endometriosis

This lectured is prepared by
Professor Nadia Saddam Fahad AL-Assady
CABOG-FIBOG
Consultant Obstetrician and Gynecologist

ILO

- K:1,4,5
- S:1,3,4,7,13, 17, 18, 21, 23, 24
- AB: 1, 3, 4, 5, 8, 9
- Spcefic objective
- To define endometriosis
- What theory behinds its etiology
- How to manage endometriosis
- What is the Adenomyosis

Endometriosis:

It is a medical condition in which tissues similar to normal endometrial tissues in structure and function found in sites outside the uterine cavity.

Sites :

a-pelvis b-ovary c-pelvic peritoneum

d-fallopian tubes e-broad ligament

f-uterosacral ligament g-umbilicus

h-abdominal scars i-nasals passages j-pleural cavity

Fate:

The ectopic endometrial tissues response to cyclic changes from ovarian hormones so at each menstrual cycle endometrial deposit proliferate then break down and the bleeding cause local inflammatory reaction followed over prolonged period of time by fibrosis, chronic repetition of this process disrupt and distort the affected tissues and typically dense scar tissues and adhesion may form.

Etiology:

It is still unknown but there are many theories and there is no single theory to explain its occurrence.

1-menstrual regurgitation and implantation theory:

Sampson's implantation theory postulates retrograde menstrual regurgitation of viable endometrial glands and tissue within the menstrual fluid and subsequent implantation on the peritoneal surface.

2-coelomic epithelium transformation theory:

Meyer's coelomic metaplasia theory describes the de-differentiation of peritoneal cells lining the mullerian duct back to their primitive origin which then transform into endometrial cells. This transformation into endometrial cells maybe due to hormonal stimuli or inflammatory irritation.

3- genetic and immunological factors:

It has been suggested that genetic and immunological factors may alter the susceptibility of a women and allow her to develop endometriosis this appears to be an increased incidence in first degree relatives of patients with this disorders and racial differences, with increased incidence among oriental women and a low prevalence in women of Afraco - Caribbean origin.

4-vascular and lymphatic spread theory:

vascular and lymphatic embolization to distant sites has been demonstrated and explains the rare finding of endometriosis in sites outside the peritoneal cavity.

Pathogenesis:

Endometriomas, known as a" chocolate cyst" it is a retention cysts that develop as a consequence of ovarian endometriosis. They commonly form when adhesions develop between endometriotic deposits on the ovary and the pelvic side wall or may result from inflammatory reaction to superficial ovarian lesion, leading to adhesion developing around the lesion, producing progressive inversion of the surrounding cortex. Endometrioma may be multiple and very large and they interfere with fertility by adhesion and distortion the fallopian tubes

In some women the endometriotic lesions affecting the utero sacral ligaments, marked fibrosis and scaring may develop, with infiltration of active endometriotic tissue into the rectovaginal septum. Dense adhesion involving the rectum may lead to partial or complete obliteration of the pouch of Douglas. Both processes associated with the development of tender nodules that easily palpable on vaginal examination and are associated with bowel symptoms. Deep nodular lesions may also be visible as small tender bluish cysts in the posterior fornix so called deep infiltrating endometriosis may present on the uterovesical fold leading to bladder involvement.

Incidence :

It is occur in [1-2%]of women of reproductive age

***Clinical feature*:**

It is usually occur in the women throughout their reproductive age, they are nulliparous and delayed their marriage , the classical feature are severe cyclical non colicky pelvic pain around the time of menstruation, sometimes associated with heavy menstrual loss. Symptoms may begin few days before menses starts until the end of menses. It is well recognized that there is poor correlation between extent of disease and the intensity of symptoms. Deep pain with intercourse can also indicate the presence of endometriosis in the pouch of Douglas.

Endometriosis in distant sits can cause local symptoms.

a-genital tract : dysmenorrhea, lower abdominal pain and pelvic pain, rupture / torsion endometrioma, low back pain, infertility .

b-urinary tract: cyclical haematuria, dysuria.

c-GIT: dyschezia [pain on defecation], cyclical rectal bleeding.

d-surgical scar / umbilicus: cyclical pain, bleeding.

e-nose and lung: cyclical epistaxis, cyclical haemoptysis and haemo pneumothorax .

physical signs:

1-hard thick nodules of variable size detected on bimanual examination in the utero sacral ligament, the pouch of Douglas, recto vaginal wall. 2-the uterus may be fixed in retroversion.
3-enlarge ovaries when endometerioma found.
4-speculum examination reveal bluish thick nodules in the posterior fornix , cervical excitation positive and there is adnexial tenderness.

D- DX:

1-adenomyosis 2-PID 3-carcinoma of ovary 4-carcinoma of rectum and colon. 5-pelvic congestion syndrome

Investigation:

a-U/S: detect endometrioma or chocolate cyst, in smaller lesion U/S is of limited value but the use of it is reassurance by excluding other gross diseases.

b-MRI: can detect lesions [>5mm] in size specially in deep tissues, for example rectovaginal septum.

c-laparoscopy: the most valuable tools for both diagnosis and treatment and can allow biopsy taken.

d-CA-125: no evidence that this test is useful as screening test but levels is likely to be raise in severe disease.

Treatment:

The aim of treatment is to relieve pain, control abnormal bleeding and promote pregnancy.

Prophylaxis:

In patient with family history of disease who seek advice we givingCOCPs to prevent progression of mild disease to severe one that cause infertility and advise for early pregnancy rather late one in view of family risk for progression of disease.

Medical management of pelvic pain associated with endometriosis:

a-NSAID: this offer a non hormonal approach and it useful in women want to conceive and are helpful in reducing the severity of dysmenorrhea and pelvic pain.

b-COCPs: it is can be used for both diagnostic and therapeutic purposes COCPs should be taken continuously for initial [6 months] to create a pseudo pregnancy and render the patient amenorrhoeic , if symptoms of cyclical pelvic pain disappear [in the absence of any gross lesion on U/S e.g endometrioma] the diagnosis is one of the minimal / mild endometriosis. If the symptoms persist then there is likely to be coexisting irritable bowel disease/ constipation which require its own treatment, if there is symptomatic relief with the continuous use of COCPs, then this therapy should be continued for several years or even longer until pregnancy is intended.

c-progestogen: they are given continuously and at high dosage inhibit ovulation and affect endometriotic implants causing decidualizationand atrophy.

both oral medroxyprogesterone acetate[100 mg daily] and depot MPA [depo provera 150 mg 3-monthly] were affective in relieving pain symptoms, the most commonly reported side affect is breakthrough bleeding others include weight gain, breast tenderness, bloating, headache and nausea. The use of levonorgestrel intrauterine system [LNG-IUS] has been affective in achieving long term therapy affect.

d-gestrinone: it is [19- nor testosterone] derivative that has progestgenic and antiprogestgenic action, at dose of [2.5mg] twice weekly for 6 months] but androgenic and hypo oestrogenic side effect are less frequent, however, there is lack of information relating to its safety for long term use.

e-danazol: it is [17-ethinyl testosterone] it is act both centrally and locally to suppress steroid genesis and induce endometrial atrophy. The dose is [400-600 mg] daily, the side effect is weight gain, limb tingling, acne, greasy skin, hirsutism, deepening of the voice and atherogenic effects on lipid profile but danazol is not suitable forlong term use.

f-GnRH analogues: are derived from native hypothalamic GnRH by peptide substitutions that increase their potency and duration of action. Both agonist analogues and antagonist have been developed, of which agonist was in established clinical practice for much longer. The antagonists act by competitive inhibition of pituitary GnRH receptors, with a rapid onset of action, whereas the agonists cause initial stimulation of gonadotrophin production followed by prolonged down regulation. The antagonists have clear advantage over the agonists for short term pituitary suppression e.g during super ovulation prior to IVF but are unlikely to take over from agonists for longer term indications such as the management of endometriosis.

Down regulation of pituitary GnRH receptors by GnRH agonists leads to inhibition of FSH and LH production and gonadal suppression. The administration of GnRH agonists is either by

a-intranasal spray: Nafarelin [200mcg]bd , Buserelin [200mcg]tid . b-depot injection: Goserelin [3.6mg s.c], Leuprorelin[3.75mg i.m ors.c]

the side effects includes:

a-hot flash b-insomnia c-vaginal dryness d-reduce of libido e-headaches

longer term use of GnRH agonists cause loss of bone mineral density and it is of major concern for this reason these should not used as single agents for longer than 6 months. In women needing longer term treatment, hormonal add back therapy can be used to reduce the bone loss and prevent unwanted side effects. Examples of add back therapy a-transdermal oestradiol [2.5mcg twice weekly] +oral MPA [5mg]daily. b-oral oestradiol [2mg]daily +norethisterone [5mg]daily . c-conjugated equine oestrogen [0.625mg] daily+ norethisterone [5mg].

Surgical management of pelvic pain associated with endometriosis

1-laproscopic local ablation by diathermy, laser vaporization or excision & adhesiolysis .
2-laproscopic uterosacral nerve ablation.
3-ovarain endometrioma excision, if large and severe unilateral oopherectomy, in severe bilateral disease bilateral oopherectomy with preservation of the uterus for donor oocytes if want pregnancy. 4-TAH+BSO if patient complete her family .

