Polycystic Ovary Syndrome

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About the Author

Marie Reilly has over 12 years experience as a Medical Herbalist, and currently practices at the Lismore Clinic in Co. Waterford. She qualified from the College of Phytotherapy in the UK, and subsequently completed the Scottish School of Herbal Medicine Masters Degree Programme, having conducted her research dissertation on the treatment of female functional infertility with herbal medicine.

Marie has also studied endobiogenic medicine with Dr. Jean Claude Lapraz, and Ayurveda with Dr. Vasant Lad. She combines these various different disciplines to form an integrated approach to healthcare.

Introduction

Polycystic ovary syndrome (PCOS) is estimated to affect around 7% of women[1]. It is the most common endocrine disorder affecting women of reproductive age, and is the most common cause of failure to ovulate.[2] Orthodox medicine has little to offer in terms of safe, effective treatments for women with PCOS, particularly those who wish to become pregnant, while herbal medicine is an effective approach to treatment in many cases.

PCOS is a complex endocrine and metabolic disorder, which is causes abnormal ovarian function, and **not** (as is commonly assumed) a syndrome caused by a disorder of the ovaries.

[1] Grant, P., & Ramasamy, S.(2012). An update on plant derived Anti-androgens. *Int J Endocrinol Metab*
[2] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.

PCOS is associated with hormonal abnormalities, hyperandrogenism, insulin resistance, metabolic syndrome, obesity, oligomenorrhea or amenorrhoea, ovarian cysts and failure to ovulate, resulting in infertility. The symptom picture can vary, and individual women may experience all or only some of these signs and symptoms.[3]

Genetic predisposition may contribute to the development of PCOS and it has also been proposed that exposure to high levels of testosterone during foetal development may increase the risk of PCOS in adulthood.[4]

Diagnosis

PCOS is diagnosed on the basis of the presence of at least 2 criteria, including: biochemical or clinical signs of androgen excess, ovulatory dysfunction, and / or polycystic ovaries.[5]

Androgens are usually only slightly to moderately raised in PCOS (if at all)[6], but LH levels are often significantly higher than FSH levels[7], ovarian oestrogen and progesterone are usually low[8], and AMH may be raised.

Hyperandrogenism may present clinically as acne, male pattern alopecia, and/or hirsutism. Hirsutism is the growth of coarse hair in a male pattern, particularly on the upper lip, chin, chest, upper abdomen, and back, and is distinguished from hypertrichosis, which involves a more uniform distribution of fine hair.

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[3] Ibid.
[4] Dumesic, D.A., Abbot, D.H. & Padmanhaban, V. (2007). Polycystic ovary syndrome and its developmental origins. *Endocr Metab Disord* 8(2):127-41
[5] Legro R.S., Arslanian S.A., Ehrmann D.A. *et al* (2013). Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*.
[6] Ibid
[7] Bone, K. & Mills, S. (2013). Principles and Practice of Phytotherapy: Modern Herbal Medicine. *Elsevier Health Sciences.*

[8] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.

However, clinical signs of androgen excess are usually mild, and virilisation (clitoromegaly, deep voice, increased musculature) is not a feature of PCOS[9].

Useful blood tests include LH, FSH, oestradiol, progesterone, total androgens, free testosterone, SHBG, Prolactin, TSH, T4, T3, AMH, lipid profile and HBA1c. Blood values of various hormones may be normal or slightly elevated in PCOS, while grossly elevated hormone levels generally suggest a different disorder. Clinical examinations should include assessment of BMI and waist to hip ratio, which should be <0.8 *(demonstrate).* Ultrasonography is used to detect polycystic ovaries (PCO).

