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ORIGINAL ARTICLE

Biopsychosocial Approach in Identifying Risk Factors of Kinesiophobia in Persons with Subacromial Pain Syndrome and Developing a Clinical Prediction Tool

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Abstract

Introduction Although the negative effects of kinesiophobia on functional status in subacromial pain syndrome (SAPS) patients are clearly demonstrated, no study examines the risk factors of kinesiophobia in individuals with SAPS from a biopsychosocial perspective. The present study aims to determine the risk factors of kinesiophobia in individuals with SAPS using a biopsychosocial approach. This study also aims to explore the compounding effects of multiple associative risk factors by developing a clinical prediction tool to identify SAPS patients at higher risk for kinesiophobia.

Materials and methods This cross-sectional study included 549 patients who were diagnosed with SAPS. The Tampa-Scale of Kinesiophobia (TSK) was used to assess kinesiophobia. Visual analog scale (VAS), The Shoulder Pain and Disability Index (SPADI), Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, the presence of metabolic syndrome, using any non-steroidal anti-inflammatory drugs, Pain Catastrophizing Scale (PCS), Illness Perception Questionnaire-revised (IPQ-R), Hospital Anxiety and Depression Scale (HADS), behavioral pattern of the patient, sociodemographic characteristics, and treatment expectancy were outcome measures.

Results Thirteen significant risk factors of having kinesiophobia were: $VAS_{at\ rest}$ (≥ 5.2), $VAS_{during\ activity}$ (≥ 7.1), DASH (≥ 72.1), presence of metabolic syndrome, $PCS_{helplessness}$ (≥ 16.1), IPQ-$R_{personal\ control}$ (≤ 17.1), IPQ-$R_{treatment\ control}$ (≤ 16.3), $HADS_{depression}$ (≥ 7.9), avoidance behavior type, being female, educational level ($\leq$ high school), average hours of sleep (≤ 6.8), and treatment expectancy (≤ 6.6). The presence of seven or more risk factors increased the probability of having high level of kinesiophobia from 34.3 to 51%.

Conclusions It seems necessary to address these factors, increase awareness of health practitioners and individuals.

Level of evidence Level IV.

Keywords Subacromial pain syndrome · Shoulder · Painful shoulder · Biopsychosocial models

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Introduction

Shoulder pain is an important musculoskeletal problem that can lead to several medical and socioeconomic issues [1]. Subacromial pain syndrome (SAPS) is a general term for mostly unilateral and non-traumatic shoulder pain, in which arm elevation aggravates symptoms and cause localized pain around the acromion [2, 3]. Problems such as subacromial bursitis, biceps tendinitis, calcific tendinitis, supraspinatus tendinopathy, partial rotator cuff tears are referred as SAPS [2, 3]. Altered movement patterns of the scapula (e.g., due to bone anomalies, decreased scapular external rotation, decreased or increased scapular upward rotation, and decreased scapular posterior tilt), decreased EMG activities of the serratus anterior, middle and lower trapezius muscles, increased EMG activities of the upper trapezius and levator scapulae muscles, shortness of the pectoralis minor muscle, and tension of the posterior capsule of glenohumeral joint are common causes of SAPS [2–4].

Despite many treatment methods such as non-steroidal anti-inflammatory drugs, exercise therapy, subacromial injections, and manipulative techniques, symptoms are present during the first 12 months in almost 60% of individuals diagnosed with SAPS [1–4]. According to Martinez-Calderon et al., other than biological and biomechanical factors, psychosocial factors also have a role in this clinical presentation [5]. From a cognitive-behavioral perspective, individuals with SAPS develop kinesiophobia in response to pain and have greater difficulty in inhibiting pain and facilitating movement [5]. Feleus et al. stated that individuals with SAPS with high kinesiophobia could not gain a desirable functional level in a 12-month period [6]. Likewise, in individuals who develop fear of movement and re-injury due to the severity of pain in the early period, chronic pain becomes more persistent in the 6-month period [5, 6].

Although the negative effects of kinesiophobia on functional status in SAPS patients are clearly demonstrated, no study in the literature examines the risk factors of kinesiophobia in individuals with SAPS from a biopsychosocial perspective. An in-depth evaluation of risk factors can assist clinicians in planning strategies to reduce the risk of kinesiophobia. Thus, the present study aims to determine the risk factors of kinesiophobia in individuals with SAPS using a biopsychosocial approach. This study also aims to explore the compounding effects of multiple associative risk factors by developing a clinical prediction tool to identify SAPS patients at higher risk for kinesiophobia.

Methods

Study Design and Participants

This cross-sectional study included patients who were diagnosed with SAPS by an orthopedic surgeon (ÇB) in the Orthopedics and Traumatology Outpatient Clinic between May 26, 2021, and March 07, 2022. A total of 549 individuals with SAPS were evaluated by an experienced physiotherapist (CK) for the variables within the scope of the study. The study was approved by local ethics committee (2021/812). Written and verbal consents were obtained from all participants and the study was conducted in accordance with the Declaration of Helsinki.

The diagnosis of SAPS was based on medical history, physical examination, and magnetic resonance imaging (MRI) of the shoulder. Eligible patients were included if they met the following criteria: at least 3 months of pain in the deltoid region; inability to lie on the affected shoulder; pain during abduction, backward flexion or internal rotation; positive Neer or Hawkins impingement test; positive lidocaine impingement test; presence of acromioclavicular (AC) joint osteoarthritis, subacromial-subdeltoid (SASD) bursitis, tendinosis in one or more tendons of the rotator cuff, partial tear in one or more tendons of the rotator cuff, and calcification in one of more tendons of the rotator cuff [7–9]. The exclusion criteria were: full thickness tear of the rotator cuff, infection, labral tear, signs of glenohumeral instability, passive restriction of glenohumeral motion, osteoarthritis of the glenohumeral joint, rheumatic diseases, cervical radiculopathy, history of shoulder trauma, synovitis, prior surgery in the affected shoulder, injection of cortisone in the affected shoulder in the last 6 weeks, and inability in filling out the questionnaires [7–9].

