CHILDHOOD VITILIGO – EPIDEMIOLOGY, CLINICAL SPECTRUM AND THERAPEUTIC RESPONSES

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(BRANCH XII A)

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CERTIFICATE

Certified that this dissertation titled **"CHILDHOOD VITILIGO – EPIDEMIOLOGY, CLINICAL SPECTRUM AND THERAPEUTIC RESPONSES"** is a bonafide work done by **Dr. M. RANGARAJ,** Post graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2009 – 2012. This work has not previously formed the basis for the award of any degree.

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CONTENTS

INTRODUCTION

Vitiligo is a common dermatologic disorder in children and one that has been observed since ancient times. It is characterized by asymptomatic, well-demarcated, ivory-white macules and patches that may be localized or generalized. Vitiligo is common in India affecting 3-4% of Indian population[2]. Childhood vitiligo is a special subtype and is seen in significant proportion of vitiligo patients. There are only a few clinical studies in the past which address the clinical spectrum of vitiligo in children.

This study on eighty cases of childhood vitiligo will cover the epidemiology, clinical spectrum and review its therapeutic responses. This study was undertaken in view of the seriousness of the problem in children.

REVIEW OF LITERATURE

DEFINITION

Vitiligo is an acquired primary, usually progressive melanocytopenia of unknown etiology, clinically manifested by circumscribed achromic macules often associated with leukotrichia and histologically by degeneration and disappearance of melanocytes in the involved skin and not uncommonly in the pigment epithelium of the eyes, leptomeninges and inner ear.

The disease affects subjects of either sex with a heritable constitutional predilection.

SYNONYMS

Sufaid Bagh	Phubhari	Phuleri
Switra	Bars	Bahak
Kilas	Palita	Kodha
Sweta Kushta	Dhawal	Kustha

HISTORY

The origin of the term vitiligo is obscure, like the disease itself. Some believe that it is derived from the Latin words ‘vitelius’ meaning

vale, i.e. pale pink flesh of calf, since the clinical lesions resembled the white patches of a spotted calf, while other believed that it originated from the Latin word ‘vitium’, meaning blemish. The Roman physician Celsus first used the term vitiligo in the second country AD. It is interesting to note that the Rigveda (6000 BC or earlier) named leukoderma as Kilas, meaning a white spotted deer[2].

EPIDEMIOLOGY

The highest incidence has been recorded in India and Mexico. Based on the studies done so far, it is roughly estimated to be 3-4% in India[2].

INCIDENCE AND HOST FACTORS

There is no particular sex predilection. Female predominance probably reflect their greater concern for cosmetic disfigurement and related to the social and marital problems.

Vitiligo mostly affects people with Fitzpatrick’s skin types III and IV[2].

The disease may start at any stage. Onset of the unilateral dematomal type is usually in childhood within 10 years of age whereas

most cases with bilateral non- dermatomal lesions (vitiligo vulgaris type) begin in the second to fourth decades of life. A few instances of vitiligo lesions present at birth have been reported as cases of congenital vitiligo[5].

Koebner's phenomenon is observed in 6-20% of cases of vitiligo vulgaris[2]. Minor trauma such as scratch mark, laceration, or stitches on the skin results in the development of a corresponding linear depigmented macule, usually in 2-4 weeks. This isomorphic phenomenon indicates an abnormal pattern of cutaneous response to trivial physical trauma.

HEREDOFAMILIAL ASPECTS

At present the consensus is that the familial incidence is between 20% and 30%[2].

VITI gene, which maps to chromosome 2p16, has also been associated with vitiligo[2].

A familial incidence of diabetes mellitus and thyroid disease has been commonly noted in cases of vitiligo. Atopy is another familial association.

ETIOLOGY:

THEORIES ON THE PATHOGENESIS:

Theories on the pathogenesis of vitiligo centered on mechanisms for the destruction of melanocytes as there are no melanocytes present in the fully evolved white macules. Traditionally there have been three hypothesis to explain vitiligo.

1. Neural Hypothesis

2. Self destructive Theory

3. Autoimmune Theory

1. NEURAL HYPHOTHESIS[8,9]

Evidences in favour of neural hypothesis include:

Stress and emotional trauma is a known initiating or precipitating factor in vitiligo, the common embryologic origin of melanocytes and the nervous system, dermatomal distribution of segmental vitiligo, demonstration of direct contact between cutaneous free nerve endings and epidermal melanocytes in vitiligo macules, demonstration of neuropeptides in the skin and their ability to regulate melanocyte differentiation has given more strength to this hypothesis.

These alterations are said to induce melanocyte dysfunction and melanocyte injury by promoting the production of melanocytotoxic compounds and by decreasing the natural detoxification.

At present, however the role of nervous system in vitiligo, if any, is poorly understood.

2. SELF DESTRUCTIVE THEORY [9]

A. B. Lerner postulated that melanocytes in vitiligo have lost an intrinsic protective mechanism that eliminates toxic intermediates or metabolites in the melanogenesis pathway.

Melanocytes synthesize melanin by oxidation of tyrosine to dihydroxyphenylalanine (DOPA) and to dopaquinone, which by a multistep reaction forms indoles. All the intermediates in the biosynthesis of melanin are phenols, excessive production or accumulation of phenolic radicals or intermediates within the melanocyte could damage the cell[10].

It has been suggested that melatonin receptor and melatonin could play a key role in vitiligo. Melatonin is known to stimulate the melanogenic pathway without the production of melanins, leading to an accumulation of toxic intermediates which causes injury to keratinocytes

and melanocytes with release of specific cellular proteins that initiate a secondary autoimmune reaction.

The presence of high levels of Hydrogen peroxide (H2O2) and low levels of catalase[11] in epidermis of vitiligenous skin suggests that there is an increased oxidative stress in vitiligo patients[12]. Several pathways could be involved in overproduction of H2O2 in vitiligo[13].

An abnormality in tetrabiopterin metabolism leads to defective recycling of 6BH4 causing formation of H2O2.

Over production of H2O2 with increased levels of monoamine oxidase A from inhibition of thioredoxin / thioredoxin reductase by calcium and increased nitric oxide synthase activities.

3. AUTOIMMUNE THEORY[14]

The association of vitiligo with autoimmune diseases suggested an immunologic basis for vitiligo.

A. Humoral immunity

There is an increased frequency of organ-specific auto antibodies in patients with vitiligo, even in the absence of any associated disease in

up to 30% of patients. Antibodies to thyroid tissue, gastric parietal cell, adrenal cytoplasm and pancreatic islet cell have been demonstrated.

More recently, autoantibodies to a transcription factor called SOX10 have been found in vitiligo associated with APECED.

B. Cell mediated immunity

In marginal skin from progressive lesions of generalized and inflammatory vitiligo, an infiltrate of skin-homing (CLA+) cytotoxic T cells expressing granzyme/perforin is often found close to the remaining melanocytes. This infiltrate is composed of CD8 T cells, CD4 T cells and subsets of macrophages, and this correlates with the increased number of CLA+ MART-1 reactive CD8 T cells in the peripheral blood of patients with progressive vitiligo. These specific cytotoxic T cells react against the melanocyte differentiating antigens in vitiligo patients[14].

4. OTHER HYPOTHETICAL THEORIES

A. Convergence theory suggests that genetic factors, stress, accumulation of toxic compounds, infection, autoimmunity, mutations, altered cellular environment, and impaired melanocyte migration and proliferation can all contribute to this disease[15].

B. An intrinsic defect of the structure and function of rough endoplasmic reticulum in melanocytes of vitiligo patients[16].

C. Deficiency of Melanocyte growth factor.

D. Viral origin.

E. Dysregulation of Melanocyte apoptosis.

CLINICAL FEATURES[7]

A typical lesion is a well defined depigmented (milky white or chalky) macule, round to oval in shape, to fairly distinct often with scalloped margins, measures from few mm to many cms in diameter, showing a variable number of depigmented (white) hairs and without any change in the skin texture.

The number, size, shape, and location of individual macules vary widely. Frequently the initial macule occurs on the exposed areas (such as the dorsal surface of hands, elbows, feet, legs, knees, neck and face), body folds (such as axillae, groin, and sub mammary region in women), lips or genitalia.

The initial unifocal lesion may be followed by the appearance of new lesions elsewhere. In less than 25% of cases the onset may be multifocal[2]. Onset of the lesions is usually insidious. The disease is progressive in nature as a rule and course is virtually unpredictable and may be quite erratic, it may be jerky, indolent or rapid. While some lesions may show signs of repigmentation, new lesions may develop on other parts of the body simultaneously. There is an episodic phase of rapid extension of lesions after remaining quiescent over a long period of time.

Although no definite precipitating factor is ascertained, many factors have been incriminated which include local trauma, itching, friction, infection, infestations, gastrointestinal disturbances, emotional upset, pregnancy, parturition and surgery.

Morphological variants[22]:

Trichrome vitiligo shows 3 different colours, the depigmented centre surrounded by an intermediate tan colour which is inturn surrounded by normal skin colour.

Quadrichrome exhibits a additional fourth colour i.e., dark brown which is either perifollicular (or) marginal,to the trichrome variety.

Pentachrome vitiligo exhibits 5 shades of colours white, tan, brown hyperpigmented, blue grey hyperpigmented and normal.

INFLAMMATORY VITILIGO

Shows erythematous raised border and may be associated with pruritus.

VALECEO VITILIGO[2]

In most instances extension of vitiligo is gradual, whereas in valeceo vitiligo there is a very rapid extension and spread of lesions.

CLASSIFICATION OF VITILIGO[7]

LOCALIZED

Focal vitiligo

usually a solitary macule or a few scattered macules in one area, most commonly in the distribution of the trigeminal nerve, although the neck and trunk are also commonly involved.

Segmental Vitiligo

Unilateral macules in a dermatomal or quasi dermatomal distribution is present. This tends to have an early age of onset and unlike

the other types, is not associated with thyroid disease or other autoimmune disease. This type occurs more commonly in children[7]. Alteration of neural peptides has been implicated in the pathogenesis of this type. More than one half of the patients with segmental vitiligo have patches of white hair, known as poliosis[7].

Mucosal Vitiligo

Involvement of the mucous membrane sites (oral, genital and anal) alone occurs.

GENERALIZED

Vitiligo Vulgaris

It is the commonest type characterized by scattered macules distributed symmetrically involving extensor surfaces of trunk, extremities, periorificial and mucosal sites.

Acrofacial

Involves distal digital and periorificial areas.

UNIVERSAL TYPE

Complete (or) Nearly complete depigmentation is seen. This type is commonly associated with multiple endocrinopathies[7].

LIP TIP TYPE

Involves periungual areas alone (or) with certain mucosal surfaces (lip, nipple, distal penis).

INVOLVEMENT OF OTHER PIGMENT EPITHELIUM

Various ocular abnormalities chiefly involving retinal pigment layers have been noted among many vitiligo patients . In the retina, the pigment layers such as the inner pigment epithelium adjacent to the neuroretina and the outer choroids may show evidence of destruction of pigment cells giving the retina a 'tigroid appearance'[2]. The commonest eye lesions are scars due to the destruction of the pigment layers[2].

ASSOCIATED SKIN DISORDERS[2]

- canities
- alopecia areata
- halo nevus
- dermatitis herpetiformis
- twenty nail dystrophy
- atopic eczema
- psoriasis
- scleroderma

- lichen planus
- lichen simplex
- discoid lupus erythematosus
- malignant melanoma
- ichthyosis.

ASSOCIATED SYSTEMIC DISORDERS

- Pernicious anemia
- Addition's disease
- Grave's disease
- hyperthyroidism
- hypothyroidism
- hyperparathyroidism
- diabetes mellitus

More than 10% of patients with pernicious anemia have been reported to develop vitiligo[2]. In a different series, vitiligo has been reported to be associated with thyroid disorders in 0.6% - 38% of patients[2]. Diabetes mellitus was reported to occur in 1%-7% of vitiligo patients[2]. An intimate relationship between vitiligo and melanoma has been reported in one study.

SYNDROMES ASSOCIATED:

Vogt-Koyanagi-Harada syndrome: It is a rare syndrome affecting children, especially of south-east Asian origin. Characteristic features are uveitis, aseptic meningitis, dysacusia, alopecia, poliosis and vitiligo. Uveitis is the presenting feature and vitiligo may appear later, during the chronic stage (fourth stage) of the disease (adolescence or adulthood). Vitiligo lesions tend to be symmetrical, involving the head, neck and trunk. The sacral region is a common site of involvement with vitiligo. Poliosis may involve the scalp, eyebrows and eyelashes.

Alezzandrini's syndrome: This syndrome is characterized by segmental vitiligo(cheek), poliosis, ipsilateral uveitis resulting in decreased visual acuity and same-sided partial hearing loss. Manifestation starts during adolescence.

PATTERN & DISTRIBUTION:

Vitiligo can exhibit 2 general pattern unilateral with a dermatomal distribution or bilateral which is very common. Although vitiligo can occur on any part of the body, there are characteristic patterns of involvement. The most frequently involved sites are the face, dorsum of

hands, axilla, umbilicus, nipples, sacrum and inguinal region. It also commonly involves sites of friction - back of hand, elbow, knee, ankles, shoulder strap and waist band areas, other commonly involved sites are scalp and neck. Palms and soles involvement is common. Mucosal involvement is frequent, the genitalia, gingiva and lips may be involved.

VITILIGO VULGARIS

It is the most common type[7]. The lesions may occur at various body sites often bilaterally, the lesions may be either symmetrical (or) asymmetrical.

SEGMENTAL VITILIGO

It is characterized by unilateral macules in a dermatomal distribution. It tends to be earlier in onset. 20% of children have this pattern [3].It is more stable. It is not familial and is unlikely to be associated with other autoimmune diseases. Koebnerization is not characteristic. Trigeminal area is the commonest site more than 50% of cases. Neck and Trunk involved in 23% and 17% respectively. Upto 13% may have multiple sites of involvement[2].

FOCAL

20% of children with vitiligo have focal pattern.

ACROFACIAL

It is the least common type in children[23].

HAIR IN VITILIGO

Depigmentation of body and scalp hair occurs in 9-45% of vitiligo patients[3]. Nearly 50% of segmental vitiligo arc associated with poliosis. The perifollicular and interfollicular skin is affected. Leukotrichia indicates poor prognosis.

HISTOPATHOLOGY:

Histopathology as a means of diagnosis is rarely employed in vitiligo but it useful when other cause of hypopigmentation needs to be excluded. The histopathological changes classically associated with vitiligo include a complete absence of melanocytes in the basal layer of the epidermis with loss of melanin in the epidermis. The upper dermis often has sparse superficial perivascular collection of lymphocytes with a few melanophages.

On H& E stained sections melanocytes are recognized as randomly dispersed cells within the basal layer with a small rounded darkly staining nucleus and a clear cytoplasm as a result of shrinkage artifact.

COURSE OF THE DISEASE

The course of the disease is unpredictable and uncertain, most often showing a tendency towards slow progression. Spontaneous repigmentation is noted in about 10-20% of patients, most frequently in sun-exposed areas and in younger patients. In vitiligo vulgaris, lesions develop on different areas in succession with varying rapidity. In some, extension of individual lesions and development of new lesions at different sites occur in episodic bouts with the intervening quiescent period varying from weeks to years. Many lesions may remain static for an indefinite period or show some degree of spontaneous regression with the development of spotty repigmentation. Sometimes residual depigmentation may be left behind after repigmentation of a large macule.

DIFFERENTIAL DIAGNOSIS

- post inflammatory hypopigmentation
- pityriasis alba
- indeterminate hansen
- pityriasis versicolor
- post kala azar dermal leishmaniasis
- chemical leukoderma
- piebaldism
- idiopathic guttate hypomelanosis
- naevus depigmentosus
- albinism
- ash leaf macule
- waardenburg syndrome
- woolf's syndrome
- halo naevus
- syphilis and yaws
- lupus erythematosus
- incontinentia pigmenti

PROGNOSIS

These factors indicate poor prognostic factors:

1) Lesions on the so-called resistant sites, such as bony prominences, non-fleshy areas, non-hairy areas and mucosal areas. They comprise the sides of the ankles, front of the wrists, back of the elbows, dorsum of feet and hands, palms, soles, nipples and areola.

2) The greater the percentage of associated white hair, the worse the prognosis.

3) Extensive long-standing disease.

4) Associated systemic disorders.

5) Familial background.

6) Old age.

7) Iatrogenic factors, including injudicious administration of topical and systemic medication, particularly photochemo-therapeutic agent)

TREATMENT MODALITIES

Treatment of vitiligo at any age remains a challenge for clinicians, more so during childhood. None of the available therapies is absolutely effective, and the disease runs a relapsing course. With any of the treatment modalities, >75% repigmentation (achieved by approximately 60% of treated children) is considered as the best therapeutic response[34].