Differential Diagnosis

Before making a diagnosis of PCOS, other conditions causing similar symptoms should be excluded[10] [11]

- **Adrenal hyperplasia** (irregular menses and hirsutism, 21-hydroxylase deficiency & *elevated 17-hydroxyprogesterone*).
- **Cushings syndrome** (Elevated cortisol & androgens, Low K+, hypertension, striae, central obesity, dorsal cervical fat pads and rounded face).
- **Drugs** (e.g. steroids such as Danazol, androgenic progestins, valproic acid).
- **HAIR-AN** Syndrome (Hyper-Androgenism, Insulin Resistance & Acanthosis Nigricans). May be associated with anti-insulin receptor antibodies or genetically reduced number of receptors.
- **Hyperprolactinaemia** (amenorrhoea/oligomenorrhoea, mild hyper-androgenic features & galactorrhoea. May be due to a pituitary tumour).

[9] Sheehan, M.T. (2004). Polycystic Ovarian Syndrome: Diagnosis and Management. *Clin Med Res* 2(1):13-27
[10] Sheehan, M.T. (2004). Polycystic Ovarian Syndrome: Diagnosis and Management. *Clin Med Res* 2(1):13-27
[11] BMJ Best Practice (2016). *Polycyctic Ovary Syndrome* [online]. Available from http://bestpractice.bmj.com/best-practice/monograph/141/diagnosis/differential.html (Accessed 30/7/16).

- **Hypothyroidism** (amenorrhoea, hair loss, obesity, fatigue, dry skin, cold intolerance, possible goiter)
- **Idiopathic or familial hirsutism**,
- **Ovarian Hyperthecosis** (androgen excess, insulin resistance)
- **Ovarian or adrenal tumour** (sudden onset of symptoms of virilisation)
- **Pregnancy** (Absent periods, weight gain. May be associated with gestational diabetes.

Use of oral contraceptives may mask many of the biochemical and clinical signs of PCOS[12]. In some women, PCOS may be masked by the co-existence of functional hypothalamic amenorrhoea. (Hypothalamic amenorrhea is associated with absent or deficient hypothalamic secretion of GnRH by the hypothalamus, resulting in low FSH.[13] It may be caused by excessive weight loss, exercise, physical illness, or emotional stress.[14] or it may also follow use of oral contraceptives[15]). In such cases, symptoms consistent with PCOS only appear when hypothalamic activity recovers.[16]

Polycystic ovaries

The term 'Polycystic Ovaries' (PCO) refers to ovaries which appear to be covered with small cysts, whereas the term 'PCOS' refers to a *syndrome*, which is characterized by various signs and symptoms such as hormonal abnormalities, menstrual cycle disturbance, hyperandrogenism, insulin resistance, and obesity.[17]

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[12] Sheehan, M.T. (2004). Polycystic Ovarian Syndrome: Diagnosis and Management. *Clin Med Res* 2(1):13-27

[13] Leyendecker, G. Wildt, L., & Hansmann, M. (1983). Induction of ovulation with chronic intermittent (pulsatile) administration of Gn-RH in women with hypothalamic amenorrhoea. *J Reprod Fertil* 69:397-409.

[14] De Souza, M.J. & Toombs, R.J. (2010). "Amenorrhea". In Nanette F. Santoro and Genevieve Neal-Perry. *Amenorrhea: A Case-Based, Clinical Guide*. Humana Press. pp..101–125

[15] Wright, K.P. & Johnson, J.V. (2008). Evaluation of extended and continuous use oral contraceptives. *Therapeutics and clinical risk management* 4 (5): 905–11.

[16] Wang, J.G. & Lobo, R.A. (2008). The complex relationship between hypothalamic amenorrhoea and polycystic ovary syndrome. *J Clin Endocrinol Metab*. (93(4):1394-7.

[17] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.

