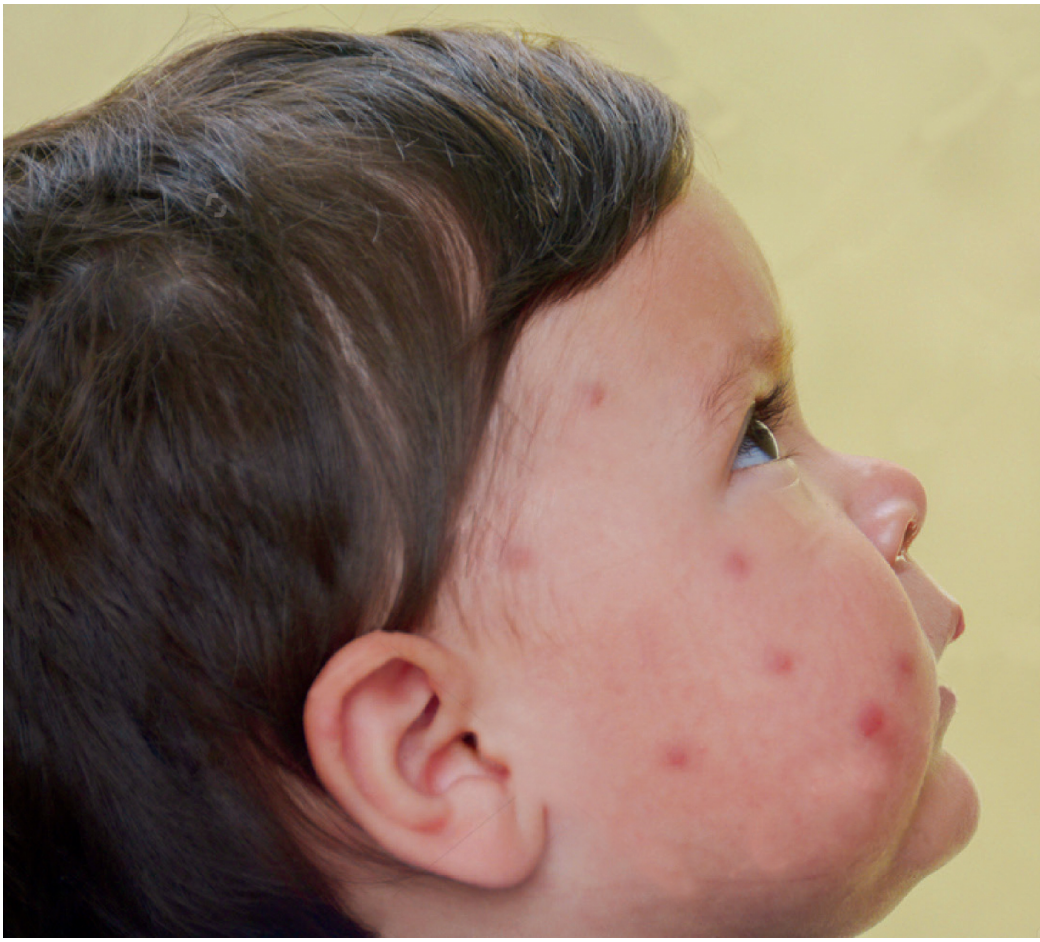
IADVL

SIG Pediatric Dermatology (IADVL Academy) Newsletter

Issue 1, April 2017



In This Issue

How I Manage Extensive Alopecia Areata in a 5-Year-Old

Dr Manish K. Shah

Associate Professor

Bai Jerbai Wadia Hospital for Children, Mumbai

The first thing I do when a parent gets a child with alopecia areata is spend some time explaining in some detail what alopecia areata (AA) is. I then inform them that children with AA have a guarded prognosis, especially if they have come with more than 50% scalp involvement, ophiasis, lesions on the body, a positive family history of AA or other autoimmune disease, a personal history of atopy, or has nail changes.

I give the parents the option of getting antimicrosomal antibody, antithyroglobulin antibody, free T4, and thyroid-stimulating hormone (TSH) tests done. I offer these mainly to parents who question persistently about investigations. I do tell them that thyroid abnormalities may be associated with AA. But the presence of thyroid abnormalities does not correlate with the severity of AA.[1] Also if thyroid antibodies are high and the patient is euthyroid, treatment is not required. Regular monitoring for free T4 and TSH is all that is required.

Since no medication alters the course of AA, I prefer to avoid systemic steroids or immunosuppressants in this situation.

I prescribe oral zinc and B-complex supplements as a co-prescription primarily because the parents do not feel satisfied unless some oral medication is prescribed. Zinc for around 3 months is quite safe.

If less than 50% scalp is involved, I prescribe topical clobetasol ointment twice a day to affected areas. I ask parents to apply a thin film for 6 weeks. Clobetasol propionate has been safely and effectively used in children more than 2 years old with AA involving more than 10% scalp.[2]

At the 6-week follow-up period, I give topical clobetasol a break. Here I introduce topical anthralin. I prescribe the 1.1% anthralin formulation that is commercially available. The instructions are as follows: Dilute one part of Derobin ointment with three parts of Vaseline petroleum jelly. Apply at night to hairless areas. Wash hands immediately after applying or use gloves, and wash off after 1 hour for 10 days. Then keep overnight. Use for 6 weeks.

By the end of 3 months, I prescribe another course of topical steroids, either topical clobetasol again or one of mometasone furoate or fluticasone propionate based on regrowth pattern and presence or absence of atrophy. After 6 weeks of repeat topical corticosteroid use, if at all there is any need it is time for another anthralin cycle. In some cases, I prescribe once a day minoxidil 2% in combination with topical steroids.

In cases with more than 50% scalp involvement, I recommend the above management quadrant-wise to limit possible side effects.

