Rosacea: classification and treatment

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Rosacea is a chronic skin disorder affecting the facial convexities, characterized by frequent flushing, persistent erythema, and telangiectases. During episodes of inflammation additional features are swelling, papules and pustules. The disease was originally called acne rosacea, a misleading term that unfortunately persists[1].

Rosacea is a common disease, especially in fair-skinned people of Celtic and northern European heritage; it has been called the curse of the Celts. It is rare in American and African blacks[2]. Women are more often affected than men, but they seldom suffer the gross tissue and sebaceous gland hyperplasia of rhinophyma. Onset is usually between ages 30 and 50. In a recent epidemiological study the prevalence was 10%, most of the patients having only a red face[3]. In young patients especially, there may be a history of acne and the conditions may coexist.

PATHOGENESIS

The exact aetiology of rosacea is unknown and theories abound[4]. Gastrointestinal disturbances, notably dyspepsia with gastric hypochlorhydria, were long suspected of being a causal factor, but controlled investigations with a gastrocamera[5] and biopsy studies[6] revealed no association. *Helicobacter pylori* has come under suspicion, but results are conflicting[7]. For example, Powell *et al.*[8] found *H. pylori* antibody in 19 of 20 patients, while Schneider *et al.*[9] found no statistical difference in positivity between patients (49%) and controls (43%). Psychogenic factors have been frequently implicated but there is no good evidence that the condition is associated with personality type or is precipitated by emotional disturbance.

The theory of hypersensitivity to *Demodex folliculorum* or its products is based primarily on the distribution of rosacea and the mite, the follicular nature of rosacea, and the finding of the mite in areas of acute inflammation in some cases of rosacea[10]. However, *D. folliculorum* mites are normal inhabitants of human follicles and sebaceous glands and application of 3% sulphur ointment, while resulting in clinical improvement of rosacea, did not affect the *Demodex* population[11]. At most, the mite induces papule or pustule formation in pre-existing rosacea. Rosacea was once regarded as a seborrhoeic disease. Seborrhoea is, however, not always present[12]. Unlike acne vulgaris, rosacea is not primarily a disease of sebaceous follicles.

The pathogenesis of rosacea thus remains obscure. What is certain, however, is that rosacea patients are constitutionally predisposed to blushing and flushing. The basic abnormality seems to be a microcirculatory disturbance of the function of the facial angular veins[13]. Statistical associations between rosacea-related flushing and migraine suggest a shared disorder of vascular regulation[14] but there is no direct evidence that rosacea is primarily a vascular disorder. The response of the facial vessels to adrenaline, histamine and acetylcholine is normal,[15] and the vessels do not seem abnormally fragile[16] so the main abnormality is probably in the dermis surrounding blood vessels rather than in vessel walls. In addition, the distribution of rosacea is not identical with the flush area. A very important background feature is sun damage. Rosacea is always associated with solar elastosis and often with heliodermatosis[17]. Fair-skinned patients with rosacea type I will often give a history of sun sensitivity.

CLINICAL FINDINGS

Rosacea is usually symmetrically distributed over the face and is particularly obvious over the nose, cheeks, chin, forehead, and glabella. Occasionally, lesions are seen at extrafacial sites including the retroauricular areas, the V-shaped area of the chest, the neck, the back, and the scalp and extremities[18]. The hallmarks of rosacea are papules and papulopustules, vivid-red erythema, and telangiectases and a history of flushing. Comedones are notably absent. In severe cases papules are numerous enough to be confluent. Granulomatous changes can develop in later stages, sometimes receiving special designations such as lupoid rosacea. Rhinophyma and other phymas are the ultimate tissue reactions. For didactic but also for therapeutic reasons rosacea is classified into stages and grades. The progression is not inevitable, and few patients experience the full course of the disease.

Episodic erythema

Most rosacea patients react with transient erythema on the central areas of the face, less often the neck and the V-shaped area of the chest. These individuals are constitutionally

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predisposed to blushing and flushing (rosacea diathesis). The reactions are more frequent and more easily induced than ordinary blushing. Numerous non-specific stimuli such as ultraviolet radiation, heat, cold, chemical irritation, strong emotions, alcoholic beverages, hot drinks, and spicy food can trigger these flares. It is a mistaken belief that tea and coffee are precipitants; the specific stimulus is heat[19]. Among the mediators proposed to be involved in the erythematous response are substance P, histamine, serotonin, and prostaglandins but the trigger remains unknown.

