QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

KRAJEWSKA, Natalia, and KALITA, Maciej. Anabolic-Androgenic Steroids and Myocardial Injury in Bodybuilders: A Narrative Review. Quality in Sport. 2026;76:75417. https://doi.org/10.12775/QS.2026.76.75417

ARTICLE TIMELINE
Received: 17.08.2026. Revised: 30.08.2026. Accepted: 13.09.2026. Published: 24.09.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Anabolic–Androgenic Steroids and Myocardial Injury in Bodybuilders: A Narrative Review

Natalia Krajewska
ORCID: 0009-0006-8360-1689
E-mail: nataliakrjwsk@gmail.com
University Clinical Centre of the Medical University of Warsaw, Central Teaching Hospital, Warsaw, Poland

Maciej Kalita
ORCID: 0009-0002-6636-0503
E-mail: kalita.maciej00@gmail.com
University Clinical Centre of the Medical University of Warsaw, Central Teaching Hospital, Warsaw, Poland

Corresponding author
Natalia Krajewska, ORCID: 0009-0006-8360-1689, e-mail: nataliakrjwsk@gmail.com

Abstract

Background. Non-medical anabolic–androgenic steroid (AAS) use is associated with several cardiovascular pathologies including dyslipidaemia, hypertension, erythrocytosis, endothelial dysfunction, thrombosis, vasospasm, and adverse cardiac remodelling. Often it is seen in bodybuilders, where abuse of AAS is prevelant. One of the life threatning consequence is myocardial infarction including myocardial infarction with non-obstructive coronary arteries (MINOCA).

Aim. To review the relationship between AAS exposure, MINOCA, and coronary microvascular dysfunction (CMD) in bodybuilders.

Material and methods. A targeted narrative search of PubMed/MEDLINE identified peer-reviewed studies including reviews, consensus documents and case reports concerning AAS, cardiovascular injury, MINOCA, and CMD.

Results. AAS may induce atherogenic, thrombotic, haemodynamic, vascular, and myocardial adverse effects. In athletes, MINOCA might clinically present itself as occult plaque disruption, transient thrombosis, epicardial or microvascular spasm, CMD, or oxygen supply–demand mismatch during severe hypertension and exertion.

Conclusions. AAS exposure should always be considered in young strength athletes with acute coronary syndrome. MINOCA requires mechanism-oriented investigation using non-invasive imaging, intracoronary imaging, coronary physiology, and vasoreactivity testing as appropriate. Current evidence supports biological plausibility but does not prove that AAS directly causes CMD.

Keywords: anabolic–androgenic steroids, bodybuilding, coronary microvascular dysfunction, myocardial infarction with non-obstructive coronary arteries, sports cardiology

1. Introduction

Bodybuilding is defined as a sport involving strenuous physical exercise to strengthen and enlarge the body's muscles. Unlike other sports, where performance is key, contestants are judged based on their physical appearance. To prepare for a competition, bodybuilders follow stringent nutritional and fluid regimens. The aim is to reduce subcutaneous water and fat and increase glycogen availability in muscles, improving muscle definition and aesthetic appearance and reducing the risk of bloating on the day of competition. Preparation typically begins 8 to 31 weeks before the competition (Rossow et al. 2013; Helms et al. 2014; Chappell et al. 2018; Bamman et al. 1993). Natural methods can be used to achieve these goals, but some athletes use diuretics, insulin, and anabolic–androgenic steroids (AAS) (Gentil et al. 2017; De Souza et al. 2018; Escalante et al. 2021; Kleiner et al. 1990). AAS misuse has been linked to a higher risk of coronary artery disease (Angell et al. 2012; Perry et al. 2020), lipid disorders, hypertension, cardiomyopathy, and myocardial infarction (Perry et al. 2020). Unfortunately, patients often do not disclose AAS use, making the prevalence of cardiovascular events difficult to estimate. There are also no randomised trials examining the association between AAS misuse and myocardial infarction; most evidence comes from case reports and observational studies.

