Clin Oral Invest (2003) 7:52–55
DOI 10.1007/s00784-002-0187-y

ORIGINAL ARTICLE

Christoph M. Ziegler · C. Haag · J. Mühling

Treatment of recurrent temporomandibular joint dislocation with intramuscular botulinum toxin injection

Received: 18 March 2002 / Accepted: 22 October 2002 / Published online: 25 January 2003

Abstract Recurrent dislocation of the mandibular condyle poses a difficult problem for affected patients. In the course of time, dislocations often become more frequent and more difficult to avoid. Even with good patient compliance, conservative treatment is often not sufficient. Operative procedures have also been described for the treatment of temporomandibular joint dislocation. However, these interventions are invasive, involving open arthrotomy with possible complications, and cannot safely guarantee a successful outcome. On the other hand, botulinum toxin injections into the lateral pterygoid muscles offer the option of a predictable and prolonged period without renewed dislocation. We present the results of this treatment carried out in 21 patients with recurrent temporomandibular joint dislocation. Four patients were treated following unsuccessful physical therapy and the use of occlusal splints. The remaining 17 patients were treated for a number of conditions resulting in dislocation, including some with senile dementia and mental impairment in whom compliance with conservative measures was poor or completely absent. Injections were given on a 3-month basis in order to have a sustained effect. Within the study period of 6 months to 3 years, only two of the 21 patients suffered further dislocation. There were no side effects recorded as a result of treatment.

Keywords Botulinum toxin · Electromyography control · Nonoperative therapy · Temporomandibular joint dislocation

C.M. Ziegler (✉) · C. Haag · J. Mühling
Department of Oral and Maxillofacial Surgery,
University of Heidelberg,
Im Neuenheimer Feld 400, 69120 Heidelberg, Germany
e-mail: cmziegler@t-online.de
Tel.: +49-6221-419213, Fax: +49-6221-419256

Introduction

Temporomandibular joint dislocation can be seen in several different conditions which include neurogenic muscular hyperactivity [4, 12, 13], pathological osseous conditions affecting the articular eminence, and connective tissue disorders such as Ehlers-Danlos disease. Patients are often unable to reduce the frequency of dislocations by themselves. Conservative treatment includes the use of physical therapy, muscle relaxants, and occlusal splints. In addition, patients are instructed to avoid wide mouth opening. However, these measures are sometimes inadequate for preventing further joint dislocations. Furthermore, conservative measures are sometimes impossible due to the lack of patient compliance resulting from mental impairment. In the past, surgical intervention was the only treatment available for these individuals [12]. In principle, operative techniques involve either an augmentation of the height of the articular eminence to prevent dislocations at all or a reduction in the height of the eminence to allow spontaneous repositioning of the condyle. A further surgical option is an extra-articular bridle suture of the temporomandibular joint ligaments to reduce the range of joint movement.

All interventions bear the risk of complications due to their invasive nature and the need of general anesthesia. Furthermore, even surgical interventions cannot totally guarantee further dislocations without reposition [7]. Treatment with botulinum toxin offers the option of minimal invasive therapy under outpatient conditions. Compared to surgical procedures, it shows comparable efficacy but very few side effects [12].

Botulinum toxin is the product of the anaerobic bacterium *Clostridium botulinum* and has one of the highest biological toxicities known. This toxin prevents the release of acetylcholine at neuronal synapses. The resulting neuromuscular blockade causes paralysis of skeletal musculature and smooth muscles supplied by the parasympathetic system. Hypohydrosis or anhydrosis results from the blockade of cholinergic sympathetic nerves. Botulinum toxin is not cytotoxic and becomes inactivat-

ed after about 3 months as a result of intraneural proteolysis and consequently restored nerve function. Persistent morphological changes and muscle atrophy resulting from treatment with botulinum toxin have not been reported [9].

Potential contraindications for treatment with botulinum toxin include neurological conditions affecting the motor endplate such as myasthenia gravis or Eaton-Lambert syndrome. Anticoagulant therapy is also a contraindication for botulinum toxin injections due to its requirement of intramuscular injection. Due to passage across the placenta, the use during pregnancy is also contraindicated [9, 12]. Furthermore, botulinum toxin should not be used in case of pre-existing dysphagia, because it may exacerbate the symptoms. Simultaneous application of muscle relaxants or antibiotics of the aminoglycoside type should also be avoided [9, 12].

