ISSN: 2766-9556

Journal of Clinical Case Studies and Reviews

Review Article

Open Access

Immune Checkpoint Inhibitor-Associated Myocarditis Presenting as Isolated High-Grade Atrioventricular Block: A Review of Surveillance Strategies

Anneke Boersma[1*], Sofie Vermeulen[2] and Tobias Reinhardt[3]

[1]Department of Medical Oncology, Zuiderveld University Medical Centre, Utrecht, Netherlands

[2]Department of Cardio-Oncology, Meridian University Hospital, Leuven, Belgium

[3]Department of Cardio-Oncology, Bavarian Institute of Cardiovascular Medicine, Regensburg, Germany

***Correspondence to:** Anneke Boersma, Department of Medical Oncology, Zuiderveld University Medical Centre, Utrecht, Netherlands

Received: 8 January 2022; **Accepted:** 25 March 2022; **Published:** 11 June 2022

Copyright: © 2022 AB, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Myocarditis is the least common and most lethal cardiovascular toxicity of immune checkpoint blockade. It declares itself early, often after a single dose, and a substantial minority of patients present with conduction disease rather than heart failure, so surveillance built around symptoms and ventricular function will miss them.

Objectives: *To review the evidence on the incidence, timing and presenting features of immune checkpoint inhibitor (ICI) myocarditis, with attention to the conduction-first phenotype, and to assess what troponin, electrocardiographic and imaging surveillance can and cannot achieve in an ambulatory oncology population.*

Material and Methods: *A narrative review. MEDLINE, Embase and the Cochrane Library were searched from January 2015 to May 2026 for registry and cohort studies, pharmacovigilance analyses, diagnostic accuracy studies, consensus definitions and society guidance. Incidence figures, event rates and diagnostic yields were extracted from the primary reports without pooling.*

Results: *Reported incidence ranges from 0.04% to 1.14% and case fatality from 25% to 50%; of 122 pharmacovigilance reports of myocarditis, 50% were fatal against 21% for pericardial disease and 6% for vasculitis. Median onset is 34 days and roughly two-thirds of patients have had only one or two cycles. Major adverse cardiac events occur in 38-46% of cases, and high-grade atrioventricular block is both a component of that composite and an independent predictor of death. Late gadolinium enhancement was present in only 48% of an international magnetic resonance cohort, with yield rising from 21.6% to 72.0% according to scan timing.*

Conclusion: *Troponin is the only instrument sensitive and practical enough to anchor a programme, but it must be paired with a baseline and serial electrocardiogram, because the conduction-first presentation can occur with modest troponin elevation and normal ventricular function. A protocolised pathway with a pre-specified action threshold is what converts surveillance into earlier treatment.*

Abbreviations: *ICI – Immune Checkpoint Inhibitor, irAE – Immune-Related Adverse Event, MACE – Major Adverse Cardiac Events, CMR – Cardiovascular Magnetic Resonance, LGE – Late Gadolinium Enhancement, STIR – Short Tau Inversion Recovery.*

Keywords: *Immune Checkpoint Inhibitor; Myocarditis; Atrioventricular Block; Troponin Surveillance*

Introduction

Immune checkpoint inhibitors have moved from salvage therapy in metastatic melanoma to first-line and adjuvant use across a widening range of tumours. The same loss of peripheral tolerance that produces the antitumour effect produces immune-related adverse events in almost every organ system [1]. Most are manageable. Myocarditis is not: it is among the rarest of these events and by some distance the most lethal [2].

The first multicentre registry to characterise it found myocarditis in 1.14% of 964 patients treated between 2013 and 2017, with major adverse cardiac events in 46% of affected patients [3]. Pharmacovigilance data assembled from the World Health Organization database over a similar period identified 122 myocarditis reports of which 61 were fatal, a case fatality of 50% against 21% for pericardial disease and 6% for vasculitis [2,4]. Single-centre series have reported incidences as low as 0.04% to 0.48%, and the honest summary of the literature is a range rather than a number [5,6].

