Chapter 20
Treatment of the Aging Face

Gulhima Arora, Sandeep Arora, and V. Sandeep Lal

20.1 Introduction

Aging is inevitable and progressive, starting in the third decade of life. Contributory factors are both intrinsic and extrinsic and hence both uncontrollable and controllable. Chronological aging encompasses involutional and structural changes and biological aging encompasses genetics as well as extrinsic factors such as ultraviolet radiation, pollution, and lifestyle habits. The two may, however, be asynchronous. The face is considered as one of the most important interfaces between the self and the outer world. Evident signs of aging pull back on one's self-esteem and image, making people seek treatment for the same [1]. This is more so with an increase in the elderly population cohort and an increase in awareness regarding the remedies for aging. There is a need for earlier interventions in aging skin in our population as Asian Indian population has been proposed to age earlier than the reported ages in Caucasian population [2].

G. Arora (✉)
Mehektagul Dermaclinic, New Delhi, India

S. Arora
Department of Dermatology, Army College of Medical Sciences, Base Hospital Delhi Cantt, New Delhi, India

V. Sandeep Lal
Victoria (Women and Child) Hospital, Kollam, Kerala, India

R. Sarkar, S. Sinha (eds.), *Skin Diseases in Females*,
https://doi.org/10.1007/978-981-16-6065-8_20

20.2 Pathophysiology of Aging

The complex biological process of aging influenced by extrinsic and intrinsic factors leads to cumulative physiological and structural changes in the face (Fig. 20.1). Senescent aging versus premature photoaging is on account of deeper structural changes as well, while the latter is confined only to skin changes.

Slower rate of epidermal cell turnover and the increase in cell cycle time, with a slower wound healing and less effective desquamation in older adults results in thin, atrophic, finely wrinkled, and intrinsically dry aged skin. Collagen production gradually reduces at the rate of 1% per year per unit area of the skin, with an enhanced rate seventh decade onwards. An altered ratio of collagen 3 to collagen 1 results from reduced, disorganized, and broken down collagen 1 [3, 4]. Elastin loss further results in reduced elasticity, wrinkling, and sagging of skin. Hyaluronic acid content in the epidermis reduces dramatically, while in the dermis remains stable. Alterations in matrisome interaction with extracellular matrix proteins underlines most of the changes in either type of aging skin, while genomic alterations may further predispose an individual to these changes [5, 6].

Photoaged skin has marked collagen and elastin degradation caused by the metalloproteinases and proteases which along with ineffective glycosaminoglycan reduces water binding. However, marked depletion of serine proteases, increase in pro-inflammatory proteases, and elastic fiber-associated proteins in photodamaged skin result in elastosis. Overall a reduction in functional elastin fibers versus an

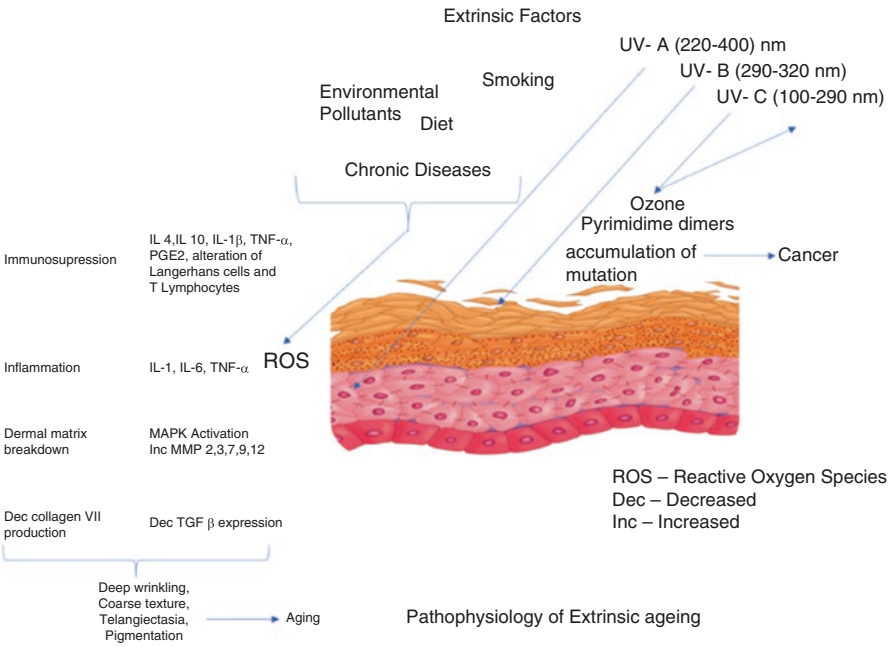

Fig. 20.1 Pathophysiology of aging

increase in non-functional elastin results in a thick mottled epidermis with deep wrinkles and a lax, dull, and rough skin.

