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Leading article

Pathogenesis of post-traumatic ankylosis of the temporomandibular joint: a critical review

Gururaj Arakeri [a,*], Atul Kusanale [b], Graeme A. Zaki [c], Peter A. Brennan [c]

[a] *Department of Oral and Maxillofacial Surgery, Sri Sai College of Dental Surgery, Vikarabad, Andhra Pradesh, India*
[b] *Oral and Maxillofacial Surgery Department, Queen Alexandra Hospital, Portsmouth PO6 3LY, UK*
[c] *Queen Alexandra Hospital, Portsmouth PO6 3LY, UK*

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Abstract

Many factors have been implicated in the development of bony ankylosis following trauma to the temporomandibular joint (TMJ) or ankylosis that recurs after surgical treatment for the condition. Although many reports have been published, to our knowledge very little has been written about the pathogenesis of the process and there are few scientific studies. Over the last 70 years various treatments have been described. Different methods have been used with perceived favourable outcomes although recurrence remains a problem in many cases, and ankylosis presents a major therapeutic challenge. We present a critical review of published papers and discuss the various hypotheses regarding the pathogenesis of the condition.

Keywords: Temporomandibular joint; Ankylosis; Review

Introduction

Temporomandibular disorder is a generic term used for any problem concerning the jaw joint.[1] Among these disorders ankylosis is one of the most debilitating and can have an adverse effect on quality of life. It is a challenging problem, and often starts during the active growth stage of early childhood.[2–6] While there have been many publications regarding the condition and its treatment, very little has been written about its pathogenesis. This review evaluates the role of various factors that are implicated in ankylosis of the temporomandibular joint (TMJ).

Aetiology of ankylosis of the TMJ

Ankylosis of the TMJ has various causes that include trauma (usually the most common), local or systemic infection, or systemic disease.[7–13] Trauma can result in an intra-articular haematoma leading to fibrosis, excessive bone formation, and ultimately to hypomobility of the joint (Fig. 1).[7] The region of the TMJ can also become infected from local sites such as otitis media and mastoiditis, or through haematogenous spread from diseases such as tuberculosis, gonorrhoea, and scarlet fever.[7] Systemic diseases that are implicated include ankylosing spondylitis, rheumatoid arthritis, and psoriasis.[7,13]

Interinsical opening is an indicator of the severity of the ankylosis, and clinically, complete ankylosis is defined as a condition when opening is less than 5 mm.[14] In unilateral cases the mandible can be forced to open because of its elasticity and the minimal mobility of the cranial sutures.[14,15]

The digastric and mylohyoid muscles produce marked notching in the lower border of the mandible in front of the insertion of the masseter and medial pterygoid muscles (Fig. 1).[14]

The notching at the antegonion and the apparent distortion of the mandibular structure (which is pathognomonic of the condylar growth arrest) are thought to be caused by continuous growth at the angle of the mandible as a result of

* Corresponding author at: Gangashri, Basaveshwara Nagar, Shahapur, Gulbarga 585223, Karnataka, India. Tel.: +91 9341428302/9347261640.
E-mail address: gururaj.arakeri@gmail.com (G. Arakeri).

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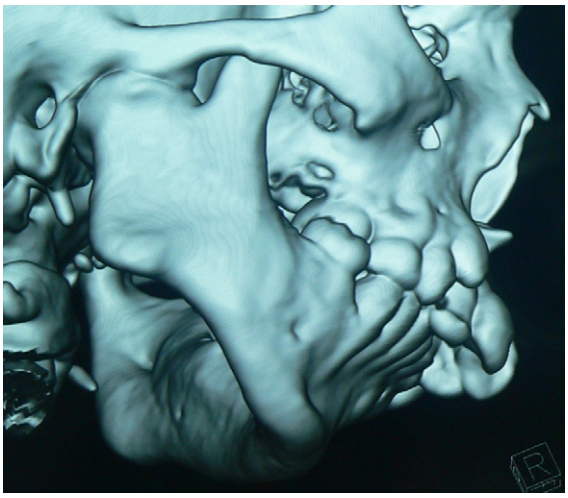

Fig. 1. Notching at the antegonion and the apparent distortion of the mandibular structure resulting in characteristic "warping" because of transarticular bony adhesion.

