Original research

Non-COVID-19 cardiovascular pathology from return-to-play screening in college athletes after COVID-19

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ABSTRACT

Objective Concerns for cardiac involvement after SARS-CoV-2 infection led to widespread cardiac testing in athletes. We examined incidental non-COVID-19 cardiovascular pathology in college athletes undergoing postinfection return-to-play screening.

Methods The Outcomes Registry for Cardiac Conditions in Athletes was a nationwide prospective multicentre observational cohort study that captured testing and outcomes data from 45 institutions (September 2020–June 2021). Athletes with an ECG and transthoracic echocardiogram (TTE) and no pre-existing conditions were included. Findings were defined as major (associated with sudden cardiac death or requiring intervention), minor (warrants surveillance), incidental (no follow-up needed) or uncertain significance (abnormal with subsequent normal testing).

Results Athletes with both ECG and TTE (n=2900, mean age 20±1, 32% female, 27% black) were included. 35 (1.2%) had ECG abnormalities. Of these, 2 (5.7%) had TTE abnormalities indicating cardiomyopathy (hypertrophic-1, dilated-1), and 1 with normal TTE had atrial fibrillation. Of 2865 (98.8%) athletes with a normal ECG, 54 (1.9%) had TTE abnormalities: 3 (5.6%) with aortic root dilatation ≥40 mm, 15 (27.8%) with minor abnormalities, 25 (46.3%) with incidental findings and 11 (20.4%) with findings of uncertain significance. Overall, 6 (0.2%) athletes had major conditions; however, coronary anatomy and aortic dimensions were inconsistently reported and pathology may have been missed.

Conclusion Major non-COVID-19 cardiovascular pathology was identified in 1/500 college athletes undergoing return-to-play screening. In athletes without ECG abnormalities, TTE's added value was limited to pathological aortic root dilatation in 1/1000 athletes and minor abnormalities warranting surveillance in 1/160 athletes. Two-thirds of findings were incidental or of uncertain significance.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Preparticipation screening in competitive athletes using history and physical examination and/or ECG is used to identify conditions associated with sudden cardiac arrest or death; however, the utility of transthoracic echocardiogram (TTE) for cardiac screening in athletes remains uncertain.

⇒ Return-to-play cardiac screening after SARS-CoV-2 infection in college athletes provides an opportunity to examine incidental non-COVID-19 cardiovascular pathology and to evaluate the incremental value of a screening TTE.

WHAT THIS STUDY ADDS

⇒ In the absence of ECG abnormalities, the added value of TTE is limited to pathological aortic root dilatation in approximately 1 in 1000 athletes and minor structural findings in 1 in 160 athletes.

⇒ The proportion of incidental and false-positive findings on TTE screening is high, and coronary artery anatomy and ascending aorta size are inconsistently evaluated.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Given the resources needed to perform TTE, and the high proportion of incidental and false-positive findings, the added value of TTE to ECG screening in college athletes is limited.

⇒ Future study will benefit from more consistent evaluation of the coronary artery anatomy and ascending aorta dimensions.

BACKGROUND

Early in the COVID-19 pandemic, concern for cardiac involvement after SARS-CoV-2 infection led to widespread return-to-play cardiac screening in competitive athletes, most commonly using serum troponin, 12-lead ECG and two-dimensional transthoracic echocardiogram (TTE).[1 2] This screening was performed in addition to standard preseason preparticipation screening (PPS) using personal/family history and physical examination (H&P), with or without ECG, in an attempt to identify competitive athletes with conditions associated with increased risk of sudden cardiac death (SCD).[3–7] While TTE is sometimes employed during PPS to identify structural pathology not detected by H&P and/or ECG, its role as an adjunct to H&P and ECG remains uncertain.[8–16]

The Outcomes Registry for Cardiac Conditions in Athletes (ORCCA) study was designed to determine the prevalence and clinical outcomes of SARS-CoV-2 cardiac involvement in competitive college athletes.[17 18] However, the data obtained

through return-to-play cardiac screening after SARS-CoV-2 infection also provide an opportunity to examine the detection of non-COVID-19 cardiovascular pathology and the diagnostic performance of TTE during PPS. The purpose of this study is to characterise non-COVID-19 cardiovascular pathology in college athletes undergoing return-to-play cardiac screening after SARS-CoV-2 infection and to examine the incremental value of TTE when added to ECG during PPS.