In cases of PCO, folliculogenesis is arrested at 5-10mm. Failure to ovulate, which may be due to PCOS, or any another cause (such as hypothalamic amenorrhoea) means that the follicles do not rupture, and instead give rise to numerous fluid filled cysts on the surface of the ovary. Women with PCOS do not always have PCO, and conversely, a finding of PCO does not necessarily mean a woman has PCOS; In fact 20% of women with normal ovulatory cycles are found to have PCO on ultrasound, due to occasional anovulatory cycles.[18]

Insulin sensitivity

Up to 75% of women with PCOS have some degree of insulin resistance, which is associated with acanthosis nigrans (hyperpigmentation of the axillae, back of the neck, and/or breast folds)[19], Increased central fat distribution[20], and elevated fasting glucose, triglycerides and cholesterol[21] Insulin resistance triggers a compensatory rise in insulin secretion, this in turn increases storage of fat, leading to obesity, which in turn further increases insulin resistance.[22]

Insulin resistance may be caused by various factors that affect the expression of insulin receptors, including a family history of type 2 diabetes, smoking, use of certain drugs (such as corticosteroids, and oestrogen-containing contraceptives), and obesity (especially central obesity)[23].

PCOS sufferers with insulin resistance have more menstrual cycle disturbance and reduced fertility when compared to those who are insulin sensitive.[24]

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[18] Clayton, R.N., Ogden, V., Hodgkinson, J. *et al.* How common are polycystic ovaries in normal women, and what is their significance for the fertility of the population? *Clin Endocrinol* 37(2):127-34.
[19] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.
[20] Kirchengast, S. & Huber, J. (2001). Body composition characteristics and body fat distribution in lean women with polycystic ovary syndrome. *Hum Reprod* 16(6):1255-60.
[21] Sheehan, M.T. (2004). Polycystic Ovarian Syndrome: Diagnosis and Management. *Clin Med Res* 2(1):13-27.
[22] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.
[23] Venkatesan, A.M., Dunaif, A., & Corbould, A. (2001). Insulin resistance in polycycstic ovary syndrome: progress and paradoxes. *Rec Prof Hor Res* 56(295-308)
[24] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.

Elevated insulin triggers hyperandrogenism by directly inducing ovarian androgen production, stimulating LH secretion, and increasing adrenal androgen production (increased activity of 11ß- hydroxysteroid dehydrogenase, 11ß-OHSD, increases cortisol clearance, with a compensatory rise in ACTH and increased adrenal androgens).[25] Insulin resistance also reduces SHBG.[26]

Insulin resistance and PCOS lead to an increased risk of Type 2 diabetes, and 40% of women with PCOS experience impaired glucose tolerance or diabetes by the age of 40.[27] Increased risk of cardiovascular disease can also be caused by impaired glucose metabolism: Insulin resistance is associated with elevated HBA1c (glycated Hb above 42 mmol/mol), elevated triglycerides, and low HDL cholesterol.[28]

Obesity

Over 28% of obese women suffer from PCOS, compared to under 6% of women with normal BMI.[29] Increasing levels of obesity are likely to increase the prevalence of PCOS and associated problems in the future. Higher levels of LH and androgens, accompanied by anovulatory cycles are normal during puberty, and it is thought that excessive weight gain during puberty may prolong these features leading to development of PCOS.[30]

Obese women with PCOS (BMI>30) suffer worse symptoms of PCOS than those with normal BMI. Obesity contributes to metabolic and reproductive abnormalities by reducing sex hormone binding globulin (SHBG), increasing insulin resistance, and independently further increases the risk of diabetes and cardiovascular disease.[31]

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[25] Ibid.
[26] Sheehan, M.T. (2004). Polycystic Ovarian Syndrome: Diagnosis and Management. *Clin Med Res* 2(1):13-27.
[27] Kidson, W. (1998). Polycystic ovary syndrome: a new direction in treatment. *MJA* 169(10):537-40
[28] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.
[29] Alvarez-Blasco, F. Botella-Carretero, J.I., San Millan, J.L. *et al* (2006). Prevalence and characteristics of the polycystic ovary syndrome in overweight and obese women. *Arch Int Med* 166(19):2081-6
[30] Pasquali, R. & Gambineri, A. (2006). Polycystic ovary syndrome: a multifaceted disease from adolescence to old age. *Ann NY Acad Sci.* 1092:158-174
[31] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.

