

ESTIMATION OF INTERLEUKIN 1-BETA IN SYNOVIAL FLUID OF TEMPOROMANDIBULAR JOINT IN CASES OF MANDIBULAR FRACTURE: AN ORIGINAL RESEARCH

Dr.Anuj Bhargava¹, Dr.Meenaxi Umarani², Dr.S.C Metgud³, Dr.S.M Kotrashetti⁴, Dr.S.D Baliga⁵

ABSTRACT

BACKGROUND : Mandibular fractures is the main type of TMJ macrotrauma. Malocclusion, unilateral mastication as well as oral habits including bruxism and clenching are included in the category of TMJ microtrauma. The articular disc in TMJ has a primary function of stress distribution. Trauma has been thought to alter the mechanical properties of the disc. TMJ Macrotrauma may cause degeneration of articular cartilage and production of the inflammatory and pain mediators. A series of cytokines and chemical mediators are reported to play a major role in various arthropathies for example rheumatoid arthritis and osteoarthritis of TMJ. Among them, typically IL-1 Beta appear to be of importance in pathogenesis of synovitis and in cartilage damage in these joint disease. IL-1Beta act to induce the production of metalloproteinases that induce matrix degradation. The inflammatory and degenerative changes of TMJ can develop after mandibular fractures.

METHOD: Analysis of TMJ status of 14 mandibular fracture patients were done for synovial fluid biochemical analysis of IL-1 Beta (IL-1 Beta ELISA kit) by a procedure of arthrocentesis. Histomorphologic examination, was also performed on the selected patients. Exclusion criteria included high condylar and neck fractures and known cases of degenerative joint disorders.

RESULTS AND OBSERVATION: In biochemical analysis considerable amount of IL- 1Beta was detected in the synovial fluid of patients. On Histomorphologic examination, degenerated cells, cartilage, inflammatory cells and crystals were observed.

CONCLUSION: Mandibular fractures can be a possible etiologic factor in cartilage destruction, biochemical and intra-articular pathology. Increased levels of IL-1 Beta could be potential catabolic marker for cartilage degradation in the TMJ. Clinicians should recognize the etiologic importance of TMJ Macrotrauma , and importance of long term evaluation of TMJ .

Keywords: TEMPOROMANDIBULAR JOINT (TMJ),ARTHROCENTESIS,IL-1 BETA

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INTRODUCTION

Facial trauma has been suggested as a possible etiologic factor of temporomandibular (TMJ) disorders (TMD). Facial trauma usually occurs as a result of traffic accident, fall, industrial injury and so on[1]. It has been reported that trauma to maxillo-mandibular complex may result in 70 % of mandibular fractures, with 26% occurring in condylar area[2].

Trauma occurring in TMJ can be classified as macrotrauma and microtrauma by the degree of intensity. Facial trauma with mandibular fractures in specific is the main type of TMJ macrotrauma. Malocclusion, unilateral mastication as well as oral habits including bruxism and clenching are included in the category of TMJ microtrauma. However, there is little information on the role of macrotrauma[1].

TMJ macrotrauma may cause degeneration of the articular cartilage and production of inflammatory and pain mediators. Articular disc in TMJ has a primary function of stress distribution. Trauma has been thought to alter the mechanical properties of the disc. However, whether these mediators are involved in pathogenesis is still unclear.

A series of cytokines and chemical mediators are reported to play a major role in various arthropathies for example rheumatoid arthritis and osteoarthritis of TMJ. Among to them, typically IL-1 Beta and tumor necrosis factor (TNF) alpha appear to be of importance in pathogenesis of synovitis and in cartilage damage in these joint diseases[3].

IL-1 which belong to the group of cytokines that mediate inflammation have been detected in Internal Derangements (IA) and Osteoarthritis (OA) of TMJ's[4,5,6,7]. The cytokines are mainly produced by monocytes/macrophages that infiltrate the synovium. A mechanism for cartilage destruction in the TMJ has been proposed which states that Endogenously produced cytokines (IL-1 or IL-6) appear to directly contribute to tissue damage by inducing the release of proteinases such as cathepsin B-14 and D15 and other collagenases. Namely, neutral matrix metalloproteinases (MMPs). These MMP's produced by synoviocytes and articular chondrocytes are reported to be possible candidates that mediate the cartilage degradation[7].