Vitiligo beginning in childhood can be associated with significant psychological trauma that may have lasting effects on the persons selfesteem. Since vitiligo is not a life threatening cutaneous condition many children with little skin involvement in cosmetically unimportant areas do not wish to treat their disease.

Few studies have assessed therapeutic responses in paediatric patients with vitiligo. Presently there is no universally effective medical or surgical modality for vitiligo. However there are number of active therapeutic approaches that are known to be effective. Most repigmentation therapies are directed to stimulate the melanocytic reserves in the hair follicle. Non surgical repigmentation therapies represent the first line active treatment modality in vitiligo[44]. If the area to

be treated is small less than 20% of the body surface, topical therapy is indicated[7]. If the area is larger than this systemic therapy is indicated. In addition to these medical and surgical treatments broad-spectrum sunscreens and cosmetic camouflage are also useful.

1. MEDICAL

a) TOPICAL

- Corticosteroids
- calcineurin inhibitors
- calipotriol
- pseudocatalase
- 5-fluorouracil
- Basic fibroblast growth factors[2]
- Human placentral extract
- Combination

b)SYSTEMIC

- Oral mini pulse therapy with betamethasone/ methylprednisolone
- Cyclophosphamide
- Azathioprine
- Levamisole

2. PHOTOTHERAPY

- Oral PUVA
- PUVASo1
- Topical PUVA
- Pseudocatalase and calcium chloride with UVB
- Narrow band UVB phototherapy
- Khellin with UVA
- Phenylalanine and UVA
- Calcipotriol with PUVA therapy
- Minoxidil and PUVA

3. SURGICAL

- Autologous mini punch grafts
- Autologous suction blister grafts
- Thiersch grafts
- Micropigmentation (tattooing)
- Therapeutic dermabrasion
- Cultured and noncultured epidermal cell transplantation
- Autologous noncultured melanocyte keratinocyte transplantation.

4. ADJUNCTIVE / ALTERNATIVE THERAPIES[4]

- Excimer laser
- Broad spectrum sunscreens
- Cosmetic camouflage
- Psychological counseling

5. DEPIGMENTATION

- 20 % Monobenzylether of Hydroquinone cream.

TOPICAL CORTICOSTEROIDS

Topical steroids are often the first treatment of choice for young patients with vitiligo because they arc easy and convenient for both doctors and patients to maintain the treatment.

MECHANISM OF ACTION

Corticosteroids cause repigmentation in vitiligo by decreasing the level of vitiligo antibodies. Locally they suppress the immunological changes allowing inactive melanocytes to effect repigmentation.

SELECTION OF PATIENTS

Age

Topical steroids are useful in children less than 10 yrs of age who are too young for PUVA therapy. They can also be considered in children less than.

Type of Vitiligo

Segmental vitiligo shows poor response. But partial or complete response is seen with Non dermatomal localized vitiligo.

Site

Lesions on the face and neck respond better[2]. Even segmental vitiligo on the face responds well. Vitiligo on the trunk and limbs show moderate response. But vitiligo involving palms, soles, fingers show poor response.

Duration of the disease

Younger lesions tend to respond better especially lesions of less than 3 months duration.

TREATMENT PROTOCOLS:

Topical therapy using 0.1% betamethasone valerate is successful in repigmenting some vitiliginous lesions. Low to mid potency steroids are used in young children. High potency preparation can be used in older children. They should be applied only to limited (or) localized areas of involvement.

Steroids should be applied two times daily for 3wks, then skipped for 1 week and then two times daily as above for 2 months. If there is no response it is discontinued. If there is good response it is continued along with monitoring every 2 months for every side effects. The earliest response is seen after 3 wks. The duration of treatment strongly depends on the response. The mean duration of treatment varies from 5 to 8 months. It can be even prolonged for a period of 21 months. An acceptable response is regarded as 50% or greater return of pigmentation to all involved skin areas. A good response occurs in 30-50% of patients.

SIDE EFFECTS

Atrophy , Striae ,Telangiectasia, Glaucoma if used for a prolonged period on eyelid, Hypothalamic - pituitary adrenal axis suppression, if used for a prolonged period.

SYSTEMIC STEROIDS

Oral minipulse therapy with betamethasone

This pulse therapy seems to be beneficial in the treatment of vitiligo by it's immunomodulatory effect.

Mechanism of Action:

Steroids prevent damage to the melanocytes in the vitiligo by decreasing levels of offending complements and antibody mediated cytotoxicity against melanocytes.

Selection of Patients

Child with extensive and rapidly spreading vitiligo.

Treatment Protocol

Tab.Betamethasone 5mg is given as a single oral dose after breakfast on two consecutive days each week. The response is evaluated every 2-4 months and the side effects are recorded.

RESPONSE TO TREATMENT

The treatment duration for oral minipulse therapy varies between 6 and 24 months. Treatment is discontinued after all depigmented areas regained pigment or when there has been no further repigmentation for

atleast 4 months. It arrests the progression of vitiligo within 1-3 months in 89% of children on treatment. Within 2-4 months 80% of the patients show spontaneous repigmentation of existing lesions which progresses with continued treatment. The extent of repigmentation varies indifferent patients and even in different lesions in the same patient. It varies from 10% to 90%.

SIDE EFFECTS

Weight gain, headache, transient mild weakness, acne, cushing's disease, diabetes mellitus, perioral dermatitis, glaucoma, cataract, herpes zoster.

CONTRAINDICATIONS

- Diabetes mellitus, tuberculosis, gastro intestinal disease, glaucoma, hypertension and osteoporosis.

INVESTIGATIONS

- Blood Sugar
- X-ray chest

Topical Tacrolimus and Topical Pimecrolimus

Tacrolimus is a macrolide lactone produced by Streptomyces tsukubaensis. Tacrolimus acts on the immune system and directly on skin cells. It binds to a receptor within the cell called the FK binding proteins. This resulting drug-protein complex inhibits calcineurin (a calcium-dependent phosphatase transmitting chemical) that in turn reduces the activity of T-lymphocytes in the immune system. As a consequence, T-cells fail to release their cytokines.

Tacrolimus by its immunomodulating activity found to be useful in treating vitiligo. It is available as 0.03% and 0.1% ointment and it has to be applied twice daily for a period of 3 to 6 months, and recent reports claimed it as a success even when used alone as a monotherapy. Pimecrolimus is available as 1% cream.

NARROW BAND UVB PHOTOTHERAPY

Narrow band UVB therapy is more effective than PUVA therapy in terms of repigmentation4. Other advantages are: (a) since no oral medication is required, no side effects of psoralens are seen; (b) it can also be used in pregnancy and childhood; and (c) no post-exposure eye

protection is necessary; and (d) exposure time is shorter than with PUVA. Hence, narrow band UVB therapy is now considered the treatment of choice for stable vitiligo. Narrow band fluorescent bulbs of Philips TL-QJ, each of 100W with an emission spectrum of 311 nm are used.

Response to treatment correlated with localization of the lesions and the patient's compliance. The risk of skin cancer with narrow-band UVB is unknown at present and there are insufficient human data available to provide recommendations. If no response is observed after 6 months, further therapy should be discouraged4 or the treatment be limited to specific areas Targeted UVB or focussed microphototherapy with a spectrum of 280 to 315 nm also has excellent results but the cost is prohibitive and the size of the area to be treated is restricted.

SURGICAL MODALITIES

MINIATURE PUNCH GRAFTING

This procedure consist of taking 1.5-2.5mm sized punch autografts from normal skin and grafting them in appropriately space and punched out chambers at the recipient site.

TEST GRAFTING

Trial grafting in a small area of vitiligo should be undertaken with 4-5 minigrafts to confirm the stability of the disease before attempting repigmentation in other involved areas4.

PROCEDURE

Give premedication (antibiotic) and calculate the approximate number of required grafts after spaced markings at the recipient site. For a large patch, multiple sittings are required.

DONOR SITE

Donor site is the extensor aspect of the thigh, gluteal region, medial aspect of the upper arm, or behind the ear lobe and retroauricular area. Prepare it surgically (viz, with shaving, cetrimide, spirit, povidone iodine) and give local anesthesia (1% lignocaine without adrenaline) intradermally to raise a wheal 5 cm in diameter or larger depending upon the number of grafts required.

Rotate a 2.5 mm punch until the cutting edge descends to the depth of the upper dermis. Take the requisite number of cuts adjacent to one

another, with intervening normal skin (rows of 10-15). Lift the edge and cut through the upper dermis close to the epidermis to free the grafts individually from the base. Transfer to a bowl containing gauze moistened with normal saline. Achieve hemostasis by pressure. Dress with a double layer of collgen mesh, gauze and Elastoplast bandage.

RECIPIENT SITE

Surgically prepare the area and give local anesthesia (1% lignocaine injected both intradermally and subcutaneously). Rotate a 2 mm punch until the cutting edge descends to the depth of the mid-dermis. Lift the edge, cut through the mid-dermis to free it from the base and discard. Take such cuts spaced 5-10 mm apart as per the geographical pattern of the patch. Transfer the stored grafts individually to these dilated punched out areas, ensuring that the dermal side of the graft is in direct contact with the recipient dermis, and even out the edges. For hemostasis and a proper take up of graft, apply firm pressure with a moist gauze piece. Dress with a double layer of framycetin tulle, gauze and elastocrepe bandage. Immobilize the part where required (rest, splints, etc.).

POST OPERATIVE MEDICATIONS

Antibiotics and anti-inflammatory drugs are administered for 8-10 days and PUVA or PUVASOL for 3-6 months is given to enhance repigmentation4. Oral and local steroids may be needed if the pigment spread is slow.

COURSE OF GRAFTS

The grafts turn pink brown, dark brown and finally black . They are taken up by 8 to 10 days. Perigraft pigmentation starts by 1 month, and spreads to cover the patch in the next 3-6 months4. The average spread varies from 5-10mm and in dark individuals up to 15 mm has been observed.

COMPLICATIONS

These include faulty or no pigmentation due to graft rejection (bleeding, shearing movement, upside-down graft, infection and improper immobilization), cobble-stoning (raised grafts), sinking pits (sunk-in grafts), polka dot appearance, hypo- or depigmented junctional line,

scarring, allergic reactions, blotchy pigmentation, color discrepancy, and reactivation of viti1igo.

Cobble-stoning and sinking pits can he prevented by ensuring that the grafts are placed at the level of the adjacent skin. If the graft is raised, either cut away its under-surface or deepen the recipient chamber.

ADVANTAGES

This is a simple, safe and inexpensive office procedure that, being an extended biopsy technique, needs no special training for a dermatologist. It has a high success rate and excellent cosmetic results. Large lesions and any site except the angle of mouth can be treated. Areas of residual vitiligo between grafts or wherever grafts are rejected can be re-grafted.

THIN THIERCH'S SPLIT THICKNESS SKIN GRAFTING

This procedure consists of grafting a very thin, split thickness skin graft consisting of the epidermis and part of the upper papillary dermis onto the dermabraded patch of vitiligo, and securing it with pressure or with surgical glue and local immobilization. It is very useful for large

patches and where immediate results are desirable[4]. Use of meshed split thickness skin grafts has also been reported to treat large areas of vitiligo.

DONOR SITE

A thin split thickness graft is obtained from routine donor sites with a simple razor blade, Silver's knife or mechanized dermatome by the basic skin grafting technique.

RECIPIENT SITE

The recipient bed is prepared in a similar fashion as in the spot and regional dermabrasion technique by evenly dermabrading the vitiliginous lesion and 1 cm of the surrounding normal skin to obtain a good raw surface. The graft is then placed with its dermal surface facing the abraded bed to extend 3-5 mm beyond its border. Its edges are evened out to prevent curling/beading and a 'stuck on' appearance. Pressure dressing with elastocrepe bandage is given after covering the graft with framycetin tulle or surgical glue is applied in droplet form all along the overlapped and free edges of the graft to ensure firm contact. A flexible collodion dressing can also be used as an alternative. The part is immobilized if required.

POSTOPERATIVE DRESSING AND MEDICATIONS

The recipient site dressing is changed after 24 hours. A seroma, hematoma or any air bubble is drained. The donor site dressing is removed after 10-15 days. Antibiotics and anti-inflammatory drugs are given for 8-10 days. Pressure garments can be given after graft uptake for 2-3 months to flatten the graft and obtain a good color match.

COMPLICATIONS

These are graft rejection (due to bleeding, movement, infection and upside down placement of graft), ‘stuck on tyre patch’ appearance, perigraft halo of depigmentation, superficial scarring at the donor site, and reactivation of vitiligo.

ADVANTAGES

Large areas can be covered in a single sitting. The grafted areas are skin colored immediately after removal of the dressing. It is a simple and safe procedure with good results. The procedure is less time consuming than other procedures of grafting for vitiligo. The graft survival chances are good because it is a thin split thickness graft.

DISADVANTAGES

Large areas have to be abraded if the geographical pattern of the vitiligo patch is to be followed. Some degree of hyperpigmentation and contracture will occur at the grafted site. Superficial scarring and vitiligo can develop at the donor site. The fingers, lips, palms and soles are difficult to graft. Benign thin, Thiersch's grafts are vulnerable to trauma. Multiple sittings are required for large lesions. Surgical expertise is required to obtain thin and adequate sized thin graft.

AIM OF THE STUDY

To study the epidemiology, clinical spectrum and therapeutic responses in childhood vitiligo with an aim to observe the following parameters:

1. Prevalence of vitiligo in children under 12 yrs.
2. Age and sex distribution, associated family history.
3. Sites of involvement and type of vitiligo.
4. Associated autoimmune disorders and syndromes.
5. Therapeutic responses to various modalities of treatment

MATERIALS AND METHODS

This was a prospective study conducted at Rajiv Gandhi Government General Hospital, Chennai in the Department of Dermatology for a period of over 1 ½ years from november 2009 to June 2011.

During this period, all children less than 12 years of age were screened for vitiligo. Only untreated patients were included in the study. A total of 80 children with vitiligo of both sexes were enrolled. They were questioned in detail regarding the age of onset, site of initial lesion, duration of disease, progression and associated cutaneous disorder.

Precipitating factors such as trauma, illness, stress and contact with chemicals were specifically asked for. History of ocular symptoms and systemic illness like diabetes, thyroid dysfunction, anaemia and Addison’s disease were recorded. History of vitiligo, premature canities or any other autoimmune disorder in the family was noted.

A detailed dermatological examination was carried out and a thorough systemic examination was made to record any associated systemic disorders.

The diagnosis of vitiligo was made based on clinical features and if needed skin biopsy. Trichrome vitiligo, quadrichrome vitiligo and associated cutaneous disorders were specifically looked for. In each case, body charting, extent of body surface involvement, leukotrichia and Kobnerization was recorded.

Each case was classified into recognized patterns of vitiligo namely vitiligo areata, segmental vitiligo, acrofacial vitiligo, lip tip vitiligo, vitiligo mucosae, vitiligo vulgaris and vitiligo universalis.

The patients in our study were divided into 3 groups.

1) Group A treated with Topical steroids and immuno modulators.

2) Group B treated with NBUVA therapy

3) Group C treated with surgical modalities with mini punch grafting and split skin grafting.

The above therapeutic modalities were adopted according to the age of the patients, type of vitiligo, site of vitiligo and extent of vitiligo.

A detailed history regarding the onset, duration and course of the disease, presence and absence of precipitating factor, family history, associated skin and systemic problems, were recorded.

Dermatological assessment of the disease was carried out using down the sites of involvement total body surface area involved, total number of factors, size and distribution of the patches, presence of white hair in the patch.

Details regarding the margin of the patch, skin texture, presence or absence of perifollicular pigmentation, Koebner's phenomenon, associated with skin and systemic problems were noted. Focal sepsis was ruled out by referring the patient to ENT and Dental OPD for checkup. Other associations if any are noted and referred to respective departments for evaluation.

After collecting the preliminary reports the patients were assessed and divided based on inclusion and exclusion criteria.

Since all the three modalities of treatment selected belong to different modes (medical, physical and surgical) they cannot be compared. So, the therapeutic response to each modality has been studied.

GENERAL PRINCIPLES

1) Patient should be explained the nature of the disease and its impredictable cause and prognosis.

2) Reassurance is essential.

3) Diet rich in proteins, Vitamin B complex, Vitamin E and minerals such as copper, iron and zinc should be supplemented.

4) Avoidance of precipitating factors and drugs like choroquine, α-interferon and β-blockers.

5) Avoidance of sun exposure, sunscreens may be used if necessary.

6) Avoidance of detergents and substances containing phenolic compounds.

EFFICACY PARAMETERS

The primary efficacy variable was the percentage change in depigmentation from the baseline to the end of study period. (i.e. 6 months)

The efficacy parameter namely the Physician's Global Improvement assessment was computed at the end of 6 months of the study.