What makes this toxicity difficult to catch is not its rarity but its timing and its presentation. Median onset is 34 days from the first dose, with an interquartile range of 21 to 75 days, and roughly two-thirds of patients have received only one or two cycles before it declares itself [3,7]. Fifty-four per cent have no other immune-related adverse event to raise suspicion [3]. Crucially, ventricular function is frequently preserved: 38% of patients with a fulminant course in the original registry had a normal ejection fraction, and 61% of an international magnetic resonance cohort had an ejection fraction of 50% or more [3,8].

A meaningful subset therefore presents not with breathlessness or shock but with conduction disease, and high-grade atrioventricular block may be the only abnormality on a first assessment. This review examines what the published evidence supports as a surveillance strategy for a toxicity that is rare, early, frequently silent and, once established, fatal in up to half of cases.

Material and Methods

Search strategy: MEDLINE, Embase and the Cochrane Library were searched from January 2015 to May 2026. Terms combined controlled vocabulary and free text for immune checkpoint inhibitor, myocarditis, immune-related adverse event, atrioventricular block, troponin and cardio-oncology. Reference lists of society guidance and consensus statements were hand-searched.

Eligibility and synthesis: registry and cohort studies reporting incidence or event rates, pharmacovigilance analyses, diagnostic accuracy studies, consensus case definitions and society guidance were eligible; isolated case reports were used only where they document a treatment approach for which no larger evidence exists. Values were extracted from the primary publications by one reviewer and checked by a second. The included studies differ in case definition, ascertainment and follow-up, so results are presented descriptively and no pooling was attempted. No individual patient data were accessed and ethics approval was not required.

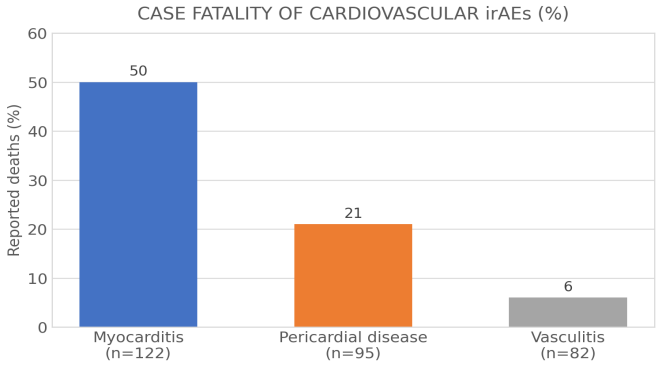

Figure 1: Case fatality of cardiovascular immune-related adverse events reported to the World Health Organization pharmacovigilance database, by event type [2].

Results

Incidence, mortality and the size of the problem

Estimates of incidence vary roughly thirty-fold across published series, driven mainly by case definition and by whether troponin was measured systematically or only on symptoms [5,6,9]. The figure most often quoted, 1.14%, comes from an eight-site registry that actively sought cases [3]; a nationwide Danish cohort and several single-centre series sit an order of magnitude lower [10,11]. What does not vary is the severity: case fatality is reported between 25% and 50%, and among cardiovascular immune-related adverse events myocarditis is markedly the most dangerous (Figure 1) [2,4,12].

Combination cytotoxic T-lymphocyte antigen-4 and programmed death-1 blockade approximately doubles the risk relative to single-agent therapy, and combination exposure was present in 34.3% of registry cases against 9.5% of controls [3,13]. Pre-existing cardiovascular disease, diabetes, obesity and sleep apnoea have all been associated with higher risk, though none is discriminating enough to define a screening population on its own [3,11].

Timing and the conduction-first presentation

The clustering of events in the first two cycles is the single most actionable fact in this literature. It means that surveillance concentrated in the first two months captures most of the risk, and that a programme which begins testing at cycle four will be too late for the majority [3,7,14]. It also means that the baseline assessment is not a formality: without a pre-treatment troponin and electrocardiogram there is nothing against which to interpret the first abnormal result [15].

Conduction disease is common and prognostically important. Electrocardiographic abnormalities in ICI myocarditis include high-grade atrioventricular block, new bundle branch block, QRS prolongation and ventricular arrhythmia, and the histopathological correlate is lymphocytic infiltration of the sinoatrial and atrioventricular nodes [16,17,18]. Complete heart block is both a

component of the standard major adverse cardiac event composite, which occurs in 38-46% of cases across published cohorts (Figure 2), and, analysed separately, an independent predictor of mortality [19]. Because nodal infiltration can dominate before ventricular involvement is apparent, an isolated high-grade block with only modest troponin elevation and a preserved ejection fraction is a recognised presentation rather than an anomaly (Table 1).