Endometriosis and infertility :

It is estimated that between [30 -40%] of patients with Endometriosis have difficulty in conceiving , in many patients there is a multifactorial pathogenesis to this sub fertility. It has yet to be shown how the presence of a few small endometriotic deposits might render a patient subfertile. In the more severe stages of Endometriosis, there is anatomical distortion with peri adnexal adhesions and destruction of ovarian tissue when endometrioma develop.

- Medical treatment of Endometriosis does not improve fertility and should not be given to patients wishing to conceive. However, surgical ablation/excision of minimal and mild Endometriosis improve fertility chances. Surgical treatment of endometriomas increases spontaneous pregnancy rates, including IVF success rates. The possible mechanisms are:
- a-ovarian function: luteolysis caused by PGF2, Oocyte maturation defects, endocrinopathies,Luteinized unrupture follicle syndrome, altered prolactin release and anovulation.

- b-tubal function: impaired fimbrial oocyte pick up, altered tubal mobility.
- c-coital function: deep dyspareunia-reduce coital frequency.
- d-sperm function: antibodies causing inactivation, macrophages phagocytosis of spermatozoa.
- e-early pregnancy failure: PG induced, immune reaction, luteal phase deficiency.

- Adenomyosis :
- Definition : it is characterized by the presence of endometrial glands & stroma in the myometrium with adjacent smooth muscle hyperplasia.
- Incidence : The incidence of Adenomyosis is unknown, it is present in (15-30%) of hysterectomy specimens. It is common in multiparous middle aged women.

Etiology:

The cause remains speculative, but the adenomyotic tissues is presumed to be derived from the endometrium. It may be triggered by a weakness in the smooth muscle of myometrium, by increased intrauterine pressure or by surgical trauma. The incidence is increased with increasing parity, a history of miscarriage, induced abortion & C/S. it is decreased in smokers compared with non-smokers. The relationship between the presence of adenomyosis & both endometrial hyperplasia & uterine fibroids has been reported, but this may be related to the age & symptomatology of the women

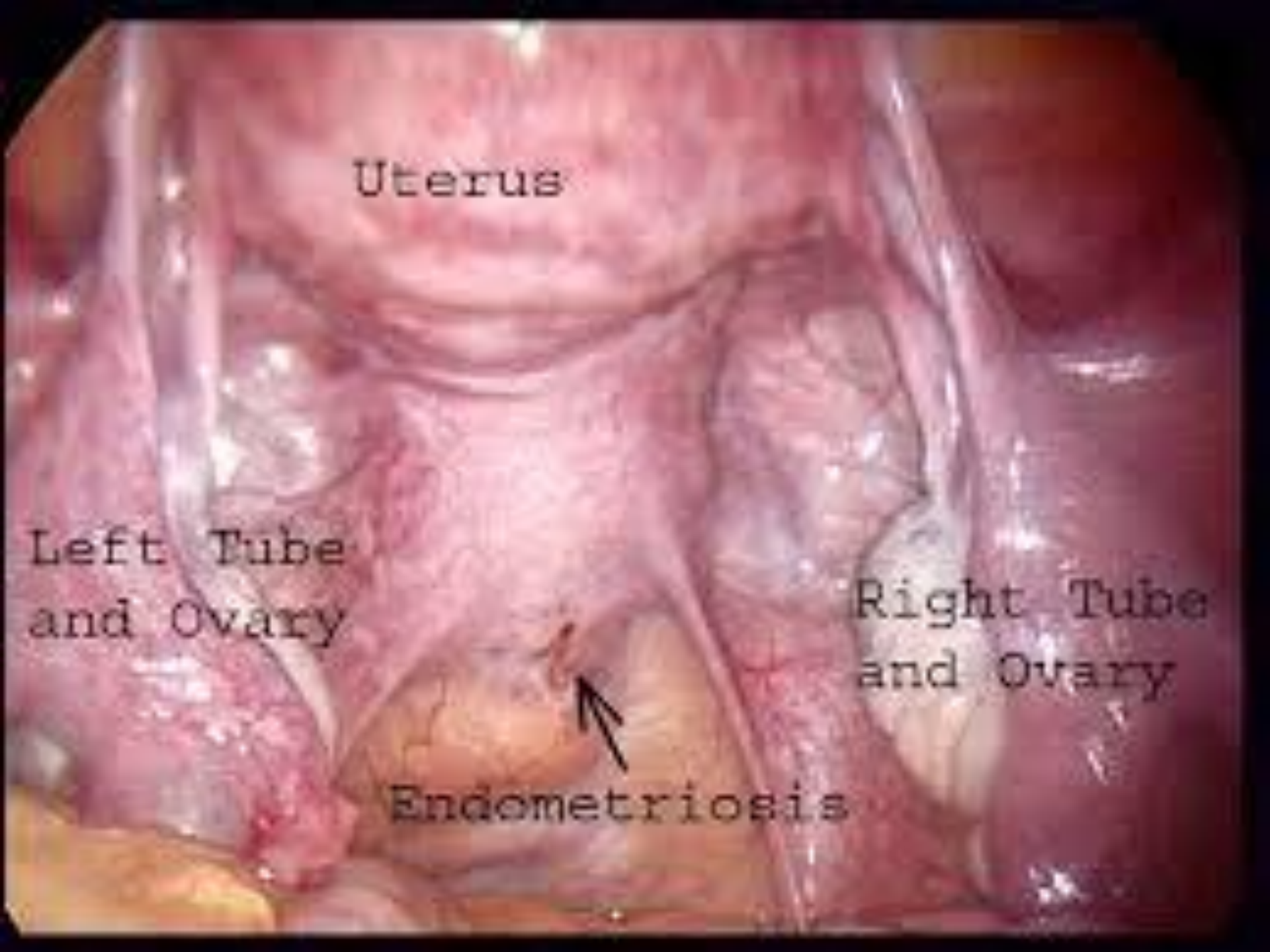
- Clinical feature :
- The women usually present with menorrhagia & dysmenorrheoa, clinically the uterus may be bulky & tender but both history & examination are very nonspecific.
- Investigation :
- 1-TVS : it is used as primary screening modality for the diagnosis & the sonographic feature includes : diffuse echogenicity, myometrial cysts, sub endometrial nodules, sub endometrial linear striations, poor definition of endometrial / myometrial border & asymmetric myometrial thickening.

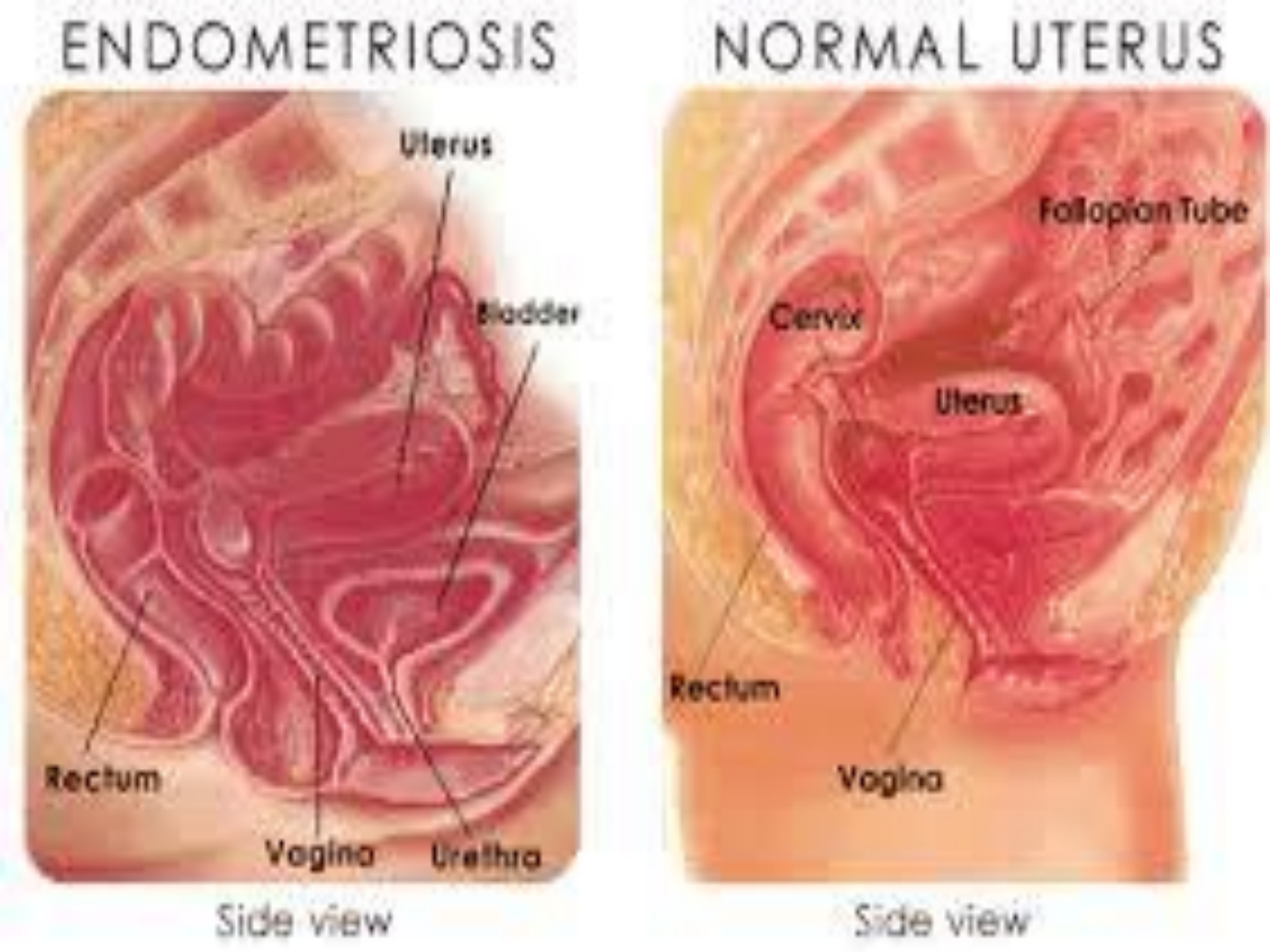
- 2-MRI : because TVS lack specificity, especially in distinguishing adenomyosis from fibroids so MRI should be used, but both techniques even when used in combination, may lack accuracy for evaluation of very large uteri with a volume greater than (400 ml).
- 3-hystroscopic & laparoscopic myometrial biopsy have been described but have limitation when compared to non-invasive methods.