In cases unresponsive to the above approach or with more extensive involvement, I have tried topical immunotherapy with diphencyprone (DPCP). The results have been mixed. DPCP is worth a trial in an institution, but very cumbersome and time consuming in private practice. Response is very slow and loads of patience with innumerable weekly visits are required.

For eyebrow involvement, I prescribe topical fluticasone or topical tacrolimus 0.1%. Sometimes I would put in a short 1-month burst of topical

mometasone. I would leave eyelid or other body lesions in this age group alone.

I further reiterate avoidance of systemic corticosteroids for childhood AA. Long-term studies using steroids, including pulse high-dose steroids have shown poor long-term outcome.[3–5] Cyclosporine, likewise, produces striking regrowth, but almost an instantaneous relapse. If really required, methotrexate is quite safe and reasonably effective.

In this age group, parents need a lot of support to deal with this kind of situation happening to their child. I encourage parents to visit www.naaf.org (National Alopecia Areata Foundation) so that they know how people throughout the world cope with this distressing condition.

TIPS AND TRICKS

1. During the initial counselling session, I prime parents that you need to give any treatment modality in AA at least a 3-month trial before discarding it as ineffective. Too often we see practicing dermatologists changing medications every 2 weeks due to pressure from the child's parents. This adds nothing therapeutically and just heightens anxiety.
2. I use the dermoscope to demonstrate early regrowth. This instils confidence in parents' minds.

REFERENCES

1. Kasumagic-Halilovic E. Thyroid autoimmunity in patients with alopecia areata. Acta Dermatovenerol Croat. 2008;16(3):123–125.
2. Lenane P, Macarthur C, Parkin PC, Krafchik B, DeGroot J, Khambalia A, Pope E. Clobetasol propionate, 0.05%, vs hydrocortisone, 1%, for alopecia areata in children: a randomized control trial. JAMA Dermatol. 2014;150:47–50.
3. Hubiche T. Poor long term outcome of severe alopecia areata in children treated with high dose pulse corticosteroid therapy. Br J Dermatol. 2008;158:1136–1137.
4. Staumont-Salle D, Vonarx M, Lengrand F, et al. Pulse corticosteroid therapy for alopecia areata: long-term outcome after 10 years. Dermatology. 2012;225:81–87.
5. Smith A, Trueb B, Theiler M, Segard M, Delaporte E. High relapse rates despite early intervention with intravenous methylprednisolone therapy for severe childhood alopecia areata. Pediatr Dermatol. 2015;32:481–487.

How I Manage Hemangioma of Infancy on the Face of a 2-Month-Old Child

Catherine S. Jury

Consultant Dermatologist

Royal Hospital for Children, Glasgow, UK

The advent of beta-blocker treatment for infantile hemangioma has revolutionized our approach to this common pediatric dermatology presentation. The dramatic responses to treatment and the relatively low incidence of side effects make it an attractive option, but it's worth remembering that, for this self-limiting condition, not all children require medical intervention.

INITIAL ASSESSMENT

- Is the hemangioma causing or likely to cause functional difficulties, for example, affecting the eyelid or lip?
- Is the hemangioma likely to result in significant cosmetic difficulties, for example, tip of nose?
- Is there a reason to be concerned about associated abnormalities, for example, plaque-like hemangioma on face raises possibility of PHACES (posterior fossa malformation, hemangioma, arterial anomalies, cardiac anomalies, eye anomalies, sternal defect) syndrome?

SITE UNLIKELY TO RESULT IN SIGNIFICANT FUNCTIONAL OR COSMETIC DIFFICULTY

No treatment is a reasonable option; allow natural involution. The majority of hemangiomas complete the involution process within the first 5 years of life. Any residual skin changes can be dealt with by surgical remodeling or laser removal should the need arise.

For parents keen on some form of treatment, topical beta blocker can help to speed the involution process and is generally well tolerated. Commonly used is Timolol gel–forming solution 0.5%–1%; one drop is applied directly to the hemangioma two times daily.[1]

SITE LIKELY TO CAUSE EITHER FUNCTIONAL OR SIGNIFICANT COSMETIC DIFFICULTY

Consider suitability for systemic beta-blocker treatment.[2] In a child over the age of 2 months, this can be safely started without admission to hospital with appropriate care and support.

PRETREATMENT EVALUATION

- History—Wheezing, cardiac abnormality, prematurity, poor feeding, hypoglycemia.
- Family history—Asthma, cardiac rhythm abnormalities of sudden death, congenital heart abnormality, maternal connective tissue disease.
- Examination—Auscultation of lungs and heart, blood pressure, weight.
- Investigation—Only if indicated by history or examination—blood glucose. Consider electrocardiogram (ECG) and cardiology review if examination or history identify any areas of concern (listed earlier).

Special consideration

Plaque hemangioma on face—consider PHACE syndrome—abnormalities in brain, heart, vasculature, and eyes. Baseline investigations should

include thyroid function, echocardiogram (ECHO), and magnetic resonance imaging/magnetic resonance angiography (MRI/MRA) brain.

INITIATION OF TREATMENT IN CHILD OVER 2 MONTHS OF AGE AND >3 KG WEIGHT

Starting dose of propanolol 1 mg/kg/day in three divided doses (consider 0.5 mg/kg in PHACE syndrome)

Reassess heart rate after 3–7 days before increase to 2 mg/kg in three divided doses.

A small number of children with large or deep hemangiomas may require up to 3 mg/kg for adequate response.

Note: Bradycardia is a much more reliable indicator of toxicity than blood pressure, which is difficult to record in infants. Although propranolol lowers blood pressure, this is almost invariably short lived and asymptomatic.

SIDE EFFECTS

Hypotension (see **Appendix 1**), bradycardia, second or third degree heart block, heart failure, hypoglycemia, wheeze, cold hands and feet, night terrors.

In practice, beta blockers tend to be well tolerated and side effects are relatively rare.

If side effects such as agitation and night terrors occur, consider changing to atenolol, which is also well tolerated but does not cross the blood–brain barrier.[3]

IMPORTANT DISCUSSION WITH FAMILY

Need for regular feeds—Omit dose if not feeding/reduced feeding.