Obesity is also associated with increased production of Leptin, and Leptin resistance, which has an adverse effect on gonadotrophin release and follicle development.[32]

Abdominal visceral is fat more likely to be associated with metabolic and reproductive problems than subcutaneous fat.[33] Women with PCOS who have a normal BMI are more likely to have increased central fat distribution (reduced waist to hip ratio) than those without PCOS. [34]

In women with PCOS who are overweight, as little as 5% weight reduction reduces insulin resistance and increases ovulation.[35] It is theorized that women with PCOS have adapted to remain fertile during food shortages, when others experience reduced fertility.

Hyperandrogenism

PCOS is often (though not always) associated with elevated ovarian and adrenal androgens. This gives rise to symptoms such as acne, hirsutism and androgenic alopecia. Elevated insulin directly triggers androgen production by the ovarian theca cells, stimulates LH secretion, and increases adrenal androgen production (due to increased activity of 11ß- hydroxysteroid dehydrogenase (11ß-OHSD), which increases cortisol clearance, and causes a compensatory rise in ACTH, which leads to increased adrenal androgen production).[36]

[32] Ibid.

[33] Zaadstra, B.M., Seidell, J.C., Van Noord, P.A. *et al.* (1993). Fat and female fecundity: Prospective study of effect of body fat distribution on conception rates. *BMJ* 306:484-7
[34] Kirchengast, S. & Huber, J. (2001). Body composition characteristics and body fat distribution in lean women with polycystic ovary syndrome. *Hum Reprod* 16(6):1255-60.
[35] Franks, S. Kiddy, D. Sharpe P. *et al.* (1991). Obesity and polycystic ovary syndrome. *Ann N Y Acad Sci* 626:201-6.
[36] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.

Genetic abnormalities of the Hypothalamus or pituitary gland may be responsible for inappropriate secretion of GnRH and /or LH, leading to increased ovarian and adrenal androgen secretion.[37]

There is also reduced activity of aromatase in ovarian granulosa cells, leading to poor conversion of androgens to oestrogen. The androgens inhibit development of follicles, resulting in anovulation, absence of the corpus luteum, and consequent low progesterone, which in turn leads to elevated LH through lack of negative feedback. The elevated LH further stimulates the ovarian theca cells, producing more ovarian androgens.

Acyclic Oestrogens

Anovulation in PCOS leads a loss of the cyclic variation in oestradiol levels. Oestrone on the other hand is derived from aromatization of androgens, primarily in adipose tissue. Women with PCOS frequently have high levels of androgen available for conversion to oestrone, and excess stores of adipose tissue where conversion can take place. Higher levels of acyclic oestrone combined with reduced endometrial shedding can lead to increased risk of endometrial hyperplasia and cancer.

Acyclic oestrogens may also cause reduced levels of FSH due to reduced negative feedback. Low FSH reduces the aromatization of androgens in the ovarian granulosa cells.

(diagram)

[37] Barontini, M., Garcia-Rudaz, M.C. & Veldhuis, J.D. (2001). Mechanisms of hypothalamic-pituitary-gonadal disruption in polycyctic ovarian syndrome. *Arch Med Res* 32(6):544-52

Infertility

PCOS accounts for 75% of anovulatory infertility. Additionally, if/when pregnancies do occur, the first trimester miscarriage rate is as high as 30% to 50% [38]

Improved insulin sensitivity is associated with improved ovulatory function and fertility in women with polycystic ovary syndrome (PCOS).[39] Fertility increases with diets which include a lower intake of intake of high glycaemic index foods with a higher intake of high-fibre, low-glycaemic index carbohydrates.[40]

Orthodox Treatment

Orthodox treatment of PCOS includes the following drugs:[41]

- Combined oral contraceptives (reduce androgens by decreasing gonadotropins, regulate menstruation and decrease the risk of endometrial hyperplasia).
- Anti-androgens (such as spironolactone: an alsdosterone antagonist, which also blocks testosterone receptors)
- Insulin-sensitizing drugs (such as Metformin)
- Clomiphene citrate (to improve fertility).