The umbrella term – myocardial infarction without or with non-significant obstruction (MINOCA) covers a spectrum of conditions causing an ischemia of the myocardium without or with non-significant obstruction in coronary arteries. It requires identification of the underlying mechanism. Potential ischaemic mechanisms include plaque disruption, coronary thrombosis or embolism, spontaneous coronary artery dissection, epicardial spasm, CMD, and supply–demand mismatch; non-ischaemic mimics such as myocarditis and Takotsubo syndrome must also be excluded (Agewall et al. 2017; Kunadian et al. 2020). This review examines how the known cardiovascular effects of AAS may intersect with the MINOCA framework, focusing on bodybuilders and the still-uncertain role of CMD.

The objective was to provide clinicians in sports medicine, emergency medicine, and cardiology with a critical, mechanism-based synthesis and a practical diagnostic perspective. The central question was: How can AAS exposure contribute to myocardial injury when there is no obstructive coronary artery disease, especially in the bodybuilder population?

2. Material and methods

For this review a systematic and comprehensive search of established scientific databases, including PubMed, Scopus, Web of Science, and Google Scholar was conducted, The search focused on peer-reviewed literature. The literature search was done using a combination of keywords to identify relevant studies. Primary search terms included: “anabolic androgenic steroids”, “bodybuilding”, “myocardial infarction”, “MINOCA”, “coronary microvascular dysfunction”, “microvascular angina”, “vasospasm”, “endothelial dysfunction”, “hypertension”, “erythrocytosis”, and “angiography-derived index of microcirculatory resistance”.

Eligible sources included peer-reviewed studies, relevant to vascular biology, consensus or guideline documents, reviews, case studies and case reports. Titles, abstracts, and full texts were assessed for relevance to the topic of the narrative review. No quantitative pooling, formal risk-of-bias assessment, protocol registration, or duplicate independent screening was performed because this was a narrative review.

3. Influence of anabolic–androgenic steroids on the cardiovascular system

AAS may affect the cardiovascular system through several connected mechanisms. Their use has been associated with substantial reductions in high-density lipoprotein cholesterol, increase in low-density lipoprotein cholesterol, elevated blood pressure, erythrocytosis, enhanced platelet activity, and impaired endothelium-dependent vasodilatation (Hartgens and Kuipers 2004; Achar et al. 2010; Angell et al. 2012; Bond et al. 2022). AAS exposure may also contribute to left-ventricular hypertrophy, myocardial fibrosis, systolic and diastolic dysfunction, and arrhythmias (Achar et al. 2010; Bond et al. 2022; Iliakis et al. 2025). A large cohort study published in 2025 found that AAS users had higher risks of myocardial infarction, venous thromboembolism, arrhythmias, cardiomyopathy, and heart failure than matched controls (Windfeld-Mathiasen et al. 2025).

All can accelerate atherosclerosis, increase myocardial oxygen demand, reduce coronary flow reserve, and create conditions favouring coronary thrombosis or vasospasm. In bodybuilders, the cardiovascular effects of AAS may be intensified by strenuous resistance exercise, Valsalva manoeuvres, dehydration, diuretic use, and concomitant use of other performance-enhancing substances. AAS-associated myocardial injury is therefore unlikely to arise from a single pathway. It may reflect the interaction of metabolic, haemodynamic, thrombotic, vascular, and

direct myocardial effects, potentially leading to infarction even with the absence of obstructive epicardial coronary disease.

Table 1. Cardiovascular effects of AAS and their potential relevance to MINOCA

CARDIOVASCULAR EFFECT	PROPOSED MECHANISM	POSSIBLE MINOCA RELEVANCE	EVIDENCE
Dyslipidemia	Marked HDL reduction and variable LDL elevation	Premature atherosclerosis or occult plaque disruption	Moderate
Erythrocytosis and thrombosis	Increased viscosity and platelet thromboxane signalling	Transient coronary thrombosis or embolic presentation	Limited-moderate
Endothelial Dysfunction	Reduced nitric-oxide-mediated vasodilatation and increased vascular tone	Epicardial or microvascular spasm	Limited
Hypertension	Sodium retention, neurohormonal activation, and microvascular remodelling	Supply–demand mismatch and CMD	Moderate
Myocardial Remodelling	Hypertrophy, fibrosis, and impaired ventricular function	Reduced coronary reserve and increased arrhythmic vulnerability	Moderate

4. Review findings

Myocardial infarction reported previously in bodybuilders with abuse of anabolic steroids usually has one of three origins: atherogenic, thrombotic or vasospastic (Christou et al. 2016).

