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Case report
GUTTATE MORPHEA: A CASE REPORT

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ABSTRACT

Scleroderma means hard, thick skin. The term "localized scleroderma" refers to the fact thatthis group of conditions is "localized" to the skin, with no internal organ involvement.Sometimes, the term "morphea" is used interchangeably with localized scleroderma. It is divided into several subtypes all of which transition through an early inflammatory stage followed by sclerosis and subsequent atrophy, guttate morhea is one of the rarest sub types, and it represent less than 1% of the cases worldwide. In our case we represent a 53 year-old male with guttate morphea. Two years ago the patient developed hypopigmented non pruritic papules on the back and shoulders. The clinical presentation, examination and histopathology of our patient were consistent with guttate morphea.

1. Introduction

Morphea is an inflammatory disorder that causes sclerotic changes in the skin. It is due toextensive storage of collagen, which leads to the thickening of skin layers. Morphea is distinctfrom systemic sclerosis (scleroderma) which is an autoimmune connective tissue disorder withfrequent systemic manifestations. The lesions are usually single or few in number. There is lack of consensus on the appropriate classification system for morphea. Several clinical forms are now recognized including:

Generalized morphea

Nodular (keloidal) morphea, subcutaneous morphea (morphea perfunda)

Linear morphea.

Guttate morphea (which may be a variant of lichen sclerosus et atrophicus, LSA)

The pathogenesis of morphea is poorly understood. Multiple factors including autoimmunity,

genetics, environment and vascular dysfunction may play a role in morphea.

CASE REPOET:
HISTORY

A 53 years old Philippine male patient live in Saudi Arabia since 1993, referred electively through OPD because he was complaining of skin legions since two years.

It started at the lower back as hypopigmented flat skin lesion noticed by his wife, associated with minimal itching.There were no scales and no change in color with sun exposure, not tender, there is no bleeding or discharge, not preceded by erythema, in the last two years the legion increased gradually in number and involved the back and shoulders, but no changes in the sizes or color of individual lesion.

Lesions were not associated with any systemic symptom.

History of exposure to polyvinyl chloride (PCV cement- used in sewage pipe). No history of similar presentation in the family or colleague. Patient sought medical advice one year back. Investigation were done; CXR, CBC and Chemistry was normal, CRP was high 26.7 (0.4-5). He was managed by antifungal cream with no improvement and he didn't use any other type of medication or herbal remedies. The patient is medically free. Not on any medication. No history of surgeries. He was moderate smoker but stopped 25 years back. He works as Plummer for 12 years. No recent travelling. No history of any known allergy.

PHYSICAL EXAMINATION

Generally patient looks well.

Skin: multiple discrete hypopigmented atrophic patches range from 1mm to 9mm over the back

and shoulder, no scales or erythema.

Extremities, trunk and face are not involved.

Nails: normal.

Genitalia: normal.

Scalp: normal.

Oral mucus membrane: normal.

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LABORATORY DATA

Hematology and chemistry: normal.

The antinuclear antibody (ANA) & antibodies to single-stranded DNA (ssDNA): no available

reagent.

HISTOPATHOLOGY

A 4mm Skin punch biopsy from the upper back taken, showed skin fragment with unremarkable

epidermis, the dermis shows minimal infiltration by chronic inflammatory cells.

The mid dermis and the deep dermis show thick collagen bundles arranged haphazardly.

No atypia or malignancy.

DIAGNOSIS

The patient clinical history, examination, labrotry findings and biopsy consistent with

Guttate morphea

DIFFERENTIAL DIAGNOSES

Mycosis fungoides, Dyscromiauniversalis, Post inflammatory hypopigmentation, Vitiligo,

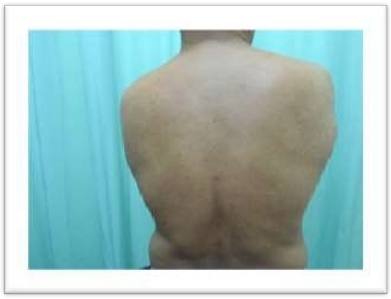
Ptryasis versicolor, Sarcoidosis, Secondary syphilis, chemically induced dermal fibrosis.

TREATMENT

The patient was treated with Mometasone furoate cream and Tacrolimus ointment.

Fig. 1, Fig. 2.: multiple, hypopigmented papules ranging from1mm to 9 mm on the shoulder and back of the patient