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[38] Homburg. R, Armar N.A., Eshel A., Adams J., Jacobs H.S. (1988). Influence of serum luteinising hormone concentrations on ovulation, conception, and early pregnancy loss in polycystic ovary syndrome. *BMJ. 297(6655):1024-6.*

[39] Chavarro, J.E., Rich-Edwards, J.W., Rosner, B.A. *et al* (2009). A prospective study of dietary carbohydrate quantity and quality in relation to risk of ovulatory infertiity. *European Journal of Clinical Nutrition* 63:78-86.

[40] Chavarro, J.E., Rich-Edwards, J.W., Rosner, B.A. *et al* (2007). Diet and Lifestyle in the Prevention of Ovulatory Disorder Infertility. *Obstetrics & Gynecology* 110(5):1050-1058.

[41] Sheehan, M.T. (2004). Polycystic Ovarian Syndrome: Diagnosis and Management. *Clin Med Res* 2(1):13-27.

Electrolysis is used to treat hirsutism by electrocoagulation of the hair follicle, which may or may not be permanent, but does not usually cause scarring. Laser treatment causes selective thermal damage to the hair follicle while avoiding surrounding tissue. It may lead to erythema, edema, blistering and/or temporary hyper- or hypopigmentation. Plucking is not generally recommended, as it can lead to folliculitis and scarring. Shaving is possibly the cheapest and simplest way to remove unwanted hair, but is not acceptable to some women.[42].

Holistic Treatment

Dietary modification and increased exercise are essential to reduce insulin resistance and obesity, and to help reduce cardiovascular risk factors.[43] Consuming only low GL carbohydrates and reducing overall intake of carbohydrates helps to increase the sensitivity of insulin receptors, promotes weight loss, and reduces cardiovascular risk factors.

Insulin resistance may also be improved using herbs such as *Cinnamonum zeylonicum, Eleutherococcus senticosus, Trigonella foenum-graecum,* and/or *Galega officinalis*.[44]Magnesium deficiency predisposes to insulin resistance and increased cardiovascular risk factors. Women with PCOS have significantly lower serum magnesium levels.[45]

Androgen production can be reduced with herbs such as *Paeonia lactiflora,* and *Glycyrrhiza glabra,* which increase aromatization and reducing 11ß-OHSD respectively[46], and both of which reduce levels of 5-alpha reductase

[42] Ibid
[43] Norma, R.J., Davis, M.J., Lord, J. *et al* (2002). The role of lifestyle modification in polycystic ovary syndrome. *Trends Endocrinol Metab* 13(6):251-7
[44] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.
[45] Muneyyirci-Delayle, O., Nacharaju, V.L., Dalloul, M. *et al.* (2001). Divalent cations in women with PCOS: implications for cardiovascular disease. *Gynecol Endocrinol* 15(3):198-201
[46] Ibid

(which converts testosterone to dihydrotestosterone)[47]. *Humulus lupulus* and *Cimicifuga racemosa* reduce both LH and androgens.

Serenoa repens Ganoderma lucidum and green tea (*Camellia sinensis*) all reduce 5-alpha reductase, and *Mentha spicata* has also been shown to reduce free testosterone and reduce symptoms of hyperandrogenism in women with PCOS, without reducing DHEA[48]

Tribulus terrestris, Asparagus racemosa or other steroidal saponin-containing herbs may be given in the early follicular phase (or for the first 10 days of the month if there is associated amenorrhoea). Phytoestrogens (such as *Cimicifuga racemosa, glycyrrhiza glabra,* and dietary beans and pulses) reduce androgens, improve lipid metabolism, and competitively inhibit oestrone at target tissues.

