

Cervical Spine Pathology Increases the Risk of Rotator Cuff Tear

A Population-Based Cohort Study

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Background: Patients with cervical radiculopathy typically present with shoulder pain and weakness; these symptoms are similar to those of rotator cuff disease. Studies investigating cervical spine pathology (CSP) as an independent risk factor for rotator cuff tear (RCT) are lacking in the literature.

Purpose: To investigate the risk of RCT among patients with CSP who have undergone cervical discectomy (CD) and to determine whether CD reduces this risk.

Study Design: Cohort study; Level of evidence, 3.

Methods: The authors queried the Taiwan National Health Insurance Research Database for patients diagnosed with CSP between 2004 and 2008 and followed up until the end of 2010. A control cohort comprised patients without CSP who were age- and sex-matched in a 4-to-1 ratio with patients with CSP through propensity score matching. A Cox multivariate proportional hazards model was applied to analyze the risk factors for RCT. After adjustment for confounders, the authors calculated the hazard ratio (HR) and adjusted HR (aHR) between the study and control cohorts. The effects of CD on the risk of RCT were also analyzed.

Results: The study included 3245 patients and 12,980 matched controls. A higher RCT incidence rate was found in the CSP cohort, with an aHR of 1.52 (95% CI, 1.22-1.89; $P < .001$). Patients with CSP who underwent CD had a risk of RCT similar to that of the controls, with an aHR of 1.65 (95% CI, 0.90-3.03; $P > .05$).

Conclusion: Patients with CSP had a 1.52-fold higher risk of RCT than healthy controls. Patients with CSP with CD did not have a high risk of RCT, possibly indicating a protective effect of discectomy against RCT.

Keywords: cervical spine pathology; rotator cuff tear; discectomy; population-based study

Severe rotator cuff disease, such as rotator cuff tear (RCT), leads to a limited range of joint motion and joint dysfunction. Rotator cuff repair surgery, postoperative rehabilitation treatment, and subsequent possible complications are cost-intensive for both patients and medical systems.¹ Moreover, restricted work ability or inability to work for 3 to 9 months after rotator cuff repair surgery may result in loss of income for patients and their families.⁵ Hence, RCT prevention and the provision of effective treatment to maintain a high quality of life among patients and enable them to continue their daily activities are key public health goals in this field.

Anatomically, the suprascapular, subscapular, and axillary nerves, which are derived from the C5, C6, and C7 nerve roots, respectively, innervate the rotator cuff muscle.

The integrity of peripheral innervation of the rotator cuff plays an important role in maintaining the normal function of the shoulder girdle.^{3,13,15,18} For example, Costouros et al² performed an electrodiagnostic test and reported that 54% of patients with massive RCT had peripheral nerve injury and that 3.8% of the RCTs were related to cervical radiculopathy. In addition, Vad et al¹⁵ reported that 7 (28%) of 25 patients with full-thickness RCT had peripheral nerve injury and that 5 of those 7 patients had proximal cervical radiculopathy or axillary nerve injury concomitant with RCT. Further, Ochiai et al¹¹ reported that 46.4% of massive RCTs coexisted with cervical spine lesions.

Zhang et al¹⁸ analyzed data from a large national database and found a significant correlation between rotator cuff disease and cervical radiculopathy. Among patients with cervical radiculopathy, 16% were diagnosed as having an RCT. The researchers also found that among patients older than 60 years of age with cervical radiculopathy, 11% were newly diagnosed with RCT in the following 5 years.

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However, cervical radiculopathy and rotator cuff disease have certain similar comorbidities. For example, patients with diabetes mellitus (DM) have a higher risk of intervertebral degeneration,^{4,7} and DM is a risk factor for RCT.⁶ Thus far, no study has investigated whether cervical spine pathology (CSP) is an independent risk factor for subsequent RCT. To determine this, the bias for comorbidities associated with RCT should be elucidated in a large-scale real-world database study. Through this population-based study, we aimed to investigate the risk of RCT among patients with CSP, with adjustments for comorbidities. Furthermore, we investigated the effect of cervical discectomy (CD) on the risk of RCT among these patients. We hypothesized that patients with CSP had a higher risk of RCT and that CD could reduce this risk.

