The most common cause of chorea in children is ARF, an autoimmune sequelae of Group A beta-hemolytic *Streptococcus* (GABHS) pharyngitis.[2] The diagnosis of ARF is based on the revised Jones Criteria.[3]

Sydenham chorea is the only major Jones criteria that can define a case of ARF on its own.[4] The pathophysiology of Sydenham chorea is only partially understood but is generally believed to be secondary to the binding of anti-N-acetyl-β-D-glucosamine antibodies with lysoganglioside and tubulin that are present in neurons of the basal ganglia.[5] Clinically, Sydenham chorea is characterized by neurologic motor symptoms with jerking movements of the arms and legs, as well as truncal restlessness. The chorea is most commonly bilateral; however, in 20%-30% of patients it can be unilateral. A classic feature is motor impersistence, which is the inability to sustain voluntary actions. This is most clearly evidenced by the darting tongue and milkmaid sign shown during our patient's examination. Dysarthria is also common, as well as emotional lability.[6] Sydenham chorea presents typically within 4-8 weeks after initial GABHS infection, but can appear up to 8 months later,[7] which makes the diagnosis more challenging as inflammatory markers may be normal and it may not be possible to document laboratory evidence of preceding GABHS infection .

The treatment of Sydenham chorea (and ARF) is supportive. Some studies have shown a hastened recovery of Sydenham chorea with the use of systemic steroids.[8] Recurrence of ARF and rheumatic heart disease can be prevented with the use of prophylactic penicillin.[9] ■

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Tense Bullae on the Hands

A 5-year-old girl presented to the dermatology clinic for evaluation of pruritic papules and blisters on the hands and forearms for three months. She was seen by her pediatrician, diagnosed with dyshidrotic eczema, and prescribed topical triamcinolone with no improvement. She reported itching everywhere, but her parents noted she most frequently scratched her hands and forearms. The itching was worse at night. No close contacts experienced pruritus. She had no history of inflammatory dermatoses or other medical problems.

Physical examination revealed tense erythematous bullae with discrete dermal-appearing papules and small nodules along the ventral aspect of the fingers, and excoriated erythematous papules on the flexor wrists (**Figure**). There was no other mucocutaneous involvement. The child was well-appearing with normal vital signs. Mineral oil preparation of scraping from the wrist papules revealed scabies mites. The child was diagnosed with bullous scabies and prescribed permethrin, with resolution of symptoms.

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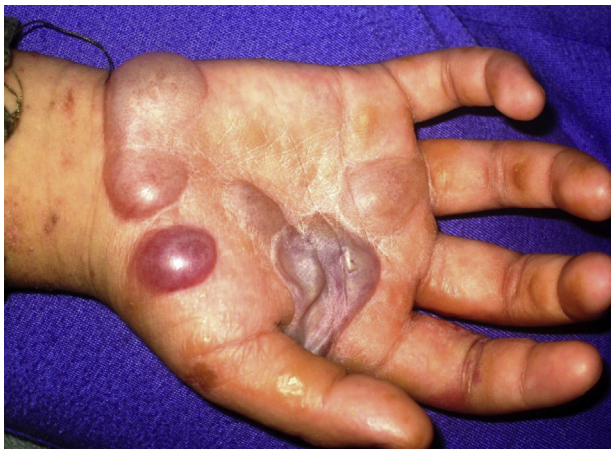

Figure. Tense bullae and papulonodules on the palm and ventral digits and erythematous excoriations on the wrist.

Scabies is a common skin infestation caused by the mite *Sarcoptes scabiei* var. *hominis* that more frequently affects the pediatric population.[1] The mite is transmitted from one person to another through direct contact. Diagnosis is confirmed by identification of mites, eggs, or fecal material of mites under microscopy. Bullous scabies, a less common presentation of scabies, has been reported in children.[2-4] One study found that 6% of children age <15 years with scabies presented with blisters.[4] Classically, scabies presents with discrete erythematous papules and nodules, excoriations, and burrows, with predilection for interdigital spaces, umbilicus, wrists, hands, axillae, and genitalia. Dermoscopy may reveal a "delta sign," which appears as a triangle corresponding to the head of the mite.[5] Bullous scabies can include all of these typical findings in addition to tense bullae, filled with clear or hemorrhagic fluid. The mechanism of bullae formation remains unknown but is thought to be direct injury by mites or immune response.[3]

The differential diagnoses for bullous scabies in children may include arthropod bite reaction, bullous impetigo, a scalding burn or chemical irritant, dyshidrotic eczema, contact dermatitis, and epidermolysis bullosa acquisita. Although much less likely, the hemorrhagic bullae on this child's hand could prompt consideration of vibrio vulnificus infection in the appropriate clinical setting (acute onset, ill-appearing, exposure to raw seafood). The child did not have a history of arthropod bite or exposure, or a history of trauma or irritant exposure. Improvement with topical steroids would be expected for a diagnosis of dyshidrotic eczema or contact dermatitis. However, the most pertinent clue to the diagnosis on physical examination in this case was the erythematous excoriated papules on her flexure wrists.

Treatment of bullous scabies is the same as that of classic scabies and usually consists of topical permethrin, sulfur, or oral ivermectin. Close contacts should be treated, even if not symptomatic, and clothing and bedding should be thoroughly washed in hot water. ■

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Axillary Calcification Due to Bacillus Calmette–Guérin Vaccination

A 10-month-old, fully vaccinated, infant boy was admitted to our hospital in rural Laos with respiratory distress that ultimately was diagnosed as due to beriberi.[1] At the time of admission, a firm mass was noted in the right axilla (**Figure 1**) during routine physical examination. His parents reported that the mass was first palpable at about 4 months of age, had progressively increased in size until about a month before admission, and had since stabilized. A chest radiograph demonstrated a calcified right axillary mass with the typical appearance of lymph nodes (**Figure 2**, arrow). A re-review of a chest radiograph obtained during a previous hospital visit at 2 months of age confirmed that this calcification was not visible at that time (**Figure 3**).

Axillary lymph node calcification is an occasional consequence of the intradermal Bacillus Calmette–Guérin vaccination of infants shortly after birth and occurs due to replication of the attenuated *Mycobacterium bovis* bacilli and clearance by regional lymph nodes. The calcification may expand over the first year of life and may subsequently regress or may remain stable in size without clinical consequence. No specific treatment or resection is necessary unless the calcified nodes become painful or otherwise cause distress. This has been

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