JMSCR Vol||07||Issue||08||Page 789-797||August 2019

http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v7i8.134

IGM Publication Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Incidence of Ocular Symptoms Following Percutaneous Coronary Procedures- A Single Centre Experience

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Abstract

Aim of the study: *To find the incidence of new ocular symptoms following transradial diagnostic and therapeutic coronary catheterization and associated risk factors.*

Materials & Methods: *Prospective observational study of 500 consecutive patients satisfying the inclusion criteria attending our tertiary health centre for transradial coronary procedures were observed for post procedural new ocular symptoms and compared with 500 patients undergoing same via transfemoral approach.*

Results: *76 patients (incidence 7.6%) developed new ocular symptoms in the form of black-outs, Amaurosis fugax and field loss. 75% of these patients belonged to radial access group. 40 patients had reversible visual symptoms as a sequalae of cerebral hypo perfusion and 36 patients had symptoms of retinal embolism. Clinically apparent changes in vision was noted in 9 [0.9%] patients. In those with new ocular symptoms, the risk factors found were age, periprocedural hypertension, vascular tortuosity, procedure done through radial artery approach in patients with height<155cm ,prolonged procedural time. 1patient had uniocular Central Retinal Artery Occlusion [CRAO] with permanent loss of vision. 3 patients had features of Branch Retinal Artery Occlusion [BRAO] and 5 patients had peripheral retinal embolus.*

Conclusions: *Although rare, incidence of ocular embolic complications were higher with percutaneous coronary procedures via radial approach. Patients with height <155 cm may be benefitted when the procedure is done via femoral approach. Cardiologists should try to avoid excessive catheter manipulations while doing procedures. Prompt diagnosis and treatment may reduce extent of damage caused by vascular embolism.*

Keywords: *Ocular embolism, coronary angiography, coronary angioplasty.*

Background

Coronary artery disease is one of the leading causes of death all over the world. Percutaneous coronary angiography and revascularisation are the standard procedures for the diagnosis and treatment of coronary artery disease. Being invasive, these procedures carry risk of thrombo embolic complications; contrast induced nephropathy or allergic reactions etc[1,2]. Commonest cause of embolization during these procedures is thought to be due to vascular atherosclerotic plaque disruption caused by catheter or wire manipulation in the ascending aorta. Other causes are thrombus formation at the catheter tip[3], air embolism [4] and rarely, foreign materials from the catheter or guidewire[5]. Cerebrovascular embolization resulting in stroke is a rare but serious complication of cardiac catheterization[6]. The incidence of clinically demonstrable cerebrovascular accident following to cardiac catheterization is reported to be between 0.11% & 0.38%[7]. But pre and post procedural magnetic resonance imaging studies have shown that asymptomatic cerebro vascular embolization can occur in up to 15% of patients[8].Study by Bruno et al showed a 10-fold increase in the incidence of stroke among men followed for a mean of 3.4 years after an index retinal embolus over the stroke incidence seen in a control group over the same period[9].Study by Klein et al showed that those with baseline retinal emboli had an increased risk of dying of stroke compared to those without even after adjustment for systemicfactors[10]. Being the first major branch of the internal carotid artery , emboli from the heart and great vessels proximal to the carotid tend to directly go to the ophthalmic artery. Retinal arteriolar emboli are generally described as discrete plaque-like lesions in the lumen of the retinal arterioles or at bifurcations. They are composed of fibrin-platelet aggregates, cholesterol fragments, or particles of calcified valves. They originate mainly from an ulcerated atheromatous carotid artery or ascending aorta plaque, calcified aortic valves, or internal carotid thrombus, and are classified as cholesterol (reflective or retractile), fibrin-platelet (dull or non-reflective) or calcific (chalky white) [11].Retinalemboli may be a simply detectable predictor of cerebrovascular embolism. Studies by Ascione et al & Blauth et al have reported rates of retinal emboli between 55% and 100% after bypass surgery, and1.25% and 13.2% after carotid stenting respectively[12,13,14]. The rate of retinal emboli reported by Kojuri et al after cardiac catheterization was 6.3%[15]. Retinal cholesterol emboli, known as Hollenhorst plaques are characterised by distinctive focal bright, refractive yellow-orange appearance. Retinal emboli are usually partially obstructive and patients won't have many symptoms. Even though rare, central retinal arterial occlusion is an emergency. Available treatments do not appear to be fully effective. An experimental study on central retinal artery occlusion showed that occlusion lasting about 97 min resulted in massive, irreversible damage. Thus, no treatment instituted much longer than 1.5 hours after the onset can be expected to restorevision[16, 17].

