

Mucin-secreting cutaneous diseases: Clinical and histopathological study in a series of 84 cases

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ABSTRACT

Background: Cutaneous mucinoses are a diverse group of skin disorders that involve the accumulation of an abnormal amount of mucin in the skin. The mucin is a jelly-like complex carbohydrate substance, called hyaluronic acid, that occurs normally in minor amounts in the dermis or mid-layer of the skin. This abnormal deposition can be localized or widespread. **Objective:** The objective was to collect the different skin diseases that introduce mucin to the skin during the course of the disease. **Patients and Methods:** This was a case series, descriptive study that was conducted during the period from 2014 to 2024 where all patients with cutaneous mucinosis were collected. Full demographic and clinical evaluation was performed. Biopsies for histopathological assessment were taken. **Results:** Eighty-four cases with cutaneous mucinosis were analyzed, with their ages ranging from 17 to 60 years, with 50 (59.5%) females and 34 (40.5%) males. The following diseases were evaluated: Granuloma annulare was observed in 50 (59.5%) cases, with ages ranging from 17 to 62 with a mean of 34 years, 42 (84%) females and 8 (16%) males; papular mucinosis in 25 (28.8%) cases, with ages ranging from 22 to 60 years, with a mean of 22 years, 19 (76%) males and 6 (24%) females; pretibial myxedema in 4 (4.8%) male patients, with ages ranging from 35 to 61, with a mean of 50 years; scleredema in 3 (3.6%) patients, with ages ranging from 36 to 45 years, with a mean of 40 years, 2 (66.66%) males and one (33.33%) female; follicular mucinosis (alopecia mucinosa) in 2 (2.3%) cases, 27-year-old female and 48-year-old male. A histopathological study of the biopsies showed obvious dermal mucin deposition using H&E stain, apart from the specific histopathology of each disease. **Conclusion:** This study showed different cutaneous diseases with mucin deposition. Some are common, such as granuloma annulare and papular mucinosis, while others are rare, like pretibial myxedema, scleroderma, and follicular mucinosis. They have diverse clinical pictures, but all create a dermal mucin deposition, with the prognosis being variable among patients.

Key words: Mucinosis, Granuloma annulare, Papular mucinosis, Scleromyxedema, Scleredema, Pretibial myxedema

INTRODUCTION

Skin mucinoses are a cluster of disorders in which abnormal amounts of mucin accumulate on the skin in a diffuse or focal pattern [1].

The fibroblasts produce mucin in small quantities as part of the dermal extracellular matrix. It is an amorphous, jelly-like compound made from acid glycosaminoglycans [2]. There are two types of acid

glycosaminoglycans: those that are interconnected, as in dermatan sulfate and chondroitin sulfate, and those that are free, as in hyaluronic acid, which is an important component of dermal mucin [3].

The water and salt equilibrium and in the dermis is maintained by mucin, which can hold water up to 1000 times its weight [4]. The presence of either a blue-staining material between collagen bundles or empty spaces within the dermis is an important indicator of

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mucin deposition in routinely stained sections [5]. Stains such as Alcian blue, toluidine blue, and colloidal iron can be used for verification [2].

Pathogenesis

Mucinosis has an unknown pathogenesis. In general, circulating cytokines, such as tumor necrosis factor-alpha, transforming growth factor beta, and interleukin-1, increase fibroblast reproduction and glycosaminoglycan production. Fibroblasts can produce more DNA when exposed to serum from patients with scleromyxedema *in vitro* [3].

Classification

There are two types of cutaneous mucinosis: primary, where mucin deposition is the major histological feature that leads to clinically unique lesions, and secondary, where mucin constitutes a secondary finding. There are two types of primary mucinosis: degenerative-inflammatory and hamartomatous-neoplastic. Degenerative-inflammatory forms are further divided into dermal and follicular forms [4].

