Read Item - Androgenetic Alopecia

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Abstract: Doctor's resource on Androgenetic Alopecia for Men and Women, Surgery, Wigs

Androgenetic Alopecia

Androgenetic alopecia is a progressive, largely irreversible patterned loss of an excessive amount of hair from the scalp. It occurs in both men and women as well as certain other primates. Prerequisites are a genetic predisposition and the presence of sufficient circulating androgens. Eunuchs do not develop androgenetic alopecia. Between 95% and 100% of the population possess the polygenic inherited predisposition, but far fewer develop significant alopecia due to variations in penetrance and expression.

Limited androgenetic hair loss affects all men and women progressively as they age. This may manifest only as an alteration in the frontal hair line. Excessive or premature hair loss, which defines androgenetic alopecia thus represents an exaggeration of a physiological event rather than a disease per se.

Paradoxically the influence of androgens on hair is site specific. Pre-pubertal pubic, axillary, beard and chest vellus hair follicles react to androgens by growing into terminal hairs. The same androgens miniaturise the pigmented terminal hairs on the frontovertical region of the scalp into non-pigmented vellus hairs. Interestingly, there is no correlation between the degree of baldness and the density of hair patterns on the trunks and limbs of males. To date there is no satisfactory explanation for this differential effect, nor for the reason many other hair follicles, including the occipital scalp hairs are much less influenced by androgens. Exploiting these site specific effects is the basis for successful hair transplantation.

Two events combine to produce the androgenetic alopecia. Firstly there is a prolongation of the telogen phase of the hair cycle with associated shortening of anagen, and secondly and more importantly the calibre of the anagen hairs produced by the follicles becomes progressively smaller. Scarring is not a feature of androgenetic alopecia and there is no actual reduction in the number of follicles on the scalp. Decreased pigmentation further diminishes the cosmetic significance of these hairs. This combination of an altered trichogram and miniaturisation of the scalp hair follicles occurs gradually over many years in a reproducible patterns. These patterns have been described and graded by Hamilton and Ludwig.

The diagnosis of androgenetic alopecia is a clinical one and is based on recognising the pattern of hair loss. A hair pull test is positive when an increased number of telogen hairs are extracted from the frontovertical region, but not the occiput, in keeping with the pattern of the altered trichogram. In doubtful cases analysis of the trichogram of clumps of plucked hairs from different regions of the scalp will further support the diagnosis. A scalp biopsy is also helpful in difficult cases, but is not required routinely.

At scanning magnification the biopsy shows an apparent decrease in the number of hair follicles, and the sebaceous glands appear relatively large. An increased proportion of the anagen follicles are vellus follicles and solar elastosis may be apparent. Streamers (remnants of involuted anagen hairs) and a lymphohistiocytic inflammatory infiltrate around the hair follicle at the level of the sebaceous duct and also around the superficial dermal capillaries are characteristic. This infiltrate appears to be important in the development of the alopecia, but the mechanism of its effect remains speculative.

The follicular miniaturisation is global affecting the papilla, the matrix and ultimately the hair shaft. Terminal hairs initially transform into indeterminate hairs and later into vellus hairs that

ultimately become so short they do not reach the follicular ostium. These 'secondary' vellus follicles differ from de novo vellus follicles in that they still have the remnants of their arrector pili muscle attached to them. Miniaturisation of the follicles appears to be due to the local effects of androgen excess and this is suggested by a number of observations.

Firstly androgenetic alopecia does not occur in eunuchs castrated prior to puberty, but can be induced by the administration of testosterone to those genetically predisposed. If the testosterone is discontinued the alopecia does not progress, although it also does not reverse.

Secondly, virilisation produces androgenetic alopecia in women with a genetic predisposition combined with acne, hirsutes and menstrual irregularity.

Thirdly antiandrogen therapy slows the progression of androgenetic alopecia. It may also partly reverse the miniaturisation.

