

# Dermatology International

## Editor-in-Chief

**Sidharth Sonthalia**

**MD, DNB, MNAMS, FISD**, Medical Director & Consultant Dermatologist,  
Skinnocence: The Skin Clinic & Research Center, Gurgaon, INDIA.

## National Editorial Advisory Board

### **Abhijeet Kumar Jha**

Assistant Professor, Dept. of Dermatology, AIIMS,  
Patna, Bihar, INDIA.

### **Ananta Khurana**

Assistant Professor, Dr RML Hospital & PGIMER,  
New Delhi, INDIA.

### **B.B. Mahajan**

Professor and Head, Dept. of Dermatology,  
Government Medical College, Amritsar, Punjab,  
INDIA.

### **Krina Bharat Patel**

Associate Professor & Head, Department of  
Dermatology, GMERS Medical College & Hospital,  
Ahmedabad, Gujarat, INDIA.

### **P. Nirmaladevi**

Professor and HOD of Dermatology, Tirunelveli  
Medical College, Tirunelveli, INDIA.

### **Pragya A. Nair**

Professor, Department of Dermatology and  
Venereology, Pramukhswami Medical College,  
Karamsad, Anand, Gujarat, INDIA.

### **R.K. Chittoria**

Additional Professor & Head, Dept of Plastic  
Surgery, JIPMER, Pondicherry, INDIA.

## Managing Editor

A. Lal

*All right reserved.* The views and opinions expressed are of the authors and not of the **Dermatology International**. **Dermatology International** does not guarantee directly or indirectly the quality or efficacy of any product or service featured in the advertisement in the journal, which are purely commercial.

### **Rajesh Sinha**

Professor & Head, Department of Dermatology, STD  
& Leprosy, AIIMS, Phulwarisarif, Patna, Bihar,  
INDIA.

### **Raksha M. Patel**

Professor and Head, Dept of Dermatology, STI and  
Leprosy, Gujarat Medical Education and Research  
Society (GMERS) Medical College and General  
Hospital, Vadodara, Gujarat, INDIA.

### **Sanjeev Gupta**

Professor, Department of Dermatology,  
MM Medical College, Ambala, Punjab, INDIA.

### **Satyadarshi Patnaik**

Professor & Head, Department of Dermatology  
MKCG Medical College, Berhampur, Odisha,  
INDIA.

### **Shyamanta Barua**

Assistant Professor, Department of Dermatology,  
Assam Medical College & Hospital, Dibrugarh,  
Assam, INDIA.

### **Supriya R. Vikhe**

Assistant Professor, Department of Dermatology,  
Padmashri Dr Vitthalrao Vikhe Patil  
Foundation's Medical College & Hospital,  
Ahmednagar, Maharashtra, INDIA.

## Publication Editor

Manoj Kumar Singh

### **Corresponding address**

Red Flower Publication Pvt. Ltd. 48/41-42  
DSIDC, Pocket-II, Mayur Vihar Phase-I Delhi -  
110 091(India)  
Phone: 91-11-22754205/45796900  
Fax: 91-11-22754205  
E-mail: info@rfppl.co.in  
Web:www.rfppl.co.in

**Dermatology International (DI)** is dedicated to the clinical and continuing education needs of the entire dermatologic community. Original, peer-reviewed articles cover clinical and investigative studies, treatments, new diagnostic techniques, and other topics relating to the prevention, diagnosis, and treatment of disorders of the skin. The article categories within the journal are: cutaneous biology; clinical and laboratory investigations; contact dermatitis & allergy; dermatological surgery & lasers; dermatopathology; epidemiology & health services research; paediatric dermatology; photobiology; and therapeutics.

#### **Subscription Information**

**India:** Institutional (1 year): INR5000

**Rest of the World:** Institutional (1 year) USD357

#### *Payment methods*

*Bank draft / cashier & order / check / cheque / demand draft / money order* should be in the name of **Red Flower Publication Pvt. Ltd.** payable at **Delhi**.

*International Bank transfer / bank wire / electronic funds transfer / money remittance / money wire / telegraphic transfer / telex*

1. **Complete Bank Account No.** 604320110000467
2. **Beneficiary Name (As per Bank Pass Book):** Red Flower Publication Pvt. Ltd.
3. **Address:** 41/48, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091(India)
4. **Bank & Branch Name:** Bank of India; Mayur Vihar
5. **Bank Address & Phone Number:** 13/14, Sri Balaji Shop, Pocket II, Mayur Vihar Phase- I, New Delhi - 110091 (India); Tel: 22750372, 22753401. **Email:** mayurvihar.newdelhi@bankofindia.co.in
6. **MICR Code:** 110013045
7. **Branch Code:** 6043
8. **IFSC Code:** BKID0006043 (used for RTGS and NEFT transactions)
9. **Swift Code:** BKIDINBBDS
10. **Beneficiary Contact No. & E-mail ID:** 91-11-22754205, 45796900, E-mail: sales@rfppl.co.in

**Online** You can now renew online using our RFPPL renewal website. Visit <http://rfppl.co.in/subscribe.php?mid=7> and enter the required information and then you will be able to pay online.

---

Send all Orders to: **Red Flower Publication Pvt. Ltd.**, 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091(India). Phone: 91-11-22754205, 45796900, Fax: 91-11-22754205, E-mail: sales@rfppl.co.in, Website: [www.rfppl.co.in](http://www.rfppl.co.in)

# Dermatology International

January - June 2017

Volume 2, Number 1

## Contents

---

---

### Original Articles

<b>Mast Cell Profile and IL-4 Levels in Borderline Spectrum of Leprosy</b> Singh Jasmeet, Gautam R.K., Khurana Ananta, Bhardwaj Minakshi, Meena Neha, Bajaj Sonali	5
<b>Epidemiological Study of Various Dermatological Conditions in Tribal Area of Valsad, India</b> Bhuvnesh Shah, Keya Sheth Shah	11
<b>RF Assisted Management of Keloids</b> Chittoria R.K., Elankumar S., Kumaran M.S., Sudhanva H.K., Preethitha B., Friji M.T., Mohapatra D.P., Dineshkumar S.	15
<b>A Clinical Study of Vitiligo at a Tertiary Care Centre of East India</b> Rajesh Sinha, Abhijeet Kumar Jha, Smita Prasad, Shweta Dipti	17
<b>Application of Low Level Laser in Management of Biofilm</b> Vinayak C., Chittoria R.K., Sudhanva H.K., Preethitha B., Kumaran M.S., Elankumar S., Sireesha K.R.	23

### Case Reports

<b>Non Responder of Alopecia Totalis to Diphencyprone Responding to Addition of Betamethasone Pulse</b> Dhurat R., Pund P., Sukesh M.S., Dandale A., Ghate S., Bhandari K.	27
<b>Anaesthetic Management of a Patient with Known Latex Allergy and Hypersensitivity to Multiple Drugs</b> Talib Khan, Rayees Najib, Arvin Preet, Zulfikar Ali, Syed Amer Zahoor, Shaql Qamar Wani, Iram Ali	31
<b>Radiofrequency Assisted Management of Trap Door Scar</b> Babu P., Chittoria R.K., Kumaran M.S., Sudhanva H.K., Kumar E., Reddy K.S., Chavan V., Friji M.T., Mohapatra D.P., Dineshkumar S.	37
<b>Guidelines for Authors</b>	41

**Subscription Information****Institutional** (1 year) INR5000/USD357**Here is payment instruction for your reference.****Check:**

Please send the US dollar check from outside India and INR check from India made:  
Payable to 'Red Flower Publication Private Limited'.  
Drawn on Delhi branch

**PayPal Instructions for the payment (only for transfer from outside India):**

Payments can be made through our PayPal account at <https://www.paypal.com>.  
Our PayPal recipient email address is [redflowerppl@gmail.com](mailto:redflowerppl@gmail.com).

**Credit Card:**

We accept Visa or MasterCard.

**Wire transfer:**

Complete Bank Account No. 604320110000467  
Beneficiary Name: Red Flower Publication Pvt. Ltd.  
Bank & Branch Name: Bank of India; Mayur Vihar  
MICR Code: 110013045  
Branch Code: 6043  
IFSC Code: BKID0006043 (used for RTGS and NEFT transactions)  
Swift Code: BKIDINBBDS

**\*\*Please kindly add bank charge at your side if you pay by check or wire transfer.**

**Payment, orders and all correspondences should be sent to:**

Red Flower Publication Pvt. Ltd.  
48/41-42, DSIDC, Pocket-II  
Mayur Vihar Phase-I  
Delhi - 110 091(India)

## Mast Cell Profile and IL-4 Levels in Borderline Spectrum of Leprosy

**Singh Jasmeet\*, Gautam R.K.\*\*, Khurana Ananta\*\*\*, Bhardwaj Minakshi\*\*\*\*, Meena Neha\*\*\*\*\*, Bajaj Sonali\*\*\*\*\***

### Abstract

**Author Affiliation:**

\*Resident \*\*Professor

\*\*\*Assistant Professor

\*\*\*\*Senior Resident,

Department of Dermatology,  
Venereology and Leprosy,  
\*\*\*\*Professor, Department of  
Pathology, Post Graduate  
Institute of Medical Education  
and Research and Dr. Ram  
Lohia Hospital, New Delhi  
110001, India.

**Reprint Request:**

Ananta Khurana, 256, Nimri  
Colony, Phase 4, Ashok Vihar,  
Delhi 110052.

E-mail:

drananta2014@gmail.com

Received on: 17.04.2017

Accepted on: 09.05.2017

**Introduction:** Mast cells are responsible for secreting cytokines and other chemical mediators, involved in immuno-inflammatory responses. However, their role has not been clearly defined in the immunopathogenesis of leprosy. **Aims and Objectives:** To count the number of mast cells in leprosy granulomas and to correlate their numbers with the serum levels of IL-4 in borderline spectrum of leprosy. **Materials and Methods:** Thirty cases of newly diagnosed, untreated patients of borderline tuberculoid (BT), borderline borderline (BB) and borderline lepromatous (BL) leprosy were included in the study. Skin biopsies were taken from the margins of the skin lesion of leprosy and stained with toluidine blue for quantification of mast cells. An average number of mast cells per granuloma was calculated after examining the granulomas in each skin biopsy. Concentration of serum IL-4 was measured quantitatively in the collected venous blood samples by ELISA method. **Results:** The mean of average number of mast cells increased from BT towards BL leprosy. Statistically significant difference was seen between BL and BT/BB leprosy. However, no significant difference was found between BB and BT Hansen's. A positive correlation was found between mast cell numbers and serum levels of IL-4 since both showed an increasing trend from BT through BL leprosy. **Conclusion:** We found a progressive rise in mast cell count and serum levels of IL-4 across the borderline spectrum of leprosy. This indicates that mast cells play a role in progression and dissemination of leprosy by proliferating at the site of inflammation and secreting an array of chemical mediators especially IL-4.

**Keywords:** Borderline Leprosy; Mast Cells; Granuloma; Interleukin 4; Tuberculoid; Lepromatous.

### Introduction

Leprosy is a chronic inflammatory disease caused by *Mycobacterium leprae*, an obligate intracellular pathogen principally affecting peripheral nerves and skin [1].

It is diagnosed by the presence of one or more of the three cardinal signs of leprosy which include; definite loss of sensation in a hypopigmented or reddish skin patch, a thickened peripheral nerve, with loss of sensation and/ or weakness of the muscles supplied by that nerve and presence of acid-fast bacilli in slit skin smear [2]. T-lymphocytes are a major source of cytokines which play a key role in immunologic, inflammatory and reparative host responses in leprosy. Further, few previous studies suggest role of mast cells in the pathogenesis and progression of leprosy [3-8].

Mast cells are derived from the myeloid stem cells and are extensively distributed in the skin, gastrointestinal tract, upper and lower respiratory tracts. They are involved in allergic inflammation and immuno-inflammatory responses [9]. Mast cells are seen in small numbers in the granulomas of leprosy and are responsible for secreting cytokines, mainly tumour necrosis factor-  $\alpha$  (TNF- $\alpha$ ) and interleukin-4 (IL-4), and other mediators (tryptase, histamine, thromboxane, prostaglandin D2 etc) [10]. The close proximity of mast cells to the peripheral nerve fibres in the tissues alongwith the shortening of the distance between mast cells and nerve fibres during inflammatory events, suggest a functional interaction between these two components [11,12]. In the present study, we attempted to count the number of mast cells in the leprosy granulomas in borderline spectrum of leprosy and correlate it with serum levels of IL-4.

## Materials and Methods

Thirty newly diagnosed, untreated patients of borderline leprosy including tuberculoid (BT), mid-borderline (BB) and borderline lepromatous (BL) as per the classification of Ridley and Jopling [13], attending the urban leprosy centre of our institute from November 2013 to February 2015 were included in the study. The study was approved by institutional ethics committee. Patients in lepra reactions, pregnant and lactating women, and patients already receiving or those who had received specific treatment for leprosy in the past were excluded. Equal number of consenting healthy, age and sex-matched volunteers were taken as controls. Informed and bilingual consent was taken from the patients and controls before inclusion in the study. A skin biopsy was taken from the margin of a representative skin lesion from the patients. The specimens were placed in 10% formalin solution and sent to the pathology department for haematoxylin and eosin (H & E) staining for diagnosis of leprosy and toluidine blue staining for quantification of mast cells. Mast cells were visualized as violet to reddish purple cells against a blue background [Figure 1]. Mast cells were counted under  $400 \times$  magnification in ten sequentially observed granulomas, and an average number of mast cells per granuloma was calculated for each case. The mean of these values for subset cases of BT, BB and BL leprosy were also determined.

Ten ml venous blood samples of patients and controls were collected in plain vacutainer tubes and allowed to stand for 30 minutes at room temperature and then centrifuged at 300 g for 5 minutes. Sera were immediately separated and stored at  $-20^{\circ}\text{C}$  until the time of analysis for the cytokine levels. Concentration of serum IL-4 was measured quantitatively in the collected serum samples by the sandwich enzyme-linked immunosorbent assay (ELISA) method (Human IL-4 ELISA kit from Krishgen BioSystems).

### Statistical Analysis

Quantitative data were presented as mean  $\pm$  SD or median and interquartile range, as appropriate.

Normality of data was checked by measures of Kolmogorov Smirnov tests of normality. For normally distributed data means of 3 groups were compared using One-Way ANOVA followed by Post Hoc Multiple Comparisons test. For skewed data Kruskal-Wallis test followed by Mann-Whitney test for two groups was applied. For categorical variables; number & percentages were calculated. Chi-square test or Fisher's exact (whichever appropriate) test was applied for categorical data. Spearman's correlation coefficient was applied to see relationship between different variables. All calculations were two-sided & were performed using SPSS version 17 (Statistical Packages for the Social Sciences, Chicago, IL). A "p" value of  $<0.05$  was considered to indicate statistical significance.

## Results

The study group comprised of 12 patients of BT, 8 patients of BB and 10 patients of BL leprosy. There were 21 (70%) males and 9 (30%) females. The mean age was  $32.51 \pm 1.78$  years among cases and  $31.17 \pm 11.74$  years among the controls. The highest number of mast cells was found in BL leprosy (mean of  $9.45 \pm 2.26$  cells per granuloma), intermediate in BB ( $3.50 \pm 1.07$  cells per granuloma) and lowest in BT patients (mean of  $2.81 \pm 0.94$  cells per granuloma) [Figure 2].

On statistical evaluation [Table 1], significant difference was seen when the mean of average number of mast cells in BL was compared with those in BT and BB; however, the difference between BB and BT leprosy was statistically insignificant.

