

1122 INADVERTENT CORONARY ARTERY DISSECTION DURING ROUTINE INVASIVE CORONARY ANGIOGRAPHY AND MANAGEMENT UNDER IVUS GUIDANCE

Introduction

Inadvertent coronary artery dissection (ICAD) is a rare and devastating complication of invasive coronary angiography (ICA) and percutaneous coronary intervention (PCI). Mechanisms include mechanical injury to the arterial wall during guide catheter/guidewire manipulation, balloon dilatation or stent deployment in a false lumen, and forceful injection of contrast in patients with ostial lesions. Other rarer conditions which have been reported to be associated with increased risk of coronary artery dissection are bicuspid aortic valves and Marfan syndrome.¹ Most occur during PCI but in this case report we describe a rare case wherein coronary artery dissection and subsequent abrupt closure occurred during routine diagnostic ICA. To our knowledge, there is a paucity of reported cases and data on ICAD during diagnostic coronary angiography, especially in the local setting.

Case Report

Our patient is a 54-year-old, post-menopausal, Filipino female who complained of a 2-year history of typical angina. Two years prior to admission, she developed substernal chest discomfort on moderate exertion which was relieved by rest. There was no consult done and no medications were taken. Six months prior to admission, there was worsening angina, now precipitated by mild exertion. On consult, work-ups were suggestive of ischemia and she was referred for ICA with intent to do revascularization.

Cardiac risk factors included her age (post-menopausal), a family history of premature coronary disease (mother died of myocardial infarction at 44 years old) and poorly controlled hypertension. She has mild bronchial asthma which is controlled with salmeterol + fluticasone inhalation. Past surgical history is unremarkable. She has no diabetes, does not smoke and has no intake of oral contraceptive pills/hormone replacement therapy or illicit drugs.

Physical examination were as follows: blood pressure = 160/80, heart rate = 84 regular, respiratory rate = 20, afebrile. BMI = 24. HEENT and chest/lung findings were unremarkable. Cardiac examination showed normal JVP, normal carotid pulse, and adynamic precordium with the apex beat at the 5th intercostal space, midclavicular line. There were no heaves, lifts, nor thrills. No murmurs were appreciated. Abdominal and peripheral vascular examination were normal as well. Electrocardiogram showed normal sinus rhythm, left axis deviation, left atrial abnormality, old anteroseptal wall infarct and high lateral wall ischemia. Chest x-ray showed borderline left ventricular cardiomegaly. Transthoracic 2-D echocardiogram showed normal LV dimension with good wall motion, contractility and normal systolic function (LVEF = 75% by Simpson's). A treadmill stress test showed a 1.5 mm ST-segment depression in the inferior and anterolateral leads at 6.6 METS (modified Bruce protocol). A Tc99m Sestamibi Stress MPI showed moderate stress-induced ischemia in the apical region and normal overall LV systolic function (LVEF at rest = 79%, LVEF post-stress = 81%). Laboratory tests were unremarkable with normal CBC and platelet counts, bleeding parameters and renal function. Working impression was atherosclerotic cardiovascular disease, hypertensive cardiovascular disease, chronic stable angina, in sinus rhythm, CCS II, NYHA FC II and coronary angiography was advised.

After informed consent was obtained, routine diagnostic ICA was done via the right transradial approach using a 6 Fr radial sheath. No difficulties were noted during cannulation of both left coronary artery (LCA) and right coronary artery (RCA) systems using a 5 Fr. Tiger diagnostic catheter. There was no left main disease. The left anterior descending (LAD) was a large-sized vessel with a significant tandem stenosis of 95% and 80% at its proximal segment; there was TIMI 2 distal flow. D1 was a small-sized vessel with an 80% stenosis at its ostium. D2 and D3 were small-sized vessels which were free of disease. The left circumflex (LCx) was a large-sized, non-dominant vessel which was free of significant disease. It gave off one small-sized OM branch with early take-off which was free of disease. The right coronary artery (RCA) was a large-sized, dominant vessel with a 70-80% stenosis at its ostium. After the initial contrast injection, there was note of a dissection plane at the RCA proximal segment (Type B), a few millimetres after the tip of the diagnostic catheter, which extended to the mid segment. There was note of extension of the dissection plane with diminishing distal flow (from TIMI 3 to TIMI 2) during the succeeding injections. Please see Appendix A for the classification of coronary dissection by Klein et. al. based on the angiographic appearance and the chance of acute closure associated with each morphology.?

Upon recognizing ICAD, immediate PCI of the RCA was pursued. A Kimny Fr. 6 guide catheter was used to cannulate the RCA ostium. A PT2 PCI wire was positioned distally into the RCA. Pre-stent dilatation of the proximal to mid RCA was done using a Maverick 2.0 x 20 mm PCI balloon at 14 atm (2.23 mm) for 12 seconds. A Firebird 3.5 x 33 mm drug-eluting stent (DES) was positioned into the proximal RCA. After pre-stent dilatation and during cine shots for stent positioning, there was noted extension of the dissection into the distal segment (Type D) followed by subsequent abrupt closure resulting to acute occlusion (Type F) of the posterior descending artery (PDA) and posterolateral artery (PLA) branches. The patient developed angina with concomitant sinus bradycardia and ST-segment elevation of the inferior leads. Intravascular ultrasound (IVUS) using an Opticross catheter showed a long segment dissection from the proximal to the distal RCA extending to immediately before the crux. There was significant reduction of the true lumen diameter with smallest MLA of 3.39 mm². Moreover, wire position in the true lumen was confirmed via IVUS. The following stents were immediately deployed: 1.) Firebird 3.5 x 33 mm DES at 9 atm (3.5 mm) for 9 seconds (proximal to mid) and 2.) Firebird 3.0 x 33 mm DES at 9 atm (3.0 mm) for 7 seconds (mid to distal). There was also significant ostial calcification and stenosis on IVUS (MLA = 3.4 mm², plaque burden = 72%), hence, another stent, a Firebird 3.5 x 13 mm DES was deployed at the ostial RCA at 16 atm (3.8 mm) for 5 seconds. Post-dilatation was done using the stent balloon. Post-stenting IVUS run showed good stent apposition, no edge dissections, and a proximal segment MLA of 9.8 mm². Final angiographic shots showed no residual stenosis and TIMI 3 flow. No residual dissection planes were noted. After RCA stenting, there was resolution of angina and inferior wall ST-segment elevation. PCI of the LAD then followed uneventfully. ?

