CASE REPORT

Severe temporomandibular dysfunction and joint destruction after intra-articular injection of triamcinolone

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BACKGROUND: Steroid injections into joints are frequently used to control symptomatic pain. Risks associated with intra-articular steroid injections are not well documented.

METHODS: We report the case of a 29-year-old woman who was referred to a dental surgeon because of a suspected relationship between persisting chronic back pain and an arthrosis of the temporomandibular joint (TMJ).

RESULTS: The dental surgeon diagnosed capsulitis of the right TMJ and injected 40 mg triamcinolone into the joint. Within 4 months the patient developed progressive pain and trismus of the right TMJ and the intra-articular injection was repeated. An occlusal splint slightly improved the patients' symptoms but induced crepitus. Magnetic resonance imaging revealed a disk dislocation in the right TMJ and severe necrosis of the condyle. The patient had persisting pain and ankylosis. Surgical restoration of the TMJ revealed a bony apposition in the fossa deformed with the socket of the joint, extensive medial erosion of the condyle and complete destruction of the disk.

CONCLUSION: This case report supports earlier observations that intra-articular glucocorticoid injections, if used in a wrong way, may cause severe destruction of a joint.

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Description of the case

Intra-articular corticosteroid injections are frequently used in patients with arthritis to achieve symptomatic pain relief. A 29-year-old woman was referred to a maxillofacial surgeon by an orthopedist who assumed that the temporomandibular joint (TMJ) could be responsible for the patient's chronic back pain. A capsulitis of the right TMJ was diagnosed but the joint had a normal range of motion and was painless. There was no history of arthritis or trauma concerning the TMJ. An intra-articular injection with 40 mg triamcinolone (Triam inject®, Lichtenstein Pharmaceuticals, Muelheim, Germany) slightly improved the back pain but the patient complained about slowly progressing pain and trismus of the TMJ. Four months later a second intra-articular injection with triamcinolone 40 mg was performed. As there was no relief of the symptoms, the patient consulted another dentist who diagnosed craniomandibular dysfunction of the right TMJ. A splint slightly improved the trismus and the pain but the patient had continuously progredient crepitus in the joint. Magnetic resonance imaging revealed anterior disk dislocation and severe necrosis of the condyle. Because of persisting pain and ankylosis in the TMJ the patient was referred to an oral and maxillofacial surgeon. The patient had a bony ankylosis of the joint with pain, clinically imposing with complete inhibition of the gliding movement and limited rotation within the joint, the maximum mouth opening was 24 mm. Functional X-ray revealed a bony apposition of the tuberculum articulare (Fig. 1). Computed tomography images showed bony enclosure of the condyle and extensive medial condylar erosion (Fig. 2). At surgery the bony apposition was found completely grown together with the socket of the joint (Fig. 3). The disk was found to be completely destroyed (Figs 4, 5) and had to be removed. The articular surface of the condyle that was covered with cartilaginous material was remodeled. Postoperatively, the maximum mouth opening improved to 34 mm without pain and the articular gliding movement was restored.

Discussion

Intra-articular corticosteroids have proved to be useful in alleviating pain, swelling and dysfunction at inflammatory diseases if indicated. In the present case it is very difficult for the authors to understand the initial

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Figure 1 Functional radiograph before surgery. The both outer X-rays show the joints in a closed position. The inner X-rays show the opened position. However, at the right temporomandibular joint (TMJ) the gliding movement is totally inhibited by a bony apposition (see arrows).



Figure 2 Computed tomography (CT)-image with severe destruction of the right temporomandibular joint (TMJ). Apposition of bone-like a cap is shown (black arrows). Furthermore, there is a deep defect on the condyle's surface (white arrow).

indication for a steroid injection into the TMJ as well as the diagnosis of capsulitis as there was neither pain nor limited range of motion within the injected TMJ. However, local side effects of corticosteroids such as destruction of articular cartilage, infections and progression of a pre-existing joint disease are welldocumented (1, 2). Three etiologic aspects may be responsible for the fatal joint destruction observed in the present case: First, improper injection technique might have caused a local infection. Secondly, as Triam inject® is a crystalloid suspension of triamcinolone, precipitation of crystals might have injured periosteum and cartilage. For intra-articular injection of small joints a water-soluble solution should be preferred (3) and the recommended intra-articular dose not exceeding 10 mg should be followed (4). Thirdly, corticoster-



Figure 3 Bony apposition covering the lateral part of the condyle and fused with the temporal component (arrow).

oid treatment may be associated with development of aseptic necrosis. A strong correlation exists between the administered dose of corticosteroid treatment and development of osteonecrosis (5). Haddad et al. (6) have recently demonstrated in a clinical study comparing histopathologic changes in TMJ treated with vs. 185



Figure 4 Joint cavity opened. The disk is replaced by fibrotic tissue (arrow).

without steroid injections that triamcinolone can act as a lytic agent. In the present case, the histologic report of the pathologist confirmed osteonecrosis and reactive osteoanagenesis.

This case report supports earlier observations that intra-articular glucocorticoid injections, if used in a wrong way, may cause severe destruction of a joint.

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Figure 5 Fibrotic tissue extirpated and articular surface of the condyle covered with a thin layer of soft tissue (according to the histologic examination of the pathologist: osteonecrosis and reactive osteoanagenesis).

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