20.2.1 Anatomy of Aging

The bones, mimetic muscles, ligaments, subcutaneous fat, epidermal and dermal layers of skin with appendages make up the facial anatomy. The progressive and accumulated changes in their shape, texture, and color result in aging of skin.

20.2.1.1 Bones

The framework of the face is provided by bones which acts as a scaffold for attachment of muscles and supporting structures. There are mainly five facial bones: skull (calvaria), nasal bone, zygoma (cheek bone), maxilla (upper jawbone), and mandible (lower jawbone). Osteoporotic changes and the loss of teeth may be the main factors resulting in the age-related effects in skull (Fig. 20.2) [7].

In old age the facial height decreases which is marked in mandible and maxilla, there is a modest increase in facial width and a minimal increase in facial depth [8].

20.2.1.1.1 Orbit

Volume of bony orbit increases with age and the curvilinear shape is distorted. Supra-medial orbital rim recedes with age in both sexes and infraorbital rim recedes laterally in women while the entire infraorbital rim recedes in men (Fig. 20.2). This loss of volume and lateral projection of orbit causes loss of support of soft tissues causing its descent and bunching. This is the reason for lateral orbital hooding and

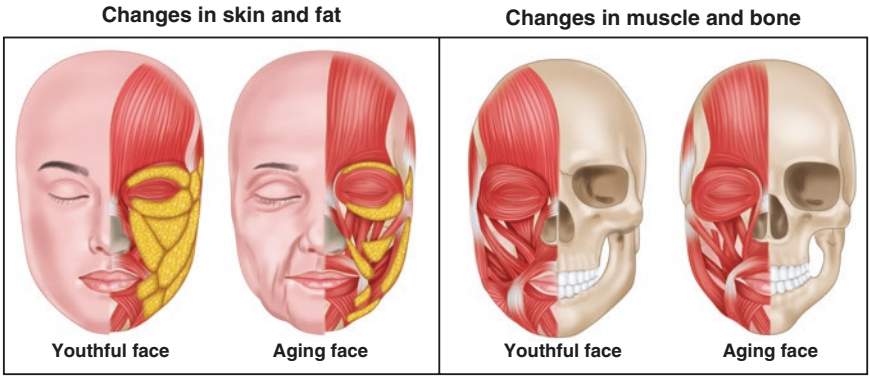

Fig. 20.2 Changes in face with aging

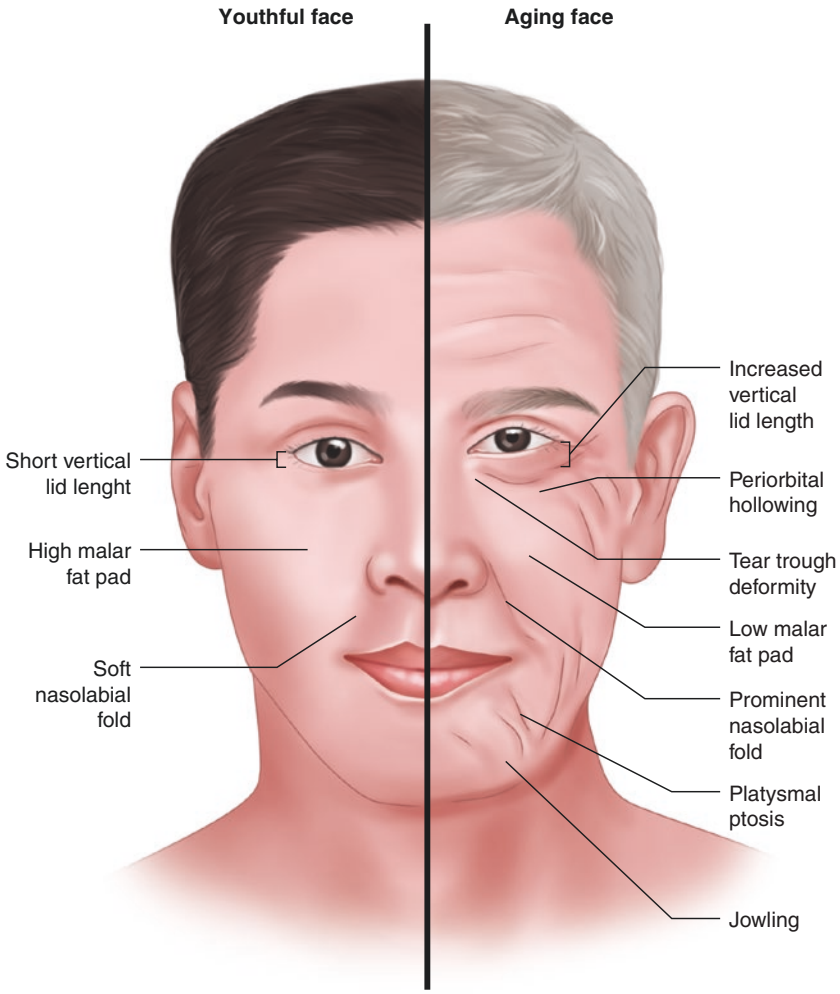

Fig. 20.3 Aging changes on the face

crow's feet. Glabellar crease is formed due to the glabellar angle being more acute with age and thereby leading to descent of medial brow. These changes give an appearance of descending brows, lateral orbital hooding of upper eye lid, and the descent of the lower lid and its junction with the cheek with deepening of the naso-jugular groove (Fig. 20.3).

20.2.1.1.2 Maxilla

The loss of teeth and bone resorption is most evident in the maxilla. The reduction in height of retrusion of lower maxillary skeleton at pyriform area causes the malar fat pad to slide downwards and forwards contributing to the prominence of nasolabial folds.

20.2.1.1.3 Mandible