Aim of the study was to find the incidence of new ocular symptoms after diagnostic &therapeutic coronary catheterization and to assess the risk factors that may increase the possibility of this complication.

Hypothesis of the study was excessive catheter manipulation near the aortic root and ascending aorta during the procedure increases the chances of thrombo embolism. The catheter manipulation difficulties could be due to age related tortuosity of aorta or its branches, short stature and difficult coronary anatomy .In the presence of above factors, the catheter manipulation is more difficult especially via the radial arterial access.

Materials & Methods

Inclusion Criteria: From march 2015 to march 2016, prospective observational study of 1000 elective [non-emergency] patients who underwent diagnostic or therapeutic coronary catheterization for standard indications in the Department of Cardiology, Government Medical College, Kottayam, Kerala were included in our study. This included equal number of patients who underwent angiogram /angioplasty via radial /femoral approach, thus constituting 4 groups [250 patients in each group].

Exclusion Criteria: known case of arrhythmia, significant valvular heart disease confirmed by transthoracic echocardiography, clinically evident peripheral vascular disease, those with history of cerebrovascular event or history of ocular thrombo embolic events, those having documented diabetic/hypertensive retinopathy, those with ocular symptoms, post CABG patients and all acute coronary syndromes were excluded.

Detailed medical history and clinical examination findings were documented for all patients, and risk factors such as dyslipidaemia, diabetes mellitus, smoking, hypertension, and obesity were recorded. All patients were given detailed description regarding the procedure and were instructed to report any symptoms especially ocular. Patients underwent planned angiography /angioplasty via radial or femoral arterial approach. Coronary angiograms and angioplasties were done via right radial or femoral artery by 7 experienced interventional cardiologists. Standard Seldinger technique was used for arterial puncture. As per the institutional protocol, 3000 international units [IU] of heparin was given for diagnostic angiography and 8000IU for angioplasty, additional doses were given after checking activated clotting time. 5F [French] catheter was used for angiography. For percutaneous transluminal coronary angioplasty with stent implantation with or without balloon dilation, a 6F catheter was used. All angioplasty patients received at least 350 mg aspirin and 600 mg clopidogrel at least 8 hours prior to the procedure. A standard non-ionic contrast medium (Omnipaque /Visipaque / Ultravist) was used. For diagnostic angiography, sheaths were removed immediately after the procedure. In patients who underwent therapeutic catheterization, the sheaths were removed 6hours after the procedure. Operators were blinded from the study.

All post procedure patients with ocular symptoms were seen by the investigator and reviewed by an experienced ophthalmologist within 24hours. Investigator also reviewed all those patients who had periprocedural new ocular symptoms & attended and managed by the operator. Visual acuity was determined with the Snellen chart and visual field was measured with the confrontation test and subjective history. Retinal examinations were done after pupillary dilation with 1.0% tropicamide. The macula and posterior pole were examined with a 90D non-contact lens through a slit lamp, and indirect ophthalmoscopy with a Volk 2.2D lens was done to visualize the peripheral portions of retina.

Patient characteristics including age, sex, disease duration, coronary risk factors, height of the patient, drug history, renal dysfunction, type of angiographic contrast used, coronary angiographic anatomy categorisation according to ACC AHA criteria, total procedural time, procedural data (arterial access, reasons for changing arterial site, reasons for guide catheter changes, use of balloons, size of stent, and the total duration of the procedure) were recorded. Ocular symptoms &signs were recorded. The initial arterial route tried by the operator was taken as the criteria to label as procedure via radial or femoral arterial approach. Reason for switching to other arterial access was also noted. The groups were subdivided in to two based on presence/absence of post procedural new ocular symptoms.

Written informed consent was obtained from all patients, and the study was approved by the research ethical committee of the Government Medical College, Kottayam, and Kerala.

Statistical analysis

For categorical and non-categorical variables, we used the chi-squared test to determine whether differences between groups were significant. P value < 0.05 was considered significant.

