Endometriosis

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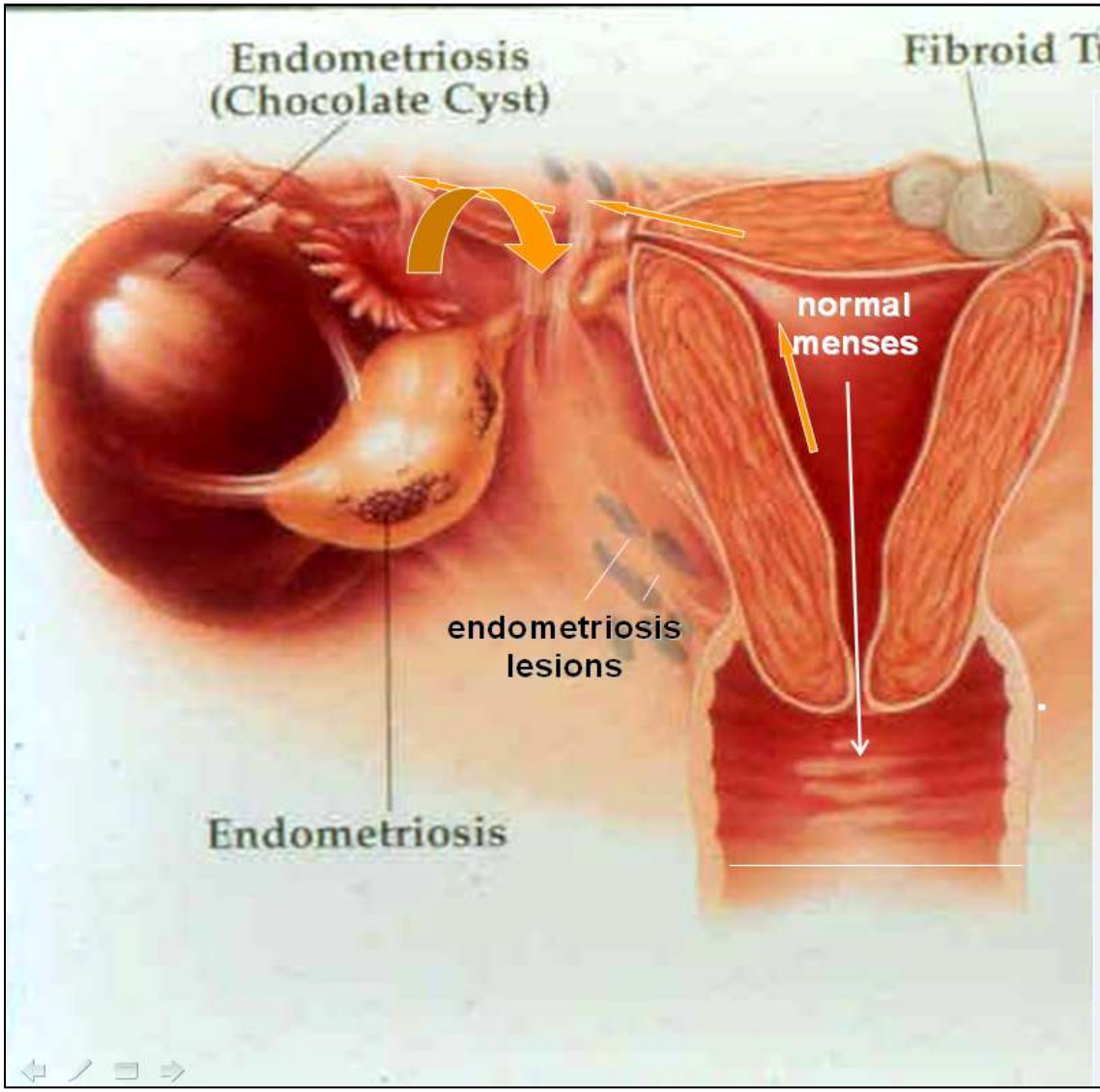
Background

- 6-10% of women of reproductive age:
  - Asymptomatic - 2 to 50%
  - Dysmenorrhea – 50 - 60%
  - Subfertility - up to 50%
- Functional endometrial glands and stroma in sites outside the uterine cavity
- Diagnosis may be delayed by up to 8 years

Etiology



Proposed Etiologies:

Retrograde menstruation/transplantation
Genetic predisposition
Immune dysfunction
Environmental triggers

Basic abnormality in eutopic endometrium - a proposed mechanism for infertility

Increasing evidence for "progesterone resistance".

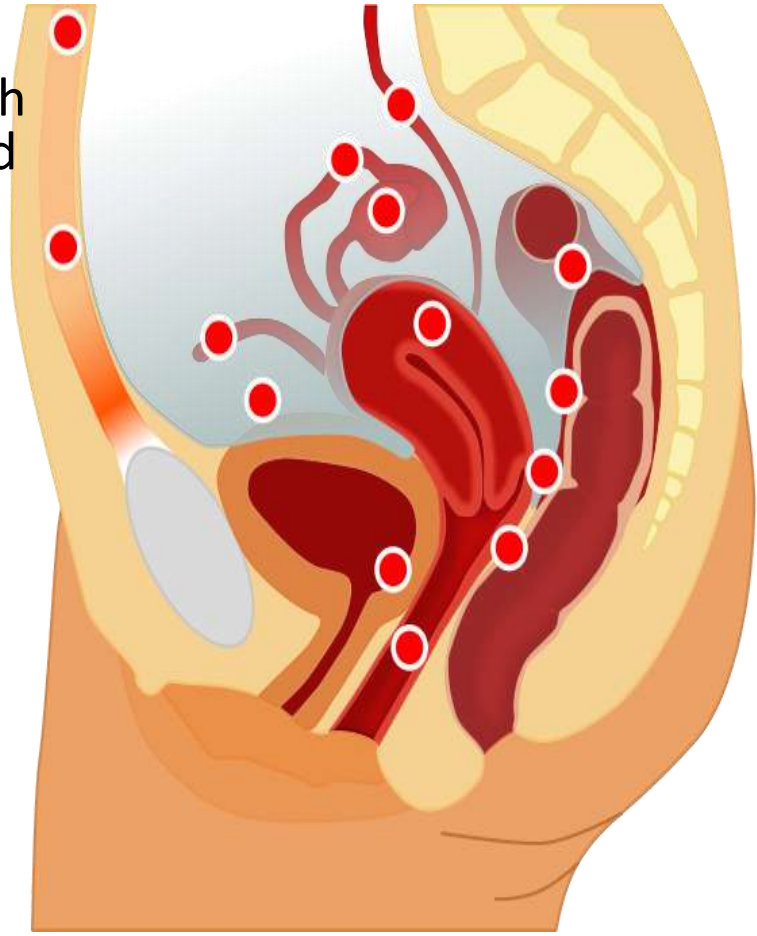
aromatase
Hox A 10,11
MMP-3,7-,11
EBAF (Lefty A)
$PR_A >> PR_B$
HGF

Pathogenesis

- Retrograde menstruation
- Implantation on peritoneal surfaces
- Inflammatory response
- Angiogenesis, adhesions, fibrosis, scarring, neuronel infiltration
- Anatomic distortion
- Pain and infertility

Theories of pathogenesis

- Retrograde menstruation (*Sampson's Theory*)
  - Endometrial fragments transported through fallopian tubes at time of menstruation and implanted at intraabdominal sites
- Müllerian (Coelomic) metapalasia (*Meyer's Theory*)
  - Metaplastic transformation of pelvic peritoneum during embryonal organogenesis
- Lymphatic spread (*Halban's Theory*)
  - Substances released/shed from endometrium induce formation of endometriosis



Risk factors

- Obstruction of menstrual outflow (mullerian anomalies)
- DES exposure
- Prolonged exposure to endogenous estrogen (early menarche, late menopause, or obesity)
- Short menstrual cycles
- Low birth weight
- Exposure to endocrine-disrupting chemicals
- Genetic component
- Consumption of red meat and trans fat

Protective factors

- Eating fruits, green vegetables, and Omega 3
- Prolonged lactation
- Multiple pregnancies

Associations

- Autoimmune diseases: IBD, MS, Fibromyalgia
- Ovarian endometrioid and clear cell cancers
- Other cancers: non-Hodgkin lymphoma and melanoma

Genetics

- Genetic predisposition:
  - low progesterone levels may be genetic
  - 10-fold increased incidence in women with an affected first-degree relative
  - Familial clustering in animal model – Rhesus monkeys
- Series of multiple hits within target genes
- Individual genomic changes:
  - Changes in chromosome 10 at region 10q26
  - Changes in the 7p15.2 region

Environmental factors

- Plastics and cooking with certain types of plastic containers with microwave ovens
- Dioxin exposure - 79% of monkeys developed endometriosis after receiving doses of dioxin
- Pesticides and hormones in our food cause a hormone imbalance
- The risk of endometriosis has been reported to be reduced in smokers (decreased estrogens)

Dioxin

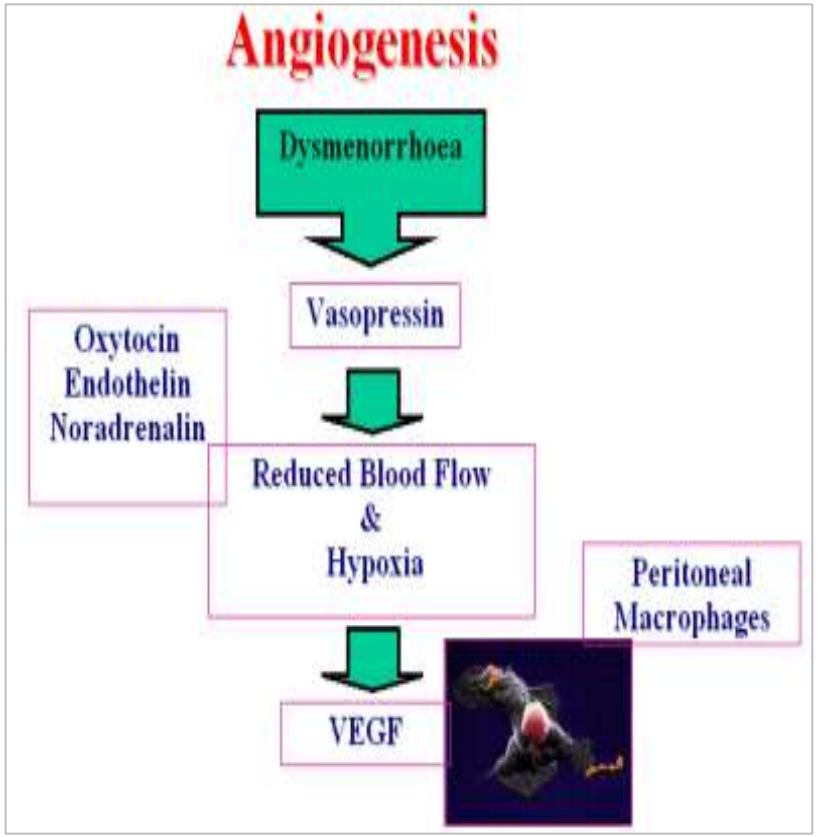
- Potent chemical toxin
- Reference compound for a large class of halogenated aromatic hydrocarbons
- 95% - incinerators burning chlorinated wastes
  - Dioxin pollution is also affiliated with paper mills, which use chlorine bleaching in their process and with the production of Polyvinyl Chloride (PVC) plastics
- The major sources of dioxin are in the diet:
  - 97.5% - meat and dairy products

Cancer biology

- Cellular proliferation and invasion
- T cell mediated invasion may be similar to that which occurs with metastatic neoplasia - immune surveillance systems are inadequate or unable to respond to the seeding tissue
- Accumulation of various growth factors and the occurrence of angiogenesis to produce a self contained blood supply are the features that implicate the relationship of cancer biology and ectopic endometriotic tissue development.

Angiogenesis

- The endometrium of women with endometriosis has an increased capacity to proliferate, implant and grow in the peritoneal cavity
- May enter a blood or lymph vessel and disseminate to distant body sites
- Endometrium is a rich source of growth factors which promote angiogenesis including the fibroblast growth factors, FGF1 and FGF2 and the vascular endometrial growth factor (VEGF)



Apoptosis

- Programmed cell death is impaired in endometriosis
- Decreased apoptosis in endometriosis cells may help an activated immune system to establish ectopic foci of disease

Immunologic dysfunction

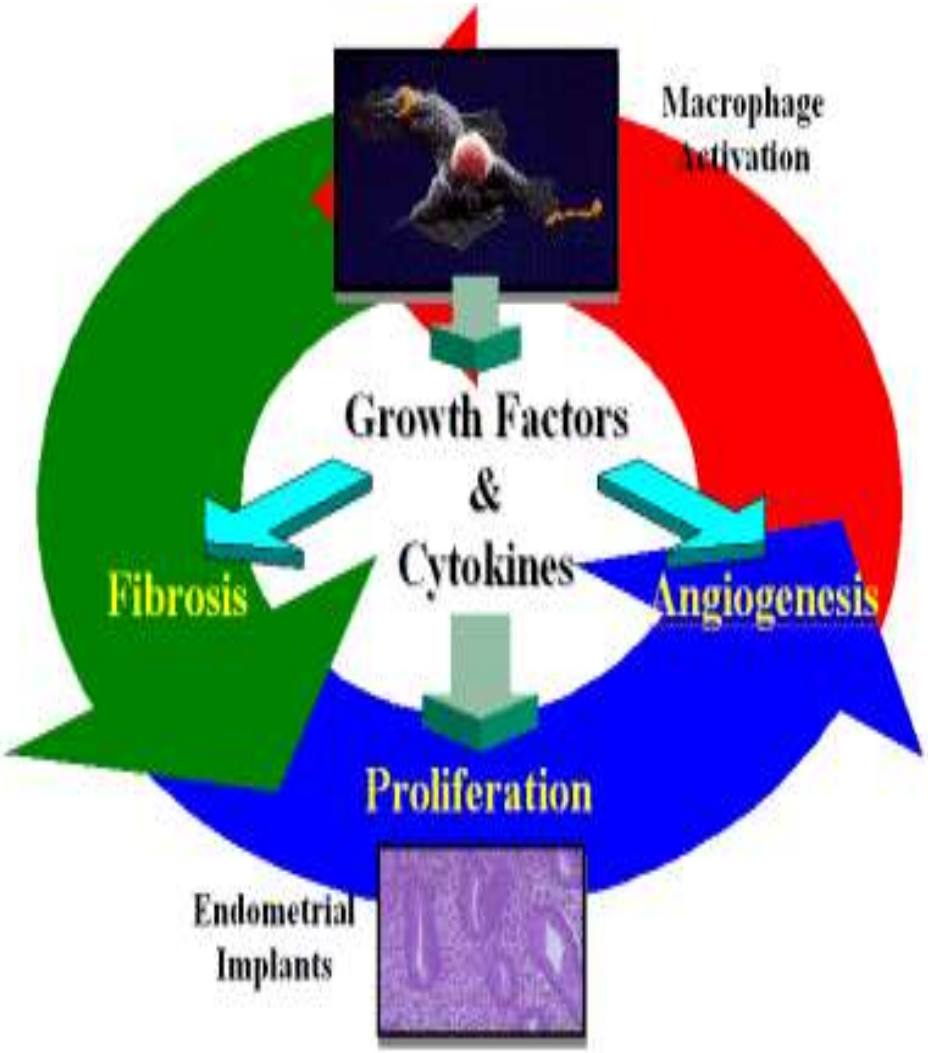
- Altered immune response to the displaced endometrial tissue
- Increased humoral immune responsiveness and macrophage activation
- Diminished cell-mediated immunity with decreased T-cell and natural killer cell responsiveness
- Humoral antibodies to endometrial tissue have also been found in sera of women with endometriosis (autoantibodies)

Altered macrophage function

- Macrophages secrete high concentration of substances such as growth factors that restrict natural killer activity, increase angiogenesis and fibrosis and induce endometrial cell proliferation in vitro
- These changes in peritoneal milieu could also be responsible for the failure of fertilization, embryo development and implantation

Paracrine changes in peritoneal fluid

- Macrophage derived substances such as prostanoids, cytokines, growth factors and angiogenic factors have been detected in the peritoneal fluid of women with endometriosis



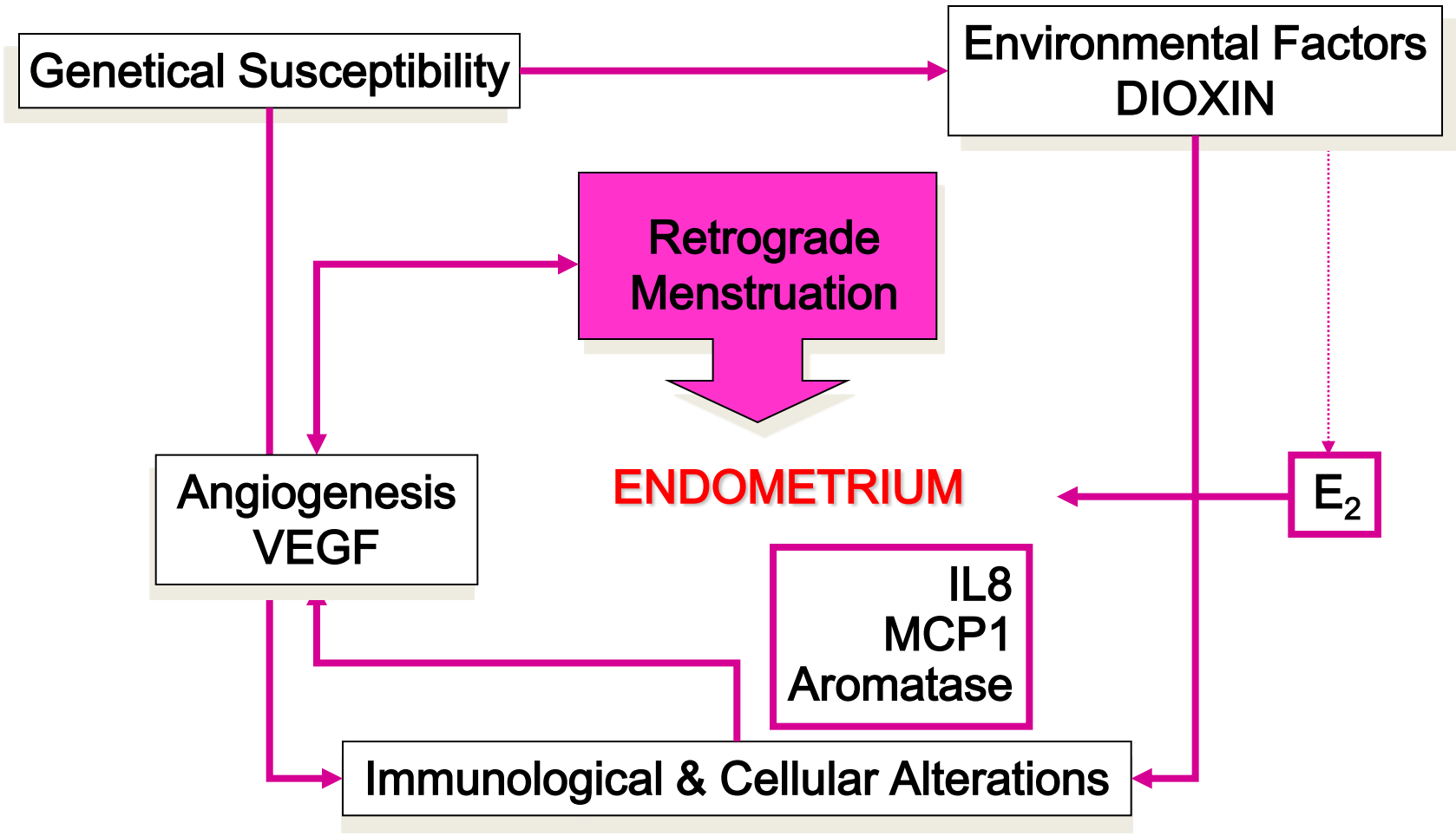
Paracrine changes

- Interleukin 8 (IL-8) is a chemoattractant and activating factor for human neutrophils and a potent angiogenic agent:
  - IL-8 concentrations in correlation with disease stage
  - Peritoneal macrophages play an important role in the initiation of the pathogenic cascade as sources of IL-1 and TNF-a in addition to IL-8
- Monocyte Chemotatctic Protein-1 (MCP-1)
  - Level significantly higher in patients with severe disease
  - Directly stimulating endometrial cell proliferation
- VEGF is a growth factor related to angiogenesis and released in response to hypoxia
  - Association between the retrograde menstruation and /or dysmenorrhoea and changes in peritoneal fluid
  - Activated macrophages in the peritoneal cavity produce large amount of VEGF

