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REARCH ARTICLE

Zero Contrast Percutaneous Coronary Intervention – A novel approach to reduce Contrast Induced Acute Kidney Injury

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ABSTRACT

Iodinated contrast media are required in all cases who undergo coronary angiography and percutaneous coronary interventions (PCI). Though the modern contrast media (both low-osmolar and iso-osmolar) have lesser adverse effects than the older generation contrast media, they may lead to life threatening complications in few cases. Contrast induced acute kidney injury (CI-AKI) is one such complication where the presentation may vary from asymptomatic mild elevation in serum creatinine to life threatening uremia. Various pre-procedural and procedural steps are followed commonly to prevent CI-AKI and reducing the contrast volume is one of the most important steps. Indeed, multiple studies have proven that reducing the contrast volume during PCI reduces the risk of CI-AKI. More recently, intra-coronary imaging guided zero-contrast PCI has emerged as an important method to prevent CI-AKI. Though randomised controlled trials comparing low-contrast PCI and zero-contrast PCI are lacking, this technique is being used widely by experienced operators. Technical expertise in complex PCI and meticulous analysis of intra-coronary imaging, particularly intravascular ultrasound (IVUS), are mandatory for this procedure.

Introduction:

CI-AKI is a serious complication of PCI and it manifests as a reduction in renal function within days after exposure to contrast media. The Kidney Disease: Improving Global Outcomes (KDIGO) defines it as an increase in serum creatinine by ≥0.3mgs% within 48 hours of contrast administration or ≥ 50% increase within 7 days.[1] Recently, the term "Contrast associated AKI (CA-AKI)" is frequently used, since the post-procedural worsening of renal function may also be caused by hypotension, drugs and athero-embolism. CI-AKI is often underdiagnosed. Though there is a descending trend in the incidence of AKI among patients hospitalised with acute myocardial infarction,[2] the incidence of AKI among patients undergoing percutaneous coronary procedures has increased significantly.[3] Furthermore, the AKI related incremental cost is estimated to be around $8,416 and $580 per inpatient procedure during index hospitalisation and readmissions respectively.[3] Additionally, CI-AKI is associated with increased mortality with 36% and 12% chances of death during hospitalisation and within 1-year of discharge respectively.[4-5] There is a large variation in the volume of contrast used among operators. Data from the National Cardiovascular Data Registry Cath-PCI (NCDR Cath-PCI) registry showed that this variation was around 23% among operators[6] and the CI-AKI risk had increased 42% for every 75ml of incremental contrast used. Hence, reducing the contrast usage during a procedure is an important way to reduce the risk of CI-AKI. The MOZART trial showed that the IVUS guidance in PCI decreases the contrast volume requirement during PCI[7]. Moreover, recent studies have shown that ultra-low contrast (Contrast volume (CV) < estimated glomerular filtration rate (eGFR)) reduces the CI-AKI risk. Zero-contrast PCI is a novel method that requires extremely skilled PCI operators and meticulous analysis of intra-coronary imaging for its successful clinical application and it is a high potential strategy to prevent CI-AKI and to improve survival after PCI.

Epidemiology and outcome of CI-AKI:

The incidence of CI-AKI ranges between 3-20%.[8] According to the NCDR Cath-PCI, the incidence of CI-AKI and AKI requiring dialysis after PCI were 7.1% and 0.3% respectively.[9] A recent meta-analysis estimated the incidence of CI-AKI and the need for dialysis after coronary angiography as 9.06% and 0.52% respectively.[10] However, the incidence of clinically significant CI-AKI after coronary angiogram in the PRESERVE trial was 1.2%. Notably, all the patients in that trial had eGFR of 15 to 44.9 ml/min/1.73 m2 or 45 to 59.9 ml/min/1.73 m2 and diabetes mellitus.[11] Moreover, the incidence of CI-AKI after coronary angiography and PCI is higher than that of Computed Tomography (CT) imaging because of varying clinical characteristics, route of administration and contrast volumes.[12] Intra-arterial contrast administration has a higher risk of CI-AKI than intra-venous contrast administration.[13] Important risk factors for CI-AKI include cardiogenic shock, ST-elevation myocardial infarction (STEMI), acute heart failure, anaemia, advanced age, pre-existing renal

dysfunction, low ejection fraction (EF), diabetes mellitus and volume depletion. CI-AKI is associated with increased short-term and long-term mortality. Furthermore, myocardial infarction, stent thrombosis, target lesion revascularisation and major adverse cardiac events are higher among patients with CI-AKI.[14] Severe CI-AKI (an increase in serum creatinine of >50% from the baseline value, an absolute increase of >1mg/dL or requiring dialysis within 7 days) is associated with 2-fold increased risk of major adverse cardiovascular events (MACE) at 5 years after PCI. Though both severe CI-AKI and mild CI-AKI are independent predictors of MACE, the association is severity dependent.[15] An analysis of the ADAPT-DES registry revealed that patients who developed CA-AKI were at higher risk of all-cause and cardiovascular mortality on 2-year follow-up and the relative risk of adverse outcome was highest among patients with pre-existing chronic kidney disease (CKD).[16] Furthermore, both transient and persistent AKI are associated with the risk of rapid CKD progression despite recovery.[17]

