Received: 29 July 2024 | Revised: 1 September 2024 | Accepted: 5 September 2024

DOI: 10.1111/dom.15965

ORIGINAL ARTICLE

WILEY

Are patients with newly diagnosed frozen shoulder more likely to be diagnosed with type 2 diabetes? A cohort study in UK electronic health records

Brett P. Dyer PhD[1] | Claire Burton PhD[2] | Trishna Rathod-Mistry PhD[3] | Miliça Blagojevic-Bucknall PhD[2] | Danielle A. van der Windt PhD[2]

[1]Griffith Biostatistics Unit, Griffith Health, Griffith University, Gold Coast, Australia

[2]Primary Care Centre Versus Arthritis, School of Medicine, Keele University, Staffordshire, UK

[3]Pharmaco- and Device Epidemiology Group, Centre for Statistics in Medicine, Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, UK

Correspondence
Brett P. Dyer, PhD, Griffith Biostatistics Unit, Griffith Health, Griffith University, Gold Coast, 4215, Australia.
Email: b.dyer@griffith.edu.au

Funding information
National Institute for Health and Care Research; Versus Arthritis, Grant/Award Number: 21899

Abstract

Aim: To estimate the association between newly diagnosed frozen shoulder and a subsequent diagnosis of type 2 diabetes in primary care.

Methods: We conducted an age-, gender- and practice-matched cohort study in UK primary care electronic medical records containing 31 226 adults diagnosed with frozen shoulder, matched to 31 226 without frozen shoulder. Patients with pre-existing diabetes were excluded. Variables were identified using established Read codes. A hazard ratio (HR) for the association between incident frozen shoulder and a subsequent type 2 diabetes diagnosis was estimated using shared frailty Cox regression, adjusted for age and gender. To determine whether the association could be explained by increased testing for type 2 diabetes based on other risk factors, a secondary analysis involved re-running the Cox model adjusting for the mean number of consultations per year, hyperlipidaemia, hypertension, obesity, thyroid dysfunction, ethnicity, deprivation, age, and gender.

Results: Participants with frozen shoulder were more likely to be diagnosed with type 2 diabetes (1559 out of 31 226 patients [5%]) than participants without frozen shoulder (88 out of 31 226 patients [0.28%]). The HR for a diagnosis of type 2 diabetes in participants with frozen shoulder versus people without frozen shoulder was 19.4 (95% confidence interval [CI] 15.6–24.0). The secondary analysis, adjusting for other factors, produced similar results: HR 20.0 (95% CI 16.0–25.0).

Conclusions: People who have been newly diagnosed with frozen shoulder are more likely to be diagnosed with type 2 diabetes in the following 15.8 years. The value of screening patients presenting with frozen shoulder for type 2 diabetes at presentation, alongside more established risk factors, should be considered in future research.

KEYWORDS
adhesive capsulitis, diabetes, diagnosis, frozen shoulder

ISAC protocol registration number 19_219R.

1 | INTRODUCTION

Frozen shoulder is a common painful condition[1,2] that restricts shoulder function[3–6] and reduces quality of life.[7,8] The exact aetiology of frozen shoulder is not known, but it is believed that diabetes is involved in the development of frozen shoulder.[9–11] A systematic review has indicated that diabetes is associated with the onset of frozen shoulder; however, due to limitations in existing studies, causality cannot be inferred from the current epidemiological evidence.[9] It is hypothesized that persistent hyperglycaemia may promote glycation,[12] which produces advanced glycation end products that could cause collagen cross-linking and the capsular fibrosis that is observed in frozen shoulder.[13–16] Additionally, a hyperglycaemic state has been shown to induce proinflammatory cytokines,[17] which would help to initiate the inflammatory process that precedes capsular fibrosis in frozen shoulder.[11,18,19]

The prevalence of diabetes in people with frozen shoulder is estimated at 30% (95% confidence interval [CI] 24%–37%).[20] Some authors have suggested that people with frozen shoulder should be tested for type 2 diabetes,[21,22] but there is a lack of high-quality evidence about whether patients who are newly diagnosed with frozen shoulder are more likely to have undiagnosed type 2 diabetes. Four studies containing 135, 88, 77 and 18 patients with frozen shoulder but no history of diabetes found that 27%, 39%, 17% and 17%, respectively, tested positive for type 2 diabetes.[21–24] The results of these four small studies motivated this study, which uses a large sample of patients that are representative of the population presenting to primary care with frozen shoulder in the United Kingdom to further investigate whether a newly diagnosed frozen shoulder could predict a future type 2 diabetes diagnosis.

