Mini Review

iMedPub Journals
http://www.imedpub.com

Journal of Clinical Nutrition & Dietetics
ISSN 2472-1921

2016
Vol. 2 No. 3: 17

DOI: 10.4172/2472-1921.100024

Undervalued Significance of Moderate Enzyme Elevation in Paradoxical Coronary Embolism Secondary to Deep Vein Thrombosis: Short Review

Puneeth Shridhar[1], David Lasorda[2] and Youngjae Chun[1,3,4]

1 Department of Bioengineering, University of Pittsburgh, Pittsburgh, USA
2 Department of Cardiology, Allegheny General Hospital, Pittsburgh, USA
3 Department of Industrial Engineering, University of Pittsburgh, Pittsburgh, USA
4 McGowan Institute for Regenerative Medicine, Pittsburgh, USA

Corresponding author: Puneeth Shridhar

pus8@pitt.edu

University of Pittsburgh, Bioengineering, 3700 O'Hara Street, Benedum Hall of Engineering, Pittsburgh, PA 15261, USA.

Tel: +16502769952

Abstract

Myocardial infarctions (MI) are relatively common in people with deep vein thrombosis (DVT). In fact, the rate of myocardial infarction has been reported to be greater in adults with DVT as compared to those without DVT and recent studies have shown interest in quantifying the relationship with MI and DVT. However, cryptogenic nature of MI in these individuals is underestimated. Approximately one-fourth of the US populations are estimated to have a patent foramen ovale (PFO). Of note, since MI being the single largest cause of mortality, a large number of these patients have a PFO. The high percentage of myocardial infarction in patients with a PFO has not been addressed in the literature appropriately before and might have been overlooked because most of the patients with infarctions had an uncomplicated course and only moderate enzyme elevation.

Keywords: Cardiac enzymes; Paradoxical embolism; Myocardial infarction

Received: July 31, 2016; **Accepted:** September 07, 2016; **Published:** September 13, 2016

Citation: Shridhar P, Lasorda D, Chun Y. Undervalued Significance of Moderate Enzyme Elevation in Paradoxical Coronary Embolism Secondary to Deep Vein Thrombosis: Short Review. J Clin Nutr Diet. 2016, 2:3.

Introduction

Patent foramen ovale (PFO) is a condition where an opening that exists between the left and right atria of the heart. This hole is present in everyone before birth, but most often the hole closes shortly after birth. As a result, shunting is seen which may be either right-left, left-right or both [1]. PFO with intracardiac shunting is associated with cryptogenic myocardial infarction (MI) in both young and elderly [2]. Many of them are found to have subclinical enzyme levels [3]. In individuals with a PFO, there are transient episodes of right-to-left intracardiac shunting associated with increased right atrial pressures that occur with Valsalva-like maneuvers, including coughing, forced expiration, or bowel movements. MI in this setting are thought to occur when a venous thromboembolism travels to the coronary arterial system after avoiding filtration in the lungs during right-to left shunting through the PFO, an event called a paradoxical coronary embolism (PCE) which was first described in 1877 [4].

Previous studies with improved imaging technology indicate that the subclinical myocardial infarctions determined in cardiac magnetic resonance imaging were observed as many as 10.8% of patients with PFO after a first cryptogenic cerebral ischemic event [5]. It is known that a MI can potentially disclose presence of PFO [6]. In addition, reports of silent myocardial infarct without overt cardiac symptoms, presumably related to patent foramen ovale have also been described [7]. The role of PFO closure in reducing silent cerebral ischemic events is well known [8]. However, not all PFOs may require treatment. Risk factors for PCE with PFO include previous or current pulmonary emboli, pregnancy, factor Leiden deficiency, ischemic stroke and various genetic mutations [9-12]. Nonetheless, treatment options for PFO remain controversial due to the risks associated with both transcatheter and surgical procedures.

An autopsy series of 1050 patients with myocardial infarctions by Prizel et al. did not find a single case of PCE among the 55 patients identified with coronary embolism [13]. The Navigator study estimated the prevalence of venous thromboembolism in patients with established cardiovascular disease or cardiovascular risk factors to be only 1.4% and the patients with venous thromboembolism had higher 5-year event rates

for the composite of death, myocardial infarction, and stroke, as compared with patients without venous thromboembolism (10.7% vs. 5.9%) [14].

