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FREQUENCY AND CAUSES OF PERIPROCEDURAL MYOCARDIAL INFARCTION IN COMPLEX CORONARY INTERVENTIONS

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Contribution

AR conceived the idea and designed study. MIH did data collection. Manuscript writing was done by KI. AH did final review. All authors contributed equally to the submitted manuscript.

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ABSTRACT

Objective: To examine the frequency and cause sof periprocedural myocardial infarction (PMI) in complex coronary interventions.

Methodology: This cross sectional study was conducted at catheterization laboratory of Gulab Devi Chest Hospital, Lahore in 6 months duration from June 2016 to January 2017. Patients aged between 30 to 80 years undergoing complex coronary interventions were included. Blood samples for creatine kinase-MB evaluation were drawn at three different times i.e. Once before procedure, 8-10 and 24 hours after the procedure. CKMB level >3× ULN was considered as PMI. Association of different factors with PMI was also observed.

Results: A total of 72 consecutive patients were included in the study.Mean age of patients was 53.65+11.84 years. There were 56(77.78%) males. PMI was detected in 14 (19.44%)patients, with 3–5× ULN in 13.88% and >5× ULN in 5.55% of patients. Statistics of study showed that revascularization of left main stem lesion (1.38 % vs 0 %, p=0.04), in-stent restenosis (2.77 % vs 1.38 %, p=0.03) and procedural complications such as side branch occlusion (1.38%vs0%, p=0.04) and slow/no reflow (4.16 % vs 1.38 %, p=0.004)were significantly associated with CKMB elevation following complex coronary intervention.

Conclusion: PMI is common in patients undergoing complex coronary interventions.

Key Words: Complex coronary intervention, Creatine kinase MB, No reflow, Periprocedural myocardial infarction.

INTRODUCTION

Percutaneous coronary intervention (PCI) is a non-surgical technique used to treat the stenotic coronary artery diseases.[1] Balloon inflation during angioplasty regularly results in transient ischemia. The most notable hazards associated with myocardial ischemia are myocardial infarction (MI),stroke, ventricular fibrillation, myocardial localized necrosis and death.[2,3] Among these events, periprocedural MI, which can range from small increase in cardiac enzymes to large sized infarct, is the most common and devastating complication. With development of PCI, especially the use of drug eluting coronary stents and anti-platelet agents during the procedure, the frequency of cardiovascular events have decreased but periprocedural MI (PMI) is still a common complication to be faced, occurring in between 5 to 44% of the cases, depending on lesion and patient characteristics and procedure related factors. About one third of all elective PCI procedures are significantly associated with PMI which has been associated with increased later mortality.[4] Necrosis may occur in significant coronary dissection or a side branch occlusion, treatment of multiple vessels, use of rotablation, slow or no reflow and distal embolization.[5-8] Cardiac biomarker elevation before PCI is primarily because of plaque rupture, epicardial thrombosis, and myocardial injury. World Health Organization meaning of MI required 2 of 3 criteria, including clinical indications, ECG variation from the normal, and creatine kinase (CK) rise. By tradition, increase of biomarkers more than 3 times have been considered as PCI-related MI.[9] CK-MB levels increase within 3-12 hours of the onset of injury, reaches to peak within 24 hours, and come back to pattern after 48-72 hours showing localized tissue necrosis.[10] Even a minor increment in the post-PCI cardiac biomarkers is significantly associated with poor short and long term results.

Frequency and association of periprocedural MI with complex coronary interventions was examined in this study. No study has been conducted on local population regarding the association of various factors with PMI. This study will also contribute to a better understanding of the causes of PMI so as to guide planning to reduce the incidence and complications rate. If PMI incidence can be reduced, clinical outcomes would be expected to improve.

METHODOLOGY

This cross sectional study was conducted at catheterization laboratory of Gulab Devi Chest Hospital, Lahore in 6 months duration from June 2016 to January 2017.Patients undergoing complex coronary interventions were included in the study.