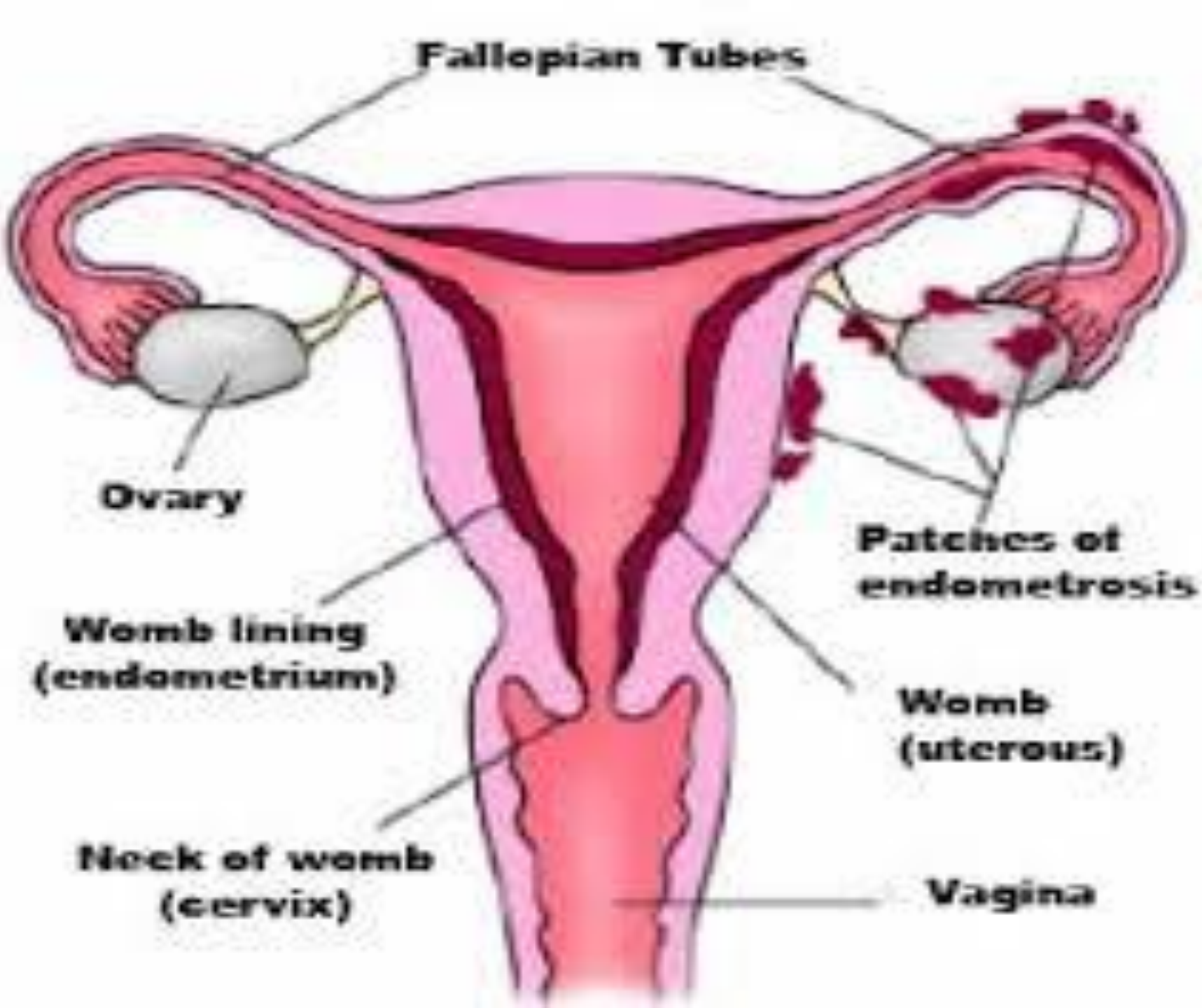
- Treatment :

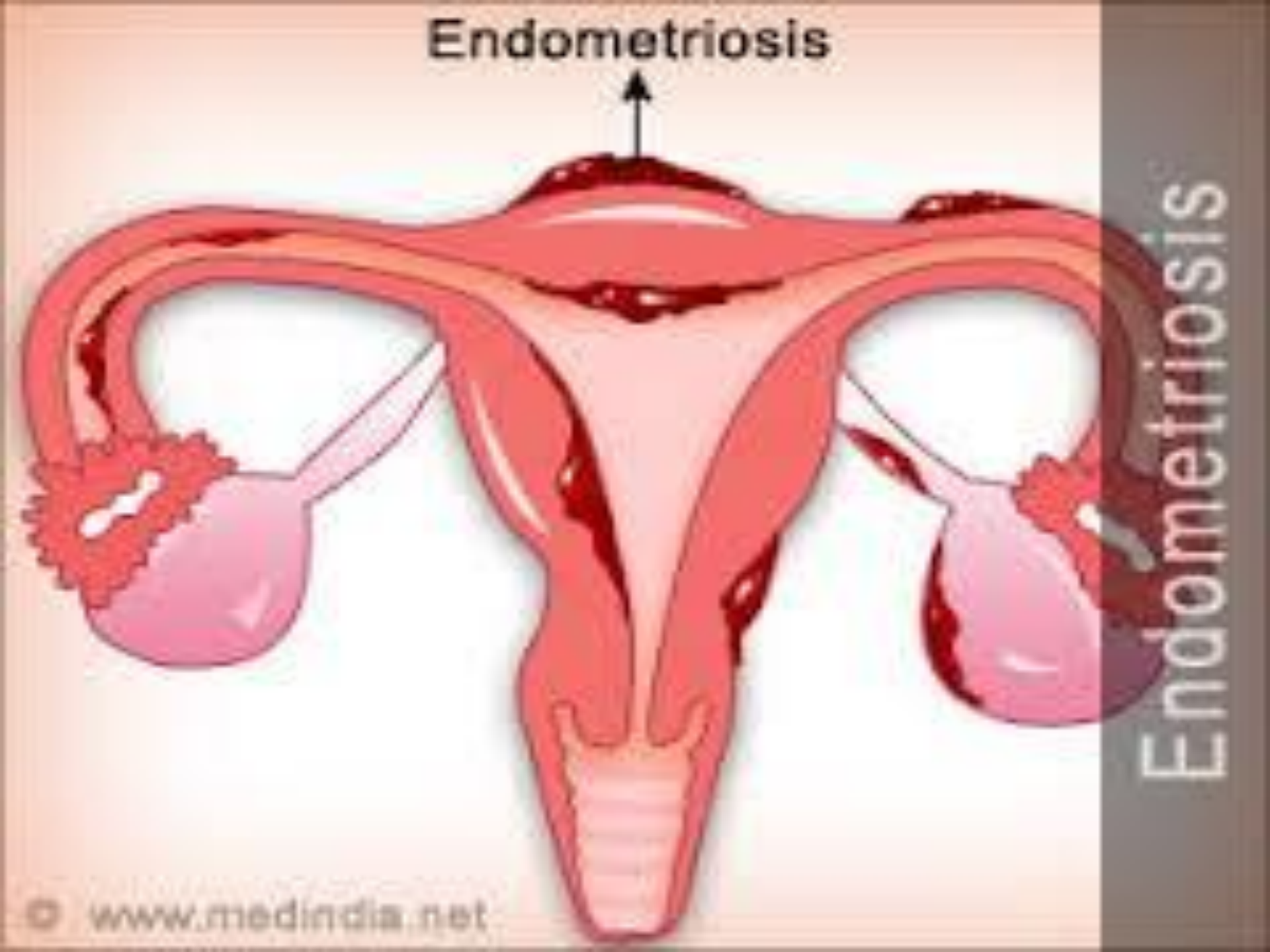
1-medical treatment: the current medical treatment of menstrual disorders includes NSAIDs, COCP, high dose progestogens & LNG-IUS. As most of these therapies are affective in the management of menorrhagia, dysmenorrhea & endometriosis they should theoretically be beneficial for adenomyosis, but only LNG-IUS is found to be affective & should be used as first line management

- 2-surgical treatment: there is some evidence that the presence of deep lesions of adenomyosis is associated with failure of endometrial ablation resulting in both regeneration of the endometrium & glandular activity within the myometrium. However at this stage it may not be possible to distinguish between preexisting & iatrogenic lesions. On the current evidence the use of LNG-IUS may preferred to endometrial ablation but the definitive treatment to adenomyosis is hysterectomy & need not be accompanied by oophorectomy unless there is a specific indication for it.

