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Autoimmune disease and aesthetic medicine: a holistic approach

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ABSTRACT

Introduction and purpose

The field of aesthetic medicine has experienced rapid advancement in recent years. It serves primarily aesthetic purposes while also supporting various medical treatments. The range of available aesthetic medicine procedures continues to expand, with numerous studies confirming their therapeutic efficacy in treating certain diseases or conditions. Conversely, a substantial list of contraindications exists, particularly in autoimmune diseases, necessitating caution and precluding the performance of certain aesthetic treatments.

Autoimmune diseases entail the body's immune system attacking its tissues, a process whose complete development and maintenance in specific diseases remains incompletely understood. Injection treatments like hyaluronic acid and mesotherapy have demonstrated considerable promise in aiding individuals with autoimmune connective tissue diseases to lead everyday lives. Laser therapy, encompassing various types of lasers with broad-ranging applications, presents a high likelihood of therapeutic success for treating diverse body areas.

This study discusses to what extent selective aesthetic medical procedures could be safely performed in patients with the most common autoimmune diseases.

Material and methods

A literature review was conducted through an extensive bibliographic search, primarily focusing on original research articles from reputable databases such as PubMed, BioMed Central, Polish Medical Platform, and Google Scholar. The search was specifically targeted towards articles discussing particular autoimmune diseases and specific aesthetic procedures.

Conclusion

Established relationships between autoimmune diseases and specific aesthetic medicine treatments have been identified, highlighting their ineffectiveness or inadvisability. In such instances, the physician's extensive knowledge and the necessity for a comprehensive, individually tailored assessment before the procedure often dictate a patient's exclusion from specific therapies.

Keywords: *aesthetic procedures, autoimmune diseases, side-effects of aesthetic procedures*

INTRODUCTION

1.1 The immune system

The immune system is responsible for numerous selective mechanisms crucial for the organism's survival. It is an integrative system throughout the body, communicating with the nervous and hormonal systems. The immune system directly and indirectly regulates various processes such as embryogenesis, carcinogenesis, and pregnancy. It can differentiate between self and foreign structures, as well as learn and remember. The immune system utilizes specific (acquired) and non-specific (innate) immunity mechanisms when encountering pathogens. Innate immunity is associated with various immune system cells and their activities, including defensin-, cytokine-, and chemokine-dependent immunity, as well as the activity of NK (Natural Killer) cells, cytotoxic complement activity, phagocytosis, and T lymphocyte reactivity towards viruses[1]. Non-specific immunity is the first defence against infections and diseases resulting from environmental factors and functions independently of previous pathogen exposure[1]. While less precise, these mechanisms enable rapid recognition and destruction of invading microorganisms.

Innate immunity is mediated by cells such as macrophages, monocytes, mast cells, granulocytes, "innate" γδ lymphocytes, NK and NKT cells, and natural lymphoid cells (ILC)[1]. Recent research suggests the involvement of additional cells in the innate response, including nTh2 (Natural T Cells), NHC (Natural Helper Cells), monocytes, and multipotent type II progenitor cells[1]. The principal components of specific immunity include T lymphocytes, B lymphocytes, antigen-presenting cells, cytokines, and antibodies. These components can generate infinite receptors and develop immunological memory upon antigen exposure, leading to an enhanced immune response upon subsequent encounters with recognized antigens. The immune response involving T lymphocytes, originating from the thymus, is a specific cellular response, while B lymphocytes, originating from the bone marrow, are involved in a specific humoral response.

Cellular immunity, an essential aspect of specific immunity, is primarily responsible for combating infections caused by intracellular microorganisms and plays a crucial role in contact reactions with chemical compounds, specific autoimmune reactions, and the rejection of transplants or cancerous tissues[2]. The humoral response involves the release of antibodies by B lymphocytes and plasma cells, followed by their binding to and neutralizing antigens[2].

