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Panic attacks and panic disorder in athletes: diagnostic challenges, exercise-related physiological symptoms, and clinical implications

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Abstract

Background. Panic attacks and panic disorder (PD) in athletes remain poorly characterised. Their assessment is complicated by the overlap between panic symptoms and physiological responses to intense exercise.

Aim. This narrative review summarises current evidence on epidemiology, interoceptive mechanisms, diagnostic challenges, clinical consequences, and management.

Materials and Methods. PubMed and Google Scholar were searched, primarily for studies published in 2020–2026; older relevant studies were also included. Athlete-specific evidence was prioritised, with general-population studies used where necessary.

Results. Epidemiological evidence is sparse and heterogeneous, and estimates still rely on a few earlier studies. Exercise-related sensations may be catastrophically interpreted in PD and contribute to avoidance, but may also have therapeutic value through interoceptive exposure. Differential diagnosis should consider respiratory and cardiac conditions, and screening should complement rather than replace clinical assessment. Treatment generally follows established PD approaches, including cognitive behavioural therapy and pharmacotherapy, but direct evidence in athletes remains scarce. Treatment decisions should account for potential effects on safety and functioning during training and competition.

Conclusions. Further athlete-specific research using standardised diagnostic approaches is needed to evaluate diagnostic and treatment strategies adapted to sport.

Keywords: panic disorder; panic attacks; athletes; exercise; interoception; mental health

1. Introduction

A panic attack is a sudden episode of intense fear or marked distress that rapidly reaches peak intensity [1]. It is accompanied by somatic and cognitive symptoms, such as palpitations, shortness of breath, dizziness, and fear of losing control [1]. Panic disorder (PD) differs from isolated panic attacks in that it is characterised by recurrent, unexpected attacks followed by persistent concern about further attacks or their consequences, or by a related change in behaviour [1]. Among athletes, panic attacks and PD should be considered within the broader context of anxiety symptoms, which are frequently reported among elite athletes [2]. In a 2026 meta-analysis including 30 studies and 9,848 active elite athletes, self-reported anxiety symptoms were present in 18.0% of participants, although substantial heterogeneity across studies warrants cautious interpretation of this estimate [2].

A particular challenge in this population is the overlap between panic symptoms and the physiological sensations associated with intense exercise [3]. Although exercise generally has anxiolytic effects, it may also provoke acute anxiety or panic attacks [3]. In athletes with PD, concern that these sensations may signal an impending panic attack can further intensify anxiety [3]. Differential diagnosis is further complicated by the fact that similar symptoms may occur in a range of medical conditions [4].

Epidemiological data on PD in athletes remain limited, and contemporary estimates of its prevalence still rely on a small number of earlier studies [5,6]. Screening tools developed for athletes assess a broader range of mental health problems and are not specific to PD [3,7]. Intervention studies addressing anxiety and related disorders in elite athletes also remain relatively scarce compared with epidemiological studies [6].

The aim of this narrative review is to summarise the current evidence on panic attacks and panic disorder in athletes. Particular attention is given to exercise-related physiological symptoms, interoceptive mechanisms, diagnostic challenges, clinical consequences, and clinical management.

2. Methods

This narrative review was based on literature identified through PubMed and Google Scholar. Searches focused primarily on literature published between 2020 and 2026, although older studies were also included when they were particularly relevant to the topics covered or addressed areas with limited recent evidence. The final search was conducted in September 2026. Various combinations of English-language search terms were used, including “panic attack”, “panic disorder”, “athlete”, “elite athlete”, “exercise”, “exercise-induced panic”, “anxiety sensitivity”, “interoception”, “EILO”, “EIB”, and “treatment”.

Priority was given to studies conducted in athlete populations. In areas where athlete-specific evidence was limited, studies from the general population were also included, particularly those addressing underlying mechanisms and treatment. Additional publications were identified from references cited in relevant systematic reviews, review articles, current position statements, and expert consensus documents.

The synthesis included publications directly relevant to the scope of the review, with particular emphasis on original studies, systematic reviews, expert consensus statements, and clinical guidelines. Given the narrative nature of the review, no formal risk-of-bias assessment or systematic review-style study selection process was undertaken. The Scale for the Assessment of Narrative Review Articles (SANRA) was used as a supplementary tool for the final quality appraisal of the review [8].

