Temporomandibular joint osteoarthrosis

Histopathological study of the effects of intra-articular injection of triamcinolone acetonide

Issa K. Haddad, *BDS, MSc.*

ABSTRACT

Objectives: The aim of this study was to investigate the histopathological changes of human temporomandibular joint osteoarthrosis following intra-articular injection of triamcinolone acetonide, and compare the results to these of control group.

Methods: One hundred and forty nine patients who complained of temporomandibular joint dysfunction underwent non-surgical treatment for at least 6 months. Twenty three patients had high condylectomy as a treatment. Forty four patients received two intra-articular injections of triamcinolone acetonide. Histological study was carried out on 44 specimens from 41 patients, the histological changes were recorded for each specimen.

Results: Patients who received temporomandibular intra-articular injection of triamcinolone acetonide showed damage to the fibrous layer (100%), to the cartilage (64%) and to the bone (42%). In those cases treated by condylectomy there were changes in the fibrous layer (100%), in the cartilaginous layer (68%) and to the bony layer (32%). Analysis of the results showed that the changes seen in the steroid (triamcinolone acetonide) group were more obvious than the changes in the non-steroid group.

Conclusion: The result of this study indicated that intra-articular injection of steroid (triamcinolone acetonide) into human osteoarthritic temporomandibular joints acts as a lytic agent.

Keywords: Osteoarthrosis, temporomandibular joint, traimcinolone acetonide.

Saudi Medical Journal 2000; Vol. 21 (7): 675-679

Intra-articular injection of steroids into the temporomandibular joint (TMJ) space is not a recent subject. Horten[1] in 1953, was the first who reported this procedure which was based on the work of Hollander et al[2] in which they described the effect of intra-articular injection of hydrocortisone in various joint disorders. Since then, a number of papers have reported varying degrees of success. The pathology and biochemical changes associated with this type of treatment remain ill-defined.[1,3]

If there is evidence of osteoarthrosis (OA), intra-articular injection of steroids may be the preferred treatment rather than surgery, and also if the non-surgical treatment failed to relive the pain for the first 6 months.[4,5] Triamcinolone acetonide which has been used for intra-articular injection is very slowly absorbed from the injection sites.[2] The dose ranges between 2 to 40 mg, depending upon the size of the joint injected.[6] In cases of TMJ, the dose is usually 10 mg.[7] Triamcinolone acetonide is a safe drug, although anaphylactic shock following injection of triamcinolone acetonide has been reported.[8,9] A repeat injection is occasionally used but the third injection should be used with caution as the expectation of further improvement decreases with successive injections.

A literature search for histopathological changes induced when animal TMJ injected with steroids

From the Department of Oral and Maxillofacial Surgery, King Hussein Medical Center, Royal Medical Services, Amman, Jordan.

Received 31st August 1999. Accepted for publication in final form 4th April 2000.

Address correspondence and reprint request to: Dr. I. K. Haddad, PO Box 1182, Zarka, Jordan. Fax: +962 (6) 533 1362 e-mail: hadaesa55@hotmail.com