Classification of Primary Mucinosis

- A. Degenerative-inflammatory mucinosis:
 1. Dermal
 - a. Scleromyxedema
 - b. Localized variants of lichen myxedematosus (LM):
 - Discrete papular mucinosis
 - Acral persistent papular mucinosis
 - Cutaneous mucinosis of infancy (CMI)
 - Nodular mucinosis
 - c. Self-healing cutaneous mucinosis
 - Juvenile type
 - Adult type
 - d. Scleredema
 - Non-diabetic (types I and II)
 - Diabetic (type III)
 - e. Mucinosis associated with altered thyroid function
 - Localized (pretibial) myxedema
 - Generalized myxedema
 - f. Reticular erythematous mucinosis
 - g. Papulonodular mucinosis associated with autoimmune connective tissue disease
 - h. Digital mucous cyst (myxoid cyst)
 - i. Cutaneous focal mucinosis
 - j. Miscellaneous mucinosis

2. Follicular
 - Follicular mucinosis
 - Urticaria-like follicular mucinosis
- B. Hamartomatous-neoplastic mucinosis:
 - (Angio)myxoma
 - Mucinous nevus

Classification of Secondary Mucinosis (Associated with Histological Deposition of Mucin)

1. Epithelial mucinosis
 - Basal cell carcinoma
 - *Rare*: Squamous cell carcinoma, verruca, seborrheic keratosis, keratoacanthoma, mycosis fungoides.
2. Dermal mucinosis
 - Granuloma annulare
 - Lupus erythematosus, scleroderma, and dermatomyositis
 - Epithelial tumors (e.g., basal cell carcinoma, eccrine carcinoma)
 - Mesenchymal tumors (e.g., myxosarcoma, myxoid lipoblastoma)
 - Neural tumors (e.g., neurofibroma, lobular neuro myxoma)
 - Other tumors (e.g., cutaneous metastases, mucinous carcinoma of the eyelid)
 - Hypertrophic scarring
 - Obesity-associated lymphedema
 - *Rare*: chronic graft-versus-host disease, cutaneous reactions to interferon, herpes zoster, venous insufficiency.
3. Follicular mucinosis
 - Eczematous dermatoses
 - Mycosis fungoides
 - *Rare*: lupus erythematosus, insect bites, familial reticuloendotheliosis, a side effect of imatinib.

Granuloma Annulare (GA)

An annular pattern of multiple papules characterizes this benign granulomatous skin disorder [5]. Ranges from skin color to red in most cases. Several variants of GA occur, including localized (subdivided into generalized annular plaques and disseminated papular) that affect children, perforating, and subcutaneous [5,6]. The incidence and prevalence of GA have not been assessed in large cohort studies [7]. As of yet, the etiology and pathogenesis of GA are unknown. Histologically, a delayed-type hypersensitivity reaction is most likely [8]. According to studies, interferon-gamma-producing T

helper I lymphocytes promote inflammation and tissue destruction by activating macrophages [6].

Although generalized GA may occur as an isolated cutaneous-limited disorder, numerous potential systemic conditions have been described in association, including diabetes mellitus, cancer, thyroid disease, rheumatoid arthritis, lipid abnormalities, HIV, hepatitis B, hepatitis C, BCG vaccination, and COVID-19. GA was recently reported following the COVID-19 vaccine, and it may also be triggered by insect bites, trauma, and herpes zoster [9].

In order to get an accurate diagnosis of GA, a skin biopsy is recommended for clinicopathological correlation. Among the differential diagnoses for GA are nummular eczema, psoriasis, sarcoidosis, necrobiosis lipoidica, tinea, lupus, eruptive xanthomas, leprosy, verruca vulgaris, and granulomatous mycosis fungoides [10,11]. Based on the clinical background, subcutaneous lesions should be distinguished from panniculitis, sarcoidosis, rheumatoid nodules, and infection.

