Hair loss in infancy and childhood

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Abstract

Hair problems can cause considerable anxiety among both parents and children. This article discusses hair growth and cycling, common presentations of hair loss and offers an approach to diagnosis. This information will equip the reader to manage common types of hair loss in the paediatric setting.

Keywords alopecia; alopecia areata; aplasia cutis; hair loss; hair shaft disorders; telogen effluvium; tinea capitis; triangular alopecia; trichotillomania

Introduction

Hair problems in children are not uncommon and can cause considerable anxiety among parents and children. Conditions such as alopecia areata and trichotillomania can present in both adults and children but in children one also needs to consider rarer congenital and hereditary causes of hair loss which can occasionally present as part of a multisystem syndrome. A practical approach to evaluating hair disorders in children is crucial to ensure the correct diagnosis is made.

Normal hair growth

During embryological development the first hair follicles start to appear at about 9 weeks of gestational age and these are fully established by 22 weeks. The follicle is conventionally divided into two regions: the upper permanent part comprising the infundibulum and isthmus and the lower cycling (growing and then regressing) part consisting of the hair bulb and suprabulbar region (see Figure 1).

Hair follicle stem cells reside in the bulge region of the isthmus. Evidence suggests that the lower part of the hair follicle is an immunologically 'privileged' site not subject to typical immune surveillance. During the hair cycle the growth phase 'anagen' is followed by an involution phase 'catagen' and then a resting phase 'telogen' (see Figure 2). The hair is then eventually shed through an active process called 'exogen'.

Lanugo hair is shed about 1 month before birth and most hairs re-enter the anagen growth phase but in the occipital scalp telogen is delayed until after birth which may give rise to a patch of occipital hair loss in the neonatal period. Hair cycling up to this stage is synchronised but beyond the neonatal period hair follicles cycle independently. Through childhood there is a gradual transition from vellus (soft, short, unmedullated and usually non-pigmented) to intermediate and then terminal hairs (longer, coarser, medullated and pigmented).

Evaluation of a child with hair loss

History

It is important to establish whether hair was normal at birth, when hair loss began and whether this was diffuse loss, patchy loss or failure to grow. Symptoms such as itch or burning are often associated with inflammation (rare) or infection or infestation (both common). Details of teeth and nail development should be ascertained as well as problems with heat and sweating if an ectodermal dysplasia is suspected. Other cutaneous lesions and rashes may be important as well as the general health of the child and achievement of developmental milestones. A family history of hair problems is likely to be relevant in inherited conditions but is also important when considering infective causes such as tinea capitis.

Clinical examination

Clinical evaluation should include an assessment of the pattern and extent of hair loss. If a patchy alopecia is apparent it is important to determine whether there are patent follicular ostia or whether these are lost suggesting a scarring condition. Signs of inflammation such as peri-follicular erythema, follicular hyperkeratosis, pustules or swelling should be sought. Any abnormalities in the skin, nails and teeth should be noted as well as any syndromic features. The hair pull technique can be used to assess hair shedding in generalised hair loss as well as disease activity in focal conditions. The number and type of hairs extracted may give clues to the underlying diagnosis (see Table 1). Anagen hairs have a pigmented bulb enclosed within its root sheath whereas telogen hairs have a de-pigmented club shaped bulb (see Figure 3). A modified hair pull test can be used to assess hair breakage.

Microscopy and scalp biopsies

Light microscopy of hairs trimmed at their bases should be used for the investigation of possible hair shaft disorders. Abnormal hair fibre production can produce unruly hair due to hairs being irregularly shaped, spangled hair where hair twists reflect light at variable angles and fragile hair. Hair fragility can lead to localised or diffuse areas of hair loss occur due to breakage of structurally weak hair. Hair shaft disorders are divided into those with or without increased fragility. Scanning electron microscopy will provide even more detailed images of the hair shaft but expertise and availability limits its use. Scalp biopsies sent for both horizontal and vertical sectioning may give useful clues to the underlying cause but requires at least two biopsies, can be challenging and ideally should be avoided in children if possible.

Hair disorders in infancy

Aplasia cutis congenita (ACC)

This condition is characterised by areas of absent or scarred skin from birth. It reflects disruption of interuterine skin development

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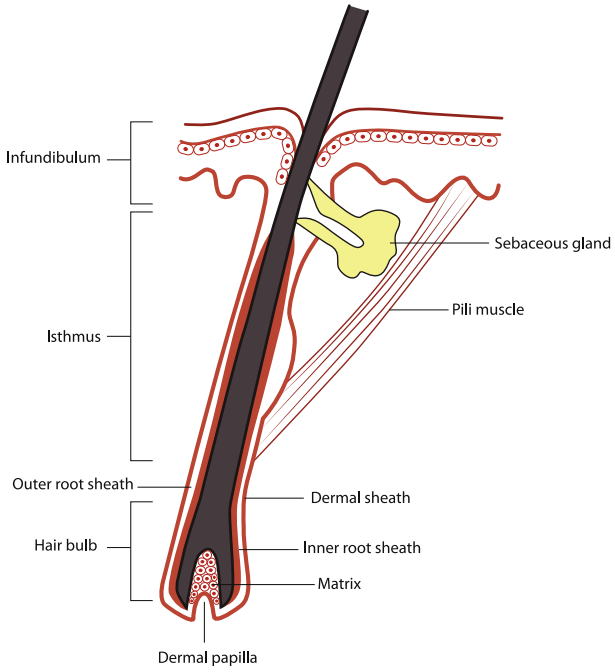

