Are occupational repetitive movements of the upper arm associated with rotator cuff calcific tendinopathies?

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Abstract Calcifying tendinopathy (CT) of the shoulder is a common painful disorder, although the etiology and pathogenesis remain largely unknown. Recent theories about the role of excessive mechanical load in the genesis of CT have been proposed. Driven by the interest for these new theories, we investigated the hypothesis of a relationship between work-related repetitive movements of the upper arm, considered a potential cause of shoulder overload, and the presence of shoulder CT. A secondary aim was to obtain data on CT prevalence in a female sample from the working-age general population, as little data currently exist. 199 supermarket cashiers and 304 female volunteers recruited from the general population underwent a high-resolution ultrasonography of the rotator cuffs of both shoulders, and the presence of tendinopathies, with or without calcification, was recorded. The prevalence of calcific tendinopathy was 22.6 % in the cashiers group and 24.4 % in the control group. There were no statistically significant differences in the prevalence of calcifications between the two groups ($p = 0.585$), either for the dominant shoulder [OR = 0.841 (95 % CI 0.534–1.326)] or for the non-dominant shoulder [OR = 0.988 (95 % CI 0.582–1.326)]. We observed bilateral calcifications in 8.5 % of cashiers, and 9.6 % of controls, and an increase in prevalence of CT with age in both groups. Work-related repetitive movements of the upper arm did not induce a higher prevalence of shoulder CT compared with the female sample from the general population. If CT etiopathogenesis is related to mechanical load, CT onset may be influenced not only by loading history, but also by individual factors.

Level of evidence Prognosis study, Level II.

Keywords Calcific tendinopathy · Rotator cuff · Repetitive movement · Prevalence · Women

Introduction

Calcific tendinopathy (CT) of the shoulder is a common disorder characterized by the presence of calcium deposits in the mid-substance of the rotator cuff tendons, particularly in the supraspinatus. Chronic activity-related pain, tenderness, local edema, and various degrees of impairment are the usual clinical features. Women between 30 and 60 years are most commonly affected, and the condition is bilateral in approximately 10 % of patients [1]. The prevalence of CT is reported to range between 2.5 and 22 % in adults, although these data are not recent and are largely based on radiographic observations of asymptomatic populations [2–5]. The recent technological advances in imaging modalities such as ultrasound and magnetic resonance...
allow a more accurate and detailed analysis of anatomic structures, and so it is reasonable to suppose that the prevalence of CT may be higher than previously reported. However, recent data are not available in the literature.

The majority of the studies conducted in the last 30 years have focussed on the pathogenesis of the disease rather than on the prevalence or the anatomic features and clinical aspects of calcifications. Several hypotheses for the pathologic process have been proposed: degenerative [6]; repetitive trauma [2]; tenocyte necrosis [7]; reactive [8]; and endochondral ossification [9, 10], although none of these explanations have proved to be completely satisfactory. There is also a lack of consensus regarding predisposing factors for CT. An association between CT and diabetes has been observed; more than 30 % of patients with insulin-dependent diabetes were found to have tendon calcifications [11–13]. It has also been reported that patients with associated thyroid disorders present an earlier onset of symptoms, a longer natural history, and undergo surgery more frequently with respect to the general population, but the precise mechanism is not clear [14]. Genetic predisposition has also been invoked [15, 16], with authors concluding that some genetic variants could increase the susceptibility of tendons to the matrix abnormalities observed in tendinopathies [17]. Due to this lack of knowledge about the pathogenesis or predisposing factors, most current treatments are neither effective nor evidence based.

