

Rhinophyma Cosmetic Disfigurement Nasal Anatomy

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Abstract

Different aspects of rhinophyma were described: history, etiopathogeny, histopathology, epidemiology, clinical, differential diagnoses, treatments and prognosis. It is emphasized that it is a localized disease in the nose associated to general disorders, and that it brings psychological and social problems. We present a case operated with plastic surgery. The authors present a hypothesis about the etiopathogeny and pathophysiology of the disease and consider rhinophyma treatments as palliative and non-curative.

Keywords: Rhinophyma; Phymatous Rosacea; Plastic Surgery; Nasal Anatomy; History of Medicine

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Introduction

The rhinophyma is a progressive and deforming enlarged of the tip and the wings of the nose. There is a thickening of the skin, usually rubicunda, with multiple nodules determined by hypertrophy of the sebaceous glands. It is generally presented in patients who have severe rosacea, hence it is also called “Fimatososa Rosacea”. However, it can also occur in isolation or independent of it.

In severe cases, nasal anatomy disfigures and hinders breathing due to the occlusion of the nose. Usually, psychological and social problems are added.

Its progression to malignant tumors, such as basal cell carcinoma and, in rare cases, to the carcinoma of squamous cells, has been described.

There is no specific treatment; And in the most serious cases, plastic surgery is necessary.

In this work the authors make some personal considerations about different aspects of the rhinophym and an operated clinical case is presented.

The term rhinophym comes from the Greek and means grown nose. Rhis: Nose and Phyma: Growth. The nose in facial aesthetics has had an important role throughout the history of humanity [1-4]. It is interesting to note that this disease has caught attention, for several centuries, both writers and painters.

William Shakespeare refers to her in her play “Enrique V” (1599).

As for the latter, the oils found in the Louvre Museum, Paris: “Portrait of an old man with his grandson” (1490) of the Italian Domenico Ghirlandaio; and that of the Prado Museum, Madrid: “Christ

before Pilatos” (1500) of the Spaniards Rodrigo and Francisco de Osona (Father and Son), who show this disease in their protagonists (Figure 1 and Figure 2).

It is well known that famous personalities such as the painter Rembrandt, the banker J.P. Morgan and actor W.C. Fields suffered from Fimatososa Rosacea.

The first Rhinophyte surgery was performed in 1629 by the German



Figure 1: “Retrato de anciano con niño” (1490), de Domenico Ghirlandaio, Louvre Museum, Paris.



Figure 2: "Cristo ante Pilatos" (1500) Rodrigo de Osonay Francisco de Osona. Museo del Prado, Madrid.

Daniel Sennert; And only in 1845, the Austrian Ferdinand von Hebra, described - for the first time - his clinical picture [5].

Morfo-physiological Review

To understand the pathophysiology of this disease it is convenient to review some aspects of nasal anatomy and its development, as well as skin histo-physiology; since it is the skin of the nose where the rhinophyma is going to manifest.

Anatomical Review

The nose is formed by osteo-cartilaginous structures, muscles, connective tissue and skin.

The structure of the nasal pyramid, in its upper third, is bone; and in its middle and lower thirds, cartilaginous. The connective, fibrous type tissue, fills the intercartilaginous and bone spaces. The muscular plane, more superficial, is made up of five delicate small skin, plans and caught muscles: one of them, in the upper third of the nasal pyramid; and the other four, in the lower two thirds and in the neighboring regions. The latter act as compressors (constrictors) and dilators of the nostrils. The skin and subcutaneous tissue have all these structures and provide insert to each of these muscles by one of its ends.

The anterior and medium edge of the nasal pyramid is called the nasal back. Down ends in a rounded eminence called Apex or nasal tip. On its sides, the two wings of the nose.

The nasal pyramid by its side edges forms a groove that changes its name according to the regions it crosses: Naso Palpebral, Naso Geniano and Naso Labial. The root of the nasal pyramid corresponds to the frontal front angle and the interiliac region. By its base, this nasal pyramid presents the nostrils (nasal holes) separated from each other by the membranous portion of the nasal septum (subtabique).

The anatomy of the tip and the wings of the nose is different from the rest of the nasal pyramid: the skin, the cartilage (with its pericondrium) and the muscles give it that particularity. In addition, it has a rich vascular plexus that explains the great vitality of these tissues, the reddish color (erythema) in some clinical situations, and the profuse bleeding in surgical interventions.