Estimates suggest that 1 million people in the United Kingdom have undiagnosed type 2 diabetes.[25] Furthermore, estimates suggest that, on average, people go undiagnosed for 7 years before being diagnosed with type 2 diabetes.[26,27] When type 2 diabetes is undiagnosed, uncontrolled hyperglycaemia can result in complications such as cardiovascular disease,[28,29] retinopathy,[30] nephropathy,[31,32] neuropathy,[33–35] and certain musculoskeletal conditions.[36] Early detection and treatment of diabetes reduces morbidity and mortality.[37] The National Institute for Health and Care Excellence (NICE) has published guidelines on the early detection of diabetes, and a national strategy is in place to prevent diabetes in those identified as being at high risk.[38,39] The NICE guidelines advise that patients with certain conditions (including polycystic cardiovascular disease, hypertension, obesity, stroke, polycystic ovary syndrome, a history of gestational diabetes and mental health problems) are risk-assessed for diabetes.[38] We therefore aimed to determine whether frozen shoulder should be considered a condition that should trigger such a risk assessment by examining whether an incident frozen shoulder diagnosis is associated with a subsequent type 2 diabetes diagnosis.

2 | METHODS

This matched cohort study was conducted in a longitudinal UK primary care electronic health record database, the Clinical Practice Research Datalink (CPRD) GOLD.[40] CPRD GOLD had 18.5 million patients in February 2020, with 3 million of those being currently registered.[41] Clinical information such as diagnoses, symptoms, prescriptions, referrals, and patient characteristics were (at the time of this study) recorded as Read codes.[40] The population of CPRD GOLD is representative of the UK population with respect to age, gender and ethnicity.[40] The CPRD data were linked to Index of Multiple Deprivation (IMD) data and Hospital Episode Statistics data to obtain patient deprivation and ethnicity data.[40,42] The study's start date was set as 1 May 2004 to coincide with the introduction of the Quality Outcomes Framework, which improved the quality of coding in primary care, in particular for diabetes care.[43,44]

The frozen shoulder group contained participants who were at least 18 years old and had their first ever frozen shoulder Read code between 1 May 2004 and 31 December 2017. The index date was defined as the date on which the patient received their first-ever frozen shoulder Read code. Each patient with frozen shoulder was age-, gender- and practice-matched to an individual without a frozen shoulder diagnosis prior to, or on, the index date of their matched pair. (Age was matched on to the same year.) The participants without frozen shoulder adopted the index date of their matched pair and needed to be alive and contributing data to a CPRD practice on their index date. Participants with pre-existing diabetes Read codes, or Read codes assigned on their index date, were excluded. Each participant was required to have at least 2 years of up-to-standard data[40] on the index date.

Patients were followed from their index date until the end of the study's follow-up (17 February 2020), date of type 2 diabetes diagnosis, date of death (derived from CPRD data), date of transfer to a non-CPRD practice, or date of last CPRD data collection.

Read code lists[45–48] can be found in Appendix A.

2.1 | Analysis

A hazard ratio (HR) for the association between a first-ever frozen shoulder diagnosis and a subsequent type 2 diabetes diagnosis was estimated using Cox regression.[49] A gamma-distributed shared frailty term was used to account for the lack of independence between matched pairs.[50] In the primary analysis, age and gender were adjusted for in the Cox model (matching was also carried out on age, gender and practice). A secondary analysis was conducted to determine whether the association between an incident frozen shoulder and a following diagnosis of type 2 diabetes could be explained by increased screening for type 2 diabetes based on other factors known to be more common in people with frozen shoulder and associated with a diagnosis of type 2 diabetes (mean number of consultations per year, hyperlipidaemia, hypertension, obesity, thyroid dysfunction, ethnicity, deprivation, gender, and age). These factors were identified via literature review and by communicating with subject-matter experts. Category levels for variables are provided in Appendix B. The missing data indicator method was used to handle missing data.[51] A complete-case analysis was performed as a sensitivity analysis to determine the impact that missing data could have on the results.