Stage I

The erythema persists for hours and days, hence the old description erythema congestivum. Erythema lasting only a few minutes is not early rosacea. Telangiectases become progressively more prominent, forming sprays on the nose, nasolabial folds, cheeks, and glabella. Most patients complain of sensitive skin that stings and burns after application of cosmetics, fragrances, and certain sunscreens. Trauma from abrasives and peeling agents readily induces long-lasting erythema. Thus the facial skin is unusually vulnerable to chemical and physical stimuli.

Stage II

Inflammatory papules and pustules develop and persist for weeks. Some papules show a small pustule at the top. The lesions are always follicular in origin—mainly sebaceous follicles but also the smaller and more numerous hair follicles. The deeper inflammatory lesions may heal with scarring, but scars are small and tend to be shallow. Facial pores become larger and more prominent. If there has been heavy solar exposure for decades, the stigmata of photodamage become superimposed—namely elastosis, solar comedones, and heliodermatosis. The papulopustular attacks become more frequent. Rosacea may extend over the entire face and even spread to the scalp, especially if the patient is balding. Itchy follicular pustules of the scalp are typical. Bacteriological studies of these pustules reveal nothing of interest. Finally the sides of the neck and the retroauricular and presternal area may be affected. Even the palms may show persistent erythema.

Stage III

A small proportion of patients progress to the worst expressions of the disease—namely, large inflammatory nodules, furunculoid infiltrations, and tissue hyperplasia. These derangements occur particularly on the cheeks and nose, less often on the chin, forehead, or ears. The facial contours become coarse, thickened, and irregular. Finally the patient shows inflamed and thickened oedematous skin with large pores, resembling the surface of an orange (*peau d'orange*). These coarse features are due to inflammatory infiltration, connective tissue hypertrophy with masses of collagen deposition, diffuse sebaceous gland hyperplasia, and overgrowth of individual sebaceous glands forming dozens of yellowish umbilicated papules on cheeks, forehead, temples and nose. Thickened folds and ridges create a grotesque appearance resembling the leonine facies of leprosy. The ultimate deformities are the phymas, of which rhinophyma is the archetype.

ROSACEA VARIANTS

The diagnosis of rosacea in its classic forms presents no difficulty. The variants, however, may be overlooked or misdiagnosed.

Persistent oedema of rosacea

The published work hardly mentions this distressing variant. It has been reported as Morbihan disease or rosaceous lymphoedema[20,21]. A hard non-pitting swelling is found on the areas involved, mainly on the forehead, glabella, nose, or cheeks. A similar oedema sometimes arises in acne and in the Melkersson–Rosenthal syndrome; it develops against a background of chronic inflammation of any cause, including bacterial infection.

Ophthalmic rosacea

Eye involvement is surprisingly common. Indeed, the disease may begin in the eye and escape diagnosis for a long time. The ophthalmic signs include blepharitis, conjunctivitis, iritis, iridocyclitis, hypopyon-iritis, and even keratitis[22]. The term ophthalmic rosacea covers all these signs. The incidence is not known but more than half of patients participating in a cooperative isotretinoin trial for the treatment of rosacea were diagnosed by ophthalmologists as having inflammatory eye involvement[23], blepharitis and conjunctivitis being the most common. The ophthalmic complications are independent of the severity of the facial rosacea but there is a strong correlation between the degree of eye involvement and a tendency to flushing[24]. Rosacea keratitis has an unfavourable prognosis, and in extreme cases leads to corneal opacity and blindness. Perhaps the most frequent eye sign is chronically inflamed margins of the eyelids, with scales and crusts, quite similar to seborrhoeic dermatitis, with which it is often confused. Pain and photophobia may be present. All patients with progressive rosacea should be seen by an ophthalmologist.

Lupoid or granulomatous rosacea

Some patients develop epithelioid (lupoid) granulomas in a diffuse pattern[25]. Clinically, dozens of brown-red papules or little nodules on a diffuse erythema, frequently involving the lower eyelids, are seen. Histopathological examination

shows perifollicular and perivascular granulomas. The concept of a rosacea-like tuberculide of Lewandowsky was based only on the tuberculoid structure found histologically in many papules; however, the condition is probably unrelated to tuberculosis. Its course is chronic and unremittent. Differential diagnosis includes lupoid perioral dermatitis, lupoid steroid rosacea, and micronodular sarcoidosis.