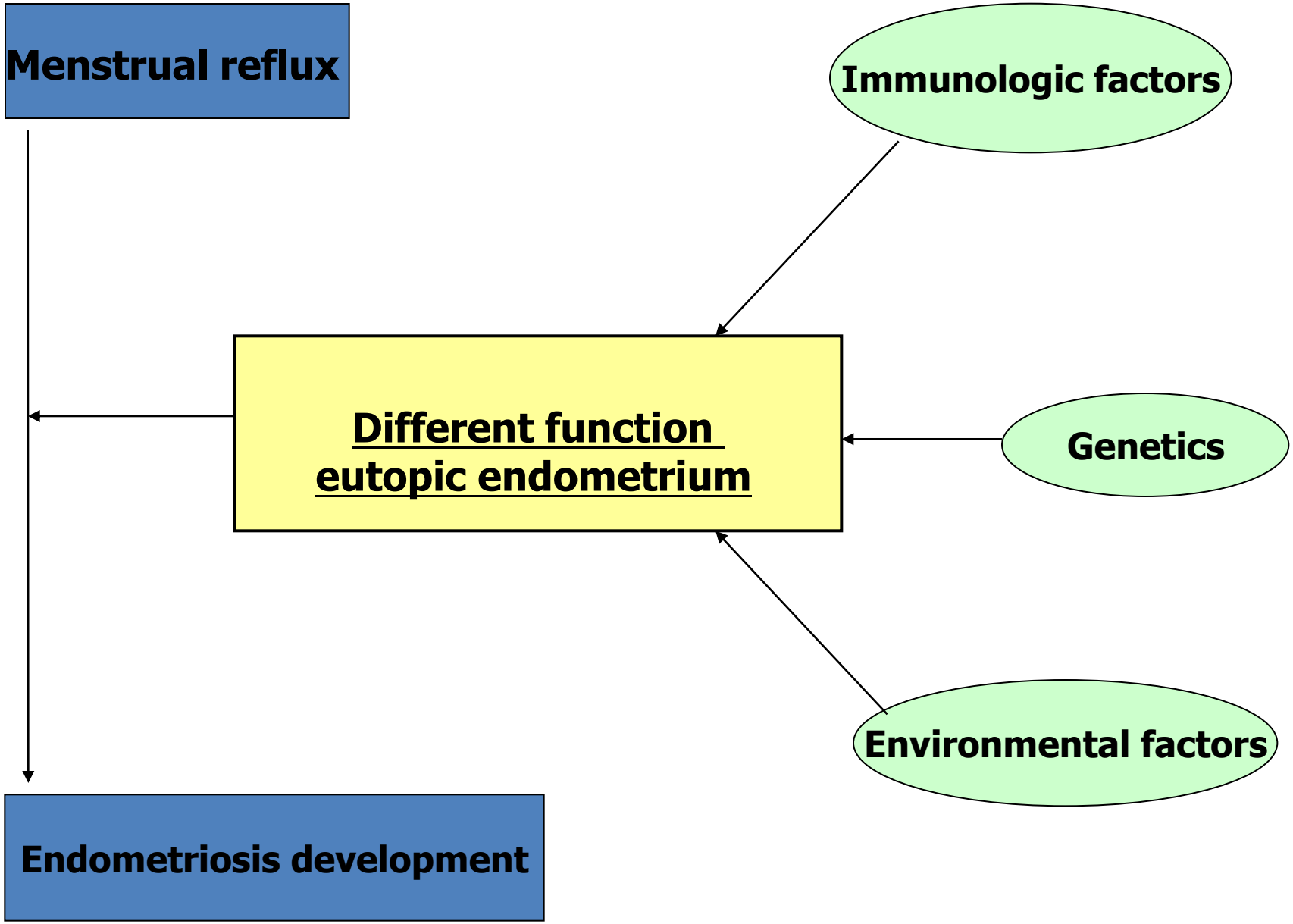
Summary of pathogenensis

- Multifactorial disease:
- Interaction between multiple gene loci and environment
- Causes of immune or inflammatory deficiency may be related to the effects of stress on immune functioning, or may be genetically determined
- Environmental factors such as Dioxin may be responsible for immunosuppressive activities and altered tissue specific responses to hormones
- Chronic immunosupression in combination with altered hormonal regulation may facilitate aberrant growth of endometrial tissue in the peritoneum
- The mechanism appears to require endometrium and retrograde menstruation in most cases of disease

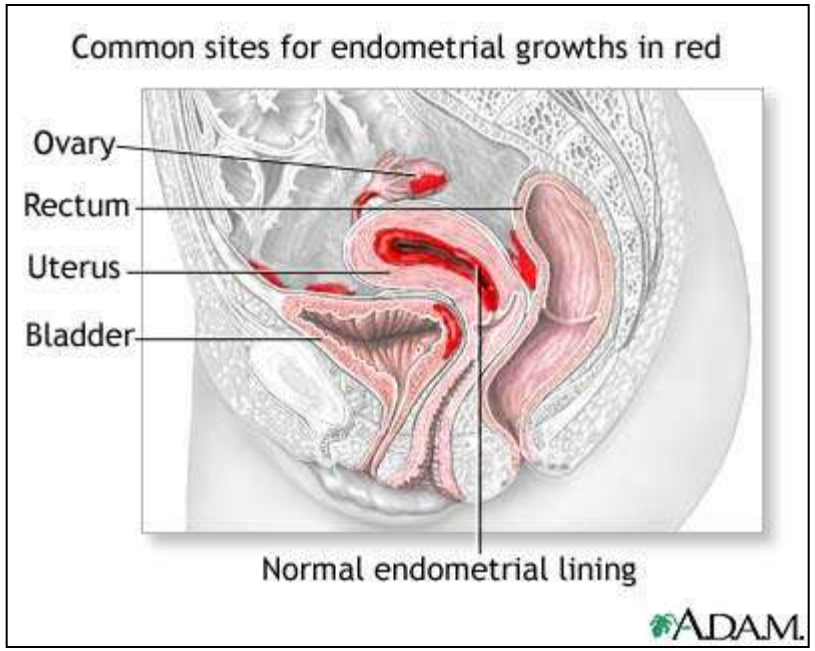
Pathogenesis

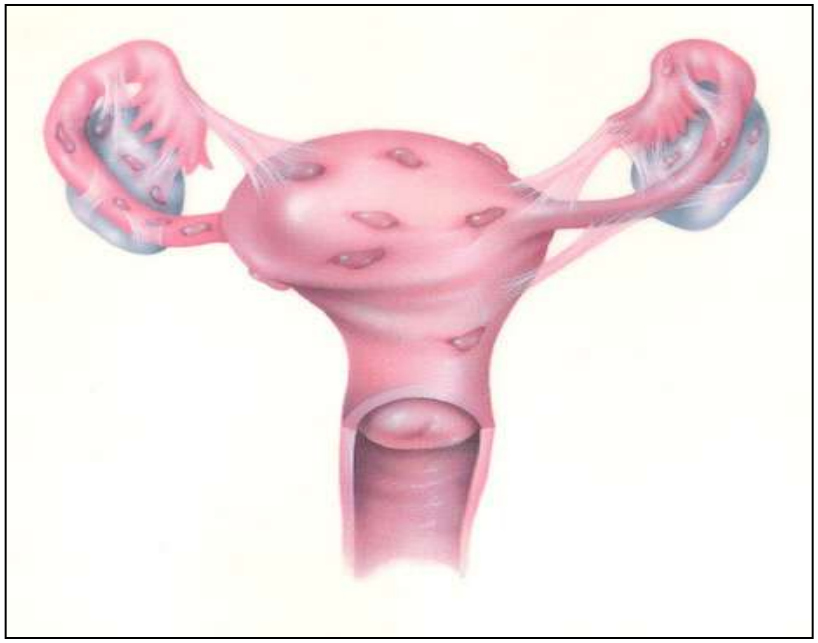


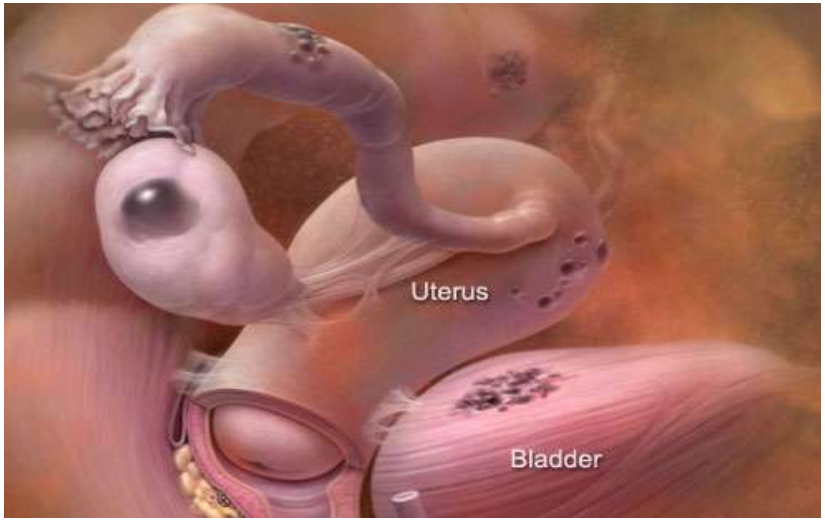
Etiology

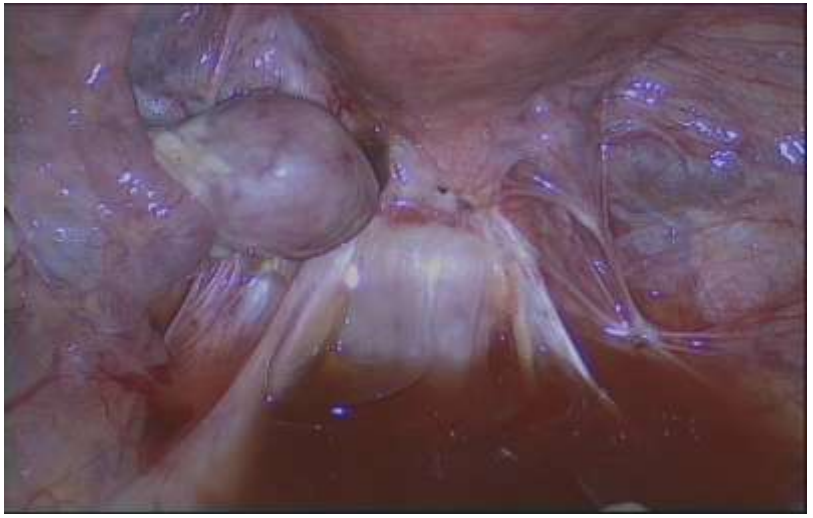

Menstrual reflux
Immunologic factors
Different function
eutopic endometrium
Genetics
Environmental factors
Endometriosis development

Disease locations









Etiology

- Peritoneal endometriosis
- Ovarian endometriosis
- Deep endometriosis

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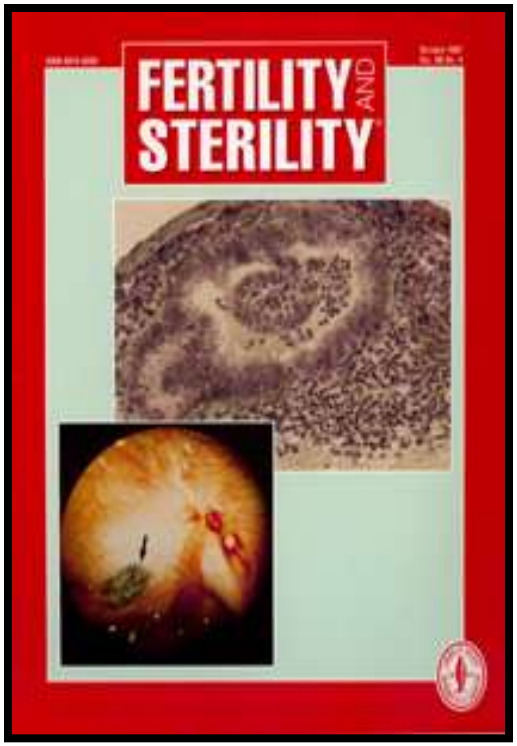


3 DIFFERENT ENTITIES

Diagnosis

- History
- Symptoms
- Clinical findings
- Ca 125
- Imaging
- Laparoscopy

Symptoms

- Chronic pelvic pain ≥ 6 months
- Dysmenorrhea – 50-90%
- Dyspareunia
- Deep pelvic pain
- Lower abdominal pain
- Pain: intermittently throughout the menstrual cycle, or continuous. Dull, throbbing, or sharp, exacerbated by physical activity
- Dysfunctional Uterine Bleeding
- Urinary symptoms (IC)
- Gastrointestinal Symptoms (IBS, IBD)
- Infertility

Assessment of Pain in Endometriosis

- Linear scales
  - Verbal Rating Scale (VRS)
  - Numerical Rating Scale (NRS)
  - A visual analog scale (VAS)
- Multidimensional Verbal Rating Scales
  - A clinician devised four point scale: Biberoglu and Behrman

    Am J Obstet Gynecol 1981;139:645-54

Findings

- Pelvic mass
- Immobile pelvic organs
- Rectovaginal nodules
- Adnexal pain
- Local tenderness
- Uterosacral ligament nodularities

Ca 125

- Biomarker
- Source – epithelium of female reproductive tract, respiratory tract, ocular surface
- Endometrium and irritated peritoneum
- Limited specificity and sensitivity, especially in premenopause
- Elevated in: endometriosis, pregnancy, ovulation, menstruation, inflammatory conditions, PID, cirrhosis, diabetes, and various epithelial cancers

Treatment - Pain management

- Repeated courses of medical therapy, surgical therapy, or both
- Pain recurs 6-12 months after completion of treatment

Empirical medical therapy

- Minimizes inflammation
- Interrupts or suppresses cyclic ovarian hormone production
- Inhibits the action and synthesis of estradiol
- Reduces or eliminates menses

Empirical medical therapy

- NSAIDS
- OCT – first line - 20-25% failure rate
- Progestins - Medroxyprogesterone acetate
- Levonorgestrel IUD (Mirena) or PO (induces endometrial atrophy and associated amenorrhea)
- GnRH agonists (hypoestrogenic state, endometrial atrophy, and amenorrhea, requires addback therapy due to bone loss over 6 mo Rx)
- Aromatase inhibitors
- Danazol – severe androgenic effects

Complementary therapies

- Acupuncture
  - Cochrane - evidence of effectiveness without side effects
- TENS – short term management
- Traditional Chinese Medicine – TCM
- Vitamins B1, B6, E
- Magnesium
- Topical heat - no evidence
- Spinal manipulations – no evidence
- Behavioral interventions

Disease progression

- 17 to 29% of lesions resolve spontaneously
- 24 to 64% progress
- 9 to 59% are stable over a 12-month period

- Major cause of disability and compromised quality of life in women and teenage girls

Surgical therapy

- Excision, fulguration, or laser ablation of endometriotic implants on the peritoneum, excision or drainage or ablation of endometriomas, resection of rectovaginal nodules, lysis of adhesions, and interruption of nerve pathways
- RCT's - 6 months, laparoscopic ablation of endometriotic implants is 65% effective in reducing pain, as compared with a 22% rate of pain reduction associated with diagnostic laparoscopy alone

Surgical therapy

- Recurrence of pain requiring therapy - 30 to 60% within 6 to 12 mos
- Interruption of nerve pathways: Presacral neurectomy (removal of the nerve bundle within the boundaries of the interiliac triangle)
- TAH BSO - pain relief in 80 to 90% but recurs in 10% of the women within 1 to 2 years after surgery
- Postoperative HRT – combined (estrogen alone may stimulate growth of microscopic disease)

