

1   **ABSTRACT**

2

3   **Background:** Calcific tendinitis affects up to 7% of the population and is the cause of about 20% of the  
4   painful shoulders.

5   Despite the high prevalence of this disease, there is a lack of studies with long term follow-up about the  
6   association of calcific tendinitis with rotator cuff tears and shoulder arthritis.

7   The primary outcome of this study was to evaluate the prevalence of the gleno-humeral arthritis in patients  
8   affected by calcific tendinitis with a minimum follow-up of 10 years.

9   Secondary outcomes were to present the long-term clinical results associated with the management of this  
10   pathology and to assess the prevalence of rotator cuff tears.

11   **Methods:** Thirty five patient were evaluated, with a mean follow up of 13 years.

12   Demographic data were recorded: affected side, age, gender, BMI, smoking, trauma, type of treatment,  
13   concomitant systemic or musculoskeletal diseases.

14   The clinical evaluation was performed using the Constant-Murley Score, American Shoulder and Elbow  
15   Surgeons Score and the Numeric Rating Scale.

16   Each patient underwent an ultrasound and a radiographic evaluation.

17   **Results:** The the mean age was 58.89 ( $\pm$  7.9) years at follow up and 45 years ( $\pm$  13.9) at diagnosis.

18   Our data showed a prevalence of diabetes of 3% and a prevalence of autoimmune diseases of 25%.

19   The prevalence of gleno-humeral arthritis was 17.14% in the study populations and the arthritis progressed  
20   in 14.29% of these shoulders.

21   Full thickness tears of the rotator cuff were not found under ultrasound evaluation.

22   The x-ray examination showed residual calcifications in 31 patients, with a diameter ranging from 2 mm to  
23   20 mm (mean diameter 5.54 mm). In 30 cases there was a reduction of the diameter, in 4 cases the  
24   calcification increased in size and in 1 case the size did not change.

25 There were no differences in terms of pain and functionality between untreated patients and those treated  
26 with corticosteroids injection and shock waves therapy.

27 **Discussion:** Results of this study did not find an increased incidence of osteoarthritis and rotator cuff tears in  
28 patients with calcific tendinitis over 10 years from the diagnosis. The lower scores obtained by our patients  
29 do not seem to be related to the presence of these pathologies, but we suppose that they could be due to the  
30 persistence of residual calcifications not efficiently treated at the onset with consequent chronicity and  
31 persistence of symptoms.

32 **Study Design:** Cohort Study, Level of evidence 3.

33 **Keywords:** shoulder, rotator cuff, calcific tendinitis, long term follow-up

34

35

36

37

38

39

40

41

42

43

44

45

46

47 **INTRODUCTION**

48

49 Calcific tendinitis, also known as Duplay disease, is a common cause of shoulder pain, especially in middle-  
50 aged female population<sup>1-4</sup>; in particular, patients typically report severe pain and significant limitation of  
51 range of motion, without an history of trauma<sup>5,6</sup>.

52 The etiopathogenesis of calcific tendinitis is still highly discussed, but is hypotized to be due to low blood  
53 perfusion in tendon tissue with decrease of local oxygen partial pressure, leading to fibroblast metaplasia  
54 with resultant production and deposition of calcium hydroxyapatite within rotator cuff tendons<sup>3,7</sup>.

55 Despite being a self-limiting disorder, whose symptom relief is usually obtained within few weeks,  
56 hydroxyapatite deposits could remain for many months and, in some cases, for years<sup>8,9</sup>. These crystals may  
57 potentially affect tendon structures, causing rotator cuff tears and subsequent arthritic evolution<sup>10,11</sup>.

58 Most of the studies in literature have explained the clinical and radiological history of calcific tendinitis at  
59 short and mid-term follow-ups<sup>12-16</sup> evaluating the biological effect of hydroxyapatite calcium on surrounding  
60 tissues and the effect on shoulder kinematics.

61 Few studies have analyzed the evolution of hydroxyapatite deposits on rotator cuff tendons at long term  
62 follow-up after the diagnosis<sup>17-19</sup>, but there are no studies that research or hypothesize the possible evolution  
63 of calcific tendinopathy of the shoulder in osteoarthritis.

