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Uterotubal transport disorder in adenomyosis and endometriosis—a cause for infertility

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Objective Uterine hyperperistalsis and dysperistalsis are common phenomena in endometriosis and may be responsible for reduced fertility in cases of minimal or mild extent of disease. Since a high prevalence of adenomyosis uteri has been well documented in association with endometriosis, we designed a study to examine whether hyperperistalsis and dysperistalsis are caused by the endometriosis itself or by the adenomyotic component of the disease.

Design A prospective observational study.

Setting University hospital, Department of Obstetrics and Gynaecology, Division of Reproductive Medicine and Gynaecologic Endocrinology with 300 *in vitro* fertilisation/intracytoplasmatic sperm injection cycles and 350 intrauterine insemination cycles/year.

Population Forty-one subjects with infertility and with laparoscopically proven endometriosis and patent fallopian tubes. Thirty-five subjects (85%) additionally showed signs of adenomyosis.

Methods All subjects underwent T2-weighed magnetic resonance imaging (MRI) and hysterosalpingoscintigraphy (HSSG) during the subsequent menstrual cycle. MRI revealed the extent of the adenomyotic component of the disease and the integrity of uterotubal transport capacity was evaluated by HSSG.

Main outcome measures Influence of adenomyosis on uterotubal transport capacity in endometriosis.

Results In 35 of the 41 subjects (85%) with endometriosis, signs of adenomyosis were detected using T2-weighed MRI. Two of six (33%) subjects with no adenomyosis (group I) showed dysperistalsis and hyperperistalsis, compared with 14 of 24 (58%) women with focal adenomyosis (group II) and 10 of 11 (91%) women with diffuse adenomyosis (seven showed a failure in transport capacity and two contralateral transport).

Conclusions Our data suggest that endometriosis is associated with impeded hyperperistaltic and dysperistaltic uterotubal transport capacity. However, adenomyosis is of even more importance, especially when diffuse adenomyosis is detected. Both forms of adenomyosis are commonly found in subjects with mild to moderate endometriosis. We suggest that the extent of the adenomyotic component in subjects with endometriosis explains much of the reduced fertility in subjects with intact tubo-ovarian anatomy.

Keywords Adenomyosis, endometriosis, hysterosalpingoscintigraphy, infertility, magnetic resonance imaging, uterotubal transport.

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Introduction

Recent findings have suggested new pathomechanisms of endometriosis and adenomyosis, as both can be regarded as variants of the same disease—a dislocation of basal endometrium.[1] The endometrium of endometriotic and adenomyotic lesions mimic the cyclical changes of the basal endometrium. It has been suggested that adenomyosis results from the infiltration of basal endometrium into myometrial dehiscences which are caused by uterine hyperperistalsis. Structural uterine wall abnormalities in T2-weighed magnetic resonance imaging (MRI) as well as in transvaginal ultrasonography have been detected in subjects with endometriosis. The muscular component of adenomyosis results secondarily from metaplasia of the infiltrating endometrial stroma.[2]

Directional sperm transport is significantly reduced in infertile women with pelvic endometriosis[3] and pregnancy rates are diminished due to this impairment.[4] These phenomena might be related to the destruction of the myometrial architecture by the spread of adenomyotic lesions. Reduced peristalsis in endometriosis can become disordered peristalsis (dysperistalsis) in the late follicular phase, and this is then associated with poor pregnancy rates.[4,5] Subjects with endometriosis show a higher frequency, amplitude and basal pressure tone compared with controls when intrauterine pressure measurements are made[6] and regularly demonstrate a retrograde (cervicofundal) contractility pattern when observed using vaginal ultrasound.[7] The technique of MRI has allowed new insights into structural uterine wall defects in adenomyosis. We therefore designed a study to examine whether uterotubal transport disorder and impeded sperm transport in subjects with laparoscopically proven endometriosis is caused by the endometriosis itself or by the adenomyotic component of the disease. Uterotubal transport was examined using hysterosalpingoscintigraphy (HSSG) and the extent of the adenomyotic disease by T2-weighed MRI.

