Short report

Recognising aponeurotic ptosis

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SUMMARY Thirteen patients who had ptosis surgery undertaken for disinsertion of the aponeurosis of the levator palpebrae superioris were reviewed. Pre-operatively all the patients had characteristic clinical signs of levator disinsertion which was confirmed at surgery and corrected by reposition of the disinserted aponeurosis. Five of these patients were initially mistakenly diagnosed as having a neurological cause for their ptosis. The diagnosis of this type of ptosis may easily be missed.

Aponeurotic ptosis is a form of acquired ptosis which is characterised by a dehiscence or disinsertion of the levator aponeurosis.¹ A recent study showed that 90% of patients with involutional or senile ptosis had an aponeurotic weakness and 10% had a normal appearing aponeurosis and marked degeneration of the anterior portion of the levator muscle.² The main features of a pure aponeurotic disinsertion^{2,3} are ptosis with a good levator excursion, a raised or absent upper lid skin crease and a thinning of the upper lid above the tarsal plate [fig 1, a, b]. A history of increasing ptosis towards the end of the day is a common finding with levator disinsertion. This reflects fatigue of Muller's muscle and is not by itself diagnostic of myasthenia gravis. Aponeurotic ptosis can also occur after trauma, ocular surgery and eyelid oedema.¹⁻⁶ Unlike ocular myopathy and myasthenia, external eye movements are not affected. The pupillary reactions are normal. Recognition of the characteristic clinical findings should enable the correct diagnosis to be made and further neurological investigation is unnecessary. Most patients with aponeurotic ptosis are asymptomatic with a mild degree of ptosis and do not require surgery. If the degree of ptosis is severe enough to disturb vision or is a cosmetic embarrassment, it can be corrected¹ by repositioning the disinserted or dehiscent levator aponeurosis onto the anterior surface of the tarsal plate (fig 1, c).

Patients

Thirteen consecutive patients underwent ptosis correction for disinsertion of the levator aponeurosis between November 1985 and June 1988. All 13 patients had typical clinical features of levator disinsertion before surgery; disinsertion was confirmed at operation, and the ptosis was corrected by repositioning the levator aponeurosis onto the anterior surface of the tarsal plate. Five of these patients, who were initially diagnosed as having a neurological cause for their ptosis, are described.

Case No 1

A 61 year old female was referred in 1979 to an ophthalmologist with a 5 year history of gradual drooping of the right upper lid. A neurological opinion was sought because the tensilon test was thought to be positive. She was admitted for investigation. The right pupil was noted to be slightly smaller but both pupillary reactions were normal. The tensilon test was repeated and was negative. A chest and radiograph CT were normal. An old photograph showed that the ptosis had been present for at least 7 years. An initial diagnosis of right Horner's syndrome of undetermined cause was made. This was subsequently revised and, because the patient felt that the tensilon injection had produced some improvement in her ptosis, she was given a therapeutic trial of pyridostigmine. This was continued and she was discharged on this medication. Six years later she was referred for consideration of right ptosis surgery. The ptosis, which had remained unchanged, showed typical features of levator disinsertion. The following year ptosis surgery was performed. The levator aponeurosis was found to be disinserted, and was repositioned with a good result. Her medication was discontinued, with no recurrence of her symptoms.

Case No 2

A 62 year old male was seen by an ophthalmologist in 1982 with a history of drooping of the left and right upper eyelid for 1 and 3 years respectively. There was no history of

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Fig 1 (a) Bilateral aponeurotic ptosis before surgery [case no. 4]. (b) Left eye of the same patient demonstrating typical features of an aponeurotic ptosis, with a raised skin crease and thinning of the upper lid above the tarsal plate. (c) The same patient after bilateral surgery for aponeurotic ptosis.

diplopia or diurnal variation in the ptosis. A bilateral ptosis and slight restriction of elevation of the left eye were noted. He was referred for neurological assessment to exclude myasthenia or ocular myopathy and was admitted for a tensilon test. This was negative, but there was a subjective improvement in the ptosis and ocular myasthenia was diagnosed. He was discharged on pyridostigmine and atropine. Two years later the ptosis had become more pronounced, but any increase in medication tended to produce unacceptable side-effects. He was then referred for consideration for ptosis surgery. In January 1985 he was still taking pyridostigmine and atropine. On examination he had bilateral ptosis, frontalis overaction, reasonable levator action and a raised skin crease. Ocular movements were full. Bilateral repositioning of the disinserted levator aponeuroses was carried out with a very good outcome.

