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Risk factors associated with degenerative glenohumeral osteoarthritis

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ABSTRACT

Objective Glenohumeral (GH) osteoarthritis (OA) is the third most common large joint disease, after hip and knee OA. This study aimed to identify risk factors for GH OA.

Methods We used data from the Dallas Shoulder cohort, including individuals aged 40–85. Those with confirmed GH OA based on X-ray were cases, and those without were controls. Univariate, least absolute shrinkage and selection operator and multivariate analyses identified risk factors, including age, body mass index (BMI), sex, work-related shoulder problems, shoulder disability, dislocation, previous trauma, surgery, smoking, hypertension, diabetes, depression, heart disease, OA, night pain and overall sleep quality.

Results A total of 1827 cases and 1556 controls were identified for GH OA. In univariate analysis, significant associations with GH OA were found for increasing age (>40 to ≤50: OR 3.29, 95% CI 2.44 to 4.45; >50 to ≤60: OR 5.90, 95% CI 4.49 to 7.77; >60 to ≤70: OR 12.18, 95% CI 9.22 to 16.08 and >70: OR 16.54, 95% CI 12.47 to 21.94), higher BMI (≤19: OR 1.44, 95% CI 1.01 to 2.04; >25 to ≤30: OR 1.57, 95% CI 1.32 to 1.86; >30 to ≤35: OR 1.85, 95% CI 1.54 to 2.22 and >35: OR 1.77, 95% CI 1.28 to 2.45), prior shoulder injury (OR 1.30; 95% CI 1.12 to 1.50), shoulder surgery history (OR 0.71; 95% CI 0.57 to 0.87), shoulder pain at night (OR 1.35; 95% CI 1.07 to 1.70) and hypertension (OR 0.70; 95% CI 0.60 to 0.81). In multivariate analysis, significant associations remained for age (>40 to ≤50: OR 2.99, 95% CI 2.21 to 4.06; >50 to ≤60: OR 5.48, 95% CI 4.14 to 7.23; >60 to ≤70: OR 11.22, 95% CI 8.44 to 14.88 and >70: OR 16.65, 95% CI 12.45 to 22.17), BMI (≤19: OR 1.49, 95% CI 1.01 to 2.20; >25 to ≤30: OR 1.45, 95% CI 1.20 to 1.77; >30 to ≤35: OR 1.70, 95% CI 1.39 to 2.09 and >35: OR 1.78, 95% CI 1.25 to 2.55) and previous shoulder trauma (OR 0.80; 95% CI 0.68 to 0.94).

Conclusion We identified increasing age and higher BMI as factors associated with GH OA. Due to the large sample size, many risk factors were assessed. Since the shoulder is not a weight-bearing joint, the BMI-GH OA link is likely molecular and systemic, warranting further investigation.

Level of evidence Prognostic level III.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Glenohumeral (GH) osteoarthritis (OA) is the third most affected joint after the hip and knee OA.
⇒ GH OA has complex and multifactorial aetiology.

WHAT THIS STUDY ADDS

⇒ Increase in age and body mass index (BMI) are significantly associated with higher odds of GH OA.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ The positive correlation between age and BMI for GH OA suggests that adopting a proactive approach may facilitate the early detection of GH OA and potentially mitigate disease progression.
⇒ The present study involving a larger cohort establish a critical foundational for future mechanistic investigations into the causal pathways associated with age and BMI, which may subsequently advance clinical practice.

INTRODUCTION

Osteoarthritis (OA) is the most common cause of chronic pain in adults and leads to disability.[1] As of 2020, an estimated 595 million people (7.6% of the world's population) had OA[2] and this is expected to increase by the year 2050.[1] OA of glenohumeral joint (GH) is the third most common large joint OA preceded by knee and hip OA.[3] GH OA is a degenerative joint disease involving articular cartilage, subchondral and periarticular bone and soft tissues such as ligaments, muscle and synovium. GH OA has a prevalence of 16%–20% in middle-aged and elderly population.[3] In the USA, for the period of 2007–2015, the surgical procedures performed to treat GH OA increased by 322%.[4]

There are multiple risk factors reported for development of GH OA. These include older age, female sex, obesity, hypertension, smoking, prior injury, genetic predisposition and lower muscle mass.[5–9] A multidisciplinary approach is needed to treat and prevent the increasing burden of GH OA by developing a thorough understanding of the risk factors involved. Prior studies reporting on

risk factors for GH OA were limited by a relatively small sample size and a lack of comprehensive analyses encompassing all potential risk factors. To address this gap, we performed an exhaustive assessment of risk factors associated with GH OA in a large sample of patients.