MMP's are secreted from the cell in latent form, bind with tissue inhibitor of metalloproteinases (TIMP'S), and become activated extracellularly by cytokine and other proteinases. The

excess amount of these activated enzymes degrades the cartilage ground substance by splitting the proteoglycan chain and thus causing a decrease in the cartilage resilience, which is followed by destruction of the cartilage compartments. Therefore, the appearance of active forms of matrix degrading proteinases in the Synovial Fluid (SF) may indicate early degenerative changes in the TMJ articular cartilage. These changes proceed to TMJ bone destruction, similar to those reported in other joints with RA and OA [7].

IL-1Beta and TNF alpha act to induce the production of metalloproteinases that induce matrix degradation[6]. Additionally it is thought that the expression levels of IL-1 beta and TNF alpha relate with the pathogenesis of synovial inflammation of TMJ.

Although it has been considered that mandibular trauma played an important role in the etiology of TMJ, There is no definite evidence and there are many controversial opinions[1]. Some authors claim that macrotrauma occurring due to mandibular fractures is a very important causative factor for pathogenesis of TMJ[1]. However there is little information on the role of macrotrauma[1]. This is the reason which forms the basis for undertaking this study.

A Multidirectional approach was applied for evaluation of changes in the microenvironment within the TMJ after macrotrauma TMJ status was analysed by histomorphologic examination and estimation of IL-1 Beta concentrations in patients with mandibular fractures. The main purpose of this study was to estimate, evaluate and analyze IL-1Beta levels in synovial fluid of TMJ of mandibular fracture patients and hypothesize the role of mandibular fractures as a potential etiologic factor for pathogenesis of TMD.

PATIENTS AND METHODS

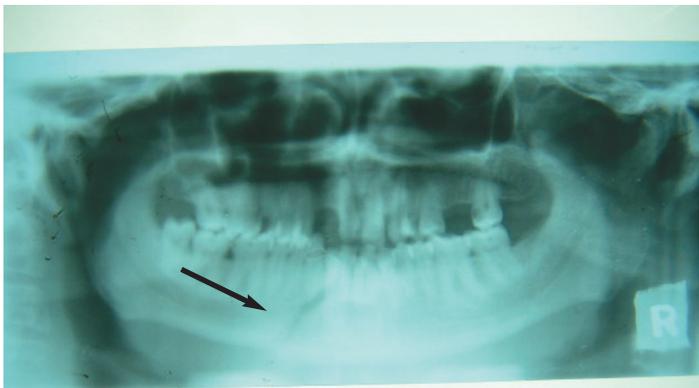


Figure.1: A Preoperative Orthopantomograph depicting Left Parasymphysis Fracture of Mandible

A total of 14 cases (28 TM joints) of Mandibular fractures admitted to KLE Hospital and Research Center Belgaum or on regular OPD basis at KLEVK Institute of Dental Sciences were selected and analyzed for the study. Patients with symphysis, Parasymphysis, Body, Angle and Subcondylar fractures were included in the study (Figure.1). Both unilateral and bilateral fractures were considered. Patients with condylar head, high condylar neck fractures, pediatric condylar fractures and known cases of

generalized joint diseases like Rheumatoid Arthritis, Osteoarthritis, internal derangement, closed lock or open lock were not included in the study. Arthrocentesis procedure was performed under aseptic precautions from both the TM Joint of the same patient and sent for laboratory investigations

PROCEDURE FOR ARTHROCENTESIS

The armamentarium was prepared as depicted in Figure.2. The points of needle insertion were marked on the skin according to the method suggested by Mc. Cain. A line is drawn from the middle of the tragus to the outer canthus.



Figure.2: Armamentarium for Arthrocentesis of TMJ

Entry points are marked along this canthotragal line. 1st point (posterior entrance point) is marked 10mm from the midtragus and 2mm below the line. 2nd point (anterior entrance point) is marked 10mm from the first point & 10mm below the line. 1st point corresponds to the glenoid fossa and 2nd to the articular eminence (Figure.3). A 19 gauge needle was then introduced through the 1st point.

5ml of saline was injected through this needle slowly with continuous positive and negative pressures alternatively to distend the joint space. Another 19 gauge needle is then simultaneously inserted in the



Figure.3 : Needles in position for Arthrocentesis of TMJ

2nd point to establish a free flow of the solution through the joint space. A total of 5ml saline solution is used to lavage the superior joint space. A total of 0.5 to 1.5 ml of diluted synovial fluid is aspirated out of the joint. Once the needles are removed, patients lower jaw is gently manipulated in the vertical, protrusive & lateral excursions to facilitate lysis of adhesions and help further free up the disc. Postoperatively Antibiotics, NSAIDs and Muscle relaxants were advised along with physiotherapy.