Recommended supplements include Omega 3 Essential Fatty Acids (at least 3g a day to help reduce visceral fat, serum triglycerides and cardiovascular risk), and Biotin (3g), Magnesium (400mcg) & GTF chromium (200mcg) to improve IR.[49]

Energetics

From an energetic perspective, PCOS is often associated with excess Kapha, It is characterized by excess mucous and a swollen tongue with white coating[50]. Pungent, astringent and bitter foods and herbs reduce Kapha. It is also important to avoid sweet, sour salty and oily and damp foods such as dairy products as they increase Kapha.[51] It is important to avoid overeating and to take regular exercise.

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[47] Grant, P., & Ramasamy, S.(2012). An update on plant derived Anti-androgens. *Int J Endocrinol Metab* 10(2):497-502
[48] Ibid
[49] Trickey, R. (2011). *Women, Hormones and the Menstrual Cycle.* Melbourne Holistic Health Group.
[50] Lad, V.D. (2006). *Textbook of Ayurveda: A Complete Guide to Clinical Assessment. Volume 2*. The Ayurvedic Press, Albuquerque, New Mexico.
[51] Tillotson,A.K. (2001). *The One Earth Herbal Sourcebook*. Kensington Books, New York.

Example prescription and recommendations

Rx. *Paeonia lactiflora* 1:3 40
Glycyrrhiza glabra FE 15
Eleutherococcus senticosus 20
Galega officinalis FE 15
Humulus lupulus 1:3 15 (or *Cimicifuga racemosa)*
105 (7.5mL bid ac)

Rx. *Mentha spicata* (infusion) – one cup bid

Rx. *Tribulus terrestris* 1:3
Sig. 5mL m. for first 10 days of each month

- Increase exercise (at least 30 minutes moderate exercise, increasing up to 1hour, at least 5 days a week)
- Drink green tea instead of regular tea/coffee
- Oatmeal with ½ tsp cinnamon, soluble fibre, phytoestrogens, avoid high GL foods
- Omega 3 EFAs (3g), Magnesium (400mg), chromium (200mcg), biotin (3g).

Case Studies

Patient A

27 y.o. female, First Visit 27/9.

PC
Diagnosed with PCOS on US Scan 4 months ago, following amenorrhoea for 14 months. (Had previously been taking OCP but menses failed to return on discontinuing).
Bilateral PCO, slightly elevated androgens.
No acne or hirsutism. Normal BMI.
Offered OCP but declined to take

PMH
Recurrent sinus infections in winter, associated with frontal headache and paranasal pain. Better for avoiding dairy products.
Insomnia (wakes at 2am and can't get back to sleep). Evening fatigue.
Poor peripheral circulation. Bowel movement every 2-3 days.

FH
Diabetes (grandfather), Hypertension (grandmother).

SH
Non-smoker, social alcohol, low stress levels

BP: 110/75, **Pulse:** 60bpm (regular)

Tongue:
Pale, red tip (Heart Fire/insomnia)
White coating (Yang Deficiency & Dampness)
Swollen edges (Spleen Qi Deficiency)

Rx

Rosmarinus officinalis	(1:3)	20	(Spleen tonic, move blood, reduce damp)
Paeonia lactiflora	(1:3)	45	(increase aromatization)
Glycyrrhiza glabra	(FE)	20	(reduce 11ß-OHSD & 5-alpha reductase)
Eleutherococcus senticosus	(1:3)	20	(Improve IR & energy levels)
Cimicifuga	(1:3)	20	(reduce LH)
Dioscorea villosa	(FE)	15	(Improve ovarian oestrogen)
		140mL	

Sig. 10 mL bid

Recommendations

- Drink green tea instead of regular tea/coffee
- Oatmeal with ½ tsp cinnamon, soluble fibre, phytoestrogens, avoid high GL foods
- Omega 3 EFAs (3g), Magnesium (400mg), chromium (200mcg).

Follow up 14/10

Sleep and energy levels improved. Prescription repeated

Follow up 4/11
Started a period on 25/10. Prescription repeated

Patient continued to have regular menses. She conceived 6 months later and subsequently gave birth to a healthy baby boy. She did not continue with herbal treatment, but later conceived and gave birth to a second child.