Risk prediction:

Female sex, older age, hypertension, diabetes mellitus, heart failure, CKD, STEMI, intra-aortic balloon pump use, Killip Class II-IV, hypotension, and number of stents were independent predictors of CA-AKI in the ADAPT DES registry.[16] Risk assessment of CI-AKI may be done using clinically validated scores like Mehran score, Blue cross blue shield of Michigan Cardiovascular Collaborative (BMC2) risk calculator and the NCDR Cath-PCI model.[9,18,19] More recently, another contemporary risk score with eight clinical and four procedural variables has been developed and it has a good discrimination power.[20] In contrast to the Mehran score, where the risk of CI-AKI can be assessed only after PCI, the BMC2 risk prediction model may be used before procedure.[21] Though many risk prediction tools have been introduced in the literature, none of them are followed systematically in clinical practice. The reasons are that most of these models are not validated externally and the therapeutic implications of various risk categories are not clearly defined.[22] Many renal biomarkers have been investigated to predict CI-AKI. Urinary dickkopf-3 (DKK3) is a stress induced renal tubular glycoprotein and pre-procedural urinary DKK3/creatinine ratio ≥322 pg/mg predicts CI-AKI in patients with CKD undergoing invasive cardiovascular procedures.[23] Neutrophil gelatinase–associated lipocalin (NGAL) is an early marker of AKI and urine NGAL <20 ng/mL and serum NGAL <179 ng/mL at 6 hours after procedure are reliable markers to rule out CI-AKI.[24] A sub study of the PRESERVE trial had shown that pre-angiography levels of injury (KIM-1, NGAL, IL-18) and repair (MCP-1, UMOD, and YKL-40) proteins, modestly predicted major adverse kidney events and only plasma KIM-1 levels were associated with CA-AKI.[25]

Prevention of CI-AKI:

Pre-procedural strategies:

Adequate hydration and reduction in contrast volume are the two main strategies suggested by the American Heart Association (AHA) to

prevent CI-AKI.[26] High dose statins and using radial access are other important steps to prevent AKI after a procedure. Various studies have shown that intravenous normal saline administration prevents CI-AKI.[27,28] Increased clearance of contrast through urine, reduced vasoconstriction and improved resistance to reactive oxidant species are the main mechanisms of the benefit of hydration.[29] Furthermore, intravenous volume expansion downregulates the renin-angiotensin-aldosterone system and suppresses tubulo-glomerular feedback by diluting the contrast within the tubular lumen.[30] However, excessive volume expansion may precipitate acute decompensation in patients with underlying left ventricular systolic dysfunction. Hence, an individualised left ventricular end-diastolic pressure (LVEDP) based hydration was administered in the POSEIDON trial and CI-AKI occurred less frequently in LVEDP based hydration than with the standard hydration. In that trial, intravenous normal saline was infused during procedure and continued till 4 hours after procedure at a rate of 5ml/kg/hr for LVEDP <13mmHg, 3ml/kg/hr for LVEDP 13-18mmHg and 15ml/kg/hr for LVEDP >18mmHg.[31] Similarly, another study has shown that central venous pressure (CVP) based hydration reduced CI-AKI more than the standard hydration. Normal saline was administered at a rate of 3ml/kg/hr if CVP <6 cm H2O, 1.5ml/kg/hr if CVP 6-12 cm H2O and 1ml/kg/hr if CVP > 12 cm H2O in that trial.[32] Another strategy called "Furosemide with matched hydration" using RenalGuard system (PLC Medical Systems, Milford, Massachusetts) has also been tried in a few trials and it has reduced CI-AKI significantly.[33-35] The 2018 European Society of Cardiology (ESC) guidelines on myocardial revascularisation advocates class I recommendation for adequate hydration before coronary angiography and class IIb recommendation for tailored hydration regimens in patients with moderate or severe CKD.[36] Similarly, pre-treatment with high dose statins is recommended by both AHA and ESC and various studies have shown that statins reduce CI-AKI significantly and consistently.[37,38] Urinary alkalinization is another proposed method to reduce oxygen free radical induced damage. However, the PRESERVE trial did not show any benefit of intravenous sodium bicarbonate over normal saline in terms of death, requirement for dialysis, decline in renal function at 90 days or CI-AKI.[39] Furthermore, this trial showed no benefit of oral N-acetyl cysteine over placebo. Similarly, the ACT trial also showed that acetylcysteine did not have any benefit in high-risk patients undergoing coronary or peripheral angiography.[40] Similar to these, many other drugs like ascorbic acid, fenoldopam, calcium channel blockers, prostaglandin analogs, atrial natriuretic peptide, endothelin antagonists, dopamine, theophylline and allopurinol have been tried and so far, none were consistent in preventing CI-AKI.[41] Nephrotoxic medications should be withdrawn 24 hours prior to procedure and 48 hours after procedure.