Results

1000 patients who satisfied all inclusion criteria were studied. 250 patients were in each group. Mean age of the patients was 44.3±6.18 years in normal group and 63±7.8 in patients with new ocular findings. Conventional coronary risk factors such as dyslipidaemia, male sex, body mass index, hyper homocysteinemia, mere presence of diabetes did not differ significantly between patients with and without new retinal emboli. Age, active smoking, elevated HbA1c >7 during the pre-procedural investigations, height less than 155 cm, Periprocedural hypertension, shorter duration of treatment with antiplatelet & hypolipidemic drugs, prolonged procedural time were showing significant differences between the two groups. Regarding the procedural characteristics stent length, stent diameter, use of balloons had no effect on the incidence of new emboli .The reasons for prolonged procedural time varied in individual cases. [Table-1]

Table 1 Baseline characteristics

Post procedure ocular findings			p value
	Normal [924]	New findings[76]	
Mean age	44.3±6.18	63±7.8	<0.01
Sex Male [n]	470	30	1.0
Female [n]	454	46	
BMI>30	24	2	1.0
Active smoking	224	26	0.05
Diabetes	314	24	0.67
HbA1c>7	76	16	0.0002
hypertension	586	63	0.0006
Periprocedural BP>160/90	84	28	<0.01
dyslipidaemia	648	48	0.2
Duration of cardiac treatment[days]	180±24	36±12	0.01
Height <155cm	114	26	<0.0001
hyperhomocysteinemia	168	14	1.0
Creatinine clearance[no:] <90[ml/mt/BSA]	96	8	1.0
Total Procedural time [minutes]			
angiogram	6.4±3.2mt	18.2±4.1mt	<0.01
angioplasty	26.2±8.2mt	35.4±7.6mt	<0.01
Stent length	28.1±12.2mm	29.2±12.4mm	0.5
Stent diameter	2.67±0.35mm	2.72±0.32mm	0.5

Table 2: Coronary lesion morphology and new ocular symptoms

	Normal/ minor CAD	New findings	Type A lesions	New ocular findings	Type B lesions	New ocular findings	Type C lesions	New ocular findings	Total
RadialCAG 250	36	1[2.7%]	104	2[1.9%]	68	10[14.7%	42	9[21.4%]	22[8.8%]
Radial PCI 250	0	0	80	4[5%]	118	13[11%]	52	18[34.6%]	35[14%]
Femoral CAG 250	8	0[0]	22	0[0]	108	0[0]	112	3[2.6%]	3[1.2%]
Femoral PCI 250	0	0	40	0	86	1[1.1%]	124	15[12.1%]	16[6.4]
Total 1000	44	1[2.3%]	246	6[2.4%]	380	24[6.3%]	330	46[13.9%]	76[7.6%]

As the complexity of the coronary artery lesion progressed from ACC AHA type B to type C, noted increase in incidence of post procedural ocular findings across all four groups, more profound effect noted in the radial group. In 44 patients who had normal epicardial coronaries, 1 patient who underwent angiogram through radial approach developed new ocular findings. The patient was having a height of 154cm, normotensive non diabetic and arterial tortuosity leading to prolonged procedural time of 20 minutes patients who underwent radial angiogram and having Type A coronary lesion, the new ocular finding was 1.9%[2 patients], which was higher than those with normal coronaries. Both the patient were having recent onset of cardiac symptoms and treatment. Both had Periprocedural hypertension, elevated HbA1c and height more than 155cm.In the femoral angiogram& angioplasty groups, none of the patients with normal coronaries and type a lesion developed new ocular findings.

Table 3 Procedural time and new ocular findings

	Radial CAG [250]		Radial PTCA[250]		Femoral approach	
	Radial CAG [232]	Crossover to femoral CAG[18]	Radial PTCA [224]	Crossover to femoral PTCA[26]	Femoral CAG[250]	Femoral PTCA [250]
Procedural time in minutes						
- In Normal	6.4±3.2 mts	6.8±2.5 mts	26.2±8.2 mts	32.3±3.4 mts	5.8±2.9 mts	22.1±4.2 mts
-In New findings	18.2±4.1 mts	19.2±3.8	35.4± 7.6 mts	44.6±4.7 mts	11.5± 3.8 mts	36.2± 4.8 mts
New Ocular findings [number]	18[7.8%]	4[22.2%]	24[10.7%]	11[42.3%]	3[1.2%]	16[6.4%]
Total	22		35		3	16

The procedure time for angiogram or angioplasty in radial and femoral groups were higher in patients with new ocular findings [P<0.01]. Procedures done via radial approach had more new ocular findings than the femoral group [p<0.01]. All these patients had prolonged procedural time due to difficulty in engaging the coronary catheter due to tortuous aorta or right brachiocephalic artery. Whenever there was difficulty in engaging the coronaries via radial approach [due to multiple reasons like radial spasm, aortic tortuosity, inadequate guide support, difficult coronary anatomy], the operator switched to femoral arterial approach. Such patients had more incidence of new ocular symptoms compared to coronary procedures done exclusively through radial or femoral artery alone. Highest incidence was seen when the angioplasty procedure was prolonged after difficult guide manipulation via radial approach and subsequently using femoral arterial approach. The effect was profound when the height of the patient was <155cm.