The mean serum levels of IL-4 were highest in patients with BL ( $76.29 \pm 37.25$  pg/ml) leprosy with intermediate levels in BB patients ( $22.99 \pm 12.93$  pg/ml) and lowest in patients with BT disease ( $4.12 \pm 2.05$  pg/ml), as shown in Table 2. On comparing mean serum levels of IL-4 of the three subsets of patients, a statistically significant difference was seen across the entire borderline spectrum (BT vs. BB,  $p = 0.001$ ; BB vs. BL,  $p = 0.003$ ; and BL vs. BT,  $p = 0.000$ ). Further, statistically significant difference was seen

**Table 1:** Comparison of mean of average number of mast cells between different groups of leprosy

Group	Mean $\pm$ SD	p value
BT vs. BB	$2.81 \pm 0.94$ vs. $3.50 \pm 1.07$	0.634
BL vs. BB	$9.45 \pm 2.26$ vs. $3.50 \pm 1.07$	0.038
BL vs. BT	$9.45 \pm 2.26$ vs. $2.81 \pm 0.94$	0.007

BB – mid-borderline; BL – borderline lepromatous; BT – borderline tuberculoid; SD – standard deviation

**Table 2:** Comparison of serum levels of IL-4 between different groups of leprosy and healthy controls

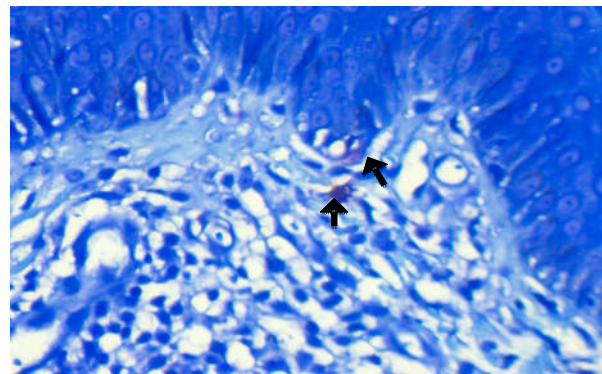
Group	Serum IL-4 levels: Mean $\pm$ SD (pg/ml)	P value
BT vs. BB	4.12 $\pm$ 2.05 vs. 22.99 $\pm$ 12.93	0.001
BL vs. BB	76.29 $\pm$ 37.25 vs. 22.99 $\pm$ 12.93	0.003
BL vs. BT	76.29 $\pm$ 37.25 vs. 4.12 $\pm$ 2.05	0.000
BT vs. healthy controls	4.12 $\pm$ 2.05 vs. 4.48 $\pm$ 2.82	0.889
BB vs. healthy controls	22.99 $\pm$ 12.93 vs. 4.48 $\pm$ 2.82	0.000
BL vs. healthy controls	76.29 $\pm$ 37.25 vs. 4.48 $\pm$ 2.82	0.000

BB – mid-borderline; BL – borderline lepromatous; BT – borderline tuberculoid; IL – interleukin; SD – standard deviation

**Table 3:** Comparison of the findings of different published studies on mast cell count in subsets of leprosy

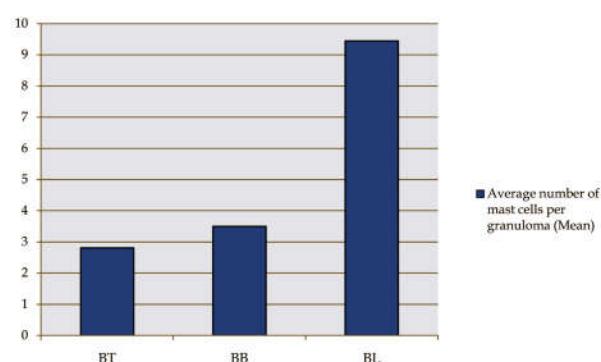
Author(s)	Number of Patients (n), Controls, and profile of patients	Mast cell staining technique	Relative mast cell density
Current study	n=30 BT-12, BB-8, BL-10	Toluidine blue	Progressive increase along the spectrum from BT to BL
Rav <i>et al</i> <sup>3</sup>	n=250 TT-50, BT-22, BB-12, BL-20, LL-36, IL-110	Toluidine blue	Progressive rise from TT to LL
Aroni <i>et al</i> <sup>4</sup>	n=28 TT-2, BT-7, BL-4, LL-11, HL-4	Chloroacetate esterase	Lower in tuberculoid than lepromatous group
Mysorekar <i>et al</i> <sup>5</sup>	n=118; controls=20 IL-25, TT-21, BT-20, BB-14, BL-20, LL-18	Toluidine blue	Progressive increase from TT to LL
Bagwan <i>et al</i> <sup>6</sup>	n=53; controls=7 IL-6, TT-9, BT-24, BB-1, BL-6, LL-7	Toluidine blue	Progressive increase from TT to LL
Sherif ES <sup>7</sup>	n=60 IL-1, TT-22, BT-13, BB-13, BL-5, LL-6	Giemsa	Progressive increase from TT to LL
Magalhaes <i>et al</i> <sup>8</sup>	n=51 BT-17, BB-17, BL+LL-17	Anti-trypsin antibody	BB and BT had higher density than BL+LL
Naik <i>et al</i> <sup>14</sup>	n=60	Toluidine blue	Progressive increase from IL to both polar TT and LL
Chatura <i>et al</i> <sup>18</sup>	n=50 TT-5, BT-24, BB-3, BL-10, LL-8	Fite-Faraco	Highest counts in BT and BL vs. their polar counterparts
Joshi <i>et al</i> <sup>19</sup>	n=37 IL-6, TT-3, BT-18, BB-2, BL-3, LL-5	Toluidine blue	BT>BL

BB – mid-borderline; BL – borderline lepromatous; BT – borderline tuberculoid; HL – histoid leprosy; IL – indeterminate leprosy; LL – lepromatous leprosy; TT – polar tuberculoid (leprosy)



**Fig. 1:** Mast cells on toluidine blue staining, highlighted by black arrows (400  $\times$ )

between mean serum levels of IL-4 of healthy controls and patients with BB and BL leprosy ( $p<0.05$ ); but not with the BT patients ( $p=0.889$ ). In our study, a trend of progressive increase in number of mast cells as well as serum levels of IL-4 was seen from BT to BL leprosy. On statistical evaluation, a positive correlation was found between mast cell count and



**Fig. 2:** Mean of average number of mast cells in each subset of leprosy

mean serum levels of IL-4 ( $r=0.664$ ,  $p<0.05$ ).

## Discussion

Mast cells are 'dynamic cells' with a central role

in allergic inflammation, protective immune response and other inflammatory responses. They are found at all strata of the skin including the dermis, around blood vessels, nerves, appendages, at dermoepidermal junction (DEJ) and also in subcutaneous tissue [9,14,15]. Mast cells can be stimulated to degranulate by direct injury, chemical agents like opioids and alcohol, certain antibiotics such as polymyxins or by cross-linking of immunoglobulin E (IgE) receptors and complement proteins [16]. Mast cells also produce IL-4, which induces naïve CD4+ T cells to differentiate into Th<sub>2</sub> cells while suppressing the development of Th<sub>1</sub> cells. They also suppress macrophage cytotoxic activity, parasite killing, and macrophage-derived nitric oxide production [17].

In this study, we found a difference in the number of mast cells in granulomas of different subsets of leprosy, with maximum number of mast cells in BL patients, lesser in BB cases and lowest in BT cases. Our findings are in agreement with findings of the study by Rav *et al* in 1990 who included 250 patients of leprosy and reported a decrease in mast cell density from lepromatous to the tuberculoid end of the spectrum [3]. They additionally reported mast cells in 100% cases of indeterminate leprosy, thereby suggesting the role of mast cells in evolution and progression of the disease [3]. Aroni *et al* in 1993 also reported a significantly lower mast cell count in tuberculoid group than the lepromatous group ( $p<0.001$ ) [4]. They suggested that the larger number of mast cells in lepromatous group may be due to increased vascularity and changes in the endothelial cells. Mast cell mediators are also known to be mitogenic for fibroblasts and endothelial cells. Thus, mast cells may also have a role in fibroblast proliferation that occurs after a lepra reaction [4]. The above findings were corroborated by Mysorekar *et al* in 2001, Bagwan *et al* in 2004, and Sherif ES in 2008 [5-7].

However, many workers have reported contradictory findings. Magalhaes *et al* found maximum density of mast cells in BB patients (73.64/ $\text{mm}^2$ ) followed by BT (65.06/ $\text{mm}^2$ ) and BL (50.43/ $\text{mm}^2$ ) [8]. Further, Naik *et al* in 2003 reported lower density of mast cells in BT (37/ $\text{mm}^2$ ) and BL (46/ $\text{mm}^2$ ) leprosy than their polar counterparts (TT-51/ $\text{mm}^2$ , LL-49/ $\text{mm}^2$ ) [14]. They suggested that periodic follow up of indeterminate and borderline lesions for mast cells might help in predicting the stability of lesions [14]. Similarly, Chatura *et al* in 2012 reported a progressive decrease in mast cell density along the borderline spectrum of leprosy [18]. Recently, Joshi *et al* and reported higher number of mast cells in BT

leprosy than in BL leprosy (BT-105.56/ $\text{mm}^2$ , BL-100/ $\text{mm}^2$ ) [19]. The reason for contradictory findings of these studies and the current study may be explained on the basis of the assertion put forward by Magalhaes *et al*. They suggested that such discrepancies might stem from the difference in the mast cell staining methods and mast cell counting technique adopted in different studies [8]. A summary of these studies has been tabulated [Table 3].

We also found a progressive rise in IL-4 levels from BT to BL leprosy, suggesting the role of IL-4 in disease progression with Th<sub>2</sub> activation. In our study, a trend of progressive increase in number of mast cells as well as the serum levels of IL-4 was seen from BT to BL leprosy. On statistical evaluation, a positive correlation was found between mast cell numbers and IL-4 ( $r=0.664$ ,  $p<0.05$ ). A few previous studies have also reported progressive rise in serum levels of IL-4 along the borderline spectrum of leprosy [7,20,21].

It is known that high levels of IL-4 in a Th<sub>2</sub> predominant milieu promotes further mast cell proliferation, thereby resulting in positive feedback for further IL-4 secretion. To summarise the results from our study and the discussed literature, it seems certain that mast cells play a role in leprosy by proliferating at the site of inflammation and secreting cytokines like IL-4 leading to Th<sub>2</sub> response, cell damage, inflammation, fibrosis, culminating into progression and downgrading of the disease.

## Conclusion

In the current study, a progressive rise in mast cell count across the borderline spectrum of leprosy with highest counts in BL leprosy was seen. This suggests a probable role of mast cells in downgrading of leprosy. The high levels of IL-4 down the spectrum of borderline leprosy suggest its role in dissemination of disease while promoting a Th2 response.

The correlation between IL-4 levels and mast cell numbers may suggest that mast cells are a major source of this cytokine in leprosy. Although the findings of this study suggest that mast cell stabilizers and anti-IL-4 therapy may have a therapeutic role in prevention of disease worsening, this hypothesis has not yet been studied. Further studies are mandated to understand the complex mechanisms involved in downgrading of leprosy and should take into account other immune cells and cytokine mediators.

## References

1. Sasaki S, Takeshita F, Okuda K, Ishii N. *Mycobacterium leprae* and leprosy: a compendium. *Microbial Immunol* 2001;45:729-36.
2. World Health Organization Regional Office for South-East Asia New Delhi. Enhanced Global strategy for further reducing the leprosy burden and sustaining leprosy control activities 2006-2010. Operational guidelines. *Lepr Rev* 2009;78:17-8.
3. Rav SD, Pratap VK, Sharma NK, Dayal SS. Mast cell in leprosy. *Indian J Lepr* 1990;62:467-72.
4. Aroni K, Kontochristopoulos G, Liossi M, Panteleos D. An investigation of mast cells in two basic leprosy groups. *Int J Lepr* 1993;61:634-5.
5. Mysorekar VV, Dandekar CP, Rao SG. Mast cells in leprosy skin lesions. *Lep Rev* 2001;72:29-34.
6. Bagwan IN, Khandekar MM, Khadana P, Jadhav MV, Deshmukh SD. A study of mast cells in granulomatous lesions of the skin, with special emphasis on leprosy. *India J Lepr* 2004;76:31-7.
7. Sherif ES. Role of mast cells and cytokine profile [TNF- $\alpha$ , IFN-  $\alpha$ , IL-4 and IL-4 mRNA] in different types of leprosy. Available from: <https://elsadany66.wordpress.com/article/role-of-mast-cells-and-cytokine-profile-i2p6c6e8rrui-3/> [Last accessed on August 16, 2016].
8. Magalhaes GO, Valentim VC, Pereira MJS, Nery JAC, Illarramendi X, Antunes SLG. A quantitative and morphometric study of tryptase-positive mast cells in cutaneous leprosy lesions. *Acta Tropica* 2008; 105:62-6.
9. Metcalfe DD, Baram D, Yoseph AM. Mast cells. *Physiol Rev* 1997; 77:1033-79.
10. Stead RH, Tomioka M, Quinonez G, Simon GT, Felten SY, Bienenstock J. Intestinal mucosal mast cells in normal and nematode infected rat intestines are in intimate contact with peptidergic nerves. *Proc Natl Acad Sci USA* 1987;84:2975-9.
11. Antunes LG, Liang Y, Costa Neri JA, Sarno EN, Frendscho MH, Johansson O. Mast cell subsets and neuropeptides in leprosy reactions. *Arq Neuropsiquiatr* 2003;61:208-19.
12. Longley J, Duffy TP, Kohn S. The mast cell and mast cell disease. *J Am Acad Dermatol* 1995;32:545-61.
13. Ridley DS, Jopling WH. Classification of leprosy according to immunity; A five group system. *Int J Lepr* 1966;34:255-73.
14. Naik R, Pai MR, Bantwal PB, Narayan S, Nayak KS, Gadhi A. Study of mast cells in non neoplastic skin lesions. *Indian J Pathol Microbiol* 2003;46:173-5.
15. Galli SJ, Metcalfe DD, Arber DA, Dvorak AM. Basophils, Mast Cells, and Related Disorders. In: Kaushansky K, Lichtman MA, Prchal JT, Levi MM, Oliver W, Press OW, Burns LJ, Caligari M, editors. *Williams Hematology*, 9<sup>th</sup> ed. New York: Mc Graw Hill 2015.p.965-82.
16. Prussin C, Metcalfe DD. IgE, mast cells, basophils, and eosinophils. *J Allergy Clin Immunol* 2003;111(2 Suppl):486-94.
17. Wang P, Wu P, Siegel ML, Egan RW, Billah MM. Interleukin (IL)-10 inhibits nuclear factor kB (NF-kB) activation in human monocytes: IL-10 and IL-4 suppress cytokine synthesis by different mechanisms. *J Biol Chem* 1995;9558-63.
18. Chatura KR, Sangeetha S. Utility of Fite-Faraco stain for both mast cell count and bacillary index in skin biopsies of leprosy patients. *Indian J Lepr* 2012;84:209-15.
19. Joshi MM, Panicker NK, Buch AC, Chandanwale SS. Mast cells in non-neoplastic skin lesions. *Indian Med Gazette* 2013;145:8-13.
20. Yamamura M, Uyemura K, Deans RJ, Weinberg K, Rea TH, Bloom BR, et al. Defining protective responses to pathogens: cytokine profiles in leprosy patients. *Science* 1991;254:277-9.
21. Misra N, Murtaza A, Walker B, Narayan NPS, Misra SR, Ramesh V, et al. Cytokine profile of circulating T cells of leprosy patients reflects both indiscriminate and polarized T-helper subsets: T-helper phenotype is stable and uninfluenced by related antigens of *Mycobacterium leprae*. *Immunology* 1995;86:97-103.

**Subscription Information****Institutional** (1 year) INR5000/USD357**Here is payment instruction for your reference.****Check:**

Please send the US dollar check from outside India and INR check from India made:

Payable to 'Red Flower Publication Private Limited'.

Drawn on Delhi branch

**PayPal Instructions for the payment (only for transfer from outside India):**Payments can be made through our PayPal account at <https://www.paypal.com>.

Our PayPal recipient email address is redflowerppl@gmail.com.

**Credit Card:**

We accept Visa or MasterCard.

**Wire transfer:**

Complete Bank Account No. 604320110000467

Beneficiary Name: Red Flower Publication Pvt. Ltd.

