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Association of calcific rotator cuff tendinopathy with nephrolithiasis and/or cholelithiasis

A case–control study

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Abstract

This study aimed to examine the association between calcific rotator cuff tendinopathy (RCT) and nephrolithiasis and/or cholelithiasis. A case–control study was conducted on patients diagnosed with RCT between June 2016 and June 2022. RCT was confirmed by ultrasound, and patients were divided into 2 groups: calcific RCT (case) and non-calcific RCT (control). Data were collected retrospectively from electronic medical records and completed by phone calls, looking for a history of nephrolithiasis and/or cholelithiasis; based on clinical features or incidental findings on abdominal and pelvic imaging. A total of 210 patients with RCT were included. Among the 95 cases of calcific RCT, 43 had a history of lithiasis (45.3%) against 23 (20%) from the non-calcific RCT group ($P < .001$); 21 patients suffered from nephrolithiasis (22.1%) and 26 had cholelithiasis (27.4%) versus 10 (8.7%) ($P = .006$) and 16 (13.9%) ($P = .015$) in the non-calcific RCT group, respectively. Logistic regression showed that the independent predictors of calcific RCT included a history of nephrolithiasis (OR, 4.38; 95% CI: 1.61–11.92, $P = .004$) and a history of cholelithiasis (OR, 3.83; 95% CI: 1.64–8.94, $P = .002$). In patients with calcific RCT, the occurrence of lithiasis was significantly associated in the bivariate analysis with higher age, body mass index, fasting blood sugar, and HbA1c (all with $P < .05$), but only with the presence of another site of calcific tendinopathy than the shoulder (OR, 3.11; 95% CI: 1.12–8.65, $P = .03$) in the multivariate analysis. Nephrolithiasis and/or cholelithiasis are associated with calcific RCT, and their presence predicts calcific RCT at least 3 times. Further research is required to determine the common risk factors and preventive measures against lithogenesis in patients with calcific RCT, nephrolithiasis, and cholelithiasis.

Abbreviations: BMI = body mass index, Ca^{2+} = calcium, CI = confidence interval, CPPD = calcium pyrophosphate deposition disease, HBA1c = hemoglobin A1c, MRI = magnetic resonance imaging, OPN = osteopontin, OR = odds ratio, Pi = inorganic phosphate, RCT = rotator cuff tendinopathy.

Keywords: biomineralization, cholelithiasis, hydroxyapatite, nephrolithiasis, rotator cuff, shoulder, tendinopathy

1. Introduction

Calcific rotator cuff tendinopathy (RCT) is a major source of pain and limited mobility of the shoulder, thus responsible for a loss of functional ability in activities of daily living.[1] Its prevalence ranges from 2.7% to 22% according to different authors.[2]

Calcific RCT is characterized by the deposition of calcium hydroxyapatite crystals in the tendons. Although it can affect any tendon in the body, it is most commonly seen in the tendons of the rotator cuff of the shoulders, in particular the supraspinatus tendon.[3] Although the progression of the disease through various stages, from formation to latent stage then to resolution, is more understood, its etiology remains unknown, and the factors associated with its pathogenesis are still unclear.

Recently, particular interest has been given to studying the comorbidities associated with calcific RCT, to better understand the pathophysiology of the disease and its management. This association has been the subject of many recent publications establishing a link between calcific RCT and endocrine diseases, including thyroid disorders,[4] hyperlipidemia,[5] and diabetes.[6] Unique is the study, carried out by Ejnisman, suggesting an association with nephrolithiasis, the latter being present in 32% of patients suffering from calcific RCT.[7]

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All data generated or analyzed during this study are included in this published article [and its supplementary information files].

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However, no association with cholelithiasis has been demonstrated to date.

This study aimed to determine the frequency of occurrence of nephrolithiasis and/or cholelithiasis in patients with calcific RCT, compared to that in patients with non-calcific RCT, and to assess the factors associated with the occurrence of nephrolithiasis and/or cholelithiasis in patients with calcific RCT.