Non-probability purposive sampling technique was used to collect data from patients aged between 30 to 80 years of either gender with coronary artery disease confirmed through clinical investigations and other diagnostic procedures (like ECG, echocardiography and coronary angiography), who had diffused coronary vessels, bifurcating lesions and were undergoing complex coronary interventions (Type B or C lesions), were included in the study. Recently preinfarcted patients, patients who had type A lesions and those who were unwilling to participate were excluded. Diabetes Mellitus was considered as already diagnosed diabetic cases or newly diagnosed cases with fasting blood sugar level >120 mg/dL at 3 separate occasions. A high systolic (140 and over) or diastolic (90 and over) on 3 separate occasions was considered as hypertension. Smoking was defined as smoking at least 100 cigarettes during one's life time. Family History was considered positive if one's father or brother has a presentation of ischemic heart disease (IHD) before 55 years of age or if mother or sister has a known history of IHD before 65 years of age. Obesity was defined as body mass index of more than 30kg/m^2. Bifurcation was defined as stenosis involving the main and side branch, if a medium or large branch (>1.5mm) originates within the stenosis and if the side branch is completely surrounded by stenotic portions of the lesion to be dilated. Diffuse disease was defined as lesion length >20 mm and >50% diameter stenosis. Left main stem disease was>50% stenosis of left main coronary artery. Ostial disease was defined as origin of lesion within 3mm of vessel origin. Chronic total occlusion (CTO) was complete or almost complete blockage of coronary artery for more than 3 months. Coronary ectasia was defined as lesion diameter greater than the reference diameter in one or more areas. Multivessel disease was defined as the involvement of more than one coronary artery. Rotablation (rotational atherectomy) is a complex procedure that effectively ablates vessel wall calcification and facilitates stent delivery and complete stent expansion. Migration of air to distally occlude the target vessel or one of its branches was considered as air embolization. Dissection was defined as discrete mobile angiographic filling defect with or without contrast staining. Instent restenosis (ISR)was defined as focal tissue growth within the stent or its margins. Strut fracture was defined as complete/incomplete separation of stent strut by a fluoroscopic image. No reflow was defined as reduced Antegrade perfusion in the absence of flow-limiting stenosis. Side branch occlusion was defined as TIMI 0,1 or 2 flow in a side branch > 1.5mm in diameter that previously had TIMI 3 flow. Complex coronary intervention was defined as the procedure treating Type B or C coronary artery lesions or management of complications (dissection, ISR). Increase in creatine kinase-MB more than 3 times of upper limit of normal was considered as PCI-related MI.

After taking ethical approval from the institutional committee and informed verbal consent from the patients, samples of 3cc were drawn from venous end, recruited to laboratory and centrifuged within 20 minutes to prevent degradation of cardiac enzymes. The sera obtained were either stored at -30 degree Celsius or the test was precipitously performed. Working reagent of CK-MB measured 500 micro litre and serum sample of 20 micro liter were mixed and kept for 5 minutes in water bath. The absorbance at 340nm wavelength was observed. Pre angioplasty, after 8 and 24 hours samples were drawn and readings of CKMB level were noted. Moreover, history regarding risk factors was also taken using a pre designed questionnaire.

Both descriptive and inferential statistical analyses were done in SPSS 21.0. The qualitative data were presented in the form of appropriate table and graphs along with its percentage. The quantitative data were presented in the form of mean and standard deviation by simple descriptive analysis. Chi Square test was applied to see the association between different variables and periprocedural MI. Independent sample t test was used to see relationship of mean stent length with PMI.