4.1. Atherogenic mechanisms

The atherogenic mechanism can be explained by the influence of AAS on athletes' lipid profiles. AAS reduce HDL cholesterol by 39–70%, which is believed to be an independent risk factor for cardiovascular disease (Hartgens and Kuipers 2004). The most pronounced suppression is observed in the HDL2 fraction, which is regarded as protective and is inversely associated with ischaemic heart disease (Sweetnam et al. 1994; Campos et al. 1995). This mechanism may explain extremely low HDL cholesterol in some AAS-using athletes. However, the absence of obstructive lesions during coronary angiography makes a fixed atherogenic obstruction less likely as the immediate cause of an infarction. It does not completely exclude plaque disruption or erosion that is not apparent on routine angiography.

4.2. Thrombotic mechanisms

The mechanism of the thrombosis under the influence of AAS remains uncertain. Several hypotheses may explain this relationship. First, stimulation of erythropoiesis by testosterone can elevate haematocrit and gradually increase blood viscosity, potentially increasing thrombus burden (Hajjar et al. 1997; Stergiopoulos et al. 2008). This effect may be intensified by fluid restriction or diuretic use during competition preparation.

Second, androgen-mediated platelet aggregation may be related to testosterone derivatives that increase thromboxane A2 signalling and human platelet thromboxane A2 receptor density (Ajayi et al. 1995). These effects have a prothrombotic potential. Nevertheless, the direct connection between AAS-induced platelet hyperaggregation and clinical thrombotic events remains incomplete.

A third theory concerns alterations in haemostatic and fibrinolytic systems. The available studies are limited and have yielded contradictory results (Vanberg and Atar 2010), making this pathway difficult to confirm. In individual patients haematocrit, haemoglobin, platelet count, angiographic evidence of thrombus, and previous thrombotic events should be assessed together. The absence of visible thrombus does not exclude transient thrombosis with spontaneous recanalisation, but it weakens this possible explanation when no other evidence supports it.

4.3. Coronary vasospasm and endothelial dysfunction

The third proposed mechanism of myocardial infarction in AAS users is coronary vasospasm. In a study of rabbits with hypercholesterolaemia, testosterone and nandrolone intensified vasoconstriction and impaired vascular relaxation (Ammar et al. 2004). Another study indicated that AAS use contributes to endothelial dysfunction. In comparison with athletes who did not use AAS, the vasodilatory response was reduced (Ebenbichler et al. 2001). Vascular reactivity was also impaired in a separate study, but the injury appeared at least partly reversible because discontinuation of AAS for three months was followed by improved vasoreactivity (Lane et al. 2006).

Vasospasm should therefore be considered as a cause of myocardial infarction in athletes without obstructive lesions on coronary angiography, after other possibilities have been excluded (Beijk et al. 2019). This conclusion must remain cautious because direct evidence concerning the influence of AAS on coronary-artery reactivity is limited. Peripheral endothelial dysfunction supports biological plausibility but is not equivalent to a positive coronary acetylcholine test.

4.4. MINOCA and coronary microvascular dysfunction

The umbrella term myocardial infarction with non-obstructive coronary arteries (MINOCA) covers a spectrum of ischaemic mechanisms occurring without a coronary stenosis of at least 50%. These include coronary spasm, spontaneous coronary artery dissection, coronary thromboembolism, plaque disruption, oxygen supply–demand mismatch, and coronary microvascular dysfunction (CMD) (Agewall et al. 2017; Kunadian et al. 2020; Khan et al. 2022). Non-ischaemic mimics, particularly myocarditis and Takotsubo syndrome, must be excluded before MINOCA is accepted as the final working diagnosis.