FOR HISTOMORPHIC CELL EXAMINATION

Each 0.5-1 ml synovial fluid Samples were collected before surgery through the procedure of arthrocentesis of the right and left TM Joint space. Samples were immediately sent to the department of diagnostic pathology for smearing. Samples were centrifuged at 1000rpm for 10 mins and analyzed by Drying and fixation with 95% methyl alcohol, Papanicalou staining was done, and examined under light (100x) and polarized microscope.

FOR IL-1 Beta ESTIMATION:

The samples which were collected by the procedure of arthrocentesis before surgery were centrifuged first at 1000 rpm for 10 mins followed by a second spin of 2000rpm for 15 mins to remove cells and then these samples were stored at -30 degree c for evaluation of IL-1 Beta concentration. IL-1 Beta samples were measured using ELISA kit (Beckman Coulter) according to the manufactures protocol (Figure.4-14).

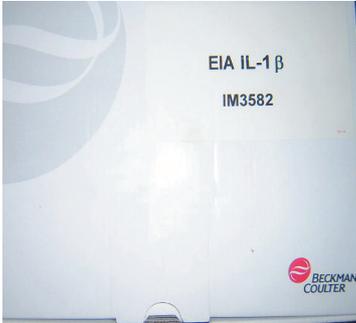


Figure.4: IL-1Beta Kit



Figure.5: Diluent & HRP Conjugate



Figure.12: Collected Samples



Figure.13 :Centrifuge



Figure.14: Spectrophotometer for reading Optical Density(OD)



Figure.6: Stop Solution and IL-1 Beta Antibody

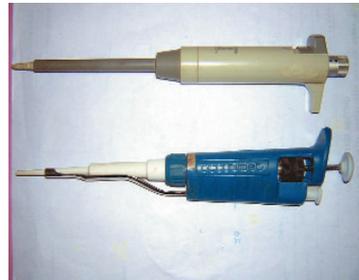


Figure.7: Micro Pippets



Figure.8: Calibrator Series



Figure.9: Uncharged Wells

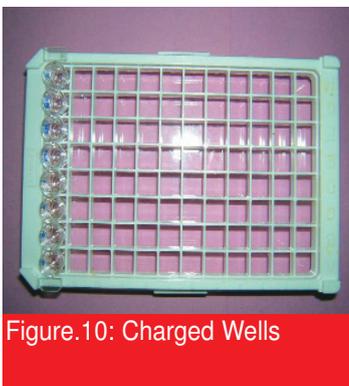


Figure.10: Charged Wells



Figure.11: Temporary storage of Samples

RESULTS

A total of 14 cases (28 TM joints) of Mandibular fractures admitted to KLE Hospital and Research Center Belgaum and on regular OPD basis at KLE VK Institute of Dental Sciences were selected and analyzed for the study. All 14cases (including both unilateral and bilateral fractures) underwent open reduction and internal fixation under general anesthesia. Two analysis were done :

1. Histomorphologic examination of the aspirated synovial fluid
2. Analysis of aspirated synovial fluid for IL-1 Beta estimation

In the 14 cases taken for the analysis of IL-1 Beta & histomorphologic examination 10 cases were of unilateral mandibular fracture (parasymphysis, angle, body & low sub condylar) constituting Group A ,This group was further divided into Fractured and the Non Fractured side subgroups.

Rest 4 cases were included in bilateral mandibular fractures comprising Group B. In the total 14 cases analyzed 9 were males and 5 were females with an age range from 20-60 years. In the Group A non fractured side 2 cases could not be analyzed for IL-1 beta concentration because of very less (<0.5ml) quantity of diluted synovial fluid. The samples were sent for histomorphologic examination only .

HISTOMORPHIC EXAMINATION

A total of 14 (28 joints)cases were analyzed and results tabulated as shown in **Table.1**. The quantity obtained from each of these joints was 0.5-1ml .The samples were thin watery in consistency and turbid in appearance. The samples were examined

with light microscope(100 X) magnification(Figure.15).