3. Panic attacks and panic disorder among athletes

Data on the prevalence of PD among athletes remain limited. In a nationwide study of 2,067 French athletes classified in the High Level or Junior categories, Schaal et al. reported current or recent PD (<6 months) in 1.2% of athletes and a lifetime prevalence of 2.8% [9]. Lifetime prevalence was higher in women than in men (4.4% vs 1.9%) [9]. In a study of 224 Australian elite athletes, Gulliver et al. found that 4.5% of participants met questionnaire-based caseness criteria for PD, while approximately 10% reported at least one full-symptom or limited-symptom panic attack during the previous week [10]. The most recent International Olympic Committee (IOC) consensus statement continues to report a PD prevalence of 2.8–4.5% among elite athletes, based on these earlier studies [6]. A broader meta-analysis of athlete mental health also indicates that female athletes report anxiety symptoms more frequently than male athletes [11].

Clinical data on PD in athletes are similarly scarce. In a two-clinic cohort of 221 elite athletes seeking psychiatric care, standardised diagnostic data were available for 180 athletes from the Stockholm clinic. In this subgroup, 69% had at least one anxiety disorder; however, PD was not reported separately and was instead included within the broader F41 diagnostic category [12]. More recent clinical evidence comes from an audit of a specialist sports psychiatry clinic in Malaysia involving 27 athletes. Five diagnoses of PD and one performance-related panic attack were recorded, and anxiety disorders were the most common diagnostic category [13]. However, given the small and highly selected clinical sample, these data cannot be used to estimate the prevalence of PD in the broader athlete population. Isolated panic attacks, by contrast, have been reported in a substantial proportion of athletes in some samples, although their occurrence should not be equated with a diagnosis of PD. In a study of 244 university athletes, DeWolfe et al. found that 38.3% of participants reported at least one panic attack during the previous year [14]. This finding should not be interpreted as an estimate of PD prevalence, as panic attacks were assessed using a single self-report item. The authors noted that this approach may overestimate their occurrence relative to estimates based on diagnostic criteria [14].

Selected athlete-specific studies relevant to panic disorder and panic attacks, together with their principal interpretive limitations, are summarised in Table 1.

Table 1. Selected athlete-specific studies relevant to panic disorder and panic attacks

Study and population	Assessment	Principal findings	Interpretive limitation
Schaal et al. (2011) [9] 2,067 French High Level or Junior athletes	Nationwide annual psychological evaluations; diagnoses based on DSM-IV or ICD-10 criteria	PD: 1.2% current/recent (<6 months) and 2.8% lifetime; lifetime prevalence 4.4% in women and 1.9% in men	Older cross-sectional data; assessment was conducted by different professionals across centres
Gulliver et al. (2015) [10] 224 Australian elite athletes	Internet-based self-report survey using symptom measures	4.5% met questionnaire-based caseness for PD; approximately 10% reported at least one full- or limited-symptom panic attack in the previous week	Questionnaire caseness and self-reported attacks do not establish a clinical diagnosis
Åkesdotter et al. (2022) [12] 221 treatment-seeking elite athletes at two Swedish clinics; diagnostic subgroup n = 180	Retrospective clinical cohort; standardised diagnostic interviews at the Stockholm clinic	69% of the diagnostic subgroup had an anxiety disorder; PD was not reported separately and was included within the broader F41 category	Highly selected treatment-seeking sample; no PD-specific prevalence estimate
Koh et al. (2026) [13] 27 athletes attending a Malaysian sports psychiatry clinic	Retrospective clinical audit	Five PD diagnoses and one performance-related panic attack were recorded	Small, selected clinical sample; findings cannot estimate population prevalence
DeWolfe et al. (2025) [14] 244 university varsity athletes	Self-report survey; panic attacks assessed with a single item	38.3% reported at least one panic attack during the previous year	Single-item self-report; panic attacks are not equivalent to PD and may be overestimated

4. Exercise-related physiological symptoms and interoceptive mechanisms

In the context of panic attacks in athletes, somatic responses and sensations elicited by intense exercise, including tachycardia, shortness of breath, dizziness, and sweating, are of particular relevance. Some of these sensations overlap with symptoms experienced during a panic attack [3,15]. In individuals with PD, not only the occurrence of these symptoms but also the way in which they are interpreted may be clinically relevant. According to the cognitive model of panic, sensations associated with physiological arousal may be interpreted as threatening [1]. Catastrophic interpretations of such sensations may in turn intensify anxiety and promote avoidance of situations in which similar symptoms occur, including exercise [14,15].