Steroid rosacea

When a rosacea patient is erroneously treated with topical steroids the disorder may at first respond, but the improvement will be followed by steroid atrophy with thinning of the skin and an increase in telangiectases[26]. The complexion becomes dark-red with a copper-like tone. Soon the surface becomes studded with follicular, round, deep papulopustules and firm nodules. The appearance is shocking, with a flaming red, scaling, papule-covered face. The distribution can extend over the entire area of application of the topical steroid, often up to the hairline. Steroid rosacea is a pitiable, avoidable condition which in addition to disfigurement is accompanied by severe discomfort and pain. Withdrawal of the steroid is followed by exacerbation of the disease.

Gram-negative rosacea

This is a newcomer among Gram-negative infections[27]. Clinically it looks like stage II or III disease. Multiple tiny yellow pustules (type I) or deep-seated nodules (type II) increase suspicion. Neither oral antibiotics nor metronidazole will control it. The diagnosis rests on demonstration of Gram-negative organisms by culturing the contents of several pustules. The disease is analogous to Gram-negative folliculitis which sometimes develops on top of acne vulgaris[28]. The organisms are the same: *Klebsiella, Proteus, Escherichia coli, Pseudomonas, Acinetobacter*, and others.

Rosacea conglobata

Rarely a patient with severe rosacea shows a reaction that mimics acne conglobata, with haemorrhagic nodular abscesses and indurated plaques. The course is progressive and chronic. This variant mainly occurs in women. It may be provoked by oral ingestion of halogen-containing preparations. Diagnostic features are pre-existing rosacea and limitation to the face, with no other signs of acne conglobata on back, chest, shoulders, or extremities.

Rosacea fulminans

This variant was first described by O'Leary and Kierland[29] under the designation pyoderma faciale. It has been a matter of controversy ever since. One can say with certainty that it is not a variant of acne; neither is it pyoderma. The name rosacea fulminans was coined by analogy with its acne counterpart, acne fulminans[30,31]. This is a conglobate, nodular disease springing up abruptly on the face of young females. Curiously, it does not occur in males. Rosacea fulminans is confined to the face. Once seen it is never forgotten. Monstrous coalescent nodules and confluent draining sinuses occupy most of the face. The main locations are the chin, cheeks, and forehead. Ripe abscesses form with multiple pustules riding on top of the carbunculoid nodules. The face is diffusely reddened. Seborrhoea is a constant feature but may be overlooked. When questioned closely, patients will often describe the development of oiliness before the onset. Previous acne or rosacea is usually denied; however, we perceived a connection to rosacea because, after the stormy blow-up, signs of rosacea often make their appearance. Some patients, too, have been flushers and blushers. Aetiology remains obscure. Often blamed is severe emotional stress, such as the death of a family member, divorce, or loss of a lover, but some patients are stress-free. The prognosis is excellent. Once the disease has been brought under control it does not recur. Differential diagnosis includes acne conglobata (young patients, mostly males, longer history, other signs of acne, comedones, scars, seborrhoea, no flushing or blushing), acne fulminans (usually seen in teenage boys), bromoderma, iododerma, and virilizing tumours.

PHYMAS IN ROSACEA

Phyma is the Greek word for swelling, mass, or bulb. Phymas occur in various areas of the face and ears, rhinophyma (rhinos=nose) being the commonest. It occurs exclusively in men and fortunately it is a rare complication. Rhinophyma may be perceived by the public as due to excessive alcohol consumption, as in the comedian W C Fields. The bulbous nose develops over many years as a result of progressive increase in connective tissue, sebaceous gland hyperplasia, ectatic veins, and chronic deep inflammation. Rhinophyma may accompany stage III rosacea but in some patients the signs of rosacea in the rest of the face are surprisingly mild. Four variants of rhinophyma can be recognized. In the glandular form, the nose is enlarged mainly because of enormous lobular sebaceous gland hyperplasia. The surface is pitted, with deeply indented and mildly distorted follicular orifices. The tumorous expansions of the nose are often asymmetrical and of varying size. Humps and sulci occur. Sebum excretion is increased. Compression by the fingers yields a white pasty substance consisting of an amalgam of corneocytes, sebum, bacteria, and sometimes *Demodex* mites. In the fibrous form, diffuse hyperplasia of the connective tissue dominates the picture[32]. The amount of sebaceous hyperplasia is variable. In the fibroangiomatous

form, the nose is copper-red to dark red, greatly enlarged, oedematous, and covered by a network of large ectatic veins. Pustules are frequently present. The actinic form is characterized by nodular masses of elastic tissue distorting the nose. These are similar to the elastomas that occur in older individuals with over-exposure to sunlight. This variety is mainly observed in people of Celtic origin.