METHODS

Study Database

The National Health Insurance (NHI) program in Taiwan has covered medical care for its citizens since 1995, and the National Health Insurance Research Database (NHIRD) contains the health insurance data of more than 99% of the population of Taiwan. The NHIRD provides anonymized data for use in epidemiological analyses, and the completeness and accuracy of the NHIRD are guaranteed by the Ministry of Health and NHI Administration Bureau of Taiwan. These data, which are maintained by the National Research Agency for research purposes, were analyzed in this cohort study. The study protocol was approved by our institutional review board. As all patient data were deidentified, the need to obtain informed consent was waived.

The data in the NHIRD include health insurance-related information of disease diagnoses, inpatient and outpatient claims data, medication prescriptions, interventions, surgeries, and arrangement of examinations. All sampled individuals were followed up for outcome identification by using the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM); however, the database does not provide detailed results or inspection reports.

Study Group Selection

The study cohort included all patients who were diagnosed as having CSP from January 1, 2004, to December 31, 2008 (ICD-9-CM codes 721.1 [cervical spondylosis with myelopathy], 722.4 [cervical disk degeneration], 722.71 [myelopathy], 723.0 [cervical spondylopathy], and 723.4 [cervical radiculopathy]). To improve the accuracy of the diagnoses, the analysis included only patients with a main diagnosis of cervical spine disease in at least 2 consecutive outpatient visits. Excluded from the study were patients diagnosed with RCT (ICD-9-CM codes 726.1 [rotator cuff syndrome], 840.4 [rotator cuff sprain], and 727.61 [rotator cuff complete rupture]) before being diagnosed as having cervical spine disease, those with missing birth- or sex-related data, those whose diagnosis was not confirmed by magnetic resonance imaging, and those younger than 18 years of age.

A total of 3245 patients with CSP met the inclusion criteria for this study. These patients were matched with controls (N = 12,980) according to age group (≤ 30 , 31-40, 41-50, 51-60, 61-70, and >70 years) and sex through propensity score matching by using the remaining patient records in the Longitudinal Health Insurance Database 2005 (LHID2005). A subset of the NHIRD, the LHID2005 comprises the claims data of 1 million people who were randomly sampled from among all those registered in the NHI program in 2005. Four control patients were matched to each patient with CSP (Figure 1).

Comorbidities as Confounders

The comorbidities of DM (ICD-9-CM codes 250 and 251), coronary heart disease (ICD-9-CM codes 410 and 412), hypertension (ICD-9-CM codes 401-405), chronic obstructive pulmonary disease (COPD; ICD-9-CM codes 490-496), hyperlipidemia (ICD-9-CM codes 272.0-272.4), autoimmune disease (rheumatoid arthritis [RA] and systemic lupus erythematosus [SLE]; ICD-9-CM codes 714.0 and 710.0), stroke (ICD-9-CM codes 430-438), disorders of the thyroid gland (ICD-9-CM codes 240-246), and gout (ICD-9-CM code 274) were analyzed as the confounders in this study.

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Ethical approval for this study was received from the University of Taipei (reference No. IRB-2018-007).