Bank &amp; Branch Name: Bank of India; Mayur Vihar

MICR Code: 110013045

Branch Code: 6043

IFSC Code: BKID0006043 (used for RTGS and NEFT transactions)

Swift Code: BKIDINBBDS

**\*\*Please kindly add bank charge at your side if you pay by check or wire transfer.****Payment, orders and all correspondences should be sent to:**

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

## Epidemiological Study of Various Dermatological Conditions in Tribal Area of Valsad, India

Bhuvnesh Shah\*, Keya Sheth Shah\*\*

### Abstract

Skin diseases and their complications are a significant burden on health system of many nations. Information on prevalence would be useful in planning an effective healthcare system. *Objective:* To identify the spectrum of dermatological disorders in the tribal area of Valsad (India). *Methods:* In this retrospective follow up, 4283 patients with skin disorders were randomly selected from dermatology outpatient department of a government centre between 1/11/2016 to 1/2/17. *Results:* A total of 4283 patient data collected which included males: 2718 (63.56%) and females: 1565 (36.54%). The patients were divided into different age groups as 1-15 (n = 259), 16-60 (n = 3685), 61 and above (n = 339). Among infective dermatological disorder, Tinea was the most frequent diagnosis, comprising 40% (1718/4283), while scabies 6.5% (279/4283), and Hansen's disease 1.9% (82/4283). The non infectious dermatological conditions largely included Urticaria 6.7% (291/4283), Eczema 6% (261/4283) and Acne Vulgaris 3.19% (137/4283). Under Infectious dermatological disorders, the male exhibited significantly higher cases of Tinea infection than females [male: (26.2%) 1126/4283 vs. female: (13.8%) 592/4283]. Under non infectious disorders, Eczema was more commonly seen in males than females [male: 161/4283 vs. female: 100/4283]. *Conclusion:* Tinea happens to be the most prominent infection among the Indian population owing to the work, working condition and Climate. This is followed by scabies and Hansen's. This study helps in creating awareness about the means of prevention and adopting certain life style. This study also helps in highlighting certain factors which can make a decisive role in planning of core health care system, prevent morbidity, mortality and also provide proper understanding of the nature and the course of disease.

**Keywords:** Epidemiological Study; Valsad; Tribal Area.

### Introduction

The exact prevalence of various dermatological condition in tribal area of Valsad (Gujarat) is still not accurately known.

The information on prevalence would be useful for planning strategies to manage the disease. Also help in creating awareness about the disease in the tribal disease.

### Material and Method

This was a retrospective study reviewing all out door patients of dermatology, at government hospital Valsad. The centre selected, was government hospital,

as it was easily accessed by the patients. All new and old cases were included for the study.

#### Data Analysis

Proper analysis was done by the staff of the hospital. The help was also taken from Department of Preventive medicine.

#### Data

- Total number of patients- 4283
- Males- 2718 (63.5%).
- Females- 1565 (36.54%)
- Patients enrolled belonged to the following age groups-

1. 1-15 years - n= 259
2. 16-60 years- n=3685
3. 60 years and above- n=339

## Results

A total of 4283 patient data collected which included males: 2718 ( 63.56 %) and females: 1565 (36.54%). The patients were divided into different age groups as 1-15yrs (n = 259), 16-60yrs (n = 3685), 61yrs onwards (n = 339). Among infective dermatological disorder, *T. corporis* with *Cruris* was the most frequent diagnosis, comprising 40% (1718/

4283), while scabies 6.5% (279/4283), and Hansens disease 1.9% (82/4283). The non infectious dermatological conditions largely included Urticaria 6.7% (291/4283), Eczema 6% (261/4283) and Acne Vulgaris 3.19% (137/4283). Under Infectious dermatological disorders, the male exhibited significantly higher cases of Tinea infection than females [male: (26.2%) 1126/4283 vs. female: (13.8%) 592/4283]. Under non infectious disorders ,Eczema was more commonly seen in males than females[ male:161/4283 vs. female 100/4283] of viral infection 9.2% (32/293) significantly higher than bacterial infection 3.8% (11/293) and fungal infection 3.4% (10/293).

**Table 1:** Non Infectious dermatosis

Condition	Male	Female	n=
Psoriasis	53	35	88
Acne vulgaris	43	94	137
Vitiligo	83	48	131
Urticaria	118	173	291
Alopecia areata	13	11	24
Eczema	161	100	261
Xerotic dermatitis	4	4	8
ICD	42	29	71
Lichenoid eczema	3	1	4
Melasma	49	24	73
PMLE	56	36	92
Seborrheic dermatitis	17	12	29

**Table 2:** Infectious dermatosis

Condition	Male	Female	n=
<i>T. Corporis,cruris</i>	1126	592	1718
<i>T.faciei</i>	66	35	101
<i>T.mannum</i>	34	10	44
<i>T.pedis</i>	17	10	27
<i>T.capitis</i>	15	17	32
Herpes	10	9	19
<i>Verruca vulgaris</i>	14	8	22
Hansens	60	22	82
Candidal intertrigo	28	24	52
Folliculitis	7	9	16
Onchomycosis	14	26	40
Scabies	173	106	279

## Conclusion

Patient with dermatological complaints is like iceberg. Only 1/3rd of actual problem is evident, while 2/3rd is submerged.

And most of the times ignored and thus making it difficult to treat. We have to emphasize on the non medical part of treatment like in treatment of tinea, advise of keeping body dry, wearing loose clothes is equally important as taking medicines.

## Acknowledgement

Dr. Harshida Shah and Dr. Bhavana Sheth.

### Conflict of Interest

None

### Statement

Manuscript has been read and approved by all

authors, that the requirement of authorship has been met, and each author believes that the manuscript represents honest work

#### *Key Message*

Epidemiological study helps in identifying various dermatological conditions in tribal area of Gujarat. thereby helping in prevention and treatment of the conditions.

#### **Reference**

1. Singh S, Beena PM, Profile of Dermatophyte infections in Baroda, Indian J Dermatol Venerol Leprol. 2003;69:281-3
2. Sackett DL et al: Clinical epidemiology- A Basic science for clinical Medicine. Boston.
3. Park JE Textbook of Preventive And Social Medicine.
4. Census of India. Office of the registrar General, India. 2A Mansingh Road, New Delhi.
5. Prevalence of Dermatological conditions in Children, Thesis by Dr.Bhuvnesh Shah.

---

## **Dermatology International**

### **Library Recommendation Form**

If you would like to recommend this journal to your library, simply complete the form below and return it to us. Please type or print the information clearly. We will forward a sample copy to your library, along with this recommendation card.

#### **Please send a sample copy to:**

Name of Librarian

Name of Library

Address of Library

#### **Recommended by:**

Your Name/ Title

Department

Address

#### **Dear Librarian,**

I would like to recommend that your library subscribe to the **Dermatology International**. I believe the major future uses of the journal for your library would provide:

1. useful information for members of my specialty.
2. an excellent research aid.
3. an invaluable student resource.

**I have a personal subscription and understand and appreciate the value an institutional subscription would mean to our staff.**

Should the journal you're reading right now be a part of your University or institution's library? To have a free sample sent to your librarian, simply fill out and mail this today!

Stock Manager

**Red Flower Publication Pvt. Ltd.**

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Phone: 91-11-45796900, 22754205, 22756995, Fax: 91-11-22754205

E-mail: [sales@rfppl.co.in](mailto:sales@rfppl.co.in)

## RF Assisted Management of Keloids

Chittoria R.K.\*, Elankumar S.\*\*, Kumaran M.S.\*\*, Sudhanva H.K.\*\*, Preethitha B.\*\*, Friji M.T.\*\*\*, Mohapatra D.P.\*\*\*, Dineshkumar S.\*\*\*

**Author Affiliation:**

\*Additional Professor & Head \*\*Senior Resident \*\*\*Associate Professor, Department of Plastic Surgery, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Pondicherry, India-605006.

**Reprint Request:**

Ravi Kumar Chittoria

Head, Department of Plastic Surgery, JIPMER, Pondicherry, India.  
E-mail: drchittoria@yahoo.com

Received on: 24.10.2016

Accepted on: 26.11.2016

**Abstract**

Intraoperative bleeding can be cumbersome while operating on scars using a scalpel. Though skin incisions by a scalpel is of common practice, incisions done by electromagnetic radiation of high frequency in the form of radiofrequency are more precise, accurate, associated with less bleeding and in turn less time consuming giving more defined result. Intra-lesional Keloid scar excisions required good haemostasis and precision leaving behind only a thin rim of tissue.

**Keywords:** Keloid; Radiofrequency; Intra-Lesional Excision.

### Introduction

Intraoperative bleeding obscures the operative field, and use of electrocautery can lead to charring of tissues. Achieving haemostasis during surgeries for keloid scars whose blood vessels have little or no contractile tissues is always a challenge to surgeons. Incision by Radiofrequency (RF) aids in a pressure less incision with no dragging or bunching of tissue with simultaneous cutting, coagulation, and precision of incision [1,2]. Radiofrequency incision causes less lateral tissue damage compared to other heat-producing devices [3]. We present use of RF probe for intra-lesional keloid excision in a 55 year old male.

### Material and Methods

A 55 year aged male presented to the outpatient department of Plastic Surgery, JIPMER, Puducherry in July 2016 with 6 years old post traumatic keloid over the presternal region with recurrent episodes of scar abscess lasting for 2 years, which was not responding to conservative measures. Treatment plan for the patient was intra-lesional excision of

keloid including all of the infected sinuses and skin grafting followed by steroid injections. Patient was evaluated and underwent intra-lesional keloid excision under local anaesthesia. The scar tissue was excised using radiofrequency probe with a power of 1.7W, instead of using scalpel. It was noticed that the resistance while cutting the scar tissue was least, bleeding was minimal and procedure was completed in minimal operative time. The skin incision had no adverse effect on the skin and the graft take was also very good. On follow-up, skin graft is taken well and there are no post-operative complications



Fig. 1: Pre op



Fig. 2: Intra -op



Fig. 3: Post op

## Discussion

Recurrent keloid scars are unsightly, may produce persistent pain, itching and are susceptible to recurrent infections. Though line of management remains conservative for most keloid scars, occasionally an intra-lesional excision may need to be considered [4]. Intra-lesional excision requires a rim of scar tissue to be left in the periphery of the scar. Surgery in keloid scars is more prone to bleeding due to lack of contractile tissues. The use of radiofrequency probe in keloid scars gives a more precise, accurate, blood less field improving the result of surgery.

Radiosurgery has been used to facilitate incisions in diverse fields of plastic surgery including blepharoplasty, face lifting, hair restoration surgery, and abdominoplasty.

Ablation effect on tissues is caused by vaporizing their water content with the help of continuous heat

application in tissues beneath the tip of the active electrode, causing cutting and the coagulation simultaneously. Radiofrequency ablation devices work by generating high frequency voltage of approximately 500 kHz. These devices cause flow of electrical currents through tissues when brought into close vicinity of tissues. The tissues provide the necessary impedance to produce heat as electrons overcome the resistance in the tissues. The patient's body functions as a part of the electrical circuit [4]. The electrical current may pass harmlessly through the patient's body without causing deleterious effects if the current alternates at the much higher frequency in the range of 330,000 cycles per second (330 kHz).

Radiofrequency has various advantages, such as ease of soft tissue ablation, hemostasis, and instant sterilization. There is minimal scar and operative time as well as post-operative pain is less in contrast to conventional scalpel incision.

## Conclusion

We suggest that radiofrequency assisted keloid excision is a better and safe alternative to scalpel/electrocautery because of more precision, better coagulation with less bleeding, less operative time and no undesirable effects on graft take.

## References

1. Hambly R, Hebda PA, Abell E, et al. Wound healing of skin incisions produced by ultrasonically vibrating knife, scalpel, electrosurgery, and CO<sub>2</sub> laser. *J DermatolSurg Oncol* 1988;14:1213-7.
2. Bridenstine JB. Use of ultra-high frequency electrosurgery (radiosurgery) for cosmetic surgical procedures. *Dermatol Surg* 1998;24:397-400.
3. Niamtu J III. 4.0 MHz radio wave applications in cosmetic facial surgery. *J CosmetDermatol* 2003; 16:33-46.
4. Wong Micheal S.M.D. Intralesional excision of keloids *Plastic and Reconstructive Surgery*, 2005 August;116(2):675.
5. Berjano, E. Theoretical modeling for radiofrequency ablation: state-of-the-art and challenges for the future, *BioMedical Engineering OnLine*, 2006;5:25.

## A Clinical Study of Vitiligo at a Tertiary Care Centre of East India

**Rajesh Sinha\***, **Abhijeet Kumar Jha\*\***, **Smita Prasad\*\***, **Shweta Dipti\*\*\***

**Author Affiliation:**

\*Assistant Professor, \*\*Senior Resident, \*\*\*Junior Resident, Department of Dermatology, STD & Leprosy, All India Institute of Medical Sciences, Patna 801507.

**Abstract**

**Background:** Vitiligo is a common skin problem affecting people globally. Though usually symptomless, it is associated with many taboos and social stigma in India. In this study, we analyzed the clinicoepidemiological profile of vitiligo patients visiting dermatology opd of a tertiary care centre of east India. **Aim:** A clinical study of vitiligo to know profile of patients suffering from vitiligo with associated cofactors. **Methods:** All patients of vitiligo visiting dermatology opd of All India Institute of Medical Sciences, Patna from March 2013 to February 2015, were included in the study. **Result:** Of 256 vitiligo patients included in the study, 51.5% (132) were males and 48.4% (124) were females. Most common morphological patterns noted were vitiligo vulgaris in 49.6% (127) patients. Most common site of onset was lower limbs in 26.56% (68) patients. Koebner phenomenon was present in 23.8% (61 cases). Leucotrichia was noted in 17.8% (46 patients). Most common associated condition noted was thyroid disorders in 6.6% (17) cases.

**Keywords:** Vitiligo; Bihar; Clinical Pattern.

**Introduction**

Vitiligo is an acquired pigmentary disorder of the skin caused by destruction or inactivation of melanocytes. It is characterised by milky white patches of different shapes and sizes. Though etiology of vitiligo has not been fully explained but significant role of genetic susceptibility, autoimmunity and oxidative stress has been implicated [1]. Prevalence of vitiligo varies from region to region and in different ethnic groups. In different studies worldwide, prevalence of vitiligo has been found to vary from 0.5 to 8% [2-5]. Different studies, carried out in different parts of India have reported prevalence rate of 0.25 to 2.5% [6-10]. Vitiligo lesions do not impair the capacity to work or expectancy of life but may cause significant influence on psychological and emotional well being of the patients. [11,12]. There are many prejudices and taboos associated with this disease that makes it a social embracement for the sufferers. In India, it is sometimes called as-sweth kusth (white leprosy) adding the stigma of leprosy to the disease. Bihar is a 3<sup>rd</sup> most populous state of India, located in the central part, with a population of 10.41 crores [13]. No study has been carried out to know the prevalence

of vitiligo in this region of the country. The aim of our study was to know the prevalence, and clinical pattern of vitiligo that affects population of central part of India.

**Material and Methods**

This prospective and observational 2 year (24 months) study was done at All India Institute of Medical Sciences, Patna, Bihar, and a tertiary care health centre in central India. All patients of vitiligo visiting dermatology opd from March 2013 to February 2015 were included in the study. Aim of the study was to know clinical profile of patients suffering from vitiligo with associated cofactors. All new cases of vitiligo (diagnosed clinically or by Woods lamp) attending dermatology OPD were included in the study. Patients suffering from depigmented lesion secondary to burns, chemical injury, scarring, physical trauma and drug intake were excluded from study. Detailed history including socio-demographic profile of each patient was recorded in the performa prepared for this purpose. Specific emphasis was given on age of onset, duration of disease, site of onset, type of vitiligo, presence of

leotrichia, presence of koebners phenomenon and clinical type of vitiligo and associated diseases. A complete history and physical examination was performed to note the characteristic of the disease and associated factors. The evolution of disease as evidenced by appearance of new lesions and the increased in the size of existing lesions, over past 3 months was noted. The clinical subtypes of vitiligo were classified as per Bordeaux classification given by vitiligo global issues consensus conference [13] into three groups, segmental, nonsegmental and unclassified vitiligo. Nonsegmental vitiligo was further classified as generalised, acrofacial, and mucosal and mixed vitiligo. Mucosal vitiligo was defined as involvement of the oral and/or genital mucosae. Acrofacial vitiligo referred to multiple, bilateral, symmetrical depigmented lesions involving acral parts of extremities and peri-orificial regions. Universal vitiligo corresponded to involvement of 80% or more body surface area. Focal vitiligo was defined as small, isolate depigmented non segmental lesion. Vitiligo vulgaris was defined as depigmented scattered lesions widely distributed and usually symmetrical. Segmental vitiligo corresponded to presence of one or more macules in a dermatomal distribution. Mixed vitiligo was defined as coexistence of segmental and non segmental vitiligo. Apart from routine blood examination, blood sugar and thyroid function test were done whenever necessary. Statistical package for social sciences SPSS version 14.0 was used to analyse the data.

