A PATIENT’S JOURNEY

Anorexia nervosa

Rebecca McKnight, Nicky Boughton

Rebecca was diagnosed with anorexia nervosa at age 15 but is now a qualified doctor who is able to maintain a healthy weight, manage her workload, and engage in normal relationships.

I was recently asked to sum up my experience of anorexia nervosa in one sentence—actually, I can do it in just one word—isolation. This may seem surprising, but when you have spent a decade fighting an illness that forces you to go against the natural instinct to protect yourself, and that creates a life so constrained by rules that it is impossible to live normally, you feel completely alone. Anorexia makes you change your view of yourself, always to one that is highly negative. Reversing this is a challenge, and one that is easily underestimated. I have had to rise to this challenge while at medical school, and now that I have finished training I have begun to reflect on how anorexia has affected me and how lucky I have been to overcome it. I wrote the following while I was very ill in hospital. It gives an insight into the pervasive nature of anorexia and why it has the highest mortality of any psychiatric condition:

“I am stuck in a black hole I have dug for myself. It is entirely my fault. I am fat, useless and hopeless. I want to get better, but it is hard to make positive decisions when the anorexic thoughts are making me obey their rules. Some describe the anorexia as a gremlin on their shoulder, but I think it’s more like something infiltrating my mind, putting in negative thoughts whenever there is a momentary gap. The ‘voice’ isn’t like a hallucination though, as the thoughts are like any others I might have, and I know that they are my own. The problem is that the urges not to eat, to stay thin and feel safe penetrate every waking moment. I have no respite; it is an endless bloody battle in which I am always on the losing side.”

The news

I was a typical victim of anorexia—a high achieving, perfectionist, middle class teenager to whom no one would have thought it would happen. But it did; a gradual two year descent into severe food restriction, overexercise, deviance, and my own little anorexic world. I was 15 years old and extremely weak by the time a diagnosis was made, and it was the first time I had heard of anorexia nervosa. When I received the news it had little effect on me—I was so entrenched in the desire to lose weight and stay inside the “safe” place that anorexia had provided that I couldn’t see beyond the next meal, let alone consider my prognosis. I was in complete denial, insisting that nothing was wrong. For my family it was an enormous shock, not just the diagnosis of a mental illness, but the feeling of helplessness at not being able to feed their own child.

An opportunity missed

I had first visited my general practitioner several months previously because I was concerned that my periods had stopped. He was very approachable and suggested I start the oral contraceptive. I left feeling confused, but dutifully redeemed the prescription and started the pill. I was extremely underweight, so it was unfortunate that the diagnosis was missed, because the anorexia was gradually getting worse. Anorexia commonly presents as amenorrhea, constipation, or chronic fatigue. Because patients with eating disorders lack insight into their condition and early diagnosis is associated with a good prognosis, doctors should always keep these conditions in mind.

Later that year I was referred as an emergency to the adolescent mental health services. There my parents and I were seen by a psychologist, who made the diagnosis.

Web based resources for patients and health professionals

Beat (www.b-eat.co.uk)—The leading UK charity providing information, help, and support for people affected by eating disorders, particularly anorexia and bulimia nervosa
National Eating Disorders Association (www.nationaleatingdisorders.org)—US charity that provides education, resources, and support to people with eating disorders
Bodywhys (www.bodywhys.ie)—The National Eating Disorder Association of Ireland offers support, information, and understanding for people with eating disorders, their families, and friends
Institute of Psychiatry (www.iop.kcl.ac.uk/sites/edu)—Website developed by the eating disorders research team at the Institute of Psychiatry in London. It has resources for patients and carers, plus the latest results of research in the field
Something Fishy (www.somethingfishy.org)—Website with extensive resources, bulletin boards, and chat facilities aimed at promoting recovery from any eating disorder. It also has a useful treatment finder, with worldwide coverage.
A CLINICIAN’S PERSPECTIVE

The eating disorders charity, Beat, estimates that more than 1.1 million people in the United Kingdom have an eating disorder. Of these, 10% are struggling with anorexia nervosa, which has the highest mortality of any psychiatric disorder in people under the age of 65. Although an average general practice will have just two patients with anorexia, the frequent lengthy consultations and cross specialty liaison needed to provide optimum management means they represent a great burden in terms of time and money.

