

Demographic and Clinical Risk Factors for Recurrent Instability after First-time Traumatic Anterior Shoulder Dislocation in Adults: A Narrative Review

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ABSTRACT

Recurrent instability following first-time traumatic anterior shoulder dislocation occurs in 19-88% of patients. It is a major clinical challenge that requires a personalised treatment decision, especially in young and active people. Younger age at first dislocation, particularly below 25 years; male sex; participation in overhead and contact sports; and generalised hypermobility independently increase recurrence rates. Key structural factors, including bony Bankart lesions, off-track Hill-Sachs lesions, and glenoid bone loss exceeding 20%, play a central role in destabilising the glenohumeral joint. The glenoid track concept provides a biomechanical basis to understand bipolar bone loss and engagement risk. Additional contributors include capsuloligamentous insufficiency and neuromuscular deficits. Current predictive models have shown modest discriminative ability, and most clinicians depend on readily identifiable risk factors to plan treatment. The correct identification of patients with numerous high-risk characteristics, especially young and active males with structural injuries, is crucial for individualised risk stratification to guide appropriate management decisions. This narrative review synthesises current evidence on demographic, biomechanical, and structural risk factors associated with recurrent anterior shoulder instability in adults to guide clinical management.

Keywords: Anterior shoulder dislocation; Bankart lesion; Joint instability; Recurrence; Risk factors.

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INTRODUCTION

Traumatic anterior shoulder dislocation is among the most prevalent types of dislocations of joints in clinical practice, with an incidence of 1-2% in the general population and 95% of all dislocations of the shoulder joint [1]. The anatomical arrangement of the glenohumeral joint ensures that mobility is maximised at the expense of stability, making it particularly susceptible to dislocation following traumatic injury [2]. Even though many people get better after their first dislocation, recurrent instability is still a considerable problem in clinical practice that can cause long-term pain, limited mobility, and joint damage

that gets worse over time [3].

The reported recurrence rates after first-time anterior traumatic shoulder dislocation show a lot of variation between studies, ranging from 19% to 88%. This variation shows how studies differ in terms of patient age, sex, activity level, associated bony and ligament lesions, occupation, and management strategy [4]. There is still considerable debate about the management of first-time anterior shoulder dislocation, particularly regarding whether to use surgery. Non-operative management has long been the standard of care, but high recurrence rates, especially in young, active patients, have led to growing support for early surgical stabilisation [5]. It is vital for clinicians to identify which patients are most likely to have a recurrence because it will affect whether conservative management with rehabilitation is the best option or whether surgical stabilisation should be considered after the first episode of dislocation [6].

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The goal of this narrative review was to address the following clinical question: In adults presenting with first-time traumatic anterior shoulder dislocation, which demographic, biomechanical, and structural risk factors are associated with an increased risk of recurrent instability? This review will be used to offer clinicians an evidence-based approach for individualised risk stratification to guide management decisions.

METHODS

This narrative review was generated from a non-systematic literature search conducted on PubMed, Scopus, and Google Scholar databases conducted from October 2025 to February 2026 (search execution dates). No lower publication date limit was applied; however, priority was given to studies published between 2015 and 2025 to ensure contemporary applicability; seminal earlier works were retained if they were historically significant. The main search keywords used were anterior shoulder dislocation, recurrent instability, Bankart lesion, Hill-Sachs lesion, bone loss of the glenoid, glenoid track, and predictive models. The search found 198 articles in all three databases. We chose 65 articles for the final narrative synthesis after getting rid of duplicates and making sure that the titles and abstracts were relevant. Systematic reviews, meta-analyses, landmark cohort studies, large retrospective studies, multivariate analyses, and important biomechanical and clinical articles were given the priority. Studies were selected based on relevance to adult patients with first-time traumatic anterior shoulder dislocation, were peer-reviewed, had clear methodology and patient descriptions, and were published in English and were prioritised. Articles focusing on atraumatic or multidirectional instability and paediatric populations were excluded. Due to the narrative design, formal systematic screening or quantitative quality assessment was not performed; however, the reporting quality of this review was assessed against all six SANRA domains, including justification of importance, PICO-structured aims, literature search strategy, appropriate referencing, scientific reasoning, and appropriate presentation of data.

