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Calcific tendinopathy of the rotator cuff

Kalcifirajuća tendinopatija rotatorne manšete

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Summary

The calcific tendinopathy of the shoulder is characterized by the presence of macroscopic deposits of calcium hydroxyapatite in the tendons of the rotator cuff, spontaneous resorption of those deposits and, consequently, the tendon healing. The evolution of calcification is divided into 4 phases: formation of the calcium deposits, resting phase, resorption phase and post-calcific stage. The incidence of this condition is, in the general population, 8-20% with the peak of incidence at the ages between 30 and 50. The calcific tendinopathy mostly affects the tendon of the supraspinatus. It affects women more often than men and right shoulder more often than left. The bilateral presentation of calcific tendinopathy occurs from 5 to 23% of patients. Different theories about etiopathogenesis of the calcific tendinopathy were described and discussed. In recent years, there have been few publications presenting chondral metaplasia as an important mechanism. Radiological and ultrasound diagnostics are the most widely used diagnostic techniques. Magnetic resonance imaging and computed tomography are rarely used, just in clinically unclear cases. Conservative treatment includes non-steroidal anti-inflammatory drugs (NSAIDs), subacromial administration of the corticosteroids, physical therapeutic intervention, needle aspiration and lavage. In patients refractory to conservative treatment, surgical treatment, primarily arthroscopy, is indicated. Defining the stage (phase) of calcific tendinopathy determines the treatment method and prognoses the course of the disease. Precisely defining the phase enables avoiding unnecessary long-lasting physical therapy and also provides using the most effective treatment.

Key words: calcific tendinopathy; shoulder; diagnosis; treatment

Sažetak

Kalcificirajuću tendinopatiju ramena karakterizira prisutnost makroskopskih depozita kalcijeva hidroksiapatita u tetivama rotatorne manšete, spontana resorpcija kalcifikata i posljedično cijeljenje tetive. Evolucija kalcifikata podijeljena je u 4 faze: formativna, razdoblje mirovanja, razdoblje resorpcije, te naposljetku postkalcificirajuća faza.

Incidencija kalcificirajuće tendinopatije ramena je u općoj populaciji 8-20%. Najviša incidencija je u odraslih u dobi od 30 do 50 godina. Najčešće mjesto kalcifikata je tetiva supraspinatusa, a rjeđe u ostalim tetivama rotatorne manšete. Žene su pogođene nešto češće od muškaraca, a desno rame nešto češće nego lijevo. Bilateralna prezentacija kalcificirajuće tendinopatije pojavljuje se u 5 do 23% bolesnika. Postoje različite teorije o etiopatogenezi, a posljednjih godina publiciraju se radovi koji govore o hondralnoj metaplaziji. U kliničkoj dijagnostici kalcificirajuće tendinopatije najviše se koristi radiološka i ultrazvučna dijagnostika. Magnetska rezonanca i kompujterizirana tomografija koriste se rijetko kod klinički nejasnih slučajeva. Konzervativno liječenje obuhvaća primjenu nesteroidnih protuupalnih lijekova (NSAID), subakromijalnu aplikaciju steroida, fizikalne terapijske intervencije, aspiraciju iglom i lavažu. U bolesnika refraktornim na konzervativno liječenje, indicirano je kiruško liječenje, prvenstveno artroskopsko.

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Definiranjem stadija (faze) kalcificirajuće tendinopatije određuje se način liječenja, te prognozira tijek bolesti. Točnim definiranjem faza izbjegava se nepotrebna dugotrajna fizikalna terapija, a ujedno omogućava učinkovito liječenje, ovisno o stadiju bolesti.