MRI Protocol for SAPS Diagnosis

An experienced orthopedic surgeon, ÇB, who was blinded to study protocol, evaluated the MRIs. The following MRI findings were examined for the diagnosis of SAPS [10]: Acromion type (I, II, III, IV), AC-joint osteoarthritis (yes/no), SASD bursitis (yes/no), tendinosis in one or more tendons of the rotator cuff (yes/no), partial tear in one or more tendons of the rotator cuff (yes/no), and calcification in one of more tendons of the rotator cuff (yes/no).

Acromion morphology: Sagittal oblique MR images were used to assess acromion shape and four morphologies were identified: Type I, flat acromion; Type II, curved acromion; Type III, hooked acromion; and in Type IV, the acromion had a curved inferior contour [12].

AC-joint osteoarthritis: Presence of joint effusion, capsular distention, joint space narrowing, marginal osteophyte, bone marrow edema or periarticular sclerosis, and subchondral cyst(s) was defined as degenerative changes of the AC joint [11].

SASD bursitis: Widening or thickening or of the bursa was defined as SASD bursitis [13].

RC tendinosis: An intermediate signal on T2-weighted images or an increased signal on low TE images (e.g., STIR, PDFS, or T2 with fat suppression) was considered as tendinosis [13].

RC partial tears: Presence of a focal liquid signal through the tendon in the absence of total extension from the articular to bursal surface was defined as an RC partial tear. Partial tears were categorized into subtypes as delamination tears or focal tears confined within tendon footprints, and as bursal-sided, articular-sided, or intrasubstance tears [13].

Calcific tendinosis: Calcium disposition around or within the rotator cuff tendons, represented as low signal density on all sequences together with edema within the tendon (and less frequently within the subjacent bone) was defined as calcified tendon [13].

Outcome Measures

The Tampa-Scale of Kinesiophobia (TSK) was used to assess kinesiophobia [14]. This 17-item scale measures the patients' beliefs about (1) underlying and major medical problems (somatic focus), and (2) (re)injury or aggravated pain (activity avoidance). The retest reliability of the TSK is moderate to good, construct, concurrent, and predictive validity is moderate, and internal consistency is good [15]. Higher scores on the scale (above 37) represent the presence of fear of movement [14, 15]. Based on their TSK scores, we divided the participants into two groups: those with high level of kinesiophobia (TSK > 37) and those with low level of kinesiophobia (TSK ≤ 37).

Suggested by Wijma et al., the PSCEBSM model (Pain–Somatic and medical factors–Cognitive factors–Emotional factors–Behavioral factors–Social factors–Motivation) was used for the biopsychosocial assessment of the participants [16]. The model focuses on examining the type of pain, identifying main factors associated with chronic pain, and determining the motivation level of the patient. Wijma et al. presented a flowchart of the model for use in clinical practice [16]. Table 1 shows reliable, valid, and culturally adapted tools used in this study.

Missing Values

Little's test for missingness showed the data were missing completely at random. We performed multiple regression-based imputation to replace missing values and pooled the results of five iterations. Upon completion, all analyses were performed on the pooled imputed dataset.

Sample Size and Statistical Analysis

The sample size was calculated by estimating the prevalence of SAPS (40.00%) in a population of 80,843 subjects with shoulder pain [1]. A confidence level of 95% ($z = 1.96$), statistical power of 80% and loss of 15% resulted in a total of 435 participants with SAPS [$z^2 * p(1\text{-}p)/d^2$]. Considering the variability of this rate, exceeding the calculated number of subjects can improve the generalization of the results.

The SPSS version 22.0 (IBM corp. Armonk, NY, USA) was used for the statistical analyses. Descriptive statistics including means and standard deviations or proportions and percentages, and when appropriate, frequencies and distributions were calculated. To investigate differences between participants with and without kinesiophobia, independent samples t-tests or Chi square tests were performed. Before the bivariate analysis, continuous variables were converted to binomials using the midpoint of the ROC (Receiver Operating Characteristic) curve.

Bivariate logistic regression model: As the initial step of clinical prediction modeling, dedicated cumulative combinations of factors related to SAPS-related kinesiophobia were evaluated [28, 29]. Thirty-one unique logistic regression analyses for the outcome variable (high level of kinesiophobia; TSK > 37) were used to analyze bivariate relationships (one predictor to a single outcome). When multiple variables measured the same construct (e.g., average sleep hours and number of sleep interruptions), we identified the single item that most accurately reflected the construct (e.g., average sleep hours) [28, 29].

Multivariate logistic regression model: The multivariate regression analysis was performed with the variables with a p value of < 0.15 in bivariate logistic regression [28, 29]. To ensure appropriate modeling, multicollinearity was checked for each of the retained variable using Phi and Cramer's V to reflect the data type (nominal) and variables with a multicollinearity R value of < 0.6 were used in the multivariate analysis. A backward conditional stepwise logistic regression was used for the multivariate analysis. Variables with 95% confidence intervals without crossing 1.0 were considered statistically significant [28, 29].

Creating the prediction tool: To understand the impact of the cumulative variable combinations on the presence of kinesiophobia, the retained variables in the multivariate model were studied. This is a feature typical to clinical prediction rules modeling [28–30]. The retained variables from the aforementioned stepwise regression were entered into 2 × 2 contingency tables such that the combination of variables (e.g., 1 of X, 2 of X and 3 of X and so on) generated specificity, sensitivity, positive and negative likelihood

