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Sir.

Regression of Remote Capillary Haemangioma after Local Intralesional Injection of Corticosteroids

Capillary haemangioma is the most common vascular tumour of the ocular adnexa and orbit in infants and children. Spontaneous involution frequently occurs by 4–7 years of age. Capillary haemangiomas of the eyelids and orbit are best managed by observation, except when vision is threatened by untoward effects of the tumour. When intervention becomes necessary, intralesional corticosteroid injection is preferred.

We report on the beneficial effect of a local intralesional corticosteroid injection on a distant capillary haemangioma in a 16-week-old infant.

Case Report

A 3-month-old infant was referred for treatment because of gradually enlarging haemangiomas of the right upper eyelid and the left parotid area which appeared at age 2 months. Initial examination bluish-purple, 'spongy' revealed subcutaneous masses in the right upper eyelid and the left parotid area with telangiectatic vessels. The masses were shown to have irregular margins and rapid uniform enhancement on dynamic computed tomography with iodinated contrast medium. Ocular examination revealed mild proptosis of the right eye with blepharoptosis. The anterior and posterior segments were normal. Cycloplegic refraction was +2.0 -2.5 \times 20° for the right eye and +0.5 -0.25 \times 180° for the left. Because the lesion in the right upper eyelid was expanding rapidly, with progression of astigmatism to $+3.5 -5.25 \times 30^{\circ}$ and the threat of amblyopia, local treatment was recommended. At the age of 16 weeks, a 50:50 mixture of triamcinolone acetonide (80 mg) and betamethasone (6 mg) was injected under general anaesthesia to the right upper eyelid mass. One week later regression of the injected lesion was noted, together with regression of the astigmatism in the right eye to $+3.0 - 2.0 \times 30^{\circ}$.

Interestingly, concomitant and significant regression of the capillary haemangioma of the left parotid area, which had been excessively enlarged prior to treatment, was also observed.

Discussion

Patients presenting with orbital capillary haemangiomas can also have coexisting capillary haemangiomas in other parts of the body. To the best of our knowledge, this is the first report of regression of a distant capillary haemangioma after local intralesional injection of corticosteroids. Although regression of the capillary haemangioma lesions may have been due to spontaneous regression, these lesions were characterised by gradual and constant growth prior to the steroid injection, with immediate regression after intralesional injection of corticosteroids. This indicates that regression was due to treatment and not to the natural history of these lesions.

We suggest two explanations for this remote effect. First, the dose of corticosteroids injected intralesionally (triamcinolone acetonide 80 mg and betamethasone 6 mg) is estimated to be 109–333 times the daily cortisol production of an age-matched infant (1.8-5.5 mg/day).² Second, the amount of corticosteroids that can be absorbed systemically from intralesional injections may be excessive.³ This combination of extremely high corticosteroid dose and excessive intravascular absorption may account for not only the response of the distant capillary haemangioma, but also the systemic side effects. Weiss³ diagnosed growth retardation in two patients who suffered adrenal suppression after corticosteroid injection into periocular haemangiomas. Therefore, clinicians should take into consideration that intralesional corticosteroid injection into capillary haemangioma may resemble systemic therapy and may cause involution of distant lesions.

David Zadok, MD Yair Levy, MD Pinchas Nemet, MD

Department of Ophthalmology Assaf Harofeh Medical Center Zerifin 70300 Tel Aviv Israel

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Sir

Pseudoretinitis Pigmentosa due to Sub-optimal Treatment of Neurosyphilis

The treatment of established neurosyphilis requires intensive therapy with frequent, high-dose intravenous penicillin G or high-dose intramuscular repository penicillin G with probenecid. However, patients may be treated for neurosyphilis with penicillin in oral or intramuscular form alone. The usual dose prescribed in these situations is inadequate for the complete eradication of the *Treponema pallidum* organisms from the eye, yet may be

sufficient to mask the signs and symptoms of infectious syphilis. This can result in luetic ocular complications. We describe a patient who illustrates this point: having previously been treated with intramuscular penicillin for neurosyphilis, he subsequently developed luetic pseudoretinitis pigmentosa.

