Endometriosis In Adolescents

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Summary: **Endometriosis is still a nightmare for the clinicians & patients alike. Although traditionally it is a disease of the 4th decade it is by far not a rare entitity in the adolescent girls. Among Australian women with endometriosis –1 in 20 is said to be a teenage girl. Pelvic pain being the major symptom in adolescents NSAID should be tried in those who have no immediate interest in fertility. Low dose OCP should be prescribed in NSAID failure cases. Before embarking on laparoscopy & associated interventions like fulguration & laser vaporization the young girl & her parents should be carefully counselled. We should respect the emotional needs of the teenagers & their consciousness of the body image problems.**

Endometriosis continues to remain a challenge for clinicians, research scientists and patients alike. The difficulties include the problems of explanation of aetiology, pathophysiology & progression, problems of its recognition both clinically & at endoscopy; and problems of ascertaining who, when, how & for how long to treat-once its presence has been confirmed. The challenge is enhanced when the sufferer is an adolescent.

Traditionally endometriosis is said to be the disease of the fourth decade of life having a peak incidence between 30 & 45 years of age (Shaw 1995). On balance the prevalance of endometriosis among women aged 15-45 years is about 10%(Barbieri 1990). However the prevalance of endometriosis in young women may not differ greatly from that in older women (Sanfilipo et al 1994). In the adolescent groups Meigs (1948) reported a 6% incidence at laparoscopy, whereas Goldstein et al in 1980 reported that 47% of adolescents referred for chronic pelvic pain have documented endomentriosis. In a series of 717 Australian women with endometriosis studied by O'Conor et al (1987) 1 in 20 sufferers were teenagers.

A major distinction between adolescent & adult endometriosis is its development in association with partial or complete obstructive Mullerian anomalies such as cervical atresia, or obstructed rudimentary uterine horns, whereby the disease is induced by severe retrograde flow(Nunley and Kitchin 1980 Huffman, 1981 Tang et al, 1986 Ugur et al, 1995). Yet in the Australian study (O'Conor 1987) only 2 out of 35 cases had existent congenital anomalies. Goldstein (1980) found congenital anomalies in 11% of 74 teenagers.

The clinical course of endometriosis associated with a reproductive tract abnormality appears to be different from that of endometriosis associated with outflow obstruction. In the former, once the patency of outflow tract is restored, reversal of the extensive pelvic endometriosis is achieved (Sanfilipo et al, 1986)

Retrograde menstruation supports the reflux implantation theory of Sampson (1927). Research to date strongly suggests that the origin of the epithelium is endometrium rather than peritoneal mesothelium as implied by Meyer's in situ metaplasia theory. (Thomas, 1992; Oral & Arici 1997). Yet Sampson's theory cannot explain endometriosis in women with primary amenorrhoea & in distant locations. No one theory provides the total answers but it is known that genetic, endocrine & immunological factors allow the growth & spread of the endometriotic implants (Ochs & Schweppe 1995).

A familial propensity to develop endometriosis has been found. The disease occurs with a frequency of 6.9% in the first degree relatives of the patients, whereas it occurs in 1% of the female relatives of the patient's husband(Sampson et al, 1981). Thus endometriosis should be kept in mind in symptomatic adolescents wh-

have a suggestive family history.

The host response to ectopic endometrium has been recently championed as an etiological factor. The major immune alterations include (1) Increased presence of circulating auto-antibodies (2) Increased numbers & activation of peritioneal macrophages & (3) Decreased T-lymphocyte reactivity and natural killing activity. Whether these changes predate the disease, are coincidental to it or result from it remains to be ascertained. Increased concentration of growth factors and cytokines found in the peritoneal fluid of patients display a dual effect: while inducing proliferation of endometriotic implants they may be inhibiting early reproductive events (Oral & Arici, 1997).

If there is some disturbance in their antigen-eliminating capabilities, this may play a role in the development of endometriosis in the adolescent as well.

Development & Evolution of Lesions : Fallon (1946) believes that development of endometriosis requires 5 or more years of menstrual cycles. The mean interval between menarche & diagnosis of endometriosis has been calculated at 4.5 years.

Goldstein et al (1979) have reported endometriosis in a 10.8 years old girl who has menarche only 5 months earlier.