Table 1 Outcome measures based on PSCEBSM model

P	Based on the criteria proposed by Nijs et al., all participants had a nociceptive pain [17] Pain severity at rest and during activity was assessed with visual analog scale (VAS, 0 = no pain, 10 = the worst pain possible) [18]. The Shoulder Pain and Disability Index (SPADI) [19] and Disabilities of the Arm, Shoulder, and Hand (DASH) [20] scores were used to assess pain-related disability
S	According to Wijma et al., other health-related conditions and medication may have an impact on chronic pain [16]. A systematic review study reports an association between metabolic syndrome and SAPS [21] As defined by the United States National Heart, Lung and Blood Institute and American Heart Association Consensus Statement, metabolic syndrome is diagnosed as a constellation of 3 or more risk factors, including abdominal obesity, high triglycerides, low- and high-density lipoprotein cholesterol, high blood pressure, and elevated fasting blood glucose [22] Therefore, we recorded the presence of metabolic syndrome and using any non-steroidal anti-inflammatory drugs
C	The participants' feelings, emotions, and thoughts related to cognitive features of pain were evaluated with the Pain Catastrophizing Scale (PCS) [23], which is a 13-item self-administered questionnaire with 3 subscales: helplessness, magnification, and rumination. Each item is scored on a 5-point scale with higher values representing greater catastrophizing. Subscale scores obtained by adding all item-scores within a subscale and the total score is the summation of all items (0–52) [23] We used the second section of Illness Perception Questionnaire-revised (IPQ-R) to assess pain perceptions of the participants [24]. This section has 38 items with a five-point Likert response format (strongly agree to strongly disagree) in seven dimensions: (1) timeline acute/chronic (beliefs about the duration of illness), (2) timeline cyclical (beliefs about stability of symptoms over time), (3) consequences (beliefs about illness severity and impact on physical, social, and psychological functioning), (4) personal control (belief about one's own ability to control symptoms), (5) treatment control (belief in cure through treatment), (6) illness coherence (comprehension or understanding of the illness), and (7) emotional representation (perception of negative emotions generated by the illness). High scores for the 1st, 2nd, 3rd, and 7th dimensions represent strong beliefs regarding the number symptoms, as well as the chronicity and the cyclical nature of the negative illness-related emotions and consequences. High scores for the 4th, 5th, and 6th dimensions reflect positive beliefs and the understanding of the illness [24]
E	Emotional factors include anxiety and depressive feelings [16], which were assessed using the Hospital Anxiety and Depression Scale (HADS) [25]. Seven items for evaluating anxiety and 7 items for depression are scored on a 4-point scale ranging from 0 (not present) to 3 (considerable) [25]
B	Patients were divided into three subgroups in terms of behavioral pattern [26]: (i) Avoidance behavior; (ii) Persistence behavior (i.e., patients who perform and complete painful activities even though the activity is perceived as too hard); and (iii) Mixed pattern (i.e., patients who avoid certain activities or movements, but persist in others) [16]
S	Age, gender, level of education, housing/living condition, daily hours of working, average sleep duration (hours), average sleep interruptions (1–5 or more), marital status or relationship with the partner, the attitudes of healthcare professionals [for instance, a former physiotherapist who told the patient that his/her shoulder was getting worse, and recovery was unlikely (yes/no)], and other non-pharmaceutical interventions were recorded [16]
M	The participants indicated their treatment expectancy on a 10-cm VAS with endpoints labeled as "0 = there will be no pain relief after the treatment" and "10 = there will be complete relief after the treatment" [27]

ratios and 95% confidence intervals. For each combination, we captured the odds ratio and 95% confidence intervals, p value and Nagelkerke R^2, which is a measure of goodness of fit to help explain the strength of the independent variable with the model [28–30]. We also included a post-test probability of a negative and positive finding using a post-test prevalence calculator (Diagnostic post-test probability disease calculator) [30].

Results

Out of 748 patients who referred to the Orthopedics and Traumatology Outpatient Clinic for shoulder problems, 549 patients were diagnosed with SAPS and met the inclusion criteria according to the evaluations by the orthopedic surgeon. These patients were assessed for the relevant measures by the physiotherapist (Fig. 1). The study was completed with 207 participants in the high kinesiophobia group (TSK > 37) and 342 participants in the low kinesiophobia group (TSK ≤ 37). Baseline and comparative characteristics of the participants are presented in Table 2.

There was no difference between the two groups in terms of age, SPADI, percentage of using NSAIDs, IPQ-$R_{\text{timeline acute/chronic}}$, IPQ-$R_{\text{timeline cyclical}}$, IPQ-$R_{\text{consequences}}$, IPQ-$R_{\text{illness coherence}}$, percentage of patients living in an apartment, working duration, percentage of relationship with the partner or marital status, percentage of attitudes and beliefs of healthcare professionals, and presence of prior/other non-pharmaceutical interventions ($p = 0.059$–0.674, Table 2).

SAPS patients with high level of kinesiophobia had a significantly higher scores for $VAS_{\text{at rest}}$, $VAS_{\text{during activity}}$, DASH, $PCS_{\text{helplessness}}$, $PCS_{\text{magnification}}$, $PCS_{\text{rumination}}$, PCS_{total}, IPQ-$R_{\text{emotional representation}}$, $HADS_{\text{anxiety}}$, $HADS_{\text{depression}}$, and lower score for IPQ-$R_{\text{personal control}}$, IPQ-$R_{\text{treatment control}}$, average hours of sleep, and treatment expectancy compared to the participants with low level of kinesiophobia ($p = < 0.001$–0.039, Table 2). Compared to the TSK ≤ 37 group, in the TSK > 37 group, there was a higher percentage for presence of metabolic syndrome, avoidance behavior

