

Case Report

ST-Segment Elevation in Methamphetamine-Users Adolescents: An Emerging Treatment Paradigm, Case Report

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Abstract

Antecedents: Objective: Present the electrocardiographic changes secondary to methamphetamine use in an adolescent. Given that this is a pioneer case in the national literature, we consider its dissemination appropriate. **Case Report:** A 17-year-old obese female adolescent, smoker and methamphetamine (crystal) user, presented with ST-segment elevation (STE) 12 hours after a binge, without accompanying anginal equivalents. With the application of anti-ischemic and antithrombotic measures, improvement of the STE was observed on the follow-up electrocardiogram (ECG) 24 hours later. The patient did not develop cardiogenic shock or arrhythmias. Thrombolysis was not performed because she did not develop pathological Q waves. Total creatine phosphokinase (CPK) and creatine phosphokinase- MB (CPK-MB) levels were elevated, due to rhabdomyolysis. **Conclusions:** ST-segment elevation in methamphetamine users has been termed benign coronary spasm by some authors; however, due to the risk of thromboinflammation at the glycocalyx level, the problem could out-come obstructive. There is no consensus regarding treatment in pediatric patients. Treatment in adults prioritizes vasodilation and antithrombotic measures, especially in presence of cardiovascular risk factors such as obesity and smoking.

Keywords

ST-segment Elevation, Methamphetamines, Electrocardiogram, Thromboinflammation

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1. Introduction

In recent decades, methamphetamine use in Mexico has been rising at an alarming rate, establishing itself as a major public health problem. According to the Epidemiological Surveillance System for Addictions (SISVEA), methamphetamine ranks among the top substances for which people seek care in emergency departments and rehabilitation centers, coming in sixth place (38.4%) behind alcohol, tobacco, and marijuana [1]. These substances, due to their low cost and easy accessibility, pose a significant risk to vulnerable populations. In the study by Uhlmann et al., which included 205 participants aged 14 to 26 years who were experiencing homelessness, the median age at initiation of substance use was 16 years. The associated factors were previous crack cocaine use, involvement in drug trafficking, and male sex. [2].

Methamphetamines are sympathomimetic amines that increase intrasynaptic levels of monoamines, primarily dopamine, norepinephrine, and serotonin. This effect produces a clinical picture characterized by intense central nervous system stimulation, euphoria, and hallucinations, as well as cardiovascular, neurological, metabolic, and gastrointestinal effects. [3].

Cardiovascular manifestations are particularly severe and represent one of the leading causes of death associated with methamphetamine use [4]. These include acute coronary syndromes: acute myocardial infarction, atrial and ventricular arrhythmias, circulatory collapse, malignant hypertension, aortic dissection, pulmonary hypertension, and cardiomyopathy in chronic users [3, 5, 6].

1.1. Pathophysiology

Among the proposed mechanisms explaining the association between methamphetamine use and the occurrence of ischemic events are the following:

1.1.1. Sympathomimetic Activation

This produces positive inotropic and chronotropic effects, thereby increasing blood pressure, cardiac output, and left ventricular wall stress. Collectively, these effects increase myocardial oxygen demand. In addition, it induces vasoconstriction, which impairs coronary blood flow and consequently promotes the development of myocardial ischemia [7, 8].

1.1.2. Endothelial Dysfunction

In the particular case of cocaine, endothelial dysfunction has been described, promoting increased platelet activation in addition to the induction of coronary vasospasm [7]. This dysfunction has been demonstrated through multiple markers, including increased endothelial secretion of Von Willebrand factor, alterations in nitric oxide production and nitric oxide

synthase activity, as well as increased tissue factor and decreased expression of its inhibitor. These changes may persist even after cessation of use, thereby contributing to the development of accelerated atherosclerosis and a prothrombotic state [7]. In addition, glycocalyx damage may cause protein extravasation and tissue edema, further promoting the aforementioned processes [9].

Assessments using the TIMi Frame Count index have documented impaired microvascular flow even though patients have coronary arteries without significant lesions and preserved epicardial flow [7].

Although the exact mechanisms of methamphetamine-induced vasoconstriction have not been fully elucidated, current evidence suggests that this phenomenon may be mediated by the endothelial release of endothelin-1 or by the activation of the trace amine-associated receptor (TAAR1) at the arterial level, as well as by catecholamine-mediated adrenergic signaling. [4].

