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Review Article

Cardiac complications of systemic drugs used in dermatology

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ABSTRACT

Within the realm of dermatology, the use of systemic drugs carries implications beyond skin health, often intersecting with cardiovascular outcomes. From antihistamines to biologics, medications prescribed for dermatological conditions may pose varying degrees of risk to the heart. Understanding these potential cardiac side effects is paramount in the comprehensive management of dermatologic patients. This journal article delves into the intricate relationship between systemic dermatologic drugs and cardiovascular health, elucidating the mechanisms underlying cardiotoxicity, identifying patient-specific risk factors, and offering insights into optimizing therapeutic regimens while safeguarding cardiac well-being. Through this exploration, we aim to equip dermatologists with the knowledge necessary for delivering safe and effective care, ensuring the holistic health of their patients.

Keywords: Adverse effects, Drugs, Cardiotoxicity

INTRODUCTION

Cardiovascular disease are one of the primary cause of morbidity and mortality in many developed countries all over the world. This makes it crucial to understand the potential effects of the drugs on the cardiovascular system.[1] There are many dermatological conditions with cardiac involvement. Certain conditions affect both the skin and the heart. And then there are drugs that are used in treating the skin conditions causing cardiac side effects. This underscores the importance of dermatologist being aware of the potential side effects of these medications.

RETINOIDS

Retinoids are known to cause dyslipidemia. Though not commonly encountered there are case reports of isotretinoin induced atrial tachycardia, pericardial effusion.[2] Acitretin can cause acute myocardial infarction and bexarotene is reported to accelerate coronary atherosclerosis.[3,4] The cardiovascular birth defects due to teratogenic action are explained by inhibition of mesoderm formation during cardiac differentiation, dysregulation of genes associated with mesoderm differentiation during cardiomyocyte differentiation and disruptions in signalling pathways during mesoderm formation.[5]

METHOTREXATE

Methotrexate use presents with both benefits and potential risks. Reduction in tetrahydrofolate results in homocysteinemia. Though there are factors that places cardiovascular system at risk some actions reduce the cardiovascular risk as discussed in Table 2.

Table 1: Classification and mechanism of cardiac toxicity.[1]

Classification	Mechanism
1.Rhythm abnormalities	Bradyarrhythmia: calcium channel blockade, sodium channel blockade, β_1 receptor antagonism, blocking I_f current, M-receptor agonism, increased vagal tone, agonism at α_2-receptors. Tachyarrhythmias: hERG channel blockade, β_1-receptor agonism, inhibition of Na^+/K^+ pump, Na+ channel blockade, M receptor antagonism.
2. Myocardial ischemia	β_1-receptor agonism, arterial thromboembolism, hyperstimulation of cardiac muscle, pronounced hypotension.
3. Left ventricular failure or dysfunction	Ca^{2+} channel blockade, Na+ channel blockade, β_1 receptor antagonism, inhibtion of HER2 signaling, VEGF signaling inhibition, tyrosine kinase blockade.
4. Pericardial disease	Immune reaction
5. Cardiac valve impairment	5-HT_{2B} receptor agonism

Table 2: Different mechanism by which methotrexate increase and decrease the cardiac risk.

Factors increasing cardiac risk[6]	Factors reducing cardiac risk[7]
Reduced endothelial nitric oxide production	Control in foam cell formation
Endothelial dysfunction due to altered oxidative stress	Induction of reverse cholesterol transport
Vascular smooth muscle proliferation	Reduction in a proatherogenic factor- serum lipoprotein(a)
Nuclear factor κB activation	Endothelial function improvement
Reduction in arterial wall elasticity	Reduction in carotid intima media thickness
Raised collagen production	Prevents rise in systolic blood pressure by controlling arterial stiffening.

Table 3: Predictive factors for cardiac complications.

Predictive factors
Cumulative dose
Route of administration
Age and sex of the patient
Pre-existing cardiac conditions
Drug interactions
History of drug allergy
Idiosyncrasy

Table 4: Warning symptoms to advice patients to report.