MATERIALS AND METHODS

Patient population and case definition

We used data from the Dallas Shoulder Cohort for this study using the Strengthening the Reporting of Observational Studies in Epidemiology guidelines.[10] Patients with shoulder pain seen in the shoulder clinic at the University of Texas Southwestern Medical Center from September 2013 to March 2023 were provided with a baseline health and shoulder questionnaire.[11] Patients aged 40–85 years who completed this questionnaire and had a confirmed diagnosis of GH OA on X-rays, either by a radiologist or a shoulder specialist, were selected as cases. Remaining patients without evidence for GH OA were controls. Rotator cuff tear is also a degenerative disease that can confound the results of an analysis comparing cases and controls of GH OA. Hence, patients with rotator cuff tears were excluded from our study.

Outcome definition

The presence of GH OA was confirmed based on X-ray imaging records. Patients with other shoulder pathologies such as, acromioclavicular OA, calcific tendinitis, scapular dyskinesis, fracture, biceps pathology, painful or failed total shoulder arthroplasty, subacromial impingement, cuff arthropathy, general shoulder pain, shoulder instability, acromioclavicular joint problem, subacromial/subdeltoid bursitis, elbow pathology, labral problem, necrosis, nerve problem, sternoclavicular joint problem and others were recruited as a control. Online supplemental table 1 represents the definition of variables used in the analysis.

Selection of risk factors

Based on earlier published literature and expert opinion, a total of 17 variables were selected as potential risk factors of interest.[5 6 8 12–15] Data for these variables were gathered from patient questionnaires and from chart review in the electronic health record system. Risk factors such as age, sex, race and ethnicity were categorised as demographic characteristics; height and weight as physical factors; marital status, alcohol consumption, smoking status, occupation and education level as social factors; hypertension, hyperlipidaemia, diabetes, rheumatoid arthritis, depression, anxiety, arthritis as comorbid conditions and duration of symptoms, history of overhead activity, dominant shoulder, affected shoulder, contralateral shoulder problems, shoulder pain at night were categorised as other shoulder-related factors.

Statistical analysis

Univariate analyses were performed for variables such as age, sex, body mass index (BMI), work-related shoulder problems, shoulder disability, shoulder dislocation, hypertension, diabetes, depression, OA, shoulder pain at night and overall sleep quality. Age was categorised into five groups: ≤40 years, >40 to ≤50 years, >50 to ≤60 years, >60 to ≤70, >70 years and older. BMI was categorised based on WHO's classification: ≤19, >19 to ≤25, >25 to ≤30, >30 to ≤35 and >35. Variables statistically significant in univariate analysis were used to construct the least absolute shrinkage and selection operator (LASSO) model to select the variables strongly associated with GH OA. In LASSO, we used indirect estimates using the Akaike information criteria method and an optimal model was selected. Further, we performed multivariate regression analysis on variables significant after LASSO. The dose–response association and potential relationship of age and BMI for GH OA were also evaluated by Bradford Hill method.[16] A $p<0.05$ was considered statistically significant. To address missing data, a test for missing data at random and non-random levels was performed. All the analyses were performed by using SAS V.9.4 and LASSO graph was prepared using R studio V.4.2.2.[17]

Patient and public involvement

No patients or members of the public were involved in the design, conduct, reporting or dissemination of the study.

Equity, diversity and inclusion statement

This study is committed to ensure representation and inclusivity across various dimensions. Our authorship team reflects a broad range of professional backgrounds and career stages, from graduate students and residents to MDs, PhDs and esteemed department chairs. The team is also demographically diverse, encompassing individuals from various racial, ethnic and gender backgrounds, with equal opportunities for women. Our patient cohort also mirrors this diversity, representing a wide range of racial, demographic, socioeconomic and occupational groups. Additionally, statistical analyses were conducted considering sex as a biological variable, with a focus on addressing gender disparities in risk factors.