People with anorexia may not want to recover. Although they want to be rid of their symptoms, their illness can give them a sense of control over their life, thus providing predictability and an identity. The idea of giving it up seems terrifying—like suggesting a mother give up her newborn baby to avoid the low mood associated with endless sleepless nights. For many patients this means that the first stage of treatment has to make them want to change, and their resistance can be frustrating for those trying to help—be they family or professionals. It is crucial that patients are not dismissed as being trivial by focusing on weight and shape to measure self worth.

Anorexia poses a considerable risk to physical and psychological health, so both aspects must be tackled together to optimise the chances of success. The physical manifestations of anorexia are classically those of starvation, but other behaviours—vomiting, taking laxatives, and overexercising—may also be presenting features. In a patient with osteoporotic fractures, subfertility, and an incompetent lower oesophageal sphincter, many specialties may be involved. A high index of suspicion by all doctors and good communication with the referring psychiatrist or general practitioner are essential. The greatest challenge for a person with anorexia is to acknowledge the condition and develop insight.

I have treated Becky for five years and have seen her at many different stages of her illness. Being in Oxford, she has had access to a specialist service that can offer patients intensive treatments and support until they are ready to change. In areas where specialist services are not available, stretched primary care trusts must decide whether to send patients out of area for expensive treatments that may not work. This inevitably occurs when life is threatened—long inpatient stays then seem the only option, because local facilities cannot provide “step-down” day patient or outpatient care. Unfortunately, the nature of anorexia means that this cycle may be repeated many times. As Becky points out, refeeding is merely the beginning, and guidelines from the National Institute for Health and Clinical Excellence recommend that psychological follow-up continues for at least one year after discharge. The current NHS emphasis on time limited treatments therefore makes appropriate treatment of anorexia challenging. Often patients are simply discharged back to the general practitioner, who not surprisingly feels abandoned with a very ill patient.

Severe anorexia is a difficult illness to treat in a health service with limited resources. However, with specialist help given over an adequate time, it is possible for the patient to recover and, like Becky, return to a normal and productive life. Nicky Boughton Nicky.Boughton@obmh.nhs.uk

What has helped and not helped me fight anorexia

**Helpful**

- Having a supportive general practitioner in recent years
- Seeing the same (excellent) psychologist—who has not given up hope in me despite several relapses—throughout my inpatient and outpatient treatment
- Learning to trust that my treatment team will not allow me to “get fat” and that their advice is only for my benefit
- My family learning about eating disorders, so they can understand my struggles
- Building up my self esteem—friends and family are important for this, especially in the competitive environment of medical school
- Realising that the anorexia won’t just disappear, that I have to ignore it until it gradually backs down
- Becoming involved with the eating disorders charity, Beat, and helping others who have eating disorders
- Broadening my world beyond my career—playing in a band again and doing gentle exercise

**Not helpful**

- Late diagnosis because the general practitioner was not “on the ball”
- Lack of specialist treatment when I was a teenager
- People telling me just to start eating again and to stop being so stubborn
- Not admitting there is a problem and burying my head in the sand
- Being abandoned by most of my peers
- Struggling with the stigma associated with the “size zero” culture and having a mental illness
- Some doctors seeing mental illness in a negative light

Anorexia poses a considerable risk to physical and psychological health, so both aspects must be tackled together to optimise the chances of success. The physical manifestations of anorexia are classically those of starvation, but other behaviours—vomiting, taking laxatives, and overexercising—may also be presenting features. In a patient with osteoporotic fractures, subfertility, and an incompetent lower oesophageal sphincter, many specialties may be involved. A high index of suspicion by all doctors and good communication

and offered us advice on home refeeding and family therapy sessions. This is standard management of an adolescent with anorexia, but my experience makes me feel that direct referral to a specialist eating disorders team would have been much more helpful. Because of the lack of inpatient facilities in my area, my family was left to manage the situation at home. My parents’ perseverance against the stubbornness and deviousness born of anorexia allowed me to reach a safe weight, but I received no help in tackling the causes behind my illness. My family had been assured that my recovery hinged on weight gain (true), with the suggestion that this would be sufficient for me to regain full health (false). When I left home for university I quickly relapsed, which was upsetting for my family. I think it is important to make it clear that recovering from an eating disorder is extremely difficult, and to warn the family of the risk of relapse.

