Intermittent superior oblique tendon sheath syndrome

A case report

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A 37-year-old married woman who had been suffering from rheumatoid arthritis for 17 years was first seen in March, 1967, complaining of intermittent diplopia for one month. When seen by the casualty officer she was found to have vertical diplopia in laevo-elevation, but surprisingly Hess screen chart plotted the next day (Fig. 1) was normal and diplopia could not be elicited.

FIG. 1 Hess charts in March, 1967

In the next few months these intermittent symptoms became more constant and the patient noticed vertical diplopia on looking down and to the left and on reading; a Hess chart plotted August 16, 1967, now showed slight but definite evidence of a right superior oblique weakness (Fig. 2, opposite).

A fortnight later she complained that the diplopia had suddenly altered, so that she was now experiencing double vision on looking up and to the left rather than down and to the left. On clinical examination she was found to have restriction of elevation in adduction of the right eye with a down-drift of the right eye on adduction, and the diagnosis of a superior oblique sheath syndrome was made; this was confirmed by the Hess chart plotted at that time (Fig. 3, opposite).

One month later, she complained that the double vision was again evident on looking down and to the left, but this gradually improved, and on January 6, 1968, she complained only of occasional diplopia on looking down and to the left. After another 3 months, however, she complained that the diplopia was more noticeable, although similar in type (*i.e.* most marked on laevo-depression) and

FIG. 2 Hess charts on August 16, 1967, showing weakness of right superior oblique muscle

FIG. 3 Hess charts on August 30, 1967, showing marked superior oblique tendon sheath syndrome

that she had also felt some clicking over the upper and inner corner of the right eye. A Hess chart (Fig. 4, overleaf) again confirmed the clinical impression of a right superior oblique weakness.

The symptoms and signs have since remained static except that during August, 1968, she had yet another brief episode of double vision on looking up and to the left which lasted for a few days.

The patient has been a known case of rheumatoid arthritis for 17 years, with numerous rheumatoid nodules on the fingers, forearms, and elbows and the typical hand deformities of this condition, but with very little pain at present. She has been treated in the past mainly with aspirin but also received a course of gold injections in 1954.

Investigations

Hb 12.9 per cent. with anisocytosis and anisochromia; white cell count 7,400 with 10 per cent. eosinophils; erythrocyte sedimentation rate of 40 mm./1st hr; positive latex-fixation test; negative reaction with antinucleo-protein reagent. Serology, serum electrolytes, and plasma proteins were all normal. Skull x ray and chest x ray normal. Tensilon test negative.

FIG. 4 Hess charts in April, 1968, showing residual weakness of right superior oblique muscle

Treatment

As the patient is able to maintain binocular vision at present except when looking down, she is being treated with 3 dioptres vertical prisms incorporated in her reading glasses; which she finds satisfactory.

To summarize this rather involved case history, this patient with chronic rheumatoid arthritis has for the past 18 months been experiencing variable vertical diplopia. Usually the clinical picture has been that of a slight and variable weakness of the right superior oblique muscle, but, in addition, there have been periods when the diplopia has suddenly changed in nature, and at these times the clinical picture has been that of a right superior oblique tendon sheath syndrome, *i.e.* restriction of elevation in adduction of the right eye.

Discussion

Since the superior oblique tendon sheath syndrome was first described (Brown, 1950) there has been considerable interest in this condition with numerous case reports (Brown, 1950, 1957; Nutt, 1955; Girard, 1956; Esterly, Nadbath, and Russell, 1960). The majority of these reported cases are congenital in origin and, of the few acquired cases, most have been due to trauma of the tendon sheath of the superior oblique either from injury or from surgery. Although the basic aetiological factors may be unknown, it has always been assumed that the mechanism of the condition is a shortening of the tendon sheath from the trochlea to its insertion in the globe.

However, as distinct from this main group, isolated reports have been made of a rather different manifestation of the superior oblique tendon sheath syndrome, which is acquired, idiopathic, and apparently intermittent. The four following cases have been described in the literature:

(1) Girard (1956) described an 18-year-old patient, showing apparent restriction of elevation in adduction of one eye but otherwise orthophoric, who by repeated attempted movements was able to overcome the restriction and produce a full range of ocular movement.