In the Cox models, participant follow-up was censored on the date of the earliest event of death, transfer to a non-CPRD practice, or end of follow-up. Participants without frozen shoulder on the index

date were censored on the date of frozen shoulder diagnosis if they were diagnosed with a frozen shoulder after the index date. Schoenfeld residual plots were used to check for signs that the proportional hazards model had been violated.[52]

Kaplan–Meier plots for participants with frozen shoulder and participants without frozen shoulder are presented.[53]

Data were manipulated and cleaned using Stata version 14.0[54] and analysed using RStudio version 1.2.5033.[55]

TABLE 1 Table summarizing characteristics of study participants.

	Frozen shoulder $n = 31\,226$ (50%)	No frozen shoulder $n = 31\,226$ (50%)	All $n = 62\,452$
Mean (SD) follow-up duration[a], years	8.58 (4.29)	8.59 (4.24)	8.58 (4.27)
Mean age (years)	59.78 (13.24)	59.78 (13.24)	59.78 (13.24)
Gender, *n* (%)			
Male	11 825 (37.87)	11 825 (37.87)	23 650 (37.87)
Female	19 401 (62.13)	19 401 (62.13)	38 802 (62.13)
Obesity, *n* (%)			
Obese	7678 (24.59)	6182 (19.80)	13 860 (22.19)
Not obese	21 094 (67.55)	20 837 (66.73)	41 931 (67.14)
Missing	2454 (7.86)	4207 (13.47)	6661 (10.67)
Hypertension, *n* (%)			
Diagnosed	9159 (29.33)	8632 (27.64)	17 791 (28.49)
Not diagnosed	22 067 (70.67)	22 594 (72.36)	44 661 (71.51)
Hyperlipidaemia, *n* (%)			
Diagnosed	4580 (14.67)	3694 (11.83)	8274 (13.25)
Not diagnosed	26 646 (85.33)	27 532 (88.17)	54 178 (86.75)
Thyroid dysfunction, *n* (%)			
Diagnosed	2986 (9.56)	2804 (8.98)	5790 (9.27)
Not diagnosed	28 240 (90.44)	28 422 (91.02)	56 662 (90.73)
Ethnicity, *n* (%)			
Bangladeshi	28 (0.09)	23 (0.07)	51 (0.08)
Black African	87 (0.28)	78 (0.25)	165 (0.26)
Black Caribbean	129 (0.41)	111 (0.36)	240 (0.38)
Black—other	38 (0.12)	38 (0.12)	76 (0.12)
Chinese	61 (0.20)	26 (0.08)	87 (0.14)
Indian	247 (0.79)	138 (0.44)	385 (0.62)
Mixed	71 (0.23)	78 (0.25%)	149 (0.24)
Other Asian	127 (0.41)	61 (0.20)	188 (0.30)
Other	220 (0.70)	222 (0.71)	442 (0.71)
Pakistani	103 (0.33)	87 (0.28)	190 (0.30)
Missing	7227 (23.14)	7407 (23.72)	14 634 (23.43)
White	22 888 (73.30)	22 957 (73.52)	45 845 (73.41)
IMD quintile, *n* (%)			
Least deprived quintile	7634 (24.45)	7875 (25.22)	15 509 (24.83)
2nd least deprived quintile	6923 (22.17)	7029 (22.51)	13 952 (22.34)
3rd least deprived quintile	6771 (21.68)	6592 (21.11)	13 363 (21.40)
4th least deprived quintile	5496 (17.60)	5341 (17.10)	10 837 (17.35)
Most deprived quintile	4378 (14.02)	4005 (12.83)	8383 (13.42)
Missing	24 (0.08)	384 (1.23)	408 (0.65)

[a]Defined as the time from the index date to the earliest of: End of study (17 February 2020), date of death, date of transfer to a non- Clinical Practice Research Datalink (CPRD) practice, or date of last CPRD data collection.
Abbreviation: IMD, Index of Multiple Deprivation.