RESULTS

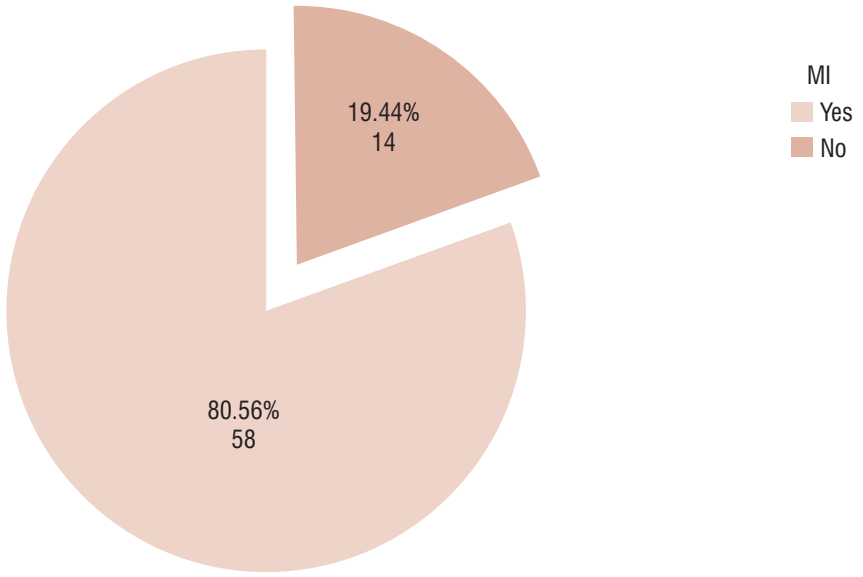
A total of 72 patients undergoing complex PCI were included in this analysis. Major clinical and demographic features of the patients are shown in table 1. The mean age of patients was 53.65+11.844 years and there were more males (77.78%) undergoing PCI. Most of the patients were diabetic (65.3%).For the devices of PCI, all patients received implantation of drug eluting stents.

Among 72 patients, CKMB elevation occurred in 14 (19.44%) patients as illustrated by figure 1,with 3–5× ULN in 13.88% and >5× ULN in 5.55% of patients.

Table 1: Demographic and Risk Factor Profile (n = 72)

Variables	Frequency n (%)	
Age (Years) (Mean ± S.D.)	53.65±11.84	
Gender n(%)	Male	56 (77.78)
	Female	16 (22.22)
Diabetes Mellitus n(%)	Yes	47 (65.3)
	No	25 (34.7)
Family History n (%)	Yes	34 (47.2)
	No	38 (52.8)
Smoking n(%)	Yes	25 (34.7)
	No	47 (65.3)
Obesity n(%)	Yes	8 (11.1)
	No	64(88.9)

Figure 1: Frequency of Periprocedural MI in Complex Coronary Interventions (n = 72)



The association of different patient and procedure related factors with CKMB elevation during complex PCI are shown in table 2. Left main stem intervention was significantly associated with the periprocedural MI (1.38% vs 0%, p=0.04). In stent stenosis (2.77%vs 1.38%, p=0.03), side branch occlusion during procedure (1.38% vs 0%, p=0.04)and slow or no-reflow(4.16% vs 1.38%, p=0.004) were also highly associated with the elevation of the CKMB after complex coronary intervention. Compared with patients without PMI, those with PMI had higher risk profiles of patient-, lesion-, and procedure-related characteristics. Comparison of our findings with different studies is shown in table 3.

Table 2: Association of Patient and Procedure Related Factors with CKMB Elevation (n = 72)

	Parameters		CKMB elevation		P-value
			Yes	No	
Lesion Related Factors	Bifurcating lesions	Yes	7	21	0.342[a]
		No	7	37	
	Diffuse disease	Yes	11	39	0.409[a]
		No	3	19	
	LMS disease	Yes	1	0	0.040[a]
		No	13	58	
	Ostial disease	Yes	7	20	0.282[a]
		No	7	38	
	CTO	Yes	6	13	0.119[a]
		No	8	45	
	Ectasia	Yes	1	5	0.857[a]
		No	13	53	
	ISR intervention		2	1	0.035[a]
			12	57	
Procedure Related Factors	Multi vessel PCI	Yes	9	28	0.282[a]
		No	5	30	
	Rotablation	Yes	2	2	0.112[a]
		No	12	56	
	Air Embolization	Yes	1	1	0.268[a]
		No	13	57	
	Dissection	Yes	0	1	0.621[a]
		No	14	57	
	Probable Strut fracture	Yes	1	1	0.268[a]
		No	13	57	
	Reflow	Normal	11	57	0.004[a]
		Slow/No	3	1	
	SBO	Yes	1	0	0.040[a]
		No	13	58	
	Mean Stent Length(mm)	Mean	75.21±42.03	58.46±28.16	0.076

[a]: Chi Square test was applied, [b]: Independent sample t test was applied, LMS: left main stem, CTO: chronic total occlusion, PCI: Percutaneous coronary intervention, ISR: in-stent restenosis, SBO: side branch occlusion.