Recent studies based on the isolation of a residential population of tendon stem cells (TSC) housed in particular spaces called “niches” of humans, rabbits, mice, and rats [18, 19] seem to shed new light on the mechanisms that could be involved in the disease, opening up possibilities for understanding—and ultimately treating and preventing—tendinopathies, including CT. A new pathogenetic model has been proposed to explain the origin of CT, based on erroneous differentiation of TSCs and failed healing in response to mechanical overload [20]. In normal conditions and after trauma, TSCs have the ability to differentiate into tenocytes and to self-renew, thus playing a key role in tendon maintenance and repair. However, in the presence of altered local conditions, such as excessive mechanical loading or the accumulation of micro-injuries, the TSCs would differentiate into chondrocytes or osteoblasts instead of tenocytes, probably through a prostaglandin E2-mediated mechanism [21, 22]. The activity of these non-tenocytes leads to chondrometaplasia and ossification or, in other words, to the production of aberrant extracellular matrix and calcific deposits within the tendon structure. As shown by Uhthoff almost 40 years ago, one key histopathologic feature of calcifying tendinopathy is the presence of chondrocyte phenotype cells surrounding the calcific deposits, suggesting that their formation is cell mediated rather than formed by precipitation of inorganic ions [10]. However, these new pathogenetic theories of CT are based exclusively on experimental evidence; we are not aware of any clinical study able to demonstrate a cause–effect relationship between mechanical overload and the development of CT in humans.

In light of these considerations, we performed a study to verify whether a relationship exists between work-related repetitive movement and the development of rotator cuff CT. With this purpose, we examined a group of female supermarket cashiers, whose repetitive movement, especially in internal and external rotation, could be reasonably supposed to induce a significant mechanical stress on the rotator cuff structures, able to induce CT. The findings of this group were compared against those of a control group of female volunteers of similar age but who were not involved in upper arm repetitive movements for professional purposes. A secondary aim was to ascertain the prevalence of shoulder calcific tendinopathy among a female sample from the working-age population, given the lack of published data on this subject.

Materials and methods

From November 2011 to March 2012, all the female cashiers from 3 supermarkets in the same province of north Italy were requested to participate in our study. 199 cashiers agreed to take part, (age range 21–60 years), while 24 were unable, or refused, to participate. Inclusion criteria for the study group were working as a cashier for at least 1 year and being female, between 19 and 60 years old. The mean duration of employment as a supermarket cashier in the cohort was 11.49 ± 6.93 years (min. 1 year, max. 31.25 years). The cashiers worked in 4-h shifts; normally they were seated, but could stand if they preferred to do so. Products arrived on a conveyor belt from the right of the cashiers, and the cashier passed them over a laser barcode scanner. The codes of heavy or awkward objects could be registered manually on a keyboard. Products were then carried to the cashiers’ left on a conveyor belt and were bagged by the customers.

Three hundred and four female volunteers from the general population (age range 19–56 years) were recruited from the customers of the same supermarkets, to form a control group. A prepaid gift card was given to reduce the selection bias, as a free ultrasonographical examination could have been a selective factor for subjects with a pre-existing pathology. The inclusion criteria for the control group were being of female sex and aged between 19 and 60 years. Exclusion criteria for the control group were performing repetitive movements of the upper arm or carrying heavy loads for professional reasons. A history of steroid injection within the last three months, previous shoulder
surgery including percutaneous needle aspiration, diagnosed rotator cuff tears, and inflammatory rheumatic disease were considered exclusion criteria for both groups. All subjects signed an informed consent document before participating in the study, and IRB approval was granted. A high-resolution ultrasonography (HRUS) of both shoulders was performed for each subject. The HRUS was performed by a single sonologist using a 15 matrix MHz linear probe (Logiq E9, G.E. Healthcare, Milwaukee, WI, USA). All examinations were performed according to a routine shoulder protocol. Investigations included transverse and longitudinal planes for the supraspinatus, the subscapularis, the infraspinatus, and teres minor tendons, with the aim of assessing the presence of abnormalities affecting the anatomic structures and identifying periarticular calcifications. The supraspinatus tendon was examined with the patient’s shoulder in hyperextension and full internal rotation, with the dorsum of the hand placed on the back in order to expose the supraspinatus from underneath the acromion. These tendons appear as a hyperechoic fibrillar layer, convex-shaped on transverse images, and convex, tapered and inserting at the greater tuberosity on longitudinal views, deep into the deltoid muscle. The infraspinatus and teres minor tendons were examined with the patient’s hand placed on the contralateral shoulder, and the transducer was oriented in the axial plane until the head of the humerus was seen adjacent to the posterior glenoid labrum. Between the deltoid muscle and the external rotator tendons, the subacromial-subdeltoid bursa could be seen, appearing as a hypoechoic line less than 2 mm wide, with a variable amount of peribursal echogenic fat. The subscapularis tendon was identified using the long head of the biceps tendon as a landmark. With the patient seated with the elbow flexed to 90° and the forearm half pronated on the lap, this tendon was easily recognizable, due to the oval-shaped echogenic structure, surrounded by a 1- to 2-mm hypoechoic halo of fluid within the synovial sheath. Anteromedial to the biceps tendon, the hyperechoic subscapularis tendon was identified with a slight external rotation of the glenohumeral joint. A dynamic view of the subscapularis tendon was obtained when the shoulder of the subject was moved into external rotation. Rotator cuff calcifications were defined as echogenic focus with or without posterior acoustic shadowing; due to the compound scanning technique—which increases image resolution by using the multiple lines of site to eliminate artifacts, shadows, and increased edge details—the acoustic extinction behind a calcific deposit was evident only in the case of really dense targets. Discontinuity of collagen fascicle and small focal areas of hypoechoigenicity defined a tendon as non-uniform and were identified as a sign of tendinopathy. The presence of calcifications, which also implies a non-uniformity of the tendon, characterized CT.

