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TRANSIENT SUPERIOR OBLIQUE SYNDROME IN SCLERODERMA A CASE STUDY

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Abstract

A 27-year-old Eurasian woman with a 13 year history of scleroderma (progressive systemic sclerosis) developed typical features of Superior oblique syndrome (Brown's syndrome), which spontaneously resolved after 12 months, leaving a palpable nodule in the left superior oblique tendon. The aetiology and associations of this disorder are briefly reviewed.

Key words: *superior oblique, Brown's syndrome, scleroderma.*

CASE REPORT

The patient, a 27-year-old Eurasian woman, had presented at age 14 with Raynaud's phenomenon and tight skin of the fingers and face. A clinical diagnosis of scleroderma (diffuse, Type III)[1] was supported by skin biopsy.

Scleroderma is a connective tissue disorder of unknown aetiology characterised by vascular insufficiency, particularly in the form of Raynaud's phenomenon, bilateral skin stiffness, various systemic disturbances associated with over-production of collagen, and features of disturbed immunity. It has been subdivided into 'types' according to the extent of the skin involvement. Although uncommon it is not excessively rare and has an estimated prevalence in Australia of approximately 10 cases per 100,000 of population.

Subsequently dysphagia, respiratory restriction and limitation of large joints developed. Her general management included oral Prednisolone, intermittent D-Penicillamine, oesophageal dilatation and Cimetidine for gastro-oesophageal reflux.

In April, 1989, at age 26, she noticed increasingly frequent vertical doubling of images on attempted upgaze, which was intermittently relieved in association with a palpable 'click' in the upper medial corner of her left orbit, and some discomfort in that region. Examination in April, 1990 revealed restriction of elevation of the left eye in adduction, with local tenderness in the region of the left trochlea. Ocular movements and Hess charts were entirely consistent with Brown's syndrome, in relation to acquired isolated inferior oblique paralysis. (Figs. 1, 2).

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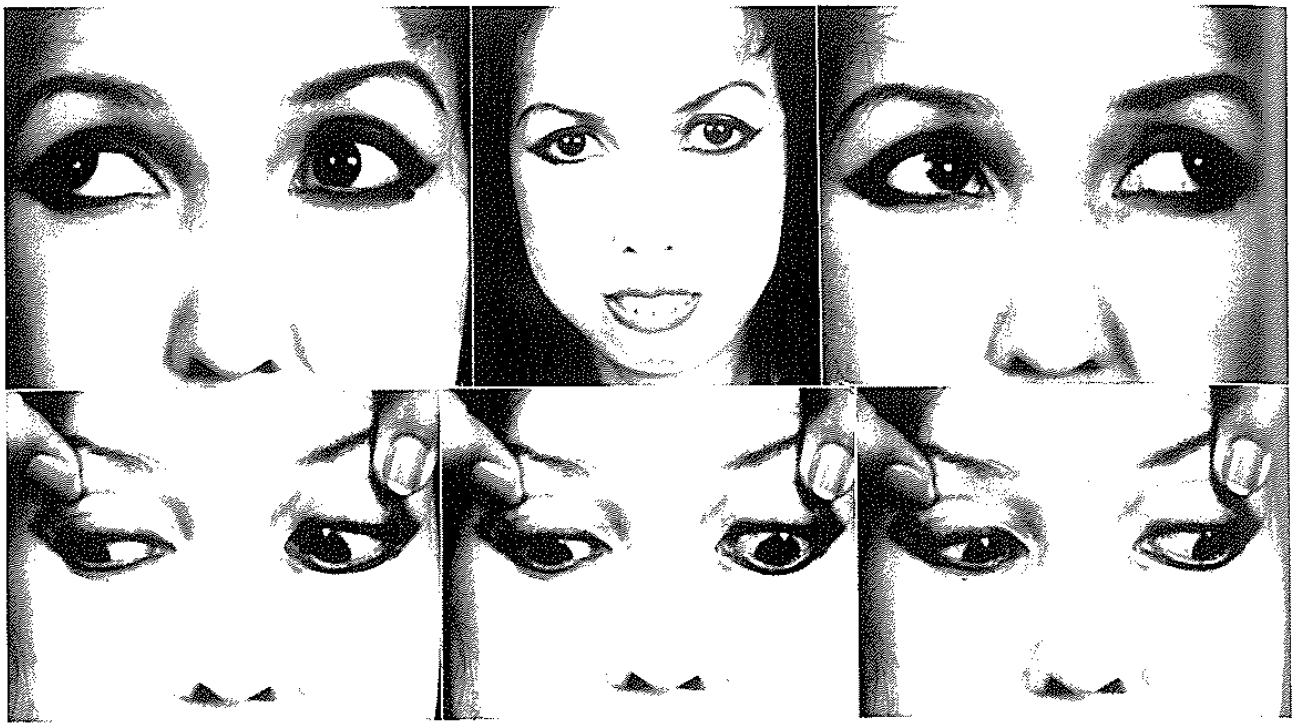

Figure 1: Facial features and restriction of left eye on dextroelevation.