64 This lack of data in literature can make it difficult to clearly establish the association of calcific tendinitis and  
65 the development of gleno-humeral arthritis.

66 On these bases, the aim of this study was to evaluate the prevalence of gleno-humeral arthritis and rotator  
67 cuff tears in a cohort of patients affected by calcific tendinitis, at least ten years after the diagnosis, and to  
68 present the long-term clinical results associated with its management.

69

70

71

72

73

74

75

76

77

78

79

80

81

82

83

84

85

86

87

88

89

90

## 91 MATERIALS AND METHODS

92

93 The primary aim of this prospective clinical trial on an historical cohort was to assess the prevalence of  
94 gleno-humeral arthritis in patients affected by calcific tendinitis at a minimum follow-up of ten years after  
95 the diagnosis using radiological evaluation.

Secondary outcomes were to evaluate the prevalence of rotator cuff tears through ultrasound examination and to assess shoulder pain and function using the Constant-Murley Score (CMS)<sup>20</sup> the American Shoulder and Elbow Surgeons Score (ASES)<sup>21</sup> and the Numerical Rating Scale (NRS)<sup>22</sup>.

102 A single researcher performed an internal database search in November 2017 to find all the patients who  
103 referred to the emergency department of our institution between August 2001 and June 2008 with clinical  
104 and radiographic diagnosis of calcific tendinitis.

105 The patients were identified using specific numeric code (ICD-9 code: 726,11) used in emergency setting to  
106 classify this pathology.

107 Patients affected by calcific tendinitis confirmed both by clinical report and x-ray, diagnosed between 30 and  
108 60 years 8 (in order to have an homogeneous sample) and with a follow-up of ten years or more were  
109 included in the study.

110 Patients were excluded when the x-rays were not available, or if the images available did not support the  
111 diagnosis of calcific tendinitis.

112 A telephone interview was conducted to inquire if each selected patient had undergone treatments on the  
113 shoulder, to collect NRS, ASES score, and to assess subjective satisfaction.

114 Patients were then invited for a functional and radiological evaluation of the affected shoulder.

115 The following data were collected: affected side, dominant side, age, gender, date of diagnosis, age at  
116 diagnosis, BMI at follow-up, smoking, type of job (light or hard physical demand), trauma, type of treatment  
117 (shock waves therapy and/or subacromial corticosteroids injections, conservative treatment such as physical  
118 therapy or NSAIDs), diabetes, bilateral shoulder pain, inflammatory diseases, and other systemic or  
119 musculoskeletal diseases.

120 During the clinical evaluation, the CMS was collected and isometric strength in shoulder forward flexion and  
121 abduction was measured.

122 All measures were performed in triplicate with a dynamometer (Kern HCB, Kern & Sohn GmbH, Germany)  
123 between October 2017 and October 2018.

124 Each patient also underwent shoulder radiographs (standard true anteroposterior and lateral views) and an  
125 ultrasound examination of the rotator cuff, performed with the high-frequency linear transducer (12.5 MHz)  
126 of a Samsung RS80A Prestige ultrasound system by a senior radiologist.

127 On standard anteroposterior shoulder x-rays views, the presence of shoulder osteoarthritis was classified  
128 according to Samilson-Prieto classification<sup>23</sup>, and the presence of calcifications and the calcium deposits  
129 were measured on x-ray with IMPAX 6.5.2 and distinguished by morphology and density using the Gärtner  
130 classification<sup>13</sup> both in baseline and final control (Fig.1).

131 The possible relationships between the evidence of arthritis progression and the following variables were  
132 investigated: a) size of calcifications, b) type of calcifications according to the Gärtner classification, c)  
133 presence of rotator cuff lesions, d) specific treatment performed.

134 The ultrasound examination was performed by a dedicated musculoskeletal radiologist (M.B.G.) and used to  
135 define rotator cuff tendons integrity with Hinsley classification<sup>24</sup>.

