ORIGINAL ARTICLE

Steroids in the Management of Synkinesis after Traumatic Oculomotor Palsy in India

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ABSTRACT

Traumatic oculomotor nerve palsies may result in aberrant regeneration and synkinesis that can impair normal facial function. We retrospectively investigated 16 patients with traumatic oculomotor nerve palsies who were treated with and without steroids to evaluate the rates of aberrant regeneration. The overall rates of aberrant regeneration were similar between the two groups and in line with other published series. Some limitations to our study may limit our ability to detect real differences, and additional studies would be beneficial.

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Introduction

Traumatic oculomotor nerve (CNIII) injury has been reported to occur in up to 0.03% of all head injuries, but represent 2.3% of all traumatic cranial nerve injuries in India.¹ Direct trauma is rare, and compression, e.g., from a subdural hematoma or extradural hematoma, is more common.² The exact site of injury to the nerve is up for debate, but has been described to by Walsh and Hoyt to occur at the point where the oculomotor nerve pierces the dura to enter the cavernous sinus, or fractured bone fragments may lacerate the nerve.³ Others have described it in the subarachnoid space coursing between the brainstem and the cavernous sinus.^{4,5} Traumatic CNIII injuries may result in aberrant regeneration or synkinesis. Synkinesis may also occur from compressive lesions, but it is rare after ischemic CNIII palsies. In this retrospective review, we investigate the effect of steroids on synkinesis after traumatic CNIII palsy.

Materials and methods

This was a retrospective chart review of 15 patients with traumatic CNIII palsy after road traffic accident evaluated between January 2016 and January 2017. All patients were seen at the outpatient department in Aravind Eye Hospital in

Coimbatore, India, between 1 week and 11 years after injury. Patients were offered steroids after review of neuroimaging and if no contraindications per neurology or neurosurgery. Presence of ptosis and subjective diplopia were documented, as was the degree of CNIII palsy (complete or incomplete) and the presence of pupil involvement. Treatments offered included physiotherapy (teaching patient to move the eye in the direction of the palsy) steroids (intravenous and/or oral) and squint surgery. Finally, the degree of recovery and development of aberrant regeneration were also documented.

Results

Patients ranged in age from 23 to 65 years with an average and mean age of 43 years, and 11 of the 15 patients were male. All patients were involved in road traffic accidents, and all but two had loss of consciousness. Neuroimaging was done for all but one patient. Of the 15 patients, eight had computed tomography (CT) imaging for review. Of these eight patients with imaging, three had skull fractures involving the temporo-parietal bone, one involving the facial bones and one involving the skull base. The other three patients with CT imaging had no fractures, but one of these had a

Table 1. Summary of patients.

Patient number	Age (y)	Sex	Eye	Complete/incomplete	Computed tomography	Treatment	F/U time (m)	Aberrant regeneration
1	28	Male	OS	Complete	Skull base, R temporal bone fx	Physiotherapy	3	No
2	23	Male	OD	Complete	Facial bone fx	Steroid/physiotherapy	5	Yes
3	40	Male	OD	Incomplete	N/A	Squint correction	17	Yes
4	57	Male	OD	Incomplete	Normal	Steroid/physiotherapy	0.25	No
5	58	Male	OD	Incomplete	N/A	Physiotherapy	24	Yes
6	65	Male	OS	Incomplete	N/A	Physiotherapy	15	Yes
7	43	Female	OD	Incomplete	SAH	Physiotherapy	1	No
8	26	Male	OS	Incomplete	N/A	Steroid/physiotherapy	6	Yes
9	43	Female	OS	Complete	R parietal bone fx, SAH	Steroid/physiotherapy	1	No
10	44	Male	OD	Incomplete	R Temporoparietal bone fx	Physiotherapy	132	Yes
11	42	Female	OS	Incomplete	N/A	Physiotherapy	4	Yes
12	37	Female	OD	Incomplete	Normal	Steroid/physiotherapy	5	Yes
13	49	Male	OS	Complete	Subdural haemorrhage	Physiotherapy	1.5	No
14	28	Male	OD	Complete	N/A	Steroid	2	Yes
15	45	Male	OD	Complete	N/A	Steroid	4	Yes

OS = left eye, OD = right eye, F/U = follow-up, fx = fracture, SAH = subarachnoid haemorrhage.

subdural haemorrhage. There was no clinical or radiographic evidence of uncal herniation or Kernohan–Woltman notch phenomenon in any patient.