Troponin as the backbone of surveillance

Cardiac troponin is the only test with the sensitivity, availability and cost profile to anchor surveillance. It is elevated in essentially all cases at presentation and the magnitude tracks with the risk of major adverse cardiac events, so it serves for both detection and triage [3,20]. Prospective experience with high-sensitivity troponin I in an ambulatory checkpoint inhibitor population confirmed that a protocolised schedule is feasible in routine clinic workflow [15].

The limitations are equally clear. Troponin lacks specificity against a background of coronary disease, pulmonary embolism, sepsis and renal impairment, so surveillance generates work; the discipline lies in pre-specifying what an abnormal result triggers. Assay choice matters too: troponin T is elevated by the skeletal myositis that frequently overlaps with this toxicity, so a raised troponin T is not by itself evidence of cardiac involvement [21,22].

Electrocardiography and the case against troponin alone

Baseline and serial electrocardiography adds little cost and captures the phenotype troponin surveillance triages least well. New conduction abnormality, PR prolongation and QRS widening have all been described as the earliest change in patients who went on to develop fulminant disease [16,17]. A programme built on troponin alone risks classifying a patient with a mild troponin rise and a new first-degree block as low-risk, when the electrocardiogram is the finding that should escalate them.

Imaging, and why a normal scan does not reassure

Echocardiography is the first-line imaging test and global longitudinal strain is reduced and independently prognostic even where ejection fraction is preserved [23]. It cannot establish the diagnosis. Cardiovascular magnetic resonance is the reference standard for myocarditis generally, but its performance in this specific toxicity is materially worse than in viral myocarditis. In an international registry of 103 patients, late gadolinium enhancement was present in only 48% and elevated T2-weighted short tau inversion recovery signal in 28%; neither predicted major adverse cardiac events (Figure 3) [8].

Timing explains part of this. In the same cohort, the yield of late gadolinium enhancement rose from 21.6% when imaging was performed early to 72.0% when it was performed from the fourth day of symptoms onward (Figure 4) [8]. The practical consequence is that a negative magnetic resonance study performed on the day

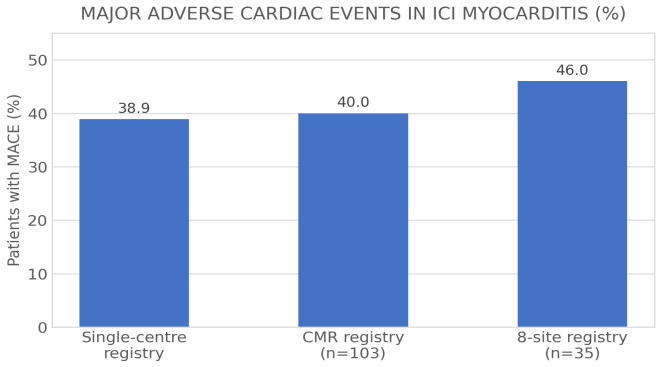

Figure 2: Reported rates of major adverse cardiac events among patients with established ICI myocarditis across published cohorts [3,6,8].

Domain	Trigger
Timing	First eight weeks of therapy; after only one or two cycles; within 24 hours of any dose
Electrocardiography	New high-grade or complete atrioventricular block; new bundle branch block; PR or QRS prolongation; ventricular arrhythmia
Biomarkers	Troponin rise above the pre-treatment baseline, however modest, without an alternative explanation
Overlap features	Concurrent myositis, ptosis, diplopia or bulbar weakness suggesting a myasthenia-like syndrome
Risk profile	Dual CTLA-4 and PD-1 blockade; concurrent cardiotoxic therapy; pre-existing cardiovascular disease

Table 1: Features that should trigger evaluation for immune checkpoint inhibitor-associated myocarditis in a patient receiving checkpoint blockade.

of presentation cannot be used to withhold treatment, and that where clinical suspicion is high and imaging is negative, endomyocardial biopsy should be considered [24,25]. Fluorodeoxyglucose positron emission tomography has been explored but is degraded by recent corticosteroid exposure and by the dietary preparation required [26].