The loss of teeth and associated resorption causes reduction in alveolar height. This resorption along with the depository effects leads to a change in the contour giving an appearance of "witch's chin." The prejowl sulcus (genio-mandibular) and increased labio-mental fold are the recognized features of aging in the lower face (Fig. 20.3).

20.2.1.2 Mimetic Muscles and Ligaments

The muscles of facial expression that are affected by aging are the frontalis, corrugators, procerus, orbicularis oculi, levator labii superioris alaeque nasi, nasalis, depressor septi nasi, masseter, orbicularis oris, mentalis, and depressor anguli oris (Table 20.1). The declining number of functional motor units and decreasing efficiency of muscles are the factors contributing to aging. Hypertrophy of orbicularis oculi is the most established reason for aging in the periorbital region. The aged periorbital region reveals progressive descent of the lid-cheek junction and lid lag with an increased scleral show, an evident nasojugal groove, malar bags, and tear trough formation.

The ligamentous attachments of face are divided into osteocutaneous and fasciocutaneous ligaments. Main osteocutaneous ligaments are the zygomatic and mandibular which originate from periosteum of malar and mandibular bones and are inserted into the dermis, the masseteric and parotid ligaments are fascial coalescence, the attenuation of these results in descent of the malar and buccal pad of fat augmenting the nasolabial fold and exacerbating the jowls (Fig. 20.4).

The SMAS described first by Mitz and Peyronie is an upward extension of superficial cervical fascia [9].

In the neck the main signs of aging are the development of platysmal bands associated with enlargement and ptosis of glands. These age-related changes cause increase of cervicomental angle and deposition of subplatysmal fat (Fig. 20.5).

Table 20.1 Mimetic muscles and their action

Muscle	Function	Result
Frontalis	Elevator of the medial and lateral brow	Horizontal ridges and frown lines
Procerus, corrugator supercilii, and the medial aspect of the orbicularis oculi	Depressors along the medial brow	Glabellar lines
Lateral half of the orbicularis oculi	Lateral depressor of the brow	Crow's feet
Nasalis	Flares and constricts the nostrils	Bunny lines