136 This classification considers three different grades of lesion: a) normal tendon, b) abnormal tendon without  
137 partial or full thickness tears, c) partial thickness tear, and d) full thickness tear (subgrouped in longitudinal  
138 tear < 2.5 cm; transverse tear > 2.5 cm and large full thickness tear > 2.5 cm).

139 The presence of subacromial bursitis, biceps tendon tenosynovitis and calcifications were also recorded.

140

141 **Statistical analysis**

142 Statistical analysis (A.M.) was performed with GraphPad Prism software (v 6.0; GraphPad Software Inc) and  
143 SAS software (v 9.4; SAS Institute, Inc).

144 The differences between the groups of patients for continuous variables were evaluated with the unpaired  
145 Student t test or Mann-Whitney test according to the characteristics of the data distribution.

146 Categorical variables were evaluated with the chi-square test or Fisher exact test.

147 For all analyses, the significance level was set at  $P \leq .05$ .

148

149

150

151

152

153

154

155

156

157

158

159

160

161

162

163

164

165

166

167 **RESULTS**

168

169 **Baseline demographics**

170 One hundred ninety one patients satisfied the age-related inclusion criteria and were eligible for the follow-up evaluation. All patients where called using the numbers available in internal database.

172 Seventy nine patients were available for a telephonic interview, and forty six patients also agreed to return to  
173 our institution for clinical and radiological evaluation.

174 Eleven patients were further excluded because of the lack of baseline radiographs. Thirty five patients were  
175 finally included in the analysis.

176 A flow diagram illustrates the grouping and flow of patients in this clinical study (Fig. 2).

177 Demographic data of the group of patients who received a clinical and radiographic assessment are reported  
178 in Table 1.

179 The 74% of included patients were women, the mean age was 58.89 ( $\pm 7.9$ ) years at the final follow-up of 13  
180 years.

181 Thirty patients were right-handed, 2 left-handed and 3 were ambidextrous.

182 The calcifications afflicted the dominant side in 21 cases, in 14 cases the non-dominant side.

183 Regarding clinical comorbidities, we found a prevalence of diabetes of 3% and a prevalence of autoimmune  
184 diseases (rheumatoid arthritis, psoriatic arthritis, Hashimoto's thyroiditis, DM1, celiac disease, psoriasis) of  
185 25%.

186 Eleven patients smoked at the follow-up and 14 at the baseline.

187 Seven patients were treated with corticosteroids injection, 8 patients underwent shock waves therapy, 11  
188 were treated with both corticosteroid injections and shock waves, and 1 patient with ultra-sound guided  
189 percutaneous lavage.

190 Eight patients were treated just with oral painkillers and anti-inflammatories or physical therapy.

191    **Gleno-humeral osteoarthritis**

192    At the baseline we found radiographic signs of gleno-humeral osteoarthritis in 4 patients (grade 1 of  
193    Samilson-Prieto Classification).

194    At the final follow-up the prevalence of gleno-humeral osteoarthritis was 17.14% (6 patients).

195    The arthritis progression, defined as an increase of at least 1 grade in the Samilson-Prieto scale from the  
196    baseline to the follow-up was find in 5 patients (14.28%). Of these, two patients showed the ex-novo  
197    appearance of gleno-humeral osteoarthritis, in the other three patients osteoarthritis was already present at  
198    the diagnosis and has worsened.

199

200    **Subgroup analysis: gleno-humeral arthritis**

201    The study population was further divided in two different groups according to the gleno-humeral arthritis  
202    progression as classified by Samilson and Prieto: patients with arthritic changes and patients without arthritic  
203    changes.

204    A stratified analysis was performed comparing demographic and clinical data to find potential risk factors for  
205    arthritic evolution (Table 2, 3).

206    Our analyses revealed that samples were homogeneous for all the considered parameters, in fact no  
207    statistically significant differences were found for age, sex, BMI, follow up, affected side, dominant side,  
208    smoking, type of job, diabetes, autoimmune diseases and traumas (Table 2 and 3).