CMD is frequently identified in patients with non-obstructive coronary disease and is associated with adverse clinical outcomes (Lee et al. 2016; Boerhout et al. 2022). Its role in MINOCA remains insufficiently studied, partly because diagnostic possibilities have only recently expanded. Previously, assessment commonly required an invasive pressure and thermodilution wire with drug administration. Angiography-derived computer calculations are now available and can estimate microvascular resistance without passage of a pressure wire. Validation studies have reported promising diagnostic performance (Fan et al. 2023). These measurements may reduce procedural complexity, but they remain model-based estimates and must be interpreted in the clinical and haemodynamic context. Contemporary reviews also

emphasise combining anatomical, physiological, and vasoreactivity testing to define CMD endotypes and guide treatment (Pompei et al. 2024).

The diagnosis of CMD is based on complementary measurements. Fractional flow reserve (FFR) excludes functionally important epicardial obstruction, coronary flow reserve (CFR) assesses the capacity to augment coronary blood flow, and the index of microvascular resistance (IMR) estimates microvascular resistance. The European Association of Percutaneous Cardiovascular Interventions consensus describes an abnormal microvascular endotype using FFR above 0.80 with CFR below 2.0 and/or IMR of at least 25 (Kunadian et al. 2020). An acetylcholine test is recommended when a vasomotor disorder is suspected because it helps differentiate microvascular angina from microvascular or epicardial vasospastic angina (Kunadian et al. 2020).

Microvascular angina is a clinical manifestation of CMD. Two broad and potentially overlapping mechanisms are described: structural and functional dysfunction (Kunadian et al. 2020; Spione et al. 2022). Structural dysfunction results from remodelling of coronary arterioles, producing luminal narrowing, limited oxygen delivery, and increased vascular tone. The vessels become less responsive to vasodilators and may be prone to inflammatory injury and microinfarction (Kunadian et al. 2020; Spione et al. 2022). In this phenotype, CFR generally decreases and IMR increases (Boerhout et al. 2022). Functional dysfunction reflects dysregulation of vasoreactivity, particularly in medium and larger arterioles. Resting coronary flow may be inappropriately high despite ordinary oxygen demand, leaving limited capacity to increase flow during maximal vasodilatation (Boerhout et al. 2022; Spione et al. 2022).

4.5. Hypertension, oxygen supply–demand mismatch, and CMD

Myocardial oxygen supply–demand mismatch can be a cause of MINOCA and may result from a hypertensive crisis (Agewall et al. 2017). Arterial hypertension has been reported frequently among patients with MINOCA and is an established cardiovascular risk factor (Buller et al. 2021). It can cause remodelling and hypertrophy of the coronary microcirculation, narrowing the lumen and contributing to microvascular angina (Buller et al. 2021; Zdravkovic et al. 2023).

Hypertension also affects the myocardium itself. It contributes to left-ventricular hypertrophy and myocardial fibrosis, both of which are important substrates for CMD (Zdravkovic et al. 2023). In addition, hypertension promotes endothelial dysfunction by increasing endothelium-derived vasoconstrictors and impairing nitric-oxide synthesis, prostacyclin availability, and endothelium-derived hyperpolarising mechanisms. The result is increased microvascular

contractility and impaired coronary flow regulation (Zdravkovic et al. 2023). During a severe blood-pressure rise, especially together with intense resistance exercise, Valsalva manoeuvres, dehydration, or stimulant exposure, established microvascular changes may facilitate acute myocardial ischaemia.

The influence of AAS on hypertension remains debated. Some studies demonstrate an association, whereas others do not (Achar et al. 2010). Proposed mechanisms include renal sodium retention and vasoconstriction mediated by activation of the renin–angiotensin–aldosterone system, increased thromboxane A2 expression, norepinephrine synthesis, and endothelin-1 activity (Achar et al. 2010; Bond et al. 2022). Persistently elevated blood pressure has been observed for months after discontinuation of AAS in some studies (Lenders et al. 1988; Urhausen et al. 2004), suggesting that cardiovascular consequences may not resolve immediately.

