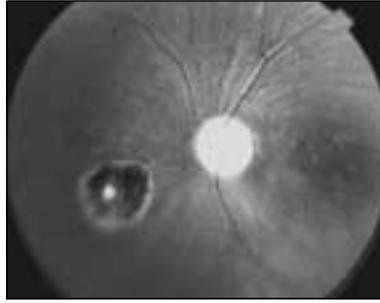


**Figure 2.** Fundus photograph of the left eye immediately after treatment of the worm with strong application of argon laser.

dren and young healthy adults. Most patients are unaware of the disease until ocular examination performed in school or an ocular examination later in life.<sup>2,3</sup> The typical late signs of DUSN predominate in these cases, and a solitary 400- to 700- $\mu\text{m}$  nematode is frequently present at this stage, even years after disease onset. According to Gass,<sup>1</sup> laser treatment of the nematode at any disease stage may improve visual acuity and inflammatory ocular signs. However, in our experience this improvement may be temporary, even if laser is applied to retinal areas simulating the presence of a worm. It is possible that some laser response in the RPE may interfere temporarily with the activity of the subretinal worm.

In 1984, Kazacos et al<sup>4</sup> showed that at least some DUSN cases are caused by *B procyonis* (Nematoda, family Ascarididae larvae), which are common intestinal roundworms of lower carnivores, including raccoons and skunks. Those authors experimentally produced DUSN in primates that were fed *B procyonis* eggs.<sup>6</sup> Additionally, the size of the intraretinal larvae and previous patients' contact with raccoons made the hypothesis that *B procyonis* was the probable cause of the disease even stronger. In their opinion, DUSN is caused by 2 species of nematodes or 2 sizes of a single species, reflecting different ages of larvae.<sup>4,5</sup> The latter seems to apply to our patient. Unlike *Toxocara* species, which do not exceed 700  $\mu\text{m}$ , *Baylisascaris* larvae grow considerably, from about 300  $\mu\text{m}$  at hatching to 2000  $\mu\text{m}$  or larger, the size most frequently recovered from clinically affected animals.<sup>4</sup> Adult *B procyonis* worms may also infect rats, squirrels, and dogs. Humans



**Figure 3.** Recent fundus photograph of the left eye showing the fundus 1 year after laser treatment. More pronounced fundus degeneration and a pale optic nerve are apparent.

and several other animals are potential intermediate hosts and become infected by ingesting *B procyonis* eggs from raccoon feces. The larvae hatch in the small intestine, enter the systemic circulation, and are distributed to various organs, including the eye.<sup>4,5</sup> In humans, *B procyonis* may cause visceral larva migrans, cerebrospinal nematodiasis, and ocular larva migrans. Fatal cases of *B procyonis* larva migrans have been reported.<sup>1,4,5</sup> Although Brazil is not an area endemic for raccoons, and cases of *B procyonis* ocular infection have not been reported in South America, we believe that our case could be the first. The presence of skunks in the peridomestic area of our patient makes this possibility likely.<sup>4,5</sup> Dogs and rats should also be considered potential sources of infection. It is important to emphasize that other species of nematodes should be considered as potential candidates for the cause of our patient's symptoms. As more local clinicians and veterinarians become aware of these larger ocular nematode infections, other important epidemiologic findings will be reported.

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## Bilateral Massive Retinal Hemorrhages in a 6-Month-Old Infant: A Diagnostic Dilemma

Retinal hemorrhages in infants sometimes pose a diagnostic dilemma for ophthalmologists.

**Report of a Case.** A 6-month-old infant was treated in the hospital for a rotavirus gastroenteritis. Two days after discharge from the hospital, he was readmitted, profoundly dehydrated and in hypovolemic shock. He had collapsed and was unresponsive. His Glasgow Coma Scale score was 4, and his pupils were fixed and dilated. He was intubated and ventilated. Serum and plasma levels were measured and revealed hypernatremic dehydration consistent with severe water loss via the gastrointestinal tract: sodium, 169 mmol/L; potassium, 7.3 mmol/L; chloride, 136 mmol/L; urea nitrogen, 25 mmol/L (70 mg/dL); and creatinine, 222  $\mu\text{mol/L}$  (2.51 mg/dL). The infant was acidotic with a pH of 6.8;  $\text{PO}_2$ , 10.6;  $\text{PCO}_2$ , 5.2; standard bicarbonate, 6.8 mmol/L; and base excess, 23 mmol/L.

Observations from funduscopy revealed massive bilateral retinal hemorrhages radiating from the posterior pole of the eyeball (**Figure 1**). Findings from coagulation studies, complete blood cell and differential cell counts, and thrombophilic screens were normal. His profiles for amino acid, fatty acid, and organic acid were normal. A computed axial tomographic scan of the brain showed diffuse cerebral edema, subdural blood in the temporal fossa, and diffuse subarach-

noid hemorrhage (**Figure 2**). The infant's clinical condition deteriorated and he died.