RESULTS

Patient characteristics

Our patient population included 1827 cases and 1556 controls. Both cases and controls had a similar distribution of males and females (table 1) . We observed less than 10% missing data in the cohort, and the missingness appeared to be random as indicated by a p>0.05.

Univariate analysis

Increasing age categories were associated with significantly higher odds for GH OA (OR 3.29; 95% CI 2.44 to 4.45 for >40 to ≤50 years vs <40 years; OR 5.90; 95% CI 4.49 o 7.77 for >50 to ≤60 years vs <40 years; OR 12.18; 95% CI 9.22 to 16.08 for >60 to ≤70 years vs <40 years and OR 16.54; 95% CI 12.47 to 21.94 for >70 years vs <40 years). An increase in BMI also showed a significant

Table 1 Descriptive statistics of the studied population

Variables	Controls N=1556 (%)	Cases N=1827 (%)	P value (χ^2)
Age (in years)			<0.0001*
≤40	452 (29.05)	83 (4.54)	
>40 to ≤50	289 (18.57)	175 (9.58)	
>50 to ≤60	363 (23.33)	394 (21.57)	
>60 to ≤70	248 (15.94)	555 (30.38)	
>70	204 (13.11)	620 (33.94)	
BMI (kg/m^2)			<0.0001*
≤ 19	87 (5.59)	95 (5.20)	
>19 to ≤25	505 (32.46)	407 (22.28)	
>25 to ≤30	496 (31.88)	629 (34.43)	
>30 to ≤35	272 (17.48)	411 (22.50)	
>35	196 (12.60)	285 (15.60)	
Sex			0.53
Male	790 (50.77)	917 (50.19%)	
Female	756 (48.59)	892 (48.82)	
Missing	10 (0.64)	18 (0.99)	
Work-related shoulder problem			0.28
No	1376 (88.43)	1603 (87.74)	
Yes	111 (7.13)	122 (6.68)	
Missing	69 (4.43)	102 (5.58)	
Shoulder disability			0.34
No	1430 (91.90)	1660 (90.86)	
Yes	57 (3.66)	66 (3.61)	
Missing	69 (4.43)	101 (5.53)	
Shoulder dislocated			0.33
No	1269 (81.56)	1525 (83.47)	
Yes	186 (11.95)	194 (10.62)	
Missing	101 (6.49)	108 (5.91)	
Previous shoulder trauma			0.0002*
No	839 (53.92)	1069 (58.51)	
Yes	607 (39.01)	594 (32.51)	
Missing	110 (7.07)	164 (8.98)	
Shoulder surgery			0.0033*
No	1333 (85.67)	1487 (81.39)	
Yes	161 (10.35)	253 (13.85)	
Missing	62 (3.98)	87 (4.76)	
Smoking			0.29
No	975 (62.66)	1175 (64.31)	
Yes	85 (5.46)	80 (4.38)	
Missing	496 (31.88)	571 (31.31)	
Heart disease			0.0225*
No	1296 (83.29)	1458 (79.80)	
Yes	144 (9.25)	218 (11.93)	
Missing	116 (7.46)	151 (8.26)	
Hypertension			<0.0001*
No	982 (63.11)	1017 (55.67)	
Yes	463 (29.76)	681 (37.27)	
Missing	111 (7.13)	129 (7.06)	
Lung disease			<0.0001*
No	1211 (77.83)	1316 (72.03)	
Yes	215 (13.82)	351 (19.21)	
Missing	130 (8.35)	160 (8.76)	
Diabetes			0.15
No	1223 (78.60)	1385 (75.81)	
Yes	211 (13.56)	284 (15.54)	
Missing	122 7.84)	158 (8.65)	
Depression			0.28
No	1174 (75.45)	1336 (73.13)	
Yes	245 (15.75)	321 (17.57)	
Missing	137 (8.80)	170 (9.30)	
Osteoarthritis			0.0042*
No	1051 (67.54)	1138 (62.29)	
Yes	351 (22.56)	495 (27.09)	
Missing	154 (9.90)	194 (10.62)	
Shoulder pain at night			0.0039*
Yes	1354 (87.02)	1619 (88.62)	
No	172 (11.05)	152 (8.32)	
Missing	30 (1.93)	56 (3.07)	
Overall sleep quality			0.77
Very good	160 (10.28)	213 (11.66)	
Fairly good	683 (43.89)	792 (43.35)	
Fairly bad	457 (29.37)	519 (28.41)	
Very bad	192 (12.34)	226 (12.37)	
Missing	64 (4.11)	77 (4.21)	