Table.1

HISTOMORPHIC ANALYSIS OF BOTH SIDES OF THE TMJ

FINDINGS	RIGHT TMJ	LEFT TMJ
INFLAMMATORY CELLS	1	1
DEGENERATED SQ CELLS	12	13
PROTEINACIOUS MATERIAL	5	8
CRYSTALS	14	14
DEGENERATED RBC'S	3	3

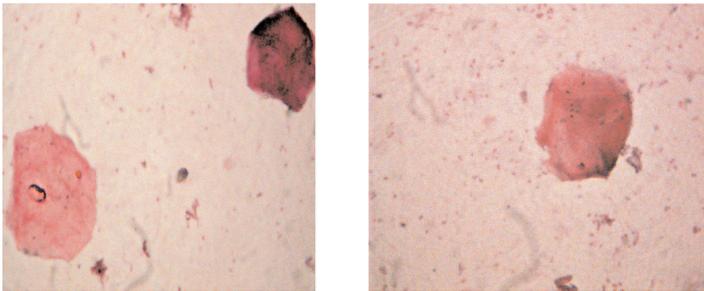


Figure.15: Histological slides showing degenerated epithelial cell and inflammatory cell (1-2 per High power field) with proteinaceous material (at magnification x 100)

IL-1 BETA ELISA EXAMINATION

The results were calculated using the individual values for each parameter. The mean values , standard deviation & coefficient of variation for each of the parameters were considered and were checked for statistical significance using the Unpaired Student t test. A calibrator series (Table 2) was prepared to calculate the levels of IL-1 Beta in pg/ml using the optical density as measured by a spectrophotometer(Figure 14).

Table.2: Calibrator Series

Optical Density (OD) Values	Concentration in (pg/ml)
0.560	250
0.230	83
0.170	28
0.125	9
0.037	0

IL-1 BETA ELISA EXAMINATION

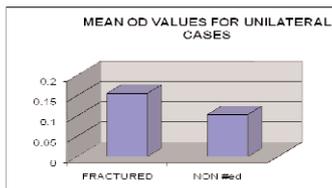


Figure.16: Group A: Mean OD Values for Unilateral Cases

The conversions were done using the LAGRANGES INTERPOLATION EQUATION.

For Group A(Unilateral Fracture Cases), on the fractured side the optical density(OD) values as depicted in Figure. 16 were in the range

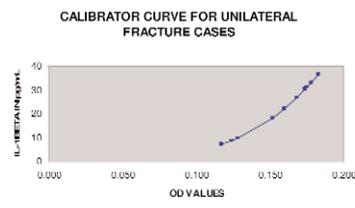


Figure.17: Group A: Calibrator Curve for Fractured Side

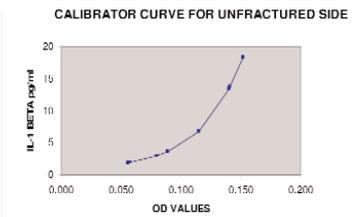


Figure.18: Group A: Calibrator Curve for Un fractured Side

of 0.117-0.183 as illustrated in the calibrator curve (Figure.17) with mean of 0.1559 whereas on the non fractured side the range of OD values (Figure 16) were 0.056-0.152 as shown in the calibrator curve(Figure.18) with mean of 0.1039.

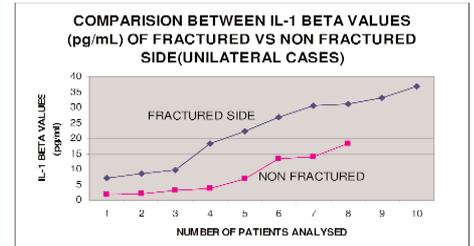


Figure .19: Group A: Comparison between IL-1Beta Values) pg/ml Fractured VS Non Fractured Side

The standard deviations calculated for both the fractured and non fractured side in unilateral cases was found to be 0.0245 and 0.0384 respectively.

The Figure.19 depicts that there is a statistically significant (p=0.003) difference between the concentration of IL-1Beta on

the fractured as compared to the non fractured side in unilateralfracture cases.