The skin has abundant sebaceous glands, which determine an increase in its thickness and gives it a unique characteristic in the organism. There is no other region of the skin that has this high gland density. This can be seen with the naked eye and is usually represented

by the comedones (or "black points") that are simply the sebaceous material in the outer hole of the gland, which act as a hyperkeratotic cap and favor the infection of the gland. When pressure is exerted on them, the sebaceous matter comes out and thus the hole is disoblotted. The face is the body region that produces the most sebaceous.

The conformation of the tip and the nostrils depend on these three structures cited (skin, muscles and cartilage) and confer multiple morphological varieties according to individuals.

While the skin of the nasal back is delicate, fine and easily sliding of the deep plane and easy dissection; The one of the tip and nasal wings is resistant, thick, intimately adhered to the deep and very irrigated planes, being difficult to dissect [6-8].

Embryological Review

The embryological origin of the skin is double: the epidermis comes from the ectoderm, while the dermis and the hypodermis (subcutaneous tissue), of the mesoderm.

In the 4th month of intrauterine life (already fetal period) the four strata are differentiated in the epidermis: basal or germinative, spiny, granular and cornea. The keratinocyte is the primordial cell of the epidermis and progresses from the deepest part to the most superficial part (vertical migration), performing a cell differentiation process. At this stage of development, the epidermis was already invaded by dendritic -looking cells that come from the neural crest and that synthesize melanin. This pigment through dendritic processes (or extensions) is transferred to epidermal keratinocytes. Those cells will then be called melanocytes and from birth they will be responsible for skin pigmentation. Each melanin supplies many keratinocytes. Also, the so -called "skin annexes" derive from ectoderm: skin glands (sweat, sebaceous), hair and nails.

In the dermis, also to the 4th month of intrauterine life, the fibroblasts originate the collagen and elastic fibers. Macrophages and mast cells are also identified. Simultaneously, the dermis originates the papillary stratum (richer in cells than fibers) and the reticular (richer in fibers than cells), which include capillaries and sensitive and motor nerve endings.

In the hypodermis an abundant adipose panicle and lax connective tissue are developed.

It is in the dermis where the histopathological changes of rhinophyma are noticed, involving the mentioned microscopic structures [9,10].

Histological Review

The epidermis is constituted by a stratified squamous, avascular and aneural epithelium such as any epithelium.

In addition to keratinocytes and melanocytes, dendrocytes (or Langerhans cells) and tactile epitheliocytes (or Merkel cells) are found in the epidermis.

The keratinocytes represent more than 90% of the epidermis cells, are linked together by the desmosomes and maintain the cyto-architecture of the epidermis. The cornea (keratin) layer and melanin protect the organism from ultraviolet rays (solar radiation). The dendrocytes originate in the bone marrow and are in charge of skin immuno-vigilance, act as a cutaneous immune sentry. The tactile epitheliocytes derive from the neural crest, are neuro-endocrine cells of the skin that probably act as mecano-receptors, since they are associated with intra-epithelial sensitive endings and often make



contact with dermal sensory axons. Simplifying, we can say that, in the epidermis the nerve endings of pain, temperature and pruritus are also observed; while in the dermis those of pressure and touch are located.

The epidermis through the dermo-epidermal union is related to the dermis. This limit is very wavy in order to increase the contact surface between the two and ensure its functionality.

The dermis, like the epidermis, has no adipose tissue, but differs from it in which it is very vascularized. For its part, the dermis is constituted by a network of collagen and elastic fibers (Figure 3).

The Sebaceous pilot unit includes the hair follicle, the erector muscle of the hair and the sebaceous gland. The Pilo-Rector smooth muscle is deeply inserted into the end of the hair (or hair bulb), located in the reticular dermis; and superficially in the dermo-epidermal union. It is innervated by sympathetic fibers and is in charge of putting “chicken skin” and probably compressing and emptying the sebaceous gland. The Sebaceous gland flows through a short excretory pipeline in the hair structure. By the mechanism that produces its secretion (sebum) it is known as Holocrine, since the entire cell dies in a differentiation process and becomes a product of secretion. Its stimulation and development are mainly in charge of androgens (Figure 4, Figure 5).

The sebaceous follicle is another glandular structure of the skin, which accompanies a rudimentary hair known as “hair.” Its sebaceous secretion (sebum) is under hormonal control. They are found in the face and in the sternal area. They are acne seat (Figure 6).