Procedural strategies:

Selection of contrast agent for the procedure is important since the risk of CI-AKI is lower

with the use of low-osmolar agents when compared to high-osmolar agents, particularly in patients with renal dysfunction.[42,43] However, the results of studies comparing low-osmolar agents with iso-osmolar agents are conflicting.[44-47] The ESC recommends the usage of low-osmolar or iso-osmolar agents for patients with moderate to severe CKD who undergo coronary angiography. Contrast volume is an independent predictor of CI-AKI after adjusting the baseline risk factors.[48,49] Hence, reduction in contrast volume is the predominant procedural strategy to prevent CI-AKI following PCI. However, the safe limit of contrast volume is still debated and various formulae have been used in various studies. The maximal acceptable contrast dose (MACD) is calculated as 5ml x body weight (kg) / serum creatinine (mgs%)[50] and it has been validated in various studies. Another method to calculate the safe contrast limit is to calculate the CV based on renal function i.e., Creatinine clearance (CrCl) or eGFR. An analysis of BMC2 registry showed that the risk of CI-AKI had increased significantly when the CV/CrCl ratio was >2.[51] Similarly, another analysis of the National Heart, Lung, and Blood Institute (NHLBI) dynamic registry showed that the CV/ CrCl ratio of > 3.7 was associated with a significant rise in serum creatinine levels. Low-contrast volume is defined as a CV/CrCl ratio of 1-3 and Ultra-low contrast volume is defined as CV/CrCl ratio <1. Ultra-low contrast volume is associated with lower incidence of CI-AKI and the need for dialysis.[52] The ESC recommends that contrast volume should be minimized with the total contrast volume to eGFR ratio of <3.7.[36] DyeVert plus system (Osprey Medical, Minneapolis, Minnesota) is a dedicated device that reduces the contrast dose while maintaining the image quality and it has been shown to reduce contrast volume by 40%.[53] Various other technical steps to reduce the contrast volume have been described. These include, using smaller catheters, diluted contrast, biplane angiography, stent enhancement techniques, high acquisition rates, IVUS etc.[54] In the MOZART trial, extensive usage of IVUS to guide PCI reduced the contrast volume significantly. Even though the procedural time had increased significantly in the IVUS guided PCI group, the cumulative air kerma and fluoroscopic time were similar to angiographic guided PCI in the MOZART trial.[55]

Other adverse effects of iodinated contrast:

Allergic reactions to iodinated contrast media are relatively uncommon with the current low-osmolality agents and most of them are mild and non-life-threatening. Acute reactions to contrast media can be allergic or physiologic reactions. Acute allergic reactions may vary from simple erythema or urticaria to life-threatening anaphylactic shock or cardiac arrest. These allergic reactions are dose and concentration independent. Acute physiologic reactions are due to direct chemotoxicity or osmotoxicity and they are dose-dependent. These physiologic reactions vary from simple vomiting, warmth and vasovagal reaction to life threatening arrhythmias, pulmonary edema and convulsions. Contrast related hyperosmolality

and hypocalcaemia due to calcium binding are the reasons for these physiologic reactions.[56] Delayed adverse effects can happen 30-60 minutes to weeks after exposure to contrast media and mostly they are cutaneous manifestations like erythema, persistent rash etc. Rarely, SLE, iodine mumps and polyarthropathy may be the manifestations of delayed reactions.