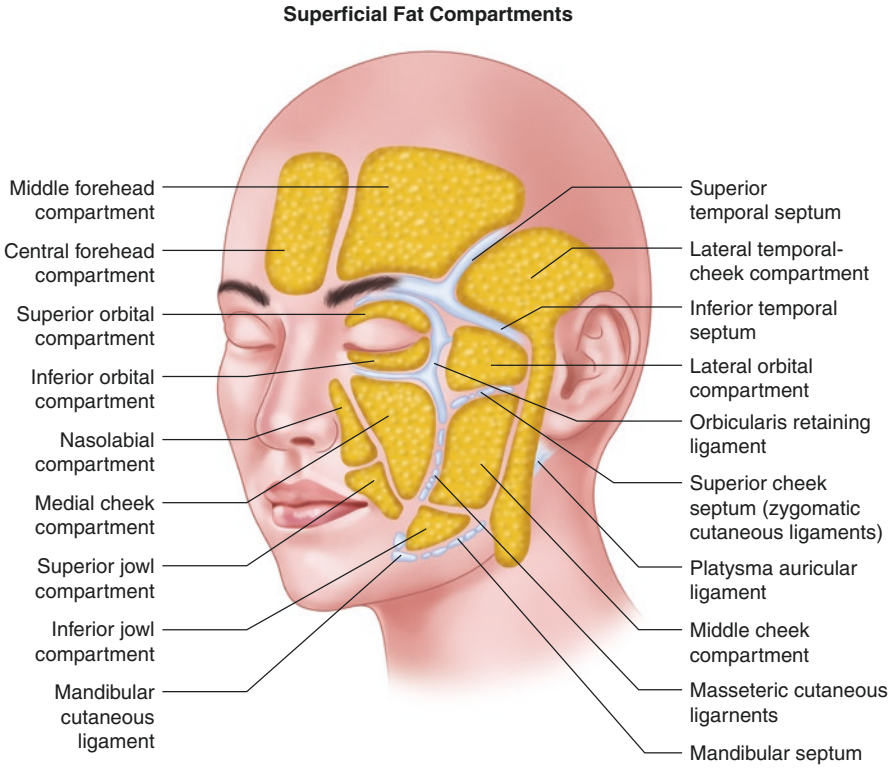

Fig. 20.4 Ligaments of the face

20.2.1.3 Subcutaneous Fat

Loss of fat plays a major role in facial aging. Rodrich and Pessa characterized subcutaneous fat compartments of face and postulated the theory of differential affection of aging in these groups [10].

The uniform diffuse and balanced distribution of fat in young is lost with aging, especially particularly around the orbit, forehead, glabella, mandible, malar, mental, and perioral regions. Dynamic rhytids which later turn into static wrinkles are due to loss of subcutaneous fat which gives prominence to contraction of the underlying muscle.

There is hypertrophy of superficial fat pads and atrophy of deep fat pads with age. Buccal pad of fat increases the marionette fold and the jowls, while malar fat pad causes prominence of the nasolabial fold and unmasking orbital fat pad.

Aging also causes reduction in hair, change in texture of skin, and pigmentary changes.