subperiosteal apposition. Because of a failure of growth at the condyle, forward and downward movement of the body of the mandible does not occur, and a localised thickening of the bone at the angle accentuates the antegonion. This, coupled with the obtuse angle formed between the cranial base and the lower border of the mandible, is responsible for the characteristic "warping" (Fig. 1).[16]

Aetiology of recurrence of ankylosis

There are many reasons why ankylosis recurs after release surgery, but those most commonly implicated include failure to have aggressive physiotherapy, and poor compliance by the patient. Kaban et al. postulated that recurrent ankylosis is primarily caused by inadequate excision of the ankylotic mass.[17] This results in failure to achieve passive mouth opening (without the need for excessive force) in theatre. If excessive force is necessary to open the jaw intraoperatively then more force will be required postoperatively. Under these circumstances, physical therapy will be very painful, and the outcome will be poor regardless of how well the patient cooperates with physiotherapy.

Methods of treatment

A variety of techniques for the treatment of ankylosis have been described including gap arthroplasty, interpositional arthroplasty, and osteotomy and excision of the ankylotic mass within the TMJ. Reconstruction of the ramus and condyle with autogenous bone, such as costochondral grafts, grafts using fibula, clavicle, iliac crest, or metatarsal head, or alloplastic material, have all been reported.[8,9,17–24] However, no single method has uniformly produced successful results.[7,8–10,12,25–31] Furthermore, there is confusion and a

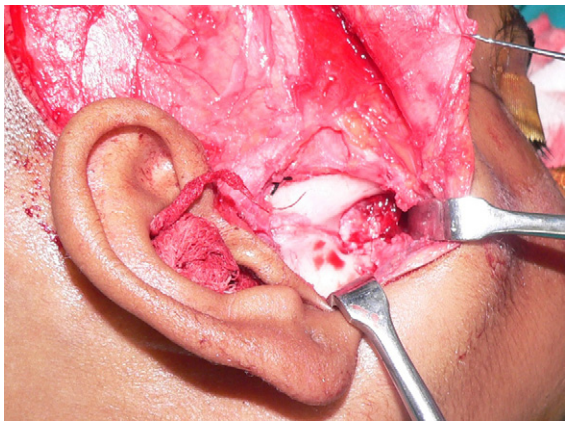

Fig. 2. Intraoperative view of post-traumatic transarticular bony adhesion (primary ankylotic mass).

lack of consistency in published data regarding treatment techniques.[17] Limited range of movement and ankylosis that recurs (usually within 6 months after operation) are the complications most often reported.[7,32,33] A poor outcome after surgery for ankylosis of the TMJ in children may be because of a lack of compliance with physical therapy, which leads to a tendency for recurrence.[17]

Concept of ankylotic mass

Salins described an ankylotic mass as being abnormal bone that replaces the articulation and results in restriction of mandibular movement.[34] Although it is not a neoplastic process, the bone is capable of continued growth, and it can be considered a reparative process similar to that found in an exuberant callus, typical of fractures in children or of fractures that have not been mobilised adequately. Remodelling does not occur in ankylosis of the TMJ, and the joint is usually surrounded by very dense fibrous tissue, particularly on its medial aspect, which further limits mandibular movement (Figs. 2 and 3).[34]

Salins challenged the hypothesis about aggressive removal of the ankylotic mass.[34] He proposed that radical removal of bone leaves opposing surfaces of healing bone that are likely to be bridged by dense fibrotic tissue that surrounds the mass and prevents unimpeded mandibular movement, and ultimately results in recurrence of the ankylosis. He suggested creating a pseudoarthrosis and leaving the ankylotic mass undisturbed. Recently, Ko et al. osteotomised the ankylotic mass instead of relieving or excising it to reduce the amount of clot formation.[35]

Is formation of an intra-articular haematoma alone the primary cause of ankylosis and its recurrence?