Fourthly scalp dermal papilla cells have the ability to activate circulating testosterone and the receptors to recognise these enhanced androgens. This is done enzymatically by 5a reductase that converts testosterone into the more potent dihydrotestosterone. Regional variation exists in both the activity of 5a reductase and the density of androgen receptors in the dermal papillae, with greatest levels being found in the frontoparietal areas of the scalp corresponding to the areas of development of androgenetic alopecia. Very high levels are also found in the pubic region and other areas of secondary sexual hair development. As mentioned previously, there is still as yet no adequate explanation for why some follicles are triggered by dihydrotestosterone to enlarge while others are stimulated to miniaturise. The possibilities that different isoenzymes of 5a reductase, different regional metabolites of DHT, different regional receptor subtypes or different second messenger processing of the signal account f
or the different effects requires further investigation.

Finally punch grafts taken from the occiput of men with androgenetic alopecia and transferred to the frontoparietal area maintain the behaviour of the donor site after transplantation. This implies that the interpretation of the androgen signal locally by the individual follicle is relatively more important than the amount of circulating androgen.

Men have sufficient circulating androgen to maximally stimulate hair follicles such that all genetically predisposed men will develop alopecia. However, androgens in the normal female range only induce balding in premenopausal women with a strong genetic predisposition. In women with a less strong genetic predisposition, baldness only develops when androgen production is increased or drugs with androgen-like activity are taken.

Seborrhoea is often observed in association with common baldness, but is not aetiologically related.

Until recently it was accepted dogma that the miniaturisation of hairs was an irreversible process. The dramatic, but exceptional regrowth that has been documented with minoxidil has led to its? reappraisal and motivated a large research effort to finding the key to unravel the complex series of events that will reverse androgenetic alopecia.

Male Patterned Baldness

Hamilton described the distinctive pattern of progression of hair loss in men and graded the severity on a scale of I to VIII. Alteration of the frontal hair line with bitemporal recession occurs first and is followed by balding of the vertex. Eventually a more uniform frontal recession joins the bald areas and the entire frontovertical region bears only an inconspicuous secondary vellus hair. The posterior and lateral scalp margins are spared even in advanced cases.

Hamilton's Type I describes the normal prepubertal pattern, and post-puberty this may still be

seen in some women, but is rare in adult men. Progression to type II occurs in at least 96% of men. It is usually detectable clinically by the age of 17, however many men do not notice it until their thirties. Type V to VIII occurs in 60% of men over the age of 50 years and the balding progresses continuously until death.

Even before alopecia is evident, an increase in the number of terminal hairs less than 4 cm long is detectable. This is due to the progressive shortening of anagen producing shorter hairs and is associated with a positive hair pull test in the affected areas. Subsequently the proportion of vellus hairs increases and the meaningful hair density (non-vellus hairs per cm2) decreases. Eventually many miniaturised vellus hairs fail to emerge from the follicle and the total hair density declines.

The prepubertal meaningful hair density of the scalp is approximately 200 non-vellus hairs per cm2 and a reduction of 30% to 140 non-vellus hairs per cm2 is required before balding is obvious. Once balding is obvious meaningful hair loss tends to progress at a rate of 10% per year and total hair loss at a rate of 5%.

The diagnosis of androgenetic alopecia is clinical and based on the pattern of hair loss. A positive hair pull test in affected areas and a positive family history support the clinical diagnosis, but are not necessary. It is very rare to require a trichogram demonstrating an increase in telogen hairs for diagnosis.

Premature balding also occurs in progeria and Werner's syndrome and early frontoparietal recession with greying is seen in myotonic dystrophy. Other signs of these disorders will allow a specific diagnosis. Occasionally a diffuse alopecia can coexist with androgenetic alopecia and will be suggested by a history of rapid deterioration and a positive hair pull test from all over the scalp. In such circumstances a drug history, thyroid function tests and a serum ferritin are indicated.

Female Patterned Baldness

Hamilton pattern balding also occurs in women. Hamilton suggested 79% of women develop Hamilton II alopecia after puberty and 25% of women develop Hamilton V by the age of 50, after which time there is minimal progression, but the true incidence is somewhat less than this. Bitemporal recession tends to be less prominent in females than males and is likely to go unnoticed until the late twenties.

Although male patterned alopecia occurs in women, more commonly females develop a hair loss over the crown with preservation of the frontal hair line. This pattern of alopecia was first described by Ludwig and the most useful grading scale for women bears his name.

The earliest change (Ludwig grade I) is thinning of the hair on the crown. This produces an oval area of alopecia encircled by a band of variable breadth with normal hair density. Frontally the fringe is narrow (1-3cm) while at the sides the margin is 4-5 cm wide. Progression to Ludwig grade II results in further rarefaction of the crown with preservation of the fringe. Grade III is near complete baldness of the crown.

