



Postural Orthostatic Tachycardia Syndrome After SARS-CoV-2 Infection in a Young Competitive Athlete: A Narrative Review of Return-to-Activity Decisions

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Abstract

A young athlete who cannot stand up without their heart rate climbing thirty beats is in a difficult position. The symptoms are disabling, the investigations are usually normal, and the advice that helps most other patients, rest and gradual return, is the advice most likely to make this condition worse.

Objectives: *To review the published evidence on postural orthostatic tachycardia syndrome following SARS-CoV-2 infection: how common it is, how it is distinguished from deconditioning and from other post-acute sequelae, what management is supported, and how return-to-activity decisions are made in athletes.*

Material and Methods: *Narrative review of systematic reviews and meta-analyses, referral-clinic cohorts, consensus statements and sports cardiology guidance indexed in PubMed and the major cardiology, neurology and sports medicine journals. No new patient data were generated; every estimate quoted is one published by the original investigators.*

Results: *Pooled across 21 observational studies, prevalence in post-acute sequelae of COVID-19 was 70.6 per cent for orthostatic intolerance, 36.2 per cent for postural orthostatic tachycardia syndrome and 18.6 per cent for orthostatic hypotension, with publication bias evident for the second. In a dedicated autonomic referral clinic, 22 of 75 individuals assessed after infection (29 per cent) received a diagnosis of the syndrome, while in 49 of 101 referrals (49 per cent) the workup excluded any cardiovascular autonomic disorder. Among those diagnosed and followed, 26 of 42 (62 per cent) improved symptomatically.*

Conclusion: *The prevalence figures differ by an order of magnitude depending on whether the population is self-selected, referred or systematically screened, and the pooled estimates should not be read as population risk. What is consistent is that the diagnosis is made by tilt or standing test rather than by symptom report, that half of those referred have something else, and that most improve.*

Abbreviations: HUTT – Head-Up Tilt Table Test, OH – Orthostatic Hypotension, OI – Orthostatic Intolerance, PASC – Post-Acute Sequelae of COVID-19, POTS – Postural Orthostatic Tachycardia Syndrome, VVS – Vasovagal Syncope.

Keywords: Postural Orthostatic Tachycardia Syndrome; Long COVID; Dysautonomia; Athletes; Return to Activity

Introduction

Postural orthostatic tachycardia syndrome is defined by a sustained rise in heart rate on standing, without orthostatic hypotension, accompanied by symptoms of orthostatic intolerance persisting for at least three months [1,2]. It predominantly affects young women, and it was recognised long before the pandemic, frequently after a viral illness [3,4].

SARS-CoV-2 gave the field an unusually large and temporally defined cohort. From mid-2020 onwards, clinics began seeing patients, often young and previously fit, with palpitations, exercise intolerance and cognitive symptoms persisting long after the acute illness [5,6,7], within the broader picture of post-acute COVID-19 syndrome [27] and of symptoms persisting at population level [28]. In a large international survey of patients with persistent symptoms, palpitations were reported by around 68 per cent and chest pain by around 53 per cent at seven months [8].

For an athlete the stakes are specific. The presenting complaint is that training has become impossible, the differential includes myocarditis and deconditioning as well as dysautonomia, and the management for one is close to the opposite of the management for another [9,10]. This review sets out what is known about how often the syndrome follows infection, how it is confirmed, and what the evidence supports in returning someone to activity.

Material and Methods

Search strategy: PubMed and the archives of the major cardiology, autonomic neuroscience and sports medicine journals were searched for studies of autonomic dysfunction after SARS-CoV-2 infection. Search terms combined postural orthostatic tachycardia syndrome, dysautonomia and orthostatic intolerance with COVID-19, post-acute sequelae, long COVID, athlete and return to play. Reference lists of consensus documents [1,2,11] were screened by hand for primary sources.

Eligibility: Studies were included if they reported the frequency, characteristics or management of autonomic dysfunction following SARS-CoV-2 infection, or addressed exercise prescription and return to activity in this population. Pre-pandemic studies of the syndrome are cited to establish the diagnostic framework and are identified as such.

Data extraction and handling: Design, setting, sample size, how cases were ascertained and the reported estimate were extracted as published. How the population was assembled is reported alongside every prevalence figure, because self-selected, referred and systematically screened cohorts give very different answers and much of the apparent disagreement in this literature is explained by that alone. No pooling or re-analysis was performed. Figures 1 to 4 and Tables 1 and 2 reproduce published values only. This is a narrative synthesis and was not registered as a systematic review.