Case definitions and what counts as a diagnosis

Two consensus frameworks now define the entity: proposed case definitions for myocarditis in the cancer-therapeutics setting, and the International Cardio-Oncology Society statement on cardiovascular toxicities [27,28]. Both grade certainty from possible to definite using troponin, electrocardiography, imaging and histology, and both accept that treatment will often begin before the definition is satisfied; oncology and immunotherapy society guidance is concordant [29,30,31].

What guidelines recommend, and what they leave open

The 2022 European cardio-oncology guidance recommends a baseline electrocardiogram for every patient starting cancer therapy and baseline natriuretic peptide and troponin where these will be

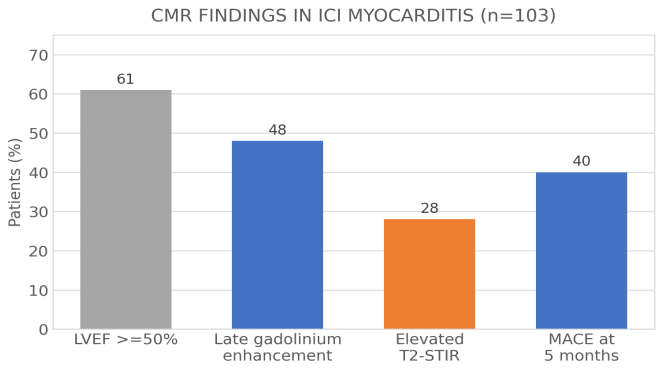

Figure 3: Cardiovascular magnetic resonance findings in an international registry of 103 patients with ICI myocarditis [8]. Neither enhancement nor oedema predicted adverse events.

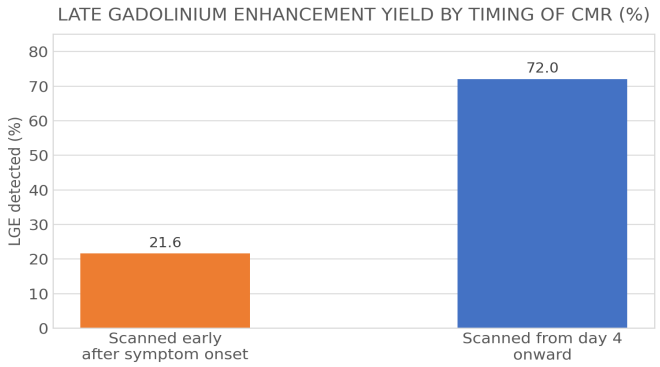

Figure 4: Yield of late gadolinium enhancement in ICI myocarditis according to when the magnetic resonance study was performed relative to symptom onset [8].

tracked, with serial troponin during checkpoint inhibitor therapy carrying a class IIa recommendation in higher-risk patients – those on dual blockade, those receiving concurrent cardiotoxic therapy, those with non-cardiac immune-related adverse events, and those with pre-existing cardiovascular disease [14,32]. A commonly cited schedule measures troponin at baseline, before doses two, three and four, and every three doses thereafter (Table 2) [15].

What the guidance does not settle is whether universal surveillance outperforms risk-stratified surveillance. A single-centre study of weekly electrocardiography and troponin in 76 asymptomatic patients on combination blockade detected no myocarditis over a median of 198 days, which illustrates both the rarity of the event and the difficulty of demonstrating benefit from screening at feasible sample sizes [12,33].

Discussion

The evidence supports a modest conclusion. Surveillance for ICI myocarditis cannot rest on a single test, but neither does it require an elaborate one. A baseline troponin and electrocardiogram before the first dose, repeated through the first two months when most events occur, with a pre-specified escalation threshold, captures the great majority of the risk at a cost ordinary oncology services can absorb.

Timepoint	Test	Action threshold
Before dose 1	ECG, troponin, natriuretic peptide	Establish the baseline; refer if ECG abnormal
Before doses 2, 3, 4	ECG and troponin	Any rise above baseline: withhold dose, urgent cardio-oncology review
Every 3 doses thereafter	ECG and troponin	As above; lower threshold if dual blockade
Any new symptom	ECG, troponin, echocardiogram	Treat on suspicion; do not wait for confirmatory imaging

Table 2: A protocolised surveillance schedule for patients starting checkpoint blockade, consolidating the 2022 European cardio-oncology recommendations and published ambulatory experience [14,15,32].