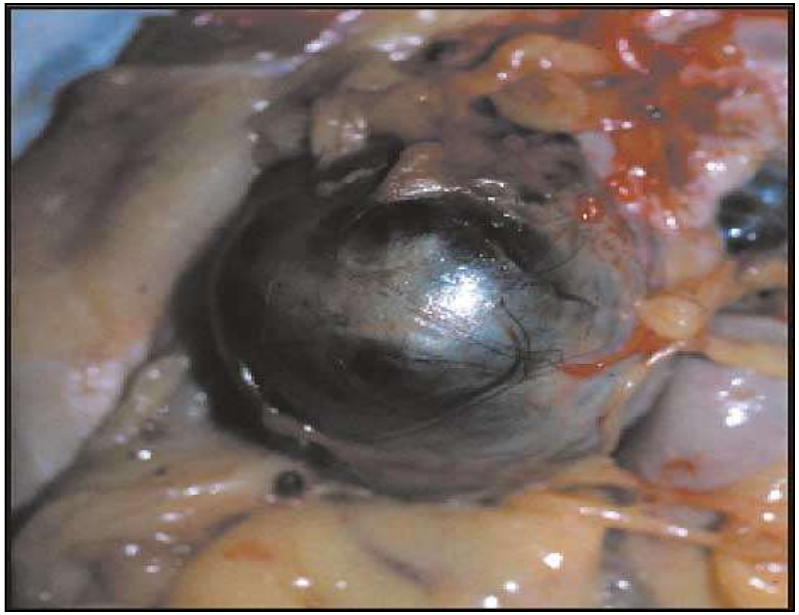
Diagnosis - surgical findings

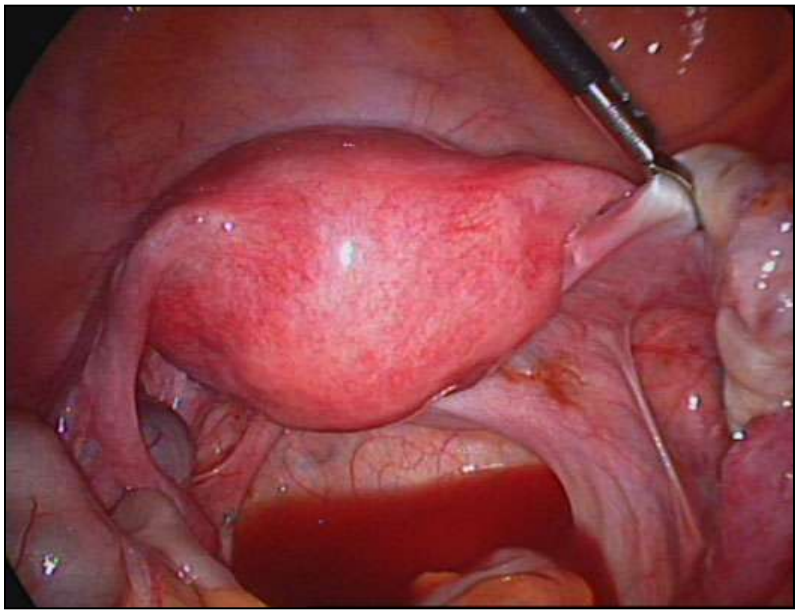
Red

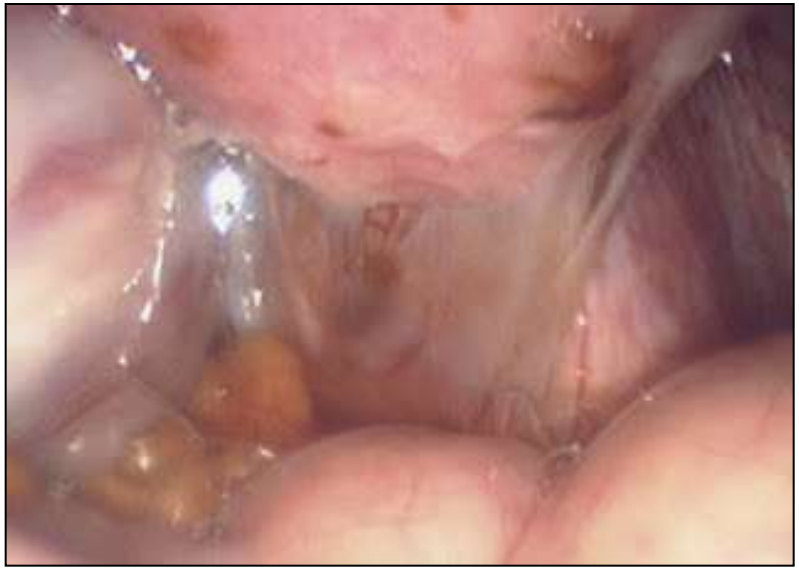
Black

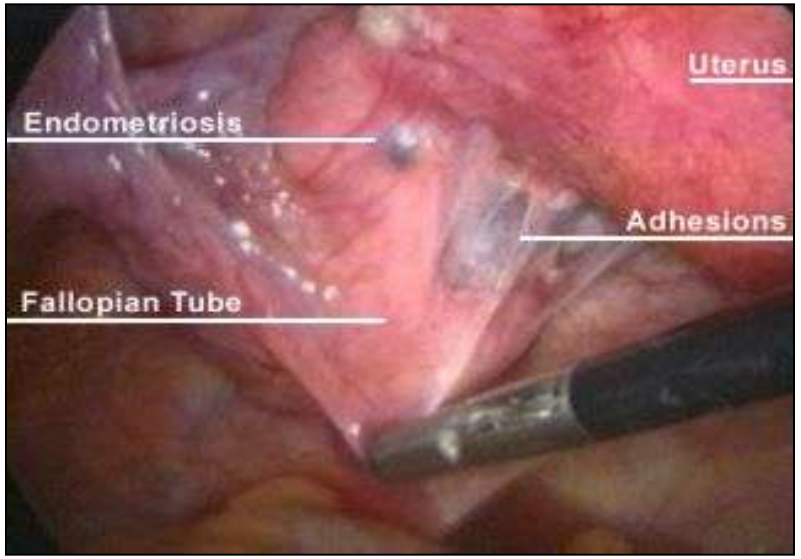
White

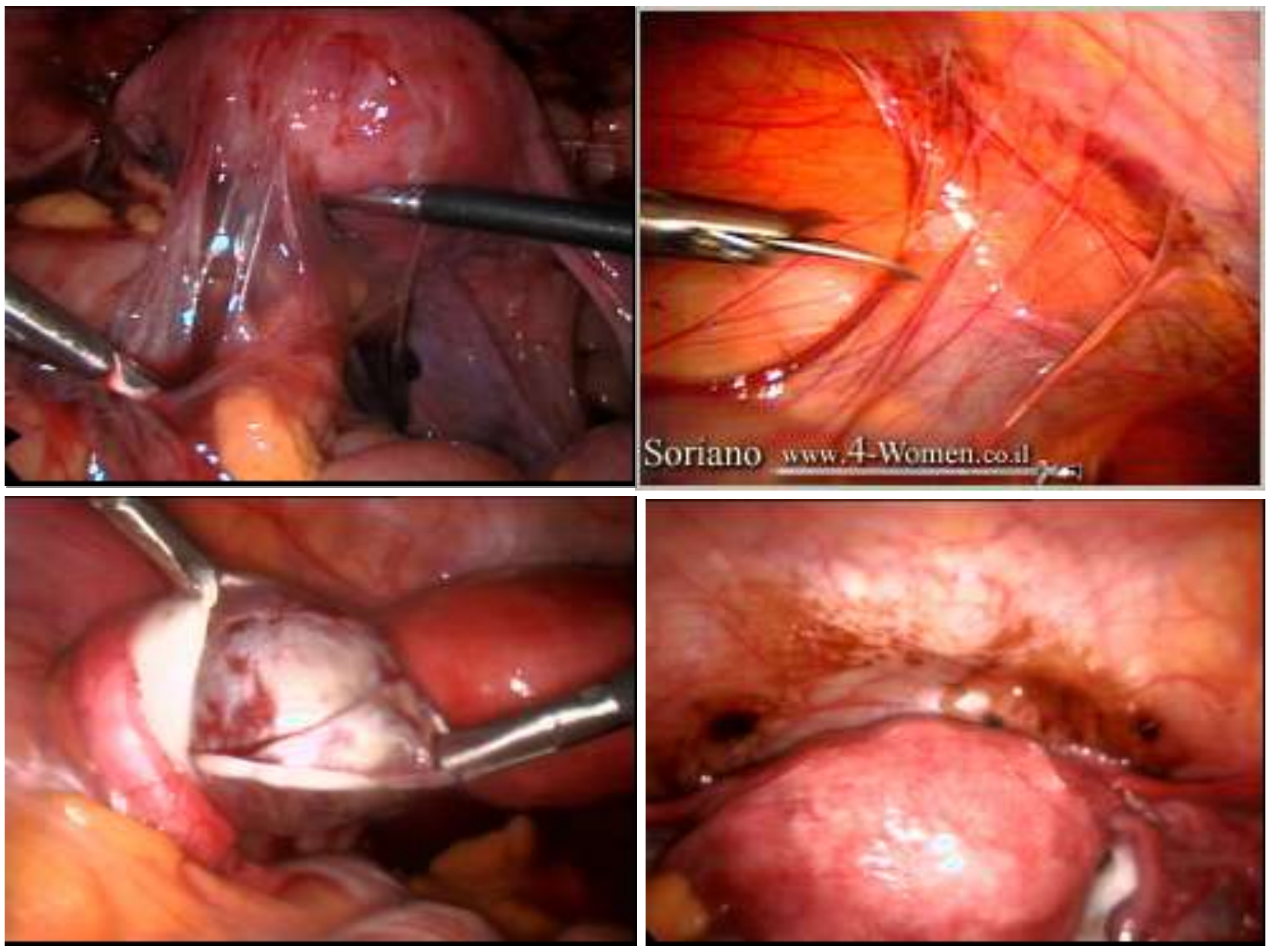
Diagnosis - surgical findings



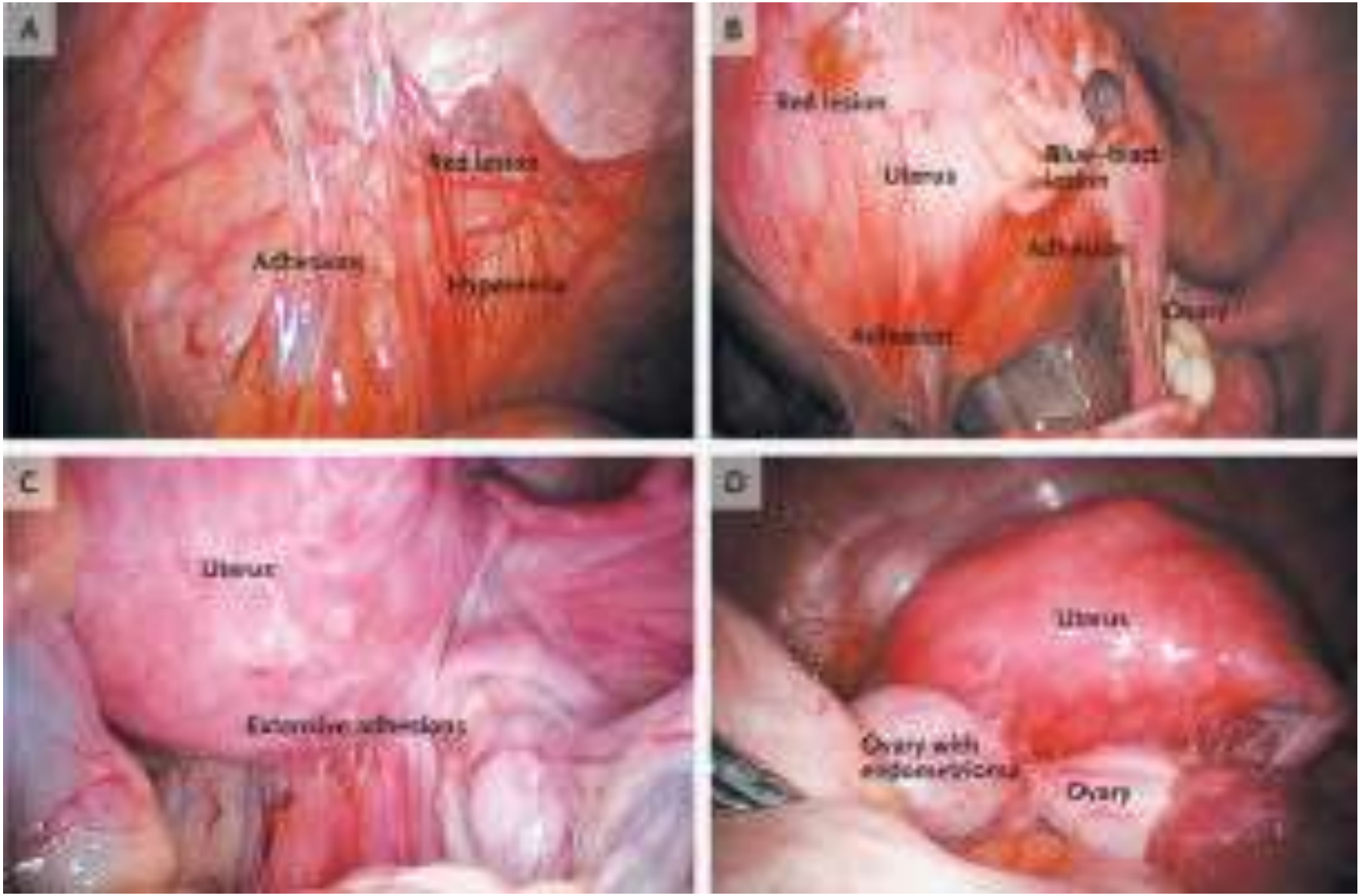




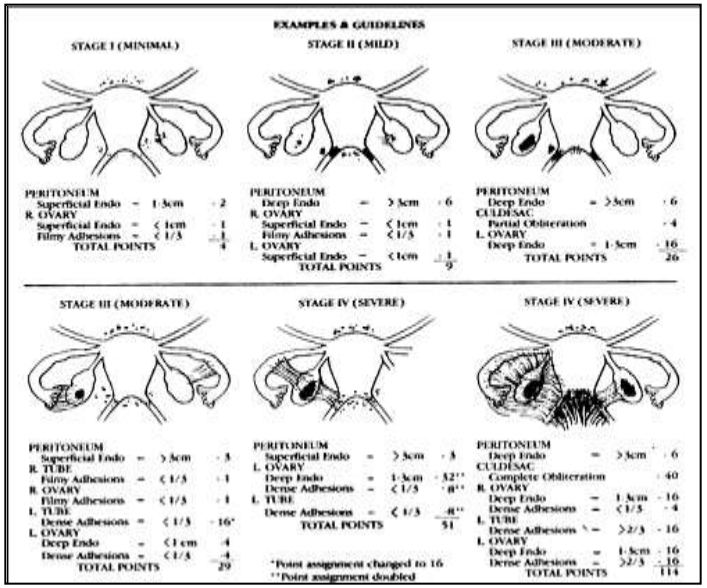


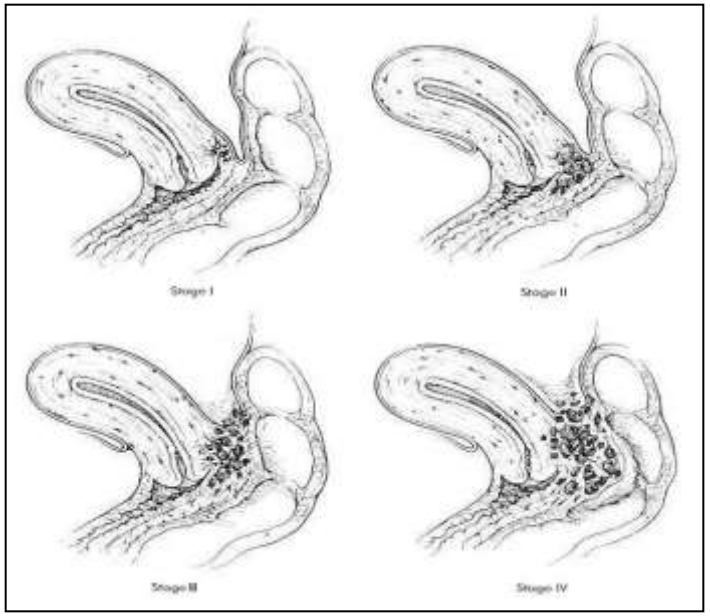

Soriano
www.4-Women.co.il

Diagnosis - surgical findings



Staging (AFS, revised ASRM)





AMERICAN SOCIETY FOR REPRODUCTIVE MEDICINE
REVISED CLASSIFICATION OF ENDOMETRIOSIS

Patient's Name _____ Date _____

Stage I (Minimal) - 1-5
Stage II (Mild) - 6-15
Stage III (Moderate) - 16-40
Stage IV (Severe) - > 40
Total _____

Laparoscopy _____ Laparotomy _____ Photography _____
Recommended Treatment _____
Prognosis _____

	ENDOMETRIOSIS	< 1cm	1-3cm	> 3cm
PERITONEUM	Superficial	1	2	4
	Deep	2	4	6
OVARY	R Superficial	1	2	4
	Deep	4	16	20
	L Superficial	1	2	4
	Deep	4	16	20

	POSTERIOR CULDESAC OBLITERATION	Partial	Complete
		4	40

	ADHESIONS	< 1/3 Enclosure	1/3-2/3 Enclosure	> 2/3 Enclosure
OVARY	R Filmy	1	2	4
	Dense	4	8	16
	L Filmy	1	2	4
	Dense	4	8	16
TUBE	R Filmy	1	2	4
	Dense	4*	8*	16
	L Filmy	1	2	4
	Dense	4*	8*	16

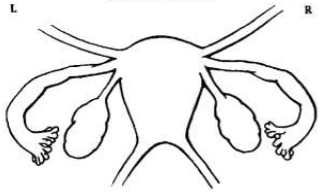
*If the fimbriated end of the fallopian tube is completely enclosed, change the point assignment to 16.

Denote appearance of superficial implant types as red [(R), red, red-pink, flamelike, vesicular blobs, clear vesicles], white [(W), opacifications, peritoneal defects, yellow-brown], or black [(B) black, hemosiderin deposits, blue]. Denote percent of total described as R___%, W___% and B___%. Total should equal 100%.

Additional Endometriosis: _____

Associated Pathology: _____

To Be Used with Normal Tubes and Ovaries



To Be Used with Abnormal Tubes and/or Ovaries

Vol. 67, No. 5, May 1997 **American Society for Reproductive Medicine** *Revised ASRM classification: 1996*

Source: Schorge JO, Schaffer JI, Halvorson LM, Hoffman BL, Bradshaw KD, Cunningham FG: *Williams Gynecology*: http://www.accessmedicine.com

Pain-Surgery vs. Medical

- Initial surgery superior with more severe disease
- No difference
  - Stage I-II endometriosis
  - Chronic Pelvic Pain
  - Previous surgery

Suggested approach to endometriosis-associated pain

- 1st line: continuous low-dose OCP with NSAIDs as needed
- 2nd line: progestins (start with oral dosing, consider switching to levonorgestrel intrauterine device or depo if well tolerated)
- 3rd line: GnRH agonist with immediate add-back therapy
- 4th line: repeat surgery, followed by 1, 2, or 3
- May consider low-dose (100–200 mg every day) danazol if other therapies poorly tolerated.

Mahutte and Arici, 2003

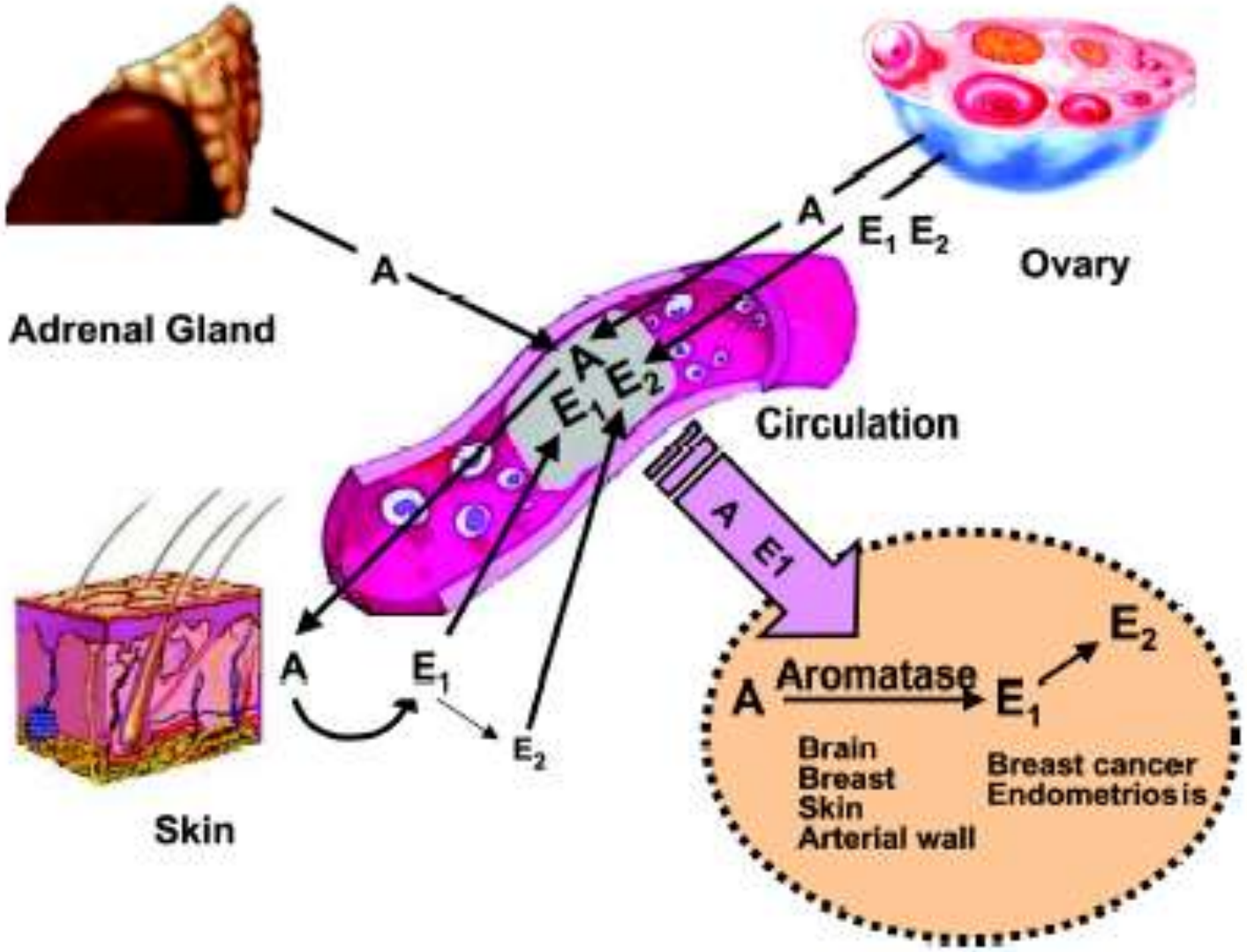
Experimental Treatments

- RU486 (mifepristone) and SPRMs
- GnRH antagonists
- TNF-α Inhibitors
- Angiogenesis Inhibitors
- MMP Inhibitors
- Immunomodulators
- Estrogen Receptor-β Agonists
- Aromatase Inhibitors

Aromatase (estrogen synthetase)

- Mediates the conversion of androstenedione and testosterone to estrone and estradiol.
- Aromatase enzyme has been demonstrated locally in endometriotic implants and a molecular etiology of endometriosis has been proposed
- The last step in steroid biosynthesis; therefore there are no important downstream enzymes to be affected.

AIs: complete inhibition in estrogen synthesis



Attar E and S.E. Bulun, Hum Reprod Update.2005; 0: 341

Management of infertility

- Gonadotropin therapy and intrauterine insemination
- In vitro fertilization (IVF)
- Cumulative pregnancy rates 4 cycles:
  - Intracervical insemination - 10%
  - Intrauterine insemination - 18%
  - Gonadotropin + intracervical insemination - 19%
  - Gonadotropin + intrauterine insemination - 33%
- Surgical:
  - Ablation of endometriotic lesions with lysis of adhesions
  - Excision of endometriomas ≥ 3 cm compared with drainage and ablation - significantly higher pregnancy rates
  - Ovarian surgery may diminish ovarian reserve in women with advanced disease

Management of infertility

- אנדומטריוזיס דרגה 1-2 – קונטרוברסיאלי האם גורם לאי פריון. טיפול כירורגי או תרופתי לא משפר פריון

- אנדומטריוזיס דרגה בינונית / חמורה :

1. אופציה ניתוחית – שיקום מבנה אנטומי. מאפשר ניסיון פריון ספונטני או עם טיפול פריון הגברת ביוץ.
2. אופציה טיפולית – הפניה לטיפול IVF ברובם דיכוי שחלתי ממושך לפני עם גלולות או GnRH.