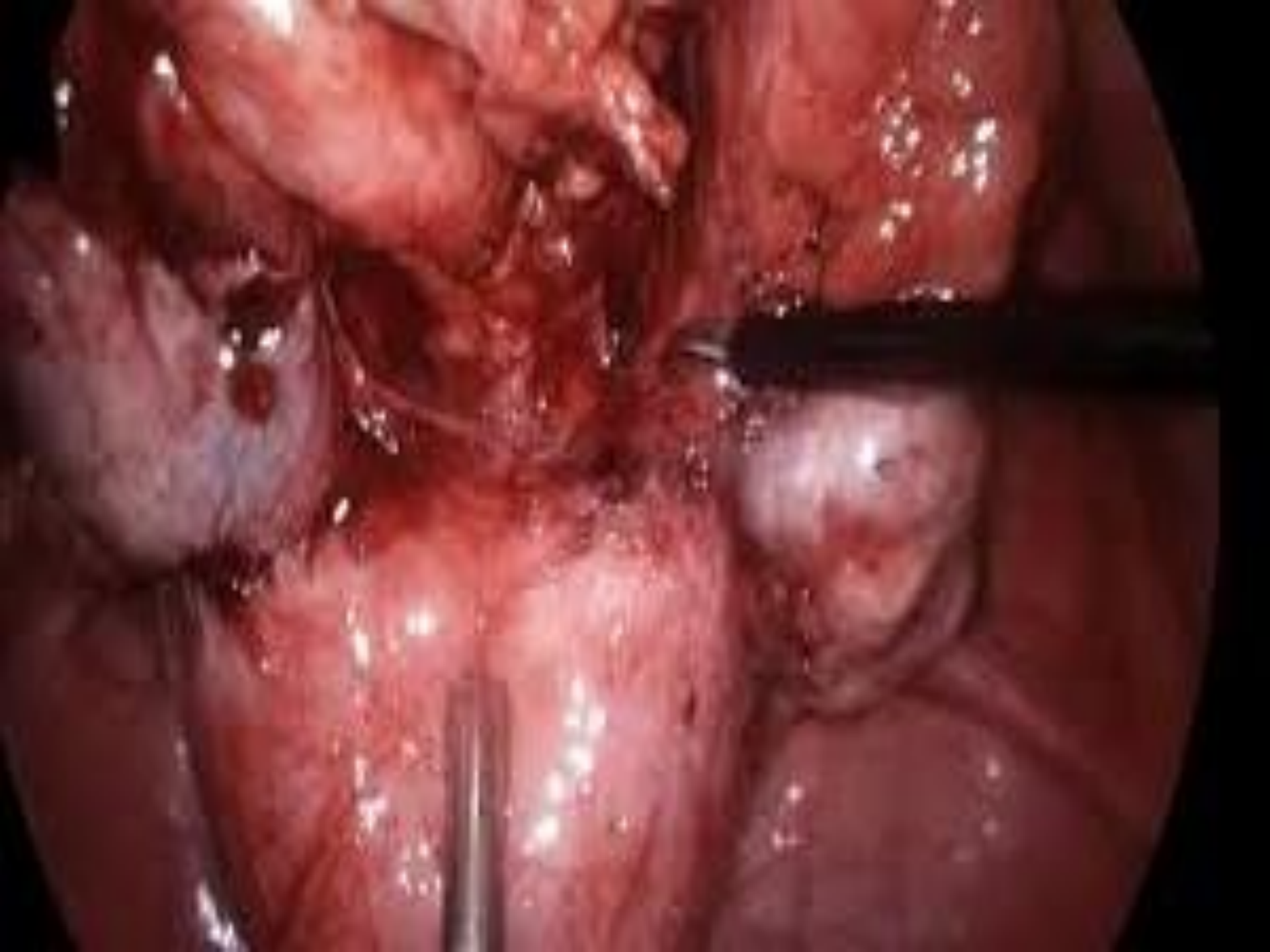

Uterus
Left Tube
and Ovary
Right Tube
and Ovary
Endometriosis

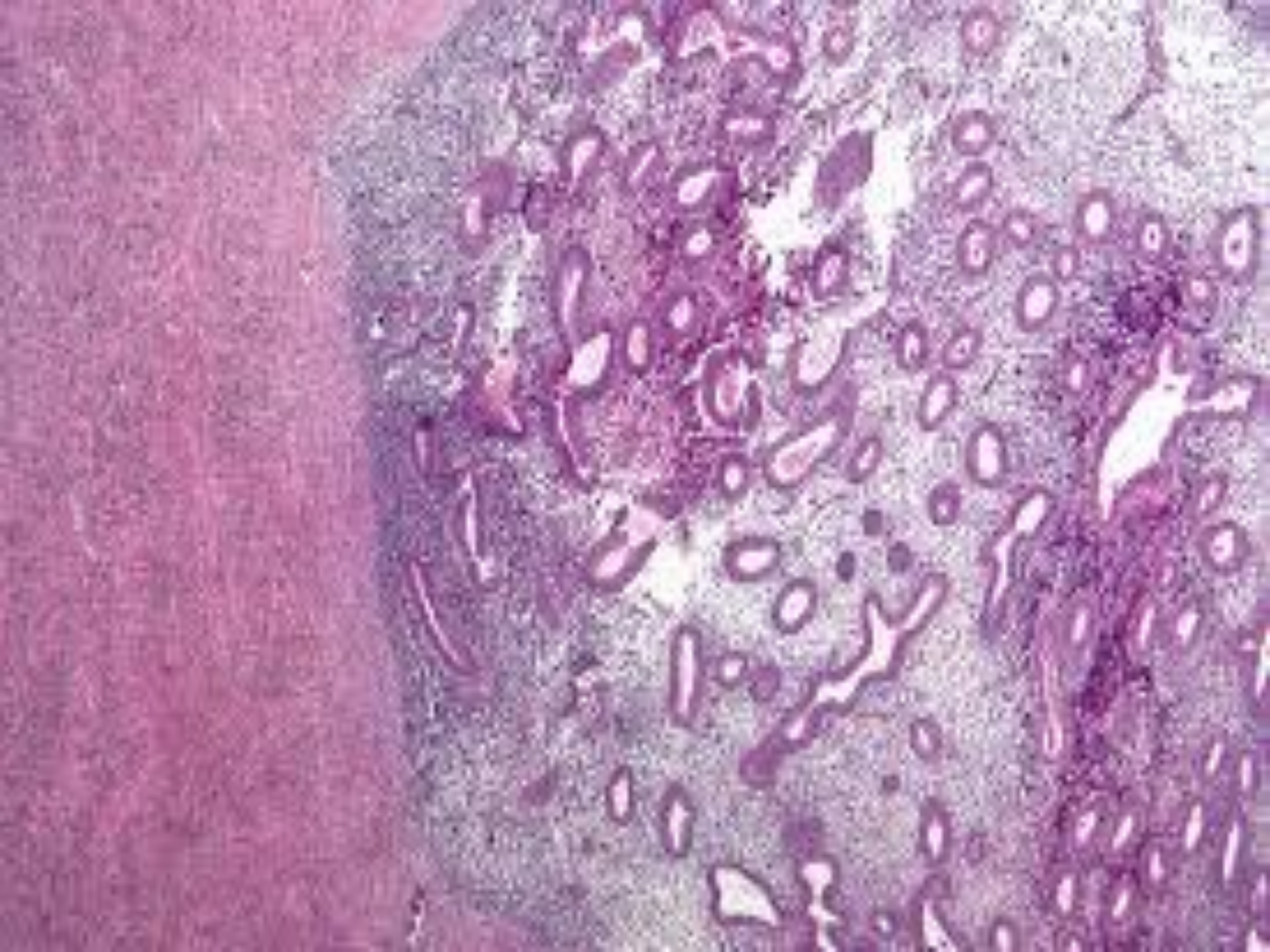

ENDOMETRIOSIS
Uterus
Bladder
Rectum
Vagina
Urethra
Side view
NORMAL UTERUS
Fallopian Tube
Cervix
Uterus
Rectum
Vagina
Side view