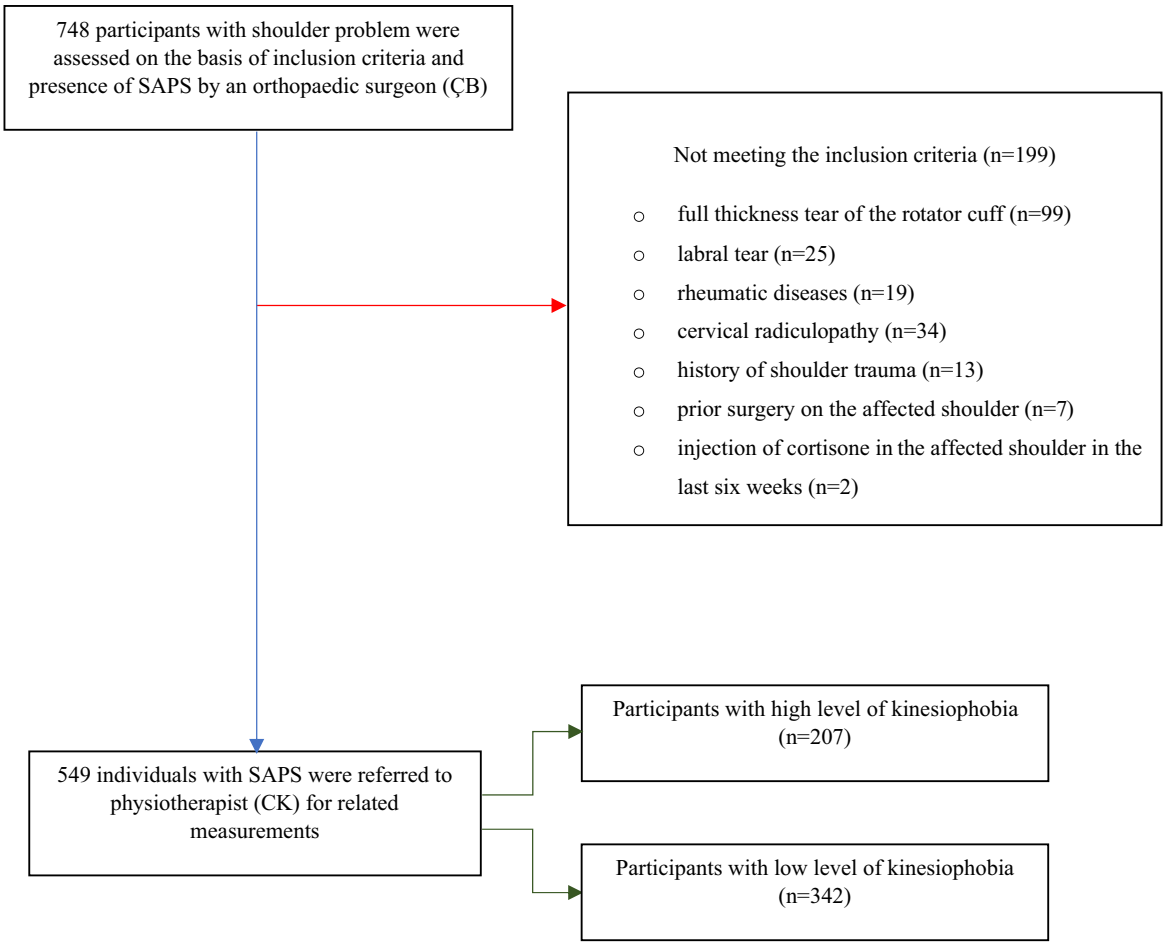

Fig. 1 Flowchart of the study

type, being female, low educational level, and 3 or more sleep interruptions ($p = < 0.001$–0.021, Table 2). In the TSK > 37 group, there was a lower percentage for persistence behavior type and mixed behavior type ($p = < 0.001$, Table 2).

Bivariate Modeling of Associative Factors and Kinesiophobia in SAPS Patients

Thirty-one variables were analyzed in the bivariate models, of which twenty-three were retained in the multivariate model ($p < 0.15$, Table 3). No variables were removed for multicollinearity (R values were all below 0.6). The multivariate analysis on the 23 variables revealed that 13 variables were associated with kinesiophobia in SAPS patients (Table 4). These were: $VAS_{at\ rest}$ (≥ 5.2), $VAS_{during\ activity}$ (≥ 7.1), DASH (≥ 72.1), presence of metabolic syndrome, $PCS_{helplessness}$ (≥ 16.1), $IPQ\text{-}R_{personal\ control}$ (≤ 17.1), $IPQ\text{-}R_{treatment\ control}$ (≤ 16.3), $HADS_{depression}$ (≥ 7.9), avoidance behavior type, being female, educational level ($\leq$ high school), average hours of sleep (≤ 6.8), and treatment expectancy (≤ 6.6).

Multivariate Modeling of Associative Factors and Kinesiophobia in SAPS Patients

Table 5 outlines the specificity, sensitivity, positive and negative likelihood ratios, and probabilities of having kinesiophobia in the presence of one or more of the 136 identified risk factors. Each cumulative risk factor increased the probability of having SAPS-related kinesiophobia. Using 7 of 13 risk factors is recommended for the clinical prediction tool, because having 7 or more risk factors increased the probability of having kinesiophobia to 51%, representing a moderate percentage of the sample ($n = 63$) with high level of kinesiophobia (Table 5).

Table 2 Descriptive statistics for the PSCEBSM model between SAPS patients with high ($n=207$) and low ($n=342$) level of kinesiophobia

