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Congenital neonatal herpes simplex retinitis

Neonatal herpes simplex virus (HSV) 2 infection seldom occurs with an incidence of 1 in 3000 live births in the United States.[1,2] Most commonly the virus is acquired through the birth canal. Very rarely the infection is developed in utero, presumably transplacental via chorionic villi.[1,3] In utero infections can cause pneumonitis, myocarditis, hepatosplenomegaly, encephalitis, hemolytic anemia, cerebral palsy, and mental delay.[1] Ocular manifestations include cataracts, corneal ulceration, anterior uveitis, vitritis, chorioretinitis, and optic atrophy.[1,3–9]

Neonatal and postnatal HSV 2 infections can cause localized cutaneous, perioral, and ocular involvement or disseminate to affect the central nervous system, adrenals, liver, and lungs.[1,3,5] Approximately 4% of all neonatal HSV infections can result in microcephaly, hydrocephalus, chorioretinitis, and vesicular skin lesions at birth.[5] When the eye is involved, the most common type of ocular involvement is usually blepharoconjunctivitis or keratitis,[4] with very few cases of chorioretinitis or optic atrophy ever reported.[3,4]

A 34-week neonate was born by C-section at University of Florida Shands Hospital to an 18-year-old southern Caucasian mother who had premature rupture of membranes at the time of birth. At birth the patient was noted to have good respirations, heart rate, colour, muscle tone, and reflexes, with a low birth weight of 1732 g. The neonate was taken to the NICU as he continued to become hypoglycaemic despite feeds. On the second day the infant was found to have vesicular lesions on his abdomen, which progressed to his oral cavity, hands, and base of feet bilaterally. The mother denied any history of oral or vaginal lesions as well as any intercourse during the pregnancy. The patient underwent a full work-up for TORCHES infection, which was negative for toxoplasma,

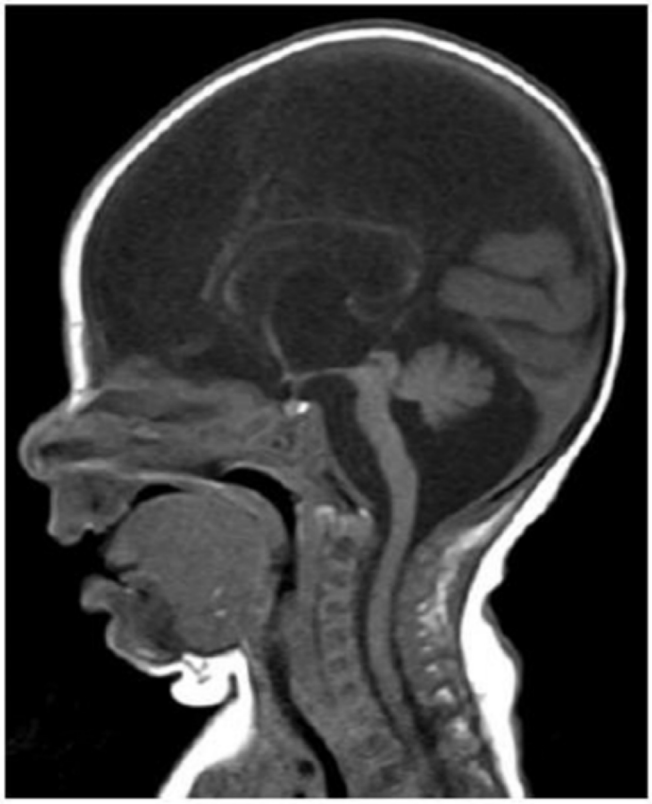

Fig. 1—Sagittal nuclear magnetic resonance image depicting hydranencephaly with complete cystic encephalomalacic loss of the bilateral frontal/temporal, and parietal lobes as well as diminution of the cerebellar hemispheres and brainstem with relative sparing of the occipital lobes.

rubella virus, cytomegalovirus, hepatitis B, HSV-1, and *Treponema pallidum*. However, HSV-2 blood and skin PCR returned positive. Cerebral spinal fluid (CSF) HSV PCR was haemorrhagic but noted to be positive for HSV 2 as well. The patient was started on IV acyclovir 20 mg/kg/day every 8 hours.