Results

How common: A systematic review and meta-analysis of 21 observational studies reported pooled prevalence in post-acute sequelae of COVID-19 of 70.6 per cent for orthostatic intolerance, 36.2 per cent for postural orthostatic tachycardia syndrome and 18.6 per cent for orthostatic hypotension (Figure 1) [12]. Prevalence of the syndrome and of hypotension was higher after mild than after moderate or severe infection, advancing age was negatively associated with both, and publication bias was evident for the syndrome though minimal for hypotension [12].

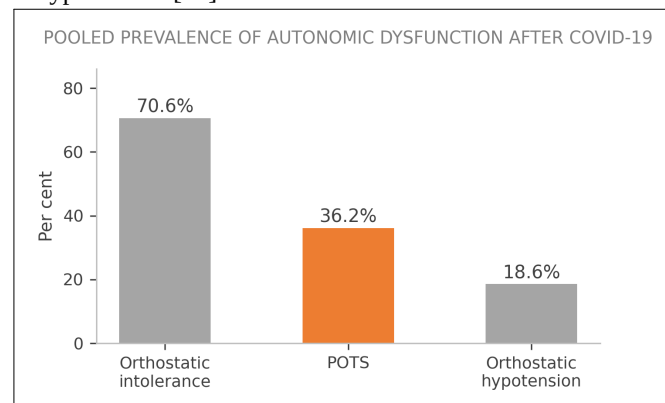


Figure 1: Pooled prevalence of autonomic dysfunction in post-acute sequelae of COVID-19 across 21 observational studies [12]

Why that figure needs care: A pooled prevalence of 36.2 per cent describes patients already identified as having post-acute sequelae, most of them recruited from specialist clinics or patient groups. It is not the risk after infection. A retrospective study of 841 hospitalised patients found autonomic dysfunction in 2.5 per cent [13], and cohorts assembled by systematic follow-up rather than referral report figures far below the pooled estimate [14,15]. Health-record analyses have compared risk after infection with risk after vaccination directly [31], while clinic-based series report high incidence by design [32]. The two numbers answer different questions and both are frequently quoted as though they answered the same one.

What a referral clinic sees: A dedicated autonomic service reported on 101 individuals newly referred after infection or vaccination. Following infection (n = 75), 22 (29 per cent) were diagnosed with the syndrome and 12 (16 per cent) with vasovagal syncope; following vaccination (n = 26), 11 (42 per cent) and 2 (8 per cent) respectively (Figure 2) [16]. The finding that matters most for practice is the one least quoted: in 49 of the 101 referrals (49 per cent), the diagnostic workup excluded any cardiovascular autonomic disorder (Figure 3) [16].

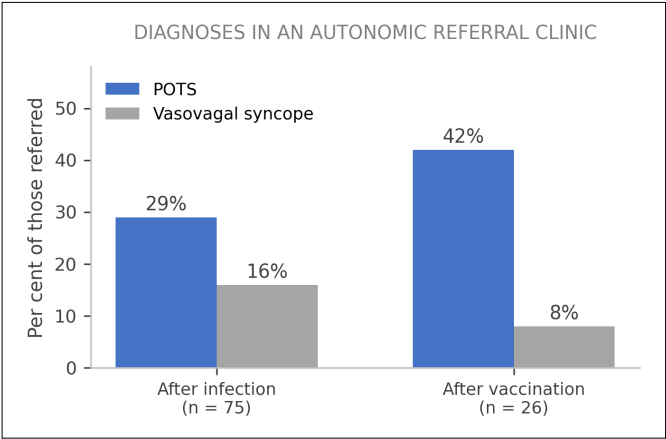


Figure 2: Diagnoses made in an autonomic referral clinic after infection and after vaccination [16]

