Simultaneous Presence of Pelvic Endometriosis and Polycystic Ovary Syndrome

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Abstract: The simultaneous presence of polycystic ovary syndrome with pelvic endometriosis presents compounded gynecological effects on women with sub-fertility and pelvic pain as the common symptoms. . We describe one such case. The molecular basis for etiology is discussed and the need for individualized treatment is suggested.

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Introduction

Polycystic ovary syndrome (PCOS) s a complex disease involving altered interactions involving the central nervous system, pituitary, ovaries, adrenals and extra-glandular steroid synthesizing tissues. It is a common endocrine disorder seen in about 10 – 15 % of women in the reproductive age with a higher prevalence in oligomenorrhoeic anovulatory cycles. The simultaneous presence of pelvic endometriosis is an uncommon combination but lends to the difficulties in treating such patients for sub-fertility. We report here a case where both conditions prevailed.

Case Report

Ms PS, a 27 year old Malay para one, presented to the fertility clinic on 17th August 2003 with a history of sub-fertility of 3 years duration and having had irregular menstrual cycles ranging from 60 – 200 days since the age of 18 years. Her BMI was 28 Kg/M^2 (overweight) and endocrine profiles showed the LH:FSH ratio to be 3.8, prolactin 12.5 ng/L, testosterone 3.0 nmol/L, DHEAS 9.2 nmol/L and serum progesterone in the calculated luteal phase to be low.

Transvaginal ultrasonography confirmed a uniformly outlined uterus of normal dimensions with an endometrial thickness of 2.5 mm. Both ovaries were enlarged and exhibited the classical features of PCOS (1). No adnexal masses were visualized except for enlarged ovaries.

A diagnostic laparoscopy was performed on 23 October 2003 as part of the work up of sub-fertility which confirmed polycystic ovaries. There were flimsy adhesions of the omentum to the anterior uterine corpus. This was easily resected. The ovaries were mobile and tubal patency was established with normal saline hydrotubation . The pelvic peritoneum showed semilunar defects with thickening of the uterosacrals. Findings were consistent with revised American Fertility Scoring System stage 1 disease.

Previously she had delivered a 3.0 Kg baby boy in August 2000 after having been on 3 cycles of clomiphene citrate for ovulation induction and intrauterine insemination.

The patient was prescribed clomiphene citrate and metformin for ovulation induction after the laparoscopy and she is being monitored for follicular stimulation. She is being followed up in the fertility clinic for further reviews and endocrine manipulation.

Discussion

We present a case where there was simultaneous presence of PCOS and endometriosis. Although sub-fertility is associated with both conditions, treatment strategies will vary according to the presenting complaint. The patient came to the fertility clinic because of anovulation and difficulty in conceiving.

The simultaneous presence of PCOS and endometriosis is challenging to the gynecologist as the etiology is difficult to explain and management can be equally difficult.

PCOS does not have a standardized criterion for diagnosis and several approaches are taken in establishing the diagnosis. Anovulation, obesity, hyperadrogenization and morphological appearance of polycystic ovaries at ultrasonography assist in establishing the diagnosis.[1] Neuro-endocrine abnormalities with increases in mean luteinising hormone levels and increased pulse amplitude is well established in PCOS. Hyperandrogenemia is relatively

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non-specific and may not be overt. The impressive effect for a primary defect of insulin action is now equally important. Increased insulin resistance has a known metabolic effect with concomitant steroidogenic and mitogenic effect on the ovaries. The increased effect on the latter and insulin growth factor proves to be augmentative to LH contributing to poor pregnancy outcome.

Brincat M 2 reviewed 192 women for the presence of both PCOS and endometriosis and found a higher prevalence of simultaneous PCOS/endometriosis compared to either of the conditions occurring alone. In their series the degree of endometriosis was worse in those with co-existing PCOS. This result was consistent with a higher incidence of sub-fertility.

Abnormal luteal phase activity has been thought to be associated with endometriosis, with a cause-result effect. A similar abnormal luteal activity is common in PCOS. Considering the higher grade of endometriosis in those with PCOS and infertility, this lends to the theory that PCOS may precede the eventual clinical state. An earlier laparoscopy done in 2000 in our, however, did not reveal the presence of pelvic endometriosis.

Whitehead SA et al[3], in studying peritoneal fluid of affected patients alluded to the 'cocktail of substances including steroids, growth factors, prostaglandins, complement factors and cytokines in the peritoneal fluid.' Both macrophages and raised cytokine concentrations in the peritoneal fluid have been shown to alter ovarian steroidogenesis.

A molecular approach to both disorders focuses on the presence of chromosomal aberration to explain the biochemical disturbances resulting in clinical manifestations of both the disorders. Chromosomal aberrations involving trisomy 11, monosomy 16 and monosomy 17 in late stage endometriosis has been alluded to. A loss of only p53 tumor suppressor gene rather than a loss (monosomy) of chromosome 17 are thought to be pivotal in the process. PCOS, on the other hand is a polygenic and multifactorial disorder where one or more dominant genes cause the condition, though the mode of inheritance is uncertain. Genes for adrenal biosynthetic enzymes, insulin receptors, leptin and leptin receptors, follistatin, activin and inhibins are implicated.[4]

In managing patients where subfetility is the presenting complaint, cognizance must be taken of the fine endocrine changes within the peritoneal fluid that may affect spontaneous ovulation and pregnancy. Our case had conceived with clomiphene citrate on a previous occasion and this is encouraging. The lack of clinical androgenisation and the lack of recognizable insulin resistance are favorable factors for ovulation with minimal stimulation in this case. Early conception should be encouraged in her in view of current research which points to worse outcomes in the presence of a higher stage of endometriosis in patients with PCOS , the former condition being envisaged (in untreated cases) in later years.

Where the primary problem is pelvic pain due to pelvic adhesive disease in pelvic endometriosis, treatment should be directed to these diseases especially when fertility and menstrual irregularity are not problems. Both surgical ablation and medical treatment for endometriosis either alone or in combination has been advocated. Whilst surgery, after a period of medical therapy, is appropriate, the use and choice of anti-estrogens to be prescribed after laparoscopic surgery is open to debate. Whilst a pseudo-pregnancy state could be created with progestogens, danazol and dimetriose, considering the simultaneous presence of PCOS, Diane 35 (Cyproterone acetate 2 mg and ethinyl estradiol 35 micrograms, Schering AG) may be considered when the primary problem is endometriosis and pelvic pain . Continuous drug treatment for 3 months with one week break may be considered a useful alternative in the presence of clinical androgenisation.

Insulin resistance is a key component in the pathogenesis of Insulin sensitizing agents like

Metformin. Metformin has been widely used to exert beneficial effects on the endocrine and metabolic disturbances that characterize PCOS. With rapid progress in the understanding of the pathogenesis, Metformin and related drugs have been extended to the management of lean PCOS patients. There are therapeutically no deleterious effects by the use of Metformin in those patients who have concomitant endometriosis.

Conclusion

The simultaneous presence of PCOS and endometriosis presents is challenging to both patient and gynecologist. The etiology is still not clear but treatment strategies need to be developed depending on the presenting complaint and the need to attain ovulation.

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