The usual course of anorexia

My illness has followed a course typical of anorexia—a slow development into the full blown clinical syndrome, followed by several relapses despite extensive treatment. Prognosis is usually quoted as the “rule of thirds”—a third fully recover, a third partially recover, and another third have chronic problems. Recovery is slow—it usually takes three to six years. I have had multiple courses of cognitive behavioural therapy and received day patient and inpatient care. As an inpatient I was put on a specialist programme combining refeeding with intensive therapy designed to reduce eating disordered behaviours and improve self esteem. With each course of treatment I made steps towards recovery, but each time I quickly relapsed, because I refused to admit that to succeed I needed to leave the illness completely behind. I believed initially that weight gain would make the “voice” go away, and I know it can happen, but for me it hasn’t. I still have the disordered thoughts, but I try not to act on them, and they are gradually weakening. I always tell others that no hospital or specialist can cure them—they can help initiate changes, but the legwork is done at home, day after day, by resisting the anorexia until you gradually start to win the battle (box).

Making the journey alone

When I was deeply entrenched in anorexia, it was difficult to motivate myself to do anything. My concurrent depression heightened this problem, and I struggled to meet the commitments needed to progress through medical school. It has taken me eight years to complete my train-
ing, during which time my friends have qualified and left me behind. Dropping behind my peers has worsened my feeling of inferiority, and I found it difficult to fit into a new year group. This made me feel very alone, and the safety of anorexia beckoned strongly. I have found that most people ignore the anorexia completely, pretending it isn’t there—this is especially true of clinicians. Those friends that have stuck by me have provided invaluable support and helped me adapt to my changed circumstances. My relationship with my family has been very strained at times, because it is hard to cope with someone who seems to be making such self-destructive choices.

The future
After 10 years, I have finally reached the stage where I can maintain a healthy weight, manage my clinical commitments, and engage in normal relationships. I am by no means completely cured of anorexia—I still fight it daily and feel enormously fat and inferior—but I have turned the corner. Recently, I fractured my patella from a fall, a painful reminder that I have been left with osteoporosis, a common consequence of malnutrition.

No one knows exactly why an eating disorder develops, but when you have endured one the answer becomes irrelevant. I have started putting large amounts of time into raising awareness of the dangers of anorexia—especially among other doctors—both as an aid to my recovery and to prevent others from having to face such an isolating experience.

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LESSON OF THE WEEK
Amennorhoea, menopause, and endocrine therapy for breast cancer

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Prolonged amenorrhoea is not necessarily indicative of menopause

Almost a third of the 1.3 million women diagnosed with breast cancer each year are premenopausal or perimenopausal, and about three quarters have endocrine sensitive disease. Many of these women are treated with cytotoxic chemotherapy. This treatment significantly improves survival outcomes, but it is also associated with an increased risk of temporary or permanent amenorrhoea. In recent years, aromatase inhibitor therapy has become a global standard of care for postmenopausal women with receptor positive disease. However, an increasing number of women who are premenopausal or perimenopausal at the time of diagnosis and whose menses stop with adjuvant chemotherapy are also being given aromatase inhibitors as monotherapy (that is, without concomitant ovarian suppression or ablation), even though their use as single agents is absolutely contraindicated in premenopausal or perimenopausal women. Therefore, accurate determination of menopausal status is vital in the effective endocrine management of women with breast cancer. We present a case of incorrect assessment of menopausal status and discuss its effects.

Case report
A 47 year old woman was diagnosed with a lymph node positive, invasive ductal carcinoma. The tumour was oestrogen receptor and progesterone receptor positive and HER-2/neu receptor negative on immunohistochemistry. At the time of diagnosis, she had not had a menstrual period for six months. She received primary chemotherapy consisting of 5-fluouracil (500 mg/m²), epirubicin (100 mg/m²), and cyclophosphamide (500 mg/m²) (FEC), followed by docetaxel (75 mg/m²) over 24 weeks. After chemotherapy she was started on 20 mg tamoxifen daily as adjuvant endocrine treatment. Tamoxifen was discontinued after 10 months because of intolerable hot flushes. By this time, she had not had a period for almost 18 months. Biochemical tests showed she had raised serum gonadotrophins (follicle stimulating hormone 70 IU/L (postmenopausal reference range 30-150 IU/L) and luteinising hormone 35 IU/L (15-64 IU/L)) and low oestradiol values (68 pmol/L (<100 pmol/L)). She was thought to be postmenopausal and was switched to an aromatase inhibitor. She tolerated this treatment well and denied any major hot flushes, arthralgia, or urogenital symptoms. Nine months later she reported having two episodes of vaginal bleeding, identical to her normal menstruation. Biochemistry showed that follicle stimulating hormone was 46 IU/L, luteinising hormone was 29 IU/L, and oestradiol was 280 pmol/L (<100 pmol/L). She was then referred for a laparoscopic oophorectomy.
Effect of menopause on management of patients with breast cancer