Ključne riječi: kalcificirajuća tendinopatija; rame; dijagnoza; liječenje

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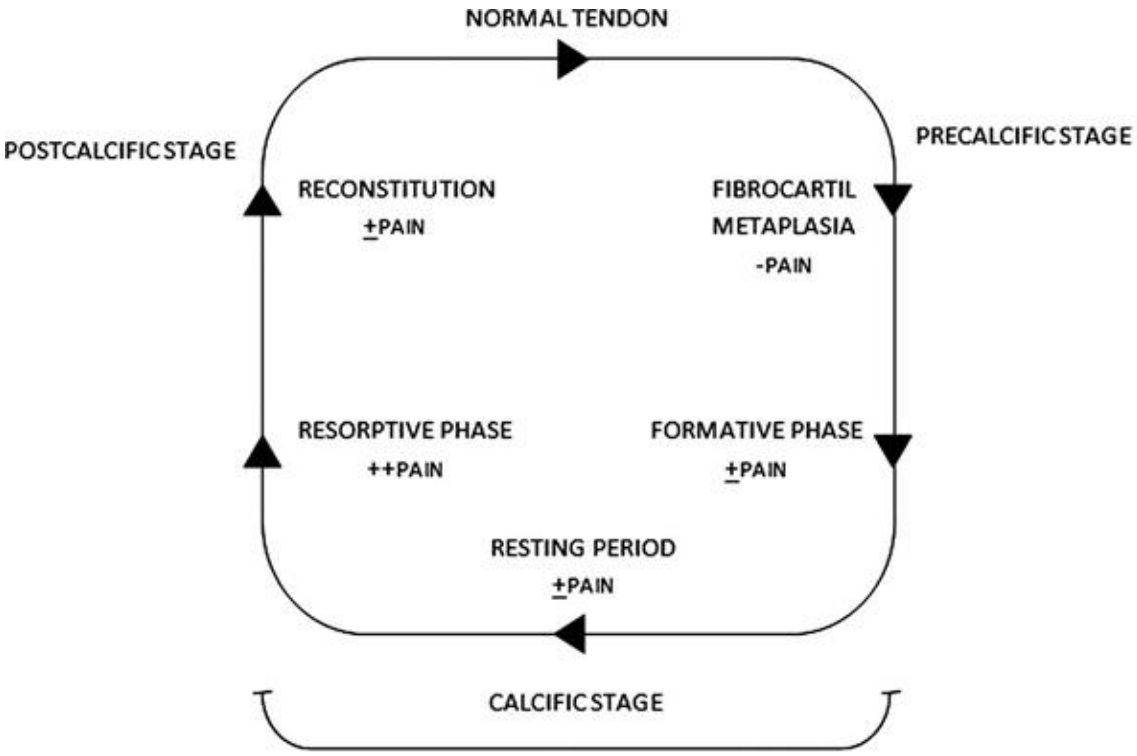
Definition of the calcific tendinopathy

The calcific tendinopathy (CT) of the shoulder is characterized by the presence of macroscopic deposits of calcium hydroxyapatite in the tendons of the rotator cuff, the spontaneous resorption of the deposits and, consequently, the tendon healing.[1] The reasons why calcification occurs are unclear. The possible causes include ischemia, metabolic disorders and fibro-cartilage transformation of the tendon tissue. During the resorption of calcific deposits, patients may experience acute symptoms, including severe pain and limitation of mobility, usually duration up to two weeks. However, if, due to the size of the deposits, subacromial impingement syndrome occurs, a state may become chronic.[2] Uhthoff Loehr described the calcific tendinopathy as a disease undergoing several phases: Formation of the calcium deposits: As a result of an unknown trigger, part of the tendon undergoes fibrocartilaginous transformation and, consequently, calcium deposits are formed. In this phase, symptoms are insignificant, just in the form of discomfort. By the time, deposits become bigger. Resting phase: Once created, a calcium deposit enters the resting period. Resorption phase: This phase starts with an inflammatory response. On the peripheral parts of the deposits, vascular tissue is generated. Macrophages and multinuclear giant cells reabsorb the deposits. Patients experience a severe pain and a limitation of mobility. Post-calfic phase: After the resorption, the collagen in tendons is reconstructed by fibroblasts (Picture 1).[3]



Picture 1 Phases/ *Slika 1. Faze* Normal tendon/*Normalna tetiva/* Precalcific stage/ *Prekalcificirajuća faza/* Fibrocartil metaplasia/ *Fibrokalrtilaginozna metaplazija/* Formative faze/*Formativna faza/* Pain/*Bol/* Resting period/*Razdoblje mirovanja/* Calcific stage/ *Kalcificirajuća faza/* Resorptive phase/ *Resorptivna faza/* Tendon reconstitution/ *Rekonstrukcija tetive/* Postcalcific stage/ *Postkalcificirajuća faza*

The two main characteristics differentiate the CT from degenerative calcification: in calcific tendinopathy, calcification is a cellular induced process in living tissue, unlike degenerative calcification where calcium salts are precipitated into a degenerative changed tendon, bounded to the bone. Histological and immunohistochemical evidence of cellular-induced resorption is supported by the clinical observation of spontaneous resorption of deposits, a phenomenon that never occurs in degenerative calcification.[4] There are four key histological findings: fibrocartilaginous metaplasia during a pre-calcific phase, deposits of calcium crystals in the fibrocartilaginous matrix during the formative phase, the cellular mediated resorption of deposits during the resorption phase and tendon restitution during post-calcific phase.[5] Calcific tendinopathy is a multifocal, cellular-mediated process in which the metaplastic transformation of tenocytes induces a calcification within the tendon. After that, the metaplastic areas are phagocyted by multinuclear giant cells. The tendon is reshaped and reformed into a normal tendon.