Treatment of endometriosis

- גינקולוג
- אורולוג
- גסטרואנטרולוג
- כירורג כללי
- מומחה לכאב
- עו"ס
- דיאטנית
- פסיכולוג
- סקסולוג

Imaging and endometriosis

- Transvaginal ultrasonography
- Magnetic Resonance Imaging
- Rectal endoscopic ultrasound
- Helicoidal CT scan
- Rectosigmoidoscopy
- Barium enema (double contrast)

- Principles:
  - Make the most accurate pre operative diagnosis:
    - Keep number of additional investigations to minimum
    - Place emphasis on least costly, least invasive if comparably efficient (Chapron 2004)

Adenomyosis

Clinical manifestations

- Heavy menstrual bleeding
- Dysmenorrhea – 25% of women
- Chronic pelvic pain
- Symptoms develop between 40 - 50 years
- Menorrhagia may be related to the increased endometrial surface of the enlarged uterus
- Pain may be due to bleeding and swelling of endometrial islands confined by myometrium
- Approximately 1/3 of women are asymptomatic

Risk factors

- Advanced age but not only
- Multiparity
- Early menarche
- Obesity
- Previous uterine surgery or intervention

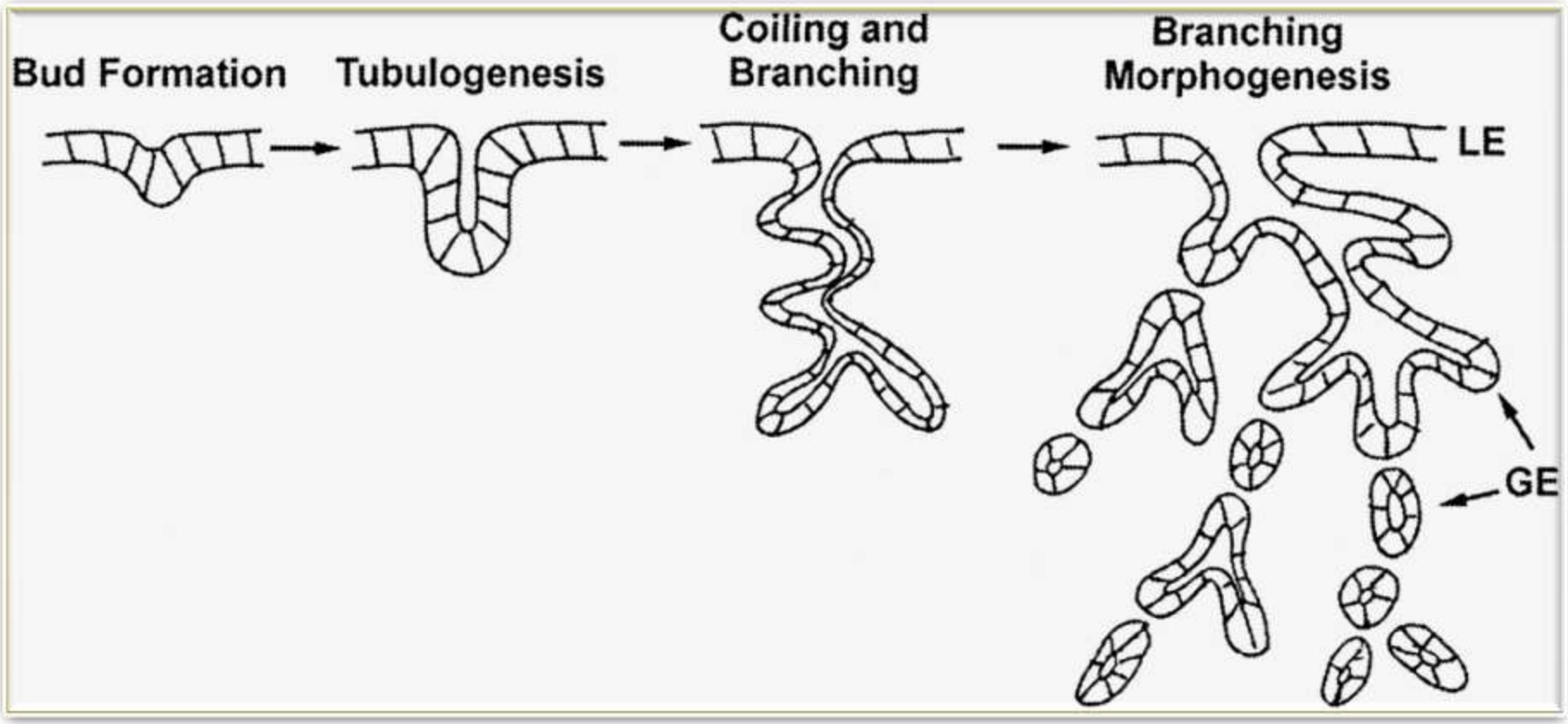
Pathological diagnosis of adenomyosis

- Previously reported prevalences vary widely (5-70%) depending on the definitions and the population studies
- Frequency of diagnosis of adenomyosis at hysterectomy 12% - 58% among 15 hospitals, and 10% - 88% among 25 pathologists
- Recent study showed adenomyosis in 25% of hysterectomies
- Associated with DIE

Pathogenesis of adenomyosis

- Uncoordinated proliferation of the inner myometrial cells - JZ hyperplasia may cause a focal or diffuse, thickened sub endometrial halo (JZ), which may be a sign of the muscular hypertrophy seen in adenomyosis
- JZ zonal hyperplasia may represent a pathological condition regardless of the presence or absence of adenomyotic foci
- Adenomyosis characterized primarily by disruption of the inner myometrial architecture and function
- Secondary infiltration of endometrial elements into the myometrium under certain circumstances of altered sex steroid milieu or altered local immunity.

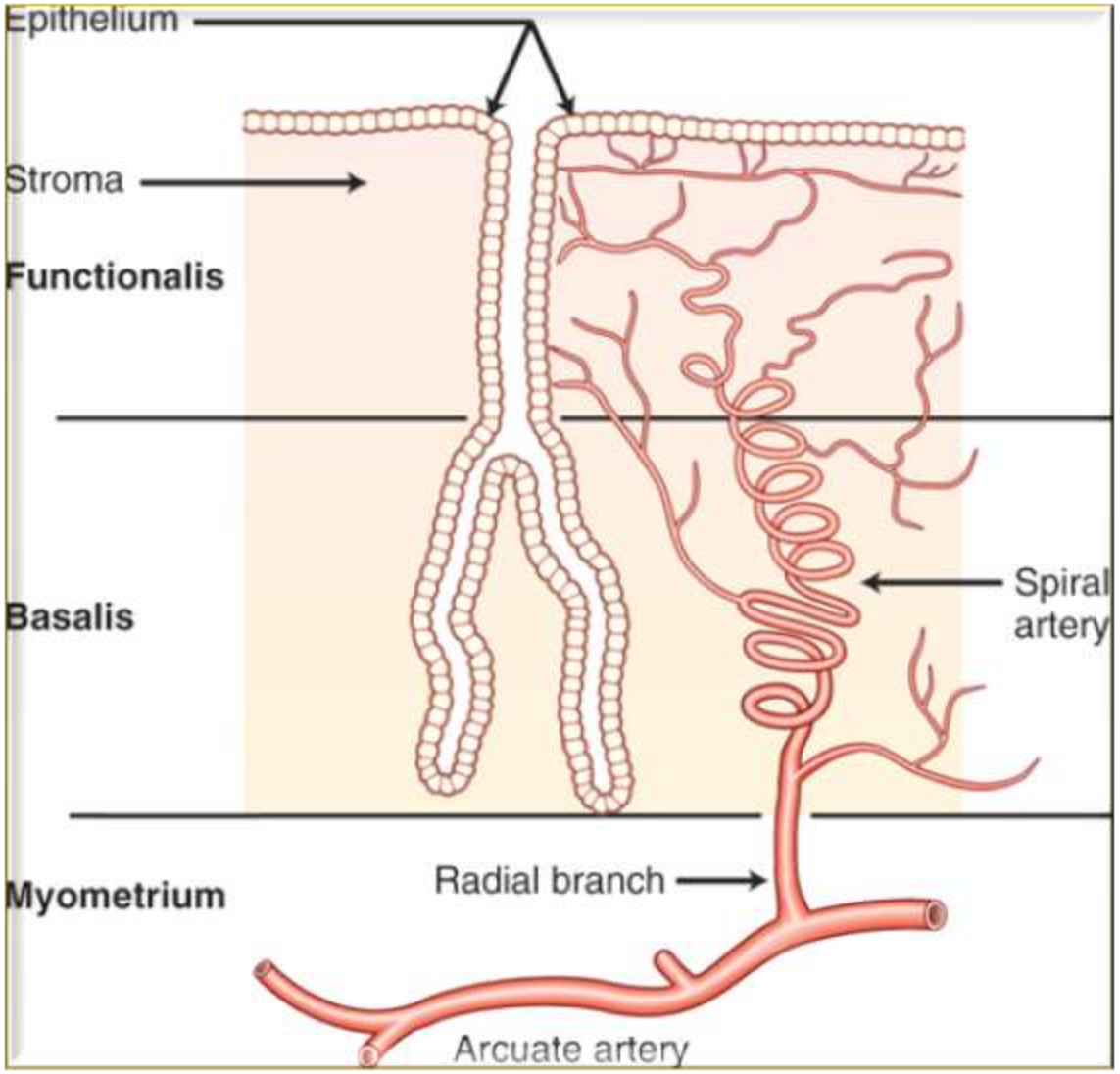
Endometrial adenogenesis in the uterine wall

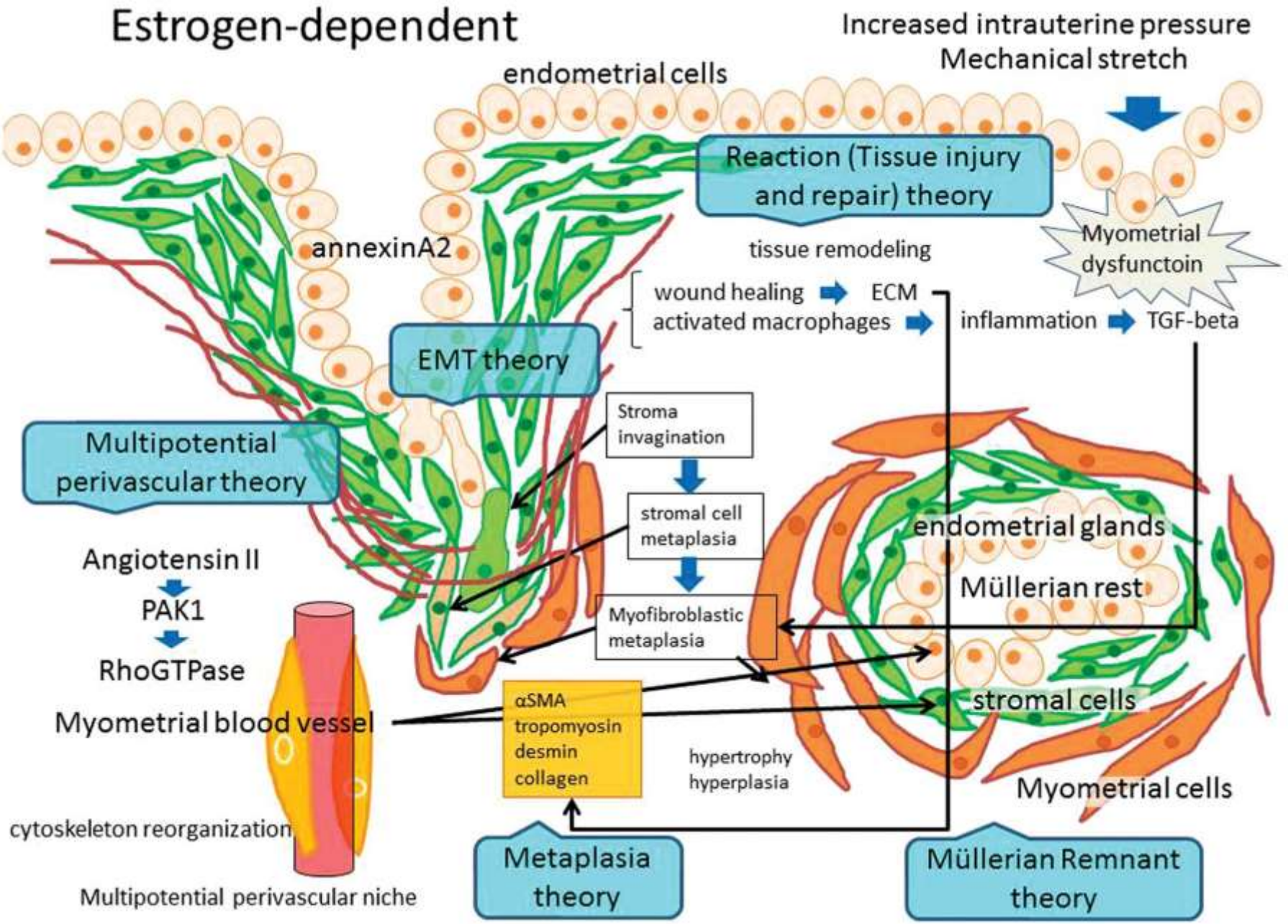


Following invagination of stromal cells, invasion of glandular cells

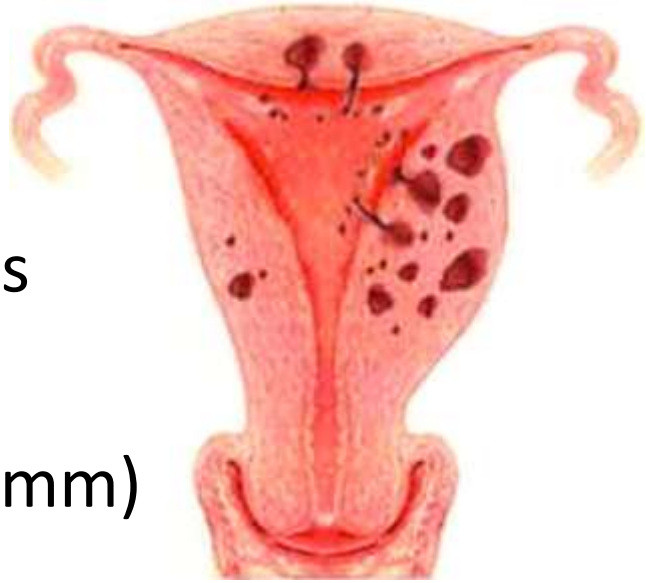
Abnormal growth and differentiation

These cells are surrounded by hypertrophic and hyperplastic myometrium




Estrogen-dependent
Increased intrauterine pressure
Mechanical stretch
endometrial cells
Reaction (Tissue injury and repair) theory
annexinA2
tissue remodeling
Myometrial dysfunctoin
wound healing
ECM
activated macrophages
inflammation
TGF-beta
EMT theory
Stroma invagination
Multipotential perivascular theory
stromal cell metaplasia
endometrial glands
Angiotensin II
Müllerian rest
PAK1
Myofibroblastic metaplasia
RhoGTPase
Myometrial blood vessel
αSMA
tropomyosin
desmin
collagen
stromal cells
hypertrophy
hyperplasia
Myometrial cells
cytoskeleton reorganization
Metaplasia theory
Müllerian Remnant theory
Multipotential perivascular niche

Sonographic criteria of adenomyosis

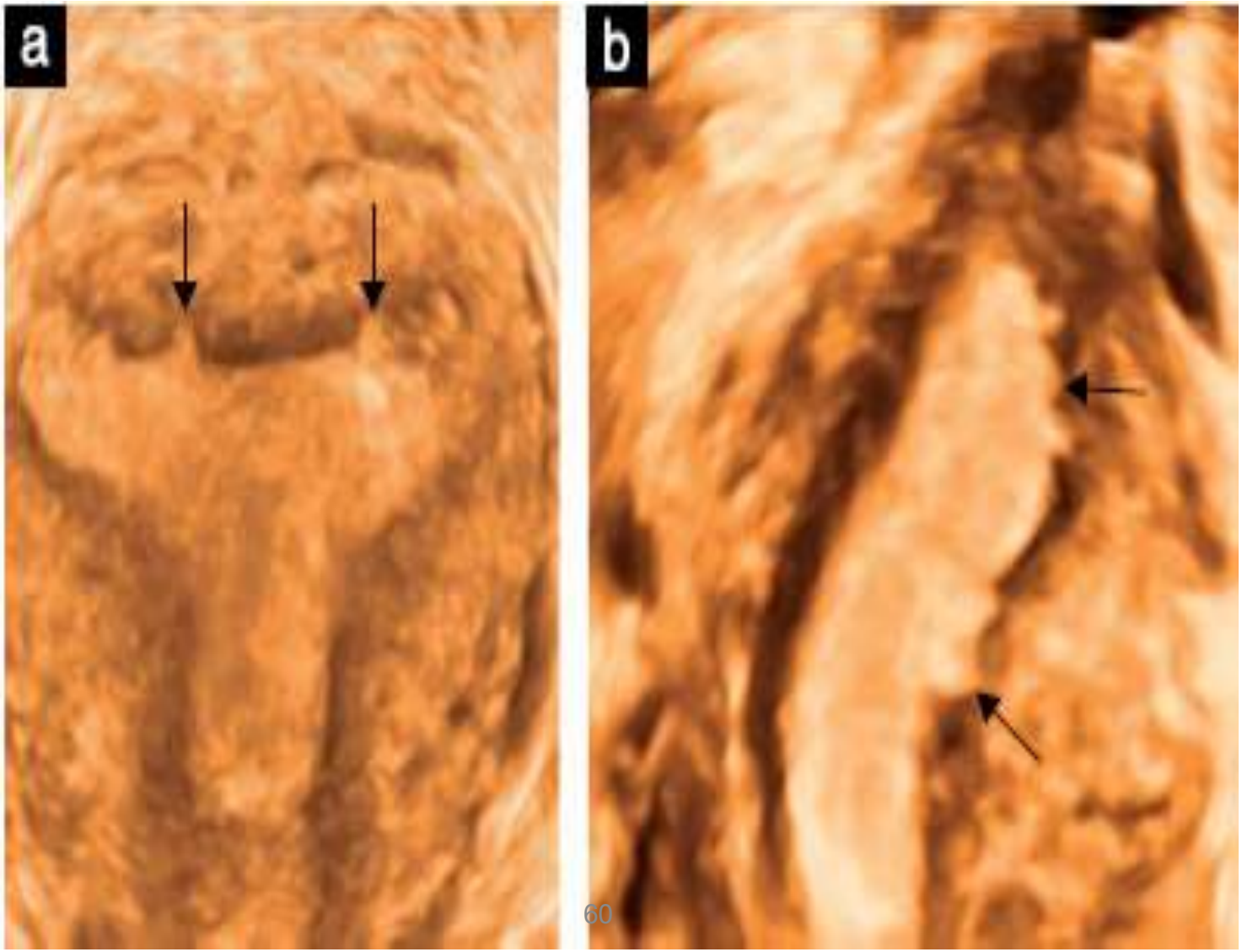


- Globular shaped uterus
- Mottled inhomogeneous myometrium
- Indistinct borders to a myometrial mass
- Indistinct endometrial stripe
- Subendometrial myometrial cysts (2-6 mm)
- Subendometrial echogenic nodules
- Subendometrial echogenic linear striations
- Asymmetric thickening of the anterior/posterior wall
- Mimimal mass effect on the endometrium or serosa
- Irregular endometrial-myometrial junction (EMJ)
- Asymmetric thickness

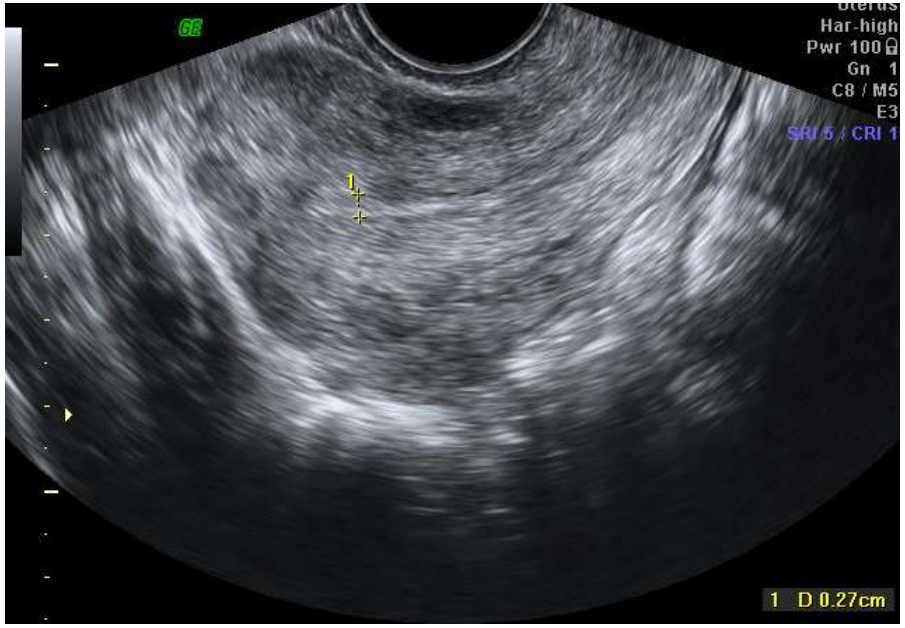
Junctional zone

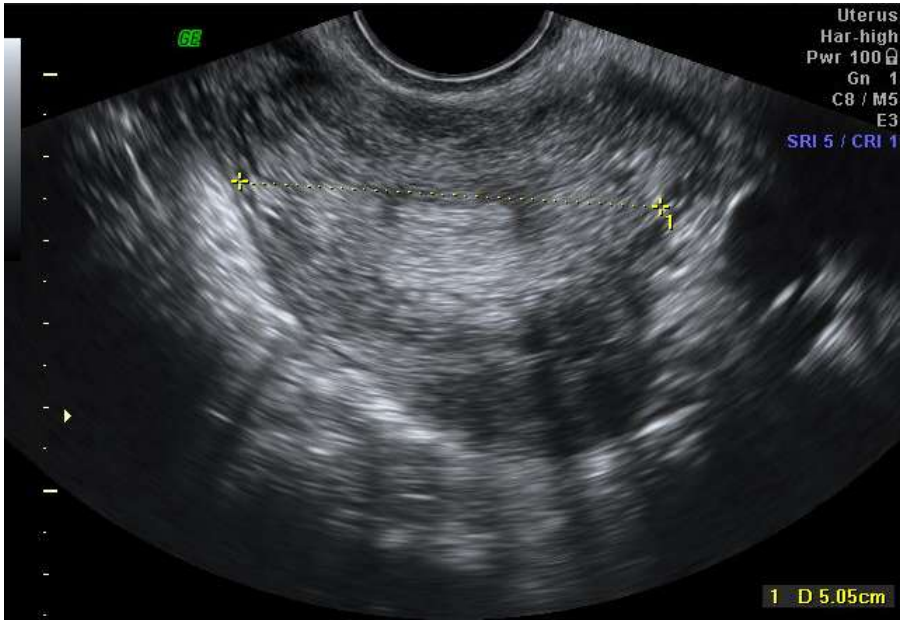
A normal junctional zone 5 mm in thickness or less