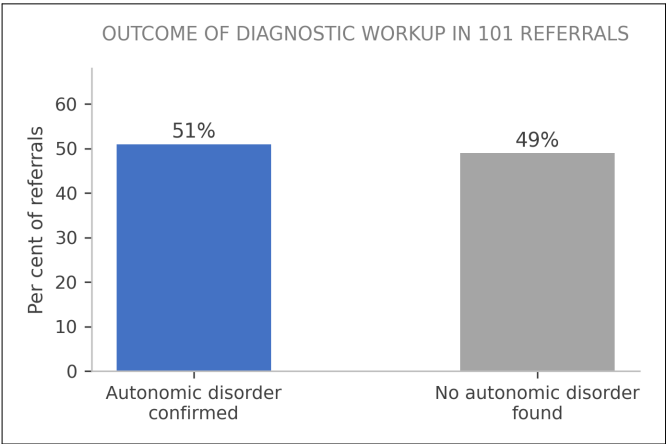


Figure 3: Proportion of referrals in whom diagnostic workup excluded any cardiovascular autonomic disorder [16]

Making the diagnosis: The criteria require a sustained heart rate increment of at least 30 beats per minute within ten minutes of standing or head-up tilt, at least 40 in those aged 12 to 19, in the absence of orthostatic hypotension, with symptoms for three months or more [1,2]. This matters because symptom report alone will not separate the syndrome from deconditioning, anxiety, anaemia or thyroid disease, all of which are commoner and some of which are treatable in other ways [3,17,18]. Reduced cerebral blood flow on standing has been documented in this population and overlaps with findings in myalgic encephalomyelitis [19,20]. Multi-disciplinary consensus guidance on assessing autonomic dysfunction in this population has since been published [29,30].

Study	Design and cohort	Principal reported finding
Meta-analysis of 21 studies [12]	Pooled prevalence in post-acute sequelae	OI 70.6%, POTS 36.2%, OH 18.6%; publication bias for POTS
Autonomic referral clinic [16]	101 newly referred individuals	POTS in 29% after infection, 42% after vaccination; 49% no disorder found
Hospitalised cohort [13]	841 patients admitted with COVID-19	Autonomic dysfunction in 2.5%
Stahlberg et al. 2021 [7]	Post-COVID tachycardia phenotype	Describes a distinct tachycardia syndrome after infection
Jamal et al. 2022 [14]	Prospective evaluation of autonomic function	Objective abnormalities in a defined post-acute cohort
Fedorowski 2019 [3]	Review of the syndrome before the pandemic	Diagnostic framework and aetiology, frequently post-viral
Vernino et al. 2021 [1]	NIH expert consensus statement	Defines the diagnostic criteria used throughout

Table 1: Principal studies reviewed, with the finding as reported by the original investigators

What helps: Management is largely non-pharmacological and was established before the pandemic: volume and salt loading, compression garments, avoidance of prolonged standing, and a structured, initially recumbent exercise programme [1,2,21]. Pharmacotherapy, most commonly beta blockade, ivabradine, midodrine or fludrocortisone, is added when non-pharmacological measures are insufficient [1,22,23]. In the referral cohort described above, non-pharmacological measures were recommended to all newly diagnosed patients and roughly one third additionally received drug treatment; among the 42 with follow-up data, 26 (62 per cent) had improved symptomatically (Figure 4) [16].

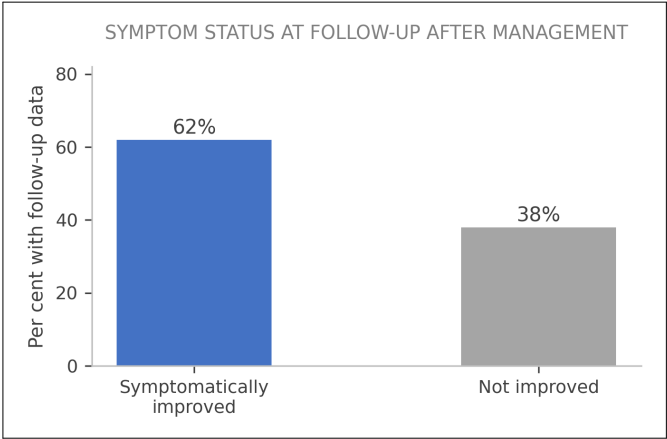


Figure 4: Symptomatic status at follow-up among patients diagnosed and managed in a referral clinic [16]

The athlete: Return-to-activity decisions in this group differ from the general post-COVID pathway. Sports cardiology guidance developed during the pandemic focused primarily on excluding myocarditis before resumption of training, and that assessment remains the first step where there were cardiopulmonary symptoms during the acute illness [9,10,24]. Once myocarditis is excluded and the syndrome is confirmed, the logic inverts: prolonged rest worsens orthostatic tolerance,

and the recommended approach is graded reconditioning beginning in recumbent or seated positions, rowing, recumbent cycling and swimming, progressing to upright work as tolerance allows [1,21,25]. Deconditioning is both a consequence of the syndrome and a contributor to it, which is why the distinction from simple detraining is often less clean in practice than in the criteria [17,26]. Reviews of the syndrome as a sequela of COVID-19 reach similar conclusions about mechanism and management [33,34,35].