Subjects and methods

Subjects

The 41 subjects studied were the subgroup of women in a larger study of 56 women with infertility associated with endometriosis, who agreed to have both HSSG and MRI (15 women declined to undergo MRI, mainly due to agoraphobia). The women were aged 25–39 years (mean: 33 years) and all gave informed consent. The study was approved by the university's local ethics committee. All subjects had a history of infertility and endometriosis diagnosed at laparoscopy. Most of the subjects were suffering from minimal or mild endometriosis ($n = 28$) and the rest from moderate to severe endometriosis ($n = 13$), according to the revised classification of the American Society of Reproductive Medicine (rAFS). In all subjects, patency of fallopian tubes was established by the passage of dye during laparoscopy.

HSSG and T2-weighed MRI were performed during the next menstrual cycle following laparoscopy. HSSG was performed only in the late follicular phase. Size and localisation of the dominant follicle at the day of HSSG were recorded.

According to the criteria of adenomyosis in T2-weighed MRI (see below), subjects were analysed in three groups: group I consisted of subjects with endometriosis without any evidence of adenomyosis, group II consisted of subjects with endometriosis and at least one area of focal adenomyosis and group III consisted of subjects with widespread diffuse adenomyosis. Subjects' age, grade of endometriosis and thickness of the 'junctional zone' (JZ) are shown in Table 1. The MRIs were all performed by the same experienced radiologist (S.Z.) and HSSG examinations were performed by two experienced specialists in nuclear medicine (N.H. and N.D.) at different sites. The analysis of the results of each of the investigations was carried out blind with respect to the other investigations.

HSSG

The HSSG was performed according to the method described by Iturralde and Venter,[8] Becker *et al.*[9] and specified by Kunz *et al.*[10] and Wildt *et al.*[11] In all 41 subjects, size and localisation of the dominant follicle were detected ultrasonographically at the day of HSSG, which were all performed in the Institute of Nuclear Medicine. The 20 MBq ^{99m}-Tc-marked macroalbuminaggregates (CIS Bio international, Gif-sur-Yvette, France) with a size of 5–20 μm, which imitates the size of sperm, were diluted with 2 ml 0.9% saline solution and then administered into the posterior vaginal fornix of the recumbent woman. Scans with a gamma camera were taken immediately after application and during various time intervals up to 30 minutes. For quantitative evaluation of HSSG, 'regions of interest' (ROIs) were determined in the area of both fallopian tubes in order to visualise the concentration of radioactivity in the area of the oviduct. By the means of ROIs, radioactivity can easily be localised to the uterine cavity or fallopian tubes. Taking into account the size and localisation of the dominant follicle, the results of HSSG were classified as follows:[4]

- Ipsilateral transport: concentration of radioactivity on the side of the dominant follicle.
- Transport on both sides: concentration of radioactivity equally distributed on both oviducts.

Table 1. Demographic data concerning age, stage of endometriosis (rAFS) and JZ thickness in MRI

	Group I	Group II	Group III	*P* value
Age, years (mean ± SD)	33.2 ± 2.9	32.4 ± 2.6	35.2 ± 4.3	n.s.
rAFS stage (mean ± SD)	1.7 ± 1.0	1.9 ± 1.0	1.9 ± 1.0	n.s.
JZ thickness, mm (mean ± SD)	3.2 ± 1.2	10.3 ± 3.1	11.2 ± 2.7	<0.001

n.s., not significant.
There was no difference concerning grade of endometriosis between the three groups, but JZ thickness differed significantly between group I and the groups of subjects with adenomyosis (focal; group II, diffuse; group III).

- Contralateral transport: concentration of radioactivity on the opposite side of the dominant follicle.
- No tubal transport (uterine cavity): concentration of radioactivity in the area of the uterine cavity without any further transport to the fallopian tubes.