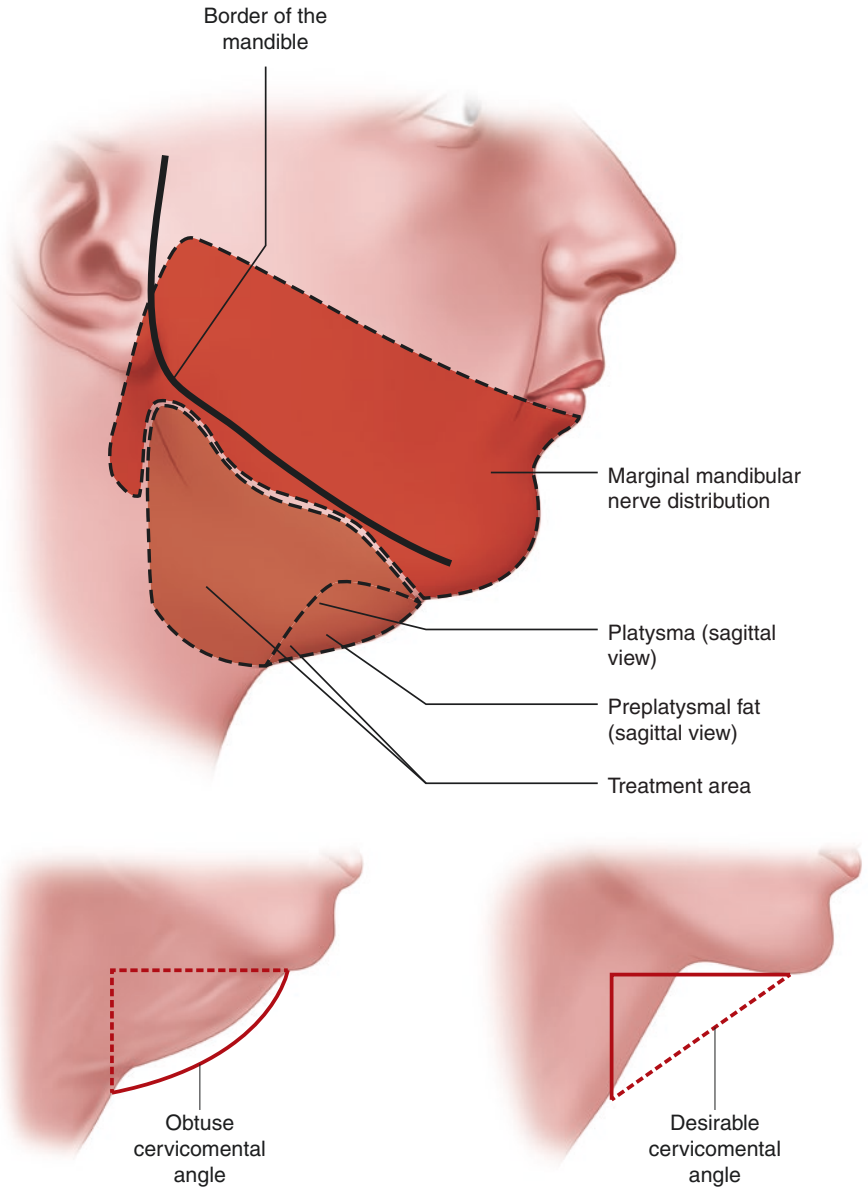

Fig. 20.5 Age-related changes of the neck

20.2.2 Clinico-Pathological Features of Aging

Aging of skin is a complex multisystem degenerative process in which the environmental changes are superimposed on the chronological senescence. These changes affect the skin, the support system and the appendages by altering its shape, texture and color. As a process aging is more pronounced and appears early in the photo-exposed areas and is of two types mainly intrinsic or extrinsic. Chronological or intrinsic aging is influenced by the genetic factors we inherit and depends on the passage of time. Intrinsic aging manifest mainly as fine wrinkles, thin transparent skin, hollow cheeks, dry skin with reduced sweating, and hair loss. This is due to progressive atrophy of skin and appendages along with the osteoporotic changes resulting from progressive senescence, defects in autophagy, and telomere shortening. The extrinsic aging, also called photoaging, is caused by environmental factors such as UV radiations, smoking, and pollutants which results in the production of reactive oxygen species (ROS) and DNA damage. This predominantly affects the photo-exposed areas manifesting as coarse wrinkling, roughness, pigmentary changes, loss of elasticity, telangiectasias, and rarely skin cancers.

Clinical	Histological
Textural changes/ roughness	Increased compaction of stratum corneum, increased thickness of granular layer, reduced epidermal thickness, and reduced epidermal mucin content
Irregular pigmentation	
Freckles	Reduced or increased number of hypertrophic, strongly dopa-positive melanocytes
Solar lentigines	Elongation of epidermal rete ridges; increases in number and melanization of melanocytes
Guttate hypomelanosis	Decreased dopa oxidase-positive, KIT+, and melanocyte sand reduction in melanocytes
Wrinkles	Thinned epidermis as well as less elastotic changes, tropoelastin, and collagen VII when compared to the surrounding photoaged skin
Sagging	Loss of elastic tissue in the dermis and the remaining fibers were disorganized, shortened, and fragmented
Inelasticity	Accumulated large amounts of homogenization and a dark blue amorphous elastotic material in the dermis, the so-called solar elastosis
Vascular changes	
Telangiectasia	Ectatic vessels often with atrophic walls
Senile purpura	Extravasated erythrocytes and increased perivascular inflammation

20.2.3 Assessment Tools for Objective Improvement of Aging

Objective assessment along with patient satisfaction is essential in esthetic practice [11–13]. A number of tools are available to assess the treatment outcome and to assess the degree of aging. The most essential among these are pre-procedural and post-procedural photographs taken with uniform lighting, patient posture, and similar photography equipment. The validated subjective scoring systems include the

wrinkle severity rating scale (WSRS), the medicis midface volume scale (MMFVS), the global aesthetic score (GAIS) and patient satisfaction score using the face visual scale. Objective assessments use instruments such as the cutometer for skin deformation, mexameter or chromatometer for color changes, sebumeter to assess sebum production, tewameter to determine the transepidermal water content, and visiometer to assess the skin topography [14]. Dermoscopy, reflectance confocal microscopy, and 3D imaging with or without projection systems are increasingly being used [15]. Histopathology remains the gold standard for assessment of tissue changes; however, it is cumbersome. Overall a combination of physician's tool along with patient satisfaction index provides the most comprehensive assessment.

20.3 Female Facial Esthetics

20.3.1 Signs of Aging

The aged skin shows the signs of photodamage pertaining to skin texture, pigmentation, and structure like atrophic, dry skin, loss of skin elasticity, uneven skin tone, pigmentation, blotchiness and telangiectasis, fine lines, and wrinkles.