Findings from histopathologic examination of the eyes revealed massive retinal hemorrhages with subhyaloid and subretinal hemorrhages in both eyes (**Figure 3**). The brain scan revealed venous and capillary congestion with subarachnoid hemorrhage, a subdural collection, and focal intracerebral hemorrhages. There was also diffuse microvessel thrombosis in many organs, including the lungs, kidneys, and myocardium, consistent

with disseminated intravascular coagulation. Nonaccidental injury (NAI) was suspected because of the findings from clinical examination; in particular, massive retinal hemorrhages in association with intracerebral hemorrhage. However, there was no evidence of trauma. A skeletal survey revealed no abnormalities. The findings from clinical examination were consistent with severe hypernatremic dehydration causing diffuse intracerebral hemorrhage, subarachnoid hemorrhage, retinal hemorrhages, and ultimately brain death.

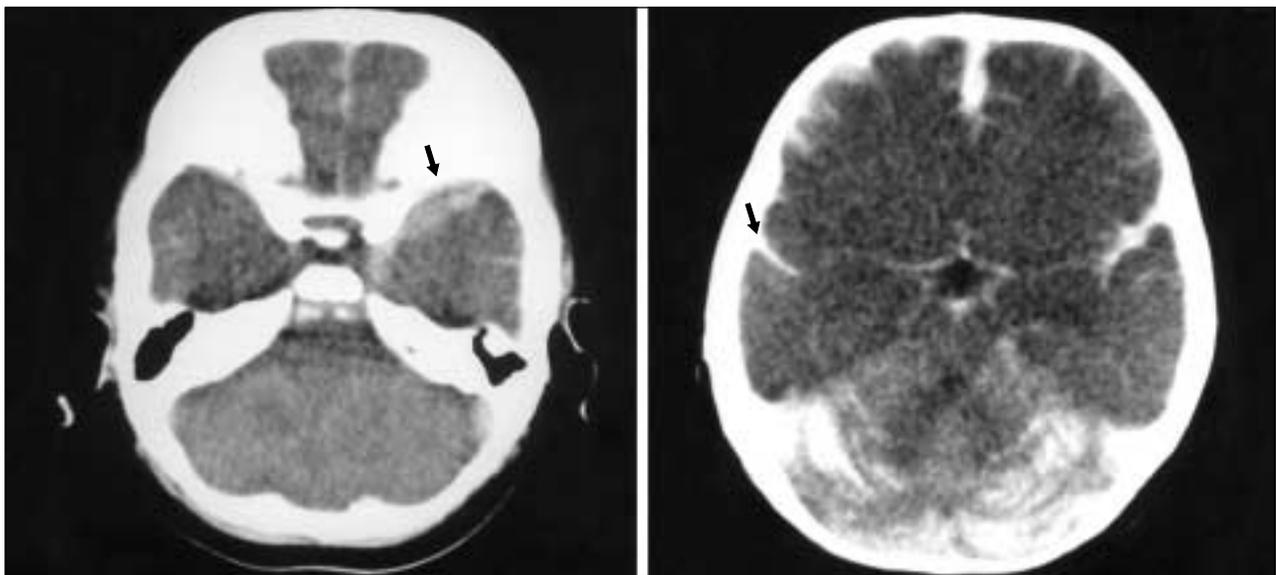
**Comment.** Retinal hemorrhages in infancy are believed to be a cardinal sign of NAI. They may occur in up to 89% of infants with NAI.<sup>1</sup> They may result from direct head trauma or the acceleration and deceleration forces generated by the shaking of the head. Shaken baby syndrome is a unique form of child abuse in which the only consistent external physical signs are ocular manifestations. Differential diagnoses of retinal hemorrhages include thrombocytopenias, leukemias, and infections such as infective endocarditis.

In this case profound electrolyte disturbance, namely, hypernatremic dehydration, caused intracerebral, subdural, and subarachnoid hemorrhages. Hypernatremia causes cerebral cellular dehydration and results in brain shrinkage. This leads to the rupture of bridging veins, causing subdural and intracerebral hemorrhages. Elevated intracranial pressure leads to increased retinal venous pressure and results in retinal hemorrhages.