1.1.3. Hyperthermia

secondary to excessive activation of the sympathetic nervous system or direct toxic effects on muscle cells. These mechanisms include the induction of oxidative stress and mitochondrial dysfunction, which may lead to apoptosis and fibrosis of cardiac myocytes, clinically manifesting as cardiotoxicity [8].

1.2. Electrocardiographic (ECG) Manifestations

In adults, tachyarrhythmias, right-axis deviation, left ventricular hypertrophy, pathological Q waves, and a prolonged QT interval have been reported. Echocardiography has documented severe systolic dysfunction, left ventricular thrombus, functional mitral regurgitation, infective endocarditis, and even severe pulmonary hypertension [10].

In a recent study, more than 71% of methamphetamine users presented with some form of ECG abnormality compared with individuals presenting with chest pain but without a history of methamphetamine use. Ischemic changes were the most frequently identified findings, including T-wave inversion in the lateral leads and Q waves in the inferior leads [10].

Electrocardiographic changes in methamphetamine users have been described in adults; however, we found no similar cases reported in the pediatric population. Therefore, we consider the presentation of this case to be warranted.

2. Case Report

A 17-year-old female patient was brought to the emergency department by paramedics after being found in her room with

loss of consciousness and vomiting. She had a history of continuous methamphetamine use since the previous afternoon.

Her parents reported that she had been using cocaine, marijuana, and lysergic acid diethylamide (LSD) for the past 6 months and had engaged in self-injurious behavior (cutting) for the past 3 years, in the context of family dysfunction, with a mother diagnosed with schizophrenia. On arrival at the emergency department, the patient was obese, with the following vital signs: weight 87 kg, height 1.72 m, body mass index (BMI) 29.4 kg/m², blood pressure (BP) 105/75 mmHg (MAP 85 mmHg), heart rate (HR) 135 bpm, respiratory rate (RR) 26 breaths/min, temperature 38.7 °C, and oxygen saturation (SpO₂) 99% with oxygen at 3 L/min via nasal cannula. Glasgow Coma Scale score was 11, with dilated and sluggishly reactive pupils. Pulmonary examination revealed no crackles or wheezing. Cardiac auscultation revealed regular heart sounds and tachycardia, without an S3 gallop. The abdomen was globose due to adipose tissue, with old linear scars consistent with cutting.

Initial laboratory tests revealed KDIGO stage 2 acute kidney injury, with a BUN/creatinine ratio of 16. Hydration was

initiated at 35 mL/kg/day due to the risk of rhabdomyolysis. The McMahon Score was 7.5 points, indicating a low risk of requiring renal replacement therapy. Fluid balance was positive at +1,715 mL. Urine output was 1,750 mL over 14 hours (1.43 mL/kg/h). Persistent fever (38.5 °C) was noted, with no active bleeding or evident infectious source. The electrocardiogram showed ST-segment elevation suggestive of subepicardial injury in leads I-III, V1-V6 and aVF. Lead II showed the presence of a J (Osborn) wave, without Q waves, and with a prolonged QTc interval (0.578 seconds) (Figure 1).

Initial creatine phosphokinase (CPK) was 4,723 U/L, with CK-MB of 126.3 U/L and a CK-MB relative index of 2.6%. Serial enzyme measurements showed an increase in CK-MB on the second and third determinations, reaching 1,533 U/L. In subsequent measurements, these enzymes could not be quantified by the laboratory because their levels exceeded the analytical range of the equipment. Elevated liver enzymes were also observed, with ALT of 59.2 U/L and AST of 237.9 U/L. Serum electrolytes: K 4.36, Cl 112.0, Na 146.9, Ca 9.8.