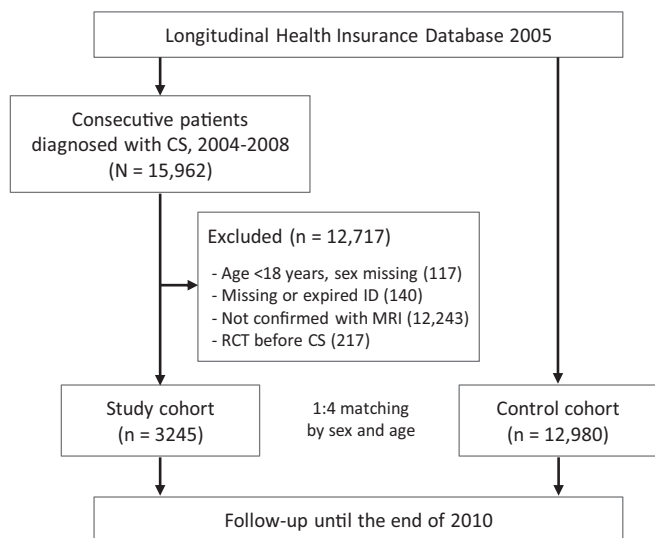


Figure 1. Flowchart of the study design. CS, cervical spine pathology; RCT, rotator cuff tear.

Outcomes

The endpoint of follow-up was the incidence of RCT (ICD-9-CM codes 726.1, 840.4, and 727.61) between the index date and endpoint or until December 31, 2010, whichever occurred first, and final-date observations were censored observations.

Statistical Analysis

The Pearson chi-square test was used to analyze the patient variables and comorbidities in the study and control groups. To analyze the risk and incidence of RCT between these 2 groups, we used the Cox model for hazard ratio (HR) and, after adjustment for possible confounders, for adjusted HR (aHR). Kaplan-Meier hazard curves were used to present the risk of RCT among the CSP cohort during the follow-up period. The influence of CD among patients with CSP was further analyzed, and the crude HR and aHR of RCT between patients with CSP who underwent CD versus those who did not were compared with those of the control cohort as the reference. All data analyses were performed using the Stata package (Version 11) and SAS Statistical Package (Version 9.1.3; SAS Institute). *P* < .05 was considered statistically significant.

RESULTS

In both the CSP and control cohorts, 47.5% of the patients were male. The prevalence of comorbidities such as DM, coronary heart disease, hypertension, stroke, COPD, hyperlipidemia, autoimmune disease (RA and SLE), thyroid disease, and gout was significantly higher in the CSP cohort than in the controls (Table 1).

The mean follow-up period was 4.52 ± 1.59 years for the CSP cohort and 5.97 ± 0.84 years for the controls. The

TABLE 1
Baseline Variables and Comorbidities for Patients With CSP and Controls (N = 16,225)^a

Baseline Variable	Patients With CSP (n = 3245)	Controls (n = 12,980)	<i>P</i>
Characteristics			
Age, y			≥.999
18-30	126 (3.9)	504 (3.9)	
31-40	320 (9.9)	1280 (9.9)	
41-50	826 (25.5)	3304 (25.5)	
51-60	908 (28.0)	3632 (28.0)	
61-70	601 (18.5)	2404 (18.5)	
>70	464 (14.3)	1856 (14.3)	
Sex			≥.999
Male	1541 (47.5)	6164 (47.5)	
Female	1704 (52.5)	6816 (52.5)	
Comorbid disorders			
DM			<.001 ^b
Yes	728 (22.4)	2278 (17.6)	
No	2517 (77.6)	10,702 (82.4)	
Coronary heart disease			
Yes	833 (25.7)	2122 (16.3)	
No	2412 (74.3)	10,858 (83.7)	
Hypertension			<.001 ^b
Yes	1441 (44.4)	4728 (36.4)	
No	1804 (55.6)	8252 (63.6)	
COPD			<.001 ^b
Yes	994 (30.6)	2919 (22.5)	
No	2251 (69.4)	10,061 (77.5)	
Hyperlipidemia			<.001 ^b
Yes	1084 (33.4)	2993 (23.1)	
No	2161 (66.6)	9987 (76.9)	
Autoimmune disease (RA and SLE)			<.001 ^b
Yes	282 (8.7)	491 (3.8)	
No	2963 (91.3)	12,489 (96.2)	
Stroke			<.001 ^b
Yes	613 (18.9)	1371 (10.6)	
No	2632 (81.1)	11,609 (89.4)	
Thyroid disorder			<.001 ^b
Yes	305 (9.4)	617 (4.8)	
No	2940 (90.6)	12,363 (95.2)	
Gout			<.001 ^b
Yes	728 (22.4)	2278 (17.6)	
No	2517 (77.6)	10,702 (82.4)	