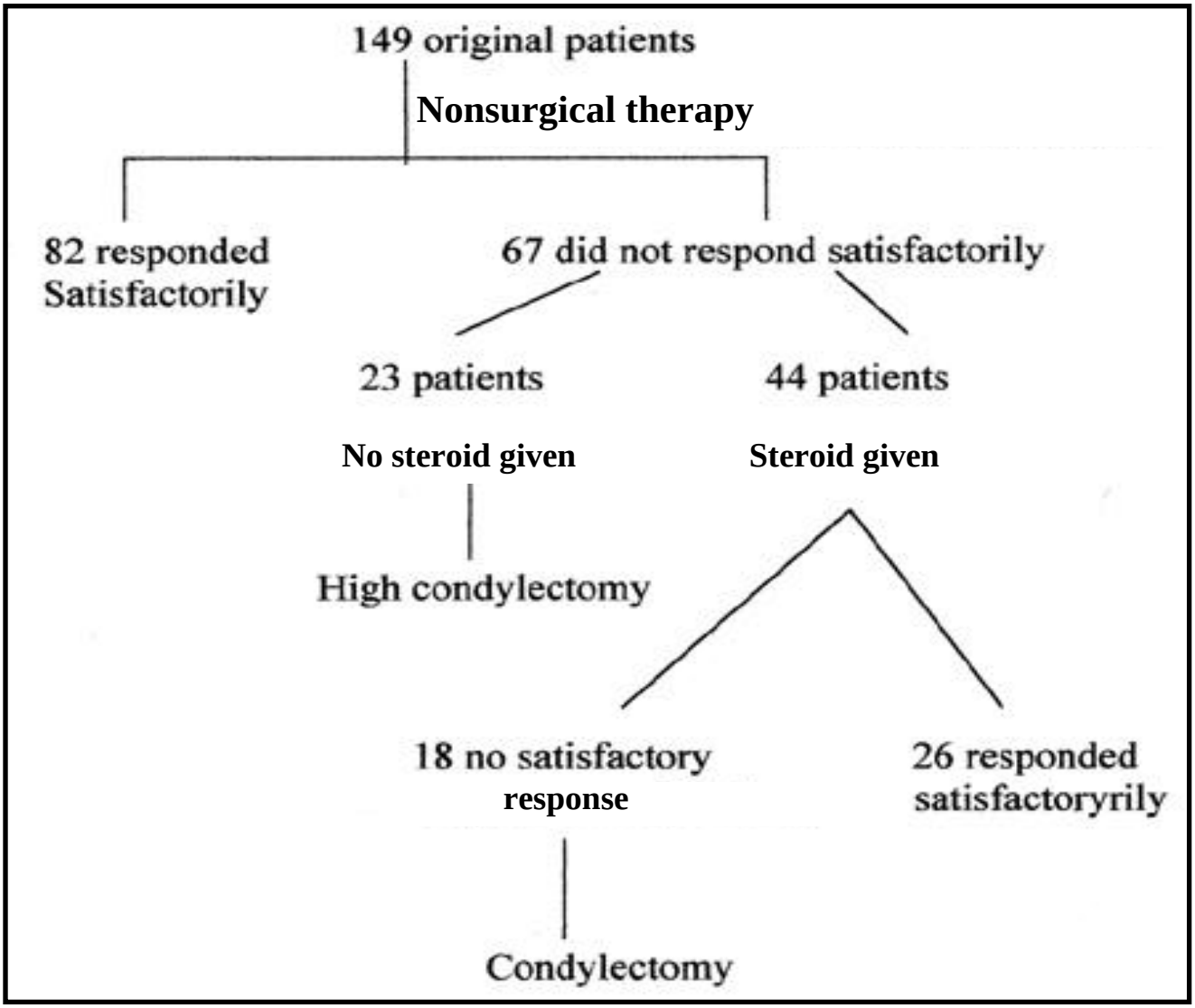

Figure 1 - Scheme represents the materials and methods of this investigation.

Figure 2 - Mild damage to the fibrous layer.

Figure 3 - Complete loss of the fibrous layer.

Figure 4 - Splitting and cracking in the cartilaginous layer.

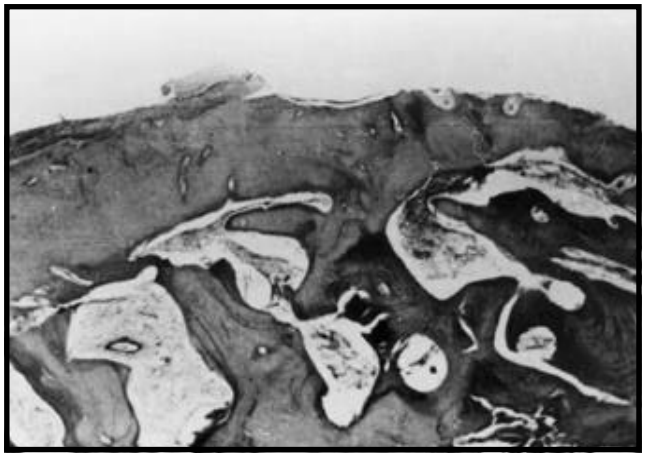

Figure 5 - Complete loss of the cartilaginous layer.