During the initial assessment, estimation of body surface area (BSA) involvement was assessed using Vitiligo Area Scoring Index (VASI). The body was divided into five separate and mutually exclusive regions: Face and Neck, Upper extremities (excluding Hands), Lower extremities (excluding Feet), Hands and Feet and Trunk. Buttocks were included with the lower extremities.

One hand unit, which encompasses the palm plus the volar surface of all the digits is approximately 1% of total body surface area and was used as a guide to estimate the baseline percentage of vitiligo involvement of any body region. To eliminate variation in hand size, we

defined a hand unit to be the volar hand, including the fingers of single investigator.

All the patients were followed up once in every two weeks, to look for any macular repigmentation and presence of adverse effects. Any new depigmented patches that developed during the study were also estimated using the hand unit method and were included in the VASI calculation.

For each body region the VASI was determined by the product of the area of vitiligo in hand units (which were set at 1% per unit) and the extent of depigmentation within each hand unit measured patch.

Standardized assessment for estimating the degree of pigmentation to derive the VITILIGO AREA SCORING INDEX (VASI).

At 100% depigmentation, No pigment is present.

At 90%, only specks of pigment are present.

At 75%, the depigmented area exceeds the pigmented area.

At 50%, the depigmented area and pigmented area are equal.

At 25%, the pigmented areas exceed the depigmented area

At 10%, only specks of depigmentation are present.

Total body VASI was then calculated using the following formula by considering the contributions of all body regions (possible 0-100)

Total Body VASI = Σ all body sites [hand units X residual depigmentation]

Clinical photographs were taken at baseline and at each monthly follow up visits as an aid to the Global clinical scoring. They were not used to derive the VASI, which was instead determined by direct clinical examination.

VASI Score	**Improvement in %**	**Comments**
1	76%-100%	Excellent improvement
2	51%-75%	Marked improvement
3	26%-50%	Definite improvement
4	1% -25%	Minimal improvement
5	0%	No change

The investigator performed the efficacy evaluation with physician’s global improvement assessment alone. VASI scoring has been used to calculate the body surface area involvement.

Limitations of the study:

- The treatment results were based only on physician's global improvement assessment score.

PATIENT SELECTION:

- In the study of 80 patients under 12 years of age,
    - As per the VASI scoring:
- 59 cases had BSA involvement <20% and
- 21 cases had BSA involvement >20%
- Out of 59 cases with BSA <20%, 17 cases were stable cases of vitiligo .
- Vitiligo cases <20% BSA were treated with topical steroids and immunomodulators.
- Stable vitiligo <20% BSA were treated with surgical modality of treatment.
- The rest 21 cases with >20% BSA were treated with physical modality namely NB UVB therapy and the responses were commuted as below.

STATISTICAL ANALYSIS

GROUP A (42 CASES OF BSA <20%)

Topical steroids and immuno modulators

42 Cases (M-20, F-22) of which

Type	Male	Female
Focal	10	8
Segmental	6	5
Mucosal	4	7
Vitiligo vulgaris	-	2

Were included in this group.

These patients received topical 0.1% betamethasone valerate in the morning and 0.03% Tacrolimus ointment in the night for 6 months. Patients were directed to apply the cream in thin layers each time.

Steroid cream and tacrolimus ointment was applied for 3 weeks and then one week of tacrolimus alone in a month period.

The same protocol was continued for a period of 6 months.

The patients were reviewed once in 2 weeks for the response and side effects. After completing the treatment they were carefully followed up for any evidence of relapse.

In this group, the mucosal vitiligo cases were treated with mild steroid like triamcinolone acetonide and tacrolimus 0.03% cream in the same manner as above for 6 months, and followed up.

GROUP (21 CASES OF BSA >20%)

NBUVB - 21 cases (M-7, F-14) of which

Type	Male	Female
Vitiligo vulgaris	5	11
Acrofacial	1	2
Liptip	1	1

Were included in this group.

These patient received NBUVB therapy for a period of 6 months. Before starting the therapy, patients were screened for any connective tissue disease. Ocular examination was carried out and any history of photosensitivity or photosensitive disorders, skin type (Fitz I-III)

concomitant radiotherapy, chemotherapy or immunosuppressive therapy and claustrophobics were excluded.

NBUVB therapy was given thrice weekly on alternate days starting with one unit dose of 0.25 J/cm2 independent of the skin type.

The dose was increased by 20% each treatment. The optimal constant dose was achieved when minimal erythema occurred in the lesions. During the treatment the eyes were protected by UV- blocking goggles. All patients were kept their underwear on to shield their genitals from radiation exposure.

Children were advised to protect their skin against excessive exposure to natural sunlight especially between 11.00am to 3.00pm during sunny days on both treatment as well as non treatment days. A sunscreen with high SPF (25 or higher) was applied on sun exposed areas.

Pre and post treatment photographs were taken to see the response to therapy.

Informed written consent was obtained from the parents. In each case, the nature of treatment was carefully explained, including details of the possible benefits and side effects.

At each visit, they are evaluated for any adverse effects like sunning sensation, itching, erythema.

GROUP C (17 CASES- STABLE VITILIGO)

Surgical modality

Minipunch grafting/ Split skin grafting

17 cases (M-8, F-9) of which

Type of vitiligo	Male	Female
Focal	1	2
Segmental	3	2
Vitiligo vulgaris	3	4
Acrofacial	1	1

Patients were selected based on the following criteria:

- Vitiligo lesions should be stable i.e no new lesions and existing lesions not enlarging in size for minimum of two years.

- For lesions present on mobile and immobile areas punch grafting was done.

- For lesions on immobile areas alone split skin grafting was done.

- Patient’s parent consent for surgery.
- Psychologically stable patients with realistic expectations.
- Out of 17 cases, 13 cases were treated with punch graft and 4 cases with split skin graft.

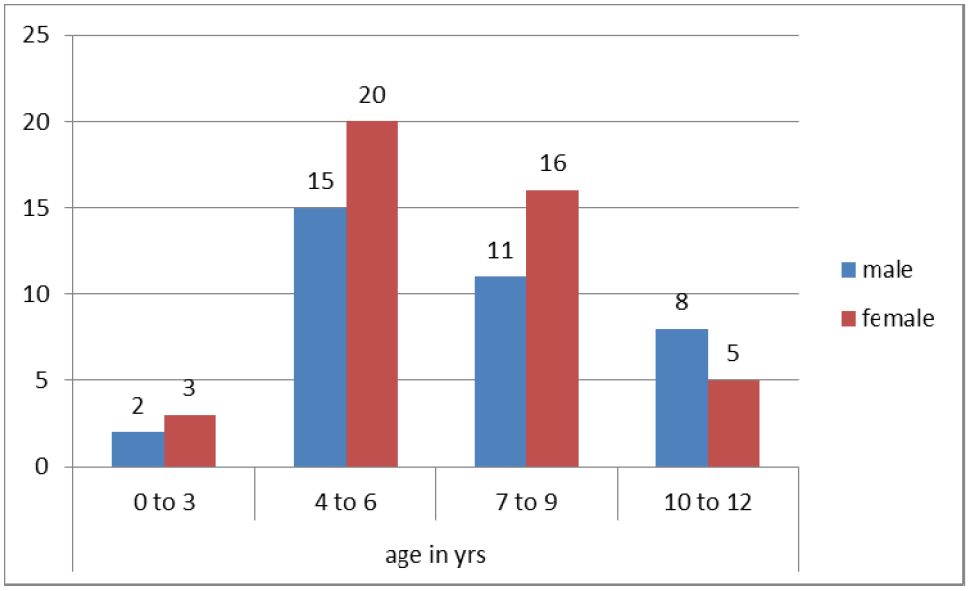
OBSERVATIONS

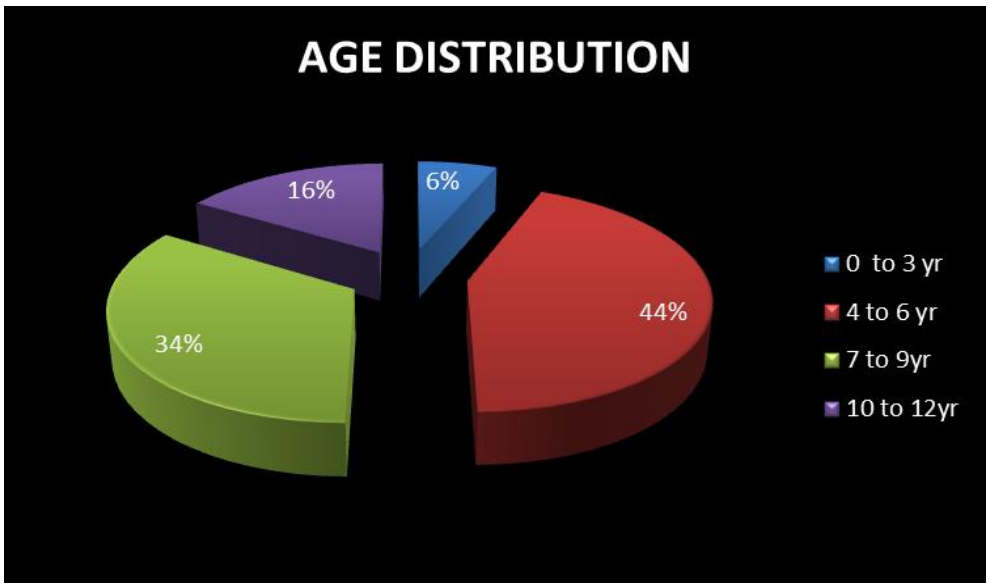
A total of 80 children were enrolled during the study period. The male to female ratio in the study was 45% to 55% [1:1.2], with females in the majority (n=44 female, and n=36 male). The mean current age of the children visiting our hospital was 6 years. Forty children (50.0%) were in the age group of 7 to 12 years. The youngest child was one and 1/2 years old.

The commonest age of onset was between 4 to 9 years.

Age and sex distribution of 80 children with vitiligo:

Age group (in years)	Number of patients		Total
	Males	Females	
0-3	2	3	5
4-6	15	20	35
7-9	11	16	27
10-12	8	5	13
Total	36	44	80

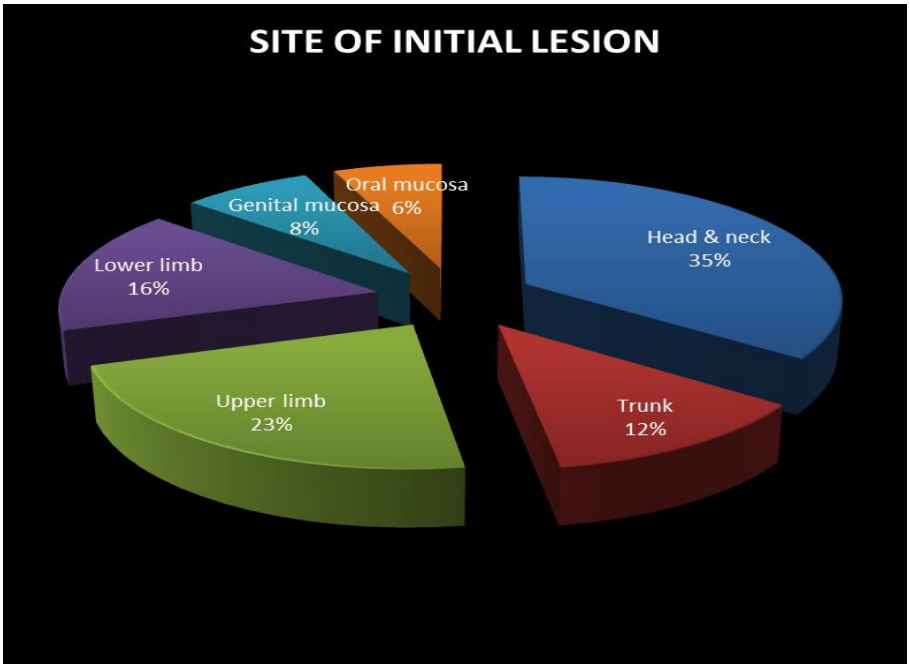




- Vitiligo was present for a mean duration of 6 months before the first consultation (range 1 month to 2 years).
- Five children (6.25%) had a history of trauma prior to onset of vitiligo.
- Ten children (12.5%) had a family history of vitiligo.

Site of initial lesion in 80 children with vitiligo:

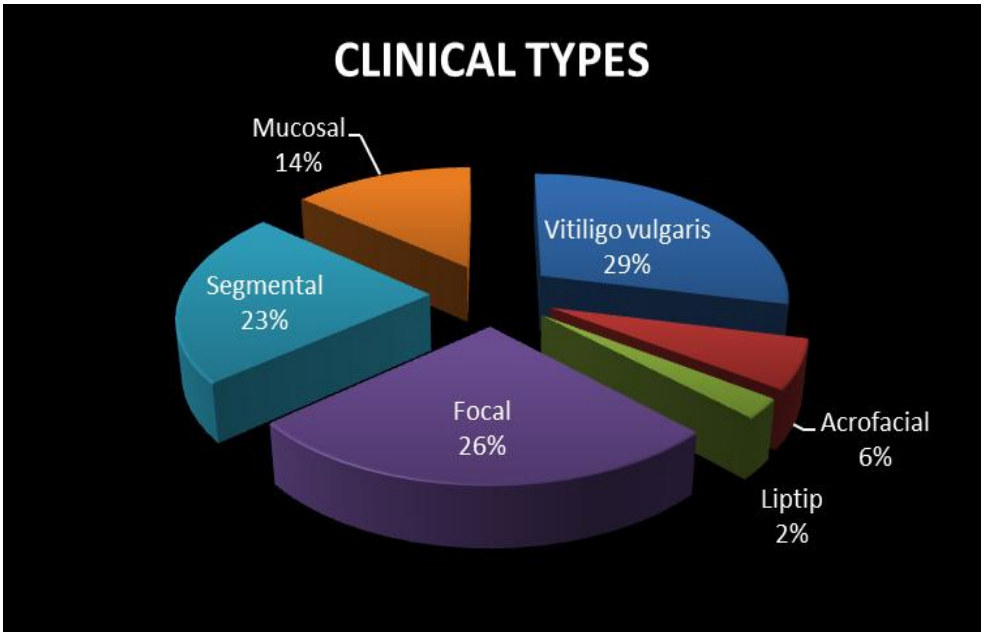
Site of initial lesion	Number of patients		total
	Male	Female	
Head & neck	13	15	28
Trunk	4	6	10
Upper limb	8	10	18
Lower limb	7	6	13
Genital mucosa	2	4	6
Oral mucosa	2	3	5
Total	36	44	80



- The most common site of initial lesion was head and neck followed by upper limbs, lower limbs and trunk.

Types of Vitiligo in 80 children with vitiligo:

Clinical type	Number of patients		Total
	Male	Female	
Vitiligo vulgaris	8	15	23
Acrofacial	2	3	5
Liptip	1	1	2
Focal	11	10	21
Segmental	10	8	18
Mucosal	4	7	11
Total	36	44	80



The most common type was

- vitiligo vulgaris seen in children 23 cases (28.75%) , followed by
- focal type in 21cases(26.25%)
- segmental type in 18 cases(22.50%)
- mucosal type in 11 cases(13.75%)
- acrofacial type in 5 cases(6.25%)
- liptip type in 2 cases(2.5%)

Among the segmental type of vitiligo in children, trigeminal dermatome was most commonly involved in 12children (15%)

In 80 children, 59 cases (73.75%) had body surface area involved less than 20%.

Leukotrichia was present in 12 children (15%), while Kobner phenomenon was observed in 17 children (21.25%).

21 children (26.25%) had an associated cutaneous disorder. These were

- Twenty nail dystrophy in 3(2.5%)
- Nail pitting in 9(11.25%)
- Halo naevi in 2(1.6%)
- Alopecia areata in 3 (2.5%)
- Premature canities in 2 (1.6%)
- lichen striatus in 2(1.6%)

Ten children (12.5%) had an associated ocular disorder. These were

- Eyelid vitiligo in 5 (6.25%)
- Reduced visual acuity (myopia) in 4 (5%)
- Conjunctivitis in 1 (1.25%) child.

SYSTEMIC ASSOCIATONS:

- Juvenile rheumatoid arthritis was seen in one (1.25%) child
- Hypothyroidism in 3(3.75%) children.

TREATMENT OBSERVATIONS

Treatment of vitiligo in children is difficult as therapeutic options are restricted when compared to that in adult patients.