Seek immediate review if excessively drowsy or clammy.

FOLLOW-UP

Every 1–2 weeks until reached adequate treatment dose of 2–3 mg/kg per day 4 weekly for 2 months, 8 weekly thereafter.

DURATION OF TREATMENT

Largely directed by response to beta blocker.

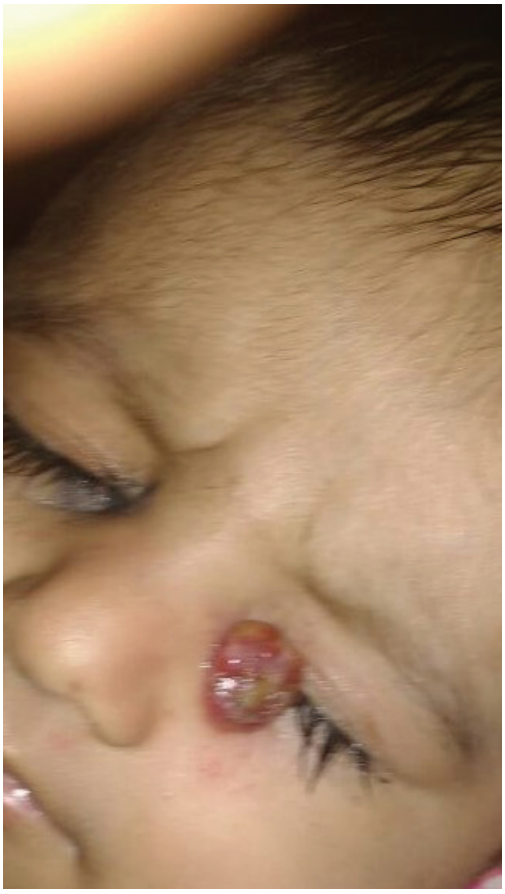
For most patients, maintain at 2 mg/kg per day until 10–12 months of age; at 12 months, stop further dose increases with a view to weaning off and stopping treatment by 18 months.

COMPLICATIONS OF HEMANGIOMAS OF INFANCY

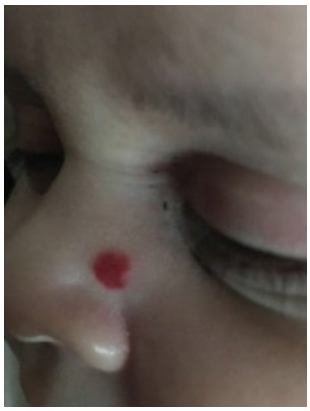
Site	Potential complications
Periocular	Obstruction of vision which can lead to amblyopia
Perioral	Difficulty in feeding, prone to ulceration
Beard area	Associated hemangioma of larynx and vocal cords
Nappy area	Prone to ulceration
Nose	Difficulty in breathing, cosmetic damage
Ear	Difficulty in hearing

Tabulation by Dr Ram Gulati.

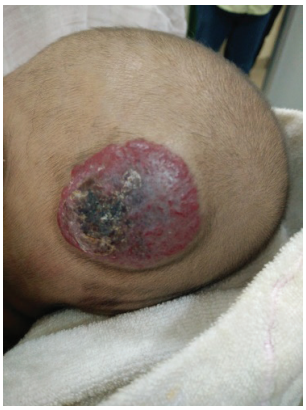
Hemangioma of Infancy—More than a Single Entity



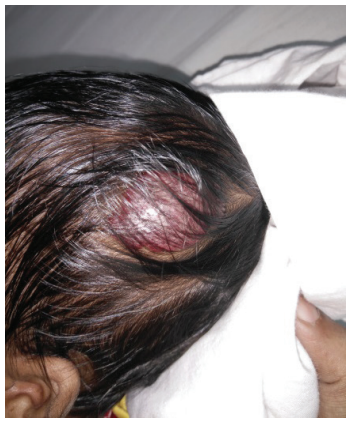
Superficial (face)
(likely to need cosmetic correction)



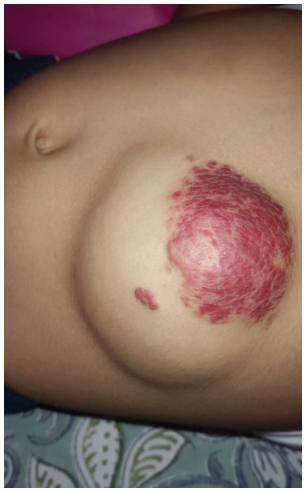
Superficial (face)
(unlikely to need cosmetic correction as would likely involute)



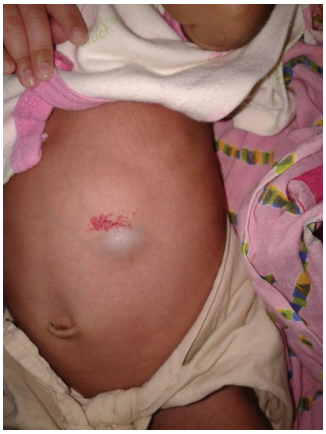
Mixed (scalp)
(likely to need cosmetic correction as scarred and likely to lead to permanent hair loss)



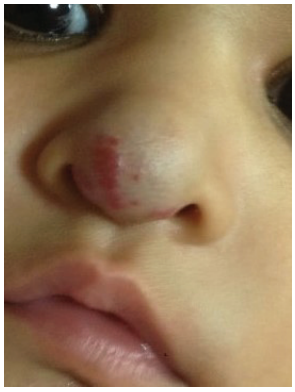
Mixed (scalp)
(unlikely to need cosmetic correction as may resolve with normal hair growth on top)



Mixed (abdomen)
(likely to leave some loose skin which may or may not need cosmetic correction)



Mixed (with a prominent deeper component)
(unlikely to need cosmetic correction)



Mixed (nose)
(started on propranolol as likely to need cosmetic correction)

REFERENCES

1. Chakkittakandiyil A, Phillips R, Frieden IJ, et al. Timolol maleate 0.5% or 0.1% gel-forming solution for infantile hemangiomas: a retrospective, multicenter, cohort study. Pediatr Dermatol. 2012;29(1):28–31.
2. Leaute-Labreze C, Dumas de la Roque E, Hubiche T, et al. Propranolol for severe hemangiomas of infancy. N Engl J Med. 2008;358(24):2649–2651.
3. Ábarzúa-Araya A, Navarrete-Dechent CP, Heusser F, Retamal J, Zegpi-Trueba MS. Atenolol versus propranolol for the treatment of infantile hemangiomas: a randomized controlled study. J Am Acad Dermatol. 2014;70(6):1045–1049.