3 | RESULTS

3.1 | Sample characteristics

The study contained 31 226 patients with their first-ever frozen shoulder Read code and 31 226 patients without a frozen shoulder Read code before or on their index date (Table 1). The participants with frozen shoulder were more likely to be female (62%) than male (38%), and the mean (SD) age of frozen shoulder diagnosis was 59.8 (13.2) years. The median (SD) duration of follow-up was 8.6 (4.3) years, which was similar in those with and without frozen shoulder.

3.2 | Association between incident frozen shoulder and a following type 2 diabetes diagnosis

Across all participants, 1647 participants (2.6%) had an incident type 2 diabetes Read code during follow-up. In those with frozen shoulder, 1559 out of 31 226 participants (5.0%) were diagnosed with type 2 diabetes during follow-up, compared to 88 out of 31 226 participants (0.3%) in those without frozen shoulder. The difference in the probability of being diagnosed with type 2 diabetes can be seen in the Kaplan–Meier plot (Figure 1). The 5-year Kaplan–Meier estimate for the frozen shoulder group was 96.2% compared to the 15.8-year estimate of 93.1%, showing that more type 2 diabetes diagnoses occurred in the 5 years after frozen shoulder diagnosis than in the following 10.8 years (Figure 1).

The association between incident frozen shoulder and a subsequent incident diagnosis of type 2 diabetes was estimated to be HR 19.4 (95% CI 15.6–24.0). In the secondary analysis, when adjusting for other factors associated with frozen shoulder and being diagnosed with type 2 diabetes, the association estimate was similar (HR 20.0, 95% CI 16.0–25.0). Complete-case analysis (76.1% of patients had complete data) provided a similar HR (21.3, 95% CI 16.5–27.6). Thus, regardless of how missing data were handled in the analysis, the results and conclusions drawn were the same.

The Schoenfeld residual plots did not show evidence that the proportional hazards assumption was violated in the Cox models (Appendix C).

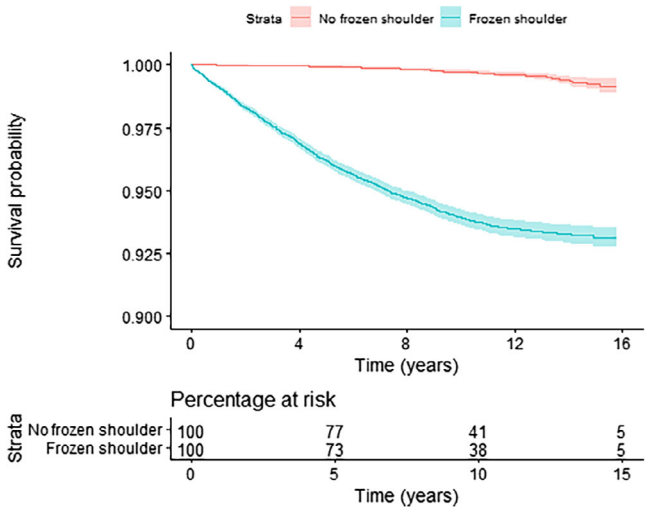

FIGURE 1 Plot of Kaplan–Meier estimates (with 95% confidence intervals) for not being diagnosed with type 2 diabetes among patients with frozen shoulder compared to patients without frozen shoulder, with a percentage at risk (of developing type 2 diabetes) table.

4 | DISCUSSION

Type 2 diabetes is highly prevalent in people with frozen shoulder. This has led to researchers hypothesizing that people presenting with frozen shoulder may be more likely to have undiagnosed type 2 diabetes than people without frozen shoulder. This study suggests that there is likely to be a strong association between a new diagnosis of frozen shoulder and a subsequent diagnosis of type 2 diabetes in the following (up to 15.8) years. The association remained when accounting for other factors known to be more common in people with frozen shoulder and associated with a diagnosis of type 2 diabetes, which further suggests that frozen shoulder may potentially be a clinically important predictor of undiagnosed type 2 diabetes. It is important to state that this research *does not* support a hypothesis that frozen shoulder is a cause of type 2 diabetes.[56,57] Rather, a potential reason why type 2 diabetes is highly prevalent in people with frozen shoulder is because it has been hypothesized that type 2 diabetes could be a cause of frozen shoulder.[9–11]

Previous studies have shown conflicting results, but this could be attributable to differences in study setting, small sample sizes, or differences in the methods used to establish existing and newly diagnosed type 2 diabetes. Previous estimates from cross-sectional studies for new diagnoses of type 2 diabetes among patients presenting with incident frozen shoulder are 27% ($n = 135$),[21] 39% ($n = 88$),[22] 17% ($n = 77$),[23] 17% ($n = 18$)[24] and 0% ($n = 122$).[58] The two studies with the largest percentages of patients with undiagnosed type 2 diabetes had 'invited patients to be tested' and thus there may be participation bias, where patients are more likely to accept the invitation to be tested if they suspect they are more at risk of having type 2 diabetes.

