Conservative Management Options for Dislocation of the Temporomandibular Joint

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5.1 Introduction

Dislocation of the TMJ affects a wide demographic (up to 5% of the population) and can be attributed to a wide variety of causes including excessive mouth opening (e.g. yawning, vomiting, seizure), trauma (e.g. flexion-extension injury to the mandible, intubation, endoscopy, dental extraction), connective tissue disorders (e.g. Ehlers-Danlos syndrome, Marfan's syndrome) and psychogenic causes (e.g. habitual dislocation, tardive dyskinesia) as possibly the majority of cases [1]. Single episodes of TMJ dislocation are often managed by manual reduction techniques (discussed elsewhere in this text) and require no further intervention. The terms "chronic", "chronic recurrent" and "habitual" are interchangeable and used for cases in which repeated episodes of dislocation occur [2]. Other authors distinguish "acute", "chronic" (i.e. persistently dislocated) and "recurrent". Recurrent dislocations can be particularly troublesome and are the subject of discussion in the current chapter, which deals with conservative interventions to address these problems.

Recurrent dislocation of the TMJ in particular is often seen in elderly patients with cerebrovascular disease, epilepsy, dementia or dystonia due to comorbidities and/or as a side effect of medications [1, 3, 4]. Guven [2] classified such recurrent dislocation into five different types:

1. Few previous episodes and condyles repositioned without anaesthesia
2. More than a few episodes and condyles repositioned with local or general anaesthesia

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N.S. Matthews (ed.), *Dislocation of the Temporomandibular Joint*,
https://doi.org/10.1007/978-3-319-62652-9_5

3. More than a few episodes due to systemic disorder requiring surgical reconstruction
4. Previous failed surgical treatments requiring surgical reconstruction
5. Patients unable to receive intermaxillary fixation due to local/systemic problems

Such causes of habitual TMJ dislocation are often the very reasons that preclude more invasive treatment strategies for these patients, where general anaesthesia and surgery may be hazardous and undesirable. These considerations have contributed to the search for more conservative treatment strategies that are discussed here.

5.2 Autologous Blood Injection

Autologous blood injection (ABI) presents a number of advantages including ease of performance, low cost and low complication rates and has recently experienced something of a resurgence in interest [5]. The protocol described by Machon et al. [6] uses 3 mL of autogenous blood, 2 mL being injected into the upper joint space and 1 mL to the pericapsular region. The importance of injecting into the pericapsular tissues has been highlighted by Yoshida et al. [7], who demonstrated higher success rates when compared with injections into the joint space alone. The procedure may or may not be preceded by arthrocentesis and is undertaken typically under conscious sedation or general anaesthesia. Patients may be advised post-operatively to restrict mandibular movement and keep to a soft diet, with some authors also recommending a head dressing to limit mandibular excursions [8].

The principle behind the technique is the inciting of a physiological response of inflammation and oedema with resultant reduction in joint motion. Histological evaluation of a porcine model by Gulses et al. demonstrated fibrotic changes in the capsule and retrodiscal tissues [9]. Inflammatory mediators released by platelets cause vasodilatation and oedema of the periarticular tissues, diminishing compliance of the joint and reducing mobility. Organized clot leads to joint stiffness over time with maturation of fibrous tissue [9]. Magnetic resonance imaging (MRI) demonstrates accumulation similar to joint effusion from 1 h after ABI with progressive reduction in condylar movement on imaging at 12 weeks [10]. Success rates of 72.7–80% have been reported, although a concomitant reduction in mouth opening is routinely described [5–8]. Concerns have been raised about the risks of joint destruction, cartilage degeneration and chondrocyte apoptosis [11]. Exposure of cartilage to blood reduces chondrocyte metabolism and may elicit concerns for safety of the procedure in younger patients. In particular, it has been demonstrated that intra-articular blood can result in the induction of chondrocyte apoptosis, potentially leading to cartilage degeneration and ultimately joint destruction [12]. The evidence base for ABI is small and reliant on single-centre case report and series, but it is gaining favour once again.

5.3 Intermaxillary Fixation

Intermaxillary fixation (IMF) may be used alone or in combination with methods such as ABI, sclerosant injection or botulinum toxin type A for TMJ dislocation [13, 14]. As a stand-alone technique, a period of immobilization for 3–6 weeks is recommended, although results are disappointing with higher recurrence rates than those seen with other conservative management strategies [14]. There is evidence to suggest that combined strategies with other treatments may achieve the highest success rates overall. IMF techniques employed may include eyelet wires, circummandibular wires, arch bars, orthodontic brackets, Leonard buttons, IMF screws and hybrid MMF systems. Methods have also been described to use IMF to relocate the condylar heads following protracted periods of dislocation, using acrylic bite blocks in the molar region as a fulcrum in concert with class III elastics applied to arch bars and IMF screws [15].