Variables		Total ($n=549$)	Participants with TSK > 37 ($n=207$)	Participants with TSK ≤ 37 ($n=342$)	p value
P	VAS at rest	4.42 ± 1.28	4.93 ± 1.44	4.01 ± 0.87	< 0.001*
	VAS during activity	5.09 ± 1.51	5.78 ± 1.86	4.49 ± 1.23	< 0.001*
	SPADI	66.47 ± 18.44	68.42 ± 17.56	65.59 ± 18.93	0.081
	DASH	61.34 ± 16.94	64.98 ± 16.44	60.59 ± 18.78	0.005*
S	Metabolic syndrome (yes)	47 (8.56)	31 (14.97)	16 (4.67)	< 0.001*
	NSAIDs (yes)	128 (23.31)	52 (25.12)	76 (22.22)	0.436
C	PCS				
	Helplessness	11.71 ± 2.74	14.87 ± 3.98	9.43 ± 2.41	< 0.001*
	Magnification	7.14 ± 2.09	7.69 ± 2.85	6.99 ± 2.43	0.002*
	Rumination	8.96 ± 2.79	11.47 ± 2.54	7.68 ± 2.68	< 0.001*
	Total	28.01 ± 7.82	34.23 ± 10.37	24.30 ± 7.72	< 0.001*
	IPQ-R				
	Timeline Acute/chronic	12.72 ± 6.74	13.49 ± 5.95	12.45 ± 8.11	0.109
	Timeline cyclical	14.89 ± 4.57	15.54 ± 4.87	14.77 ± 4.48	0.059
	Consequences	16.01 ± 5.35	16.68 ± 4.47	15.81 ± 5.99	0.071
	Personal control	20.29 ± 5.83	18.21 ± 4.64	21.29 ± 7.13	< 0.001*
	Treatment control	18.64 ± 4.21	17.79 ± 3.68	19.18 ± 4.87	< 0.001*
	İllness coherence	16.89 ± 4.63	16.65 ± 4.44	16.93 ± 4.98	0.506
	Emotional representation	17.35 ± 5.13	17.94 ± 5.08	17.01 ± 5.14	0.039*
E	HADS				
	Anxiety	8.69 ± 2.48	9.84 ± 3.41	8.14 ± 2.65	< 0.001*
	Depression	6.14 ± 2.67	6.98 ± 2.81	5.61 ± 2.14	< 0.001*
B	Patients demonstrating				
	Avoidance	178 (32.42)	123 (59.42)	55 (16.08)	< 0.001*
	Persistence behavior	93 (16.93)	10 (4.83)	83 (24.26)	< 0.001*
	Mixed pattern	278 (50.63)	74 (35.74)	204 (59.64)	< 0.001*
S	Age	47.72 ± 13.47	48.15 ± 13.41	47.65 ± 13.57	0.674
	Sex (female)	314 (57.19)	147 (71.01)	167 (48.83)	< 0.001*
	Educational level (≤ high school)	268 (48.81)	128 (61.83)	140 (40.93)	< 0.001*
	Housing or living situation (apt)	347 (63.20)	120 (57.97)	222 (64.91)	0.104
	Working duration (years)	16.89 ± 11.48	15.96 ± 12.69	17.58 ± 10.85	0.112
	Average hours of sleep (hours)	6.67 ± 1.13	6.41 ± 1.24	6.84 ± 1.17	< 0.001*
	Average sleep interruptions (≥ 3)	142 (25.86)	65 (31.40)	77 (22.51)	0.021*
	Relationship with the partner or being married (yes)	483 (87.97)	185 (89.37)	298 (87.13)	0.434
	Attitudes and beliefs of healthcare professionals (yes)	30 (5.46)	13 (6.28)	17 (4.97)	0.513
	Prior/other non-pharmaceutical interventions (yes)	157 (28.59)	53 (25.60)	104 (30.40)	0.228
M	Treatment expectancy (0–10)	6.42 ± 2.41	3.79 ± 1.28	7.93 ± 2.49	< 0.001*

*Significant $p < .05$; variables represent number (%); Independent samples t-tests or Chi square tests were performed to understand differences between SAPS patients with and without kinesiophobia

TSK tampa-scale of kinesiophobia, *VAS* visual analog scale, *PSCEBSM* pain-somatic and medical factors-cognitive factors-emotional factors-behavioral factors-social factors-motivation, *SPADI* shoulder pain and disability ındex, *DASH* disabilities of the arm, shoulder and hand (DASH) score, *NSAIDs* non-steroidal anti-inflammatory drugs, *PCS* pain Catastrophizing Scale, *IPQ-R* ıllness perception questionnaire-revised, *HADS* hospital anxiety and depression scale, *apt* apartment

Discussion

This is the first study investigating risk factors for presence of high level of kinesiophobia in patients with SAPS. Creating a clinical prediction tool enabled us to provide information regarding the effect of multiple associative variables and high level of kinesiophobia. Thirteen significant risk factors of having kinesiophobia were: $VAS_{at\ rest}$ (≥ 5.2),

Table 3 Bivariate relationship between risk factors and having high level of kinesiophobia in patients with SAPS

Variable (Binomial distinction)		Odds ratio (95% CI)	*p* value	Nagelkerke R^2
P	VAS at rest (≥ 5.2)	3.08 (1.90–5.00)	< 0.001*	0.448
	VAS during activity (≥ 7.1)	3.31 (2.05–5.34)	< 0.001*	0.472
	SPADI (≥ 70.1)	1.82 (1.08–3.07)	0.023*	0.292
	DASH (≥ 72.1)	2.10 (1.26–3.50)	0.004*	0.337
S	Metabolic syndrome (yes)	3.58 (1.91–6.74)	< 0.001*	0.496
	NSAIDs (yes)	1.17 (0.78–1.75)	0.436	0.224
C	PCS			
	Helplessness (≥ 16.1)	1.95 (1.26–3.02)	0.002*	0.296
	Magnification (≥ 8.8)	1.48 (0.94–2.33)	0.085*	0.262
	Rumination (≥ 11.9)	1.80 (1.12–2.91)	0.015*	0.288
	Total (≥ 37.0)	1.69 (1.04–2.74)	0.031*	0.275
	IPQ-R			
	Timeline acute/chronic (≥ 13.8)	1.27 (0.86–1.87)	0.222	0.242
	Timeline cyclical (≥ 15.8)	1.33 (0.90–1.96)	0.143*	0.247
	Consequences (≥ 16.5)	1.30 (0.88–1.92)	0.180	0.245
	Personal control (≤ 17.1)	2.13 (1.46–3.09)	< 0.001*	0.354
	Treatment control (≤ 16.3)	2.04 (1.41–2.97)	< 0.001*	0.311
	İllness coherence (≤ 15.9)	0.79 (0.52–1.20)	0.281	0.189
	Emotional representation (≥ 18.7)	1.46 (0.99–2.14)	0.052*	0.258
E	HADS			
	Anxiety (≥ 10.7)	1.53 (0.97–2.40)	< 0.063*	0.269
	Depression (≥ 7.9)	2.00 (1.30–3.09)	0.001*	0.303
B	Patients demonstrating			
	Avoidance	7.64 (5.12–11.40)	< 0.001*	0.549
	Persistence behavior	0.15 (0.08–0.31)	< 0.001*	0.121
	Mixed pattern	0.37 (0.26–0.53)	< 0.001*	0.142
S	Age (≥ 52.4)	1.19 (0.81–1.76)	0.363	0.227
	Sex (female)	2.56 (1.77–3.70)	< 0.001*	0.419
	Educational level (≤ high school)	2.33 (1.64–3.32)	< 0.001*	0.399
	Housing or living situation (apt)	0.74 (0.52–1.06)	0.104*	0.179
	Working duration (years, ≥ 17.3)	1.21 (0.82–1.79	0.330	0.235
	Average hours of sleep (hours, ≤ 6.8)	2.07 (1.45–2.95)	< 0.001*	0.316
	Relationship with the partner or being married (yes)	1.24 (0.72–2.13)	0.435	0.239
	Attitudes and beliefs of healthcare professionals (yes)	1.28 (0.60–2.69)	0.513	0.243
	Prior/other non-pharmaceutical interventions (yes)	0.78 (0.53–1.16)	0.227	0.185
M	Treatment expectancy (0–10, ≤ 6.6)	2.16 (1.51–3.07)	< 0.001*	0.378