QUIZ Time!

Dr Tanumay Raychaudhury

AU: Please provide degree and affiliation of author "Dr Tanumay Raychaudhury."

Let's see how well you know children's skin
(Answers on the last page)

1. A couple present with history of two children who have died with blistering skin disorders in the first month of life. They have a newborn at 5 days of life with new erosions. They want to know about prenatal testing. How will you counsel them?
   a. Prenatal testing cannot be done in India.
   b. Collect blood now for DNA tests on present child and do chorionic villi sampling at 10–12 weeks.
   c. Fetal skin biopsy at 24 weeks of next pregnancy.
   d. Collect skin from this child for immunofluorescence and predict next depending on autosomal dominant/recessive.
2. Neonate presents with vesicular rash at birth. There are few superficial pustules and occasional pigmented macules seen. Lesions are predominantly over chin, trunk, and buttocks. Tzanck test is negative for multinucleate giant cells, yeast, or bacteria but has neutrophils. Gram stain is negative. Your primary diagnosis is:
   a. Transient pustular melanosis
   b. Erythema toxicum
   c. Neonatal varicella
   d. Congenital syphilis
3. A 6-year-old nonatopic child presents with oval hypopigmented, well-defined macule, over the left cheek with mild scaling. Mother complains of dandruff. Lesion is asymptomatic. Stretch test is negative for pityriasis versicolor. How will you treat?
   a. Selenium sulfide shampoo
   b. Ketoconazole cream
   c. Low-potent steroid
   d. Emollient cream
4. Regularly arranged small clumps of melanin are seen in which silvery hair syndrome?
   a. Elejalde
   b. Chédiak–Higashi
   c. Griscelli
   d. Phenylketonuria
5. Molluscum contagiosum has highest clearance rates to
   a. Curettage
   b. Cantharidin
   c. Imiquimod
   d. Salicylic + lactic acid
6. Child with palmoplantar hyperhidrosis is advised botulinum toxin injection. Which statement is false about this?
   a. About 50 injections per palm, subepidermally placed, 2 U in each inject
   b. Each injection results in a 1.2-cm anhidrosis
   c. Mild transient thumb weakness can occur which lasts 3 days
   d. Action of anhidrosis is by acting at postganglionic sympathetic cholinergic nerves in the sweat glands
7. Which of these is not a part of the expanded criteria for juvenile dermatomyositis?
   a. Dysphonia
   b. Dysphagia
   c. Calcinosis
   d. Nailfold capillaroscopic abnormalities
8. A 7-year-old girl is brought with depigmented plaques over the vulval area involving the introitus. There is atrophy and mild hemorrhagic spots seen. History of application of unknown topical medicines for 1 week. Your provisional diagnosis is
   a. Lichen sclerosus
   b. Genital vitiligo
   c. Child abuse
   d. Steroid atrophy
9. Child with mild atopic dermatitis, parents ask you about dietary restrictions. You would
   a. Suggest no restriction
   b. Ask for a skin prick testing
   c. Ask for a radioallergosorbent test (RAST) serum IgE–specific test and follow-up with oral food challenge
   d. Blanket restriction of milk, eggs, fish, and seafood

10. A child presents at 2 years of age with recurrent sheets of monomorphic discrete pustules in crops, severe nail pitting and onycholysis, ichthyosis, oral mucositis, and conjunctivitis. Also has severe pain with restricted joint movement at both ankles. No high fever recorded. Multiple antibiotics used without any growth on pus culture. Chest X ray shows balloon-like swelling at the ends of ribs. Diagnosis includes

a. Muckle wells syndrome
b. Acute generalized exanthematous pustulosis (AGEP)
c. Generalized pustular psoriasis with arthritis of childhood
d. Deficiency of interleukin-1 receptor antagonist

Upcoming Events in Pediatric Dermatology

1. 13th World Congress of Pediatric Dermatology, Chicago, July 6–9, 2017
   wcpd2017.com
2. ISPD Annual Conference, Delhi, August 18–20, 2017
   www.peddermindia.org
3. 17th European Society of Pediatric Dermatology Annual Meeting, Palma de Mallorca, Spain, October 19–21, 2017
   www.espd.info

Interesting Excerpts from Recent Literature

TO TREAT OR NOT TO TREAT MOLLUSCUM CONTAGIOSUM

Molluscum contagiosum (MC) is a common viral disease primarily affecting children. The objective was to compare the effectiveness of curettage as a treatment modality for MC with no treatment.

A retrospective study of 2022 children with MC between 2008 and 2012.

A total of 1879 patients underwent curettage; 70% were cured after one treatment and 26% after two treatments. Satisfaction was high: 97% of children and parents.

COMMENT

Most current guidelines including those from Centers for Disease Control and Prevention advise against active treatment for a self-resolving viral infection such as MC among healthy children. This paper discusses how active treatment should be offered to achieve high patient satisfaction and faster cure. Unfortunately, as a retrospective study, the comparison is highly skewed in favor of curettage since a high majority were subjected to the procedure than left alone. From a practical point of view, active treatment should be only offered after adequate counselling that the disease is self-resolving.