209    No statistically significant association was encountered between the gleno-humeral arthritis progression and  
210    the evaluated variables.\_

211    Although patients with arthritic changes showed a more relevant mean reduction of calcification diameter  
212    compared with non-arthritic patients (10.2mm vs 7.7mm), the differences in means between the two groups  
213    were not statistically significant.

214    Under ultrasound evaluation, patients with arthritis progression showed a higher percentage of degenerative  
215    manifestations on rotator cuff tendons, but the difference was not statistically significant.

216    No full thickness tears of the rotator cuff were found.

217

218    **Subgroup analysis: type of treatment**  
219    Given the extreme treatment variability, patients were subsequently divided in two groups (treated:  
220    corticosteroid injections and/or shock waves therapy and percutaneous lavage; untreated: physical therapy,  
221    NSAIDs or nothing) and their clinical and radiological data were compared.  
222    The mean variation of the calcification maximum diameter was 8.74 mm in the treated group compared to  
223    5.63 mm in the untreated group, but no significant differences were noted. (Table 4).

224

## 225    **Calcifications**

226    At the diagnosis the calcifications had a diameter ranging from 5 mm to 27 mm with an average diameter of  
227    12.9 mm.

228    The most affected tendon was the supraspinatus, followed by subscapularis.

229    Residual calcifications were observed in 31 patients (88.57%) with a diameter ranging from 2 mm to 20 mm  
230    (mean diameter 5.54 mm).

231    At the final follow-up, the residual calcifications in 8 cases had a diameter of less than 2 mm , in 13 cases  
232    had a diameter between 3 mm and 9 mm and in 4 cases a diameter greater than 10 mm.

233    In 30 cases we found a reduction of the diameter (with a mean percentage of reabsorption of 57%), in 4 cases  
234    the calcification increased in size and in 1 case the size did not change (Fig.3) .

235

## 236    **Subgroup analysis: persistence of calcifications**

237    Calcifications with a maximum diameter of less than or equal to 2 mm were compared to those greater than 2  
238    mm in diameter for the following variables.

239    The mean ASES score was 74.1 in the group with larger calcifications and 89.4 in patients with smaller  
240    calcifications (p 0.08, n.s.).

241    The NRS was 2.7 in the larger calcification group compared to 1.0 in the smaller calcification group (p 0.22,  
242    n.s.). (Table 5).

243

244

245

## 246    **DISCUSSION**

247 Primary outcome of this study was the evaluation of the prevalence of gleno-humeral osteoarthritis in patient  
248 affected by calcific tendinitis at 10 years from the diagnosis.

249 Radiological results did not show an increased prevalence of gleno-humeral osteoarthritis in our patients..

250 We found a prevalence of gleno-humeral osteoarthritis of 17.14%, similar to that estimated in the general  
251 Japanese population by Kobayashi et al.<sup>25</sup>

252 These authors observed a prevalence of about 15% in 541 subjects between 40 and 75 years old according to  
253 the Samilson-Prieto classification .

254 These results support that patients with calcific tendinitis seem to have the same risk of arthritic  
255 degeneration compared to the general population of the same age.

256 This study also investigated the prevalence of partial and full thickness rotator cuff tears.

257 The evaluation of rotator cuff tears using ultrasound did not showed full thickness tears in any patient, but  
258 only signs of cuff degeneration in ten patient.

259 Our results have shown a persistence of the calcification in the vast majority of the shoulder evaluated  
260 (88.57%) with a mean percentage of reabsorption of 57%.

261 This is a relevant finding in contrast to the belief that the natural history of this pathology leads to full  
262 reabsorption of the deposits<sup>3</sup>.

263 Better outcomes were reported in the 4 patients without calcifications compared to those who had residual  
264 deposits, and their dimension have influenced negatively the outcome. It has been reported that patients with  
265 radiological improvement over years report better clinical outcomes<sup>18</sup>.

266 We have supposed that the presence of calcific deposits alters shoulder biology leading to inflammation as  
267 synovitis and subacromial bursitis, influencing shoulder kinematics and bringing to slower or incomplete  
268 recovery.