In 1987, Redwine drew our attention to age related evolution in color aprearances of endometriosis. He found the mean age of endometriosis patients to increase from 21.5 years, for those with clear papules only to 26.3 years for those with red lesion to 29.5 years for those with white lesion to 31.9 years for those with black lesion. The age range of patients with clear papules only was 17-26 years, of patients with black plus any other lesion was 17-43 & of patients with black lesion only was 20-52 years. These findings suggested that endometriosis in many patients is a self limiting disease. The early nonpigmented papules evolved into well vascularised hyperaemic red lesions & finally are subdued by the peritoneal defence system & develop into familiar black & blue powder burn lesions poorly vascularised, quiescent & surrounded by white fibrotic tissue. Adolescents usually have earlier lesions. Endometriosis should not be treated simply because it is there. The final answer to the question whether endometriosis is a progressive disease will have to come from long term prospective invesigation studying spontaneous evolution of peritoneal lesions without therapeutic interference. This will require performing a second look laparoscopy in a patient who has not received any treatment since her first laparscopy. As of now, pain is the main indication for treatment.

Clinical presentation: The cardinal symptom associated with endometriosis in the adolescent is pelvic pain, primarily in the form of dysmenorrhoea. The suspicion is raised especially when the pain is not relieved by NSAIDS & ovulation inhibition. Chatman & Ward(1982) noted dysmenorrohea (78%) followed by bowel symptoms (37%) & dyspareunia.

While vaginal examination is often difficult & unsatisfactory in the unmarried teenager rectal examination is more rewarding & tenderness with or with out nodularity along the uterosacral ligaments & in the pouch of douglas is almost diagnostic. (Examination during menses enhance the pelvic findings). Anomalies of the vagina & cervix should be searched for. Examination under anaesthesia may be required.

Ultrasonography may be helpful especially for documenting adnexal masses such as a chocolate cyst. Serial USG & surgical evaluation in selected cases may be required.

Pre-operative assessment of patients with chronic pelvic pain should include complete blood count, urine analysis & cervical cultures for gonorrhoea & chlamydia in

selected cases. Laparoscopy is the gold standard for diagnosis & the early haemorrhagic & polypoid lesion should be kept in mind. The disease may be staged according to AFS revised classification.

Management: The specific emotional needs of teenagers, their body image problems, difficulties with regard to self-esteem & self-confidence, fears for reproductive future, & coincidental parental & family conflicts, make it essential to explain patiently to these girls the real nature of the disease process & the procedures to be carried out. A genuine sympathy for adolescents is essential & 'active listening' is a critical part of the consultation. Adolescents are notoriously fickle in their compliance to any sort of regimentation including regular taking of medication. The need to establish a mutual bond of trust & reliability cannot be over emphasised.

Treatment for an adolescent who has no immediate interest in fertility, centers primarily on control of pain. NSAID's may be first line drugs given immediately before or at the onset of menstruation & continued until dysmenorrhoea has subsided.

For those who do not respond to NSAID's low dose O.C.P. should be prescribed in the usual cyclic manner. Failure of these primary measures to relive symptoms indicates a need for future evaluation including laparoscopy.

Before any surgical intervention, the doctor should counsel the patient & the parents about the possible need for intra-operative therapeutic alternatives such as laparoscopic fulguration or laser vaporization of the endometriotic foci. Improvement in symptoms seems greater when fulguration & vaporisation of implants & lysis of adhesions are preformed during laparsoscopy (Sanfilippo, 1994). Rarely conservative surgery may be necessary. Large chocolate cyst should be excised & as much functional ovarian tissue as possible should be preserved.

No well designed prospective studies have been conducted with respect to treatment of adolescent endometriosis. After laparoscopic or conservative surgery a form of medical therapy is often advised to prevent progression or to treat recurrence. The choice of medical regimens include O.C.P, progestogens, danazol & Gn RH agonists.

If primary laparoscopic destruction is not done, symptomatic patients may be treated medically especially if endometriotic cysts are less then 3cms. As there is little to choose between the different drugs in term of efficacy, the choice should be based on side-effect profile, patient choice & cost (Lindsay, 1995). It should be emphasised that medical treatment leads to regression rather than elimination.

At present treatment is given for a finite period (say 6 months) because of the potential harm from metabolic side effects. Future research needs to address long-term medical treatment effective in controlling symptoms arresting progress & acceptable in terms of side effects. As of now it must be remembered that there is a recurrence rate of 30-53% within 1-5 years of any medical management (Waller & Shaw, 1993 Bayer & Seibel, 1989). Since adolescents quickly tire of prolonged oral medication, 3 monthly injections of medroxy progesterone acetate may have a role as a medical therapeutic agent.

Conclusion: Evaluation of pelvic pain in the adolescent requires a systematic approach. Cyclic pain should be treated with NSAID's failing which OCP may be used. In refractory cases the possibility of endometriosis must be excluded by laparoscopy. The traditional model of diagnostic laparoscopy followed by medical tretment now requires re-evaluation as laser laparoscopy, offers the

potential for cost effective 1 step treatment (at the same time as diagnosis) (Stones & Thomas, 1995) Controversies continue. It may yet be proved that minimal endometriosis seen at laparoscopy is coincidental rather than causal in women with pelvic pain.

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