Anthropometric characteristics, arm dominance, and systemic diseases that could affect the musculoskeletal system (i.e., diabetes and thyroid disease) were recorded, and all subjects completed the Baecke questionnaire, which measures habitual physical activity levels [23] combining 3 indices—work, sport, and leisure activity. Subjects then responded to a questionnaire that investigated pain of the upper limb, based on the criteria of the Nordic Musculoskeletal Questionnaire [24]. The questionnaire was administered by an orthopedic specialist, to ensure that each subject fully understood the questions. This questionnaire asked whether the subject had shoulder pain: in order to capture chronic, acute, and continuous symptoms; “pain” in our questionnaire was defined as being pain for at least one day a month, or for at least seven consecutive days, in the past year. A positive response was defined as a “symptomatic shoulder.” A negative response was deemed an “asymptomatic shoulder.”

Statistical analysis

The sample size calculation was performed hypothesizing a 20 % presence of calcifications in the non-exposed (control) group. With a sample of 500 subjects (200 exposed, 300 non-exposed), a 1.5 difference in prevalence between the groups can be assessed with sufficient power (80 %, one-tail test—hypothesis that the exposed sample percentage is either greater or lesser than the non-exposed sample value). Risk analyses and the odds ratio with 95 % confidence interval were used to analyze the association between work-related repetitive movement and the presence of shoulder calcifications. All analyses were conducted for both dominant and non-dominant arm separately. The nonparametric Mann–Whitney U test was used to compare the distribution of non-normal variables (age, height, weight, BMI, and physical activity scores) between the two groups. The Chi-square test was used to compare the imaging results of the two groups. Statistical significance was set at \( p < 0.05 \). SPSS 21 for Windows was used for statistical analyses.

Results

As shown in Table 1, there were no statistically significant differences in the anthropometric characteristics of the two groups \( (p < 0.05) \). The right arm was dominant in 93 % of the cashiers and in 93.7 % of the controls. Two cases of Type 2 diabetes were reported in the control group and 1 case among the cashiers. There was 1 case of Type 1 diabetes in the control group and 1 case of thyroid disease in the cashiers group.
Prevalence of CT on ultrasound

The prevalence of calcific tendinopathy was 22.6 % (45 subjects) in the cashiers group and 24.4 % (74 subjects) in the control group. Specifically, CT was present in approximately 20 % of the dominant shoulders of both cashiers and controls and in 13 % of the non-dominant shoulders of both groups (Table 2). We observed 46 subjects with bilateral calcifications, 17 subjects (8.5 %) in the cashiers, and 29 subjects (9.6 %) in the control. There were no statistically significant differences in the prevalence of calcifications between the two groups ($p = 0.585$), either for the dominant shoulder [OR = 0.841 (95 % CI 0.534–1.326)] or for the non-dominant shoulder [OR = 0.988 (95 % CI 0.582–1.326)].

None of the 5 subjects with systemic diseases were found to have CT on ultrasound.

Calcifications and age

There was no statistical difference between the study and control groups when analyzing the mean age and standard deviation of the subjects with calcific tendinopathy in at least one shoulder (Table 3). The subjects with calcifications were significantly older than those without shoulder calcific deposits in both groups ($p < 0.001$). Our results show the prevalence of rotator cuff calcific tendinopathy increasing with age. A rising trend in the prevalence of calcifications in at least one shoulder was noticeable in both groups when divided into 10-year age-classes (Fig. 1). The same rising trend was present also in dominant and non-dominant shoulder separately (Figs. 2, 3).