Ipsilateral or both-sided transport can be summarised as physiologic uterotubal transport capacity, contralateral transport or no uterotubal transport in HSSG can be interpreted as pathophysiologic transport phenomena. In a previous publication, a healthy control group was established showing physiologic ipsilateral or bilateral uterotubal transport in 68% of cases and showing pathologic contralateral transport or a failure in transport in 32%.[4]

T2-weighed MRI

MRI was performed using a 1.5-Tessla unit (Magnetom Symphony™; Siemens, Erlangen, Germany) with the use of an anterior-body-phased array coil, with the subject in a supine position. After a gradient-echo localiser sequence had been performed to check the subject position, fast T2-weighted turbo spin-echo sequences were acquired (repetition time [TR]/echo time [TE] = 3500/100; 5.5-mm section thickness, 0.8-mm intersection gap, 512 × 256 matrix) at transverse and sagittal section orientations. Additional coronal T2-weighted fat suppressed spin-echo sequences (TR/TE = 2500/100; 5.5-mm section thickness, 0.8-mm intersection gap, 512 × 256 matrix) were acquired.

Supplementary transverse T1-weighted spin-echo imagings (TR/TE = 550/10; 5.5-mm section thickness, 0.8-mm intersection gap, 512 × 256 matrix) were performed. During the examination, no medications for suppression of peristaltic artefacts were administered.

The measurements of total myometrium and the diameters of the subendometrial myometrium (JZ) were performed in a midsagittal plane on the height of the transition between the upper and the lower half of the dorsal wall of the uterine corpus. The radiologic sign of the JZ is characterised by a three-fold increase in nuclear area and decreased extracellular matrix per unit volume on the cellular level. The increased nuclear area reflects both a higher smooth muscle density and an increased nucleocytoplasmatic ratio of the monocytes.[12] Several studies since then have demonstrated MRI to be highly accurate compared with histological findings in the diagnosis of adenomyosis with a sensitivity and specificity of 86–100% and an overall accuracy of 85–90.5%.[13,14] The predominant lesion of adenomyosis at MRI consists of a low signal-intensity area on T2-weighed images, which frequently enlarges the JZ in a diffuse or focal pattern. These areas of low signal intensity have been shown to correspond to the smooth muscle hyperplasia accompanying heterotopic endometrial tissue. There is still a continuing debate about the cutoff value for JZ thickness to differentiate subjects with adenomyosis from those without. In the first publications,[15,16] a JZ thickness greater than 5 mm was believed to be diagnostic for adenomyosis, whereas a thickness of 3–5 mm was considered indeterminate. In another study,[14] it was reported that when the JZ thickness was 12 mm or greater, adenomyosis was diagnosed with a high degree of accuracy. By contrast, a maximal JZ thickness of 8 mm or less allowed exclusion of the disease. In subjects with a maximal JZ thickness of 8–12 mm, secondary findings such as relative thickening of the JZ in a localised area, poor definition of borders or high signal-intensity foci on T2-weighed images can be used to diagnose adenomyosis.[14] In another study,[17] an MRI diagnosis of adenomyosis was made with confidence when the JZ thickness was greater than 10 mm. According to these data, in the study presented here, findings were classified as adenomyosis when the maximal JZ thickness was 9 mm or greater in T2-weighed images. A maximal JZ thickness of 8 mm or less in the images was assessed as exclusion of the disease or subjects showed secondary findings like a localised thickening of the JZ, poor definition of borders or high signal-intensity foci.

Examples of both the focal and diffuse forms of adenomyosis in MRI are demonstrated in Figure 1.

Statistical analysis

Patient data were stored in a personal computer using Microsoft® Excel 2000 tables. Comparisons between the three groups regarding uterotubal transport capacity were calculated using the chi-square test. The Tukey–Kramer test was used for the comparison of demographic data. JZs are presented as median (25th percentile, 75th percentile). Comparisons within the patient groups concerning transport capacity and JZ thickness were performed using the nonparametric Wilcoxon–Whitney *U* test.