METHODS

This was a secondary analysis of a nationwide prospective multicentre observational cohort study. College athletes included in this study had undergone return-to-play cardiac screening after a confirmed SARS-CoV-2 infection with both ECG and TTE. Athletes with a known cardiovascular diagnosis and those diagnosed with possible, probable or definite myocarditis and/or pericarditis on cardiac MRI were excluded. Study enrolment procedures and definitions have been previously described.[17]

Data collection was performed between September 2020 and June 2021. Participating institutions submitted de-identifed data via a standardised data capture tool, which captured demographic information and cardiovascular testing results. Copies of de-identified ECGs and clinical reports of other testing were also requested for each athlete. Each institution maintained a link to patient identifiers which was not provided nor accessible to the study investigators.

Abnormal ECG and/or TTE findings were characterised by severity, as adjudicated by an investigator panel of cardiologists and sports medicine physicians with expertise in sudden cardiac arrest (SCA) or death in athletes: 'major'—clinically actionable disorders associated with an increased risk of SCA/SCD, or requiring intervention[19]; 'minor'—not associated with SCA/SCD but warranting continued surveillance; 'incidental'—not associated with SCA/SCD and not warranting surveillance or 'uncertain significance'—abnormal TTE report with subsequent normal follow-up testing, suggesting a false-positive initial interpretation or transient abnormality (figure 1). Standard descriptive statistics were used to describe the study population; continuous variables were summarised via means and SD and categorical variables were described with frequencies and percentages. The prevalence estimates for cardiovascular pathology findings are reported as percentages using the total number of athletes identified with the condition, divided by the study population.

Patient and public involvement

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

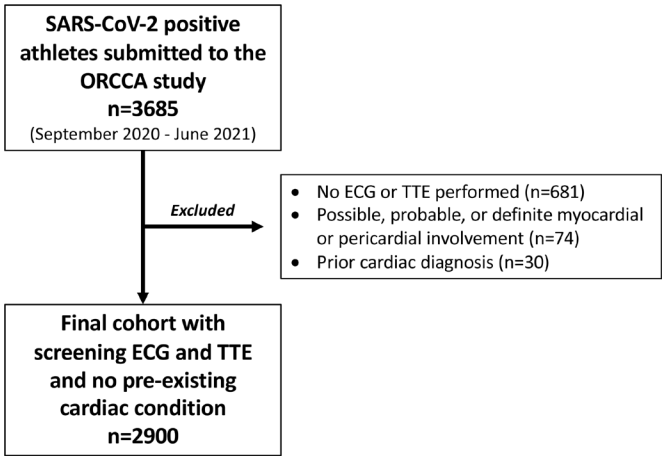

Figure 2 Study design and selection of athlete cohort. ORCCA, Outcomes Registry for Cardiac Conditions in Athletes; TTE, transthoracic echocardiogram.

RESULTS

Study population

Forty-five participating institutions submitted data regarding 3685 athletes to the ORCCA registry during the study period, including 3004 athletes who had undergone both ECG and TTE screening. One-hundred four athletes (3%) were excluded from this analysis: 30 with pre-existing cardiovascular diagnoses, and 74 with possible, probable or definitive myocardial and/or pericardial SARS-CoV2 involvement (figure 2). Table 1 provides baseline demographic characteristics of the final cohort, comprising 2900 athletes (mean age 20±1, 32% female, 64% white, 27% black). Athletes participated in 26 different sports. The most commonly represented sports were football (n=1010, 34.8%), baseball (n=272, 9.4%) and track and field (n=218, 7.5%).

Structural and non-structural cardiovascular findings

Structural and non-structural findings are summarised in figure 3. Thirty-five (1.2%) athletes had an abnormal ECG. Three athletes had major findings: paroxysmal atrial fibrillation requiring cardioversion (n=1); apical-variant hypertrophic cardiomyopathy (n=1) and suspected cardiomyopathy characterised by left ventricular dilation and non-compaction (n=1). The remaining 32 athletes with an abnormal ECG had no structural or functional findings on TTE, thus representing a false-positive ECG result (1.1%).