2. Methods

2.1. Study design and sample selection

This case–control study was conducted on patients diagnosed with an RCT between June 2016 and June 2022 and followed in a rheumatology clinic in Beirut, Lebanon.

The inclusion criteria were Lebanese nationality, age over 18 years at diagnosis, and confirmation of the diagnosis of RCT on ultrasound. Exclusion criteria were a history of previous shoulder trauma or surgery or a diagnosis of a malignant lesion affecting the shoulder.

The study population was composed of 2 groups of patients as shown in Figure 1. The first group (cases) comprised patients with calcific RCT, confirmed by ultrasound imaging showing the presence of at least 1 calcification in at least 1 tendon of the rotator cuff. The second group (control) included patients with non-calcific RCT based on the result of ultrasound. Patients in the control group who have calcific tendinopathies on other sites than the shoulder, such as calcific tendinopathy of the gluteus medius, Achilles, and triceps tendons, were excluded.

The study protocol was approved by the ethics committee of the Hôtel-Dieu de France University Hospital in Beirut, Lebanon.

2.2. Data collection

Age at diagnosis, sex, occupation, body mass index (BMI), smoking status, comorbidities such as diabetes, dyslipidemia, thyroid disorders, hyperparathyroidism, gout, calcium pyrophosphate deposition disease (CPPD), and blood test results available at the time of diagnosis were collected from the electronic medical records.

The rest of the data was collected prospectively. Patients included in the study were contacted, and after giving their consent to participate, they answered a questionnaire during a phone call. The purpose of this questionnaire was to look for a history of symptomatic or asymptomatic kidney stones and gallstones, based on the patients' previous symptoms (renal or biliary colic), previous abdominal and pelvic imaging tests (ultrasound or scanner or magnetic resonance imaging (MRI)), and eventual therapeutic procedures (lithotripsy, ureteral stenting, nephrostomy, cholecystectomy, endoscopic retrograde cholangio-pancreatography) which could confirm the diagnosis of nephrolithiasis and/or cholelithiasis.

2.3. Statistical analysis

All data were analyzed using the SPSS software program (Version 26.0, IBM Corp., Armonk, NY, USA).

Statistical analyses were conducted to compare the different parameters, first between the groups of calcific RCT and non-calcific RCT, then within the group of calcific RCT between the patients with and those without a history of lithiasis.

Patients' characteristics are presented using descriptive statistics. The Shapiro–Wilk test was used to test continuous variables