Cranial ultrasonography performed showed hydranencephaly, which is the absence of the cerebral hemispheres with CSF in its place. The ultrasonography noted complete cystic encephalomalacic loss of the bilateral frontal/temporal, and parietal lobes with relative sparing of the occipital lobes. The cerebellar hemispheres and brainstem were noted to be markedly diminutive bilaterally. Magnetic resonance imaging confirmed these findings and additionally showed a posterior fossa hygroma, nearly absent corpus callosum, and haemorrhage and/or calcification in the right temporal lobe and dural surfaces with diminution of the carotid and vertebrobasilar vessels (Fig. 1).

On initial undilated ophthalmic consultation examination of the infant, the anterior segment showed no corneal dendritic lesions or epithelial or stromal corneal disease. The anterior chamber was quite with no signs of inflammation, no iris atrophic patches, and clear lens. Dilated eye examination was not performed at the initial visit. He was later seen in clinic at 8 weeks of age. The baby fixed and followed light from the right eye but not with the left eye. In the left eye, shimmering nystagmus was noted. The cornea, lens, and anterior chamber were clear and quite in both eyes. Cycloplegic refraction was +1.50 diopters of sphere in both eyes. Dilated fundus examination of the right eye showed clear vitreous with normal optic disc, small scar temporal to the fovea, and multiple pigmented well-defined chorioretinal scars in the nasal retina (Fig. 2A, B). The left eye fundus examination showed clear vitreous cavity, with normal disc and a large chorioretinal pigmented scar involving the macula up to the superior and inferior arcades as well as peripheral scars superonasally (Fig. 3). No signs of active vitritis or retinitis was noted in either eye. Repeat blood HSV 2 PCR performed after the clinic visit remained positive and the patient was continued on systemic antivirals.

The baby was then scheduled for an examination under anaesthesia, and the examination findings were confirmed

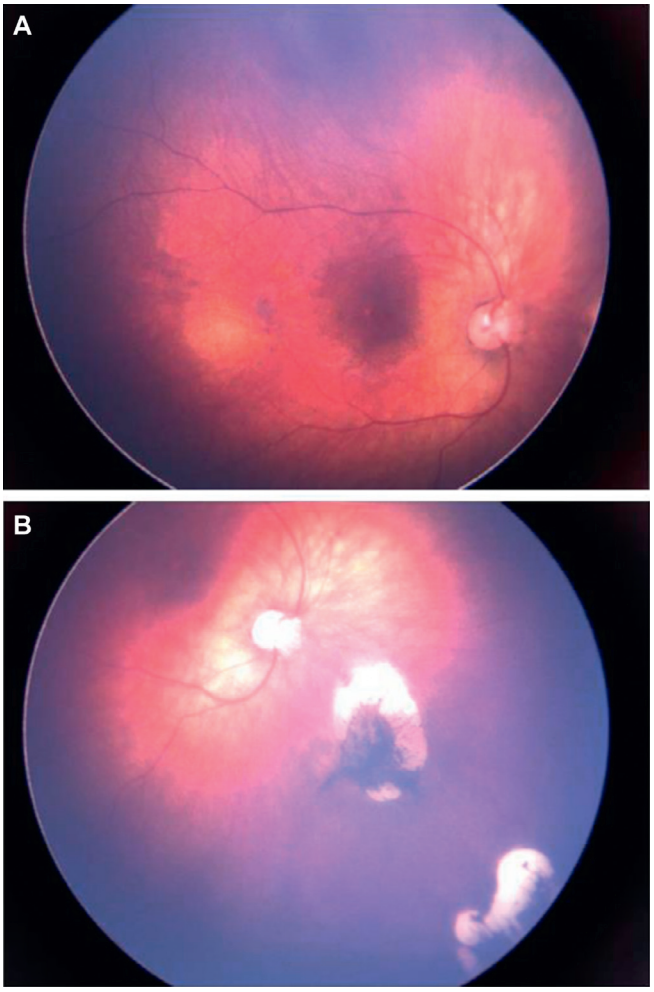

Fig. 2—(A) Fundus photograph of the right eye showing clear vitreous with normal optic disc and small scar temporal to the fovea. (B) Fundus photograph of the right eye showing multiple pigmented well-defined chorioretinal scars in the nasal retina.