Warning symptoms
Palpitations
Chest pain/epigastric pain
Light headedness
Sweating
Swelling of the legs
Breathlessness

AZATHIOPRINE

Azathioprine, though the cardiac risk is very low, cases of hypotension, cardiogenic shock and atrial fibrillation are reported.[8,9]

MYCOPHENOLATE MOFETIL

Mycophenolate mofetil (MMF) is said to reduce the progression of atherosclerosis due to decreased vascular oxidative stress and its anti-inflammatory action.[10] In contrast, there have been case reports documenting MMF-induced supraventricular tachycardia.[11]

CYCLOSPORINE

Animal studies have demonstrated a correlation between cyclosporine use and adverse cardiovascular effects, including coronary vasospasm and ventricular hypertrophy.[12,13] Additionally, clinical case reports have documented instances of atrial fibrillation associated with cyclosporine toxicity in human subjects.[14]

BLEOMYCIN

Bleomycin use has been associated coronary artery disease, myocardial ischemia, myocardial infarction and rarely pericarditis and acute chest pain syndrome. These adverse events are not associated with any consistent signs and symptoms.[15]

CYCLOPHOSPHAMIDE

Cyclophosphamide, when given in low doses not induce cardiotoxicity. High dose cyclophosphamide use in the management of Mycosis fungoides, lymphoma etc has been associated with tachyarrhythmias, decreased amplitude of QRS complex, heart block and nonspecific T wave or ST segment abnormalities.[16]

ANTIFUNGALS

Itraconazole has been linked to adverse cardiovascular events, encompassing congestive heart failure, premature ventricular contractions, systemic hypertension and, in severe cases, ventricular fibrillation.[17-21] Fluconazole is known to prolong the QT interval and result in Torsade de pointes (TDP) even at low doses, necessitating the need of serial electrocardiographic monitoring in patients with increased risk of ventricular arrythmias.[18,22] Griseofulvin causes peripheral vasodilation and decreases the blood pressure when given intravenously. It also acts directly on the myocardial fibres and vessel wall resulting in increased heart rate.[23] Ketoconazole can also prolong the QT interval and cause TDP.[24] Amphotericin B is associated with ventricular arrhythmias, bradycardia, hypokalaemia. Though cardiac toxicity is rare with liposomal preparation of Amphotericin B bradycardia, Atrioventricular block (AV block) are reported.[25]

ERTHYROMYCIN

Erythromycin is reported to increase the risk of ventricular arrhythmias, QT prolongation and Torsades de pointes. In addition, patients who are on erythromycin with concomitant CYP3A inhibitors has five times more risk of sudden death due to cardiac causes than those who are not on these drugs.[26]

FOSCARNET

It's mainly used in acyclovir resistant Herpes simplex infection and cytomegalo virus infection. Foscarnet forms drug-calcium ion complex resulting in hypocalcemia, hypomagnesemia (hypoparathyroid state) and hypokalaemia (renal wastage of potassium). This in turn leads to cardiac arrythmias. Hypokalaemia is commonly seen during the induction phase of Foscarnet therapy.[27]

ANTIHISTAMINES

Due to profound cardiac adversities two first generation antihistamines, astemizole and terfenadine are banned from use.[28] First generation H1 antihistamine is associated with higher risk of cardiovascular events like arrhythmias especially in pediatric population.[29]

TRICYCLIC ANTIDEPRESSANTS

Amitriptyline, doxepin, nortriptyline are associated with life threatening rhythm abnormalities especially wide QRS complex tachycardia and hypotension.[1]

HYDROXYCHLOROQUINE

Hydroxychloroquine (HCQ) causes cardiomyopathy with the electron microscopic changes similar to that seen Fabry disease. HCQ induced cardiomyopathy may manifest as ventricular hypertrophy, valvular dysfunction, hypokinesia and potentially pulmonary arterial hypertension.[30] Acute intoxication of HCQ can lead to QTc prolongation, ventricular tachycardia, ventricular fibrillation, rarely prolonged QRS and Torsades de pointes. However prolonged use of over 10 years results higher incidence of arrhythmias and cardiomyopathies than in acute intoxication. Atrial fibrillation, bundle branch block, left-sided heart failure and QT prolongation are seen with prolonged use of HCQ.[31]

CORTICOSTEROIDS

Corticosteroids are widely used in day to day practice. They exert their action via genomic action or via non genomic action such as activating the membrane bound glucocorticoid receptor or via direct physiochemical interaction with the cell wall elements.[32] While the genomic effects pose the cardiac related adverse effects, the non-genomic effects are responsible for the beneficial effects of steroids on vascular inflammation, inhibiting the leucocyte endothelial interaction and also activating the endothelial nitric oxide synthase.[33,34] The common adverse effect of glucocorticoids on Cardiovascular system is dyslipidemia and hypertension, atrial fibrillation and also pose a significant risk of heart failure.[35-37] Though it was of the belief that low dose steroids was comparatively safer, its now been proved that doses even less than 5 mg of prednisolone doubles the risk of cardiovascular diseases.[36]