The relative incidence of Ludwig versus Hamilton pattern alopecia among balding women has been determined. Ludwig pattern I-III occurs in 87% of premenopausal women while Hamilton stage II-IV occurred in 13%. Among postmenopausal women, Ludwig I-III occurred in 63% while Hamilton II-V occurred in 37%.

The presence or absence of virilisation cannot be inferred from the pattern of alopecia, be it Hamilton or Ludwig. More useful is the rate of development of the alopecia, its severity and any associated evidence of androgen excess such as hirsutes, acne, menstrual irregularities and cliteromegaly. The vast majority of women do not require any investigation for virilisation other than a directed history and examination. The causes and investigation of virilisation are discussed elsewhere. It is usually more relevant to direct investigations towards excluding other causes of a diffuse alopecia, especially in patients with early androgenetic alopecia

when the pattern of loss is difficult to discern.

The diagnosis of androgenetic alopecia in a female is a clinical one based on the pattern of hair rarefaction. It is supported by a positive hair pull test restricted to the crown and a strong family history of baldness, but their absence does not exclude the diagnosis. A trichogram will distinguish telogen effluvium. Thyroid function tests and a serum ferritin estimation will exclude other most causes of a diffuse alopecia. Only occasionally is a scalp biopsy required.

Pharmacological Therapy for Men

Camouflage is the simplest, easiest, cheapest and most effective way of dealing with mild androgenetic alopecia. Balding becomes most noticeable when the scalp can be seen through the hair. Camouflage treatments dye the scalp the same colour as the hair, and give the illusion of thicker hair. Numerous brands are available in pressurised spray cans in a number of different colours and they are often combined with a holding hair spray (and sunscreen). The hair is dried and styled before the dye that matches the patients hair colour is sprayed onto the base of the hair. Although many of the newer agents are water resistant, problems may still arise in the rain if the hair gets wet and the dye runs. In addition patients should avoid touching their hair as the dye will colour their hands. Towels and pillow cases may stain, but these come out in the wash. Patients are advised to remove the dye each night by shampooing and to reapply the dye each morning.

Antiandrogens feminise men and so are not an appropriate therapy for balding men. The only effective pharmacological treatment for men is topical minoxidil. Minoxidil is a vasodilator that was developed for the treatment of hypertension. Hypertrichosis mainly of the body and to a lesser extent of the scalp was noticed as a side-effect, and subsequently a topical preparation for use in androgenetic alopecia was developed.

Numerous dosing studies have been done. The minimum concentration that will increase non-vellus hair counts is 0.1%. The minimum concentration that will produce cosmetically acceptable regrowth is 1% and the optimal result is obtained with 2%. There is no added benefit from using 3%. One millilitre should be applied directly to the bald area twice daily and gently massaged in. The scalp must be dry when minoxidil is applied and the hair should not be wetted for 1 hour after the application. Once daily use is not sufficient for maintenance, and there is no extra benefit with applications more frequently than twice daily.

If successful, after 2 months of continuous minoxidil use hair shedding decreases and hair regrowth may be detected at 4 to 8 months. The hair counts usually stabilise after 12 to 18 months whereas control groups continue to progressively lose hair at 5% per year. Occasionally regrowth does not begin for up to 12 months and the treatment should not be abandoned due to lack of efficacy before then. Most people will get a regrowth of indeterminate hairs, but for many this is not cosmetically significant and insufficient to warrant the expense of treatment. Very occasionally there is a dramatic response with near reversion to normal, but this is unpredictable.

Useful prognostic factors for regrowth are the severity and the duration of the alopecia. Good prognostic factors are (1) a brief history of balding (fewer than 5 years), (2) limited alopecia on the vertex (less than 10cm diameter), and (3) more than 100 indeterminate hairs in the treated area.

If successful treatment needs to be continued indefinitely because if stopped any new hair will fall out and regression to the pre-treatment state will occur within three months. Minoxidil has been used together with tretinoic acid to enhance penetration and initial trials show promise for this combination.

