

1 **ABSTRACT**

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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

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47 **INTRODUCTION**

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49 Calcific tendinitis, also known as Duplay disease, is a common cause of shoulder pain, especially in middle-
50 aged female population¹⁻⁴; in particular, patients typically report severe pain and significant limitation of
51 range of motion, without an history of trauma^{5,6}.

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^{3,7}.

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^{8,9}. These crystals may
57 potentially affect tendon structures, causing rotator cuff tears and subsequent arthritic evolution^{10,11}.

58 Most of the studies in literature have explained the clinical and radiological history of calcific tendinitis at
59 short and mid-term follow-ups¹²⁻¹⁶ 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 diagnosis¹⁷⁻¹⁹, 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.

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91 **MATERIALS AND METHODS**

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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.

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

99 The study protocol was approved by the Regional Ethical Committee ([REDACTED]
100 [REDACTED]
101 [REDACTED]).

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 classification²³, 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 classification¹³ 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 classification²⁴.

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.

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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 $P \leq .05$.

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167 **RESULTS**

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169 **Baseline demographics**

170 One hundred ninety one patients satisfied the age-related inclusion criteria and were eligible for the follow-
171 up evaluation. All patients were 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 found 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.

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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.

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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).

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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) .

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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).

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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.²⁵

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 deposits³.

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 outcomes¹⁸.

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^{18,26}, where a close association between
273 better clinical outcomes and absence of calcifications at long-term follow-up was observed.

274 Porcellini et al²⁶ 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.¹⁷ 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.²⁷

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^{28,29}.

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).

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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^{16,30}.

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-up²⁶.

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.

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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)**

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431 **Fig.2.** Flowchart.

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433 **Fig. 3.** Bilateral calcific tendinitis 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)**

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452 **TABLE LEGENDS**

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

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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.*

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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.*

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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.*

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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.*

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