For Group B (Bilateral Fracture Cases) the mean value of optical density(OD) as depicted in Figure.20 was calculated for the right side of TMJ (Bilateral cases) was 0.1123 whereas the

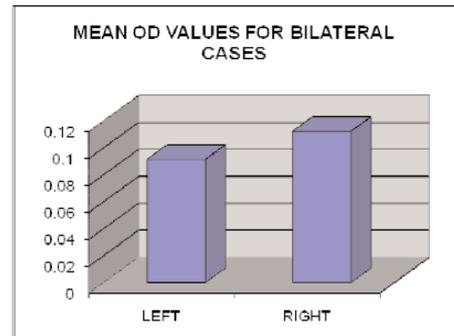


Figure.20: Group B:Mean OD Values for Bilateral Cases

mean value calculated for the left side of TMJ (Bilateral cases) was 0.0915.The standard deviation values calculated were found out to be 0.0579 and 0.0503 respectively.

The Figure.20 and 21 clearly illustrates that the mean OD values and their corresponding IL-1Beta concentrations for both the right and left side cases are almost the same but with a slight increase on

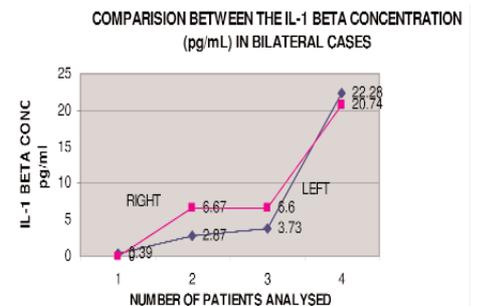


Figure.21: Group B: Comparison between IL-1Beta Concentration (pg/ml) in Bilateral Cases

the right side.

The p value was calculated by unpaired student t test for both unilateral and bilateral cases. There was a statistically significant result obtained with $p=0.0030$ in unilateral cases (Group A).

The p value calculated for the bilateral cases (Group B) was found to be 0.6078, suggesting that the results were statistically not significant, (if $p<0.05$ then statistically significant result).

DISCUSSION

It has been suggested by many authors that disc displacement or tearing, acute synovitis, TMJ ankylosis, traumatic arthritis, or effusion can develop after facial trauma [8,9,10]. A study reported that history of facial trauma was closely related to frequency and intensity of TMJ pain [10].

Three mechanisms of injury leading to degenerative TMJ disease have been proposed by Milam and Schmitz [11]: 1) direct mechanical injury 2) hypoxia-reperfusion injury 3) neurogenic inflammation. Neurogenic inflammation hypothesis as proposed by Milam & Schmitz has been the mainstay of our study as it suggests that inflammatory cytokines like IL-1 Beta detected in the SF might be feasible to diagnose the changes associated with TMJ internal derangements, especially cartilage degeneration and potential predisposition to inflammatory arthritis of TMJ after macrotrauma to the TMJ.

Neuropeptides released from activated peripheral nerve terminals because of mechanical stress -trauma into the surrounding tissue can evoke an inflammatory response. Substance P and calcitonin gene-related peptide are examples of neuropeptides [12] that can evoke an inflammatory response ie activation of proinflammatory cytokines (IL-1 Beta, TNF alpha) by the local cell population. These cytokines may in turn stimulate the production, release and /or activation of matrix degrading enzymes as well as activate arachidonic acid catabolism (prostaglandins and leukotrienes). Prostaglandins such as PGE2 may sensitize peripheral nerve terminals in the region leading to a continued release of proinflammatory neuropeptides with normal function of the joint. This may potentially lead to self perpetuating cycle that can amplify the inflammatory responses that are evoked by this mechanism [11].

In our study we have estimated the levels of IL-1 Beta cytokine synthesized and activated as hypothesized by this mechanism. These neuropeptides are typically found in the central and peripheral terminals of c-fibre neurons. 70 % to 80% of SP and cGRP synthesized by C-fibre neurons is transported to peripheral rather than the central terminals and this axonal transport to peripheral terminals is increased in neurons supplying inflamed tissue. Nerve terminals containing these proinflammatory neuropeptides (SP and cGRP) have been detected in various tissues of TMJ complex [13]. Highest density of these neurons is found in the anterior aspect of the capsular ligament and in the retrodiscal tissue.

Samples of lavage fluid from human TMJ's have been analyzed for the presence of neuropeptides, and substance P, cGRP, substance Y [12]. The concentrations of these neuropep-

tides in the TMJ have been reported to be substantially higher than those observed in other articular joints [11]. This estimation is consistent with immunohistochemical studies that have demonstrated a higher density of proinflammatory neuropeptides containing neurons in the TMJ compared with other studied body joints [11].