## Result

A total of 256 vitiligo patients were examined during the study. They accounted for 2.78 % of the total number new patients, who attended dermatology opd clinic during the study period. Among these, 51.5% (132) were males and 48.4% (124) were females. The male to female ratio was 1.06. Mean age at presentation was 24.5 year and age at presentation ranged from 3 months to 79 years. Most common morphological pattern noted was vitiligo vulgaris in 127 (49.6%) of cases followed by acrofacial 66 (25.8%), focal 29 (11.3%), segmental 27, (10.5%), mucosal 4 (1.6%), mixed 2 (0.8%) and universal 1 (0.4%) (Fig.-1). Most common site of onset was lower limbs in 26.56% (68) patients. This was followed by head and neck 24.60% (63), trunk 18.75% (48); mucosal 16.01% (41) cases, upper limb 14.06 (36) (Figure 2). Duration of vitiligo at the time of presentation ranged from 1 month to 50 years. Maximum number of patients had disease duration of 1 to 5 years (53.12%), at the time of hospital visit (Fig.-

3). A positive family history was observed in 37 (14.4%) of patients. It was observed that 136 (53.1%) patients had a body surface area involvement of less than 2 %, 76 patients (30.15%) had a body surface involvement of 2-5% and 44 patients (17.46%) had more than 5% body surface area involvement (Figure 4). Koebner phenomenon was present in 23.8% (61 cases). Leucotrichia was noted in 17.8% (46 patients). Other associated conditions that were noted in our study included alopecia areata in 2.6%, psoriasis 0.8%, eczema 3.1%, halo nevus in 1.1%, lichen planus 0.3% and thyroid disorders in 6.6% cases (Table 1)

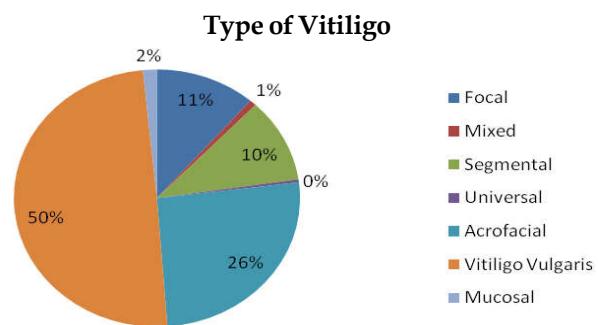


Fig. 1: Chart showing type of Vitiligo

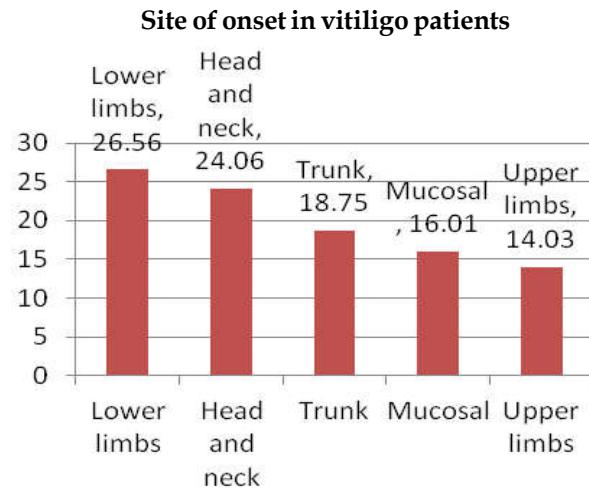


Fig. 2: Chart depicting site of onset of vitiligo

## Duration of Disease

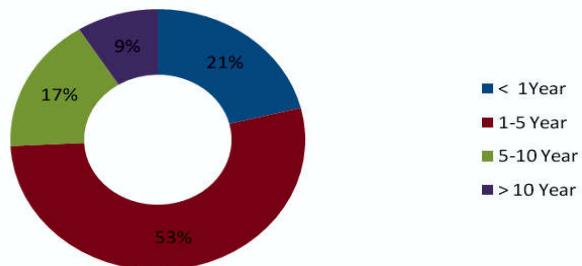
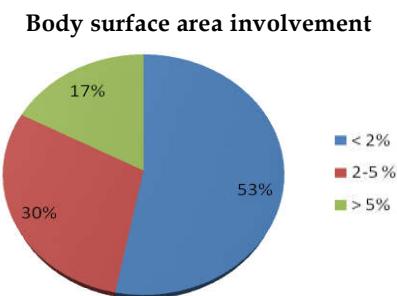


Fig. 3: Duration of Disease

**Table 1:** Associated Conditions

Alopecia Areata	6 (2.4%)
Thyroid Disorder	17 (6.6%)
Eczema	8 (3.1%)
Halo nevus	3 (1.1%)
Lichen Planus	1 (0.3%)
Psoriasis	2 (0.8%)
Diabetes	11 (4.3%)
Atopic Dermatitis	4 (1.6%)

**Fig. 4:** Chart showing body surface area involvement

## Discussion

Vitiligo is an acquired depigmentary disorder that is caused by loss of melanocytes, the pigment producing cells of skin. It presents as chalky white spots over skin which are more noticeable in dark skin people. It causes many social and psychological problems especially in regions with dark skinned people as they are more noticeable. The exact etiology of vitiligo is still not known.

Prevalence of vitiligo varies from country to country and from region to region and in different ethnic group. In our study the prevalence was 2.78%. Most studies from India have reported prevalence of 1-3% [6,7,8,9,10]. All these studies are hospital based studies. A community based study from China found the prevalence to be as low as 0.56% [2]. A Korean study showed annual prevalence of vitiligo determined by hospital visiting rate to be 0.12% to 0.13% over a period of three years [15]. The varying ethnic backgrounds of the population residing in different geographic region with varying environmental conditions may contribute to the wide variation in the prevalence in India. The incidences of vitiligo reported in other studies are 2.5% by Handa and Kaur, 1.84% by Martis et al and 1.4% by Sehgal [6,16,17].

Male to female ratio in our study was 1.2. Most of other studies report near equal incidence in males and females [8,18,19]. A study conducted by Handa and Kaur reported female to male incidence of 1.2 [6]. The predominance in females could be attributed to higher aesthetic concern among female population

[20]. In males, focal and acrofacial variants were more common where as in females, vitiligo vulgaris was more commonly reported. The number of females affected with vitiligo was higher than males in our study. This may be because of earlier reporting by female patients because of more concern and matrimonial issues. Most of the Indian studies have documented similar result of higher prevalence in female population [9,16]. In contrast to above studies Handa et al found higher prevalence of vitiligo in male population [6].

The duration of disease ranged from 3 months to 50 years with a mean duration of  $6.4 \pm 8.1$  years. Most of the cases (74.1% patients) had disease duration of less than 5 years. Progressive disease was reported in 59.2% patients at the time of presentation. Asymptomatic nature and slow response to medications could be the reason for long duration of the disease. The longer duration of disease could be attributed to the slow progressive nature of the disease and slow response to most of the therapeutic modalities available. Most other studies have reported similar mean duration of disease ranging from 1-5 years [6,8,9]. Disease duration of less than 1 year was present in 21.1%, where as 8.9% patients presented with disease duration of greater than 10 years. 16.8% patients had disease duration between 5-10 years.

Most common clinical type of vitiligo noted in our study was vitiligo vulgaris (127 patients, 49.6%). This was followed by acrofacial, focal, segmental, mucosal, mixed and universal. Vitiligo vulgaris has been reported as commonest type of vitiligo in various studies [3,6,9,16]. In contrast, very few studies have reported acrofacial to be the commonest type of vitiligo [8,21]. A study from Japan reported segmental as second most common type of vitiligo [23].

Most common age of onset in our study was 2<sup>nd</sup> decade (10-20 age group). There was wide variation in age of onset. It ranged from 3 months to 87 years. On comparing age of onset in male and females, we found that female patients presented more commonly 2<sup>nd</sup> decade where as male patients presented more commonly in 3<sup>rd</sup> decade. Age of onset was also found to be lower in cases of segmental vitiligo. Other

studies have also reported 2<sup>nd</sup> -3<sup>rd</sup> decade as most common age of onset [6,8,9,16]. Wide variation has also been reported in age of onset [2,18].

Family history was noted in 9.4% of patients. Most common type of vitiligo associated with positive family history was arofacial (3.2%), followed by segmental (2.8%), vulgaris (1.9%) focal (1.1%) and mucosal (0.4%). Zhang et al reported highest familial clustering of segmental vitiligo [2]. In most studies, family history in vitiligo patients varies from 8-20% [6,8,9,16]. A study from Pakistan has reported a high family history of 27.8%. [22].

Lecotrichia was noted in 7.5% of patients. Handa and Kaur reported incidence of lecotrichia to be 11.5% [6]. Vora et al reported lecotrichia in 33.5% of patients [9]. A study from Turkey reported leukotrichia in only 3.38% of patients [24]. Leukotrichia was associated most commonly with vitiligo vulgaris (3.4%) followed by segmental vitiligo (2.9%) and focal vitiligo (1.2%). Presence of lecotrichia is significant because it is associated with poor response to medical therapy. Koebnerisation was noted in 6.2% of patients. Koebnerisation was more common in progressive form of disease than in non progressive form. Incidence of koebnerisation phenomenon ranged from 5-31% in different studies [6,8,16].

Systemic diseases like hypo/hyperthyroidism, diabetes, hypertension, pernicious anaemia, autoendocrinopathy, Sjogren syndrome can occur in patients of vitiligo. A retrospective population-based study conducted in Taiwan showed a significant association between vitiligo and several comorbid diseases, including alopecia areata, Hashimoto thyroiditis, myasthenia gravis, psoriasis, Graves' disease, Sjögren's syndrome, systemic lupus erythematosus and atopic dermatitis [25]. In our study, thyroid disorder was noted in 6.6% of patients. Incidence of thyroid disorders has been reported to vary between 0.2-9% in various studies [3,8,16]. Diabetes was noted in 0.8% of patients.

Various cutaneous diseases and findings like alopecia areata, halo nevus, atopic dermatitis, psoriasis, eczema, premature greying can occur in vitiligo [26, 27]. In our study vitiligo was associated with alopecia areata (2.4%), atopic dermatitis (2.6%), halo nevus (1.1%), eczema (3.1%), psoriasis (0.08%). Handa and Kaur reported alopecia areata in 0.4% and atopic dermatitis in 1.4% in their study [6]. Yazanpanath et al reported coincidence of vitiligo and psoriasis in 0.19% of patients. [28]

Though we didn't screen all patients for audiometric abnormality, 6.2% (16) patients reported impaired hearing, which was confirmed by test. Akay

et al reported that sensineural hypoacusis was found in 37.7% of vitiligo patients [29]. In a study from south India hypoacusis was present in 10% of cases [30]. Melanocytes play an important role in hearing process as melanocytes reside in the cochlea and loss of these melanocytes leads to deafness [31].

Though the result of our study was similar to studies from other parts of India, this is first study on the clinical and epidemiological aspect of vitiligo from this part of India. A limitation of our study was that it was hospital based study which may not reflect the true prevalence rate of vitiligo in the general population.

## References

1. Halder RM, Taliaferro SJ. Disorders of melanocytes. In: Wolff K, et al. editor. Fitzpatrick's Dermatology in general medicine. 7th ed. New York: McGraw-Hill; 2008; p616-618.
2. Wang X, Du J, Wang T, Zhou C, Shen Y, Ding X, Tian S, Liu Y, Peng G, Xue S, Zhou J, Wang R, Meng X, Pei G, Bai Y, Liu Q, Li H, Zhang J. Prevalence and clinical profile of vitiligo in China: a community-based study in six cities. *Acta Derm Venereol*. 2013 Jan;93(1):62-5.
3. Alissa A, Al Eisa A, Huma R, Mulekar S. Vitiligo-epidemiological study of 4134 patients at the National Center for Vitiligo and Psoriasis in Central Saudi Arabia. *Saudi Med J*. 2011 Dec;32(12):1291-6.
4. Howitz J, Brodthagen H, Schwartz M, Thomsen K. Prevalence of vitiligo. Epidemiological survey on the Isle of Bornholm, Denmark. *Arch Dermatol*. 1977 Jan;113(1):47-52.
5. Alikhan A, Felsten LM, Daly M, Petronic-Rosic V. Vitiligo: a comprehensive overview Part I. Introduction, epidemiology, quality of life, diagnosis, differential diagnosis, associations, histopathology, etiology, and work-up. *J Am Acad Dermatol*. 2011 Sep;65(3):473-91.
6. Handa S, Kaur I. Vitiligo: clinical findings in 1436 patients. *J Dermatol*. 1999 Oct;26(10):653-7.
7. Shahil E M, Agrawal D, Vagadia K, Marfatia Y S, Begum R. Vitiligo: Clinical profiles in Vadodara, Gujarat. *Indian J Dermatol* 2006;51:100-4.
8. Agarwal S, Ojha A, Gupta S. Profile of vitiligo in kumaun region of Uttarakhand, India. *Indian J Dermatol*. 2014 Mar;59(2):209.
9. Vora RV, Patel BB, Chaudhary AH, Mehta MJ, Pilani AP. A Clinical Study of Vitiligo in a Rural Set up of Gujarat. *Indian J Community Med*. 2014 Jul;39(3):143-6.
10. Shah H, Mehta A, Astik B. Clinical and sociodemographic study of vitiligo. *Indian J Dermatol*. 2014 Jul;39(3):143-6.

Dermatol Venereol 2008;74:701.