*Met criteria ($p < 0.15$) for inclusion in multivariate model

VAS visual analog scale, *SPADI* shoulder pain and disability ındex, *DASH* disabilities of the arm, shoulder and hand (DASH) score, *NSAIDs* non-steroidal anti-inflammatory drugs, *PCS* pain catastrophizing scale, *IPQ-R* ıllness perception questionnaire-revised, *HADS* hospital anxiety and depression scale, *apt* apartment, *CI* confidence intervals

$VAS_{during\ activity}$ (≥ 7.1), DASH (≥ 72.1), presence of metabolic syndrome, $PCS_{helplessness}$ (≥ 16.1), $IPQ\text{-}R_{personal\ control}$ (≤ 17.1), $IPQ\text{-}R_{treatment\ control}$ (≤ 16.3), $HADS_{depression}$ (≥ 7.9), avoidance behavior type, being female, educational level (≤ high school), average hours of sleep (≤ 6.8), and treatment expectancy (≤ 6.6). According to the model, presence of seven or more risk factors increased the probability of having high level of kinesiophobia from 34.3 to 51%. Although the model lacks validation in a longitudinal cohort study to determine its predictivity, the current results provide a deep insight to the risk factors of SAPS-related kinesiophobia. This can assist educating SAPS patients and developing interventions to tackle and cope with kinesiophobia.

Consistent with previous studies [32–34], avoidance behavior pattern was found to have one of the highest odds for developing high level of kinesiophobia in SAPS patients.

Table 4 Results of final multivariate model (backwards stepwise) demonstrating variables that are associated with high level of kinesiophobia in patients with SAPS ($R^2 = 0.166$)

Variable		Odds ratio (95% CI)	*p* value
P	VAS at rest (≥ 5.2)	3.16 (2.01–5.43)	< 0.001*
	VAS during activity (≥ 7.1)	3.26 (1.94–5.18)	< 0.001*
	DASH (≥ 72.1)	2.28 (1.41–3.62)	0.001*
S	Metabolic syndrome (yes)	3.71 (2.28–7.14)	< 0.001*
C	PCS		
	Helplessness (≥ 16.1)	1.83 (1.03–2.97)	0.008*
	IPQ-R		
	Personal control (≤ 17.1)	2.22 (1.51–3.39)	< 0.001*
	Treatment control (≤ 16.3)	2.13 (1.59–3.24)	< 0.001*
E	HADS		
	Depression (≥ 7.9)	2.14 (1.43–3.37)	< 0.001*
B	Patients demonstrating		
	Avoidance	7.41 (4.81–10.67)	< 0.001*
S	Sex (female)	2.61 (1.87–3.91)	< 0.001*
	Educational level (≤ high school)	2.40 (1.74–3.51)	< 0.001*
	Average hours of sleep (hours, ≤ 6.8)	2.13 (1.45–3.18)	< 0.001*
M	Treatment expectancy (0–10, ≤ 6.6)	2.24 (1.67–3.42)	< 0.001*

*Significant $p < 0.05$

CI confidence intervals, *VAS* visual analog scale, *DASH* disabilities of the arm, shoulder and hand (DASH) score, *PCS* pain catastrophizing scale, *IPQ-R* ıllness perception questionnaire-revised, *HADS* hospital anxiety and depression scale

Steimer considered moderate avoidance behaviors in acute stage as a normal response to stressful events [31]. Nevertheless, recent reviews stated that abnormal pain beliefs such as fear or catastrophizing lead to maladaptive escape behaviors and cause high level of kinesiophobia, all of which abet the transition from acute to chronic pain [32–34]. Furthermore, negative pain-related beliefs can disturb certain pain-processing regions of brain. Consistent with Malfliet et al. [35], it seems that SAPS patients with avoidance behavior pattern are more likely to refuse certain movements/activities erroneously believing that they will cause a (re)injury.

In the present study, presence of metabolic syndrome was found to be a significant risk factor for high level of kinesiophobia. However, there is no study in the relevant literature investigating the potential relationship between metabolic syndrome and kinesiophobia in patients with SAPS. Metabolic syndrome has been investigated in different shoulder pathophysiologies such as shoulder arthroplasty [36, 37], reverse shoulder arthroplasty [38], adhesive capsulitis [39], and posterosuperior rotator cuff tears [40]. A systematic review has suggested an association between metabolic syndrome and SAPS, however, the included studies had low to moderate quality of evidence [21]. Some studies associate SAPS with metabolic risk factors, such as obesity, body fat, and body mass index [41–43]. In their cross-sectional study, Miranda et al. reported a link between hyperglycemia and higher risk of shoulder pain [44]. According to a recent meta-analysis, in patients with diabetes mellitus, the prevalence of tendinopathy and tendon thickening is higher

Table 5 Clinical prediction tool for SAPS-related high level of kinesiophobia in the presence of different numbers of risk factors