Phymas occur in other locations such as chin, forehead, eyelids and ears[33,34].

HISTOPATHOLOGY

Histopathology of rosacea varies with the stage and type of disease[35]. The changes often resemble those of other chronic disorders. Skin damage by sunlight is a common background feature, so severe elastosis is often present. In stage I rosacea there are ectatic venules and lymphatics, slight oedema, and sparse lymphatic perivascular infiltration. Moderate hyperplasia of the elastic tissue is present with increased curled, thickened elastic fibres (elastolysis). In stage II there is increasing lymphohistiocytic perivascular and perifollicular infiltration. Intrafollicular collections of neutrophils are often found, and are always present when pustules are observed. The infiltrate likewise surrounds sebaceous ducts and glands. The veins are thickened and grossly dilated. Elastosis is more advanced. In stage III there is diffuse expansion of the connective tissue, accompanied by hyperplasia of sebaceous follicles with long, distorted follicular canals, and large, irregular sebaceous acini. Epithelialized tunnels undermine the hyperplastic tissue and are filled with inflammatory debris. The elastotic changes are prominent, often evident as amorphous masses of degenerated elastic tissue. *D. folliculorum* mites are often found within the follicular infundibula and sebaceous ducts. They are merely commensals. This is different from true *Demodex* folliculitis (demodicosis). Epithelioid granulomas of the non-caseating type with multiple foreign-body multinucleated cells are the histopathological equivalent of lupoid rosacea. Abscesses with pseudoepitheliomatous hyperplasia, widespread necrosis, and lakes of granulocytes are characteristic of rosacea fulminans.

DIFFERENTIAL DIAGNOSIS

Acne vulgaris is distinguishable from rosacea by its lack of vascular component. Telangiectases and erythema are usually absent. Comedones and cysts are not seen in rosacea. Acne lesions may resolve into small fibrotic nodules and scars (which are absent in rosacea). *Perioral dermatitis* is a symmetrical eruption consisting of tiny vesicles and micropapules on an erythematous background. These lesions may appear in crops, sometimes with scaling. Occasionally, perioral dermatitis is accompanied by a similar eruption in the glabellar, malar, and periorbital regions. Rebound after discontinuation of potent topical corticosteroids seems to play a role in many cases. *Seborrhoeic dermatitis*, which may coexist with rosacea, is polymorphous, as are the other eczemas. Thus scaling and even mild vesiculation may occur. In contrast to rosacea, seborrhoeic dermatitis involves the paranasal area, the nasolabial grooves, the retroauricular region, and several areas beyond the face. *Photodermatitis* can be toxic or allergic in nature. When allergic, the polymorphic feature of eczema occurs and the eruption is usually present in extrafacial photo-distribution areas. When toxic, the eruption resembles a sunburn. *Polymorphous light eruption* is a photosensitive eruption usually appearing in spring or early summer, consisting of erythematous papules or eczematous plaques. Unlike in rosacea, itching is common after intensive sun exposure and before the eruption. The diagnosis is confirmed by phototesting. *Chronic discoid lupus erythematosus* has pigmentary changes, atrophy, and scarring which rule out rosacea. When lupus erythematosus is systemic and there is a symmetrical erythema especially intense over both malar areas, the distinction may be difficult. This difficulty is compounded by a positive lupus band test in the facial skin of some patients with rosacea[36]. A thorough evaluation for other evidence of systemic lupus erythematosus is indicated in these cases. *Haber's syndrome* is a rare familial rosacea-like genodermatosis with persistent facial erythema, telangiectases, follicular and verrucous papules, and atrophic pitted scars[37]. The patients often have dry facial skin and xerosis of their body. Finally, in patients with the sudden onset of severe flushing, *systemic flushing disorders*, such as carcinoid syndrome, must be considered. The 24-h urinary excretion of 5-hydroxyindoleacetic acid is normal in rosacea[38].

TREATMENT

Rosacea is treatable but seldom curable. Treatment schedules are determined by the stage and severity of the disease.