11. Pahwa P, Mehta M, Khaitan BK, Sharma VK, Ramam M. The psychosocial impact of vitiligo in Indian patients. Indian J Dermatol Venereol Leprol. 2013 Sep-Oct;79(5):679-85.
12. Osman AM, Elkordufani Y, Abdullah MA. The psychological impact of vitiligo in adult Sudanese patients. Afr J Psychiatry (Johannesbg). 2009 Nov;12 (4):284-6.
13. <http://www.census2011.co.in/census/state/bihar.html> (last accessed on 27 Aug 2016).
14. Ezzedine K, Lim HW, Suzuki T, Katayama I, Hamzavi I, Lan CC, Goh BK, Anbar T, Silva de Castro C, Lee AY, Parsad D, van Geel N, Le Poole IC, Oiso N, Benzekri L, Spritz R, Gauthier Y, Hann SK, Picardo M, Taieb A; Vitiligo Global Issue Consensus Conference Panelists. Revised classification/nomenclature of vitiligo and related issues: the Vitiligo Global Issues Consensus Conference. Pigment Cell Melanoma Res. 2012 May;25(3):E1-13.
15. Lee H, Lee MH, Lee DY, Kang HY, Kim KH, Choi GS, Shin J, Lee HJ, Kim DH, Kim TH, Lee AY, Lee SC, Lee S, Kim KW, Hann SK, Park CJ, Oh SH. Prevalence of vitiligo and associated comorbidities in Korea. Yonsei Med J. 2015 May;56(3):719-25.
16. Martis J, Bhat R, Nandakishore B, Shetty JN. A clinical study of vitiligo. Indian J Dermatol Venereol Leprol. 2002 Mar-Apr;68(2):92-3.
17. Sehgal VN, Rege VL, Mascarenhas F, Kharangate VN. Clinical pattern of vitiligo amongst Indians. J Dermatol. 1976 Apr;3(2):49-53.
18. Howitz J, Brodthagen H, Schwartz M, Thomsen K. Prevalence of vitiligo. Epidemiological survey on the Isle of Bornholm, Denmark. Arch Dermatol. 1977 Jan;113(1):47-52.
19. Das SK, Majumder PP, Chakraborty R, Majumdar TK, Haldar B. Studies on vitiligo. I. Epidemiological profile in Calcutta, India. Genet Epidemiol. 1985; 2(1):71-8.
20. Nunes DH, Esser LM. Vitiligo epidemiological profile and the association with thyroid disease. An Bras Dermatol. 2011 Mar-Apr;86(2):241-8.
21. Singh M, Singh G, Kanwar AJ, Belhaj MS. Clinical pattern of vitiligo in Libya. Int J Dermatol. 1985 May;24(4):233-5.
22. Habib A, Raza N. Clinical pattern of vitiligo. J Coll Physicians Surg Pak. 2012 Jan;22(1):61-2. doi: 01.2012/JCPSP.6162.
23. Ohguchi R, Kato H, Furuhashi T, Nakamura M, Nishida E, Watanabe S, Shintani Y, Morita A. Risk factors and treatment responses in patients with vitiligo in Japan – A retrospective large-scale study. Kaohsiung J Med Sci. 2015.
24. Kalkanli N, Kalkanli S. Classification and comparative study of vitiligo in Southeast of Turkey with biochemical and immunological parameters. Clin Ter.2013;164(5):397-402.
25. Chen YT, Chen YJ, Hwang CY, Lin MW, Chen TJ, Chen CC, Chu SY, Lee DD, Chang YT, Liu HN. Comorbidity profiles in association with vitiligo: a nationwide population-based study in Taiwan. J Eur Acad Dermatol Venereol. 2015 Jul;29(7):1362-9.
26. Mohan GC, Silverberg JI. Association of Vitiligo and Alopecia Areata With Atopic Dermatitis: A Systematic Review and Meta-analysis. JAMA Dermatol. 2015 May;151(5):522-8.
27. Ingordo V, Cazzaniga S, Raone B, Digiuseppe MD, Musumeci ML, Fai D, Pellegrino M, Pezzarossa E, Di Lernia V, Battarra VC, Sirna R, Patrizi A, Naldi L. Circulating autoantibodies and autoimmune comorbidities in vitiligo patients: a multicenter Italian study. Dermatology. 2014;228(3):240-9.
28. Yazdanpanah MJ, Banihashemi M, Pezeshkpoor F, Moradifar M, Feli S, Esmaeili H. Evaluation between Association of Psoriasis and Vitiligo. J Cutan Med Surg. 2015 Mar-Apr;19(2):140-3.
29. Akay BN, Bozkir M, Anadolu T, Gullu S. Epidemiology of vitiligo, associated autoimmune diseases and audiological abnormalities: Ankara study of 80 patients in Turkey. J Eur Acad Dermatol Venereol 2010;24:1144-50.
30. Shankar DS, Shashikala K, Madala R. Clinical patterns of vitiligo and its associated co morbidities: A prospective controlled cross-sectional study in South India. Indian Dermatol Online J. 2012 May;3(2):114-8.
31. Ravinder K, Prasad D. Melanocyte. In: Lahiri K et al. Editors. Pigmentary disorders a comprehensive compendium. 1<sup>st</sup> ed. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd. 2014.p.13-21.

**STATEMENT ABOUT OWNERSHIP AND OTHER PARTICULARS**  
**“Dermatology International” (See Rule 8)**

1. Place of Publication : Delhi

2. Periodicity of Publication : Quarterly

3. Printer's Name : **Asharfi Lal**  
 Nationality : Indian  
 Address : 3/258-259, Trilok Puri, Delhi-91

4. Publisher's Name : **Asharfi Lal**  
 Nationality : Indian  
 Address : 3/258-259, Trilok Puri, Delhi-91

5. Editor's Name : **Asharfi Lal**  
 Nationality : Indian  
 Address : 3/258-259, Trilok Puri, Delhi-91

6. Name & Address of Individuals : **Asharfi Lal**  
 who own the newspaper and particulars of : 3/258-259, Trilok Puri, Delhi-91  
 shareholders holding more than one per cent  
 of the total capital

I Asharfi Lal, hereby declare that the particulars given above are true to the best of my knowledge and belief.

Sd/-  
**(Asharfi Lal)**

## Application of Low Level Laser in Management of Biofilm

**Vinayak C.\*, Chittoria R.K.\*\*, Sudhanva H.K.\* , Preethitha B.\* , Kumaran M.S.\* , Elankumar S.\* , Sireesha K.R.\***

**Author Affiliation:**

\*Senior Resident \*\*Professor, Department of Plastic Surgery, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER) Pondicherry, India-605006.

**Reprint Request:**

**Ravi Kumar Chittoria**

Professor, Department of Plastic Surgery, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER) Pondicherry, India-605006.

Email: drchittoria@yahoo.com

**Received on:** 27.02.2017

**Accepted on:** 17.03.2017

**Abstract**

Management of biofilm in chronic wounds is rapidly becoming a primary objective of wound care. However management of biofilm is an undeniably complex task. Beyond the basic steps of initial prevention (use of anti-biofilm agents), removal (debridement, desloughing) and prevention of reformation (use of antimicrobial agents), there are myriad patient, environmental and clinical parameters that must be considered when identifying a tailored solution. Systemic treatment strategies are required for infected chronic wounds, whereas in non-infected wounds where the presence of biofilm is impeding healing, strategies can be adopted to break up the biofilm. The antimicrobial effects of various lasers have been studied in vitro, and most reports have indicated that laser irradiation is useful for bacterial suppression. In the present case report we discuss the use of low level laser therapy in chronic leg wound.

**Keywords:** Biofilm; Chronic Wound; Low Level Laser Therapy.

### Introduction

Biofilms are described as bacteria attached to surfaces, encapsulated in a self-produced extracellular matrix and tolerant to antimicrobial agents (this includes antibiotics and antimicrobials). Less than 10 studies have visualised biofilms in non-healing chronic wounds using the accepted approaches of microscopy with or without molecular analysis [1,2]. These studies identified the presence of biofilms in 60% to 100% of samples. Currently, there is no 'gold standard' diagnostic test to define the presence of wound biofilms and no quantifiable biomarkers. Clinicians should 'assume all non-healing, chronic wounds that have failed to respond to standard care have biofilms' and, therefore, treatments should be targeted towards this [3]. Targeted therapies could be used to improve healing in cases where microbial biofilms is a causal component of chronic wounds as unique strategies to make microbes more susceptible to antimicrobials for clearance by the host immune system and therapies directed at preventing a prolonged inflammatory component of wound healing.

Low level laser therapy (LLLT) is the application

of light usually allow power laser or LED in the range of 1mW - 500mW. The light is typically of narrow spectral width in the red or near infrared(NIR) spectrum (600nm - 1000nm), with a power density (irradiance) between 1mw-5W/cm<sup>2</sup>. LLLT is not an ablative or thermal mechanism, but rather a photochemical effect comparable to photosynthesis in plants whereby the light is absorbed and exerts a chemical change [4]. In our case report we discuss the application of LLLT in chronic leg wound and the changes in the wound leading to effective treatment of a debilitating condition.

### Material and Methods

57 year male with wounds over right leg since 8months post traumatic injury which was non progressive in nature with minimal, non-foul smelling discharge with no associated fever. On examination wounds were of size 6 x 5cm and 12x8cm over medial and anterior aspect of lower one-third of leg respectively, both covered by pale, slimy granulation tissue with slough, surrounding skin hyper pigmented and indurated. No evidence of arterial or venous insufficiency confirmed by

Doppler study. Patient tissue culture was positive for pseudomonas and proteus. Biopsy showed no evidence of malignancy.

Patient underwent LLLT therapy using Gallium Arsenide laser for 10 minutes over the raw area of the wound over the medial surface and saline dressing of wound for 2 weeks every alternate day. At the end of 2 week wound over medial surface showed pink granulation tissue with reduction of surrounding oedema whereas wound over anterior surface remained pale. During this period patient was splinted, no debridement of wound performed and systemic antibiotics were administered as per sensitivity report. No local antibiotics used.



Fig. 1: Wound at presentation



Fig. 2: LLLT of wound over medial surface



Fig. 3: wound at the end of second week

## Discussion

The prevention and management of biofilms in chronic wounds is rapidly becoming a primary objective of wound care, with the presence of biofilms acknowledged as a leading cause of delayed wound healing. The biofilms interfere with normal wound healing, apparently by 'locking' the wound bed into a chronic inflammatory state that leads to elevated levels of proteases (matrix metalloprotease and neutrophil elastase) and reactive oxygen (ROS) that damage proteins and molecules that are essential for healing, biofilms maintain localised low oxygen tensions in the wound, thus contributing to chronicity [5,6]. A large percentage of bacteria in biofilms communities are metabolically dormant, which generates tolerance to antibiotics. Identification of biofilms in clinical practice is difficult, with few guidelines available to facilitate its recognition Keast et al. propose four main features that may increase suspicion of the biofilm presence, as follows:

1. Antibiotic failure
2. Infection of >30 days duration
3. Friable granulation tissue
4. A gelatinous material easily removed from wound surface that quickly rebuilds<sup>7</sup>.

Once the likelihood of biofilms presence is established, an appropriate treatment strategy should be determined with aims to reduce burden and prevent its reconstitution.

Low level laser therapy is irradiation of pathology (raw area) with light of near infrared radiation spectrum affects the biofilm and the wound. LLLT causes to disaggregation of microorganisms which form the biofilm and lose of adherence but doesn't reduce the number of micro-organisms. Different microbial species have different susceptibility to LLLT [8]. Nussbaum et al. also demonstrated that LLLT ( $\lambda=810$  nm) at 5, 10, 20 and 50 J/cm<sup>2</sup> doses was effective in inhibiting growth of *Pseudomonas aeruginosa* and *Staphylococcus aureus* [9].

LLLT not only affects biofilm but has effect on surrounding tissue. The molecular and cellular mechanisms LLLT suggest that photons are absorbed by the mitochondria; they stimulate more ATP production and low levels of ROS, which then activates transcription factors, such as NF- $\kappa$ B, to induce many gene transcript products responsible for the beneficial effects of LLLT. ROS are well known to stimulate cellular proliferation of low levels, but inhibit proliferation and kill cells at high levels. Nitric

oxide is also involved in LLLT, and may be photo-released from its binding sites in the respiratory chain and elsewhere. It is possible that NO release in low amounts by low dose light may be beneficial, while high levels released by high dose LLLT may be damaging. The third possibility is that LLLT may activate transcription factors, up regulating protective proteins which are anti-apoptotic, and generally promote cell survival [10,11].

Hence LLLT can be an effective tool in management of chronic wound. Further research is needed to evaluate the behaviour of different microorganisms, and their interaction in biofilms when subjected or not to LLLT and substantiate benefit of LLLT on wound healing.

## Conclusion

LLLT an evolving option for chronic wounds and biofilm management can be considered during routine wound care.

## References

1. Swanson T, Grothier L, Schultz G. Wound infection made easy. *Wounds International*. 2014.
2. Wolcott R, Dowd S. The role of biofilms: are we hitting the right target?. *Plastic and reconstructive surgery*. 2011 Jan 1;127:28S-35S.
3. Høiby N, Bjarnsholt T, Moser C, Bassi GL, Coenye T, Donelli G, Hall-Stoodley L, Holá V, Imbert C, Kirketerp-Møller K, Lebeaux D. ESCMID guideline for the diagnosis and treatment of biofilm infections 2014. *Clinical microbiology and infection*. 2015 May 1;21:S1-25.
4. Huang YY, Chen AC, Carroll JD, Hamblin MR. Biphasic dose response in low level light therapy. *Dose-Response*. 2009 Oct 1;7(4):dose-response.
5. Wolcott RD, Rhoads DD, Dowd SE. Biofilms and chronic wound inflammation. *J Wound Care*. 2008 Aug 1;17(8):333-41.
6. James GA, Ge Zhao A, Usui M, Underwood RA, Nguyen H, Beyenal H, deLancey Pulcini E, Agostinho Hunt A, Bernstein HC, Fleckman P, Olerud J. Microsensor and transcriptomic signatures of oxygen depletion in biofilms associated with chronic wounds. *Wound Repair and Regeneration*. 2016 Feb 1.
7. Keast D, Swanson T, Carville K, Fletcher J, Schultz G, Black JM. Ten Top Tips... Understanding and managing wound biofilm. *Journal of Lymphoedema*. 2014;5(2):20-4.
8. Krespi YP, Kizhner V, Nistico L, Hall-Stoodley L, Stoodley P. Laser disruption and killing of methicillin-resistant *Staphylococcus aureus* biofilms. *American journal of otolaryngology*. 2011 Jun 30;32(3):198-202.
9. Nussbaum EL, Lilge L, Mazzulli T. Effects of 630-, 660-, 810-, and 905-nm laser irradiation delivering radiant exposure of 1-50 J/cm<sup>2</sup> on three species of bacteria in vitro. *Journal of clinical laser medicine & surgery*. 2002 Dec 1;20(6):325-33.
10. Karu T. Laser biostimulation: a photobiological phenomenon. *Journal of Photochemistry and Photobiology B: Biology*. 1989 Aug 31;3(4):638.
11. Chen AC, Arany PR, Huang YY, Tomkinson EM, Sharma SK, Kharkwal GB, Saleem T, Mooney D, Yull FE, Blackwell TS, Hamblin MR. Low-level laser therapy activates NF- $\kappa$ B via generation of reactive oxygen species in mouse embryonic fibroblasts. *PloS one*. 2011 Jul 21;6(7):e22453.

## Instructions to Authors

Submission to the journal must comply with the Guidelines for Authors.

Non-compliant submission will be returned to the author for correction.

To access the online submission system and for the most up-to-date version of the Guide for Authors please visit:

<http://www.rfppl.co.in>

Technical problems or general questions on publishing with DI are supported by Red Flower Publication Pvt. Ltd's Author Support team ([http://rfppl.co.in/article\\_submission\\_system.php?mid=5#](http://rfppl.co.in/article_submission_system.php?mid=5#))

Alternatively, please contact the Journal's Editorial Office for further assistance.

Editorial Manager

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Phone: 91-11-22754205, 45796900, 22756995, Fax: 91-11-22754205

E-mail: [author@rfppl.co.in](mailto:author@rfppl.co.in)

## Non Responder of Alopecia Totalis to Diphenycyprone Responding to Addition of Betamethasone Pulse

**Dhurat R.\*, Pund P.\*\*, Sukesh M.S.\*\*, Dandale A.\*\*\*, Ghate S.\*\*\*\*, Bhandari K.\*\***

### Abstract

**Author Affiliation:**  
\*Professor and HOD \*\*Senior Resident \*\*\*Lecturer \*\*\*\*Associate Professor, Department of Dermatology, LTM Medical College and General Hospital, Sion, Mumbai - 400 022, Maharashtra, India.

**Reprint Request:**  
**Rachita Dhurat**, B 14/2 Maitri Park CHS, Sion Trombay Road, Chembur, Mumbai - 400 071, Maharashtra India.  
E-mail:  
rachitadhurat@yahoo.co.in  
**Received on:** 29.04.2017  
**Accepted on:** 09.05.2017

**Introduction:** Contact sensitizers remain the mainstay of treatment of alopecia areata totalis (AT). Systemic steroids in different regimes have been tried in AT with variable success. Many authors consider that addition of systemic steroids to the contact sensitizing protocol may mitigate the effect of the latter. However, there is scarcity of studies evaluating the response to combination therapy of systemic steroids with contact sensitizers. **Case Summary:** We report the case of a 19-year-old boy with AT of 5 years duration. Diphenylcyclopropenone (DPCP) was applied to right half of the scalp as per the standard protocol. With no response even after six months of this treatment, DPCP application was stopped and oral betamethasone pulse at the dose of 0.1 mg /kg weekly was started. Within 4 weeks of Betamethasone pulse, new hair growth was visible only on the right half of the scalp. DPCP was re-instituted on the same side of the scalp along with continuation of systemic steroids. Left half of the scalp served as control. At 8 weeks, DPCP treated side showed excellent growth while the left side responded poorly. **Conclusion:** This singular split scalp treatment outcome suggests that the response of contact sensitizers like DPCP is not suppressed by systemic corticosteroids; rather seems to have a synergistic effect in the treatment of AT.