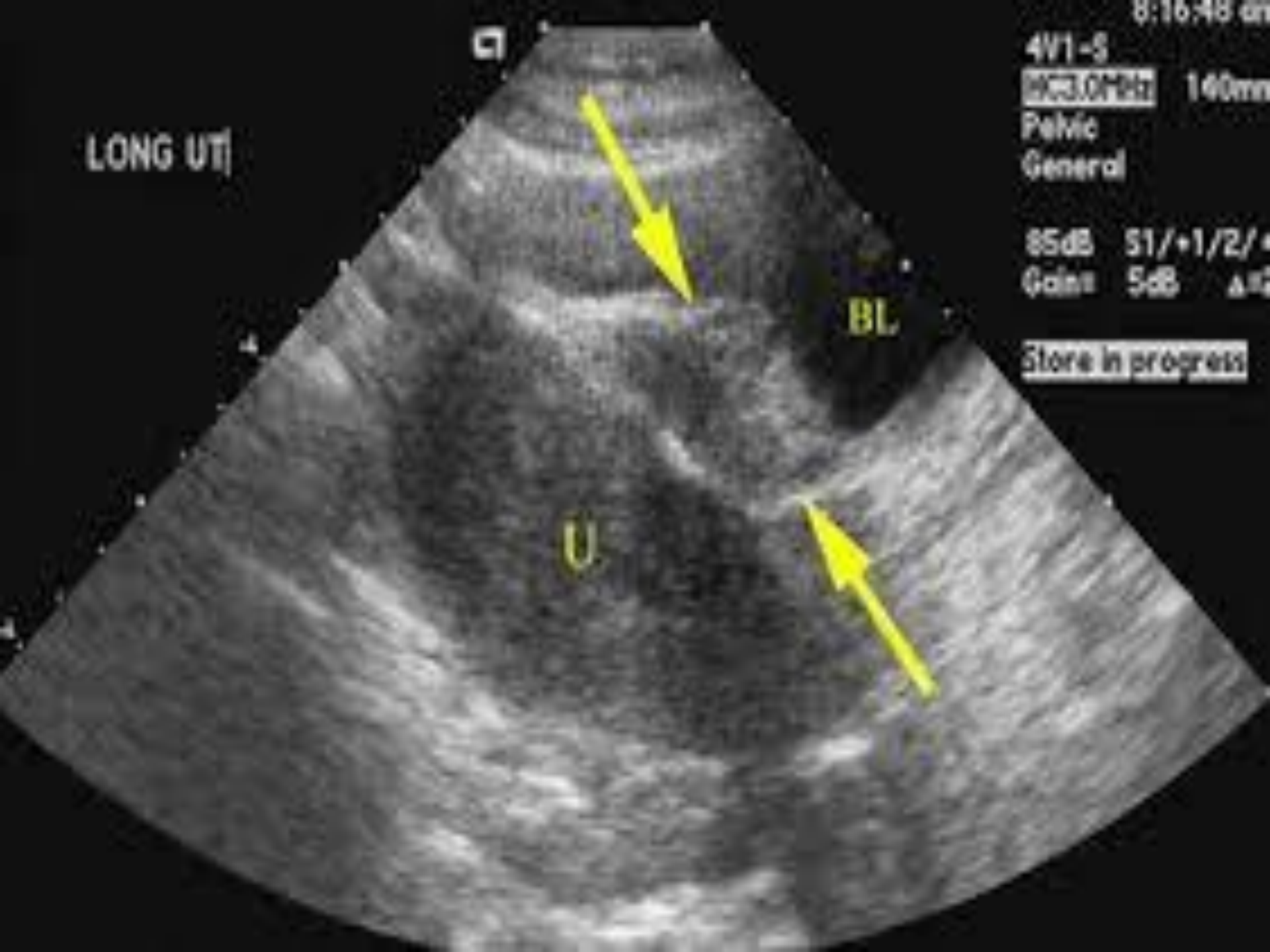

Fallopian Tubes
Ovary
Patches of endometrosis
Womb lining (endometrium)
Womb (uterous)
Neck of womb (cervix)
Vagina

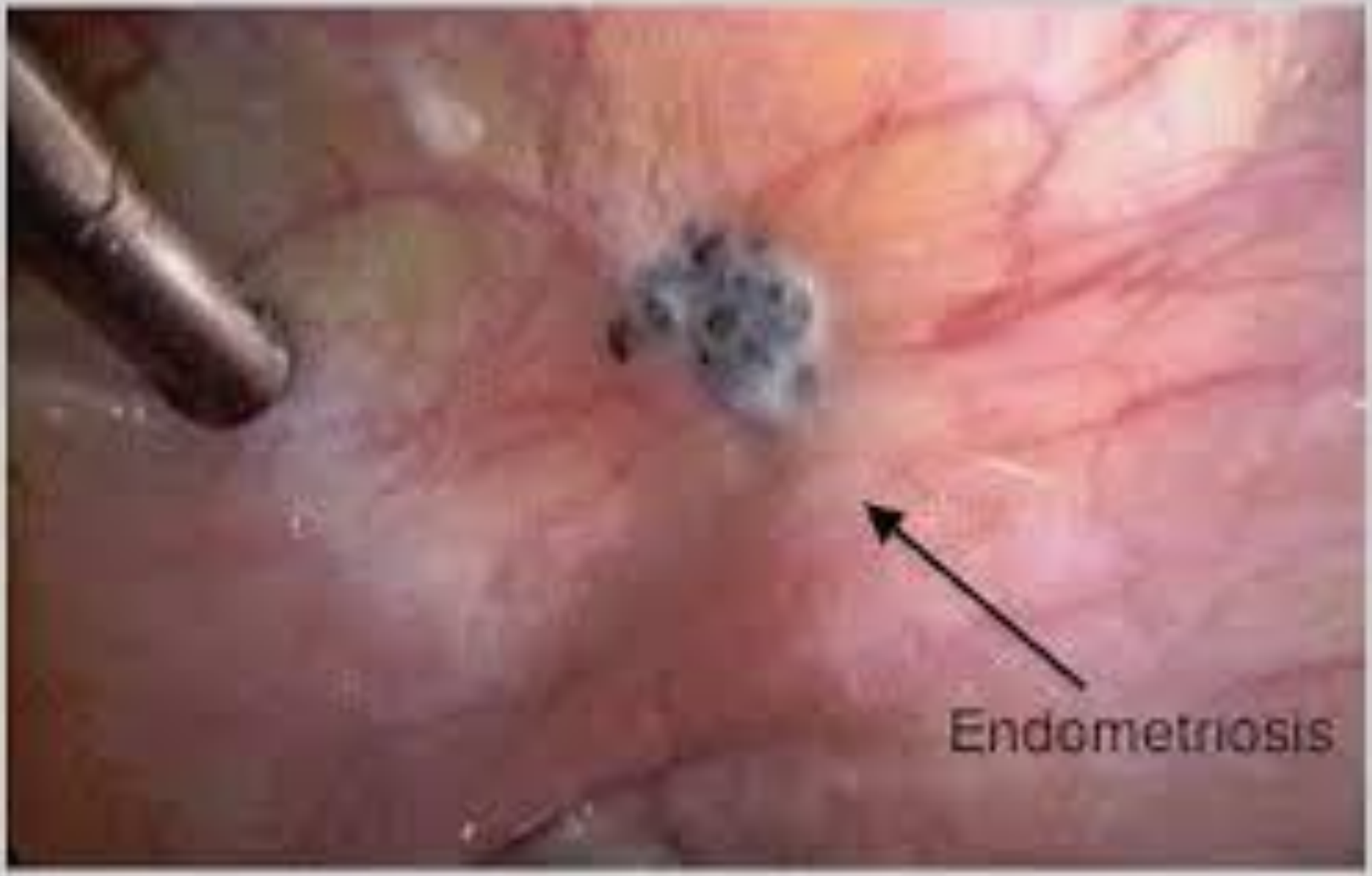

Endometriosis
Endometriosis
© www.medindia.net


Fallopian tubes
Ovary
Endometriosis
Womb lining
(endometrium)
Womb (uterus)
Neck of womb
(cervix)
Vagina