In order to diagnose GA, histopathology presents distinctive features including dermal palisading granulomas with collagen degeneration in the center, mucin, and lymphohistiocytic infiltration [12]. There are four different patterns of histiocytes in GA: interstitial, palisading granulomas, nodule formation similar to sarcoidosis, and a combination of these [11]. In contrast to generalized GA, localized GA usually exhibits more prominent collagen necrosis [12]. A perforating GA differs from other variants in that collagen is expelled through the epidermis [13].

Generally, granuloma annulare does not require treatment because the patches disappear on their own within several months [10]. Sometimes, however, they persist for a long time [11]. An individual lesion may benefit from treatment, but the treatment is not curative [12]. An effective treatment includes topical corticosteroid ointment under occlusion, intralesional steroid injections, cryotherapy or laser ablation, imiquimod cream, and topical calcineurin inhibitors (tacrolimus and pimecrolimus) [13]. In widespread granulomas annulare, systemic steroids, isotretinoin, methotrexate, potassium iodide, dapsone, hydroxychloroquine, pentoxifylline, and phototherapy may be considered [12].

Lichen Myxedematosus (Lichen Myxedematosus)

This is also known as *papular mucinosis*, a rare skin disorder characterized by mucin deposits in the skin [14]. Lichen myxedematosus has localized and generalized forms [1]. Localized scleromyxedema is more favorable than generalized scleromyxedema, which can involve other organs and sometimes be fatal [3].

Lichen myxedematosus and scleromyxedema are often used interchangeably, but scleromyxedema refers to the generalized form [4].

Lichen myxedematosus has no known cause [14]. Monoclonal gammopathy, in which blood levels of an immunoglobulin called paraprotein are abnormally high, is almost always associated with scleromyxedema [15]. IgG-lambda light chain molecules are usually involved [4]. There may be a small increase in plasma cells in the bone marrow [3]. Other cases of scleromyxedema have been linked to cancers of the bone marrow, such as myeloma, lymphoma, and leukemia [1]. There have been several cases of localized lichen myxedematosus associated with HIV infection, hepatitis C infection, toxic oil exposure, and contaminated L-tryptophan [16].

Adults between the ages of 30 and 50 are often affected by scleromyxedema [1]. Both men and women are affected equally [4]. Scleromyxedema is characterized by skin-colored papules on the face, trunk, and extremities [14]. The mucous membranes and scalp are not affected [15]. The papules are 2–3 mm in diameter, waxy, and closely arranged [4]. Their color may change to red-brown over time. The brow may develop deep furrows as the condition progresses [1]. Skin stiffening, fingers (sclerodactyly), and reduced mobility of the mouth can occur, similarly to systemic sclerosis scleroderma, without telangiectasia or calcinosis [15].

Small, firm, waxy papules are confined to a few sites in the localized forms of lichen myxedematosus [15]. The distribution and course of each localized form subtype differ [14]. There is no hardening of the skin, and the disease is usually stable. In the blood, there are no abnormal levels of protein [16].

The main diagnostic test for suspected lichen myxedematosus is a skin biopsy, which shows characteristic pathological signs. Among other

tests, serum and urine protein electrophoresis for paraproteins, thyroid function, and auto-antibodies, including antinuclear factor, may also be performed [3].

It is possible to refer the patient to a hematologist and undergo a bone marrow biopsy if paraprotein is present. In patients with widespread skin involvement, a general physician may assess the involvement of internal organs [14].

There are difficulties and disappointments associated with treating scleromyxedema. Treatments have included isotretinoin, corticosteroids, methotrexate, UVA1 phototherapy, PUVA, intravenous immunoglobulin, plasmapheresis, electron beam radiation, and dermabrasion [17].

Treatments that interfere with the development of precursor cells of the bone marrow have been tried, but they are usually reserved for patients with severe and rapidly progressive diseases [17]. The most common of these therapies are: cyclophosphamide, melphalan, and chlorambucil. As a result of its ability to modulate the immune system, thalidomide has emerged as a potential treatment in recent years. Skin induration (hardening) may be reduced with topical corticosteroid creams and oral isotretinoin [18].