Contributed by Dr Tanumay Raychaudhury

Source: Harel A, Kutz AM, Hadj-Rabia S, Mashiah J. To treat molluscum contagiosum or not—curettage: an effective, well-accepted treatment modality. Pediatr Dermatol. 2016; 33:640–645.

Active treatment should be offered despite the fact that MC is self-limiting. Curettage in an appropriate setting is very effective, with high patient satisfaction and fast cure rates.

DOES CHILDHOOD VITILIGO DIFFER WHEN HALO NEVI ARE PRESENT?

Halo nevi (HN) are much more common in vitiligo patients, especially children, but they do not significantly alter treatment response or risk for disease progression.

Vitiligo, characterized by depigmented patches due to loss of melanocytes, affects up to 4% of individuals worldwide. Childhood vitiligo differs from adult vitiligo regarding higher prevalence of segmental disease, history of atopy, and family history of autoimmune disease. HN with an acquired depigmented peripheral zone that occur in 1% of the general population, usually on the trunk, are more common in vitiligo patients than in the general population and are considerably more common in children with vitiligo than in adults.

Over a 24-year period, 208 children presented with vitiligo, 55 (26%) of whom had HN. Children with vitiligo-associated HN were more likely to be male (62%; $p = 0.03$) and older at presentation than those with vitiligo alone (5.8 vs. 7.3 years; $p = 0.01$).

Children with vitiligo-associated HN were more likely to have the generalized vitiligo subtype. No between-group differences were observed in body surface area affected or family history of vitiligo or autoimmune diseases. Patients with HN were no more likely to develop new vitiligo over a mean of 1.9 years, and there were no significant differences in repigmentation.

COMMENT

HN are up to 10 times more common in vitiligo patients than in the general population, and even more common in children versus adult vitiligo patients. These findings reassure us that HN do not significantly alter treatment response or risk for disease progression.

Contributed by Dr Tanumay Raychaudhury

Source: Cohen BE, Mu EW, Orlow SJ. Comparison of childhood vitiligo presenting with or without associated halo nevi. Pediatr Dermatol. 2016;33:44.

POTENTIAL CENTRAL NERVOUS SYSTEM EFFECTS OF PROPRANOLOL: NEED SAFER OPTIONS

Given its improved safety profile compared with systemic corticosteroids, propranolol has become the mainstay treatment of infantile hemangioma (IH) worldwide. There is evidence, mainly from adult volunteer studies, that propranolol use is associated with central nervous system (CNS) effects. Impairment to short- and long-term memory, psychomotor function, sleep quality, and mood with relatively low doses and durations of treatment have been reported. The exact magnitude of CNS effects resulting from propranolol use, especially in the early developmental stages and for prolonged periods of use, is not currently known. These effects may not be readily recognizable and require specialized assessment of cognitive function not routinely performed. Furthermore, there may be a delay between exposure and cognitive defects. The evidence to date provides a strong rationale to proceed with caution when prescribing propranolol for IH: treatment should be used only when indicated (in the presence of ulceration, impairment of a vital function, or risk of permanent disfigurement) and for a limited duration, and the benefits of treatment should be weighed carefully against potential adverse events before treatment is initiated. This narrative review describes the evidence for an effect of propranolol use on CNS function from volunteer and patient studies, including IH.

COMMENT

Since the serendipitous finding of usefulness of propranolol in IHs in 2008, there has been a flurry of case series and drug trials describing its efficacy, due to which propranolol has become the first line of management of IHs. However, being a lipophilic nonselective beta blocker, it crosses the blood–brain barrier and is associated with restless sleep, and other neurological side effects. The exact magnitude of CNS effects resulting from propranolol use, especially in the early developmental stages, is not currently known. Few studies have reported effects on memory, psychomotor function, and sleep quality. Hence, despite the good efficacy and relatively good adverse effect profile of propranolol, efforts should be continued for an even safer option, and if we aim to use a beta blocker in the treatment of hemangiomas, it seems judicious to use one that does not cross the blood–brain barrier such as atenolol or acebutolol.

The current review gives a detailed account of the various potential adverse effects on the developing brain due to beta blockers. It also provides the physiologic basis and mechanism by which propranolol may mediate these adverse effects such as its interactions with the noradrenergic system and the serotonin pathways. There may be a delay between exposure and cognitive defects. Although much data are based on the adult volunteer studies, it needs to be taken seriously as most of these studies were well-designed randomized controlled trials and crossover studies. Concluding, the infants who are initiated on propranolol need to be followed up over a long time to observe for these effects. These side effects would theoretically be alleviated with the use of a beta blocker to which the blood–brain barrier is impermeable.

Contributed by Dr Rahul Mahajan

Source: Langley A, Pope E. Propranolol and central nervous system function: potential implications for paediatric patients with infantile haemangiomas. Br J Dermatol. 2015; 172(1):13–23.

EXPLORING BABY OILS: THE EVIDENCE-BASED METHODS

Topical oils on baby skin may contribute to development of childhood atopic eczema. A pilot, assessor-blinded, randomized controlled trial assessed feasibility of a definitive trial investigating their impact in neonates. One-hundred and fifteen healthy, full-term neonates were randomly assigned to olive oil, sunflower oil, or no-oil group, twice daily for 4 weeks, stratified by family history of atopic eczema. We measured spectral profile of lipid lamellae, transepidermal water loss (TEWL),

stratum corneum hydration, and pH and recorded clinical observations, at baseline and 4 weeks post-birth. Recruitment was challenging (recruitment 11.1%; retention 80%), protocol adherence reasonable (79%–100%). Both oil groups had significantly improved hydration but significantly less improvement in lipid lamellae structure compared to the no-oil group. There were no significant differences in TEWL, pH, or erythema/skin scores. The study was not powered for clinical significance, but until further research is conducted, caution should be exercised when recommending oils for neonatal skin.