There are also sweat glands, located in the deep dermis or hypodermis, which are classified into merocrins and apocrines. The merocrins are distributed in greater proportion in the skin, while the apocrine are only located in the axillary, perineal and mamillary (or nipples) regions. The sweat duct of the latter flows into a hair follicle. The sweat that produces suffers a process of bacterial decomposition, so its unpleasant smell. The sudoriparous glands of merocrin type, are independent of the hairproof, and participate in thermoregulation [7,11, and 12].

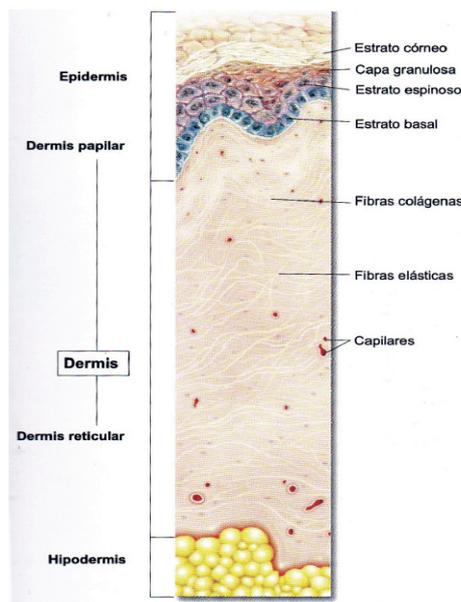


Figure 3: Skin histology. Epidermis (flat epithelium) stratified avascular), dermis and hypodermis (11).

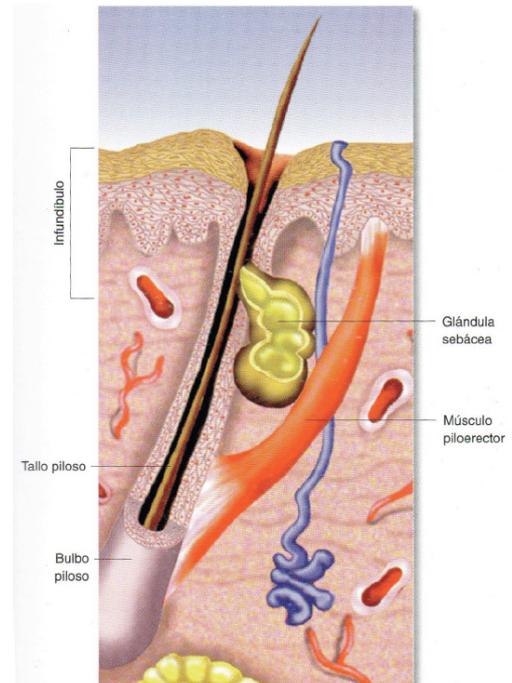


Figure 4: Pilosebaceous unit. Hair follicle, muscle arrector pili and sebaceous gland (11).

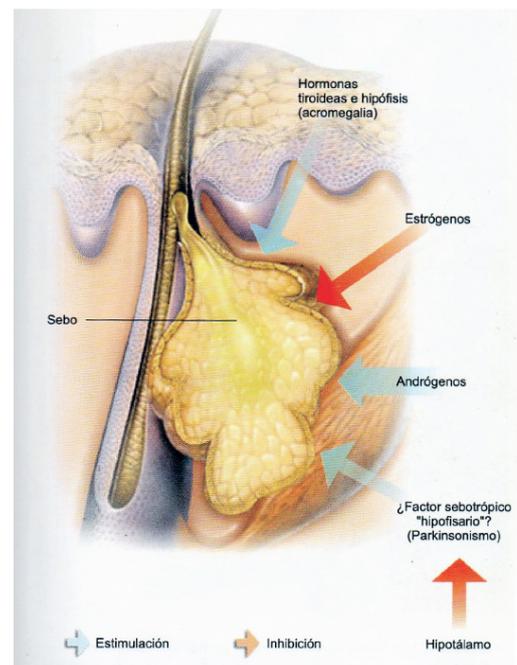


Figure 5: At higher magnification: sebaceous gland opening in the hair infundibulum (11).