Number (of 13*) risk factors present	Sensitivity (95% CI)	Specificity (95% CI)	Positive likelihood Ratio (95% CI)	Negative likelihood Ratio (95% CI)	Post-test probability when finding is positive (%)**	Post-test probability when finding is negative (%)
1 or more	98.5 (96.1–99.8)	4.2 (3.1–5.9)	1.03 (1.0–1.07)	0.36 (0.12–0.44)	35	16
2 or more	90.4 (88.8–94.7)	15.9 (13.8–17.7)	1.07 (1.03–1.11)	0.60 (0.51–0.67)	36	24
3 or more	83.5 (81.2–86.1)	25.8 (21.4–28.9)	1.15 (1.12–1.16)	0.62 (0.54–0.69)	38	23
4 or more	75.3 (71.2–78.6)	37.6 (34.8–39.9)	1.21 (1.17–1.28)	0.64 (0.60–0.73)	39	24
5 or more	66.2 (63.5–68.6)	50.8 (47.2–52.7)	1.35 (1.30–1.40)	0.65 (0.61–0.71)	41	26
6 or more	57.7 (54.1–59.4)	63.2 (61.0–65.3)	1.57 (1.51–1.65)	0.67 (0.62–0.73)	45	27
7 or more	48.6 (45.8–52.7)	75.8 (72.8–77.8)	2.01 (1.93–2.19)	0.68 (0.65–0.73)	51	29
8 or more	40.4 (36.8–43.7)	82.4 (80.4–84.7)	2.30 (2.23–2.36)	0.72 (0.66–0.76)	55	30
9 or more	33.8 (30.8–36.4)	88.5 (86.4–90.3)	2.94 (2.81–3.12)	0.75 (0.71–0.80)	61	31
10 or more	27.6 (25.7–29.2)	92.8 (90.6–94.7)	3.83 (3.62–4.19)	0.78 (0.73–0.84)	67	32
11 or more	19.5 (16.7–22.8)	95.6 (93.5–97.7)	4.43 (4.35–4.59)	0.84 (0.79–0.90)	70	33
12 or more	9.8 (6.7–11.9)	99.2 (98.1–100.0)	12.25 (12.12–12.42)	0.91 (0.85–0.95)	86	34
13 of 13	0.4 (0.00–0.7)	100.0 (99.2–100.0)	Inf (0.11-Inf)	0.99 (0.97–1.00)	~ 100	35

CI confidence intervals

*Thirteen significant variables: VAS at rest (≥ 5.2); VAS during activity (≥ 7.1); DASH (≥ 72.1); Metabolic syndrome (yes); PCS helplessness (≥ 16.1); IPQ-R personal control (≤ 17.1); IPQ-R treatment control (≤ 16.3); HADS depression (≥ 7.9); Demonstrating avoidance; Being female; Educational level (≤ high school); Average hours of sleep (≤ 6.8); Treatment expectancy (≤ 6.6)

**Pre-test probability was 34.3% before statistical analysis was performed to evaluate cumulative effects of associated variables

[45]. Goodson et al. highlighted the increased prevalence of severe chronic pain in people with metabolic syndrome [46]. Based on these findings, the association between metabolic syndrome and high level of kinesiophobia may stem from increased shoulder pain and impaired tendon structure, and thus increased fear of movement [36–46].

VAS at rest and VAS during activity with cut-off values of ≥ 5.2 and ≥ 7.1, respectively, were found to be associated with higher level of SAPS-related kinesiophobia in the current study. This is consistent with previous studies investigating the association between pain and kinesiophobia [47–51]. According to a recent systematic review including cross-sectional studies, there is an association between higher level of kinesiophobia and higher pain intensity, pain severity, and disability in patients with chronic musculoskeletal pain [47]. The Cognitive Fear Avoidance Model suggests that a threatening painful experience can generate catastrophizing beliefs that certain activities will cause more pain and lead to re-injury [52]. The subsequent avoidance behavior pattern causes disuse, disability, and depression in the long run. Patients are then trapped in the vicious cycle of increased fear of pain, more pain, and disability.

A higher DASH score was associated with greater kinesiophobia in SAPS patients. Das De et al. investigated the relationship between upper extremity-specific disability and kinesiophobia [53]. They reported a significant correlation between DASH scores and depressive symptoms, kinesiophobia, catastrophic thinking, and pain anxiety [53]. The study revealed that kinesiophobia and catastrophic thinking were the most prominent predictors accounting for half of the variance in upper extremity-specific disabilities [53]. Consistent with our findings, previous studies [54, 55] have demonstrated that the magnitude of upper extremity disability is associated with modifiable psychological factors; mainly misinterpretation of nociception (i.e., catastrophic thinking and kinesiophobia). Vincent et al. reported that in patients with chronic low back pain, kinesiophobia increased disability independent of pain scores, notably in obese patients [56]. This association is validated in other studies including individuals with neck-shoulder pain [6, 15] and chronic low back pain [57]. In a study by Crombez et al., kinesiophobia score was found to be a better predictor of disability than the pain anxiety score, even after adjusting for sociodemographic factors [55].

PCS helplessness, IPQ-R-personal control, and IPQ-R-treatment control (with cut-off values of ≥ 16.1, ≤ 17.1, and ≤ 16.3, respectively) were associated with higher odds for high level of SAPS-related kinesiophobia. Likewise, previous studies have reported an association between cognitive psychologic factors (such as negative pain perceptions and catastrophizing [6, 58, 59, 63, 64], and low pain self-efficacy [62]) and higher degrees of kinesiophobia [6, 59–61], and subsequently higher prevalence of shoulder pain and disability [6, 61, 62]. Given the fact that illness perception and cognition are modifiable psychological features, patients with SAPS can be educated about the stand-alone and combination of risk factors for kinesiophobia as well as modifying and/or mitigating strategies.

High level of depression (≥ 7.9) was found to be a significant risk factor for SAPS-related kinesiophobia. The association between depression and longer duration of shoulder symptoms, higher levels of shoulder disability, fear of movement, and poorer quality of life is reported in previous studies [64, 67, 68]. Bilgin et al. concluded that 1 unit increase in depression level increased the risk of kinesiophobia by 1.10 times in individuals with chronic neck and low back pain [69]. Similarly, Vlaeyen et al. highlighted the link between kinesiophobia and depressive symptoms [65]. The recent guidelines emphasize early detection and treatment of depression as it is the determinant for poor recovery [66].