Table 4: Height of the patient and new ocular symptoms

	Radial CAG 250		Radial PTCA 250		Femoral procedures 500	
	Radial CAG N=232	Cross over to femoral 18	Radial PTCA N=224	Cross over to femoral 26	Femoral CAG N=250	Femoral PTCA N=250
New findings [n=76]	18[7.8%]	4[22.2%]	24[10.7%]	11[42.3%]	3[1.2%]	16[6.4%]
Height>155cm[n=860]	206	14	208	15	212	210
Normal [n=810]	188	14	184	15	209	200
New findings [n=50]	18	0	21	0	3	10
Height<155cm[n=140]	26	4	16	11	38	40
Normal [n=114]	0	0	16	0	38	34
New findings[n=26]	26	4	0	11	0	6

Table 5: Ocular symptoms, etiology&findings

Symptoms	76	Etiology	Ocular fundus	Ocular fundus findings		
				CRAO	BRAO	Peripheral Retinal embolus
Black outs	40	Cerebral hypo perfusion	Normal in all	0	0	0
Amaurosis fugax	30	Ocular embolism	Abnormal in 4	0	0	4
Field loss	5	Ocular embolism	Abnormal in 5	0	2	3
Loss of vision	1	Ocular embolism	Abnormal in 1	1	0	0

Ocular Symptoms & Findings

76 patients [7.6%] were having post procedural ocular symptoms. 40 patients had reversible vision loss described by majority as blackouts affecting both eyes within 24 hours after the procedure. All of them had normal ocular fundus or had no evidence of fresh ocular thromboembolism, which pointed towards cerebrovascular accident due to hypo perfusion. The causes of cerebral hypo perfusion was found as transient bradycardia & hypotension in 12, bleeding related hypotension in 1, transient bradycardia in 3,and CT proven cerebral thromboembolism in 24 ..30 patients had uniocular Amaurosis fugax as post procedural ocular symptom.17 had right eye & 13 had left eye involvement. Among those with Amaurosis fugax, only 4 had demonstrable ocular fundus findings in the form of peripheral retinal embolism. None of them had demonstrable Central Retinal Artery occlusion [CRAO] or Branch Retinal Artery Occlusion [BRAO]. 5 patients had uniocular field defects. 2 patients had BRAO, 3 had peripheral retinal artery embolism. Of the 76 patients, only one patient had unilateral irreversible total loss of vision [right eye] immediately after the procedure, subsequently confirmed as CRAO. Of these patients, 25 had undergone cardiac catheterization for diagnostic angiography (32.9%) and the remaining 51 (67.1%) for coronary revascularisation

Discussion

Coronary angiogram and percutaneous coronary angioplasty are the standard non-surgical procedure for diagnosis and treatment of coronary heart disease. Percutaneous coronary procedures poses a risk of vascular embolism. Sources of embolism can be degenerative aortic valve, vascular atherosclerotic plaques, left main coronary artery disease, catheter related thrombus etc.[1,18]

On analysis of various clinical and procedural variables in 1000 patients, following findings were noted. As expected those with new ocular findings, the mean age was higher. This is comparable to all available literature on vascular thromboembolism[3]. Arterial atherosclerosis starts as early as 2 years of age. The progression of atherosclerosis is then modified by factors like age, presence of vascular risk factors. Out of the 30 males who developed new ocular symptoms, 26[86.7%] were active smokers or quit smoking only few days prior to the elective procedure. One interesting finding was that mere presence of diabetes didn't have any significant association with new ocular findings, but elevated HbA1c had correlation, pointing towards the need for strict diabetic control while doing all elective vascular procedures[10]. History of hypertension, especially the periprocedural hypertension was one of the strongest predictor of new ocular/systemic embolic findings in our study, which is consistent with available studies[4].It was noted that those who developed the new ocular findings were diagnosed to have coronary artery disease & put on medications approximately one to two months prior to the procedure. The duration of treatment was much less compared to the group, who had no new ocular symptoms. Longer duration of statin therapy could have helped in better plaque stabilisation. Height less than 155cm was found to be an important predictor for development of new ocular symptoms, especially when the procedure was done through the radial artery approach. Even though none of the patients had true arterial tortuosity syndrome, patients with height 155 cm had difficulty in negotiating the wire, catheters through the subclavian artery and ascending aorta. Multiple wire and guide exchanges to negotiate the curve might have increased plaque rupture from the ascending aorta and subsequent embolization. Those who developed new ocular findings had more Type B & C ACC AHA coronary artery lesions. In such patients, thromboembolism from ascending aorta could be due to generalised arteriosclerotic process in relation to atherosclerotic coronary artery disease. Another etiology in such patients could be requirement of more hardware manipulations during PCI to tackle more complex procedure leading to plaque rupture and embolization. The reasons for switching arterial access from radial artery to femoral artery included radial spasm, inadequate guide support, tortuous/curved course of arch branches ,difficult coronary anatomy and periprocedural hemodynamic alterations that prompted the operator to switch the arterial access .

