontinence scoring system was used; total score ranging between 0 and 2420. Symptoms of POP and sexual dysfunction (SD) were evaluated using the International Consultation on Incontinence Questionnaire Vaginal Symptoms (ICIQ-VS) questionnaire: question 5 and 6 for POP symptoms (range 0-28) and question 11-13 for SD (range 0-36)21. All questionnaires included questions on frequency and bother, and higher scores indicated poorer outcomes.

Pelvic floor muscle strength (PFMS) was assessed by digital palpation, inserting the index-finger approximately 4 cm into the vagina. The six-point Modified Oxford Scale (MOS) was used to grade PFMS22. POP was assessed using the validated International Continence Society POP-Q staging method23. The 3D/4D TPUS was performed using the GE Voluson I system (4–8 MHz curved array volume transducer, acquisition angle 85°) with women in the supine position, knees semi-flexed and an empty bladder. Images were acquired at rest, maximum pelvic floor contraction and Valsalva manoeuvre. Offline analysis was carried out using 4D VIEW version 10.2.

The LAM attachment to the pubic bone was assessed in the plane of minimal hiatal dimensions using Tomographic Ultrasound Imaging at maximum contraction with 2.5 mm slide intervals as described previously24,25.In the three central slices,left and right side were scored separately and the final unilateral score ranged from 0 (no avulsion) to 3 (complete avulsion)7,9,10,24,25. A summed total score was assigned for both sides, and classified as no LAM avulsion (summed score 0), minor LAM avulsion (summed score 1–3) or major LAM avulsion (summed score 4–6, or unilateral score 3)7,9,10. The hiatal area was measured in the rendered axial plane at rest, squeeze and valsalva.

The four-year follow-up was performed by IvG: clinical data collection and TPUS. Hiatus measurements were performed by one investigator (IvG). Two independent investigators (IvG, KvD) reviewed the ultrasound images to assess LAM avulsion, blinded to clinical assessment and delivery details. Discrepancies were reviewed by a third blinded investigator (RT).

*Statistical analysis*

To assess the effect of LAM morphology on the development of signs and symptoms of PFD four years after the first delivery, only women who had one delivery (vaginal or caesarean) at baseline were selected. These women were separated into four groups; one group of women who had a caesarean section (group 1, control group), and women who delivered vaginally (normal or instrumental) were separated in three groups according to LAM morphology at four years; intact LAM at all visits (group 2), LAM avulsion no longer evident (intact at 4Y but previous LAM avulsion) (group 3) and persistent LAM avulsion (avulsion at 4Y and at any of the previous visits) (group 4).

To assess the effect of subsequent deliveries on the development of PFD, women with ≥2 deliveries at the four-year follow up visit were selected and subdivided in groups as described above. Women with a caesarean section after a vaginal delivery or a vaginal birth after caesarean section (VBAC) were excluded from statistical analysis to avoid heterogeneity of the groups; moreover, women with a VBAC had a vaginal delivery not at index, so development of PFD could be on a different time scale. Women >24 weeks of pregnancy at follow-up visits were excluded from analyses as ongoing pregnancy may influence outcome measures.

Pattern differences of signs and symptoms of PFD over time were evaluated using a linear mixed model in women with one delivery and ≥2 deliveries separately. A mixed model on prospectively gathered longitudinal data of clinical and ultrasound findings was used to minimise the effects of missing data. The likelihood-based estimation method underlying this statistical model can handle missing data if data are missing at random. Therefore, women who attended the four-year follow-up visit but missed either three months or one-year follow-up or both were included in the analyses. An unstructured covariance matrix was used to account for the repeated measures design of the study. All available data from the four visits for the four groups were used and outcome was modeled as a function of group, visit and interaction between group and visit. If the interaction between group and visit was statistically significant, individual changes within group or visit were assessed. Outcome measures were means including standard deviation (tables) and estimated marginal means with 95% confidence intervals (figures). Changes between antenatal and four-year follow-up (for women with one delivery) and between one-year and four-year follow-up (for women with ≥2 deliveries) were compared.

The initial power calculation was based on the incidence of LAM avulsion after first delivery. Statistical analysis was performed using SPSS software version 23 (IBM, Armonk, NY, USA). A *P*-value <0.05 was considered statistically significant for all analyses.

**Results**

*Baseline characteristics*

Of the 269 nulliparous women, 191 (71%) were followed up at three months postpartum, 147 (55%) at one and 147 (55%) at four years (Figure 1). At four years, the mean age was 34 (SD 5) years and the mean BMI 26.6 (SD 6.0). The mean time after first delivery was 3.8 (0.4 SD) years. 74 women (50%) had a subsequent delivery (70 had a second and 4 had a third delivery) with a mean follow-up time of 1.4 (SD 0.8) years after second delivery. Three women had a VBAC and seven women had a caesarean after vaginal delivery.

*LAM avulsion*

At four years, 20/31 (65%) women diagnosed with LAM avulsion either at three months or one year postpartum attended for follow-up. One minor and one major LAM avulsion were diagnosed at four years in women who did not attend previous follow-ups. Of these 22 women with LAM avulsion, at the four-year follow-up 8 (36%) were no longer evident and 14 (64%) had a persistent avulsion. The overall prevalence of LAM avulsion four years following first vaginal delivery was 14/108 (13%); 12/108 (11%) were major and 2/108 (2%) were minor (Table 1, supplemental digital content 1).

At four years, among the 12 women with only one vaginal delivery, 6 out of 9 (67%) major LAM avulsions found at three months or one year postpartum persisted and the remaining 3 (33%) where no longer evident. Of the minor LAM avulsions, 1 out of 3 (33%) persisted and the remaining 2 (67%) were no longer evident. Although not significant, LAM avulsions caused by forceps delivery were more likely to persist than LAM avulsions sustained during spontaneous or ventouse delivery (4/5 (80%) vs 3/7 (42.9%), *P*=0.293). Prevalence of any LAM avulsion four years after one vaginal delivery was 12.7% (7/55) (Table 1, supplemental digital content 1).