The conduction-first presentation is the strongest argument against a troponin-only programme. A patient whose only abnormality is new high-grade atrioventricular block sits outside the pattern that troponin thresholds and ejection fraction cut-offs are designed to detect, yet complete heart block independently predicts death in this population [19]. Adding an electrocardiogram to each troponin draw costs almost nothing and closes that gap. Where block is present, temporary pacing may be required, but the decision to implant a permanent device should be deferred, since conduction disease that resolves with immunosuppression is well described [34,35].

Treatment once the diagnosis is suspected

Guidance is uniform that checkpoint inhibitor therapy should be withheld and high-dose methylprednisolone started on suspicion rather than confirmation, and that earlier, higher initial dosing is associated with fewer major adverse cardiac events [29,32,36]. For steroid-refractory disease, second-line options include abatacept, which blocks the CD80/CD86 co-stimulatory interaction, the Janus kinase inhibitor ruxolitinib, antithymocyte globulin and plasma exchange (Table 3) [37,38]. A phase 3 trial of abatacept added to corticosteroids is the first prospective test of any second-line agent here.

Rechallenge remains contentious. Registry data on resumption after immune-related adverse events show a substantial recurrence rate, and for myocarditis most authorities advise permanent discontinuation, any exception being a multidisciplinary decision [30,39].

Study limitations

This is a narrative rather than a systematic review: studies were selected against stated criteria but without duplicate screening of the full yield, and no risk-of-bias instrument was applied. Incidence and fatality figures derive from registries and spontaneous reporting systems subject to ascertainment bias, and pharmacovigilance case fatality in particular overstates the true rate because severe cases are

Agent	Mechanism	Evidence
Methylprednisolone	Broad immunosuppression	First line in all guidance; earlier and higher dosing linked to fewer events [36]
Abatacept	CD80/CD86 co-stimulation blockade	Preclinical survival benefit and case-level response; phase 3 trial ongoing [37]
Ruxolitinib	JAK inhibition	Reported in combination with abatacept for steroid-refractory fulminant disease [38]
ATG or plasma exchange	T-cell depletion; antibody removal	Case-level only; reserved for refractory disease [34]

Table 3: Immunosuppressive options for confirmed or strongly suspected ICI myocarditis. Evidence beyond corticosteroids is drawn from case series and preclinical work; a randomised trial of abatacept is in progress.

preferentially reported. Accuracy data come predominantly from referral centres. The review addresses surveillance and initial management rather than long-term follow-up of survivors.

Conclusion

Immune checkpoint inhibitor myocarditis is rare, early and lethal, and a meaningful proportion of patients present with conduction disease rather than the heart failure phenotype surveillance programmes are usually designed around. Troponin is the only practical backbone for detection, but it must be paired with baseline and serial electrocardiography, concentrated in the first two months and read against a pre-treatment baseline. Magnetic resonance imaging cannot exclude the diagnosis, particularly when performed early. A protocolised pathway with a pre-specified action threshold is what turns surveillance into earlier treatment.

References

1. Postow MA, Sidlow R, Hellmann MD. "Immune-related adverse events associated with immune checkpoint blockade". N Engl J Med 378(2018):158-168.

2. Salem JE, Manouchehri A, Moey M, Lebrun-Vignes B, Bastarache L, et al. "Cardiovascular toxicities associated with immune checkpoint inhibitors: an observational, retrospective, pharmacovigilance study". Lancet Oncol 19(2018):1579-1589.

3. Mahmood SS, Fradley MG, Cohen JV, Nohria A, Reynolds KL, et al. "Myocarditis in patients treated with immune checkpoint inhibitors". J Am Coll Cardiol 71(2018):1755-1764.

4. Moslehi JJ, Salem JE, Sosman JA, Lebrun-Vignes B, Johnson DB. "Increased reporting of fatal immune checkpoint inhibitor-associated myocarditis". Lancet 391(2018):933.

5. Palaskas N, Lopez-Mattei J, Durand JB, Iliescu C, Deswal A. "Immune checkpoint inhibitor myocarditis: pathophysiological characteristics, diagnosis, and treatment". J Am Heart Assoc 9(2020):e013757.