The synthesis of the evidence was conducted in a descriptive manner and categorised thematically, including demographic, biomechanical, structural, and predictive models, as well as risk stratification tools. All sections summarised the evidence, reviewed the underlying mechanisms, and speculated on the clinical implications for patient counselling and treatment choice.

DEMOGRAPHIC RISK FACTORS

Age at first dislocation

Age at first shoulder dislocation is a highly significant and reliably confirmed predictor of recurrent instability. An inverse relationship between age and recurrence risk has been demonstrated across multiple populations and treatment strategies, with younger patients facing substantially higher rates of recurrence [7, 8].

Following non-operative treatment, Lill et al. identified recurrence rates of 89% in patients aged ≤ 30 years, in contrast to 26% in those older than 30 years [9]. Other series similarly demonstrate markedly higher recurrence in patients aged ≤ 40 years (approximately 44%) versus those older than 40 years (approximately 11%) [8]. Patients experiencing their first dislocation prior to the age of 20 show recurrence rates of 66-94%, but those older than 40 years typically demonstrate rates below 15-20% [1, 10, 11].

In a landmark study, the prognosis of 257 patients who suffered from their first episode of shoulder dislocation was monitored after 25 years, during which the worst prognostic factor for the patients was found to be aged under 25 years, of whom half eventually needed surgical stabilisation. This finding is supported by numerous systematic reviews that found the best predictor of recurrent shoulder instability is age under 20-25 years. Thus, clinicians need to take this age threshold into account when deciding between non-operative rehabilitation and early surgical intervention [12, 13].

The high recurrence rate of shoulder instability in younger patients can be explained by the combination of behavioural and biological factors. Frequent participation in high-impact sports puts a lot of mechanical stress on the joints, and intrinsic ligamentous laxity and immature muscular stabilisation further undermine the integrity of the joints [8, 9]. Unlike older patients, who benefit from "protective" fibrotic scarring and capsular thickening that limit translation, younger individuals often heal with redundant, elastic tissue that fails to provide sufficient static stability [8, 14].

Sex

Male sex is now recognised as an independent risk factor for recurrent instability in multiple studies, though the magnitude of this effect is generally less pronounced than that of age. Relevant epidemiological research constantly shows that in any population and irrespective of age, recurrence of anterior shoulder dislocation is two to four times higher in males compared to females [7, 13, 15].

The sex-based difference may relate to several factors. Anatomically, males typically demonstrate different glenoid morphology, which may suggest that sex-specific anatomy may differentially influence instability risk between males and females [16]. In addition, males demonstrate a higher likelihood of participating in high-impact and contact sports, as well as an increased incidence of associated traumatic lesions, such as labral tears and glenoid bone loss, both of which are well-established contributors to recurrent instability [17]. Sex-related differences in muscle mass distribution, neuromuscular control, and shoulder girdle biomechanics may further influence the dynamic stabilising mechanisms of the glenohumeral joint [18]. Notably, even after adjustment for activity level and injury severity, male sex has remained an independent predictive value for recurrence in several analyses, suggesting that biomechanical or biological factors beyond exposure alone may contribute to the increased risk [7].

Activity level and occupation

High levels of physical exercise, especially when engaging in contact or collision sports, are associated with an increased risk of recurrent shoulder instability that can be attributed to repeated exposure to high-risk injury mechanisms [7]. American football, rugby, ice hockey, and martial arts are all contact sports that can cause direct injury to the shoulders and put them in vulnerable positions. Similarly, athletes involved in overhead sports like volleyball, swimming, baseball, and tennis are more likely to experience recurrent shoulder injuries. This happens due to the constant strain on the anterior capsulolabral structures in positions of external rotation and abduction that destabilize the anterior shoulder [19, 20]. Another factor that determines the risk of recurrence is occupational demands. People who perform heavy manual labour and military personnel are exposed to significant repetitive

and traumatic shoulder loads, placing them at increased risk of recurrent instability [21, 22]. It is necessary to acknowledge that such a relationship has a component of bidirectionality, where increased activity levels automatically increase the probability of encountering the specific forces that lead to re-dislocation.