Deep vein thrombosis (DVT) is associated with pathologic changes and physiologic adaptations that can lead to cryptogenic myocardial infarction in the setting of a PFO. For example, erythrocyte adhesion, endothelial damage, and platelet and coagulation activation in DVT result in a hypercoagulable state and increased susceptibility to venous thrombosis. Recently, coronary microvascular dysfunction has been linked to conditions that predispose to venous thrombosis such as myeloproliferative neoplasms (PV), and there is growing evidence that more and more venous hypercoagulable states increase the risk of myocardial infarction [15]. In the presence of PFO, the underlying hypercoagulability could lead to PCE as per our literature review **(Table 1)**. Loscalzo et al. has noted 9% of paradoxical emboli go to coronaries [16]. In addition, the presence of "thrombus in transit" in patients with DVT, either at baseline or during an episode of acute chest syndrome, could further increase the risk of PCE [17,18]. Indeed, till date, more than 100 cases of thrombus in transit have been described in literature. Most of the investigators concluded that further studies are warranted to determine if PFO is an independent risk factor for MI with DVT. Another study found that PFOs may be underestimated as possible causes of coronary embolic events even without venous hypercoagubilities [19].

Based on literature review of six isolated cases, we found out a large number of PCE can coexist or co-present with pulmonary embolism (PE) in patients with underlying DVT with varying cardiac enzyme values [17-23]. However, in few patients, MI can present independently without PE. In one of the cases, non-surgical closure of PFO was performed [19]. There are also studies which indicate coronary heart disease is a risk factor for PE in absence of clinically diagnosed DVT [24].

Future studies are needed to investigate the benefit and outcomes of nonsurgical PFO closure in patients with DVT and MI. It will also be important to examine the relationship between DVT and PFO in both young adults and elderly. Unfortunately, current approaches to MI prevention for elderly with DVT have been limited to secondary prevention because of the lack of methods for identifying patients at increased risk for developing their first MI. PCE prevention is especially important in young adults given the impact on physical function. For young adults, DVT and MI can lead to disability, morbidity and mortality. The devastating outcomes of acute MI in patients with DVT demand further study, not only to define additional risk factors, but also to identify successful interventions. Although further research is needed, the positive outcome of our patients with DVT suggests that identifying PFOs and closing them non-surgically could be beneficial in selected patients.

Conclusion

Similar to cryptogenic stroke secondary to deep vein thrombosis (DVT), patients with DVT associated cryptogenic myocardial infarction are at higher risk for recurrent paradoxical embolism and they should be considered for PFO closure. Presence of moderate enzyme elevation should not be a ground for denial for closure.

Table 1 Hypercoagulability and patent foramen ovale leading to paradoxical coronary embolism.

Author	Age/Sex	Associated Emboli	Risk Factor	Closure	Stent	Deep Vein Thrombosis
Farhang P	43/M	PE	Smoking	No	No	No
Ramineni	29/F	PE	Pregnancy	Yes	Angiojet, DES	IVC Filter
Murthy A	29/F	Presumed PE	-	No	Aspiration Alone	Warfarin
Edibam C	30/F	PE, Cerebral and Splenic emboli	Delayed Hip Fracture Reduction, Prothrombin 20210 Mutation	No	-	IVC Filter
Haghi	61/F	PE	Factor Leiden	No	LMW Heparin	-
de Swiet J	23/F	PE, cerebral emboli	Oral Contractive pills	-	-	Vein ligation

References

1 Di Tullio M, Sacco RL, Gopal A, Mohr JP, Homma S (1992) Patent foramen ovale as a risk factor for cryptogenic stroke. Ann Intern Med 117: 461-465.

2 Neisius U, Northridge DB, Cruden NL, Denvir MA (2015) Myocardial infarction associated with patent foramen ovale and paradoxical embolism: A case series. Int J Cardiol 180: 34-37.

3 Bruch L, Parsi A, Grad MO, Rux S, Burmeister T, et al. (2002) Transcatheter closure of interatrial communications for secondary prevention of paradoxical embolism: single-center experience. Circulation 105: 2845-2848.

4 Wachsman DE, Jacobs AK (2002) Paradoxical coronary embolism: a rare cause of acute myocardial infarction. Rev Cardiovascular Med 4: 107-111.