Pretibial Myxedema

This is a form of diffuse mucinosis characterized by the accumulation of glycosaminoglycans in the dermis and subcutis [19]. Glycosaminoglycans, also known as mucopolysaccharides, are complex carbohydrates that maintain tissue hydration and lubrication [3]. A major glycosaminoglycan in pretibial myxedema is hyaluronic acid, which is created by fibroblasts [19].

Pretibial myxedema is also known as localized myxedema, thyroid dermopathy, and infiltrative dermopathy [4]. Lower leg swelling and lumpiness are most commonly seen on the shins (pretibial areas) [20].

Up to 13% of people with severe eye disease suffer from tibial myxedema, which affects 0.5–4.3% of patients with Graves' disease. Also seen in patients with Hashimoto thyroiditis, primary hypothyroidism (underactive thyroid), and euthyroidism (normal thyroid function) [3]. TSH-R antibodies are present in high concentrations in the serum. The most common age group for this condition is between 40 and 60 years

of age. It more commonly affects females, with a female-to-male ratio of 3.5:1 [21].

Scleredema

An unknown cause of cutaneous mucinosis. There is a difference between scleredema and scleroderma, in which the skin is fibrotic (morphea and systemic sclerosis).

There is an adult population affected by scleredema [22]. Many people with scleredema have underlying systemic diseases. Diabetes mellitus, hyperparathyroidism, Sjögren's syndrome, rheumatoid arthritis, multiple myeloma, malignant insulinoma, and HIV infection are among them [23].

During a skin biopsy, mucin deposits are found between collagen bundles in the dermis, confirming the diagnosis [23]. Since scleredema is rare, there is no established treatment. PUVA, cyclophosphamide, oral corticosteroids, ciclosporin, UVA1 phototherapy, and electron beam radiation have all shown some benefits [22-24].

Alopecia Mucinosis (follicular mucinosis)

Under a microscope, follicular mucinosis is the appearance of mucin around the hair follicles [25]. Bald patches of skin with prominent hair follicles are characteristic of the condition [1]. In the dermis, mucin looks like stringy, clear, or whitish goo largely composed of hyaluronic acid. A number of other types of mucinosis are described and classified: a primary and acute condition that occurs in children and adolescents (Pinkus type), a primary and chronic condition that affects people over 40 years old, a secondary condition associated with benign or malignant skin conditions, and a rare condition called urticaria-like follicular mucinosis [26].

In the case of alopecia mucinosis, there is no known cause, but it is believed that mucinous material deposits and accumulates in hair follicles and sebaceous glands, resulting in an inflammatory condition that subsequently destroys hair follicle function [27].

Most commonly, alopecia mucinosis affects the face, neck, and scalp, but it can affect any part of the body [25]. It appears as grouped follicular papules within reddened, dry patches or plaques. The diameter of patches or plaques is usually 2–5 cm, but they can

be larger [3]. There may be one lesion at the onset, or multiple lesions may develop over time. The early stages of hair loss are non-scarring and reversible, but in more advanced stages, the hair follicles are destroyed, resulting in scarring [27].

On biopsy, alopecia mucinosa is diagnosed by its clinical appearance and histopathological findings: accumulation of mucin in sebaceous glands and pilosebaceous follicles, keratinous debris within cystic cavities, inflammation, and degeneration of follicular structures [28].

Currently, there is no proven treatment for mucinous alopecia [27]. Primary and acute alopecia mucinosis in children usually resolves spontaneously [26]. In other forms of the disease, spontaneous resolution is rare, making it difficult to assess the effect of treatment [25]. In addition to topical, intralesional, and systemic corticosteroids, oral antibiotics such as minocycline, dapsone, indomethacin, interferons, topical and systemic, photochemotherapy (PUVA), topical nitrogen mustard, radiation, UVA1 phototherapy, and topical bexarotene 1% gel have been tried with limited success [28,29]. If the underlying skin disease is cutaneous T-cell lymphoma, secondary alopecia mucinosis should be treated appropriately [29].