5. Practical diagnostic approach

MINOCA should be regarded as a working diagnosis rather than a final explanation for myocardial infarction. When a bodybuilder with the history of AAS abuse presents with myocardial infarction and non-obstructive coronary arteries, coronary angiography should not be considered the end of the investigation. Once obstructive coronary disease has been excluded, further investigations should be directed at identifying the underlying mechanism. Cardiac magnetic resonance imaging is recommended to confirm an ischaemic pattern of myocardial injury and to exclude alternative diagnoses, particularly myocarditis and Takotsubo syndrome. Intracoronary imaging with optical coherence tomography or intravascular ultrasound may identify plaque disruption, coronary dissection or intramural haematoma that was not visible during routine angiography (Agewall et al. 2017; Kunadian et al. 2020).

If the patient continues to experience angina and CMD or coronary vasospasm is suspected, invasive coronary functional testing should be considered. This may include measurement of coronary flow reserve and microvascular resistance during adenosine administration, together with acetylcholine provocation testing for epicardial or microvascular spasm. Anti-anginal treatment may be started while the diagnostic assessment is being completed, but it should subsequently be adjusted to the identified mechanism. Beta-blockers or calcium-channel blockers may be used in patients with microvascular angina, depending on heart rate, blood pressure and comorbidities. When vasospasm is suspected or confirmed, calcium-channel blockers are generally preferred, and long-acting nitrates may be added if symptoms persist.

Treatment should also include strict blood-pressure and lipid control, management of other cardiovascular risk factors and discontinuation of AAS and other potentially harmful performance-enhancing substances (Kunadian et al. 2020; Pompei et al. 2024).

The literature supports AAS as a potential contributor to myocardial infarction in young athletes through atherogenic, thrombotic, vasospastic, hypertensive, and microvascular pathways. It does not yet establish AAS as a direct cause of CMD. Thorough investigation is essential because identifying the mechanism guides secondary prevention and prognosis. A confidential, non-judgmental history of AAS, diuretics, stimulants, and other performance-enhancing substances should be part of the evaluation of acute coronary syndrome in strength athletes.

6. Future directions

Future research should determine whether supraphysiological AAS use independently causes persistent coronary microvascular dysfunction. Prospective studies should compare current AAS users, former users, and strength-trained non-users. Exposure should be characterised by compound, dose, duration, cumulative exposure, and concomitant use of diuretics, stimulants, growth hormone, insulin, and other performance-enhancing substances.

Future studies should combine ambulatory blood-pressure monitoring, lipid and haematological assessment, echocardiography, cardiac magnetic resonance, coronary flow reserve, IMR, and acetylcholine provocation testing in patients with history of AAS abuse. Repeated assessment after AAS discontinuation could help distinguish reversible endothelial or vasomotor abnormalities from permanent structural microvascular remodelling (Pompei et al. 2024).

Angiography-derived IMR and quantitative flow ratio calculations may provide useful physiological information without a pressure wire (Fan et al. 2023). More evidence is warranted. As for now their results should be interpreted as supportive evidence rather than a complete diagnosis. A high resistance estimate cannot identify whether the abnormality is structural or functional, whether it preceded the infarction, or whether it was caused by AAS. Combining myocardial imaging, vessel-wall imaging, coronary flow assessment, and vasoreactivity testing provides a more defensible mechanism-based diagnosis.

Registries of young patients with MINOCA should systematically record strength-sport participation, competition-preparation practices, dehydration, and performance-enhancing drug use. Standardised, non-judgmental questionnaires and, when feasible, biochemical

exposure verification would reduce underreporting. Longitudinal studies are also needed to establish the incidence, prognosis, and recurrence risk of MINOCA among AAS users and to inform cardiovascular follow-up and return-to-training guidance.

7. Limitations of the evidence

Most evidence on cardiovascular injury in AAS users comes from case reports, small physiological studies, cross-sectional comparisons, and narrative reviews. Exposure is frequently self-reported, doses and products vary, and concomitant use of stimulants, diuretics, growth hormone, or other substances is common. Findings from therapeutic testosterone, peripheral arteries, or animal models cannot be transferred directly to supraphysiological AAS regimens. Because this narrative review did not use duplicate screening, formal quality appraisal, or quantitative pooling, it is vulnerable to selection bias and cannot quantify MINOCA risk or establish that AAS directly cause CMD. It supports biological plausibility and a mechanism-based diagnostic framework rather than a definitive causal claim.