Although botulinum toxin is the active agent of botulism, intoxication after medical application has never been recorded. About 3–10% of patients are refractory to therapy due to the presence of neutralizing antibodies against the toxin. The frequent administration of botulinum toxin in high doses can cause increased resistance against the toxin [1, 9]. Of the seven serotypes (A to G), only type A has been studied and applied in medical therapy. Commercially available preparations are Dysport (Ipsen Pharma, Ettlingen, Germany) and Botox (Allergan, USA, distributed in Germany by Merz). Clinical applications of botulinum toxin type A include dystonias, spastic and autonomic disorders, and tremor disorders [9]. In the field of maxillofacial surgery, the use of botulinum toxin has been associated with the treatment of oromandibular dyskinesia, myofascial pain dysfunction syndrome, and deinnervation of the facial muscles in cosmetic corrections [2, 10, 11, 13, 14, 15]. In addition, botulinum toxin has been injected into the lateral pterygoid muscles bilaterally to reduce muscular activity to prevent recurrent temporomandibular joint dislocations [3, 5, 8].

The purpose of our botulinum toxin applications in patients with recurrent dislocations of the temporomandibular joint was to reduce the frequency of luxations to a maximum of one dislocation per month. The group of interest was comprised especially of patients with insufficient compliance to conservative treatment and at high risk for surgical interventions. Therefore, it was also important to avoid severe side effects. As joint dislocations are in principle connected with pain, a reduction of complaints should occur simultaneously with reduced luxation frequency. Whenever possible in our patient sample, we attempted to measure the degree of pain relief after treatment.

Material and method

Between March 1998 and December 2001, 21 patients (23 to 91 years old) were treated with bilateral botulinum toxin A injections (Dysport) into the lateral pterygoid muscles (Fig. 1). The injections were repeated at 3-month intervals. The indication for treatment with botulinum toxin was mandibular dislocation four times in the preceding 2 months or twice in the preceding 4 weeks. The diagnosis of recurrent dislocation was confirmed by the referring physician and at least two maxillofacial surgeons in our department. It was based on patient history (inability of the patient to close the mouth due to a locked jaw) and additional findings such as a palpable dislocation of the condyle from the fossa, tenderness of the joint capsule and the masticatory muscles, and occlusal disturbance.

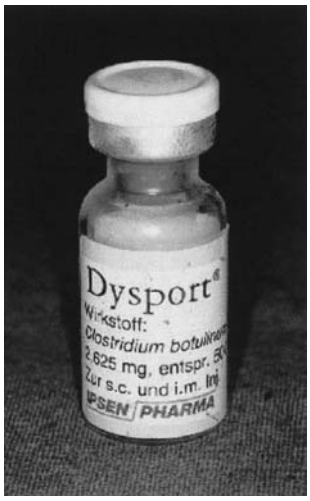

Fig. 1 Botulinum toxin A. (Dysport) (Ipsen Pharma, Ettlingen, Germany)

Radiographic examination (pantomogram) was possible in 16 of the 21 patients. In the other five cases, radiography was not feasible due to the lack of patient compliance. Additional inclusion criteria were failure of conservative therapy and patients in whom compliance to conservative therapy was not possible. Compliance lacked in 17 of the 21 patients suffering from organic psychosis (especially presbyophrenia) and other diseases connected with mental retardation such as juvenile cerebral sclerosis, Hallervorden-Spatz disease, and crying cat syndrome. In these cases, informed consent was given by the legal guardians after informing the patients about the risks inherent to the local application of botulinum toxin. The botulinum toxin was administered with no additional treatment. Four patients with habitual dislocation filled out a visual analog scale (VAS) from 0 (no pain) to 10 (most imaginable pain) before and 1 week after the treatment to measure reductions in pain intensity.

Injections were performed with a Teflon-coated needle (lumen electrode 50×0.45 mm) and an electromyographic (EMG) controlling device (Keypoint version 2.12, Medtronic, Düsseldorf, Germany) during the procedure (Fig. 2). The ground was located in the cervical region. After skin disinfection, bilateral intramuscular injections of 50–100 mouse units (MU) Dysport were performed into the lateral pterygoid muscles (Table 1). If possible, patients