Significance was assumed when $P < 0.05$. All calculations were performed using GraphPad InStat® (GraphPad Software Inc., San Diego, CA, USA) and BIAS (Copyright Epsilon 1989–2002).

Results

In 29 of 41 investigated subjects (71%), definite signs of adenomyosis could be detected on MRI. Only 12 subjects (29%) showed a thickness of the JZ below 7 mm and did therefore not fulfil the criteria for the diagnosis of adenomyosis by the enlargement of the JZ of more than 8 mm. However, 6 of these 12 subjects (50%) had features suggestive of adenomyosis using secondary criteria such as a relative thickening of the JZ in a localised area, poor definition of borders or high signal-intensity foci. Thus, 35 of 41 studied subjects (85%) with endometriosis had features diagnostic or suggestive of adenomyosis uteri.

Subjects without any signs of adenomyosis (group I) showed physiologic uterotubal ipsilateral or bilateral transport capacity in four of six individuals (67%), while two/six subjects (33%) had pathophysiologic transport contralateral

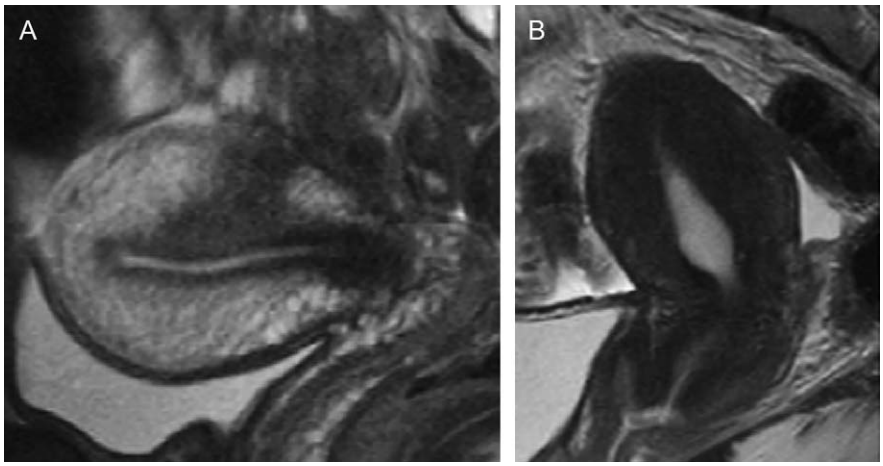

Figure 1. T2-weighed sagittal turbo spin-echo sequences of subjects with symptomatic endometriosis. (A) Woman with radiologic signs of focal adenomyosis of the posterior uterine wall involving innermost myometrial layers as well as subserosal layers. (B) Diffuse adenomyosis of the anterior and posterior uterine wall.

to the dominant follicle. There were no cases of a failure of transport (all HSSGs were positive).

In subjects with focal adenomyosis (group II), 10/24 subjects (42%) showed ipsilateral or bilateral intact transport, 8/24 subjects (33%) showed contralateral transport and in 6/24 subjects (25%) a failure in transport was observed.

In subjects with diffuse adenomyosis (group III), 8/11 subjects (73%) showed a negative HSSG, 2/11 subjects (18%) revealed contralateral transport and only 1/11 subjects (9%) showed intact, unimpaired uterotubal transport. These differences in uterotubal transport capacity in HSSG were significantly different ($P = 0.02$, Figure 2).

A typical example for a woman showing signs of diffuse adenomyosis in MRI and a failure of transport capacity in HSSG is demonstrated in Figure 3.

Although diffuse adenomyosis leads to failure in intact transport, in our study, contralateral transport or failure in transport were not significantly dependent on an increase of JZ's thickness per millimetre. These data were analysed using the Wilcoxon–Whitney test and are listed in Table 2.