*Significant variable.
BMI, body mass index.

association with the likelihood of diagnosis GH OA (OR 1.57; 95% CI 1.32 to 1.86 for >25 to ≤30 vs >19 to ≤25; OR 1.85; 95% CI 1.54 to 2.22 for >30 to ≤35 vs >19 to ≤25 and OR 1.77; 95% CI 1.28 to 2.45 for >35 vs >19 to ≤25). However, sex was not significantly associated with GH OA (OR 0.98; 95% CI 0.85 to 1.12).

Prior shoulder injury (p=0.0002), a history of shoulder surgery (p=0.0034), a report of shoulder pain at night (p=0.0042) and the presence of OA in other joints (p=0.0043) were significantly associated with GH OA. Comorbid conditions such as hypertension did show a significant association (p<0.0001), but smoking (p=0.2918) and diabetes (p=0.1520) did not show any significant difference (table 2).

Table 2 Univariable analysis of the studied population in GH OA cases and controls

Variables		Estimate (SE)	OR	95% CI	P value‡
Age	≤40*				<0.000†
	>40 to ≤50	1.19 (0.15)	3.29	2.44 to 4.45	
	>50 to ≤60	1.77 (0.13)	5.90	4.49 to 7.77	
	>60 to ≤70	2.50 (0.14)	12.18	9.22 to 16.08	
	>70	2.80 (0.14)	16.54	12.47 to 21.94	
BMI	≤19	0.36 (0.17)	1.441	1.01 to 2.04	<0.0001†
	>19 to ≤25*				
	>25 to ≤30	0.45 (0.08)	1.570	1.32 to 1.86	
	>30 to ≤35	0.62 (0.09)	1.854	1.54 to 2.22	
	>35	0.57 (0.16)	1.774	1.28 to 2.45	
Sex	Female*				0.53
	Male	0.01 (0.06)	1.01	0.88 to 1.16	
Work-related shoulder problem	No*				0.28
	Yes	0.05 (0.13)	1.06	0.81 to 1.38	
Shoulder disability	No*				0.35
	Yes	0.00 (0.18)	1.00	0.69 to 1.43	
Shoulder dislocation	No*				0.33
	Yes	0.14 (0.10)	1.15	0.93 to 1.42	
Previous shoulder trauma	No*				0.0002†
	Yes	0.26 (0.07)	1.30	1.12 to 1.50	
Shoulder surgery	No*				0.0034†
	Yes	−0.34 (0.10)	0.71	0.57 to 0.87	
Smoking	No*				0.29
	Yes	0.24 (0.16)	1.28	0.93 to 1.75	
Heart disease	No*				0.0229†
	Yes	−0.29 (0.11)	0.74	0.59 to 0.92	
Hypertension	No*				<0.0001†
	Yes	−0.35 (0.07)	0.70	0.60 to 0.81	
Lung disease	No*				0.0001†
	Yes	−0.40 (0.09)	0.66	0.55 to 0.80	
Diabetes	No*				0.15
	Yes	−0.17 (0.09)	0.84	0.69 to 1.02	
Depression	No*				0.28
	Yes	−0.14 (0.09)	0.86	0.72 to 1.04	
Osteoarthritis	No*				0.0043†
	Yes	−0.26 (0.08)	0.76	0.65 to 0.90	
Shoulder pain at night	No*				0.0042†
	Yes	0.30 (0.11)	1.35	1.07 to 1.70	
Overall sleep quality	Very good	−0.13 (0.11)	0.87	0.69 to 1.09	0.77
	Fairly good*				
	Fairly bad	0.02 (0.08)	1.02	0.86 to 1.20	
	Very bad	−0.01 (0.11)	0.98	0.79 to 1.22	

*Reference group.
†Significant variable.
‡P value obtained from type 3 analysis.
BMI, body mass index; GH, glenohumeral; OA, osteoarthritis.