Physiological Review

The skin establishes a boundary between the environment and the interior of the body, so it is called “organera organ.” It is the first barrier, of a mechanical type, which the organism has before the external aggression and is the place of exchange between the two media, that of particular metabolic processes (for example, synthesis of vitamin D) and the expression of visceral manifestations. Collaborates in the photo-protection and thermal-regulation. It is

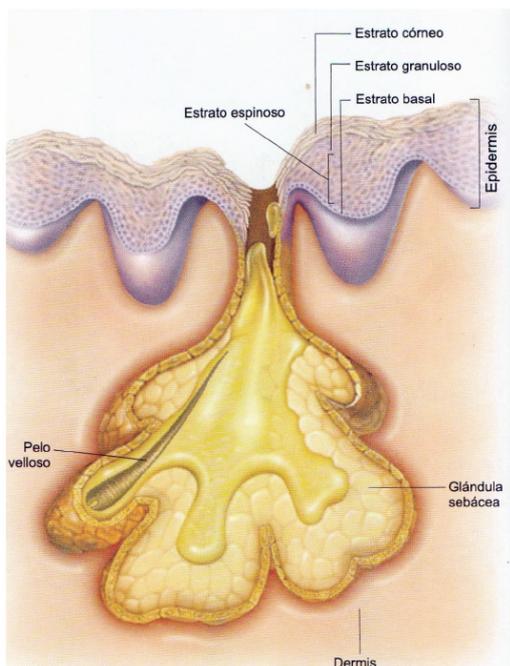


Figure 6: Sebaceous follicle: sebaceous gland accompanied rudimentary hair (vellus) (11).

the first immune barrier that the organism has. Recall that allergens also act on the skin and they trigger reactions of skin hypersensitivity. Finally, in it a nerve activity develops through sensitive and motor endings. Through the former, it is responsible for the tactile, thermal, algic or painful sensitivities, of pressure and pruritus; Thus, the skin performs the external functions that the organism needs. Through the second, belonging to the sympathetic (adrenergic and cholinergic) fibers that innervate the hair follicle, the sweat and sebaceous glands. A dysfunction of this autonomic nervous system modifies the normal physiological mechanism of these three structures. In the rhinophyma some of the functions set forth here can be altered [8,11-13].

Etiopathogenesis

Its origin is unknown and it may be multifactorial. Different hypotheses are postulated that include some influence of: vascular alterations (angioneurotic), degeneration of the dermal matrix, genetic predisposition, excessive steroid doses, androgenic hormonal influences, environmental factors (exposure to sunlight and heat), chronic alcoholism, Vitamin D Deficiency, Emotional situations (stress) and chronic infections for microorganisms such as *Demodex Folliculorum* (mite) and *Helicobacter pylori* (bacteria) [5,14-17].

Let us remember that in the skin a resident flora (bacteria, fungi and parasites) inhabits that it lives as a saprophyte but that can become pathogenic and that fulfills an important role in protection and cutaneous barrier (among them, exercising bacterial interference with external noxas).

In the hair follicles and in the Sebaceous glands inhabit the demodex, the *propionibacterium* (bacteria) and the *manerazia* (fungus), among others [13]. When the "follicular microenvironment" is altered, a conducive condition for excessive proliferation of these microorganisms is installed. As a result, a local inflammatory picture is generated with the release of chemical mediators (histamine, serotonin, prostaglandins, complement fragments, cytokines, etc.) and a hypersensitivity response against them is triggered. These phenomena

are noticed in the rhinophyma. This would be a part of the immune hypothesis [11].

In addition, molecular studies suggest that in patients with fimatous rosacea there is an alteration of the immune response in the pathogenesis of this "inflammatory disease" [18]. Some propose that there is a deregulation of the mediators and receptors involved in neurovascular and neuroimmune communication, so sometimes the drugs that act at that level are beneficial [19].

On the other hand, in patients with hemochromatosis, where excess iron that circulates in the blood can result in tissue damage in the skin (one of the organs where it is deposited), and that manifests clinically in a classical hyperpigmentation. In these cases, the iron deposit in the nasal region can contribute to the pathogenesis of the rhinophyma [20,21].

In summary: Three subtypes are distinguished in the rosacea and it is the third one called Fimatosa or Rhinophym Rosacea. It is likely that it is determined by a patient sensitivity to some of these triggers mentioned above. Thus the Rosacea progresses towards a rhinofima [22] (Figure 7).