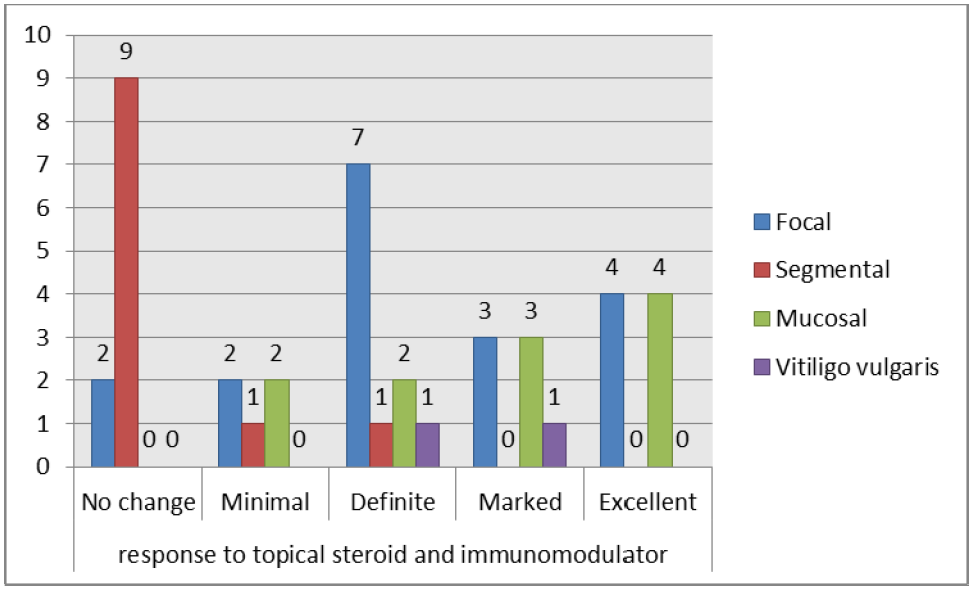
GROUP-A TOPICAL STEROIDS&IMMUNNOMODULATORS

Repigmentation was observed in 31out of 42 children treated with topical 0.1% Betamethasone valerate cream with immunomodulator 0.03% tacrolimus ointment.

The response of different types of vitiligo in this group with reference to clinical distribution and response grading as per the physician's global assessment scoring is given in the table below:

CLINICAL TYPES:

Response grading	Focal	Segmental	Mucosal	Vitiligo vulgaris	total	Percentage
No change	2	9	-	-	11	26.19%
Minimal	2	1	2	-	5	11.90%
Definite	7	1	2	1	11	26.19%
Marked	3	-	3	1	7	16.67%
Excellent	4	-	4	-	8	19.04%



- Face, neck and mucosal lesions responded maximally to this modality of treatment.

- Definite to marked repigmentation was observed in the lesion over the trunk and limbs.

- Minimal repigmentation was observed in the lesions over the extremities like hands and feet.

- No change in pigmentation was seen in the lesions associated with leukotrichia.

- Good (> 50% pigmentation) response was observed in 10 of the 12 children with lesions involving single site and 2 out of 3 children with lesions involving multiple sites.

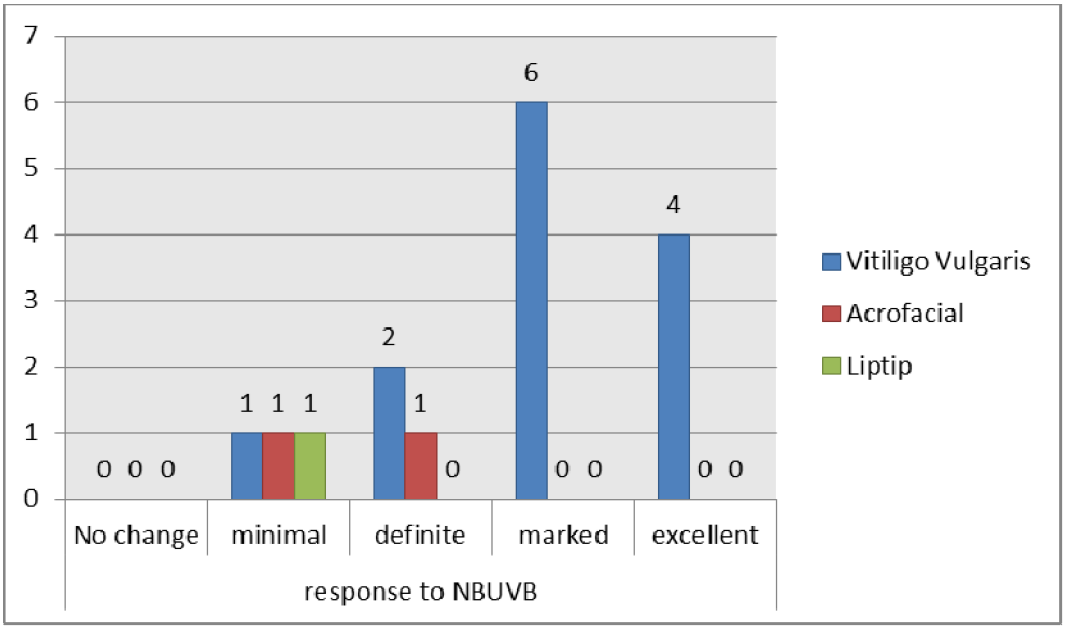
- Better response was observed in lesions involving <5% skin surface overall in this group.
- No significant difference in response was observed in relation to duration of lesions.
- No drop outs were found in this group. No adverse effects (atrophy, telangiectasia) were seen. No relapse was observed during the follow up period.
- Out of the 42 cases treated in this group, 10 cases (all focal cases) had rapid progression of lesions with onset of new lesions. They were treated with oral mini pulse with tab. Prednisolone 30mg (as betamethasone in oral form was not available in our hospital) divided into two doses 15mg each on two consecutive days every weekend. The progression of lesions were arrested within 1-3 months in 8 out of 10 cases and within 4 months in the rest 2 cases.
- The patients were continuing the topical steroids and immunomodulators simultaneously during this intermittent therapy. These patients on mini pulse steroid therapy not only responded by the progression of lesions but had earlier repigmentation of lesion as well compared to others not receiving mini pulse therapy in 80% of cases.

GROUP-B: NBUVB THERAPY (21 CASES)

With NBUVB therapy repigmentation was observed in all cases receiving the treatment, following the protocol strictly.

REPIGMENTATION ALONG WITH CLINICAL GRADING TO NBUVB (N=16)(5 drop outs)

Improvement	Vitiligo Vulgaris	Acrofacial	Liptip	Total Number	Percentage
No change	-	-	-	-	-
minimal	1	1	1	3	18.75%
definite	2	1	-	3	18.75%
marked	6	-	-	6	37.50%
excellent	4	-	-	4	25.0%



Out of 21 children, 5 children discontinued (VV-3, Acrofacial-1, Liptip-1) therapy as they were not able to attend the phototherapy centre three times a week and were school going. Rest 16 children managed to complete 6 month of therapy regularly.

Out of 16 children excellent repigmentation was observed in 4 out of the 13 (3 dropouts) children with vitiligo vulgaris type, marked repigmentation was observed in rest 7 children of vitiligo vulgaris type and 1 out of 2 (one drop out) cases of acrofacial type, minimal to definite repigmentation was seen in one acrofacial type and one liptip types (one dropout).

GROUP-C: SURGICAL THERAPY

Out of 17 cases who had stable vitiligo lesions, 13 cases were treated with miniature punch grafting for patients having lesions in both mobile and immobile areas and the rest 4 cases with spit skin grafting for those having lesions in immobile areas alone.

PUNCH GRAFTING

Pigmentation started appearing in 1½ to 2 months after grafting. Uniform perigraft and perifolicular pigmentation was seen and several such pigmented islands coalesced together to cover the affected area within 3-6 months. Majority of cases showed 7-10mm pigmentation.

All the patients were followed up for 6 months and the results were observed after 6 months. 9 out of 13 cases showed fair to marked cosmetic matching with the normal surrounding skin.

Almost all the donor sites healed with scarring within 1-2 months. The scars were superficial and acceptable to the patients.

Cobble stoning was seen in 5 patients in the initial stages but slowly faded within a period of 4-6 months . Keloidal tendency was noted in 2 cases later subsided with application of topical steroids.

Depigmentation of grafts were seen in 2 cases associated with leukotrichia.

Response	Type of vitiligo			Total	Percentage
	Vitiligo vulgaris	Segmental	Mucosal		
No change	-	1	-	1	7.69%
Minimal	1	1	1	3	23.07%
definite	4	2	1	7	53.84%
Marked	1	1	-	2	15.38%
excellent	-	-	-	-	-

OTHER OBSERVATIONS

1) Dark complexion patients pigmentation was better compared to fair subjects.
2) Good cosmetics results were observed in cases where two stage surgery was planned for bigger patches.

SPLIT SKIN GRAFTING

4 cases (focal vitiligo) were chosen for split skin grafting .

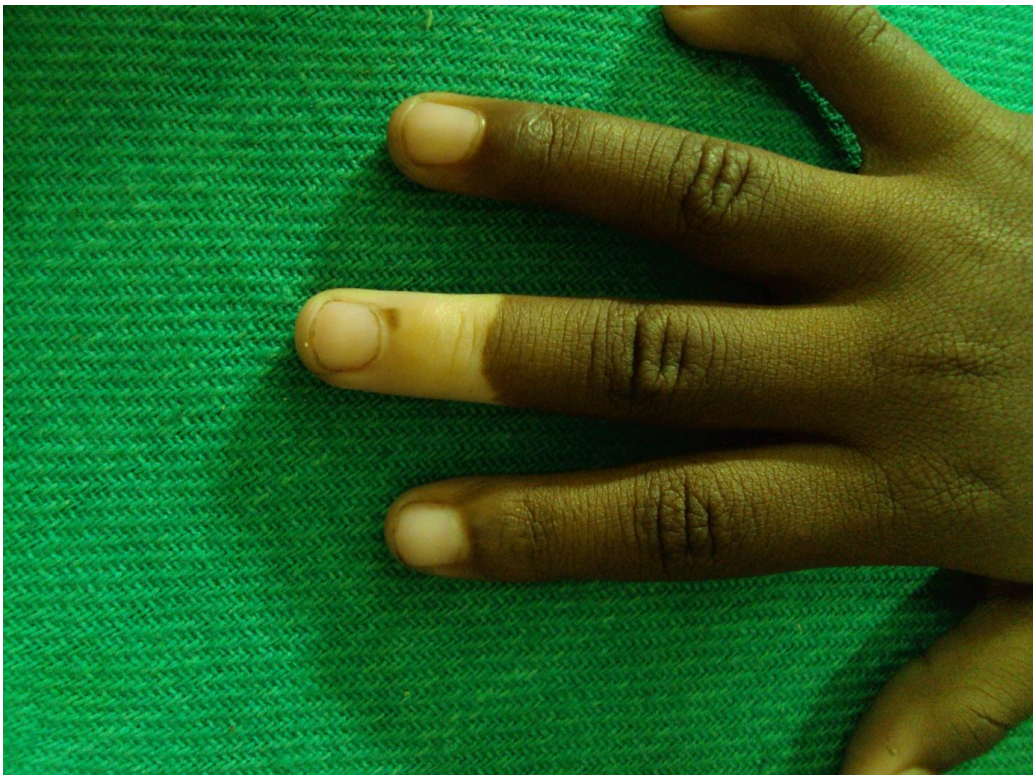
There was appearance of colour change as soon as the graft is taken up. So early colour change is an advantage here over punch grafting.

This modality was best for facial lesions and lesions close to the hair line and poor at the extremities. Amongst the clinical types, focal and segmental showed better response than acral vitiligo.

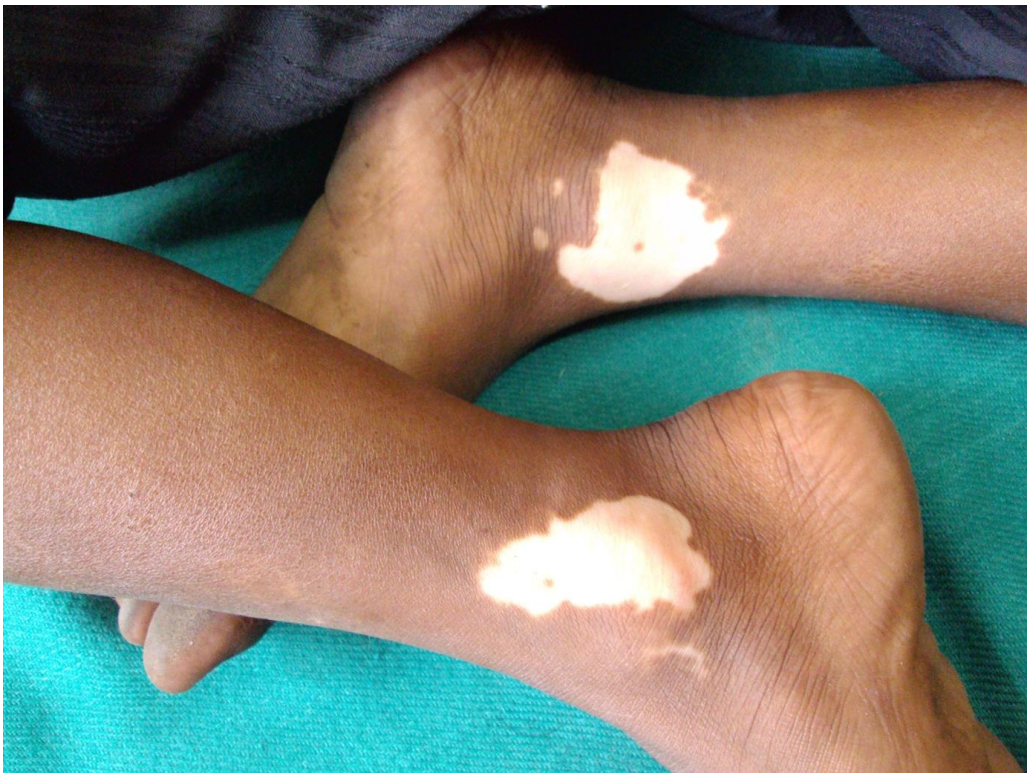
Tire-pattern appearance was seen in 2 cases and donor sites showed scaring in all cases.

Postoperative phototherapy in the form of NBUVB was given to the graft site 3 times a week and better results were seen after 6 months during follow up.