Studies in patients with PD have also demonstrated greater fear and avoidance of exercise. Muotri and Bernik found that patients with PD had significantly higher scores on the Questionnaire for Assessment of Physical

Activity Avoidance (QAPAA), which assesses fear and avoidance of exercise. Mean fear-of-exercise scores were 6.71 in the respiratory subtype, 5.50 in the non-respiratory subtype, and 0.46 in the control group, with significant differences between both PD groups and healthy controls ($p < 0.001$). A similar pattern was observed for exercise avoidance, with corresponding mean scores of 6.00, 5.50, and 1.40 ($p < 0.001$). In the same sample, patients with PD also had lower maximal oxygen uptake (VO_2max) than healthy controls: $26.85 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ in the respiratory subtype, $26.28 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ in the non-respiratory subtype, and $33.97 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ in the control group ($p < 0.001$) [16].

In a separate cardiopulmonary exercise study, Muotri et al. also identified differences in perceived exercise intensity. At a workload corresponding to 90% of individual VO_2max , patients with PD reported higher ratings of perceived exertion on the Borg scale than healthy controls (18.93 ± 0.55 vs 16.67 ± 0.60 ; $p < 0.05$). At the same relative workload, their heart rate was slightly lower (160.97 ± 6.55 vs 164.73 ± 4.48 beats/min; $p < 0.05$) [15]. These findings suggest that perceived exercise intensity in patients with PD may not fully correspond to physiological measures of exertion. Similar differences were reported by Caldirola et al.; in a small sample, patients with PD showed lower oxygen consumption and higher ratings of perceived exertion than healthy controls, with no significant associations between these parameters and the psychological variables assessed [17].

The same physiological sensations that may intensify anxiety during exercise can also be used therapeutically as part of interoceptive exposure [3,18,19]. In a study by Smits et al., 20 minutes of moderate exercise performed before a 35% CO_2 challenge was associated with lower panic and anxiety reactivity to the challenge compared with rest in healthy individuals with no history of panic attacks [20]. Repeated exposure to interoceptive sensations such as tachycardia or shortness of breath may help reduce catastrophic interpretations of these sensations and the anxiety associated with them [18,19,21]. In another study, six brief exercise sessions produced a significant reduction in anxiety sensitivity among individuals with elevated anxiety sensitivity at baseline, supporting the potential role of exercise as a form of exposure to interoceptive sensations [22]. Exercise intensity may also be relevant. In an exploratory study by Wintermann et al., low-intensity exercise increased heart rate in patients with PD but did not produce a significant increase in anxiety or cortisol levels [23]. However, the experimental studies cited above were not conducted in athletes, limiting the extent to which their findings can be directly applied to this population.

A systematic review by Machado et al. of eight studies of regular exercise in patients with PD found more consistent improvements in overall anxiety; effects on panic symptoms remained inconclusive [24]. Muotri et al. [19] randomised 102 sedentary patients with PD to 12 weeks of brief intermittent intense exercise (BIE) or relaxation training; outcomes were reported for 72 completers (BIE, $n = 37$; relaxation, $n = 35$). After 12 weeks, symptom severity assessed using the Panic Agoraphobia Scale (PAS) was significantly lower in the BIE group than in the relaxation group (14.9 ± 5.3 vs 23.1 ± 9.4 ; $p < 0.001$). This difference was maintained at week 24 (14.2 ± 5.5 vs 24.7 ± 8.5 ; $p < 0.001$) [19]. In addition, at week 24, the frequency of panic attacks was significantly lower in the BIE group than in the relaxation group (0.7 ± 0.6 vs 1.5 ± 1.0 ; $p = 0.003$) [19]. Results for completers suggest potential value for BIE in PD; applicability to athletes remains uncertain because participants were sedentary.

The proposed bidirectional role of exercise-related bodily sensations in the maintenance and treatment of panic symptoms is illustrated in Figure 1 [1,3,18–22].