Among athletes with a normal ECG, major/clinically actionable findings were confined to three individuals with aortic root

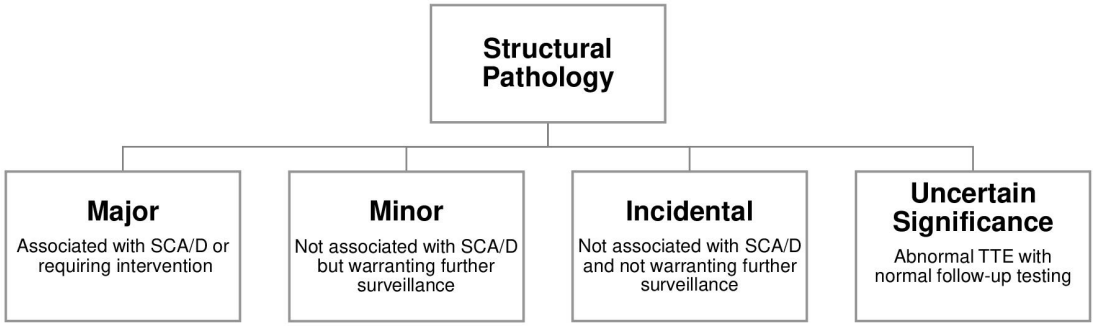

Figure 1 Classification of echocardiographic findings by severity. SCA/D, sudden cardiac arrest or death; TTE, transthoracic echocardiogram.

Table 1 Demographics

Patient characteristics*	Total cohort (n=2900) N (%)
Female	925 (32)
Age (years), mean (SD)	20 (1)
BMI (kg/m^2), mean (SD)	
Male	27 (5)
Female	23 (3)
White non-Hispanic	1864 (64)
Black	783 (27)
White Hispanic	82 (3)
Mixed	63 (2)
Asian	32 (1)
Pacific Islander	15 (0.5)
American Indian	7 (0.2)
Unknown race	54 (2)
Non-cardiac pre-existing conditions	
Sickle cell trait	35 (1)
Diabetes	11 (0.4)
Hypertension	11 (0.4)
Hyperlipidaemia	6 (0.2)
Asthma (mild-intermittent)	147 (5)
Asthma (mild-persistent or greater)	65 (2)
Immunosuppressive agent	4 (0.1)
Cardiovascular testing performed	
ECG+TTE	2599 (90)
ECG+TTE+CMR	301 (10)

*Partial data were available for the following characteristics: age (n=2887), sex one patient responded as non-binary (n=2899), BMI (n=2623), pre-existing conditions (n=2732).
BMI, body mass index; CMR, cardiac magnetic resonance; TTE, transthoracic echocardiogram.

dilatation ≥40 mm (table 2). Fifteen (0.5%) athletes exhibited minor cardiovascular findings, most commonly bicuspid aortic valve without haemodynamically significant regurgitation or stenosis and borderline aortic enlargement <40 mm (table 3). Twenty-five (0.8%) athletes had incidental findings including patent foramen ovale (PFO), PFO versus atrial septal defect (ASD) and reduced global longitudinal strain (table 4). Eleven (0.4%) athletes had findings of uncertain significance, with abnormal initial findings on TTE and normal follow-up testing suggesting an initial false-positive finding or the presence of transient abnormalities (table 5).

Additional cardiovascular testing and follow-up

Twenty-four athletes received additional cardiovascular testing following screening ECG and TTE, including 23 with abnormal TTE findings (online supplemental table S1). Cardiovascular magnetic resonance (CMR) was performed in 21 athletes, and stress testing (including cardiopulmonary exercise testing and stress echocardiography) and outpatient cardiac event monitoring were conducted in three athletes each. In nine athletes, follow-up testing confirmed TTE findings. CT angiography found one new incidental diagnosis of a bovine aortic arch. The remainder of follow-up testing did not reveal any significant structural or electrical pathology.

Two athletes were restricted from competition. One athlete with dilated cardiomyopathy and suspected left ventricular non-compaction was restricted with results of genetic testing for cardiomyopathy pending at time of data acquisition. Another athlete with mild left ventricular dilation and an ejection fraction of 47% was restricted, with CMR results interpreted as non-ischaemic cardiomyopathy versus normal physiological adaptation; these findings were subsequently classified as being of uncertain significance.