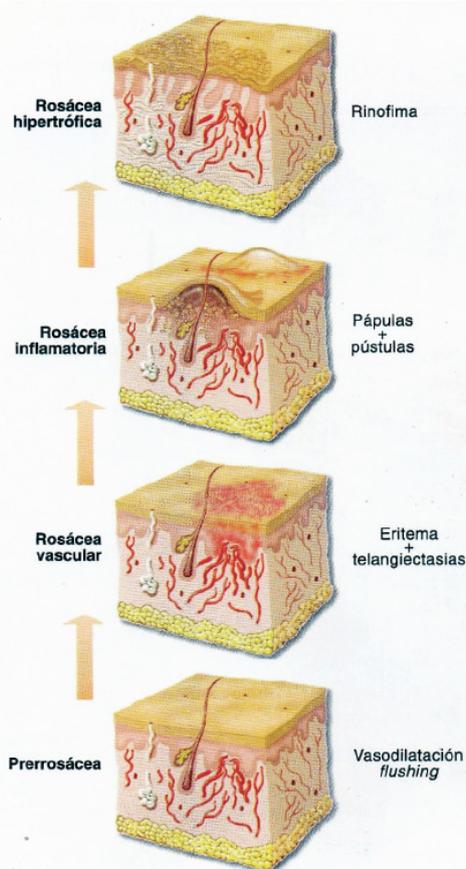


Figure 7: Rosacea: progressive clinical periods (evolutionary stages) (11).

Histopathology

In the rosacea, the histological study demonstrates the presence of a chronic inflammatory reaction of a productive type, mainly constituted of lymphocytes with some macrophages and plasmocytes, around hair follicles and sebaceous glands. Sometimes, epithelioid cells and giant cells identical to true tubers are noticed. In these cases, the differential histological diagnosis with skin tuberculosis is often confused. They



can be accompanied by interfollicular micro-Absites with pustules and capillary dilation (erythema and telangiectasias). Sometimes, there is hypertrophy of the sebaceous glands and the adjacent connective tissue with inflammation. In summary: the histological painting is not pathognomonic, except in the rhinophym, in which it is very suggestive [23].

In the rhinofima, the skin of the nose shows a nodular thickening, characterized by the hypertrophy and hyperplasia of the sebaceous glands. Dilated sebaceous ducts and hypertrophic acinos, contain epithelial deaths and thick sebaceous substance. Sometimes, the glandular epithelium can suffer a squamous metaplasia. The neighboring dermis can show signs of chronic inflammatory reaction and fibrosis. In general, the underlying cartilaginous structures are not involved in the process [24].

We must remember that every inflammatory process has regressive phenomena (death and cell damage), vascular and humoral; and proliferative reactions (which lead to repair or healing), the connective tissue being the battlefield of inflammation [25,26]. Then, this fabric will be the most committed in the rhinophine.

In summary: in rhinophyme it is observed hypertrophy and hyperplasia of the sebaceous glands (glandular component), fibrovascular proliferation of the dermis with signs of angiogenesis (fibrous and vascular components) and acanthosis of the epidermis (acantic component). Therefore, four histological variants are described in the rhinophym: glandular, fibrous, fibro-angiomatous and acantic.

Epidemiology

There are no world incidence and prevalence data. Rhinophym is unusual and usually appears in maturity, between 50 and 60 years of age, being more frequent in male sex and in the white race. In the female sex it appears earlier. Few cases have been described in black and yellow races.

In the bibliography it is highlighted that the white race is more frequent among Celtic ethnicity in Scotland. In general, it is presented in fatty complexions and in the first three of the six cutzpatrick skin phototypes. A hereditary or family influence has also been suggested [5,19,20,27, and 28]. Recall that the Celts came from Anatolia (Asia Minor) and then occupied the center of Europe. With their migrations they toured her almost completely and reached the British islands. The rosacea and the rhinophine have been considered “the curse of the Celts” [29-31].

Clinic

The rhinophine can occur in a pink or independent field [29,32]. It is a chronic inflammatory picture (which lasts for months or years), of slow and insidious evolution, with repeated infectious episodes.

Rosacea, of unknown etiology, of chronic evolution that alternates with acute outbreaks, usually occurs in cases of seborrhea. It affects the central portion of the face and has a slight similarity with acne. Classically, three stages or periods are described: first, erythema and telangiectasias (vascular rosacea) appear; then, papules and pustules of variable degree (inflammatory rosacea); Finally, thickening and skin hypertrophy (hypertrophic rosacea).

When the pathological state affects the nose and is serious, clinically it is called Fimatoso or rhinophima rosacea. It is always limited to the lower half of the nose [23]. In some cases, the fimatous rosacea can extend and concomitantly affect the forehead (methophym) and the

chin (gnatophym), due to its characteristic of central-family dermatosis [33].

Rhinophym can present variable clinical forms and a natural progression determined by different stages or periods. The painting extends from the slight cosmetic discomfort to the serious disease that disfigures nasal anatomy and makes breathing difficult, either by the weight of the hypertrophic mass that hangs or by the collapse of the nasal valves (expression at the level of the nasal mucosa of the upper edge of the major alar cartilage).