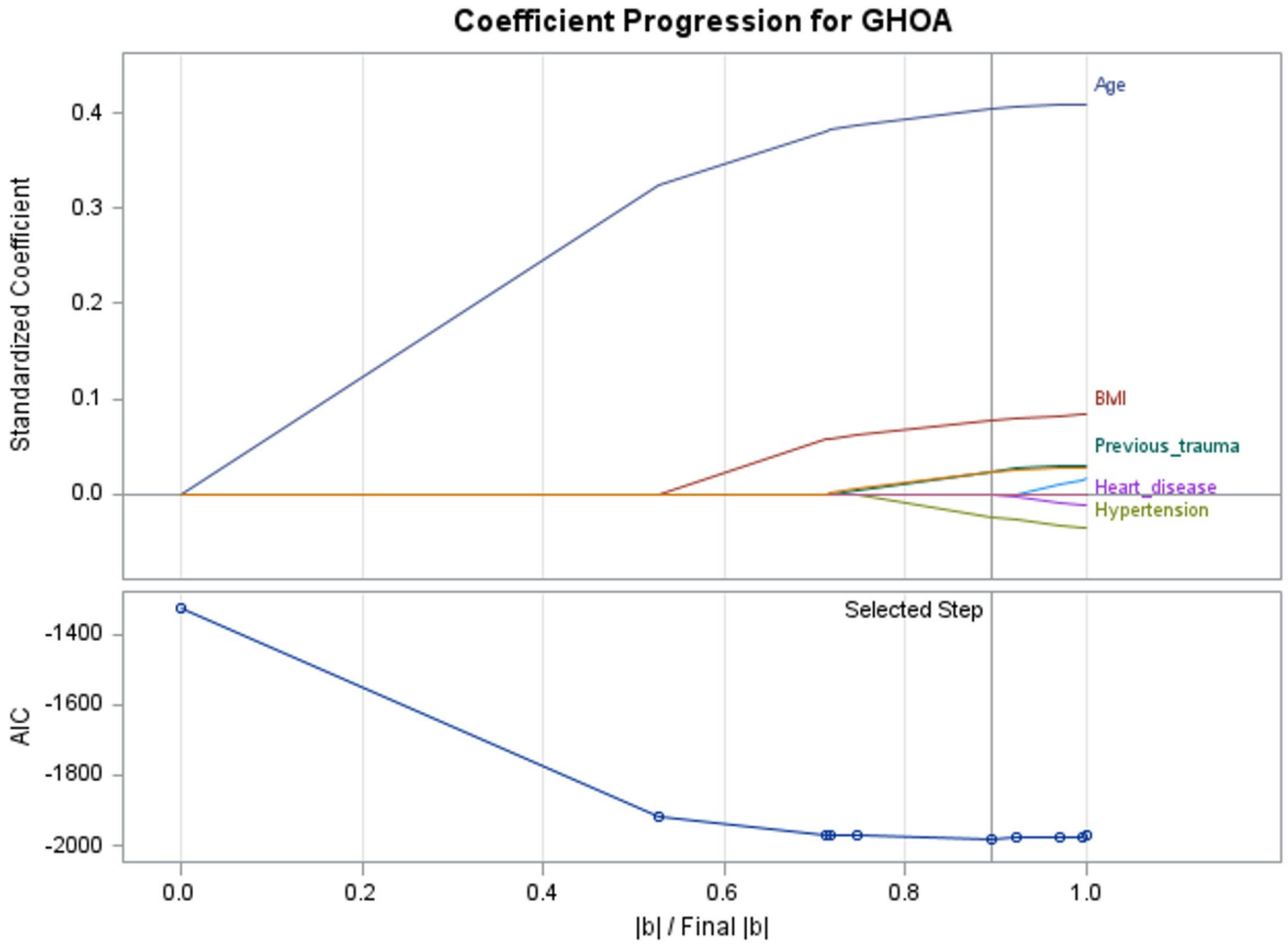

Figure 1 Least absolute shrinkage and selection operation (LASSO) model using Akaike information criteria (AIC) method to select the variables. In the above LASSO, optimal model was selected by indirect estimation using AIC method. GH, glenohumeral; OA, osteoarthritis.

In LASSO model, we observed that age, BMI, previous trauma, heart disease and hypertension were significantly associated with the diagnosis of GH OA (figure 1).