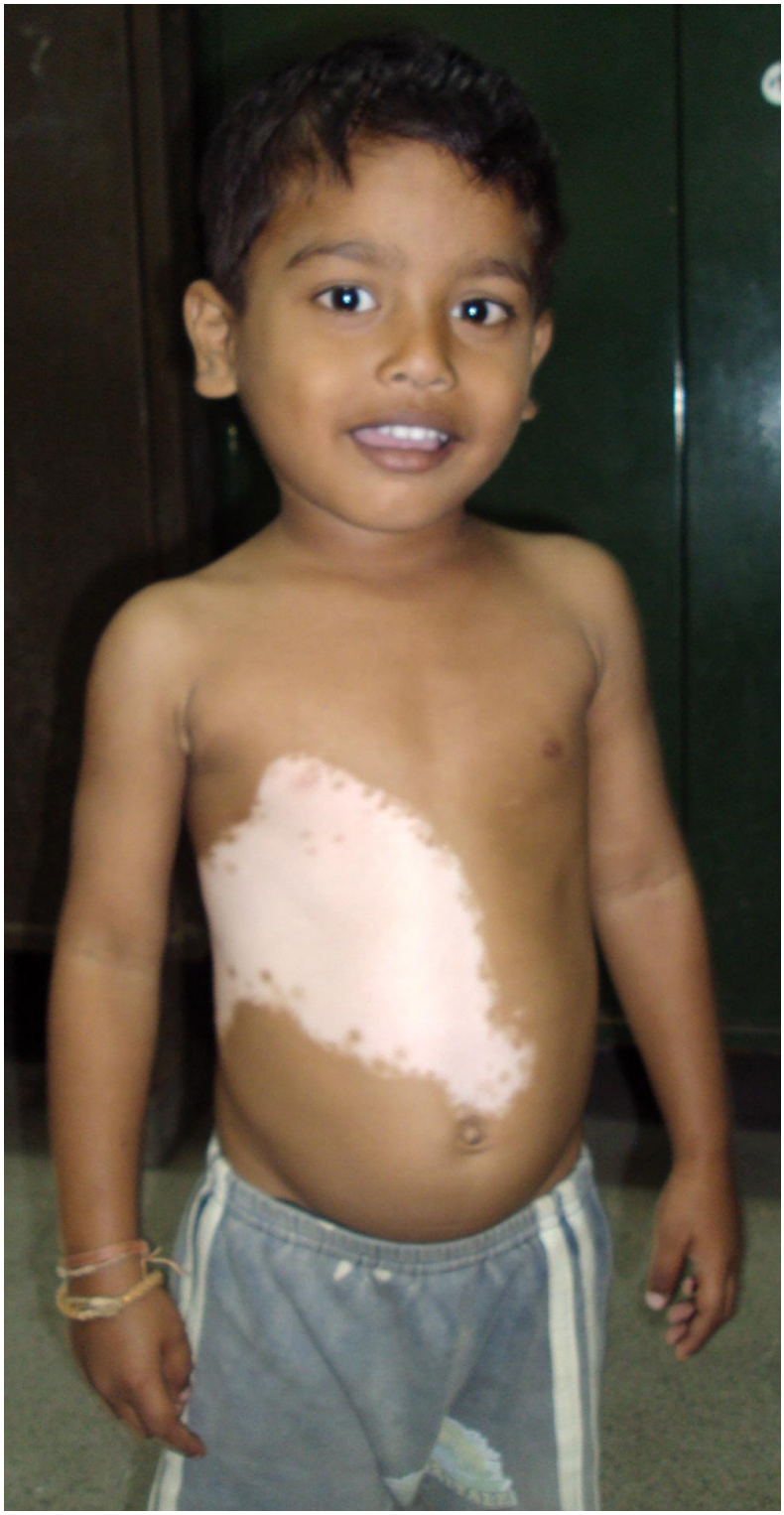
Focal vitiligo



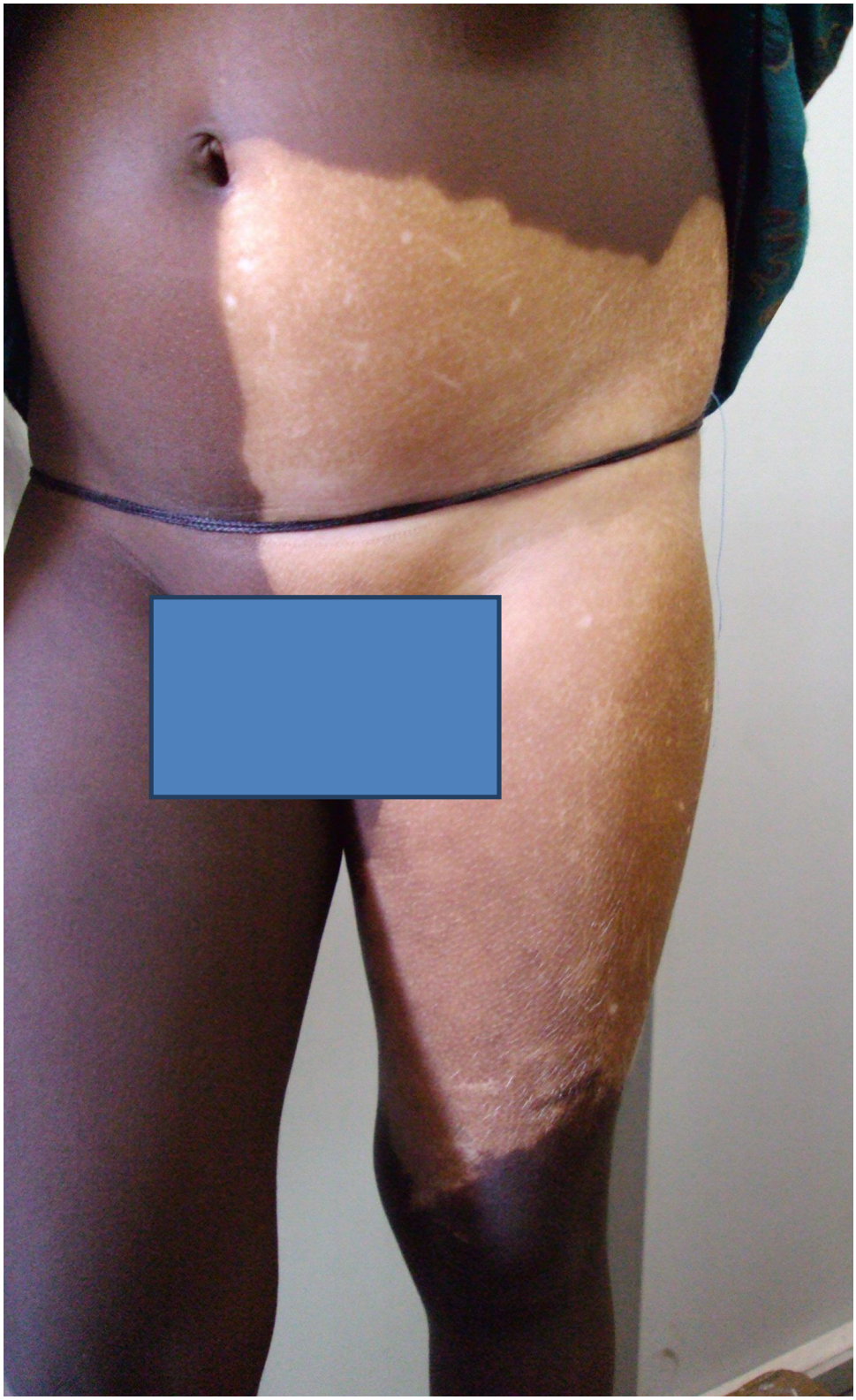
Focal vitiligo



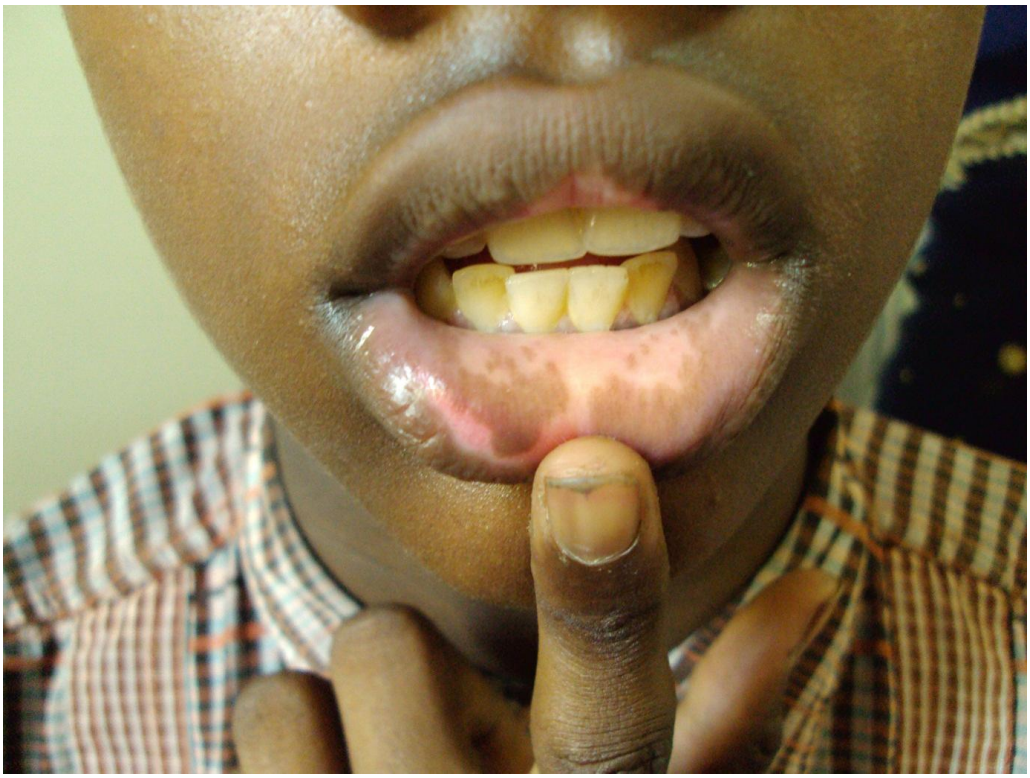
Segmental vitiligo



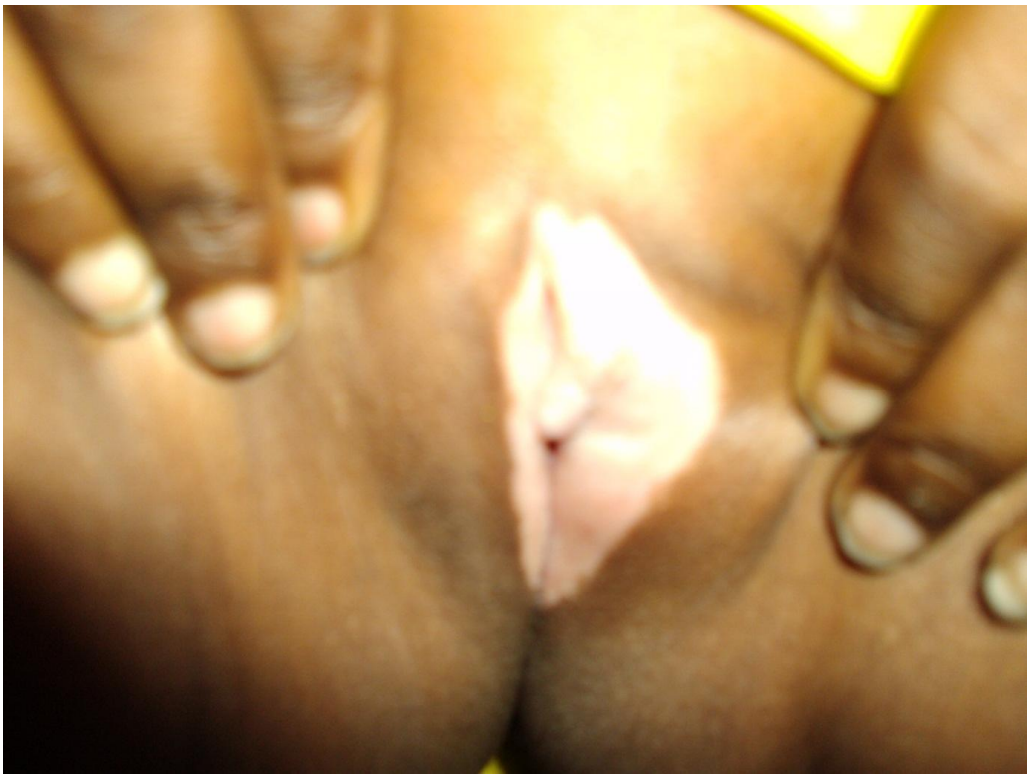
Segmental vitiligo



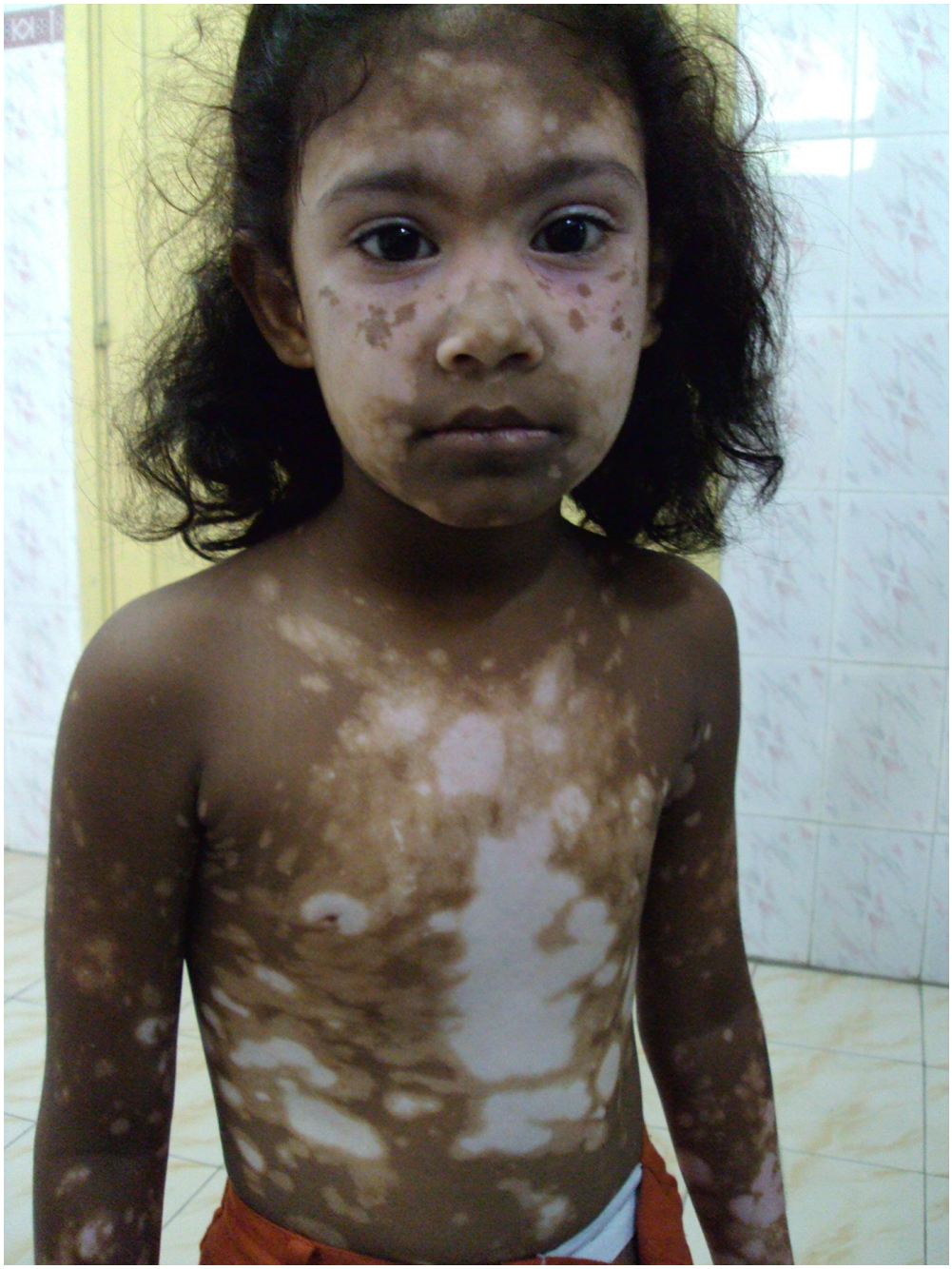
Oral mucosal vitiligo



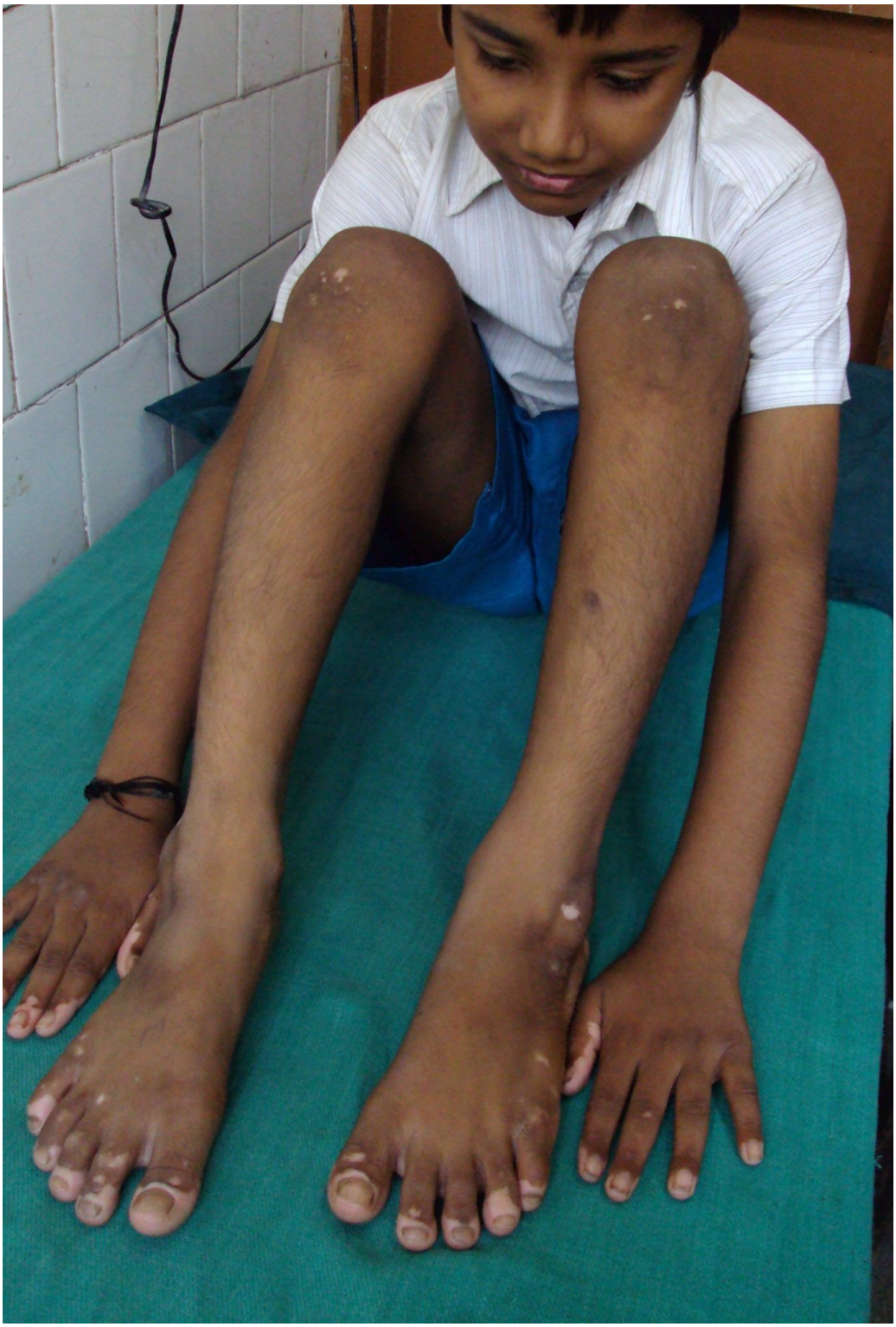
Genital mucosal vitiligo



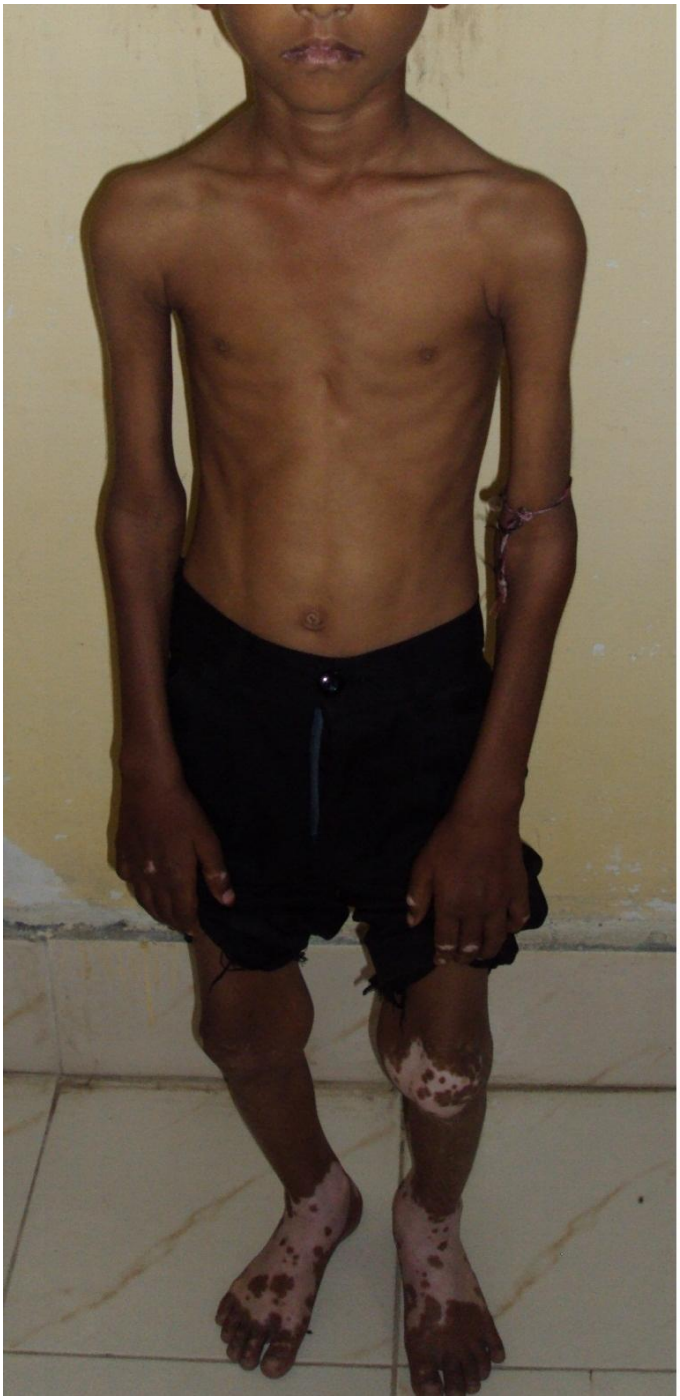
Vitiligo vulgaris



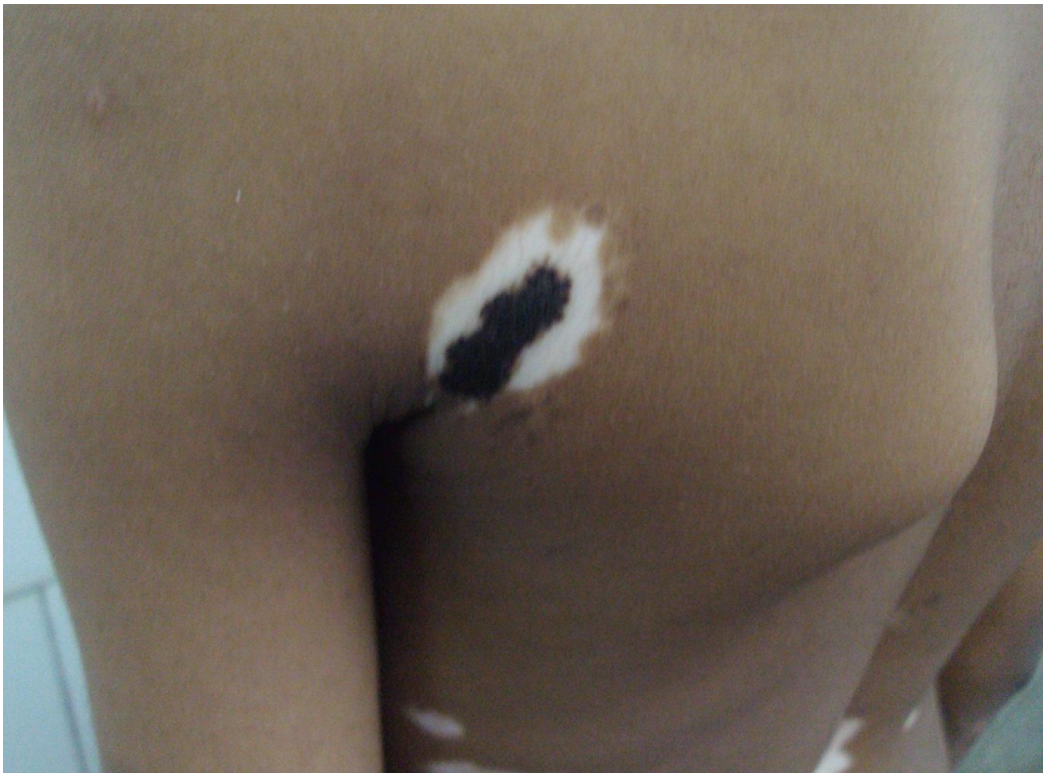
Acrofacialvitiligo



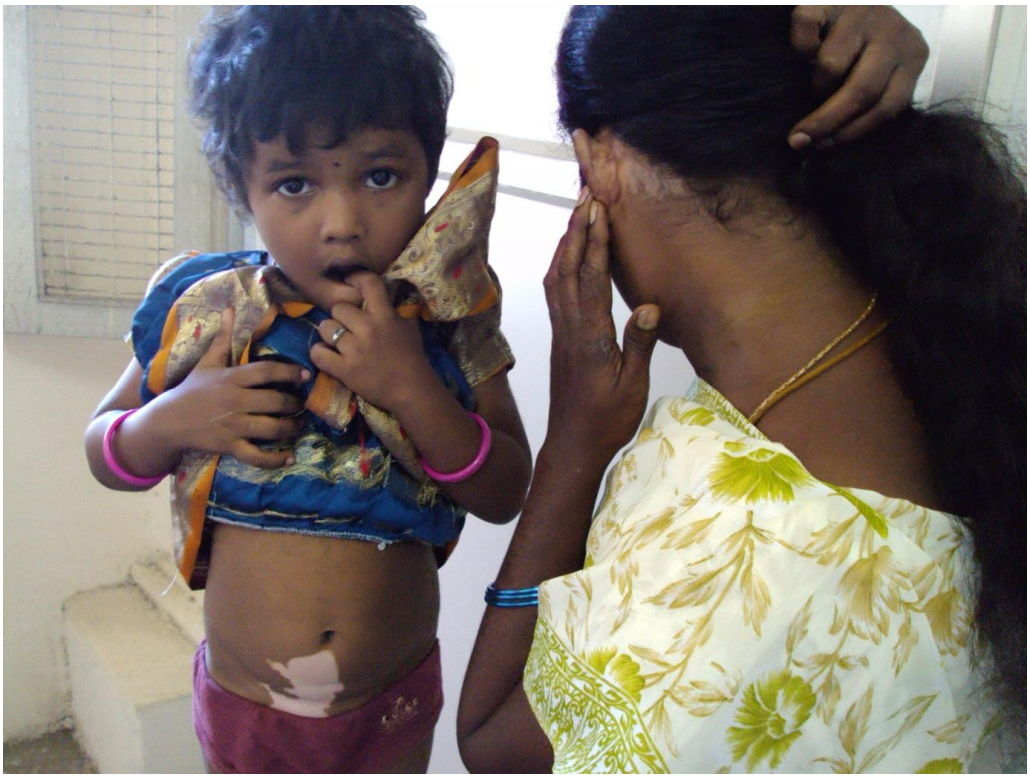