Several authors have postulated that intra-articular haematoma alone may lead to ankylosis of the TMJ

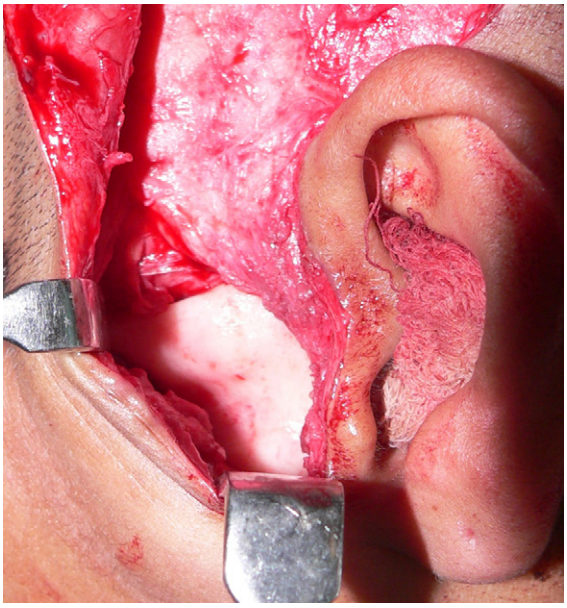

Fig. 3. Intraoperative view of recurrence of ankylosis (secondary ankylotic mass).

based on organisation and subsequent ossification of an intracapsular haematoma.[2,36–40] This hypothesis is based on animal experiments and may be different from that seen in vivo.[39] Oztan et al. concluded that trauma causing haemorrhage in the joint space may not give rise to ankylosis as it does not always progress to form bone.[2]

Does the meniscus play a prime role in the adhesion of articular surfaces?

The meniscus normally serves as a barrier to prevent fusion of the condyle with the glenoid fossa.[41] If the disc is damaged, the condyle and glenoid may fuse if the bony surfaces are also damaged and there is a haematoma between them – for example, when a fracture-dislocation is accompanied by displacement of the meniscus from the glenoid fossa.

Other contributing factors are direct bone-to-bone contact, close approximation of the articular components, and prolonged mechanical immobilisation or muscular splinting.

However, ankylosis still occurs, particularly in children, after fractures of the condylar head even though the meniscus is present and undamaged. Also, there are reports of remnants of fibrocartilage (presumably of disc origin), and a persistent rudimentary joint space in many ankylosed TMJs.[10,36,42]

It is very difficult to understand why a natural disc-ligament complex that is interposed between the condyle and glenoid fossa adheres even after a mild degree of injury (many patients give a history of a fall and mild injury) when in contrast, a gap arthroplasty or an interpositional arthroplasty does not lead to ankylosis despite inducing much trauma to the region.

The principle behind release of an ankylotic mass with an interpositional graft (either autogenous or alloplastic) is to prevent adhesion between the two bony cut surfaces. However, if such a graft can prevent ankylosis, then logically it should not occur after trauma, since the meniscus and capsule act as natural interpositional materials. The intact disc may act as a physical impediment to transarticular bony fusion.[36,43] Aggressive excision of the mass alone with no interposition might leave a dead space and allow a haematoma to form, which, together with opposing cut bony surfaces would lead to scarring and repeated adhesion or ankylosis.

Does ankylosis of the TMJ follow the characteristic steps of distraction osteogenesis because of traction of the lateral pterygoid muscle?

Recently, it has been postulated that distraction osteogenesis caused by traction of the lateral pterygoid muscle on the bone after sagittal fracture of the mandibular condyle is an important factor in the genesis of traumatic ankylosis of the TMJ.[39] However, it may be that the role of the lateral pterygoid muscle in distraction osteogenesis is overstated because the attachment of the muscle is at the neck of the condyle and not at the head.[15] To induce distraction osteogenesis at the condylar head, the traction force needs to be directed towards the head rather than the condylar neck. After a sagittal split of the condyle, the lateral pterygoid muscle pulls the bony fragment in a medial and upward direction away from the condylar region, so its role in ankylosis is not convincing.