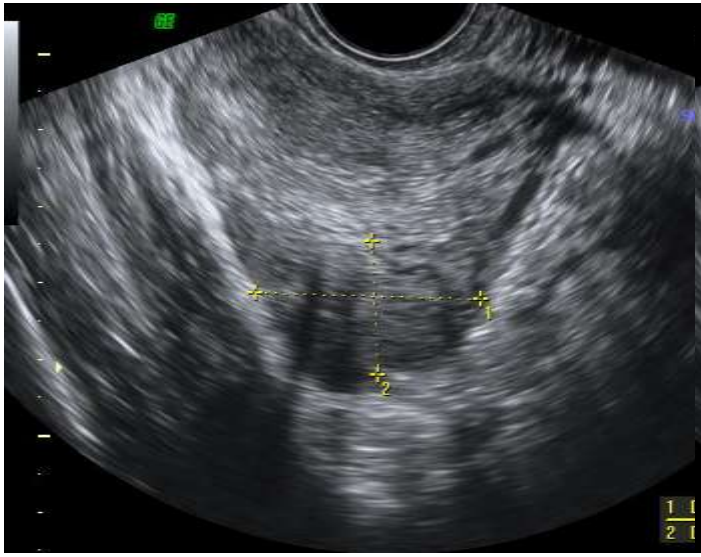
Early adenomyosis

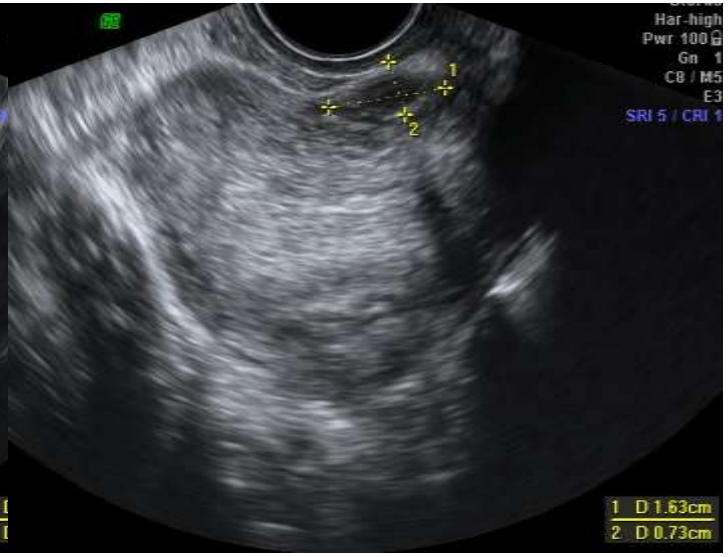


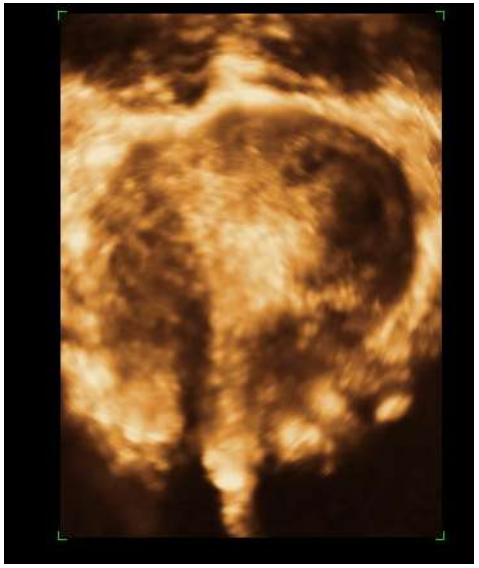
Adenomyosis











Malignant transformation of adenomyosis

- **The pathological criteria used for case identification are:**
- i) evidence of pre-existing adenomyosis at the site of the malignant lesion
- ii) presence of glandular cells and/or endometrial stromal cells supporting a diagnosis of adenomyosis
- iii) evidence of transitions between benign and malignant glandular structures
- iv) carcinoma must be absent from invasion or metastasis from another source
- v) carcinoma must be absent from the eutopic endometrium

Malignant transformation of adenomyosis

- Malignant changes in adenomyosis were present in 6.8% of patients with endometrial cancer
- A majority of cases with adenocarcinoma arising in adenomyosis were associated with adjacent endometrial adenocarcinomas
- Adenocarcinomas developing within adenomyosis often originate from endometrial carcinomas which arise from the eutopic endometrium, then invade into pre-existing adenomyosis

Adenomyosis and infertility

- There are good biological reasons to suspect that adenomyosis may have the potential to impair the implantation of good quality embryos transferred during IVF treatment
- A recent observational study clearly linked adenomyosis diagnosed on magnetic resonance imaging (MRI) with an increase in macrophage and natural killer cells in the endometrium of women experiencing infertility

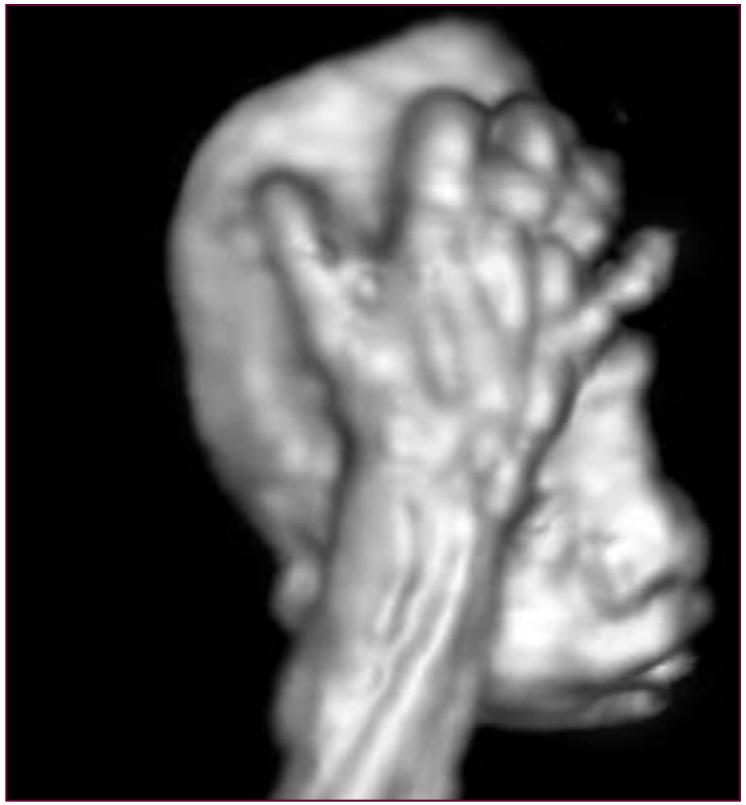
Human Reproduction, Vol.27, No.12 pp. 3487–3492, 2012

Adenomyosis and infertility

- Macrophages are known to release chemicals which may be detrimental to embryos, such as cytokines tumour necrosis factor a (TNFa) and interferon g (IFNg), plus harmful reactive oxygen species (‘free radicals’)
- Earlier studies have linked the presence of adenomyosis with increased ‘free radical’ concentrations in the endometrium , providing a possible mechanism by which adenomyosis may impair implantation and cause miscarriage.

Human Reproduction, Vol.27, No.12 pp. 3487–3492, 2012

Thank you

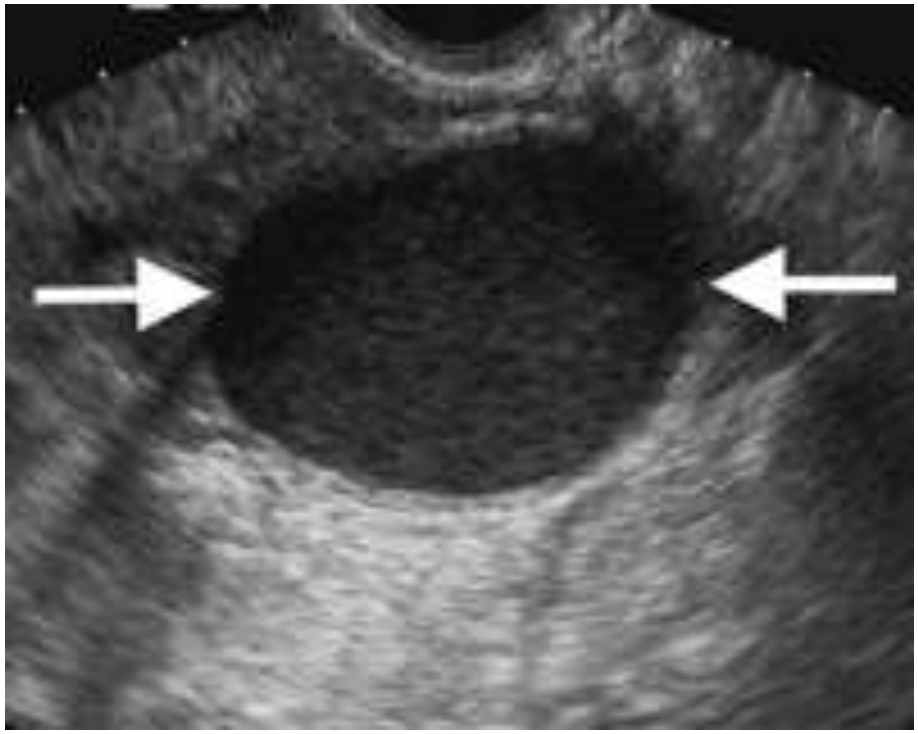


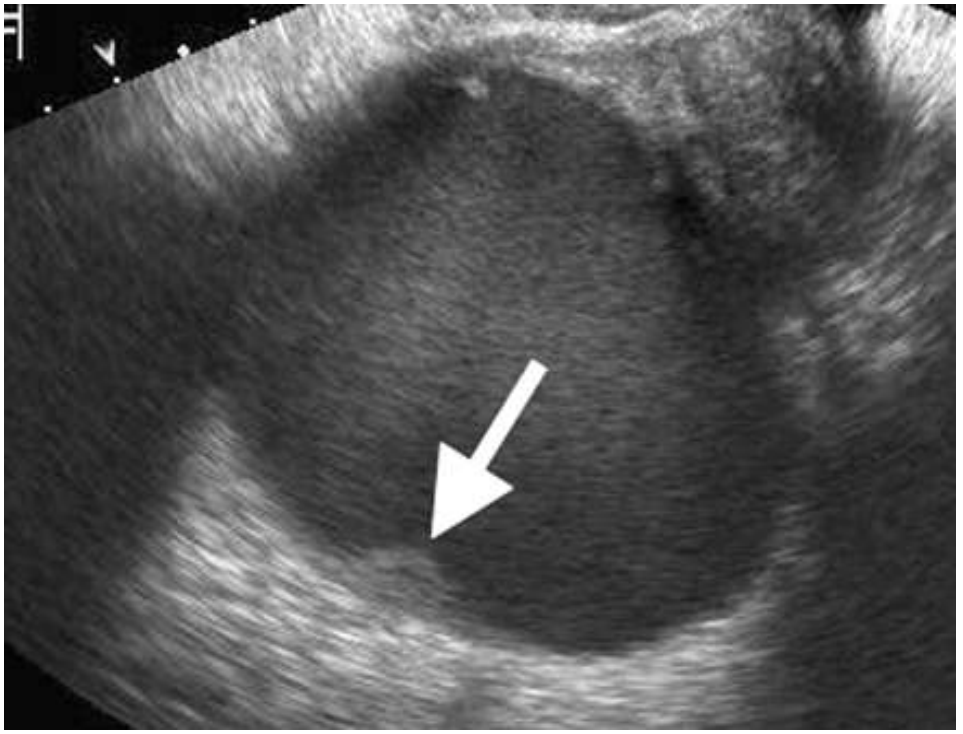
veredeis@bezeqint.net

Typical transvaginal ultrasonographic appearance of an endometrioma

- A mass characterized by circular homogeneous, hypoechoic “tissue” without papillary proliferations and a clear demarcation from the ovarian parenchyma
- “ground glass appearance” (chocolate cyst)
- Diffuse, low-level echoes (82-95%)
- Small, 30-59 mm – 80%

Typical endometriomas





- Wall nodularity – 20%
- Hyperechoic wall foci result from cholesterol crystals break-up from chronic hemorrhage - 30% (old cysts)

Typical vs. atypical Endometrioma

- Typical endometrioma:
  - Unilocular
  - Ground glass (homogenous)
  - +/- wall nodularity
- Atypical endometrioma:
  - Bi or multilocular
  - Not ground glass
  - Retracted blood clots
  - Calcifications
  - Papillary projections with vascularization in pregnancy, calcified
  - Completely atypical
- Malignization: 0.3-0.8%

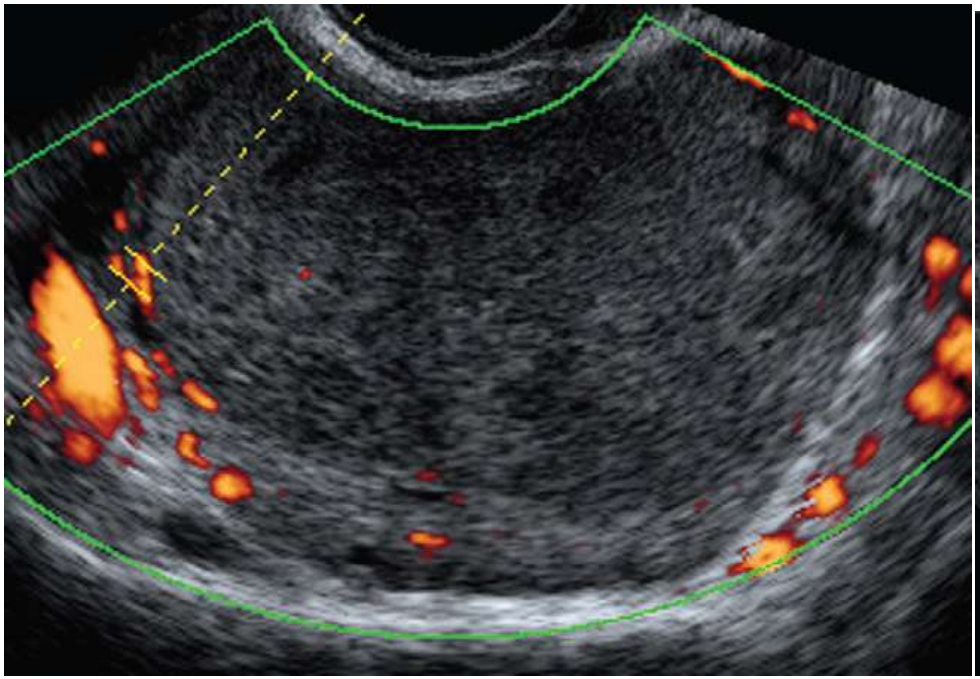
Optimal rule for endometriomata

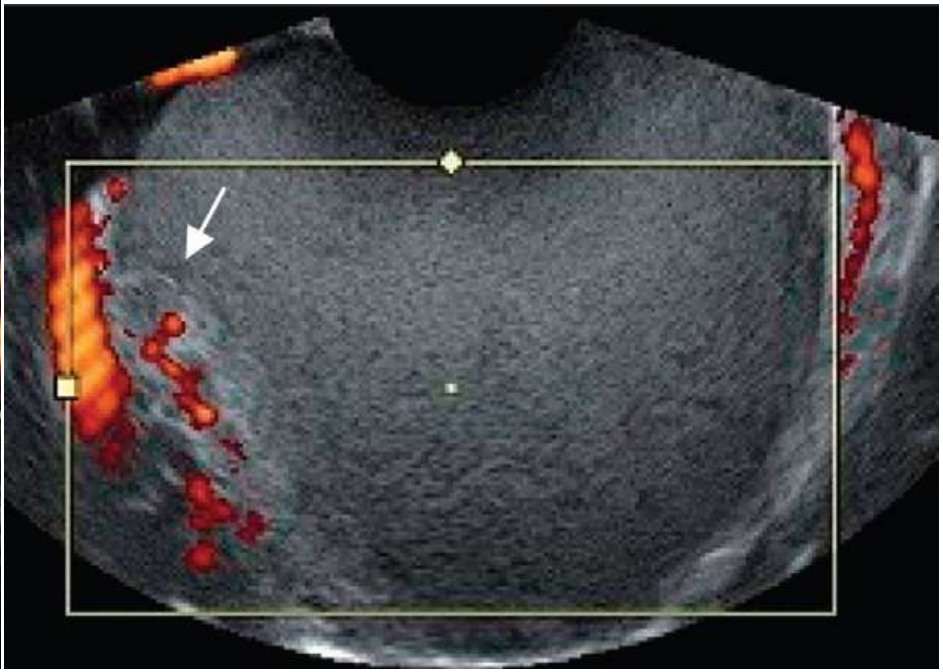
- **“adnexal mass in a premenopausal patient with ground glass echogenicity of the cyst fluid, one to four locules, without a solid component”**
- When tested on the whole IOTA dataset, this rule gave a specificity of 98%

Van Holsbeke UOG 2011

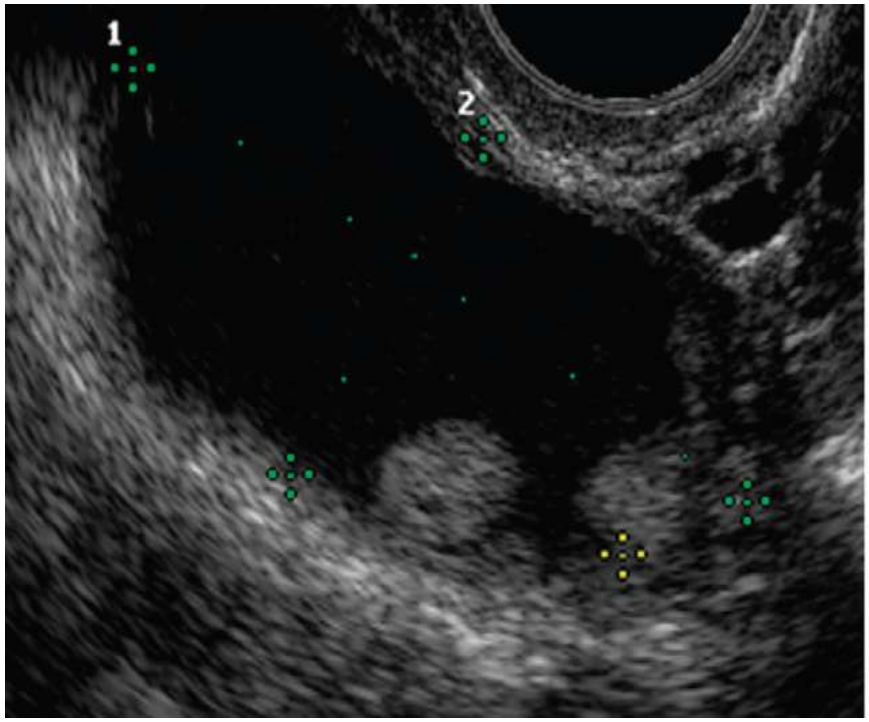
Endometriomas in pregnancy

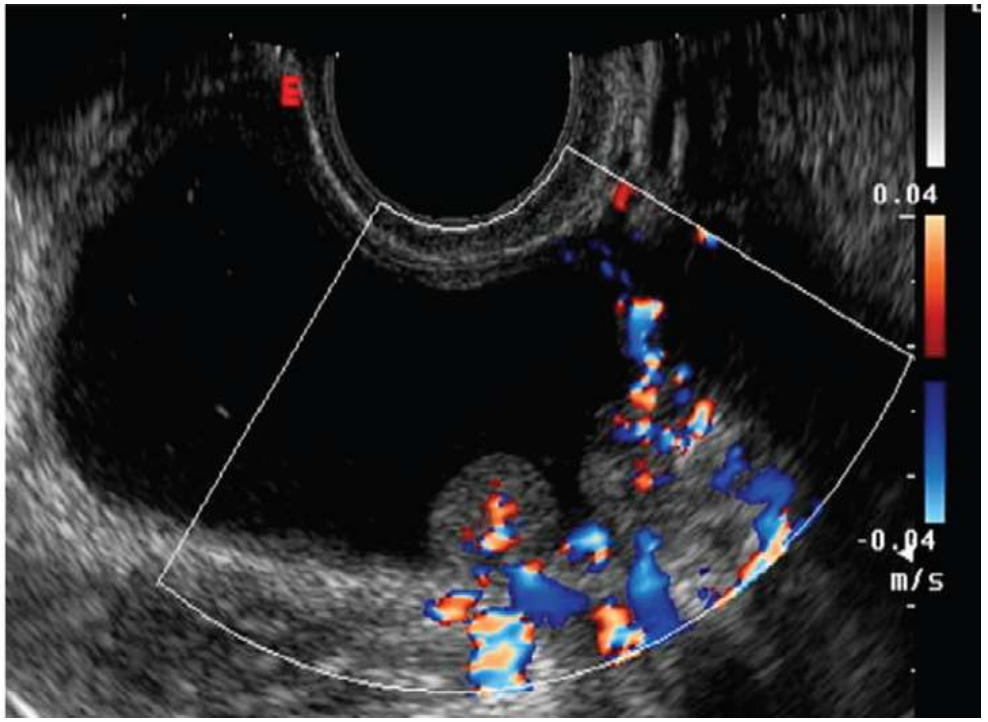
- Most decidualized endometriomas (82%) - vascularized rounded papillary projections with a smooth contour in an ovarian cyst with one or a few cyst locules and ground-glass or low-level echogenicity of the cyst fluid





