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Atopic Dermatitis With Group A β -Hemolytic *Streptococcus* Skin Infection Complicated by Posterior Reversible Encephalopathy Syndrome

Report of a Case. An 11-year-old boy was hospitalized for atopic dermatitis with secondary bacterial infection. He showed multiple erythematous crusted patches on both legs for 2 weeks (**Figure 1**). On initial admission, the patient denied having headaches or a sore throat; he had no fever. His antistreptolysin-O titer was normal (127 IU/mL; normal range, 0-243 IU/mL), and group A β -hemolytic *Streptococcus* (GABS) was identified through cultures of the cutaneous lesions. Although the skin lesions improved under treatment with intravenous cefazolin sodium, he complained of a headache and was subsequently found to have high blood pressure (150/90 mm Hg) from hospital day 3. After the blood pressure was controlled with sublingual nifedipine, he was discharged on hospital day 5.

On the day of discharge, he returned to the ER after a generalized tonic-clonic seizure. He also had visual disturbance and vomiting. Initial blood pressure was 150/90 mm Hg, and body temperature was normal. The antistreptolysin-O titer was rechecked and found to be within normal limits. Serum urea nitrogen and creatine levels were normal, and C3 and C4 levels were low. On urinalysis, mi-

croscopic red blood cells were noted with gross hematuria. A brain computed tomographic (CT) scan (**Figure 2**) demonstrated cortical and subcortical attenuation in the occipital lobes. Electroencephalography (EEG) findings suggested a diffuse encephalopathy with a severe focal encephalopathy.

He was diagnosed as having acute poststreptococcal glomerulonephritis (APSGN) complicated by posterior reversible encephalopathy syndrome (PRES). His blood pressure was controlled under treatment with amlodipine besylate, hydralazine, and enalapril maleate; his blurred vision and/or neurologic symptoms improved under treatment with sodium valproate. One week later, his EEG trace was normal. At 1 month follow-up, the brain CT was normal, and his general condition was good without neurologic complications.

Comment. Posterior reversible encephalopathy syndrome, a term first used by Hinchey et al,¹ is a reversible neurologic disorder that manifests as a headache, visual disturbances, focal neurologic deficits, and seizures, usually in the setting of hypertension.¹ It can occur in diverse clinical settings, including with eclampsia, cyclosporine toxic effects, autoimmune diseases, and infections. Acute poststreptococcal glomerulonephritis is characterized by gross hematuria, edema, and hypertension. Cerebral vasculitis, hypertensive encephalopathy, and hyperperfusion have been suggested as possible mechanisms of



Figure 1. Multiple erythematous crusted patches on popliteal fossae bilaterally.

