Infantile haemangioma	
ICD 10 code	D18
Synonyms	Strawberry birth mark, strawberry naevus, strawberry haemangioma, capillary naevus/hemangioma, haemangioma simplex, proliferative haemangioma. Deeper lesions can be called cavernous haemangiomas.
Differentials	Vascular malformation.
What is it?	Infantile haemangioma is a proliferative vascular lesion that usually develops shortly after birth, and grows rapidly before undergoing variable degrees of spontaneous involution. It is distinct from vascular malformation, which is more stable.
Frequency	10% of infants.
Description	Up to 80% of infantile haemangiomas occur on the head and neck area. They can be classified as: ▸ Superficial: dilated blood vessels in the uppermost layers of the dermis. Infantile haemangiomas may look like a strawberry. ▸ Deep: dilated blood vessels are deeper in the dermis and subcutis. They appear as a bluish, soft to firm swelling. ▸ Mixed: a 'strawberry' naevus overlies a bluish swelling. ▸ Segmental: more serious than localised haemangioma. They may be associated with other congenital anomalies such as PHACE (posterior fossa, haemangioma, arterial, cardiac and eye abnormalities) or PELVIS syndromes (perineal haemangioma, external genital malformation, lipomyelomeningocoele, vesicorenal abnormalities, imperforate anus, and skin tag).
Clues to diagnosis	▸ Appears days to weeks after birth. ▸ May appear like a strawberry or bluish swelling.
Aetiology	Infantile haemangiomas are due to proliferating endothelial cells. Hypoxia is now considered the likely cause. Endothelial progenitor cells circulate in a fetus and cause new blood vessels to form in response to hypoxia. Normally, endothelial progenitor cells have gone by birth, but may still be present in low birth-weight or premature babies. As these endothelial progenitor cells disappear, the haemangiomas regress. Infantile haemangiomas express glucose transporter 1 (GLUT 1) protein. Segmental infantile haemangiomas are thought to arise early in gestation (6–8 weeks), as a developmental error.
Aggravating factors	Risk factors for developing infantile haemangiomas include low birth-weight, particularly if female, caucasian race, prematurity, multiple births, family history of infantile haemangioma, and in association with advanced maternal age.
Investigations	Usually none needed.
Prognosis	Infantile haemangiomas generally grow to 80% of maximum size in the first 3–4 months and most stop growing by 5 months. However, some may keep growing for up to 18 months. Regression or involution may take 3–10 years. Most superficial infantile haemangiomas involute and disappear without treatment. However, regression of deep haemangiomas tends to be incomplete, and they may leave an irregular atrophic scar or anetoderma in at least 50% of cases.

Infantile haemangioma

Treatment

As most superficial infantile haemangiomas improve or regress completely with time, treatment is often not needed. However, if treatment is indicated, this should preferably be started before 3–6 months of age. Treatment should be considered in the following circumstances:

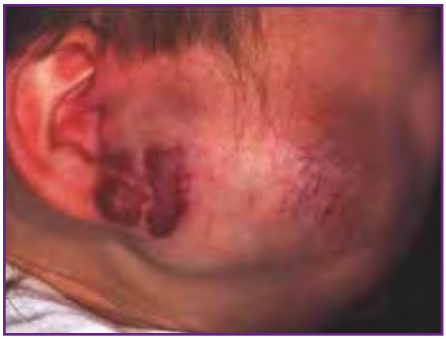
- Very large and unsightly lesions.
- Ulcerating haemangiomas.
- Lesions that impair vision, hearing, breathing, defaecation, urination or feeding.
- Nasal tip lesions.
- Lesions overlying the trachea.
- If haemangioma has failed to resolve by school age.

Treatments include:

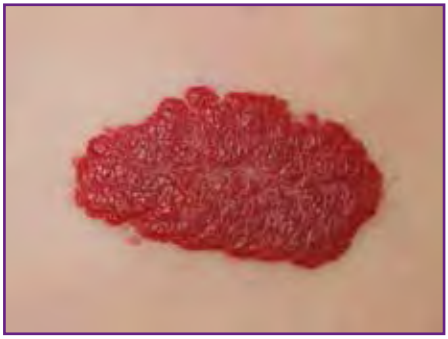
- Oral β-adrenergic blocking agents (e.g. propranolol) (treatment of choice).
- Topical β-adrenergic blocking agents (e.g. timolol) for small superficial haemangiomas.