During the immune response, various classes of antibodies appear, including IgE, IgM, and IgG, each stimulated by specific antigens[2]. Lymphoid organs, comprising immune system cells, serve as sites for antigen recognition and lymphocyte residence. Highly specialized lymphocytes capable of recognizing antigens and producing antibodies are central to the immune response. Natural Killer (NK) cells, lacking markers or antigen receptors, contribute to the immune response by inducing cytotoxic damage, particularly to cancer cells[2]. With their phagocytic ability, macrophages remove dead and worn-out cells, neutralize phagocytosed microorganisms, and release biologically active substances that modulate lymphocyte activity and inflammation[2]. Dendritic cells

(APC), vital in innate immunity, absorb antigens, activate lymphocytes, and secrete INF-α, thereby stimulating antigen presentation. Granulocytes, including neutrophils, eosinophils, and basophils, also play essential roles in specific and non-specific defence mechanisms[2]. The complement system, comprising approximately 35 proteins, is crucial in protecting the body against microbial attacks by supporting phagocytosis and phagocyte chemotaxis[2].

1.2. The immune system of the intestinal mucosa

The mucous membranes of the digestive, respiratory, and integumentary systems are the primary interfaces between the body and the external environment. As such, these membranes are equipped with specific and non-specific defence mechanisms to protect against external factors. The immune system of the mucous membranes plays a crucial role in this defence, with lymphatic tissue in the form of solitary and organized clusters of lymphatic follicles representing its primary component.

Mucosa-Associated Lymphoid Tissue (MALT) encompasses the tissue in the respiratory tract within the bronchi, Bronchus-Associated Lymphoid Tissue (BALT) includes the mucosal and submucosal tissue of the digestive system, Gut-Associated Lymphoid Tissue (GALT) represents the tissue of the throat and nose, and Nose-associated Lymphoid Tissue (NALT) comprises the tissue of the salivary glands, lacrimal glands, mammary glands, and glands of the urogenital system[3].

The intestines are considered the most extensive human organs, with a surface area ranging from 200 to 400 m^2, and rely on specific and non-specific immune mechanisms for proper function[3]. Critical components of this defence include the microflora, lysozyme, low pH, antimicrobial peptides, and proteolytic enzymes[3]. Effector cells, such as secretory IgA immunoglobulins, inhibit bacterial cell adhesion, absorb antigens, and neutralize viruses and toxins[3]. The cylindrical epithelium, comprising absorbing cells like enterocytes, goblet cells, and epithelial leukocytes, constitutes an additional barrier against invading pathogens[3].

In the small intestine, Paneth cells, located at the base of the crypts, play a critical role in controlling microbial populations and protecting neighbouring stem cells by releasing lysozyme, phospholipase, α-defensins, and cathelicidins (antimicrobial peptides)[3]. The lamina propria of the mucosa, containing T and B lymphocytes, macrophages, granulocytes, eosinophils, and mast cells, also serves an equally important function. Mast cells, for instance, contribute to fighting infections and enhancing the mucosal microenvironment mainly through the chemotaxis of neutrophils and eosinophils, stimulating mucus secretion and increasing epithelial permeability[3].

Peyer's patches, as lymphatic structures, represent another fundamental component in the intestines, serving as the site for induction of the immune response leading to general immunity[3]. Furthermore, the gastrointestinal mucosa contains a substantial number of intraepithelial (scattered) lymphocytes, primarily T lymphocytes (90%) and γδ T lymphocytes (10%)[3]. These cells possess cytotoxic properties, exert suppressive effects, prevent immune reactions to food antigens, and regulate intestinal epithelium renewal[3].

The immune system's primary function is safeguarding the body against infections, cancer, and various diseases. Any disruptions in the proper functioning of this system, whether in the form of deficiencies or over-reactivity, can lead to abnormalities in the division, maturation, and interaction of immune system cells. Numerous factors, including genetic predisposition, age, gender, nutrition, physical activity, and stress, influence the proper functioning of the immune system and other bodily systems[3].

1.3. Autoimmunity

Autoimmunity refers to the immune system's response against one's antigenic cells or autoantigens. This process is commonly initiated when acquired immunity is compromised, leading to a failure to distinguish between "self" and "foreign" antigens[4]. Presently, approximately 70 autoimmune diseases have been identified through research[4]. These diseases typically follow a chronic course and may result in disability or fatality. Autoimmune diseases are categorized based on the location of the autoantigen, leading to either organ-specific diseases involving a particular organ or systemic diseases affecting the entire body. Furthermore, autoimmune diseases are also classified based on the predominant effector mechanisms, which can be cellular or humoral[4]. It is important to note that both these classifications coexist and complement each other in the context of autoimmune diseases.