8. Conclusions

Non-medical AAS exposure can generate a convergence of dyslipidaemia, hypertension, erythrocytosis, platelet activation, endothelial dysfunction, vasospasm, and adverse myocardial remodelling. In a bodybuilder with acute myocardial infarction and non-obstructive coronary arteries, these pathways make AAS exposure clinically relevant even when no epicardial culprit lesion is visible.

MINOCA should trigger a structured search for mechanism using cardiac magnetic resonance, intracoronary imaging, coronary physiology, and vasoreactivity testing as appropriate. CMD is a plausible component of AAS-associated myocardial injury, particularly in the presence of severe hypertension, but current evidence does not establish direct causation. Accurate exposure history, cautious interpretation of angiography-derived indices, and long-term risk-factor treatment are essential.

Disclosure

Supplementary Materials: None.

Author Contributions: Conceptualization, NK; methodology, NK nad MK; software, NK and MK; check, writing - rough preparation, NK and MK; writing - review and editing, NK and

MK; visualization, NK and MK; supervision, NK; project administration, Nk and MK. All authors have read and agreed with the published version of the manuscript.

Funding: None.

Institutional Review Board Statement: Not applicable, this article is a narrative review.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created or analysed in this study.

Acknowledgements: None.

Conflicts of Interest: None.

Use of Artificial Intelligence: A generative artificial-intelligence tool was used to assist with language refinement and manuscript organisation under human supervision. The author is responsible for literature selection, verification, interpretation, and the final content.

References

Achar, S., Rostamian, A., & Narayan, S. M. (2010). Cardiac and metabolic effects of anabolic-androgenic steroid abuse on lipids, blood pressure, left ventricular dimensions, and rhythm. American Journal of Cardiology, 106(6), 893–901. https://doi.org/10.1016/j.amjcard.2010.05.013

Agewall, S., Beltrame, J. F., Reynolds, H. R., et al. (2017). ESC working group position paper on myocardial infarction with non-obstructive coronary arteries. European Heart Journal, 38(3), 143–153. https://doi.org/10.1093/eurheartj/ehw149

Ajayi, A. A., Mathur, R., & Halushka, P. V. (1995). Testosterone increases human platelet thromboxane A2 receptor density and aggregation responses. Circulation, 91(11), 2742–2747. https://doi.org/10.1161/01.CIR.91.11.2742

Ammar, E. M., Said, S. A., & Hassan, M. S. (2004). Enhanced vasoconstriction and reduced vasorelaxation induced by testosterone and nandrolone in hypercholesterolemic rabbits. Pharmacological Research, 50(3), 253–259. https://doi.org/10.1016/j.phrs.2004.03.010

Angell, P., Chester, N., Green, D., Somauroo, J., Whyte, G., & George, K. (2012). Anabolic steroids and cardiovascular risk. Sports Medicine, 42(2), 119–134. https://doi.org/10.2165/11598060-000000000-00000

Bamman, M. M., Hunter, G. R., Newton, L. E., Roney, R. K., & Khaled, M. A. (1993). Changes in body composition, diet, and strength of bodybuilders during the 12 weeks prior to competition. J Sports Med Phys Fitness, 33(4), 383-391. https://www.ncbi.nlm.nih.gov/pubmed/8035587

Beijk, M. A. M., Vlastra, W., Delewi, R., et al. (2019). Myocardial infarction with non-obstructive coronary arteries: A focus on vasospastic angina. Netherlands Heart Journal, 27(5), 237–245. https://doi.org/10.1007/s12471-019-1232-7

Boerhout, C. K. M., de Waard, G. A., Lee, J. M., et al. (2022). Prognostic value of structural and functional coronary microvascular dysfunction. EuroIntervention, 18(9), 719–728. https://doi.org/10.4244/EIJ-D-22-00043

Bond, P., Smit, D. L., & de Ronde, W. (2022). Anabolic–androgenic steroids: How do they work and what are the risks? Frontiers in Endocrinology, 13, 1059473. https://doi.org/10.3389/fendo.2022.1059473