Soft tissue aging gives the appearance of dynamic static wrinkles and folds, volume depletion, and ptosis of the facial tissues with sagging [8, 16].

20.4 Preventative Treatment of Aging

Beauty and youthfulness are intricately related to health. Taking care of the facial skin from the early years has proved to be beneficial in prolonging biological aging to a great extent. The authors describe a few treatments which they describe as "preventative," which can delay aging to an extent.

20.4.1 Skin Care

Dermaceuticals which involve the use of SPFs and barrier retaining creams would logically be the first-line preventative treatments in the second and third decades. Maintaining a healthy skin barrier protects against dehydration, harmful effects of environmental insults, allergens, and microbes. The use of creams with ceramides, neutral lipids, and natural moisturizing factors proves useful.

The use of sun protection factors both chemical and physical is also preventative in causing premature photoaging and the initial molecular damage by the reactive oxygen species (ROS) and the cascade of chronic photodamage that follows [17].

"Prejuvenation" is the term that is used to delay the onset of photodamage of the skin by the harmful effects of ultraviolet radiation.

Alpha hydroxy acids can be added to the skin care regime in the fourth decade, and retinoids in the late fourth and fifth decades. Their mechanism of actions are to regulate the growth and differentiation of the epithelial cells, improving the epidermal barrier and evening the skin tone [18], acting as an antioxidant, reducing fine lines and inflammation [19], especially in combination with vitamin E [20], maintaining under-eye pigmentation, and suppressing the effect of collagenase following UV radiation, respectively [21]. Preventative injectables are also being used to maintain the early signs of aging.

Botulinum toxin is used for treating motor wrinkles in the early stages before they become static, and hence can be used preventatively.

Collagen induction therapies to maintain skin elasticity can also be done. Monofilament polydioxanone threads can also be used for rejuvenation by increasing the vascularization and collagen production.

The use of regenerative medicine procedures like platelet rich plasma or injectable platelet rich fibrin are also done to maintain the biological skin health. Autologous micrograft transplant using multipotent progenitor cells can also be used.

Lifestyle modifications such as avoidance of smoking, managing stress, and weight reduction are of equal value in slowing down the aging process.

Systemic antioxidants which scavenge the free radicals, neutralize and decrease ROS by quenching iron are also used as preventative and antiaging treatment options. Oral vitamin C, vitamin E, trace elements like selenium which increases glutathione peroxidase, carotenoids, and copper are a few of them [22–24].

Chemical peels are also preventative in delaying and reversing the early signs of photodamage.

20.4.2 Topical Antiaging Agents

Antioxidants in different formulations can be used as antiaging applications. They quench and reduce the free radicals in the skin and thus reduce collagen degradation. Polyphenols and flavonoids along with topical vitamins are the most commonly used antioxidants.

The power of topical vitamins to skin care is beneficial when started at any age. The commonly used topical vitamins are vitamins A, B, C, E, and K and Co-enzyme Q10. Vitamin C is used in the concentration of 5–15%. Studies have proved that in combination with vitamin E it acts as a stronger antioxidant. Vitamin B3 or niacinamide is used in the concentration of 5%, and vitamin E in 2–20% as antiaging agents. Vitamin A or retinol and its derivatives, retinaldehyde, and tretinoin are among the most powerful antiaging agents. In the concentration of 0.05% tretinoin is approved as an antiaging treatment in the USA. It has also been shown to reduce the early signs of photodamage [25]. Their mechanism of actions are to regulate the growth and differentiation of the epithelial cells, improving the epidermal barrier and evening the skin tone [18], acting as an antioxidant, reducing fine lines and inflammation [19, 20], maintaining under-eye pigmentation, and suppressing the effect of collagenase following UV radiation, respectively [21].

Other antiaging agents such as polypeptides and growth factors can induce the production of collagen and decrease the effect of matrix metalloproteinases [26].

20.4.3 Minimally Invasive Antiaging Treatments

20.4.3.1 Treatment of Structural Changes

20.4.3.1.1 Treatment of Wrinkles

The facial expression lines are usually tackled with botulinum toxin. It is not completely therapeutic for static wrinkles, but it does soften them. The injections are intramuscular, relaxing the muscles responsible for rhytid formation by preventing the release of acetylcholine at the neuromuscular junction. The number of units needed depend on the muscle bulk. Females need lesser units compared to males.

Static wrinkles need filling with hyaluronic acid, biologic materials, or other synthetic fillers.

Energy-based devices like radiofrequency can also be used to soften wrinkles.