**Keywords:** Alopecia Totalis; Areata; Diphenylcyclopropenone; Oral Minipulse; Steroid.

### Introduction

Alopecia areata (AA) is relapsing disease with a variable course. Its management is guarded with a highly variable response to DPCP.

Systemic corticosteroids have been used in severe forms of alopecia areata [1]. To minimize the side effects of daily systemic corticosteroids, oral minipulse (OMP) therapy with various steroids has been used with success in alopecia areata [2,3,4]. Topical immunotherapy has been variably advocated in extensive alopecia areata, efficacy of which varies from 5 to 85%. Non responders to DPCP have also been reported. The reason for non response to DPCP has not been studied extensively. There aren't any reports of combination of DPCP and systemic steroids in AA.

Hereby, we report a case of alopecia totalis (AT) a non responder to DPCP monotherapy who responded to addition of betamethasone pulse; contradicting to the assumption that steroids negate the effect of contact sensitizers.

### Case Report

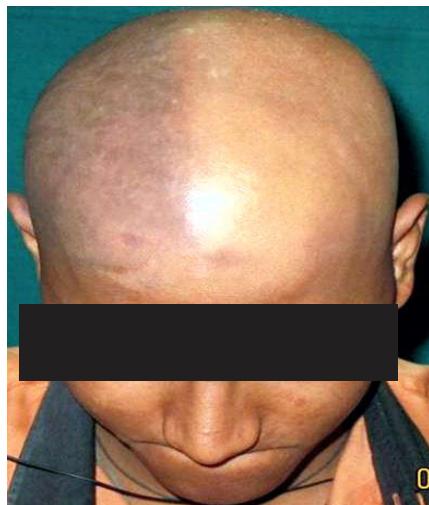
A nineteen year old Asian boy had total baldness on the scalp since five years. On examination, he had complete alopecia over the scalp with presence of few terminal hairs and his body hair was normal. Skin over the scalp showed no evidence of atrophy or scarring (Figure 1). The patient did not receive any treatment in the past. A clinical diagnosis of AT was made and patient was started with topical immunotherapy in the form of DPCP.

The patient was sensitized with DPCP 2% on the occipital bald area of 4cm. A mild to moderate irritant reaction as expected was noted after 48 hours. After 2 weeks of sensitization, a single coat of 0.001% DPCP was applied to the right half of the scalp using cotton tipped applicator in view of conducting a split scalp placebo control comparative study. Application was repeated weekly with gradual increase in the DPCP concentration (0.001 %, 0.01 %, 0.05 %, 0.1 %) according to the patient's response; the aim being to maintain erythema and pruritus for 48 hrs. The

patient developed mild erythema at concentration of 0.1%; therefore this concentration of DPCP was maintained for the next 6 months. However, there was no response in terms of hair growth (on the right side of the scalp) (Figure 2). Therefore, DPCP was discontinued and patient was shifted to oral minipulse (OMP) steroids in the form of oral minipulse betamethasone 0.1 mg /kg/ week in two divided doses, on two consecutive days. Within four weeks of betamethasone pulse therapy, new hair growth was noted on the DPCP treated side (Figure 3). Having noticed such a dramatic response in a short period of time, DPCP was reintroduced in order to evaluate the synergistic effect of DPCP and oral steroids. In this way, the right half the scalp received DPCP while left half served as control along with continuation of oral betamethasone. Within 8 weeks of treatment, right side of the scalp (DPCP+ OMP) showed significant regrowth as compared to the left side (DPCP only) which showed poor response (Figure 4).



**Fig. 1:** Patient of alopecia totalis with presence of few terminal hairs and no evidence of atrophy or scarring



**Fig. 2:** No response in terms of hair growth noted on the DPCP treated right side of the scalp at the end of 6 months. Only mild erythema of DPCP seen



**Fig. 3:** New hair growth noted on the DPCP treated side within four weeks of betamethasone pulse therapy



**Fig. 4:** Right side of the scalp (DPCP+ OMP) showing significant regrowth as compared to the left side (DPCP only) at the end of 8 weeks of treatment.

## Discussion

Pathogenesis of alopecia areata is multifactorial. The recent theory on pathogenesis of alopecia areata as per Paus et al [5] focuses on a breach of immune privilege as main cause of AA, the anagen hair bulb represents an immune privileged site characterized by the absence of major histocompatibility complex (MHC) class I expression and the presence of immunosuppressive cytokines such as TGF- $\beta$  (transforming growth factor  $\beta$ ). There is induction of CD8+ and CD4+ T cells targeted to newly exposed follicular antigens, which are normally sequestered from immune recognition. This immune

dysregulation could induce hair loss in AA through multiple mechanisms including: Direct cytotoxicity by CD8+ T cells, natural killer cells (NK), or NK-T-cell activity; Antibody dependent cell-mediated cytotoxicity (ADCC) etc. Immunotherapy reverses these changes. Skin treated with topical sensitizers shows a decrease in peribulbar CD4D CD8 ratio. There is a shift in the position of T lymphocytes from the perifollicular to the interfollicular area.

Systemic steroids for AA have been used in various forms. Sharma [3] administered oral prednisolone as pulses in doses of 300 mg at 4 weeks intervals. Cosmetically acceptable hair growth was seen in 58.3% patients at 4 months of treatment and relapse was seen in 2 patients after stoppage of therapy at 3 and 9 months respectively. In another study, Sharma et al [4] have reported complete hair growth in 26.6% of patients and a good response in 36.6% of patients treated with oral mini-pulse with dexamethasone.

Topical immunotherapy has been used for the treatment of severe alopecia areata. Immune modulating effect of topical immunotherapy is unclear. Response rate with diphenycprone ranges from 5% to 85% [6-11] which has led to considerable confusion surrounding its therapeutic value and efficacy. Several prognostic criteria have been identified: presence of nail changes, personal history of atopy, duration and extent of AA, age at onset of disease. In contrast to these parameters, histopathological features that may exert an influence on the therapeutic outcome of topical immunotherapy in AA have not been much explored. Paul et al [12] have examined histopathological changes in scalp biopsy obtained from 85 patients with severe AA before initiation of Diphenycprone treatment and concluded that non responder to topical sensitizers tend to have rather pronounced inflammatory reaction with dense perifollicular lymphocytic infiltrate.

A similar study showed that the combination of corticosteroids and contact sensitizer (anthralin) showed good response in treatment resistant, extensive alopecia areata. Out of eight patients with alopecia universalis/alopecia totalis, two attained cosmetic response as early as three months, two at six months and one showing partial response; thus concluding that the combination therapy was synergistic, safe and effective [13].

This study showed synergistic response with anthralin as a contact sensitizer; whereas in our study, DPCP was used. This explains the augmented effect of corticosteroids with topical sensitizers where corticosteroids may help in reducing the pronounced inflammation around the bulb leading to a more

favourable response to contact sensitizers.

This split scalp study similarly proves the synergistic effect of corticosteroids with contact sensitizers in treatment of alopecia totalis.

## Conclusion

The efficacy of DPCP in the treatment of AT is poor. This split scalp study proves that the addition of systemic steroids augments the effect of DPCP.

## References

1. Simpson NB. Alopecia areata. In: Rook A, Dawber R, editors. Diseases of the hair and scalp. 2nd Ed. London: Blackwell Scientific Publications; 1991.p. 324-33.
2. Pasricha JS, Kumrah L. Alopecia totalis treated with oral mini-pulse (OMP) therapy with betamethasone. Indian J Dermatol Venereol Leprol 1996;62:106-9.
3. Sharma VK. Pulsed administration of corticosteroids in the treatment of alopecia areata. Int J Dermatol 1996;35:133-6.
4. Sharma VK, Gupta S. Twice weekly dexamethasone oral pulse in the treatment of extensive alopecia areata. J Dermatol 1999;26:562-5.
5. Paus R, Nickoloff BJ, Ito T. A 'hairy' privilege. Trends Immunol 2005;26:32-40.
6. Hull SM, Norris JF. Diphenycprone in the treatment of long-standing alopecia areata. Br J Dermatol 1988; 119:367-374.
7. Van der Steen PH, van Baars HM, Perret CM, Happle R. Treatment of alopecia areata with diphenylcyclopropenone. J Am Acad Dermatol 1991;24:253-257.
8. Happle R, Hausen BM, Wiesner-Menzel L. Diphenycprone in the treatment of alopecia areata. Acta Derm Venereol 1983;63:49-52.
9. Van der Steen PH, van Baars HM, Happle R, Boezeman JB, Perret CM. Prognostic factors in the treatment of alopecia areata with diphenylcyclopropenone. J Am Acad Dermatol 1991;24:227-230.
10. Van der Steen PH, Boezeman JB, Happle R. Topical immunotherapy for alopecia areata: re-evaluation of 139 cases after an additional follow-up period of 19 months. Dermatology 1992;184:198-201.
11. Ashworth J, Tuyp E, Mackie RM. Allergic and irritant contact dermatitis compared in the treatment of alopecia totalis and universalis: a comparison of the value of topical diphenycprone and tretinoin gel. Br J Dermatol 1989;120:397-401.
12. Pronounced perifollicular lymphocytic infiltrates in alopecia areata are associated with poor

treatment response to diphenycprone. Eur J Dermatol. 1999 Mar;9(2):111-4.

C. Extensive alopecia areata: Not necessarily recalcitrant to therapy!. Int J Trichol 2011;3:80-3.

13. Deshpande D, Dhurat R, Saraogi P, Mishra S, Nayak

---

## **Anaesthetic Management of a Patient with Known Latex Allergy and Hypersensitivity to Multiple Drugs**

**Talib Khan\*, Rayees Najib\*\*, Arvin Preet\*\*\*, Zulfikar Ali\*, Syed Amer Zahoor\*\*\*\*, Shaqul Qamar Wani\*\*\*\*\*, Iram Ali\*\*\*\*\***

**Author Affiliation:**

\*Assistant Professors \*\*Senior Resident  
\*\*\*Post Graduate Scholar \*\*\*\*Additional Professor, Department of Anaesthesiology and Critical Care, SKIMS, Soura, J&K, India. \*\*\*\*\*Assistant Professor, Deptt. of Radiation Oncology, SKIMS, Srinagar, J&K, India. \*\*\*\*\*Registrar, Deptt. of Pediatrics GMC, Srinagar, G&K, India.

**Abstract**

The first case report of natural rubber-associated allergic reaction was described in Germany in the year 1927: a case of latex associated allergy by type I or immediate hypersensitivity [1]. Despite the increase of latex associated allergy in general population, severe anaphylactic reactions during some surgical procedures are still rare; however, they are associated with increased morbidity and mortality. Up to now, natural latex rubber is still the second most common cause for anaphylactic reactions during anesthesia [2]. Prevention, diagnosis, treatment, and follow-up of patients affected by this event represent a big challenge for anesthesiologists. The objective of this report was to describe the anesthetic management of a patient with known latex allergy and associated allergy to multiple drugs, food and fibres and to take measures to prevent life threatening allergic emergencies.

**Reprint Request:**

**Rayees Najib**, Senior Resident, Department of Anesthesia and Critical Care, SKIMS, Srinagar, J&K.190005.  
E-mail: rayeesnajib@yahoo.com

**Received on:** 01.05.2017

**Accepted on:** 16.05.2017

**Keywords:** Anaphylaxis; Contact Dermatitis; Latex Rubber.

**Case Report**

The following manuscript describes the perioperative management of a healthy 32 year old married Asian female, health worker by profession with a known history of latex hypersensitivity and allergy to multiple drugs and other allergens (food and fibres) diagnosed by skin prick and patch testing (Table 1) on allergist follow-up and was on immunotherapy posted as an elective case for laparoscopic Cholecystectomy. The patient had no other associated co-morbidities. The Patient was diagnosed for latex hypersensitivity 10 years back when she developed urticaria on multiple exposures to latex gloves as well as to the powder used in rubber gloves.

This patient was planned for an elective cholecystectomy one month back at a District hospital in view of her chronic cholecystitis with multiple gall stones. But she developed a severe anaphylactic reaction on the day of surgery in the preoperative room two hours after she was cannulated by a nurse in the ward without any intravenous drugs being given. The patient was resuscitated immediately with boluses of IV adrenaline, antihistamines and

corticosteroids and was discharged after an overnight observation in that hospital. The cause of this severe allergic reaction was attributed to her previous history of latex allergy as none of the medical equipments used were confirmed to be latex free and that she did not receive any other drugs before the anaphylaxis occurred. This patient was then referred to our superspeciality referral hospital for the perioperative management of her laparoscopic cholecystectomy in view of her being a high risk case for developing latex associated severe allergic reactions.

At our hospital a thorough workup about the patients history and investigations was done in the preanesthesia evaluation clinic, the patient was referred to the immunologist for further evaluation and to know about cross sensitivity and reactions to other drugs to be used during her stay in the hospital. Skin prick testing and patch testing were done to ascertain and document the allergic reaction to latex and other allergens. On evaluation she was found to be allergic to a multitude of drugs, food and fibres (Table 1) was put on immunotherapy by the allergist for a period of 4 weeks before giving clearance for the anesthesia and surgery. She was also evaluated for allergy to anesthetic drugs which was found to be

negative. Since, this patient had a uneventful history of tooth extraction under local anesthetic lidocaine 7 years back.

The patient was given clearance for anesthesia and a high risk informed consent was taken for anesthesia and surgery. The theatre staff was notified a day before the surgery and all necessary preparations were done to minimize latex exposure to the operation theatre. The patient was kept first in the list in order to minimize the use of latex containing substances in the theatre to get a latex safe environment. On the morning of surgery patient received oral premedication Montelukast 10 mgs, Levocetirizine 10mg, Ranitidine 150 mgs and Metoclopramide 10mgs with a sip of water one hour before the scheduled surgery. The patient's latex allergy was documented in the case notes and the operation theatre was sealed for minimum flow of human traffic and the main door of theatre was notified as a restricted zone in order to minimize airborne latex exposure. All items containing latex were removed from the patient care area. Only non-

latex medical supplies were used including, (Figure 1, 2 &3) but not limited to

- Gloves
- IV equipment
- Ventilation and airway equipment
- Catheters
- Surgical tape
- Tourniquets
- Medication containers without latex stoppers.

Anaesthetic circuit which was used was made of silicon and latex free PVC face mask were used. Patient was induced after attaching all the standard ASA monitoring. Airway was secured with size 4 latex free LMA, paper dressings were used for securing the intravenous cannula and fixing the LMA. A resuscitation trolley with resuscitation drugs and equipments was kept standby with prior notification to the ICU staff in order to avoid any catastrophe.