Other treatments include:

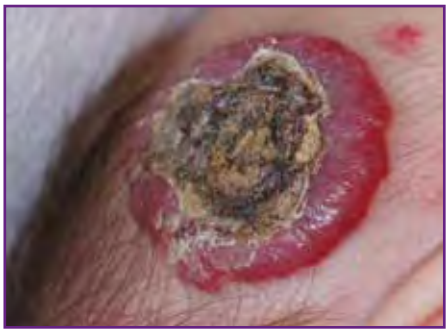
- Ultrapotent topical corticosteroid ointments.
- Intralesional corticosteroids.
- Oral corticosteroids (2 mg/kg/day).
- Pulsed dye laser.
- Imiquimod cream.



Mixed segmental haemangioma



Superficial infantile haemangioma



Ulcerated haemangioma

Pharmaceutical name	Dapsone
Class	Dapsone is a sulfone drug, belonging to the sulfonamide family.
Indications	Treatment of dermatologic diseases involving neutrophilic infiltrates. Dermatitis herpetiformis, linear IgA disease, bullous systemic lupus erythematosus, erythema elevatum diutinum, pemphigus foliaceus, bullous pemphigoid, neutrophilic dermatoses (e.g. Sweet syndrome, pyoderma gangrenosum) and various vasculitic disorders. It is also a key component of combination therapy for leprosy. Topical dapsone is used to treat acne vulgaris.
Mode of action	Dapsone inhibits bacterial synthesis of dihydrofolic acid, via competition with para-aminobenzoate for the active site of dihydropteroate synthetase. It also has anti-inflammatory properties: dapsone inhibits neutrophil myeloperoxidase and neutrophil chemotaxis, thus reducing damage from neutrophils. It also inhibits eosinophil myeloperoxidase activity.
Routes	Topical, oral.
Precautions	Absolute: hypersensitivity to dapsone. Relative: low glucose-6-phosphate dehydrogenase (G6PD) level, significant cardiopulmonary disease, sulfonamide allergy.
Monitoring	Pre-treatment: complete blood count (CBC), liver function tests (LFT); consider G6PD screen. Consider ongoing monitoring: ▸ CBC with platelet count every week for 1 month, then every 2 weeks for 2 months, then every 3–4 months. ▸ Reticulocyte count and methaemoglobulinaemia if anaemia develops. LFT after one week, one month, then every 3–4 months. Urinalysis every 3–4 months. Each visit should include an assessment of peripheral motor function and an assessment for the signs and symptoms of methaemoglobinaemia and peripheral neuropathy.
Adverse effects	Common: dose-dependent methaemoglobinaemia and haemolytic anaemia. Rare: agranulocytosis, peripheral neuropathy and dapsone hypersensitivity (e.g. toxic epidermal necrolysis or drug hypersensitivity syndrome (DHS/DRESS)).

Pharmaceutical name	Dapsone
Drug interactions	Increase dapsone levels: probenecid, trimethoprim and folate antagonists. Reduce dapsone levels: activated charcoal, para-aminobenzoic acid (PABA) and rifampicin. Notes: › Cimetidine increases absolute levels of dapsone, but reduces methaemoglobinaemia. › Sulfonamides and hydroxychloroquine may worsen haemolysis.

Pharmaceutical name	Erythromycin
Class	A macrolide antibiotic derived from a strain of the actinomycete *Saccharopolyspora erythraea*.
Indications	Erythromycin is active against many gram-positive organisms (including *Staphylococcus aureus*, *Streptococcus pyogenes*, corynebacteria and clostridia) and some Gram-negative organisms (*Neisseria gonorrhoeae*). It is also effective for mycoplasma infections, syphilis and chlamydia. Erythromycin is particularly useful in individuals that are allergic to penicillin. Topical: acne vulgaris. Off-label: has been used for a variety of inflammatory dermatoses such as acne, rosacea and pityriasis lichenoides.
Mode of action	Erythromycin displays bacteriostatic activity.
Routes	Oral, intravenous, topical.
Precautions	Severe hepatic impairment; drug interactions (e.g. terfenadine, astemizole, pimozide, cisapride, ergotamine, dihydroergotamine).
Monitoring	Consider liver function tests if prolonged course (>12 weeks) or high dose.
Adverse effects	Common: diarrhoea, nausea, abdominal pain, vomiting, superinfection (including candidiasis). More serious: arrhythmia (prolonged QTc interval), reversible deafness, allergic reactions (urticaria, anaphylaxis). Rare: cholestasis, Stevens-Johnson syndrome/toxic epidermal necrolysis, pyloric stenosis (young infants).
Drug interactions	Erythromycin is an inhibitor of the cytochrome P450 system (CYP3A). Terfenadine, astemizole, pimozide, cisapride, ergotamine, dihydroergotamine. It may alter the effectiveness of combined oral contraceptive pills.