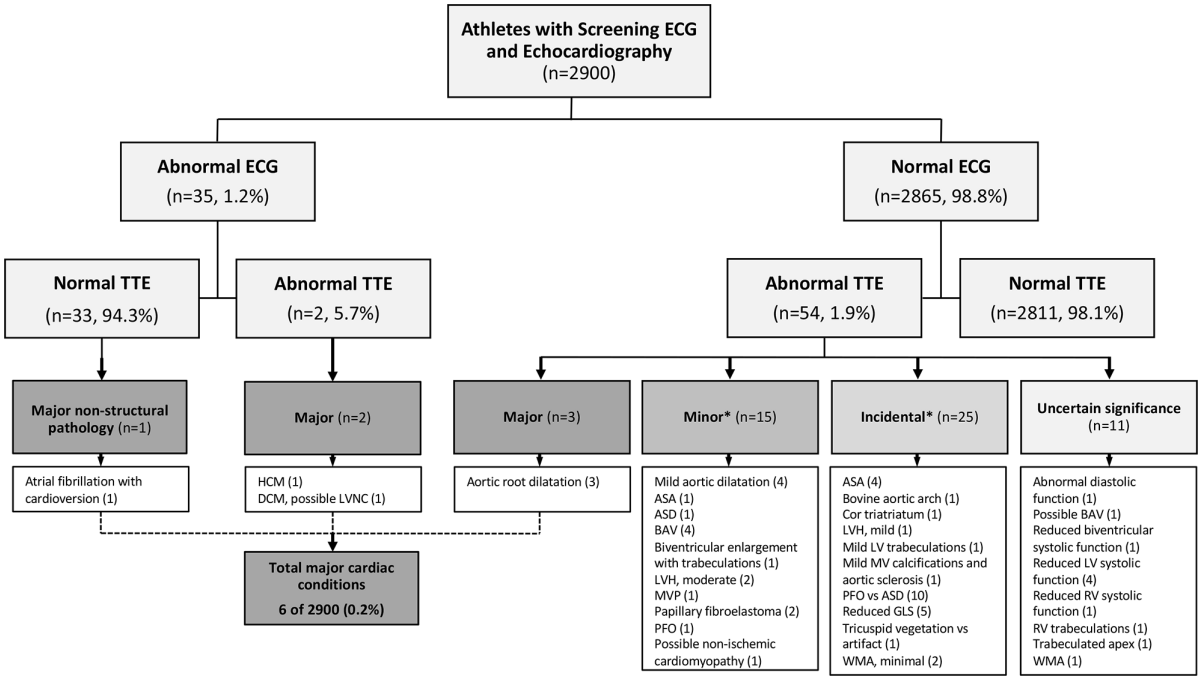

Figure 3 Summary of structural and non-structural findings in patients undergoing ECG and TTE screening. ASA, atrial septal aneurysm; ASD, atrial septal defect; BAV, bicuspid aortic valve; DCM, dilated cardiomyopathy; GLS, global longitudinal strain; HCM, hypertrophic cardiomyopathy; LV, left ventricle; LVH, left ventricular hypertrophy; LVNC, left ventricular non-compaction; MV, mitral valve; MVP, mitral valve prolapse; PFO, patent foramen ovale; RV, right ventricle; TTE, transthoracic echocardiogram; WMA, wall motion abnormalities. *Some athletes had more than one minor or incidental finding.

Table 2 Clinical characteristics for athletes with major cardiovascular diagnoses

Case	Age (years)	Sex	ECG	TTE	Additional testing	Final diagnosis
1	21	M	TWI V3-V6, LVH	Moderate LVH	► CMR: apical HCM ► 48-hour Holter monitor: normal ► Exercise stress test: normal	HCM
2	23	M	Normal	Dilated aortic root (42 mm, z-score 3.0)	CMR: normal	Aortic root dilatation
3	24	M	LBBB	Moderate LV dilation, LVEF 25%–30%, apical and inferoseptal wall dyskinesia	► CMR: severe LV enlargement, LVEF 28%, thinning of compacted myocardium apically ► Restricted from sport undergoing genetic cardiomyopathy workup	Dilated cardiomyopathy, possible LVNC
4	19	M	Normal	Dilated aortic root (40 mm, z-score 2.8)	–	Aortic root dilatation
5	20	M	Atrial fibrillation	Normal	► CMR: normal ► Cardioverted in emergency department	Atrial fibrillation
6	22	M	Normal	Dilated aortic root (40 mm, z-score 2.7)	–	Aortic root dilatation

CMR, cardiac magnetic resonance; F, female; HCM, hypertrophic cardiomyopathy; LBBB, left bundle branch block; LV, left ventricle; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; LVNC, left ventricular non-compaction; M, male; TTE, transthoracic echocardiography; TWI, T wave inversions.