Table 3: Frequencies of Occurrence of Periprocedura IMI, Reported in Different Studies.

Author	Year of Publication	Country	Sample Size	Frequency (%)
Bertinchant et al.[26]	1999	France	105	1.9%
Gruberg et al.[27]	2002	USA	116	43.1%
Saadeddin et al.[28]	2002	Saudi Arabia	96	27%
Nageh et al.[29]	2003	UK	109	25.6%
Mandadi et al.[6]	2003	USA	405	26.9%
Drzewiecka-Gerberet al[30]	2004	Poland	90	8.9%
Okmen et al.[31]	2006	Turkey	100	34%
Hoole et al.[24]	2010	UK	243	25.0%
Magro et al.[18]	2013	Europe	52	5.76%
Sheikh et al.[32]	2013	Pakistan	100	5.0%
Mehta et al.[33]	2016	India	302	2.0%

DISCUSSION:

This is a study of association between complex coronary intervention and periprocedural myocardial infarction. Many Studies have been done earlier demonstrating increased cardiac biomarkers after PCI, illustrated in table 3.[3,11-13]

In the present study, periprocedural myocardial infarction, defined by the most commonly accepted criteria (CKMB >x3 ULN without new Q waves) occurred in 19.44% patients which is similar to other published studies.

More extensive CAD is associated with a higher frequency anda larger degree of PMI. Presence of multivessel disease at the time of revascularization increases the chance of PMI by 1.3-1.8 fold.5Multivessel CAD is significantly associated with PMI in a study done by Kini et al. ($p=0.04$)[14]. Ourstudy results did not correlate with the study done by Kini et al. ($p=0.282$).

CKMB release strongly correlated with diffuse coronary disease; diffuse disease had a CKMB release of 24.5% versus 16.2 in focal lesions ($p<0.001$). While ostial lesions did not correlate with CKMB release.14In this study, both diffuse disease and Ostial lesions were not associated with PMI, $p=0.409$ and $p=0.282$ respectively.

Stone et al. found cardiac biomarker elevation in total 17.9% patients and it was more likely to occur after Rotablation ($p<0.001$)[12]. While this study results showed that there is no association between Rotablationand periprocedural MI($p=0.857$). This is most likely because plaque modification technique was used i.e. use of smaller burr at low speed ablation and it was mostly done at 1,40,000 to 1,50,000 rpm. With low speed Rotablation, there is less incidence of slow or no reflow and distal embolization hence low incidence of PMI.

PMI is significantly associated with complex bifurcating interventions ($p=0.001$).[15] This study did not correlate with the study of Hildick-smith et al.as results showed that periprocedural MI was not associated with bifurcating lesions intervention ($p=0.342$). Various bifurcation techniques are associated with various degree of PMI. Patients with true bifurcation were included and various two stent techniques with proper lesion preparation and use of intravenous GP IIb/IIIa inhibitors during PCI were used, which reduced the risk of PMI as shown in this study.

PMI is among the most common complications of CTO PCI. Patel et al. showed that it occurred in 2.5% patients undergoing CTO intervention and was significantly associated with it ($p<0.0001$)[16] While Suero et al. demonstrated thatMI occurred in 0.5% of CTO lesions' interventions whereas 0.6% for non-CTO lesions ($p=0.6$).[17] Statistics of this study demonstrates that Periprocedural MI is not associated with CTO lesions intervention ($p=0.119$) and correlates with the results of Suero et al.

Periprocedural MI occurred in 5.76% patients undergoing PCI for left main stem disease in a study done by Magro et al.[18] This study showed that, MI occurred in 1(1.78%) patient and is associated with left main stem intervention ($p=0.04$), most probably due to distal embolization of atheromatous plaque.