Pulse therapy with corticosteroids is used in the management of autoimmune disorders. Sinus tachycardia, the most common cardiac event is followed by atrial fibrillation, sinus bradycardia and ventricular

tachycardia.[38] Arrhythmias occur in 1% to 82% patients on intravenous methylprednisolone pulse.[39] One study suggests the mean time for development of bradycardia as 42-80hours from the initiation of the pulse.[40]

ORAL MINOXIDIL

Minoxidil, a systemic vasodilator, triggers myocardial ischemia by reflex tachycardia and fluid retention by modulating renal hemodynamics. Thus, making it contraindicated in patients with angina or recent history of myocardial ischemia, heart failure and left ventricular hypertrophy.[41] There are case reports of Pericardial effusions due to low dose oral minoxidil.[42]

RITUXIMAB

Rituximab is a chimeric human/murine IgG monoclonal antibody that binds specifically to CD 20 antigen on normal and malignant B lymphocytes. In patients aged more than 60 years with a known history of cardiovascular disease are at a higher risk of developing arrythmias. Cardiac adverse effects of rituximab include arrythmias, hypotension, cardiogenic shock, reduction in ejection fraction, non-ischemic cardiomyopathy, Takotsubo's cardiomyopathy During infusion reaction, side effects like angina, acute coronary syndrome (ACS), and arrhythmias are reported.[43,44] Benign cardiac side effects of rituximab include hypertension (between approximately 6%-12% of patients) and transient hypotension (approximately 10% of patients).[45]

OMALIZUMAB

Omaliuzmab is a humanized monoclonal antibody that prevents activation by allergens and reduces chronic inflammation. Cardiomyopathy, MI (myocardial infarction), arrythmias, heart failure, syncope, Arterial and venous thrombosis have been reported in patients on omalizumab compared to untreated group.[46,47] Exposure to omalizumab is associated with an increased risk of arterial thrombotic events in asthmatic patients, predominantly cardiovascular death and stroke.[46] Omalizumab may be a cause of Takotsubo syndrome, a reversible form of dilated cardiomyopathy mimicking acute MI associated with sympathetic stimulation.[48]

ETANERCEPT

It's a tumor necrosis factor α (TNF-α) antagonist. Etanercept blocks the deleterious effects of TNF at the level of cell surface receptors. TNF can dissociate from this cytokine-drug complex leading to increased peripheral bioactivity that adversely affect the cardiomyocytes.[49] Though it is claimed to be safe from cardiac front, there are case reports of heart failure, non-ischemic cardiomyopathy.[50-52]

ADALIMUMAB

In vitro studies showed improvement in congestive heart failure (CHF) symptoms in mice cardiomyocytes. There are no large scale human studies to support this finding. There are case reports of CHF due to adalimumab.[53] However among etanercept, infliximab and adalimumab, adalimumab has better cardiac safety.[54]

INFLIXIMAB

A popular clinical trial on the effects of infliximab in class III-IV heart failure has been stopped prematurely with a conclusion that there was increased hospitalization and deaths. This was seen in patients on a dose of 10 mg/kg, while 5 mg/kg showed no evidence of reduction in cardiac symptoms.[55] Therefore infliximab should be avoided in symptomatic moderate to severe congestive heart failure.

INTERFERON

Interferon therapy is associated with arrythmias, dilated cardiomyopathy, hypotension, ischemic heart disease and pericarditis.[56,57]

INTRAVENOUS IMMUNOGLOBULIN THERAPY

Intravenous immunoglobulin therapy (IVIg) infusion is associated with high risk for cardiac arrest and supraventricular tachycardia. It can also result in sick sinus syndrome, atrial flutter, atrial fibrillation and bradycardia.[58,59]

CONCLUSION

Dermatologist should be aware of the cardiac side effects of the drugs as well as the cardiac involvement in a particular condition. It always starts with eliciting a proper history to rule out any pre-existing cardiac conditions and starting on the appropriate dose according to the age and weight of the patient. In instance of adverse events prompt intervention from withholding the drug to taking a cardiologist opinion is paramount in preventing morbidities.