Among other athletes diagnosed with major cardiovascular diagnoses, the athlete with HCM returned to play and one athlete underwent cardioversion for paroxysmal atrial fibrillation. No restrictions were placed on athletes with aortic root dilatation and no other cardiovascular interventions were conducted during the study period.

DISCUSSION

In a cohort of 2900 college athletes without known cardiovascular pathology who underwent ECG and TTE screening for return to play, 57 (2.0%) had electrocardiographic or echocardiographic abnormalities, including 5 (0.2%) with major structural abnormalities associated with an elevated risk of SCA/SCD and 15 (0.5%) with conditions warranting further surveillance. Of 54 athletes with normal ECG, the added value of TTE was limited to the identification of presumably pathological but mild aortic root dilatation in three (0.1%) athletes.

The low prevalence of structural pathology in this cohort is consistent with prior studies using preparticipation echocardiography at American universities. Screening TTE in 964 athletes at

Table 3 Clinical characteristics for athletes with minor cardiovascular diagnoses

Case	Age (years)	Sex	ECG	TTE	Additional testing	Final diagnosis
1	20	M	Normal	Mild hypokinesis, LVEF 45%	CMR: global hypocontractility, LVEF 44%	Possible non-ischaemic cardiomyopathy
2	21	F	Normal	Papillary fibroelastoma	–	Papillary fibroelastoma
3	21	M	Normal	Mildly dilated aortic root, possible BAV	–	Mild aortic root dilatation, possible BAV
4	21	M	Normal	Papillary fibro-elastoma, ASA, possible PFO	–	Papillary fibroelastoma, ASA, possible PFO
5	18	M	Normal	LVH (IVS 15 mm)	–	LVH, moderate
6	22	M	Normal	Increased LVWT	–	Increased LVWT
7	17	M	Normal	BAV	–	BAV
8	21	M	Normal	BAV	–	BAV
9	19	F	Normal	ASD	–	ASD
10	19	M	Normal	BAV	Pulmonary function testing: normal	BAV
11	17	M	Normal	Dilated aortic root (39 mm, z-score 1.3)	CMR: normal	Mild aortic root dilatation
12	20	M	Normal	Mild aortic dilatation	CMR: normal	Mild aortic dilatation
13	17	M	Normal	BiV enlargement, prominent trabeculations	► CMR: severe BiV enlargement, prominent trabeculations ► No sport restrictions, continued follow-up every 6 months	BiV enlargement with prominent trabeculations
14	22	M	Normal	MVP	–	MVP
15	21	M	Normal	Dilated ascending aorta (39 mm, z-score 3.2)	► CMR: normal ► CTA chest: normal, maximal thoracic aortic diameter 36 mm (z-score 2.9)	Mild ascending aorta dilatation

ASA, atrial septal aneurysm; ASD, atrial septal defect; BAV, bicuspid aortic valve; BiV, biventricular; CMR, cardiac magnetic resonance; CTA, CT angiography; F, female; IVS, intraventricular septum; LV, left ventricle; LVEF, left ventricular ejection fraction; LVWT, left ventricular wall thickness; M, male; MVP, mitral valve prolapse; PFO, patent foramen ovale; TTE, transthoracic echocardiography.

Table 4 Clinical characteristics for athletes with incidental cardiovascular diagnoses