It is characterized clinically by nodular thickening of the nasal skin, hyperplastic sebaceous glands with dilated follicular holes, erythema and telangiectasias, papules and pustules. The abundant sebaceous secretion and the presence of bacteria and other microorganisms lead to chronic dermatitis with over-infection episodes. Tubers, nodules and/or vegetation are observed in the skin of the nose, of variable size, isolated or converged, smooth or ulcerated, red-vinous, with bad smell, which can infiltrate and compromise neighboring structures (cartilage and muscles and muscles nasal). These lesions have a gummy consistency to palpation [30,34].

Differential Diagnosis

The deformity caused by rhinophym can complicate the precise examination of nasal skin and certain pathologies within hypertrophied and inflamed skin can go unnoticed [35-38]. For example, the incidence of hidden basocellular carcinoma is 3-10% in patients with rhinophym [38-40].

Other lesions can coexist or imitate rhinophimics: lipoma, eosinophilic granuloma, facial granuloma of lung and breast, etc. Therefore, it is convenient to carry out the deferred histopathological study of the dry tissue.

In the differential diagnosis, some skin conditions must be taken into account, such as: vulgar acne; Seborrheic dermatitis, by contact and atopic; bacterial and icing folliculitis. They should not also forget some systemic diseases that study with erythema or facial telangiectasias such as systemic lupus erythematosus (Les), pheochromocytoma, mastocytosis and carcinoid syndrome, among others.

Treatments

For rhinophyme there is no effective treatment and a multidisciplinary approach is required [2]. The therapeutic options range from clinical treatments, with topical and oral administration of different drugs and antibiotics to reduce local inflammatory reaction, to plastic surgery [41-44]. In the most serious cases, with the latter, it is tried to recover the normal morphology of the nose.

Surgical Procedures

In the moderate and serious cases of rhinophyme, various methods have been used to remove hyperplastic tissue with excisional surgery and/or decortication, which is the election procedure, either with: cold, electro-bisturi or electro-cautery, cryocirgery and /or laser, etc. [44,45].

It is recommended not to ablod all the sebaceous glands to avoid healing defects. That is, to leave a minimal dermal layer so that from there the healing of second intention can occur through granulation tissue: “tissue that cleanses, fights and fill in the battlefield where inflammation was carried out” [25]. However, even so, healing disorders can be observed, which result in asymmetry and/or hypertrophy.

On the other hand, by leaving this thin layer of sebaceous glands,



the recurrence phenomenon observed can be explained when applying this technique. Therefore, this type of surgery requires a “direct visual control” and a certain manual ability to find that section plane. Hemorrhage is always important in this operation because the tissue is highly vascularized. Electrophulguration is better than electrocoagulation, since, although both produce good results in the control of hemostasis, the second is indicated that the thermal damage produced, in some cases uncontrollable, can generate a longer healing process and more prolong low quality; And in addition, a damage to the underlying nasal cartilage (which includes its perforation) [2,44].

In the manner of historical review of surgical procedures, let's point out that: Dieffenbach, in 1847, described the surgical treatment of the exeresis of the nasal hyperplastic tissue and the primary closure of the wound. In 1851, von Langenbeck made the exeresis of the total skin thickness, leaving the closure for second intention. In 1864, Stromeyer communicated a more conservative resection, the splitting of the partial skin thickness involved, allowing reepithelization through the Sebaceous glands by second intention. In 1876, Ollier called this “decortication.” In 1912, Wood introduced the skin graft for the closure of the post-casting defect of the rhinophym. In 1946, Douglas Macomber proposed the use of skin graft of the supraclavicular region, as a giver zone with better aesthetic results than those taken from other regions of the body [27].

In conclusion: Surgical decortication and secondary healing, through granulation tissue, is one of the most used treatments for patients with severe rhinophym, obtaining quite satisfactory results in the attempt of an aesthetic rhinomodation. In addition, there are evidence that shows that healing by second intention can bring superior results to those obtained with those of the primary closure or with the placement of a graft. The healing of the bloody bed is convenient to make them with wet priests instead of dry [14,26, and 45-47].

Mild rhinophine cases can also be treated surgically with the “shaving” technique (shaving or tangential split) with cold scalpel, dermatomo or radiofrequency scalpel (or radio-white). Others have used ultrasonic (or harmonic) scalpel to improve intraoperative hemostasis. Finally, also with a non -surgical procedure, which is dermabrasion [48,49].