The side effects of topical minoxidil include pruritus, a contact irritant dermatitis and occasionally contact allergic dermatitis can develop. Hypotension does not occur with topical treatment because there is minimal systemic absorption. Oral minoxidil has also been used, however it appears to be no more effective than topical minoxidil. In addition the systemic side-effects contraindicate its routine use for androgenetic alopecia.

Recently low dose Finasteride, a selective inhibitor of 5a reductase has be used in pilot studies to treat androgenetic alopecia in men with promising results and minimal side-effects. Circulating levels of testosterone are unaffected and there is no obvious feminisation or alteration of libido. Placebo controlled human studies are now in progress, but the results are not yet available, and finasteride is not currently licensed for this indication.

Pharmacological Therapies for Women

Minoxidil also works for women. Most women notice decreased hair shedding and some regrowth of non-vellus hairs, however in many the regrowing non-vellus hairs are indeterminate hairs rather than terminal hairs. Only one-third of women notice cosmetically significant regrowth.

Spironolactone, an aldosterone antagonist used primarily as a potassium sparing diuretic also has antiandrogenic properties that can be used to treat androgenetic alopecia. It appears to inhibit the interaction between dihydrotestosterone and the intracellular receptor, as well as inhibiting ovarian androgen production. The dose range is 50-200 mg per day however, the optimal dose of aldosterone is 100 mg daily. This tends to slow the progression of balding without reversing the process. Much of the data on aldosterone relates to its use in hirsutes and few trials have been conducted on its use in androgenetic alopecia. A contraceptive pill is not mandatory with this agent, but women of child bearing age should be warned against becoming pregnant while on this medication due to the risks of feminising a male child.

Systemic antiandrogen therapy with cyproterone acetate (as described for hirsutes) decreases hair shedding but there is generally no cosmetically significant regrowth. In premenopausal women a contraceptive pill should be used with this agent. The effect are generally not noticed for 3 to 6 months after commencing treatment and they tend to continue only for as long as the tablets are taken. About one-third to one half of women taking 100 mg of cyproterone acetate notice a major reduction in hair fall. Lower doses do not appear to work as effectively. The trial data presently available is limited and should be interpreted cautiously. Comparative trials are required to judge the relative benefits of aldosterone and cyproterone acetate.

The combination of topical minoxidil with systemic anti-androgens could be more efficacious than either used alone. Properly conducted trials to verify this clinical suspicion are awaited. Despite considerable effort, there is as yet no effective topical anti-androgen available.

Hair Transplantation and Scalp Surgery

Androgenetic alopecia in women presents with a thinning of hair over the vertex. It rarely produces bald patches suitable for corrective surgery, and the techniques discussed here apply predominantly to men.

All surgical procedures aim to use androgen unresponsive parietal and occipital hairs to cover the bald areas. Relocated hairs behave as they did prior to moving, showing little tendency to miniaturise in their new home. Numerous different techniques have been used and include:

1. Scalp reduction surgery involves the excision of an ellipse of central bald skin. Tissue expanders can be used to increase the harvest, but require insertion up to 3 months prior to the procedure to give the tissue time to expand. Post operatively some of the initial gain is ultimately lost as scalp laxity returns and the area of alopecia enlarges due to 'stretch-back?. This technique is ideal for patches of non-progressive scarring alopecia.

2. Rotation flaps, such as the Juri flap are used to swing in vascularised tissue to recreate the frontal hair line. Flaps have the advantage of achieving a high density of hair growth, although sometimes it is too dense and looks unnatural. In addition there is less post-operative telogen

effluvium than occurs post-transplantation. One disadvantage of flaps is that they represent an uneconomical use of a restricted supply of donor tissue. Furthermore, if the patient has a large bald spot there may not be enough parietal and occipital skin available to cover the defect. The same applies if the patient returns 5 or 10 years later with progression of his androgenetic alopecia looking for a second graft. Another problem with flaps is with their orientation. Hairs grow in their original direction, and simple rotation directs hair growth posteriorly, exposing the scar and appears unnatural. Newer techniques, such as tunnel flaps are designed to address this. Potential complications of the procedure inclu
de unsightly donor scars and flap devitalisation with consequent loss of valuable donor tissue.