At the same time, the desire to return to sport before the injured tissue has fully healed or regained the muscle control it needs increases the risk of re-dislocation even more [23]. In addition, the risk factors for recurrent instability include young age, participation in contact or overhead sports, and having a structural injury. These factors will influence decisions about when to return to play. Each case needs to be evaluated individually for the best balance between tissue healing, functional recovery, and the physical demands of the specific sport or job [19, 23]. These factors show the importance of structured risk assessment to ensure safe and timely return to activity for those individuals who have their first anterior shoulder dislocation.

BIOMECHANICAL RISK FACTORS

Hypermobility and ligamentous laxity

Generalised joint hypermobility, which is assessed through a standardised tool called the Beighton score, with scores of ≥ 4 indicating hypermobility, is associated with increased recurrence risk, with pooled estimates indicating nearly three-fold increased odds after a first dislocation [24]. The increased risk can be explained by the excess capsular redundancy and worsening of the stability of the joints in the stationary position [8, 24].

Hypermobility in recurrent shoulder dislocation has been associated with poorer outcomes following non-operative management and a higher likelihood of failure after soft-tissue stabilisation procedures in the absence of bony or capsular augmentation [25, 26].

Neuromuscular factors

Neuromuscular factors are critical determinants of the risk of recurrence following shoulder dislocation [27]. Coordinated activation of the rotator cuff and periscapular musculature is important to achieve dynamic shoulder stability [28]. Deficits in rotator cuff strength and scapular control, as well as proprioception, after shoulder dislocation can compromise the dynamic stability of the shoulder [28].

The loss of proprioception reduces the ability of the shoulder to sense unstable postures before soft-tissue failure, and this elevates the chances of recurrent instability [29]. Research that focuses on addressing both recurrent instability and neuromuscular exercises has shown that the presence of neuromuscular deficits is associated with a higher incidence of recurrence and functional disability [30]. These clinical findings focus on the need to ensure that rehabilitation is effective in addressing rotator cuff strengthening and scapular stabilisation, as well as proprioceptive training to minimise the risk of further exacerbation [31].

STRUCTURAL RISK FACTORS

Bankart lesions

The Bankart lesion, distinct as an avulsion of the anterior-glenoid labrum from the glenoid rim, is the most common structural injury associated with anterior shoulder dislocation and is an integral part of the so-called "essential lesion" in

the context of an anteriorly dislocated shoulder [32]. Imaging modalities and arthroscopic studies have consistently shown that this lesion occurs in approximately 85-90% of people with their first traumatic dislocation, and the incidence reaches 95-100% in patients presenting with recurrent instability [33]. These data, therefore, highlight the gradual degeneration of the labral complex with every recurring dislocation episode, hence the clinical significance of addressing this pathology in the overall management of shoulder instability [34].

It creates a pathological condition that undermines the static stability of the joint by disrupting the inferior glenohumeral ligament complex attached to the glenoid labrum, which acts as the main constraint to anterior translation in the abducted and externally rotated posture. This will compromise the labral bumper effect and ligamentous restriction of anterior humeral head displacement, which creates a mechanical circumstance causing recurrent instability [35].

With the biomechanical implications of the lesion, the spontaneous anatomical healing ability of the Bankart lesion was limited, a finding that can be explained by multiple pathophysiological mechanisms. The presence of synovial fluid in the intersection of the labrum and glenoid prevents the development of fibrinous haemostatic clots, and the poor vascularity of the area is intrinsically restrictive to the regenerative capacity of the tissue [36].

Furthermore, traditional sling immobilisation often fails to maintain adequate reduction of the detached labral portion on the glenoid rim, and the early onset of shoulder mobilisation endangers fragile reparative tissues before the formation of a stable reattachment. All these biological and mechanical factors explain the great likelihood of non-anatomic healing because of conservative treatment approaches [37]. Makaram and colleagues demonstrated, using multivariate analysis, that bony Bankart lesions, which are fractures of the anterior glenoid rim with labral avulsion, are an independent predictor of recurrent instability [7].

The combination of soft tissue and osseous injury leads to a more severe compromise of anterior stability, and it makes this combination less amenable to healing with conservative management [38]. Arthroscopic studies have also supported the fact that Bankart lesions rarely heal in an anatomic position following conservative management, often resulting in the medialisation or malposition of the labral healing. This explains the high rates of recurrence observed with non-operative management and supports early surgical intervention in appropriate patient populations [39].