Case no 3

This patient had a history of depression and childhood migraine. In 1977, when she was aged 61 years, she was noted by a psychiatrist to have a mild right ptosis, with apparent weakness of up-gaze and medial gaze of the right eye. The right pupil was slightly larger than the left, but both pupils reacted briskly and equally to light and accommodation. She would not allow fundal examination because of intense photophobia. She was referred for neurological investigation. Right carotid angiography was advised to differentiate between ophthalmoplegic migraine and a posterior communicating artery aneurysm, as a cause of a third nerve

palsy, but the patient refused further investigation. In 1984 she was receiving psychiatric care and was reviewed again by a neurologist. CT was normal and further investigations were considered unnecessary in view of her age and unchanging clinical condition. In 1986 the right ptosis was described as complete and an ophthalmic opinion was sought. On examination, there was no diplopia when the ptotic lid was raised. A right latent divergent strabismus was present, but no evidence of a third nerve palsy. Her intense photophobia was considered to be functional in origin. The right upper eyelid skin crease was markedly raised. In February 1988 surgical exploration revealed disinsertion of the levator aponeurosis. The levator was repositioned and a very satisfactory result obtained.

Case no 4

This patient was referred for neurological assessment of her bilateral ptosis in 1965, when she was aged 52 years. A tensilon test was negative and progressive ocular myopathy was diagnosed. In 1980 she was admitted for reassessment and again the tensilon test was negative; electromyographic studies revealed no evidence of myopathy, and nerve conduction studies were normal apart from a suggestion of right ulnar sensory neuropathy. Ocular myopathy was considered to be the most likely diagnosis. In 1985 her ptosis had increased and she was referred for consideration of surgery. She had a severe bilateral ptosis which forced her to walk with her chin raised. Typical features of a levator disinsertion were present [fig 1a, b] and ocular movements were full. In

March 1988 at surgery the right levator was found to be disinserted and it was repositioned. Two months later the same procedure was carried out on the left side [fig 1c]. The patient was delighted with the surgical outcome.

Case no 5

This female patient aged 69 years had a history of a drooping right eye for 6 years and was referred by an ophthalmologist in 1986 for neurological evaluation. A tensilon test was negative, although there was some subjective improvement in the ptosis. CT showed no abnormality. In view of the subjective improvement with edrophonium, ocular myasthenia was considered a likely diagnosis and she was given a therapeutic trial with pyridostigmine. She remained on treatment for 6 months. One year later she was referred to a second ophthalmologist who found clinical evidence of levator disinsertion, which was confirmed at surgery. A very good post-operative result was achieved.

Discussion

The importance of reaching the correct diagnosis in cases of levator disinsertion is illustrated by the five case histories above. In particular, in case no 4, the patient was labelled as having ocular myopathy for over 20 years before her symptoms were effectively cured by ptosis correction. It is also important to differentiate levator disinsertion from other causes of unilateral ptosis, in particular, Horner's syndrome and a third nerve palsy. The recognition of the characteristic features of aponeurotic ptosis will save the patient unnecessary investigations such as CT or carotid angiography. A history of ocular surgery, lid oedema or trauma to the upper lid may support the diagnosis, but in the majority of cases the cause of the

ptosis is involutional and there is no such history. Although this paper reports on patients who have come to surgery, it is important to emphasise that, as already mentioned, most patients with aponeurotic ptosis do not require surgery. Before referral for neurological investigation, the ophthalmologist has a responsibility to ensure that a local cause for ptosis has been excluded. A subjective improvement in the ptosis after a tensilon test is not an indication that the patient has myasthenia, unless there is an accompanying objective, measurable decrease in the ptosis.

Ten per cent of involutional ptoses were found in one study to be due to fatty degeneration of the anterior levator muscle.² These patients tended to have a bulky eyelid, a normal skin crease and good levator function. None of the 13 patients we have studied fell into this group. Ptosis surgery can be carried out on these patients if the ptosis proves to be troublesome.

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