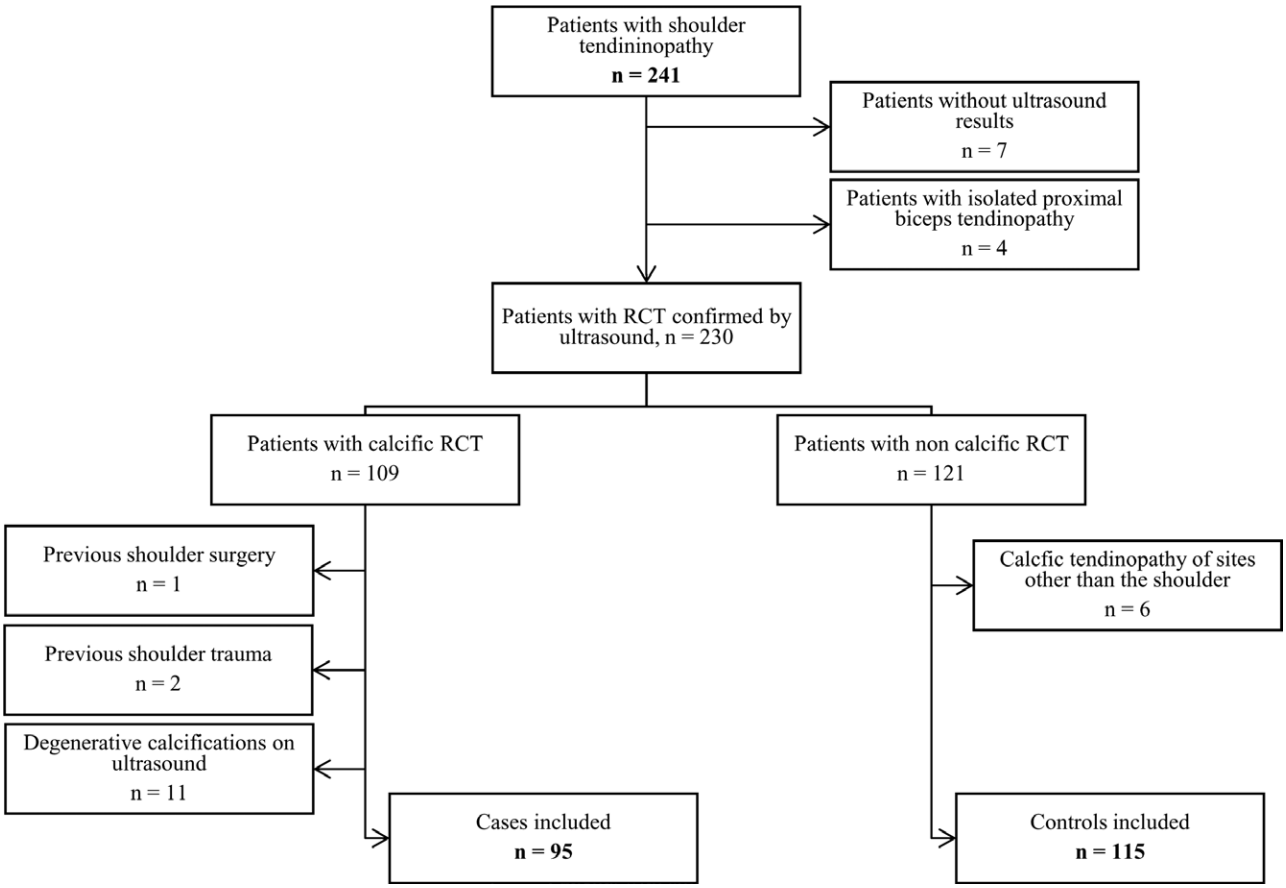

Figure 1. Flowchart of study sample selection.

for normality. Normally distributed continuous variables were expressed as mean ± standard deviation (SD); non-normally distributed continuous variables as median and range; and categorical data were presented using absolute frequencies and percentages. The Student *t* test was used for normal variables, and the nonparametric Mann–Whitney *U* test for non-normal variables. As for the qualitative variables, the Chi-square and Fisher exact tests were used.

Finally, a multivariate analysis by binary logistic regression was performed to identify the independent factors associated with calcific RCT, then those associated with the occurrence of lithiasis. Were included in each multivariate analysis model the explanatory variables that were thought to influence the dependent variable, while respecting the absence of multicollinearity between the independent variables. The statistical significance threshold was set for a *P* value < .05 and the confidence interval (CI) was calculated at 95%.

3. Results

3.1. Baseline patient characteristics

A total of 210 patients diagnosed with RCT were included, 95 with calcific RCT and 115 with non-calcific RCT. The 2 groups of patients were comparable in terms of demographic characteristics with no statistically significant difference as shown in Table 1.

3.2. Ultrasound results

Among the cases of calcific RCT, we noted 98 localized calcifications in the supraspinatus (79.7%), 20 in the subscapularis (16.2%), 5 in the infraspinatus (4.1%), and none in the teres minor tendons. The right side was affected in 66 patients (53.7%), the left side in 57 (46.3%), and bilateral involvement was observed in 28 patients (29.5%). The average size of the calcifications was 11.9 mm, for an extent ranging from 2 to 37 mm.

Table 1

Comparison of demographic characteristics and comorbidities between patients with and those without calcific rotator cuff tendinopathy.