vitiligo associated with juvenile rheumatoid arthritis



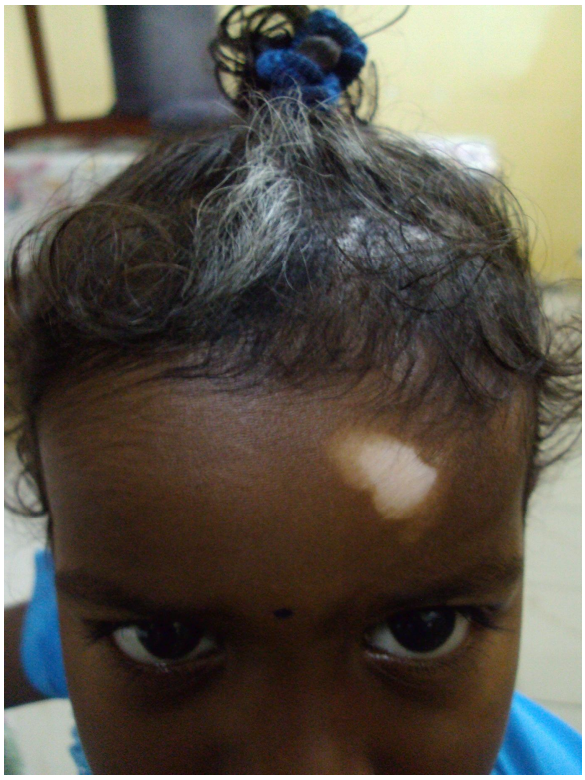
Vitiligo associated with epidermal nevus



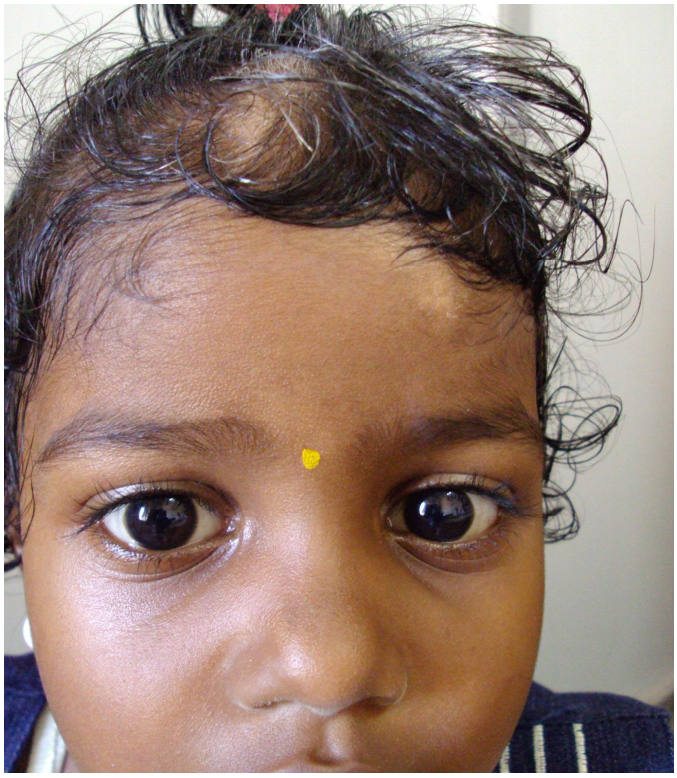
Familial
association



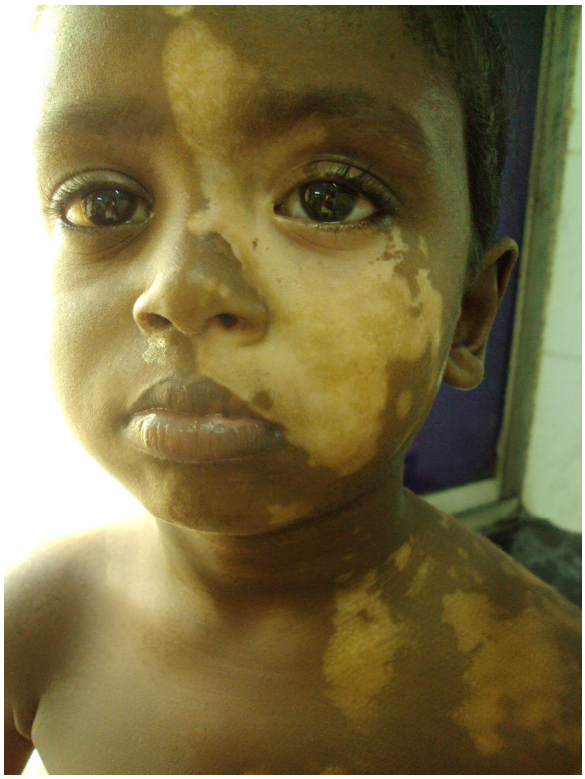
Before topical steroid and immunomodulator



After topical steroid and immunomodulator



Before NBUVB



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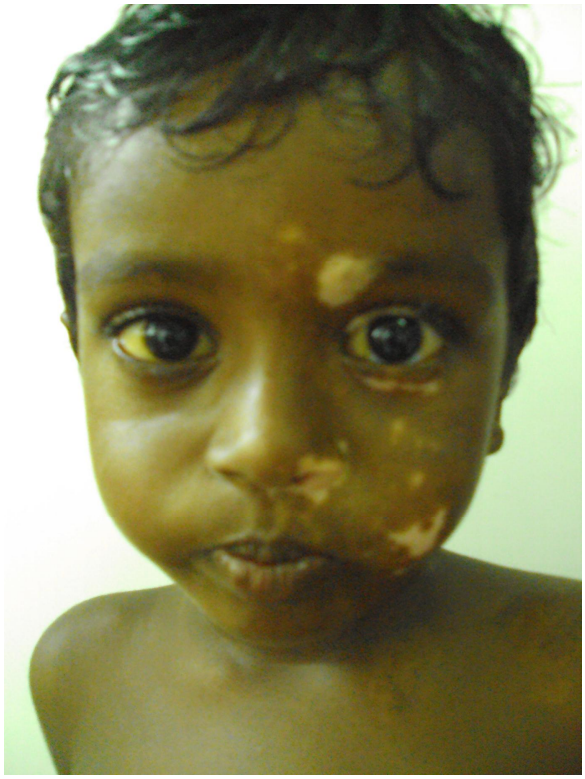
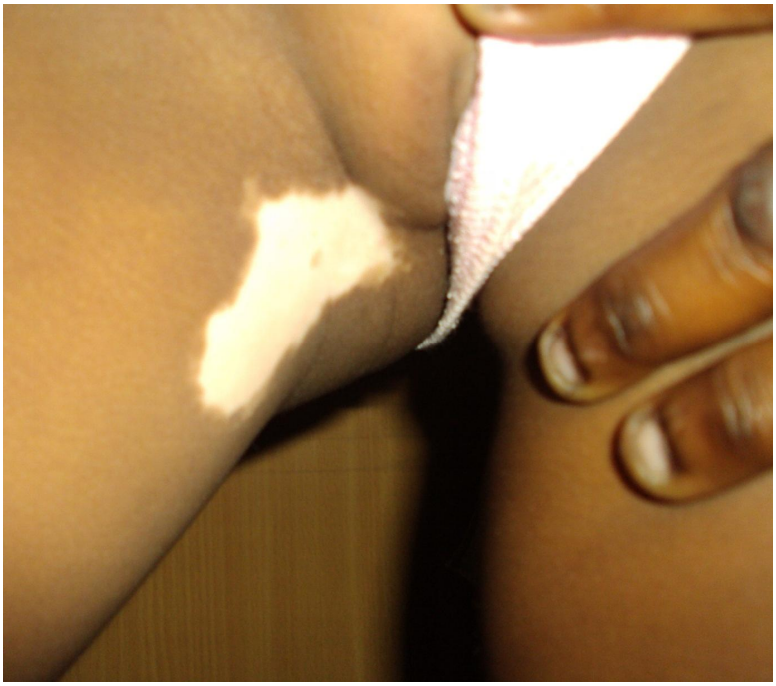
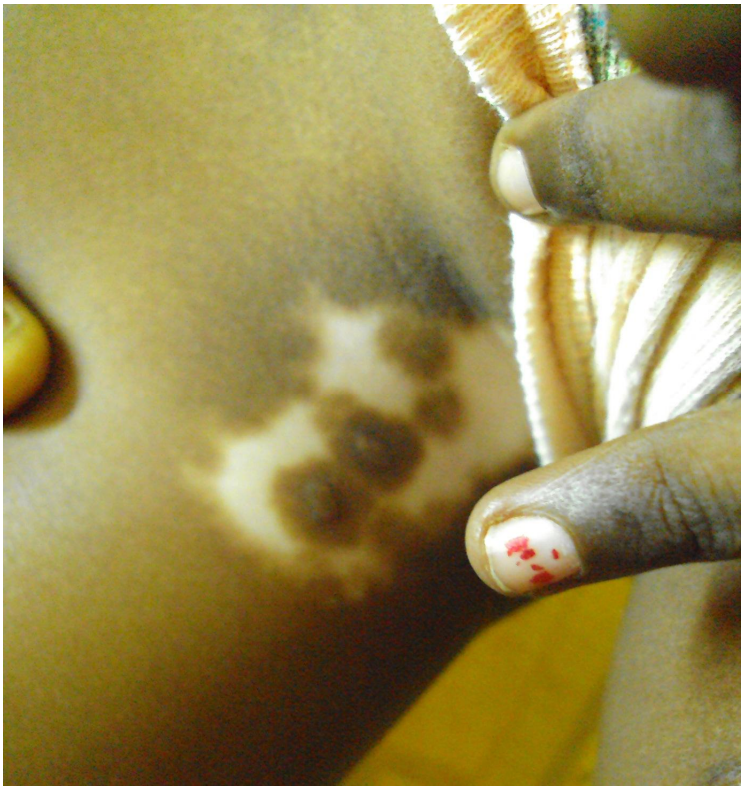