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REFERENCES

1. Mladěnka P, Applová L, Patočka J, Costa VM, Remiao F, Pourová J, et al. TOX-OER and CARDIOTOX Hradec Králové Researchers and Collaborators. Comprehensive review of cardiovascular toxicity of drugs and related agents. Med Res Rev. 2018;38(4):1332-403.
2. Güler E, Babur Güler G, Yavuz C, Kızılırmak F. An unknown side effect of isotretinoin: pericardial

effusion with atrial tachycardia. Anatol J Cardiol. 2015;15(2):168-9.
3. Zito PM, Mazzoni T. Acitretin. In: StatPearls. Treasure Island (FL): StatPearls Publishing. Available at: https://www.ncbi.nlm.nih.gov/books/NBK519571/. Accessed on 25 September 2022.
4. DeAngelo S, Mann KI, Abdulbasit M, Ahnert A, Sundlöf DW. A case report: rapid progression of coronary atherosclerosis in a patient taking Targretin (Bexarotene). Cardio-Oncology. 2020;6:31.
5. Liu Q, Van Bortle K, Zhang Y, Zhao MT, Zhang JZ, Geller BS, et al. Disruption of mesoderm formation during cardiac differentiation due to developmental exposure to 13-cis-retinoic acid. Sci Rep. 2018;8(1):12960.
6. Zhang S, Bai YY, Luo LM, Xiao WK, Wu HM, Ye P. Association between serum homocysteine and arterial stiffness in elderly: A community-based study. J Geriatr Cardiol. 2014;11:32-8.
7. Bălănescu AR, Bojincă VC, Bojincă M, Donisan T, Bălănescu SM. Cardiovascular effects of methotrexate in immune-mediated inflammatory diseases. Exp Ther Med. 2019;17(2):1024-9.
8. Brown G, Boldt C, Webb JG, Halperin L. Azathioprine-induced multisystem organ failure and cardiogenic shock. Pharmacotherapy. 1997;17:815-8.
9. Dogan P, Grbovic E, Inci S, Bayraktar F, Cagli K. Azathioprine-induced atrial fibrillation. Intractable Rare Dis Res. 2015;4(4):207-9.
10. Allison AC, Eugui EM. Preferential suppression of lymphocyte proliferation by mycophenolic acid and predicted long-term effects of mycophenolate mofetil in transplantation. Transplant Proc. 1994;26:3205-10.
11. Mbaebie N, Shaw P. Chaos in an Attempt to Appease- A Case of Mycophenolate Induced Supraventricular Tachycardia. J Clin Stud Med Case Rep. 2018;5:055.
12. Braun-Dullaeus RC, Feussner M, Walker G, Hopmann H, Kraemer HJ, Grimminger F, et al. Cyclosporine-induced Coronary Artery Constriction-Dissociation between Thromboxane Release and Coronary Vasospasm. J Heart Lung Transplant. 1999;18(4):328-35.
13. Ding B, Price RL, Borg TK, Weinberg EO, Halloran PF, Lorell BH. Pressure Overload Induced Severe Hypertrophy in Mice Treated with Cyclosporine, an Inhibitor of Calcineurin. Circ Res. 1999;84:729-34.
14. Sanghi P, Ahmad M. A case of atrial fibrillation from cyclosporine toxicity. Indian Pacing Electrophysiol J. 2004;4(1):40-2.
15. Floyd JD, Nguyen DT, Lobins RL, Bashir Q, Doll DC, Perry MC. Cardiotoxicity of cancer therapy. J Clin Oncol. 2005;23(30):7685-96.