For evaluation of radionuclides' transport velocity, the uterotubal ratio of the ROIs was analysed by focussing on the first scan after 30 minutes. In the subjects showing intact, strictly ipsilateral uterotubal transport, there was no positive

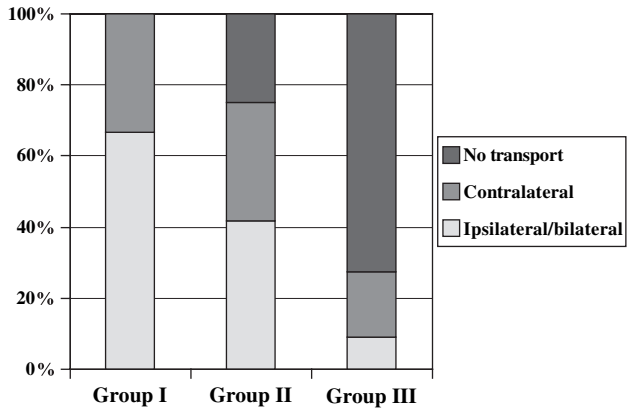

Figure 2. Uterotubal transport disorder in endometriosis (I), focal adenomyosis (II) and diffuse adenomyosis (III). Subjects of group I (endometriosis of the pelvis without signs of adenomyosis) demonstrate physiologic uterotubal transport in 67%, while 33% of the subjects show transport strictly contralateral to the dominant follicle. Failure in transport (negative HSSG) was not observed. In group II (endometriosis and focal adenomyosis), 42% of subjects showed physiologic ipsilateral or bilateral transport, 33% revealed contralateral uterotubal transport and in 25% of women a failure of transport could be detected. The proportion of pathophysiologic transport phenomena overweighs if diffuse adenomyosis is detected (group III); while 73% of the subjects demonstrate a failure in transport (negative HSSG), 18% of the subjects show contralateral and 9% ipsilateral intact transport mechanism ($P = 0.02$).

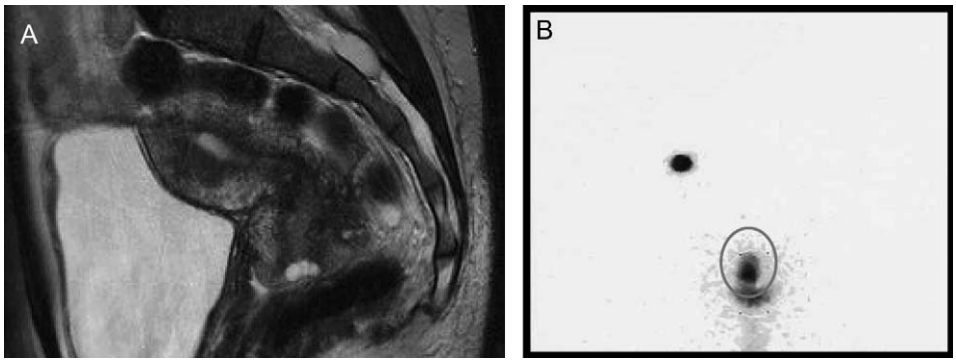

Figure 3. MRI in adenomyosis and HSSG. (A) A woman with diffuse adenomyosis and enlargement of the JZ >11 mm in T2-weighed images and endometriosis of the pelvis rAFS I. The woman suffered from primary infertility for 3 years. (B) No further uterotubal transport could be detected in HSSG. Radionuclides remain in the uterine cavity (mark). The spot above the uterine cavity is a defined mark on the women's right side.

correlation between thickness of the JZ and the velocity of radionuclides' transport ($r = 0.4$, 95% CI: –0.42 to 0.86).

In all the three groups, the mean value for the grade of endometriosis was between 1.6 and 1.9 (rAFS stage, Table 1, n.s.). Focal or diffuse forms of adenomyosis can therefore be observed in mild to moderate forms of endometriosis and are not correlated to a particular stage of pelvic endometriosis.