Table 1 Characteristics of the patients included in this study. *MU* mouse units

Patient	Sex	Age	Disease	Duration of treatment	Injection interval (*n* injections)	Dosage (MU Dysport)
B.H.	F	34	Crying cat syndrome	8/98–1/00	3–6 months (4)	50–75
P.S.	F	30	Crying cat syndrome	12/98–12/99	3 months (4)	75
S.T.	M	36	Hallervorden-Spatz syndrome	7/99–9/99	2 months	75
A.M.	F	34	Habitual dislocation	5/99–8/99	3 months (2)	75
C.W.	F	42	Habitual dislocation	3/98–9/98	3 months (3)	75–100
S.M.	F	26	Juvenile cerebral sclerosis	4/98–10/99	3–6 months (5)	50–75
S.R.	M	23	Juvenile cerebral sclerosis	1/99–5/00	3–4 months (5)	50–75
S.A.	M	24	Juvenile cerebral sclerosis	1/00–5/01	3–4 months (5)	50–75
A.M.	F	44	Habitual dislocation	4/01–11/01	3 months (2)	75
C.S.	M	53	Habitual dislocation	5/00–12/01	3–4 months (5)	50–75
M.S.	F	77	Organic psychosis	2/01–10/01	3–5 months (2)	50–100
H.D.	F	79	Organic psychosis	10/00–10/01	3 months (4)	75
D.E.	M	83	Organic psychosis	6/01–11/01	2–3 months (2)	75
S.W.	M	89	Organic psychosis	4/98–7/99	3 months (2)	75–100
M.N.	F	73	Organic psychosis	7/99–1/00	3 months (2)	75
E.R.	M	81	Organic psychosis	5/98–12/99	3–4 months (5)	50–75
M.S.	F	82	Organic psychosis	10/98–2/00	3–4 months (5)	75
W.N.	F	91	Organic psychosis	6/98–11/98	2–3 months (2)	50–75
E.W.	F	86	Organic psychosis	5/99–3/00	3 months	75
B.N.	F	89	Organic psychosis	8/99–4/00	3 months (2)	75
E.M.	F	78	Organic psychosis	2/99–4/00	3–6 months (3)	50–100

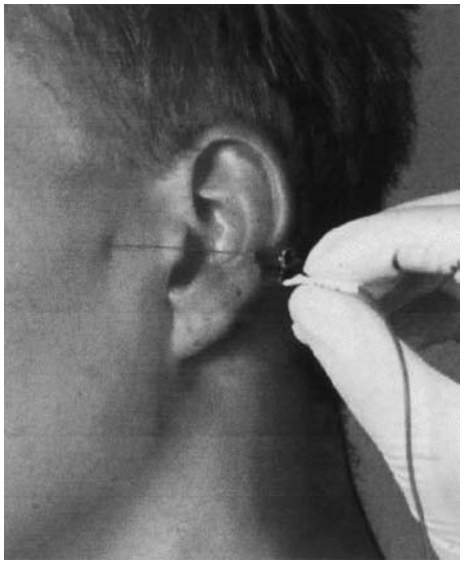

Fig. 2 EMG-controlled application device with lumen electrode

had to open their mouths slightly during the injections. The injection needles were percutaneously pushed forward through the incisura semilunaris dorsocranially of the processus pterygoideus. The correct intramuscular position was controlled by EMG under active lateral jaw movements to the contralateral side without occlusal contact.

For outcome measurement, the occurrence of dislocations before and during therapy as well as of potential side effects were recorded by the patients or their guardians using a luxation protocol. Reinvestigations (clinical examination) took place after 1 week, 3 and 6 months, and 1 year. To avoid further radiation exposure, radiographs were not taken.

Results

No patients sustained dysphagia or a significant reduction in masticatory function after the treatment with botulinum toxin. Other possible side effects such as allergic reactions or resistance against the toxin were also not recorded. In spite of the often reduced patient compliance, no complications such as bleeding, nerve damage, or iatrogenic injuries of neighbouring anatomic structures happened. In eight patients, EMG monitoring was not possible due to reduced cooperation resulting from mental impairment. In the other cases, the correct position of the injection needle could be ascertained via the EMG pattern during jaw movement.

The treatment lasted from 6 to 18 months, in which five patients each received a total of five bilateral injections. The dosages included 50 MU to 100 MU of toxin per side, in which 50 MU were given as repeated injections and 100 MU only in severe cases with several luxations per week. Under this regimen, 19 of the 21 patients showed no further dislocations for at least 8 months after the final injection. Four patients had single dislocations nine, 11, 14, and 17 months after the last botulinum toxin application.

Two patients showed further dislocations during the treatment with botulinum toxin. One of them suffered from crying cat syndrome and sustained a further dislocation 2 months after the second treatment. The other one, diagnosed with organic psychosis, had a single further dislocation 6 weeks after the first injection of botulinum toxin. As a result, the interval between treatments was reduced from 3 months to 2.5 months. Both patients remained symptom-free afterwards without any further luxations in the following year.

The four patients with habitual dislocation, no mental impairment, and therefore good compliance showed

distinct pain reduction between 3 and 4 points on the 11-point VAS. In addition, they reported better quality of life due to the lack of further joint luxations. As a tolerable side effect, the mean range of mouth opening decreased in these patients from 40.5 mm to 33 mm, connected with a slight subjective weakness in maximal biting force. In the other patients, pain intensity measurements were not possible due to reduced compliance. Still, relatives and guardians reported distinct subjective relief of complaints in these cases as judged from the patients' behavior.