Variables	Groups		*P* value
	Calcific RCT (n = 95)	Non-calcific RCT (n = 115)	
Age at diagnosis (yrs), mean ± SD	55.8 ± 11.6	54.4 ± 14.6	.431
Female, N (%)	65 (68.4)	80 (69.6)	.858
BMI (kg/m^2), mean ± SD	27.4 ± 3.8	27.59 ± 4.4	.696
Occupation, N (%)			
Manual work	26 (27.4)	21 (18.3)	.177
Office work	22 (23.1)	26 (22.6)	
Housework	32 (33.7)	37 (32.2)	
Retirement	15 (15.8)	31 (26.9)	
Smoking, N (%)	38 (40)	30 (26.1)	**.032****
Serum Calcium (mg/dL), mean ± SD	9.5 ± 0.5	9.6 ± 0.5	.11
Diabetes, N (%)	20 (21.1)	26 (22.6)	.786
Dyslipidemia, N (%)	39 (41.1)	41 (35.7)	.422
Thyroid disorders, N (%)	18 (18.9)	12 (10.4)	.079
Hyperparathyroidism, N (%)	7 (7.4)	10 (8.7)	.726
Gout, N (%)	1 (1.1)	1 (0.9)	.892
CPPD, N (%)	1 (1.1)	0 (0)	.270
History of lithiasis, N (%)	43 (45.3)	23 (20)	**<.001*****
Nephrolithiasis	21 (22.1)	10 (8.7)	**.006*****
Cholelithiasis	26 (27.4)	16 (13.9)	**.015***
Renal and biliary lithiasis	4 (4.2)	3 (2.6)	.704

BMI = body mass index, CPPD = calcium pyrophosphate deposit, RCT = rotator cuff tendinopathy.
*P < 0.10; **P < 0.05; ***P < 0.01.

3.3. Clinical results

Among the 95 cases of calcific RCT, 43 had a history of lithiasis (45.3%) against 23 (20%) in the non-calcific RCT group (P < .001); 21 suffered from nephrolithiasis (22.1%) and 26 had cholelithiasis (27.4%), versus 10 (8.7%) (P = .006) and 16 (13.9%) (P = .015), respectively (Table 1).

Multivariate analysis (Table 2) highlighted that a history of lithiasis was an independent predictor of calcific RCT: patients with a history of nephrolithiasis were 4.38 times more likely to develop RCT of a calcific nature (OR, 4.38, 95% CI: 1.61–11.92, P = .004), and those with a history of cholelithiasis were 3.83 times more likely (OR, 3.83, 95% CI: 1.64–8.94, P = .002).

Among patients with calcific RCT and nephrolithiasis and/or cholelithiasis (n = 43), the average age of onset of calcific RCT was 59.3 years compared to 53 years in patients without a history of lithiasis (P = .008), mean BMI was 28.3 kg/m^2 versus 26.6 (P = .02), mean fasting blood sugar was 110 mg/dL versus 97 mg/dL (P = .02), and mean hemoglobin A1c (HbA1c) was 6% versus 5.6 % (P = .032). This group also included 14 obese (32.6%) versus 8 (15.4%) (P = .048), 13 diabetics (30.2%) versus 7 (13.7%) (P = .046), and 16 patients with another site of calcific tendinopathy than the shoulder (37.2%) versus 9 (17.3%) (P = .028).

On the other hand, among patients with calcific RCT, those with a history of lithiasis did not show any statistically significant difference compared to those without a history of lithiasis, in terms of sex, size of calcification, bilaterality of tendinopathy, and prevalence of dyslipidemia, thyroid disorders and hyperparathyroidism (Tables 3 and 4).

Finally, the variables showing significant results in the bivariate analysis were included in a multivariate analysis model. Based on this model, the presence of another site of calcific tendinopathy remained the only predictive factor for the occurrence of nephrolithiasis and/or cholelithiasis among patients with calcific RCT (OR, 3.11, 95% CI: 1.12–8.65, P = .03).