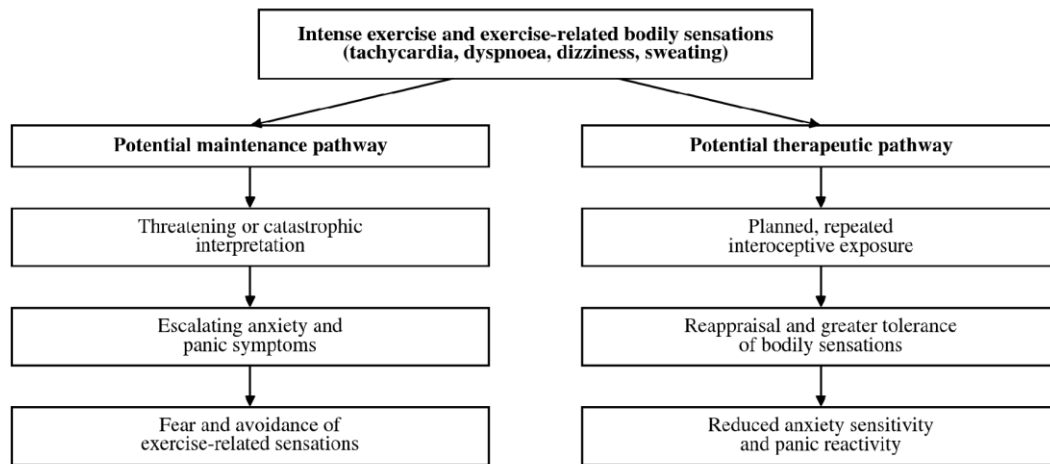


Figure 1. Proposed bidirectional role of exercise-related bodily sensations in panic disorder

Note: This conceptual synthesis is based primarily on evidence from non-athlete populations; direct evidence in athletes remains limited [1,3,18–22].

5. Diagnostic challenges in athletes

The overlap between exercise-related sensations and panic symptoms complicates diagnostic assessment in athletes. Similar symptoms may arise from medical conditions, including exercise-induced laryngeal obstruction (EILO) and exercise-induced bronchoconstriction (EIB) [3,25].

EILO is a transient obstruction of the upper airway at the level of the larynx. Symptoms typically occur at peak ventilatory demand, are most pronounced during inspiration, and usually resolve within 2–3 minutes after exercise cessation [25]. A similar pattern was observed in a retrospective study of competitive athletes referred for evaluation of EILO. Among 73 athletes with confirmed EILO, 66 reported symptoms during high-intensity exercise, 65 indicated that symptoms were most severe during inspiration, and 49 reported stridor [26].

When differentiating EILO from a panic attack, the timing of symptoms, their relationship to the respiratory phase, and the presence of stridor are therefore particularly relevant. The presence of accompanying anxiety alone is insufficient to distinguish between these conditions [3,25].

EILO and asthma may coexist. Among 55 athletes with EILO for whom objective test results for asthma were available, 22 had findings supporting a diagnosis of asthma [26]. In contrast to EILO, individuals with asthma and/or EIB generally tolerate ongoing exercise relatively well and predominantly report expiratory symptoms, while wheezing typically peaks 3–15 minutes after exercise cessation [25]. Because of the overlap in clinical presentation, diagnostic assessment should not rely solely on reported symptoms. When EILO is suspected, continuous laryngoscopy during exercise (CLE) is the diagnostic gold standard, whereas the diagnosis of EIB requires objective assessment of pulmonary function [25,26].

In addition to respiratory causes, cardiac conditions should be considered in the differential diagnosis. Symptoms such as palpitations, dizziness, and chest discomfort or pain may occur both during panic attacks and in

cardiovascular disease [3,27]. However, the sensation of a rapid heartbeat does not necessarily indicate an arrhythmia, as palpitations may also accompany normal physiological responses [27]. Features that increase suspicion of a cardiac cause include a sudden onset and termination of palpitations, particularly when accompanied by chest pain, presyncope, or syncope [27]. In athletes presenting with palpitations, the initial evaluation should include a detailed history, physical examination, and resting 12-lead ECG. When symptoms are exercise-related, evaluation should include exercise testing aimed at reproducing the conditions under which symptoms occur or rhythm monitoring during activity [27].