The aim of the present study is to examine the different skin diseases containing mucin in the skin over a ten-year period, trying to find the frequency of these diseases.

PATIENTS AND METHODS

Eighty-four patients complaining of cutaneous mucinosis gathered during the period from June 2014 to 2024 years at the outpatient dermatology unit of Baghdad Teaching Hospital, and a private clinic (KES), in Baghdad, Iraq, were involved in this case-series, descriptive, observational, clinical-histopathological study. The study was conducted following the Declaration of Helsinki. After discussing the nature of the study with all patients, informed consent forms were obtained from all. The close-up picture of the object was taken at the same place with a fixed distance and lighting in the same direction. Also, all patients included in the study accepted the idea of sharing their photos. A full epidemiological and demographic profile was recorded during the study. In order to establish the right clinical diagnosis,

a detailed history of the patient was taken along with a thorough physical examination, in addition to their name, age, sex, residence, occupation, duration of the disease, site of involvement, distribution, or number of lesions. Medical and drug history, investigations for underlying diseases such as diabetes mellitus, thyroid disease, malignancy, lipid abnormalities, associated viral infection (HIV, hepatitis B & C), and rheumatoid arthritis were done when suspected. Biopsies for histopathological assessment were taken from the patients.

RESULTS

Eighty-four patients complaining of cutaneous mucinosis were considered in the present work, with their ages ranging from 17 to 60 years, with a mean of 38 years, with 50 (59.5%) females and 34 (40.5%) males. The following diseases were evaluated: Granuloma annulare was observed in 50 (59.5%) cases, with their ages ranging from 17 to 62, with a mean of 34 years, 42 (84%) females, and 8 (16%) males. Papular mucinosis in 25 (29.8%) cases, with ages ranging from 22 to 60 years, with a mean of 22 years, 19 (76%) males and 6 (24%) females. Pretibial myxedema in 4 (4.8%) male patients, with ages ranging from 35 to 61, with a mean of 50 years. Scleredema in 3 (3.6%) patients, with ages ranging from 36 to 45 years, with a mean of 40 years, 2 (66.66%) males and one (33.33%) female. Follicular mucinosis (alopecia mucinosis) in 2 (2.3%) cases, a 27-year-old female and a 48-year-old male. A histopathological study of biopsies showed obvious dermal mucin deposition using H&E stain, apart from the specific histopathology of each disease (Tables 1 – 3) (Figs. 1 – 7).

DISCUSSION

The term *cutaneous mucinosis* refers to a group of uncommon skin disorders where mucin accumulates abnormally in the skin in all of these conditions [1]. Normally, hyaluronic acid occurs as a jelly-like substance in the dermis or mid-layer of the skin as part of the connective tissue [2]. As a result of mucinosis, abnormal deposits may appear locally or all over the body [3]. In terms of severity, they can range from minor cosmetic inconveniences to potentially severe conditions that affect the internal organs. There is no clear understanding of the underlying causes of this group of disorders [4].

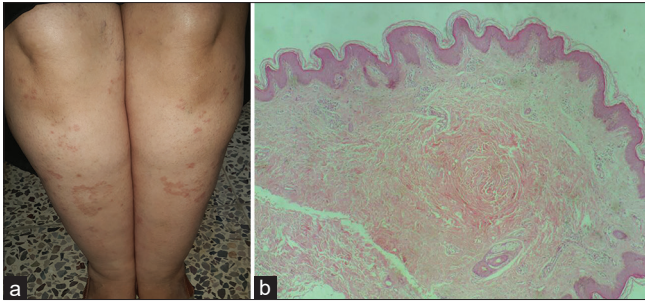


Figure 1: a) Case of granuloma annulare in a 45-year-old female. b) Case of granuloma annulare in a 25-year-old male; mainly mucin deposition and degenerative necrosis of collagen (H&E; 10x).