Hill-Sachs lesions

Hill-Sachs lesions are described as posterolateral compression fractures of the humeral head that occur when the humeral head hits against the anterior glenoid rim during anterior shoulder dislocations. These lesions are found in 40-90% first-time anterior dislocations and up to 100% in cases of recurrent instability [40]. Hill-Sachs lesions have also surfaced as an important predictor of recurrence risks, with the size, location, and orientation of the Hill-Sachs lesions being critical in determining the recurrence risks [41].

The effect of this lesion can be evaluated by the "glenoid track" concept, which refers to the "functional area of contact" between the humeral head and glenoid during shoulder motion and classifies Hill-Sachs lesions into two types: on-track and off-track [42]. This basis has been supported by large retrospective cohort studies, including that

of Buyukkuscu et al., which identified Hill-Sachs lesions as an independent risk factor for recurrent shoulder dislocation [11].

In the on-track position, the Hill-Sachs lesion stays lateral to the glenoid track during shoulder motion. Therefore, it will never engage with the anterior glenoid rim. While in the off-track position, the Hill-Sachs defect is medial enough to engage the glenoid edge during abduction/external rotation, forming bone-on-bone contact that increases instability recurrence despite soft tissue repair (Figure 1) [43].

Track status is determined via 3D computerized tomography (CT) scan or magnetic resonance imaging (MRI) using the idea of glenoid track (GT) width, which is estimated via the subsequent formula: $GT = 0.83D - d$, where D is the diameter of the inferior glenoid and d is the glenoid bone defect width [45]. The Hill-Sachs interval (HSI) is measured from the rotator cuff attachment to the medial edge of the Hill-Sachs lesion. When HSI exceeds GT, the lesion is off-track and poses a high risk for engagement and recurrent instability [45]. Categorisation of Hill-Sachs Lesions Based on the concept of GT width is summarised in Table 1.

Treatment for Hill-Sachs lesions differs according to their size and engagement risk. Small, on-track lesions need only Bankart repair, while large or off-track lesions may require remplissage (filling the defect with infraspinatus tendon/capsule) and, in severe cases, need humeral bone grafting [46, 47].

GLENOID BONE LOSS

The glenoid offers the concave surface, which is essential in glenohumeral joint stability. Recurrent subluxations can cause anterior-inferior glenoid fractures or chronic erosion that reduces this critical bony architecture [48]. Recently, it was a standard practice to quantify glenoid bone loss. A variety of measurement methods have been outlined, with the best-fit circle and Pico methods being the two validated methods using CT scan [49]. And it is usually presented as a proportion of the inferior glenoid’s width, with bone loss beyond 20-25% of the glenoid width representing a critical threshold beyond which soft tissue repair alone shows high failure rates, resulting in a need for bone grafting procedures [50]. Subcritical bone loss (10-20%) increases the risk of recurrence and, in such cases, will warrant surgical consideration in patients presenting with additional risk factors [51].

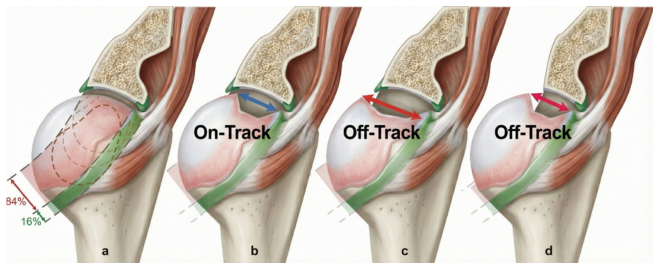


Figure 1. Schematic representation of the glenoid track concept and the relationship between the Hill-Sachs interval and glenoid track width. Adapted and modified from Tuite MJ & Pfirrmann CWA, in *Musculoskeletal Diseases 2021–2024: Diagnostic Imaging* (Hodler J et al., eds), Springer, 2021, Chapter 1. © 2021 The Author(s); CC BY 4.0. [44].

Table 1. Categorisation of Hill-Sachs lesions based on the glenoid track width concept*.

Condition	Result	Clinical implication
HSI < GT	On track	The Hill-Sachs lesion remains confined to the glenoid track. A standard Bankart repair is usually sufficient.
HSI > GT	Off-track	The lesion is too wide/medial and will "fall off" the glenoid rim. A high potential failure with just soft tissue reconstruction.