4. Discussion

Our study confirmed, similarly to previous findings,[7] that nephrolithiasis is significantly more common in patients with calcific RCT. However, to the best of our knowledge, this is the first study to reveal a significant positive association between calcific RCT and cholelithiasis. This double association is nevertheless not surprising, since the association between nephrolithiasis and cholelithiasis is already known, but the physio pathological mechanism in question is under investigation.[8,9]

Our study allowed to assess properly the prevalence of nephrolithiasis and cholelithiasis in the study samples since it was not limited to the symptomatic history of the patients, but it also looked for asymptomatic nephrolithiasis and cholelithiasis discovered incidentally on abdominal and pelvic imaging.

Various metabolic diseases, including diabetes and obesity, are risk factors for nephrolithiasis and cholelithiasis,[9] and are also closely linked to the formation of tendon calcifications in the shoulder.[6,10] This is consistent with the results of the present study, which show a higher prevalence of diabetes and obesity, as well as higher BMI, fasting blood glucose, and HbA1c among patients with both calcific RCT and lithiasis. In addition, these patients are older compared to those without lithiasis, consistent with age being a risk factor for kidney stones and gallstones.[9] Moreover, the multivariate analysis allowed us to retain the presence of another site of calcific tendinopathy

Table 2

Binary logistic regression analysis for predictors of calcific tendinopathy among patients with rotator cuff tendinopathy.

Variables	OR	95% CI	*P* value
Age, ≤60 years old	**2.07**	1.01 to 4.27	**.048****
Sex, Female	1.016	0.52 to 2.01	.962
BMI[a]			.242
Overweight	0.69	0.33 to 1.45	.326
Obesity	0.45	0.18 to 1.14	.092
Smoking	**2.01**	1.06 to 3.80	**.032****
Diabetes	0.90	0.40 to 1.99	.786
Dyslipidemia	1.85	0.88 to 3.9	.103
Thyroid disorders	**2.54**	1.05 to 6.17	**.039****
Hyperparathyroidism	0.61	0.19 to 1.99	.412
Lithiasis type[b]			**<.001****
Nephrolithiasis	**4.38**	1.61 to 11.92	**.004****
Cholelithiasis	**3.83**	1.64 to 8.94	**.002****
Renal and cholelithiasis	3.81	0.70 to 20.60	.12

BMI = body mass index, CI = confidence interval, OR = odds ratio.

*$P < 0.10$; **$P < 0.05$; ***$P < 0.01$.

[a]Overweight is defined by a BMI ≥ 25 and < 30 kg/m^2, and obesity by a BMI ≥ 30 kg/m^2. The reference category is a BMI < 25 kg/m^2.

[b]The reference category is the absence of history of lithiasis.

Table 3

Comparison of age, sex, and calcification characteristics among patients with calcific rotator cuff tendinopathy according to the presence of a history of lithiasis.

Variables	History of lithiasis		
	Yes (n = 43)	No (n = 52)	*P* value
Age at diagnosis (yrs), mean ± SD	59.3 ± 12	53 ± 10.7	**.008*****
Female, N (%)	30 (69.8)	35 (67.3)	.797
Calcification size (mm), mean ± SD	11.8 ± 6.9	12.4 ± 6.6	.486
Bilateral involvement, N (%)	13 (30.2)	15 (28.9)	.883
Another calcification site than the shoulder, N (%)	16 (37.2)	9 (17.3)	**.028****

*$P < 0.10$; **$P < 0.05$; ***$P < 0.01$.

than the shoulder, as an independent predictive factor of lithiasis among patients with calcific RCT.

This study failed to show a significant association between calcific RCT and the presence of both nephrolithiasis and cholelithiasis at the same time. However, this lack of association is not to be retained given the limited number of patients with both types of stones in our sample, so it is crucial to include a larger number of patients, or even to systematically perform abdominal and pelvic imaging in all participants to demonstrate whether patients with both types of stones are more likely to be diagnosed with calcific RCT compared to those with only 1 type of stones.