Calcifications and symptoms

Ninety-four cashiers (47.2 %) reported shoulder pain, compared with 77 subjects (25.4 %) in the control group ($p < 0.001$, $\chi^2 = 25.47$), and this difference was also statistically significant both for the dominant and the non-dominant arm ($p < 0.001$).

Sixty percent of the cashiers with CT were symptomatic, as compared to 43.2 % of the controls with CT, but this difference was not significant ($p = 0.08$, $\chi^2 = 3.14$).

In the CT subgroups, there was a significantly higher prevalence of subjective pain in the control subjects with CT as compared to controls without CT ($p < 0.001$, $\chi^2 = 16.42$), although this clear significant difference was not observed in the cashier CT subgroup ($p = 0.051$, $\chi^2 = 3.80$). However, when the CT subgroups were analyzed according to arm dominance (rather than by subject), the results showed a significantly higher prevalence of pain in shoulders with CT versus shoulders without CT, both for cashiers and controls, and dominant and non-dominant shoulders (all $p < 0.001$, except cashiers dominant shoulder where $p = 0.027$).

Calcifications and Baecke questionnaire scores (physical activity levels)

The mean Baecke score [23] was significantly higher ($p < 0.001$) in the control group compared with the cashiers ($8.3 \pm 0.84$ vs $7.8 \pm 0.91$). Similarly, in the subgroup of subjects with shoulder CT, we observed significantly higher physical activity scores in the controls versus the cashiers ($8.3 \pm 0.91$ vs $7.6 \pm 0.96$, $p = 0.002$).

Table 1  Age and anthropometric characteristics of cashiers and control groups

<table>
<thead>
<tr>
<th></th>
<th>Cashier group ($n = 199$)</th>
<th>Control group ($n = 304$)</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>$37.7 \pm 7.7$ (95 % CI 36.72–38.68)</td>
<td>$38.5 \pm 9.5$ (95 % CI 37.37–39.52)</td>
<td>0.46</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>$163.5 \pm 6.5$ (95 % CI 162.54–164.36)</td>
<td>$163.5 \pm 6.1$ (95 % CI 162.83–164.21)</td>
<td>0.98</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>$61.3 \pm 11.1$ (95 % CI 59.73–62.83)</td>
<td>$62.2 \pm 12.0$ (95 % CI 60.79–63.51)</td>
<td>0.09</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>$22.9 \pm 3.9$ (95 % CI 22.38–23.48)</td>
<td>$23.2 \pm 4.2$ (95 % CI 22.75–23.70)</td>
<td>0.86</td>
</tr>
</tbody>
</table>

Table 2  Prevalence of shoulder calcific/non-calcific tendinopathy on ultrasound in female cashier and control groups (female sample from working-age general population), by arm dominance

<table>
<thead>
<tr>
<th></th>
<th>Cashier ($n = 199$) No. of subjects</th>
<th>Control ($n = 304$) No. of subjects</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dominant shoulder</td>
<td>Calcific tendinopathy 36 (18.1 %)</td>
<td>63 (20.8 %)</td>
<td>0.75</td>
</tr>
<tr>
<td></td>
<td>Tendinopathy 3 (1.5 %)</td>
<td>4 (1.3 %)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Healthy tendons 160 (80.4 %)</td>
<td>236 (77.9 %)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Calcific tendinopathy 26 (13.1 %)</td>
<td>40 (13.2 %)</td>
<td>0.83</td>
</tr>
<tr>
<td>Non-dominant shoulder</td>
<td>Tendinopathy 1 (0.5 %)</td>
<td>3 (1.0 %)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Healthy tendons 172 (86.4 %)</td>
<td>260 (85.8 %)</td>
<td></td>
</tr>
</tbody>
</table>
Discussion

