EVALUATION OF REPLACEMENT GRAFT AND PUNCH GRAFTS IN THE TREATMENT OF VITILIGO

AJIT SINGH KUMAR,* R. C. SARIN † AND V. K. PURI ‡

Summary

Thirty cases of vitiligo each with minimum of two lesions underwent replacement graft and multiple punch grafts in one lesion each. Complications observed at the recipient site like infection and raised rugosed surface were significantly more in replacement grafts. Hypopigmentation of the graft was significantly more when the disease was progressive.

Vitiligo is a scintillating problem. Not merely that its etiology is enigmatic, its treatment is also in a doldrum phase, though certain drugs have gained significant importance in its therapy. The defect in vitiligo is lack of melanin pigment in the area affected, and this may be due to exhaustion of melanocytes, blockade of formation of melanin or disappearance of melanocytes. Neurochemical factors, auto-immunity, metabolic factors or self-destructive process by the melanocytes. themselves are all considered as having a possible etiological role in this disease.

Since the demonstration of recipient site dominance in vitiligo,[1,2,3] surgical measures like replacement grafts have not been much in vogue. In the recent years it has been felt that the phenomenon of recipient site dominance depends on the nature of the disease with regard to the progressive or stationary state of the disease[4,5]. Multiple punch grafts have been favoured over replacement graft as punch grafts can take easily and spread of pigmentation can occur around individual grafts[6,7].

The present study has been undertaken to find out whether autograft is cosmetically helpful, etiopathogenesis can be sorted out and how far punch grafts can be utilized advantageously. Many a time proper matching pigment skin is not easily available in these patients.

Material and Method :

Thirty cases suffering from vitiligo were selected at random from the Department of Skin & S. T. D. of Shri Guru Tegh Bahadur Hospital, Amritsar during the years 1977 and 1978. Cases selected were equally divided into two groups of fifteen patients ; both groups having equal numbers of progressive and stationary cases. The disease process was considered stationary if the disease had not progressed with appearance of new patches or enlargement of existing patches for atleast one year preceding the date of admission into the study. Patients selected were not given any specific medication for vitiligo during the period of study.

* Registrar in Skin & S.T.D. Department
† Professor, Head of Skin & S.T.D. Department
‡ Assistant Professor, Plastic & Reconstructive Surgery, Medical College/S. G. T. B. Hospital, Amritsar.

Received for publication on 22—2—1979.

In each case, history was recorded with special reference to mode of onset, nature of disease and treatment taken. Results of examination were recorded with special reference to sites involved, hyperpigmented borders, leucotrichia and color.

In each case autografting was carried out. Out of two lesions demarcated for grafting, replacement graft after excision of whole lesion was done in one. In the other, multiple punch excisions were made in the vitiliginous patches which were then covered with an equal sized and equal number of punch grafts (Photograph). Grafting was conducted under general anaesthesia in eleven cases, spinal anaesthesia in thirteen cases and local anaesthesia in six cases.

The grafted sites were immobilised for about a week. Systemic antibiotics were given to prevent any secondary infection. Dressings were changed on 7th day. After discharge from the hospital, the patients were followed up weekly for 4 weeks and monthly for a total period of 3 months. At follow up each grafted site was examined for presence of any infection, loss of graft, alteration in colour, nature of surface, formation of any keloid and spread of pigmentation around punch grafts.

Donor area was medial aspect of thigh in twenty six cases and legs in 4 cases. These areas healed in 7–15 days and at follow up were examined for any evidence of hypopigmentation, hyperpigmentation, infection or keloid formation.

Observations : General

Out of 30 cases studied, 13 were females and 17 males. Their ages ranged between 8 years and 70 years. 23 cases (76.60%) were in the age group of 11-30 years. Duration of the disease varied from one month to 22 years. In 10 cases (33.3%) it was more than 5 years. 27 cases (90%) were of multifocal(M) type (M. acrofacialis 20 cases and M. vulgaris 7 cases) and 3 of unifocal (U) type (U. areata 2 cases and U. zosteriformis 1 case) as per classification of Dutta et al,[8] and Sarin and Kumar[9].

Sites selected :

Sites selected for replacement and punch grafts were on lower extremities in 24 cases, upper extremities in 4 cases and trunk in 2 cases. 17 sites had stationary lesions and thirteen sites had progressive ones.

Replacement graft :

The lesions selected for replacement graft ranged in size between 0.8 — 227.5 sq. cm. In 15 cases it was less than 10 sq. cm in area. Grafts took in 7–15 days. Complications developed in 26 cases singly or in combinations and were infection in 8, raised rugosed borders and/or surface in 20, loss of graft, keloid and leucotrichia in one each and hypopigmentation in 6 cases.

Punch grafts :

The lesions ranged in size between 1.4 sq. cm. and 37.5 sq cm. The size of punch graft ranged between 2 mm and 1.2 cm. In 25 cases the size of the graft was 4 mm each. A total of 185 punch grafts were implanted in 30 lesions. The number of punch grafts varied between 2 and 11 in individual cases – 10 cases received 6 to 11 punch grafts. These punch grafts were made equidistant. Punch grafts took in 7–14 days. Complications observed in 21 cases singly or in combinations were, raised rugosed surface in 7, hypopigmentation in 13 (out of 91 punch grafts in them 54 developed hypopigmentation), hyperpigmentation in 2 and infection in 1. Spread of pigment around punch grafts occurred in 7 cases and was of the range of 1–2 mm in 3 months (Table 1).