Epidemiology and pathogenesis of the calcific tendinopathy

Reports of the total frequency of calcific tendinopathy vary significantly. The incidence of the calcific tendinopathy of the shoulder is, in the general population, 8-20% with the peak of incidence at the ages between 30 and 50.[1] The calcific tendinopathy mostly affects the tendon of the supraspinatus muscle; infraspinatus, subscapularis and the teres minor tendons are rarely affected, less than 10% of the calcific tendinopathy includes the subscapular tendinopathy.[6] It affects women more often than men, and the right shoulder more often than left. The bilateral presentation of calcific tendinopathy of the shoulder is not uncommon and occurs from 5 to 23% of patients.[7-9] The etiology of the calcific tendinopathy remains unknown. Possible pathoetiological factors are inflammation, degeneration, fibrocartilaginous transformation, overload or insufficient load, repetitive motion, external irritation, a combination of internal and external factors, postural variations, nutritional and systemic factors and genetics.[3] It is very likely that pathoetiology and pathohistology of pain and dysfunction of the tendons of the rotator cuff are complex and multifactorial. There are different theories about etiopathogenesis. In recent years, there have been few publications presenting chondral metaplasia as an important mechanism.[10] Pre-clinical and clinical studies have clearly shown that the formation of the calcium deposits in calcific tendinopathy is a cellular induced process. The possible pathogenetic mechanism of the heterotopic ossification is a disturbed differentiation of the stem cells of the tendon origin.[11] The mechanical overload or repeated microtrauma leads to an increased expression of the bone morphogenetic protein (BMP-2), and increase in the activity of the alkaline phosphatase which leads to a disturbed differentiation of the stem cells in chondorcytes and osteoblasts instead of differentiation in tenocytes. BMP-2 was detected as a key element in the pathogenesis of calcific tendinopathy.[12] On the animal models, it has been shown that the injection of the recombinant human bone morphogenetic protein (rhBMP) in the tendons leads to the ectopial bone formation.[13]

Recent studies have demonstrated the presence of thyroid receptors in tenocytes.[14] Their role in the proliferation and apoptosis of the tenocytes has not yet been determined. Hypothyroidism causes the accumulation of glycosaminoglycans in the extracellular matrix, which may facilitate the calcification in the tendon.[15] Sengar et al. discovered a correlation between the HLA-A1 gene and calcific tendinitis, indicating the genetic predisposition of the disease.[16] Further investigations should determine the possibility of incorrect transdifferentiation of the tendon fibroblasts and the role of mechanical load, growth factors, cytokines and extracellular matrix in the pathogenesis of this disease. Furthermore, an association between calcific tendinopathy and diabetes has been observed; there are reports in which > 30% of patients with insulin-dependent diabetes have calcifying deposits in tendons. There are also reports that patients with associated thyroid disorders show earlier symptoms, longer duration of the disease, and undergo surgery more easily than others undergo, but the precise mechanism is still unclear. An increased incidence of class A1 human leukocyte antigen serotype was observed in patients with calcific tendinopathy, suggesting a possible genetic predisposition to the disease.[17]

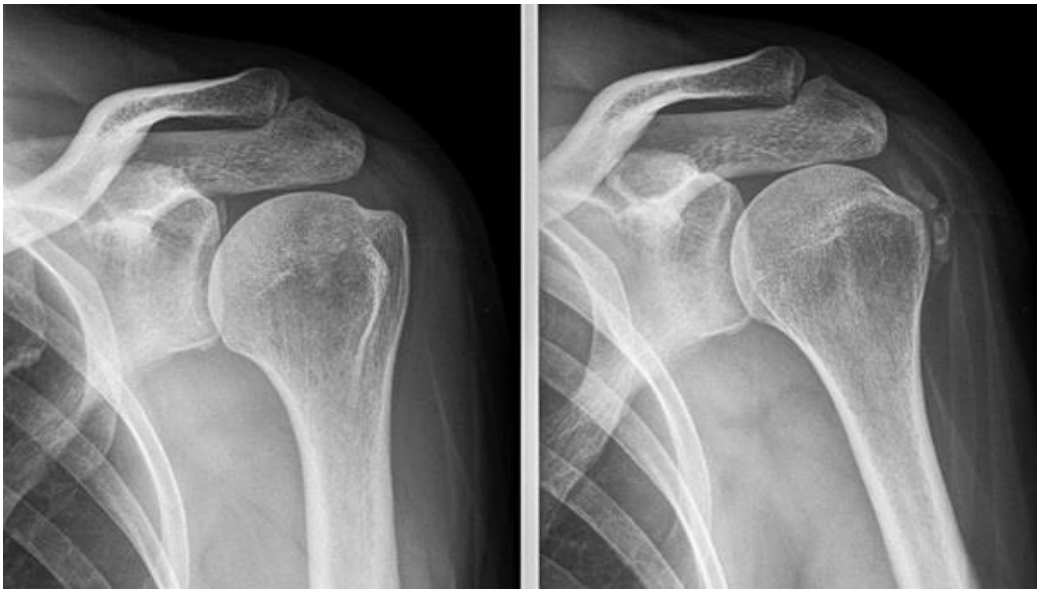
Clinical diagnosis of calcific tendinopathy

The initial stage of the formation of calcium deposits causes minor symptoms in the form of discomfort. Vascular proliferation occurs during the resorption of the deposits and, consequently, an increase in the intertendinous pressure, causing pain. Due to the increase in the volume of the tendon, it collides with the coracoacromial arch and causes more pain. During the formative phase, the patient complains of pain and local sensitivity that irradiates in the area of the deltoid muscle. Shoulder mobility is

