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A CASE STUDY ON RETINOID INDUCED DARIER'S DISEASE

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ABSTRACT

Darier's disease is a rare cutaneous disease with an autosomal dominant mode of inheritance. Greasy papules and plaques arise on the seborrheic areas and in the flexures and almost all patients have nail abnormalities. Darier's disease is an example of a dominantly inherited disease caused by haplo-insufficiency. Oral retinoids are the most effective treatment but their adverse effects are troublesome. Topical retinoids, topical corticosteroids, surgery, and laser surgery have their advocates but evidence for efficacy is sparse. Pre-therapy investigations of cell-mediated immunity showed no severe immunological dysfunction, although high responses to supra-optimal Con A concentrations suggested abnormalities in immunoregulatory lymphocyte subpopulations. In addition, patients showed enhanced LMIF production upon stimulation with Con A, PWM and PPD. Retinoid therapy decreased the number of peripheral blood total leukocytes, lymphocytes and T-cells, normalized the LMIF production, and decreased the lymphocyte responses to mitogens.

Key words: Darier's Disease, Dermal Infections, Retinoid, Adverse Drug Reaction.

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INTRODUCTION

Darier's disease is a rare cutaneous disease with an autosomal dominant mode of inheritance. Greasy papules and plaques arise on the seborrheic areas and in the flexures and almost all patients have nail abnormalities. Acantholysis and dyskeratosis are the typical histological findings[1]. The underlying defect is a result of mutations in the ATP2A2 gene on chromosome 12q23-24 that encodes for a sarco/endoplasmic reticulum calcium ATPase (SERCA 2).Acantholysis is thought to result from desmosome breakdown. Darier's disease is an example of a dominantly inherited disease caused by haplo-insufficiency. Oral retinoids are the most effective treatment but their adverse effects are troublesome. Topical retinoids, topical corticosteroids, surgery, and laser surgery have their advocates but evidence for efficacy is sparse [2, 3].

CASE REPORT:

Female patient aged 28 years and had history of joint pains on and off oral lesions present since 16 yrs. Patient had complaints on darreris disease due to T. isotretinoin 20mg once daily and also complained flare up of disease since 3 months, rough yellowish colored raised lesions all over body, scalp and face more at forearm and legs with fissure and oozing of blood and water discharge since 2months itchy all over body more at groins since months.

Patient also had lymphocyte responsiveness to phytohaemagglutinin (PHA), concanavalin A (Con A), pokeweed mitogen (PWM) and purified protein derivative of tuberculin (PPD), leukocyte migration inhibitory factor (LMIF) production and suppressor cell activities were studied before and during oral etretinate treatment. Pre-

therapy investigations of cell-mediated immunity showed no severe immunological dysfunction, although high responses to supra-optimal Con A concentrations suggested abnormalities in immunoregulatory lymphocyte subpopulations. In addition, patients showed enhanced LMIF production upon stimulation with Con A, PWM and PPD. Retinoid therapy decreased the number of peripheral blood total leukocytes, lymphocytes and T-cells, normalized the LMIF production, and decreased the lymphocyte responses to mitogens.

Clinical Findings:

GENITALS:

Watery lesions present over groin and pubis

Right Axillary Lymedenopathy +

SYSTEM	Day 1-DAY15
CVS	S1S2+
CNS	NORMAL
R.S	B/LAE+
P/A	SOFT

CUTANEOUS EXAM:

Greasy yellowish brown papules and diffuse hyperkeratotic plaques with scaling + over trunk, UL and LL, groins palms, hair normal oral cavity-fissured tongue+, nails-longitudinal ridged white plaque + over tongue, cobble stone over palate appearance.

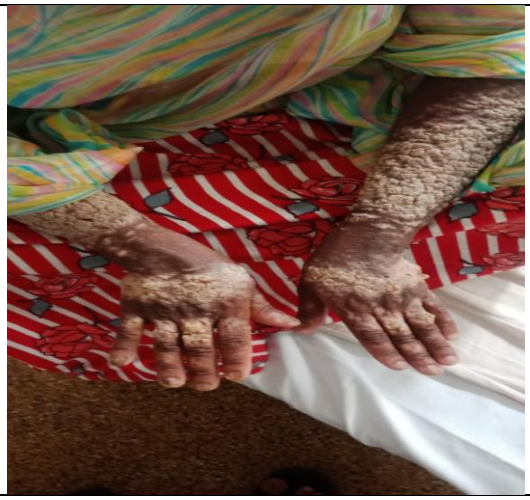
DRUG – DRUG INTERACTION:

On Day 8 patient had complaints of white discharge po curdy associated with itching. Complains of watery loose stools occurred on Day 10 due to intake of Amoxyclav and loose stools are subsided after stoppage of Amoxyclav drug.