Zero contrast PCI:

Though iodinated contrast media is considered essential to perform coronary procedures, these procedures may also be done without contrast media. Zero-contrast PCI is feasible with the use of intra-coronary imaging. Though both IVUS and optical coherence tomography (OCT) may be used for zero-contrast PCI guidance, IVUS is commonly used. The first feasibility study on IVUS guided Zero-contrast PCI was done by Ziad Ali et al., among patients with advanced renal dysfunction and none of these patients developed CI-AKI or MACE on follow-up.[57] However, their study was done on relatively fewer complex lesions with a median Synergy Between PCI With Taxus and coronary artery bypass surgery (CABG) (SYNTAX) score of 12 and most of the patients received a single stent. Further studies with more complex lesions were done later and most of these studies had high success rates and good short-term outcomes.[58,59] Apart from these, many case reports on highly complex lesions like rotational atherectomy, left main bifurcation PCI, Chronic total occlusion (CTO) etc. have been published and all these suggest that even in complex lesion subsets, zero-contrast PCI is feasible.[60-62] Zero-contrast PCI under OCT guidance may also be done using dextran 40 or hydroxy ethyl starch.[32,64] Patient selection and timing of the procedure are also important. We followed a simple approach for patient selection in our study[58] where patients with renal dysfunction were considered for zero-contrast PCI when they had met the following criteria:

(1) eGFR < 30 ml/min/1.73 m2;

(2) Serum creatinine > 1.5 mgs% or eGFR 30-45 ml/min/1.73 m2 **and** any of the following

- (a) Age > 75 years
- (b) Left ventricular ejection fraction (LVEF) < 35%
- (c) Diabetes mellitus
- (d) Recent hypotension or shock.

Timing of the procedure:

An initial coronary angiogram should be performed with ultra-low contrast volume (CV <eGFR) and subsequent PCI can be staged in stable patients. The duration between a coronary angiogram and subsequent zero-contrast PCI is variable and it is dependent on the patient's clinical condition and the operator preference. There is no uniform time interval followed in most of the studies but many studies had an average time interval of one week.[57,59] Most of the low osmolar contrast media have a half-life of 2 hours and in patients with normal renal function, it takes 20 hours for the contrast to be eliminated. Hence, it is logical to give an interval of at least 24 hours, but this interval is not recommended by the ACR committee on contrast media.[56] Furthermore, this interval may vary in patients with renal dysfunction. Hence, this approach cannot be universalised.

Additionally, patients with acute coronary syndrome and ongoing angina may require urgent revascularisation and we have demonstrated in our study that immediate zero-contrast PCI following coronary angiography is feasible and it is associated with low incidence of CI-AKI.[58] Hence, a personalised decision should be taken regarding the time interval between the coronary angiogram and zero-contrast PCI.

Procedural steps:

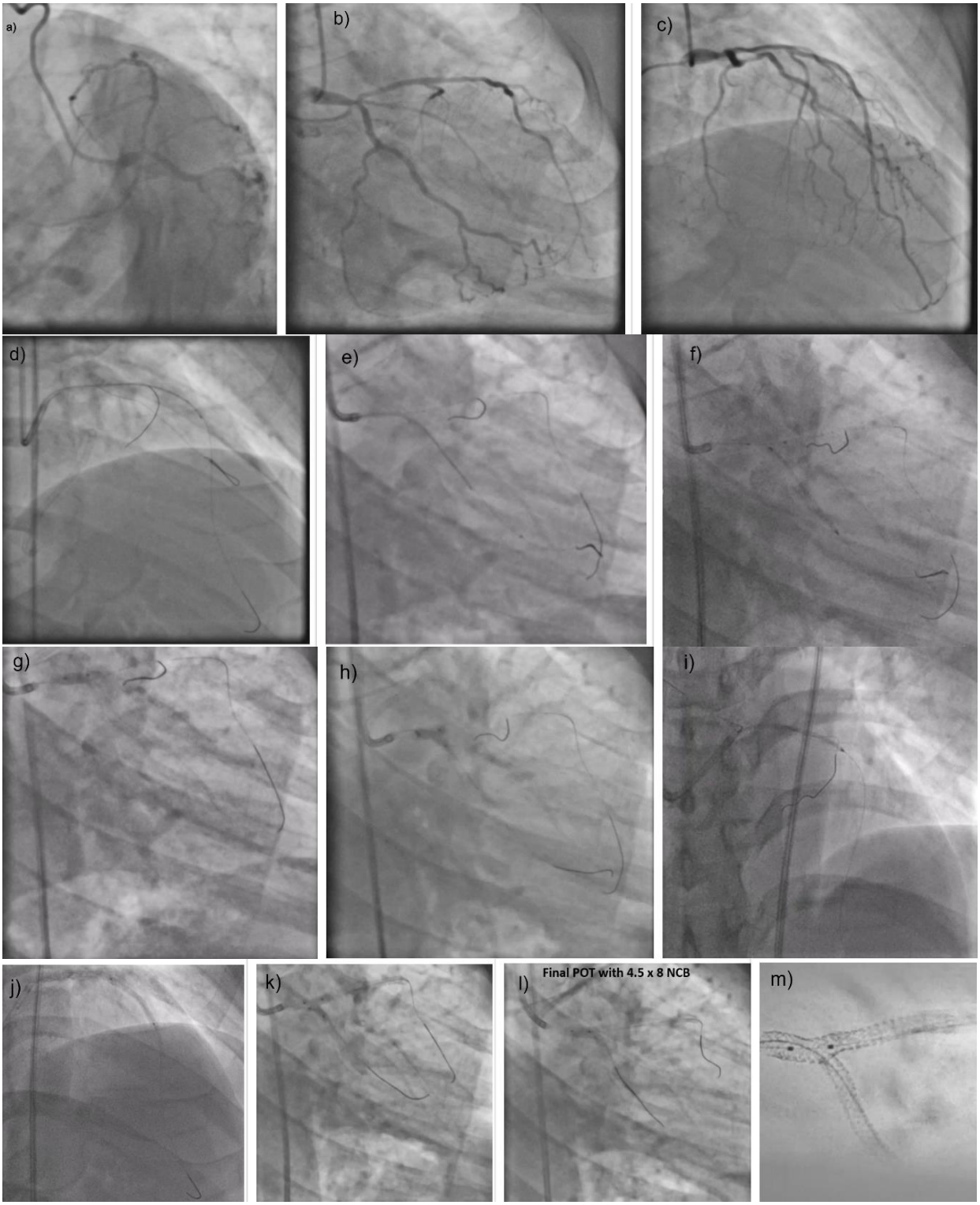
IVUS guided Zero-contrast PCI is feasible and safe in most patients who are at high risk of CI-AKI and studies have shown that even complex cases can be done without complications.[58] Hydration with normal saline should be started as per the standard protocols. In emergency procedures, hydration may be started as soon as possible according to the hemodynamic status of the patient. Baseline eGFR, electrocardiogram and echocardiogram should be recorded before the procedure. The femoral arterial approach allows us to use larger guiding catheters with better support for complex PCI, though the radial approach is also feasible in most cases. After guiding catheter engagement, a metallic silhouette of the vessel is created using multiple wires as markers for side branches and the ostium of the main vessel. Left circumflex artery (LCX) wire acts as a marker to locate left anterior descending artery (LAD) ostium and LAD wire acts as a marker to locate LCX ostium. The left main coronary artery (LMCA) ostium is located by a floating wire in the aortic sinus. It is important to note that the guidewire insertions should be done in the same fluoroscopic projections as that of the previous angiogram and the IVUS run confirms the luminal position of the guidewires. After metallic silhouette creation, an IVUS run is done to select the appropriate strategy for lesion preparation, to select the stent size and to identify the landing zones. Proximal and distal landing zones are identified fluoroscopically by their distances from the nearest marker wires and these distances are measured during analysis of the initial IVUS run. After stenting, an IVUS run is done again to identify the complications and for stent optimisation. During the procedure, strict monitoring of the patient's symptoms, electrocardiogram and echocardiogram is required. If complications are suspected, check angiogram using minimal contrast should be taken immediately to avoid serious consequences. After procedure, serial echocardiographic monitoring is essential to rule out pericardial effusion.

Case example:

A 59-year-old male, with a long-standing history of hypertension, diabetes mellitus and dyslipidaemia, was admitted in our hospital with rest angina for 3 days. His serum troponin level was elevated and the serum creatinine and eGFR were 1.3mgs% and 56.5ml/min/1.73m^2 respectively. His coronary angiogram showed significant LMCA stenosis with 3-vessel disease and the SYNTAX score was 24. Since the patient was not willing to go for CABG, staged PCI was planned. Initially, PCI to right coronary artery (RCA) was done with 2 drug eluting stents (DES). Post