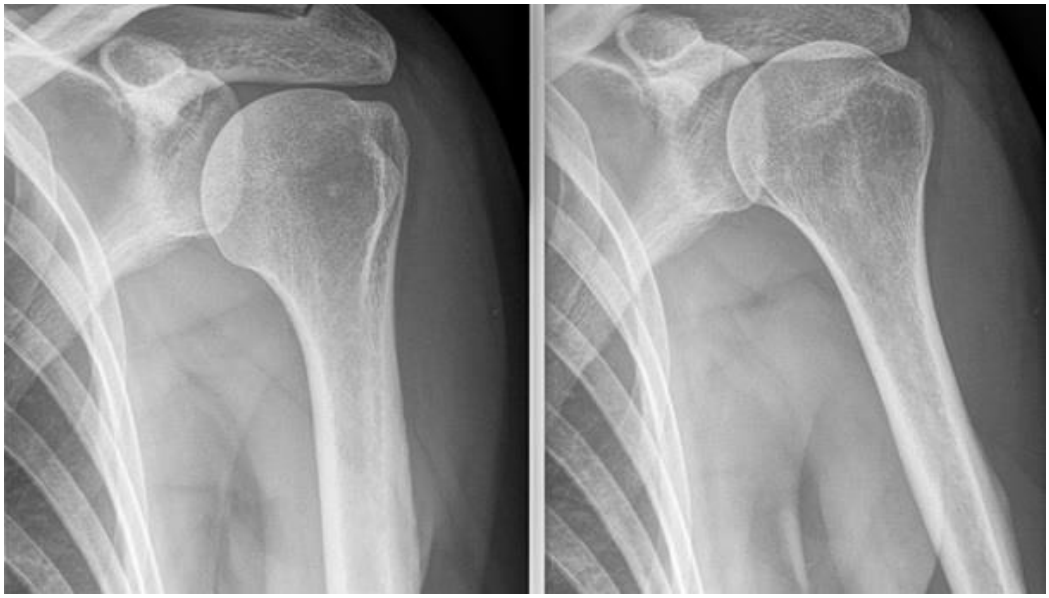
reduced due to pain; the patient cannot lie on the affected side. During the acute resorption phase, the pain is so severe that the patient keeps his hand to the body and does not allow any movement.[18] The pain can become chronic which is associated with the size of the deposits and the development of the subacromial impingement syndrome.[19]

Radiological diagnostics of calcific tendinopathy

A standard radiograph in an antero-posterior view and recordings in internal and external rotation enables localization and evaluation of texture and morphology of deposits. The deposit of calcium in the acute (resorption) phase is blurry, poorly limited, and inhomogeneous. In a chronic, formative phase, the deposit is dense, well-limited and homogeneous (Picture 2, Picture 3).[20]



Picture 2 X-ray in AP view and in internal rotation, resorptive phase (original data)
Slika 2. Radiološka anteroposteriorna snimka u unutrašnjoj rotaciji, resorptivna faza (vlastiti izvor)



Picture 3 X-ray of the same patient, one year later (original data)
Slika 3. Radiološka snimka istog bolesnika, godinu dana kasnije (vlastiti izvor)

There are various radiological classifications based on the size of deposits, the morphology of the deposits and the stage of the disease. Several classifications have been proposed. However, the fact that there are many classifications indicates that there is no classification which perfectly corresponds to the radiological image and the symptomatology of the patient. DePalma distinguishes two types of the calcific tendinopathy: Type I: flaky and amorphous, type II: defined and homogenous. Type I is most common in patients with acute symptoms, and type II is commonly found in patients with chronic symptoms.[21]

The French Arthroscopic Society has defined 4 types of deposits:

Type A: sharp lines, dense and homogenous

Type B: sharp line, thick and of multiple fragments

Type C: heterogeneous appearance, flaky

Type D: distrophytic calcification at the insertion point of the tendon

Gärtner proposed a classification: Type I with sharp lines and dense structure, type II with blurry lines and transparent structure and type III with characteristics of both of these types.[22]

Ultrasound in diagnostics of calcific tendinopathy

In recent years, ultrasound has become an important method of reviewing and monitoring patients with damaged movement system because it is widely available, inexpensive, displays changes in para-articular structures, inflammatory changes in the sinovia and swelling in the joint, muscles, tendons and the surface of the bone. Ultrasound is a diagnostic method that can accurately determine the size and localization of calcium deposits. It has the advantage in diagnoses because it helps to detect other related conditions such as rotator cuff rupture and tendovaginitis of the long head of the biceps muscle. The deposits of calcium are shown as a hyperechogenic focus in the rotator cuff tendons. The presence of the acoustic shadow depends on the density and size of the calcification. The homogenous calcification in the chronic phase is clearly visible as a hyperechogenic focus which makes an acoustic shadow. The inhomogeneous calcification in the acute phase contains a lower amount of calcium and is shown by decreased echogenicity.[19] According to the morphology of calcifications, ultrasound was used to classify different types of calcifications. Chiou et al. describe four forms of the deposits: arch-shaped (hyperechogenic arch with shadow), fragmented or puncture-shaped (at least two separated echogenic places with or without shadow), nodular-shaped (echogenic node without shading) and cystic-shaped (thick echogenic wall with an anechogenic area or layered content).[23]