Type of disorder	Example	Effect of disorder	Autoantibodies present	Autoreactive T cells
Organ specific	autoimmune hemolytic anemia	destruction of erythrocytes	antibodies to erythrocyte antigens	
	autoimmune thyroiditis	hypothyroidism	antibodies to thyroglobulin and thyroid peroxidase	T_H1 cells specific for thyroid antigens
	Addison's disease	adrenal insufficiency	antibodies to cytoplasmic antigens of cells of adrenal cortex	infiltration of adrenal cortex with autoreactive T cells
	type 1 diabetes mellitus	destruction of insulin-producing cells in pancreas; serious metabolic disturbances	antibodies to islet cells found in classical juvenile form	infiltration of pancreas with autoreactive T cells
	Goodpasture's syndrome	progressive kidney and lung damage	antibodies to basement membrane antigens of kidney and lung	
	Graves disease	hyperthyroidism	antibodies to thyroid stimulating hormone receptors	destruction of thyroid cells by autoreactive T lymphocytes
	myasthenia gravis	progressive muscle weakness	antibodies to acetyl choline receptors on muscle cells	
	pernicious anemia	failure to absorb vitamin B_{12} in the stomach (,)	antibodies to intrinsic factor	
Systemic disease	rheumatoid arthritis (RA)	inflammatory disorder affecting joints, skin and internal organs	antibodies to IgG (rheumatoid factor)	infiltration of joints with autoreactive T lymphocytes
	systemic lupus erythematosus (SLE)	inflammatory disorder affecting multiple organ systems	antibodies to DNA, chromatin and histones; rheumatoid factor in some individuals	evidence of T cell reactivity in some of the many organs affected
	multiple sclerosis (MS)	inflammatory disorder affecting central nervous system	antibodies to myelin basic protein	destruction of myelin membrane by autoreactive T lymphocytes

Table 1. Examples of autoimmune diseases classification based on location of the antigen[5].

2. ETIOPATHOGENESIS OF SELECTED AUTOIMMUNE DISEASES

2.1 Type I diabetes

Type 1 diabetes mellitus is a condition characterized by the destruction of β cells, resulting in a complete deficiency of insulin production, and is primarily attributed to an immunological process. This form of diabetes, which was previously referred to as insulin-dependent diabetes, type 1 diabetes, or juvenile diabetes, affects only 5-10% of the population[6]. The onset of the disease is caused by cellular autoimmunity leading to the destruction of pancreatic β cells. Critical indicators of cell destruction include antibodies against islet cells and insulin, as well as antibodies against glutamic acid decarboxylase (GAD65) and tyrosine phosphatases IA-2 and IA-2b[6]. The rate of β-cell destruction varies widely, with rapid destruction occurring primarily in infants and children and a slower progression in adults. Children and adolescents often present with ketoacidosis as the initial symptom, while others may experience mild hyperglycemia, which can progress to severe hyperglycemia and ketoacidosis during times of stress or infection[6]. In the advanced stages of the disease, insulin secretion by the pancreas is either completely absent or significantly reduced. Both genetic and environmental factors influence autoimmune destruction of pancreatic β cells. Additionally, this disease is frequently associated with other autoimmune conditions such as Graves' disease, Hashimoto's disease, Addison's disease, vitiligo, celiac

disease, autoimmune hepatitis, or pernicious anemia[6]. Type 1 diabetes mellitus is a condition characterized by the destruction of β cells, resulting in a complete deficiency of insulin production, and is primarily attributed to an immunological process[6]. This form of diabetes, which was previously referred to as insulin-dependent diabetes, type 1 diabetes, or juvenile diabetes, affects only 5-10% of the population[6]. The onset of the disease is caused by cellular autoimmunity leading to the destruction of pancreatic β cells[6]. Key indicators of cell destruction include antibodies against islet cells and insulin and against glutamic acid decarboxylase (GAD65) and tyrosine phosphatases IA-2 and IA-2b[6]. The rate of β-cell destruction varies widely, with rapid destruction occurring primarily in infants and children, and a slower progression in adults[6]. Children and adolescents often present with ketoacidosis as the initial symptom.