Dorros et al. reported that coronary artery dissection was noted in 9.2% patients undergoing PTCA. Coronary artery dissection was a complication in 31% of these patients who suffered from a major associated complication i.e. MI in 11.6%, emergency bypass in 26% and in-hospital death in 4% patients.19This study showed that coronary artery dissection occurred in only 1(1.38%) patient without any other complication and it was covered with a stent later. With the development of low profile balloons and thin strut stents in the present era, the frequency of coronary artery dissection is very low as supported by this study.

Coronary air embolism occurred in 0.13% patients undergoing PTCA in a study done by Dorroset al.[19] while in our study only 2 (2.78%) patients in whom only 1 (1.38%) patient suffered from MI. This is a rare complication that can be occurred due to improper flushing of system as occurred in present study and that is managed with standard protocolfollowed by stenting. But as air embolism results in no flow to the vessel, it can result in various degrees of myocardial injury and increase in CKMB levels, as occurred in one of the patients.

Side branch occlusion can occur during PCI especially when subintimal strategies are used in CTO intervention and is

associated with higher frequency of Post PCI MI.[20] Occlusion of a side branch has been reported in 12.5-19% of cases in which a stent was placed over a major side branch (>1mm).[21,22] According to the study of Talasz et al, side branch occlusion occurred in 23.8% patients undergoing percutaneous PCI and CKMB mass concentrations were substantially higher than normal in all of these patients.[23] In present study, side branch occlusion occurred in only 1(1.38%) patient and it was significantly associated with CKMB elevation (p=0.040). These findings may additionally emphasize that careful review of side branch anatomy and optimal side branch protection during complex PCI is required to minimize procedural infarction.

Longer stent length is also implicated for increased myocardial enzyme release in a recent study by Hoole et al. They also reported that diminished myocardial perfusion was also significantly associated with cardiac enzymes elevation (p=0.006).[24] No reflow/ slow flow is one of the most potent predictor of PMI (p<0.001).[5,14] Slow/No reflow was significantly associated with CKMB elevation in present study (p=0.04) while mean stent length was not a statistically significant factor associated with PMI (p= 0.07). Cardiac enzyme elevation in slow/no reflow cases may be attributable to microembolization of thrombotic or atherosclerotic material which can be undetectable in coronary angiogram.

Mechanism of in stent restenosis is multifactorial. Other than procedural factors (stent mal apposition and under expansion), neo atherosclerosis is most common cause of ISR especially in diabetics. With use of new generation DES, the incidence of neo intimal proliferation and neo atherosclerosis is quite low but still it is the main contributing factor for target lesion reinfarction.[25] In this study 2 patients had PMI who underwent re intervention for in stent restenosis. We are not able to define the mechanism completely, it may be an incidental finding or neo atherosclerosis may behave differently than the atherosclerosis in native coronary arteries. This question remained unanswered unless we utilize intracoronary imaging to exactly define the characteristics of neo intimal proliferation and neo intimal atherosclerosis.

Although PMI is highly associated with complex interventions, but when its association in various subgroups is seen, the significance is altered. This could be due to the characteristic features of plaques such as large plaque volume and necrotic core areas, often associated with embolic events, indicative of periprocedural MI. Embolization of lipid core of stenotic plaques after PCI has been considered as an important cause of no reflow and MI. Modern imaging techniques such as Optical Coherence Tomography, Intravascular ultrasound and near infrared spectroscopy are the need of time in this setting. Rises in inflammatory markers such as IL-6 and CRP levels post PCI are also responsible for significant CKMB elevation. Studies are being conducted to estimate pre and post lesion temperatures and inflammatory markers levels so as to find out the exact inflammatory area to deploy stents there.

LIMITATIONS

The main limitations of the study were small sample size as compared to the disease burden and the use of 2D X-ray imaging technique which do not comment about the plaque morphology. Intra coronary imaging (OCT, IVUS) was not used in this study as without intracoronary imaging, plaque morphology cannot be determined exactly hence can't predict the risk of PMI. Further studies are required with intracoronary imaging to identify the plaque characteristics and risk of PMI

CONCLUSION

Periprocedural MI is common in patients undergoing complex coronary interventions. These findings have important clinical implications for practice and may help in deciding further management of the patients.