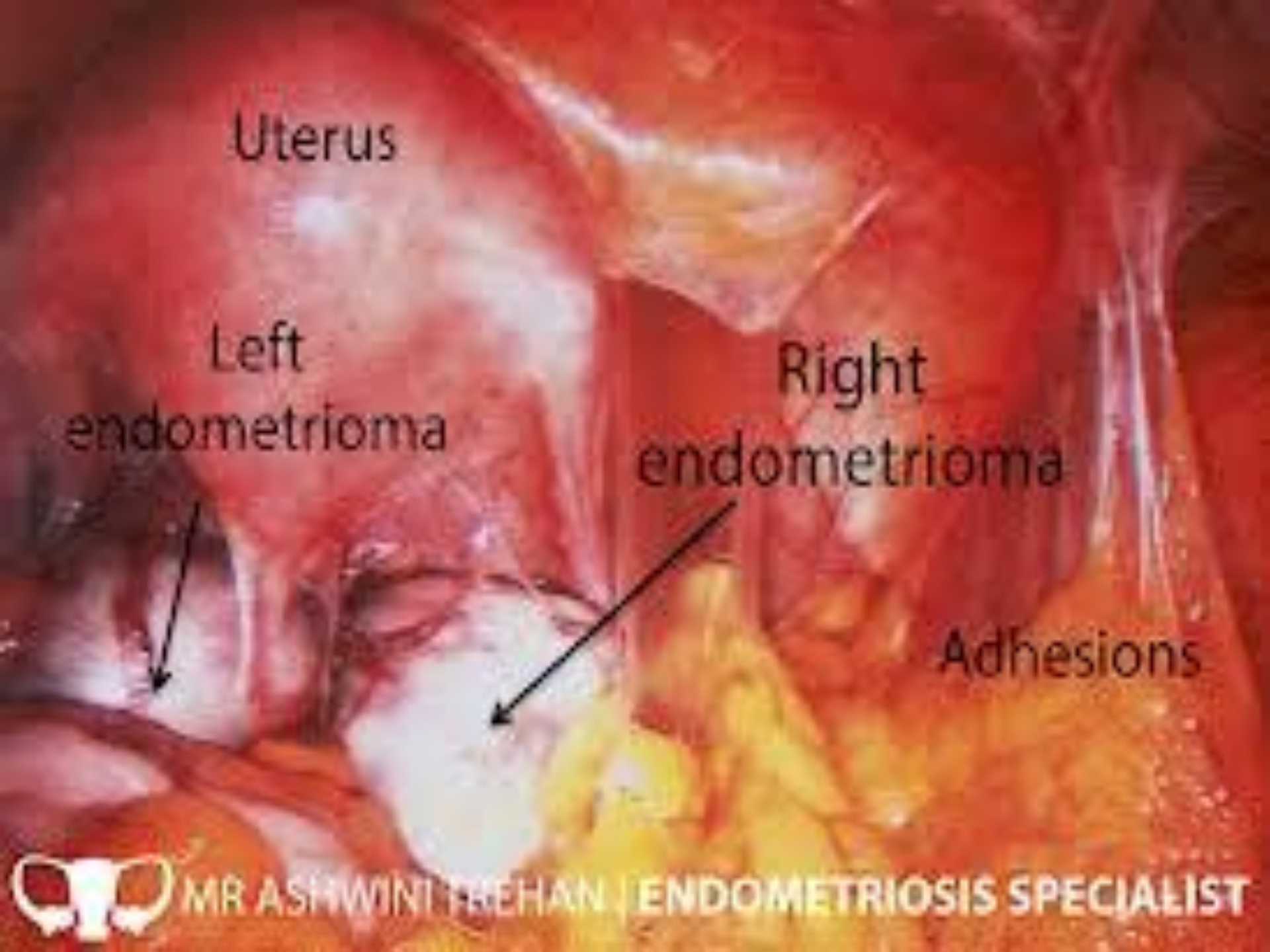


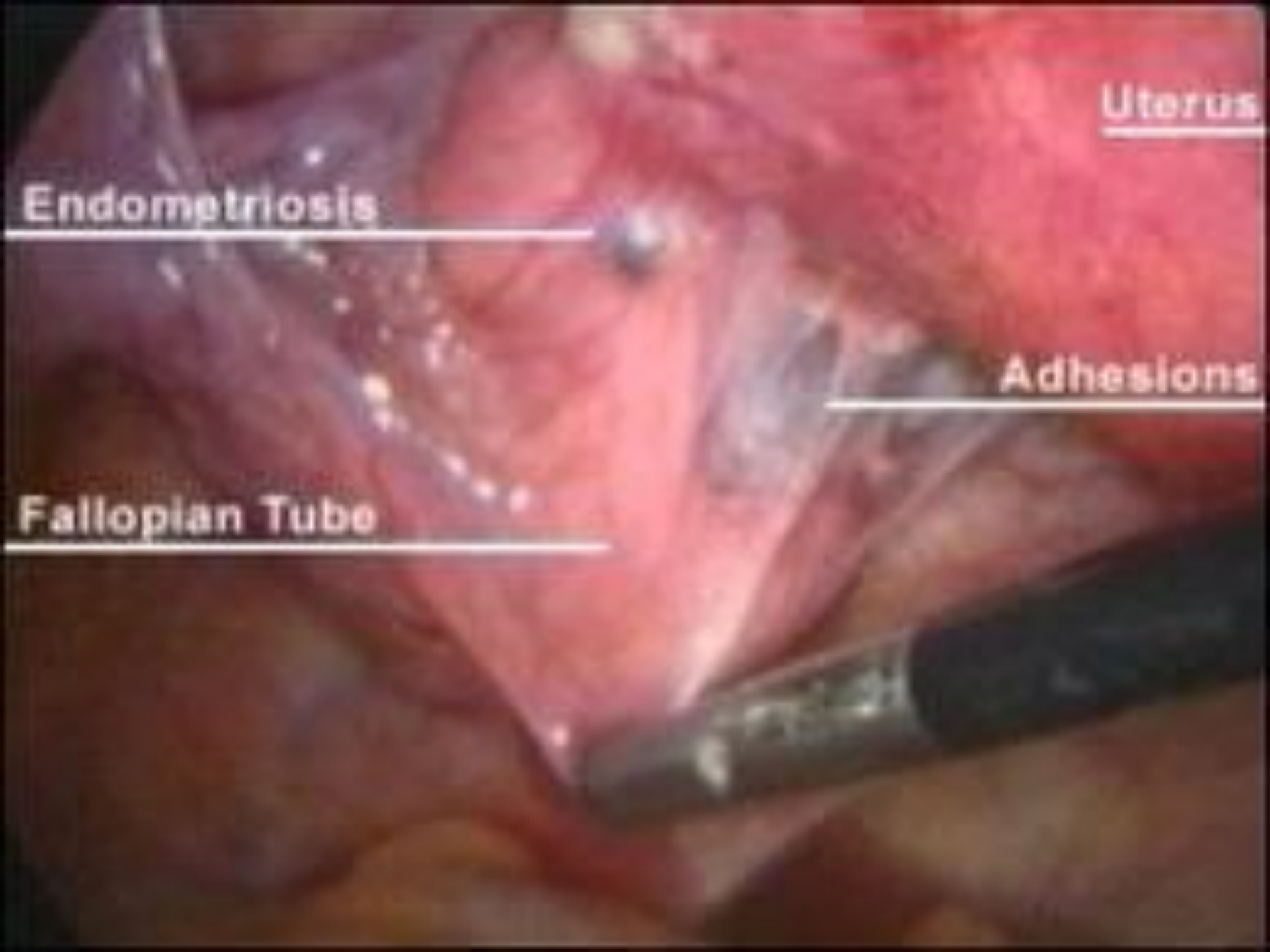



LONG UT
BL
U
4V1-3
Pelvic
General
85dB
Gain 5dB
Store in progress

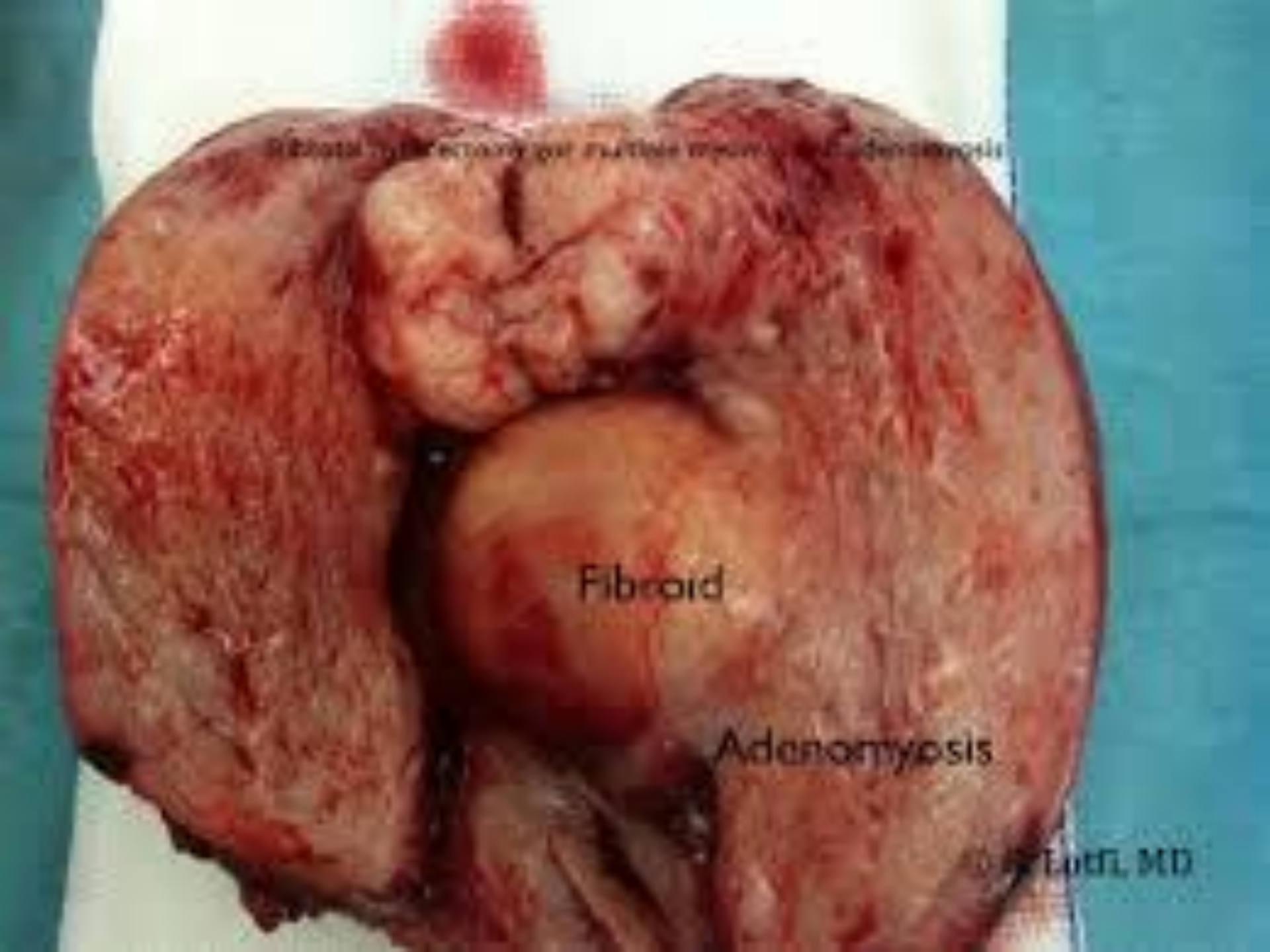