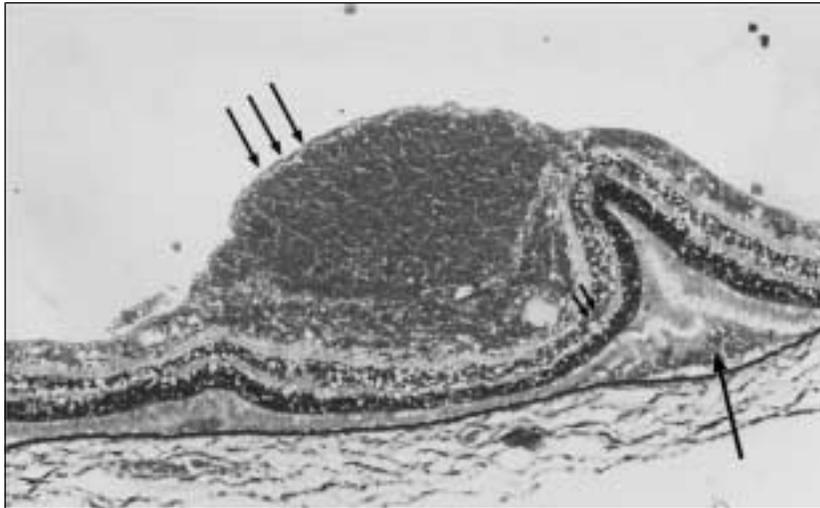
Finberg<sup>2,3</sup> reported 12 cases of subarachnoid or subdural hemorrhage from hypernatremic dehydration in infants, 2 of whom died and 10 who had severe residual neurological damage. Pathologic effects of hypernatremia were also reported following a nursery disaster in which an improper infant food mixture containing an excess of sodium chlo-



**Figure 1.** Retinal hemorrhages radiating from the posterior pole of the eyeball in a 6-month-old infant.



**Figure 2.** Noncontrast computed axial tomographic scans of the brain. Left, The arrow indicates subdural hemorrhage in the temporal fossa. Right, Diffuse cerebral edema and subarachnoid hemorrhage are indicated by the arrows.



**Figure 3.** Findings from histopathologic examination of the retina show subretinal (single arrow), intraretinal (double arrows), and subhyaloid hemorrhage (triple arrows).

ride was administered to infants. Findings from autopsy showed subarachnoid hemorrhage, intracerebral hemorrhage, cortical venous thrombosis, and venous infarctions. Similar cases in adults have shown widespread cerebral hemorrhage.<sup>4</sup> Infants are more susceptible to hypernatremia because of their large surface area and poor renal concentrating ability. The typical radiological findings from computed tomographic scans of infants with hypernatremia include brain parenchymal abnormalities, multifocal areas of hemorrhage, and infarction.<sup>5</sup> Retinal hemorrhages were not documented in these cases.

In conclusion, we report a case of massive bilateral retinal hemorrhages and intracranial hemorrhages attributable to profound hypernatremic dehydration in an infant. The findings from clinical examination are similar to those seen in NAI. It is important to highlight this to avoid potential mistaken diagnoses. Unexplained retinal hemorrhages in infancy mandate a full clinical workup. The NAI remains high on our list of differential diagnoses, but other pathologic conditions can mimic NAI and have an identical clinical presentation.

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### Retinal Periphlebitis in a Patient With Pineal Germinoma

Patients with pineal germinomas commonly show signs and symptoms related to increased intracranial pressure and direct compression of the upper brainstem or cerebellum.<sup>1</sup> Ocular manifestations typically include papilledema and extraocular movement disturbances. We describe a patient with a pineal germinoma who had posterior segment inflammatory changes.

**Report of a Case.** A 14-year-old boy of East Indian descent sought care because of a 2-month history of floaters and difficulty with visual tasks. The patient complained of headaches, nausea, vomiting, and an intermittent auditory bruit in the right ear. Systemic examination disclosed an unsteady gait and tremor. Ophthalmic examination revealed a

visual acuity of 6/12 OU. Convergence-retraction nystagmus, pupillary light-near dissociation, and slight limitation of upward gaze were present bilaterally. Diplopia was noted on right gaze with evidence of slight underaction of the left superior oblique. Findings from slit-lamp examination were normal. Dilated fundus examination revealed bilateral 2+ vitreous cells and marked optic nerve head edema (**Figure 1**). Tortuosity of the retinal vessels with areas of focal sheathing and exudates in a "candle wax dripping" configuration were also present. Large clumps of vitreous cells were noted in the inferior vitreous base bilaterally.

Fluorescein angiography revealed numerous areas of segmental hyperfluorescence of the retinal venules along the temporal arcades and leakage from both discs bilaterally. Neither capillary nonperfusion nor retinal neovascularization was noted. Laboratory investigations indicated a minimally elevated erythrocyte sedimentation rate and a normal serum angiotensin-converting enzyme level. Findings from tuberculin skin test, rapid plasma reagin test, and chest x-ray film were normal. A preliminary diagnosis of sarcoidosis was suspected. The patient was referred to the neurology service for evaluation of possible central nervous system involvement.

Computed tomographic scans and magnetic resonance images revealed a partially calcified homogenous 3-cm pineal mass and obstructive hydrocephalus. The neurology service felt the clinical and radiographic findings were consistent with a germinoma and recommended radiotherapy. Based on the clinical examination and the presence of the sarcoidlike appearance in the fundus, the ophthalmology service recommended biopsy to obtain a tissue diagnosis.

A ventriculostomy of the third ventricle with endoscopic biopsy was performed. Findings from histologic examination confirmed the diagnosis of a germinoma. A cerebrospinal fluid specimen was analyzed, and the results were negative for tumor markers and inflammatory cells. Fractionated focal radiotherapy (45