^aData are reported as n (%). COPD, chronic obstructive pulmonary disease; CSP, cerebral spine pathology; DM, diabetes mellitus; RA, rheumatoid arthritis; SLE, systemic lupus erythematosus.
^bStatistically significant difference between groups.

average time from diagnosis of CSP to the diagnosis of RCT was 1.28 ± 0.97 years. During the follow-up period, the incidence of RCT was 39 per 10,000 person-years in the control cohort and 84 per 10,000 person-years in the CSP cohort. A significantly higher risk of RCT was found in the CSP cohort, with a crude HR of 1.72 (95% CI, 1.39-2.12; *P* < .001) and an aHR of 1.52 (95% CI, 1.22-1.89; *P* < .001) compared with the controls (Table 2). Figure 2 presents the Kaplan-Meier hazard curves for the risk of RCT in the CSP and control cohorts during the follow-up period.

Among the CSP group, 2986 (92%) did not undergo CD, and the crude HR (1.71; 95% CI, 1.37-2.12; *P* < .001) and

TABLE 2

Incidence and Hazard Ratios for RCT in Patients With and Without CSP During the Follow-up Period (up to 7 Years)^a

	Patients With CSP (n = 3245)	Controls (n = 12,980)
Presence of RCT during follow-up, n (%)	124 (4)	304 (2)
Person-years	14,669	77,507
Incidence per 10,000 person-years	84	39
Crude HR (95% CI)	1.72 ^b (1.39-2.12)	1.00
Adjusted HR (95% CI)	1.52 ^b (1.22-1.89)	1.00

^aAdjusted for patient age, sex, autoimmune disease, diabetes mellitus, hypertension, hyperlipidemia, coronary heart disease, disorders of the thyroid, gout, stroke, and chronic obstructive pulmonary disease. CSP, cervical spine pathology; HR, hazard ratio.

^bStatistically significant difference compared with controls ($P < .001$).

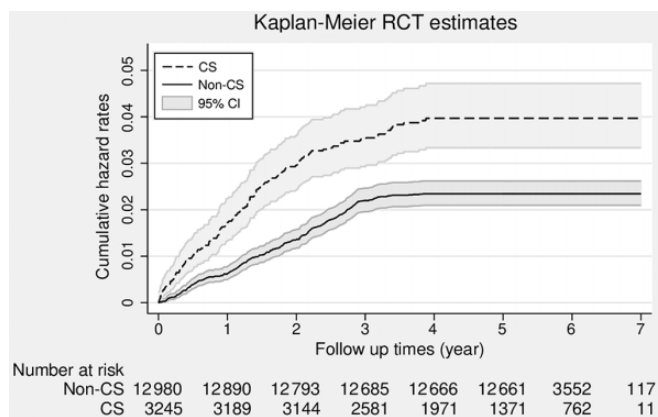


Figure 2. Kaplan-Meier hazard curves for rotator cuff tear (RCT) in patients with cervical spine pathology (CS) and controls over a follow-up period of up to 7 years. Log-rank test, $P < .001$.

aHR (1.51; 95% CI, 1.21-1.88; $P < .001$) indicated a significantly higher risk of RCT in these patients compared with the control cohort (Table 3). The remaining 259 (8%) did undergo CD, and the crude HR and aHR were 1.84 (95% CI, 1.02-3.37; $P < .05$) and 1.65 (95% CI, 0.90-3.03; $P > .05$), respectively, indicating that patients with CSP who underwent CD did not have a significantly elevated risk of RCT compared with controls (Table 3).