REFERENCES

1. Torpy JM, Lynm C, Glass RM. Percutaneous coronary intervention. JAMA 2004;291(6):778.
2. Li J, Zhang Y, Cui L, Wang J, Pang X, Lai Y, et al. Mechanical stretch changes coronary artery fibroblasts function by upregulating HSF1 protein expression. Int J Biol Macromol 2013;59:105-10.
3. Tardiff BE, Califf RM, Tcheng JE, Lincoff AM, Sigmon KN, Harrington RA, et al. Clinical outcomes after detection of elevated cardiac enzymes in patients undergoing percutaneous intervention. J Am Coll Cardiol 1999;33:88-96.
4. Katritsis DG, Ioannidis JP. Percutaneous coronary intervention versus conservative therapy in nonacute coronary artery disease. Circulation 2005;111:2906-12.
5. Herrmann J. Peri-procedural myocardial injury: 2005 update. Eur Heart J 2005;26:2493-519.
6. Mandadi VR, DeVoe MC, Ambrose JA, Prakash AM, Varshneya N, Gould RB, et al. Predictors of troponin elevation after percutaneous coronary intervention. Am J Cardiol 2004;93:747-50.
7. Blankenship JC, Haldis T, Feit F, Hu T, Kleiman NS, Topol EJ, et al. Angiographic adverse events, creatine kinase-MB elevation, and ischemic end points complicating percutaneous coronary intervention (a REPLACE-2 substudy). Am J Cardiol 2006;97:1591-6.
8. Kugelmass AD, Cohen DJ, Moscucci M, Piana RN, Senerchia C, Kuntz RE, et al. Elevation of the creatine kinase myocardial isoform following otherwise successful directional coronary atherectomy and stenting. Am J Cardiol 1994;74:748-54.
9. Lansky AJ, Stone GW. Periprocedural myocardial infarction prevalence, prognosis, and prevention. Circ Cardiovasc Interv 2010;3(6):602-10.
10. Jaffe AS, Apple FS, Lindahl B, Mueller C, Katus HA. Why all the struggle about CK-MB and PCI? Eur Heart J 2012;33:1046-8.
11. Ellis SG, Chew D, Chan A, Whitlow PL, Schneider JP, Topol EJ. Death following creatine kinase-MB elevation after coronary intervention. Circulation 2002;106:1205-10.
12. Stone GW, Mehran R, Dangas G, Lansky AJ, Kornowski R, Leon MB. Differential impact on survival of

electrocardiographic Q-wave versus enzymatic myocardial infarction after percutaneous intervention. Circulation 2001;104:642-7.