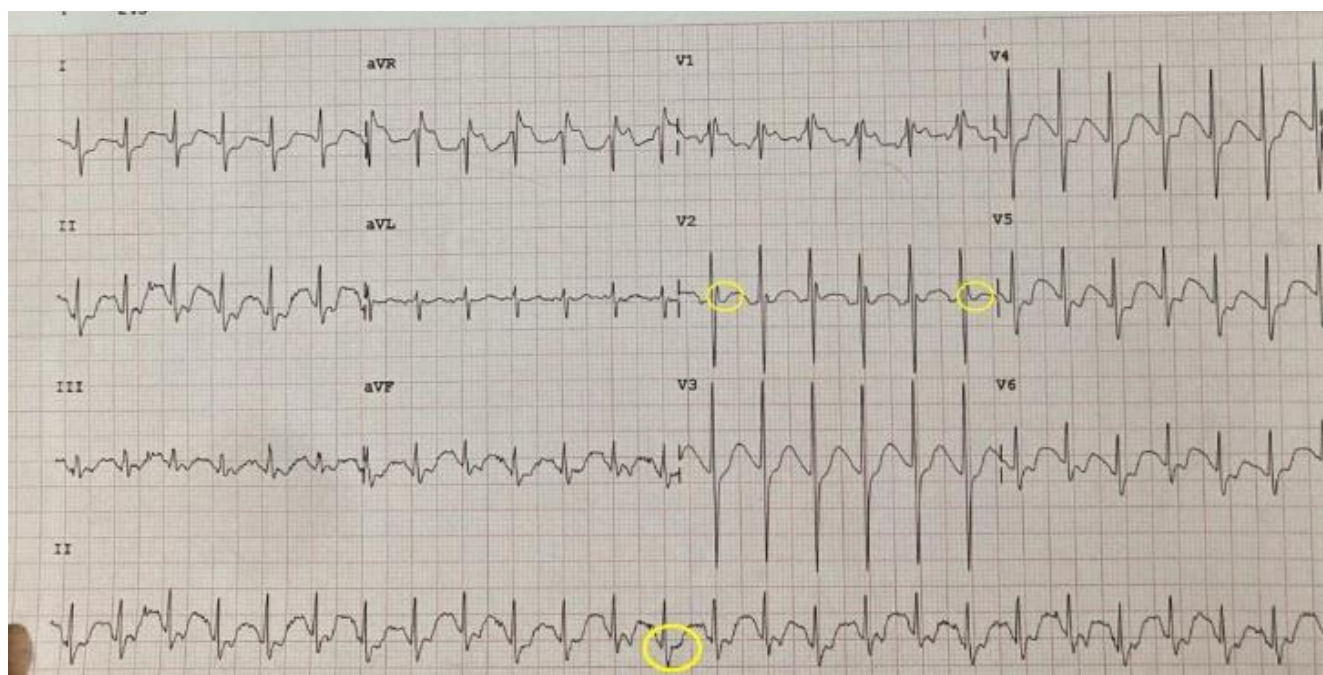


Figure 1. First electrocardiogram (June 7, 2023). Significant ST-segment elevation in the inferior leads (I, II, III, and aVF) and in leads V1-V6, with no Q waves. Sinus rhythm with an average ventricular rate of 125 beats/min and a prolonged QTc interval (0.578 seconds). An Osborn J wave is observed in lead II.

Due to persistent fever above 38.5 °C, a dual antipyretic regimen (metamizole and paracetamol) was initiated, and intravenous fluids were adjusted to 70 mL/kg/day. Urine toxicology screening was positive for methamphetamine. Given the ST-segment elevation, coronary protective measures were initiated with a 300 mg loading dose of clopidogrel, followed by

75mg/24 hours; atorvastatin 80mg/24 hours, heparin 80mg/24 hours, and a nitroglycerin patch. A reduction in ST-segment elevation was observed after 24 hours, with no pathological Q waves. QTc prolongation persisted, but no arrhythmias developed (Figure 2).