Case	Age (years)	Sex	ECG	TTE	Additional testing	Final diagnosis
1	19	F	Normal	ASA	–	ASA
2	22	M	Normal	Cor triatriatum	–	Cor triatriatum
3	22	F	Normal	PFO	–	PFO
4	18	F	Normal	Possible PFO	–	Possible PFO
5	19	F	Normal	Possible PFO	–	Possible PFO
6	21	F	Normal	Possible PFO	–	Possible PFO
7	20	M	Normal	Mild MV calcifications, aortic sclerosis without stenosis	–	Mild MV calcifications, aortic sclerosis
8	20	M	Normal	Inferoapical LV trabeculation, mild global hypokinesis	–	Possible limited LVNC, LV trabeculation
9	20	M	Normal	Mild concentric LVH (IVS 13 mm)	–	LVH, mild
10	19	M	Normal	PFO versus ASD	–	PFO versus ASD
11	18	F	Normal	PFO versus ASD	–	PFO versus ASD
12	19	F	Normal	ASA	CMR: moderate-to-severe BiV enlargement	ASA
13	20	F	Normal	ASA	CMR: normal	ASA
14	19	M	Normal	Aortic arch outpouching	CTA chest: bovine aortic arch	Bovine aortic arch
15	19	F	Normal	PFO, ASA	–	PFO, ASA
16	19	F	Normal	PFO	–	PFO
17	21	M	Normal	PFO	–	PFO
18	22	M	Normal	PFO	–	PFO
19	21	F	Normal	Tricuspid vegetation versus artefact	TEE recommended, not done	Tricuspid vegetation versus artefact
20	19	M	Normal	GLS 14%	–	Normal/Athlete's heart
21	19	M	Normal	GLS 13%	–	Normal/Athlete's heart
22	20	M	Normal	Mildly reduced GLS	–	Normal/Athlete's heart
23	19	M	Normal	Mildly reduced GLS	–	Normal/Athlete's heart
24	21	F	Normal	GLS 15.7%; minimal basal inferior/inferoseptal WMA	–	Normal/Athlete's heart
25	22	M	Normal	Septal hypokinesis	–	Normal/Athlete's heart

ASA, atrial septal aneurysm; ASD, atrial septal defect; BiV, biventricular; CMR, cardiac magnetic resonance; CTA, CT angiography; F, female; GLS, global longitudinal strain; IVS, intraventricular septum; LV, left ventricle; LVEF, left ventricular ejection fraction; LVNC, left ventricular non-compaction; M, male; MV, mitral valve; PFO, patent foramen ovale; TEE, trans-oesophageal echocardiography; TTE, transthoracic echocardiography; WMA, wall motion abnormalities.

Table 5 Clinical characteristics for athletes with findings of uncertain significance

Case	Age (years)	Sex	ECG	TTE	Additional testing	Final diagnosis
1	20	F	Normal	RV trabeculation	CMR: normal	RV trabeculation
2	18	M	Normal	Trabeculated apex	Repeat TTE: mild BiV enlargement, partial scarring of papillary muscles	Normal/Athlete's heart
3	19	M	Normal	Abnormal diastolic function	CMR: normal	Normal/Athlete's heart
4	18	M	Normal	Possible BAV	CMR: normal	Normal/Athlete's heart
5	20	M	Normal	LVEF 48%; moderately reduced RV function, FAC 24.2%	► CMR: LVEF 48%, RVEF 42%, global hypokinesis ► Stress VO_2: normal ► Stress echo: mildly reduced BiV function ► Ziopatch: normal ► Cardiopulmonary stress test: normal	Normal/Athlete's heart
6	20	M	Normal	LVEF 45%–50%	CMR: normal	Normal/Athlete's heart
7	21	M	Normal	LVEF 46%	► CMR: reduced LVEF ► Repeat TTE: normal	Normal/Athlete's heart
8	21	F	Normal	Septal WMA	CMR: normal	Normal/Athlete's heart
9	19	M	Normal	Abnormal LV mid-septal motion; moderate LA dilation	► CMR: mild global hypokinesis ► 24-hour Holter: rare PACs and ventricular ectopic beats ► Stress echo: normal	Normal/Athlete's heart
10	19	M	Normal	Mildly reduced RV systolic function	CMR: normal	Normal/Athlete's heart
11	20	M	Normal	LVEF 47%, mildly increased LV size	► Restricted from sport pending CMR ► CMR: LVEF 48%, moderately enlarged LV	Non-ischaemic cardiomyopathy versus athlete's heart

BAV, bicuspid aortic valve; BiV, biventricular; CMR, cardiac magnetic resonance; CTA, CT angiography; F, female; FAC, fractional area change; LA, left atrium; LV, left ventricle; LVEF, left ventricular ejection fraction; M, male; PAC, premature atrial contraction; RV, right ventricle; RVEF, right ventricular ejection fraction; TEE, trans-oesophageal echocardiography; TTE, transthoracic echocardiography; WMA, wall motion abnormalities.