Exertional syncope warrants particular caution, especially when it occurs without prodromal symptoms, as potentially serious cardiac causes should be excluded in this setting [27]. In a pooled analysis of six studies including 82 athletes with exertional syncope, no structural heart disease was identified in 75 athletes (91.5%), whereas cardiovascular disease was identified in 7 (8.5%) [27].

The principal clinical features that may help distinguish panic attacks from common respiratory and cardiac mimics are summarised in Table 2 [3,25–27].

Table 2. Differential diagnosis of panic-like symptoms during or after exercise in athletes

Condition	Typical timing and pattern	Key clinical features	Diagnostic assessment
Panic attack	Sudden onset with a rapid peak; may occur during exercise, after exercise, or independently of sport.	Intense fear or distress with palpitations, dyspnoea, dizziness, sweating, and fear of losing control. Symptoms are not consistently linked to the inspiratory or expiratory phase.	Clinical psychiatric assessment after relevant medical causes have been considered; screening tools are supportive, not diagnostic [1,3].
EILO	Usually occurs during high-intensity exercise and is maximal at peak ventilatory demand; symptoms commonly resolve within 2–3 minutes after exercise cessation.	Predominantly inspiratory dyspnoea, throat tightness, and stridor. Anxiety may accompany the episode but does not establish a panic diagnosis.	Continuous laryngoscopy during exercise (CLE) is the diagnostic gold standard [25,26].
EIB/asthma	Symptoms often become most pronounced after exercise; wheezing typically peaks 3–15 minutes after exercise cessation.	Predominantly expiratory dyspnoea, wheeze, cough, or chest tightness. Athletes may tolerate ongoing exercise better than those with EILO.	Objective pulmonary function testing, with bronchoprovocation when indicated [25,26].
Cardiac cause	May occur during exertion or abruptly at rest. Sudden onset and termination, particularly with exertional symptoms, increase suspicion.	Palpitations, chest pain or discomfort, dizziness, presyncope, or syncope. Exertional syncope without prodromal symptoms is a red flag.	History, physical examination, and resting 12-lead ECG; exercise testing or ambulatory/activity rhythm monitoring when indicated [27].

Similar symptoms may also arise from other medical conditions, including anaemia, hypoglycaemia, and thyroid dysfunction [3]. Caffeine use should also be considered, as it may cause symptoms such as nervousness, restlessness, and tachycardia [3,27]. History-taking should address the use of stimulants and performance-

enhancing substances with potential arrhythmogenic effects [27]. Psychosocial factors should likewise be considered, particularly in younger athletes; a recent case report illustrates how anxiety-related symptoms may complicate the assessment of exertional dyspnoea [28].

6. Clinical consequences

A panic attack during athletic activity may directly impair an athlete's functioning by affecting breathing, heart rate, cognition, and perceived control [6].

Broader evidence on anxiety in athletes also suggests that greater anxiety symptom severity may impair attention, executive functioning, and information processing [3,29]. Among elite athletes, higher levels of self-reported anxiety have also been associated with poorer performance and a greater number of performance errors [3,29,30]. Depending on the severity of symptoms and their impact on the athlete's safety, stability, and functioning, temporary modification of participation in training or competition may be necessary. In more severe cases, temporary withdrawal from sport may be required [6,31].

7. Clinical management

Accurate diagnosis and assessment of the impact of symptoms on an athlete's functioning form the basis of subsequent management. Treatment generally follows standards used in the general population and may include psychotherapy, pharmacotherapy, or a combination of both, depending on the clinical presentation and symptom severity [3,30]. Management should nevertheless be adapted to the specific demands of athletic participation, including training requirements and competition schedules [3,6]. In PD, cognitive behavioural therapy (CBT) may incorporate exposure-based techniques, including interoceptive exposure [3,32].

Selective serotonin reuptake inhibitors (SSRIs) are generally preferred in athletes [3,6,33]. This preference is also consistent with the World Federation of Societies of Biological Psychiatry (WFSBP) guidelines for the treatment of PD in the general population. These guidelines recommend SSRIs and venlafaxine, a serotonin-norepinephrine reuptake inhibitor (SNRI), as first-line pharmacological treatments, with level A evidence and recommendation grade 1 [32]. A Cochrane network meta-analysis including 70 randomised clinical trials in adults with PD found that all medication classes examined were more effective than placebo in terms of treatment response, while no significant differences were identified between drug classes [34]. In a survey of sports psychiatrists, Reardon et al. found that sertraline and escitalopram were the most frequently selected medications for treating PD in athletes (33% each; n = 13), followed by fluoxetine (13%; n = 5) [35].