Topical

All sources of local irritation, such as soaps, alcoholic cleansers, tinctures and astringents, abrasives and peeling agents must be avoided. Only very mild soaps or properly diluted detergents are advised. Protection against sunlight is important: sunscreens with a protecting factor (SPF) of 15 or higher are always recommended, preferably of the broad spectrum UV-A plus UV-B type. For some it may be hard to find a sunscreen which is tolerated without burning or irritation. Female patients can be encouraged to hide telangiectases with cosmetic bases having a green tint.

The antibiotics used in acne are sometimes effective. Tetracyclines, clindamycin, and erythromycin, usually in

concentrations from 0.5% or 2.0%, are commercially available. In one study, twice daily application of 1% clindamycin phosphate lotion in an aqueous base was equivalent in efficacy to 250 mg oral tetracycline twice a day, without important side-effects over a 12-week trial[39]. The mechanism of antibiotics may be anti-inflammatory rather than antibacterial. Tetracyclines and erythromycin reduce leucocyte migration and phagocytosis[40].

Metronidazole is a synthetic nitroimidazole-derivative antibacterial and antiprotozoal agent, available in the USA and the UK as a 0.75% aqueous gel (MetroGel). In clinical studies metronidazole 0.75% topical gel or 1.0% cream improved inflammatory lesions in 68–96% of patients[41]. The gel is applied twice daily and is most effective in papular and pustular rosacea. It does not alter erythema, telangiectases, or flushing. When the drug is withdrawn, symptoms commonly recur. Where a registered preparation is not available we suggest the following prescription: metronidazole 2.0; Eucerin lotion ad 100.0. This 2% metronidazole lotion is applied once or twice daily as long as necessary. The principal adverse effects of topically applied metronidazole are local reactions such as burning and stinging.

Imidazoles are increasingly used for treatment of rosacea[42]. In our experience, not substantiated by clinical trials, ketoconazole cream (Nizoral cream) is the best choice, applied once or twice daily. The mechanism may be anti-inflammatory or immunosuppressive rather than bactericidal. Imidazoles seem to have low toxicity and are well tolerated by patients with sensitive skin.

Old-time remedies such as drying lotions should not be forgotten[43]. A very thin application at night is recommended. One can use commercial precipitated sulphur lotions. We prescribe: hydragyrum sulphur rubr 0.5; sulphur praecipitati 2.0; Lotio zinci ad 100.0.

Topical retinoids provide another option. The acneiform component responds to tretinoin[44]. Isotretinoin is worth a trial. There is preliminary evidence that 0.2% isotretinoin in a bland cream is helpful[45]; it is less irritating than tretinoin and suppresses inflammatory lesions in stage II and III.

In a double-blind randomized half-face comparison between 20% azelaic acid cream preparation and its identical-appearing vehicle as placebo, azelaic acid was effective against papules, pustules, and erythema in rosacea[46]. The only adverse effect over 9 weeks was minor skin irritation. Probably the anti-inflammatory activity of azelaic acid accounts for a substantial proportion of the therapeutic effect.

As stated above, we do not think that *Demodex folliculorum* mites have a causal role in rosacea. However, massive infestations may aggravate the condition. A check for mites is best done with the skin-surface biopsy technique (place a drop of cyanoacrylate on a glass slide, cover with immersion oil, and examine with the 10 × or 20 × objective in the light microscope)[47]. The mites are satisfactorily controlled with lindane (γ-hexachlorocyclohexane), crotamiton, or benzyl benzoate once daily for two to five days. Topical corticosteroids should not be used, except in rosacea fulminans[30,31]. In these patients short courses of high-potency topical corticosteroids are a reasonable option to reduce the inflammation.

Systemic

Rosacea generally responds well to oral antibiotics. Erythromycin is often effective, but tetracyclines are preferable[48]. Tetracycline-HCl, oxytetracycline, doxycycline, and minocycline are usually effective in controlling papulopustular rosacea and even reducing erythema. One should start with large doses—1.0–1.5 g tetracycline-HCl or oxytetracycline per day. 50 mg of minocycline or doxycycline twice daily can be given. As soon as papulopustules are fully controlled (usually after two to three weeks) doses of 250–500 mg tetracycline-HCl or oxytetracycline, or 50 mg minocycline or doxycycline, per day are generally sufficient. Rosacea patients often titrate doses according to disease activity and should be encouraged to do so. Some get by with 250 mg tetracycline-HCl on alternate days. Oral tetracyclines are effective in ophthalmic rosacea[49]. Antibiotic use in rosacea is often not sufficiently monitored. The disease has its ups and downs. Too often patients are on a fixed oral dose for many years when topical drugs might be sufficient.