5 Wöhrle J, Kochs M, Hombach V, Merkle N (2010) Prevalence of myocardial scar in patients with cryptogenic cerebral ischemic events and patent foramen ovale. JACC Cardiovasc Imaging 3: 833-839.

6 Trabattoni D, Zaro T, Garducci S (2012) A myocardial infarction may disclose patent foramen ovale. J Cardiol Cases 5: 80-82.

7 Duman D, Demirtunc R, Duman D, Sungur F, Agirbasli M, et al. (2006) Paradoxical mesentery embolism and silent myocardial infarction in primary antiphospholipid syndrome: a case report. Heart Surg Forum 9: 592-594.

8 Vigna C, Inchingolo V, Giannatempo G, Pacilli MA, Di Viesti P, et al. (2008) Clinical and brain magnetic resonance imaging follow-up after percutaneous closure of patent foramen ovale in patients with cryptogenic stroke. Am J Cardiol 101: 1051-1055.

9 Windecker S, Stortecky S, Meier B (2014) Paradoxical embolism. J Am Coll Cardiol 64: 403-415.

10 Croft AP, Khan JN, Chittari MV, Varma C (2012) Paradoxical coronary artery embolism causing acute myocardial infarction in a young woman withfactor V Leiden thrombophillia. J R Coll Physicians Edinb 42: 218-220.

11 Lee HY, Yoo SM (2013) A case of paradoxical air embolism in the coronary artery through a patent foramen ovaledemonstrated by coronary CT angiography. Clin Imaging 37: 167-169.

12 Carano N, Agnetti A, Hagler DJ, Tchana B, Squarcia U, et al. (2004) Acute myocardial infarction in a child: possible pathogenic role of patent foramen ovale associated with heritable thrombophilia. Pediatrics 114: 255-258.

13 Prizel KR, Hutchins GM, Bulkley BH (1978) Coronary artery embolism and myocardial infarctions. Ann Intern Med 88: 155-161.

14 Katz M, Califf RM, Sun J, McMurray JJ, Thomas L, et al. (2014) Venous Thromboembolism and Cardiovascular Risk: Results from the NAVIGATOR Trial. Am J Med 9343: 788-788.

15 Vianello F, Cella G, Osto E, Ballin A, Famoso G, et al. (2014) Coronary microvascular dysfunction due to essential thrombocythemia and policythemiavera: The missing piece in the puzzle of their increased cardiovascular risk? Am J Hematol 90: 109-113.

16 Loscalzo J (1986) Paradoxical embolism: clinical presentation, diagnostic strategies, and therapeutic options. Am Heart J 122: 141-145.

17 Farhang P, Ahmad A, Vazquez-Figueroa JG, Douglass P (2013) Simultaneous Pulmonary and Coronary Embolism in a patient with Patent Foramen Ovale and Deep Venous Thromboses: A Case Report. J Clin Trials 3: 130.

18 Edibam C, Morgan DJ, Rao SV, Keren A, Deague J (2008) Deep vein thrombosis and fatal paradoxical embolism after delayed reduction of a traumatic hip dislocation. J Trauma 64: 1133-1135.

19 Rigatelli G, Giordan M, Braggion G, Aggio S, Chinaglia M, et al. (2007) Incidence of extracerebral paradoxical embolisms in patients with intracardiac shunts. Cardiovasc Revasc Med 8: 248-250.

20 Ramineni R, Daniel GK (2010) Association of a patent foramen ovale with myocardial infarction and pulmonary emboli in a peripartum woman. Am J Med Sci 340: 326-328.

21 Murthy A, Shea M, Karnati PK, El-Hajjar M (2009) Rare case of paradoxical embolism causing myocardial infarction: successfully aborted by aspiration alone. J Cardiol 54: 503-506.

22 Haghi D, Sueselbeck T, Papavassiliu T, Haase KK, Borggrefe M (2004) Paradoxical coronary embolism causing non-ST segment elevation myocardial infarction in a case of pulmonary embolism. Z Kardiol 93: 824-828.

23 de Swiet J (1979) Paradoxical embolism associated with oral contraceptives: an underdiagnosed lesion? Postgrad Med J. 55: 419-420.

24 Sørensen HT, Horvath-Puho E, Lash TL, Christiansen CF, Pesavento R, et al. (2011) Heart disease may be a risk factor for pulmonary embolism without peripheral deep venousthrombosis. Circulation 124: 1435-1441.