Multivariate analysis

In multivariate analysis, increasing age (p<0.0001), increasing BMI (p<0.0001) and a history of previous trauma (p=0.0034) were significantly associated with GH OA. An elevated risk of developing GH OA was noted across age groups: >40 to ≤50 years (OR 2.99; 95% CI 2.21 to 4.06), >50 to ≤60 years (OR 5.48; 95% CI 4.14 to 7.23), >60 to ≤70 years (OR 11.22; 95% CI 8.44 to 14.88) and over 70 years old (OR 16.65; 95% CI 12.45 to 22.17). Likewise, an increased risk of GH OA associated with higher BMI categories: >25 to ≤30 (OR 1.45; 95% CI 1.20 to 1.77), >30 to ≤35 (OR 1.70; 95% CI 1.39 to 2.09) and >35 (OR 1.78; 95% CI 1.25 to 2.55). Among comorbid conditions prior shoulder trauma did show a significant association. Other risk factors such as hypertension, shoulder pain at night and heart disease were significantly associated in univariate analysis but did not show a significant association in multivariate analysis (table 3).

Considering age as a confounding variable and incremental increases of BMI (a modifiable factor) as exposure to GH OA, the dose–response relationship analysis showed that the risk for GH OA increases along with the increase in age (0.01±0.00; p<0.0001) and BMI (0.006±0.00; p<0.0001).

DISCUSSION

Our study assessed risk factors associated with GH OA in a large cohort (1827 cases and 1556 controls) of patients with shoulder issues. Higher risk for GH OA was observed across increased age groups (>40 to ≤50 years: OR 2.99; >50 to ≤60 years: OR 5.48; >60 to ≤70 years: OR 11.22 and >70 years old OR 16.65) and higher BMI categories (>25 to ≤30: OR 1.45; >30 to ≤35: OR 1.70 and >35: OR 1.78).

Our findings from multivariate analysis showed that an increase in age is associated with an increased risk of diagnosis for GH OA. Kobayashi *et al* also observed an increasing risk of developing GH OA across different age groups. Specifically, individuals aged 60–69 years had a risk approximately 5.59 times higher, those aged 70–79

Table 3 Multivariable regression analysis of studied population in GH OA cases and controls

Variables		Estimate (SE)	OR	95% CI	P value‡
Age					<0.0001†
	<40*				
	>40 to ≤50	1.09 (0.15)	2.99	2.21 to 4.06	
	>50 to ≤60	1.70 (0.14)	5.48	4.14 to 7.23	
	>60 to ≤70	2.41 (0.14)	11.22	8.44 to 14.88	
	>70	2.81 (0.14)	16.65	12.45 to 22.17	
BMI					<0.0001†
	≤19	0.31 (0.19)	1.49	1.01 to 2.20	
	>19 to ≤25*				
	>25 to ≤30	0.37 (0.09)	1.45	1.20 to 1.77	
	>30 to ≤35	0.53 (0.10)	1.70	1.39 to 2.09	
	>35	0.58 (0.18)	1.78	1.25 to 2.55	
Sex					0.24
	Female*				
	Male	0.12 (0.07)	1.12	0.96 to 1.31	
Previous shoulder trauma					0.0034†
	No*				
	Yes	−0.22 (0.08)	0.80	0.68 to 0.94	
Hypertension					0.32
	No*				
	Yes	−0.05 (0.08)	0.95	0.80 to 1.12	
Shoulder pain at night					0.16
	No*				
	Yes	−0.17 (0.13)	0.83	0.64 to 1.09	
Heart disease					0.75
	No*				
	Yes	0.09 (0.12)	1.09	0.85 to 1.41	

*Reference group.
†Significant variable.
‡P value obtained from type 3 analysis.
BMI, body mass index; GH, glenohumeral; OA, osteoarthritis.

years had a risk of 11.59 times high and those >80 years old had a risk of 10.77 times higher when compared with younger age groups.[6] One of the studies in a Korean population also reported that risk for GH OA increases from age group of 70–74 years to >75 years old.[18] On the contrary, another study from South Korea reported that ageing was associated with spine, knee and hand OA but not with the OA of hip and shoulder.[19] A study from a US population on prevalence of GH OA in unstable shoulders reported that older age is associated with diagnosis of GH OA.[20] The likely mechanism for the association of increasing age with GH OA is that ageing of extracellular matrix leads to thinning of articular cartilage, reduces hydration and causes a build-up of proteins containing advanced glycation end-products (AGEs), making it more prone to develop OA.[21 22]