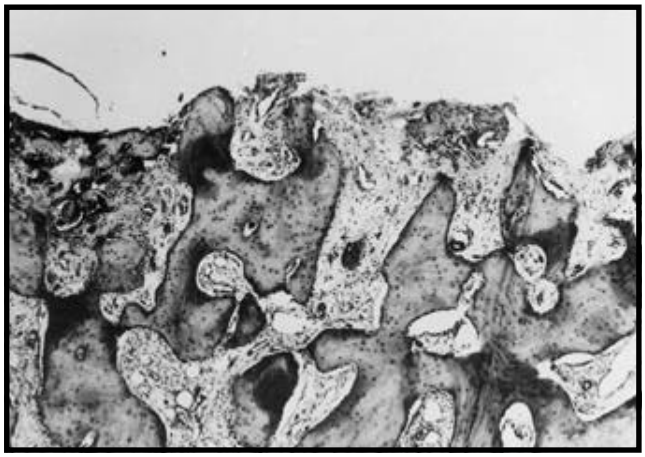

Figure 6 - Gross damage to the bony layer with cystic formation.

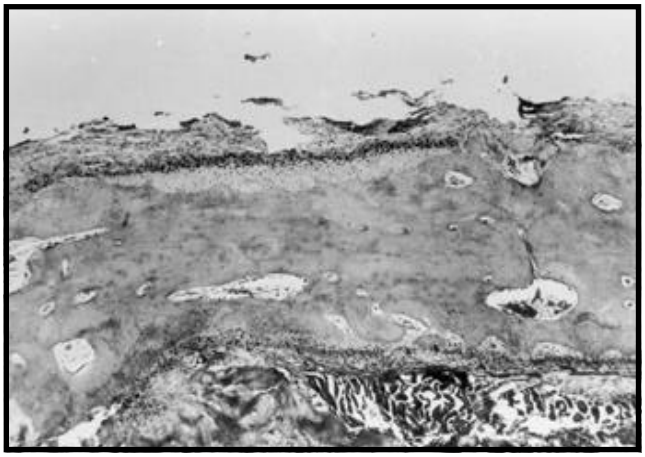

Figure 7 - Damage and clefing in the fibrous layer.

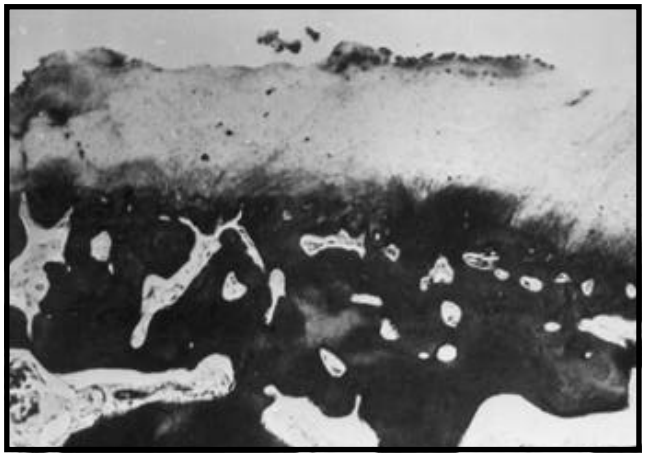

Figure 8 - Complete loss of the fibrous layer.

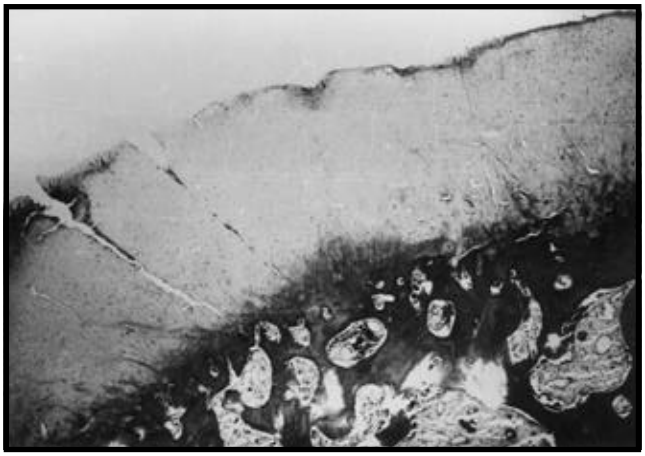

Figure 9 - Splitting and cracking of the wole thickness of the cartilaginous layer.