**Table 1: Allergic Skin Prick Testing Report:** Showing patient strongly positive for multiple allergens viz; D. Mite, F Salavius, R. nigricans, Penicillin Sp., few pollens, house dust, epithelial and Insects

**DEPARTMENT OF IMMUNOLOGY AND MOLECULAR MEDICINE  
SHER-I-KASHMIR INSTITUTE OF MEDICAL SCIENCES**

**ALLERGEN SKIN PRICK TESTING REPORT**

Name of the patient: Bhat Haseena Age/Sex: 32/F MRD: 024010

CC NO. \_\_\_\_\_ Ward/Bed: OPD Previous Diag: Urticaria under Evaluation

Date of Testing: 09/12/2015 Referred by \_\_\_\_\_ Lab. No. 581

S No.	Name of The Food Allergen	Result	S No.	Name of The Food Allergen	Result
1	Mite( D.pteronyssinus)	P 6x6mm	12	R.Nigricans	P 5x2.5mm
2	Pollens	P 5x5mm	13	Penicillin Sp.	P 6x5mm
3	Fungi	P 5x5mm	14	Asp. Few pollens	P 5x5mm
4	Dust	P 5x2.5mm	15	House dust	P 8x6mm
5	Dog epithelia	P 8x6mm	16	Mosquito	P 5x5mm
6	Sheep wool	P 6x6mm	17	Cucumber	N
7	Cockroach	P 5x5mm	18	lemon	N
8	Spinach	N	19	Chocolate	N
9	Cheese	N	20	Cat epithelia	P 5x5mm
10	Eggwhite	N	21	Peanuts	N
11	Peas	N	22	Prawns	N
	Positive Control (Histamine)			Negative Control (Normal Saline)	

POSITIVE : **Multiple Allergens viz; Drugs, Food, fibres, dust, pollens, insects and fibres.**

Sign of Resident



Sign of Consultant



**Table 2: Food Allergen, Skin Prick and Patch Testing Report: Suggests Latex allergy positive report**

**DEPARTMENT OF IMMUNOLOGY AND MOLECULAR MEDICINE  
SHER-I-KASHMIR INSTITUTE OF MEDICAL SCIENCES**

**FOOD ALLERGEN SKIN PRICK TESTING REPORT**

Name of the patient: Bhat Haseena Age/Sex: 32/F MRD: 193017

CC NO. \_\_\_\_\_ Ward/Bed: OPD Previous Diag: Chronic Urticaria, Asthma

Date of Testing: 07/09/2016 Referred by \_\_\_\_\_ Lab. No.90

S No.	Name of The Food Allergen	Result	S No.	Name of The Food Allergen	Result
1	Walnut	N	10	Egg white	N
2	Pista	N	11	Garlic	N
3	Raddish	N	12	Orange	N
4	Curd	N	13	Carrot	N
5	Almonds	N	14	Tomato	N
6	Fish	N	15	Rice	N
7	Saffron	N	16	milk	N
8	Spinach	N	17	Peanuts	N
9	Cheese	N	18	Latex	P6mmX5mm
	Positive Control (Histamine)	15mm P		Negative Control (Normal Saline)	NIL

\*P-positive \*N-Negative

Positive : **Latex**

Comments



Sign of Resident



Sign of Consultant



Fig 1: Anesthetic Drugs and antibiotics being used without latex impregnated rubber caps and (b) latex free Intravenous cannula



Fig. 2: Rubber material removed from the intravenous drip set to make it latex free. (b)Latex free syringes and arterial cannula



Fig. 3: Anesthetic Trolley with latex free drugs and equipments

Surgery was started 20 minutes after the initiation of anesthesia. No reactions or any other complications to anesthesia occurred during that period. After extubating, patient was shifted to the postoperative recovery (PACU) area for monitoring where she remained stable and was shifted to general surgery ward after being observed for 2 hours postoperatively. Patient was discharged after two days in a stable condition with uneventful post operative course.

## Discussion

The clinical manifestations of exposure to latex is a well established spectrum ranging from mild allergic contact dermatitis, which is not mediated by the immune associated response, to late hypersensitivity reaction (type IV mediated by T cells and immediate hypersensitivity reaction type I), also known as anaphylactic or IgE-mediated reaction [3]. The degree of severity, type I reactions can be divided into: I) cutaneous-mucous reactions II) moderate multivisceral reactions III) life-threatening with mono- or multi-visceral reactions IV) cardiorespiratory arrest V) death occurring because of inadequate response to cardiorespiratory resuscitation maneuvers [4].

Severe allergic manifestations to natural latex rubber have been documented in the literature since the last two decades. Since then, the incidence of allergic reactions could be decreased considerably by avoiding rubber exposure during surgeries in the operation theatres, which is considered to be the most important measure to reduce such reactions in the vulnerable population. Despite this knowledge and documentation, natural latex rubber products are still

in use because of the higher costs of latex-free products and their inferior quality. Incomplete manufacturer's specifications or labels on the surgical anesthetic equipments pose yet another problem in identifying the products containing latex.

The other important reasons for reducing the use of natural latex rubber were high-risk groups, such as patients with atopic syndrome or Spina bifida, surgery in infancy, and health professionals [5]. Prevalence is very high, and allergic reactions to natural latex rubber represent potentially life-threatening intraoperative complications [6]. Allergic reactions to natural latex rubber during anesthesia still amount a mortality rate from 5% to 7% [7]. Patients with atopic syndrome show a predisposition towards sensitization to natural latex rubber significantly, meaning a constant repeated contact with rubber containing materials [8]. Many affected patients have had history of multiple surgeries because of Spina bifida or anomalies of the urogenital tract in early infancy and thus high natural latex rubber exposure [9].

The medical history itself was conclusive taken from this patient as documented in this case report. The fact that the patient had been previously exposed to natural latex rubber- because she was a healthcare worker was ascertained on history and the available investigations and that she had received multiple immunotherapies so far (Table 1 and 2). Early exposure to natural latex rubber is a relevant factor for developing an allergy to latex in later life [9]. For preventing allergies to natural latex rubber, premedication with antihistamines and corticosteroids has been suggested. Premedication with histamine receptor antagonists (Ranitidine-H2 blocker + Levocetirizine-H1 blocker) and leukotriene (LT4) receptor antagonist (Montelukast) was used in

our case. The cost of extensive pre-surgery screening is also not relevant for every patient [10]. However, the diagnosis was well established in our case pre operatively by skin prick and patch testing guided by strong positive allergic episodes. Preoperative diagnosis by means of 'skin prick method' is not required always but can aid in the diagnosis, particularly for patients with spina bifida (upto 44% show latex allergies), dysplasia of the genitourinary tract and atopic dermatitis aswell as for the patients with occupational exposure to latex and allergies to food( eg; figs, papayas, chestnuts and kiwis) [10].

In case of all precautionary measures, if an intraoperative anaphylactic reaction occurs, it is crucial to first stop the contact with the allergen. The following list of measures shows a short overview of necessary interventions to be done:

- Stop contact with the allergen:
- Gloves have to be taken off outside the theatre and clothes must be changed.
- All personnel wearing latex gloves have to leave the operating theatre immediately or as quickly as possible.
- All items containing latex should be removed from the theatre.
- Recruitment of personnel, such as physicians and nursing staff.
- Priorities are 1st securing the airways, oxygen administration and maintaining normal haemodynamics by increasing volume intake and early adrenaline administration.

Simultaneous corticosteroids and antihistamine administration. The authors of this case report suggest the following measures to be taken in case of an acute anaphylaxis- As per the hospital protocol in case of a severe allergic reaction of any kind.

- In case of a suspected airway swelling, administer nebulised adrenaline via a mask, intravenous corticosteroids and intubate as soon as possible if already not done so.
- Oxygenation with sufficient FiO<sub>2</sub>, bronchoconstriction needs to be treated with nebulised bronchodilators.

Additional intravenous cannulae, volume replacement, adrenaline boluses and cardiopulmonary resuscitation if necessary.

Second line-therapy- with H1/H2-antihistaminics.

Further physical examinations- reevaluation.

## Conclusion

Regular and systematic education of patients, their families, healthcare workers and employers is an integral part of the management of latex allergy.

Patients and their family members who are diagnosed with latex allergy should be educated about the common symptoms and the management of this condition. They should also be made aware about the preventive measures to avoid future exposure of the offending allergen.

Healthcare workers should be imparted knowledge and trained about how to recognise the signs and symptoms of an allergic reaction and to treat an anaphylactic reaction.

Hospitals and healthcare centres should have set protocols and policies to ensure safety of both patients and the healthcare workers.

Natural latex rubber products can cause allergic reactions with a wide range of clinical signs and symptomatology, the spectrum ranging from mild eczema to severe and life threatening anaphylactic reactions. Furthermore, intraoperatively the diagnosis might get impeded by highly variable clinical symptoms, the ill response of patients, anesthesia-induced change in haemodynamics, blood loss during surgery. Therefore, prevention of exposure particularly in the high risk group seems to be even more important than raising awareness for allergies to natural latex rubber.

- Stopping the use of natural latex rubber or its by-products is the 1st step to prevent a latex associated anaphylactic reaction.
- A standardized questionnaire on preoperative assessment about latex allergy or any previous exposures to latex should be documented before any anesthetic procedure.
- A list of latex containing equipments in the operation theatre should be compiled and replaced by non-latex containing material.
- A hospital based standardized protocol must be laid down about the treatment should a anaphylactic reaction occur at anytime in the perioperative period and such a policy be written down in a standard operating procedure (SOP).
- Simulations and continuous medical education about the management of an anaphylaxis can be exercised along with the

basic and advanced life support training in a hospital setting.

## References

1. Valls A, Pascual CY, Caballero MT et al. Alergia al latex. *Allergol Immunopathol (Madr)*, 2004;32: 295-305.
2. Nel L, Eren E. Peri-operative anaphylaxis. *Br J Clin Pharmacol*. 2011;71:647-58.
3. Allarcon JB, Malito M, Linde H et al. Alergia ao latex. *Rev Bras Anestesiol*, 2003;53:89-96.
4. Ring J, Messmer K. Incidence and severity of anaphylactoid reactions to colloid volume substitutes. *Lancet*, 1977; 1:466- 469.
5. Sampathi V, Lerman J. Case scenario: perioperative latex allergy in children. *Anaesthesiology*. 2011;114: 673-80.
6. Dewachter P, Mouton-Faivre C, Emala CW. Anaphylaxis and anesthesia: controversies and new insights. *Anaesthesiology*. 2009;111:1141-50.
7. De Queroz M, Combet S, Berard J, Pouyau A, Genest H, Mouriquand P, et al. Latex allergy in children: modalities and prevention. *Pediatr Anesth*. 2009; 19:313-9.
8. Charous BL, Blanco C, Tarlo S, Hamilton RG, Baur X, Beezhold D, et al. Natural rubber latex allergy after 12 years: recommendations and perspectives. *J Allergy Clin Immunol*. 2002;109:31-4.
9. Murat I. Latex allergy: where are we? *Paediatr Anaesth*. 2000;10:577-97.
10. Caimmi S, Caimmi D, Cardinale F, Indinnimeo L, Crisafulli G, Peroni DG, et al. Perioperative allergy: uncommon agents. *Int J Immunopathol Pharmacol*. 2011;24:61-8.

---

## Radiofrequency Assisted Management of Trap Door Scar

Babu P.\*, Chittoria R.K.\*\*, Kumar M.S.\*, Sudhanva H.K.\*, Kumar E.\*, Reddy K.S.\*,  
Chavan V.\*, Friji M.T.\*\*\*, Mohapatra D.P.\*\*\*, Dineshkumar S.\*\*\*

### Author Affiliation:

\*Senior Resident \*\*Additional Professor & Head  
\*\*\*Associate Professor, Department of Plastic  
Surgery, Jawaharlal Institute of Postgraduate  
Medical Education and Research (JIPMER)  
Pondicherry, India-605006.

### Abstract

Scar revision forms a major part of plastic surgery practice. Various methods of scar revision have been described including revision by Z Plasty, W Plasty, serial excision, dermabrasion of the scar etc. the final outcome of scar revision depends on various factors like type of scar, skin type of the patient, orientation of the scar, technique used for revision and instrument used for revision. In this article we describe the use of the radiofrequency probe for revision of a trapdoor scar in a 32 year old patient and its benefits.

**Keywords:** Scar; Radiofrequency.

### Reprint Request:

Ravi Kumar Chittoria

Head, Department of Plastic Surgery,  
JIPMER, Pondicherry, India.  
E-mail: drchittoria@yahoo.com

Received on: 31.10.2016

Accepted on: 26.11.2016

### Introduction

The Radiofrequency cautery is also known as "poor man's laser" or "cold cautery". It uses alternating current at very high frequencies (500-4000 KHz) for producing ablation of tissues. It can be used for electrofulguration, electrodessication or electrocoagulation [1,2]. The radiofrequency probe ensures precise cutting with minimal collateral damage producing more aesthetically pleasing scars. This also ensures that healing is faster. It is highly effective in causing cutting of the skin at the same time coagulating tissues ensuring good hemostasis. Because of the simultaneous effects of cutting and coagulation, time taken to finish surgery is also faster. Procedures can be performed under local anesthesia. Lesions that would otherwise require the use of expensive ablative lasers can be treated in a fast, cost effective manner by the use of radiofrequency ablation [3,4,5,6]. It can be used to do full thickness excisions or to superficially shave off lesions. Its cosmetic uses include excision of naevi, removal of vascular lesions like hemangiomas, face lifts, abdominoplasty etc [7].

Here we present the case of a 32 year old male patient with a post traumatic, post operative trapdoor scar on the right cheek for whom scar revision was

done with the use of radiofrequency cautery.

### Case Report

A 32 year old male patient presented to the plastic surgery outpatient department with history of post traumatic, post operative unsightly scar over the right cheek since 30 years. Patient did not complain of pain, pruritis or altered sensation in the region of the scar.

On examination, a curvilinear, hyperpigmented, mature, depressed scar was present over the right cheek measuring 8 cm in length and 4mm wide in the broadest part of the scar. There were multiple



**Fig. 1a and b:** Preoperative pictures showing unsightly scar over right cheek

hatch marks running across the central scar. A trapdoor component was also noted to be present. There was no tethering to the underlying structures. The Vancouver scar scale (VSS) score was 3. The patient had Fitzpatrick type 5 skin. The patient was informed about various treatment modalities available and a final plan for serial excision of the scar was made.

After investigating the patient and obtaining informed written consent, the patient underwent Radiofrequency assisted excision of the central part of the scar under local anesthesia. The power of the radiofrequency used was 1W. The wound was closed with absorbable subcuticular sutures. Intraoperatively it was noted that the use of the radiofrequency probe provided good cutting of the scar tissues with adequate hemostasis. This ensured faster completion of the surgical procedure. The first post op check dressing was done on post op day 3 which showed good wound healing with a fine scar. After 10<sup>th</sup> post operative day, the patient was advised silicone gel application and sunscreen application to prevent hypertrophy and hyperpigmentation of the scar. The patient was followed up for a total period of 2 weeks and the follow up was uneventful. The postoperative VSS score was 0.



Fig. 2: Intraoperative picture showing markings for excision



Fig. 3: Use of Radiofrequency for excision of scar



Fig. 4a and b: Intraoperative pictures showing scar after excision and immediate postoperative appearance following suturing.



Fig. 5: Appearance at 10 days postop

## Discussion

Since the establishment of electricity, electrosurgery has been used in medical practice. Electrosurgical instruments have evolved from more basic instruments to better developed machines [8]. The radiowaves created by the radiofrequency unit travel through the cautery tip to the patient and back to the unit through a plate antenna placed close to the patient. When the waves pass through the body, the body tissues offer resistance which in turn generates heat which causes boiling of the tissue water and rupture of the cell membrane producing a cutting effect. The heat causes denaturation of the body proteins causing a coagulation effect. The various waveforms used causes three main patterns of its action. Fully rectified and filtered waveform causes cutting. Fully rectified waveform causes partial cutting and partial coagulation. Partially rectified waveform causes predominantly coagulation [9,10,11].

The electrodes in the Radiofrequency unit cause very minimal collateral damage (upto 75 micrometre) as the electrodes remain cold during the procedure and only the tip of the electrode comes in contact with the skin for a very short period of time. As the diameter of the electrode tip is small, the electrode-tissue interface is small. The Radiofrequency unit uses high frequency but at low intensity [9,10,11].

Scar revision forms one of the procedures performed routinely in plastic surgery. The ultimate result after revision depends on many factors. One of

those factors is the trauma caused to the tissue intraoperatively. The radiofrequency probe, by ensuring good cutting with minimal collateral damage ensures an aesthetically satisfying scar appearance after revision. The use of scalpel causes bleeding when the incision is made and obscures the surgical field prolonging surgical time. The radiofrequency probe ensures adequate hemostasis intraoperatively and prevents complications such as hematoma formation, postoperative wound gape and infection ensuring a good final outcome after surgery.

## Conclusion

Through this case report we would like to suggest that the use of radiofrequency for scar revision is a better option to conventional methods such as revision with scalpel or electrocautery. It is precise with minimal collateral damage and good hemostasis ensuring faster surgery with better postoperative outcomes.