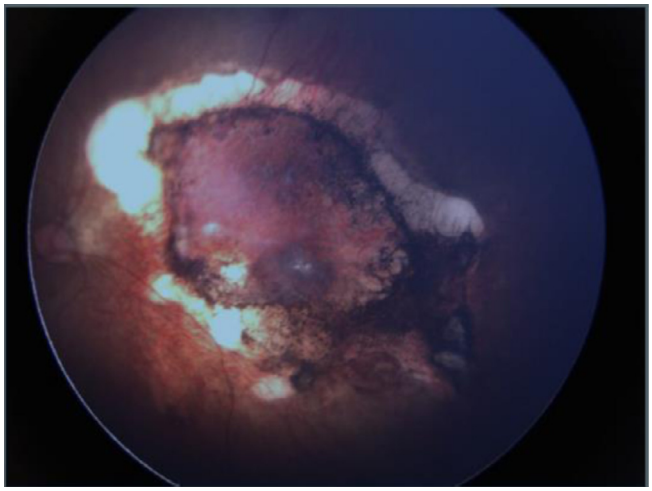

Fig. 3—Fundus photograph of the left eye showing clear vitreous cavity, with normal disc and a large chorioretinal pigmented scar involving the macula up to the superior and inferior arcades.

with presence of multiple pigmented chorioretinal scars and no signs of active vitritis, retinitis, vasculitis, or optic neuropathy in either eye.

The infant was continued on oral antivirals until his next HSV 2 blood PCR, which was negative at 7 months of age. The acyclovir was stopped and the patient was placed on valacyclovir PRN for recurrence of skin lesions.

Neonatal HSV 2 infection can be devastating and many infants are born premature with low birth weight. There are many reports of acute retinal necrosis (ARN) in younger adult patients associated with congenital or neonatal HSV 2 infection, but few cases of ARN or chorioretinal atrophy are reported in neonates.

In one of the reports by LaMattina et al,[6] 2 premature infants born at 27 and 26 weeks postmenstrual age with congenital HSV 2 were found to have active chorioretinitis lesions, which progressed to ARN in both eyes of the infants. Both infants were treated with oral acyclovir, with one infant requiring laser demarcation of the ARN. The infants, followed until 4 years of age, were noted to have optic neuropathy with nystagmus and strabismus. The authors did not provide retinal pictures or detailed the extent of the retinal findings. One of the few cases we found was in 1964, and it reported a neonate who developed fever, irritability, and seizures at 3 weeks. The infant was found to have massive macular haemorrhage and exudates and subsequently went blind with absence of cerebrocortical function.[9]

In 2 cases reported in a 37-week-old infant and a 4-week-old infant born with congenital HSV 2 infection, both infants were noted to have chorioretinal scars in the posterior fundus in both eyes with no signs of active infection or inflammation.[3,4] Our case is very similar to the above 2 cases, presenting with multiple pigmented chorioretinal scars involving the nasal retina in the right eye and macula in the left eye. No active retinitis, vasculitis, or vitritis was noted in our case either.

This is very unusual and further supports that congenital HSV 2 infections should be considered in patients with bilateral hyperpigmented scars regardless of their location in the retina, besides the more common congenital toxoplasmosis as the cause of posterior retinal scars. Even with treatment, the infant will be at risk of reactivation and will need frequent close eye examinations. We are not aware of the role of prophylactic systemic acyclovir to prevent reactivation of retinitis in these rare cases presenting with chorioretinal scars.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.jcjo.2018.08.009.