This study utilized CPRD data to attempt to understand whether the results from these smaller studies could be replicated in a large UK primary care electronic health record database that simulates usual care. The results of this study are consistent with previous studies, but the interpretation of results differs due to differences in the way this study was designed and the healthcare system in which it was set. The use of electronic health records was beneficial to provide a large, representative sample of people presenting to primary care with frozen shoulder, but electronic health records have limitations. The main limitation is that, unlike previous studies, not every participant in this study was tested for type 2 diabetes. Read codes were used to identify the presence of type 2 diabetes, but this relies on type 2 diabetes being diagnosed either through symptomatic presentation or health screening. This is especially problematic as type 2 diabetes is a disease that can go undetected for substantial periods of time; however, strategies are continuously being put in place to

identify and prevent diabetes. It is likely that the true number of patients that developed type 2 diabetes during follow-up was underestimated given the lower estimate of the incidence of type 2 diabetes of 5% in this cohort. Further, the participants with frozen shoulder may already have been undergoing testing more frequently for type 2 diabetes due to general practitioners (GPs) being aware that frozen shoulder is associated with type 2 diabetes. In a survey of 714 UK GPs, 60% reported that they would run blood tests on patients presenting with frozen shoulder. Although the survey response rate was low (14.7%) and the respondents may not conduct screening in all patients with frozen shoulder, the survey results would suggest that our results may contain bias.[59] If participants with frozen shoulder were being tested more frequently than people without frozen shoulder, then the association would have been exaggerated. Adjusting for other risk factors associated with type 2 diabetes may only partly have accounted for this risk of detection bias. The absence of a short, sharp drop in the Kaplan–Meier plot during the first few months of the study shows that people presenting with frozen shoulder continued to be diagnosed with type 2 diabetes at later time points. Given the later time of diagnosis, these participants would have been less likely to have been diagnosed as a result of a test conducted when initially presenting with frozen shoulder. It may need further study to determine whether adding a diagnosis of frozen shoulder will increase the performance of existing diabetes risk assessment tools (such as QDiabetes[60–63]) and to what extent it will be cost-effective to test every patient presenting with frozen shoulder for type 2 diabetes. However, this work motivates this to be considered, particularly within the high-priority context of diabetes detection and prevention.

In conclusion, this study has shown that people presenting to primary care with frozen shoulder are more likely to have a subsequent diagnosis of type 2 diabetes than people without frozen shoulder. The results of this study suggest patients presenting with frozen shoulder should be considered for risk assessment and/or testing for diabetes, although further research is needed to determine the added predictive performance of a frozen shoulder diagnosis alongside other risk factors, and the cost-effectiveness of implementing screening of this additional group.

AUTHOR CONTRIBUTIONS

All authors contributed to the conception and design of the study. Data cleaning and analysis was conducted by Brett Dyer. All authors contributed to the editing and approval of the final manuscript.

ACKNOWLEDGEMENTS

This study was supported by the Versus Arthritis PhD scholarship scheme (grant number 21899). Claire Burton is funded by a National Institute for Health Research (NIHR) Clinical Lectureship. The views expressed are those of the authors and not necessarily those of the National Health Service (NHS), the NIHR, or the Department of Health and Social Care.

This study is based in part on data from the CPRD obtained under licence from the UK Medicines and Healthcare products Regulatory Agency. The data are provided by patients and collected by the NHS as part of their care and support. The interpretation and conclusions contained in this study are those of the author/s alone.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest.