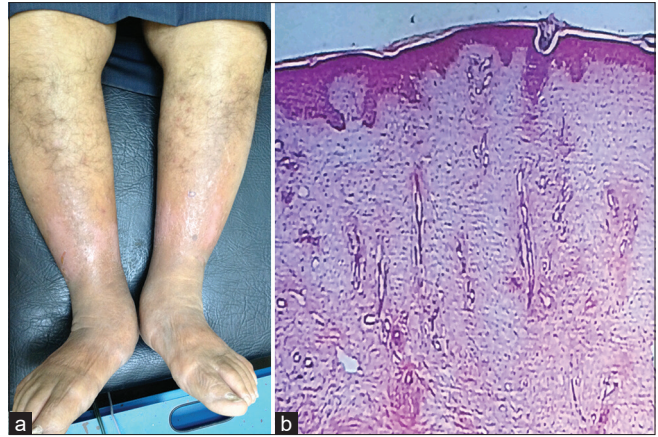


Figure 4: a) Case of pretibial myxedema on both shins. b) Histopathology of pretibial myxedema showing dermal mucinosis (H&E; 10x).

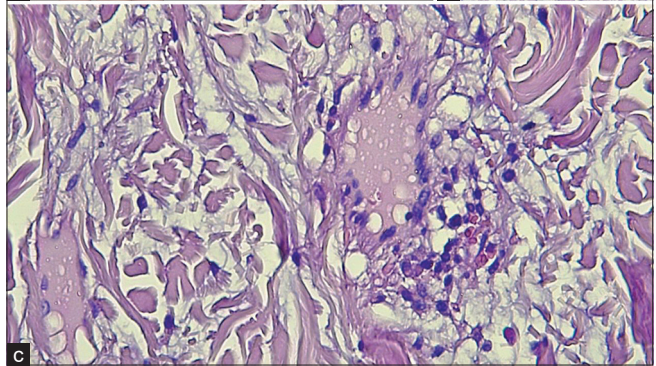
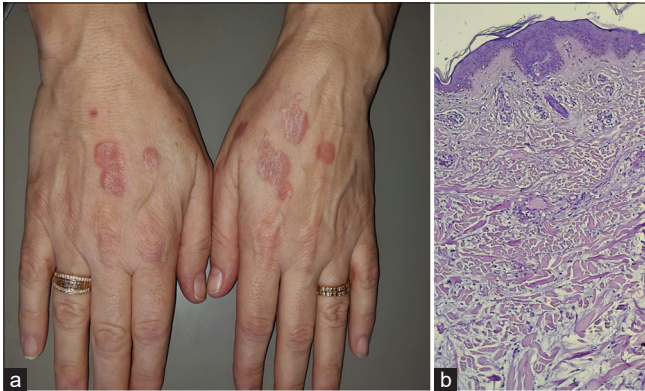


Figure 2: a) 36-year-old female with granuloma annulare. (b and c) Same patient with granuloma annulare showing a dermal interstitial granulomatous reaction with mucin deposition in addition to giant cells (H&E; 10x, 40x).

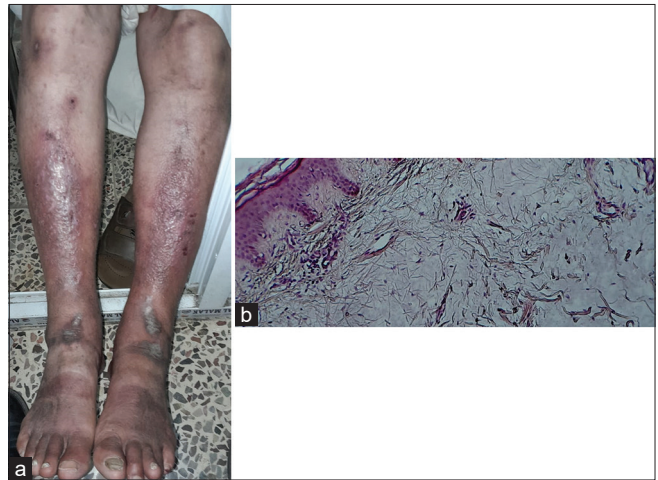


Figure 5: a) 60-year-old male patient with pretibial myxedema of the legs and feet. b) Histopathology of pretibial myxedema showing massive dermal mucin deposition (H&E; 10x).