At presentation, 12 patients had ptosis, 11 had diplopia, and six had complete CNIII palsies. Seven patients received steroid therapy: four intravenous steroids from outside institutions (details unknown), and two oral prednisolone 40 mg daily for 5 days followed by taper of 10 mg every 5 days. In four cases, steroids were initiated within 3 months of injury. All but four patients also received physiotherapy. Only one patient had squint surgery, and this patient did not receive steroids or physiotherapy. Patients were followed for between 1 week and 11 years. Recovery in all patients was partial. Aberrant regeneration was present in 10 patients. Of the seven patients who received steroids, five developed aberrant regeneration. Of the eight who did not receive steroids, five developed aberrant regeneration. As a result, in our series, we found an aberrant regeneration rate of 71% with steroids and 63% without it. See [Table 1](#) for summary.

Discussion

Aberrant regeneration, or synkinesis, refers to misdirection of nerve fibre growth after insult or injury.

Common patterns include lid elevation during adduction or depression of the eye (pseudo-Graefe phenomenon) or depression and miosis of a pupil unreactive to light during adduction.^{6,7} Aberrant regeneration after trauma to CNIII is likely related to damage to the endoneurium that permits the regenerating nerve to follow an aberrant course outside its protective sheath.⁸

Aberrant regeneration can be seen in over half of patients with traumatic CNIII palsies,⁹ consistent with our series in which it was found in 10/15 or 67% of patients. The time to discovery of aberrant regeneration, however, varies from 36 days to more than 39 years, depending on when patients were followed. If followed by an ophthalmologist within 6 months of injury, the mean time to first appearance of aberrant regeneration was 116 days, while if followed at 1 year, it was 247 days.⁸ In our series of patients who received steroids, the aberrant regeneration rate was 71%, while it was 63% in the patients who did not receive steroids.

There is a dearth of data on the effects of steroids on aberrant regeneration after traumatic CNIII palsy. In recent years, the reluctance to use steroids could be due to the results from the CRASH study which showed no reduction in mortality with methylprednisolone in the 2 weeks after head injury, but an increased risk of death within 2 weeks.¹⁰ As a result, steroids are often not given

to patients with traumatic brain injuries with Glasgow coma score of 14 or less, which may often be the case in many patients with traumatic cranial nerve palsies. However, in patients with stable and reassuring neurological findings, the use of steroids has been described after traumatic CNIII,¹¹ traumatic abducens palsy,¹² and traumatic facial nerve palsy,¹ all generally with good results. Steroid use has also been described after presumed inflammatory CNIII palsy secondary to norovirus.¹³ Probably the best model for steroids in peripheral nerve injury is after Bell's palsy,¹⁴ though this is believed to be an inflammatory nerve injury rather than traumatic.

Unfortunately, this series did not find any benefit to using steroids to prevent aberrant regeneration, and in fact, the steroid group had a higher aberrant regeneration rate than did the non-steroid group. One postulated reason for our findings is that while the steroids may reduce inflammation of a traumatized nerve, once there is trauma to it, steroids have no effect on subsequent aberrant regeneration itself since that process is not an inflammatory one. However, other studies have demonstrated that, if given early enough, the steroids may limit the degree of inflammation and secondary damage to the endoneurium, and thus reduce the rate of aberrant regeneration.^{11–13}

Limitations of our study include its retrospective nature, small number of patients, variable or lacking steroid dosing regimens and timing, variable follow-up, and oculomotor measurements done without prism diopters. This study was not powered for statistical calculations, so no statement about statistical significance can be made. Further studies may be beneficial to better understand the role of steroids in management of traumatic oculomotor nerve palsies.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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