### *Conflicts of Interest*

None

### *Source of Funding*

None

*Disclosures:* None

---

## References

1. Boughton RS, Spencer SK. Electrosurgical fundamentals. *J Am Acad Dermatol* 1987;16:862-7.
2. Sebbon JE. Electrosurgery: High-frequency modalities. *J Dermatol Surg Oncol* 1988;14:367-71.
3. Bridenstine JB. Use of ultra - high frequency electrosurgery (radiosurgery) for cosmetic surgical procedures. *Dermatol Surg* 1998;24:397-400.
4. Chiarello SE, Radiovaporization RF. Cutting to vaporize and sculpt skin lesions. *Dermatol Surg* 2003;29:755-8.
5. Pollock SV. Electrosurgery of the skin, New York: Churchill Livingston; 1991.
6. Sachdeva S, Dogra A. Radiofrequency (RF) ablation in dermatology. *Indian J Dermatol Venereol Leprol* 2007;52(3):134-7.
7. Hainer BL. Electrosurgery for the skin. *Am Fam Physician* 2002;66:1259-66.
8. Mutualik S. Standard guidelines for electrosurgery with radiofrequency current. *Indian Journal of Dermatology, Venereology, and Leprology*. 2009;75(8):83.
9. Sebbon JE. Electrosurgery principles: Cutting current and cutaneous surgery - Part-1. *J Dermatol Surg Oncol* 1988;14:29-31.
10. Sebbon JE. Electrosurgery principles: Cutting current and cutaneous Surgery - Part-2. *J Dermatol Surg Oncol* 1988;14:147-50.
11. Hainer BL. Fundamentals of electrosurgery. *J Am Board Fam Pract* 1991;4:419-26.

**Revised Rates for 2017 (Institutional)**

<b>Title</b>	<b>Frequency</b>	<b>Rate (Rs): India</b>	<b>Rate (\$):ROW</b>
Community and Public Health Nursing	3	5000	4500
Dermatology International	2	5000	4500
Gastroenterology International	2	5500	5000
Indian Journal of Agriculture Business	2	5000	4500
Indian Journal of Anatomy	4	8000	7500
Indian Journal of Ancient Medicine and Yoga	4	7500	7000
Indian Journal of Anesthesia and Analgesia	4	7000	6500
Indian Journal of Biology	2	5000	4500
Indian Journal of Cancer Education and Research	2	8500	8000
Indian Journal of Communicable Diseases	2	8000	7500
Indian Journal of Dental Education	4	5000	4500
Indian Journal of Emergency Medicine	2	12000	11500
Indian Journal of Forensic Medicine and Pathology	4	15500	15000
Indian Journal of Forensic Odontology	2	5000	4500
Indian Journal of Genetics and Molecular Research	2	6500	6000
Indian Journal of Hospital Administration	2	6500	6000
Indian Journal of Hospital Infection	2	12000	9000
Indian Journal of Law and Human Behavior	2	5500	5000
Indian Journal of Library and Information Science	3	9000	8500
Indian Journal of Maternal-Fetal & Neonatal Medicine	2	9000	8500
Indian Journal of Medical & Health Sciences	2	6500	6000
Indian Journal of Obstetrics and Gynecology	4	9000	8500
Indian Journal of Pathology: Research and Practice	4	11500	11000
Indian Journal of Plant and Soil	2	65000	60000
Indian Journal of Preventive Medicine	2	6500	6000
Indian Journal of Research in Anthropology	2	12000	11500
Indian Journal of Surgical Nursing	3	5000	4500
Indian Journal of Trauma & Emergency Pediatrics	4	9000	8500
Indian Journal of Waste Management	2	9000	8000
International Journal of Food, Nutrition & Dietetics	3	5000	4500
International Journal of Neurology and Neurosurgery	2	10000	9500
International Journal of Pediatric Nursing	3	5000	4500
International Journal of Political Science	2	5500	5000
International Journal of Practical Nursing	3	5000	4500
International Physiology	2	7000	6500
Journal of Animal Feed Science and Technology	2	78000	70000
Journal of Cardiovascular Medicine and Surgery	2	9500	9000
Journal of Forensic Chemistry and Toxicology	2	9000	8500
Journal of Geriatric Nursing	2	5000	4500
Journal of Medical Images and Case Reports	2	5000	4500
Journal of Microbiology and Related Research	2	8000	7500
Journal of Nurse Midwifery and Maternal Health	3	5000	4500
Journal of Organ Transplantation	2	25900	25000
Journal of Orthopaedic Education	2	5000	4500
Journal of Pharmaceutical and Medicinal Chemistry	2	16000	15500
Journal of Practical Biochemistry and Biophysics	2	5500	5000
Journal of Social Welfare and Management	3	5000	4500
New Indian Journal of Surgery	4	7500	7000
New Journal of Psychiatric Nursing	3	5000	4500
Ophthalmology and Allied Sciences	2	5500	5000
Otolaryngology International	2	5000	4500
Pediatric Education and Research	3	7000	6500
Physiotherapy and Occupational Therapy Journal	4	8500	8000
Psychiatry and Mental Health	2	7500	7000
Urology, Nephrology and Andrology International	2	7000	6500

**Terms of Supply:**

1. Agency discount 10%. Issues will be sent directly to the end user, otherwise foreign rates will be charged.
2. All back volumes of all journals are available at current rates.
3. All Journals are available free online with print order within the subscription period.
4. All legal disputes subject to Delhi jurisdiction.
5. Cancellations are not accepted orders once processed.
6. Demand draft / cheque should be issued in favour of "Red Flower Publication Pvt. Ltd." payable at Delhi
7. Full pre-payment is required. It can be done through online (<http://rfppl.co.in/subscribe.php?mid=7>).
8. No claims will be entertained if not reported within 6 months of the publishing date.
9. Orders and payments are to be sent to our office address as given above.
10. Postage & Handling is included in the subscription rates.
11. Subscription period is accepted on calendar year basis (i.e. Jan to Dec). However orders may be placed any time throughout the year.

**Order from**

Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091 (India), Tel: 91-11-22754205, 45796900, Fax: 91-11-22754205. E-mail: [sales@rfppl.co.in](mailto:sales@rfppl.co.in), Website: [www.rfppl.co.in](http://www.rfppl.co.in)

Manuscripts must be prepared in accordance with "Uniform requirements for Manuscripts submitted to Biomedical Journal" developed by international committee of medical Journal Editors.

## Types of Manuscripts and Limits

Original articles: Up to 3000 words excluding references and abstract and up to 10 references.

Review articles: Up to 2500 words excluding references and abstract and up to 10 references.

Case reports: Up to 1000 words excluding references and abstract and up to 10 references.

## Online Submission of the Manuscripts

Articles can also be submitted online from [http://rfppl.co.in/customer\\_index.php](http://rfppl.co.in/customer_index.php).

1) First Page File: Prepare the title page, covering letter, acknowledgement, etc. using a word processor program. All information which can reveal your identity should be here. use text/rtf/doc/PDF files. Do not zip the files.

2) Article file: The main text of the article, beginning from Abstract till References (including tables) should be in this file. Do not include any information (such as acknowledgement, your name in page headers, etc.) in this file. Use text/rtf/doc/PDF files. Do not zip the files. Limit the file size to 400 Kb. Do not incorporate images in the file. If file size is large, graphs can be submitted as images separately without incorporating them in the article file to reduce the size of the file.

3) Images: Submit good quality color images. Each image should be less than 100 Kb in size. Size of the image can be reduced by decreasing the actual height and width of the images (keep up to 400 pixels or 3 inches). All image formats (jpeg, tiff, gif, bmp, png, eps etc.) are acceptable; jpeg is most suitable.

Legends: Legends for the figures/images should be included at the end of the article file.

If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks from submission. Hard copies of the images (3 sets), for articles submitted online, should be sent to the journal office at the time of submission of a revised manuscript. Editorial office: Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091, India, Phone: 91-11-22754205, 45796900, 22756995. E-mail:

author@rfppl.co.in. Submission page: [http://rfppl.co.in/article\\_submission\\_system.php?mid=5](http://rfppl.co.in/article_submission_system.php?mid=5).

## Preparation of the Manuscript

The text of observational and experimental articles should be divided into sections with the headings: Introduction, Methods, Results, Discussion, References, Tables, Figures, Figure legends, and Acknowledgment. Do not make subheadings in these sections.

## Title Page

The title page should carry

- 1) Type of manuscript (e.g. Original article, Review article, Case Report)
- 2) The title of the article, should be concise and informative;
- 3) Running title or short title not more than 50 characters;
- 4) The name by which each contributor is known (Last name, First name and initials of middle name), with his or her highest academic degree(s) and institutional affiliation;
- 5) The name of the department(s) and institution(s) to which the work should be attributed;
- 6) The name, address, phone numbers, facsimile numbers and e-mail address of the contributor responsible for correspondence about the manuscript; should be mentioned.
- 7) The total number of pages, total number of photographs and word counts separately for abstract and for the text (excluding the references and abstract);
- 8) Source(s) of support in the form of grants, equipment, drugs, or all of these;
- 9) Acknowledgement, if any; and
- 10) If the manuscript was presented as part at a meeting, the organization, place, and exact date on which it was read.

## Abstract Page

The second page should carry the full title of the manuscript and an abstract (of no more than 150 words for case reports, brief reports and 250 words for original articles). The abstract should be structured and state the Context (Background), Aims, Settings and Design, Methods and Materials, Statistical analysis used, Results and Conclusions. Below the abstract should provide 3 to 10 keywords.

## Introduction

State the background of the study and purpose of the study and summarize the rationale for the study or observation.

## Methods

The methods section should include only information that was available at the time the plan or protocol for the study was written such as study approach, design, type of sample, sample size, sampling technique, setting of the study, description of data collection tools and methods; all information obtained during the conduct of the study belongs in the Results section.

Reports of randomized clinical trials should be based on the CONSORT Statement (<http://www.consort-statement.org>). When reporting experiments on human subjects, indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000 (available at [http://www.wma.net/e/policy/1\\_7-c\\_e.html](http://www.wma.net/e/policy/1_7-c_e.html)).

## Results

Present your results in logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations. Extra or supplementary materials and technical details can be placed in an appendix where it will be accessible but will not interrupt the flow of the text; alternatively, it can be published only in the electronic version of the journal.

## Discussion

Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying

mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

## References

List references in alphabetical order. Each listed reference should be cited in text (not in alphabetic order), and each text citation should be listed in the References section. Identify references in text, tables, and legends by Arabic numerals in square bracket (e.g. [10]). Please refer to ICMJE Guidelines ([http://www.nlm.nih.gov/bsd/uniform\\_requirements.html](http://www.nlm.nih.gov/bsd/uniform_requirements.html)) for more examples.

### Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. *J Oral Pathol Med* 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Kälestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. *Acta Odontol Scand* 2003; 61: 347-55.

### Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. *Dermatology* 1997; 195 Suppl 2: 3-9.

### Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. *J Periodontol* 2000; 71: 1792-801.

### Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. *Dent Mater* 2006.

### Personal author(s)

[6] Hosmer D, Lemeshow S. *Applied logistic regression*, 2<sup>nd</sup> edn. New York: Wiley-Interscience; 2000.

### Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. *Dental caries: The disease and its clinical management*. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

### No author given

[8] World Health Organization. *Oral health surveys - basic methods*, 4<sup>th</sup> edn. Geneva: World Health Organization; 1997.

### Reference from electronic media

[9] National Statistics Online – Trends in suicide by method in England and Wales, 1979-2001. [www.statistics.gov.uk/downloads/theme\\_health/HSQ\\_20.pdf](http://www.statistics.gov.uk/downloads/theme_health/HSQ_20.pdf) (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

More information about other reference types is available at [www.nlm.nih.gov/bsd/uniform\\_requirements.html](http://www.nlm.nih.gov/bsd/uniform_requirements.html), but observes some minor deviations (no full stop after journal title, no issue or date after volume, etc).

### Tables

Tables should be self-explanatory and should not duplicate textual material.

Tables with more than 10 columns and 25 rows are not acceptable.

Table numbers should be in Arabic numerals, consecutively in the order of their first citation in the text and supply a brief title for each.

Explain in footnotes all non-standard abbreviations that are used in each table.

For footnotes use the following symbols, in this sequence: \*, ¶, †, ‡,

### Illustrations (Figures)

Graphics files are welcome if supplied as Tiff, EPS, or PowerPoint files of minimum 1200x1600 pixel size. The minimum line weight for line art is 0.5 point for optimal printing.

When possible, please place symbol legends below the figure instead of to the side.

Original color figures can be printed in color at the editor's and publisher's discretion provided the author agrees to pay.

Type or print out legends (maximum 40 words, excluding the credit line) for illustrations using double spacing, with Arabic numerals corresponding to the illustrations.

### Sending a revised manuscript

While submitting a revised manuscript, contributors are requested to include, along with single copy of the final revised manuscript, a photocopy of the revised manuscript with the changes underlined in red and copy of the comments with the point to point clarification to each comment. The manuscript number should be written on each of these documents. If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks of submission. Hard copies of images should be sent to the office of the journal. There is no need to send printed manuscript for articles submitted online.

### Reprints

Journal provides no free printed reprints, however a author copy is sent to the main author and additional copies are available on payment (ask to the journal office).

### Copyrights

The whole of the literary matter in the journal is copyright and cannot be reproduced without the written permission.

### Declaration

A declaration should be submitted stating that the manuscript represents valid work and that neither this manuscript nor one with substantially similar content under the present authorship has been published or is being considered for publication elsewhere and the authorship of this article will not be contested by any one whose name (s) is/are not listed here, and that the order of authorship as placed in the manuscript is final and accepted by the co-authors. Declarations should be signed by all the authors in the order in which they are mentioned in the original manuscript. Matters appearing in the Journal are covered by copyright but no objection will be made to their reproduction provided permission is obtained from the Editor prior to publication and due acknowledgment of the source is made.

but no objection will be made to their reproduction provided permission is obtained from the Editor prior to publication and due acknowledgment of the source is made.

### Abbreviations

Standard abbreviations should be used and be spelt out when first used in the text. Abbreviations should not be used in the title or abstract.

### Checklist

- Manuscript Title
- Covering letter: Signed by all contributors
- Previous publication/ presentations mentioned, Source of funding mentioned
- Conflicts of interest disclosed

### Authors

- Middle name initials provided.
- Author for correspondence, with e-mail address provided.
- Number of contributors restricted as per the instructions.
- Identity not revealed in paper except title page (e.g. name of the institute in Methods, citing previous study as 'our study')

### Presentation and Format

- Double spacing
- Margins 2.5 cm from all four sides
- Title page contains all the desired information. Running title provided (not more than 50 characters)
- Abstract page contains the full title of the manuscript
- Abstract provided: Structured abstract provided for an original article.
- Key words provided (three or more)
- Introduction of 75-100 words
- Headings in title case (not ALL CAPITALS). References cited in square brackets
- References according to the journal's instructions

### Language and grammar

- Uniformly American English
- Abbreviations spelt out in full for the first time. Numerals from 1 to 10 spelt out
- Numerals at the beginning of the sentence spelt out

### Tables and figures

- No repetition of data in tables and graphs and in text.
- Actual numbers from which graphs drawn, provided.
- Figures necessary and of good quality (color)
- Table and figure numbers in Arabic letters (not Roman).
- Labels pasted on back of the photographs (no names written)
- Figure legends provided (not more than 40 words)
- Patients' privacy maintained, (if not permission taken)
- Credit note for borrowed figures/tables provided
- Manuscript provided on a CDROM (with double spacing)

### Submitting the Manuscript

- Is the journal editor's contact information current?
- Is the cover letter included with the manuscript? Does the letter:
  1. Include the author's postal address, e-mail address, telephone number, and fax number for future correspondence?
  2. State that the manuscript is original, not previously published, and not under concurrent consideration elsewhere?
  3. Inform the journal editor of the existence of any similar published manuscripts written by the author?
  4. Mention any supplemental material you are submitting for the online version of your article. Contributors' Form (to be modified as applicable and one signed copy attached with the manuscript)