recognizing that the absence of a bite or scratch does not preclude RBF. Moreover, clinicians with a high degree of suspicion for RBF should immediately notify the laboratory to prepare for the unique collection and culture requirements of *S moniliformis* and, while awaiting the PCR or blood culture results, consider empirical antibiotic treatment.5,6 With increased awareness and understanding of RBF, timely diagnosis and treatment are likely to improve patient outcomes.

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**Anagen Effluvium Caused by Thallium Poisoning**

Anagen effluvium is the abrupt loss of hair during the growth phase due to an insult that impairs mitotic or metabolic activity of the hair follicle. This form of hair loss is essentially synonymous with chemotherapy-induced alopecia. Less common causes include medications, radiation, toxic chemicals, and inflammatory disease. Thallium, once considered the “poisoner’s poison,” is an odorless, flavorless, colorless heavy metal and a rare cause of anagen effluvium.

**Report of a Case** | A 25-year-old man with history of depression had repeatedly presented to urgent care with flu-like symptoms. Five days after one of these visits, he returned with hypertension and tachycardia. Within 3 days he had difficulty moving from his bed and was admitted with weight loss, peripheral neuropathy, and continued fatigue. Initial workup, including magnetic resonance imaging of the brain, spinal radiography, lumbar puncture, and testing for heavy metals (including lead, mercury, cadmium, and arsenic), revealed nothing abnormal. Additional testing was performed for pheochromocytoma, Guillain-Barré syndrome, lupus, streptococcal infection, human immunodeficiency virus, legionella, and thyroid disease, and results were negative. During admission, he developed diffuse hair loss and dermatology was consulted.

On physical examination, diffuse alopecia with preserved follicular ostia was noted (Figure 1), and a positive
finding on pull test with dermoscopic examination confirmed anagen hairs. His medications at that time were paroxetine, vitamin D, quetiapine, and docosanol cream, gabapentin, metoprolol, pantoprazole, and enoxaparin. He denied herbal or dietary supplement use. Workup for anagen effluvium included repeat heavy metal testing for arsenic, mercury, and lead, results of which were all negative; iron studies, which showed low iron and iron saturation but normal ferritin levels; and a tick-borne disease panel, results of which were positive for *Rickettsia typhi* group IgG. A scalp biopsy showed a nonscarring, noninflammatory alopecia (Figure 2).

Over the course of his month-long hospitalization, his peripheral neuropathy worsened, and decreased vision was noted in the left eye. Further questioning revealed that the patient was a graduate chemistry student. His advisor stated that he worked with copper, and the patient indicated that he was also helping with studies on thallium and thorium. Copper and ceruloplasmin levels were normal, but blood and urine thallium levels were greater than 100 μg/L and greater than 25 μg/L (upper limits of normal, 2 and 5 μg/L), respectively. He was immediately treated with hemodialysis and Prussian blue. Ophthalmologic consultation confirmed optic neuropathy from thallium. Psychiatric consultation for chronic depression was refused by the patient. The patient did not want a criminal investigation into the matter, and the primary medical team deferred investigation.

The patient was discharged to acute rehabilitation with a blood thallium level of 25 μg/L.

Discussion | Thallium was used in the past to treat gonorrhea, syphilis, tuberculosis, ringworm, and even as a depilatory to remove excess hair. It was commonly used as a rodenticide, but household use was banned in 1965 owing to multiple reported poisonings. Today it is used in a number of industrial products from semiconductors to thermometers. Thallium is absorbed through the skin and the respiratory and gastrointestinal tracts. 