A higher BMI was associated with GH OA in our study. Our results are similar to prior studies in this area.[7–9 23] The prevalence of OA among normal/underweight individuals has been reported to be 16% and tends to increase to 23% in overweight people and 31% obese categories.[24] The likely reason for a higher BMI leading to GH OA is due to low grade inflammation as a result of obesity.[25] Obesity also leads to the expression of adipokines which affects cartilage, bone and synovium in the joints.[26] One of these adipokines called leptin is thought to increase the expression of enzymes (matrix metalloproteinases), nitric oxide, produce proinflammatory cytokines that breakdown joint tissue as well as other substances that cause inflammation.[27–29] This suggests that metabolic factors like obesity, hypertension, hypercholesterolaemia and high blood glucose might also play a role in GH OA by affecting these pathways in the body.[30–34] Since the GH joint is not a weight-bearing joint, these systemic and molecular mechanisms for the association of BMI with GH OA may direct to therapeutic pathways that can be targeted in future studies.

Our study found that prior shoulder trauma is negatively associated with GH OA. This association may be attributed to various secondary causes such as osteonecrosis,[35] fracture or dislocation,[3] septic arthritis,[36 37] glenoid dysplasia[38] and previous arthroscopic surgery. These secondary causes can contribute to develop GH OA possibly through mechanisms such as joint instability, altered biomechanics or an inflammatory process. An understanding of these associations will be crucial for both preventive measures and treatment strategies for individuals at higher risk of GH OA due to prior trauma.

Clinical implications

GH OA is a multifaceted condition involving pathological changes in the cartilage, subchondral bone and synovial fluid.[28 29] Its progression is highly variable making it challenging to predict disease trajectory. Known risk factors for development of degenerative GH OA include age, gender, obesity and genetic predisposition, though the relative impact of these factors on GH OA remains incompletely understood. The present study shows a significant association with increasing age and BMI with GH OA.

Ageing is thought to influence GH OA through alterations in the extracellular matrix, which results in thinning of articular cartilage, reduced hydration and accumulation of AGE products.[30 31] Additionally, while elevated BMI is a well-established risk factor for weight-bearing joints, it may also contribute to GH OA through systemic inflammation. Macrophages in the adipose tissue produce proinflammatory cytokines such as IL-6 and TNF-α which may exacerbate joint degradation.[36 37] Given the established relationship between age and BMI with GH OA, age may serve as a key factor for early diagnosis, while BMI represents a modifiable risk factor for managing the disease burden.

Overall, this study lays a foundational basis for future mechanistic research aimed at elucidating the causal pathways involved in GH OA. The data-driven from this larger cohort provide a critical stepping stone for developing preventive strategies and proactive diagnostic approaches.

Strength and limitations

The major strength of the present study is an analysis based on a large sample size that allowed for assessment of multiple factors with adequate statistical power. Limitations of our study include the lack of measurements of physical activity and clinical markers such as fasting blood glucose and lipid panels. Another limitation is that controls in our study were not individuals without GH OA in the general population, but those presenting with other shoulder issues. An ideal study would compare cases to population-based controls, but such a study design would be difficult from a feasibility standpoint given that these patients would need shoulder X-rays for confirmation of their case–control status. Third, we did not use CT or MRI images to classify cases and controls and even X-ray findings were not classified into Kellgren-Lawrence or Samilson-Prieto classification which could have benefit to perform more robust analysis.

CONCLUSION

It is important to identify factors associated with GH OA to help with disease prevention by creating public health initiatives that focus on educating the population about the link between obesity and joint health. By addressing specific risk factors, we can effectively reduce the overall incidence and burden of GH OA. In our cohort of 3383 patients, many potential risk factors were assessed. An increasing age and a higher BMI were significantly associated with GH OA. Given that the shoulder is not a weight-bearing joint, the mechanisms for the association of BMI with GH OA are likely molecular and systemic. This may direct towards potential therapeutic targets in the future.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval The present study was in compliant with the Declaration of Helsinki and was approved by the Institutional Review Board (IRB) at the University of Texas Southwestern Medical Center (IRB No.: STU 062016-012). A written informed consent was obtained from all patients at the time of enrolment.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement All data relevant to the study are included in the article or uploaded as supplementary information. The data supporting the results

are presented within the manuscript in the form of tables and figures. No specific datasets were generated during the study.

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