COMMENT

Emollients are among the most commonly used agents in the routine care of the newborn as well as in the treatment of atopic eczema. This is not only due to the traditional practice of their use across different cultures worldwide but also due to various scientific studies that have promoted the use of emollients in the newborn. Among the emollients, the most commonly used are the vegetable oils such as olive oil, coconut oil, and sunflower seed oil. However, there is scarce literature proving their efficacy as an effective and safe emollient. Few previous studies comparing different vegetable oils concluded that sunflower seed oil is a better oil compared to olive oil, as the latter causes a significant reduction in stratum corneum integrity and thickness, does not significantly hydrate stratum corneum, and induces mild erythema in subjects with and without a history of atopic eczema. This is because of the higher content of linoleic acid in sunflower seed oil through its activation of peroxisome proliferator-activated receptor alpha (PPAR-α) receptor.

The "OBSeRvE" study, a very well-designed study, shows that there was no significant difference in the TEWL, skin erythema, and pH scores among various treatment groups. Although application of vegetable oils led to improved hydration, it also led to a significantly reduced improvement in lamellar structure compared to no-oil application as assessed by attenuated total reflectance Fourier transform infrared (ATR-FTIR) spectroscopy. This suggests that topical oil application may have negative impact on the baby's skin and may impede development of the lamellar lipid structures of the permeability barrier from birth. The present study contradicts previous traditional beliefs and literature by showing that no-oil application may be a better option than vegetable oil application. However, since the study was not adequately powered, more data need to be generated before arriving at a final conclusion. Further, similar studies need to be undertaken on coconut oil, which is one of the most commonly used vegetable oil in India.

Contributed by Dr Rahul Mahajan

Source: Cooke A, Cork MJ, Victor S, et al. Olive oil, sunflower oil or no oil for baby dry skin or massage: a pilot, assessor-blinded, randomized controlled trial (the Oil in Baby SkincaRE [OBSeRvE] Study). Acta Derm Venereol. 2016;96(3):323–330.

MAINTENANCE THERAPY WITH TOPICAL CALCINEURIN INHIBITORS: ARE OUR CONCERNS VALID?

In children with atopic dermatitis (AD), long-term maintenance therapy using topical corticosteroids (TCSs) and/or topical calcineurin inhibitors (TCIs) has been an issue of controversy. Traditionally, TCSs have been used, though associated with adverse effects including atrophy, rebound flares, and potential to cause systemic adverse effects by percutaneous absorption. Over past 20 years or so, TCIs have been used, though in a limited way owing to a debated boxed warning based on theoretical risk of malignancy (e.g., skin and lymphoma). To look into the safety profile of TCS and TCI, a comparative systematic search of PubMed was done for long-term (≥12 week) clinical trials of TCS or TCI treatment in patients <12 years of age with AD. MeSH terms, abstracts, and relevant article text were used as determinants to review the citations, and studies were excluded if they did not encompass subjects <12 years, or were <12 weeks' duration, retrospective, meta-analyses, or limited to anecdotal case reports. Twenty-seven trials met criteria, of which 21 included 5825 pediatric patients treated with TCIs, and 6 included 1999 patients treated with TCS. TCS studies were limited to low- to mid-potency steroids, and only one study included a vehicle control. Of the 21 TCI studies, 8 were vehicle controlled with well-reported safety data, and with only ≤5% of patients reporting discontinuation due to adverse effects (DAEs). Both TCI and vehicle groups showed similar cutaneous and systemic adverse events (AEs) with none reporting lymphoma. Safety data in TCS trials were less well reported and incidence of DAE was addressed in just two trials, while systemic and cutaneous AEs were mostly not reported.

Thus, the data overall support the long-term use of TCIs with evidence being robust, documenting safety and efficacy, while data supporting long-term TCS use are limited to low- to mid-potency products.

COMMENT

The above systematic review shows robust evidence in favor of using TCI as the preferred maintenance topical therapy in pediatric patients with AD. With the proactive approach favored over the more traditional reactive approach, this becomes even more relevant. The boxed warning based on the theoretical risk of developing malignancy with TCI seems to be ill-founded, and largely been extrapolated from the oral use of tacrolimus and that too in situations like solid organ transplant, where other factors contribute to development of secondary malignancies. It is therefore high time that the boxed warning is looked into and reframed, so that TCI can be used as standard-of-care maintenance therapy in pediatric patients with AD.

Contributed by Dr Ram Gulati

Source: Siegfried EC, Jaworski JC, Kaiser JD, Hebert AA. Systematic review of published trials: long-term safety of topical corticosteroids and topical calcineurin inhibitors in pediatric patients with atopic dermatitis. BMC Pediatr. 2016;16:75.

TESTING FOR SERUM IGE AND MONITORING FOR SEVERE ATOPIC DERMATITIS: BUSTING THE MYTH

Atopic dermatitis (AD) is a common chronic cutaneous condition with recognized association with asthma, allergic rhinitis (AR), and allergies. However, just like the pathophysiology of AD, these associated conditions are complex in nature and not fully understood. Though evidence exists for efficacy of specific immunotherapy (SIT) in pediatric asthma and AR, no robust data exist for such use in AD. Elevated IgE is seen in many patients with AD; however, it is an unreliable biomarker, as its value shows quite marked variability and fluctuation over time, has a poor positive predictive value as far as clinical relevance is concerned, and does not strongly correlate with disease activity. Despite this, many studies on SIT use either positive skin prick testing (SPT) or serum-specific IgE levels to monitor therapy. Barring few exceptions, allergen avoidance is usually not effective at controlling AD in children. The few studies that have investigated the efficacy of SIT in children with AD are fraught with conflicting results and lack of reproducibility with a standard treatment protocol. Limited studies have shown clinical improvement in mild-to-moderate AD cases, but no effect on more severe patients. As far as uncontrolled studies are concerned, they are difficult to interpret, due to the natural history of remission of AD over time in many patients without specific interventions. Disadvantages with implementing SIT are multitude including length of treatment, poor compliance, cost, and potential side effect profile.