No click was detected on examination. Rose Bengal staining and Schirmer test demonstrated a mild lacrimal deficiency. Contraction and stiffening of her eyelids and lips were also consistent with the primary diagnosis of scleroderma.[2]

Computerised topography of the orbits and brain was performed, and showed no abnormality.

Twelve months after the onset of her ophthalmic symptoms, the patient was woken at

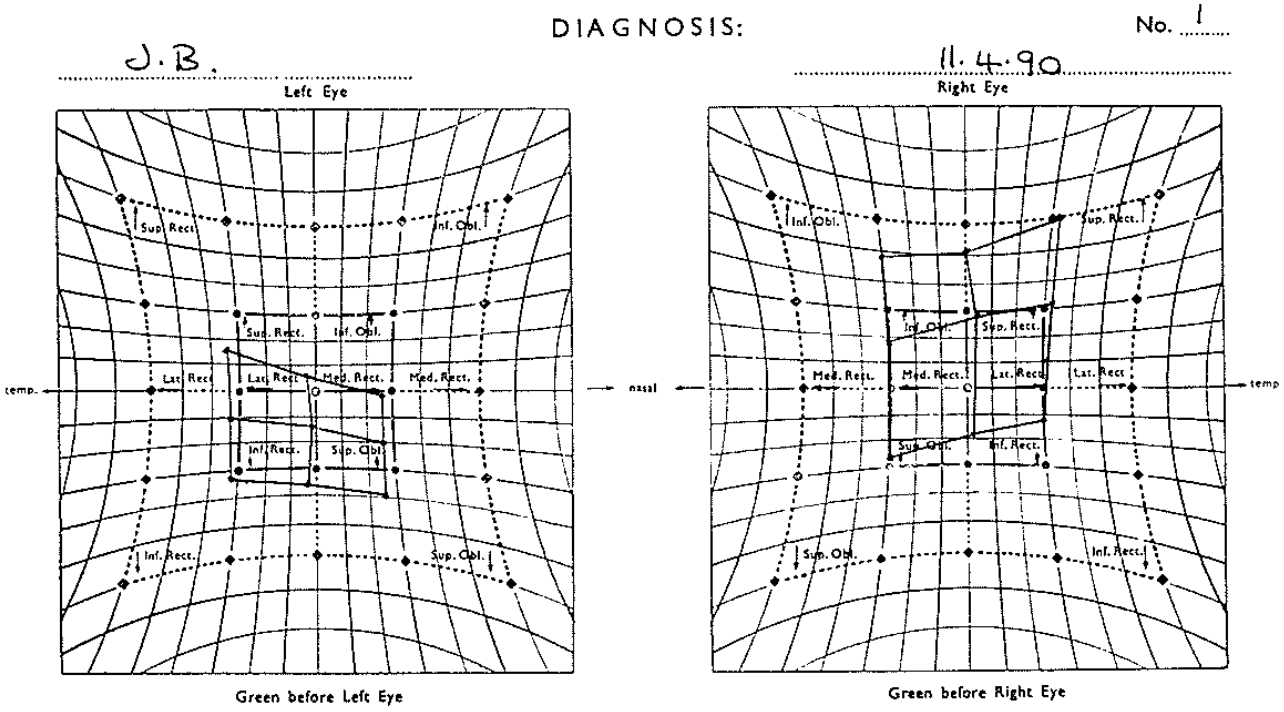

Figure 2: Hess chart 10 months after onset.