The most commonly reported reasons for selecting these medications were efficacy, overall tolerability, and minimal impact on athletic performance [35]. However, SNRIs were not among the three most frequently preferred medications for treating PD in this survey [35]. In clinical practice, medication choice depends not only on expected efficacy and potential effects on athletic performance but also on the adverse-effect profile [6,35]. Among the 20 adverse effects assessed in the same survey, sedation or drowsiness (73%), weight gain (58%), and impaired athletic performance (27%) were most frequently identified as factors limiting medication use [35].

Available evidence on the effects of selected SSRIs on physical performance remains inconclusive [6,36,37]. Studies of fluoxetine found no significant effects on selected measures of physical performance or muscle strength, whereas findings for paroxetine were inconsistent [37]. Interpretation of these findings is limited because studies examining the effects of antidepressants on physical performance have often focused on short-term outcomes and

included individuals without a diagnosed mental disorder. In addition, the dosing regimens used did not always reflect routine clinical practice [36,37].

Benzodiazepines may also be used in the treatment of PD, although their role is more limited [32]. Despite a high level of evidence for efficacy (level A), the WFSBP assigns benzodiazepines a lower recommendation grade (grade 2) because of the risk of dependence [32]. Their rapid onset of action supports short-term use during the first weeks of SSRI/SNRI therapy, before the full antidepressant effect develops. They may also be used for severe acute panic attacks [32].

In athletes, benzodiazepine-related effects that may impair psychomotor performance, such as slowed reaction time, cognitive impairment, and muscle relaxation, are particularly relevant [3,33,37]. However, the extent to which these effects translate into impaired athletic performance has not been clearly established. Negative effects may be more pronounced with longer-acting agents [6]. Treatment decisions in athletes should therefore balance clinical benefits against the potential effects of treatment on functioning during training and competition [3,6].

8. Discussion

Panic attacks and panic disorder in athletes remain poorly characterised, and the limited number of studies specific to this population makes it difficult to reliably estimate the prevalence of PD and determine optimal treatment approaches [3,6]. Interpretation of symptoms is further complicated by their substantial overlap with physiological responses to intense exercise, particularly tachycardia, shortness of breath, and dizziness [3,6]. These interoceptive sensations may intensify anxiety in individuals with PD, but may also serve as targets for interoceptive exposure [18,19]. In clinical practice, particular attention should therefore be paid to distinguishing panic attacks from respiratory and cardiac conditions that may produce similar symptoms [25,27]. Management should also account for the demands of training and competition [3].

Contemporary estimates still rely primarily on the studies by Schaal et al. and Gulliver et al., although these were conducted in 2011 and 2015, respectively [6,9,10]. Comparisons across studies are further complicated by heterogeneity in the measures used, including PD diagnoses, questionnaire-based caseness, broader anxiety-disorder categories, and self-reported panic attacks [10,12–14]. More recent clinical data, such as the audit by Koh et al., document PD diagnoses among athletes seeking psychiatric care, but the small and highly selected clinical sample precludes reliable estimation of prevalence in the broader athlete population [13]. As a result, the true prevalence of PD in this population remains uncertain.

The relationship between exercise and interoceptive mechanisms in PD remains insufficiently understood. Available studies suggest that patients with PD may perceive exercise-related sensations as more intense, while fear of these sensations may contribute to avoidance of physical activity [15–17]. Conversely, controlled exposure to similar sensations may reduce anxiety sensitivity and, in some studies, panic symptom severity [19,20,22]. The applicability of these findings to athletes is limited because none of the cited studies directly examined this population, and some were conducted in physically inactive individuals. It therefore remains unclear whether regular exposure to tachycardia, increased ventilation, and other exercise-related sensations influences how athletes interpret such sensations or their susceptibility to panic symptoms.

The overlap between exercise-related sensations and panic symptoms complicates differential diagnosis. Shortness of breath, palpitations, dizziness, and chest discomfort may occur both during a panic attack and in respiratory or cardiac conditions [3,25,27]. In clinical practice, not only the type of symptoms but also the circumstances in which they occur, their temporal relationship to exercise, and the presence of features suggesting a specific medical

cause are relevant [25–27]. Over-attributing such symptoms to anxiety may lead to clinically important medical conditions being overlooked, whereas focusing exclusively on medical causes may hinder recognition of coexisting panic symptoms [3,4].