At four years, 9 women with LAM avulsion diagnosed three months after first delivery had a second vaginal delivery. Of the 7 women with a major avulsion, 2 persisted before and after the second delivery, in 3 the major avulsion was no longer evident before and after second delivery, and in 2 one minor and one major avulsion were visible after second delivery which was not evident before (i.e. at one year visit). Of the 2 minor LAM avulsions found three months after the first delivery, both deteriorated to a major avulsion after second vaginal delivery (Figure 2). In total 4/9 (44%) LAM avulsions worsened. One major LAM avulsion was found at four-years in a woman who did not attend previous follow-up visits. Prevalence of any LAM avulsion after two vaginal deliveries was 13.2% (7/53) (Table 1, supplemental digital content 1). The prevalence of LAM avulsion was not higher after a second vaginal delivery than after only one vaginal delivery (13.2% vs 12.7%, *P*=0.941).

No new LAM avulsions were found after the second vaginal delivery in women diagnosed with an intact LAM three months after their first delivery. One new major LAM avulsion was diagnosed in a woman who had a VBAC.

*Pelvic Floor Dysfunction*

Mean values for signs and symptoms of PFD at each visit of women with only one delivery are provided in Table 2 and of women with ≥2 vaginal deliveries in Table 3.

For women with only one delivery, data at four-years were compared to the antenatal assessment at 36 weeks of pregnancy (Table 2, supplemental digital content 2). No significant difference in symptoms of UI, AI and SD were found irrespective of mode of delivery and LAM morphology. Symptoms of POP were significantly worse shortly after vaginal delivery in women with persistent LAM avulsion, however this did not remain significant after four years (*P*=0.125-0.297). PFMS was significantly reduced in women with persistent LAM avulsion compared to women with CS, intact LAM and LAM avulsion that was no longer evident; diff. -1.8 vs. +0.1, +0.1, -0.3 MOS point resp. (*P*=<0.001-0.025). In women with a previous LAM avulsion that was no longer evident at four years, anterior vaginal wall prolapse was worse than for women with CS (diff. -0.7 vs -0.1 cm; *P*=0.047) and posterior vaginal wall prolapse was worse than for women with CS (diff. -0.5 vs +0. 1 cm, *P*=0.006) and intact LAM (diff. -0.5 vs -0.1 cm, *P*=0.048). Women with a persistent LAM avulsion did not have worsening of anterior (diff. -0.4 vs -0.1 and -0.2 cm) and posterior vaginal wall prolapse (diff. -0.0 vs +0.1 and -0.1 cm) as compared to women with an intact LAM. No significant changes in apical prolapse were observed. The hiatal area on ultrasound was significantly increased in women with a persistent LAM avulsion compared to women with CS and intact LAM at squeeze (diff. +2.5 vs -2.1 and -2.1 cm2; *P*<0.001) and Valsalva (diff. +5 vs -1.3 and -1.7 cm2; *P*=0.004-0.006).

For women with a second delivery, data after second delivery (4Y FU) were compared to data before the second delivery (1Y FU) (Table 3, supplemental digital content 2). No significant difference in symptoms of PFD (UI, AI, POP, SD) was found irrespective of mode of delivery and LAM morphology. PFMS was significantly worse in women with LAM avulsion after first delivery, but only a trend to deterioration after second delivery was noticed compared to the other groups (diff. -0.7 MOS point vs -0.1, +0.2, 0.0; *P*=0.103-0.405). Anterior vaginal wall prolapse was significantly worse in women with LAM avulsion after the first delivery compared to women with CS and intact LAM; however it did not deteriorate after the second delivery compared to the other groups (diff. -0.1 vs +0.2, +0.1, 0.0; *P*=0.613-0.973). This indicates that the worsening of PFMS and anterior vaginal prolapse occurred within the first year after levator avulsion. No significant difference in apical and posterior vaginal prolapse was seen. Hiatal area on ultrasound (rest, squeeze and Valsalva) increased significantly after the first delivery in women with LAM avulsion compared to the other groups. Hiatal area deteriorated after second delivery in women with persistent LAM avulsion compared to women with an intact LAM at rest (diff. +0.5 vs -1.7 cm2; *P*=0.024) and valsalva (diff. +4.1 vs -1.5 cm2; *P*=0.026).

In the seven women who had a caesarean section after a previous vaginal delivery, three (42%) had been diagnosed with LAM avulsion at three months after first vaginal delivery. In none of them was the reason for the caesarean section due to symptoms of PFD and none had signs or symptoms of PFD at four-year follow-up.

**Discussion**

*Main findings*

Four years after one vaginal delivery, 42% of LAM avulsions diagnosed with TPUS three months postpartum were no longer evident; minor avulsions were more likely to improve than major avulsions (67% vs 33%). No new LAM avulsions were diagnosed after a second vaginal delivery, however in 44% of women who were previously diagnosed with LAM avulsion it became more extensive. The prevalence of LAM avulsion four years following the first vaginal delivery was 13% and it was not different for women with one or two vaginal deliveries.

Four years after one vaginal delivery, in women with an intact LAM, no changes in symptoms and signs of PFD were found. In women with a previous LAM avulsion that was no longer evident, worsening of POP-Q measurements were found, although symptoms of PFD, PFMS and LAM area on ultrasound did not change. In women with persistent LAM avulsion, reduced PFMS and an enlarged LAM area were found, but this was not associated with symptoms of PFD and POP on clinical examination.

A second vaginal delivery had no effect on symptoms and signs of PFD in women with an intact LAM or LAM avulsion that was no longer evident. After second vaginal delivery, hiatal area increased in women with persistent LAM avulsion, without changes in symptoms of PFD, PFMS and POP on clinical examination.