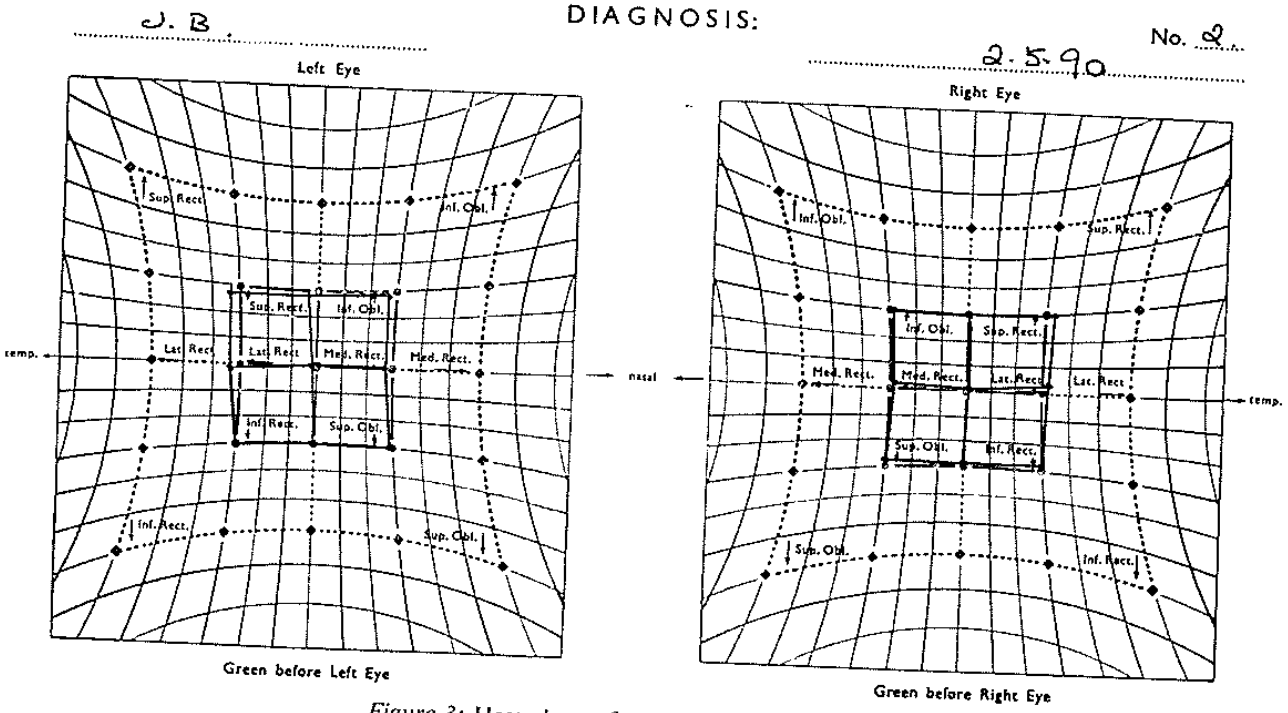

Figure 3: Hess chart after resolution of diplopia.

night by a pain above her left eye, and found that the diplopia had resolved, which has not re-occurred. Subsequent examination showed normal ocular muscle balance and motility (Fig. 3). A mobile nodule could be palpated in the antero-medial part of the superior oblique tendon.

DISCUSSION

In an early description of the syndrome bearing his name, H.W. Brown[3] proposed shortening of the anterior tendon sheath of the superior oblique muscle as the cause of this musculo-fascial disorder seen most often in children, and often resolving spontaneously. However, the most effective surgical treatment was found not to be excision of the tendon sheath, but tenotomy or tenectomy of the superior oblique tendon,[4] a finding consistent with other theories of pathogenesis such as reduced elasticity or shortening of the tendon[5], or a nodular thickening of the tendon obstructing its free movement through the trochlea.

The most common systemic association of this condition is with rheumatoid arthritis,[7] implying an increased frictional resistance to tendon movement. Local treatment by cortico-steroid injections has been helpful to a varying degree in such cases.[8,9,10] Local inflammatory[11] and neoplastic disease,[12] and surgical trauma[13] have been implicated rarely. A bilateral case developing in pregnancy has been reported.[14] Current understanding of the causes and mechanism has been comprehensively reviewed by Wilson et al.[15]

The late onset of this patient's disorder is unusual,[6] and supports the assumption that her systemic illness was a causative factor. It is suggested by Weilby that, in analogy with trigger finger,[17] the nodular thickening of the tendon may be secondary to frictional resistance to movement through the trochlea, with over-riding and bunching of concentric layers of collagen within the tendon. Spontaneous resolution could thus occur through the restoration of the normal arrangement of the tendon fibres, perhaps accompanied by some reduction of swelling within the tendon. This suggestion is consistent with the common experience of release with a 'click',[18] and in the case described here, by the residual palpable thickening of the tendon.

The specific relationship of Brown's syndrome with scleroderma or other rheumatic disorders

is not clear. The superior oblique tendon does not have a true synovial sheath, so that the syndrome is not a teno-synovitis. Localised infiltration and/or oedema is seen in other tendons and ligaments in rheumatoid arthritis, more rarely in scleroderma; involvement of the superior oblique tendon may be rare simply because of its smallness in proportion to the bulk of other tendinous structures. If frictional damage is a factor in nodule formation, the immobilisation of the tendon when totally trapped within the trochlea will tend to encourage spontaneous recovery, which is frequently observed.

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