It is likely that all three mechanisms - direct mechanical injury, hypoxia reperfusion injury and neurogenic inflammation - are involved in the degenerative process affecting the TMJ. The mechanisms include the synthesis and activation of proinflammatory cytokines (IL-1 beta, IL - 6, TNF alpha), enhanced arachidonic acid catabolism with the production of prostaglandins and leukotrienes and synthesis and activation of matrix degrading enzymes (collagenases, stromelysins) [11].

Considering mechanical injury to the TMJ, synovial pathology can be explained as follows. Overloading to the TMJ directly or indirectly, beyond limitation of functional adaptability may cause TMJ disease such as osteoarthritis, internal derangements, synovitis, joint pain and adhesions. Joint overloading causes synovial fluid and tissue changes including proteoglycan degradation, increased inflammatory mediators [1].

In our study we have found statistically significant levels of IL-1 Beta in TMJ synovial fluid aspirates on the unilateral fracture cases - Group A and detectable levels of IL-1 Beta by spectrophotometer in bilateral fracture cases - Group B. A similar study was conducted which performed biochemical analysis of (PGE2 and LTB4), and detected considerable amount of prostaglandin E2 and leukotriene B4 in the synovial fluid. This Study concluded that trauma could be one of the etiologic causes for TMJ disorders [1].

Many related factors of TMD have been suggested, including neuromuscular disharmony, developmental disharmony, psychological stress, maltreated restoration as well as oral habits like bruxism. It is possible that if trauma occurred in the TMJ combined with these many factors there will be over loading beyond the healing ability of TMJ and eventually, TMD [1].

In our study we have estimated the concentrations of IL-1 beta in an acutely traumatized TMJ. The concentration of IL-1 beta in TMJ SF after mandibular trauma in both unilateral fracture (9- 83 pg/ml) -Group A and bilateral fracture-Group B (Right side fracture= 9-28 pg/ml & Left side fracture =0-9 pg/ml) cases. In both the groups the concentrations have been found to be above the detectable levels by the enzyme linked immunoassay technique (pg/ml). These results suggest the impact of trauma on the TMJ.

Our study excludes those patients with history of underlying degenerative joint disease like osteoarthritis, internal derangements. Proinflammatory cytokines are not a part of the normal constitution of healthy TMJ synovial fluid, They are rarely found in normal healthy joints.

There has been a study on co-expression of IL-1 Beta and TNF alpha in synovial tissue of TMJ with internal derangement in which normal control groups were taken and they found the average concentration of IL-1 Beta in normal healthy volun-

teers to be 0.003+- 0.006 (pg/100 microgram protein)[6]. In another study biochemical markers in the synovial fluid for detecting intraarticular inflammation and early cartilage degradation of the TMJ were examined in patients with internal derangement or osteoarthritis and normal volunteers. In normal control group the IL-1 beta concentration was found to be (76.7+- 95.3 pg/100 microgram of SF protein).

In this study we have considered the non fractured side TMJ in unilateral fracture group as a normal healthy joint. The IL-1 Beta levels have been in the range of 1-13 pg/ml (0-9 pg/ml according to the calibrator series) which is extremely low but still in detectable range by Enzyme-linked immunosorbent assay technique except in one case where the levels are found to be 18.35 pg/ml. The above mentioned findings suggests that IL-1 Beta can be detected in a healthy TM Joint as a consequence of trauma to the mandible. The results also infer that macrotrauma may cause slight degree of transmission of mechanical stresses to the non fracture side also in unilateral fracture cases apart from transferring a greater magnitude of mechanical stresses to the fracture side .

Production of IL-1 in synovial macrophages and synovial fibroblast and chondrocytes also may induce the release of proteinases that destroy the joint cartilage.

A study which has identified the proteinases by zymography in the TMJ SF to be MMP's[7]. MMP's are considered to regulate TMJ tissue remodeling under normal physiologic conditions ,but in exacerbation of condition, the enzyme may cause the degradation of collagen and proteoglycan molecules, which constitute the articular cartilage[7].

IL-1 Beta has been noted by researchers to be absent in diseases involving local Inflammation. This does not exclude the possibility that IL-1 Beta is involved in local joint inflammation, but it may rather indicate that rapid turnover or consumption may occur within the joint cavity. IL-1 beta receptors that bind IL-1 and inhibit its action have been found in experimental rheumatoid arthritis. Soluble IL-1 receptors have yet to be found in human SF[13].