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Bilateral ocular ischemic syndrome as a manifestation of Takayasu arteritis in children

Takayasu arteritis (TA) is a chronic, autoimmune, granulomatous, inflammatory disease of the aorta and its major branches at their origin, which results in dilatation, occlusion, stenosis, and (or) aneurysm formation of the affected arteries.[1,2]

TA could be seen in different races, but the incidence is higher in the Southeast Asian population. The onset of disease is most frequent under 40 years old, generally in the third and fourth decade of life, and there is a predilection for women.[3,4]

TA in childhood could affect young infants to late adolescents, with the youngest patient diagnosed at 6 months old. It is reported as the third most common cause of vasculitis in the pediatric patients and is the only large vessel vasculitis in this group.[3,4]

The incidence of TA in the pediatric population has been estimated at 1.2 to 2.6 million per year.[4,5] Around 5% of patients with TA are children and adolescents. The majority of children are diagnosed between 8 and 13 years old, and as well as adults, there is a predilection for female patients, with a ratio of 3:1.[4]

We present a case of a pediatric patient who was diagnosed with bilateral ocular ischemic syndrome (OIS), as an initial manifestation of childhood Takayasu arteritis (c-TA).

Case report

A 12-year-old Peruvian female complained of headache, several episodes of left eye amaurosis fugax, right hemiparesis, with spontaneous recovery, and bradylalia for 15 days. On examination, visual acuity was 6/6 in both eyes (BE). Anterior segment and gonioscopy were within normal limits. Dilated retinal examination revealed venous dilation, some arteriolar narrowing, cotton wool spots, and widespread microaneurysm formation in BE (Fig. 1). Fluorescein angiography (FFA) showed patchy choroidal filling, delayed arm-to-retina circulation time, prolong arteriovenous time, staining of retina vessels, and nonperfusion areas in BE (Fig. 2). The diagnosis of bilateral OIS was done. A complete work-up was required in order to establish the cause of OIS. Angiogram revealed a severe stenosis in both subclavian arteries, stenosis of the right common carotid artery, occlusion of the left common carotid artery, severe stenosis of the right and left vertebral arteries (Fig. 3), and she had also abdominal aorta involved. Laboratory studies: C-reactive protein was 54, erythrocyte sedimentation rate of 84, and infection causes were ruled out. The diagnosis of TA was established. She started on corticosteroids and mycofenolate with good response, with a partial reperfusion of carotids and good perfusion of both eyes.

Discussion

The pathologic course of TA starts in the adventitia, which progresses to the intima with marked proliferation and fibrosis, eventually causing vascular narrowing, occlusion, with or without thrombosis, and as a result, compromise of the blood flow occurs through the involved vessels. Aneurismal formation can be seen later.[5]

c-TA starts with a nonspecific and acute inflammatory phase (anorexia, fever, night sweats, weight loss, arthralgia, and skin rash).[3,4] After that, one-third of children experience effects of tissue ischemia with significant vascular sequelae. The average time between the symptoms until the final diagnosis is 19 months, almost four times longer than adults.[3–5]

The spectrum of clinical features varies in pediatric and adult patients, being hypertension the most common symptom in both groups (73%), followed by headache (53%), constitutional symptoms (53%), and fever (45%). Bruit and claudication pain are uncommon in c-TA.

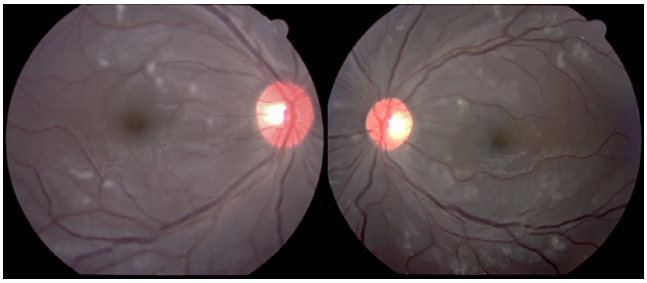

Fig. 1—Bilateral color fundus pictures showing venous dilatation, arteriolar narrowing, cotton wool spots and widespread microaneurysm formation.