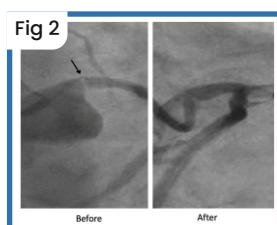
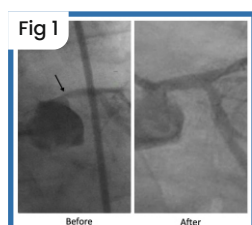
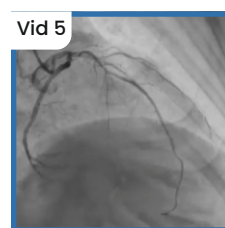
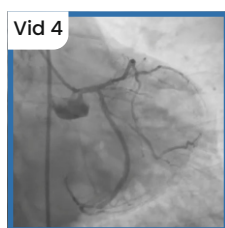
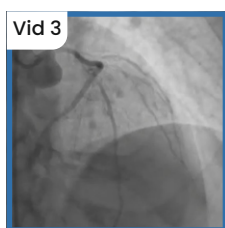
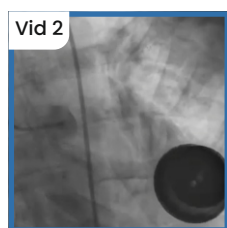
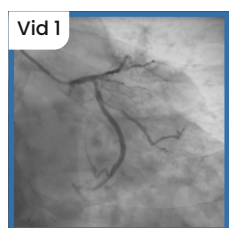




Catheter Induced Spasm of Left Main

A 58 y old male was taken for angioplasty of a 90% stenosis of a large obtuse marginal (OM) artery (Video 1). He had recent onset rest angina with elevated Troponin and "T" inversion in lateral leads. His LVEF was normal on echo. Right radial approach was used. A 6F XB catheter was used to engage the left main. A few seconds after the left main was engaged there was flat line on cardiac monitor indicating complete absence of electrical activity of heart. There was no pulse and blood pressure. The guide catheter was pulled out, cardiopulmonary resuscitation initiated and endotracheal intubation was performed immediately. One milligram of adrenaline was given intravenous every 2–3 minutes. Electrical activity resumed after 3 doses of adrenaline. The ECG on monitor showed "tombstone" type ST segment elevation. Blood pressure was about 40 mm Hg. Intravenous fluids and noradrenaline infusion were started. Echo showed a dilated LV with LVEF of 0.20. There was severe metabolic acidosis on ABG. A cuspal coronary injection showed discrete spasm of left main (Video 2,3 and Fig 1 & 2 arrow). Pressor support, correction of acidosis, ventilatory support was continued. Blood pressure increased to 70–80 mm Hg and ECG showed decrease in ST segment elevation. A repeat coronary injection revealed disappearance of left main spasm with TIMI-3 flow in the coronary (Video 4,5 and Fig 1,2). He was managed in CCU and was extubated after 48 hours. LV function was restored to normal and renal function remained normal. His angioplasty and stenting was performed via femoral route on day 5 after the episode with excellent angiographic result (Video 6)



Discussion

The left main supplies approximately 75–100% of the left ventricular myocardium (depending on coronary dominance). Severe spasm can therefore produce:

- Abrupt reduction or cessation of flow to both the LAD and LCx territories
- Profound ischemia
- Severe chest pain and ST-segment elevation
- Hypotension or cardiogenic shock
- Ventricular tachycardia/fibrillation
- Cardiac arrest in extreme cases

Mechanism

The spasm is usually triggered by:

- Mechanical irritation of the LM ostium by the guide catheter
- Deep intubation
- Repeated engagement/disengagement
- Pre-existing vasospastic tendency
- Large or aggressively shaped guides

Recognition

Warning signs during angiography include:

- Sudden LM narrowing immediately after engagement
- Pressure damping or ventricularization
- Loss of distal coronary flow
- ST-segment changes
- Hemodynamic deterioration

Immediate management

1. Withdraw/disengage the guide catheter immediately.
2. Administer intracoronary nitroglycerin (or occasionally verapamil/nicardipine if needed) if blood pressure permits
3. Provide hemodynamic support if necessary.
4. Repeat angiography to confirm resolution.
5. If the appearance persists, consider LM dissection and evaluate with IVUS/OCT as appropriate.

Distinguishing from left main dissection

A critical point is that both conditions may occur after catheter manipulation:

- Spasm: smooth concentric narrowing that rapidly resolves with nitrates.
- Dissection: persistent narrowing, intimal flap, contrast staining, worsening flow, and no resolution with nitrates.

Historical perspective

Several case reports and series have documented severe LM spasm causing:

- Transient cardiogenic shock
- Ventricular fibrillation
- Need for temporary circulatory support

Fortunately, when recognized promptly and treated with catheter withdrawal and intracoronary vasodilators, the spasm usually resolves completely within seconds to a few minutes.

For an interventional cardiologist, any new severe LM stenosis appearing immediately after guide engagement should be assumed to be spasm or dissection until proven otherwise, because both can rapidly become catastrophic if not addressed.



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