Buller, P., Rosiak, W., Tyczyński, M., Gil, R., & Bil, J. (2021). Arterial hypertension as a risk factor for myocardial infarction with non-obstructive coronary arteries. Arterial Hypertension, 25. https://doi.org/10.5603/AH.a2021.0009

Campos, H., Roederer, G. O., Lussier-Cacan, S., Davignon, J., & Krauss, R. M. (1995). Predominance of large LDL and reduced HDL2 cholesterol in normolipidemic men with coronary artery disease. Arteriosclerosis, Thrombosis, and Vascular Biology, 15(8), 1043–1048. https://doi.org/10.1161/01.ATV.15.8.1043

Chappell, A. J., Simper, T., & Barker, M. E. (2018). Nutritional strategies of high-level natural bodybuilders during competition preparation. Journal of the International Society of Sports Nutrition, 15, 4. https://doi.org/10.1186/s12970-018-0209-z

Christou, G. A., Christou, K. A., Nikas, D. N., & Goudevenos, J. A. (2016). Acute myocardial infarction in a young bodybuilder taking anabolic androgenic steroids: A case report and critical review of the literature. European Journal of Preventive Cardiology, 23(16), 1785–1796. https://doi.org/10.1177/2047487316651341

De Souza, D. C., Dos Santos, J. A. B., De Jesus, D. M., & Gentil, P. (2018). Biochemical profile and body composition alteration of amateur bodybuilders during the pre-contest period. Journal of Functional Morphology and Kinesiology, 3(2), 26. https://doi.org/10.3390/jfmk3020026

Ebenbichler, C. F., Sturm, W., Gänzer, H., et al. (2001). Flow-mediated, endothelium-dependent vasodilatation is impaired in male body builders taking anabolic-androgenic steroids. Atherosclerosis, 158(2), 483–490. https://doi.org/10.1016/S0021-9150(01)00465-8

Escalante, G., Stevenson, S. W., Barakat, C., Aragon, A. A., & Schoenfeld, B. J. (2021). Peak week recommendations for bodybuilders: An evidence-based approach. BMC Sports Science, Medicine and Rehabilitation, 13(1), 68. https://doi.org/10.1186/s13102-021-00296-y

Fan, Y., Li, C., Hu, Y., et al. (2023). Angiography-based index of microcirculatory resistance for the assessment of microvascular dysfunction. Quantitative Imaging in Medicine and Surgery, 13(6), 3556–3568. https://doi.org/10.21037/qims-22-961

Gentil, P., de Lira, C. A. B., Paoli, A., et al. (2017). Nutrition, pharmacological and training strategies adopted by six bodybuilders: Case report and critical review. European Journal of Translational Myology, 27(1), 6247. https://doi.org/10.4081/ejtm.2017.6247

Hajjar, R. R., Kaiser, F. E., & Morley, J. E. (1997). Outcomes of long-term testosterone replacement in older hypogonadal males. Journal of Clinical Endocrinology & Metabolism, 82(11), 3793–3796. https://doi.org/10.1210/jcem.82.11.4387

Hartgens, F., & Kuipers, H. (2004). Effects of androgenic-anabolic steroids in athletes. Sports Medicine, 34(8), 513–554. https://doi.org/10.2165/00007256-200434080-00003

Helms, E. R., Aragon, A. A., & Fitschen, P. J. (2014). Evidence-based recommendations for natural bodybuilding contest preparation: Nutrition and supplementation. Journal of the International Society of Sports Nutrition, 11, 20. https://doi.org/10.1186/1550-2783-11-20

Iliakis, P., Stamou, E., Kasiakogias, A., et al. (2025). Anabolic-androgenic steroids induced cardiomyopathy: A narrative review of the literature. Biomedicines, 13(9), 2190. https://doi.org/10.3390/biomedicines13092190

Khan, A., Lahmar, A., Riasat, M., et al. (2022). Myocardial infarction with non-obstructive coronary arteries: An updated overview. Cureus, 14(3), e23602. https://doi.org/10.7759/cureus.23602