Patient B

26 y.o. female, First Visit 5/5.

PC
Recurrent sinus infections (every 3-4 months, lasting for 2-3 weeks).
Symptoms include low energy, frontal headaches, eye pressure and paranasal pain.
Occasional chest infections (in winter) with thick sticky mucous.
Exacerbated by working in 30 degree temperatures on maternity ward.
3 courses of antibiotics in past 12 months – no improvement (caused vaginal candida)

PMH
Generally feels sluggish.
Intolerant to wheat and dairy products (but does not avoid them).
Obesity, dark hair growth on body, thinning hair on head, acne on back.
TFTs and HBA1c normal
Periods pill-controlled but were previously regular (5/21), heavy and painful.

DH
OCP (Dianette) past year (for treatment of acne)

FH
Hypertension, hypercholesterolaemia

SH
Smokes up to 5 cigarettes a day
Alcohol 7-8 units once every 2-4 weeks
Walks 3-4 miles 3 times per week
Works 13 hour shifts, 3 or 4 days per week

Diet

Breakfast	Porridge made with low fat milk & honey or brown toast with butter & tea with milk
Snack	Chocolate biscuits
Lunch	Often skips lunch or has chicken salad/bagel (regularly goes without eating for up to 8 hours)
Dinner	Meat, veg, potatoes

Tongue
Blue tongue body colour, red tip, wet.

Rx

Echinacea purpurea (rad.)	(FE)	20	(Improve immunity)
Tanacetum parthenum	(1:2)	10	(Reduce sinus pain and inflammation)
Hydrastis canadensis	(1:3)	20	(Tone MMs, reduce infection)
Cinnamonum zeylonicum	(1:3)	30	(Improve IR, antimicrobial)
Paeonia lactiflora	(1:3)	45	(Increase aromatization)
Glycyrrhiza glabra	(FE)	15	(Reduce 11ß-OHSD & 5-alpha reductase)
		140mL	

Sig. 10 ml bid

Rx. *Mentha spicata* (infusion) – one cup bid

Recommendations
Magnesium (400mg), chromium (200mcg). probiotic
Eat regular meals and healthy snacks, Increase fluid intake.
Add ½ teaspoon cinnamon powder to porridge
Avoid gluten, dairy products and High GL foods

Patient subsequently diagnosed with PCOS following blood tests and Ultrasound scan
(Treatment on-going)

Patient C

44 y.o. female. First visit 4/2

PC
Abdominal pain, bloating and nausea onset 5-6 weeks previously. Worse after breakfast.
Epigastric and bilateral hypochondrial pain (constant but worse for hunger)
Normal daily bowel movements
Fatigue, SOBOE, palpitations, light-headedness
Blood tests: Hb 10.8.
X-ray clear
Ultrasound showed PCOS, fibroids, and fecal impaction.

PMH
Depression
Lower left quadrant pain. (Constant, worse at ovulation)
PCOS diagnosed over 20 years ago – obesity, acne, hirsutism
Periods regular (7/28), heavy, clotty, some pain (better for movement)
PMS with irritability, mastalgia and constipation

SH
Non smoker, social alcohol
Swims 3-4 times per week

FH
Ovarian cancer

Diet

Breakfast	Porridge or branflakes Occasional poached eggs or bacon with low GI brown bread
Snack	Banana / apple or almonds
Lunch	Soup and bread or beans on toast Tea and biscuit
Snack	Tea and biscuit or apple
Dinner	Fish / chicken, veg, potatoes / rice / pasta

DH

Cymbalta	120mg	(SSNRI)
Ramil	5mg	(ACE inhibitor)
Omeprazole	40mg	(PPI)
Maxalon	10mg	(to increase gastrointestinal motility)
Spasmonil	20mg/500mg	(Dicyclomine/dimethicone - GI antispasmodic)
Magnesium	300mg	(for left sided migraines – effective)