In our study, female participants were found to have increased odds for SAPS-related kinesiophobia. Likewise, Luque-Suarez et al. reported that female sex and kinesiophobia had an adverse impact on shoulder pain and disability [70]. In contrast, Rovner et al. reported that when both males and females experience the same pain severity, women report significantly higher activity level, pain acceptance, and social support, while men report higher kinesiophobia, mood disturbances, and lower activity levels [71]. In their study investigating sex-related differences in patients with chronic pain, Racine et al. stated that their male participants had higher levels of kinesiophobia, were more likely to perceive their pain as being harmful, and used more activity pacing when performing daily activities, whereas women were more likely to exhibit an overdoing activity pattern than men [72]. However, we do not agree with the results of these studies based on different pathophysiological definitions [71, 72]. Because sex difference is reflected in our findings and others studies in the relevant literature in terms of the location/etiology of the pain [73]. This can be attributed to a combination of different aspects. When describing sex disparities in pain experience, Melchior et al. reported that the pain sensitivity and risk of chronic pain and kinesiophobia are higher in women compared to men [74]. The likelihood of experiencing greater pain intensity is higher in women as their pain tolerance and thresholds are lower [75]. Sex hormones, endogenous opioid functioning, and genotype influence pain sensitivity and thereby have a causal role in gender differences [75].

Lower educational level imposed a higher risk of kinesiophobia in patients with SAPS. Previous studies have shown educational level as an important parameter in determining pain severity, functional level, and quality of life in individuals with musculoskeletal problems [49, 76]. In these studies, compared to primary, secondary, and high school graduates, college graduates had higher functional status and

quality-of-life scores and lower pain severity. Researchers suggested that better financial and social conditions acquired by education can positively influence one's lifestyle and increase their quality of life [49, 76, 77]. A previous study reported no differences between higher and lower educated participants in terms of pain-related disability [78]. As mentioned earlier, individuals with higher educational levels are reported to have better functional level; thus, it is likely that they may have lower degrees of kinesiophobia. Individuals with higher educational and financial levels can access pain management methods easier and they can adapt these methods to their daily life [79, 80]. They have more opportunities to alleviate pain (such as visiting a doctor or searching on the internet) and to reduce pain-related psychological burden by participating in social activities more frequently [69].

Shorter sleep duration was also associated with SAPS-related kinesiophobia in this study. Prevalence of clinical pain and changes in pain-processing patterns increase in cases of sleep deprivation [81, 82]. Chronic sleep insufficiency can lead to sensitization [83]. To our knowledge, no studies have investigated the relationship between sleep deprivation and kinesiophobia in SAPS patients. Studies investigating this relationship in other pathologies (such as obstructive sleep apnea syndrome [84], systemic lupus erythematosus [85], and temporomandibular disorders [86]) have reported that sleep deprivation affects pain alleviating agents and the immune system and can hinder muscle recovery and damage repair. Sleep screening should be included in the management of SAPS and patients should be educated on the importance of sufficient sleep durations and strategies to prevent sleep-related problems.

Low treatment expectancy ($\leq$6.6) was associated with increased odds of SAPS-related kinesiophobia. Previous research highlighted that treatment expectancy is associated with both physical and cognitive-behavioral treatment outcomes in patients with chronic musculoskeletal pain [87, 88]. Higher pain-related fear was associated with lower treatment expectancy and lower credibility, whereas higher internal control was associated with higher treatment credibility [87, 88]. Goossens et al. reported that treatment expectancy could significantly predict the treatment outcome [89].

Limitations

As the study has a cross-sectional correlational design, only non-causal associations can be inferred from the findings. There are limitations with the methodology we used to develop the clinical decision tool, methods that are traditionally used to develop clinical prediction rules. However, these methods best reflected our purpose of combining parsimonious factors related to kinesiophobia. Using a factor analysis or a cluster analysis could be considered, but these approaches fail to identify the variables associated with kinesiophobia, instead they can only detect variables that have similar constructs (independent of kinesiophobia). Further, since a clinical prediction tool is generally developed from longitudinal modeling, we would need to follow-up our sample over time to establish evidence for the identified predictors of kinesiophobia.

Conclusion

This study created a clinical prediction tool that identified the cumulative effect of 13 risk factors [VAS at rest ($\geq$5.2), VAS during activity ($\geq$7.1), DASH ($\geq$72.1), presence of metabolic syndrome, PCS helplessness ($\geq$16.1), IPQ-R-personal control ($\leq$17.1), IPQ-R-treatment control ($\leq$16.3), HADS depression ($\geq$7.9), avoidance behavior type, being female, educational level ($\leq$high school), average hours of sleep ($\leq$6.8), and treatment expectancy ($\leq$6.6)] associated with high level of kinesiophobia in patients with SAPS. Presence of seven or more risk factors would increase the probability of having high level of kinesiophobia from 34.3% to 51%. Kinesiophobia may hinder rehabilitation adherence in SAPS patients. However, it is a modifiable factor that if minimized/eliminated, earlier pain relief and functional recovery will be facilitated. In this sense, clinicians are recommended to identify the presence of kinesiophobia prior to prescribing any intervention, since kinesiophobia may require a different and more specific approach than standard rehabilitation programs. Furthermore, although rather speculative, SAPS patients with high levels of kinesiophobia could be more likely to search for biomedical solutions for their pain, due to their fear of doing exercises, giving rise to more comorbid disorders. In this context, the frustration caused by kinesiophobia can negatively affect the therapeutic relationship between patients and physiotherapists and considerably restrict rehabilitation efforts. Ideally, the presence of kinesiophobia should be detected during the first assessment, to plan biopsychosocial treatment strategies focused on modifying kinesiophobia. Setting functional goals, educating patients on safe behavior management, and graded exposure to feared activities as behavioral experiments can be effective ways of modifying kinesiophobia. Proper selection of candidates, proper counselling, giving moral support, avoiding and treating modifiable factors, and treating comorbidities should be key strategies against high level of kinesiophobia.

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Data Availability The data that support the findings of this study are available from the corresponding author, upon reasonable request.

Declarations

Conflict of interest The authors have no conflicts of interest to declare.

Ethical approval This study was approved by Selçuk University Medical Faculty Clinical Research Ethics Committee (2021/812).

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