* Abbreviations: Glenoid track (GT) width, Hill-Sachs interval (HSI).

Recurrent dislocation episodes cause progressive erosion of the anterior glenoid rim, which eventually develops the classic "inverted pear" glenoid deformity, which severely compromises anterior stability [52]. Glenoid bone loss combined with Hill-Sachs' lesions places bipolar bone loss together, hence synergistically increasing instability [53]. Three-dimensional CT reconstruction and MRI have now become standard tools to assess both sides of the joint in preoperative planning to accurately assess the bone loss for precise measurement and guidance in surgical decision-making [54].

CAPSULAR AND LIGAMENTOUS INJURIES

Capsular and ligamentous injuries are the main contributing factors of recurrent anterior shoulder instability following first-time dislocation [55]. The inferior glenohumeral ligament (IGHL) complex, which is the major static stabiliser in abduction-external rotation, is often torn or attenuated in traumatic anterior dislocation; thereby, residual laxity in the capsule is acquired with the loss of resistance to anterior translation [1].

Specific injury patterns, involving humeral avulsion of the glenohumeral ligament (HAGL) lesions and the stripping of the capsule, further compromise static restraint and are often underdiagnosed on routine imaging. Poor healing of such structures after conservative management puts the shoulder at risk of recurrent instability, especially in high-demand and [56]. These variants can be detected with modern imaging, especially MRI arthrography, but the arthroscopic examination is the gold standard for definitive characterisation. Failure to recognise and address capsuloligamentous insufficiency is considered a significant cause of persistence and recurrent anterior shoulder instability [57]. The other labral injuries, including anterior labroligamentous periosteal sleeve avulsion (ALPSA) lesions, characterised by medialised non-anatomic healing of the labroligamentous complex, carry higher recurrence rates than classic Bankart lesions, while the glenolabral articular disruption (GLAD) lesion, involving superficial labral tearing with chondral injury, is less likely to be a cause of recurrent instability but is associated with persistent post-reduction pain [55, 58].

GREATER TUBEROSITY FRACTURES AS PROTECTIVE FACTORS

Greater tuberosity fractures often occur concomitantly with anterior shoulder dislocations for the first time, par-

ticularly in older adults, and these fractures may act as a protective factor against recurrent instability [7]. Observed data indicate that the presence of a greater tuberosity fracture reduces recurrent instability by more than seven times [8].

The reduced risk of recurrence is likely due to several interconnected mechanisms. First, during fracture dislocations, it has different biomechanics than pure dislocations, as traumatic energy dissipates through the bone rather than the soft tissue, resulting in less severe capsulolabral damage [59]. Second, following fracture healing, fibrotic tethering may restrict terminal abduction and external rotation, the two most common provocative postures in anterior dislocation [59].

PREDICTIVE MODELS AND RISK STRATIFICATION TOOLS

Since the aetiology of recurrent anterior shoulder instability is multifactorial, various predictive models and risk-stratification tools have been developed to integrate demographic, biomechanical, and structural risk factors, thereby enabling clinical decision-making [60]. The main objective of these tools is to distinguish the persons at increased likelihood of recurrence that might possibly undergo early operative intervention, whereas those who are low risk can proceed with conservative treatment safely [60].

PREDICTING RECURRENT INSTABILITY OF THE SHOULDER (PRIS)

The concept of the PRIS tool was developed as a multivariate logistic regression model, which predicts who will not have further shoulder instability in twelve months after suffering an initial traumatic anterior shoulder dislocation. PRIS is a probability-based risk estimate and not a categorical score like the additive, point-based system of the Instability Severity Index Score [60].

PRIS has integrated a multitude of variables, including age (16-40 years), dominant-arm involvement, immobilisation, bony Bankart lesion presence, Shoulder Pain and Disability Index (SPADI) score, and Tampa Scale of Kinesiophobia (TSK-11) scores, to produce a continuous probability estimate, which is calculated using the following formula: Risk of recurrence = $-4.73 + 1.06 \times (\text{age } 16\text{-}25 \text{ years}) + 1.80 \times (\text{bony Bankart lesion}) + 0.80 \times (\text{impacted dominant side}) - 1.27 \times (\text{immobilised}) + 0.03 \times (\text{SPADI total score}) + 0.13 \times (\text{TSK-11 total score})$ as illustrated in Table 2 [60].