TABLE 1

Showing complications in replacement graft versus punch grafts

Complications	Replacement graft		Punch grafts	
	No. of cases	%	No. of cases	%
(a) Infection*	8	26.7	1	3.3
(b) Keloid formation	1	3.3	0	0
(c) Raised surface*	20	66.7	7	23.3
(d) Loss of graft	1	3.3	0	0
(e) Hypopigmentation	6	20.0	13	43.3

* Difference is statistically significant.
(a) $p < 0.05$
(c) $p < 0.001$

TABLE 2

Showing hypopigmentation in graft versus nature of disease

Grafts	Number of cases				Total
	Lesions		Disease		
	Prog.	Staty.	Prog.	Staty.	
(a) Whole graft	1	1	1	1	2
(b) Punch grafts	5	4	6	3	9
(c) Both grafts	2	2	4	0	4
Total	8	7	11	4	15

Prog. = Progressive
Staty. = Stationary

Hypopigmentation developed in 15 patients (19 lesions) out of a total of 30 (60 lesions) who had punch grafting. Among these 15 cases, 11 had progressive disease, but 5 lesions selected for grafting had become stationary (Table 2).

Donor area:

Complications were observed in 28 cases in the form of hypopigmentation in 18 cases, hyperpigmentation in 13 cases, keloid in 5 cases and infection in 1 case.

Comparative observations:

Significant hypopigmentation of the graft was observed when the disease (50% cases) or the lesion (38.5% cases) was progressive. Hyperpigmentation in punch grafts occurred only in progressive disease or lesion. Spread of pigmentation around punch grafts was also more often seen when the disease or lesion was progressive (Table 3).

Discussion:

The reported incidence of vitiligo in various dermatological clinics in India varies from 3.5% to 4.3%[8,10]. The incidence of this disorder among our patients during the period of the present study was about 1%.

Behl et al[4] and Gopinathan[5] had confuted the recipient site dominance on autografting reported earlier[1,2,3] and on the basis of their experience concluded that activity of disease in general and that of the lesion in particular would have an important bearing on the behaviour of the graft. In the present

TABLE 3

Showing comparative observations on pigmentation in progressive vs. stationary cases and lesions

	Progressive			Stationary		
	No. of cases	Total No.	%	No. of cases	Total No.	%
Graft :						
* (a) Hypopigmentation	15	30	50	4	30	13.3
(b) Hyperpigmentation	2	15	13.3	0	15	0
(c) Spread of pigment	4	15	26.7	3	15	20.0
Donor Area :						
(a) Hypopigmentation	8	15	53.3	10	15	66.7
(b) Hyperpigmentation	7	15	46.7	6	15	40.0
		LESION				
Graft :						
(a) Hypopigmentation	10	26	38.5	9	34	26.5
(b) Hyperpigmentation	2	13	15.4	0	17	0
(c) Spread pigmentation	4	13	30.8	3	17	17 6

* Difference is statistically significant $p < 0.01$.

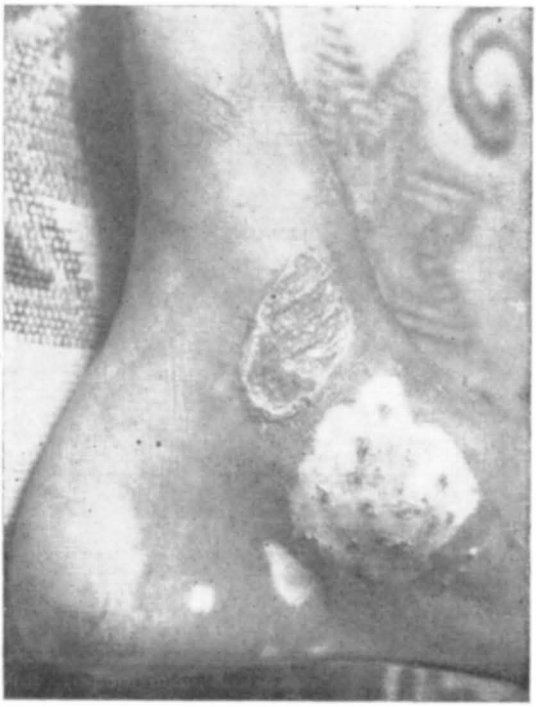

Fig. Photograph showing replacement graft and punch graft at 3 wks

study out of the 60 lesions in 30 cases, who underwent full thickness and punch grafts, in 19 lesions in 15 cases hypopigmentation developed. It was noted that the ultimate behaviour as regards development or otherwise of hypopigmentation of the graft depends on the progressive or static nature of the disease.