*Strengths and Limitations*

This study is the first to prospectively assess LAM integrity and correlation with PFD over a four-year period.Analysis was performed based on parity, and groups were homogenous as women with two different modes of delivery were excluded from analyses. Signs and symptoms were assessed using validated methods and LAM avulsion was assessed by two observers to minimise performance bias. We acknowledge that the four-year follow-up was performed by a different research fellow (IvG), but consistency was maintained as both fellows were trained by the principal investigator (RT). Although a large number of nulliparous women were recruited of which 55%attended the four-year follow-up, the number of women with LAM avulsion was low, leading to small subgroup analyses. Perhaps there are more women with non-persisting avulsion as some have not attended previous follow-ups.

*Interpretation*

After the first delivery, the prevalence of LAM avulsion decreased from 21% at three months to 8% at one year postpartum as 62% of LAM avulsion were no longer evident10. Although no new LAM avulsions were diagnosed, some old avulsions became apparent or deteriorated after second vaginal delivery. This could be a genuine effect, but we cannot exclude the possibility of a misdiagnosis of an intact LAM at one year; however, the risk of misinterpretation was minimised as all volumes were assessed by two blinded investigators and any discrepancies were resolved by a tertiary investigator. Other studies assessing the LAM condition before and after a second vaginal delivery did not find any deterioration of LAM avulsion13,18.

Shek et al. found anatomical improvement in 17% of women with LAM avulsion 2.6 years after first vaginal delivery, but they had a low follow-up rate of women with LAM avulsion (38%)26. Chan et al. found improvement in 13%, 3.8 years following first vaginal delivery13. Both improvement rates are lower than our 36% using a similar methodology to diagnose LAM avulsion. A possible explanation could be the differences in ethnicity and use of forceps. Moreover, they did not include minor avulsion, which is more likely to improve compared to more extensive injury10.

No significant difference in the prevalence of LAM avulsion was found after one or two vaginal deliveries, as confirmed by two others14,16,17. Similar to Horak et al. no new cases of avulsion were found after a second vaginal delivery16.Chan et al. found one new LAM avulsion after a second vaginal delivery13.The incidence of LAM avulsion after second vaginal delivery is low and the prevalence of LAM avulsion is similar after one or two vaginal deliveries, confirming that the first vaginal delivery is at greatest risk of LAM trauma15,16,18.

The difference in hiatal area between antenatal and four years was significantly larger in women with persistent LAM avulsion, which could be explained by a small hiatal area at 36 weeks gestation. This is in keeping with the study of Siafarikas et al who found a smaller hiatal area at rest and during Valsalva in late pregnancy to be associated with LAM avulsion postpartum8. We found a significant deterioration of hiatal area after a second vaginal delivery in women with LAM avulsion. One study found a greater hiatal area at rest in multiparous compared to primiparous women14, butothers did not find an enlargement of hiatal area after the second vaginal delivery15,18.

Objective measurements (PFMS and hiatal area) significantly changed in women with persistent LAM avulsion, but this was not paralleled with a subjective change in POP symptoms. Handa et al. showed that LAM avulsion is strongly associated with prolapse beyond the hymen (OR 2.7) and symptoms of prolapse (OR 3.0) at a mean of 11 years postpartum28, which they attributed to a larger hiatal area and weaker pelvic muscles29. This suggests that it may take more than four years for symptoms of POP to develop.

Considering women with LAM avulsion are at increased risk to develop PFD, it would be of value to identify them, although the best timing for this assessment has yet to be established. More importantly, every effort must be made to prevent LAM avulsion during first vaginal delivery, for instance by reducing the number of forceps deliveries and duration of second stage of labour7,8,13,30.

In conclusion, the first vaginal delivery is at greatest risk for LAM avulsion to occur, with an impact on PFD four years later. A second vaginal delivery could result in deterioration of LAM avulsion, but no new avulsions were found. Larger longer term prospective studies of primiparous and multiparous women are needed to establish whether a caesarean section would benefit women who have sustained a previous LAM avulsion. Attention needs to be focused on preventative strategies to minimise the risk of sustaining LAM avulsion.

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**Disclosure of interests**

The authors declare that they have no conflict of interest

**Contribution to authorship**

IMA van Gruting Data collection and management, Data analysis, Manuscript writing

KWM van Delft Project development, Data collection and management, Manuscript writing

R Thakar Protocol and project development, Manuscript editing

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**Ethics Approval**

This study was approved by the National Research Ethics Service South West London committee (REC 10/H0806/87) on 17 November 2010.

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**References**

1. DeLancey JO, Morgan DM, Fenner DE, Kearney R, Guire K, Miller JM, Hussain H, Umek W, Hsu Y, Ashton-Miller JA. Comparison of levator ani muscle defects and function in women with and without pelvic organ prolapse. Obstet Gynecol 2007;109:295–302.

2. Dietz HP, Simpson JM. Levator trauma is associated with pelvic organ prolapse. BJOG 2008;115:979–84.

3. Dietz HP, Shek C, De Leon J, Steensma AB. Ballooning of the levator hiatus. Ultrasound Obstet Gynecol 2008;31:676–80.

4. Oversand SH, Staff AC, Sandvik L, Volløyhaug I, Svenningsen R. Levator ani defects and the severity of symptoms in women with anterior compartment pelvic organ prolapse. Int Urogynecol J. 2018 Jan;29(1):63-69.

5. Friedman T, Eslick GD, Dietz HP. Risk factors for prolapse recurrence: systematic review and meta-analysis. Int Urogynecol J. 2018 Jan;29(1):13-21.

6. Vergeldt TF, van Kuijk SM, Notten KJ, Kluivers KB, Weemhoff M. Anatomical Cystocele Recurrence: Development and Internal Validation of a Prediction Model. Obstet Gynecol. 2016 Feb;127(2):341-7.