Curnier and Choudhary first performed the tangential split with scalpel or dermatomo for volume reduction, then the use of sculpts to sculpt and finally the dermabrasion of the bloody surface to contour it [27,41, and 50]. Cornier also performed the tangential split with scalpel or dermatomo and on the bloody bed placed an alginate dressing obtaining good results in terms of hemostasis and reepithelization. According to his experience, the use of alginate was superior to gauze impregnated with soft paraffin, to oxidized cellulose dressings, to the porch collagen patches or membranes and the waterproof dressings. Healing time was lower, as well as the risk of hypertrophic scars [51].

Non-surgical Therapeutic Approaches

With another therapeutic, non -surgical approach: diet, herbs, vitamins and minerals have been tested, as well as vapors, and also fibrolisin and steroid injections, but all without success. The administration of antibiotics and retinoids, oral and/or topics, have not proven to stop the progression of rosacea to rhinophine or cause regression of the existing rhinophine [41,44]. In 1920, radiotherapy became fashionable, but because of its complications it fell into disuse [52].

With respect to two drugs we want to clarify the following.

Isotretinoin, a retinoid drug, administered orally and for 4-5 months, has shown some benefits in the early stages of the rhinophym and in young men, as well as in chronic stages decreasing the intensity of exacerbations. The combined therapy of isotretinoin and dermabrasion, in some cases reduced the volume of the rhinophym and sebaceous and follicular acinos; the hyperplasia of the sebaceous glands, papules and pustules; edema and inflammation; and telangiectasias [52].

The Tamoxifen, an anti-steroid drug, which acts on the beta transformative growth factor, it is believed that the fibrotic activity of the disease can decrease [44].

As for therapy using new technologies, such as laser: Shapshay, in 1980, pointed out the use of carbon dioxide laser, CO₂. He used the “continuous wave” for the destruction and elimination of hyperplastic tissue, while the “pulsed” (or pulses) to level and match the shape of the residual tissue at the expense at which secondary healing will occur. Henning and Van Gemert, in 1983, used Argon's laser, but as caused tissue necrosis, was abandoned. The Erbio laser has also been used: ititrio-aluminum-sharing (ER: YAG), with a thermal collateral effect on the tissues more controllable than the CO₂ laser. In all of them, the depth of penetration of the ray to the tissues must be calibrated, in order not to damage the cartilaginous-fibrous plane of the tip and wings of the nose. The main advantage of these lasers is that they handle an exangue bloating bed or field [44,45,54, and 55]. Har-el and others, after comparing laser versus surgical split they indicated the same postoperative and aesthetic results (56,57,58). Currently, a new generation of lasers tries to look for better therapeutic results.

Anatomo-patological Study

It is convenient that the resected material be sent to deferred histopathological study to rule out possible hidden evil processes.

The coexistence of leather carcinomas, both basocellular and squamous, in a rhinophine is a fact described in medical literature. It is not clear if it is coincidence or it is causality [36,38]. Supporting this last hypothesis, it is believed to be a degeneration of the tissues undergoing chronic inflammation.

Procedures such as laser, cryocirugia, dermabrasion and others that destroy the tissue prevent this anatomo-patological exam.

Aesthetic Result

In the Posoperatory, in general, there is a good acceptance of patients with the aesthetic results achieved, which is greater the more serious it is the case of Rhinopime, where the disease is more spectacular. However, for the plastic surgeon of our time - expended with outstanding results - the final aesthetic effect is usually regular or simply good; to which their transience must be added due to the possibility of recurrence of the disease.

Forecast

The rhinophine has a chronic and progressive natural course evolution. In many patients who receive treatment, clinical and/or surgical, a stabilization with variable residual symptoms and signs is achieved. In other cases, the treatment fails and the picture is recurring.

In short, rhinophym as a disease honors French aphorism: “Medicine sometimes cures, often relieves and always comforts.” As for plastic surgery in rhinophym, it has more “repairing” effects than “curative.”



Clinical Case

The case of a 60-year-old male is presented, with a history of Rosacea with Severe Rhinophyma, 15 years of evolution, with little response to topical and oral pharmacological treatments. Other diseases: diabetes and arterial hypertension. Belfalpebral and bilateral blepharitis history. He presents a clinical picture of generalized facial rosacea with erythema, telangiectasias, pruritus, papules, pustules and dilated pores. At the nasal level, thickening of the tip and wings of the nose is observed, with the presence of multiple nodules, which increase the volume and weight of the nose, without generating respiratory obstruction but that deforms its normal morphology. There is impossibility of correctly hygienizing the area, with bad smell due to sebaceous secretion and accumulated detritus.