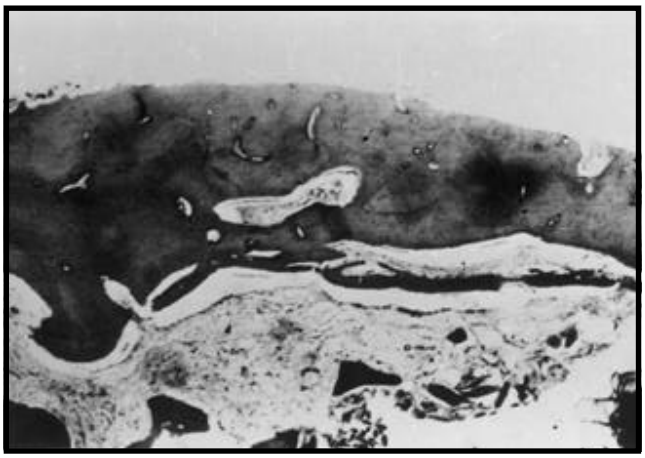

Figure 10 - Complete loss of the cartilaginous layer.

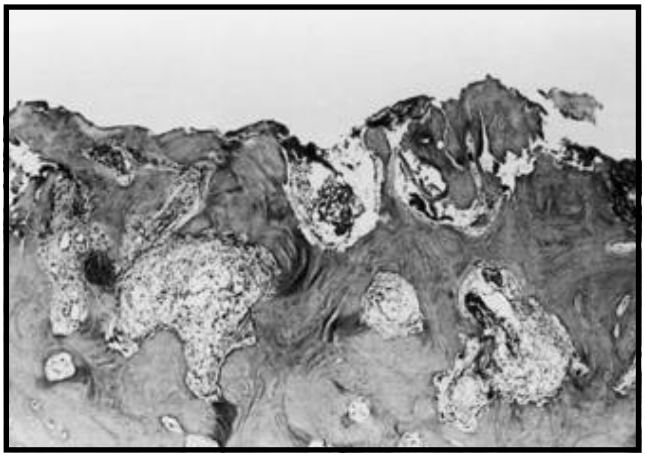

Figure 11 - Gross damage and cystic formation in the bony layer.

intra-articulately, has been reported. Histological changes of human TMJ-OA, following intra-articular injection of steroids did not yield any published information up to the time of writing this report. This study could very well be the first investigation of the histological changes of human TMJ-OA following injection of triamcinolone acetonide intra-articularlly. The results were compared to those of a group of patients with TMJ-OA who did not receive steroids injection.

Methods. The subjects of this study consisted of 149 male and female patients who complained of temporomardibular joint "osteoarthrosis" (TMJ-OA). Their ages ranged from 27 to 79 years, with an average of 48 years. Osteoarthrosis (OA) was diagnosed on clinical basis as described by Gray et al.[8] Radiological examination revealed no obvious changes in either transpharynyeal or panoramic radiographs.

All cases underwent non-surgical treatment for at least 6 months, which included occlusal adjustment, bite raising, replacing the missing teeth, and physiotherapy. Sixty seven patients who did not improve by non-surgical treatment were divided into two groups.

The first group (non-steroidal) comprised of 23 patients (25 joints), whose ages ranged from 27 to 77 years, with an average of 48 years. High condylectomy was performed on each group.

The number of patients in the second group (steroidal) was 44, their ages ranged from 26 to 79 years, with an average of 51 years. All received 2 intra-articular injections of triamcinolone acetonide (20 mg) at two-monthly intervals. Eighteen patients did not respond satisfactorily to the injections, and were therefor treated by high condylectomy.

The surgical specimens comprising 44 tissues (41 patients) were fixed in 10% buffered formal saline. Each specimen was cut into 5-8 pieces in the sagittal plane and decalcified in formic acid. An end point of decalcification was determined by radiography. At least one section from each specimen was stained with hematoxylin and eosin, and examined by light microscope. The presence and degree of histological changes were recorded on all layers of TMJs. Summary of all patients is seen in Figure 1.

Results. ***Non-steroid group.*** The joints that received no intra-articular injections of triamcinolone acetonide, showed damage to the articular fibrous zone in all specimens. This damage ranged from mild damage in 4 joints (Figure 2), to complete loss of the articular fibrous zone in 21 joints (Figure 3). Damage to the cartilaginous layer was found in 17 joints (68%). This effect ranged from erosion in 5 specimens, splitting and cracking in 3 joints (Figure 4), to complete loss of the cartilaginous zone in 9

specimens (Figure 5). Subchondral bony layer was affected in 8 joints (32%). This damage ranged from erosion in 6 specimens,to cystic formation in other two specimens (Figure 6).