7. van Delft K, Thakar R, Sultan AH, Schwertner-Tiepelmann N, Kluivers K. Levator ani muscle avulsion during childbirth: a risk prediction model. BJOG. 2014 Aug;121(9):1155-63; discussion 1163.

8. Siafarikas F, Staer-Jensen J, Hilde G, Bø K, Ellström Engh M. The levator ani muscle during pregnancy and major levator ani muscle defects diagnosed postpartum: a three- and four-dimensional transperineal ultrasound study. BJOG. 2015 Jul;122(8):1083-91.

9. van Delft K, Sultan AH, Thakar R, Schwertner-Tiepelmann N, Kluivers K. The relationship between postpartum levator ani muscle avulsion and signs and symptoms of pelvic floor dysfunction. BJOG. 2014 Aug;121(9):1164-71; discussion 1172.

10. van Delft KW, Thakar R, Sultan AH, IntHout J, Kluivers KB. The natural history of levator avulsion one year following childbirth: a prospective study. BJOG. 2015 Aug;122(9):1266-73.

11. Halle TK, Staer-Jensen J, Hilde G3, Bø K, Ellström Engh M, Siafarikas F. Change in prevalence of major levator ani muscle defects from six weeks to one year postpartum, and maternal and obstetric risk factors: a longitudinal ultrasound study. Acta Obstet Gynecol Scand. 2020 Apr 22. doi: 10.1111/aogs.13878.

12. Glazener C, Elders A, MacArthur C, Lancashire RJ, Herbison P, Hagen S, Dean N, Bain C, Toozs-Hobson P, Richardson K, McDonald A, McPherson G, Wilson D; ProLong Study Group. Childbirth and prolapse: long-term associations with the symptoms and objective measurement of pelvic organ prolapse. BJOG. 2013 Jan;120(2):161-168.

13. Chan SSC, Cheung RYK, Lee LL, Choy RKW, Chung TKH. Longitudinal follow-up of levator ani muscle avulsion: does a second delivery affect it? Ultrasound Obstet Gynecol. 2017 Jul;50(1):110-115.

14. Chan SSC**,** Cheung RYK, Lee LL, Chung TKH. Longitudinal pelvic floor biometry: which factors affect it? Ultrasound Obstet Gynecol. 2018 Feb;51(2):246-252.

15. Kamisan Atan I, Gerges B, Shek KL, Dietz HP. The association between vaginal parity and hiatal dimensions: a retrospective observational study in a tertiary urogynaecological centre. BJOG. 2015 May;122(6):867-72.

16. Kamisan Atan I, Lin S, Dietz HP, Herbison P, Wilson PD; ProLong Study Group. It is the first birth that does the damage: a cross-sectional study 20 years after delivery. Int Urogynecol J. 2018 Mar 21.

17. Dietz HP, Walsh C, Subramaniam N, Friedman T. Levator Avulsion and Vaginal Parity: Do Subsequent Vaginal Births Matter? Int Urogynecol J. 2020 May 30. doi: 10.1007/s00192-020-04330-4.

18. Horak TA, Guzman-Rojas RA, Shek KL, Dietz HP. Pelvic floor trauma: does the second baby matter? Ultrasound Obstet Gynecol. 2014 Jul;44(1):90-4.

19. Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P. ICIQ: a brief and robust measure for evaluating the symptoms and impact of urinary incontinence. Neurourol Urodyn 2004;23:322–30.

20. Vaizey CJ, Carapeti E, Cahill JA, Kamm MA. Prospective comparison of faecal incontinence grading systems. Gut 1999;44:77–80.

21. Price N, Jackson SR, Avery K, Brookes ST, Abrams P. Development and psychometric evaluation of the ICIQ Vaginal Symptoms Questionnaire: the ICIQ-VS. BJOG 2006;113:700–12.

22. Laycock J. Clinical evaluation of the pelvic floor. In *Pelvic floor re‐education, Principles and practice*, Schüssler B, Laycock J, Norton PA, Stanton SL, eds. Springer‐Verlag: London, 1994; 42–48

23. Hall AF, Theofrastous JP, Cundiff GW, Harris RL, Hamilton LF, Swift SE, Bump RC. Interobserver and intraobserver reliability of the proposed International Continence Society, Society of Gynecologic Surgeons, and American Urogynecologic Society pelvic organ prolapse classification system. Am J Obstet Gynecol 1996;175:1467–70.

24. Dietz HP. Quantification of major morphological abnormalities of the levator ani. Ultrasound Obstet Gynecol. 2007 Mar;29(3):329-34.

25. Dietz HP, Bernardo MJ, Kirby A, Shek KL. Minimal criteria for the diagnosis of avulsion of the puborectalis muscle by tomographic ultrasound. Int Urogynecol J 2011;22:699–704.

26. Shek KL, Chantarasorn V, Langer S, Dietz HP. Does levator trauma 'heal'? Ultrasound Obstet Gynecol. 2012 Nov;40(5):570-5.

27. Branham V, Thomas J, Jaffe T, Crockett M, South M, Jamison M, et al. Levator ani abnormality 6 weeks after delivery persists at 6 months. Am J Obstet Gynecol 2007;197:65.e1–6

28. Handa VL, Blomquist JL, Roem J, Muñoz A, Dietz HP. Pelvic Floor Disorders After Obstetric Avulsion of the Levator Ani Muscle. Female Pelvic Med Reconstr Surg. 2019 Jan/Feb;25(1):3-7.

29. Handa VL, Roem J, Blomquist JL, Dietz HP, Muñoz A. Pelvic organ prolapse as a function of levator ani avulsion, hiatus size, and strength. Am J Obstet Gynecol. 2019 Jul;221(1):41.e1-41.e7.