Discussion

With the improvement of high-resolution imaging techniques such as MRI and transvaginal sonography (TVS), uterine wall architecture can be assessed *in vivo*. TVS scans show more interindividual variability than MRI scans. The value of MRI to detect endometriosis is, however, still a matter of debate: although preoperative MRI detected fewer endometriotic lesions of the pelvis than laparoscopy or histopathology,[18] obliteration of the cul-de-sac can be imaged with high accuracy via the MRI jelly method.[19] By contrast, the method of T2-weighed MRI to diagnose adenomyosis is much more reliable. The radiologic sign of the JZ is characterised by a threefold increase in nuclear area and decreased extracellular matrix per unit volume on the cellular level. The increased nuclear area reflects both a higher smooth muscle density and an increased nucleocytoplasmatic ratio of the monocytes.[12] Several studies have demonstrated MRI to be highly consistent with the gold standard of histology in the diagnosis of adenomyosis, with a sensitivity and specificity of 86–100% and an overall accuracy of 85–90.5%.[13,14] The predominant lesion of adenomyosis at MRI consists of a low signal-intensity area on T2-weighed images, which frequently enlarges the JZ in a diffuse or focal pattern. There is still a continuing debate about the cutoff value for JZ thickness to differentiate subjects with adenomyosis from those without. In initial publications,[15,16] a JZ thickness greater than 5 mm was believed to be diagnostic for adenomyosis, whereas a thickness of 3–5 mm was considered indeterminate. In another study,[14] it was reported that when the JZ thickness was 12 mm or greater, adenomyosis was diagnosed with a high degree of accuracy. By contrast, a maximal JZ thickness of 8 mm or less allowed exclusion of the disease. In subjects with a maximal JZ thickness of 8–12 mm, secondary findings such as relative thickening of the JZ in a localised area, poor definition of borders, or high signal-intensity foci on T2-weighed

Table 2. Uterotubal transport capacity depending on JZ thickness

	Ipsilateral and bilateral	Contralateral	No transport	*P* value
n	15	12	14	
JZ thickness (mm)				
Minimum	2.0	4.0	7.0	Group I:II = 0.9, n.s.
Maximum	17.0	16.0	15.0	Group I:III = 0.1, n.s.
Median	9	9.5	11.0	Group II:III = 0.1, n.s.
25th percentile	3.0	6.0	9.0	
75th percentile	12.0	11.0	14.0	

n.s., not significant.
Wilcoxon–Mann–Whitney test did not reveal statistical significance comparing the integrity of uterotubal transport depending on the absolute thickness of the JZ in millimetre.

images was used to diagnose adenomyosis.[14] In another study,[17] an MRI diagnosis of adenomyosis was made with confidence when the JZ thickness was greater than 10 mm. In keeping with these data, in our study, findings were classified as adenomyosis when the maximal JZ thickness was 9 mm or greater in T2-weighed images. A maximal JZ thickness of 8 mm or less in the images was assessed as exclusion of the disease.

Our data suggest that endometriosis leads to a significant restriction in uterotubal transport capacity. Uterine hyperperistalsis and dysperistalsis in endometriosis as a dysfunction of the mechanism of rapid sperm transport has been reported before,[3] but our data additionally show that subjects with endometriosis and early adenomyosis start to develop hyperperistalsis reflected by an increase of contralateral tubal demonstration. Dysperistalsis dominates in subjects with endometriosis and diffuse adenomyosis proven by no further tubal transport (negative HSSG).

While in subjects with endometriosis and a single focus of adenomyosis predominantly hyperperistalis with contralateral sperm transport prevails, diffuse thickening of the JZ leads to failure of uterotubal transport capacity. It is known from earlier studies that both the transport phenomena (contralateral and no further transport) can be classified as pathologic uterotubal transport.

In our study, we were not able to show that the transport capacity or velocity of transport decreased in direct correlation to the expansion of the JZ per millimetre, but transport phenomena were significantly different when focal or diffuse adenomyosis were diagnosed.