20.4.3.1.2 Treatment of Folds

Direct filing of folds can be done with the use of biodegradable or synthetic fillers. Autologous lipofilling can also be done. A retrograde or antegrade technique can be used. Direct filling can also be achieved with the use of "filler" or "broom" threads, which stimulate collagenization. Platelet rich fibrin when done over sessions, can also stimulate collagen, thus making them shallow.

Indirect softening of the nasolabial or marionette lines can be done by repositioning the soft tissues in vectors opposite to the aging vectors with injections of fillers on the lateral side of the face.

Sutures or thread lifts can also cause attenuation of the folds by lifting and stretching the tissues adjacent to them.

Energy-based devices like high intensity focused ultrasound for lifting the facial tissues also soften them.

20.4.3.1.3 Volume Restoration

Biodegradable or non-biodegradable synthetic fillers can be used to restore volume that gets depleted with age. Instant volumization occurs with the use of these cross-linked gels. Natural fillers like biofillers, which are platelet poor plasma gels, can also be used with short-term longevity. Collagen induction with threads, platelet rich plasma, or bioactive hyaluronic acid gels can also be done, but the process takes several sittings along with minimal stimulation.

Injections can be supraperiosteal for structural support and greater longevity or in the deep atrophic fat pads for contouring and filling. The former requires gels with a high cohesivity, and the latter can be done with gels having greater viscosity and tissue integration.

20.4.3.1.4 Treatment of Sagging

Lifting of ptotic tissues against the vectors of aging is best done with thread lifts. Usually threads with barbs or cones are used as lifting threads. The insertion is in vectors perpendicular to the ptosis of the tissue. Insertion is always in the subcutaneous plane.

Peripheral injections of fillers can also stretch and lift the face against the aging vectors. Injecting in the lateral zygoma, for example, lifts the face and softens the nasolabial fold.

Energy-based devices are also very useful to lift sagging tissues. The use of high intensity focused ultrasound (HIFU) when used in the depths of 4.5 and 3 mm can target the SMAS and the deep dermis, respectively. Monopolar and some good bipolar radiofrequency devices can also tighten the sagging skin, but they do not penetrate as deep as HIFU. These work on the principle of stimulating the extracellular matrix proteins and improving collagenization and elastogenesis.

20.4.3.2 Laser and Energy-Based Devices

With the demand for non-aggressive, non-invasive, non-surgical procedures with minimal downtime increasing lasers and energy-based devices have a special place in management of the aging skin.

The lasers used may be of differing wavelengths as ablative or non-ablative in fractionated or non-fractionated forms. These lasers function to target pigmentation, erythema, irregular vessels, sebaceous changes, rhytids, and other senescent changes.

Ablative lasers are more aggressive with a longer downtime e.g., carbon dioxide, erbium YAG laser. They function by causing skin resurfacing by epidermal removal and dermal remodeling by dermal heating causing collagen denaturation and subsequent resynthesis. They are best suited for severe facial rhytids and pigmentary changes. The superficial rhytids are targeted directly while the deeper ones improve with subsequent dermal remodeling.

Non-ablative lasers cause the above dermal remodeling without or minimal epidermal damage, e.g., erbium glass, pulse dye laser, Nd: YAG, diode, and intense pulse light.

Ablative and non-ablative lasers may be fractionated or non-fractionated wherein the laser beam is fractionated into columns. This prevents excessive epidermal damage and dermal overheating.

Ablative lasers generally have been reported to have a better clinical response compared to non-ablative lasers.

20.5 Conclusion

Treatment of the aging face is a part of "successful aging." It boosts the self-esteem of an individual and hence the overall well-being. Preventative and restorative esthetic dermatology thus play a convincing role in healthy aging. Preventative anti-aging strategies can even prevent certain cutaneous malignancies. Treatment of the aging face is not just to look younger, but to look better with an even tone and a good skin texture, free from pigmentary conditions. Antiaging encompasses a holistic and combination approach from lifestyle modification to targeted treatments.