30. Dietz HP. Forceps: towards obsolescence or revival? Acta Obstet Gynecol Scand. 2015 Apr;94(4):347-51.

**Table 1** Prevalence of LAM avulsion 4 years following first vaginal delivery

|  |  |  |  |
| --- | --- | --- | --- |
|  | **Total (*n*)** | **1 VD (*n*)** | **≥ 2 VD (*n*)** |
| **LAM avulsion**  **present at 4 years** | 14 (64%) | 7 (58%) | 7 (70%) |
| 12 major | 6 major | 6 major |
| 2 minor | 1 minor | 1 minor |
| **LAM avulsion**  **no longer evident at 4 years** | 8 (36%) | 5 (42%) | 3 (30%) |
| 6 major | 3 major | 3 major |
| 2 minor | 2 minor | 0 minor |
| **Total number of LAM avulsion at any time point** | 22 (100%) | 12 (100%) | 10 (100%) |
| 17 major# | 9 major | 8 major# |
| 5 minor\* | 3 minor | 2 minor\* |
| **Number of women with VD** | 108 | 55 | 53 |
| **Prevalence** | 13% (14/108) | 12.7% (7/55) | 13.2% (7/53) |

LAM = levator ani muscle

VD = vaginal delivery

\* Two minor avulsions deteriorated to major avulsion due to second delivery

# Two major avulsions were no longer evident 1 year after vaginal delivery but after second delivery a minor and a major avulsion were seen again

**Table 2** Signs and symptoms of PFD over a four-year time period in women with one delivery

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Variable** | **Group** | **N (min-max)** | **Antenatal**  **n=73** | **3M PP**  **n=60** | **1Y PP**  **n=56** | **4Y PP**  **n=71** |
| **Symptoms PFD** | | | | | | |
| **Urinary incontinence (range 0-21)**  *Mean (SD)* | 1 CS | 20-25 | 4.2 (5.4) | 2.0 (4.7) | 3.6 (5.8) | 3.9 (5.9) |
| 1 SVD LAM intact | 32-39 | 2.7 (3.4) | 1.6 (3.3) | 2.4 (3.2) | 3.0 (3.7) |
| 1 SVD LAM no longer evident | 3-4 | 1.8 (2.1) | 1.5 (3.0) | 1.7 (2.9) | 0.8 (1.5) |
| 1 SVD LAM avulsion | 3-5 | 3.6 (3.5) | 4.7 (5.7) | 5.3 (4.7) | 5.2 (4.1) |
| **Anal incontinence**  **(range 0-24)**  *Mean (SD)* | 1 CS | 20-25 | 0.5 (1.5) | 0.1 (0.2) | 0.2 (0.5) | 0.3 (0.9) |
| 1 SVD LAM intact | 30-39 | 0.6 (1.6) | 0.7 (2.0) | 0.2 (1.0) | 0.2 (0.8) |
| 1 SVD LAM no longer evident | 3-4 | 0.3 (0.5) | 0.5 (1.0) | 0.0 (0.0) | 0.3 (0.5) |
| 1 SVD LAM avulsion | 3-5 | 0.2 (0.4) | 0.7 (1.2) | 0.3 (0.6) | 1.4  (3.1) |
| **POP symptoms**  **(range 0-28)**  *Mean (SD)* | 1 CS | 21-25 | 1.2 (3.5) | 0.2 (0.8) | 0.6 (2.8) | 2.3 (6.2) |
| 1 SVD LAM intact | 32-39 | 0.8 (3.4) | 1.1 (3.9) | 0.0 (0.0) | 0.9 (2.6) |
| 1 SVD LAM no longer evident | 3-4 | 3.5 (7.0) | 0.0 (0.0) | 0.7 (1.2) | 1.3 (2.5) |
| 1 SVD LAM avulsion | 3-5 | 2.8 (6.3) | 9.0 (15.6) | 11.0 (14.2) | 7.2 (10.7) |
| **Sexual dysfunction**  **(range 0-36)**  *Mean (SD)* | 1 CS | 12-24 | 1.8 (2.8) | 4.3 (10.7) | 1.8 (4.1) | 5.1 (10.9) |
| 1 SVD LAM intact | 21-30 | 2.2 (6.7) | 5.0 (7.2) | 4.5 (9.7) | 2.6 (5.7) |
| 1 SVD LAM no longer evident | 1-4 | 1.0 (1.7) | 5.3 (6.8) | 0.0 (na) | 2.5 (5.0) |