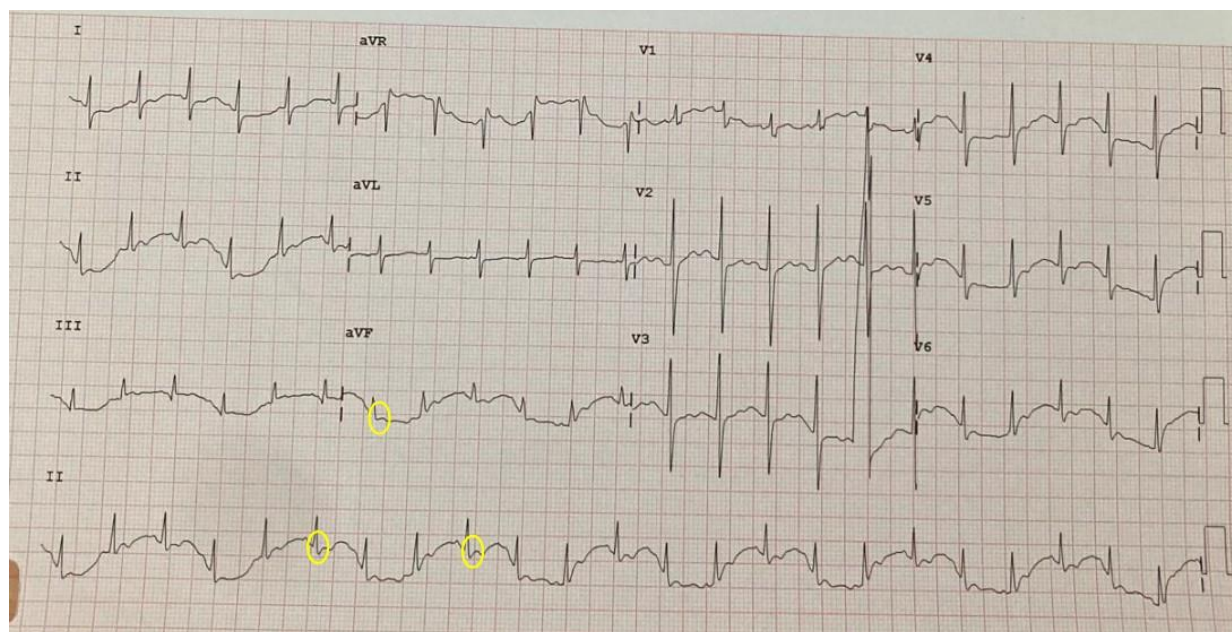


Figure 2. Follow-up electrocardiogram at 24 hours. A decrease in ST-segment elevation in DI and inferior leads were observed, with persistent QTc prolongation and no Q waves.

3. Discussion

In adult methamphetamine users, cardiovascular complications are well documented as one of the most common reasons for seeking care in emergency departments [11]. ST-segment elevation in the absence of acute myocardial infarction (AMI) has been described as “benign coronary spasm” [3].

In a series of young adults and adolescents conducted by Menyar Ayman A. [13], with a minimum age of 18 years, the prevalence of AMI was 11%; among these patients, 5% had normal coronary arteries, as demonstrated by imaging studies.

In the case that we presented, the electrocardiogram (Figure 1) showed extensive ST-segment elevation, along with an Osborn J wave and QTc prolongation (0.578 seconds) [15], in leads I, II, III, aVF, V3-V6, without pathological Q waves [12-14]. This electrocardiographic pattern was compatible with benign coronary spasm. In this setting, the literature recommends prioritizing coronary vasodilation, while the use of coronary-protective interventions should be guided by the experience and protocols of the treating institution. Thrombolysis is recommended in selected cases [13, 19].

During the inflammatory state induced by chemical injury, the glycocalyx undergoes damage and degradation, leading to thromboinflammation, which has been associated with intravascular thrombosis and acute myocardial infarction (AMI) [16]. As noted in the literature, the therapeutic approach should focus on coronary vasodilation. However, given that the patient had two cardiovascular risk factors—smoking and obesity—coronary-protective measures were initiated [17, 18].

A vasodilator, topical nitroglycerin, and anti-ischemic ther-

apy—including statins, low-molecular-weight heparin, aspirin, and clopidogrel—were administered. The follow-up electrocardiogram at 24 hours showed improvement in ST-segment elevation, although the Osborn deflection persisted in lead II. [15]. This positive deflection occurring before the T wave has been associated, among other conditions, with myocardial ischemia. Thrombolysis was not performed because the patient did not develop pathological Q waves [13, 19, 20].

Although the QTc remained prolonged (0.578 seconds), the patient did not develop any arrhythmias. Methamphetamine users have been reported to exhibit QTc interval abnormalities in up to 22% of cases [10].

Due to the patient’s altered neurological status, it was not possible to determine whether she experienced chest pain or anginal equivalents. In a series by Turnipseed SD, 8% of methamphetamine users presented with chest pain; furthermore, 25% developed acute coronary syndrome and 8% experienced cardiac complications [6].

Several case reports in young adults support the occurrence of acute myocardial infarction (AMI) following methamphetamine use. One such report described a 28-year-old patient who developed an extensive anterior AMI secondary to coronary thrombosis and amphetamine-induced coronary spasm, ultimately requiring heart transplantation [21-23].

Global refractory microvascular spasm has also been described as a cause of AMI in young patients without angiographic evidence of coronary occlusion [21]. Extreme hyperthermia may also cause ST-segment elevation in the absence of AMI among chronic methamphetamine users, as reported by Schussler et al. in a 37-year-old male patient with a temperature of 42.4 °C [8, 24, 25].

4. Conclusions

ST-segment elevation without pathological Q waves secondary to methamphetamine use may be associated with transient benign coronary spasm. In pediatric population, there is currently no consensus regarding its management; based on experience in adults, treatment prioritizes coronary vasodilation, prevention of thromboinflammation and IAM as result as obstructive coronary. In settings where cardiac imaging studies are unavailable, management remains challenging and should be guided by the experience and protocols of each healthcare center. An electrocardiogram should be obtained in every pediatric patient with methamphetamine exposure to facilitate the systematic assessment and management of potential cardiac abnormalities.