Doppler in diagnostics of calcific tendinopathy

The Doppler can reveal vascularization, and numerous publications were published in which color and power Doppler are used to detect, visualize and quantify changes in the vascularization of joints in various inflammatory diseases of the musculoskeletal system.[24,25,2,19]

The Doppler shows increased vascularization around the deposits in certain disease phases which corresponds to the histopathological findings such as phagocytosis in the reabsorption phase and a proliferation of the vascular channels around the deposits, described in the paper of Uthoff and co (Picture 4).[3]

Magnetic Resonance Imaging

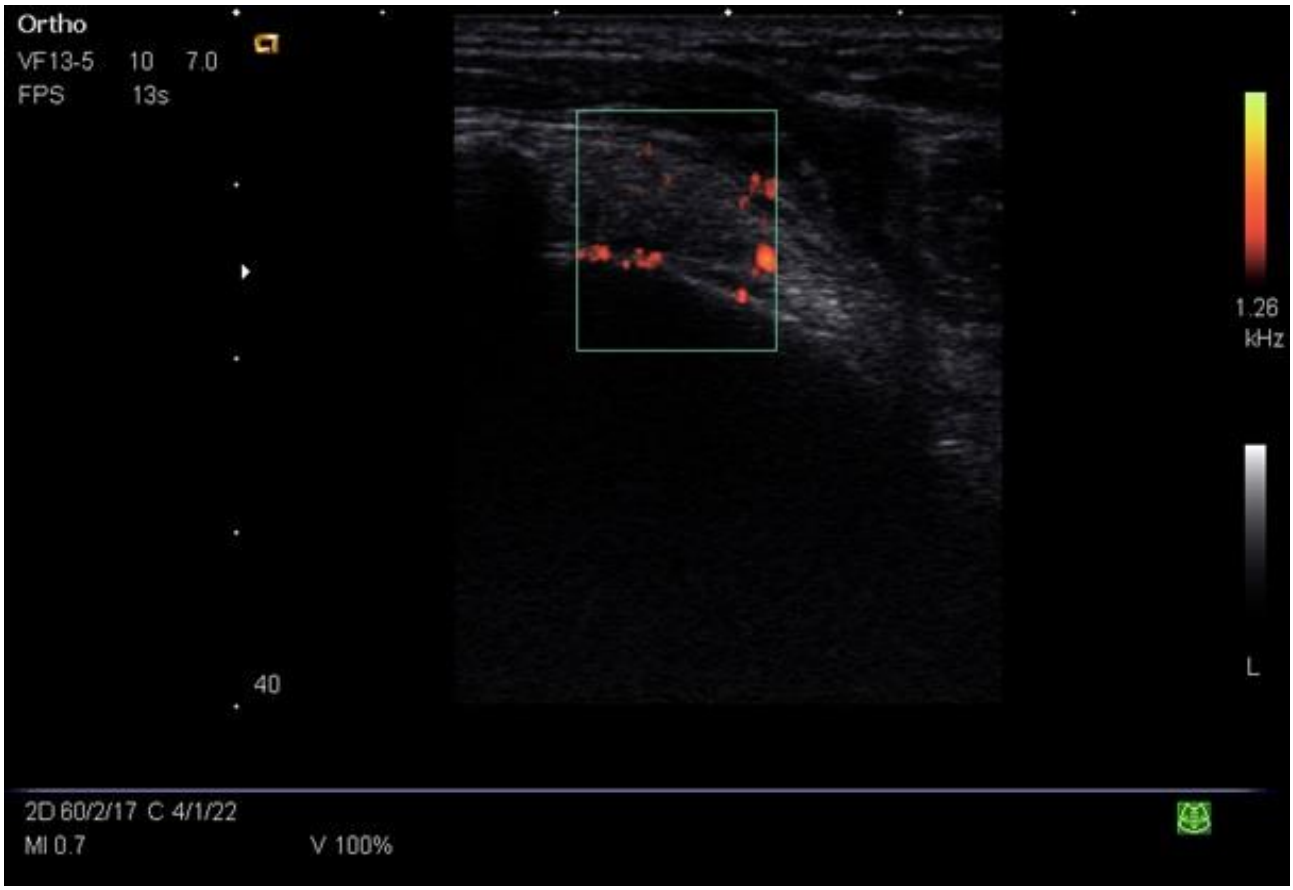
The MRI should not be used as the first diagnostic choice, because the deposits appear as unclear areas of low-intensity signals on the T1 and T2 images, or may not be recognized. The MRI is an additional, but not an essential tool, considering that in most cases it does not provide any additional information. The areas of increased intensity can be found around the deposits in the T2 view and indicate the edema around the deposits in the resorption phase, which can be misinterpreted as a rotator cuff rupture.[26] Amorphous deposits may be wrongly declared as a neoplastic process due to hypervascular absorption.