In group B of the 4 bilateral mandibular fracture cases analyzed the concentration of IL-1 Beta were found to be (Right side fracture=9-28 pg/ml and Left side fracture =0-9pg/ml) with a mean of (left side fracture=.0915 and Right side fracture =.1123) and no statistical significance was found (p=0.6078) . In all these cases the concentrations of IL-1 Beta were above detectable range. There was a higher concentration of IL-1 beta found on the right side fracture cases suggesting that trauma can lead to generation of mechanical stresses within the TMJ which gets equally distributed on both sides. A greater propensity of mechanical stress concentration for the right side was observed in our study. Results also suggest that the concentration of IL-1 beta in 3 out of 4 bilateral cases are considerably increased when the fracture is in subcondylar region of the mandible. Although the sam-

ple size is too low to comment on the findings. The result indicate that macrotrauma to the TMJ can lead to an increase in the proinflammatory mediators

In our study on histomorphologic cell examination considerable number of degenerated epithelial, proteinaceous material, casts and few inflammatory cells (lymphocytes) were seen suggestive of inflammatory changes in the TMJ post trauma. A study has been conducted which Correlated the amount of Joint Effusion on T2 weighted image of TMJ on MRI scan and the concentrations of total protein and IL-1 Beta, IL-6, IL-8, and TNF Alpha in the synovial fluid Joints with JE had on average significant higher concentrations of total protein., Furthermore there was significant correlation between JE grade[14] and concentration of total protein ,IL-6, IL-1 Beta, IL-8. The study concluded that JE may contain the released products when there is pronounced synovitis. It is probably composed of high concentrations of total protein with inflammatory cytokines[14].

Similar study has been conducted on Role of facial trauma as a possible etiology in TMJ disorders and they found out considerable amounts of PGE2 and Leukotriene B4 ,degenerative cartilage and cells in the SF of patients suggesting that the inflammatory and degenerative changes of TMJ can develop after macrotrauma[1].

In general IL-1 Beta, TNF alpha, IL-6 which are proinflammatory cytokines ,are induced by inflammatory stimuli. These cytokines have been detected in the SF from not only diseased knee joints but also TMJs with internal derangements and osteoarthritis. The main role of IL-1 Beta is destruction of the cartilage by degradation of proteoglycan[10,15].

The results of our study suggests that the IL-1 beta could be possible marker to assess early micro environmental inflammation or cartilage degradation in the TMJ. The results are in concomitance to a study that suggests an increase in the levels of cytokines (IL-1 Beta and IL-6) and active forms of MMP's could be potential catabolic markers of cartilage degradation in the TMJ [7]. Our results are encouraging and have been promising. Still Further research into Temporomandibular Joint which is an ever perplexing subject may evolve newer horizons in the pathogenesis ,diagnosis and treatment of Temporomandibular Joint disorders.

CONCLUSION

The changes in articulating joints caused by traumatic injuries appear to be diverse and unpredictable, and their causes have begun to be addressed only recently.

Macrophage like cells (synovial cells/synovial macrophages/synovial fibroblast) are key immunoregulators that layer the synovial membrane and are likely to play a pivotal role in initiation of inflammation and subsequent tissue destruction. During this process the activated macrophage like cells release cytokines such as IL-1 Beta ,TNF alpha, IL-6 which may in turn stimulate the production ,release and /or activation of matrix degrading enzymes as well as activate both phospholipase A2 , lipoxygenase and cyclooxygenase pathway

leading to production of prostaglandins and leukotrienes. PGE2 may sensitize peripheral nerve terminals in the region leading to a continued release of proinflammatory neuropeptides with normal function of TMJ. This may potentially lead to a self-perpetuating cycle that can amplify the inflammatory response.

Complications of trauma to TMJ are far reaching in their effects and not always immediately apparent. Disturbance of occlusal function, deviation of mandible, internal derangements of the TMJ and ankylosis of the joint with resultant inability of jaw movements are all sequelae of this injury. If aggressive functional physical therapy and long term follow up is performed, the favorable functional recovery of TMJ can be obtained.