Abbreviations

STE	ST-segment Elevation
ECG	Electrocardiogram
CPK	Creatine Phosphokinase
CPK-MB	Creatine Phosphokinase-MB
SISVEA	Epidemiological Surveillance System for Addictions
TAAR1	Trace Amine-associated Receptor
LSD	Lysergic Acid Diethylamide
BMI	Body Mass Index
BP	Blood Pressure
HR	Heart Rate
RR	Respiratory Rate
SpO ₂	Oxygen Saturation

Author Contributions

Eduardo Quintero: Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Project administration, Supervision, Validation, Writing – review & editing

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Carolina Herrera: Data curation, Formal Analysis, Visualization, Writing – original draft

Ingrid Cabrera: Writing – original draft, Data curation, Formal Analysis

Ivonne Lopez: Data curation, Formal Analysis, Validation, Writing – review & editing

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Conflicts of Interest

The authors declare no conflicts of interest.

References

- [1] Secretaría de Salud, Dirección General de Epidemiología. (2026). Informe SISVEA 2024: Sistema de Vigilancia Epidemiológica de las Adicciones. Gobierno de México. https://www.gob.mx/cms/uploads/attachment/file/1053432/Informe_SISVEA2024.pdf
- [2] Uhlmann S, DeBeck K, Simo A, Kerr T, Montaner JS, Wood E. Crystal methamphetamine initiation among street-involved youth. *J Psychoactive Drugs*. 2014; 46(3): 198-207. <https://doi.org/10.3109/00952990.2013.836531>
- [3] Paratz E, Cunningham N, MacIsaac A The Cardiac Complications of Methamphetamines Heart, Lung and Circulation, 2015; 25, 325-332 Miranda CH, Schmidt A, Filho AP, Borges.
- [4] Kevil CG, Goeders NE, Woolard MD, Bhuiyan MS, Dominic P, Kolluru GK, et al. Methamphetamine use and cardiovascular disease. *Arterioscler Thromb Vasc Biol*. 2019; 39(9): 1739-46. <https://doi.org/10.1161/ATVBAHA.119.312461>
- [5] Chen J. P. (2007). Methamphetamine-associated acute myocardial infarction and cardiogenic shock with normal coronary arteries: refractory global coronary microvascular spasm. *The Journal of invasive cardiology*, 19(4), E89-E92.
- [6] Turnipseed, S. D., Richards, J. R., Kirk, J. D., Diercks, D. B., & Amsterdam, E. A. (2003). Frequency of acute coronary syndrome in patients presenting to the emergency department with chest pain after methamphetamine use. *The Journal of emergency medicine*, 24(4), 369-373. [https://doi.org/10.1016/s0736-4679\(03\)00031-3](https://doi.org/10.1016/s0736-4679(03)00031-3)
- [7] Fuenzaiida A, Valdebenito M, Fajuri A. Acute Myocardial infarction associated with cocaine use: a different entity? *Chilean Journal of Cardiology*. 2014; 33(2): 123-126. <https://doi.org/10.4067/S0718-8562014000200006>
- [8] Schussler JM, Tomson C, Dresselhouse MP. Extreme Hyperthermia From Methamphetamine Toxicity Presenting as ST-Elevation Myocardial Infarction on Electrocardiogram: A Case Report Written With ChatGPT Assistance. *Cureus*. 2023 Mar 13; 15(3): e36101. <https://doi.org/10.7759/cureus.36101>
- [9] Miranda, C. H., Schmidt, A., Filho, A. P., & Borges, M. C. (2015). Endothelial glycocalyx damage in patients with acute coronary syndrome. *Critical Care*, 19(Suppl 2), P4. <https://doi.org/10.1186/cc14660>
- [10] Paratz ED, Zhao J, Sherwen AK, Scarlato RM, MacIsaac AI. Is an abnormal ECG just the tip of the ICE-berg? Examining the utility of electrocardiography in detecting methamphetamine-induced cardiac pathology. *Heart Lung Circ*. 2017 Jul; 26(7): 684-9. <https://doi.org/10.1016/j.hlc.2016.11.004>
- [11] Quintero-Aguirre, E., Balbuena-Robles, L. H., Barrera-Villagas, G., & Serna, D. (2015). Dolor precordial en los adolescentes y diagnostico diferencial de la onda T. *Revista Mexicana de Pediatr* 82(1), 14-17. <https://www.medigraphic.com/pdfs/pediat/sp-2015/sp151d.pdf>