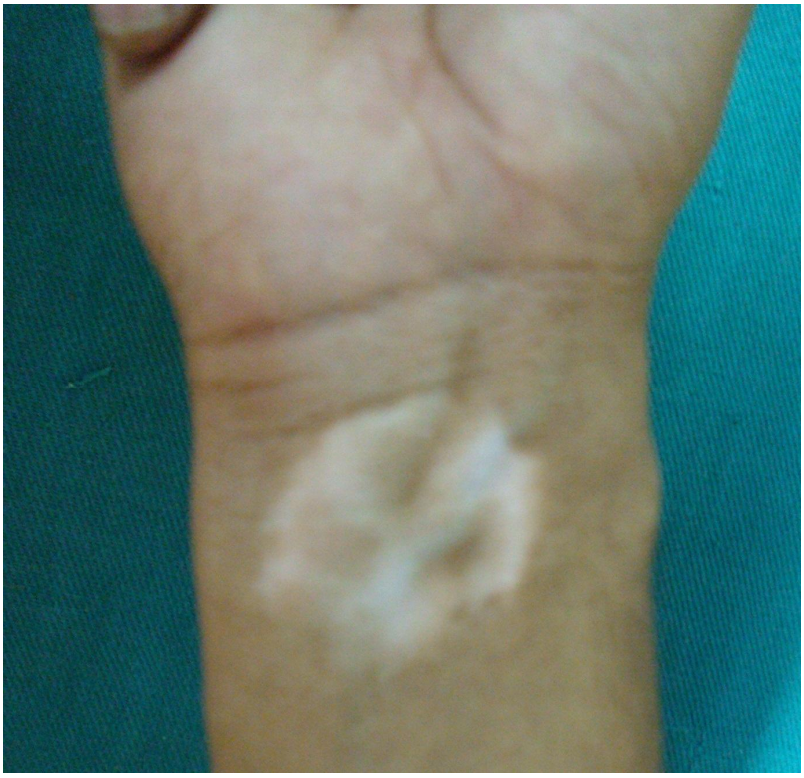
Before punch grafting



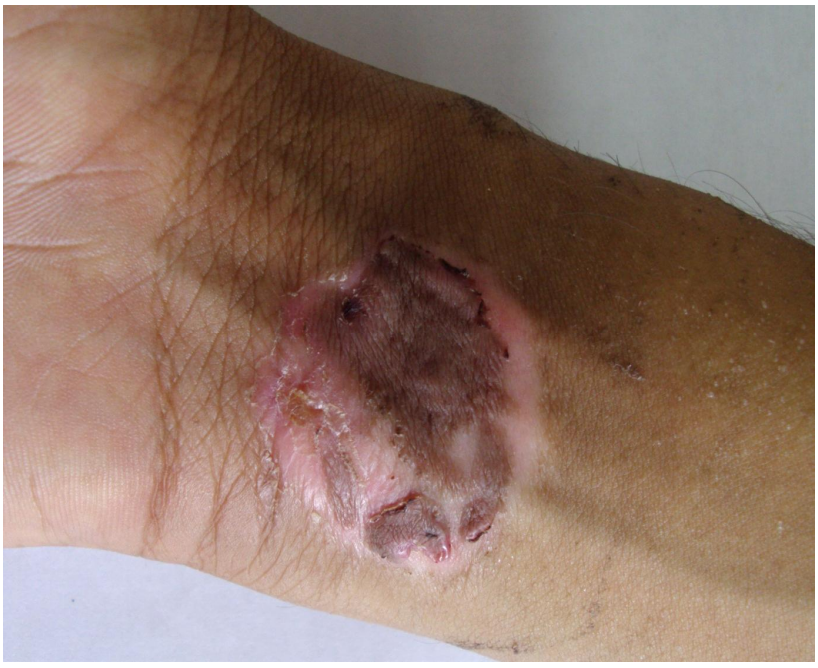
After punch grafting



Before split skin grafting



After split skin grafting



DISCUSSION

Vitiligo is a common disease in India having a prevalence of 0.46 – 8.8%[73]. Majority (>50%) of this population develop the disease before 20 years of age group making vitiligo an important aspect in Paediatric Dermatology. Indian studies on childhood vitiligo have reported the prevalence to be 2.6%[76]

AGE DISTRIBUTION:

In our study of 80 children, the commonest age of presentation was between 4 and 9 years which is in contrast to the study of Belliappa et al[75] where the commonest age of presentation was between 7 and 12 years.

The youngest child in our study was 1 ½ years old similar to Belliappa et al study where the youngest child was 1 year old. Earlier studies have reported cases of congenital vitiligo. But our study did not have any case of congenital vitiligo.

SEX DISTRIBUTION:

The Prevalence of vitiligo was found to be higher in girls than in boys 44 Vs 36 in our study of 80 cases.

The male to female ratio was 1: 1.2.

In earlier studies as well, girls were affected more than boys. However boys and girls were affected equally in Zhi Hu[85] et al study.

AGE OF ONSET:

In our study, the commonest age of onset of the disease was between 4 to 9 years constituting 77.5% of the cases. Belliappa et al reported that 68.9% of cases had onset of disease between 4 and 8 years of age similar to our study.

When comparing the age at presentation and age of onset, age of presentation in most cases is between 4 and 9 years and age of onset is also between 4 and 9 years. This shows that the patients and their parents are more aware of the nature of the disease and its course and present to the physician earlier to seek treatment.

DURATION AND PROGRESSION:

The duration of depigmentation varied from 1 month to 2 years. The mean duration before they first seek treatment was 6 months. This is in contrast to Belliappa et al study where mean duration of disease was 14 months.

SITE OF INITIAL LESION:

In our study the most common site of initial lesion was head and neck followed by upper limbs, trunk and lower limbs in that order. Belliappa et al also reported that the most common site of onset was head and neck. Jaisankar et al[76] reported the various sites of onset as lower limbs, head and neck, upper limbs and thorax in that order.

FAMILY HISTORY:

In our study, 12.5% of children had family history of vitiligo. In Belliappa et al study family history was present in 14.8% of children, Jaisankar et reported very low incidence as 3.3% and in Halder et al[33]study there was 35% family history.

TYPE OF VITILIGO:

In our study, vitiligo vulgaris was the most common clinical type seen in 28.75% closely followed by focal type in 26.25%, segmental type in 22.50%. In earlier studies on childhood vitiligo as well, vitiligo vulgaris was the most common type reported. Belliappa et al in their study of 122 children reported was the most common type is 36.9% and segmental vitiligo as the second most frequent type occurring in 27%.

Lip-Tip vitiligo was the least common type seen in our study population which is similar to study of Belliappa et al ,Halder et al but Jaisankar et al reported acrofacial type as the least common.

Mucosal Vitiligo was seen in 13.75% in our study, Belliappa et al also had similar figures of 13.10%.

Among segmental type, trigeminal dermatome was most commonly involved in 15% in our study consistent with all other studies done earlier.

BODY SURFACE AREA INVOLVEMENT :

Our study showed Body surface area of less than 20% in 73.75% of children in contrast to 95.9% in Belliappa et al study.

SPECIAL FEATURES:

Leucotrichia was present in 15% of children with vitiligo in contrast to 41.8% in Belliappa et al study. Kobner phenomenon was observed in 21.25% and Belliappa et al reported in 24.6% of patients.

CUTANEOUS & AUTO IMMUNE DISORDERS ASSOCIATED:

In our study, Alopecia areata was seen in 2.5%, Halo Nevi in 1.6% of children. Belliappa et al reported 2.5% of alopecia areata similar to our study and 4.9% Halo Nevi.

OTHER CUTANEOUS DISORDERS:

Premature canities was seen in 1.6%, epidermal nevus in 3.75%, Lichen striatus in 1.6%, Twenty nail dystrophy in 2.5% and nail pitting in 15%. Belliappa et al reported premature canities in 1.6% similar to our study.

TREATMENT

The treatment modalities selected here were based on the available medications and facilities in our hospital set up.

Group A: Topical steroid and immunomodulator

Repigmentation was seen in 73.80% of cases treated with both topical 0.1% betamethasone and 0.03% tacrolimus ointment. To the best of our knowledge, no other study in childhood vitiligo has combined both topical steroid and immunomodulator for treatment , rather compared the efficacy of both proving that topical immunomodulator is an effective alternative to topical steroid[60].

A good response (>50% pigmentation) was found in 35.71% in our study. Response was very good for focal and mucosal lesions.

Segmental patches and those associated with leukotrichia did not respond well to this modality.

Mucosal vitiligo usually a resistant form, responded well on combining both topical steroid and immunomodulator , so this modality is best suited for mucosal lesions.

Oral minipulse steroid with prednisolone is an effective treatment option for controlling the rapid disease spread in childhood vitiligo and with addition of topical steroid and immunomodulator the extent of repigmentation was also significant consistent with the previous studies of Pasricha[64] et al and Majid[62] et al.

GROUP B (NBUVB)

A good response (>50%repigmentation) was observed in 62.50% of treated cases. After a mean interval of 32 times of phototherapy 50% repigmentation was achieved similar to A.J kanwar65 et al study. The adverse effects were mild and transient and none discontinued the therapy because of side effects. Vitiligo vulgaris type responded well and liptip type responded least similar to other studies on this modality.

GROUP C

PUNCH GRAFTING: In our study over 69.22% cases showed definite to marked repigmentation as compared to Khandpur et al study of 83.3%. The graft site showed appreciable perigraft spread of pigmentation and good cosmetic colour match to the surrounding skin in

70% of treated cases. Vitiligo vulgaris responded well and segmental type responded least.

SPIT SKIN GRAFTING: Excellent cosmetic matching was seen in 75% of grafts involving larger areas. Obvious colour change seen after grafting is an advantage in this procedure.

Further similar such studies need to be done at regular intervals with newer modalities of treatment.

CONCLUSION

- Vitiligo incidence during the study period was 0.27%.
- The incidence of vitiligo in children was 15.87% of the total number of vitiligo patients over a period of 1 ½ years of study.
- Females were predominantly affected than males which might be due to their increased cosmetic concern.
- There was a positive family history in 12.5% of children.
- Most common age group affected include 4-6 years.
- Most common site of initial lesion was head and neck followed by upper limb, lower limb and trunk.
- Most common clinical type was vitiligo vulgaris followed by focal type then segmental. Lip tip type was least common type.
- Cutaneous association was seen in 26.25%.
- Body surface area involving < 20% was found in 73.75%.

- Localized facial and mucosal lesions best respond to topical steroid and immunomodulator combination. The compliance of the patients was very good with this treatment modality.

- NBUVB therapy is an effective and safe modality to treat generalized vitiligo with cosmetically acceptable repigmentation.

- Punch grafting proves to be easier, faster and least expensive method of treatment in stable vitiligo cases.

- Split skin grafting carries a distinct advantage over mini punch grafting in producing excellent cosmetic matching over larger areas using fewer grafts.

Further several such studies need to be done on a larger scale to compare the epidemiology, clinical spectrum and various newer modalities of treatment ,their responses, adverse effects and to derive a standard protocol for treating this less studied entity childhood vitiligo.

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Sl.No.	age in years	sex	age of onset in yrs	DU in mths	FH	SK	SYM	ASYM	KB	H in P	H in G	Nail	Mucosa	Type
1	4	m	4	6	-	+	✓		-	N	N	N	-	V.V
2	12	f	11	12	-	+		✓	-	N	N	N	-	SM
3	5	f	5	5	-	+	✓		-	N	N	N	+	AF
4	10	m	10	1	-	M	✓		-	N	N	N	-	V.V
5	9	f	9	4	-	+		✓	-	L	N	N	-	F
6	9	m	9	5	+	+		✓	-	N	N	P	+	SM
7	4	f	4	7	-	+		✓	-	N	N	N	-	SM
8	5	m	5	3	-	+		✓	-	N	N	N	-	M
9	11	m	11	1	-	+	✓		-	N	N	N	-	V.V
10	3	f	3	3	-	+	✓		-	N	N	N	-	LT
11	11	f	11	5	-	+	✓		-	N	N	N	-	V.V
12	6	f	6	1	-	M		✓	-	N	N	N	+	SM
13	6	m	6	6	-	+	✓		-	N	N	P	-	AF
14	8	f	8	4	-	+	✓		-	N	N	N	-	V.V
15	5	m	5	1	+	+		✓	-	L	N	N	-	SM
16	5	f	5	3	-	+		✓	-	N	N	N	-	F
17	8	m	7	12	-	+	✓		-	N	N	N	-	V.V
18	4	f	4	1	-	M	✓		-	N	N	N	-	V.V
19	6	f	5	12	-	+		✓	-	L	N	N	-	F
20	10	f	10	2	-	+		✓	-	N	N	N	-	SM
21	9	m	9	3	-	+		✓	-	N	N	P	+	M
22	3	m	3	6	-	M	✓		-	N	N	N	-	V.V
23	5	m	5	1	-	+		✓	-	N	N	N	-	SM
24	11	f	10	10	+	+	✓		-	N	N	N	-	V.V
25	5	f	5	3	-	+		✓	-	N	N	N	-	F
27	6	f	6	5	+	+	✓		-	N	N	N	+	V.V
28	8	f	8	2	-	+		✓	-	N	N	N	-	M
29	9	m	9	6	-	+		✓	-	N	N	N	-	SM
30	4	m	4	3	-	+	✓		-	N	N	N	-	V.V
31	9	f	9	7	-	+		✓	-	N	N	P	+	F
32	6	f	6	5	-	+		✓	-	N	N	N	-	SM
33	12	m	11	10	-	M		✓	-	L	N	N	-	F
34	7	f	7	5	-	+	✓		-	N	N	N	-	V.V
35	5	f	5	3	-	+		✓	-	N	N	N	-	F
36	7	m	7	6	-	+	✓		-	N	N	N	+	V.V
37	11	f	11	2	-	+		✓	-	L	N	N	-	F
38	6	f	6	2	-	+	✓		-	N	N	P	-	AF
39	8	f	8	5	+	+		✓	-	N	N	N	-	SM
40	2	m	2	2	-	M	✓		-	N	N	N	-	V.V

