RM3.01 ENDOSCOPIC MANAGEMENT OF ENDOMETRIOSIS

RM3.01.02

EXCISION OF PELVIC ENDOMETRIOSIS

David B. Redwine, Endometriosis Institute Of Oregon, Bend, OR, United States

No medicine eradicates endometriosis, and medicine treats only the symptoms, not the disease. For these reasons, surgery is the treatment of choice. Because endometriosis can invade beneath the visible pelvic surface to varying degrees, thermal ablation with laser vaporization or electrocoagulation may leave disease incompletely treated. Excision of endometriosis gives the surgeon a better chance of completely removing all disease, including deeply invasive disease. Mature excisional techniques have been described and evaluated for endometriosis of any location and any depth of invasion. Excision can be performed with sharp scissors, monopolar or bipolar electrosurgery, or laser. Monopolar electrosurgical excision is the most versatile technique, since the scissors can be used to palpate, grasp and rearrange tissue, cut sharply, cut with electrosurgery, coagulate bleeders, blunt dissect, and retract. This eliminates most instrument changes and speeds surgery along. Excision results histologic confirmation of endometriosis which enhances the scientific process, since the visual identification of endometriosis is not always correct. The technique of monopolar electroexcision of endometriosis is simple in concept. Abnormal peritoneum is grasped and elevated away from underlying vital structures. 90 watts of pure cutting current passed down 3 mm scissors is used to incise normal peritoneum around the lesion of interest. Retroperitoneal blunt dissection is used to separate fibrosis associated with endometriosis from vital structures. Remaining tendrils of connective tissue held on stretch can then be quickly cut with electrosurgery. Parenchymal disease, such as of the uterosacral ligaments, can be quickly resected using 50 watts of coagulation current, which has a higher voltage and therefore a more powerful cut. Recurrent disease after complete excision is uncommon, calling into question Sampson's theory of origin of endometriosis. Symptom relief is excellent and fertility is not compromised by excision.

RM3.01.03

THE ENDOMETRIOTIC DISEASE THEORY

P.R. Koninckx, University Hospital Gasthuisberg, Department Obstetrics-Gynaecology, Division Endoscopic Surgery, Catholic University of Leuven, Leuven, Belgium

The Sampson and metaplasia theories consider endometriosis as normal glands and stroma outside the uterus. The key phenomenon is the implantation/metaplasia, whereas the subsequent growth and development is considered inevitable, albeit modulated by peritoneal and immune factors. The disease therefore is considered progressive and recurrent.

EDT considers retrograde menstruation of viable endometrial cells and occasional implantation of these cells a normal physiologic phenomenon. These nonimplanted or implanted cells are normally removed by the defence mechanisms of the body, such as macrophages. Attachment and implantation is favoured when the mesothelial layer is damaged by trauma, infection, or even by low-grade inflammation (e.g. irritation caused by CO2 pneumoperitoneum) or by abundant retrograde menstruation. It seems logical to postulate that by mere statistical mechanisms, attachment and implantation must occur more frequently when more viable cells are present in peritoneal fluid. Although these cells can temporarily grow and develop depending upon the environment, their ultimate fate when left alone will be a spontaneous regression. This can be complete disappearance/removal or this can result in some fibrotic or scar tissue as the remnant of local inflammation, containing eventually some endometrial cells, shielded from the bloodstream and from immunocompetent cells, comparable to the bacteria in an abscess.

ED is caused by a cellular modification, e.g. a genetic mutation, as observed in many benign tumours. This cellular accident will happen more frequently in genetically predisposed persons, and will be favoured by other factors such as total body irradiation, or chemical pollutants such as dioxins. It seems logical that the probability of such a cellular accident increases when more cells are present. The type of cellular modification, together with local factors such as the peritoneal fluid microenvironment or the intra-ovarian milieu, will determine whether they will develop into typical lesions, deep endometriosis or cystic ovarian endometriosis, and whether the morphologic characteristics will be chocolate cysts, endometrial glands and stroma, or adenomyosis externa. Typical lesions are considered either as the remnant of a normal physiologic condition, or as a benign tumour with such a low invasiveness and growth potential that the lesions remain inactive over longer periods.

The EDT has the following clinical implications. According to the implantation theory endometriosis is a recurrent disease and women in whom the diagnosis of (minimal) endometriosis is made are considered at risk for developing severe endometriosis later in life. According to the EDT, minimal endometriosis, especially subtle or nonpigmented lesions, are no longer considered a pathologic condition and these women should not be considered as having an increased risk to develop severe endometriosis. Since the EDT considers endometriotic disease as a benign tumour, complete surgical excision would eradicate the disease with no risk of recurrence. The actual data showing low recurrence rates after excision of severe and cystic ovarian endometriosis are consistent with this view. According to the EDT the increase with age and heriditary aspects can be interpreted as the probability for a cellular incident to happen.

The importance of the EDT as opposed to the Sampson and metaplasia theories, is that implantation is viewed as relatively unimportant, whereas the driving motor become the cellular changes. This obviously is crucial for research and treatment which should reorient from preventing implantation to understanding, preventing or treating cellular damage or the consequences. According to the EDT endometriosis is entering the era of tumour biology.

RM3.02 NEW METHODS OF CONTRACEPTION

RM3.02.01

NESTORONE ROD: A SECOND GENERATION SUBDERMAL IMPLANT

R. Massai[1], Moo-Young A[2], [1]Instituto Chileno de Medicina Reproductiva (ICMER), Santiago, Chile. [2]The Population Council, New York, USA.

The Nestorone (NES) implant is a second generation implant for female contraception. It is easy to insert and remove and very efficacious and safe. Its adverse effects are minimal. This implant contains NES, a potent 19 Nor-progesterone derivative, which does not affect lipoproteins and does not bind to SHBG. The steroid is not biologically active when administered by the oral route due to its rapid biotransformation by first-pass hepatic metabolism. Thus NES is ideally suited for breastfeeding and non-breastfeeding women.

In previous dose-finding studies in non-breastfeeding women, only one pregnancy was reported, and it occurred in the 24th month of use of a single implant (100 μg/d). Experience with this implant in 3576 woman-months of use demonstrated high contraceptive efficacy for nearly two years (Pearl Index: 0.34). The mode of action is primarily ovulation inhibition and secondarily, increased viscosity of the cervical mucus.

The contraceptive effectiveness of the implant was also assessed in breastfeeding women during lactation and after weaning, for up to two years. In 2195 woman-months of use, there were no pregnancies and no adverse effects on breastfeeding or infant growth and health.

In order to provide a greater margin of safety beyond two years, this implant was reformulated to deliver a higher dose of NES (125 μg/d) for a longer time. This reformulated version is currently being tested in a multicenter Phase II clinical study designed to measure its contraceptive efficacy beyond two years.

RM3.02.02

IMPLANON – A SINGLE ROD SUBDERMAL IMPLANT

J. Newton, Birmingham University/Birmingham Women`s Hospital, Birmingham, United Kingdom.

Implanon is a single rod subdermal implant containing 68mg Etonogestrel. It lasts for at least 3 years and should only be be administered by those familiar with the insertion technique.

In our centre, we have been working with this implant for the last 10 years and have published the results of the Pase 2 clinical studies of the pharmacodynamics of leeched implant system, the endometrial response in Implanon users, ultrasound and endocrine parameters, and the bleeding patterns associated with Implanon use. In addition, we participated in the multi-centre clinical tudy of Implanon use and the comparative study of Implanon versus the 6 rod Norplant system. This