In our study group, 76 patients had new ocular symptoms. Highest incidence of new ocular symptoms was in the radial angioplasty group and least in the femoral angiogram group. None of the cerebral hypo perfusion induced blackouts were permanent. Out of 76 patients who developed new ocular findings, one had permanent loss of vision, reason being CRAO. Field defects in BRAO patients improved, but not completely reversed.

Segal et al noted that plaque rupture from the ascending aorta or the aortic arch can be the main source of systemic emboli [7] .Klein et al [10] suggested higher pulse pressure, hypertension, cardiovascular disease, diabetes mellitus and past & current smoking increased the chances of retinal emboli. Thyer et al[19] reported no evidence suggesting that coronary catheterization contributes to retinal embolism shortly after the procedure. Kreis et al[20] reported 2% incidence of acute retinal embolism after coronary catheterization. In their study the main method of retinal examination was digital retinal photography. New ocular symptoms after cardiac catheterization were seen in 7.6% of our patients, higher than that noted by Kreis and colleagues. Busing et al[8] found that diagnostic and interventional cardiac catheterization increased the risk of silent cerebral infarction to 15%.

Almost all patients who developed ocular symptoms due to retinal emboli developed the symptoms during or immediately after the procedure. Time period of development of ocular symptoms due to cerebral hypo perfusion varied from immediately after the procedure up to initial 12 hours post procedure. The visual disturbances due to cerebral hypo perfusion was transient and needed no specific ocular treatment.

Treatment received in cerebral hypo perfusion group included intravenous fluids, pressor, atropine when the cause was predominantly vasovagal. Transient atrial fibrillation was noted in one patient. None of the patient had ventricular arrhythmias. None had evidence of cerebral bleed. For those with CT proven infarct, after getting neurology consultations, addition of heparin was done as per activated clotting time, even though no definite neurological deficits could be elicited. Treatment tried for unilateral ocular symptoms, which were due to ocular embolism included ocular massage, reduction of intraocular pressure by intravenous acetazolamide, paracentesis, as well as carbon dioxide (CO2) rebreathing bag respiration. The treatment was given to the patient after seen by the ophthalmologist. The time of starting of treatment varied from half an hour to 24 hours, due to delay in the diagnosis. Visual outcome was poor in the CRAO patient and only minimal improvement in the BRAO patients. Retinal ischemic damage lasting up to 240 min is usually irreversible [21]. The conventional treatment methods include ocular massage; reduction of intraocular pressure by various medical and surgical means; vasodilatation by sublingual isosorbide dinitrate and CO_2 rebreathing bag; antiplatelet and heparin therapy. Other treatments reported with variable success include isovolemic hemodilution; hyperbaric oxygen; intravenous steroid to reduce vascular endothelial; Nd: YAG laser arteriotomy and embolectomy[22]; surgical embolectomy [23]; and intra-arterial fibrinolysis delivered directly into the ophthalmic artery. However, none of these methods are proven to be 100% effective. Hence, early diagnosis helps to identify the source of the problem, thus preventing further damage or recurrence to other important organ. CT and MRI scan can help whenever there is a diagnostic confusion between ocular and cerebral embolism.

Limitations of the study

Study patients didn't undergo ocular examination prior to the planned elective procedure as it was an observational study. Pre procedural ocular examination could have helped in finding patients with baseline retinal vascular compromise and helped in identifying additional risk factors if any. Post procedural detailed ocular examination was done only in patients complained of visual disturbances. Totally asymptomatic ocular thromboembolic events might have been missed by the same.

Conclusion

Age, periprocedural hypertension, height less than 155 cm, elevated HbA1c,active smoking, shorter gap between diagnosis and coronary procedure ,radial arterial access, longer procedural time are independent predictors of retinal emboli after percutaneous coronary interventions. Unless recognised and treated vigorously in the initial few hours of retinal embolus causing CRAO, prognosis is poor.

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