The patient was closely monitored in the coronary care unit for 24 hours and remained stable with no recurrence of angina. She was discharged asymptomatic a few days following the procedure and were given the following medications: aspirin 80 mg/tab OD, ticagrelor 90 mg/tab BID, rosuvastatin 20 mg/tab ODHS, telmisartan 80 mg/tab OD, verapamil 80 mg/tab BID, ISMN 30 mg/tab OD and was advised cardiac rehab and regular OPD follow-up.

Discussion

Inadvertent coronary artery dissection (ICAD) is a rare but potentially catastrophic complication of coronary angiography or angioplasty. The incidence of ICAD for ICA and PCI is 0.008-0.02% and 0.06%-0.15%, respectively.^{2,3} The incidence is somewhat higher in the setting of acute coronary syndromes and is approximately 0.19%.² Although ICAD more commonly involves the LCA, some have studied the risk factors and causes for ICAD of the RCA and have identified the following: 1) the LCA subtends an acute angle (20° - 25°) with the ascending aorta, whereas RCA is relatively more straight (60° - 88°); thus, LCA catheterization is more co-axial when compared to the RCA, 2.) the ostium of the LCA is greater than that of the RCA in >76% of cases, and 3.) histologically, the ostium of the LCA has more Type I than Type III collagen (results in more tensile strength), while the opposite is that of the RCA.² Risk factors identified for ICAD include hypertension, aging, increased calcification of the aortic root or coronary lesion site, recent ACS, and intra-aortic balloon pump support.⁵ In our patient, in addition to the risk factors innate to the RCA mentioned above, age and ostial lesion location and calcification might have contributed to the increased the risk for ICAD as aging is associated with cystic medial degeneration and ostial lesion calcification with increased risk of dissection.^{2, 5} Selection of appropriate diagnostic/guiding catheters, careful manipulation, optimal coaxial alignment of the catheter, and gentle contrast injection are recommended to prevent ICAD.⁶ In our case, there was no difficulty during RCA cannulation but there was noted sudden deep intubation even when careful clockwise rotation was done while cannulating the RCA ostium. The diagnostic catheter flicked and reached up to >10 mm past the RCA ostium (not a rare occurrence due to the horizontal orientation of the RCA ostium and small size of the diagnostic catheter). Although diagnostic catheters are softer than guide catheters, the sudden deep intubation may have caused an endothelial break which created an intimal flap and exposed the media causing intramural hematoma formation and subsequent mechanical obstruction causing decreased distal flow. A cycle of further stasis, thrombosis, and to some extent vasoconstriction then occurred.⁹

Moreover, in our case, serial pre-stent dilation was done and several cine shots were made before stent deployment which could have possibly caused expansion of the intramural hematoma and spiralling of the dissection to the distal segments causing abrupt closure and TIMI 0 distal flow to the PDA and PLA branches (progression of initial Type B to Type D and then Type F coronary artery dissection, increasing the chances of abrupt closure from 2-4% to 30% and 69%, respectively⁷). During ICAD with abrupt closure, expeditious efforts to restore antegrade flow must be quickly attempted and urgent stenting of the point of origin is usually required to avoid distal propagation and stabilize dissections. The use of intravascular imaging such as IVUS or optical coherence tomography (OCT) should be considered to clearly define the pathology and intervention.^{7,8} In our case, IVUS played a vital role as it confirmed intraluminal wire placement and provided guidance for stent sizing which permitted successful obliteration of the false lumen and restoration of antegrade flow. In addition, IVUS also confirmed optimal stent expansion after PCI.

Management following successful reestablishment of antegrade flow include admission to a coronary care/telemetry unit for close observation and supportive treatment of other complications (i.e. if an intra-aortic balloon pump or pulmonary artery catheter was inserted, these should remain in place until clinical stability is ensured). Glycoprotein IIb/IIIa inhibitors and anticoagulants may be administered for 12-24 hr, but most data are anecdotal. If a thrombus remains at the completion of the procedure, long-term therapy with DAPT (aspirin

+ clopidogrel) and anticoagulation (warfarin and/or enoxaparin) may be considered, but most evidence are primarily anecdotal as well. Serial ECGs and cardiac enzymes may aid in assessing the size and extent of the myocardial damage. An elevation of creatine kinase or creatine kinase-MB (>3x normal) has been associated with worse long-term outcomes in some studies. Recurrent chest pain/ischemia should be managed urgently and the benefits and disadvantages of coronary artery bypass graft surgery (CABG) must be considered in a case-to-case basis. Irreversible closure of a small branch without significant effect