2.2. Rheumatoid arthritis

Rheumatoid arthritis (RA) is a chronic autoimmune disease affecting connective tissue, with its initial impact seen in the synovium of the joints. This leads to the destruction of joint tissues and eventual structural damage, resulting in progressive disability. While categorized as a connective tissue disease, RA can also have systemic effects, impacting various systems and organs throughout the body. Initially, patients may experience different forms of joint pain, which can be mistaken for typical degenerative joint changes in older individuals[7]. The recurrent nature of the symptoms and their manifestation in other systems or organs often prompt a broader diagnostic approach, ultimately leading to a confirmed diagnosis of RA[7]. Severe joint pain is the most common reason for patients to seek medical assistance. Many symptoms appear similar in the early stages of the disease, making an accurate diagnosis challenging. The diagnostic process involves thorough questioning to guide the diagnosis and minimize unnecessary laboratory and imaging tests[7]. Like other autoimmune conditions, ongoing inflammation and the immune system's attack on its cells in RA can lead to prolonged impact periods, potentially weakening other bodily processes[7].

2.3 Systemic lupus erythematosus

Systemic lupus erythematosus is an autoimmune disease dating back over seven centuries. Despite continuous research, the causes of the disease remain unknown, and its numerous varieties make it challenging to diagnose and classify[8]. Initially considered a dermatological disease, it is now recognized as a systemic, multi-system disease, as it presents with skin symptoms, fever, anaemia, weight loss, enlarged lymph nodes, and arthritis[8]. Kidney complications, in extreme cases, can be fatal. In recent years, the immune system's involvement has been suggested in the disease's development, adding complexity[8]. Advances

in molecular biology and immunology have led to the detection of new autoantibodies against various cellular elements, none of which are currently specific for systemic lupus erythematosus, resulting in significant diagnostic challenges[8].

2.4. Systemic sclerosis

Scleroderma, or Morphea, encompasses several subtypes and typically progresses from an initial inflammatory phase to skin hardening and subsequent tissue degeneration. From 1960 to 1993, the annual incidence was 2.7 per 100,000 individuals, with the majority (56%) experiencing lamellar Morphea, followed by 20% with linear Morphea, 13% with generalized Morphea, and 11% with profound Morphea[9]. The most common variant, plaque Morphea (limited), typically manifests as an erythematous or hyperpigmented plaque that gradually becomes hardened and spreads outward[9]. Linear Morphea and Parrye syndrome Romberg's disease are characterized by atherosclerotic plaques in a linear distribution, often affecting the scalp and hemiface[9]. Generalized scleroderma presents as plaques occurring in various anatomical locations, most commonly appearing on the trunk, with a notable absence of involvement of the hands. A rare form includes atrophoderma Pasini and Pierini and deep morph[10]. Systemic sclerosis, limited or diffuse, primarily impacts the distal limbs and face or the distal and proximal limbs, trunk, and face, respectively[10]. Both subtypes may affect internal organs, particularly the lungs, joints, and gastrointestinal tract[10]. Throughout the progression of the disease, skin changes such as microstomia, discolouration, and telangiectasia are commonly observed[10].

2.5. Graves' disease

Graves' disease stands as the primary cause of hyperthyroidism and is identified as an autoimmune disorder. The condition is characterized by the stimulation of the thyroid gland by one's antibodies to produce hormones[11]. Despite ongoing research, the precise cause and full etiopathogenesis of Graves' disease remain incompletely understood. Current understanding suggests a predominant role of genetic and epigenetic factors, with potential contributions from environmental factors such as smoking, stress, excessive iodine intake, certain medications, and infectious agents in disease development[11]. The spectrum of thyroid function disorders associated with Graves' disease includes thyrotoxicosis, less commonly euthyroidism, parenchymal vascular goitre, thyroid orbitopathy, and pretibial oedema[12]. It is noted that these symptoms may not manifest simultaneously[12]. The symptoms of Graves-Basedow's disease share similarities with hypothyroidism, with the additional presence of

visual swelling in the thyroid area and thyroid goitre contributing to an aesthetically unappealing appearance[13].