Intermaxillary traction with elastics is of particular value in the management of chronic protracted TMJ dislocation, as opposed to chronic recurrent dislocation or habitual dislocation. In these instances, there can be considerable difficulty in overcoming spasm of the pterygomasseteric sling, temporalis muscle fibrosis and impingement of the coronoid process, and traditional methods of manually relocating the TMJ such as the Hippocrates manoeuvre will often fail. A combination of posterior bite blocks and class III elastic traction on arch bars has been described as successfully reducing isolated cases over a period of weeks [16].

5.4 Botulinum Toxin

Botulinum toxin is produced by the anaerobic bacterium *Clostridium botulinum* and acts by blocking the release of acetylcholine into the synaptic cleft at the motor end plate, thus interrupting neuromuscular transmission. Licenced indications for medical use in the United Kingdom are varied and include strabismus, blepharospasm, hemifacial spasm, cervical dystonia, focal spasticity in cerebral palsy, upper limb spasticity in adult stroke patients and axillary hyperhidrosis [17]. The use of botulinum toxin for TMJ dislocation is an unlicenced indication and was first described by Daelen and colleagues [18].

For recurrent TMJ dislocation, botulinum toxin type A is injected extraorally through the sigmoid notch into the lateral pterygoid muscles bilaterally (Figs. 5.1 and 5.2), with or without concomitant injection into the masseter muscles bilaterally [19]. An intraoral route of injection has also been described by some authors, although this is less commonly reported [20, 21]. Electromyographic (EMG) control can be used to facilitate a more accurate injection [22, 23]. The senior author has also often injected the anterior fibres of the temporalis muscles, as the vector of pull of these fibres may serve to contribute to protrusion of the mandible and a predisposition to condylar dislocation.

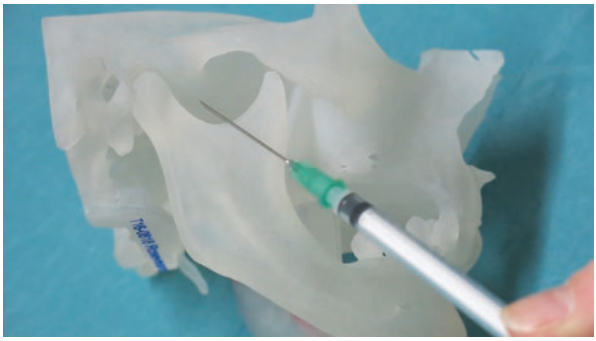

Fig. 5.1 Injection of Botox® through the sigmoid notch into lateral pterygoid muscle schematically represented on a stereolithographic model

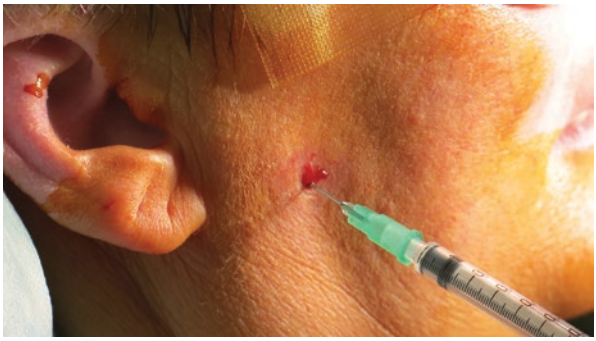

Fig. 5.2 Injection of Botox® through sigmoid notch into lateral pterygoid muscle demonstrated on a patient

Doses are variable, but typically when using Botox® (Allergan), 25–75 U are injected into each side. Importantly, units of Dysport® are not equivalent to units of Botox®. Dysport® is around four times less active than Botox®, and doses should be adjusted accordingly, although the literature generally describes the use of Botox® [17]. Typical doses for Dysport® as used by the authors are around 500 U into each muscle.