COMMENT

Though subcutaneous and sublingual immunotherapy is being forwarded by few as helpful in AD patients with food allergy, it has not shown to alter the clinical outcomes in severe disease. Also, relying on IgE level, which in itself is quite a variable biomarker with poor correlation with disease activity, makes monitoring of improvement with SIT difficult. Apart from the possibility of adverse effects, SIT is a prolonged therapy, and with reasonable cost involved and dubious evidence, it is not recommended as a valid treatment for treating AD patients with food allergies. In fact, this may lead parents to believe that food is the most important trigger, and this may reduce their effort on keeping up with topical therapy which is the mainstay for AD management.

Contributed by Dr Ram Gulati

Source: Ginsberg DN, Eichenfield LF. Debates in allergy medicine: specific immunotherapy in children with atopic dermatitis, the "con" view. World Allergy Organ J. 2016; 9:16.

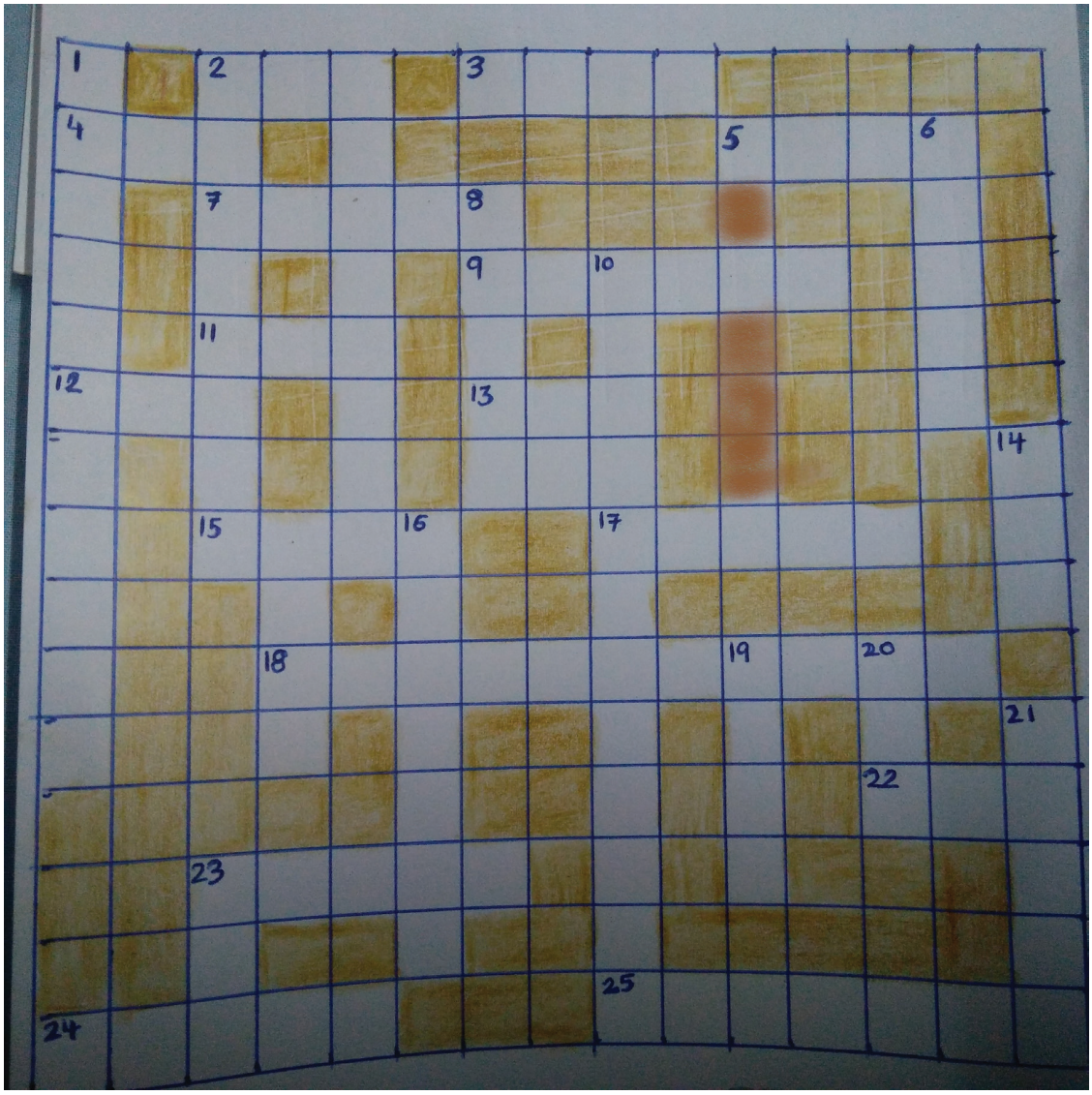
AU: Please provide degree and affiliation of author "Dr Ram Gulati."

Crossword Time!

Dr Ram Gulati

You know your children's skin well, but can we fit the right word/abbreviation in the provided squares is a trick not every mind is endowed with. Best of luck!