13. Cavallini C, Savonitto S, Violini R, Arraiz G, Plebani M, Olivari Z, et al. Impact of the elevation of biochemical markers of myocardial damage on long-term mortality after percutaneous coronary intervention: results of the CK-MB and PCI study. Eur Heart J 2005;26:1494-8.
14. Kini A, Marmur JD, Kini S, Dangas G, Cocke TP, Wallenstein S, et al. Creatine kinase-MB elevation after coronary intervention correlates with diffuse atherosclerosis, and low-to-medium level elevation has a benign clinical course: implications for early discharge after coronary intervention. J Am Coll Cardiol 1999;34:663-71.
15. Hildick-Smith D, De Belder AJ, Cooter N, Curzen NP, Clayton TC, Oldroyd KG, et al. Randomized trial of simple versus complex drug-eluting stenting for bifurcation lesions. Circulation 2010;121:1235-43.
16. Patel VG, Brayton KM, Tamayo A, Mogabgab O, Michael TT, Lo N, et al. Angiographic success and procedural complications in patients undergoing percutaneous coronary chronic total occlusion interventions: a weighted meta-analysis of 18,061 patients from 65 studies. JACC Cardiovasc Interv 2013;6(2):128-36.
17. Suero JA, Marso SP, Jones PG, Laster SB, Huber KC, Giorgi LV, et al. Procedural outcomes and long-term survival among patients undergoing percutaneous coronary intervention of a chronic total occlusion in native coronary arteries: a 20-year experience. J Am Coll Cardiol 2001;38:409-14.
18. Magro M, Girasis C, Bartorelli AL, Tarantini G, Russo F, Trabattoni D, et al. Acute procedural and six-month clinical outcome in patients treated with a dedicated bifurcation stent for left main stem disease: the TRYTON LM multicentre registry. EuroIntervention 2013;8(11):1259-69.
19. Dorros G, Cowley M, Simpson J, Bentivoglio L, Block P, Bourassa M, et al. Percutaneous transluminal coronary angioplasty: report of complications from the National Heart, Lung, and Blood Institute PTCA Registry. Circulation 1983;67:723-30.
20. Michael TT, Papayannis AC, Banerjee S, Brilakis ES. Subintimal dissection/reentry strategies in coronary chronic total occlusion interventions. Circ Cardiovasc Interv 2012;5(5):729-38.
21. Aliabadi D, Tilli FV, Bowers TR, Benzuly KH, Safian RD, Goldstein JA, et al. Incidence and angiographic predictors of side branch occlusion following high-pressure intracoronary stenting. Am J Cardiol 1997;80:994-7.
22. Kralev S, Poerner TC, Basorth D, Lang S, Wolpert C, Haghi D, et al. Side branch occlusion after coronary stent implantation in patients presenting with ST-elevation myocardial infarction: clinical impact and angiographic predictors. Am Heart J 2006;151:153-7.
23. Talasz H, Genser N, Mair J, Dworzak EA, Puschendorf B, Friedrich G, et al. Side-branch occlusion during percutaneous transluminal coronary angioplasty. Lancet 1992;339:1380-2.
24. Hoole SP, Heck PM, Sharples L, Dutka DP, West NE. Coronary stent length predicts PCI-induced cardiac myonecrosis. Coron Artery Dis 2010;21:312-7.
25. Komiyama H, Takano M, Hata N, Seino Y, Shimizu W, Mizuno K. Neoatherosclerosis: coronary stents seal atherosclerotic lesions but result in making a new problem of atherosclerosis. World J Cardiol 2015;7:776-83.
26. Bertinchant JP, Polge A, Ledermann B, Genet LC, Fabbro-Peray P, Raczka F, et al. Relation of minor cardiac troponin I elevation to late cardiac events after uncomplicated elective successful percutaneous transluminal coronary angioplasty for angina pectoris. Am J Cardiol 1999;84:51-7.
27. Gruberg L, Fuchs S, Waksman R, Pichard AD, Kent KM, Laird JR, et al. Prognostic value of cardiac troponin I elevation after percutaneous coronary intervention in patients with chronic renal insufficiency: a 12-month outcome analysis. Catheter Cardiovasc Interv 2002;55:174-9.
28. Saadeddin SM, Habbab MA, Sobki SH, Ferns GA. Biochemical detection of minor myocardial injury after elective, uncomplicated, successful percutaneous coronary intervention in patients with stable angina: clinical outcome. Ann Clin Biochem 2002;39:392-7.
29. Nageh T, Sherwood RA, Harris BM, Byrne JA, Thomas MR. Cardiac troponin T and I and creatine kinase-MB as markers of myocardial injury and predictors of outcome following percutaneous coronary intervention. Int J Cardiol 2003;92:285-93.
30. Drzewiecka-Gerber A, Drzewiecki J, Wita K, Krauze J, Jaklik A, Trusz-Gluza M, et al. Prognostic value of troponin I after elective percutaneous coronary interventions. Kardiol Pol 2004;61:122-5.
31. Okmen E, Cam N, Sanli A, Unal S, Tartan Z, Vural M. Cardiac troponin I increase after successful percutaneous coronary angioplasty: predictors and long-term prognostic value. Angiology 2006;57:161-9.
32. Shaikh MM, Lashari MN, Soomro K. Frequency of periprocedural myocardial infarction in patients undergoing elective percutaneous coronary intervention. Pak Heart J 2013;46:108-12.
33. Mehta V, Sukhija R, Mehra P, Goyal A, Yusuf J, Mahajan B, et al. Multimarker risk stratification approach and cardiovascular outcomes in patients with stable coronary artery disease undergoing elective percutaneous coronary intervention. Indian Heart J 2016;68:57-62.