Although the precise mechanisms that induce tendinopathy remain unclear, there is wide consensus on the key role of mechanical stimuli on tendon tissue [25]. It is well known that adequate mechanical loads have beneficial, anabolic effects, whereas excessive mechanical stress results in tendon injuries [26]. The ability of tendon tissue to respond to mechanical stimuli is due to the cells present in tendons. Tenocytes are relatively abundant in tendon tissues and are responsible for the maintenance of tendon homeostasis. Another recently identified tendon cellular type is tendon stem cells (TSCs) that have all the common features of adult stem cells, including self-renewal, clonogenicity, and multipotency. Experimental evidence suggests that moderate mechanical loading is able to promote cellular proliferation in TSC cultures, whereas overloading causes tendinopathy by reducing the number of TSCs and inducing them to differentiate into non-tenocytes (adipocytes, chondrocytes, and osteocytes). According to Wang et al., such non-tenocyte differentiation may explain the histopathologic changes observed at advanced stages of tendinopathy, as lipid deposition, proteoglycan accumulation, and calcification [27]. Thus, tendinopathy may be considered as a cell-mediated failed healing process in which TSCs play a major role, by experiencing aberrant non-tenocyte differentiation under excessive mechanical loading conditions, although a concomitant role of biologic factors, acting together with mechanical components, cannot be excluded.

An analogous pathogenetic model can be hypothesized for calcific tendinopathies, which have similar histologic features to those observed in tendinopathy without calcifications. Calcific deposits are frequently surrounded by chondrocytes in a fibrous, metachromatic, and glycosaminoglycan-rich extracellular matrix [28], suggesting that CT and tendinopathy without calcifications may share common initial pathological mechanisms.

This model for CT may also help to explain the role of the known predisposing factors, whose contribution to the disease is at present unclear. TSCs reside within a niche that is a three-dimensional, specialized microenvironment. It houses stem cells and maintains a balance of quiescence,
self-renewal, and cell-fate commitment [29]. Bi et al. showed that the tendon stem cell niche is composed predominantly of extra cellular matrix (ECM) and that biglycan and fibromodulin are two critical components for the organization of this structure [18]. Any changes in matrix composition affect TSC pool size and divert TSCs from tenogenesis to osteogenesis, leading to ectopic ossification in the tendon [18]. The most important known predisposing factor for CT is diabetes. It has been postulated that exposure to high levels of glucose may cause the glycosylation of several matrix proteins, as observed also in in vitro studies [30]. It is reasonable to speculate that these ECM changes can impact on the structure and functioning of the TSC niches; however, due to the very small number of cases we observed in both groups, we were unable to investigate this theory.

On the basis of these recent theories about the pathogenesis of tendinopathies and in particular, CT, we aimed to investigate if work-related repetitive movements are involved in the development of shoulder CT. Supermarket cashiers seemed to be a suitable study population for this purpose, due to the type of upper limb movements they are required to perform. All three of the etiological factors identified for occupational shoulder disorders (flexed and/ or abducted shoulders, repetitive arm work, and high work speed) are present in cash register operation [31]. Furthermore, the age profile of the majority of the subjects and their sex reflect the population most commonly affected by CT (females between 30 and 60 years).

Our results did not confirm the hypothesis that work-related repetitive movements can induce CT of the rotator cuff tendons. Indeed, we did not observe any statistically significant difference in the prevalence of calcifications between supermarket cashiers and the general population, either by subject, or if dominant and non-dominant arm were considered separately. A first and obvious interpretation of this result may be that the repetitive movements performed by the cashiers do not overload the rotator cuff. At the present time, the overall amount of load that induces tendon pathology has not been estimated, but an excessive mechanical stress may also take the form of “overuse” during time [32]. Indeed, a repetitive load applied to a musculoskeletal structure has to be considered in all its components: intensity (amount of strain), volume (duration of application), and frequency (number of repetitions and sessions per day or week). It can be postulated that if all the load components are not disproportionate, the stress on the rotator cuff does not exceed the capacity of tendons to tolerate load [33]. Although excessive load intensity is unlikely to be generated during the normal cashier activity, high volume and frequency loading might induce relevant stress on the shoulder tendons. In the study population, it seems likely that the duration of work sessions (maximum 4 h) and time intervals between sessions (minimum 2 h) are adequate for tendon recovery. Nevertheless, in the interpretation of our results, it should be borne in mind that load is almost certainly modulated by the interaction of local factors (cytokine and growth factor production) and general characteristics (sex, age, genes, and joint morphology).

