



PCOS – Poly Cystic Ovarian Syndrome and Gardasil®

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Polycystic ovarian syndrome (PCOS) is a disorder characterized by laboratory and/or clinical findings of higher levels of androgens (male sex hormones), difficulty with ovulating and menstrual irregularity, and polycystic appearance to ovaries on ultrasound. Its prevalence is nearly 10 percent among reproductive-age women and may represent the largest underappreciated segment of the female population at risk for cardiovascular disease.

Most women suffering from PCOS present with one or more of the following symptoms or characteristics: irregular periods, excessive facial hair, alopecia (loss of hair), high levels of androgens or specific hormones on lab testing, and obesity, although as many as 20 percent of women with PCOS are not obese. Many women also present with a variety of menstrual related complaints, from not having a period for years to heavy menstrual bleeding. Infertility is also a common finding among PCOS sufferers and the diagnosis is often made during an infertility evaluation.

Estrogen Dominance and PCOS

Estrogen dominance describes a condition where you can have normal or excessive estrogen, but have too little progesterone to balance estrogen's effects in your body.

An important cause of low progesterone occurs when the follicle does not release the egg. The follicle becomes a cyst and the normal progesterone surge does not take place. The lack of increase in progesterone signals the hypothalamus to produce more LH and FSH, which stimulates the ovary to make more estrogen and androgens, which in turn stimulates more follicles toward ovulation. If these additional follicles are also unable to produce a matured ovum and make progesterone, the menstrual cycle is dominated by estrogen and androgen production without progesterone. This failure to ovulate contributes to a condition called "estrogen dominance."

Women with PCOS (polycystic ovarian syndrome) frequently do not produce enough progesterone.¹

Excessive Estrogen Symptoms

Long-term estrogen dominance leads to a vast array of possible health problems:

- Acceleration of aging
- Allergies or asthma
- Autoimmune disorders
- Bone loss
- Breast cancer

- Breast tenderness or fibrocystic disease
- Cervical dysplasia
- Decreased sex drive
- Depression/anxiety/irritability
- Dry eyes
- Endometrial (uterine) cancer
- Fat gain (abdomen, hips thighs)
- Fatigue
- Fibroids
- Gallbladder disease
- Hair Loss
- Headaches
- Hypoglycemia
- Increased blood clotting
- Infertility
- Insomnia
- Irregular menstrual periods
- Memory loss or foggy thinking
- Mood swings
- PMS
- Polycystic ovaries
- Thyroid dysfunction or sluggish metabolism
- Uterine cancer
- Water retention, bloating ¹.

There are 404 PCOS reports filed with the U.S. Vaccine Adverse Event Reporting System – (VAERS). It is estimated that only 1 to 10% of the adversely injured HPV vaccinated population is reporting.

SANE Vax Inc. is very concerned that vaccinating adolescent girl's at the most fragile time of their lives – menarche/puberty is causing them great harm. Until young girls develop an endocrine rhythm of their own, their hormones are in a constant state of fluctuation. For some girls, the introduction of the HPV vaccine is obviously wreaking havoc on their endocrine systems. Many of the adversely injured have reported menstrual irregularities post-vaccination.

One of the grave concerns about Gardasil is the addition a L-Histidine – a synthetic form of histamine already produced in the body. Gardasil is the first vaccine to contain this synthetic amino acid. Histamine is stimulated by estrogen production – and if a young woman is experiencing annovulatory cycles; her estrogen levels are already elevated – producing excess histamine. Too much histamine will cause the body's immune system to start attacking itself...and one of the other major side effects reported to VAERS are the prevalence of autoimmune diseases.

The FDA has acknowledged studies on the interaction between Gardasil, estrogen and HPV stimulation have not been conducted.

Related Studies

Histamine is an organic nitrogen compound involved in local immune responses as well as regulating physiological function in the gut and acting as a neurotransmitter. Histamine triggers the inflammatory response. As part of an immune response to foreign pathogens, histamine is produced by basophils and by mast cells found in nearby connective tissues. Histamine increases the permeability of the capillaries to white blood cells and some proteins, to allow them to engage pathogens in the infected tissues. It is found in virtually all animal body cells. <http://en.wikipedia.org/wiki/Histamine>

Histamine metabolism during the menstrual cycle.

The urinary excretion of histamine and its metabolites methylhistamine (MeHi) and methylimidazoleacetic acid (MelMAA) were measured during the menstrual cycle in 9 healthy women, 1 allergic woman, and 3 nonpregnant women with anovulatory regular cycles. Simultaneous urinary analyses of luteinizing hormone (LH) and total estrogens were performed. The healthy women showed individual variations in the excretion of histamine, MeHi and MelMAA. This observation has been interpreted as an expression of minor individual differences in the catabolism of histamine. At midcycle an increase in the urinary excretion of histamine metabolites was sometimes evident and a statistically significant correlation could be established between MeHi and estrogen in urine. These results may support previous findings of histamine release by estrogens in uterine tissue but may also reflect an elevated histamine formation.