procedure, the serum creatinine increased to 2.8mgs% and he was managed conservatively for CI-AKI. He recovered from the renal injury over a period of 2 weeks and the serum creatinine level came down to 1.6 mgs%. In view of this CI-AKI, further PCI to left system was planned as IVUS guided zero-contrast procedure. Furthermore, the LMCA bifurcation had medina (1,1,1) lesion *(Figure 1)* and hence an upfront 2-stent strategy with DK-crush technique was planned for LMCA bifurcation. Through right femoral artery access, a 7F EBU 3.5 guiding catheter was used to engage LMCA. Then, keeping the previous angiogram as a roadmap, both LAD and LCX were wired with the Fielder-FC guidewires in the same angiographic projections. Another Fielder-FC wire was parked in a septal artery as a marker wire. Then IVUS runs were done from LAD-LMCA and major obtuse marginal (OM)-LMCA and the parameters are given below (Table 1 & Figure 2). IVUS runs confirmed the luminal position of both LAD and LCX guidewires. Then the LAD lesion was pre-dilated with a 2.5x12mm non-compliant (NC) balloon and a 3x6mm cutting balloon was used at the ostial LAD and distal LMCA. Similarly, the LCX lesion was pre-dilated with 2x9mm NC balloon and 3x6mm cutting balloon was used at the ostial LCX. Then a 3x33mm DES was deployed at LCX-OM keeping the LAD wire as a marker to locate LCX ostium. The LCX stent was crushed using 3.5x12mm & 4x 8mm NC balloons and it was recrossed with Fielder-FC wire. First kissing balloon inflation was done using 3.5x12mm & 3.5x8mm NC balloons. Then the LCX wire was removed and it was kept in the left aortic sinus as a marker for the LMCA ostium. Then a 3.5x 38mm DES was deployed from the ostial LMCA-LAD, till the septal marker wire. After LMCA POT with a 4.5x8mm NC balloon, a 3x28mm DES was deployed from the distal end of the LMCA-proximal LAD stent with a 2mm overlap. LCX was recrossed again and the second kissing balloon inflation was done using 4x8mm & 3.5x8mm NC balloons. The final proximal optimisation technique (POT) was done using a 4.5x8mm NC balloon. Then IVUS runs were done from LAD-LMCA and OM-LMCA and the parameters are given below (Table 2 & Figure 3). IVUS showed adequate stent expansion without edge dissection or malapposition. The entire procedure was done without contrast usage and the guide catheter was flushed with saline intermittently to avoid thrombus formation. Hydration with normal saline was started 6 hours prior to the procedure at the rate of 1.5ml/kg/hr and it was continued till 12 hours after the procedure. After the procedure, serial echocardiogram was done to rule out pericardial effusion and there was no significant rise in serum creatinine level. On 3 months follow-up, the patient remains symptom free.

Figure 1: Procedural steps of zero-contrast LMCA bifurcation PCI



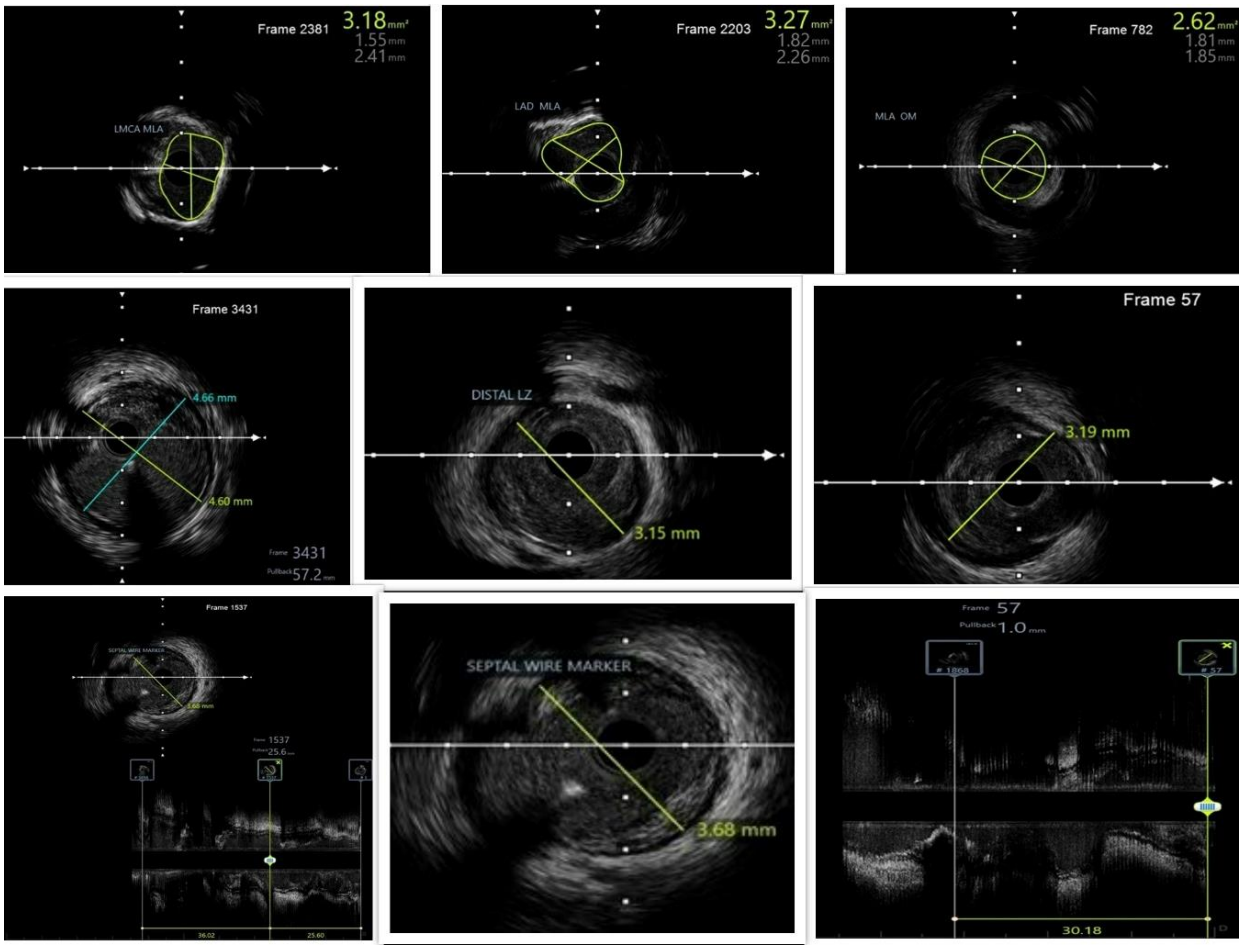
a, b, c): Angiographic images show significant LMCA bifurcation lesion with medina (1,1,1).
d) Both LAD & LCX were wired and a marker wire was kept in septal artery. IVUS run from LAD-LMCA was done.