2.6. Hashimoto's disease

Autoimmune thyroiditis, also called chronic lymphocytic thyroiditis and Hashimoto's disease, is an autoimmune condition characterized by gradual damage to the gland's structure, leading to progressive impairment of its endocrine function. This disease encompasses various forms, including IgG4-dependent, fibrous, juvenile, and postpartum thyroiditis, with thyroid peroxidase (TPO) and thyroglobulin (TG) antigens playing pivotal roles[14].

Since the 1940s, Hashimoto's disease has emerged as one of the most frequently diagnosed autoimmune and endocrine disorders[14]. In the 1970s, thyroid peroxidase (TPO) was identified as the primary antigen in Hashimoto's disease, and genetic factors were also implicated in its development[14].

Diagnosis primarily relies on detecting elevated levels of thyroid antibodies and a distinctive ultrasound image. Monitoring the disease's progression and evaluating thyroid function necessitates comprehensive testing of thyroid parameters and thyroid-stimulating hormone (TSH) levels, forming the basis of substitution therapy. However, in the case of the IgG4-dependent form, short-term treatment with glucocorticosteroids may be considered[14].

SELECTED AESTETIC TREATMENTS

3.1. Hyaluronic acid

As the human body ages, there is a decrease in the volume and elasticity of body tissues, leading to dehydration. This ageing process is particularly noticeable on the face. Hyaluronic acid (HA) is the primary natural molecule responsible for retaining water in the skin[15]. The first commercially available HA filler for aesthetic use was Zyderm, approved by the FDA in 1981[15]. Initially made of bovine collagen, it was associated with numerous allergic reactions, which led to introducing an artificial hyaluronic acid molecule in 2003. Currently, over 160 products from more than 50 different manufacturers are available on the market[15].

Hyaluronic acid is a glycosaminoglycan naturally released into the extracellular matrix by various cells, including skin fibroblasts, endothelial cells, smooth muscle cells, adventitious cells, and oocytes. It has minimal risk of immunogenicity and is stable at the implantation site, making it the molecule of choice for use as a dermal filler in aesthetic treatments[16]. Due to its rheological and viscoelastic properties, hyaluronic acid influences the extracellular environment through complex molecular interactions with cellular and matrix receptors[16]. Its hydrophilic nature allows hyaluronic acid to form extensive gel-like conformations with water

molecules, resulting in tissue hydration and increased skin turgor[16]. Hyaluronic acid comes in three forms with different molecular weights, each with different effects[16].

The use of aesthetic therapies based on hyaluronic acid is consistently increasing, with over 4.3 million aesthetic treatments using it performed in 2019, according to the International Society of Aesthetic Plastic Surgery, marking a 15.7% increase from 2018[17]. However, with this increase in usage, there are also growing reports in the literature of possible side effects, resulting therapeutic problems, and more or less severe consequences for patients. Minimizing the risk of complications involves proactive patient management and monitoring, alongside careful patient selection, to ensure that only suitable patients are treated[15].

It is crucial to avoid treating patients with pre-existing conditions that specifically prohibit the use of HA-based dermal fillers. Such conditions include skin atrophy, anetoderma, athroderma, vermiculation, thinning of the skin, and active infections in the treated region, such as viral, bacterial, and fungal infections[17].

Physicians should carefully evaluate patients with active inflammatory dermatitis, including atopic dermatitis. In patients with existing autoimmune connective tissue diseases, such as systemic lupus erythematosus or active scleroderma, administering fillers with hyaluronic acid may worsen the existing pathology[17]. Hyaluronic acid's molecular weight and shape indicate healthy or inflamed tissue and its interactions with immune cells may influence their response. Higher molecular weight hyaluronic acid predominates in healthy tissues, whereas in the event of tissue damage or infection, hyaluronic acid is broken down, resulting in an inflammatory response. Immune cells interact with hyaluronic acid in tissues and lymphatic organs and react to it in different ways depending on its molecular weight[17]. In particular, hyaluronic acid fragments present in inflamed tissues are associated with the spread of the inflammatory response[17]. Therefore, treatments based on hyaluronic acid are strictly prohibited as they might increase inflammation in patients with active phases of autoimmune diseases. In non-active phases, the usage of the product is debatable and left for consideration by the patients and doctors.