269 Indeed, patients with smaller calcifications (diameter < 2 mm) showed better functionality of the shoulder  
270 during clinical evaluation and lower inflammatory signs on ultrasound images than those with bigger ones,  
271 but no significant difference between the groups was recorded.

272 Our findings are in agreement with those reported by other authors<sup>18,26</sup>, where a close association between  
273 better clinical outcomes and absence of calcifications at long-term follow-up was observed.

274 Porcellini et al<sup>26</sup> reported similar results evaluating, clinically and with ultrasound, 63 patients affected by  
275 calcific tendinitis and treated by arthroscopic needling with a follow-up of 3 years.

276 They found the persistence of calcifications in 45 patients who presented the lower mean clinical scores, and  
277 they assessed that the outcome was inversely related to the number and size of residual calcifications

278 In our cohort of patients no statistically significant difference in terms of pain and functionality was found  
279 between patients subjected to conservative treatment and those who underwent to corticosteroids injections  
280 and shock waves therapy.

281 The latter group had only little more reabsorption of the calcification compared to untreated (66.9% of the  
282 original deposit in the treated versus 59.3% in the untreated).

283 Results about pain relief and recovery are in line to those obtained by De Witte et al.<sup>17</sup> who assessed the  
284 evolution of calcific tendinitis with a long-term follow-up and whose patients reached lower outcomes  
285 regardless of the treatment performed.

286 Regarding demographic characteristics, an increased prevalence of diabetes was not observed, whereas an  
287 association between these two pathologies is reported.<sup>27</sup>

288 The high prevalence of autoimmune diseases of about 25% in the included patients and an increased number  
289 of smokers compared to the general population (40%) are relevant data, reported in few previous  
290 manuscripts<sup>28,29</sup>.

291 This group of pathologies and habits could probably influence the vascularity of the tendons leading to an  
292 increase of calcium deposits and to a decrease of tendons healing.

---

293 Our study has some limitations: first of all is the lack of a control group. The prevalence of gleno-humeral  
294 osteoarthritis, as well as that of cuff lesions, increases physiologically with age therefore the lack of a control  
295 group of healthy patients makes it difficult to demonstrate its appearance as an evolution of shoulder calcific  
296 tendinopathy.

297 Furthermore there is a lack of information regarding the prevalence of rotator cuff tears and shoulder  
298 osteoarthritis in a population similar for age and origin to our patients therefore we can't compare what we  
299 have found with this data.

300 The study design (prospective clinical trial on an historic cohort) did not allow to include a clinical and  
301 ultrasound assessment at the diagnosis that would have lead to a more accurate comparison with follow-up  
302 evaluation.

303 Furthermore included patients have been subjected to many different treatments over a long period of time  
304 (10 years). To obtain substantial results authors divided the patients in two groups, comparing those who  
305 underwent conservative treatment and those who were subjected to corticosteroids injections, shock waves  
306 therapy or needling. These groups include different treatments and a selection bias is present.

307 Another limitation of this study is the high number of patients unwilling or unable to return to our institution  
308 for the clinical and radiological evaluations: a study conducted with a larger sample population probably  
309 would have given more significant results. Therefore conclusions must be drawn with caution specially for  
310 subgroups which present a small patient cohort (i.e.: arthritic progression group, untreated group).

311

312

### 313 CONCLUSIONS

314 Results of this study did not show an increased prevalence of gleno-humeral osteoarthritis in patients  
315 affected by calcific tendinitis of the shoulder.

316 Tendon calcifications are present at more than 10 years of follow-up in the majority of the patients, in  
317 contrast with common knowledge of a self-limiting pathology<sup>16,30</sup>.

318 The lower clinical outcomes obtained by patients included in this study do not seem to be related to the  
319 presence of gleno-humeral joint pathologies such as osteoarthritis or rotator cuff tears.

320 The persistence of residual calcifications could be the cause of persistence of minor symptoms at long term  
321 follow-up<sup>26</sup>.

322 If the association between persistence of calcifications and long term follow-up symptoms will be confirmed,  
323 it would be interesting to see if a faster and more aggressive approach, such as percutaneous lavage or  
324 arthroscopic needling, could give better results than the conservative treatments to which our cohort of  
325 patients underwent.