LAB INVESTIGATION REPORT

PARAMETER	LAB VALUES	REFERENCE RANGE
HB	9g/dl	12-14g/dl
WBC	4900cumm	4,500-11,000cumm
PLT	1 lakhs/cumm	1.5lakhs-4.5lakhs/cumm
S.Creatinine	0.6mg/dl	0.8-1.2mg/dl
SGOT	132IU/L	5-40 IU/L
SGPT	19 IU/L	7-56 IU/L

Figure 1 & 2: Adverse reaction of Isotretinoin



DISCUSSION:

The use of the Retinoids, in particular Isotretinoin (Roaccutane), in the management of severe recalcitrant acne and other seborrhoeic skin conditions has to some extent revolutionised their management, producing rapid clearing and prolonged periods of remission.' One course is often enough to achieve a satisfactory disease free state. Residual facial scarring, however, can be a problem and for this reason, patients may present to the plastic surgery department for facial dermabrasion [4]. It should be remembered that a significant number of these patients may be on concurrent Isotretinoin therapy, or may have recently finished a course of treatment [5,6].

Various reporters have encountered undesirable complications following dermabrasion of patients either on, or having recently finished, a course of treatment [7]. The main problems identified with dermabrading such patients are those of delayed wound healing and, more importantly, atypical keloid scarring appearing two to four months after the initial dermabrasion. Interestingly, the keloid scars occur in unusual sites, for example the cheeks and forehead [8].

DRUG CHART:

DRUGS	DOSE	ROA	FREQ	DURATION
T.B COMPLEX		PO	BD	D1-D15
T.IFA		PO	OD	D1-D15
T.PCT	500mg	PO	TID	D1-D15
T.CETRIZINE	10mg	PO	BD	D1-D15
TAB AMOXYCLAV	625mg	PO	TID	D2-D10
T.FLUCONAZOLE	200mg	PO	OD	D7-D15
T. ISOTRETIONIN	20mg	PO	OD	D3-D15
INJ METROGYL	500mg	IV	TID	D10-D15
INJ CIFRAN	500mg	IV	BD	D10-D15
INJ PANTOP	40mg	IV	BD	D1-D15
INJ ONDANSETRON	8mg	IV	SOS	D10-D15
T.RACECADOTRIL	100mg	PO	BD	D12-D15
T.SPOROLAC		PO	TID	D12-D15
CAP A &D		PO	OD	D1-D15
CLOBETASOL		TOPICAL		D2-D15
MICANOZOLE CREAM		TOPICAL		D1-D15
EMOLLIENT		TOPICAL		D1-D15
ORS SACHETS		PO		D12
CLOTRIMOXAZOLE CREAM		TOPICAL		D10-D15

Isotretinoin and its derivatives have been shown to have diverse effects on the metabolic activity of the skin and, in particular, on fibroblast activity. Its potency as a drug against acne lies in its ability to depress the activity of the pilo-sebaceous unit dramatically. It has been postulated that this may be an important factor in delayed wound healing - the pilo-sebaceous unit being important in the reepithelialisation process. Isotretinoin has also been shown to suppress collagenase activity in keloid fibroblast cultures, a fact which has led to the speculation that this may be a mechanism by which keloid formation is promoted, or, at least, not inhibited [9].

It is difficult to explain the abnormal scar sites, however, it is possible that in altering the biochemical and physiological nature of the skin, its mechanical properties are also affected and this interaction may contribute to abnormal scar formation [10].

CONCLUSION:

Medical experts advocate varying time intervals between stopping Isotretinoin treatment and commencing dermabrasion. Some have advocated a wait of as long as one to two years before undertaking such treatment.' Though this may appear to be a long time, it should be remembered that the effects of Isotretinoin are long lasting and it would seem prudent to wait for a period of at least three to six months before undertaking dermabrasion in a patient who has been on Isotretinoin.

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