In external validation, a cut-off probability of approximately 0.89 demonstrated high specificity (95%) for ruling out recurrence. Scores lower than this value indicate a low probability of recurrent instability, and scores higher than this value indicate an increased estimated risk of recurrence. Despite this utility, the overall discriminative performance of the model remains modest, with an area under the curve of 0.69, limiting its capability to accurately distinguish between patients who will and will not experience recurrent instability [60].

INSTABILITY SEVERITY INDEX SCORE (ISIS)

This score was developed to predict failure after arthroscopic Bankart repair rather than recurrence following traumatic dislocation for the first time. This score is composed of six preoperative factors, including age, type of sport, hyperlaxity, Hill-Sachs lesion, glenoid bone loss, and loss of external rotation. A score of 6 or higher is often used to suggest open

Table 2. Variables of the Predicting Recurrent Instability of the Shoulder (PRIS) tool*.

Variable	Scoring/Description
Age	16-40 years (16-25 years = 1 point; 26-40 years = 0 points)
Bony Bankart lesion	Yes = 1; No = 0 (proved by X-ray)
Affected dominant side	Yes = 1; No = 0
Immobilisation after initial FTASD	Yes = 1; No = 0
Shoulder pain and disability index total score	0-100% (0% = no shoulder pain or disability)
Tampa scale of Kinesiophobia-11 total score	11-44 (a higher score indicates greater kinesiophobia)

* Abbreviations: FTASD: First-time traumatic anterior shoulder dislocation.

or bone-block procedures (Table 3) [61]. Although the individual elements of the ISIS score are associated with the risk of recurrence, applying the score to a patient with their first dislocation represents an extrapolation that has not been rigorously investigated for this use. Studies assessing the predictive performance of ISIS for recurrent shoulder instability report area under the curve (AUC) values ranging from 0.526 to 0.792, thus showing variable performance and creating significant debate about its clinical usefulness [62, 63].

MACHINE LEARNING AND ALTERNATIVE APPROACHES

Lu et al. developed machine learning models to estimate recurrence, advancement to surgical intervention, and the occurrence of arthritis after first-time anterior dislocation [64].

Table 3. Instability severity index score (ISIS) components and scoring.

Variable	Parameter	Score
Age	< 20 years	2
	≥ 20 years	0
Sports participation level	Competitive	2
	Recreational/none	0
Sport category	Contact or forced overhead	1
	Other sports	0
Shoulder hyperlaxity	Anterior and/or inferior hyperlaxity	1
	Normal	0
Hill-Sachs lesion(AP X-ray)	Visible on external rotation	2
	Not visible on external rotation	0
Glenoid contour(AP X-ray)	Loss of the inferior glenoid contour	2
	No lesion	0
Total	-	10

Table 4. Predictive models for recurrent instability*.

Model	Type	Variables included	AUC	Key threshold	Limitations
Predicting Recurrent Instability of the Shoulder	Probability-based logistic regression	Age (16–25 years), dominant arm, immobilisation, bony Bankart, SPADI score, and TSK-11 score	0.69	>0.89 = high risk <0.89 = low risk	Modest discriminative ability
Machine learning	AI algorithms	Age, activity level, imaging data, multiple variables	Variable	N/A	Not widely validated
Makaram model	Multivariate regression	Male sex, younger age, contact sports, bony Bankart	0.672	N/A	Modest discriminative ability

* Abbreviations: AI: Artificial intelligence; AUC: Area under the curve; SPADI: Shoulder pain and disability index; TSK-11: Tampa scale of Kinesiophobia-11.

Table 5. Risk Stratification after primary anterior shoulder dislocation.

Variable	High-risk profile (Consider early surgery)	Low-risk profile (Consider conservative.)
Age	< 25 years	> 40 years
Sex	Male	Female
Sport/Job	Contact, collision, overhead	Sedentary, non-contact
Bone loss	Bony Bankart or off-track Hill-Sachs	None or greater tuberosity fracture
Laxity	Generalised hypermobility	Normal

The models use various factors, such as age, activity level, and imaging data, to produce personalised risk predictions. Unlike traditional regression-based tools, machine learning approaches can account for nonlinear relationships and offer the potential to identify complex interactions between risk factors [64].