| Health related factor       | Management issues                                                                 |
|----------------------------|----------------------------------------------------------------------------------|
| Sexual health              | Libido; marital relations; vaginal health; gynaecological symptoms (questions about causes of non-cyclical vaginal bleeding after chemotherapy) |
| Reproductive health        | Fertility and likelihood of future pregnancy; need for contraception               |
| Bone health                | Need for prevention of postmenopausal and treatment associated bone loss           |
| Cancer management          | Need for and timing of use of aromatase inhibitor; need for ovarian ablation or suppression |

ing ovarian function after chemotherapy rely on menstruation as the only surrogate marker of ovarian function. However, menstrual history can be misleading, because spontaneous conception can occur in women with amenorrhea secondary to premature ovarian failure induced by chemotherapy.

Menopause is classically defined as the absence of menstrual periods for 12 consecutive months with no cause. Accurate assessment of menopausal status is crucial in view of its effect on patient management (see table). The treatment of premenopausal or perimenopausal women with aromatase inhibitors can have serious consequences. These can include pain from ovarian hyperstimulation induced by aromatase inhibitors, as well as the increased risk of unplanned pregnancy.

Aromatase inhibitors can also trigger a reflex increase in gonadotrophins, which causes an increase in ovarian production of aromatase and oestrogens, and can lead to an increased risk of breast cancer recurrence. It is difficult to measure this risk owing to a paucity of published data in this area. However, studies of different durations of adjuvant endocrine therapy have shown that effective oestrogen suppression for five years reduces the risk of disease recurrence by about 25% compared with shorter durations.

It can be difficult to define the menopause in breast cancer patients, especially in those who have received chemotherapy, tamoxifen, or both. A review of breast cancer patients with chemotherapy induced amenorrhea who were aged 40 or more and were started on aromatase inhibitors showed that 27% regained ovarian function after initiation of treatment at a median of 12 months (range 4-59 months). An arbitrary threshold of 12 months of amenorrhea in women who are premenopausal and perimenopausal before chemotherapy is therefore inappropriate. The picture is further complicated by the use of tamoxifen after chemotherapy, a treatment associated with amenorrhea in up to 90% of premenopausal or perimenopausal women. Moreover, switching from tamoxifen to aromatase inhibitors can also result in the return of menstrual bleeding even after a prolonged period (for example, five years) of amenorrhea.

In addition, the hot flushes that many women on tamoxifen experience can also give the false impression of established menopause.

Clinicians often try to verify the clinical diagnosis of menopause with routine biochemical tests such as raised gonadotrophins or depressed concentrations of oestradiol. Unfortunately, these biomarkers have no single cut-off point, with evidence supporting a cyclical process occurring during the perimenopause and early years of the menopause. The variability of these markers in the perimenopausal period means that even if serum markers are assessed before chemotherapy, they might not reflect a permanent menopausal state. These tests can also be difficult to interpret—both follicle stimulating hormone and oestradiol have poor predictive power for ovarian function in patients who have had chemotherapy and in those taking tamoxifen. Therefore, the use of gonadotrophins and oestradiol as surrogate markers for the menopause in these patients is unclear.

