Use of the hyaluronic acid to prevent bursitis after percutaneous treatment of the rotator cuff calcific tendinitis.

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Aims and objectives

The aim of this work is to evaluate the role of Hyaluronic Acid (HA) in the subacromion-subdeltoid bursa (SSB) to prevent the persistent bursitis as a complication of post-US-guided percutaneous treatment in patients with rotator cuff calcific tendinitis (RCCT).

RCCT refers to the intratendinous deposition of calcium, predominantly hydroxyapatite, a commonly seen condition, with prevalence in the III, IV, and V decade of life, occurring in up to 20% of painful shoulders. It is asymptomatic in a variable percentage of 3-30%.

The supraspinatus tendon (80% of cases), followed by the infraspinatus (15% of cases) and subscapularis (5% of cases) tendons, is the most commonly affected cuff tendon.

This pathological condition is a dynamic process that evolves through four stages: pre-calcific, calcific, resorptive, and post-calcific. In the pre-calcific stage, microtraumatic factors associated with a local decrease in blood supply can lead to intratendinous fibrocartilaginous metaplasia, with resulting calcification. The subsequent calcific phase is considered as a resting period, generally asymptomatic. Eventually, triggered by unknown factors, there is resorption of the deposit, accompanied by vascular invasion, the migration of phagocytes with dissolution of the calcific focus (resulting in a "toothpaste" appearance of the calcific deposit), dispersion of its contents within the tendon and in the SSB, and edema from intratendinous pressure, such that the condition becomes symptomatic with pain, even violent, with increased night-time intensity associated with reduction of joint motion. After resorption, in the post-calcific or reparative phase (from a few months to ten years), fibroblasts restore the normal tendinous collagen pattern.

Asymptomatic calcifications should not be treated, instead treatment becomes necessary and urgent in the phase of resorption with the occurring of hyperalgesic crisis with severe functional impairment.

Images for this section:
Fig. 6: Anterior view
Methods and materials

The percutaneous treatment of the RCCT is performed under US-guidance using a high-resolution linear probe by two radiologists in conditions of perfect sterility. A preliminary US examination of the shoulder is required to localize the calcification. The anesthesia is performed subcutaneously, along the path of the needles, in SBB and around the calcification. Then two 18 G needles are introduced within the calcification. A 20 ml syringe filled with saline solution is connected to one of the needles. The saline is injected into the calcification and exits from the other needle, getting out the calcific material. This washing is repeated several times, checking the result by US.

At the end of the washing a needle is extracted while the other can be used to introduce HA in the calcified shell, which is subsequently reabsorbed spontaneously, and in the SSB, where is also introduced slow-release corticosteroid to obtain a local anti-inflammatory effect and to prevent the onset of adhesive bursitis. The introduction of HA into the calcification shell also has the purpose of preventing new deposition of calcium.

At the end of the procedure dry ice is applied locally and the patient stays under observation about 30 minutes. A broad-spectrum antibiotic is administered for at least three days, usually starting in the evening previous the treatment.

A cycle of physiotherapy is recommended in order to promote proper recovery of the rotator cuff activity.

HA is a polysaccharide member of the group of glycosaminoglycans. It is a polymer with a very high molecular mass and consists of repeating units of N-acetylglucosamine and glucuronic acid linked together by glycosidic bonds. It is present in the superficial layers of cartilage, the intercellular matrix of the joint capsule, synovial tissue, and synovial fluid. Hyaluronic acid is highly absorbent with visco-elastic properties: viscosity (lubrication) in case of static compressive strength and elasticity (shock-absorbing) in response to dynamic shear and compressive forces. The administration of HA with US-guided intra-articular injection, referred to as viscosupplementation (pure mechanical effect), has the aim to reduce disability and pain by restoring the physiological properties of the synovial fluid and thereby improving articular function.

The intra-articular administration of HA has a role not only in restoring the viscoelastic properties of synovial fluid, but also in stimulating the endogenous production of hyaluronic acid by articular chondrocytes and synoviocytes through the release of products based on hyaluronic acid.
We retrospectively evaluated the clinical outcome of post-US-guided percutaneous treatment in 246 consecutive patients (between May 2011-January 2013) with painful RCCT, splitted into two groups: Group A (127/246) without infiltration of HA; Group B (119/246) with infiltration of HA in the SSB.

Images for this section:

Fig. 4: Equipment: - Two 18 G needles - Inox bowl (to collect the washing fluid) - Sterile saline solution - Syringes (20 ml and 10 ml) - Lidocaine (10 ml) - Steroid (1 ml, 40 mg/ml) - Low molecular mass (<1000 kDa) HA - Plaster - Ice pack.
**Fig. 3:** US-guided puncture of the calcification with two 18 G needles for arthrocentesis so that the tips are faced to enable the continuous washing with isotonic solution up to subtotal dissolution and aspiration of the calcific material.
Fig. 2: Needle in the SSB: injection of corticosteroid and HA.
Fig. 1: A case of bursitis after percutaneous treatment of calcifying tendinitis.
Results

Group A: we observed pain recurrence in 23/127 patients. In 8 there were residual calcifications treated with a second procedure; in the other 15 cases the pain was due to the development of significant bursitis, then re-treated with lavage and US-guided anti-inflammatory infiltration in the bursa.

Group B: the recurrence of pain occurred in 15/119 patients, due to residual calcifications (7 cases), treated with a second procedure, or to the development of significant bursitis (8 cases) re-treated with lavage and infiltration of anti-inflammatory under US-guidance.

Comparing the two groups, the difference of post-procedural bursitis incidence was not statistically significant ($\chi^2 = 1.88; p=0.17$). No significant overall differences were also observed between the two groups regarding the persistence of calcifications.

Images for this section:
Fig. 5: Table of results. Group A: without infiltration of hyaluronic acid. Group B: with infiltration of hyaluronic acid.
Conclusion

The only technical-procedural difference between the two groups of patients was the HA infiltration. Despite the lack of statistical significance, our study underlines a probable role of HA in reducing the occurrence of postprocedural bursitis due to its trophic as well as mechanical action. On the basis of these data and the review of the poor available literature, even requiring further studies with larger number of patients, the procedure seems feasible, since it has no side effects or complications.

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References