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/dom.15965.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

ORCID

Brett P. Dyer https://orcid.org/0000-0001-8039-7631
Claire Burton https://orcid.org/0000-0003-4688-3075
Trishna Rathod-Mistry https://orcid.org/0000-0002-6369-4746
Miliça Blagojevic-Bucknall https://orcid.org/0000-0001-7230-7771
Danielle A. van der Windt https://orcid.org/0000-0002-7248-6703

REFERENCES

1. van der Windt DA, Koes BW, de Jong BA, Bouter LM. Shoulder disorders in general practice: incidence, patient characteristics, and management. *Ann Rheum Dis*. 1995;54(12):959-964.
2. Walker-Bone K, Palmer KT, Reading I, Coggon D, Cooper C. Prevalence and impact of musculoskeletal disorders of the upper limb in the general population. *Arthritis Care Res*. 2004;51(4):642-651.
3. Cho C-H, Jin H-J, Kim DH. Comparison of clinical outcomes between idiopathic frozen shoulder and diabetic frozen shoulder after a single ultrasound-guided IntraArticular corticosteroid injection. *Diagnostics*. 2020;10:370.
4. Ando A, Hamada J, Hagiwara Y, Sekiguchi T, Koide M, Itoi E. Short-term clinical results of manipulation under ultrasoundguided brachial plexus block in patients with idiopathic frozen shoulder and diabetic secondary frozen shoulder. *Open Orthop J*. 2018;12(1):99-104.
5. Duzgun I, Baltacı G, et al. Manual therapy is an effective treatment for frozen shoulder in diabetics: an observational study. *Jt Dis Relat Surg*. 2012;23(2):94-99.
6. Vastamaki H, Ristolainen L, et al. Range of motion of diabetic frozen shoulder recovers to the contralateral level. *J Int Med Res*. 2016;44(6):1191-1199.
7. Bagheri F, Ebrahimzadeh MH, Moradi A, Bidgoli HF. Factors associated with pain, disability and quality of life in patients suffering from frozen shoulder. *Arch Bone Jt Surg*. 2016;4(3):243-247.
8. Harris AH, Youd J, Buchbinder R. A comparison of directly elicited and pre-scored preference-based measures of quality of life: the case of adhesive capsulitis. *Qual Life Res*. 2013;22:2963-2971.
9. Dyer BP, Rathod-Mistry T, Burton C, van der Windt D, Bucknall M. Diabetes as a risk factor for the onset of frozen shoulder: a systematic review and meta-analysis. *BMJ Open*. 2023;13:e062377.