Endometriomas in pregnancy





Endometriomas and malignancy

- Subjective impression – misclassification of malignancies as endometriomas in 0.2-0.9%
- Characteristics differ in pre-menopausal and post-menopausal women
- Postmenopausal with ground glass – high malignancy risk
- Precursors of endometrioid BOT which may progress to low-grade invasive carcinoma
- Associated clear-cell BOT

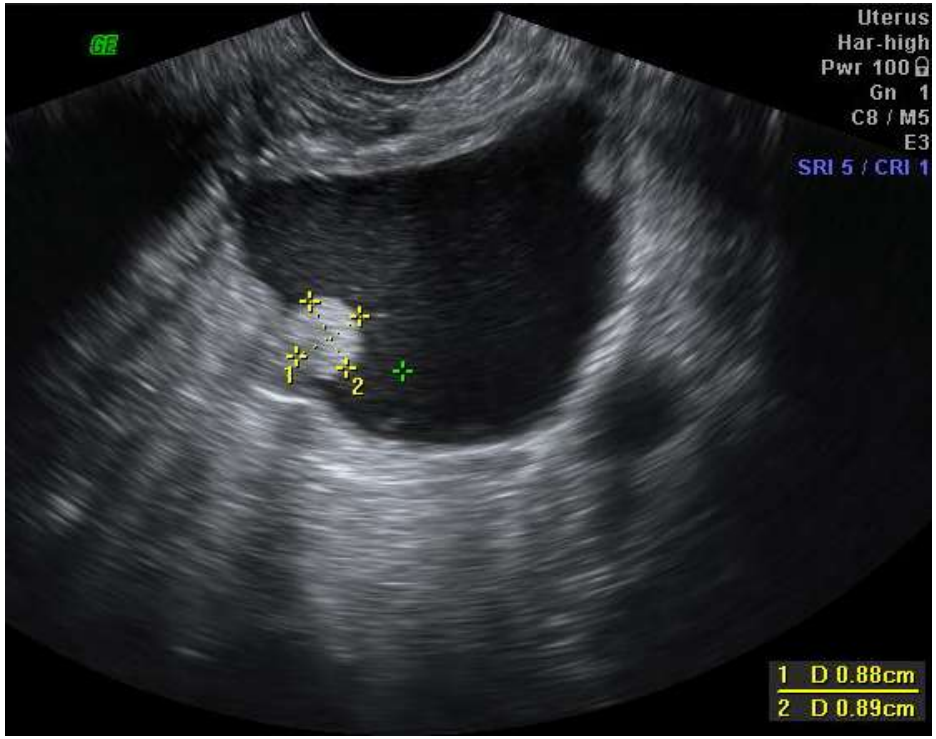
Testa 2011, Van Holsbeke 2010, Lalwani 2010

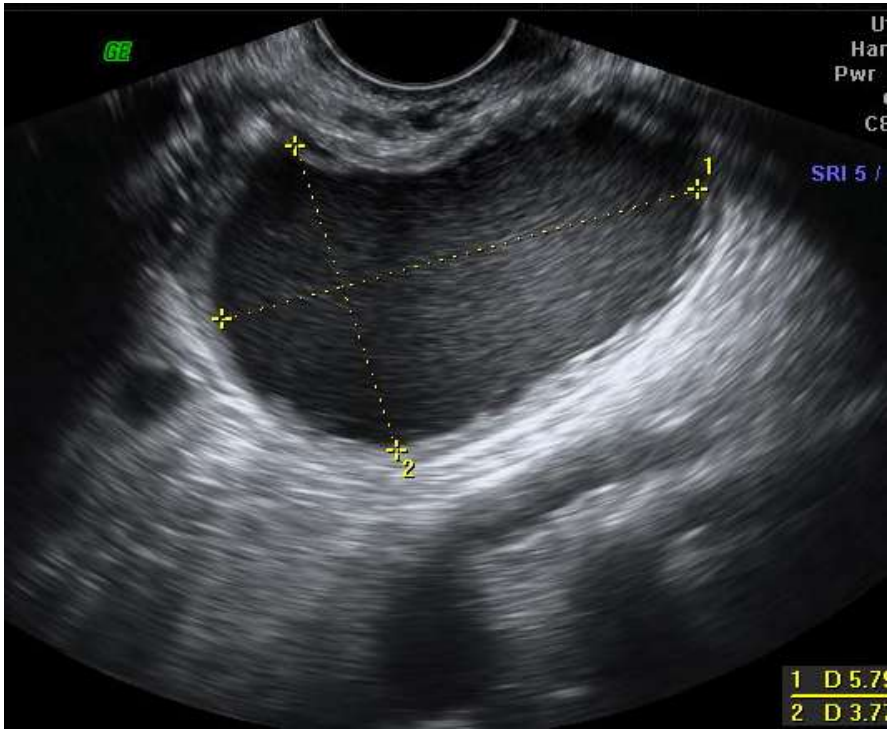
Endometriomas and malignancy

- Vascularized solid component
- In pregnancy difficult differentiation between BOT and decidualised endometriotic cysts
- Decidualised endometriomas – 82% vascularised rounded papillary projections with a smooth contour in an ovarian cyst with one or more cyst locules and ground glass or low level echogenicity of the cyst fluid

Fruscella 2004, Mascilini 2014 online

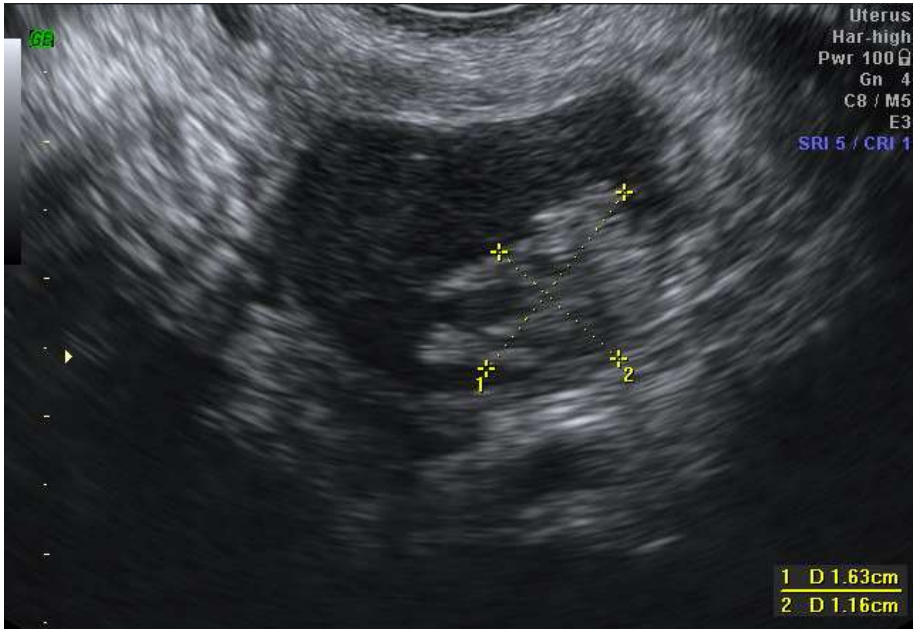
?


GE
Uterus
Har-high
Pwr 100
Gn 1
C8 / M5
E3
SRI 5 / CRI 1
1
2
1 D 0.88cm
2 D 0.89cm


GE
1
2

BOT serous

?



Invasive micropapillary serous Ca arising in BOT serous

Superficial endometriosis

- Up to 15% of normal asymptomatic healthy women
- Not visible by imaging?
- Almost 100% of patients with endometriomas have superficial disease elsewhere
- But in the absence of endometrioma?

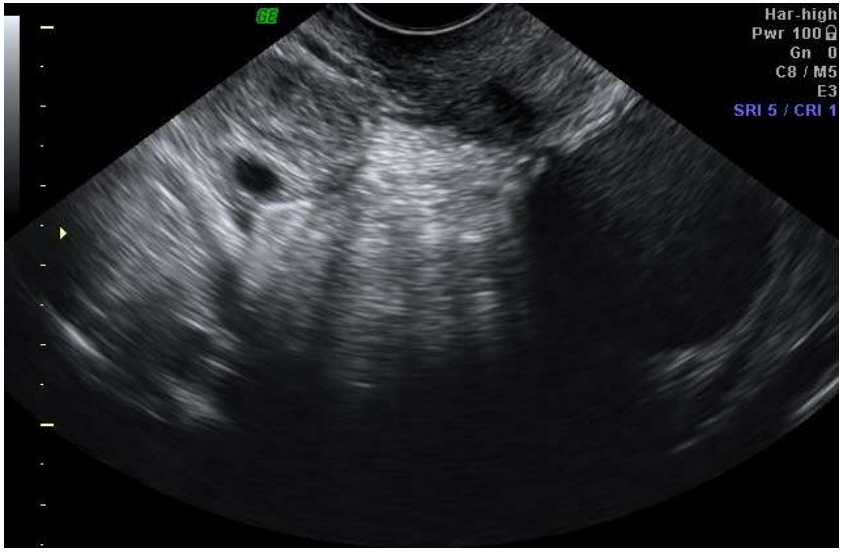
Superficial endometriosis

- Up to 15% of normal asymptomatic healthy women
- Not visible by imaging?
- Almost 100% of patients with endometriomas have superficial disease elsewhere
- But in the absence of endometrioma?

Diagnosis of adhesions to ovaries

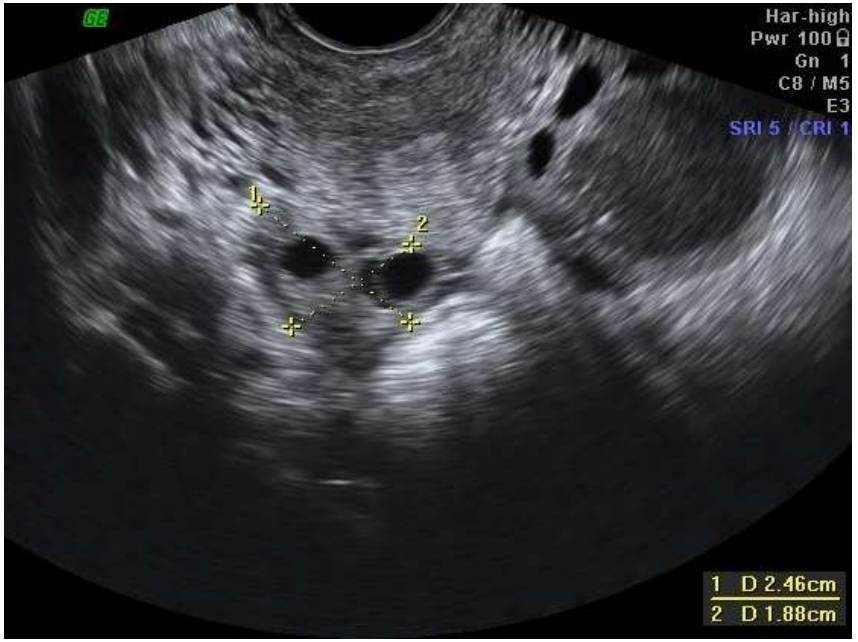
- Fixation to the uterus of at least one ovary on US: (Guerriero, 2009)
  - Sensitivity 89%, specificity 90%, LR+ 8.92, LR-0.12
  - 96% probability of adhesions (27% when absent)

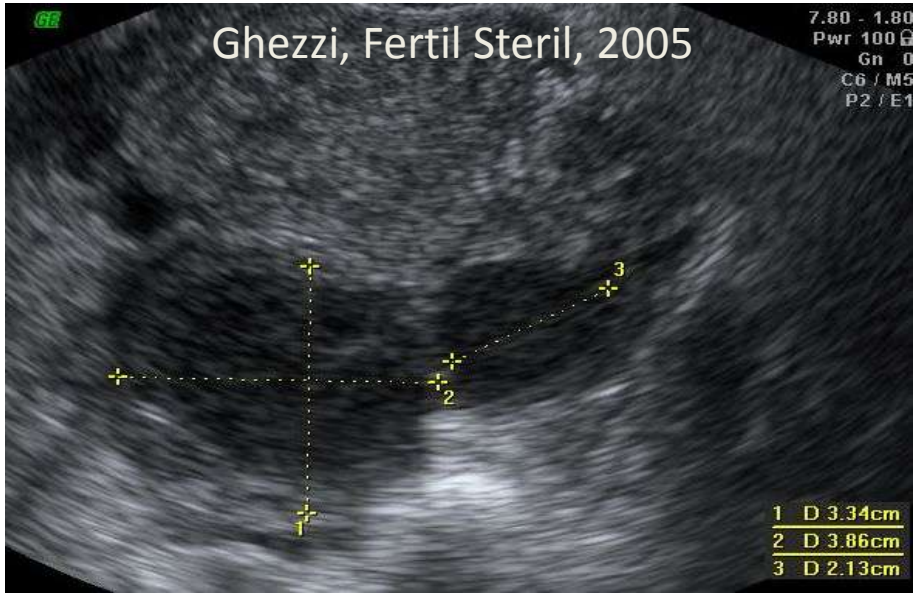
Adhesions small bowel to adnexa and uterus

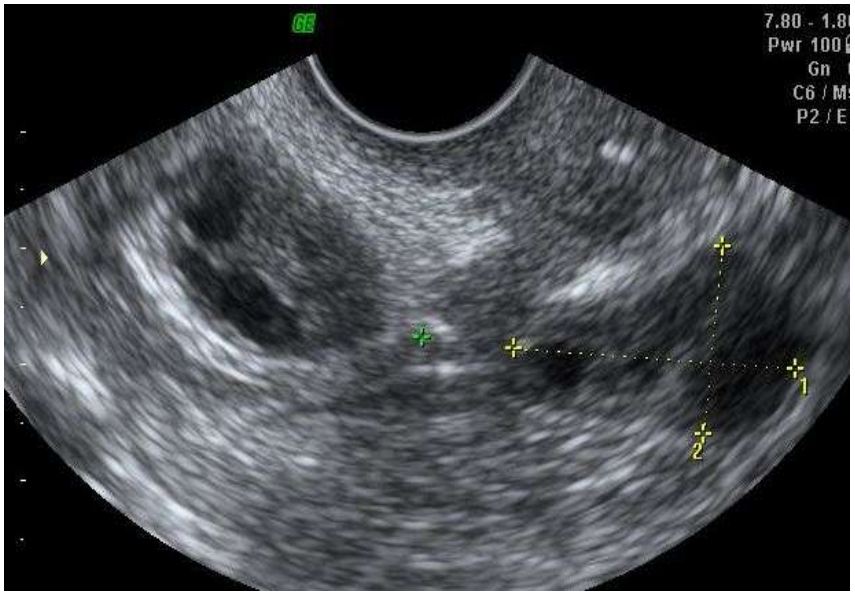


Kissing ovaries and deep endometriosis

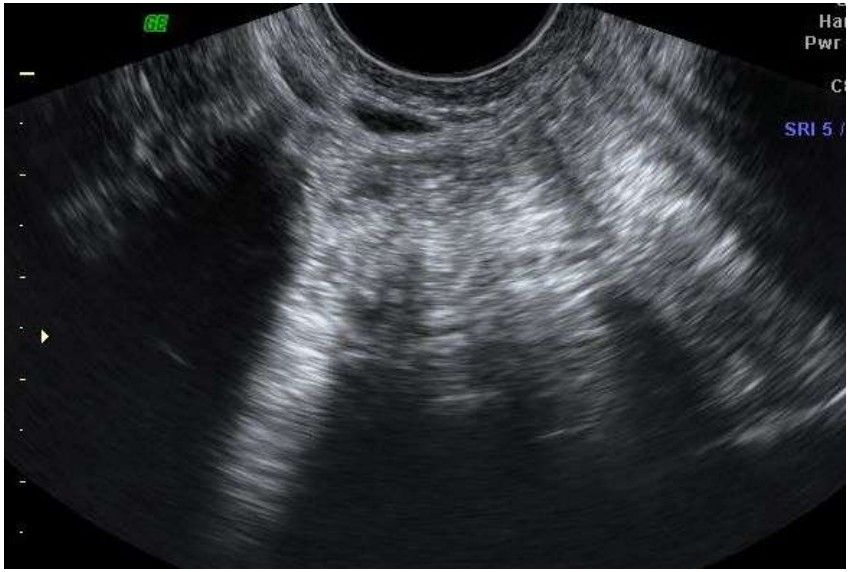
Criteria	Kissing ovaries	Non kissing ovaries
Bowel involvement	18.5	2.5
Fallopian tube obstruction	80	8.6
AFS score	74	35
Operating time	115 min	50 min