Fig. 3a,



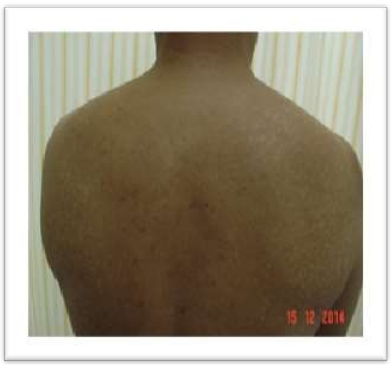
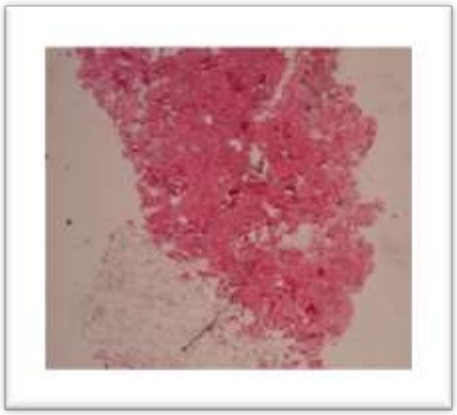
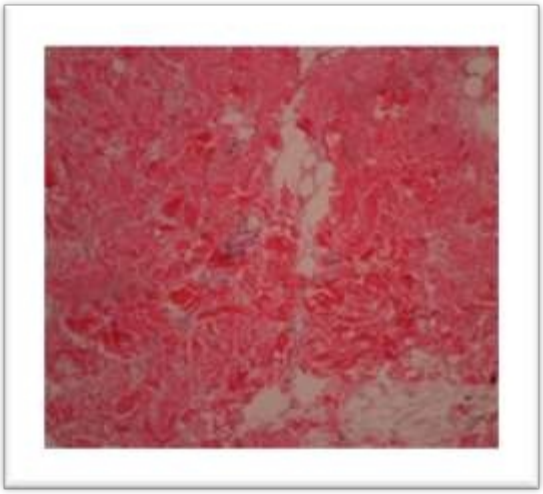
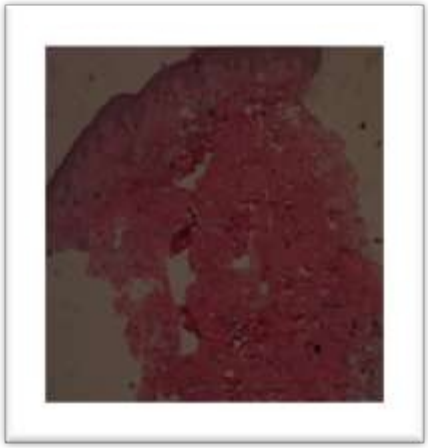


Fig. 3a, 3b & 3c: The mid dermis and the deep dermis show thick collagen bundles, crowded and arranged haphazardly.







DISCUSSION

Sclerotic lesions in the skin manifest as firm, bound-down plaques or nodules. However, in morphea, the initial sign of disease is often an inflammatory, erythematous patch or edematous plaque. Some patients may note unexplained pain or itching at the site of disease prior to the development of a

clinically evident lesion. Sclerosis usually begins in the center of inflammatory lesions, initially leaving an erythematous or violaceous border. Hypopigmentation, hyperpigmentation, alopecia secondary to loss of hair follicles, and a shiny cutaneous surface are common additional features of sclerotic lesions.

After a period of months to years, sclerotic plaques soften and transition into hypopigmented or hyperpigmented atrophic plaques .Atrophy in lesions that primarily involve the dermis may present as areas of fine, cigarette-paper like skin or shallow depressions. The depth of involvement may be superficial (primarily dermal) or deep (involving the deep dermis plus the subcutis, fascia, and/or bone).

Although morphea occurs in persons of all races, it appears to be more common in whites, who comprise 73-82% of patients seen. Females are affected about three times as often as males, including children. Morphea is classified in several types, such as patchy, generalized, linear and deep form. This classification is based on the clinical presentation and extensiveness of the pathologic processthat affects the tissue. [1] There are other different classifications depend on: the shape and amount of affected skin.

The Etiology is unknown. A variety of factors, including autoimmunity, genetics, and vascular dysfunction may play a role in morphea. Multiple environmental factors (e.g., radiation, infections, skin trauma or environmental exposures) also have been proposed as contributors to disease expression.

Multiple blood tests may be performed, which help to determine how active the disease is and how it will progress. These blood tests include: Eosinophils, immunoglobulins, and auto antibodies: antinuclear antibodies (ANA), antibodies to single-stranded DNA (ssDNA) and antihistones antibodies. These tests are not specific for localized scleroderma and may be positive in other diseases. However, specific antibodies that are common in systemic sclerosis (typically anti-centromere antibodies or anti-topoisomerase or SCL70) are expected to benegative in patients with localized scleroderma.

Antinuclear antibodies are present in approximately 20-80% of morphea patients, typically with a homogeneous, speckled, or nucleolar pattern. The prevalence is higher in patients with generalized, linear, and deep subtypes. Anti–single-stranded DNA antibodies are present in 25% of patients with plaque-type morphea, levels correlate with extensive, active disease and joint contractures. [1] In our case antinuclear antibodies and Anti–single-stranded DNA antibodies were requested however reagents were not available.

The treatment of generalized morphea is challenging. High-potency topical glucocorticoids may be applied locally. Systemic glucocorticoids, antimalarials, colchicines, and azathioprine are usually ineffective. D-penicillamine (2-5mg/kg) may halt the formation of new lesions and induce the softening of the older lesions. Oral calcitriol (0.50-0.75 mg daily) may improve skin extensibility in adult patients with generalized morphea. UVA1 (340-450nm) phototherapy may also be helpful. Finally, oral methotrexate (15-25 mg per week) may provide some benefit to a subset of patients. [2] Our patient was treated with Mometasone furoate cream and Tacrolimus ointment. As a result of this treatment the lesions slightly improved.

CONCLUSION:

In conclusion we represent a case of 53 years male patient with multiple discrete hypopigmented atrophic patches on the back and shoulder confirming the diagnosis of guttate morphea one of the rare subtypes of localized scleroderma

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