In acute thallium poisoning, alopecia typically occurs within 2 to 3 weeks of exposure, with symptoms of gastrointestinal upset, painful polyneuropathy, visual effects, tachycardia, hypertension, fatigue, and/or encephalopathy. Diffuse loss of scalp hair and lateral aspects of eyebrows has been reported, with sparing of the eyelashes, axillary, and pubic hair. Diffuse skin pigmentation, Mees lines, perioral dermatitis, stomatitis, scaling of palms and soles, and acneiform, pustular, or nonspecific erythematous eruption may also be seen. Thallium poisoning is treated with Prussian blue, which binds thallium, promoting gastrointestinal excretion, and hemodialysis until plasma and urinary thallium levels are near normal. Dermatologic symptoms typically subside without sequelae, but painful polyneuropathy and ocular disease persist.

We present this case to remind clinicians that anagen effluvium can be associated with toxic chemical exposures.

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**Treatment of Skin Ulcers Secondary to Sneddon Syndrome With Alprostadil (Prostaglandin E1)**

Sneddon syndrome (SS) is a rare noninflammatory systemic vascular disease that clinically presents with cerebrovascular disease and racemous livedo.1 This condition is characterized by obstructive vasulopathy of cutaneous microcirculation, which can lead to the development of painful skin ulcers.

**Report of a Case** | A woman in her 50s with a history of SS of 20 years' evolution came to the dermatology department with ulceration on both legs. The patient had a history of lacunar infarction and was taking acetylsalicylic acid for prevention of cerebral ischemic events. Skin ulcers were previously treated with systemic corticosteroids and clopidogrel with no response. Physical examination revealed the presence of ulcerations of an irregular shape 1 to 5 cm in diameter with retiform purpura in both lower limbs (Figure, A). In addition, racemous livedo was observed on almost all the body surface. Peripheral pulses in upper and lower limbs were preserved bilaterally. No signs of severe chronic venous insufficiency or palpable purpura were observed. Laboratory findings for differential diagnosis of SS were normal or negative. Dermatopathologic analysis showed presence of fibrin thrombi and wall thickening in the papillary dermis with neovascularization phenomena. No fibrinoid necrosis of the vessel wall, neutrophilic infiltrate, or leukocytoclasia was observed. A diagnosis of SS was confirmed.

Treatment with intravenous alprostadil (prostaglandin E1 [PGE-1]) (Prostavasin; Gebro Pharma GmbH) was started at doses of 60 μg every 24 hours for 5 days and then a dose of 60 μg every 24 hours monthly as maintenance. From the first infusion, the patient showed rapid improvement in pain. After 3 months of treatment, complete healing of the skin ulcers was observed (Figure, B). At last follow-up, the patient had been treated for 6 months with a monthly infusion of alprostadil and remained asymptomatic. No adverse effects were observed.

**Discussion** | There are no established guidelines regarding the treatment of SS. Steroids and immunosuppressants have been used, but their effectiveness is still controversial, given the absence of vascular inflammation. Anticoagulant and antiplatelet agents have been used for the long-term prevention of cerebral ischemic events. Antiplatelets are more suitable when findings of antiphospholipid antibodies (aPL) are negative; anticoagulants are more suitable when aPL findings are positive.2 Different therapies such as intravenous immunoglobulins, rivaroxaban, nifedipine, or those similar to prostaglandin have been used to treat the cutaneous symptoms. Mittag et al3 reported the first case of SS treated with iloprost in cycles also obtaining a remission in pain and clearing of the skin ulcers. Mofarrah et al4 described a case of livedoid vasculitis successfully treated with PGE-1 at dose of 60 μg for 5 days followed by a dose of 60 μg of monthly maintenance. The pain disappeared in the first 2 days, and ulcers healed after 3 weeks of treatment.

Both livedoid vasculitis and SS are cutaneous microcirculation abnormalities characterized by obliterative phenomena in the vessels. Noting this similarity, we used the same treatment regimen for SS. Alprostadil seems to be more effective in occlusive vasculopathy with racemous livedo. In the case reported by Mofarrah et al,4 iloprost was not as effective as

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**Figure. The Leg of a Patient With Sneddon Syndrome**

A. Before treatment, the leg shows multiple ulcerations. B. The ulcerations are cleared after 3 months of alprostadil treatment.