DO MAJOR DEFECTS OF THE LEVATOR ANI MUSCLE HEAL WITHIN THE FIRST 6 MONTHS POSTPARTUM?

Hypothesis / aims of study
The levator ani muscle (LAM) is an important part of the pelvic floor support. During vaginal delivery the LAM, especially the most medial part, is stretched, which may result in muscle tearing.

Studies examining women many years after delivery have found that women with POP have a higher prevalence of LAM defects than women without POP. LAM defects were diagnosed in 13-39.5% (1) of women after vaginal delivery. The highest prevalence was found in women as early as 48-72 hours postpartum (1). This might suggest rehabilitation potential for the LAM in line with other injured striated muscles in the body. MRI studies indicate that such LAM defects can recover, although the greatest potential for recovery is when there is no major pelvic floor muscle damage (2).

The large variation in reported prevalence of LAM defects may be explained by lack of consistency in the definition and classification of LAM defects (3). We used three central slices of tomographic ultrasound imaging (TUI) recorded using 3D/4D transperineal ultrasound, which is one of the best validated methods for diagnosing LAM defects. Our intention was to study whether LAM defects diagnosed early after delivery persisted at 6 months postpartum.

The aim of the present study was to use transperineal 3D/4D ultrasound to compare the status of LAM defects at 6 weeks and 6 months postpartum in women delivering their first child.

Study design, materials and methods
This prospective cohort study was conducted at a university hospital between July 2010 and March 2012. Primiparous women having delivered vaginally not earlier than gestational week 32 were included. All nulliparous pregnant women were invited to participate when they attended their routine second trimester ultrasound examination around gestational week 20. At six weeks and six months postpartum the women underwent a 3D/4D transperineal ultrasound examination in dorsal lithotomy position after voiding, using the GE Kretz Voluson E8 system (GE Medical Systems) with 4-8MHz curved array 3D/4D ultrasound transducer (RAB4-8/obstetric). The ultrasound images were stored offline by anonymous code numbers and analyzed using 4D View (v. 7.0 and 10.0; GE Healthcare). The examination was performed by two investigators who also analyzed the images for defects of the levator ani. Good to excellent inter- and intra-rater agreement for detecting defects of levator ani muscle 6 weeks after delivery was found (kappa 0.79).

Assessment of LAM defects was performed at maximal pelvic floor muscle contraction. Major levator ani muscle defects were defined using tomographic ultrasound imaging (TUI) when an abnormal muscle insertion was present in three central slices; at the plane of minimal dimensions and 2.5 mm and 5 mm cranially to it. The plane of minimal dimensions was defined in the midsagittal plane as the shortest distance from the inferoposterior margin of the symphysis pubis to the levator ani at maximal pelvic floor muscle contraction. In doubtful cases the levator–urethral gap (LUG) was used, with values > 2.5 cm being regarded as abnormal. Partial defects were defined when an abnormal muscle insertion was present in less than all three central planes. The examiners were blinded to all previously collected data, the women’s obstetric history and each others’ results. Statistical analysis was performed using SPSS v 15. Mean values for demographic data are presented as mean and standard deviation (SD). Frequencies are used to describe numbers of defects.

Results
The mean age of the 173 women was 29.7 years (SD 4.1). Seven women chose not to continue at 6 months, leaving 166 cases for follow-up. None of the women who chose not to continue in the study had been diagnosed with LAM defects at 6 weeks postpartum. Mean examination time after delivery was 6.2 weeks (SD 1.0) and 26.6 weeks (SD 2.6), respectively. BMI was 25.1 kg/m² at 6 weeks and 24.4 kg/m² at 6 months postpartum. 82.6% had normal vaginal delivery, and 17.4% had instrumental vaginal delivery.

At 6 weeks postpartum 29 women were diagnosed with major defects. Of those women, 22 (76%) were found to persist at 6 months postpartum. Five of the seven defects seen at 6 weeks postpartum did not fulfil the criteria for major defects at 6 months postpartum, but were found to have detachment of LAM in two of the three central planes, thus fulfilling the criteria for partial defects. In the remaining two cases no LAM defect was diagnosed at 6 months postpartum. No major defects were diagnosed only at 6 months.

Interpretation of results
Most major defects persist at 6 months after delivery. However, muscle recovery may happen after delivery. We found that two cases (7%) seemed to have recovered completely, whereas 5 (17%) had recovered partially. This is in accordance with other findings, showing recovery of LAM defects in the postpartum period.

Previous studies have shown that LAM defects diagnosed shortly after delivery may be difficult to distinguish from hematoma or oedema, which can persist up to three months postpartum. This might imply that the recovery found could partially be due to false positive diagnoses 6 weeks postpartum. The majority of healed defects, however, had signs of detachment, indicating some injury. No major defects of LAM were diagnosed only at 6 months, suggesting that a false negative diagnosis is relatively unlikely early postpartum.

Early diagnosis of LAM injuries may be important, as early intervention could enhance the healing process for LAM injuries as for all other striated muscles in the body. If this is so it may be that even more LAM defects would recover with early intervention, although further high quality randomized controlled trials are needed to clarify this.
Concluding message
Most defects persist at 6 months after delivery. However, when diagnosing major defects of LAM early postpartum. It must be borne in mind that natural recovery can take place within the first 6 months postpartum. Whether this healing process can be enhanced by early intervention remains to be studied.

References

Disclosures
Funding: Norwegian South-Eastern Regional Health Authority Clinical Trial: Yes Registration Number: NCT01045135 RCT: No Subjects: HUMAN Ethics Committee: REK Sør-Øst D 2009/170 Helsinki: Yes Informed Consent: Yes