In our understanding of the disease of endometriosis, dysperistalsis is almost certainly caused by the diffuse adenomyotic component of the woman suffering from endometriosis. Uterotubal transport disorder as proven by negative HSSG is associated with very low pregnancy rates,[5,11] while the integrity of rapid sperm transport through the female genital tract can well be documented by the means of HSSG.[10,11] A massive restriction of the integrity of the uterotubal transport capacity in correlation with low pregnancy rates can be observed in endometriosis,[4] which is increased further when adenomyosis is diagnosed.

The impact of unimpaired tubo-ovarian anatomy and function in low-grade endometriosis and its influence upon infertility and subfertility is still controversial. Medical or surgical eradication of peritoneal lesions does not improve[20–22] or normalise fertility[23] in women with peritoneal endometriosis alone without any tubo-ovarian pathology. This suggests that peritoneal endometriosis might not serve as a major factor causing subfertility and infertility in these women. Our data suggest that most of the subjects with endometriosis develop adenomyosis even in its mild or moderate form, i.e. stages I or II of the disease. This might serve as an explanation for infertility in women with only minimal peritoneal endometriosis and intact uterotubal anatomy.

In another study, the most striking observation was that the mean diameter of the posterior JZ was significantly larger in women with endometriosis and fertile partners than those with subfertile or infertile partners. Similarly to our presented data, enlargement of the JZ was independent of the grade of endometriosis and the age of the subjects. Consistent with our data, the authors found a prevalence of adenomyosis in subjects with endometriosis in MRI scans of up to 90%.[24] Since the subjects were young (mean: 32.5 years) and suffering from infertility, MRI findings were not correlated to histological findings.

Our findings are supported by the findings that basal endometrium mimics the cyclical changes in estradiol and progesterone receptors of endometriotic and adenomyotic tissue, whereas functional endometrium and myometrium reacts completely differently.[1] Since the proportion of basal endometrium with stem cell potential is significantly increased, the observation might be explained by the observation that intrapelvic injection of menstrual endometrium causes pelvic endometriosis in the baboon model.[25]

Other groups have previously reported remarkable changes in uterine peristalsis associated with endometriosis. It has been reported that endometriosis is associated with an increase of intrauterine pressure,[26] with higher frequency, amplitude and basal tone of contractions, especially during menstruation.[6] These phenomena might equally be explained by the developing adenomyotic component of the disease.

Our data strongly suggest that adenomyosis and endometriosis are variants of the same disease with uterine hyperperistalsis and dysperistalsis being of critical importance.

As dysperistalsis in endometriosis, reflected by negative HSSG, has been proven generally to be associated with poor pregnancy rates even in subjects with normozoospermia of their partners, the additional performance of MRI allows completely new insights into changes of the uterine wall architecture caused by the adenomyotic process.

Since there was no positive correlation between the severity of pelvic endometriosis and the severity of adenomyosis in our subjects, we recommend MRI scans even in subjects with mild or moderate forms of endometriosis. Histological findings confirm MRI findings concerning adenomyosis in 85–90.5% of women.[14]

The pathomechanism of endometriosis and adenomyosis as variants of the same unique disease process probably explains the high prevalence of adenomyosis in subjects with endometriosis.

In combination with HSSG, our data suggest impeded hyperperistaltic sperm transport in endometriosis and focal adenomyosis, while uterine transport results in dysperistalsis when diffuse adenomyosis is diagnosed. The diagnosis of diffuse adenomyosis is significantly associated with dysperistalsis.

As dysperistalsis, reflected by negative HSSG, is associated with poor pregnancy rates, this examination method seems to be the only way to evaluate the integrity of a woman's uterotubal sperm transport capacity.[4] Especially in endometriosis and adenomyosis, infertility in mild to moderate forms of the disease can therefore be well understood by the means of HSSG.

According to our data, in subjects with laparoscopically proven endometriosis, the combination of T2-weighed MRI and HSSG allows completely new insights into the pathomechanisms of infertility in endometriosis and adenomyosis. ■

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