for management of AIS among pediatric patients (younger than 18 years of age) are based on data extrapolated from adult data. The use of intravenous thrombolytic agents such as tPA is considered experimental given the lack of clinical trials and prospective studies of its use in the pediatric population. However, results of the Thrombolysis in Pediatric Stroke (TIPS) trial,4 a 5-year prospective trial examining the use of intravenous tPA in children aged 2 to 17 years, will be crucial for creating evidence-based recommendations for treating pediatric patients with AIS.

REFERENCES


Benign Primary Milia

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A 17-year-old presented with multiple white papules on her face that had been first noted approximately 4 years ago. These asymptomatic papules had appeared gradually over 18 months’ time. Her medical history was otherwise unremarkable. In particular, there was no history of trauma, and she was not on any topical or oral medication. A diagnosis of benign primary milia of children was made.

Milia are small (generally less than 3 mm), white, benign, dome-shaped, superficial keratinous cysts. Histologically, they appear as small infundibular cysts that are lined with stratified squamous epithelium with a granular cell layer.1 The cyst contains laminated layers of keratin. Milia may arise spontaneously without known cause (primary milia) or secondary to various processes (secondary milia). Primary milia may be congenital or have onset later in life.

Congenital primary milia are present in approximately 40% of newborn infants, with no sex predilection. The condition is less common and is delayed in onset in premature infants.1 While congenital primary milia favor the nose, benign primary milia of children and adults favor the eyelids. Other unusual sites of involvement include the areola, nasal crease, glans penis, and vulva.1 The presence of primary milia may be a feature of Rombo syndrome, Brooke-Spiegler syndrome, Bazex-Dupré-Christol syndrome, Basan syndrome, orofaciodigital syndrome type 1, basal cell nevus syndrome, and Nicolau-Balus syndrome.1
The differential diagnosis of primary milia includes sebaceous hyperplasia, syringomas, molluscum contagiosum, lichen nitidus, closed comedones, cutaneous myxomas, and calcinosis cutis.¹

Milia en plaque and multiple eruptive milia are considered variants of milia.²³ Milia en plaque is characterized by a plaque formed by a confluence of closely grouped, tiny, skin-colored to white papules, often with an erythematous base.³ The condition is most common in middle-aged individuals, with a female predominance and no predilection for race.³ The lesions usually are asymptomatic. The postauricular area is most commonly affected. Other sites of involvement include the pericircular area, supraclavicular area, submandibular area, cheeks, nasal bridge, trunk, and lower limbs.³ Multiple eruptive milia are characterized by a sudden onset of multiple milia over a period of weeks to months, mainly on the head, neck, and trunk.² The lesions usually are asymptomatic.²

Secondary milia may occur in association with disease (eg, epidermolysis bullosa, porphyria cutanea tarda), medication (eg, oral cyclosporine, penicillamine, 5-fluorouracil, topical corticosteroids), or trauma (dermabrasion, chemical peels, burns, skin grafts, tattoos).⁴

Primary congenital milia often exfoliate and resolve spontaneously in the first few weeks to months of life, thus requiring no treatment. Benign primary milia of children and adults tend to persist longer. The lesions of milia en plaque and multiple eruptive milia usually persist without treatment.³ For secondary milia, the underlying cause has to be removed if possible. For persistent lesions, treatment options include simple mechanical evacuation or extraction, topical retinoids, electrocautery, electrodesiccation, cryosurgery, or carbon dioxide laser vaporization.³

REFERENCES