Computed Tomography

It has an excellent resolution for detecting deposits, but the cost and the exposure to the radiation restrict its use. Computerized tomography is indicated in cases where ultrasound diagnostics are unavailable or when the sonography is negative and does not agree with the positive clinical findings or in younger patients after trauma and complex shoulder injuries.[27]

Treatment

The first-line therapy is a conservative treatment involving non-steroidal anti-inflammatory drugs (NSAIDs), subacromial administration of the corticosteroids, physical therapeutic intervention, needle aspiration, and lavage. In patients with severe pain and dense well-limited calcium deposits, refractory to conservative treatment, surgical treatment is indicated, primarily arthroscopy, sometimes combined



Picture 4 Positive power Doppler in the resorptive phase (original data)
Slika 4. Pozitivan nalaz power Dopplera u resorptivnoj fazi (vlastiti izvor)

with acromioplasty and/or reconstruction of the rotator cuff. The first goal of treatment is to release pain using physical modalities to reduce pain and inflammation. After the inflammation is reduced, the next step is preservation and regeneration of movement and strengthening of the muscles. The ultimate goal of rehabilitation is to achieve functional restitution of the affected shoulder and to enable the patient to perform daily activities without restriction. The predictors of poor prognosis are female sex, affected dominant arm, bilateral disease, longer duration of symptoms (> 3 months) and multiple calcifications, and, according to some authors, localization of deposits in the front part of the acromion and the existence of subacromial impingement.[28,29] The treatment approach depends on the severity of the symptoms and the patience of the doctor and the patient (Table 1).

Table 1 Therapeutic approach algorithm
Tablica 1. Algoritam terapijskog pristupa

Therapy/ *Terapija*	Effect/*Učinci*
Chronic pain/ *Kronična bol*	
Conservative treatment: avoid corticosteroids *Konzervativna terapija; izbjegavati kortikosteroide* Surgical procedure: if conservative treatment is ineffective, if pain interferes with ADL *Operativno liječenje ako je konzervativna terapija neuspješna i ako postoje ograničenja za rad i ADL*	Maintain ROM and strength *Održavati opseg pokreta i snagu*
Acute pain/*Akutna bol*	
Needling and lavage/*Needling i ispiranje*	Decompression/*Dekompresija*

Corticosteroid injections/*Jedna kortizonska injekcija*	Reduce phagocytosis, reduce hyperemia/*Smanjenje fagocitoze; smanjenje hiperemije*
ROM exercises/*Vježbe opsega pokreta*	Prevent development of „frozen shoulder“/*Spriječiti pojavu „smrznutog ramena“*
Subacute pain/*Subakutna bol*	
Abduction/ *Mirovanje u abdukciji*	Reduce the pressure, increase the blood flow/*Istezanjem smanjiti pritisak. povećati protok krvi*
ROM and strength exercises/V*ježbe opesga pokreta i jačanja*	Prevent development of „frozen shoulder“ and strength loss/ *Spriječiti pojavu smrznutog ramena i slabosti mišića*
Avoid corticosteroids/*Izbjegavati kortikosteroidne injekcije*	

ADL - activities of daily living/*aktivnosti dnevnog života*; ROM- a range of motion/*opseg pokreta*

Conservative treatment

Pharmacological treatment

The usage of non-steroidal anti-inflammatory drugs is indicated in the acute and subacute stages. An anti-inflammatory dose is applied in a period of 5-10 days. In the case of chronic symptoms, NSAIDs are prescribed as needed. Other analgesics are rarely prescribed unless satisfying analgesia is not achieved with NSAIDs.[30,31]

Local injections

Local subacromial injections of corticosteroids are used in the acute phase and reduce pain significantly.[32,33] Local intrabursal corticosteroid injections are not used in patients with chronic symptoms. If there is a subacromial impingement syndrome, corticosteroid injection is applied with a lidocaine. Subacromial injections have several advantages: cheap treatment, low risk of complications and regression of the symptoms after a short period.