3.2 Botulinum toxin

Botulinum toxin, known as Botox, is considered one of the most potent biological substances. It is a neurotoxin produced by the anaerobic, gram-positive bacterium Clostridium botulinum and is associated with eight antigenically distinguishable exotoxins[18]. This toxin interferes with nerve transmission, explicitly blocking the release of acetylcholine, a key neurotransmitter at the neuromuscular junction[18]. The administration of botulinum toxin results in muscle paralysis by inhibiting the release of acetylcholine from presynaptic motor neurons. Its effects are irreversible, although nerve regeneration and the formation of new synaptic contacts can eventually restore function. In addition to its medical applications in treating conditions such as strabismus, focal dystonias, and excessive sweating, botulinum toxin has gained popularity in cosmetic procedures, particularly for reducing wrinkles and lines on the face and neck[21]. Despite its widespread use, concerns have been raised regarding potential immune reactions in certain individuals, such as those with thyroid autoimmune diseases, following the administration of botulinum toxin as the toxin may cause thyroid complications based on molecular mimicry with thyroid autoantigens[19]. As described by the authors, some antibodies produced after injection of botulinum toxin (anti- Btx) may bind to the thyrotropin receptor (TSH-R)[19]. This may therefore lead to the induction of anti-TSH-R antibodies that inhibit TSH-R signaling (TSH-R- blocking antibodies - TSHR-Bab), which leads to

increased TSH levels in the blood[19]. Apart from this one scientific report, the authors of the study did not identify any other studies confirming the thesis about the influence of botulinum toxin on thyroid function[20]. However, botulinum toxin is used to treat eye complications in thyroid diseases, such as strabismus or upper eyelid retraction. In addition to the effectiveness of ophthalmological treatment with botulinum toxin, its safety is also emphasized, even in the acute inflammatory phase of the disease[21].

3.3. Autologous platelet

In recent years, numerous scientific studies have illustrated the presence of cells in the adult human body capable of repairing and regenerating damaged tissues. Collecting and processing blood using an advanced extraction system makes it feasible to generate platelet-rich plasma (PRP), which harbours several vital factors conducive to supporting wound healing, rebuilding degraded cells, and nourishing various structures[22].

PRP, owing to its heightened content of platelets and growth factors such as epidermal growth factor (EGF), insulin-like growth factor 1 (IGF-1), platelet-derived growth factor (PDGF-AA, -AB, and -BB), transforming growth factor (TGF-ß1 and -ß2), and vascular endothelial growth factor (VEGF A and C), has been utilized in medicine for years[23]. These components stimulate fibroblasts, adipocytes, and endothelial cells, fostering regenerative processes and accounting for their wide application in wound healing and joint treatment[22]. The popularity of PRP treatments is on the rise, with these procedures increasingly employed in treating various diseases spanning different medical specialities. In addition to aesthetic medicine and plastic surgery applications, PRP has gained particular traction in orthopaedics[23].

Furthermore, prospective randomized clinical trials have demonstrated superior outcomes with PRP compared to steroids in treating osteoarthritis[23]. At the same time, both animal and human studies have shown promising effects of PRP in treating rheumatoid arthritis[23]. Research on using PRP in treating neurological diseases, especially multiple sclerosis, has also shown promise in animal models[23]. Moreover, there have been reports at conferences regarding the use of intrathyroid injections of PRP in the treatment of hypothyroidism, with some authors suggesting that this form of therapy reduces the levels of thyroid-stimulating hormone (TSH) and anti-thyroglobulin (anti-TG) antibodies[24]. Nevertheless, it is essential to consider that thyroid diseases can adversely affect platelet function, which is critical for the effectiveness of this treatment[24]. Thyroid hormones exert their primary effects on receptors located in the cell nucleus, while platelets lack a nucleus; however, the influence of thyroid hormones on the cytoplasmic elements of the cell is recognized[24].

Additionally, it is postulated that thyroid hormones influence nucleated stem cells (megakaryocytes) that give rise to platelets. Increased megakaryocytes in the bone marrow have also been observed in patients with hypothyroidism. Thyroid diseases also impact mean platelet volume (MPV) and platelet distribution width (PDW), with elevated measurements noted in Hashimoto's thyroiditis, hyperthyroidism, and subclinical hypothyroidism[24].