16. Okuyan H, Altın C. Heart failure induced by itraconazole. Indian J Pharmacol. 2013;45(5):524-5.
17. Ahmad SR, Singer SJ, Leissa BG. Congestive heart failure associated with itraconazole. Lancet 2001;357:1766-7.
18. Fung SL, Chau CH, Yew WW. Cardiovascular adverse effects during itraconazole therapy. Eur Res J. 2008;32(1):240.
19. Okamoto J, Fukunami M, Kioka H. Frequent premature ventricular contractions induced by itraconazole. Circ J. 2007;71:1323-5.
20. Sharkey PK, Rinaldi MG, Dunn JF, Hardin TC, Fetchick RJ, Graybill JR. High dose itraconazole in the treatment of severe mycoses. Antimicrob Agents Chemother. 1991;35:707-13.
21. Nelson MR, Smith D, Erskine D, Gazzard BG. Ventricular fibrillation secondary to itraconazole induced hypokalaemia. J Infect. 1993;26:348.
22. Tholakanahalli VN, Potti A, Hanley JF, Merliss AD. Fluconazole-induced torsade de pointes. Ann Pharmacother. 2001;35(4):432-4.
23. Aris P, Wei Y, Mohamadzadeh M, Xia X. Griseofulvin: An Updated Overview of Old and Current Knowledge. Molecules. 2022;27(20):7034.
24. Mok NS, Lo YK, Tsui PT, Lam CW. Ketoconazole induced torsades de pointes without concomitant use of QT interval-prolonging drug. J Cardiovasc Electrophysiol. 2005;16(12):1375-7.
25. Sanches BF, Nunes P, Almeida H, Rebelo M. Atrioventricular block related to liposomal amphotericin B. BMJ Case Rep. 2014;2014:bcr2013202688.
26. Ray WA, Murray KT, Meredith S, Narasimhulu SS, Hall K, Stein CM. Oral erythromycin and the risk of sudden death from cardiac causes. N Engl J Med. 2004;351(11):1089-96.
27. Jayaweera DT. Minimising the dosage-limiting toxicities of foscarnet induction therapy. Drug Saf. 1997;16(4):258-66.
28. Olasińska-Wiśniewska A, Olasiński J, Grajek S. Cardiovascular safety of antihistamines. Postepy Dermatol Alergol. 2014;31(3):182-6.
29. Kim JH, Cha HR, Ha EK, Kwak JH, Kim H, Shin J, et al. Association between First-Generation Antihistamine Use in Children and Cardiac Arrhythmia and Ischemic Heart Disease: A Case-Crossover Study. Pharmaceuticals. 2023;16(8):1073.
30. Gagnon LR, Sadasivan C, Yogasundaram H, Oudit GY. Review of Hydroxychloroquine Cardiotoxicity: Lessons From the COVID-19 Pandemic. Curr Heart Fail Rep. 2022;19(6):458-66.
31. Fram G, Wang DD, Malette K, Villablanca P, Kang G, So K, et al. Cardiac Complications Attributed to Hydroxychloroquine: A Systematic Review of the Literature Pre-COVID-19. Curr Cardiol Rev. 2021;17(3):319-27.
32. Buttgereit F, Scheffold A. Rapid glucocorticoid effects on immune cells. Steroids. 2002;67:529-34.
33. Pitzalis C, Pipitone N, Perretti M. Regulation of leukocyte-endothelial interactions by glucocorticoids. Ann N Y Acad Sci. 2002;966:108-18.
34. Hafezi-Moghadam A, Simoncini T, Yang E, Limbourg FP, Plumier JC, Rebsamen MC, et al. Acute cardiovascular protective effects of corticosteroids are mediated by non-transcriptional