DISCUSSION

Through this population-based cohort study, we found that CSP is a risk factor for RCT. Evidence regarding the association between CSP and RCT is limited; however, we observed that patients with CSP are at a 1.52-fold risk (95% CI, 1.22-1.89; $P < .001$) of RCT compared with those without CSP. Clinically, patients with CSP can be

TABLE 3

Incidence and Hazard Ratios for RCT in Patients With CSP and Without CD During the Follow-up Period (up to 7 Years)^a

	Patients With CSP		
	Without CD (n = 2986)	With CD (n = 259)	Controls (n = 12,980)
Presence of RCT during follow-up, n (%)	113 (4)	11 (4)	304 (2)
Person-years	13,397	1271	77,507
Incidence per 10,000 person-years	84	86	39
Crude HR (95% CI)	1.71 ^c (1.37-2.12)	1.84 ^b (1.02-3.37)	1.00
Adjusted HR (95% CI)	1.51 ^c (1.21-1.88)	1.65 (0.90-3.03)	1.00

^aAdjustment for patient age, sex, autoimmune disease, diabetes mellitus, hypertension, hyperlipidemia, coronary heart diseases, disorders of the thyroid, gout, stroke, and chronic obstructive pulmonary disease. CD, cervical diskectomy; CSP, cervical spine pathology; HR, hazard ratio; RCT, rotator cuff tear.

^bStatistically significant difference compared with controls ($P < .05$).

^cStatistically significant difference compared with controls ($P < .001$).

evaluated with shoulder weakness and limited range of motion. In addition to cervical spine-related lesions, a diagnosis of RCT should be considered in such cases. Our findings further show that compared with controls, patients with CSP who underwent CD did not have a significantly elevated risk of RCT after adjustment for possible confounders ($P > .05$). Thus, CD could reduce the risk of symptoms of RCT in patients with CSP.

Cervical radiculopathy and rotator cuff disease are associated with similar comorbidities. For example, patients with DM have a higher risk of intervertebral degeneration,^{4,7} and DM is a risk factor for RCT.⁶ In addition, hyperlipidemia may be a risk factor for intervertebral pathology,¹⁰ and a previous study has reported an association between hyperlipidemia and rotator cuff disease.¹⁷ Further, patients with RA have a higher risk of RCT.¹⁶ RA often affects the cervical spine and may manifest as radiculopathy, myelopathy, quadriplegia, and, in extreme circumstances, sudden death.¹² Thus, to eliminate the effect of comorbidities, in the present study, we used the Cox proportional hazards model after adjusting for possible confounders to provide a more convincing conclusion.

Our findings were compatible with those of Zhang et al,¹⁸ who analyzed data from a large national database and found a significant correlation between rotator cuff disease and CSP and increasing patient age. Concomitant diagnoses of CSP and rotator cuff disease were identified in 86,928 patients; 13% of 679,112 patients were diagnosed as having rotator cuff disease, and 16% of 531,177 patients were diagnosed as having CSP. The association between CSP and rotator cuff disease increased significantly with age; 13% of patients with CSP who were younger than 60 years had rotator cuff disease, but this percentage increased to 25% in

patients with CSP who were older than 60 years ($P < .0001$). Among patients older than 60 years who were newly diagnosed as having CSP, 11% either were newly diagnosed with or underwent an operation for rotator cuff disease within 5 years.