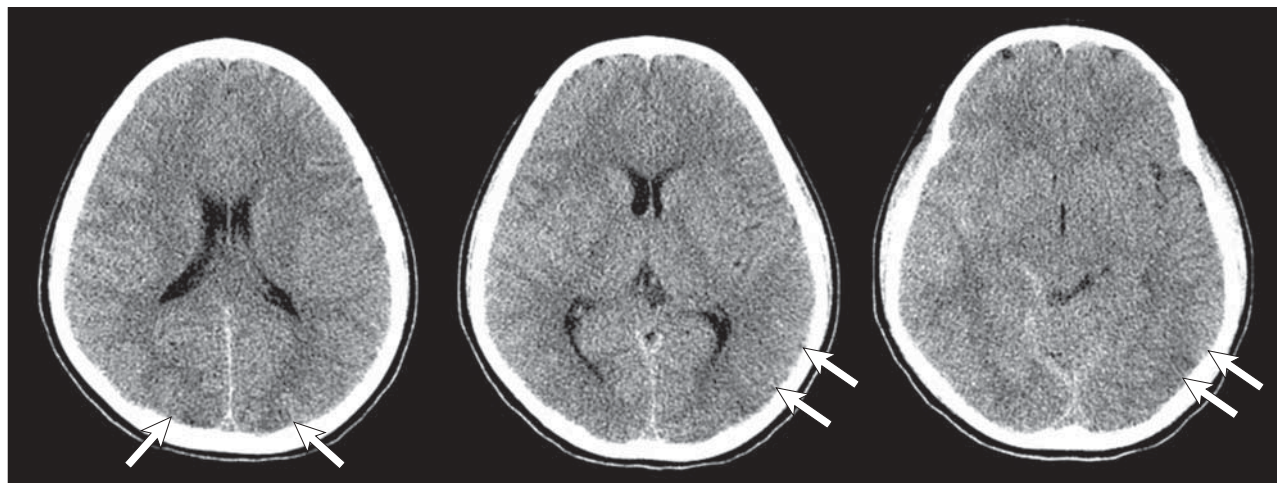


Figure 2. Axial computed tomographic scans of the brain demonstrate low density in the cortex and subcortical white matter in the parieto-occipital lobes bilaterally (arrows).

the neurologic complications associated with APSGN.² Acute poststreptococcal glomerulonephritis caused by GABS infection is an important cause of PRES in children and typically appears 1 to 3 weeks after pharyngitis and 3 to 6 weeks after impetigo.³ In contrast to M types 1 and 12 in APSGN following pharyngitis, M types 2, 49, 55, 57, and 60 were recognized in APSGN following pyoderma.⁴

Although GABS was identified in our patient through the culture of skin lesions, we did not check the strain of *Streptococcus*. However, because our patient had a normal antistreptolysin-O titer without tonsillitis, APSGN following skin infection was suspected. Cases of APSGN complicated by PRES usually occur after respiratory tract infection. To our knowledge, only 2 cases following impetigo without underlying atopic dermatitis have been reported.^{2,5}

Physicians could easily overlook high blood pressure and agitation in a young patient, and this would lead to a delay in the diagnosis of PRES. In conclusion, if a child with atopic dermatitis has cutaneous GABS infection, physicians should check the blood pressure and neurologic symptoms and perform laboratory tests such as a urinalysis and C3 level assay.

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Multifocal Tufted Angiomas in 2 Infants

Tufted angiomas (TAs) are uncommon, benign vascular tumors that show tufts of capillaries or “cannonballs” in the dermis on histologic analysis.¹ These tumors most commonly manifest in early childhood, with rare reports of congenital or adult-onset cases. Clinically, TAs typically present as solitary, dusky red to violaceous plaques or nodules, most commonly occurring on the trunk and upper extremities.¹ The natural history of TA is generally of slow enlargement, but spontaneous regression has been reported in lesions of congenital or infantile onset. To our knowledge, we report herein the first 2 cases of multiple TAs in young infants.

Report of Cases. *Case 1.* A 1-month-old Hispanic girl presented for evaluation of multiple vascular skin lesions. Two large lesions were present on her back at birth, and she subsequently developed multiple smaller lesions. Liver ultrasonography and complete blood cell count findings at birth were normal. Skin examination showed a firm, 4-cm violaceous nodule on her right middle back overlying a large, dark red plaque (**Figure 1**). On her left middle back was a dark red patch. In addition, on her trunk and extremities were multiple pinpoint to 3-mm red papules. Analysis of skin biopsy specimens from both the congenital nodule and a smaller acquired lesion showed characteristics consistent with TA. At her 6-month follow-up, significant regression was seen.

Case 2. A 7-month-old boy was seen for evaluation of vascular lesions first noted at age 2 months. Skin examination showed erythematous to brown papules and plaques on the right side of his face (**Figure 2**), with noncontiguous papules on the right neck, chest, right upper back, and right earlobe. Evaluation of a biopsy specimen from his right upper back showed characteristics consistent with TA. Results of a complete blood cell count and assays for prothrombin and partial thromboplastin times were normal. At age 10 months, his lesions had substantially regressed.