Recurrent ptosis

In this Journal, Sieb and Hartmann described two patients with intermittent and alternating ptosis [1]. Intermittent sympathetic dysfunction causing a "partial" Horner's syndrome was suggested as the underlying pathogenic feature.

The photograph of the patients eye lid shows a complete ptosis which is not seen in Horner's syndrome because Müller's muscle contributes only to about 1.5 mm lid elevation [2]. More than sympathetic dysfunction alone is needed to cause the presented ptosis.

We recently reported a similar case in a 41 year old woman demonstrating involvement of both Müller's muscle and levator palpebrae superioris clinically and pharmacologically [3]. Orbital imaging showed enlargment of the levator palpebrae/rectus superior complex which also suggests a local pathology. We proposed a local, possibly inflammatory process of the lid surface anatomy as described by Rice and Gray [4]. A similar explanation might account for the mild aching at the frontal region of the affected side in Sieb and Hartmann's patient.

More recently a 62 year old man presented to our clinic with a 3 year history of recurrent right complete ptosis lasting 7-10 days duration, occurring once or twice a year with full recovery. The onset of the ptosis was associated with erythema and mild periorbital aching and swelling.

Unfortunately we have not yet been able to find an appropriate treatment for this condition. An initial trial with pyridostgmine using the rationale that 15-20% of patients with myasthenia gravis have negative acetyl choline antibody [5] was disappointing [2], as was treatment with non-steroidal anti inflammatory drugs and oral prednisolone [1,2]. Sieb and Hartmann tried the serotonin antagonist pizotifen and

prednisolone also without significant improvement.

We do not know the reason for the recurrent complete ptosis in our two patients. Neither can we be sure that the siblings described by Sieb and Hartmann have the same disorder, particularly as in these cases the side of the ptosis alternated [1]. However we suggest that the syndrome must be due to local disease causing loss of function of the levator palpebrae superioris muscle either alone or in addition to Müller's muscle.

A Petzold, GT Plant

National Hospital for Neurology and Neurosurgery

Queen Square

London WC1 N3BG

References

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