Physical therapeutic interventions

Extracorporeal shockwave therapy (ESWT)

In the last 25 years, ESWT has been successfully used in people with tendon and muscle diseases.[34,35] The application of a shockwave in the treatment of calcific tendinopathy was firstly described by Dahmen in 1992.[36] Shockwaves can be generated by electrohydraulic, electromagnetic or piezoelectric mechanisms. The high amplitude pressure pulse is generated outside the patient's body and energy is focused on the target area of the body. There are two types of the shockwave applicator: focused and radial. Kinetic energy is transformed into the acoustic wave energy of high amplitude, short duration and sudden increase in pressure. Focused shock waves converge into a focal point or spot. The penetration depth can be precisely determined in a zone between 5-60mm below the skin surface, depending on the applicator diameter and frequency. In non-focused radial shockwave therapy the energy from the surface of the applicator radially extends into the tissue. The largest energy is on the surface of the applicator and is proportionally reduced to the maximum depth of up to 35mm. The destructive potential of the shockwave is thus not exploited as much as the ability of a shockwave to cause hyperemia. The aim of the treatment is to intensify vascularization and to promote the natural washout of the organism.[37] The mechanism by which ESWT produces a clinical effect is still unknown. Several theories are suggested: a mechanical effect that causes fragmentation of deposits, a molecular effect with induction of inflammatory response with neovascularization, chemotactic activity and phagocytosis, and analgesic efficacy and inhibition of serotonergic system activation and peripheral denervation. The biological effects of the shockwave therapy are hyperemia, angiogenesis, and stimulation of fibroblasts, tenocytes, osteoblasts and chondorcytes, and mechanism of mechanical destruction. The overall effect on the target tissues is probably achieved by the combination of described effects.[38-44]

In the paper written by Farr and co., energy of the waves is classified as a low energy (below 0.08 mJ/mm^2), medium energy (0.08-0.28 mJ/mm^2) or high

energy (0.28-0, 60 mJ/mm^2).[39] The side effects reported as a result of the activity of ESWT between 0.04 and 0.22 MJ / mm^2 are very rare, such as pain, local soft tissue edema, skin erosion, erythema, petechial hemorrhage, and subcutaneous hematoma. If high energy ESWT is applied, local anesthesia is required. In literature, there is only one case of osteonecrosis of the head of the humerus after the ESWT.[45] Contraindications for ESWT are divided into general and local contraindications. General contraindications are infections, implanted electrical stimulator, pregnancy and local tumor. Local contraindications are avascular necrosis of the head of the humerus, heterotopic ossification, ostomyelitis and non fused growth plates.

Several studies have confirmed the benefits of ESWT for the treatment of calcific tendinopathy of the shoulder. It is noted that high energy ESWT (EFD ≥ 0.28 mJ/mm2) is more effective than low energy ESWT (EFD < 0.28 mJ/mm^2) in improving shoulder function and reducing pain. Gerdesmayer et al. conducted a study on 144 patients treated with randomized energy (high or low). Both types of ESWT resulted in significant improvements in the 6-month evaluation, but higher ESWT energy resulted in better results. Measured with Constant Murley Score (CMS)[46,47] by analyzing 54 randomized clinical trials, it was found that the usage of focused and radial ESWTs was effective when applying high levels of energy, leading to pain reduction and deposit resorption with a better response of type II patients to the Gartner Classification.[48] It has also been found that the results are comparable to the results achieved by surgical procedure. The role of ESWT as one of the physical therapies can only be discovered by further well-designed researches. Phase-dependent indications (formative, resorptive phase) are still not clearly defined.

Therapeutic ultrasound

Some studies have reported the efficacy of ultrasound therapy: pain relief and functional improvement within 2 months of application.[49] There are also studies in patients with calcific tendinopathy where ultrasound therapy has not achieved greater effect than placebo.[50] There is no evidence of calcification reabsorption or better recovery of function.[51]

Laser

In pain management, the laser is a generally accepted modality although, due to the inability to define the mechanism of pain suppression, some doubts about the actual effect are present.[52,53] Laser photons are absorbed by chromophores, resulting in target heating and localized damage. Laser irradiation alters cellular metabolism and cellular functions.[54] Usage of lasers in the treatment of shoulder pain has more efficacy than placebo. However, there is no difference between the usage of laser therapy + physiotherapy and placebo + physiotherapy.[54]

TENS

Transcutaneous electrical stimulation (TENS) is useful for pain relief in the acute phase.[55] The results of the Cochrane review, "Physiotherapy interventions for shoulder pain", where the synthesis of available evidence of the advantages and disadvantages of electrotherapy modalities for the treatment have not shown that TENS or any other electro-procedure, such as low-intensity laser, ultrasound, pulse electromagnetic therapy have had better results than placebo.[56]

Therapeutic exercises

Well-conceived therapeutic programs have the goal of reducing pain, reflecting/increasing the range of motion, and achieving/maintaining independence in daily activities. In an acute phase, physical activity should be reduced to avoid damaging healthy anatomical structures. After pain relief, therapeutic exercises should be prescribed: passive and active assistive to increase the range of motion. Later, the introduction of resistance training exercises is required to strengthen the muscles of the rotator cuff. Therapeutic exercise programs, if they are well-conceived, can lead to improvements similar to those achieved by surgery. Simple range of motion exercises and resistance exercises performed at home with periodic professional examination can be more effective than intense physiotherapy.[57]

Ultrasound-guided percutaneous aspiration and washing (needling and lavage)