Figure 1 Schematic diagram of basic components of the hair follicle.

and potential causes include vascular compromise, trauma, interuterine infection or teratogenic medication. It may be incorrectly attributed to obstetric trauma such as forceps or fetal scalp electrodes.

A solitary erosion, deep ulceration or scar affecting the scalp is the most common form. These lesions are usually located on the vertex of the scalp lateral to the midline and vary in size from 1 to

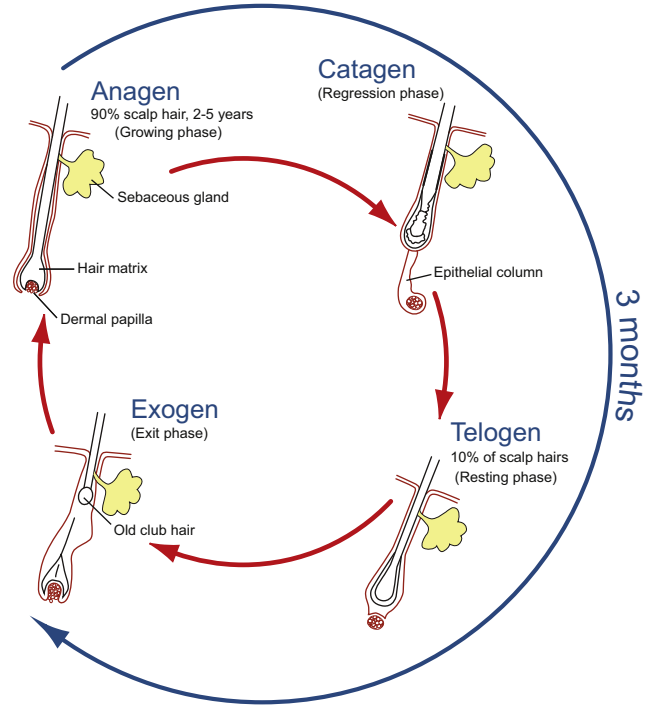

Figure 2 Stages of the hair cycle.

Potential hair pull findings

Condition	Positive hair pull findings
Telogen effluvium	Increased telogen hairs extracted from all areas
Alopecia areata	Increased telogen hairs or dystrophic hairs from affected areas
Primary scarring alopecias	Increased anagen hairs extracted
Loose anagen syndrome	Painless extraction of dysplastic anagen hairs (may lack root sheath and have a hockey stick appearance)

Table 1

10 cm in diameter. Distorted hair growth around the lesion is known as the hair collar sign. The abnormality is usually limited to the epidermis and dermis. Most lesions heal spontaneously within a few months leaving hairless scars which generally become less noticeable as the child grows.

Temporal triangular alopecia

This is a relatively common non-scarring form of alopecia. The majority present around 2–6 years of age with a well-circumscribed triangular or oval patch of alopecia in the fronto-temporal region (see Figure 4). Most are unilateral with the base of the triangle orientated forwards and fine vellus hairs are present in the affected area. Due to the location and typical lancet shape the diagnosis of triangular alopecia is usually easily made clinically, with alopecia areata being the main differential.

Occipital neonatal alopecia

This common form of alopecia develops in the occipital region in the first few months of life (see Figure 5). It occurs due to alterations in hair cycling. Unlike hairs at other sites the occipital hairs don't move into telogen until after birth and therefore shedding in this area commonly occurs 2–3 months later. Friction of the head on the pillow may contribute to the shedding but the alopecia will resolve spontaneously.

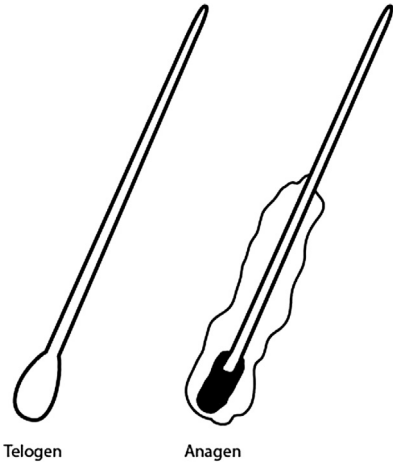

Figure 3 Telogen hair with de-pigmented bulb and anagen hair showing pigmented bulb enclosed within its root sheath.