Our study concludes that the inflammatory and degenerative changes of TMJ can develop after macrotrauma especially to mandible. IL-1 beta could be possible marker to assess early micro environmental inflammation or cartilage degradation in the TMJ. The production of IL-1 beta by synovial macrophages and its role in inflammatory and degenerative process in the TMJ warrants further investigation. The study suggests that Arthrocentesis is a simple and minimally invasive diagnostic as well as therapeutic procedure, with little risk of complications. Lavage of superior joint space with saline exerts its effects via its ability to eliminate joint effusion. Various proteins and biochemical mediators causing pain are washed away, healthy synovial fluid production is promoted and hydraulic pressure release adhesions allowing some component of repair & adaptation.

The study also suggests that Trauma can be a possible etiologic factor in cartilage degeneration and biochemical and intrarticular pathology. Clinicians should recognize the etiologic importance of the macrotrauma to the mandible. Long term follow up and evaluation of the TMJ as well as adequate treatment is required for patients with macrotrauma to the mandible.

REFERENCES

1. Yun.P.Y, Kim Y.K : "The role of facial trauma as a possible etiological factor in temporomandibular joint disorder " J Oral Maxillofac Surg 2005; 63(11): 1576-83.
2. Alling C, Osborn D (1998):Maxillofacial Trauma. Philadelphia, PA , Lea and Febiger p 238.
3. Segami N, Nishimura M, Suzuki T : "Co-expression of interleukin-1beta and tumor necrosis factor alpha in synovial tissues and synovial fluids of temporomandibular joint with internal derangement: comparison with histological grading of synovial inflammation" J Oral Pathol Med 2002;31(9):549-57.
4. Kubota E, Kubota T, Matsumoto J : "Synovial fluid cytokines and proteinases as markers of temporomandibular joint disease" J Oral Maxillofac Surg 1998;56(2):192-8.
5. Shafer DM ,Assael L,White LB, et al:"Tumor necrosis factor alpha as a biochemical marker of pain and outcome in temporomandibular joints with internal derangements." J Oral Maxillofac Surg 1994;52:786.
6. Fu K, Ma X, Zhang Z ,et al : " Tumor necrosis factor in synovial fluid of patients with temporomandibular joint disorders" J Oral Maxillofac Surg 1995; 53:424.
7. Kubota E, Imamura H, Kubota T et al : "Interleukin 1 beta and stromelysin (MMP3) activity of synovial fluid as a possible markers of osteoarthritis in temporomandibular joint" J Oral Maxillofac Surg 1997;55:20.
8. Pullinger AG, Seligman DA: "Trauma history in diagnostic groups of temporomandibular disorders" Oral Surg Oral Med Oral Pathol 1991;71:529.
9. Howard P, Hatsell Charles P, Guzman Herbert : "Temporomandibular joint injury potential imposed by the low velocity extension -flexion maneuver" J Oral Maxillofac Surg 1995;53:256-262.
10. Plesh O, Gansky SA: The relation between chronic facial pain and history of trauma and surgery Oral Surg 1999;88:16.
11. Milam S, Schmitz John P : " Molecular biology of temporomandibular joint disorders: proposed mechanism of disease" J Oral Maxillofac Surg 1995; 53:1448-1454.
12. Holmlund A, Ekblom A, Lind J: Concentrations of neuropeptides substance P, neurokinin A, calcitonin gene related peptide, neuropeptide Y and vasoactive intestinal polypeptide in synovial fluid of the human temporomandibular joint .Int .J.Oral Maxillofac .Surg 1991;20:228-231.
13. Sandler A, Buckley M, Braun T : "Correlation of inflammatory cytokines with arthroscopic findings on patients with temporomandibular joint internal derangement" J Oral Maxillofac Surg 1998;56:534-543.
14. Segami N, Nishimura M, Suzuki T, Murakami K : "Comparison of concentration levels of proinflammatory cytokines and total protein in synovial fluid of the temporomandibular joint with internal derangements and osteoarthritis" Oral surg Oral Med Oral Pathol 2002;94(4) :515-21.
15. David E. Frost, Barry D Kendell: "The use of arthrocentesis for treatment of Temporomandibular joint disorders. J. of Oral Maxillofac Surgery 1999.

AUTHORS

1. Dr. Anuj Bhargava, MDS

Reader

Department of Oral and Maxillofacial Surgery
Bhabha College of Dental Sciences
Bhopal-462026
Madhya Pradesh

2. Dr. Meenaxi Umarani, MDS

Professor

Department of Oral and Maxillofacial Surgery
KLE V.K Institute of Dental Sciences
KLE University
Belgaum- 590010
Karnataka