Lavage can be effective only in the case of radiological or ultrasound proved resorption. Ultrasound-guided fine needle aspiration procedure is preferred for better results. Some authors suggest administration of corticosteroids, while others warn that corticosteroids prevent vascular proliferation, local hyperemia, and macrophage activity, which returns calcium deposits to the resting phase. De Witte et al. described the differences between groups of

patients treated with ultrasound-induced percutaneous aspiration and lavage and groups of patients treated with administered subacromial corticosteroid injection. After one year of treatment, the function was evaluated by Constant Murley Score (CMS) and better results were observed in a group of patients treated with needling and lavage. Furthermore, in patients treated with percutaneous aspiration (13 out of 23 patients), full resorption occurred more frequently than in those treated with corticosteroid injection.[58] The Cochrane review on 1450 patients established that ultrasound-guided percutaneous aspiration of the deposits is a safe and effective procedure with an estimated significant reduction in pain.[29]

Systemic review and meta-analysis of comparison of ESWT, ultrasound-guided percutaneous lavage - UGPL, corticosteroid injections and combined treatments showed that UGPL + corticosteroid injections treatment achieved the best results, according to CMS, visual analog scale and the decrease in deposit size.[59]

Other forms of treatment

Platelet-rich plasma – PRP

Platelet-rich plasma has become a popular option for the treatment of injured tendons. The efficacy of the PRP therapy in rotator cuff calcific tendinitis is uncertain. In literature, there are only a few studies regarding PRP application in rotator cuff tendinopathy. Future studies will standardize the preparation and frequency of PRP injections in this condition. The PRP injection can be an effective treatment option, especially in refractory cases of rotator cuff calcific tendinitis.[60] Rha et al found that the effects of PRP injection on shoulder pain and function exceeded those of dry needling in a 6-month follow up. By contrast, Kesikburun et al. have compared the effects of PRP to placebo effects and found no significant difference between them in pain relief and functional improvement.[61] Seijas et al. presented a case of a 44-year-old woman treated with platelet-rich plasma injections (three treatments applied at two-week intervals). All previous treatments in these patients failed. Regression of symptoms occurred within six weeks, and the benefit was maintained in one year.[62]

Surgical treatment

Approximately 10% of patients are resistant to the conservative treatment and, for those patients, an operation is required. Surgical treatment is indicated in patients with severe symptoms lasting more than six months. Two types of surgical procedures are available: arthroscopic removal of calcium deposits and open surgery, which have become less frequently used in recent years.[63-65] Indications for the surgery are symptom progression, constant pain that interferes with daily activities and the absence of reduction of symptoms after conservative therapy. Recovery time after surgery is surprisingly long, in 30% pain and shoulder dysfunction is present 12 weeks after surgery.[66]

Arthroscopy

Arthroscopy has become a commonly used technique for removing calcium deposits, although the arthroscopic approach is demanding. There are several possible advantages over open surgery: less pain, visualization of additional pathology, and a potentially shorter recovery period after surgery. Subacromial decompression is performed only when in presence of some additional pathology, such as a sharp acromion, osteophytes or subacromial syndrome. Arthroscopic removal of calcium deposits without acromioplasty yields results in a rapid pain relief, regardless of acromion morphology. Recovery of the functions requires almost 3 months.[67,68] Postoperatively, the range of motion is maintained. Rehabilitation consists of a range of motion exercises 24 hours after arthroscopy, first passive exercises, than combined with active assistive. Based on the patient's ability, active exercises are introduced on the third day. Patients may return to work after 3 months, and full functional recovery is achieved in 6 months. Arthroscopy is a gold standard for surgical treatment, but two questions remain: whether acromioplasty is needed and is it required to completely remove a deposit.[69]

The senior author, orthopaedic shoulder surgeon (NC) in the last 10 years artroscopically removed calcium deposit without acromioplasty in 159 patients. Acromioplasty is not needed during removal of calcium deposit from the tendon and authors do not recommend acromioplasty in patients with calcium deposit. In the follow up of patients, we recommend using VAS pain or Numeric pain scale for pain assessment, inclinometer for range of motion measuring and Constant Shoulder score, Oxford shoulder score and Quick- DASH (short version of the questionnaire Disabilities of the arm, shoulder and hand) for assessment of shoulder function.[23,70-77]

Conclusion

The successful treatment of patients with calcific tendinopathy depends on the understanding of the pathophysiology of the disease. It is proven, in various clinical observations, that spontaneous resorption exists, but the cause of the resorption remains unclear. The influence of morphology of certain calcium deposits on the symptoms, pain and disturbed function, should be differentiated, which would help to distinguish the phase of the calcific tendinopathy. There is also a great importance in determining the correlation between the radiological and ultrasound characteristics of the deposit and the positive color and power Doppler and clinical finding. The introduction of Doppler findings in the follow-up algorithm is significant. Accurately staging of the disease enables avoiding unnecessary long-lasting physical therapy and usage of effective treatment.

Factors responsible for fibrocartilaginous metaplasia, as well as those that trigger the resorption process, are still unknown and further investigation is required.

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