Among the 15 cases who showed recipient site dominance, 11 had progressive disease. In other words out of a total of 15 cases with progressive nature of disease, hypopigmentation of replacement or punch or both grafts occurred in 11 cases (73.3%) and out of 15 cases in which the disease was stationary either of the grafts became hypopigmented in 4 cases (26.7%). A disease like vitiligo has a chronic course and often shows intermittent activity. It is possible that the 4 cases thought to be stationary at the time of grafting were really

not entirely stationary. Behl[11] selected 107 very carefully screened stationary cases for autografting and reported depigmentation of graft in 10 (9.3%).

Haxthausen[1] had stated that loss of pigmentation in the graft in his cases appeared gradually all over the grafts, whereas in the reports of Behl and Gopinathan[5] the cases which developed hypopigmentation did so from the margins of the graft giving an impression as of something nibbling at the pigment from periphery towards the centre.

This recipient site dominance observed in present series also indicates that certain local factor or factors are involved in the etiopathogenesis of vitiligo. The nature of such factors remains conjectural at this stage. Altered neurotrophic influence was propounded by Haxthausen[1]. Altered immune response of the melanocytes may be a factor such as is seen in fixed drug eruption or graft rejection.

Trauma in predisposed individuals may induce vitiligo as a Koebner isomorphic response[12,13,14]. Behl et al[4] reported development of depigmentation at abrasion sites on normal skin in vitiligo in 8 (8%) out of 100 cases. Gopinathan[5] reported development of hypopigmentation in 67% sites close to the lesion and 38% sites at distant sites in 13 vitiligo cases. He further concluded that epidermodermal trauma was necessary to produce depigmentation.

In the present series hypopigmentation on donor sites developed in 18 (60%) cases. Nature of the disease had no influence on the development of hypopigmentation. The trauma had been epidermodermal. It is difficult to say that trauma was a contributory factor as the depigmentation started at the margins of the graft and then gradually spread towards the centre.

Orentreich and Selmanowitz[7] reported spread of pigmentation upto 1 mm irrespective of the size of the graft. In the present series in 7 lesions out of 30 which were punch grafted the spread of pigmentation in 3 months' time ranged between 1–2 mm. This spread of pigmentation did not appear around all the punch grafts in a lesion. In one case some punch grafts became hypopigmented even while other showed spreading pigmentation. Evidently pigment spread is controlled by some remote intricate factors and therefore preoperative planning for spacing of the grafts is not possible.

Replacement grafts took on an average a little longer time (8.9 days) as compared to punch grafts (7.8 days) as expected[6]. Complications singly or in combinations were more in replacement grafts (26 cases) as compared to punch grafts (21 cases).

Infection was significantly more in replacement grafts. No doubt this complication is preventable by using better measures of asepsis. In the present series both grafting methods were employed at the same sitting in the same patient by the same persons from the same donor areas. The size of the graft thus seems to be a determining factor as regards infection which led to loss of replacement graft in one case. No punch graft was lost.

Raised rugosed margins were more common in replacement grafts (20 cases) as compared to punch grafts (7 cases). Infection, application of stitches to the margin or type of dressings used appeared to have no bearing on the occurrence of this complication. The period of follow up had been only 3 months which is too short a period for a final assessment. Such a complication was observed only in 3 cases (2.8%) by Behl[11].

References :

1. Haxthausen H : Studies on the pathogenesis of morphoea, vitiligo and acrodermatitis atrophicans by means of transplantation experiments, Acta Dermat Venereol, 27 : 352, 1947.
2. Comel M (1948) : Quoted by Rothman S : Physiology and Biochemistry of Skin. The University of Chicago Press, Chicago and London, 1965, p 540-541.
3. Spencer GA and Tolmach JA : Exchange grafts in vitiligo, J Invest Derma, 19 : 1, 1952.
4. Behl PN, Agarwal RS and Singh G : Aetiological studies in vitiligo and therapeutic response to standard treatment, Indian J Dermat 6 : 101, 1961.
5. Gopinathan T : A study of the lesions of vitiligo, Arch Derm, 91 : 397, 1965.
6. Orenteich N : Hair transplantation – The punch graft technique, Surg Clin N Amer, 51 : 511, 1971.
7. Orentreich N and Selmanowitz VJ : Autograft repigmentation of leukoderma, Arch Derm 105 : 731, 1972.
8. Dutta AK and Mandal SB : A clinical study of 650 vitiligo cases and their classification, Indian J Dermat, 14 : 103, 1969.
9. Sarin RC and Kumar AS : A clinical study of vitiligo, Indian J Derm Ven Lepr 43:311, 1977.
10. Dutta AK : Studies on vitiligo with special reference to neural concept, Indian J Dermat Ven Lepr, 43 : 190, 1977.
11. Behl PN : Treatment of vitiligo with homologous thin Thiersch skin grafts, Curr Med Pract 8/4 : 218, 1964.
12. Lerner AB, Case JD : Pigment cell regulatory factors. J Invest Derm 32 : 211, 1959.
13. Fitzpatrick TB and Mihm MC : Abnormalities of the melanin pigmentary system, Dermatology in General Medicine, Mc Graw Hill Co. Blackiston Publication. New York Ed 1, 1971. p 117
14. Coondoo A, Sen N and Panja RK : Leucoderma of lips, Indian J Derm, 21:29, 1976.