e) IVUS run from OM-LMCA was done.
f) LCX-OM stenting was done with 3x33mm DES. A 3.5x 12mm NC balloon was kept in LAD for crush.
g) 4x8mm NC balloon was used for repeat crushing.
h) First kissing balloon inflation was done with 3.5x12mm & 3.5x8mm NC balloons.
i) A floating wire was kept in left aortic sinus as a marker for LMCA ostium. A 3.5x38mm DES was deployed from LMCA ostium to septal marker wire.
j) Mid LAD was stented with 3x28mm DES overlapping the first stent.
k) Second kissing balloon inflation was done with 4x8mm & 3.5x8mm NC balloons.
l) Final POT was done with 4.5x8mm NC balloon.
m) 'Clear stent' image of LMCA bifurcation stenting.

Table 1: IVUS analysis before stenting:

LMCA diameter	4.60mm
Proximal LAD diameter	3.60mm
Distal reference segment diameter at LAD	3.10mm
Distal reference segment diameter at major OM	3.10mm
Distal LMCA luminal area	3.18mm^2
Ostial LAD luminal area	3.27mm^2
Ostial LCX luminal area	2.60mm^2
Length between ostial LMCA to septal marker wire	36.0mm
Length between septal marker wire to distal LAD reference segment	25.60mm
Length between ostial LCX and Distal OM reference segment	30.10mm

Figure 2: IVUS analysis before stenting:



Top row shows MLA at distal LMCA, LAD and OM as 3.18mm^2, 3.27mm^2 and 2.62mm^2 respectively.