10. Hsu CL, Sheu WH. Diabetes and shoulder disorders. *J Diabetes Investig*. 2016;7(5):649-651.
11. Cucchi D, Marmotti A, de Giorgi S, et al. Risk factors for shoulder stiffness: current concepts. *Joints*. 2017;5(4):217-223.
12. Goldin A, Beckman JA, Schmidt AM, Creager MA. Advanced glycation end products. *Circulation*. 2006;114:597-605.
13. Paul RG, Bailey AJ. Glycation of collagen: the basis of its central role in the late complications of ageing and diabetes. *Int J Biochem Cell Biol*. 1996;28(12):1297-1310.
14. Negre-Salvayre A, Salvaryre R, et al. Hyperglycemia and glycation in diabetic complications. *Antioxid Redox Signal*. 2009;11(12):3037-3096.
15. Hwang KR, Murrell GA, et al. Advanced glycation end products in idiopathic frozen shoulders. *J Shoulder Elbow Surg*. 2016;25(6):981-988.
16. Hsu JE, Anakwenze OA, Warrender WJ, Abboud JA. Current review of adhesive capsulitis. *J Shoulder Elbow Surg*. 2011;20(3):502-514.
17. Shanmugam N, Reddy MA, Guha M, Natarajan R. High glucose-induced expression of proinflammatory cytokine and chemokine genes in monocytic cells. *Diabetes*. 2003;52(2):1256-1264.
18. Pietrzak M. Adhesive capsulitis: an age-related symptom of metabolic syndrome and chronic low-grade inflammation? *Med Hypotheses*. 2016;88:12-17.
19. Lho YM, Ha E, Cho CH, et al. Inflammatory cytokines are overexpressed in the subacromial bursa of frozen shoulder. *J Shoulder Elbow Surg*. 2013;22(5):666-672.
20. Zreik NH, Malik RA, et al. Adhesive capsulitis of the shoulder and diabetes: a meta-analysis of prevalence. *Muscles, Ligaments Tendons J*. 2016;6(1):26-34.
21. Rai SK, Kashid M, Chakrabarty B, Upreti V, Shaki O. Is it necessary to screen patient with adhesive capsulitis of shoulder for diabetes mellitus? *J Family Med Prim Care*. 2019;8(9):2927-2932.
22. Tighe CB, Oakley WS. The prevalence of a diabetic condition and adhesive capsulitis of the shoulder. *South Med J*. 2008;101(6):591-595.
23. Maini S, Bansal A, Arora R, Bansal S. Prevalence of diabetes in patients with frozen shoulder. *J Evol Med Dent Sci*. 2019;8(11):804-806.
24. Withrington RH, Girgis FL, et al. A comparative study of the aetiological factors in shoulder pain. *Br J Rheumatol*. 1985;24:24-26.
25. Whicher CA, O'Neill S, et al. Diabetes in the UK: 2019. *Diabet Med*. 2020;37:242-247.
26. Harris MI, Klein R, Welborn TA, Knuiman MW. Onset of NIDDM occurs at least 4–7 yr before clinical diagnosis. *Diabetes Care*. 1992;15(7):815-819.
27. Thompson TJ, Engelgau MM, Hegazy M, et al. The onset of NIDDM and its relationship to clinical diagnosis in egyptian adults. *Diabet Med*. 1996;13(4):337-340.
28. Low Wang CC, Hess CN, Hiatt WR, Goldfine AB. Atherosclerotic cardiovascular disease and heart failure in type 2 diabetes mellitus—mechanisms, management, and clinical considerations. *Circulation*. 2016;133:2459-2502.
29. Petrie JR, Guzik TJ, Touyz RM. Diabetes, hypertension, and cardiovascular disease: clinical insights and vascular mechanisms. *Can J Cardiol*. 2018;34(5):575-584.
30. Duh EJ, Sun JK, Stitt AW. Diabetic retinopathy: current understanding, mechanisms, and treatment strategies. *JCI Insight*. 2017;2(14):e93751.
31. Gross JL, de Azevedo MJ, Silveiro SP, Canani ĹH, Caramori ML, Zelmanovitz T. Diabetic nephropathy: diagnosis, prevention, and treatment. *Diabetes Care*. 2005;28(1):164-176.
32. Lim AKH. Diabetic nephropathy—complications and treatment Diabetic nephropathy—complications and treatment. *Int J Nephrol Renov Dis*. 2014;7:361-381.
33. Feldman EL, Callaghan BC, Pop-Busui R, et al. Diabetic neuropathy. *Nat Rev Dis Primers*. 2019;5(41):41.
34. Said G. Diabetic neuropathy—a review. *Nat Rev Neurol*. 2007;3:331-340.
35. Callaghan BC, Cheng H, et al. Diabetic neuropathy: clinical manifestations and current treatments. *Lancet Neurol*. 2012;11(6):521-534.
36. Sozen T, Başaran NÇ, Tınazlı M, et al. Musculoskeletal problems in diabetes mellitus. *Eur J Rheumatol*. 2018;5(4):258-265.
37. Herman WH, Ye W, Griffin SJ, et al. Early detection and treatment of type 2 diabetes reduce cardiovascular morbidity and mortality: a simulation of the results of the Anglo-Danish-Dutch study of intensive treatment in people with screen-detected diabetes in primary care (ADDITION-Europe). *Diabetes Care*. 2015;38(8):1449-1455.