References

1. Baker L, Gringart E. Body image and self-esteem in older adulthood. Ageing Soc. 2009;29:977–95.
2. Shome D, Vadera S, Khare S, Ram MS, Ayyar A, Kapoor R, et al. Aging and the Indian face: an analytical study of aging in the Asian Indian face. Plast Reconstr Surg Glob Open. 2020;8:e2580.
3. Varani J, Dame MK, Rittie L, Fligiel SEG, Kang S, Fisher GJ, et al. Decreased collagen production in chronologically aged skin: roles of age-dependent alteration in fibroblast function and defective mechanical stimulation. Am J Pathol. 2006;168:1861–8.
4. Oikarinen A. Aging of the skin connective tissue: how to measure the biochemical and mechanical properties of aging dermis. Photodermatol Photoimmunol Photomed. 1994;10:47–52.
5. Kołodziej-Wojnar P, Borkowska J, Wicik Z, Domaszewska-Szostek A, Połosak J, Cąkała-Jakimowicz M, et al. Alterations in the genomic distribution of 5hmC in in vivo aged human skin fibroblasts. Int J Mol Sci. 2020;22:78.
6. McCabe MC, Hill RC, Calderone K, Cui Y, Yan Y, Quan T, et al. Alterations in extracellular matrix composition during aging and photoaging of the skin. Matrix Biol Plus. 2020;8:100041.
7. Ozturk CN, Ozturk C, Bozkurt M, Uygur HS, Papay FA, Zins JE. Dentition, bone loss, and the aging of the mandible. Aesthet Surg J. 2013;33:967–74.
8. Coleman S, Grover R. The anatomy of the aging face: volume loss and changes in 3-dimensional topography. Aesthet Surg J. 2006;26:S4–9.
9. Mitz V, Peyronie M. The superficial musculo-aponeurotic system (SMAS) in the parotid and cheek area. Plast Reconstr Surg. 1976;58:80–8.
10. Rohrich RJ, Pessa JE. The fat compartments of the face: anatomy and clinical implications for cosmetic surgery. Plast Reconstr Surg. 2007;119:2219–27.
11. Day DJ, Littler CM, Swift RW, Gottlieb S. The wrinkle severity rating scale: a validation study. Am J Clin Dermatol. 2004;5:49–52.
12. Shoshani D, Markovitz E, Monstrey SJ, Narins DJ. The modified Fitzpatrick Wrinkle Scale: a clinical validated measurement tool for nasolabial wrinkle severity assessment. Dermatol Surg. 2008;34:S85–91.
13. Sen S, Choudhury S, Gangopadhyay A, Halder C, Biswas P, Jain A. A clinical rating scale for the assessment of facial aging in Indian population. Indian J Dermatol Venereol Leprol. 2016;82:151.

14. Hersant B, Abbou R, SidAhmed-Mezi M, Meningaud JP. Assessment tools for facial rejuvenation treatment: a review. Aesthet Plast Surg. 2016;40:556–65.
15. Verhulst A, Hol M, Vreeken R, Becking A, Ulrich D, Maal T. Three-dimensional imaging of the face: a comparison between three different imaging modalities. Aesthet Surg J. 2018;38:579–85.
16. Kaur M, Garg RK, Singla S. Analysis of facial soft tissue changes with aging and their effects on facial morphology: a forensic perspective. Egypt J Forensic Sci. 2015;5:46–56.
17. Baumann L. Skin ageing and its treatment. J Pathol. 2007;211:241–51.
18. Bissett DL, Oblong JE, Berge CA. Niacinamide: a B vitamin that improves aging facial skin appearance. Dermatol Surg. 2006;31:860–6.
19. Gaspar L, Campos P. Photostability and efficacy studies of topical formulations containing UV-filters combination and vitamins A, C and E. Int J Pharm. 2007;343:181–9.
20. Lin F-H, Lin J-Y, Gupta RD, Tournas JA, Burch JA, Angelica Selim M, et al. Ferulic acid stabilizes a solution of vitamins c and e and doubles its photoprotection of skin. J Invest Dermatol. 2005;125:826–32.
21. Choi CM, Berson DS. Cosmeceuticals. Semin Cutan Med Surg. 2006;25:163–8.
22. Terada A, Yoshida M, Seko Y, Kobayashi T, Yoshida K, Nakada M, et al. Active oxygen species generation and cellular damage by additives of parenteral preparations: selenium and sulfhydryl compounds. Nutrition. 1999;15:651–5.
23. Fusco D, Colloca G, Lo Monaco MR, Cesari M. Effects of antioxidant supplementation on the aging process. Clin Interv Aging. 2007;2:377–87.
24. Marini A. Beauty from within: does it really work? Hautarzt. 2011;62:614–7.
25. Ascenso A, Ribeiro H, Marques H, Oliveira H, Santos C, Simões S. Is tretinoin still a key agent for photoaging management? Mini-Rev Med Chem. 2014;14:629–41.
26. Page-McCaw A, Ewald AJ, Werb Z. Matrix metalloproteinases and the regulation of tissue remodelling. Nat Rev Mol Cell Biol. 2007;8:221–33.