The Baecke scores (which measure habitual physical activity) were significantly higher both in the control group versus cashiers, and in the control CT subgroup versus the cashier CT subgroup. Nevertheless, there was no significant difference in the prevalence of CT between cashiers and controls. As with cashier activity, the load caused by overall physical activity (work, leisure and sport) does not seem to be excessive, causing an overload of the shoulder tendon structures.

Interestingly, there was a significantly higher prevalence of subjective shoulder pain in the cashiers compared with the control group, both when the groups were analyzed as a whole, as well as in the two subgroups of subjects with CT. A possible explanation of this difference may be the multifactorial origin of shoulder pain, which is hypothesized to include not only alterations to the anatomic structures, but also the psychosocial work environment [34–36].

A potential limitation of this study is that we have only captured a moment in time, as this is an observational transversal study. Therefore, we cannot draw conclusions regarding the possible evolution of the disease in these two populations, nor exclude that CT could be a late stage of an insidious degenerative tendinopathy induced by overuse. A longitudinal prospective study would be necessary to investigate this hypothesis. However, had this been the case, our imaging results should have at least revealed the initial stages of tendinopathy, whereas only approximately 1% of non-calcific tendinopathies were observed in the cashier group (Table 2). Furthermore, given the relatively long mean employment as a cashier (11.49 ± 6.93 years), this hypothesis would imply that more subjects would have reached this late calcific stage.

In the literature, the data regarding the prevalence of shoulder calcifications focus almost exclusively on populations that are either specifically symptomatic or asymptomatic, rather than investigating the prevalence of CT in general populations. Furthermore, it seems there has been nothing published on the prevalence of CT in women with which to compare our findings. We identified only 2 studies that report prevalence of CT in a “general” population (i.e., the subjects were not chosen on the basis of being either symptomatic or asymptomatic): Bosworth’s 1941 study of 6061 insurance office workers, where CT was found in 2.7% of subjects [2]; and Mavrikakis et al. who observed a 10.3% prevalence in 320 normal subjects [13]. In studies of asymptomatic populations, the reported prevalence of CT was as follows: 7.3% in 1,276 subjects [37]; 7.5%
in 200 subjects [5]; and 20 % in 100 shoulders [3]. These observations contrast with the findings of our study, as we saw a noticeably higher prevalence of CT in both the study and control groups (22.6 and 24.4 %, respectively). This discrepancy may be due firstly to the advances in imaging technology since the above studies were performed, and secondly to our exclusively female populations.

However, if we consider other aspects of CT, rather than just the percentage prevalence, our results were comparable with the literature. Firstly, we observed a prevalence of bilateral calcifications in our cohorts that is similar to the 10 % commonly reported (8.5 %—cashiers, 9.6 %—control) [1]. Our data also support the role of aging in the formation of calcifications, as the percentage of subjects with calcific deposits increased with age in both groups. The overall increase observed with age is comparable with the existing data regarding rotator cuff injuries and aging both in the general population [38, 39] and among women [40]. In the oldest age-class of cashiers, the results for the dominant arm did not show the same steady increase of CT observed in their non-dominant shoulder and in the control group, although this difference was not statistically significant. This may be explained by the fact that despite the large size of our sample, the last age-class was relatively underrepresented.

In the absence of recent data regarding the relationship between TSC and aging, the observed increase of CT with age may be explained on the basis of the current knowledge about stem cells. As TSC can be considered to be a mesenchymal stem cell population, it seems plausible that the findings related to mesenchymal stem cells (MSCs) may also relate to TSC. It has been demonstrated that aging affects MSC potential and consequently alters tissue homeostasis and organ function. In particular, both in animals (rodents and monkeys) and in humans, BMSCs (bone marrow/stem/stromal cells) show impairment in terms of differentiation properties [41, 42].

Conclusion

In this comparison between a group of female cashiers and a control group of female volunteers from the general population, we found that the role of load induced by repetitive movements in the formation of shoulder calcifications had no detectable effect, as calcifications were similarly present in the two groups. The formation of calcifications appeared to be age related, like other features of tendinopathy. To our knowledge, this is the first study that investigates the potential role of occupational repetitive movement, in the development of CT, and that evaluates the prevalence of CT in a specifically female sample of the working-age general population.

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Conflict of interest The authors declare that they have no conflict of interest.

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