Steroid group. All specimens that received intra-articular injection of triamcinolone acetonide showed damage to the fibrous layer. This ranged from clefting in 3 joints (Figure 7) to complete loss of this zone in 16 specimens (Figure 8). The cartilaginous layer was affected in 14 specimens (74%). Erosion was found in 3 joints, splitting or cracking of the whole thickness of cartilage in two joints (Figure 9), and partial or complete loss of this layer in 9 joints (Figure 10). Damage to the subchondral bony layer was found in 8 specimens (42%). Erosion was demonstrated in 4 specimens, and cystic formation was recorded in the other 4 specimens (Figure 11).

Discussion.

The response of animal joints to steroids injection intra-articularly revealed significant histological changes. The fibrous layer had been lost over the whole of the condylar head.[3] The response of the cartilaginous zone was erosion of the superficial strata, linear splitting along the longitudinal axis, clefts, and shreds of cartilage were found lying within the articular space. Osteoporosis was noticed underneath the damaged cartilage, extending from the subchondral area down into the condylar neck.[6,10]

Up to this date, there is no study on the histopathological changes of human TMT-OA injected intra-articularly with steroids. This study undertakes what is believed to be the first investigation into histological changes of human TMT-OA following intra-articular injection of triamcinolone acitonide. The results were compared to a similar series of human TMJ-OA in which only non-surgical treatment had been given, without intra-articular injection of steroids.

The results of this study revealed higher destruction to all layers of the joints that received intra-articular injection of triamcinolone acetonide, when compared to the group of joints, which received no steroid injections. This finding firmly supports the hypothesis; intra-articular injection of steroids acts in joints suffering from OA as a lytic agent with the potential to produce a pharmacological arthroplasty.

In this study the complete loss of fibrous layer was found in 84% (16 specimens), other studies showed the loss of the fibrous layer in all cases.[3] This is simply because the joints in this investigation received only 2 injections of steroids, meanwhile the joints in Poswillo's study received 6 injections of steroids. This indicates the direct effect of steroids on the articular fibrous layer. Continued pain in proportion of cases, in spite of intra-articular injection of steroids, indicates that steroids therapy alone may be not sufficient to control the osteoarthotic changes in those joints, and surgery could not be avoided in some cases.

Further results could be obtained from this investigation. As this study is limited to a microscopic level, electron microscopic investigation would be very helpful in gaining further information and more detailed results. Clinical and radiological follow up, which are not included in this study, could be the subject of a further paper.

References

1. Horten CP. The Treatment of Arthritic Temporomandibular Joints by Intra-articular Injection of Hydrocortisone. Oral Surg 1953; 6: 826-829.
2. Hollander JL, Brown EM, Jessar RA, Brown CY. Hydrocortisone and Cortisone injected into arthritic joints: Comparative effect of and use of hydrocortisone as a local antiarthritic agent. JAMA 1951; 147: 1629-1236.
3. Gibson T, Burry HC, Poswillo D, Glass J. Effect of intra-articular Corticosteroid injections on primate cartilage. Ann Rheum Dis 1976; 36: 74-78.
4. Goss AN. Towards an international consensus on temporomandibular joint surgery. Int J Oral Maxillofac Surg 1993; 22: 78-81.
5. Derendorf H. Pharmacokinetics of triamcinolone acetonide after intravenous, oral, and inhaled administration. J Clin Pharmacol 1995; 35: 302-305.
6. Silbermann M, Moredohvich D, Toister Z, Azaria N. Mechanisms involved in mandibular condylopathy secondary to intra-articular injections of glucocorticoids. J Oral Surg 1978; 36: 112-117.
7. Gray RJM, Davies SJ, Quayle AA. A clinical approach to temporomandibular disorders: Treatment planning, general guidelines and case histories. Br Dent J 1994; 171: 171-178.
8. Poswillo D. Experimental investigation of the effects of intra-articular hydrocortisone and high condylectomy on the mandibular condyle. Oral Surg Oral Med Oral Path 1970; 30: 161-173.
9. Larsson L. Anaphylactic shock after administration of triamcinolone acetonide in a 35 year old female. Scand J Rheamtol 1989; 18: 441-444.
10. Creamer P, Flores R, Hochberg MC. Management of