The protocol consisted of the following steps:

1. Perioperative indomethacin for 2 weeks;
2. The creation of a minimal gap of 5 to 10 mm;
3. Ipsilateral coronoidectomy and (if required) contra lateral coronoidectomy;
4. Pterygomasseteric sling and temporalis muscle release;
5. Interpositional dermis fat graft fixed to the condylar stump;
6. Insertion of a suction drain;
7. Immediate aggressive physiotherapy for at least 6 months;
8. Immediate aggressive physiotherapy for at least 6 months;
9. Immediate aggressive physiotherapy for at least 6 months;
10. Immediate aggressive physiotherapy for at least 6 months.

Recent research has shown that prolonged TMJ ankylosis inhibits the normal range of mouth opening by shortening. These effects are generally more evident when joint pathology exists for a longer period [1]. Two different pathologic changes could be associated with the prolonged TMJ ankylosis which includes degenerative changes or hypertrophy.

It is a well-established phenomenon that skeletal muscles undergo disuse atrophy when they are not in use [2]. In TMJ Ankylosis, where the mandible is in a chronic state of closure, the logical inference would thus be that the elevator muscles would undergo degenerative changes or disuse atrophy. It has been suggested that prolonged TMJ ankylosis inhibits the normal use of the joint, are associated with degenerative changes in the masseter, medial pterygoid and temporalis muscles inducing reflex muscle splinting to protect the joint [1].

While Vinay et al evaluates thickness and cross-sectional areas of jaw elevator muscles and indicates that muscle hyperactivity might be associated with ankylosis. A probable hypothesis would be that regular mandibular movements are more difficult to perform due to the stiffness in the joint, which subsequently resulted in increased thickness of the muscles due to the increased functional demand. It could also be that the increased muscle thickness is due to shortening of the muscle, because the height and length of the affected mandibles are smaller than normal [3].

Recently, a protocol for management of TMJ ankylosis consisted of nine steps based on the Pathogenesis of Ankylosis and Re-Ankylosis have been published.

The protocol consisted of the following steps:

- Perioperative indomethacin for 2 weeks;
- The creation of a minimal gap of 5 to 10 mm;
- Ipsilateral coronoidectomy and (if required) contra lateral coronoidectomy;
- Pterygomasseteric sling and temporalis muscle release;
- Interpositional dermis fat graft fixed to the condylar stump;
- Insertion of a suction drain;
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- Immediate aggressive physiotherapy for at least 6 months.

Botulinum toxin type A (BTA) are purified substances that derived from clostridium botulinum, and can block muscular nerve signals. Injection of very small amounts of BTA into specific facial muscles can block the muscle’s impulse and temporarily weakens the contraction of muscles by blocking the presynaptic cholinergic nerve endings, thus causing relaxation of the voluntary muscle [4]. Therefore, it will results in reducing the activity of elevator muscles of the mandible. It has been shown clinically that injection of Botulinum toxin, as an adjunct to surgical therapy, in the masseter muscles of patients operated for TMJ Ankylosis has improved outcomes [5]. Based on this concept, I would like to add the preoperative injection of Botulinum toxin in the masseter muscles, as an adjunct pre-operative step to the surgical protocol to surgical therapy, to improve the outcomes.

References