Discussion

According to our experience and also the literature [6, 9, 12], intramuscular injections of botulinum toxin are safe and reliable as a treatment with minimal side effects for recurrent temporomandibular joint dislocations. None of our patients suffered reduced jaw opening, dysphagia, or allergic reaction as results of treatment, and all remained at least dislocation-free. Using EMG, controlled injections of the toxin can be safely placed into the lateral pterygoid muscles. By doing so, the activity of the lateral pterygoid muscle can be reduced and temporarily weakened [8].

Initially, patients required repeated injections on a frequent basis, but after at least four injections, they remained symptom-free for over half a year after cessation of the injection therapy. This suggests that the lateral pterygoid muscle does not regain its original degree of hyperactivity and may indeed undergo involution. Similar effects can also be seen in other fields of botulinum toxin use [13]. For ethical reasons, it was not possible to use a control group, but as our treatment group included several noncompliant patients, interferences could be largely excluded. In another study, Porta [10] showed the superior efficacy of botulinum toxin compared with steroid treatment in a patient with masticatory muscle pain.

Muscular hypervalence and discoordination of the lateral pterygoid muscles are aggravating causes of dislocation of the temporomandibular joint [12]. A further causal factor seems to be a neurogenic disorder [4, 12]. Therefore, intramuscular injections with botulinum toxin A represent a therapeutic option for recurrent temporomandibular joint dislocation. Especially when conservative measures fail, this procedure could be an alternative to invasive surgical interventions.

The use of botulinum toxin can also be of value in reducing the chronic facial pain associated with temporomandibular dysfunction [2, 14]. Due to the types and composition of our treatment group, in only four patients showing full compliance and no mental impairment could reductions in pain intensity be shown with an objective measuring method (VAS). However, based on the literature [2, 10, 12, 14], improvements in complaints could also be speculated in the other cases.

In conclusion, we can recommend this therapy for the treatment of recurrent temporomandibular joint dislocation. Our report on 21 patients with temporomandibular dislocation is one of the largest yet reported in the literature. Nevertheless, further studies involving larger numbers of patients and, if possible, control groups are desirable to confirm these initial findings.

References

1. Borodic G, Johnson E, Goodnough M, Schantz E (1996) Botulinum toxin therapy, immunologic resistance, and problems with available materials. Neurology 46: 26–29
2. Borodic GE, Acquardo MA (2002) The use of botulinum toxin for the treatment of chronic facial pain. J Pain 3: 21–27
3. Daelen B, Thorwirth V, Koch A (1997) Treatment of recurrent dislocation of the temporomandibular joint with type A botulinum toxin. Int J Oral Maxillofac Surg 26: 458–460
4. Daelen B, Koch A, Thorwirth V (1998) Botulinumtoxinbehandlung der neurogenen Kiefergelenkluxation. Mund Kiefer GesichtsChir 2: 125–129
5. Freund B, Schwartz M, Symington IM (1999) The use of botulinum toxin for the treatment of temporomandibular disorders: preliminary findings. J Oral Maxillofac Surg 57: 916–921
6. Freund B, Schwartz M, Symington JM (2000) Botulinum toxin: new treatment for temporomandibular disorders. Br J Oral Maxillofac Surg 38: 466–471
7. Holmlund AB, Gynther GW, Kardel R, Axelsson SE (1999) Surgical treatment of temporomandibular joint luxation. Swed Dent J 23: 127–132
8. Moore AP, Wood GD (1997) Medical treatment of recurrent temporomandibular joint dislocation using botulinum toxin A. Br Dent J 183: 415–417
9. Naumann M, Hefter H, Heinen F (1999) Dysport. Klinische Anwendung von Botulinumtoxin. Uni-Med, Bremen
10. Porta M (2000) A comparative trial of botulinum toxin type A and methylprednisolone for the treatment of myofascial pain syndrome and pain from chronic muscle spasm. Pain 85: 101–105
11. Roser M, Cornelius CP, Topka H (1988) Neuerungen in der MKG-Chirurgie. Lokale Injektionsbehandlung mit Botulinumtoxin A. Mund Kiefer Gesichtschir 2: 121–124
12. Umstadt HE (2002) Botulinum toxin in craniomaxillofacial surgery. Mund Kiefer GesichtsChir 6: 236–240
13. Von Lindern JJ, Niederhagen B, Bergé S, Appel T, Reich RH (2000) Die Behandlung der muskulären Hyperaktivität der Kaumuskulatur mit Botulinumtoxin Typ A. Dtsch Zahnärztl Z 55: 26–29
14. Von Lindern JJ (2001) Type A botulinum toxin in the treatment of chronic facial pain associated with temporo-mandibular dysfunction. Acta Neurol Belg 101: 39–41
15. Zuber M, Sebald M, Bathien N, de Recondo J, Rondot P (1993) Botulinum antibodies in dystonic patients treated with type A botulinum toxin. Frequency and significance. Neurology 43: 1715–1718