| 1 SVD LAM avulsion | 1-4 | 0.0 (0.0) | 8.0 (na) | 14.0 (11.1) | 7.5 (11.9) |
| **Signs of PFD on clinical examination** | | | | | | |
| **MOS**  **(range 0-5)**  *Mean (SD)* | 1 CS | 20-25 | 3.5 (1.3) | 3.9 (1.3) | 3.3 (1.3) | 3.6 (1.6) |
| 1 SVD LAM intact | 30-39 | 3.6 (1.2) | 3.2 (1.2) | 3.2 (1.1) | 3.7 (1.0) |
| 1 SVD LAM no longer evident | 3-4 | 3.3 (1.7) | 3.3 (1.7) | 3.7 (1.2) | 3.0 (1.4) |
| 1 SVD LAM avulsion | 3-5 | 3.4 (1.5) | 1.7 (1.2) | 1.7 (0.6) | 1.6 (1.1) #^\* |
| **Ba in cm**  **(range -3 to +10)**  *Mean (SD)* | 1 CS | 20-25 | -2.8 (0.4) | -2.9 (0.4) | -2.7 (0.6) | -2.7 (0.5) |
| 1 SVD LAM intact | 30-39 | -2.7 (0.5) | -2.2 (0.7) | -2.3 (0.8) | -2.5 (0.6) |
| 1 SVD LAM no longer evident | 3-4 | -2.5 (0.6) | -1.8 (1.5) | -1.7 (1.5) | -1.8 (1.3) # |
| 1 SVD LAM avulsion | 3-5 | -3.0 (0.0) | -1.7 (0.6) | -2.0 (1.0) | -2.6 (0.5) |
| **C in cm**  **(range -10 to +10)**  *Mean (SD)* | 1 CS | 20-24 | -8.7 (0.6) | -8.5 (1.1) | -8.1 (1.5) | -7.0 (1.5) |
| 1 SVD LAM intact | 30-39 | -8.6 (0.8) | -8.1 (1.3) | -8.5 (0.9) | -7.5 (1.6) |
| 1 SVD LAM no longer evident | 3-4 | -8.8 (0.5) | -6.8 (2.1) | -7.3 (2.9) | -5.5 (2.5) |
| 1 SVD LAM avulsion | 3-5 | -8.2 (1.1) | -9.0 (0.0) | -8.7 (0.6) | -7.4 (1.1) |
| **Bp in cm**  **(range -3 to +10)**  *Mean (SD)* | 1 CS | 20-25 | -2.9 (0.3) | -2.9 (0.3) | -2.9 (0.3) | -3.0 (0.2) |
| 1 SVD LAM intact | 30-39 | -2.9 (0.4) | -2.6 (0.8) | -2.6 (0.7) | -2.8 (0.6) |
| 1 SVD LAM no longer evident | 3-4 | -3.0 (0.0) | -2.5 (1.0) | -2.7 (0.6) | -2.5 (0.6) #^ |
| 1 SVD LAM avulsion | 3-5 | -3.0 (0.0) | -2.3 (0.6) | -2.0 (1.7) | -3.0 (0.0) \*§†‡ |
| **Signs of PFD on Ultrasound** | | | | | | |
| **Hiatal area in cm3** *- Rest*  *Mean (SD)* | 1 CS | 20-25 | 15.5 (4.3) | 13.6 (3.6) | 14.5 (3.9) | 13.7 (3.5) |
| 1 SVD LAM intact | 30-39 | 16.5 (3.3) | 14.9 (3.3) | 16.1 (3.0) | 14.1 (3.3) |
| 1 SVD LAM no longer evident | 3-4 | 14.9 (4.7) | 14.7 (2.7) | 16.7 (2.5) | 13.7 (2.8) |
| 1 SVD LAM avulsion | 3-5 | 13.3 (2.3) | 13.8 (2.5) | 15.7 (2.9) | 15.3 (3.5) |
| **Hiatal area in cm3** *- Squeeze*  *Mean (SD)* | 1 CS | 20-25 | 12.3 (2.7) | 10.6 (2.1) | 11.5 (3.0) | 10.2 (2.3) |
| 1 SVD LAM intact | 30-39 | 12.8 (2.4) | 12.7 (3.3) | 12.4 (2.7) | 10.7 (2.8) |
| 1 SVD LAM no longer evident | 3-4 | 11.6 (4.4) | 12.4 (1.8) | 11.8 (1.2) | 10.9 (1.6) |
| 1 SVD LAM avulsion | 3-5 | 10.5 (2.1) | 13.5 (3.4) | 14.9 (3.5) | 13.0 (3.6) \*^# |
| **Hiatus area in cm3** *- Valsalva*  *Mean (SD)* | 1 CS | 20-24 | 19.6 (6.3) | 15.9 (3.4) | 17.8 (4.3) | 18.3 (5.0) |
| 1 SVD LAM intact | 30-38 | 21.2 (5.2) | 20.0 (4.9) | 22.4 (7.6) | 19.5 (6.5) § |
| 1 SVD LAM no longer evident | 3-4 | 19.9 (4.8) | 22.4 (10.8) | 24.8 (7.6) | 22.1 (8.1) |
| 1 SVD LAM avulsion | 3-5 | 16.1 (1.7) | 21.9 (10.4) | 23.1 (8.3) | 21.1 (5.5) ^# |