In the setting of fertility and assisted reproduction, non-invasive investigations of ovarian reserve are used to help predict the outcome of assisted reproduction techniques. A recent study of predictors of ovarian reserve in premenopausal women with breast cancer evaluated several biochemical and biophysical parameters of ovarian reserve. Results showed that the sonographic measurement of antral follicle count, as well as basal and stimulated serum antimüllerian hormone, follicle stimulating hormone, inhibin B, and oestradiol were different in breast cancer patients compared with age matched controls who had proved fertility, normal menstrual history, and no medical illness. A further study of premenopausal breast cancer survivors who were all more than one year from diagnosis and had no evidence of recurrence concluded that premenopausal breast cancer survivors had reduced ovarian reserve compared with controls, and that the largest differences between survivors and controls were seen in the antral follicle count and antimüllerian hormone concentration. However, the value of these markers in breast cancer has been tested only in case-control studies. This method has inherent weaknesses, so more robust data from prospective studies are needed before these tests can be recommended for routine use. Such studies are ongoing.

We recommend that doctors who regularly prescribe endocrine therapy to premenopausal or perimenopausal women with breast cancer be wary of using menstruation history or biochemical tests to diagnose ovarian failure. It is difficult to confirm the menopause in this setting, and the consequences of an incorrect diagnosis can be serious. If an aromatase inhibitor is indicated, we therefore recommend the use of ovarian ablation or suppression. Clinicians should discuss the relative benefits of the different methods of ovarian ablation (oophorectomy or ovarian radiotherapy) and suppression (use of luteinising hormone releasing hormone agonists) with their patient. Tamoxifen is the only safe endocrine treatment in women who decline ovarian ablation or suppression.
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1 Ferlay J, Bray F, Pisani P, Parkin DM. GLOBOCAN 2002 Cancer Incidence, Mortality and Prevalence Worldwide. IARC CancerBase No 5, version 2 Lyon: IARCPress, 2004.

2 American Cancer Society. Breast cancer facts and figures. 2007-8. www.cancer.org/downloads/STT/bcf7-final.pdf

3 Cancer Care Ontario and the Public Health Agency of Canada. Cancer in young adults in Canada. http://www.phac-aspc.gc.ca/publicat/ cync-cjac06/pdf/cync-cjac-2006_e.pdf

4 Early Breast Cancer Trialists’ Collaborative Group. Tamoxifen for early breast cancer: an overview of the randomised trials Lancet 1998;351:1451-67.

5 Early Breast Cancer Trialists’ Collaborative Group. Polychemotherapy for early breast cancer: an overview of the randomised trials. Lancet 1998;352:930-42.

6 Winer EP, Hudis C, Burstein HJ, Bryant J, Chiebowski RT, Ingle JN, et al. American Society of Clinical Oncology technology assessment working group update: use of aromatase inhibitors in the adjuvant setting. J Clin Oncol 2003;21:2597-9.

7 Luitchman Singh K, Mutukrishna S, Stein RC, McGarrigle HH, Patel A, Parikh B, et al. Predictors of ovarian reserve in young women with breast cancer. Br J Cancer 2007;96:1806-16.

8 Anderson RA, Themmen AP, Al-Qahatani A, Groome NP, Cameron DA. The effects of chemotherapy and long-acting gonadotrophin suppression on the ovarian reserve in premenopausal women with breast cancer. Hum Reprod 2006;21:2583-92.

9 Petrek JA, Naughton MJ, Case LD, Paskett ED, Naftalis EZ, Singletary SE, et al. Incidence, time course, and determinants of menstrual bleeding after breast cancer treatment: a prospective study. J Clin Oncol 2006;24:1045-51.

10 Tham YL, Sexton KE, Weiss H, Ellerage R, Friedman LC, Kramer R. The rates of chemotherapy-induced amenorrhea in patients treated with adjuvant doxorubicin and cyclophosphamide followed by a taxane. Am J Clin Oncol 2007;30:126-32.

11 Vital-Reyes V, Tellez-Velasco S, Chihdiq D, Grizzle W, Reyes-Fuentes A. Spontaneous pregnancy in a woman with premature ovarian failure: a case report. J Reprod Med 2004;49:969-91.

12 Schinzinger A. Ueber carcinoma mammae [abstract]. 18th Congress of the German Society for Surgery. Beilage zum Centralblatt fur Chirurgie 1889;16:55-6.

13 Smith IF, Dowsett M. Aromatase inhibitors in breast cancer. N Engl J Med 2003;348:2431-42.

14 Mitwalli MF, Casper RF. Use of an aromatase inhibitor for induction of ovulation in patients with an inadequate response to clomiphene citrate. Fertil Steril 2001;75:305-9.

