AIM OR OBJECTIVE

The occurrence of cardiac arrest among endurance athletes is relatively high. A study investigating the amount of cardiac related deaths in triathletes from 1985-2016 found that cardiac related deaths are seen more frequently in middle-aged to older men, with 44% of these deaths resulting from underlying cardiovascular disease (Harris et al., 2018). The current hypothesis surrounding the prevalence of cardiac related deaths in endurance athletes involves noticeable cardiovascular changes from physical training. This study seeks to examine the reported causes of cardiac related deaths in U.S triathletes to identify a common pathophysiology that can inform preventative measures to reduce the incidence of sudden cardiac death in triathletes.

INTRODUCTION

• The American Heart Association (AHA) recommends 150 minutes of moderate exercise a week, due to the observed benefits on health including lower blood pressure, decreased cholesterol, and reduced risk for type 2 diabetes which are all known risk factors for cardiovascular disease.
• Observed physiologic changes that exercise has on patients with cardiovascular disease include reduced atherosclerosis in the vasculature, improved autonomic balance in the heart, and improved regeneration of myocardium.
• Although the impact of exercise is shown to have positive effects on cardiac health there has been a reported increase in incidence of adverse cardiac events among athletes, more prominently in endurance athletes.
  
Search Terms:
  
• Triathletes AND cardiovascular disease,
• Hypertrophy, ischemia, arrhythmia, sudden cardiac death

Identifying Search Terms and Databases

Search Terms:
• Triathletes AND cardiovascular disease,
• Hypertrophy, ischemia, arrhythmia, sudden cardiac death

Databases:
• Four Databases were searched using the above phrase above resulting in 227 results:
  
BioMed Central – 10 articles
MedLine Complete – 37 articles
PubMed – 115 articles
Scopus – 65 articles

Selection of Articles Reviewed

227 articles met initial search criteria
40 articles excluded as duplicates
187 unique articles
139 articles excluded for relevance
48 articles included
15 articles excluded on full text review
33 articles included in final review

METHODS

Establishing Inclusion and Exclusion Criteria

Inclusion criteria:
• Male
• Triathletes
• Diagnosed with CVD
• Ischemic changes
• Hypertrophic changes
• Arrhythmic changes
• Sudden Cardiac Death

Exclusion Criteria:
• Presence of autoimmune disease
• Positive family history of cardiac disease
• Known genetic cause of cardiovascular disease
• Female

ANALYSIS AND DISCUSSION

Cardiac hypertrophy
• Relative Blood Volume (rBV) at rest differentiates LVH in physiologic athlete’s heart and pathologic hypertrophic cardiomyopathy and hypertensive heart disease. rBV at rest below 0.114ml has a sensitivity of 93% and specificity of 100% for disease.
• Normal LV diastolic function differentiates physiologic LVH in athlete’s heart from pathologic LVH in valvular disease and hypertensive heart disease.
• LV mass >220g is associated a significant increase in BP values at anerobic threshold. Septal wall thickness >1.2cm was associated with increased BP.
• Presence of Premature Ventricular Contractions (PVC) at maximal exercise could be evidence of evolution to pathologic cardiomyopathy.

Fibrosis & Arrythmia
• Fibrotic changes seen were not associated with pathological changes leading to adverse cardiac events
• Supraventricular arrhythmias and AV nodal dysfunction were associated with right atrial dilation.
• Left ventricular hypertrophy was associated with higher risk for fatal ventricular arrhythmias such as VFib and VTach

Ischemia
• Significant coronary atherosclerosis was observed in several cases of SCD upon autopsy.
• The coronary artery calcium score was shown to be a more accurate predictor of CAD in asymptomatic triathletes.

Pulmonary Edema, Hypertension, Arterial Dilation
• Abnormal left ventricular diastolic compliance associated with increased risk of swimming induced pulmonary edema.
• Increase peak systolic blood pressure in response to exercise may result in myocardial injury causing LA dysfunction

REFERENCES

• Pathologic hypertrophy: Myocardial Contrast Echocardiogram (MCE), Echocardiogram with pulsed doppler tissue imaging, Maximal exercise cardiac stress test, and Electrocardiogram.
• Fibrosis: Cardiovascular Magnetic Resonance
• Arrythmia: Electrocardiogram, Holter monitor
• Ischemia: Coronary Calcium Score, single-photon emission tomography (SPECT)
• Swimming-Induced Pulmonary Edema: Autopsy
• Exercise-Induced Arterial Hypertension: Spiroergometry, Echocardiography
• Peripheral Arterial Dilation: Ultrasound

Recommendations for Prevention and Management

• For athletes with LVH and exercise induced arterial hypertension, treat with an ACE inhibitor or AT1 blocker before training or competition is recommended.
• For athletes with a coronary calcium score > 100 Angiostin units treatment with prophylactic Aspirin is recommended.
• For athletes with exercised induced SVT a heart rate monitor is recommended to detect periods of arrhythmia. This was shown to assist the athlete in adjusting the intensity of exercise.
• Other recommendations include, decreasing or terminating training and increased medical teams at triathlon events to decrease incidence of SCDS in triathletes.

CONCLUSION

• Several pathologic changes were identified including cardiac hypertrophy, ischemia, fibrosis, and hypertension, with associated increased risk for adverse cardiac events.
• Diagnostic tools varied based on pathologic changes identified.
• Current recommendations for prevention of adverse cardiac events in triathletes are prophylactic medical management, decreased intensity of training, and continuous monitoring.
• Further research assessing pathologic changes can provide evidence-based guidelines for preventative screening tools for triathletes to decrease incidence of cardiac related deaths.