# Significant difference between AN and 4 year follow up compared to CS

^ Significant difference between AN and 4 year follow up compared to SVD LAM intact

\* Significant difference between AN and 4 year follow up compared to SVD LAM no longer evident

¥ Significant difference between AN and 4 year follow up compared to SVD LAM avulsion

§ Significant difference between 1 year and 4 year follow up compared to CS

† Significant difference between 1 year and 4 year follow up compared to SVD LAM intact

‡ Significant difference between 1 year and 4 year follow up compared to SVD LAM no longer evident

¶ Significant difference between 1 year and 4 year follow up compared to SVD LAM avulsion

POP = pelvic organ prolapse

MOS= modified oxford score

Ba = position on anterior vaginal wall (POP-Q)

Bp = position on posterior vaginal wall (POP-Q)

C= position of cervix (POP-Q)

LAM= levator ani muscle

PP= postpartum

**Table 3** Signs and symptoms of PFD four years after first delivery in women with two or more deliveries

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Variable** | **Group** | **N**  **(min-max)** | **Before 1st delivery**  n=64 | **3 months after 1st delivery**  n=57 | **1 year after 1st delivery**  n=47 | **After 2nd delivery (4 years after 1st delivery)**  n=64 |
| **Symptoms PFD** | | | | | | |
| **Urinary incontinence (range 0-21)**  *Mean (SD)* | 2 CS | 9-11 | 3.0 (4.1) | 1.4 (3.1) | 1.7 (2.6) | 3.6 (3.8) |
| 2 SVD LAM intact | 34-43 | 2.9 (3.6) | 1.9 (2.9) | 2.3 (2.7) | 4.2 (4.6) |
| 2 SVD LAM no longer evident | 3 | 1.0 (1.7) | 2.0 (1.7 | 0.0 (0.0) | 1.0 (1.7) |
| 2 SVD LAM avulsion | 4-7 | 6.0 (4.4) | 2.3 (3.7) | 2.7 (3.2) | 4.1 (4.8) |
| **Anal incontinence**  **(range 0-24)**  *Mean (SD)* | 2 CS | 7-11 | 0.3 (0.9) | 0.0 (0.0) | 0.7 (1.9) | 0.1 (0.3) |
| 2 SVD LAM intact | 33-43 | 0.6 (1.8) | 1.5 (3.2) | 0.4 (1.1) | 0.4 (1.4) |
| 2 SVD LAM no longer evident | 3 | 0.0 (0.0) | 0.0 (0.0) | 0.0 (0.0) | 0.0 (0.0) |
| 2 SVD LAM avulsion | 4-7 | 1.0 (1.7) | 1.7 (4.1) | 0.5 (1.0) | 1.1 (2.0) |
| **POP symptoms**  **(range 0-28)**  *Mean (SD)* | 2 CS | 9-11 | 0.2 (0.6) | 0.0 (0.0) | 0.0 (0.0) | 0.0 (0.0) |
| 2 SVD LAM intact | 33-43 | 0.7 (2.5) | 1.0 (3.0) | 1.0 (4.0) | 1.4 (3.2) |
| 2 SVD LAM no longer evident | 3 | 0.0 (0.0) | 2.7 (4.6) | 0.0 (0.0) | 7.7 (13.3) |
| 2 SVD LAM avulsion | 4-7 | 0.3 (0.8) | 2.7 (6.5) | 3.5 (7.0) | 3.1 (5.6) |
| **Sexual dysfunction**  **(range 0-36)**  *Mean (SD)* | 2 CS | 7-10 | 6.9 (11.0) | 10.0 (9.4) | 5.1 (6.4) | 7.3 (11.0) |
| 2 SVD LAM intact | 25-40 | 2.4 (4.8) | 4.9 (5.8) | 3.0 (5.5) | 6.8 (9.3) |
| 2 SVD LAM no longer evident | 1-2 | 6.5 (9.2) | 4.0 (na) | 0.0 (0.0) | 1.0 (1.4) |
| 2 SVD LAM avulsion | 4-5 | 0.8 (1.5) | 2.3 (4.5) | 0.0 (0.0) | 0.6 (1.3) |
| **Signs of PFD on clinical examination** | | | | | | |
| **MOS**  **(range 0-5)**  *Mean (SD)* | 2 CS | 7-11 | 3.0 (1.5) | 3.6 (1.6) | 3.7 (1.4) | 3.6 (1.3) |
| 2 SVD LAM intact | 33-43 | 3.6 (1.3) | 2.9 (1.3) | 3.2 (1.2) | 3.4 (1.2) # |
| 2 SVD LAM no longer evident | 3 | 3.3 (2.1) | 3.7 (2.3) | 3.0 (1.7) | 3.0 (2.6) |
| 2 SVD LAM avulsion | 4-7 | 2.9 (1.4) | 2.3 (1.2) | 2.8 (1.7) | 2.1 (1.1) # |
| **Ba in cm**  **(range -3 to +10)**  *Mean (SD)* | 2 CS | 7-11 | -2.8 (0.4) | -2.9 (0.3) | -2.6 (0.5) | -2.8 (0.4) |
| 2 SVD LAM intact | 33-43 | -2.7 (0.5) | -2.3 (0.7) | -2.4 (0.5) | -2.5 (0.6) |
| 2 SVD LAM no longer evident | 3 | -2.7 (0.6) | -1.7 (0.6) | -2.3 (0.6) | -2.3 (1.2) |
| 2 SVD LAM avulsion | 4-7 | -2.6 (0.5) | -1.5 (1.0) | -1.8 (1.0) | -1.7 (1.3) #^ |
| **C in cm**  **(range -10 to +10)**  *Mean (SD)* | 2 CS | 7-11 | -8.9 (0.3) | -9.0 (0.0) | -8.7 (0.8) | -7.3 (1.6) |
| 2 SVD LAM intact | 33-43 | -8.4 (1.0) | -7.9 (1.4) | -8.1 (1.4) | -7.3 (1.4) |
| 2 SVD LAM no longer evident | 3 | -7.7 (1.5) | -7.3 (2.1) | -8.3 (1.2) | -6.3 (1.2) |
| 2 SVD LAM avulsion | 4-7 | -8.6 (0.8) | -7.8 (0.8) | -9.0 (0.0) | -6.7 (1.5) |
| **Bp in cm**  **(range -3 to +10)**  *Mean (SD)* | 2 CS | 7-11 | -3.0 (0.0) | -2.8 (0.4) | -2.7 (0.5) | -3.0 (0.0) |
| 2 SVD LAM intact | 33-43 | -3.0 (0.2) | -2.8 (0.4) | -2.9 (0.3) | -2.8 (1.0) |
| 2 SVD LAM no longer evident | 3 | -3.0 (0.0) | -2.0 (1.0) | -3.0 (0.0) | -3.0 (0.0) |
| 2 SVD LAM avulsion | 4-7 | -2.9 (0.4) | -2.5 (0.8) | -2.3 (1.5) | -2.9 (0.4) |
| **Signs of PFD on Ultrasound** | | | | | | |
| **Hiatal area in cm2** *- Rest*  *Mean (SD)* | 2 CS | 7-11 | 16.2 (3.7) | 13.0 (3.1) | 14.4 (1.2) | 13.8 (2.6) |
| 2 SVD LAM intact | 33-43 | 15.8 (3.3) | 15.3 (3.3) | 16.0 (3.3) | 14.3 (2.9) § |
| 2 SVD LAM no longer evident | 3 | 18.8 (8.3) | 17.0 (7.5) | 17.4 (5.0) | 18.0 (4.5) |
| 2 SVD LAM avulsion | 4-7 | 13.5 (2.5) | 16.6 (3.6) | 16.9 (3.7) | 17.4 (3.9) †\* ^# |
| **Hiatal area in cm2** *- Squeeze*  *Mean (SD)* | 2 CS | 7-11 | 12.9 (3.0) | 9.5 (1.8) | 10.6 (1.3) | 9.9 (2.2) |
| 2 SVD LAM intact | 33-43 | 12.3 (2.7) | 12.4 (2.5) | 12.4 (2.7) | 11.2 (2.3) # |
| 2 SVD LAM no longer evident | 3 | 12.9 (3.4) | 11.4 (1.3) | 13.1 (1.2) | 13.0 (1.3) # |
| 2 SVD LAM avulsion | 4-7 | 11.7 (2.8) | 15.2 (2.6) | 14.7 (4.4) | 15.7 (6.5) \*^# |
| **Hiatus area in cm2**  *- Valsalva*  *Mean (SD)* | 2 CS | 7-11 | 23.2 (6.7) | 19.1 (5.9) | 23.6 (5.5) | 19.8 (7.0) |
| 2 SVD LAM intact | 33-43 | 20.1 (6.7) | 20.4 (5.6) | 21.5 (6.2) | 20.0 (5.6) |
| 2 SVD LAM no longer evident | 3 | 28.6 (16.3) | 24.6 (11.1) | 23.5 (4.5) | 26.1 (9.9) |
| 2 SVD LAM avulsion | 4-7 | 15.8 (2.3) | 20.9 (4.0) | 25.8 (7.7) | 29.9 (11.3) #^\*† |