Some studies have used nerve electrodiagnostic tests to confirm neuropathy in patients with RCT. Costouros et al² reported that among 26 patients with severe RCT, 14 (54%) had accompanying peripheral nerve injury, 7 (27%) had suprascapular nerve neuropathy, 4 (15.4%) had axillary nerve neuropathy, 2 (7.7%) had an injury to the upper trunk of the brachial plexus, and 1 (3.8%) had cervical radiculopathy. Vad et al¹⁵ examined 25 patients with RCT and found that 4 (16%) patients had an injury to the upper trunk of the brachial plexus and 3 (12%) had suprascapular nerve neuropathy or C6 cervical radiculopathy. Patients with combined RCT and neuropathy have been found to have significantly higher levels of muscle atrophy than patients with RCT. Ochiai et al¹¹ investigated 341 patients with symptomatic RCTs and found that 82 of them were evaluated with pseudoparalysis. Of these 82 patients, 40 (48.8%) had cervical spondylotic amyotrophy, 16 (19.5%) had suprascapular neuropathy, and 1 (1.2%) had axillary nerve palsy. Therefore, a high prevalence of neuropathy combined with massive RCT is observed in pseudoparalyzed shoulders. These reports indicate a strong correlation between CSP and RCT and that their combined occurrence increases the severity of RCT. However, these retrospective studies have not investigated any causal relationship between these 2 diseases.

The pathogenetic mechanism of higher RCT risk among patients with CSP could be explained by impaired stability or shoulder strength due to neuropathy. Several animal studies have investigated the relationship between rotator cuff rupture and nerve damage,^{8,9,13,14} and results have shown that the severity of tendon tear and fatty degeneration is related to the size of the rupture and accompanying nerve damage.^{8,9} In a rodent model in which neurectomy was performed proximal to the suprascapular nerve, Sasaki et al¹⁴ found that the rotator cuff tendon became stiffer and that less force was required to tear it. They concluded that neuropathy proximal to the suprascapular nerve, such as cervical spine or brachial plexus neuropathy, may lead to weakness and stiffness of the rotator cuff tendon, which may accelerate tendon degeneration and rupture. Therefore, neuropathy may be the cause of RCT.

In this analysis, we also found that patients with CSP who had not undergone CD had a higher risk of subsequent RCT than the controls did. Compared with the control group, the rate of future RCT was not statistically significant in patients who had undergone CD. Our explanation for this phenomenon is that if a patient does not undergo CD, neuropathy from the herniated disk of the spine cannot be relieved, resulting in weakening of the rotator cuff muscle and an inability to resist the upward force provided by the deltoid. This ultimately results in subacromial impingement syndrome, which gradually leads to tendon wear, tear, and rupture.

This study has several possible limitations. First, this analysis used ICD-9-CM codes from the NHIRD to determine

the diagnoses of CSP and RCT. The NHI administration has established several verification committees that regularly review medical records and verify the accuracy of the diagnostic codes to enhance diagnostic accuracy. In addition, we used only consecutively coded cases to avoid using inaccurate codes in the database records. Therefore, although the accuracy of the diagnoses in the database cannot be confirmed, the registered diagnoses of CSP and RCT can be considered to have high accuracy. Second, our study design was a retrospective cohort study, and information on possible confounders (such as lifestyle, obesity, smoking, and alcohol consumption), the severity of cervical spine disease, and nerve electrodiagnostic study data cannot be obtained from the study database.

A third limitation was that the definition of RCT was mainly based on clinical symptoms with diagnostic codes, and not all the patients with RCT were confirmed by MRI. This may mean that some patients with asymptomatic RCT could not be identified in our study. Finally, our study was based on diagnostic and intervention coding from the health insurance database, and information regarding the severity and cause of CSP and the detailed surgical methods used could not be retrieved from the database. Detailed information such as the level of surgery, whether fusion was performed, and type of decompression could not be analyzed in this study. Further prospective studies are warranted to determine the association between the type of intervention used or the severity of CSP and the risk of RCT.

CONCLUSION

The results of our longitudinal population-based cohort study revealed that patients with CSP have a 1.52-fold higher risk of RCT than those without CSP do. In addition, compared with controls, a higher incidence of RCT is observed among patients with CSP who have not undergone CD; however, no difference in the incidence was observed between the controls and patients with CSP who had undergone CD. This indicates that patients with CSP who have undergone CD do not have a high risk of RCT.

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