Rx

Vitex agnus castus	(FE)	7	(treat PMS & Mastalgia)
Alchemilla vulgaris	(1:3)	20	(improve progesterone, reduce bleeding)
Paeonia lactiflora	(1:3)	45	(Aromatization, blood tonic, reduce ov. pain)
Glycyrrhiza glabra	(FE)	15	(Reduce11ß-OHSD, 5-a reduct., protect stomach)
Filipendula ulmaria	(1:3)	25	(treat gastritis)
Zingiber officinale	(1:2)	3	(antimicrobial, move blood, reduce nausea)
Rumexcrispus	(1:3)	30	(reduce constipation, blood tonic)
Cinnamonum zeylon.	(1:3)	20	(Improve insulin resistance, antimicrobial)
		160	

Sig. 7.5mL td i.c.

Recommendations
Magnesium citrate (400mg), Probiotic. Floradix (Florvital)
Low FODMAP Diet (Gluten and dairy free).

Follow up 19/2

Still has fatigue, SOBOE and lightheadedness
Less sugar cravings
No epigastric or hypochondrial pain, no LLQ pain, bloating reduced
Discontinued Maxalon and spasmonil but still taking PPI.
Getting night sweats.

Follow up 12/3

GI symptoms resolved
Hb level now 12. No fatigue, SOBOE and lightheadedness.
Less PMT & Mastalgia

Patient continued to feel well with no further abdominal pain or bloating, but eventually had radical hysterectomy due to ovarian cancer risk.

Patient D

35 y.o. female, First visit 10/12

PC
Trying to conceive past 4 years. Progesterone 15 (on day 21). Other bloods NTN.
Pelvic US clear
Fungal folliculitis on chest (following use of Clomiphene). Worse for heat.
Folliculitis initially exacerbated by inappropriately prescribed antibiotics.
Subsequently tried oral Sporanox and topical nizoral to little or no effect.
Dark hair growth, thinning hair on head, Sugar cravings, hot flushes.
Periods regular (5-6/28-30), PM mastalgia, irritability and aggression

PMH
Eyes feel tired for past 6 years. Wears glasses (Recent eye test) tried eye drops – no effect
Gets stressed easily. Feels unable to relax

DH
B complex, folic acid, garlic

Tongue
Reddish purple tongue body (Heat and blood stagnation)
Red and swollen sides and tip (Liver and heart fire)
Dry yellow coating down both sides (Heat drying fluids)

BP: 135/85, **Pulse:** 100bpm regular and narrow

Rx

Vitex agnus castus	FE	5	(PMS, mastalgia)
Bupleurum chinense	1:3	35	(Improve liver function, reduce Liver Fire)
Anemone pulsatilla	1:3	10	(Eye pain, nervine)
Paeonia lactiflora	1:3	45	(Reduce androgens)
Glycyrrhiza glabra	FE	15	(Reduce androgens, Yin tonic)
Melissa officinalis	1:1	20	(Nervine – Reduce Heart Fire (or *Humulus* etc.)
Cimicifuga racemose	1:3	15	(Reduce LH, reduce hot flushes)
Berberis aquifolium	1:3	35	(depurative for skin (or *Trifolium, Arctium* etc.)
Centella asiatica	1:3	30	(Adaptogen, move Blood, improve skin)
		210	

Sig 10ml tds a.c. cum aq.

Recommendations
Low GL diet
Vitamin B Complex, Magnesium, Evening primrose oil Probiotics

After 6 months
Eye symptoms and PMS resolved, stress levels much reduced
Folliculitis improved but still has breakouts. Still has hirsutism
Progesterone 23 on day 21
BP reduced to 70 bpm

After a further 6 months
Progesterone 56 on day 21

Patient subsequently tried other treatments including bioidentical hormones, Dianette and spironolactone, and each time experienced significant worsening of symptoms after discontinuing the treatment.