# Significant difference between AN and 4 year follow up compared to CS

^ Significant difference between AN and 4 year follow up compared to SVD LAM intact

\* Significant difference between AN and 4 year follow up compared to SVD LAM no longer evident

¥ Significant difference between AN and 4 year follow up compared to SVD LAM avulsion

§ Significant difference between 1 year and 4 year follow up compared to CS

† Significant difference between 1 year and 4 year follow up compared to SVD LAM intact

‡ Significant difference between 1 year and 4 year follow up compared to SVD LAM no longer evident

¶ Significant difference between 1 year and 4 year follow up compared to SVD LAM avulsion

POP = pelvic organ prolapse

MOS= modified oxford score

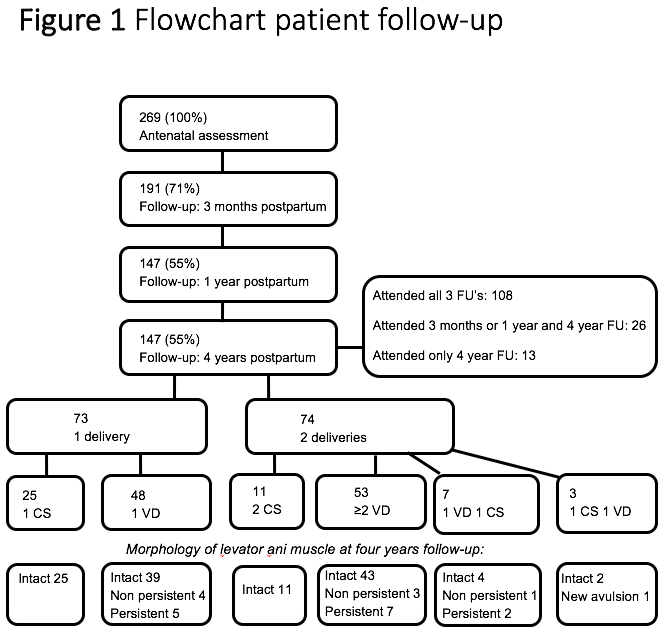
Ba = position on anterior vaginal wall (POP-Q)

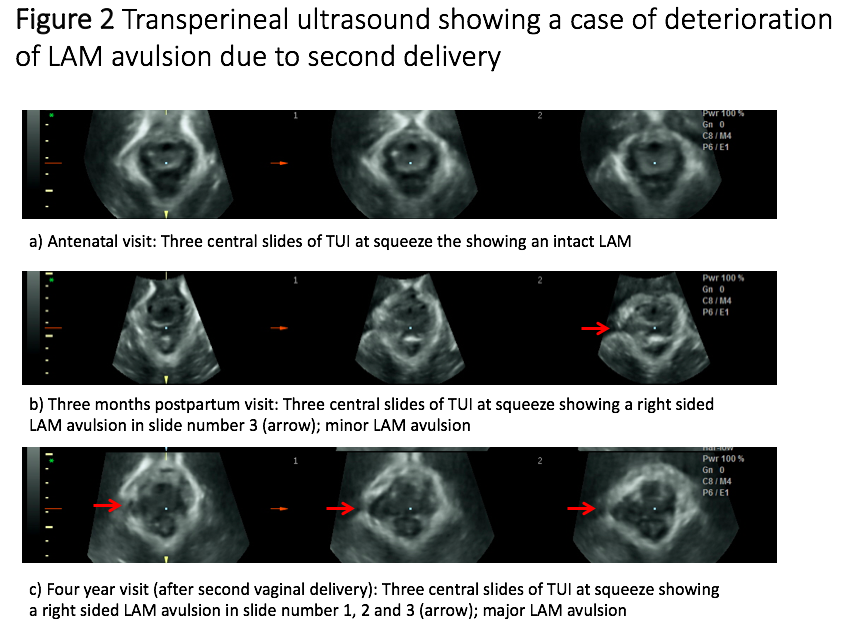
Bp = position on posterior vaginal wall (POP-Q)

C= position of cervix (POP-Q)

LAM= levator ani muscle

PP= postpartum

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**Supplemental digital content 1** Overview of patients with LAM avulsion

**Supplemental digital content 2** Graphs of symptoms and signs of pelvic floor dysfunction four years after first delivery in women with either one or two or more deliveries

a) Urinary incontinence

b) Faecal incontinence

c) Pelvic Organ Prolapse symptoms

d) Sexual dysfunction

e) Pelvic floor muscle strength

f) Anterior vaginal wall prolapse

g) Middle compartment prolapse

h) Posterior vaginal wall prolapse

i) Hiatal area at rest

j) Hiatal area at squeeze

k) Hiatal area at Valsalva