The allergic woman excreted constantly increased amounts of histamine and its metabolites, especially when her allergic symptoms became aggravated premenstrually. She was without any change in MelMAA excretion at midcycle but the MeHi excretion varied with the excretion of estrogens in the urine. The subjects with anovulatory menstrual cycles had low values of histamine and metabolites although within the normal variations.²

Over the past year, numerous studies have been published citing estrogens role in HPV stimulation, and HPV-related cancers. In addition, documented studies prior to the approval of the HPV vaccines also shows estrogens role in Oral Contraceptives as a possible HPV stimulator.

National Cancer Institute

Oral Contraceptives and Cancer Risk: Questions and Answers

Reviewed: 05/04/2006

Although OC use may increase the risk of cervical cancer, human papillomavirus (HPV) is recognized as the major cause of this disease. Approximately 14 types of HPV have been identified as having the potential to cause cancer, and HPVs have been found in 99 percent of cervical cancer biopsy specimens worldwide.

A 2003 analysis by the International Agency for Research on Cancer (IARC) found an increased risk of cervical cancer with longer use of OCs. Researchers analyzed data from 28 studies that included 12,531 women with cervical cancer. The data suggested that the risk of cervical cancer may decrease after OC use stops. In another IARC report, data

from eight studies were combined to assess the effect of OC use on cervical cancer risk in HPV-positive women. Researchers found a fourfold increase in risk among women who had used OCs for longer than 5 years. Risk was also increased among women who began using OCs before age 20 and women who had used OCs within the past 5 years. The IARC is planning a study to reanalyze all data related to OC use and cervical cancer risk.³

Effect of oral contraceptives on risk of cervical cancer in women with human papillomavirus infection: the IARC multicentric March 2002

Background

Use of oral contraceptives could increase risk of cervical cancer; however the effect of human papillomavirus (HPV), the main cause of cervical cancer, is not usually taken into account. We aimed to assess how use of oral contraceptives affected risk of cervical cancer in women who tested positive for HPV DNA.

Interpretation

Long-term use of oral contraceptives could be a cofactor that increases risk of cervical carcinoma by up to four-fold in women who are positive for cervical HPV DNA. In the absence of worldwide information about HPV status, extra effort should be made to include long-term users of oral contraceptives in cervical screening programmes.⁴

In October 2010, the International Journal of Cancer published this study on ***The association of hormonal contraceptive use and HPV prevalence:***

Women diagnosed with cervical cancer report longer duration and more recent use of combined oral contraceptives (COCs). It is unclear whether COC use is associated with upstream events of human papillomavirus (HPV) infection prior to development of clinical disease.” The studies objective...“was to assess the association of contraceptive use on the risk for prevalent HPV infection in a cohort of long-term hormonal contraceptive (HC) users.

The authors found: a demonstrable “association between the use of COCs for >6 years and prevalent HPV infection among 20- to 37-year-old women from Thailand after controlling for sexual behavior and cytological abnormalities. This finding is in agreement with other smaller cross-sectional studies conducted among college age women and women 20–29 years of age who report COC use for >4 years.” The summary “observed that long-term use of COCs of >6 years is associated with an increased risk of prevalent HPV infection in a cohort of HC and NHC users in Thailand.¹

“Longitudinal studies examining the risk of HPV acquisition and particularly HPV persistence by contraceptive use are therefore urgently needed.”⁵

Estrogen was again implicated with HPV stimulation in an article that appeared in Health Day News in January of this year: ***Estrogen May Play Role in Rising Rates of Head, Neck Cancer***; Health Day News; January 4, 2011

Rates of head and neck cancer are rising among some groups of people, including young women without any known risk factors. Now, a study suggests that estrogen may help the cancer spread by boosting the movement of precancerous cells in the mouth.⁶

Another concern SANE Vax Inc has about Gardasil is the addition of Polysorbate 80 or 'Tween 80' to the vaccine. This 1993 study documents that 'Tween 80' can cause chronic estrogen stimulation.

Delayed effects of neonatal exposure to Tween 80 on female reproductive organs in rats⁷.

Abstract

Neonatal female rats were injected ip (0.1 ml/rat) with Tween 80 in 1, 5 or 10% aqueous solution on days 4-7 after birth. Treatment with Tween 80 accelerated maturation, prolonged the oestrus cycle, and induced persistent vaginal oestrus. The relative weight of the uterus and ovaries was decreased relative to the untreated controls.

Squamous cell metaplasia of the epithelial lining of the uterus and cytological changes in the uterus were indicative of chronic oestrogenic stimulation. Ovaries were without corpora lutea, and had degenerative follicles.

Sources

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