3. Hair transplantation takes advantage of donor dominance (the Orentreich principle), which is the tendency of transplanted hairs to maintain the growth characteristics of their original (the donor) site, independent of the character of the recipient site. Thus occipital and parietal hairs from the scalp margin do not fall victim to androgenetic alopecia when placed on the crown. Punch grafting used to be the most commonly used technique. Multiple 4mm punch biopsies are taken from the scalp margins and inserted into the bald areas. The donor sites can be individually sutured or left to heal by secondary intention. The recipient site is prepared to receive the grafts by creating 3.5mm circular holes in the bald skin with another punch biopsy. Slightly better results are achieved with smaller recipient holes, as the grafts tend to shrink after they are removed, while the recipient holes tend to enlarge slightly. Because the blood supply to the grafts is compromised if they are placed too close togeth
er, the final result may look artificial with discrete tufts of hair separated by bald areas.

This technique can be used to provide hair cover for large areas of bald scalp, the limiting factor being the availability of donor tissue. It is axiomatic that donor plugs should be taken from the hairy areas with the best prospect of retaining their hair during the patients lifetime. The zone of androgen responsive hair is variable between individuals and may be too narrow to provide complete coverage of the defect. Often doubtful hair has to be used and for this reason balding recurs in the transplanted hair in the ensuing 5 to 10 years necessitating a further procedure. This possibility should be carefully explained to the patient in advance. Nevertheless many patients feel 5 to 10 years of hair in some areas is worth the moderate discomfort and significant expense.

The state of the art surgery for baldness is single follicle transplantation. This is gradually replacing punch grafting that tended to produce an unsatisfactory appearance reminiscent of a toothbrush or a doll's head. The technique involves harvesting strips of hair bearing skin from areas likely less sensitive to androgenic alopecia. When harvesting the donor tissue, the surgeon must be careful to angle the incision to conform to the direction of hair growth so as to not transect follicles. The donor site is sutured and the grafts are then dissected by a technician into individual hairs using a scalpel and jeweller's forceps. These hairs are then placed obliquely into holes made with a 18-gauge hypodermic needle or slits made with a fine scalpel blade that are oriented according to the desired direction of hair growth. Three or four sessions (of 300 to 600 grafts) are usually required to achieve the desired hair density, producing a more gradual return of hair growth. The cost to the patient is appro
ximately $US 5-10 per hair.

Single follicle grafts are particularly useful when treating 'early' androgenetic alopecia in men and can also be used for females with androgenetic alopecia as the grafts can be fed in between existing follicles to increase hair density. There is minimal damage to the recipient site using this technique, while punch grafting would require removal of some hair bearing scalp tissue.

However, used as the sole method of covering a large patch of alopecia, single follicle grafts are very time consuming and expensive. Others prefer to use a combination of small punch grafts and single follicle grafts, with the single hairs placed in between the punch grafts to soften the effect. Alternatively single grafts can be used to recreate the fringe, and punch grafts used for the main defect.

The best candidates for grafting are those with light, fine hair, good residual hair density over frontal regions, and minimal contrast between the colour of the hair and the skin. Orientation

of the grafted hairs is important as the hairs will grow in the direction they have been inserted. Shortly after inserting the grafted hairs they undergo a telogen effluvium and it takes between 6 and 12 months before a good cosmetic result is achieved.

Apart from early mild pruritus and scalp oedema post-operative problems are uncommon. The major complications of grafting include hypertrophic scarring, hyperaesthesia, haematoma formation, arteriovenous fistula formation and post-operative infection.

Attempts to increase the pool of donor hairs have not yet proved successful. Hairs grown in culture are only viable for a few weeks and hair cloning in still hazardous. Hair cloning involves transversely bisecting a single donor hair into two pieces through the bulge and implanting the two bisected donors into the scalp in the hope that two new hairs will regenerate from a single donor. The problem with hair cloning is the low survival rate of the transected donors, and this technique need further refinement.

Instructions For Patients Following Hair Transplantation

THE FIRST 24 HOURS
1. You will feel drowsy after the operation so you should make arrangements for someone else to drive you home.
2. Take care getting in and out of the car while your scalp is numb. If you bump your head you may dislodge some grafts.
3. On reaching home rather than lying down it is suggested you rest in an upright position until your normal bedtime.
4. Pot-operative pain is not usually a problem, but if necessary you can take any of the commonly available ?pain killers.? The donor site at the back and side of your scalp is often the most uncomfortable and may remain tender for some days.
5. The bandage and dressing can be removed on the day following the operation. This is best done in the shower and you can lightly shampoo the scalp. An electric drier should be used to dry the hair.
6. If you have a hair piece this can be replaced after drying the scalp, but try to leave this off as much as possible during the first 2 to 3 weeks.
7. Some of the grafts may appear a little raised at first. This is quite normal and generally subsides after a few weeks. The grafts will usually become flat within 6 months, but occasionally this may take longer.
8. Rarely there is some spotting of blood through the bandages overnight. It is usually only necessary to add a further layer of padding and bandages. If the bleeding persists please call me.