6. Ganatra S, Neilan TG. "Immune checkpoint inhibitor-associated myocarditis". Oncologist 23(2018):879-886.

7. Johnson DB, Balko JM, Compton ML, Chalkias S, Gorham J, et al. "Fulminant myocarditis with combination immune checkpoint blockade". N Engl J Med 375(2016):1749-1755.

8. Zhang L, Awadalla M, Mahmood SS, Nohria A, Hassan MZO, et al. "Cardiovascular magnetic resonance in immune checkpoint inhibitor-associated myocarditis". Eur Heart J 41(2020):1733-1743.

9. Hu JR, Florido R, Lipson EJ, Naidoo J, Ardehali R, et al. "Cardiovascular toxicities associated with immune checkpoint inhibitors". Cardiovasc Res 115(2019):854-868.

10. D'Souza M, Nielsen D, Svane IM, Iversen K, Rasmussen PV, et al. "The risk of cardiac events in patients receiving immune checkpoint inhibitors: a nationwide Danish study". Eur Heart J 42(2021):1621-1631.

11. Mir H, Alhussein M, Alrashidi S, Alzayer H, Alshatti A, et al. "Cardiac complications associated with checkpoint inhibition: a systematic review of the literature in an important emerging area of cancer". Can J Cardiol 34(2018):1059-1068.

12. Ganatra S, Parikh R, Neilan TG. "Cardiotoxicity of immune therapy". Cardiol Clin 37(2019):385-397.

13. Wang DY, Salem JE, Cohen JV, Chandra S, Menzer C, et al. "Fatal toxic effects associated with immune checkpoint inhibitors: a systematic review and meta-analysis". JAMA Oncol 4(2018):1721-1728.

14. Lyon AR, Lopez-Fernandez T, Couch LS, Asteggiano R, Aznar MC, et al. "2022 ESC guidelines on cardio-oncology developed in collaboration with the EHA, ESTRO and IC-OS". Eur Heart J 43(2022):4229-4361.

15. Waliany S, Neal JW, Reddy S, Wakelee H, Shah SA, et al. "Myocarditis surveillance with high-sensitivity troponin I during cancer treatment with immune checkpoint inhibitors". JACC CardioOncol 3(2021):137-139.

16. Power JR, Alexandre J, Choudhary A, Ozbay B, Hayek SS, et al. "Electrocardiographic manifestations of immune checkpoint inhibitor myocarditis". Circulation 144(2021):1521-1523.

17. Zlotoff DA, Hassan MZO, Zafar A, Alvi RM, Awadalla M, et al. "Electrocardiographic features of immune checkpoint inhibitor associated myocarditis". J Immunother Cancer 9(2021):e002007.

18. Moslehi J, Lichtman AH, Sharpe AH, Galluzzi L, Kitsis RN. "Immune checkpoint inhibitor-associated myocarditis: manifestations and mechanisms". J Clin Invest 131(2021):e145186.

19. Escudier M, Cautela J, Malissen N, Ancedy Y, Orabona M, et al. "Clinical features, management, and outcomes of immune checkpoint inhibitor-related cardiotoxicity". Circulation 136(2017):2085-2087.

20. Thuny F, Naidoo J, Neilan TG. "Cardiovascular complications of immune checkpoint inhibitors for cancer". Eur Heart J 43(2022):4458-4468.

21. Anquetil C, Salem JE, Lebrun-Vignes B, Johnson DB, Mammen AL, et al. "Immune checkpoint inhibitor-associated myositis: expanding the spectrum of cardiac complications of the immunotherapy revolution". Circulation 138(2018):743-745.

22. Lehmann LH, Cautela J, Palaskas N, Baik AH, Meijers WC, et al. "Clinical strategy for the diagnosis and treatment of immune checkpoint inhibitor-associated myocarditis: a narrative review". JAMA Cardiol 6(2021):1329-1337.

23. Awadalla M, Mahmood SS, Groarke JD, Hassan MZO, Nohria A, et al. "Global longitudinal strain and cardiac events in patients with immune checkpoint inhibitor-related myocarditis". J Am Coll Cardiol 75(2020):467-478.