(2) Mein (1964) described a case in a 17-year-old girl who showed a similar type of defect, which also could be overcome by attempting to elevate and depress the eye in adduction. In this case the defect also appeared intermittently in the other eye after her first attendance and, in addition,

she sometimes experienced a clicking sensation on full elevation and on depression of the eye in adduction.

(3) Clark (1966) reported a case in a 31-year-old man who had a left hyperphoria and recurrent episodes when the left eye became hypotropic and intorted on adduction.

(4) Mills and Coate (1967) reported a case in a 22-year-old woman with intermittent diplopia, who was found to have an intermittent limitation of elevation in adduction of one eye which later became permanent. At operation the sheath was explored between the globe and the trochlea, and was found to be thickened towards the trochlea although the tendon was normal. Both the tendon and the sheath were divided and the symptoms improved for a short time, but recurred later, though less severely.

In case reports 1, 2, and 4 it was postulated hypothetically that a mechanical obstruction of some sort was present in the superior oblique tendon intermittently preventing movement around the trochlea. In the following discussion further evidence for this hypothesis will be given and the condition examined in more detail.

In the case described in this paper there are three essential features, all of which are intermittent:

(1) A gross restriction of elevation in adduction of the affected eye, *i.e.* a failure of the superior oblique tendon and its sheath to be passively stretched. This was also shown in all four cases mentioned above.

(2) A clicking sensation in the upper and inner angle of the orbit of the affected eye (this was also shown by Case 2 and possibly Case 1).

(3) A slight but definite weakness of the superior oblique in the affected eye which always appeared consecutively but not simultaneously with the restriction of stretching of the superior oblique. It is the first time that this feature has been commented upon but, in fact, Case 3 demonstrated an identical condition.

Anatomically the tendon of the superior oblique has a loose fascial covering as it passes forwards from the muscle belly, but this becomes a firm fibrous sheath-like tunnel at the region of the trochlea and continues as such while the tendon bends around the trochlea and passes on to the globe. The only satisfactory explanation for these combined phenomena is that there is a swelling in the tendon of the superior oblique just behind the trochlea at the mouth of the fibrous sheath. Intermittently, on stretching the muscle, the swelling is unable to pass into the sheath, thus causing the clinical manifestation of the tendon sheath syndrome. Intermittently again the swelling passes into the sheath but on contraction of the muscle causes some slight resistance, thus producing the variable and slight superior oblique weakness. Obviously the swelling cannot be restricted in both places simultaneously, so the symptoms are of either one type or the other. The mechanism of the clicking produced as the swelling crosses the entrance of the fibrous sheath at the trochlea is obvious.

In the author's case it is likely that the swelling of the tendon is associated with chronic nodular rheumatoid arthritis, but definite proof of this is unfortunately not available. It might be argued from Case 4, in which the tendon and sheath were explored and the sheath found to be thickened and the tendon normal, that there is no evidence for this hypothesis. However, according to the hypothesis, the swelling should be found on the orbital side of the trochlea and in this case the tendon and sheath were explored only on the ocular side of the trochlea. The thickening of the sheath near the trochlea, however, is of considerable significance (see below).

Although the superior oblique tendon is unique amongst the tendons of the extraocular muscles in that it sharply changes its direction of pull, a similar situation is quite common in other parts of the body, especially in the tendons in the hand. Indeed, whenever a tendon changes its direction, there must be some fibrous or osseous canal to hold it in place, and important evidence comes from considering the pathology of some of the tendon sheath complexes in the hand, where the anatomy of the tendon and the sheath is similar to that of the superior oblique tendon and sheath. The long flexor tendons of the fingers enter a fibrous tendon sheath at the heads of the metacarpal bones and with the fingers in flexion make a right-angled bend as they enter the sheath. The flexor pollicis longus tendon to the thumb enters a fibrous sheath at the head of the first metacarpal bone and bends similarly on flexion of the thumb.