38. National Institute for Health and Care Excellence. *Type 2 Diabetes: Prevention in People at High Risk*. National Institute for Health and Care Excellence (NICE); 2017 20/11/2023; Available from: https://www.nice.org.uk/guidance/ph38/chapter/Recommendations.
39. NHS England. *NHS Diabetes Prevention Programme National Service Specification*. NHS England; 2023; Available from: https://www.england.nhs.uk/publication/nhs-dpp-national-service-specification/.
40. Herrett E, Gallagher AM, Bhaskaran K, et al. Data resource profile: clinical practice research datalink (CPRD). *Int J Epidemiol*. 2015;44(3):827-836.
41. CPRD, *Release Notes: CPRD GOLD February 2020. [Unpublished Report]*. CPRD; 2020.
42. Padmanabhan S, Carty L, Cameron E, Ghosh RE, Williams R, Strongman H. Approach to record linkage of primary care data from clinical practice research datalink to other health-related patient data: overview and implications. *Eur J Epidemiol*. 2019;34:91-99.
43. Kontopantelis E, Reeves D, Valderas JM, Campbell S, Doran T. Recorded quality of primary care for patients with diabetes in England before and after the introduction of a financial incentive scheme: a longitudinal observational study. *BMJ Qual Saf*. 2013;22(1):53-64.
44. Khunti K, Gadsby R, Millett C, Majeed A, Davies M. Quality of diabetes care in the UK: comparison of published quality-of-care reports with results of the quality and outcomes framework for diabetes. *Diabet Med*. 2007;24:1436-1441.
45. B P Dyer, C Burton, et al. *Frozen Shoulder—Read Codes*. Keele University; 2023; Available from: https://researchdata.keele.ac.uk/78/.
46. B P Dyer, C Burton, et al. *Type 2 Diabetes*. Keele University; 2023; Available from: https://researchdata.keele.ac.uk/80/.
47. B P Dyer, C Burton, et al. *Diabetes (all Types)—Read Codes*. Keele University; 2023; Available from: https://researchdata.keele.ac.uk/77/.
48. B P Dyer, C Burton, et al. *Thyroid Dysfunction—Read Codes*. Keele University; 2023; Available from: https://researchdata.keele.ac.uk/79/.
49. Cox DR. Regression models and life-tables. *J R Stat Soc B: Methodol*. 1972;34(2):187-220.
50. Gutierrez RG. Parametric frailty and shared frailty survival models. *Stata J*. 2002;2(1):22-44.
51. Groenwold RHH, White IR, Donders ART, Carpenter JR, Altman DG, Moons KGM. Missing covariate data in clinical research: when and when not to use the missing-indicator method for analysis. *CMAJ*. 2012;184(11):1265-1269.
52. Lee M, Han J. Statistical methods and models in the analysis of time to event data. *Ann Transl Med*. 2020;8(4):73.
53. Rich JT, Neely JG, Paniello RC, Voelker CCJ, Nussenbaum B, Wang EW. A practical guide to understanding Kaplan-Meier curves. *Otolaryngol Head Neck Surg*. 2010;143(3):331-336.
54. StataCorp. *Stata Statistical Software: Release 16*. StataCorp LLC; 2019.
55. RStudio Team. *RStudio: Integrated Development Environment for R*. RStudio, Inc.; 2019.
56. Losciale JM, Bullock GS, et al. Description, prediction, and causation in sport and exercise medicine research: resolving the confusion to improve research quality and patient outcomes. *J Orthop Sports Phys Ther*. 2023;53(7):375.
57. Hernán MA, Hsu J, Healy B. A second Chance to get causal inference right: a classification of data science tasks. *Chance*. 2019;32(1):42-49.
58. Gundtoft PH, Attrup ML, et al. Diabetes mellitus affects the prognosis of frozen shoulder. *Dan Med J*. 2020;67(10):A02200071.

59. Artus M, van der Windt DA, Afolabi EK, et al. Management of shoulder pain by UK general practitioners (GPs): a national survey. *BMJ Open*. 2017;7(6):e015711.
60. QDiabetes. 09/01/2024; Available from: https://qdiabetes.org/index.php.
61. Hippisley J, Coupland C, et al. Development and validation of QDiabetes-2018 risk prediction algorithm to estimate future risk of type 2 diabetes: cohort study. *BMJ*. 2017;359:j5019.
62. Hippisley-Cox J, Coupland C, Robson J, Sheikh A, Brindle P. Predicting risk of type 2 diabetes in England and Wales: prospective derivation and validation of QDScore. *BMJ*. 2009;338:b880.
63. Collins GS, Altman DG. External validation of QDSCORE for predicting the 10-year risk of developing type 2 diabetes. *Diabet Med*. 2011;28(5):599-607.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Dyer BP, Burton C, Rathod-Mistry T, Blagojevic-Bucknall M, van der Windt DA. Are patients with newly diagnosed frozen shoulder more likely to be diagnosed with type 2 diabetes? A cohort study in UK electronic health records. *Diabetes Obes Metab*. 2024;1-7. doi:10.1111/dom.15965