24. Champion SN, Stone JR. "Immune checkpoint inhibitor associated myocarditis occurs in both high-grade and low-grade forms". Mod Pathol 33(2020):99-108.

25. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, et al. "Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations". J Am Coll Cardiol 72(2018):3158-3176.

26. Ederhy S, Devos P, Pinna B, Funck-Brentano E, Abbar B, et al. "18F-fluorodeoxyglucose positron emission tomography/computed tomography imaging for the diagnosis of immune checkpoint inhibitor-associated myocarditis". Arch Cardiovasc Dis 115(2022):114-124.

27. Bonaca MP, Olenchock BA, Salem JE, Wiviott SD, Ederhy S, et al. "Myocarditis in the setting of cancer therapeutics: proposed case definitions for emerging clinical syndromes in cardio-oncology". Circulation 140(2019):80-91.

28. Herrmann J, Lenihan D, Armenian S, Barac A, Blaes A, et al. "Defining cardiovascular toxicities of cancer therapies: an International Cardio-Oncology Society (IC-OS) consensus statement". Eur Heart J 43(2022):280-299.

29. Brahmer JR, Abu-Sbeih H, Ascierto PA, Brufsky J, Cappelli LC, et al. "Society for Immunotherapy of Cancer (SITC) clinical practice guideline on immune checkpoint inhibitor-related adverse events". J Immunother Cancer 9(2021):e002435.

30. Schneider BJ, Naidoo J, Santomasso BD, Lacchetti C, Adkins S, et al. "Management of immune-related adverse events in patients treated with immune checkpoint inhibitor therapy: ASCO guideline update". J Clin Oncol 39(2021):4073-4126.

31. Naidoo J, Murphy C, Atkins MB, Brahmer JR, Champiat S, et al. "Society for Immunotherapy of Cancer (SITC) consensus definitions for immune checkpoint inhibitor-associated immune-related adverse events". J Immunother Cancer 11(2023):e006398.

32. Curigliano G, Lenihan D, Fradley M, Ganatra S, Barac A, et al. "Management of cardiac disease in cancer patients throughout oncological treatment: ESMO consensus recommendations". Ann Oncol 31(2020):171-190.

33. Nso N, Antwi-Amoabeng D, Beutler BD, Ulanja MB, Ghuman J, et al. "Cardiac adverse events of immune checkpoint inhibitors in oncology patients: a systematic review and meta-analysis". World J Cardiol 12(2020):584-598.

34. Cautela J, Zeriouh S, Gaubert M, Bonello L, Laine M, et al. "Intensified immunosuppressive therapy in patients with immune checkpoint inhibitor-induced myocarditis". J Immunother Cancer 8(2020):e001887.

35. Axelrod ML, Meijers WC, Screever EM, Qin J, Carroll MG, et al. "T cells specific for alpha-myosin drive immunotherapy-related myocarditis". Nature 611(2022):818-826.

36. Zhang L, Zlotoff DA, Awadalla M, Mahmood SS, Nohria A, et al. "Major adverse cardiovascular events and the timing and dose of corticosteroids in immune checkpoint inhibitor-associated myocarditis". Circulation 141(2020):2031-2034.

37. Salem JE, Allenbach Y, Vozy A, Brechot N, Johnson DB, et al. "Abatacept for severe immune checkpoint inhibitor-associated myocarditis". N Engl J Med 380(2019):2377-2379.

38. Salem JE, Bretagne M, Abbar B, Leonard-Louis S, Ederhy S, et al. "Abatacept/ruxolitinib and screening for concomitant respiratory muscle failure to mitigate fatality of immune checkpoint inhibitor myocarditis". Cancer Discov 13(2023):1100-1115.

39. Dolladille C, Ederhy S, Sassier M, Cautela J, Thuny F, et al. "Immune checkpoint inhibitor rechallenge after immune-related adverse events in patients with cancer". JAMA Oncol 6(2020):865-871.

Citation: Boersma A, Vermeulen S, Reinhardt T. "Immune Checkpoint Inhibitor-Associated Myocarditis Presenting as Isolated High-Grade Atrioventricular Block: A Review of Surveillance Strategies." J Clin Case Stud (2022): 001-006 doi: 10.5281/zenodo.22205984