Derived by the dermatologist with a diagnosis of severe rhinophyma for surgical treatment. Cold and electro-coagulator bisturí was used, with local and outpatient anesthesia. Surgery was performed in three times, taking into account the aesthetic units of the nose, so to know the patient's own scar response, starting with the region of the wings, right and left, and ending with the back and nasal tip.

An excisional resection of the multinodular hyperplastic tissue of both nasal wings and a partial decortication at the nasal tip (to the pilot-sebaceous units, that is, in depth, the resection of the two surface thirds of the dermis) was performed. This was done in three surgical times: first the most compromised nasal wing, then the other and finally the nasal tip. The hemostasis was performed with electro-flouration. It was allowed to heal for the second intention in the three surgical times: the surgical beds of the nostrils, right and left, delayed 15 days each; and that of the nasal tip, of greater extension, 25 days. There were no complications. The controls were newspaper and the discharge took a year. It should be borne in mind that the patient had pre-existing systemic diseases that altered the third phase of the inflammatory process (healing). The pathological anatomy reported: epidermoid cysts, sebaceous hyperplasia, tumor of granular cells and actinic keratosis. Malignity was not diagnosed. A good result was obtained, recovering the form and nasal contour (Figure 8, 9, 10, 11, 12 and Figure 13).



Figure 8: Clinical case. Pre and postoperative.



Figure 9: Preoperative. Third time - Nose tip.



Figures 10 and 11: Intraoperative of the third time - Nose tip and Removal of rhinophymatous tissue.



Figure 12 and Figure 13: Final postoperative - At six months.

Discussion

Etiological hypothesis of the authors

What phenomena and circumstances develop rhinophyma? What factors determine it? We have not found, in the database consulted, works that give reliable answers to these questions.

In our hypothesis, three forces would converge that would install a chain, chronic and evolutionary reaction.

On the one hand, we believe that there may be a general factor, such as an alteration in the general metabolism of the patient, with changes at the cellular and molecular level in distant organs, which would produce a series of activators or inductors that would act on the skin of the nose, which is the tissue that would react ("white organ"), altering its morphology, micro and macroscopically. There would be an "affinity" for nasal skin, more precisely its dermis (mesodermic component of the skin).

On the other, we believe that a local, irritative or traumatic factor can coexist with an infectious aggregate that generates chronic inflammation.

And finally, we think that a genetic factor can also be present.

In this way: biological, chemical and physical factors, whether distant or local, intervene and generate an alteration of normal nasal



skin. The epidermis and the dermis, both at the nasal level and anywhere in the body, maintain a balance with each other. In it, there are also regulatory components of its cell renewal. In the rhinophyma there would be a failure in that “dermal-epidermal” balance and an error in that regulation, generating the structural changes that characterize this disease.

For this reason, the treatments that arise in this work, which are those collected in the different texts and articles, are of partial or incomplete results since they treat only the tissue where this metabolic imbalance acts or reacts and not its cause. Therefore, we can say that they are “palliative” and “non-curative” treatments; Concept that we have not seen clearly indicated in the literature consulted. The clarification of its etiology and pathogenesis must be awaited to find effective treatment.

The total eradication of the sick tissue (chronically inflamed) is only possible when the tissue that is removed includes all the dermis and reaches the cartilaginous-withoneal plane. In that case, the regeneration by second intention from the bloody bed will not be possible and a skin graft must be placed in the nasal defect, preferably of total thickness, which provides a relative aesthetic result. Thus, the total removal of sick skin is inversely proportional to the postoperative aesthetic result.

Conclusion

Rhinophyma is a rare inflammatory pathology. Its ethiopathogenesis is unknown, but it is associated with different disorders and habits. It is of chronic and progressive evolution.

The initial lesion based on the dermal layer of the skin of the tip and the wings of the nose, affecting the sebaceous glands and the connective tissue. It alters nasal morphology and this aesthetic disfiguration causes psychological and social problems.

No specific treatment is available. Those that exist are palliative and non-healing, as is the case with plastic surgery. Basically, the objective of the treatment is to eliminate a significant amount of hypertrophic nasal skin and leave a minimum dermal layer, by the skin annexes that are found in it, from which the wound can be re-epithelialized and achieve an improvement in the nasal contour. However, that same remaining fabric is also responsible for its recurrence.

The deferred histopathological study of the resected tissue is convenient to rule out other pathologies such as skin carcinomas.

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Declarations

The authors declare that they have no conflicts of interest, that the work has been approved by the ethics committee responsible in the workplace, and do not declare means of financing of the work carried out.

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