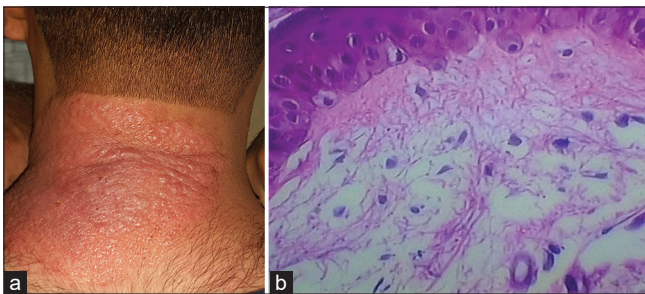


Figure 3: a) Papular mucinosis of the neck with a shiny pea-de-orange appearance. b) Papular mucinosis of the neck showing massive deposition of dermal mucin (H&E; 10x).

Table 1: Sociodemographic characteristics of the study sample (n = 84)

Characteristic	n	(%)
Age (yrs.):		
- Min.	22	
- Max.	60	
Sex:		
- Male	50	(59.5)
- Female	34	(40.5)

The present study reported 84 patients complaining of cutaneous mucinosis, ranging in age from 17 to 60 years, with a mean of 38 years, 59.5% females and 40.5% males. 59.5% of the patients exhibited granuloma annulare; the average age of the patients was 34 years, 84% were females, and 16% were males. Meanwhile, 29.8% of the patients had papular mucinosis, with a mean age of 22 years, 76% were male

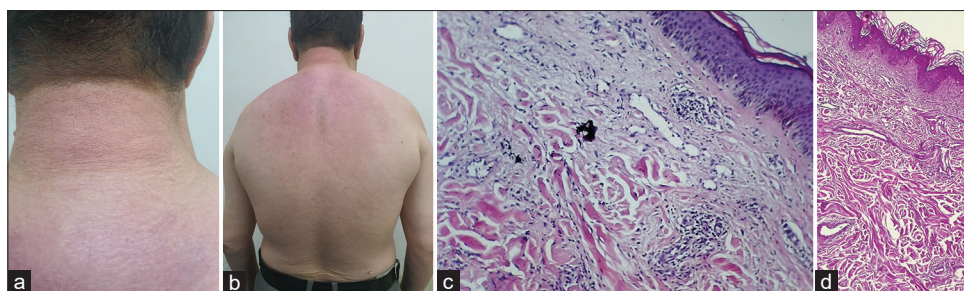


Figure 6: a) Case of scleredema of the upper back. b) Case of scleredema affecting the upper back. c) Histopathology of scleredema showing dermal mucin deposition with dermal lymphocytic infiltrate (H&E; 10x). d) Histopathology of scleredema consisting of mucin deposition with separation of collagen bundles (H&E; 10x).