Medially dislocated condylar fracture and ankylosis of the TMJ

Ferretti et al. suggested that dislocation of the condylar head creates a cavity that is rapidly filled by blood creating a large haematoma which, because of disruption of the periarticular anatomical boundaries spreads to extracapsular sites, and thereby bypasses any impediment that the articular disc may affect ensuring its extravasation beyond the condylar head. Ossification occurs when the haematoma is populated by endosseous vessels and there is a sufficient degree of immobility. Several molecular pathways (including the expression of several genes belonging to the tumour growth factor (TGF) family, and bone morphogenic proteins (BMP) are activated following fracture. Expression of these proteins at the fracture site will activate osteoblasts and culminate in the formation of bone. Thus, the vascularisation of a haematoma is the critical first step to its ossification, and vigorous mobilisation after injury is critical in preventing the complication of ankylosis after condylar fracture.[36,44,45]

Although the hypothesis is interesting when compared with previous theories, it is essentially based on observations following dislocation of condylar fractures and therefore does

not explain the sequence of events for ossification in non-dislocated fractures of the condyle or condylar head.

Does the formation of ankylosis after trauma follow the characteristic events of fracture healing?

Conceptually, ankylosis has been regarded as the fusion of two approximated and injured bony surfaces, and has also been regarded as inappropriate tissue differentiation after fracture.[40] Even so, for a process to be labelled as a fracture, there should firstly be discontinuity in a bone, and secondly, two exposed bony edges. Ankylosis cannot be correlated with fractures because it involves the fusion of two different bony surfaces (condyle with cancellous, and glenoid with compact bone); one static, and one constantly mobile. Furthermore, in most cases of trauma, the condyle (head or neck) is injured more severely than the bony glenoid fossa, which is protected by an interposed articular disc, so the sequence of events of a healing fracture cannot be extrapolated to the process of ankylosis.

Role of the sagittally split condyle in transarticular adhesion of the TMJ

In 1982, Rowe reported that ankylosis can be a result of anteroposterior (sagittal) fracture of the condyle.[46] Displacement of the lateral fragment upward over the outer rim of the glenoid fossa, with associated displacement of the intra-articular disc, and the accompanying loss of mobility, were responsible for the condition in these patients.

In 2008, He et al. suggested a strong relation between sagittal split condylar fractures and simultaneous mandibular fractures in the pathogenesis of ankylosis of the TMJ.[47] This mechanism increases the width between the condyles or rami at the level of the lateral segment. The fractured surface of the ramus or lateral condylar pole displaces laterally and possibly superiorly to the glenoid fossa and comes into close contact with the zygomatic arch, which causes a juxta-articular type of ankylosis (ankylosis that occurs lateral to the articulation itself[36]). A study of 206 patients with ankylosis of the TMJ by Sarma and Dave showed that 93% were juxta-articular with a medially positioned non-ankylosed condylar head,[48] although in their series there were no simultaneous fractures. Ferretti et al.[36] reported only three simultaneous mandibular fractures among 26 patients with ankylosis of the TMJ, so the role of such fractures in patients with sagittal split condylar fracture remains controversial in the aetiology of ankylosis.

Is protein-energy malnutrition a predisposing factor for traumatic adhesion in the TMJ?

It is interesting that the incidence of patients who go on to develop ankylosis of the TMJ is high in some parts of the world. The most striking finding is the perceived incidence of ankylosis in some countries, particularly in developing countries, and the relative scarcity of the disorder in developed countries.[47,49] Why would there be a higher incidence in China, Africa, or India, than in the United States or Europe?[47]

Possible reasons why developing countries have an increased number of such patients are larger populations, genetic predisposition, the lack of availability of proper care, or inadequate care because of poorly equipped hospitals.[47] Recently, it has been shown in rats that malnutrition results in impaired callus formation, as well as fibrous ankylosis in the TMJ on healing of a displaced condylar process.[49] In this study repair of the fracture was impaired, there was proliferation of connective tissue, and atrophy of condylar fibrocartilage that led to transarticular fibrous adhesion. As protein-energy malnutrition affects roughly half the population worldwide, particularly in developing countries,[49] malnutrition could also be a predisposing cause of ankylosis. Finally, one further explanation may be poor economic status as possible neglect of minor facial trauma could progress to ankylosis of the TMJ.

Conclusion

Many factors have been suspected to affect the development of ankylosis of the TMJ after trauma, and its recurrence after surgical treatment. While there exists a clear association between ankylosis and traumatic injuries of the mandibular condyle, authors do not agree about its management and aetiology. Further research is needed to understand the debilitating condition more fully, and additional treatments need to be developed to reduce the incidence of recurrence after surgery.

Conflict of interest

None.

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