Case Report

A 67-year-old man presented with a 2 year history of progressive decrease in visual acuity and nyctalopia. He had had no previous visual symptoms. His visual acuity was 6/18 right eye and 6/9 left eye. Argyll-Robertson pupils were present.

Ophthalmoscopy showed pale optic discs and marked attenuation of retinal vessels. There was a diffuse, granular appearance of the retinal pigment epithelium throughout both fundi and drusen present at the maculae. Intraretinal bone corpuscle pigmentation and choroidal atrophy were particularly prominent temporal to the macula in the right eye.

Humphrey perimetry showed peripheral constriction of the visual field in each eye and a marked nasal scotoma in the right eye. An electroretinogram (ERG) showed normal rod b wave latencies but grossly reduced b wave amplitudes in each eye. A cone ERG was abnormal in the right eye and normal in the left. An electro-oculogram was abnormal in both eyes but more so in the right.

General examination revealed no stigmata of congenital syphilis. Cardiovascular examination was unremarkable. Neurological examination revealed Argyll-Robertson pupils and absent ankle jerks. Serum VDRL was negative, but serum TPHA and FTA-ABS were positive. A diagnosis of luetic pseudoretinitis pigmentosa was made.

The patient then disclosed that he had had previous treatment of syphilis. Fourteen years earlier he had presented with 'shooting pains' in his legs. The only abnormal neurological feature at that time Argyll-Robertson pupils; dilated examination had revealed no retinal abnormality. Cardiovascular examination, the chest radiograph and electrocardiogram had been normal. Serum VDRL, TPHA and FTA-ABS were positive and analysis of cerebrospinal fluid (CSF) was normal with negative CSF VDRL, TPHA and FTA-ABS serology. Treatment had consisted of a 14 day course of intramuscular procaine penicillin (0.9 mega-units daily) without probenecid. He had failed to attend subsequent review appointments.

In view of the development of luetic pseudoretinitis pigmentosa he was admitted for further investigation and treatment. CSF analysis was repeated and showed a mildly elevated protein at 0.5 g/l, no pleocytosis and negative CSF VDRL, TPHA and FTA-ABS serology. Intravenous penicillin G, 4 mega-units 4 hourly, was administered for 14 days without complications.

Discussion

Syphilitic retinopathy can mimic retinitis pigmentosa.¹ The ERG may help in differentiating the two conditions, as in this case. In retinitis pigmentosa the B wave latency as well as its amplitude are reduced whereas in syphilitic retinopathy the B wave latencies remain normal and the amplitudes are reduced in proportion to the amount of damaged retina.¹

Recently, some authors have reported a preponderance of meningovascular and vascular syphilis as the presentation of symptomatic neurosyphilis. This may represent an increase in partially treated syphilis. As in this case, hyporeflexia, absent ankle jerks and pupillary abnormalities are the commonest signs.^{2,3}

Adequate treatment of neurosyphilis is essential. The WHO recommends that 0.018 µg/ml be accepted as a minimal treponemicidal concentration. Treatment with procaine penicillin (2.4 mega-units daily by intramuscular injection) plus probenecid (0.5 g 6 hourly) is accepted as achieving treponemicidal levels in CSF but only inconsistently.⁴ In the case described the patient received suboptimal treatment resulting in treatment failure and later ocular complications. Syphilis remains an important disease and should still be considered in the differential diagnosis of apparent retinitis pigmentosa syndrome.

A. J. Lotery M. O. McBride C. Larkin J. A. Sharkey

Departments of Ophthalmology and Genito-urinary Medicine

Royal Victoria Hospital Belfast

Northern Ireland

Correspondence to: Mr A. J. Lotery Department of Ophthalmology Royal Victoria Hospital Belfast B12 6BA

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