Isotretinoin (13-*cis*-retinoic acid) is exceptionally effective though far more risky than tetracyclines[23,50–52]. It may be appropriate for all forms of severe or therapy-resistant rosacea, especially the variants unresponsive to antibiotics, e.g. lupoid rosacea, stage III rosacea, Gram-negative rosacea, rosacea conglobata, and rosacea fulminans. It is particularly helpful in patients who have oily, wide-pored skin and multiple sebaceous gland hyperplasias. In addition, all forms of phyma are indications. The dose required for control of rosacea varies. Three treatment schedules will be outlined here. The dose of isotretinoin is 0.5–1.0 mg/kg per day as used in acne. Side-effects on the eyes make this dose intolerable for many patients. Ophthalmic rosacea may get worse and likewise dry eye and blepharitis. Patients may become unable to use contact lenses. The high dose is used only in rosacea fulminans, or preoperatively for a couple of months to shrink rhinophyma before surgical reduction. Low-dose isotretinoin is much better and safer. 0.1–0.2 mg/kg per day is usually effective in severe rosacea, though clearing may take longer.

Minidose isotretinoin is 2.5 mg or 5.0 mg daily (not adjusted to bodyweight). This dose is surprisingly helpful in

many forms of the disease, especially stage III rosacea, lupoid rosacea, and persistent oedema in rosacea. Side-effects on the eyes are negligible. Duration of therapy is longer than with the other doses, about 6 months. The cumulative dose, however, is low. The usual precautions apply. Isotretinoin is a teratogen and is contraindicated in women of childbearing age unless they meet all the requirements on the package label. Aminotransferases, cholesterol, and triglycerides must be measured before therapy and monthly or bimonthly thereafter. With minidose isotretinoin no laboratory abnormalities have been observed.

In an uncontrolled study of 13 Japanese male rosacea patients[53] good results were reported with spironolactone. 50 mg daily over four weeks seemed to improve itching and erythema, perhaps via inhibition of epidermal cytochrome P-450. Further studies are necessary to confirm this. Clonidine, in a limited trial, reduced facial flushing[54], but doses which do not decrease blood pressure have little or no effect[55]. Metronidazole is licensed for the treatment of infections caused by *Trichomonas vaginalis, Entamoeba histolytica*, and *Giardia intestinalis*. The usual dose is 500 mg twice daily for six days. Oral metronidazole is effective in all types of rosacea, including stages II and III[56]. However, 20 to 60 days may be needed to achieve control with a daily dose of 500 mg. It should be regarded as a second-line drug.

Rosacea fulminans requires special care[30,31]. Treatment starts with oral corticosteroids (e.g., prednisolone 1.0 mg/kg per day), for one week to cool down the fire. Then isotretinoin is added, at around 0.2–0.5 mg/kg, rarely 1.0 mg/kg, per day, with a slow tapering of the corticosteroid over the next two to three weeks. Isotretinoin is continued until all inflammatory lesions have disappeared. This may require three to four months. Draining abscesses should not be incised. Warm compresses can be applied, together with a potent corticosteroid cream (for the first 2 weeks only).

Miscellaneous

Facial massage has long been recommended. This is the so-called Sobye's massage[57]. Controlled studies are lacking. Twice daily gentle circular massage is given for several minutes to nose, cheeks, and forehead.

There is no specific rosacea diet. Dietary limitations relate only to factors which provoke erythema, flushing and blushing such as alcoholic beverages, hot drinks, and spicy food. The patients themselves may find out which dietary items are troublesome.

Obliteration of ectatic vessels, particularly on the nose, can be achieved by intravascualr insertion of a fine diathermy needle or by argon or pulsed dye lasers[58]. In expert hands these work well.

Rhinophyma needs surgical intervention[59]. Excellent cosmetic results can be obtained by various techniques including scalpel or razor modelling, electrocoagulation, elevation of the intact epidermis with debridement of excess tissue below it, and lasers. Much depends on the training and preferences of the physician. Isotretinoin may be used successfully before the operation to shrink the bulbous portions, and postoperatively also[60].

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