THE FIRST 3 WEEKS
9. No special treatment is required for the grafts, but it is suggested that you wash your scalp daily with shampoo until the crusts have separated. Normal scalp washing can then be resumed.
10. There may be slight oozing from the donor region at night so it is suggested that you cover your pillow with a towel until this settles. The stitches in the donor site will dissolve in time. If they become annoying these can be removed after 7 days.
11. After grafting to your frontal region you might expect some swelling around the eyebrows, forehead and occasionally the eyelids. On rare occasions one or two black eyes develop. The swelling usually develops on the third day after the operation and subsides over the next 2 or 3 days. The swelling is a natural response to the operation and no treatment is necessary although the regular application of ice packs will give relief.
12. Light jogging can be resumed two days after surgery. Swimming in the sea is permissible after 5 days, but be careful not to sunburn the areas. More vigorous sports should not be resumed before 10 days.
13. Infection of the grafts is rare but if you should develop swelling or tenderness beneath any of the grafts after a few days, sponge the area with an antiseptic solution and squeeze gently as for a pimple or boil. Antibiotics are rarely necessary but if the condition is not improving please call me.

14. Occasionally some tender swellings are detected behind the ears. These are enlarged lymph glands and will subside in a few days.
15. Crusts will form over each graft within 2 to 3 days. These generally come away after 2 to 3 weeks taking many of the graft hairs with them. You can assist the process by gently rubbing but do not use force. Temporary loss of the graft hairs is expected and these hairs will regrow.
16. If vigorous bleeding should occur at any time apply a clean handkerchief or towel and apply firm pressure to the bleeding point for 10 minutes. Please call me if the bleeding persists.

THE FIRST 12 MONTHS
17. After the graft hairs have been lost there will be a period of several weeks when the transplants are bare and dormant. The new hair will begin to appear after approximately 12 weeks. At first only a few hairs will be seen and these will then grow at the normal rate of one centimetre per month. The number of new hairs will continue to increase for 12 months. Occasionally the graft hairs do not fall out but continue to grow.
18. With second and subsequent operations the onset of the new hairs can be delayed and on rare occasions may not commence until 18 weeks.
19. Often the new hair is a little darker and coarser than the pre-existing hair. The new hair may also have a slight crinkle but the texture returns to normal with continued growth.
20. You may have some persistent numbness over the donor nd newly grafted regions. This will gradually improve over several months as the cut nerve fibres regenerate.

Wigs

Many men and women with diffuse alopecia prefer wigs to scalp surgery. Wigs can either be interwoven with existing hair or worn over the top of existing hair. Interwoven wigs tend to lift as the hair beneath grows and require adjustment every few weeks. This may add considerably to the expense.

Wig hair is made from either a synthetic acrylic fibre that withstands wear and tear very well, or natural fibre (usually human hair). Natural fibre wigs look better but are more expensive and tend to wear out after 6 months to a year. Wigs can be styled and washed and modern wigs provide excellent coverage that looks natural. Among the better wigs are those made by taking a mould of the completely bald (or shaved) skull upon which a hairpiece is constructed by hand. Such wigs tend to remain fixed to the skull by suction and can be worn during all activities including water sports. The draw back of these wigs is that they get excessively hot in the summer. A good wig can save a patient multiple visits to the surgeons.

Unfortunately because the only wigs people see are the bad ones they have a poor reputation. Many people require coaxing to visit a wig maker. Excellent advice on wigs is usually available from patient support groups such as hairline international in the U.K. and the alopecia society in Australia. Clinicians are wise to familiarise themselves with the services local organisations offer.

Diagrams are located in the sections on common baldness for patients.

Download attachments: BMJrevPPC.pdf ; redandrogenicalopecia.PDF ; redbaldness in women.PDF