External validation must be made before these models can be used in routine clinical decision-making. Similarly, Makaram et al. applied a multivariate regression model in 1,293 patients who had primary traumatic anterior dislocation of the shoulder to create a clinical prediction model [7, 62]. The model identified male sex, younger age, participation in contact sports, and bony Bankart lesions as independent predictors of recurrence. The model, however, had moderate ability to discriminate between outcomes (AUC 0.672), highlighting the ongoing challenges in accurately predicting individual patient outcomes [7]. The predictive models for recurrent instability are summarised in Table 4.

CLINICAL ADOPTION AND PRACTICAL APPLICATION

Despite the emergence of advanced predictive tools, their use in clinical practices is limited. Most clinicians still use individual risk factors, especially age, activity level, and structural injuries that can be detected on standard imaging, rather than including complex prediction models [7]. Although it is not as accurate as the formal predictive models, it provides simple methods that can be used in a busy clinical environment where complex risk calculations take time and may not be practical.

PRACTICAL RISK STRATIFICATION

The idea of risk stratification based on easily identifiable factors remains clinically useful. High-risk profiles typically include young age (particularly <25 years), male sex, involve-

ment in contact or overhead sports, and the existence of significant structural injuries (bony Bankart, large Hill-Sachs, and glenoid bone loss) [22]. Patients meeting multiple high-risk criteria have recurrence rates exceeding 80-90% with conservative management and represent clear candidates for early surgical stabilisation [22, 65].

Conversely, low-risk profiles include older age (>40 years), lower activity demands, female sex, and the presence of protective factors such as the greater tuberosity fracture [22]. These patients have lower recurrence rates with conservative management, making initial non-operative treatment reasonable, according to the risk stratification framework (Table 5).

LIMITATIONS

The current narrative review is subject to various natural limitations. Specifically, the use of non-systematic search strategies could lead to the exclusion of relevant studies, and the lack of formal quality assessment or risk-of-bias evaluation could be a source of selection bias. The heterogeneity inherent in the included studies, in terms of patient groups, treatment strategies, imaging modalities, and follow-ups, makes it difficult to compare directly and makes quantitative synthesis impossible. Some of the studies considered using retrospective designs, which are prone to bias. Current predictive models demonstrate only modest discriminative ability and limited external validation, thus limiting their generalisability. Finally, patient-specific factors such as compliance with rehabilitation, psychological conditions, and socioeconomic factors are not well represented in the existing literature, but they are likely to effect on recurrence risk.

CONCLUSION

The recurrent instability following a first-time traumatic anterior shoulder dislocation is explained by several factors that interact in a complex manner. The most significant pre-

dictor is age. Additional independently increased risks are male sex, contact sports participation, hypermobility, bony Bankart lesions, and critical glenoid bone loss. The concept of the glenoid track has identified high-risk bipolar bone loss, where an off-track Hill-Sachs lesion significantly increases the recurrence rate despite soft-tissue repair. Modern predictive models have modest discriminative ability, and most clinicians are thus guided by easily observable high-risk features in making treatment plans. An in-depth understanding of these risk factors enables the development of personalised, evidence-based therapeutic choices, thus, maximises outcomes and reduces the risk of cumulative joint damage, resulting from repeated instability. Further research is required to improve predictive models, determine the best surgical timing, investigate biologic enhancement strategies, and establish long-term relationships between treatment strategies and outcomes.

ETHICAL DECLARATIONS

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None.

Ethics Approval and Consent to Participate

Not applicable.

Consent for Publication

Not applicable.

Availability of Data and Material

All data supporting the findings of this study are derived from previously published articles, which are cited within the manuscript. No new datasets were generated or analyzed during the current study.

Competing Interests

The authors declare that there is no conflict of interest.

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Authors' Contributions

Kadhim AS: Conceptualization; Methodology; Literature Search; Data Curation; Software; Visualization; Resources; Project Administration; Writing – Original Draft; Writing – Review and Editing; Supervision. Saadoon RA: Literature Search; Data Curation; Investigation; Writing – Original Draft; Writing – Review and Editing. Alardi IM: Literature Search; Data Curation; Supervision; Validation; Writing – Review and Editing. All authors read and approved the final version of the manuscript.

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