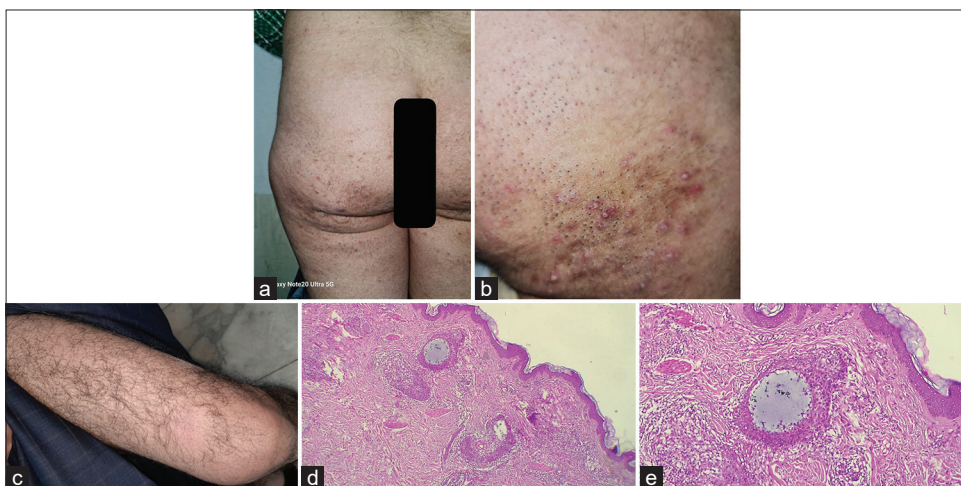


Figure 7: a) Patient with follicular mucinosis. b) Patient with follicular mucinosis. c) Patient with follicular mucinosis. d) Histopathology of follicular mucinosis showing the accumulation of mucin in hair follicles (H&E; 10x). e) Histopathology of follicular mucinosis under higher magnification (H&E; 40x).

Table 2: Disease description of the study sample (n = 84)

Characteristic	n	(%)
Disease description:		
- Granuloma annulare	50	(59.5)
- Papular mucinoses	25	(29.8)
- Pretibial myxedema	4	(4.8)
- Scleredema	3	(3.6)
- Follicular mucinosis	2	(2.3)

Table 3: Distribution of the study sample based on sociodemographic characteristics and disease description (n = 84)

Characteristic	Age (yrs.)			Sex	
	Min.	Max.	Mean	Male	Female
Disease description:					
- Granuloma annulare (n = 50)	17	62	34	8 (16%)	42 (84%)
- Papular mucinoses (n = 25)	22	60	22	19 (76%)	6 (24%)
- Pretibial myxedema (n = 4)	35	61	50	4 (100%)	0
- Scleredema (n = 3)	36	45	40	2 (66.6%)	1 (33.3%)
- Follicular mucinosis (alopecia mucinosis) (n = 2)	48	27	37.5	1 (50%)	1 (50%)

and 24% were female. An estimated 4.8% of the male patients, aged 35–61 with an average age of 50, had

pretibial myxedema. Scleredema was noticed in 3.6% of the patients, whose age ranged from 36 to 45 years, with a mean age of 40, 66.66% males and 33.33% females. There were two cases of follicular mucinosis (alopecia mucinosis) in 2.3% of the cases, a 27-year-old female and a 48-year-old male. Apart from the specific histopathology of each disease, H&E stain analysis of the biopsies showed obvious dermal mucin deposition.

In a study performed by Sharquie et al., they found that annular granuloma annulare usually presented with typical annular beaded lesions and was classified as generalized (87.23%), localized (8.51%), or profundus (4.25%). Meanwhile, some patients had only one site affected, such as the scalp and penis. The histopathological picture showed palisading granulomas. In both clinical and histopathological cases, it can mimic numerous granulomatous diseases, but sarcoidosis is the most significant mimic.

In another study conducted by Sharquie et al. [29], a total of nine patients with papular mucinosis were

reported, ranging in age from 20 to 56 years, with a mean age of 35 years, and only one 4-year-old child. There was a female predominance (70% females). The face was the most common site of involvement, but the upper arms and necks were also affected. As a result of the rash, skin-colored or red fleshy papules and plaques were observed, as well as diffuse erythematous orange peel-like forms. In most cases, the rash was asymptomatic. The pathology of the disease revealed diffuse mucin deposition in the dermis [29].

Currently, this study is considered to be the first work that examined the frequency of important cutaneous mucinoses in one collective study.

CONCLUSIONS

This study showed different cutaneous diseases with mucin deposition. Some are common, like granuloma annulare and papular mucinosis, while others are rare, like pretibial myxedema, scleroderma, and follicular mucinosis. They have diverse clinical pictures but all share one pathological feature: dermal mucin deposition. Prognosis is variable among patients depending on the variety of mucinosis.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

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