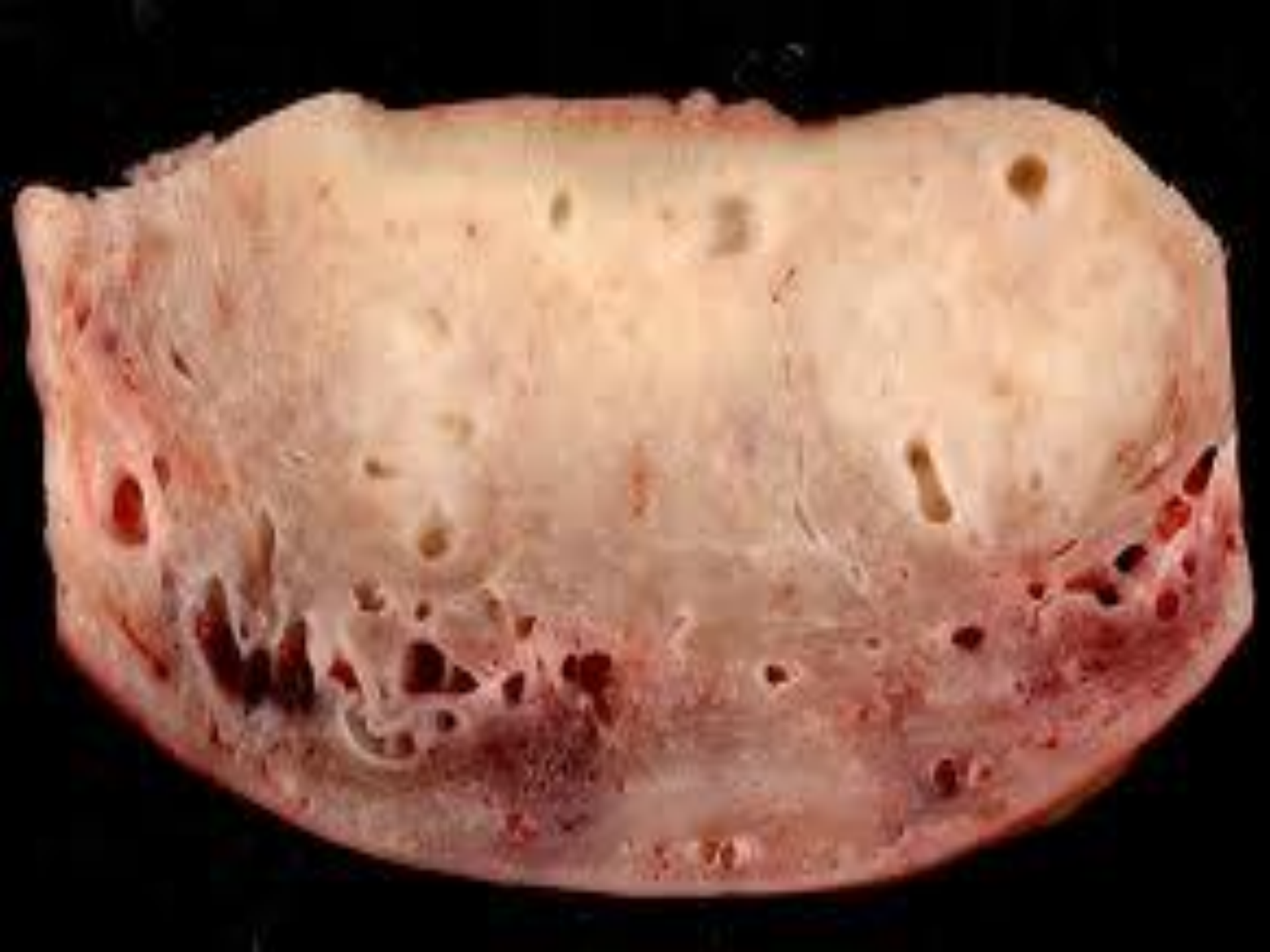
Endometriosis

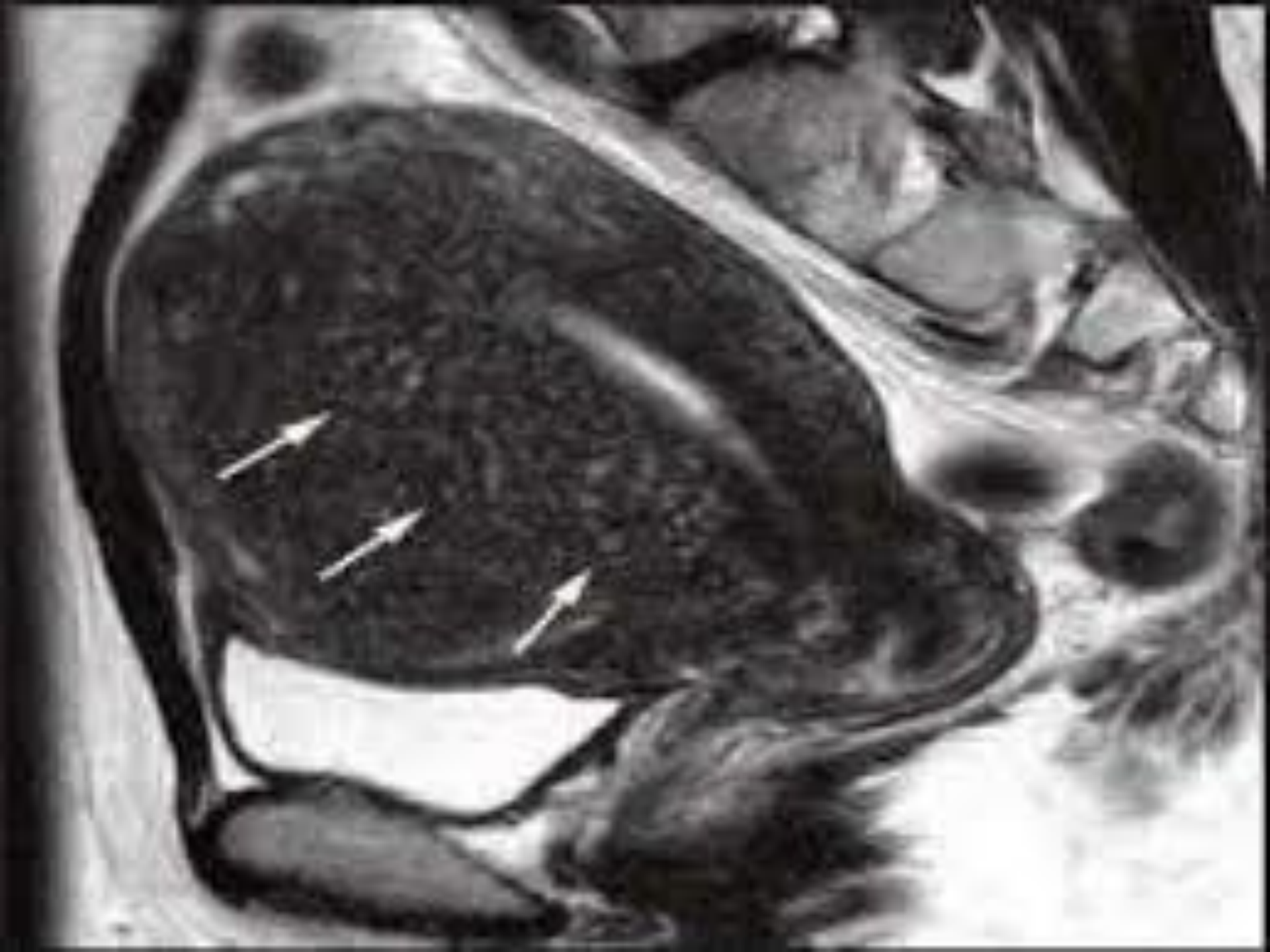

Uterus
Left endometrioma
Right endometrioma
Adhesions
MR ASHWINI REHAN | ENDOMETRIOSIS SPECIALIST