15 Smith IF, Dowsett M, Yap YS, Walsh G, Lanning PE, Santen RJ, et al. Adjuvant aromatase inhibitors for early breast cancer after chemotherapy-induced amenorrhea: caution and suggested guidelines. J Clin Oncol 2006;24:2444-7.

16 Sacco M, Valentinii M, Beffiglio M, Pellegrini F, De Berardis G, Franciosi M, et al. Randomized trial of 2 versus 5 years of adjuvant tamoxifen for women aged 50 years or older with early breast cancer: Italian Interdisciplinary Group Cancer Evaluation Study of Adjuvant Treatment in Breast Cancer 01. J Clin Oncol 2003;21:2276-81.

17 International Breast Cancer Study Group; Colleoni M, Gelber S, Goldhirsh A, Aebi S, Castiglione-Gertsch M, et al. Tamoxifen after adjuvant chemotherapy for premenopausal women with lymph node-positive breast cancer. International Breast Cancer Study Group Trial 13-93. J Clin Oncol 2006;24:1332-41.

18 Hargis JB, Nakajima ST. Resumption of menses with initiation of letrozole after five years of amenorrhea on tamoxifen: caution needed when using tamoxifen followed by aromatase inhibitors. Cancer Invest 2006;24:174-7.

19 Ovelrie I, Moen MH, Morkrid L, Skjaeraasen JS, Holte A. The endocrine transition around menopause—a five years prospective study with profiles of gonadotropines, estrogens, androgens and SHBG among healthy women. Acta Obstet Gynecol Scand 1999;78:642-7.

20 Delrio G, De Placido S, Pagliarulo C, d’Istria M, Fasanò S, Marinelli A, et al. Hypothalamic-pituitary-ovarian axis in women with operable breast cancer treated with adjuvant CMF and tamoxifen. Tumori 1996;72:53-61.

21 Lum SS, Woltering EA, Fletcher WS, Pommier RF. Changes in serum estrogen levels in women during tamoxifen therapy. Am J Surg 1997;173:399-402.

22 Bukulmez O, Arić A. Assessment of ovarian reserve. Curr Opin Obstet Gynecol 2004;16:231-7.

23 Kwee J, Schats R, McDonnell I, Themmen A, de Jong L, Lambalk C. Evaluation of anti-Mullerian hormone as a test for the prediction of ovarian reserve. Fertil Steril 2008;90:737-43.

24 Clemons M, Simmons C. Identifying menopause in breast cancer patients: considerations and implications. Breast Cancer Res Treat 2007;104:115-20.

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Do you see what I see?

I lost my right eye in 1949—or rather it was enucleated on the advice of an eminent eye surgeon to prevent my developing sympathetic ophthalmoplegia in the other eye. To my parents, my losing an eye was a total disaster, but to me it has been rather fun and without doubt a positive factor in my development. My parents brought me up to accept that I was “different” but “special” different. I must have had some difficulties with depth perception but I was so young at the time of my accident that I have largely learnt to compensate. I drive, play a reasonable game of tennis, and only occasionally miss a glass when pouring out the wine. Having been called names such as boss-eyed or Cyclops and believing that I would never be pretty, I determined to work hard and from an early age decided to be a doctor.

There have been few occasions when I have felt myself to be “handicapped.” When my classmates were all raving about their new Viewmaster toys I (literally) couldn’t see what all the fuss was about. Likewise, magic pictures never worked for me, and at medical school the second eyepiece on a microscope was a waste of time. As a GP, I can’t examine fundi with the correct eye and checking visual fields requires some imagination. The ophthalmic registrar who was giving me a hard time over an urgent referral was a bit taken back by my reply to his “all you need is two fingers and two eyes” statement.

Recently, all these experiences came back to me as I sat with my grandchildren watching a 3D presentation of the film UP, which had received such an excellent review in the BMJ. My grand-daughter was right when she loudly announced to all and sundry that I wouldn’t need the glasses because I only had one eye. It was an excellent film, but when everyone else was “oohing and aahing” I found myself asking the kids “what’s happening?”

I wondered if I should have asked for a discount, as I did when the optician ordered two very expensive varifocal lenses for my glasses when only one was needed. Mainly though, I wondered about the fact that none of us really know how what we see compares with what other people see—the old “is my red the same as your red” dilemma.

