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A Picture Says a Thousand Words
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A Picture Says a Thousand Words

Multiple telangiectasias in a child

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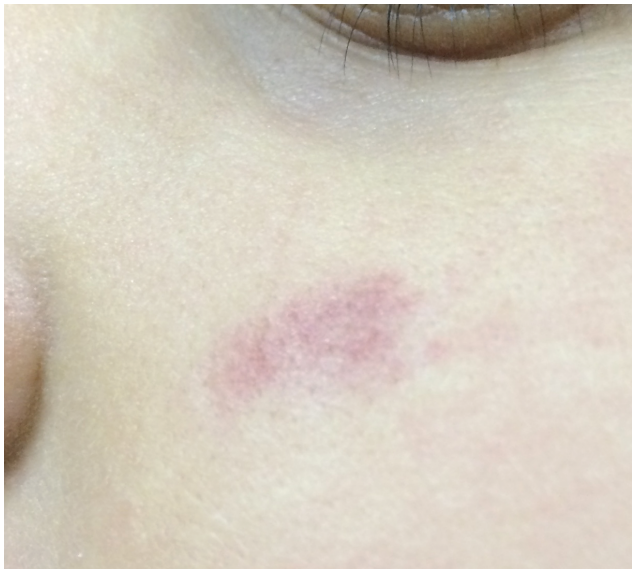
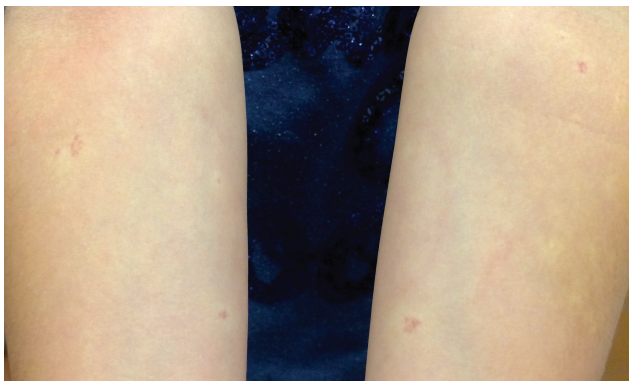
A healthy 8-year-old girl presented with multiple punctate and arborizing telangiectasias, which were surrounded by anemic halos, developed progressively from 2 months of age. They favoured sun-exposed areas of the face, neck, upper, and lower extremities. There was no associated mucosal involvement, clinical evidence of bleeding diathesis, or systemic involvement. There was no family history of similar lesions. Dermoscopy showed a linear branched vascular pattern that disappeared with compression. No homogeneous brownish background was observed on dermoscopy. What is the diagnosis?





ANSWER: HEREDITARY BENIGN TELANGIECTASIA (HBT)

HBT is an uncommon, genetically inherited, benign skin disorder with onset in childhood (1).

Clinically, HBT is characterized by the presence of telangiectasias with different morphological features, such as punctate, linear, or arborizing. They are surrounded by anemic halos on sun-exposed areas of the face, neck, upper trunk, and upper extremities. With the exception of the vermilion lips and palate, mucous membranes are not involved and there is no visceral involvement (2,3).

The main differential diagnoses include capillary malformations–arteriovenous malformation (CM-AVM) syndrome, an autosomal dominant disease due to a mutation in the RASA1 gene. This condition is characterized by capillary malformations often present at birth, telangiectasias with a background of hyperpigmentation and surrounding pale halo scattered throughout the body, as well as fast-flow AVMs and AV fistulas that can be presented in approximately 35% of patients (3–5). Due to the similar morphology and distribution of lesions between CM-AVM syndrome and HBT we cannot completely exclude the former. Still, in this case, telangiectasias favoured sun-exposed areas. This distribution of cutaneous lesions is often found in HBT (1,3). On dermoscopy, we did not observe thumbprint-like capillary malformations, which is a characteristic skin finding in CM-AVM syndrome (5). Also, we did not observe a homogeneous brown background in telangiectasias, which is a dermoscopic characteristic of cutaneous CM in CM-AVM syndrome that may result from involvement of mast cells (4). Moreover, we excluded the presence of intracranial AVMs by brain MRI and there is no evidence of other AVMs. These findings make CM-AVM syndrome less likely than HBT. Another diagnoses ruled out includes hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease), which presents with mucosal and visceral involvement associated with repeated episodes of epistaxis in children, before the appearance of cutaneous telangiectasias during adolescence or even later in life. Additionally, essential generalized telangiectasia was also ruled out since it typically affects adult women with blanchable telangiectasias of the distal lower extremities, that progresses upward toward trunk and upper extremities (1,5,6).

We considered this case a nonhereditary form of HBT given the absence of family history of HBT (2).

HBT has a good prognosis (1). If the lesions constitute a cosmetic issue, electrocautery, laser treatments, and intense pulsed light are possible treatment approaches (7).

Since it is not possible to completely exclude the diagnosis of CM-AVM syndrome, we keep this child in a close medical follow-up. This question may be clarified if genetic testing for RASA1 gene becomes available in the future.

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