41	6	m	6	4	-	+		✓	-	N	N	N	-	M
42	5	m	5	7	-	+		✓	-	L	N	N	-	SM
43	9	f	9	3	-	+		✓	-	N	N	N	-	F
44	6	f	6	4	-	+	✓		-	N	N	N	+	V.V
45	10	m	10	1	+	+		✓	-	N	N	N	-	F
46	11	m	10	9	-	+	✓		-	N	N	P	-	AF
47	4	f	4	3	-	M	✓		-	N	N	N	+	V.V
48	7	f	7	6	-	+	✓		-	N	N	N	-	V.V
49	8	m	8	4	-	+	✓		-	N	N	N	-	V.V
50	1.5	f	1	5	-	+		✓	-	N	N	N	-	SM
51	8	f	8	2	-	+		✓	-	L	N	N	-	M
52	9	m	8	10	+	+	✓		-	N	N	N	-	V.V
53	6	f	6	3	-	M		✓	-	N	N	N	-	F
54	9	f	9	4	-	+		✓	-	N	N	N	+	SM
55	4	m	3	9	-	+		✓	-	N	N	N	+	M
56	12	m	11	10	-	+		✓	-	N	N	N	+	F
57	6	f	6	3	-	+		✓	-	N	N	N	-	SM
58	5	f	5	4	-	M		✓	-	N	N	N	-	F
59	12	m	11	12	-	+		✓	-	N	N	P	-	M
60	7	f	7	6	-	+	✓		-	N	N	P	-	V.V
61	6	m	6	3	-	+	✓		-	N	N	N	-	V.V
62	7	f	7	5	-	+		✓	-	L	N	N	-	F
63	8	m	7	10	+	M	✓		-	N	N	N	-	V.V
64	5	f	5	3	-	+		✓	-	N	N	N	-	SM
65	4	m	4	4	-	M		✓	-	N	N	N	-	F
66	5	m	5	6	-	+	✓		-	N	N	N	+	AF
67	7	m	7	2	-	M		✓	-	N	N	P	-	F
68	2	f	1.5	5	-	+	✓		-	N	N	N	-	V.V
70	7	f	7	2	-	+		✓	-	N	N	N	-	M
71	5	f	5	7	+	+		✓	-	N	N	N	+	F
72	8	f	8	5	-	+		✓	-	N	N	P	-	SM
73	6	m	5	9	-	+		✓	-	L	N	N	-	F
74	6	m	5	8	-	+		✓	-	N	N	N	+	M
75	8	f	7	10	-	+	✓		-	N	N	P	-	LT
76	5	f	5	4	-	+		✓	-	N	N	N	-	F
77	6	m	6	1	-	+		✓	-	N	N	N	-	F
78	10	m	10	3	-	+		✓	-	L	N	N	-	SM
79	6	f	5	8	-	+		✓	-	N	N	N	+	F
80	7	f	6	6	-	+		✓	-	N	N	N	-	SM

Sl.No	ENT	Eye	Dental	Endo	Hb in gms%	TC	Nt in %	Lym in %	Es in %	mono in %	motion O/C	RBS in mg%	LFT	X- ray chest	S.A in %	Tt	Du in mths	EXC	MKD	DEF	MIN	NR	S/E
1	N	N	N	N	11	7500	55	34	3	-	-	82	WNL	N	>20	NUVB	6	✓					B
2	N	N	C	N	13	6700	65	32	4	-	-	98	-	-	5	S	6				✓		
3	N	N	N	N	12	8900	57	40	6	2	-	122	-	-	1	TS+IM	6	✓					
4	T	N	G	N	11	11000	56	31	2	-	-	120	-	-	1	TS+IM	6			✓			
5	N	N	C	N	11	8700	67	36	3	-	-	87	-	-	>20	NUVB	6		✓				
6	N	N	N	N	10	10700	72	35	7	-	-	94	-	-	4	S	6				✓		
7	T	EV	N	N	13	9800	57	37	10	-	O,C	87	-	-	>20	NUVB	6	✓					
8	N	N	C	N	12	5600	64	29	3	-	-	90	-	-	2	TS+IM	6					✓	I
9	N	N	N	N	13	10600	73	33	6	-	-	89	-	-	>20	NUVB	6		✓				
10	P	N	G	N	10	3500	72	36	7	-	-	100	-	-	5	S	6				✓		
11	N	M	G	N	11	13500	78	31	4	-	-	123	WNL	N	5	S	6			✓			
12	N	N	N	N	9	8900	65	38	5	-	-	140	-	-	2	TS+IM	6	✓					
13	T	N	C	N	12	7500	56	37	5	-	-	112	-	-	>20	NUVB	6					-	
14	N	EV	N	N	10	4700	77	34	4	-	-	87	WNL	N	3	TS+IM	6			✓			I,B
15	N	N	C	N	13	6700	67	40	3	-	-	98	-	-	>20	NUVB	6					-	
16	P	N	N	N	12	8900	56	39	8	-	-	113	-	-	4	TS+IM	6			✓			
17	N	N	N	N	10	4800	76	32	5	-	-	142	-	-	5	S	6					✓	
18	N	N	C	N	9	11800	78	36	9	-	-	136	-	-	5	S	6			✓			
19	N	N	C	N	12	10500	65	38	3	-	-	123	-	-	<1	TS+IM	6	✓					
20	T	N	N	H	13	6700	76	33	4	-	-	107	-	N	<1	TS+IM	6			✓			
21	P	N	N	N	11	9800	58	41	5	-	-	89	-	N	>20	NUVB	6	✓					B
22	T	EV	C	N	11	7800	76	34	6	-	-	93	-	-	1	TS+IM	6					✓	
23	N	N	N	N	11	11900	67	36	2	-	-	123	-	-	>20	NUVB	6		✓				
24	N	N	G	N	12	12500	55	32	8	-	-	154	-	-	1	TS+IM	6					✓	
25	N	N	N	N	13	4500	78	36	5	-	-	117	-	-	>20	NUVB	6					-	
26	N	my	C	N	10	12000	71	33	4	-	-	143	WNL	N	1	TS+IM	6	✓					
27	N	N	N	N	12	5600	63	41	6	-	-	132	-	-	<1	TS+IM	6	✓					
28	P	N	N	N	10	7600	68	36	7	-	-	165	-	-	1	TS+IM	6			✓			
29	N	N	N	N	11	5600	73	33	4	-	-	134	-	-	>20	NUVB	6				✓		
30	N	N	N	N	9	8700	59	38	5	-	-	123	-	-	1	TS+IM	6					✓	
31	N	N	C	N	12	8000	73	34	7	-	-	143	-	-	<1	TS+IM	6			✓			
32	N	N	N	N	11	6700	65	35	8	-	-	135	-	-	>20	NUVB	6				✓		
33	T	N	N	N	10	7800	72	39	3	-	-	121	WNL	-	1	TS+IM	6		✓				
34	N	C	G	N	12	9500	62	33	4	-	-	111	-	N	1	TS+IM	6	✓					
35	N	N	C	N	13	10200	65	36	6	-	-	87	-	-	1	TS+IM	6			✓			
36	N	N	N	N	11	11200	78	38	5	-	-	97	-	-	>20	NUVB	6		✓				
37	P	N	C	N	10	9800	75	39	3	-	O,C	88	-	N	1	TS+IM	6					✓	
38	N	N	N	H	9	5600	54	34	7	-	-	90	-	-	5	S	6			✓			
39	N	EV	N	N	11	7600	68	33	8	-	-	85	-	-	>20	NUVB	6					-	
40	N	N	G	N	11	3400	71	36	4	-	O,C	90	-	-	1	TS+IM	6					✓	
41	N	N	N	N	11	5800	68	32	5	-	-	110	-	-	>20	NUVB	6				✓		
42	T	co	N	N	11	8700	57	31	9	-	-	120	-	N	>20	NUVB	6			✓			B

43	N	N	N	N	12	5600	75	38	7	-	O,C	111	-	N	<1	TS+IM	6					✓	
44	N	N	G	N	13	9000	66	34	8	-	-	132	WNL	-	1	TS+IM	6			✓			
45	T	N	C	N	10	12900	59	35	5	-	O,C	143	-	-	1	TS+IM	6			✓			
46	N	N	N	N	9	13400	60	36	6	-	O,C	154	-	-	<1	TS+IM	6	✓					
47	N	N	N	N	12	6700	70	33	4	-	-	165	-	-	1	TS+IM	6				✓		
48	N	my	N	N	11	9700	56	30	5	-	-	123	-	-	>20	NUVB	6		✓				
49	N	N	C	N	10	9500	76	33	6	-	-	132	-	-	1	TS+IM	6					✓	
50	T	N	N	N	12	10400	56	36	3	-	-	143	-	-	<1	TS+IM	6		✓				
51	N	N	N	N	13	12300	76	34	7	-	-	112	-	-	1	TS+IM	6			✓			
52	N	N	G	N	14	9800	57	35	3	-	-	121	-	N	2	S	6		✓				
53	N	N	C	N	11	7800	59	34	8	-	-	89	-	-	>20	NUVB	6			✓			
54	N	N	C	N	10	6800	65	33	5	1	O,C	132	WNL	N	2	S	6		✓				
55	P	N	N	N	9	9500	66	39	6	-	-	90	-	-	2	S	6			✓			
56	T	N	N	N	12	5600	72	38	4	-	-	89	-	-	<1	TS+IM	6	✓					
57	N	N	G	N	11	6700	59	33	8	-	-	134	-	-	2	S	6					✓	
58	N	N	N	N	13	8700	67	32	3	-	-	154	-	-	>20	NUVB	6		✓				
59	N	N	C	N	10	11300	66	31	4	3	-	121	-	-	3	S	6	✓					
60	N	N	N	N	13	9800	75	34	6	-	O,C	109	-	-	1	TS+IM	6		✓				
61	T	N	N	N	12	7600	67	41	8	-	-	105	-	N	1	TS+IM	6				✓		
62	N	N	G	N	13	12900	56	40	6	-	-	123	-	-	<1	TS+IM	6					✓	
63	N	EV	C	N	11	13600	67	39	7	-	-	154	-	-	1	S	6	✓					
64	P	N	N	N	11	10400	68	33	7	-	-	176	-	-	<1	TS+IM	6					✓	
65	N	N	N	N	10	4500	57	32	10	-	-	88	-	N	1	S	6		✓	✓			
66	T	N	C	N	9	7800	66	36	3	-	-	132	-	-	5	S	6			✓			
67	N	EV	N	N	12	9500	78	35	4	-	-	89	-	-	1	TS+IM	6					✓	
68	N	N	C	N	11	11000	82	31	5	1	-	90	WNL	-	<1	TS+IM	6		✓				
69	P	my	N	N	10	6700	76	33	6	-	-	94	-	-	1	TS+IM	6				✓		
70	N	N	G	H	11	8700	58	36	7	-	-	123	-	-	>20	NUVB	6			✓			
71	N	N	N	N	12	9500	66	38	8	-	-	143	-	-	5	S	6					✓	
72	N	N	C	N	13	10900	74	32	3	-	-	133	-	N	1	TS+IM	6		✓				
73	T	N	N	N	11	12000	63	35	5	-	O,C	127	-	-	5	S	6			✓			
74	N	my	N	N	13	13500	55	36	4	1	-	109	-	-	1	TS+IM	6				✓		
75	P	N	C	N	12	12000	67	37	7	-	-	123	-	-	<1	S	6		✓				
76	N	N	N	N	10	14600	80	32	8	-	-	143	-	-	>20	NUVB	6	✓					
77	N	N	C	N	9	3700	67	31	9	-	-	123	-	N	1	TS+IM	6				✓		
78	P	N	N	N	12	5600	58	36	4	-	-	143	-	-	>20	NUVB	6					-	B
79	N	N	N	N	11	7800	60	32	6	-	-	123	-	-	1	TS+IM	6		✓				
80	T	N	C	N	10	9300	65	31	8	1	-	156	WNL	-	5	TS+IM	6		✓				I,B

KEY T0 MASTER CHART

m	-	MALE
F	-	FEMALE
YR	-	YEAR
DU	-	DURATION
FH	-	FAMILY HISTORY
SK	-	SKIN LESION
SYM	-	SYMMETRICAL
ASYM	-	ASYMMETRICAL
Kb	-	KOEBNERIZATION
H IN P	-	HAIR IN PATCH
H IN G	-	HAIR IN GENERAL
M	-	MUCOSAL
N	-	NORMAL
P	-	PITTING
VV	-	VITILIGO VULGARIS
SM	-	SYMMETRICAL
AF	-	ACROFACIALVITILIGO

F	-	FOCAL
LT	-	LIPTIP
T	-	TONSILLITIS
P	-	PHARYNGITIS
EV	-	EYELID VITILIGO
MY	-	MYOPIA
CO	-	CONJUNCTIVITIS
C	-	CARIES
G	-	GINGIVITIS
H	-	HYPOTHYROIDISM
ENDO	-	ENDOCRINE
HB	-	HEMOGLOBIN
TC	-	TOTAL COUNT
NT	-	NEUTROPHIL
LYM	-	LYMPHOCYTE
ES	-	EOSINOPHIL
MONO	-	MONOCYTE
O/C	-	OVA / CYST

LFT	-	LIVER FUNCTION TEST
RBS	-	RANDOM BLOOD SUGAR
WNL	-	WITHIN NORMAL LIMITS
SA	-	SURFACE AREA
TT	-	TREATMENT
NBUVB	-	NARROW BAND ULTRAVIOLET B RAYS
TS+IM	-	TOPICAL STEROIDS AND IMMUNOMODULATOR
S	-	SURGERY
EXC	-	EXCELLENT
MKD	-	MARKED
DEF	-	DEFINITE
MIN	-	MINIMAL
NR	-	NO RESPONSE
S/E	-	SIDE EFFECTS
B	-	BURNING
I	-	ITCHING

PROFORMA

Name	:	
Age/sex	:	
Serial no	:	
Date	:	
Address	:	
Complaints	:	
H/o present illness	:	
Age of onset	:	
Duration of skin lesions	:	
Duration of mucous membrane lesions	:	
Stationary or progressive	:	
H/o itching	:	YES/NO
H/o photosensitivity	:	YES/NO
H/o drug intake	:	YES/NO
H/o trauma	:	YES/NO
H/o previous skin lesions	:	YES/NO
H/o of contact with chemicals/cosmetics	:	YES/NO
H/o ocular symptoms	:	YES/NO
H/o impaired hearing	:	YES/NO

Past H/o:

H/o previous treatment	:	
Duration of treatment	:	
H/o pulmonary tuberculosis	:	YES/NO

Family H/o:

Any other family members affected	:	YES/NO
Dietary habits	:	

General examinations:

Nutrition	:	
Height	:	
Weight	:	

Systemic examination:

CVS	:	
RS	:	
ABDOMEN	:	
CNS	:	
OPTHALMOLOGY	:	
ENT	:	
ENDOCRINE	:	

Dermtological examination

Scalp	Face		Neck	Trunk		Flexures	
	ERt	LLt		Front	Back	Right	Left

Upper Limb		Lower Limb		Palm Sole		Mucous Membrane	
Rt	Lt	Rt	Lt	Rt	Lt	Oral	Genital

Morphology	:	
Size	:	Macule/patch
Surface area	:	%
Number	:	single/multiple
Border	:	inflammatory/non-inflammatory
u/l or b/l	:	
Sym/asym	:	
Koebnerization	:	yes/no
Hair in the lesional skin	:	normal/ depigmented
Nail	:	
Special types	:	trichrome/ quadrichrome

Clinical types

1.Localised	:	Focal / segmental / mucosal
2.Generalised	:	vitiligo vulgaris/acrofacial/mixed
3.Universal		
4. lip tip		

Other associated dermatological conditions

thyroid disorders	:
pernicious anemia	:
diabetes mellitus	:
Alopecia areata	:
Halo naevus	:
Morphoea	:
Lichen sclerosus	:

Investigations

Hb%	:
TC	:
DC	:
Blood urea	:
Blood sugar(f), (pp)	:
Motion -ova, cyst	:
ENT	:
Eye	:
Dental	:

For selected cases only

Endocrine assessment :

Liver function test :

x-ray chest PA view :

IQ assessment :

CT brain :

Duration of treatment :

Response

Excellent :

Marked :

Definite :

Minimal :

Absent :

Side effects :

Follow up :

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Rangaraj M
PG in MDDVL
Madras Medical College, Ch-3

Dear Dr. Rangaraj.M

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " Childhood Vitiligo – Epidemiological study , Clinical spectrum and Therapeutic Responses" No. 14112010.

The following members of Ethics Committee were present in the meeting held on 24.11.2010 conducted at Madras Medical College, Chennai -3.

1. Prof. S.K. Rajan, MD	-- Chairperson
2. Prof. J. Mohanasundaram, MD,Ph.D,DNB Dean, Madras Medical College, Chennai -3	-- Deputy Chairman
3. Prof. A. Sundaram, MD Vice Principal , MMC, Chennai -3	-- Member Secretary
4. Prof R. Sathianathan, MD Director, Institute of Psychiatry, MMC, Ch-3	-- Member
5. Prof. R. Nandhini, MD Director, Institute of Pharmacology, Ch-3	-- Member
6. Prof. Pregna B. Dolia , MD Director, Institute of Biochemistry, MMC, Ch-3	-- Member
7. Prof. C. Rajendiran , MD Director, Institute of Internal Medicine, MMC, Ch-3	-- Member
8. Thiru. S. Govindasamy BA.BL	-- Lawyer
9. Tmt. Arnold Soulina	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee