EDITORIAL

Thyroid Eye Disease

Thyroid eye disease is an enigma; the cause is unknown and the treatment empirical. If double vision occurs it can usually be successfully managed surgically when the disease process is inactive, but this may mean waiting many months and effective treatment in the interim would therefore be welcome. The paper by Lyons and colleagues in this issue addresses this problem, and raises two interesting questions.

The first concerns the pathological changes in the eye muscles that give rise to the characteristic muscle swelling, restriction of movement and strabismus. Does the substantial reduction in squint angle that occurred after three quarters of the botulinum toxin injections imply, as the authors suggest, that an element of reversible inflammatory muscle spasm is contributing to the strabismus in the early stages of the disease? Although an inflammatory component can be prominent early in the natural history of the disease,[1] extra-ocular muscle spasm due to local factors is not normally an accepted concept. It certainly does not appear to occur in other orbital inflammatory conditions such as idiopathic myositis, when the involved muscles are under-active. However, the best explanation of Simonsz and Kommerell's work, on the length–tension characteristics of dysthyroid muscle (albeit only 3 patients) is that active contraction is contributing to the muscle stiffness.[2] The effect of botulinum is further evidence to support the hypothesis. Why such contraction should occur is another matter. It would be interesting to know if the authors found any EMG abnormalities in the course of the localisation of the injection indicative of abnormal muscle activity. Their suggestion that it may be due to a change in muscle fibre type is not convincing since this change has been described only in chemically induced thyrotoxicosis in experimental animals. Not all patients with thyroid eye disease are thyrotoxic, and it is well recognised that thyroid orbitopathy may occur many years after the patient has suffered from hyper-thyroidism, and also before its onset. The spasm must therefore be a local secondary response to changes in the muscle produced by the ill-understood primary immunological disturbance. It is possible, for example, that lymphocytic infiltration of the muscles alters muscle spindle sensitivity characteristics which could generate inappropriate motoneuron activity.

The second question is whether botulinum toxin treatment is symptomatically beneficial in the active phase of the disease when surgery is contra-indicated. The evidence here is not conclusive. Although 75% of the injections reduced the squint by a mean of 14 prism dioptres (75% of the initial angle, with over-correction in nine out of 48 cases or nearly 20%) the effect of the toxin was undetectable by two months, so the patients must have gone through a period of relatively rapid return to *status quo* during this period. On this basis, despite the safety and ease of the procedure, it would be difficult to recommend. However, an interesting point emerges; unless combined with surgery, in most circumstances realignment of the visual axes with botulinum is temporary.[3] An unexpected long-term benefit was evident in six of the treated cases in this series. Rarely, reduction or abolition of strabismus can occur spontaneously in Graves' disease because of the development of symmetrical involvement of the eye muscles. This was seen in one of the six cases, and may have contributed, with improvement of fusional control, in others. Another possible explanation is that, as a result of the injection, contracture occurred preferentially in the ipsilateral antagonist (in most cases the superior rectus) during the period of agonist palsy. By redistributing the impact of contracture, the treatment may have contributed to a long-term result. It does not appear to be possible to predict a successful outcome, but the treatment can be offered to symptomatic patients on the basis of a small—10–15%—chance of permanent improvement.

This paper provides some interesting and important insights into thyroid eye disease. It confirms the impression that botulinum toxin treatment is not simply an alternative to strabismus surgery, as was originally argued, but a different form of treatment with different indications.[4] Unfortunately, under most circumstances the thyroid patient is probably best advised to wait for the strabismus to stabilise when definitive surgery can be carried out.

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References

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