Kleiner, S. M., Bazzarre, T. L., & Litchford, M. D. (1990). Metabolic profiles, diet, and health practices of championship male and female bodybuilders. J Am Diet Assoc, 90(7), 962-967. https://www.ncbi.nlm.nih.gov/pubmed/2365938

Kunadian, V., Chieffo, A., Camici, P. G., et al. (2020). EAPCI expert consensus document on ischaemia with non-obstructive coronary arteries. European Heart Journal, 41(37), 3504–3520. https://doi.org/10.1093/eurheartj/ehaa503

Lane, H. A., Grace, F., Smith, J. C., et al. (2006). Impaired vasoreactivity in bodybuilders using androgenic anabolic steroids. European Journal of Clinical Investigation, 36(7), 483–488. https://doi.org/10.1111/j.1365-2362.2006.01667.x

Lee, J. M., Jung, J. H., Hwang, D., et al. (2016). Coronary flow reserve and microcirculatory resistance in patients with intermediate coronary stenosis. Journal of the American College of Cardiology, 67(10), 1158–1169. https://doi.org/10.1016/j.jacc.2015.12.053

Lenders, J. W. M., Demacker, P. N. M., Vos, J. A., et al. (1988). Deleterious effects of anabolic steroids on serum lipoproteins, blood pressure, and liver function in amateur body builders. International Journal of Sports Medicine, 9(1), 19–23. https://doi.org/10.1055/s-2007-1024972

Perry, J. C., Schuetz, T. M., Memon, M. D., Faiz, S., & Cancarevic, I. (2020). Anabolic steroids and cardiovascular outcomes: The controversy. Cureus, 12(7), e9333. https://doi.org/10.7759/cureus.9333

Pompei, G., Ganzorig, N., Kotanidis, C. P., et al. (2024). Novel diagnostic approaches and management of coronary microvascular dysfunction. American Journal of Preventive Cardiology, 19, 100712. https://doi.org/10.1016/j.ajpc.2024.100712

Rossow, L. M., Fukuda, D. H., Fahs, C. A., Loenneke, J. P., & Stout, J. R. (2013). Natural bodybuilding competition preparation and recovery: A 12-month case study. International Journal of Sports Physiology and Performance, 8(5), 582–592. https://doi.org/10.1123/ijspp.8.5.582

Spione, F., Arevalos, V., Gabani, R., Sabaté, M., & Brugaletta, S. (2022). Coronary microvascular angina: A state-of-the-art review. Frontiers in Cardiovascular Medicine, 9, 800918. https://doi.org/10.3389/fcvm.2022.800918

Stergiopoulos, K., Brennan, J. J., Mathews, R., Setaro, J. F., & Kort, S. (2008). Anabolic steroids, acute myocardial infarction and polycythemia. Vascular Health and Risk Management, 4(6), 1475–1480. https://doi.org/10.2147/VHRM.S4261

Sweetnam, P. M., Bolton, C. H., Yarnell, J. W., et al. (1994). Associations of the HDL2 and HDL3 cholesterol subfractions with the development of ischemic heart disease in British men. Circulation, 90(2), 769–774. https://doi.org/10.1161/01.CIR.90.2.769

Urhausen, A., Albers, T., & Kindermann, W. (2004). Are the cardiac effects of anabolic steroid abuse in strength athletes reversible? Heart, 90(5), 496–501. https://doi.org/10.1136/hrt.2003.015719

Vanberg, P., & Atar, D. (2010). Androgenic anabolic steroid abuse and the cardiovascular system. Handbook of Experimental Pharmacology, 195, 411–457. https://doi.org/10.1007/978-3-540-79088-4_18

Windfeld-Mathiasen, J., Heerfordt, I. M., Dalhoff, K. P., et al. (2025). Cardiovascular disease in anabolic androgenic steroid users. Circulation, 151(12), 828–834. https://doi.org/10.1161/CIRCULATIONAHA.124.071117

Zdravkovic, M., Popadic, V., Klasnja, S., et al. (2023). Coronary microvascular dysfunction and hypertension: A bond more important than we think. Medicina, 59(12), 2149. https://doi.org/10.3390/medicina59122149