In both these situations there is a very common pathological condition consisting of a swelling of the tendon at the mouth of the fibrous sheath associated with hypertrophy of the proximal end of the sheath. The conditions are grouped together as stenosing tenovaginitis or tenosynovitis and known respectively as trigger or snapping finger and trigger or snapping thumb. The clinical picture of these conditions matches precisely the clinical picture described in this paper as regards the superior oblique (Apley, 1968):

- (1) There is resistance to extension of the fingers (thumb), analogous to the resistance of the superior oblique to being passively stretched (producing restriction of elevation of the eye in adduction), and caused by the inability of the swelling in the tendon to enter the thickened fibrous sheath. This resistance can often be overcome by vigorous extension of the fingers, and in Cases 1 and 2, described above, this resistance was similarly overcome by vigorous movement of the eyes.
- (2) There is often a slight difficulty of flexion in the fingers (thumb), analogous to the superior oblique weakness demonstrated and caused by the slight resistance of the swelling in the tendon which had entered the sheath, to sliding out of the sheath again.
- (3) There is a snapping or clicking in the finger (thumb) analogous to that in the eye and caused by restriction of free movement of the tendon within the sheath with sudden release on overcoming the restriction.

It is interesting too that stenosing tenovaginitis is most common in young adults, which would agree with the age distribution of the series of five cases collected here. There is a further point of considerable interest, namely that the condition of stenosing tenovaginitis occasionally presents in early childhood especially in the thumb; and in these instances the deformity is often more marked with total inability to extend the thumb. It may well be that many of the typical congenital cases of the superior oblique tendon sheath syndrome are, in fact, similar to this condition in the thumb, with a swelling of the tendon behind the trochlea and with a thickening of the tendon sheath at its proximal (*i.e.* trochlear) end. The latter has frequently been recorded as an operative finding and the former may well not have been found owing to the difficulty of exploring the superior oblique tendon beyond the trochlea. There is indirect evidence for this in that there have been reports in the literature of typical cases of the superior oblique sheath syndrome in young children which have spontaneously recovered (Costenbader and Albert, 1958; Adler, 1959). This is, of course, inexplicable by any of the other theories of the aetiology of the tendon sheath syndrome.

For these reasons it is suggested that a proportion of cases of the superior oblique tendon sheath syndrome are caused by a mechanism identical to the common orthopaedic con-

dition of stenosing tenovaginitis, *i.e.* a swelling of the tendon of the superior oblique associated with hypertrophy of the fibrous tendon sheath near the trochlea.

The characteristic features of such cases are:

- (1) An intermittent picture of the superior oblique sheath syndrome which may become permanent.
- (2) A clicking, snapping, or pulling sensation over the trochlea.
- (3) An intermittent, variable, and slight weakness of the superior oblique which would be seen consecutively, but not simultaneously, with the sheath syndrome.

An individual case need not demonstrate all three of these features, but their presence is strong evidence that stenosing tenovaginitis is the underlying cause of the condition. It is also possible that many straightforward cases of the superior oblique tendon sheath syndrome presenting in children are caused by this condition.

Summary

A case of "acquired intermittent superior oblique tendon sheath syndrome" associated with consecutive superior oblique weakness and intermittent clicking is described. Similar cases in the literature are reviewed and the underlying cause discussed. There is strong evidence for considering this syndrome to be caused by a localized swelling of the superior oblique tendon and hypertrophy and constriction of the tendon sheath. The similarity of this condition to stenosing tenovaginitis is shown and it is suggested that it is also a cause of the more common type of superior oblique sheath syndrome presenting in children.

I wish to thank the Orthoptic Department of the Bristol Eye Hospital and Mr. N. L. Dallas for permission to publish details of a case under his care.

References

- ADLER, F. H. (1959) *A.M.A. Arch. Ophthalm.*, **61**, 1006
- APLEY, A. G. (1968) "A System of Orthopaedics and Fractures", 3rd ed., p. 198. Butterworths, London
- BROWN, H. W. (1950) In "Strabismus Ophthalmic Symposium (1)", ed. J. H. Allen, p. 205. Mosby, St. Louis
- (1957) *Trans. Amer. ophthalm. Soc.* **55**, 415
- CLARK, E. (1966) *Brit. orthopt. J.*, **23**, 116
- COSTENBADER, F. D., and ALBERT, D. G. (1958) *A.M.A. Arch. Ophthalm.*, **59**, 607
- ESTERLY, H. D., NADBATH, R. P., and RUSSELL, J. S. (1960) *Ibid.*, **63**, 997
- GIRARD, L. J. (1956) *Sth. med. J.*, **49**, 342
- MEIN, J. (1964) *Brit. orthopt. J.*, **21**, 116
- MILLS, P. V., and COATE, M. A. (1967) *Ibid.*, **24**, 132
- NUTT, A. B. (1955) *Trans. roy. Coll. Surg. Engl.*, **16**, 30