326

327

328

329

330

331

332

333

334

335 **BIBLIOGRAPHY**

336 1. Bosworth B marsh. Calcium deposits in the shoulder and subacromial bursitis. *J Am Med Assoc.*  
337 1941;116(22):2477. doi:10.1001/jama.1941.02820220019004

338 2. Elshewy MT. Calcific tendinitis of the rotator cuff. 2016;7(1):55-60. doi:10.5312/wjo.v7.i1.55

339 3. Uhthoff, Loehr. Calcific Tendinopathy of the Rotator Cuff: Pathogenesis, Diagnosis, and

- 340 Management. *J Am Acad Orthop Surg.* 1997;5(4):183-191. doi:10.5435/00124635-199707000-00001
- 341 4. Clavert P, Sirveaux F, Société française d'arthroscopie. [Shoulder calcifying tendinitis]. *Rev Chir*  
342 *Orthop Reparatrice Appar Mot.* 2008;94(8 Suppl):336-355. doi:10.1016/j.rco.2008.09.010
- 343 5. Uhthoff HK, Sarkar K. Calcifying tendinitis. *Baillieres Clin Rheumatol.* 1989;3(3):567-581.  
344 doi:10.1016/s0950-3579(89)80009-3
- 345 6. Speed CA, Hazleman BL. Calcific tendinitis of the shoulder. *N Engl J Med.* 1999;340(20):1582-1584.  
346 doi:10.1056/NEJM199905203402011
- 347 7. Merolla G, Singh S, Paladini P, Porcellini G. Calcific tendinitis of the rotator cuff: state of the art in  
348 diagnosis and treatment. *J Orthop Traumatol.* 2016;17(1):7-14. doi:10.1007/s10195-015-0367-6
- 349 8. Daecke W, Kusnierzak D, Loew M. Long-term effects of extracorporeal shockwave therapy in  
350 chronic calcific tendinitis of the shoulder. *J Shoulder Elb Surg.* 2002;11(5):476-480.  
351 doi:10.1067/MSE.2002.126614
- 352 9. Bannuru RR, Flavin NE, Vaysbrot E, Harvey W, McAlindon T. High-Energy Extracorporeal Shock-  
353 Wave Therapy for Treating Chronic Calcific Tendinitis of the Shoulder. *Ann Intern Med.*  
354 2014;160(8):542. doi:10.7326/M13-1982
- 355 10. Gotoh M, Higuchi F, Suzuki R, Yamanaka K. Progression from calcifying tendinitis to rotator cuff  
356 tear. *Skeletal Radiol.* 2003;32(2):86-89. doi:10.1007/s00256-001-0472-x
- 357 11. Jim YF, Hsu HC, Chang CY, Wu JJ, Chang T. Coexistence of calcific tendinitis and rotator cuff tear:  
358 an arthrographic study. *Skeletal Radiol.* 1993;22(3):183-185. doi:10.1007/bf00206150
- 359 12. Balke M, Bielefeld R, Schmidt C, Dedy N, Liem D. Calcifying tendinitis of the shoulder: midterm  
360 results after arthroscopic treatment. *Am J Sports Med.* 2012;40(3):657-661.  
361 doi:10.1177/0363546511430202
- 362 13. De Witte PB, Selten JW, Navas A, et al. Calcific tendinitis of the rotator cuff: A randomized