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10-MINUTE CONSULTATION

Hirsutism

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A 20 year old woman of Indian origin attends your surgery with an escalating excess growth of dark hair on her face, arms, and thighs. She has a body mass index of 27.

What issues you should cover

Hirsutism is the presence of terminal hair (long, coarse, and pigmented) in women and girls, in a male androgen sensitive pattern. This must be differentiated from hypertrichosis, which is generalised hair growth not exclusive to androgen sensitive areas (caused by—for example, the use of Minoxidil). Hirsutism affects 5-15% of women of reproductive age. Androgens, principally testosterone (from the adrenal glands and ovaries), increase hair growth by converting fine, unpigmented vellus hair to terminal hair in androgen sensitive areas such as the face, chest, and abdomen.

What you should do

Ask about:
• Why the patient has come to see you now—cosmetic appearance or psychological distress may be factors
• History of hirsutism—gradual or abrupt onset, extent, and progression
• Reproductive and menstrual history
• Family history of hirsutism, diabetes, or polycystic ovary syndrome
• History of acne
• Male pattern frontal balding
• Deepening of the voice
• Clitoromegaly
• Drug history—for example, progestogenic contraceptive pills, anabolic steroids, and sodium valproate

Patient presents with hirsutism

Mild hirsutism, regular menses

Moderate or severe hirsutism, with or without irregular menses

No further investigations

Measure free testosterone (check local lab for availability)
Prolactin, luteinising hormone, follicle stimulating hormone (if oligomenorrhoea or amenorrhoea is present)
Ovarian ultrasound scanning (if polycystic ovary syndrome (PCOS) is suspected)

Manage hirsutism (and PCOS if present) in primary care

Free testosterone ≥ 4 nmol/l rapid and abrupt progression of hirsutism; virilisation
Hyperpigmentation
Cushingoid features
Galactorrhoea or raised prolactin
Hirsutism resistant to treatment in primary care
Fertility treatment associated with PCOS

Refer to a specialist

USEFUL READING

Information for clinicians

Martin KA. Evaluation and treatment of hirsutism in premenopausal women: an endocrine society clinical practice guideline. J Clin Endocrinol Metab 2008;93:1105 (doi:10.1210/jc.2007-2437)
Yildiz BO. Assessment, diagnosis and treatment of a patient with hirsutism. Nat Clin Pract Endocrinol Metab 2008; 4:294-300 (doi:10.1038/ncpendmet0789)

Information for patients

Patient UK (www.patient.co.uk/showdoc/40000957)—Comprehensive health information

• Age—in young children it may indicate precocious puberty.
• Assess the extent of the hirsutism using the modified Ferriman-Gallwey score. Examine hair growth in these nine sites—upper lip, chin, chest, upper back, lower back, upper abdomen, lower abdomen, arm, and thigh) and rate it from 0 to 4, where 0 indicates no terminal hair growth and 4 indicates complete and heavy cover. The maximum score possible is therefore 36; a score greater than 6 indicates hirsutism. This is a subjective assessment so also consider your patient’s perception of excess hair when making your diagnosis.
• Measure body mass index, waist circumference, and blood pressure. Look for signs of acne and temporal balding. Do not examine the genitalia unless gross signs of virilisation are present.
• Consider the possibility of polycystic ovary syndrome, and make the diagnosis if two of the following three phenomena are present:
  • Oligo-ovulation or anovulation manifesting as oligomenorrhoea or amenorrhoea
  • Clinical (for example, hirsutism) or biochemical (for example, raised testosterone) signs of hyperandrogenaemia
  • Polycystic ovaries on ultrasound scanning.
• Consider suggesting depilatory treatments or bleaching. Say that more permanent methods such as electrolysis or laser removal have no guarantee of success. Advise weight loss, which can be driven by insulin resistance. Address any psychological issues.
• If appropriate, in moderate to severe cases prescribe the oral contraceptive pill to target the underlying hormonal imbalance. For facial hair, give Vaniqa (eflornithine hydrochloride), a hair growth enzyme inhibitor. Advise the patient not to get pregnant while using the oral contraceptive pill or Vaniqa. Follow-up after three months.

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