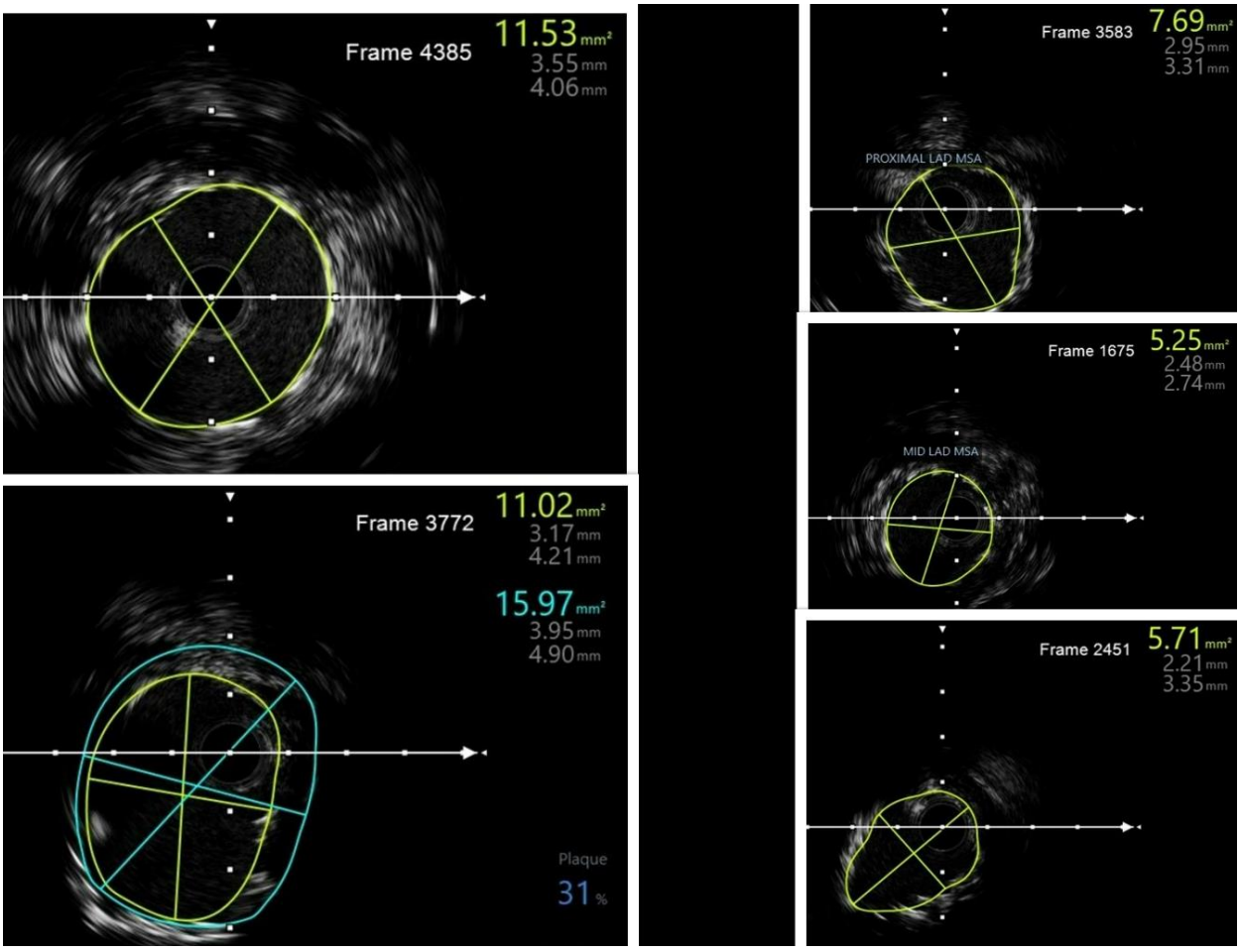
Middle row shows vessel diameters at LMCA, distal LAD and OM as 4.6mm, 3.1mm and 3.19mm respectively.

Bottom row shows length measurements in LAD and OM. *Bottom left* image shows the length from LMCA ostium to septal marker wire as 36mm and septal marker wire to distal landing zone as 26.5mm. Similarly, *bottom right* image shows the length from ostial LCX to distal landing zone at OM as 30.1mm.

Table 2: IVUS analysis after stenting:

Mid LAD MSA	5.25mm^2
Proximal LAD MSA	7.69mm^2
Distal LMCA stent area	11.02mm^2
Proximal LMCA stent area	11.53mm^2
LCX MSA	5.71 mm^2

Figure 3: IVUS images after stenting:



Left upper and lower images show MSA at proximal and distal LMCA as 11.53mm^2 and 11.02mm^2 respectively.

Right panel shows MSA at proximal LAD, mid LAD and OM as 7.69mm^2, 5.25mm^2 and 5.71mm^2 respectively.

Conclusion:

Patients with CKD are at high risk of cardiovascular events and 50% of all deaths in stage 4 & 5 of CKD patients are due to cardiovascular mortality.[65] However, the utilization of coronary angiogram and PCI is less among CKD patients, particularly those who are not on renal replacement therapy.[66] Fear of precipitating CI-AKI is the predominant reason for this under-utilization of revascularisation procedure. Advances in technology and intra-coronary imaging help modern operators to perform PCI without contrast media. Operator experience in understanding the coronary anatomy, tactile feedback of guidewires and intra-coronary image interpretation are essential for the successful completion of this procedure. The widespread use of this technique has the potential to decrease mortality by reducing CI-AKI and to provide a better quality of life for patients with advanced CKD.

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