- 363 controlled trial of ultrasound-guided needling and lavage versus subacromial corticosteroids. *Am J*  
364 *Sports Med.* 2013;41(7):1665-1673. doi:10.1177/0363546513487066
- 365 14. Bazzocchi A, Pelotti P, Serraino S, et al. Ultrasound imaging-guided percutaneous treatment of  
366 rotator cuff calcific tendinitis: Success in short-term outcome. *Br J Radiol.* 2016;89(1057).  
367 doi:10.1259/bjr.20150407
- 368 15. Galletti S, Magnani M, Rotini R, et al. The echo-guided treatment of calcific tendinitis of the  
369 shoulder. *Chir Organi Mov.* 2004;89(4):319-323.
- 370 16. Castagna A, De Giorgi S, Garofalo R, Conti M, Tafuri S, Moretti B. Calcifying tendinitis of the  
371 shoulder: arthroscopic needling versus complete calcium removal and rotator cuff repair. A  
372 prospective comparative study. *Joints.* 2015;03(04):166-172. doi:10.11138/jts/2015.3.4.166
- 373 17. de Witte PB, van Adrichem RA, Selten JW, Nagels J, Reijnsierse M, Nelissen RGHH. Radiological  
374 and clinical predictors of long-term outcome in rotator cuff calcific tendinitis. *Eur Radiol.*  
375 2016;26(10):3401-3411. doi:10.1007/s00330-016-4224-7
- 376 18. Witte PB De, Kolk A, Overes F. Rotator Cuff Calcific Tendinitis : Ultrasound-Guided Needling and  
377 Lavage Versus Subacromial Corticosteroids Five-Year Outcomes of a Randomized Controlled Trial.  
378 2017;45(14):6-8. doi:10.1177/0363546517721686
- 379 19. Serafini G, Sconfienza LM, Lacelli F, Silvestri E, Aliprandi A, Sardanelli F. Rotator Cuff Calcific  
380 Tendonitis: Short-term and 10-year Outcomes after Two-Needle US-guided Percutaneous  
381 Treatment— Nonrandomized Controlled Trial. *Radiology.* 2009;252(1):157-164.  
382 doi:10.1148/radiol.2521081816
- 383 20. McLean JM, Awwad D, Lisle R, Besanko J, Shivakkumar D, Leith J. An international, multicenter  
384 cohort study comparing 6 shoulder clinical scores in an asymptomatic population. *J Shoulder Elb*  
385 *Surg.* 2018;27(2):306-314. doi:10.1016/j.jse.2017.08.016
- 386 21. Padua R, Padua L, Ceccarelli E, Bondi R, Alviti F, Castagna A. Italian version of ASES questionnaire

- 387 for shoulder assessment: Cross-cultural adaptation and validation. *Musculoskelet Surg.*  
388 2010;94(SUPP). doi:10.1007/s12306-010-0064-9
- 389 22. Hawker GA, Mian S, Kendzerska T, French M. Measures of adult pain: Visual Analog Scale for Pain  
390 (VAS Pain), Numeric Rating Scale for Pain (NRS Pain), McGill Pain Questionnaire (MPQ), Short-  
391 Form McGill Pain Questionnaire (SF-MPQ), Chronic Pain Grade Scale (CPGS), Short Form-36  
392 Bodily Pain Scale (SF. *Arthritis Care Res.* 2011;63(SUPPL. 11):240-252. doi:10.1002/acr.20543
- 393 23. Samilson RL, Prieto V, D M. Dislocation arthropathy of the shoulder. *J Bone Joint Surg Am.*  
394 1983;65(4):456-460. doi:10.1302/0301-620X.96B11.34133
- 395 24. Hinsley H, Nicholls A, Daines M, Wallace G, Arden N, Carr A. Classification of rotator cuff  
396 tendinopathy using high definition ultrasound. *Muscles Ligaments Tendons J.* 2014;4(3):391-397.  
397 doi:10.11138/mltj/2014.4.3.391
- 398 25. Kobayashi T, Takagishi K, Shitara H, et al. Prevalence of and risk factors for shoulder osteoarthritis  
399 in Japanese middle-aged and elderly populations. *J Shoulder Elb Surg.* 2014;23(5):613-619.  
400 doi:10.1016/j.jse.2013.11.031
- 401 26. Porcellini G, Paladini P, Campi F, Paganelli M. Arthroscopic treatment of calcifying tendinitis of the  
402 shoulder: Clinical and ultrasonographic follow-up findings at two to five years. *J Shoulder Elb Surg.*  
403 2004;13(5):503-508. doi:10.1016/j.jse.2004.04.001
- 404 27. Mavrikakis ME, Drimis S, Kontoyannis DA, Rasidakis A, Moulopoulou ES, Kontoyannis S. Calcific  
405 shoulder periarthritis (tendinitis) in adult onset diabetes mellitus: a controlled study. *Ann Rheum Dis.*  
406 1989;48(3):211-214. doi:10.1136/ard.48.3.211
- 407 28. Sengar D. Increased frequency of HLA-A1 in calcifying tendinitis. *TissueAntigens.* 1987;29(3):173-  
408 174. doi:10.1111/j.1399-0039.1987.tb01571.x
- 409 29. Oudelaar BW, Ooms EM, M.H.A Huis in 't Veld R, Schepers-Bok R, Vochteloo AJ. Smoking and  
410 morphology of calcific deposits affect the outcome of needle aspiration of calcific deposits (NACD)