the University of Kansas found only 8 (0.8%) with major structural abnormalities[11]; a pilot study of a limited '5 min' TTE at the University of Wisconsin noted minor abnormalities in 5 of 395 (1.3%) athletes screened,[16] and a later retrospective study there reported 49 of 2898 (1.7%) athletes undergoing TTE had findings requiring follow-up and 3 (0.1%) had major findings meriting exclusion from competition.[13] Similar echocardiographic findings are also present in large cohorts of preadolescent and adolescent soccer players in the USA and Europe (1.8%–4.5% minor findings, 0.1%–0.3% major findings),[8 9 12 15] as well as professional athletes in Spain and Italy (0.4%–5.7% minor findings, 0.2–0.3% major findings).[10 14]

A key consideration to using TTE in during PPS among competitive athletes is its incremental value above the standard of H&P and/or ECG, which are relatively inexpensive and readily available compared with echocardiography.[20 21] In prior studies employing a preparticipation TTE, 15 of the 38 (39.5%) athletes excluded from competition had antecedent H&P or ECG findings.[8–16] Additionally, a prospective study of young competitive athletes with SCA/SCD by Peterson *et al* reported that 71% of aetiologies were potentially detectable on ECG, whereas approximately 20% (coronary artery anomalies, aortopathies, valvular disorders and congenital heart defects) would likely show TTE abnormalities in the setting of a normal ECG.[22]

Our findings suggest the added value of TTE to preparticipation ECG is low, particularly for the detection of cardiomyopathies. Both athletes in this cohort diagnosed with cardiomyopathies had an abnormal ECG. In athletes with a normal ECG, the added value of TTE was limited to pathological aortic root dilatation in 0.1% and minor structural abnormalities in 0.5% of athletes. While aortic dilatation has been associated with an elevated risk of SCD via aortic rupture or dissection,[12 20 21 23] outcomes data among athletes with aortic dilatation in the absence of an underlying genetic or hereditary aortopathy are limited. One small longitudinal study of athletes with isolated aortic dilatation suggests that clinically significant complications may be rare.[24–26] Indeed, aortic rupture and dissection represent only a small proportion (3%–5%) of SCD cases in US athletes.[19 22] Notably, all athletes with an enlarged aorta in our cohort continued sports participation after testing.

Importantly, the rate of incidental TTE findings and findings of uncertain significance in this cohort was high: 36 of 54 (67%) athletes with an abnormal TTE but normal ECG had clinically insignificant findings or normal follow-up testing, yielding a positive predictive value of only 35.7% for structural findings warranting surveillance. Additional testing prompted by TTE findings was also of limited diagnostic benefit: only 1 of 23 athletes who underwent follow-up testing had findings not already suggested by TTE.

Study limitations

A key limitation of this study was the inconsistent evaluation of coronary artery anatomy and ascending aorta size. As previously reported, only 10 of 56 (18%) echocardiography laboratories used by the 45 institutions providing data to the ORCCA study regularly commented on coronary anatomy,[27] and although aortic root size was commonly reported, only 15 of 45 institutions consistently measured ascending aorta size. As a result, it is possible that structural abnormalities in this cohort were missed and the incremental value of a screening TTE is actually higher than reported in this cohort. Anomalous origin of a coronary artery is among the top three reported causes of SCD in athletes, comprising up to 17% of cases, and is the most common cause of SCD/SCA not typically detectable on ECG.[21 28 29] Large cohorts have demonstrated that coronary artery origins are detectable in 96%–99% of cases using TTE,[13–16] and American College of Cardiology/American Heart Association guidelines state that any echocardiographic evaluation of athletes should attempt to characterise coronary anatomy.[30] Thus, inconsistent coronary evaluation represents a notable limitation in this study and gap in current TTE interpretation protocols.