Furthermore, a study has revealed that platelet aggregation was increased in patients with untreated primary hypothyroidism and decreased in patients with untreated Graves disease. As platelet aggregation is a direct measure of platelet activation and its release of autologous growth factors and cytokines to tissues, it can be

postulated that the efficacy of PRP treatment in hyperthyroidism may be diminished, whereas in hypothyroidism it may remain unaltered. However, further comprehensive research is warranted to substantiate these conclusions. Moreover, in the context of extensive procedures involving PRP, it is advisable to monitor coagulation parameters in patients with thyroid disorders due to the heightened risk of bleeding[24].

Additionally, a study has assessed a novel regenerative approach entailing the transplantation of adipose tissue-derived stem cells and PRP injection, which led to the resolution of symptoms and reduction of atrophy and scleroderma in 15 patients with a histological diagnosis of lichen sclerosus (LS) who had not responded to previous local steroid therapy[24]. The potential utilization of PRP in treating autoimmune skin diseases such as LS has been deliberated in the literature; however, it remains unclear whether PRP is a sufficiently effective treatment to supplant topical steroids[24].

3.4 Laser treatments

Laser therapy has been widely utilized in various medical disciplines for years. Lasers and intense pulsed light (IPL) have recently gained significant popularity in dermatology and aesthetic medicine. This method involves directing a beam of light with a specific intensity onto a targeted body area for a specific duration. The lasers used in such treatments can emit continuous beams or pulses[25]. In contrast, IPL comprises a broad spectrum of light akin to incandescent light, which can impact larger areas of the skin.

Dermatological lasers are classified into different categories based on the type of active medium responsible for generating the laser beam. Solid-state lasers utilize a crystal matrix with a metal ion component that absorbs and re-emits photons[26]. Examples include neodymium-YAG, erbium-YAG, and ruby lasers, each serving distinct purposes in skin treatments[26]. On the other hand, gas lasers replace the crystal with gas and encompass lasers such as CO2, argon, and helium-neon, each with specific applications in dermatology. Additionally, semiconductor (diode) lasers are employed for hair removal[26].

To date, no studies have been identified demonstrating a negative impact of dermatological laser therapy or IPL treatment on patients with autoimmune thyroid and connective tissue diseases[25]. Moreover, certain types of laser therapies, notably low-level laser therapies (LLLT), positively affect the treatment of autoimmune thyroid diseases. Scientific reports also indicate the favourable impact of laser and IPL treatments on patients with autoimmune connective tissue diseases such as scleroderma[26]. Pulsed dye laser and intense pulsed light are commonly used to treat morphea telangiectasia and systemic sclerosis, often requiring multiple treatment sessions[26]. Both treatments have shown objective functional improvement, with potential side effects including pain, bruising, swelling, hypopigmentation, and scarring[26].

Additionally, IPL can induce collagen formation, improving conditions such as microstomia[27]. Furthermore, fractional and fully ablative lasers are utilized to address cutaneous fibrosis associated with morphea and SSc, with immediate improvement post-treatment attributed to mechanical relaxation of atherosclerotic tissue and delayed improvement linked to tissue response and upregulation of growth factors and cytokines that modulate healing[28]. In specific cases, the CO2 laser has been observed to be superior to phototherapy for various types of

morphea, including active disease[28]. It has effectively treated scleroderma-related conditions such as heel contractures, digital calcinoses, and perioral rhythms, resulting in notable improvement[27].