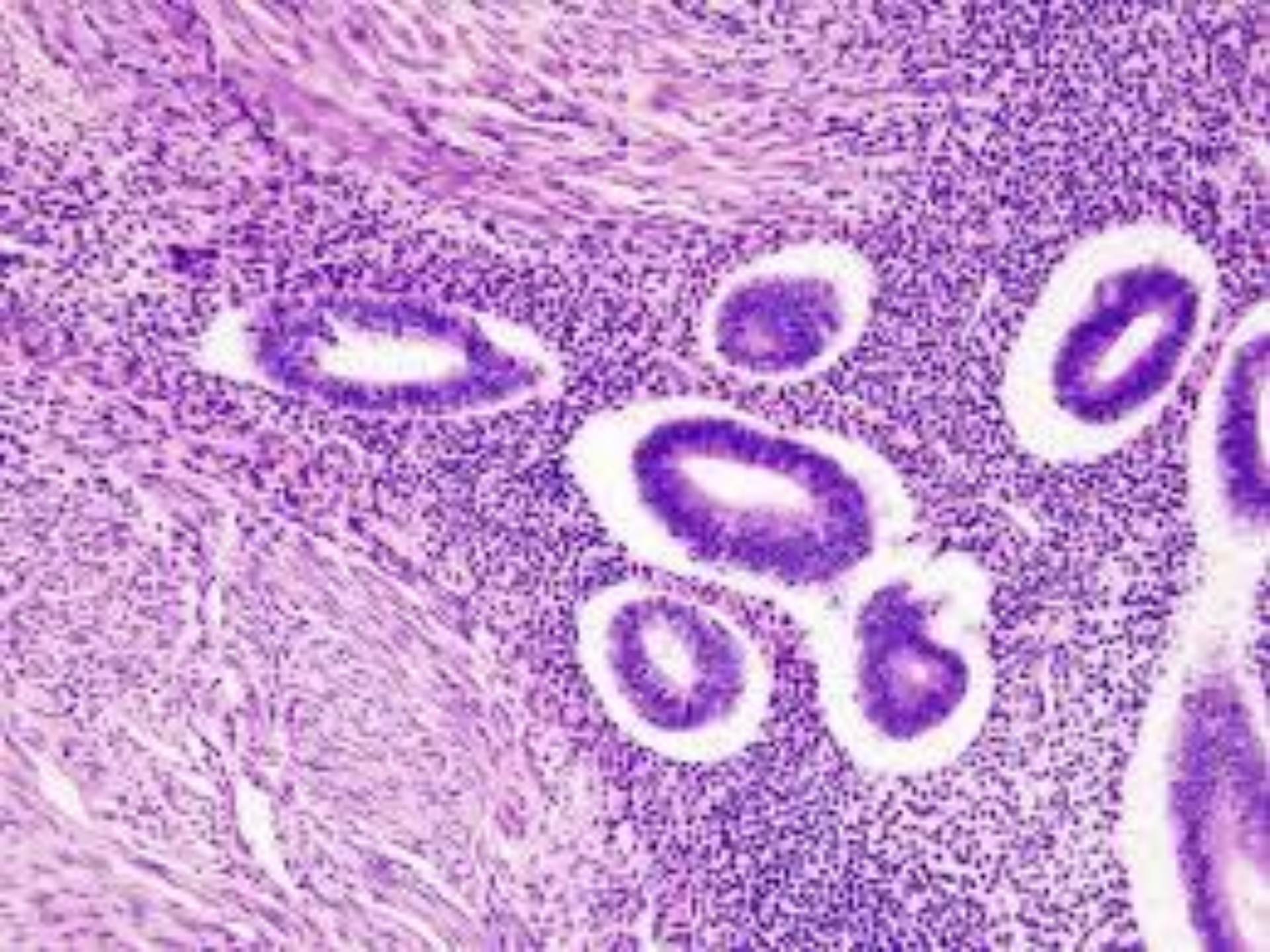

Uterus
Endometriosis
Adhesions
Fallopian Tube

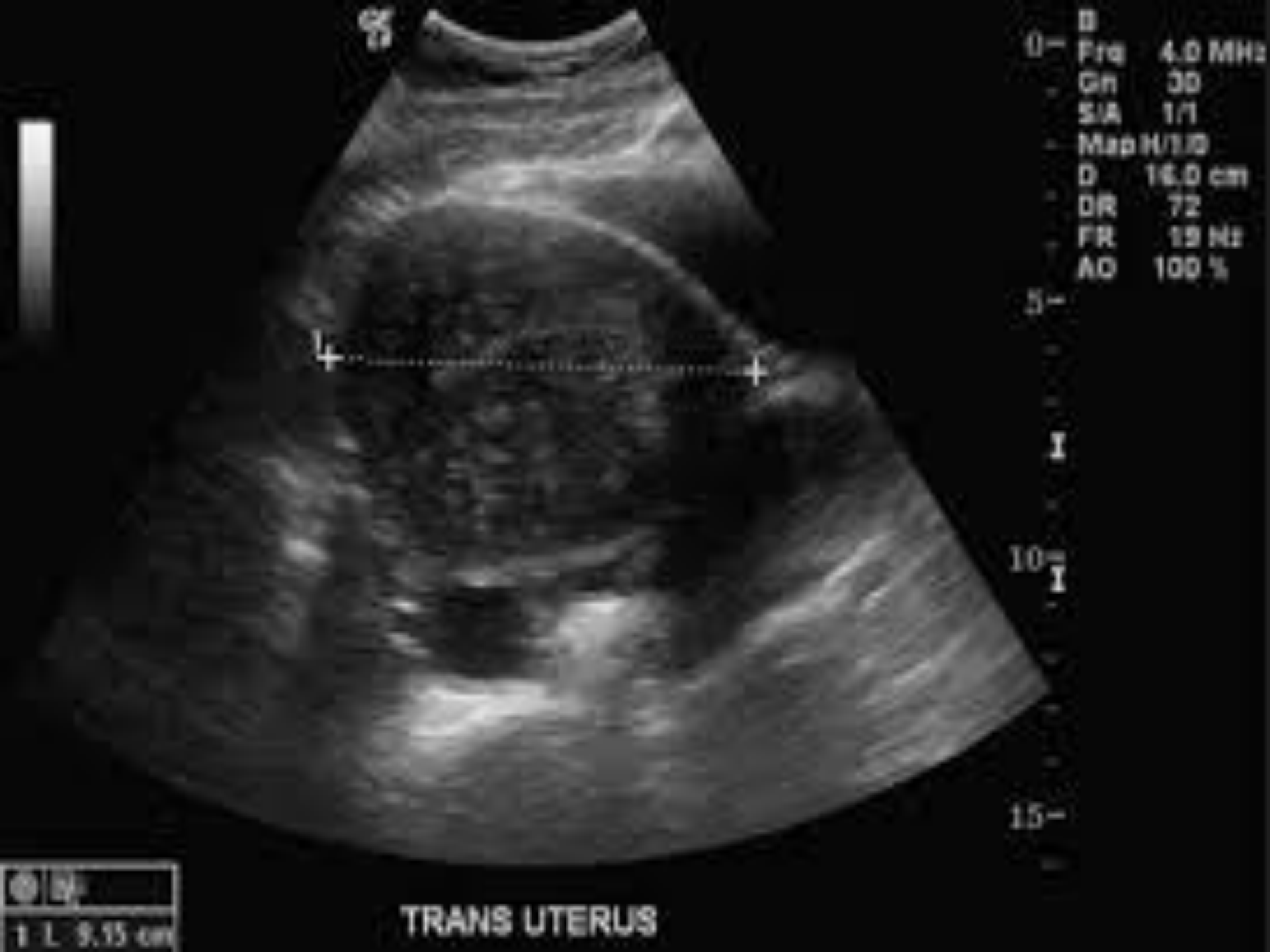
Comparison of Normal Uterus with Adenomyosis


Fibroid
Adenomyosis





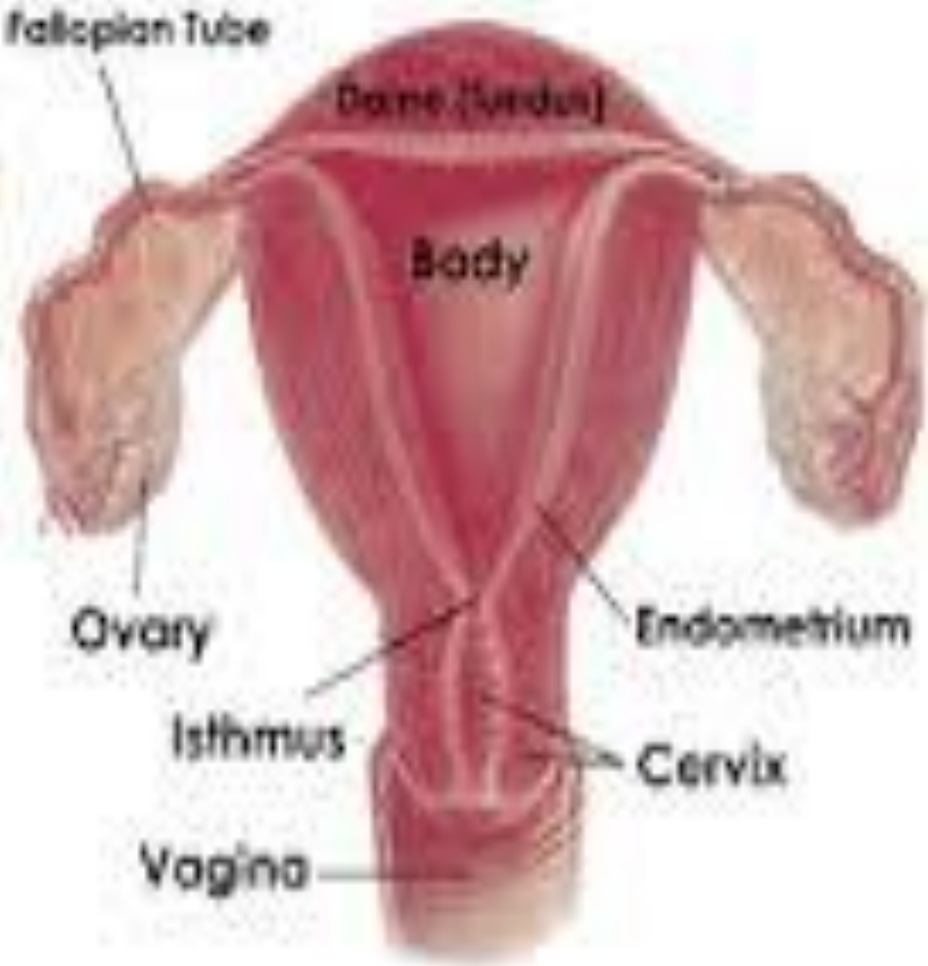



B
Frq 4.0 MHz
Gn 30
S/A 1/1
Map H/1/D
D 16.0 cm
DR 72
FR 19 Hz
AD 100 %
0
5
10
15
TRANS UTERUS

ADENOMYOSIS

Front view - Adenomyosis

NORMAL UTERUS



Front view of healthy uterus

Thank you

Any questions