Question	What the evidence supports	Caution
How common after infection?	Depends entirely on how the cohort was assembled	36.2% pooled vs 2.5% in hospitalised screening [12,13]
Is symptom report enough?	No; requires tilt or standing test	49% of referrals had no autonomic disorder [1,16]
First step in an athlete	Exclude myocarditis where indicated	Sports cardiology guidance takes precedence [9,10]
Does rest help?	No; prolonged rest worsens orthostatic tolerance	Opposite of general convalescent advice [1,21]
Exercise prescription	Graded, initially recumbent reconditioning	Evidence largely pre-pandemic and uncontrolled [21,25]
Prognosis	Most improve with management	62% improved, in a followed subgroup only [16]

Table 2: Practical questions in this presentation and what the evidence currently supports

Discussion

The most useful thing this literature offers is not a prevalence figure but a warning about prevalence figures. A pooled estimate of 36.2 per cent and a screened estimate of 2.5 per cent are both published, both defensible, and describe different populations [12,13]. The first counts people already identified as having persistent symptoms; the second counts everyone admitted. Quoting either without its denominator produces a misleading impression, in opposite directions.

The second useful finding is that half of the patients referred to a specialist autonomic service with suspected dysautonomia after COVID-19 turned out not to have one [16]. That is not a reason to dismiss the symptoms, which were real enough to prompt referral. It is a reason to do the test. A syndrome diagnosed on symptom pattern alone, in a population where symptoms are near-universal and specific findings are not, will be substantially over-diagnosed, and over-diagnosis carries its own harms: unnecessary drug treatment, an identity built around a condition, and the real diagnosis missed.

For the athlete specifically, the clinically important point is the inversion of standard advice. Post-viral convalescence usually means rest and gradual return. In confirmed postural orthostatic tachycardia syndrome, prolonged rest worsens the physiology, and the recommended approach is structured reconditioning in positions that avoid orthostatic stress [1,21]. Getting the sequence wrong, resting a patient with the syndrome or exercising a patient with myocarditis, produces harm in both

directions, which is why the order of assessment matters more than any individual intervention.

The evidence for management deserves an honest label. Most of it predates the pandemic, is uncontrolled, and consists of small series and consensus opinion [1,21,22]. The 62 per cent symptomatic improvement figure comes from 42 patients with follow-up data in a single clinic, without a comparison group, over a period in which spontaneous improvement is common [16]. It supports optimism in the conversation with a patient. It does not establish that the management caused the improvement.

Study limitations

The limitations belong to the underlying literature. Case ascertainment differs fundamentally between studies, and the pooled prevalence estimates carry high heterogeneity and demonstrated publication bias [12]. Referral cohorts describe who was referred, not who has the condition [16]. Few studies include pre-infection autonomic assessment, so causation cannot be separated from unmasking of pre-existing tendency [3,14]. Control groups are usually absent, in a condition where spontaneous improvement is common. Management evidence is largely pre-pandemic, uncontrolled and extrapolated [21,22,23]. Almost nothing addresses athletes specifically, so the return-to-activity recommendations here are inferred from general guidance and sports cardiology consensus rather than tested in this population [9,10,25]. And the literature is dominated by high-income settings and by the earlier variants of the virus [12].

Conclusion

A young athlete with persistent exercise intolerance after SARS-CoV-2 infection needs myocarditis excluded first and then a standing or tilt test, because half of those referred with suspected dysautonomia after COVID-19 do not have it, and because the management for what they do have may be the reverse of what they have been told. Where the syndrome is confirmed, the treatment is unglamorous and mostly not pharmacological, the evidence for it is weak but the direction consistent, and most patients improve. The figures for how often this follows infection range from 2.5 to 36 per cent depending on who was counted, and anyone citing one of those numbers should say which population it came from.

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Conflict of Interest

The authors declare no competing interests.

Ethics Statement

This article reviews previously published studies only. No new patient data were collected and no participants were recruited, so ethical approval was not required.

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