Patients with known calcifications of tendons other than the rotator cuff were excluded from the control group, but not from the first group. Our choice is justified by the fact that calcific tendinopathies all belong to the hydroxyapatite deposition disease, whatever the site affected is[11] and that they can all have a dysmetabolic character, as suggested by a recent work from Giai Via et al[12] associating calcific tendinopathy of the Achilles tendon with diabetes, hypothyroidism, and obesity. This choice is reinforced by the hypothesis that calcific tendinopathy and lithiasis are closely linked but reduces the specificity of the study to calcific tendinopathy of the shoulder.

All these data are consistent with the theory of common pathophysiology between calcific RCT and lithiasis, mainly the pathological biomineralization, which is a physiological process, by which living organisms use mineral materials, including

Table 4

Comparison of modifiable factors among patients with calcific rotator cuff tendinopathy according to the presence of a history of lithiasis.

Variables	Groups		
	History of lithiasis (n = 43)	No history of lithiasis (n = 52)	*P* value
BMI (kg/m^2)	28.3 ± 3.9	26.6 ± 3.5	**.02****
Obesity (%)	14 (32.6)	8 (15.4)	**.048***
Smoking (%)	18 (41.9)	20 (38.5)	.736
Calcemia (mg/dL)	9.5 ± 0.5	9.4 ± 0.2	.437
Diabetes (%)	13 (30.2)	7 (13.7)	**.046****
Fasting blood sugar (mg/dL)	110 ± 32	97 ± 18	**.02****
HbA1c (%)	6 ± 1.3	5.6 ± 0.6	**.032****
Dyslipidemia (%)	20 (46.5)	19 (36.5)	.325
Thyroid disorders (%)	9 (20.9)	9 (17.3)	.654
Hyperparathyroidism (%)	3 (7)	4 (7.7)	>.999

*$P < 0.10$; **$P < 0.05$; ***$P < 0.01$.

calcium (Ca^{2+}) and inorganic phosphate (Pi), to produce mineralized deposits. Most human tissues are supersaturated with Ca^{2+} and Pi ions, but biomineralization only occurs in limited sites, namely cartilage, bones, and teeth.[13] On the contrary, soft tissues have regulatory mechanisms that inhibit the formation of mineral crystals. When these mechanisms are dysfunctional, pathological uncontrolled biomineralization is observed.[14]

Several conditions must be met for pathological biomineralization to occur, and it is possible to identify among these conditions some common features between calcific RCT, nephrolithiasis, and cholelithiasis. Oversaturation is the primary condition in any phenomenon of pathological crystallization. A solution is considered oversaturated when it contains more substance than the solvent can dissolve under normal circumstances. The crystallizing substance differs according to the microcrystalline pathology in question: it is the oversaturation of the extracellular matrix of tendons with calcium and phosphate in the calcific RCT, of the urine with calcium, phosphate, oxalate, uric acid or cystine for the nephrolithiasis, and of the bile with cholesterol or free bilirubin for cholelithiasis. It is this first step which determines the kinetics of crystallization, and consequently, the nature, the number, and the size of the crystals created,[15] but it is not sufficient for the formation of abnormal deposits.

Nucleation is the second step in pathological biomineralization. It is defined by the formation of small nucleus from the oversaturated substance.[15] In calcific RCT, the sites of nucleation are the matrix vesicles. They are extracellular, bilamellar vesicles, approximately 50 to 200 nm in diameter, which provide an ideal microenvironment for biomineralization, as they contain calcium and phosphate transporters, and alkaline phosphatase which induces hydroxyapatite formation.[16] Matrix vesicles are also believed to cause kidney stones, as they have been identified in a mineralized subepithelial tissue of the renal papilla, called Randall plaque.[17] Recent findings show that human renal interstitial fibroblasts undergo osteogenic differentiation and participate in the formation of Randall plaque calcifications,[18] just as shoulder tenocytes undergo metaplasia into chondrocytes to produce hydroxyapatite crystals in the calcific RCT.[19] As for cholelithiasis, its pathophysiology also includes a nucleation stage.[20] However, the vesicular structures involved in biliary lithogenesis do not resemble the matrix vesicles known in calcific RCT or in nephrolithiasis.

