A 9 year old girl presents to the emergency department with a painful lesion on her scalp. It was initially a small, erythematous, scaly, pruritic eruption but gradually increased in size over the previous six weeks despite several courses of antibiotics from her general practitioner. Physical examination showed a girl in good health, with a 15 cm by 15 cm extremely tender, boggy inflammatory mass on the crown of her head accompanied by localised alopecia (hair loss) and cervical lymphadenopathy (fig 1). She is apyrexial with a white cell count 16.5×10^9/l and C reactive protein 28 mg/l.
Questions

- (1) What is your working clinical diagnosis?
- (2) What organism is most likely to be responsible?
- (3) What investigations would you like to perform?
- (4) What is the treatment?

Answers

(1) This is an example of a fungal kerion, which is an inflammatory reaction to tinea capitis (scalp ringworm). The differential diagnosis of a kerion includes scalp abscess and dissecting cellulitis. The differential diagnosis of non-inflammatory tinea capitis is seborrhoeic dermatitis, atopic dermatitis, impetigo, and pustular or plaque psoriasis.

(2) The predominant pathogen causing tinea capitis in the United Kingdom is *Trichophyton tonsurans*, which is a dermatophyte fungus of the genus *Trichophyton*.

(3) Obtain plucked hairs and scalp brushings from the infected area and send for mycological analysis, and examine the infected area with a Wood’s lamp (table).

(4) Treatment consists of a systemic antifungal, such as griseofulvin, combined with a topical agent, such as ketoconazole shampoo.

Discussion

Fungal infections in humans are seen commonly in primary and secondary care and are mostly caused by two groups of fungi. Dermatophytes consist of multicellular filaments known as hyphae, and yeasts are unicellular and replicate by budding (for example, candidiasis). Dermatophytes require keratin for growth and therefore cause superficial infections of the skin, hair, and nails. They are transmitted by direct contact with other humans (anthropophilic), animals (zoophilic), or soil (geophilic), as well as by indirect contact with fomites (that is, inanimate objects capable of carrying infection such as combs, hats, and towels). The various types of dermatophytoses (fungal infections) are referred to as “tineas” or ringworm. Clinically they are classified by anatomical site or structure affected.[1]

Tinea capitis is the most common dermatophytosis in children but is relatively uncommon in adults. The exact incidence remains unknown but is higher in developing countries and inner city areas and tends to be associated with overcrowding and low socioeconomic status.[2] It was a common childhood disease in the UK in the 1950s and ‘60s, but incidence fell after the introduction of oral griseofulvin and school surveillance programmes. However, in the past 20 years the incidence has increased, particularly among African-Caribbean children in inner city areas.[3] [4] [5] [6] The reason is unclear, but hairdressing practices such as shaving the scalp
(clippers and razors transfer viable dermatophytes) and plaiting (causing traction of the hair shaft allowing invasion of dermatophytes) may increase transmission.[3]

Infection is initiated when spores from another person, animal, or fomite come into contact with the scalp of the patient and implant in the stratum corneum (the outermost layer of skin), facilitated by minor scalp or hair trauma. Immune response to fungal invasion varies among individuals and determines the clinical picture and chronicity of infection. Primary infection causes mild erythema and scale, resulting from increased keratinocyte turnover. The formation of a kerion represents the most exaggerated host response to dermatophyte. The exact mechanism of the immune response is unclear, but type IV (delayed) hypersensitivity, which is cell mediated plays a pivotal role in combating dermatophytes.[7]

Clinical diagnosis of tinea capitis can be challenging because presentation varies from a non-inflammatory lesion with minimal pruritis and slight hair loss, to a fiercely inflammatory kerion (box).[2] An estimated 50% of patients are prescribed inappropriate treatment in the community or refer themselves to hospital.[2] [3] Kerion can be confused with scalp abscess, and it is important to differentiate between the two. Although incision and drainage is an appropriate treatment for scalp abscess, it is unnecessary and ineffective for kerion. Scalp abscesses are usually associated with severe constitutional upset and are rare, unless there is immunodeficiency or penetrating trauma.[8] If in doubt, consult a dermatologist. The clinical diagnosis should be confirmed by either microscopy, culturing samples of the affected area or using a Wood’s lamp (table).[2] [7]

<table>
<thead>
<tr>
<th>Test</th>
<th>Method</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microscopy</td>
<td>Place sample of hair, or skin scraping on a glass slide and add a few drops of 10-20% KOH. Examine under microscope for presence of branching rod shaped filaments (hyphae)</td>
<td>Quick and inexpensive</td>
<td>Sensitivity depends on operator so cases may be missed (false negative)</td>
</tr>
<tr>
<td>Wood’s lamp</td>
<td>Filtered ultraviolet light applied directly to the affected area. Infection due to Microsporum canis, Microsporum audouinii will produce characteristic bright green fluorescence. Trichophyton tonsurans (most common) does not fluoresce</td>
<td>Quick and non-invasive</td>
<td>Most dermatophytes do not fluoresce</td>
</tr>
<tr>
<td>Fungal culture</td>
<td>Obtain samples by scraping affected area with scalpel or with moistened cotton swab or toothbrush and inoculate onto mycological media. The aim is that the dermatophyte will grow on media</td>
<td>Most sensitive method, determines antifungal susceptibilities</td>
<td>Takes 7-14 days to be declared positive and 21 days to be declared negative</td>
</tr>
</tbody>
</table>
Six main clinical patterns of tinea capitis (from most to least common)

- Grey type circular patches of alopecia, marked scaling
- Moth eaten patchy alopecia, generalised scale
- Kerion boggy tumour with pustules and lymphadenopathy
- Black dot patches of alopecia, dotted with broken hair stubs
- Diffuse scale widespread scaling, dandruff-like
- Pustular alopecia, scattered pustules, lymphadenopathy

The goals of treatment are to provide a quick cure with minimum adverse affects, while controlling the spread of infection. Most superficial infections can be treated topically, but tinea capitis requires systemic treatment (to ensure penetration to the root of the hair follicles) and topical treatment (to reduce the risk of transmission). Our patient was treated with oral griseofulvin and ketoconazole shampoo for two months and made an excellent recovery.

Griseofulvin is currently the only licensed treatment for tinea capitis in the UK. It is fungistatic (stops growth of fungus), arresting cell division and impairing fungal cell wall synthesis. Newer agents are increasingly being considered but a recently published Cochrane systematic review recommends that griseofulvin remains the drug of choice, until randomised, comparative studies have clarified the role of newer agents.[9]

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References


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