Because available screening tools for athletes are not specific to PD, their results should be interpreted cautiously when assessing for this disorder [3,7]. In a study of 1,121 Polish elite athletes, 72.4% of participants exceeded the triage threshold in the IOC Sport Mental Health Assessment Tool 1 (SMHAT-1), and 51% screened positive on at least one of the subsequent screening tools. Following a brief clinical interview, only 9.5% of athletes had persistent mental health concerns requiring referral to a specialist [38]. This substantial discrepancy between screening results and clinical assessment indicates that a positive screening result should not be equated with a diagnosis. A similar distinction is reflected in the IOC Sport Mental Health Assessment Tool 2 (SMHAT2), in which the Generalized Anxiety Disorder-7 (GAD-7) is used to screen for anxiety symptoms, while a positive result leads to further assessment, including clinical evaluation [7].

Evidence for treating PD in athletes remains much more limited than the broader literature on psychological interventions for anxiety in athletes. In a meta-analysis by Li et al. including 24 studies and 853 athletes, psychological interventions significantly reduced state anxiety, with a large overall effect ($SMD = -0.99$; 95% CI -1.16 to -0.81) [39]. However, these studies focused primarily on sport-related and situational anxiety rather than clinically diagnosed PD, limiting the extent to which their findings can be directly applied to the treatment of PD in athletes.

Evidence on pharmacological treatment of PD in athletes also comes largely from studies conducted in the general population [3,6]. The network meta-analysis by Guaiana et al. included 70 studies and 12,703 adults with PD. No differences in treatment response were found between the main drug classes, while benzodiazepines ranked highest for acceptability, as measured by treatment discontinuation [34]. Interpretation of these findings is nevertheless limited by the low quality of evidence for benzodiazepine comparisons and the short duration of follow-up [34]. WFSBP guidelines recommend SSRIs and venlafaxine as first-line treatments, while benzodiazepines receive a lower recommendation grade because of the risk of dependence [32]. In athletes, benzodiazepine-related effects such as sedation, muscle relaxation, and slowed reaction time are particularly relevant [3]. Treatment choice should therefore take into account both clinical efficacy and the potential impact of treatment on training and competition [3,6].

Future research should use structured diagnostic instruments based on standard diagnostic criteria, clearly distinguish panic attacks from PD, and include larger and more diverse athlete samples. Brief screening tools, such as the Athlete Mental Health Screener-18 (AMHS-18), may facilitate longitudinal monitoring of athletes' mental health, but they do not replace clinical diagnosis and are not specific to PD [40]. Prospective studies examining the role of interoceptive sensations in trained individuals are particularly needed, as are intervention studies evaluating CBT, interoceptive exposure, and pharmacotherapy directly in this population. Another priority is the development of more effective approaches to distinguishing panic symptoms from exercise-related respiratory and cardiac conditions.

This review has several limitations inherent in its narrative design. The literature search was limited to PubMed and Google Scholar, and no formal risk-of-bias assessment was performed. Evidence from the general population was also included because of the limited number of athlete-specific studies. The findings should therefore be

interpreted in light of the heterogeneity of the available literature and the limited extent to which some results can be directly generalised to athletes.

9. Conclusions

Evidence on panic attacks and panic disorder in athletes remains limited, and current epidemiological estimates are based largely on a small number of methodologically heterogeneous studies. Of particular clinical importance is the overlap between panic symptoms and the physiological responses associated with intense exercise, which complicates diagnosis and requires consideration of psychiatric as well as respiratory, cardiac, and other medical causes. Screening tools may help identify athletes who require further assessment, but they should not replace clinical evaluation. Interoceptive mechanisms may play an important role both in the maintenance of panic symptoms and in their treatment. However, most evidence on interoceptive exposure and pharmacotherapy comes from the general population. Management in athletes should therefore combine established standards for the treatment of PD with careful consideration of the effects of treatment on safety and functioning during training and competition. Future research should focus on studying PD directly in athlete populations and on the effectiveness of diagnostic and therapeutic strategies tailored to the specific demands of sport.

Disclosure

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