411 for calcific tendinitis of the rotator cuff. *Eur J Radiol*. 2015;84(11):2255-2260.

412 doi:10.1016/j.ejrad.2015.07.030

413 30. De Carli A, Pulcinelli F, Rose GD, Pitino D, Ferretti A. Calcific tendinitis of the shoulder. *Joints*.  
414 2014;2(3):130-136. doi:10.11138/jts/2014.2.3.130

415

416

417

418

419

420

421

422

423

424

425

426

427 **FIGURE LEGENDS**

428 **Fig.1.** Residual calcification (Gartner Type 1). This patient was clinically and radiographically assessed after  
429 ten years from the diagnosis. The x-ray shows the absence of arthritic signs. (**FIGURA DA ELIMINARE**)

430

431 **Fig.2.** Flowchart.

432

433 **Fig. 3.** Bilateral calcific tendenitis showed on x-rays performed at the diagnosis (A-C) and after 10 years (B-  
434 D). The same treatment produced a different evolution: in the right shoulder there was the persistence of the  
435 calcification while in the left the treatment led to a complete resorption. **(FIGURA DA ELIMINARE)**

436

437

438

439

440

441

442

443

444

445

446

447

448

449

450

451

452 **TABLE LEGENDS**

453 **Table 1. Patients' demographics.**

454

455 *Data are reported as mean ( $\pm SD$ ), median [Q1-Q3] or frequency/ratio. BMI: body mass index; F/M:*  
456 *females/males; N: No; L/R: left/right, Q1: first quartile; Q3: Third quartile; SD: standard deviation; Y: yes.*

457

458 **Table 2. Subgroup analysis: arthritic progression and patient characteristics.**

459 *Data are reported as mean ( $\pm SD$ ), median [Q1-Q3] or frequency/ratio. BMI: body mass index;*  
460 *F/M: females/males; N: No; n.s.: non-significant; R/L: left/ right, Q1: first quartile; Q3: Third quartile;*  
461 *SD: standard deviation; Y: yes.*

462

463

464 **Table 3. Subgroup analysis: arthritic progression and clinical and radiological results.**

465 *Data are reported as mean ( $\pm SD$ ), median [Q1-Q3] or frequency/ratio. n.s.: non-significant; Q1: first*  
466 *quartile; Q3: third quartile; RC: Rotator cuff; SD: standard deviation; Y: yes; N: No.*

467

468 **Table 4. Subgroup analysis: type of treatment and clinical and radiological results.**

469 *Data are expressed as mean ( $\pm SD$ ),or frequency/ratio. ASES: American Shoulder and Elbow Surgeons*  
470 *score; CMS: Constant-Murley Score; mm: millimeters; NRS: Numeric Rating Scale; n.s.: not significant;*  
471 *SD: Standard Deviation; Y: yes; N: No.*

472

473 **Table 5. Subgroup analysis: calcification diameter and clinical and radiological results.**

474 Data are expressed as mean ( $\pm$  SD), median [Q1-Q3] or frequency/ratio. ASES: American Shoulder and  
475 Elbow Surgeons score; CMS: Constant-Murley Score; NRS: Numeric Rating Scale; n.s.: not significant; Q1:  
476 first quartile; Q3: third quartile; SD: Standard Deviation; Y: yes; N: No.

477  
478

479

480

481

482