activation of endothelial nitric oxide synthase. Nat Med. 2002;8:473-9.
35. Sholter DE, Armstrong PW. Adverse effects of corticosteroids on the cardiovascular system. Can J Cardiol. 2000;16(4):505-11.
36. Pujades-Rodriguez M, Morgan AW, Cubbon RM, Wu J. Dose-dependent oral glucocorticoid cardiovascular risks in people with immune-mediated inflammatory diseases: a population-based cohort study. PLoS medicine. 2020;17(12):e1003432.
37. Souverein PC, Berard A, Van Staa TP, Cooper C, Egberts ACG, Leufkens HGM, et al. Use of oral glucocorticoids and risk of cardiovascular and cerebrovascular disease in a population based case-control study. Heart. 2004;90:859-65.
38. Jain R, Bali H, Sharma VK, Kumar B. Cardiovascular effects of corticosteroid pulse therapy: A prospective controlled study on pemphigus patients. Int J Dermatol. 2005;44:285-8.
39. Akikusa JD, Feldman BM, Gross GJ, Silverman ED, Schneider R. Sinus bradycardia after intravenous pulse methylprednisolone. Pediatrics. 2007;119:e778-82.
40. Dashore S, Pande S, Borkar M, Pande A. Late onset bradycardia: An unusual Side-Effect of high dose dexamethasone pulse therapy in patients of pemphigus vulgaris: A case series of five patients. Indian J Drugs Dermatol. 2015;1:23-6.
41. Weber MA, Schiffrin EL, White WB, Mann S, Lindholm LH, Kenerson JG, et al. Clinical practice guidelines for the management of hypertension in the community: a statement by the American Society of Hypertension and the International Society of Hypertension. J Clin Hypertens (Greenwich). 2014;16:14-26.
42. Dlova NC, Jacobs T, Singh S. Pericardial, pleural effusion and anasarca: A rare complication of low-dose oral minoxidil for hair loss. JAAD Case Rep. 2022;28:94-6.
43. Patil VB, Lunge SB, Doshi BR. Cardiac side effect of rituximab. Indian J Drugs Dermatol. 2020;6:49-52.
44. Verma SK. Updated cardiac concerns with rituximab use: A growing challenge. Indian Heart J. 2016;68 Suppl 2:S246-8.
45. Rajagopalan M, Vasani R. Rituximab in the treatment of skin diseases. Indian J Drugs Dermatol. 2017;3(2):105.
46. Iribarren C, Rahmaoui A, Long AA, Szefler SJ, Bradley MS, Carrigan G, et al. Cardiovascular and cerebrovascular events among patients receiving omalizumab: Results from EXCELS, a prospective cohort study in moderate to severe asthma. J Allergy Clin Immunol. 2017;139(5):1489-1495.e5.
47. Narukonda S, Vinod NR, Joshi M. A Case of Pulmonary Vein Thrombosis Associated with Treatment of Omalizumab. J Investig Med High Impact Case Rep. 2017;5(3):2324709617724176.
48. Aguiar-Ricardo I, Nunes-Ferreira A, Roda Â, Bras-Rosario L. Omalizumab induced Takotsubo syndrome: case report. Eur Heart J Case Rep. 2019;3(1):yty155.
49. Mann DL. Targeted anticytokine therapy and the failing heart. Am J Cardiol. 2005;95:9C-16C.
50. Senel S, Cobankara V, Taskoylu O, Karasu U, Karapinar H, Erdis E, et al. The safety and efficacy of etanercept on cardiac functions and lipid profile in patients with active rheumatoid arthritis. J Investig Med. 2012;60(1):62-5.
51. Khan MA, Dormand H, Neyses L, Mamas MA. Heart Failure with Etanercept Therapy: A Case Report. J Clin Exp Cardiol. 2013;4:236.
52. Martini, S, Masood, F, Chaus, A. Bone fixer or heart breaker: new onset heart failure in the setting of etanercept therapy. J Am Coll Cardiol. 2020;75 (11_Suppl_1):2588.
53. Mansitó López C, Torres Laboy P, Ortiz Bou M, Quintero Noriega A, Cintron Rivera V. Fatal New-Onset Congestive Heart Failure Related to Adalimumab Use in a Patient with Relapsing Hidradenitis Suppurativa: A Case Report. Am J Case Rep. 2021;22:e929148.
54. Kavanaugh A, Keystone EC. The safety of biologic agents in early rheumatoid arthritis: Clin Exp Rheumatol. 2003;21(5 Suppl. 31);S203-8.
55. Chung ES, Packer M, Lo KH, Fasanmade AA, Willerson JT, et al. Randomized, double-blind placebo-controlled pilot trial of infliximab, a chimeric monoclonal antibody to tumor necrosis factor-alpha, in patients with moderate-to-severe heart failure: Results of the anti-TNF Therapy Against Congestive Heart Failure (ATTACH) trial. Circulation. 2003;107(25):3133-40.
56. Fukuhara M, Matsumura K, Ohmori S, Yanai T, Tsubota Y, Abe I, et al. Effects of interferon on circadian changes in blood pressure and heart rate variability in patients with chronic hepatitis. Am J Hypertension. 1999;12(5):519-23.
57. Nishio K, Arase T, Tada H, Tachibana H. Interferon related pericarditis: Review. World J Cardiol. 2017;9(6):553-7.
58. Tufekci S, Coban A, Bor M, Yasa B, Nisli K, Ince Z. Cardiac rhythm abnormalities during intravenous immunoglobulin G(IVIG) infusion in two newborn infants: coincidence or association? Clin Case Rep. 2015;3(9):731-4.
59. Melhem RA, Mustafa A, Majzoub M, Jalloul Y, Lafferty J, Grovu R, et al. Cardiac sequelae of intravenous immunoglobulin infusion. J Allerg Clin Immunol. 2023;151(2):AB330.

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