3.5. Mesotherapy

Mesotherapy is a cosmetic and dermatological procedure that involves the intradermal or subcutaneous injection of active substances to stimulate the regenerative processes of the skin and subcutaneous tissue[29]. The primary goal of this treatment is to deliver active substances to the dermis to enhance collagen production and fibroblast activity while also retarding the degeneration of elastin and reducing epidermal water loss[29]. In addition to the healing properties of the injectable agents, skin puncturing itself stimulates proteins and genes involved in the wounding process, such as VEGF, β-catenin, Wnt3a, and Wnt3b[29]. Data on the safety of mesotherapy in autoimmune diseases are limited. Many substances used in mesotherapy contain various mixtures, the effects of which on the human body have not been thoroughly researched. Although no alarming reports have been identified after mesotherapy, assessing the effects of such mixtures on patients with autoimmune thyroid diseases is challenging. The literature describes the exclusive use of thyroid hormones as components of a mesotherapy cocktail due to their lipolytic activity. However, this use is controversial, as cases of thyrotoxicosis have been reported following the use of a cocktail containing triiodothyronine. There are few reports on the positive effects of using steroids administered in mesotherapy for the treatment of alopecia areata, an autoimmune disease, contrary to other authors who do not recommend mesotherapy for this condition[30]. Thyroid functions returned to normal after discontinuation of therapy[30]. One of the substances administered intradermally is poly-L-lactic acid (PLLA). This biocompatible and immunologically neutral synthetic polymer stimulates fibroblast proliferation and collagen formation, thus improving skin thickness and quality. However, the potential resorption of PLLA limits long-term results to approximately two years. PLLA has been used in patients with PRS and facial linear sclerosis, resulting in subjective improvement of aesthetic deficits[31]. Its use in scleroderma patients revealed that the initially hard, fibrotic skin made injection difficult in the first two sessions but improved in subsequent sessions, limiting the injection volume to 1 to 1.5 ml per session[31]. Furthermore, PLLA has been employed as an adjuvant treatment in combination with structural fat transfer in patients with ulcers as a skin thickening agent prior to eventual fat transfer and in the case of minor volume deficits in a combined PLLA-fat transfer-IPL treatment regimen[31].

CONCLUSION

Despite numerous studies and the ongoing advancement of aesthetic medicine, there still needs to be more information regarding the safety and efficacy of treatments for autoimmune diseases. The most extensive research focused on thyroid diseases and their correlation to various aesthetic procedures- the summary is in Table 1. Research on patients with various autoimmune conditions, including rheumatoid arthritis, ankylosing spondylitis, and systemic connective tissue diseases such as scleroderma, suggests that non-invasive or minimally invasive cosmetic dermatological procedures are generally safe and do not exacerbate systemic autoimmune diseases during periods of remission. However, certain treatments, such as mesotherapy and botox, are not recommended due to insufficient safety studies and a higher potential for side effects. Nonetheless, research has demonstrated the positive impact of mesotherapy, hyaluronic acid therapy and platelet-rich plasma therapy in patients with scleroderma, significantly improving their quality of life. It is essential for the safe use of aesthetic medicine treatments in patients with autoimmune diseases to involve a thorough patient interview and for specialists to have the necessary knowledge to select appropriate treatments for individual patients

Procedure type	**Procedural indications**	**Efficacy and safety in patients with autoimmune thyroid diseases**
Autologous platelet -rich plasma	Androgenetic alopecia, acne scars , skin enlargement (facial wrinkles), therapies combined with fractional laser (resurfacing), autologous fat graft (tissue augmentation), radio waves (skin density)	Secure; however, it may be effective decreased due to platelet dysfunction, especially at hyperthyroidism
Botulinum toxin	Correction of wrinkles, strabismus, blepharospasm, chronic migraine	Relatively safe but it may increase inflammation cascade
Hyaluronic acid	Restoration of tissue volume, facial	Relatively safe, but

	modeling, wrinkle correction, non-surgical rhinoplasty	inflammation of the nodes may occur
IPL and laser treatment	resurfacing, extramuscular wrinkles , telangiectatic nevi , rosacea, radiodermatitis , neck erythrosis , nevus of Ota , Becker's nevus, liver spots, Hyponatremia (EAH) skin-epidermal changes	Safe and efficient
Mesotherapy	Wrinkles on the face, insufficiently moisturized and nourished skin, rejuvenation, skin firming, photoaging, alopecia, cellulite, local fat deposits, discoloration and melasma , telangiectasia, vitiligo, eczema	Safety unknown, procedure not recommended

Table 2: Efficacy and safety of selected aesthetic medicine treatments in patients with autoimmune thyroid diseases.

Disclosures

Author's contribution:

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31. https://img.brainkart.com/imagebk17/j2lQzVL.jpg (TABLE 1)