(Answers on the last page)



Clues (some straightforward, some cryptic; but surely with minds as sharp as our brilliant dermatologists, cracking the code should be no trouble at all)

Across

2. Dry deaf with problem in connexin 26
3. Bald boy with rough skin hates light
4. Dysphagia in a pigmented child who doesn't seem to cry
5. Glanzmann–Riniker syndrome
7. Component of WHIM
9. Mutation in AIRE gene causes this
11. SLICC criteria define it
12. Proteins found in saline extract of cell nuclei
13. Kind of fever associated with atopic eczema
15. X-linked immune dysfunction with eczematous rash
17. Bacteria cause the tongue to go black and
18. Miliary papulovesicles with staphylococcal infection
22. This pigmented condition is usually seen on cheeks
23. Associated with Rombo and Bazex–Dupre–Christol syndromes
24. Series of this makes the genetic code
25. Think of liver when prescribing it to children

Down

1. Telecanthus and hearing loss; may be caused by PAX3 mutation
2. Suspect early and push in IVIg
6. Neck in atopic dermatitis
8. Chamot looked at skin and bones
10. Eczema is the commonest cause
14. Hay–Wells syndrome
16. Cutaneous feature of hypothyroidism
19. Painless facial nodule with spontaneous healing
20. Dermal melanocytosis
21. GHR gene mutation causes short stature, delayed puberty, and thin hair

23 Check this out before you initiate NBUVB

A Brief Summary of the 'GENODEMA' Program, A Unique Event on February 3–5, 2017, at D Y Patil Hospital, Pune

Dr Nilendu Sarma

AU: Please provide degree and affiliation of author "Dr Nilendu Sarma."

It was a different stroke. A conference and hands-on training workshop on genodermatosis is difficult to conceptualize and execute. Dr Aayush and his team have done that flawlessly.

The program consisted of 1-day hands-on training on cell culture techniques on melanocyte, keratinocyte, and fibroblast. Sufficient skin samples and all the required instruments were kept ready. The team from Institute of Genomics & Integrative Biology (IGIB) led by Dr Archana Singh, another dynamic scientist, and her team taught all the trainees in a lucid manner. Many young and senior people from dermatology and different medical disciplines including biotechnology came to learn the procedure.

The conference duration was of one and half day, where the first day was devoted to basic genetic science related to dermatology. Finest quality of the speakers and the full house created the right academic milieu. Renowned speakers from both fields such as Dr Shubhu Phadke (HOD, Department of Genetics, SGPGI), Dr Sridhar Sivasubhu (IGIB), Dr Chaitanya Datar (Pune), Dr Prameela (Manipal), and few clinicians such as Dr Rahul Mahajan (PGI, Chandigarh) and Dr Rajendra Sinde enriched the audience. Dr Ravi Hiremangalore (Manipal) discussed about EB via webinar. There were discussions on latest advances in different genodermatosis such as EB, ichthyosis, TSC, NF, and various other rare genetic conditions such as infantile systemic hyalinosis. Different vital and practical aspects like preparing a genetic disease registry, pedigree chart preparation, DNA sequencing like NGS, microarray, and other methods of genetic diagnosis were discussed at length.

AU: Please spell out EB, TSC, NF, NGS.

I must say about the good number of audience. It was truly satisfying to experience their prolific knowledge on the subject.

I was chosen the moderator of the panel discussion possibly to get an unbiased moderation by the clinician without any genetic knowledge! I possibly represented the larger section of our fraternity! Many practical issues were discussed including the collaboration between the geneticist and clinicians and bilateral expectation.

Second day was on vitiligo with molecular advancement in this field. Dr Davinder Parsad and Dr Ravinder stunned again all of us with their work on dermal fibroblast and its role in vitiligo. Dr Archana presented the cell culture technique. There was award paper presentation too, and the quality of papers by the pediatrician, dermatologists, and basic research scholars were really impressive.

The entire program was conducted in DY Patil Institute, Pune. It was an amazing experience to be there. Food was perfect and tasty; hospitality was profuse.

The only thing that could have been better was time management. But for this kind of unprecedented initiative, this was more than expected.

Young energetic and dynamic Dr Aayush Gupta conceptualized this event. I saw huge potentiality in him. It will be surprising if I do not see him as the leading dermatologist in India in near future. He was very well supported by a mix of senior and young colleagues of him. The program was successful because of the synchronous work of his colleagues like Dr Sanjeev Gupta, Dr Kalyan Davale, Dr Avinash Jadav, Dr Rashi, Dr Dhara, Dr Preeti, Dr Sweety, and Dr Sagar. Possibly there were many I did not know. Dr Y. K. Sharma was simply amazing and led the team from the front. Dr Shubhu Phadke, HOD, was present actively despite his busy departmental schedule.

Precisely, it was nearly a complete academic event that I shall remember for long.

Appendix 1

Normal Blood Pressures in Children by Age

Age	Systolic blood pressure (mm Hg)		Diastolic blood pressure (mm Hg)	
	Female	Male	Female	Male
Neonate (1 day)	60–76	60–74	31–45	30–44
Neonate (4 days)	67–83	68–84	37–53	35–53
Infant (1 month)	73–91	74–94	36–56	37–55
Infant (3 months)	78–100	81–103	44–64	45–65
Infant (6 months)	82–102	87–105	46–66	48–68
Infant (1 year)	86–104	85–103	40–58	37–56
Child (2 years)	88–105	88–106	45–63	42–61

Key to Quiz

1b, 2a, 3d, 4b, 5a, 6c, 7b, 8a, 9a, 10d

Key to the Crossword

ACROSS

2. KID (keratitis deafness ichthyosis)
3. IFAP (ichthyosis follicularis, alopecia, photophobia)
4. AAA (achalasia, Addison's disease, alacrima)
5. SCID (severe combined immunodeficiency)
7. WARTS
9. APECED (autoimmune polyendocrinopathy–candidiasis–ectodermal dystrophy/dysplasia)
11. SLE (systemic lupus erythematosus)
12. ENA (extractable nuclear antigens)
13. HAY
15. IPEX (immunodysregulation polyendocrinopathy enteropathy X-linked syndrome)
17. HAIRY
18. PERIPORITIS
22. OTA
23. MILIA
24. CODON
25. ASPIRIN

DOWN

1. WAARDENBURG
2. KAWASAKI
6. DIRTY
8. SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis)
10. ERYTHRODERMA
14. AEC (ankyloblepharon-ectodermal defects-cleft lip/palate)
16. XEROSIS
19. IFAG (idiopathic facial aseptic granuloma)
20. ITO
21. LARON
23. MED (minimum erythema dose)