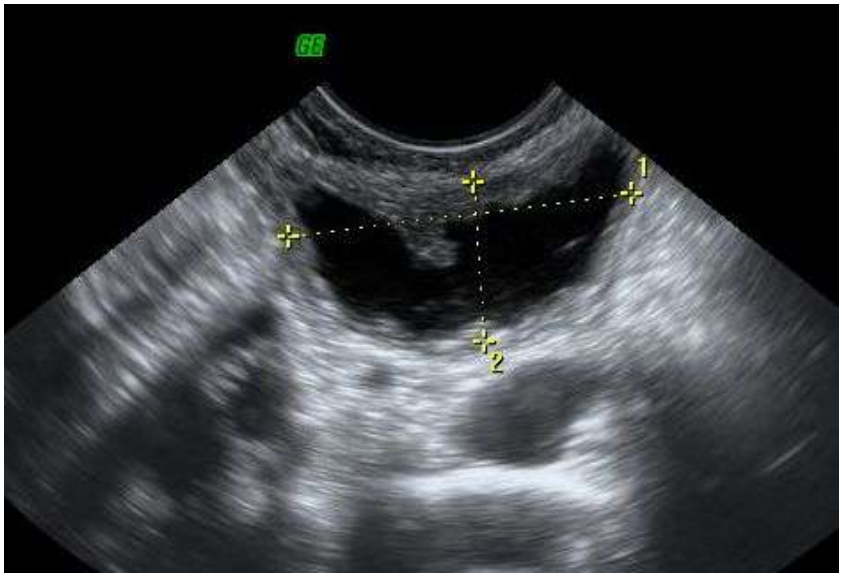


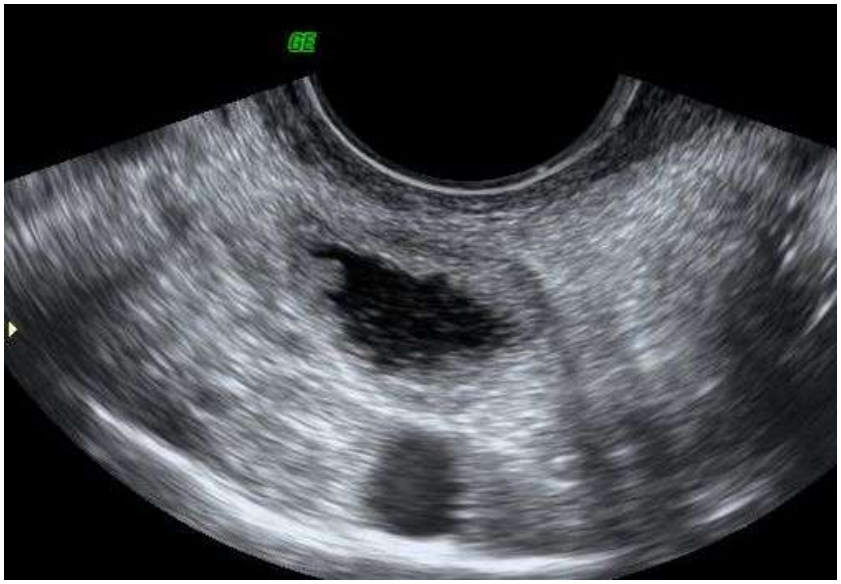


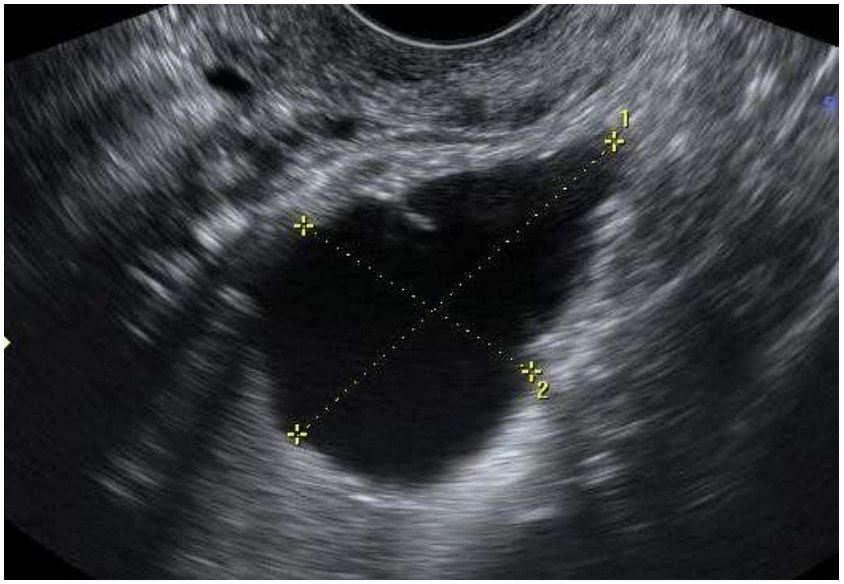
Intestinal adhesions



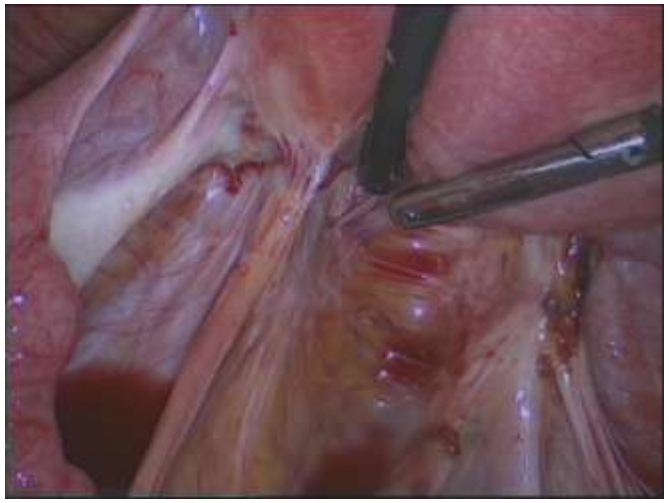
Tubal disease



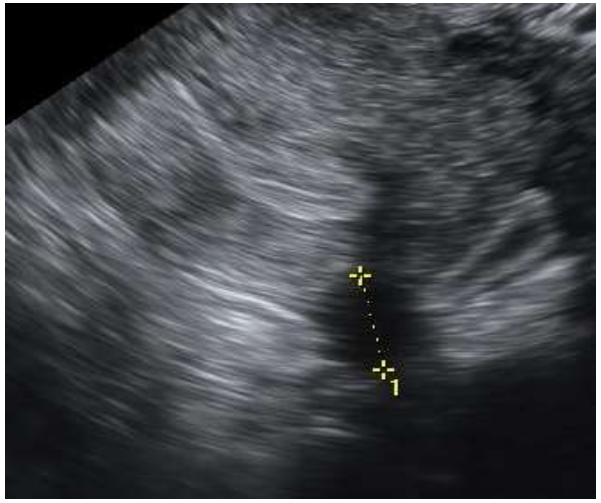


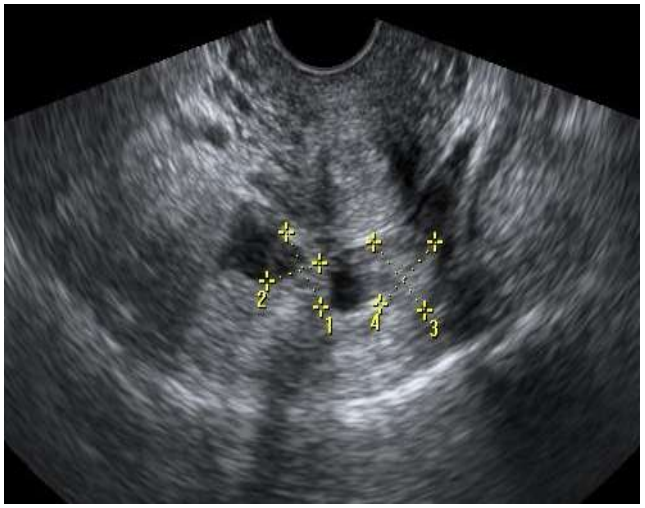


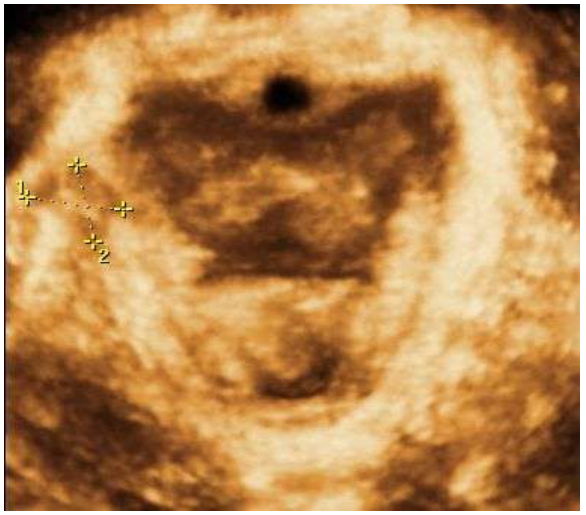
Sacrouterine involvement



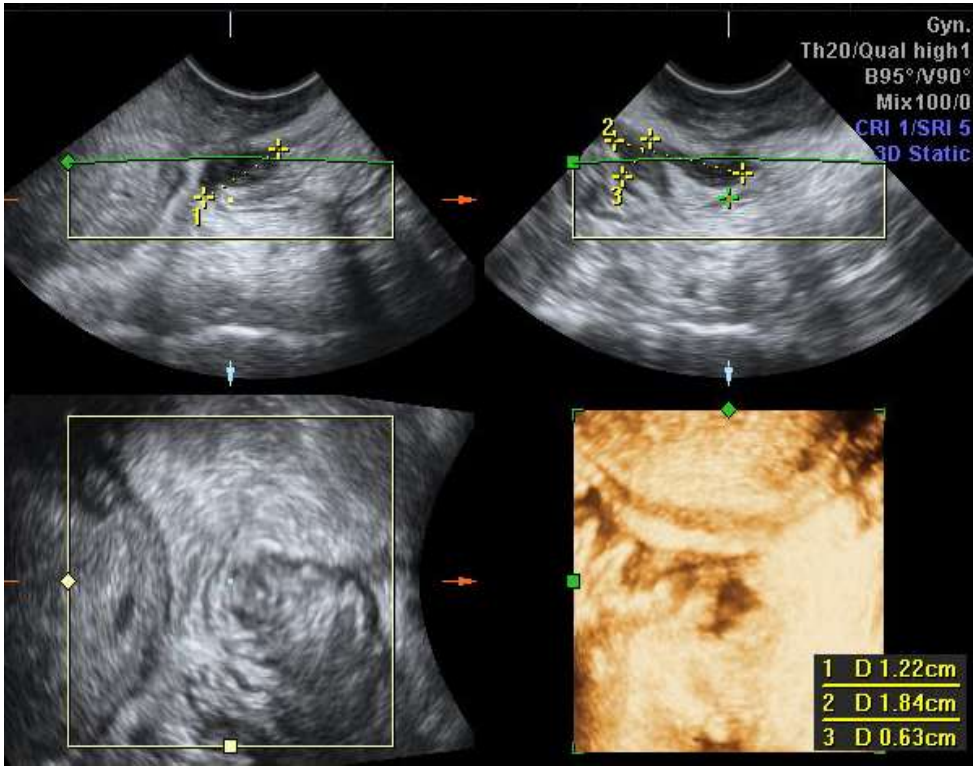
Involvement of the recto-vaginal space with both uterosacral ligaments involved and the normal anatomy of both ureters disturbed

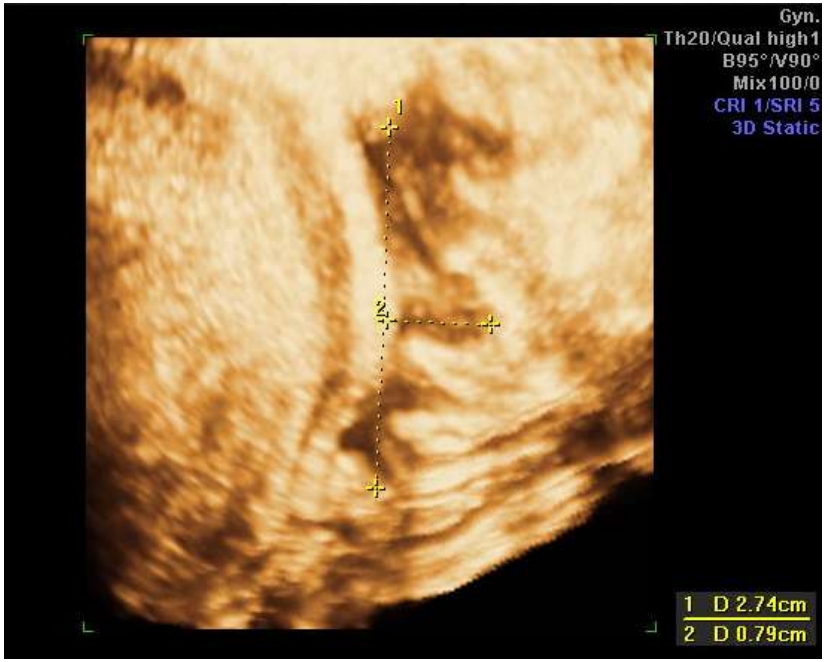






Rectosigmoid nodules

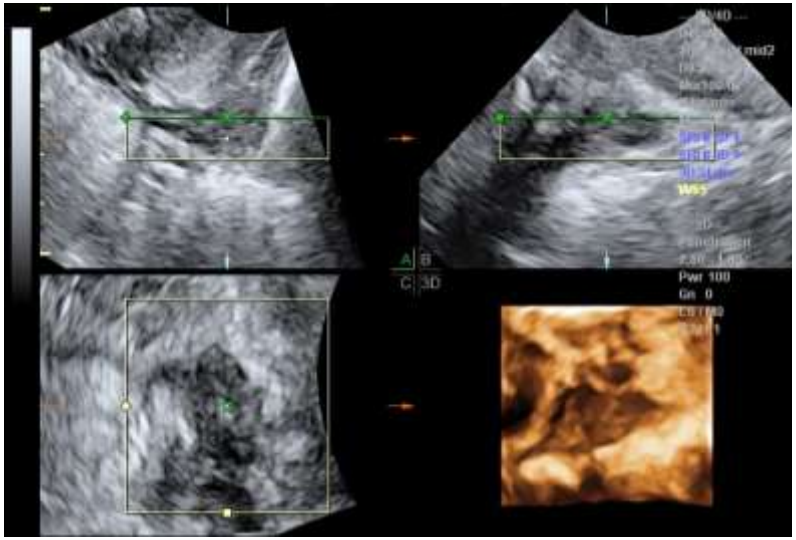




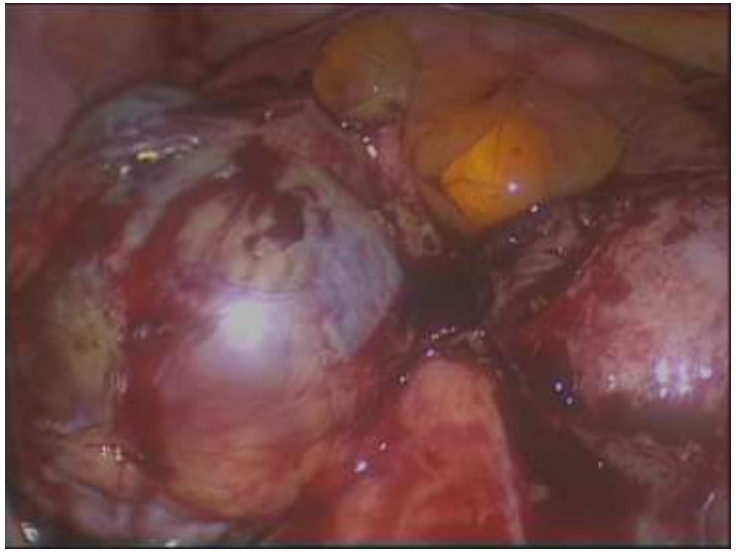
Indian headdress sign

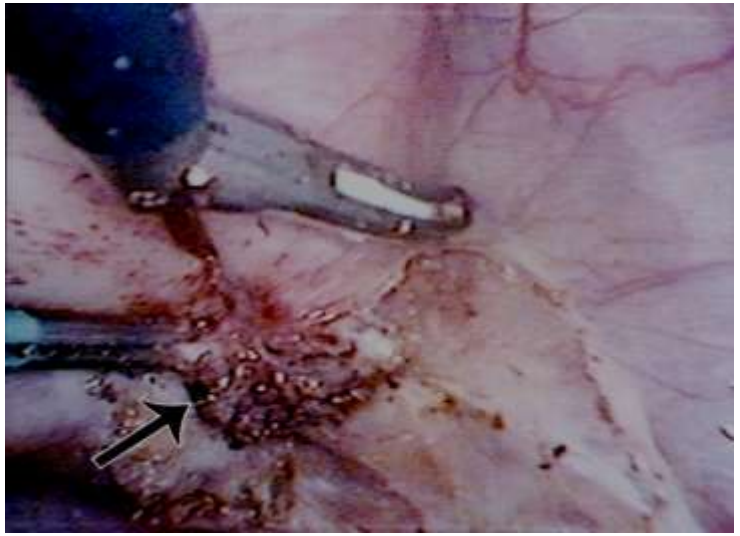
Rectosigmoid nodules

US diagnosis

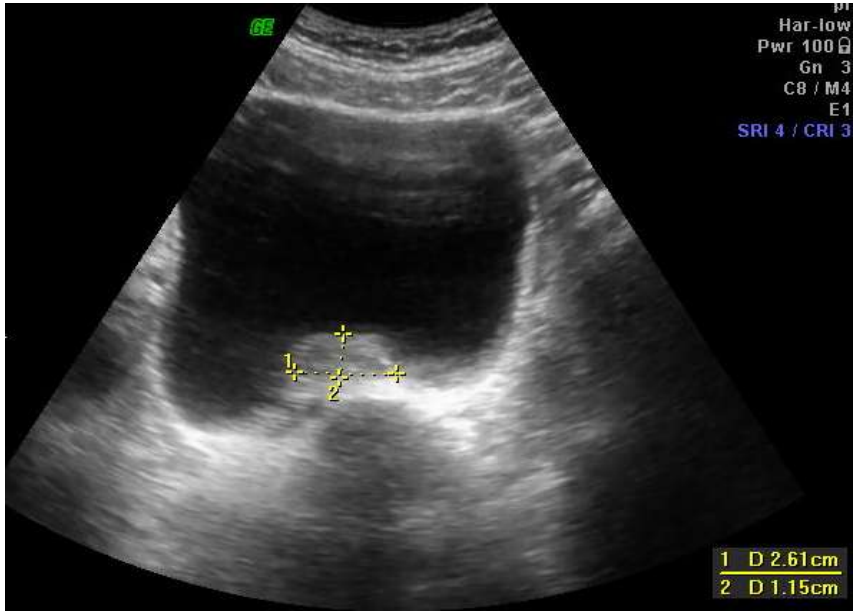


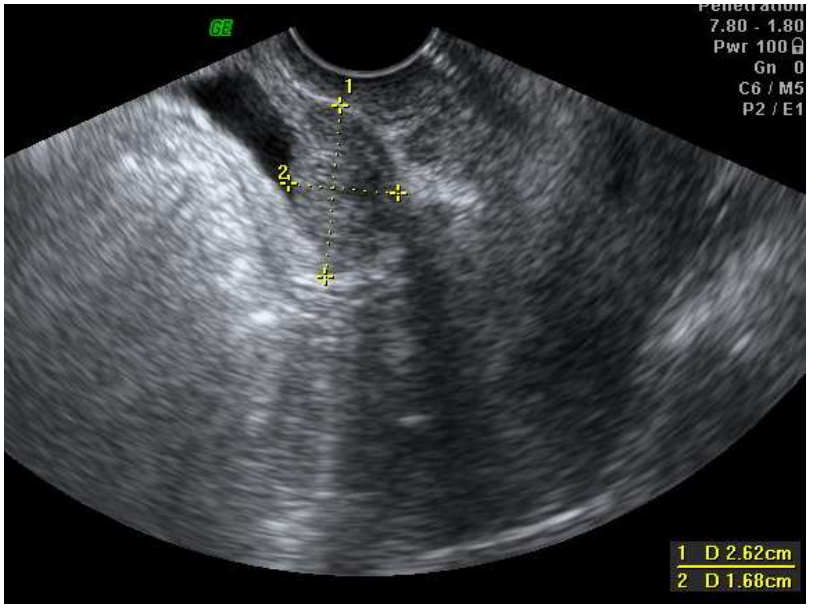
Surgical diagnosis

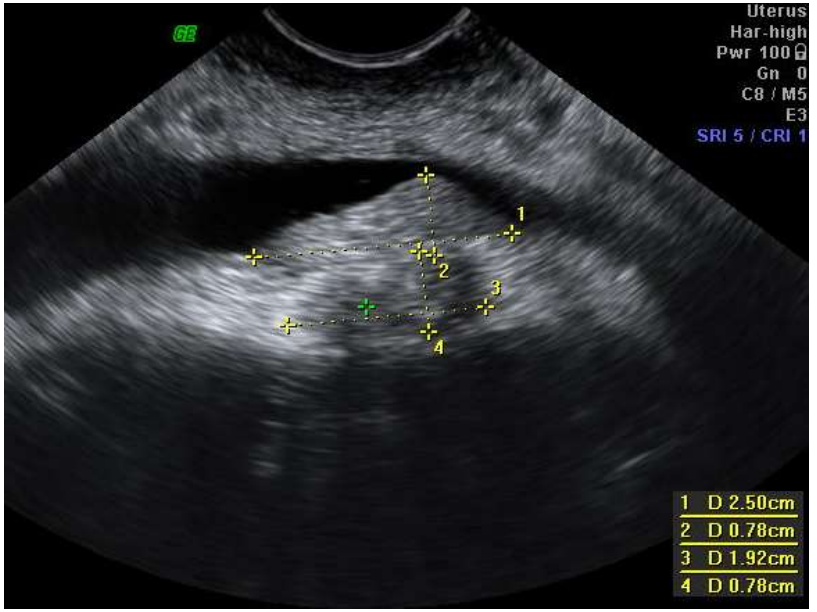




Anterior compartment involvement

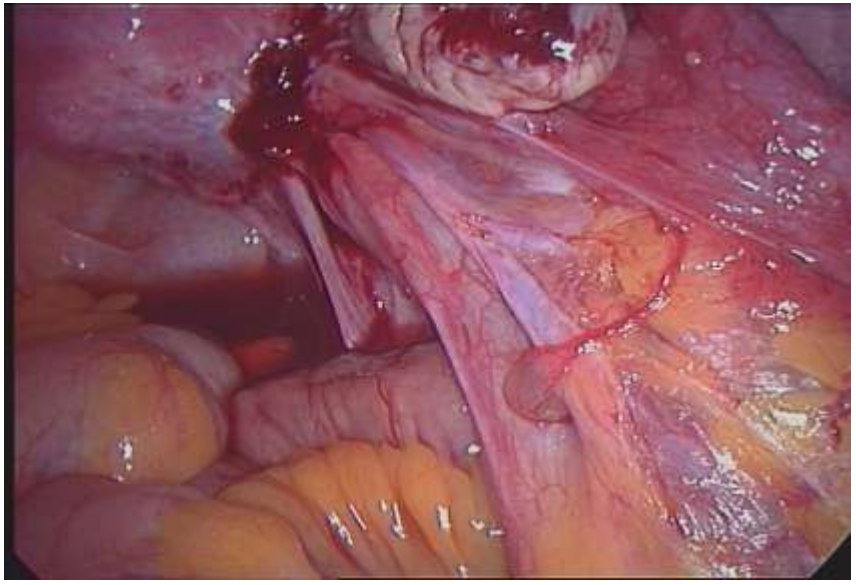


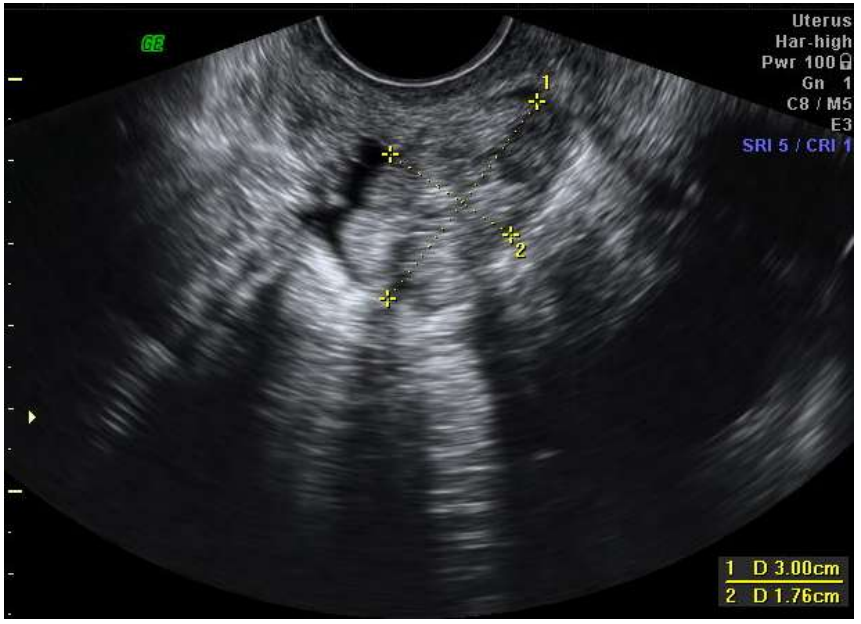


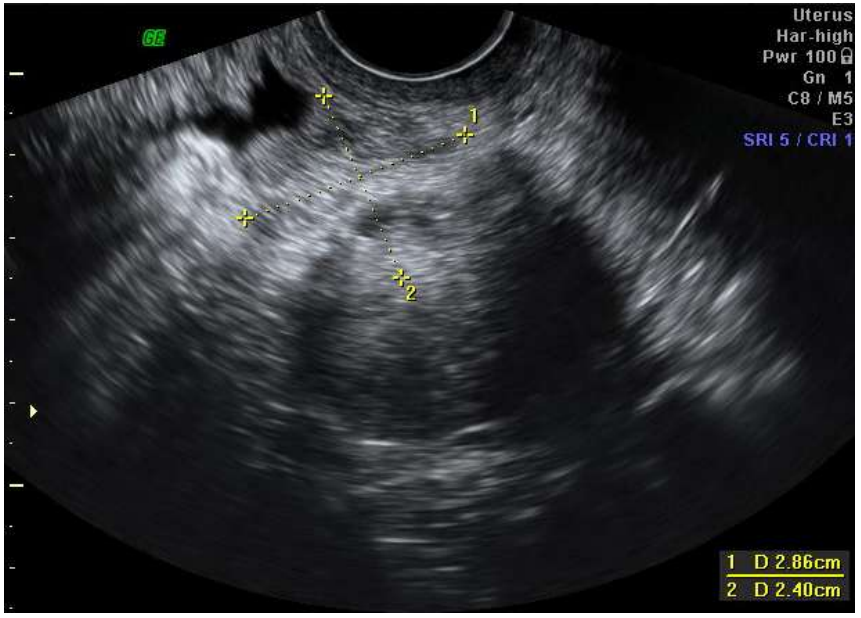


Anterior compartment involvement

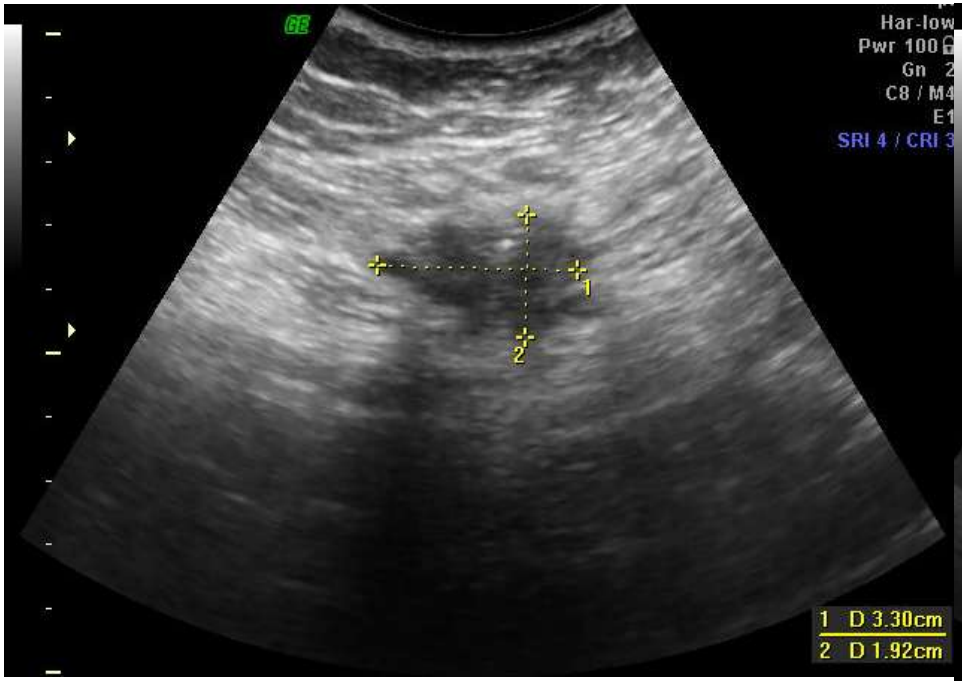
Bladder plica nodule

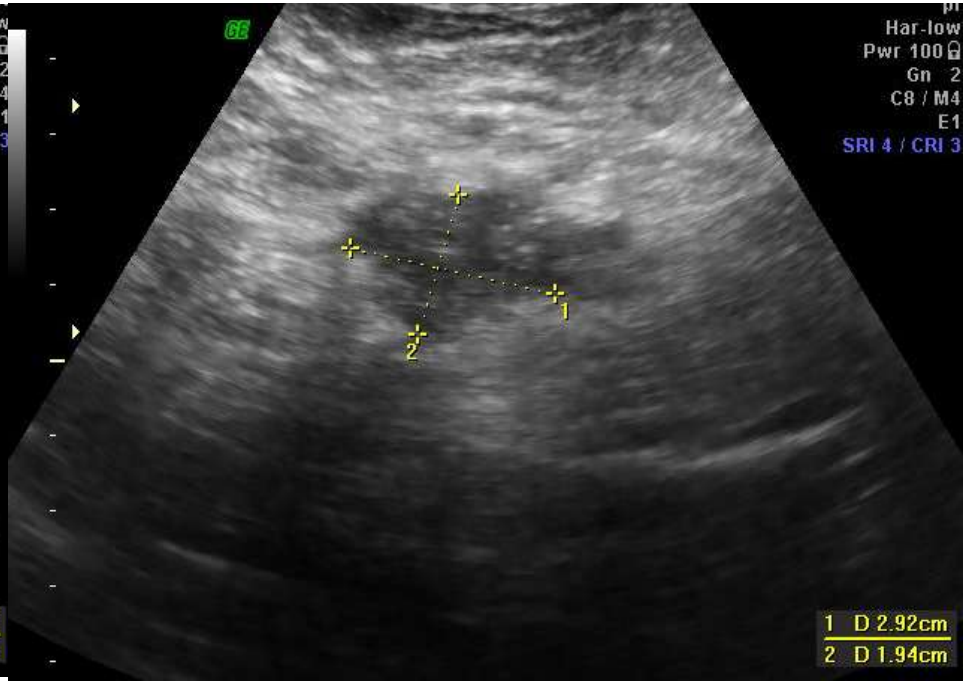






Abdominal wall endometriosis





Comparison between modalities

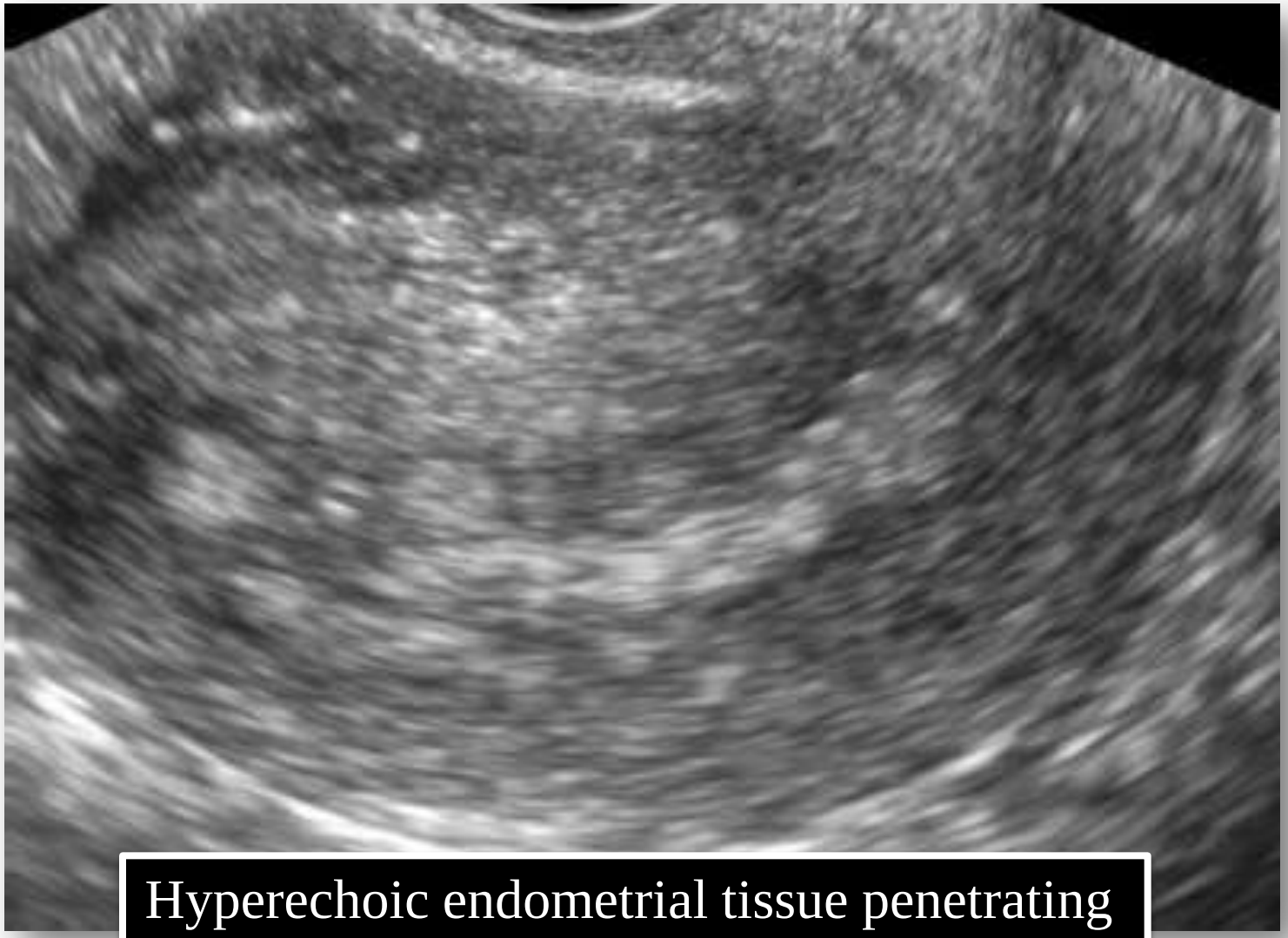
Location	Test	PE (%)	TVUS (%)	RES (%)	MRI (%)
Overall	Sensitivity	83	86	73	95
Uterosacral	Sensitivity	73	78	48	84
	Accuracy	74	77	47	85
Rectosigmoid	Sensitivity	46	94	89	87
	Accuracy	54	96	89	87
Vaginal	Sensitivity	50	47	7	80
	Accuracy	75	79	70	84
Rectovaginal	Sensitivity	18	9	18	55
	Accuracy	87	88	86	94

Measurement of the junctional zone

Progression of adenomyosis

Well-defined myometrial cysts

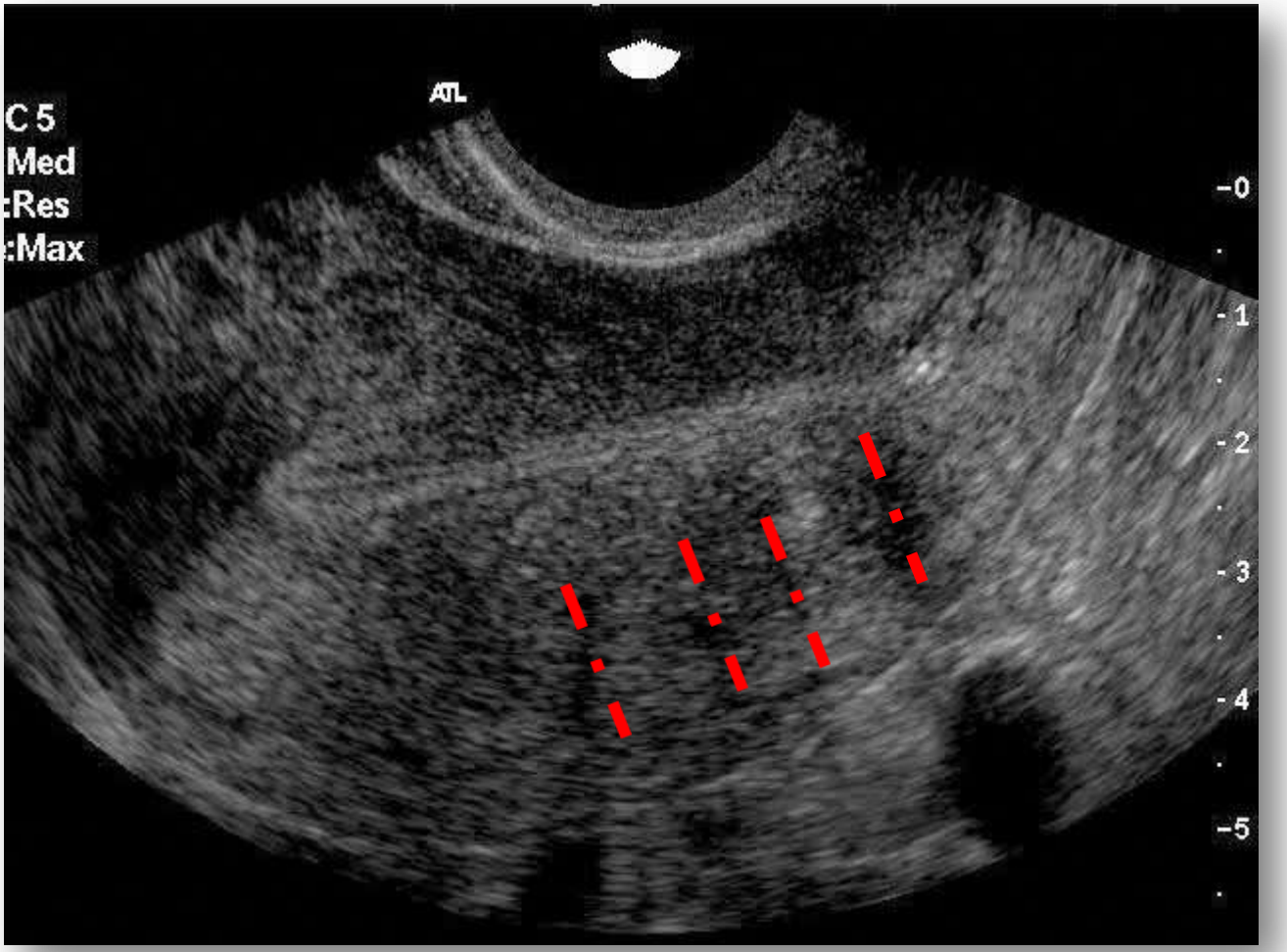
Irregular border



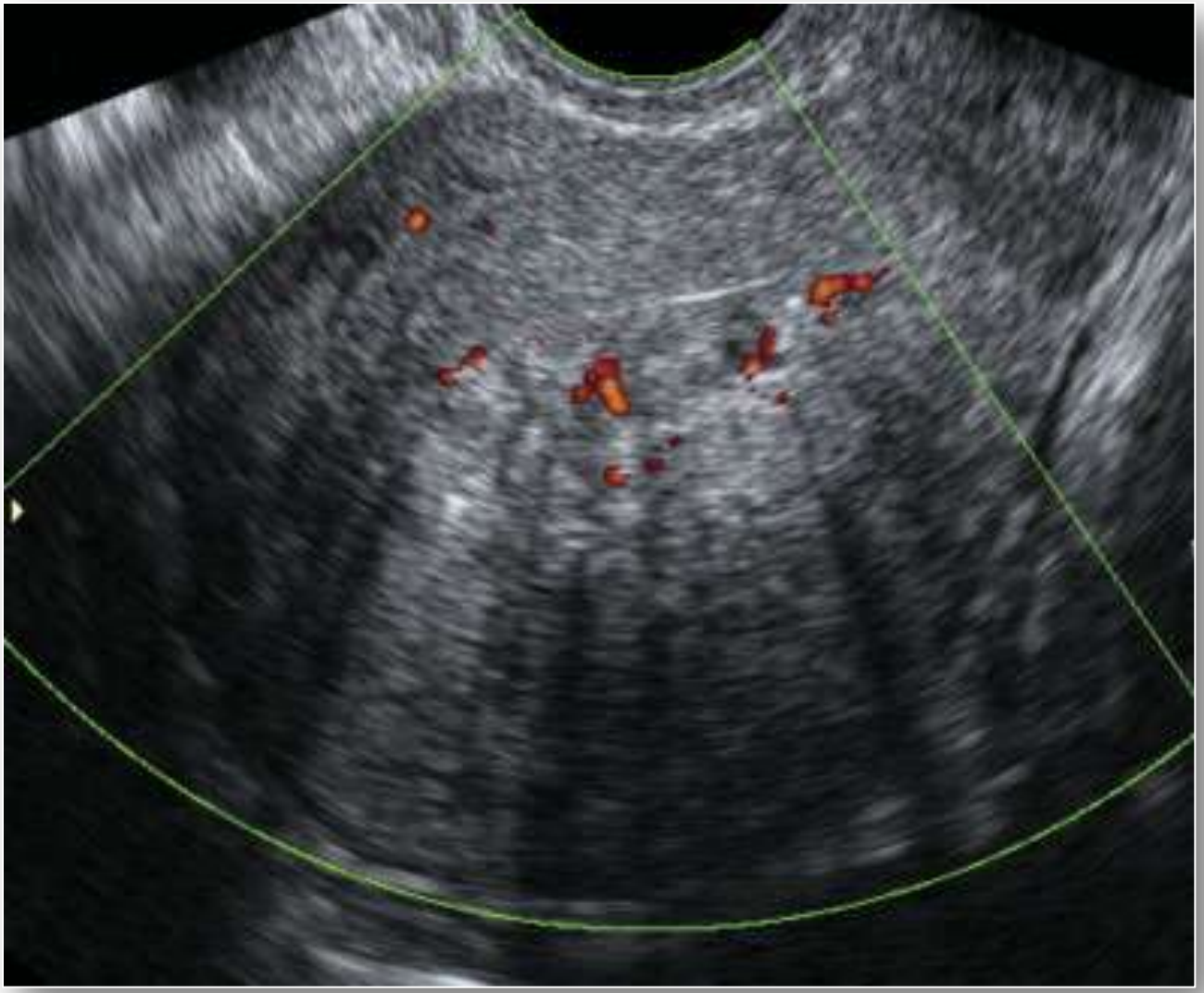
Hyperechoic endometrial tissue penetrating into the inner myometrium

Echogenic linear striations

Linear shadows



Few diffuse vessels



Lateral infiltration of the junctional zone