As with many of the options discussed in this chapter, evidence is sparse and confined to case series of small numbers. The largest cohort in the literature that we could find is that of Ziegler and colleagues [4], who demonstrated complete cure of recurrent TMJ dislocation in 19 out of 21 patients over a follow-up time of up to 3 years. Adverse effects are infrequent, with the most common being transient dysphagia in 8% of patients as the result of diffusion of the drug into adjacent muscles [20]. Other potential side effects include nasal regurgitation, nasal speech, painful chewing and dysarthria [20]. Careful injection technique and appropriate dosing should minimize these problems that are short-lived when they do occur. Risks must be discussed and documented, and written informed consent provided to encompass those risks mentioned, as well as the possibility of transient facial weakness which may persist for months due to inadvertent distribution of the toxin adjacent to branches of the seventh cranial nerve.

5.5 Exogenous Sclerosants and Prolotherapy

A wide variety of exogenous sclerosants has been tried including iodine, ethanolamine oleate, alcohol (100% ethanol), bleomycin, tetracycline, cyclophosphamide and OK-432 (Picibanil), among others [3]. The senior author has also successfully employed sodium tetradecyl sulphate (STS), more widely used in the management of venous varicosities as a sclerosant.

The use of intra-articular sclerosant has been a part of the armamentarium for dealing with recurrent TMJ dislocation for some time and was first described as early as 1950 by McKelvey [24]. Whilst various agents have been tried, most recently OK-432 has been highlighted by Matsushita and colleagues as a possible agent of choice [3]. The drug is a streptococcal derivative inactivated by penicillin G. Originally developed as an anticancer agent, maxillofacial surgeons are often more familiar with its use in treating lymphatic malformations and chyle leaks following neck dissections [3, 25]. Prepared by diluting the drug with equal parts of saline and 2% lignocaine, it can be introduced into the superior joint compartment and pericapsular tissue. The authors highlighted two cases that exhibited no further episodes of dislocation at 6 months but acknowledged the risks of anaphylaxis, interstitial pneumonia and acute renal failure.

"Prolotherapy" is a variation on this theme that involves the intra-articular injection of non-sclerosants such as dextrose to initiate an inflammatory response. Zhou and colleagues [26] described the use of 50% hypertonic dextrose with 2% lignocaine as an intra-articular injection in 45 patients suffering from recurrent dislocation. Success was achieved in 91% of cases by 12 months, although a few of the patients received more than one round of treatment during that period. A small randomized controlled trial conducted by Refai et al. [27] showed promise in the use of prolotherapy for TMJ hypermobility, although they used 10% dextrose with 2% mepivacaine, and the follow-up period was much shorter (12 weeks).

5.6 Mental Health and Recurrent TMJ Dislocation

There is no doubt that in a subset of patients with recurrent or habitual dislocation of the TMJ, there is a functional component to all or part of their presentation, with an overlap between mental health disorders and TMJ dislocation. A substantial portion of patients with chronic TMJ disorders have been shown to have moderate to severe depression with somatization in cohort studies [28, 29]. Furthermore, mental health patients taking neuroleptic medications or antipsychotics have been shown to develop TMJ dislocation as a result of dystonia secondary to their medications [30–32]. Converting patients to different medications or seeking different treatment modalities may have a role to play in reducing episodes of habitual dislocation in such patients, and clinicians should work closely with the psychiatric services responsible for their care.

Orofacial pain and TMJ disorders have been reported as being far more common in patients with eating disorders, particularly those patients exhibiting binge eating

and self-induced vomiting [33]. In one study, eating disorder patients practising self-induced vomiting were shown to have higher muscle sensitivity on palpation alongside expected higher rates of dental erosion and attrition, and these clinical findings are worth noting [34].

Perhaps more worryingly is the trend for some patients to deliberately induce recurrent subluxation or dislocation of the TMJ as part of attention-seeking behaviour or Munchausen syndrome [35]. Such patients may present on a regular basis to emergency departments and become well known to local maxillofacial services. Named after the Baron von Munchausen and his fantastical stories, Munchausen syndrome is said to be characterized by a triad of pathological lying, simulated illness and wandering from hospital to hospital (peregrination) [36]. In such patients, the emphasis should be on "compassionate and firm limits" to the level of care provided, and clearly in such a group, conservative and non-surgical treatments are likely to be the ceiling of care [35]. It is important that all members of the team are aware of such patients and are consistent in their communication.

We have encountered a small number of younger patients who were referred to us with recurrent TMJ dislocation, either unilateral or bilateral, in whom it eventually became evident that there were underlying psychological issues which merited referral on to clinical psychologists or psychiatrists. These issues concerned bereavement reactions, issues of conflict with one or both parents and complex psychological and metabolic problems akin to anorexia, the latter compounded by periods of intermaxillary fixation. We have failed to find any studies in the maxillofacial or psychiatric literature exploring these issues.

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