Another characteristic observation in pathological biomineralization is the decrease in the expression of calcification inhibitors. Osteopontin (OPN) is a non-collagenous protein identified in samples studying calcific RCT.[1] It is thought to be a powerful inhibitor of calcification because the OPN-positive

multinucleated cells phenotypically resemble osteoclasts and contribute to the resorption of hydroxyapatite deposits from calcified tendon areas.[13] Similarly, renal OPN would play a protective role against nephrolithiasis, by inhibiting the nucleation, growth, and aggregation of calcium oxalate crystals in vitro.[21] Animal studies have also revealed that OPN-deficient mice have an increased risk of kidney crystal deposition. Whereas OPN would have an opposite role in biliary lithogenesis, a recent study found that hepatic OPN deficiency in mice impaired biliary homeostasis and protected against the formation of gallstones.[22]

Another interesting molecule in its inhibitory role is phytate. Grases et al[13] detected a significantly lower phytate concentration in the urine of patients with calcific RCT, compared to healthy controls. Since the concentrations of phytate in the blood and tissues are correlated with those in the urine, then these patients suffering from calcific RCT had a tissue deficiency in this crystallization inhibitor. The prevalence of nephrolithiasis in these patients was also high (19%). Moreover, phytate deficiency has also been observed in the urine of patients with calcium kidney stones.[23]

To sum up, these recent theories of pathological biomineralization make the link between calcific RCT, nephrolithiasis, and cholelithiasis more plausible. The mechanisms presented above must benefit from more precision with scientific advances. In this context, it would be advantageous to propose a prevention plan against stone formation in the management of calcific RCT patients. Obesity and diabetes are modifiable factors on which we could act in this preventive perspective. Practically, the actions to be advocated in patients with calcific RCT would include lifestyle changes, diet, sports activity, maintenance of a normal BMI and close control of blood sugar and HbA1c. In addition, it is known that a higher dietary calcium and phytate intake is associated with a reduced risk of kidney stones,[24] we can speculate from these results that such a diet might be also beneficial in preventing calcific RCT. It is therefore important to add this point to the agenda of future research.

4.1. Limitations of the study

Our study has some limitations that should be discussed. Although we attempted to limit the memorization bias by the collection of available abdominal and pelvic images from the electronic medical records, the information about the history of nephrolithiasis or cholelithiasis was primarily based on the patient recall.

Patients diagnosed with CPPD or gout were not excluded from our study. These pathologies can be confounding factors. In fact, 3 of the patients included are diagnosed with CPPD or gout and have nephrolithiasis or cholelithiasis. It is therefore not possible to know whether the presence of stones in these 3 patients is linked to calcific tendinopathy, CPPD, or gout. However, whereas the association between gout with nephrolithiasis is validated in the literature,[25] its association with cholelithiasis has never been demonstrated. Similarly, CPPD has never been linked to kidney stones or gallstones.

While calcium supplementation has no effect on the development of kidney stones,[24] it was shown that it is associated with a higher risk of calcific RCT.[26] Although no association between the serum calcium level and the presence of calcific RCT was found in our study, we did not assess the association between the calcium supplementation and the presence of lithiasis in patients with calcific RCT.

5. Conclusion

Nephrolithiasis and/or cholelithiasis are associated with calcific RCT, and their presence predicts a calcific type of the RCT by at least 3 times. Further research is required to determine the common risk factors and preventive measures against lithogenesis in patients with calcific RCT, nephrolithiasis, and cholelithiasis.

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