Other limitations in this study include the reliance on imaging reports, rather than investigator review and adjudication of original images, for interpretation of TTE and CMR results. As such, inter-reader reliability of echocardiographic interpretation could not be performed. Similarly, all screening and diagnostic testing was performed and interpreted by the institutions submitting data to the ORCCA study, and the lack of a standardised interpretation and reporting protocol across all institutions could have impacted results. Additionally, the proportion of athletes in our cohort who had previously undergone PPS, and the results of that screening, were unknown. This may have impacted pathological findings in our athlete cohort, similar to Italian studies that found lower rates of echocardiographic findings in athletes who had previously undergone PPS.[14 15] Likewise, the population of athletes who participated in this registry, although large, may not be fully representative of a non-selected population of athletes. Finally, all diagnostic testing performed in this cohort occurred during return-to-play screening after SARS-CoV-2 infection, and therefore may not represent standard PPS protocols or timing.

CONCLUSIONS

Return-to-play cardiac screening after SARS-CoV-2 infection revealed major non-COVID-19 cardiac pathology in approximately 1 in 500 athletes. In the absence of ECG changes, however, the added value of TTE was limited to pathological aortic root dilatation in approximately 1 in 1000 athletes and minor structural findings warranting surveillance in 1 in 160 athletes. Two-thirds of TTE abnormalities had no clinical significance. Given the resources needed to perform TTE, and the high proportion of incidental and false-positive findings in this cohort, the incremental value of TTE as currently practised remains small. Further study, including standardised aorta and coronary artery evaluation, is necessary to better inform the potential role of screening TTE.

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Contributors CFK and JAD were involved in the conception, design, drafting, revision and final approval of the manuscript. BJP, NM, ALB, TWC, KGH, SAK and MRP were involved in the design, revision and final approval of the manuscript. All authors agreed to be accountable for the accuracy and integrity of all aspects of the work. JAD accepts full responsibility for the overall content and conduct of the study.

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Competing interests None declared.

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

Patient consent for publication Not applicable.

Ethics approval This study was approved by Massachusetts General Brigham Institutional Review Board (protocol #2020P002667). Participating institutions provided de-identified data to the ORCCA study, and each institution maintained their own link to patient identifiers which was not provided nor accessible to the study investigators. Individual consent was not required nor provided consistent with our approved Human Subjects protocol.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available on reasonable request. ORCCA data are available on reasonable request.

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Table S1. Further Testing and Interventions based on Echocardiographic and Electrocardiogram Findings

Testing/Intervention	Indication	Additional Findings
Cardiac Monitoring (n=3)	LVH (n=1) Reduced biventricular systolic function (n=1) Reduced LV systolic function (n=1)	
Cardioversion (n=1)	Atrial fibrillation (n=1)	
CMR (n=21)	Abnormal diastolic function (n=1) Ascending aorta dilatation (n=1) Aortic dilatation (n=1) Aortic root dilatation (n=2) Atrial fibrillation (n=1) ASA (n=2) BAV, possible (n=1) Biventricular enlargement and trabeculations (n=1) LV dilation with reduced EF (n=2) LVH (n=1) Reduced biventricular systolic function (n=1) Reduced LV systolic function (n=4) Reduced RV systolic function (n=1) RV trabeculation (n=1) Wall motion abnormalities (n=1)	Ascending aorta dilatation (n=1) Aortic root dilatation (n=2) Biventricular enlargement and trabeculations (n=1) Dilated cardiomyopathy (n=1) HCM (n=1) Possible LVNC (n=1) Possible nonischemic cardiomyopathy (n=1)
CTA Chest (n=2)	Aortic arch outpouching (n=1) Ascending aorta dilatation (n=1)	Ascending aorta dilatation (n=1) Bovine aortic arch (n=1)
Genetic Testing (n=1)	LV dilation with reduced EF (n=1)	Dilated cardiomyopathy (n=1)
Repeat TTE (n=2)	Reduced LV systolic function (n=1) Trabeculated apex (n=1)	
Restriction from Sport (n=2)	Dilated cardiomyopathy (n=1) LV dilation and reduced EF (n=1) LVH (n=1)	
Stress Testing (n=3)	Reduced biventricular systolic function (n=1) Reduced LV systolic function (n=1)	

ASA = atrial septal aneurysm, BAV = bicuspid aortic valve, CMR = cardiac magnetic resonance imaging, CTA = computed tomography angiography, EF = ejection fraction, HCM = hypertrophic cardiomyopathy, LV = left ventricle, LVH = left ventricular hypertrophy, LVNC = left ventricular noncompaction, RV = right ventricle, TTE = transthoracic echocardiography