- [12] El Menyar A. A. (2006). Drug-induced myocardial infarction secondary to coronary artery spasm in teenagers and young adults. *Journal of postgraduate medicine*, 52(1), 51-56.
- [13] Towbin J, Bricker J, Garson A. Electrocardiographic criteria for diagnosis of acute myocardial infarction in childhood. *American Journal of Cardiology*, 69, 1545-1548.
- [14] Machado Villarreal, L., & Olmos Granados, H. M. (2022). Marcadores Biologicos en el diagnostico del infarto agudo al miocardio. *Expresiones Medicas*, 9(3), 7-13. Recuperado a partir de. <https://erevistas.uacj.mx/ojs/index.php/expemed/article/view/5368>
- [15] Maruyama, M., Kobayashi, Y., Kodani, E., Hirayama, Y., Atarashi, H., Katoh, T., & Takano, T. (2004). *Osborn waves: History and significance*. *Japanese Heart Journal*, 45(4), 505-511.
- [16] Iba T, Maier CL, Helms J, Ferrer R, Thachil J, Levy JH. Managing sepsis and septic shock in an endothelial glycocalyx-friendly way: from the viewpoint of surviving sepsis campaign guidelines. *Ann Intensive Care*. 2024; 14: 64. <https://doi.org/10.1186/s13613-024-01301-6>
- [17] Nehus, E., & Mitsnefes, M. (2019). Childhood Obesity and the Metabolic Syndrome. *Pediatric clinics of North America*, 66(1), 31-43. <https://doi.org/10.1016/j.pcl.2018.08.004>
- [18] Franks, P. W., Hanson, R. L., Knowler, W. C., Sievers, M. L., Bennett, P. H., & Looker, H. C. (2010). Childhood obesity, other cardiovascular risk factors, and premature death. *The New England journal of medicine*, 362(6), 485-493. <https://doi.org/10.1056/NEJMoa0904130>
- [19] Suryawanshi, S. P., Das, B., & Patnaik, A. N. (2011). Myocardial infarction in children: Two interesting cases. *Annals of pediatric cardiology*, 4(1), 81-83. <https://doi.org/10.4103/0974-2069.79633>
- [20] Akhter MW, Aronow WS, Rashid M, Fredrick T, Campbell M. Amphetamine-related acute myocardial infarction requiring cardiac transplant. *Int J Clin Pract*. 2003 Jan; 57(1): 9-1.
- [21] Yeo KK, Mooney J, Wyrick K, Amsterdam EA. Methamphetamine-associated acute coronary syndrome: spasm versus thrombosis. *Int J Cardiol*. 2007 Apr 4; 121(1): e3-e4. <https://doi.org/10.1016/j.ijcard.2006.09.014>
- [22] Khareshi I, Mahjoob MP, Esmaeeli S, Eslami V, Haybar H. Simultaneous Thrombosis of the Left Anterior Descending Artery and the Right Coronary Artery in a 34-Year-Old Crystal Methamphetamine Abuser. *Korean Circ J*. 2015 Mar; 45(2): 158-160. <https://doi.org/10.4070/kcj.2015.45.2.158>
- [23] Westover, A. N., Nakonezny, P. A., & Haley, R. W. (2008). Acute myocardial infarction in young adults who abuse amphetamines. *Drug and alcohol dependence*, 96(1-2), 49-56. <https://doi.org/10.1016/j.drugalcdep.2008.01.027>
- [24] Khattab, E., & Shujaa, A. (2013). Amphetamine abuse and acute thrombosis of left circumflex coronary artery. *International Journal of Case Reports and Images*, 4(12), 698-701. <https://doi.org/10.5348/ijcri-2013-12-414-CR-8>
- [25] Abdullah, C. S., Aishwarya, R., Alam, S., Morshed, M., Remex, N. S., Nitu, S., Kolluru, G. K., Traylor, J., Miriyala, S., Panchatcharam, M., Hartman, B., King, J., Bhuiyan, M. A. N., Chandran, S., Woolard, M. D., Yu, X., Goeders, N. E., Dominic, P., Arnold, C. L., Stokes, K., Bhuiyan, M. S. (2020). Methamphetamine induces cardiomyopathy by Sigmar1 inhibition-dependent impairment of mitochondrial dynamics and function. *Communications biology*, 3(1), 682. <https://doi.org/10.1038/s42003-020-01408-z>