INTRODUCTION
Percutaneous transluminal coronary angioplasty (PTCA) is the treatment of choice for high risk unstable angina \(^1\). Though PTCA is a safe procedure complications are not uncommon \(^2\). We describe a patient who developed complications that were recognised and managed appropriately.

CASE REPORT
A 63-year-old male presented with recent onset angina of 20 days duration. He is a known hypertensive for the past six years, on irregular medication. He is not a known diabetic or smoker. There was no family history of coronary artery disease or sudden cardiac death. On admission he was haemodynamically stable with normal precordial examination. Electrocardiogram revealed ‘T’ wave inversion in anterior leads but cardiac biomarkers were negative. After initiation of antiplatelet and antithrombotic therapy there were no further episodes of angina.

Coronary angiogram demonstrated 70% eccentric, calcified stenosis in the proximal left anterior descending (LAD) \(\text{Figure 1}\).
The left coronary system was cannulated with 6F, JL 3.5 guiding catheter and the lesion was crossed with 0.014” Hi torque floppy guidewire (Abbott Vascular, Santa Clara, California). The strategy was to deploy a short stent to the proximal LAD lesion and antero-posterior 10° cranial (AP cranial) angiographic projection was used for the entire procedure. A 2.75X13 mm Angstrom stent (Vasmed technologies Ltd, UAE) was deployed denovo after proper positioning. During deployment a waist was noted and stent had to be deployed at a high atmosphere (atm) of 18, to break the waist. Following stenting a small dissection was noted at the distal edge of the stent (Figure 2).

Figure 2: Dissection seen at the distal edge of the stent. Note the landing zone of stent to be in the diseased mid LAD segment

In view of calcium in the dissected segment, this was dilated with a 2.5X10mm Sprinter balloon (Medtronic, Inc., Minneapolis, Minnesota) at 10 atm. A 2.75X12 mm Vision stent (Abbott Vascular, Santa Clara, California) was delivered, to cover the dissection flap and overlap with the distal end of proximal stent, at 12 atm. Contrast injection after stenting showed another dissection at the distal edge of the second stent (Figure 3).

At this point, patient had sensation of fullness of urinary bladder and was told to wait for a couple of minutes. Another 2.75X23 mm Vision stent was delivered at 10 atm to cover the dissected segment. Angiogram done after stenting showed no-flow distal to the third stent. After 100 µg of intracoronary nitroglycerine and sodium nitroprusside there was TIMI 3 flow in the LAD. The entire stented segment of LAD was post-dilated with 3.0X10 mm non-compliant balloon at 12-16 atm. Up to this stage of the procedure the patient was haemodynamically stable, inspite of dissections and no-flow phenomenon. As the patient was about to be shifted out of the catheterisation lab, he complained of fatigue and had profuse sweating. The cardiac monitor showed a heart rate of 22 beats per minute followed by asystole. Immediate CPR followed by intravenous (IV) atropine and bolus IV fluid restored the heart rate to 96/minute and he became stable. Check coronary angiogram showed patent stent with TIMI 3 flow. The bladder distension had probably resulted in vasovagal reaction and transient asystole. Bladder catheterisation done on table resulted in drainage of about 2200 ml of urine. He received IV infusion of eptifibatide for 24 hours. The further in hospital course was uneventful and patient is asymptomatic at 6 months follow-up.

DISCUSSION

PTCA with stenting is an effective treatment option for patients with acute coronary syndrome (1). Though it is a safe procedure in experienced hands, life threatening complications like dissections are not uncommon. The reported incidence of periprocedural dissection is 2% (2). In the drug eluting stent (DES) era, this has been reported to be about 2.8% (3). Dissections are likely to occur following balloon dilation or at the edges of stent. The predictors of periprocedural dissection are enumerated in Table 1 (4-6).

Table 1: Predictors of periprocedural coronary artery dissection

- Calcified lesion
- Eccentric lesion
- Long lesion
- Tortuosity
- Type B, C ACC/AHA lesion
- Balloon to artery ratio >1.2
- Guiding catheter E.g. Amplatzer

**Figure 3: Image demonstrating dissection at the distal end of second stent**
In our patient the LAD lesion showed fluoroscopic evidence of calcification and was eccentric. Predilation and preparation of the coronary bed is necessary for successful outcome in this situation. Being a short segment with only 70% stenosis, we opted for direct stenting. On careful analysis, it can be seen that mid LAD has diffuse, tubular narrowing of 30% distal to the proximal lesion. Deploying the stent at high pressure (to break the waist), coupled with the distal landing zone of stent being in diseased segment further increased the propensity to dissection. The stenting associated dissection could have been avoided in this case by predilating the lesion adequately and by choosing a longer stent which lands in normal healthy segment.

As untreated edge dissections predisposes to stent thrombosis, which is associated with adverse outcome, deploying an overlapping stent to cover the dissected segment is the preferred treatment strategy (7). The stent should be of sufficient length to cover the dissected segment and allow for over lap of few millimeters with the initial stent. Though repeated contrast injection predisposes to its propagation, at least two perpendicular views should be taken to determine the extent of dissection (8). In our case the entire procedure was done in the AP cranial view. This could have probably led to underestimation of extent of dissection following stenting. Only after assessing the true extent of dissection from two orthogonal planes, a stent of appropriate size should be chosen. Intravascular ultrasonography would have provided precise information regarding the extent of dissection, stent size required as well as adequacy of stent deployment.

The no-flow phenomenon associated with PTCA has been reviewed by Hiroshi Ito (9). The causes of no-flow are spasm, dissection, thromboembolism and microvascular dysfunction. In our patient, the probable cause of the observed no-flow phenomenon was vasospasm, as there was prompt response to intracoronary nitroglycerine and sodium nitroprusside.

Cardiac arrest during PTCA can be devastating and requires prompt initiation of resuscitative measures. The periprocedural causes of cardiac arrest are listed in Table 2.

### Table 2: Periprocedural causes of cardiac arrest

- Abrupt vessel closure
- Stent thrombosis
- Vasospasm
- Coronary dissection
- Coronary perforation
- Air embolism
- Ventricular arrhythmias
- Vasovagal reaction
- Anaphylaxis

As the check angiogram following resuscitation showed TIMI 3 flow, the most probable cause of asystole was a vasovagal response to urinary bladder retention (10). Contrast used during angioplasty coupled with maintenance IV fluids and prolonged procedure leads to urinary bladder distension. Simple steps like ensuring the patient voids urine prior to entering the cathlab suite and prompt bladder drainage when patient feels bladder fullness will prevent bladder distension induced vasovagal reaction and its consequenc- es. Every effort should be taken to avoid vasovagal reaction in the periprocedural period as the associated hypotension predisposes to stent thrombosis. Preventing complications, prompt recognition when it occurs and initiating corrective measures rapidly are key to successful angioplasty and stenting.

### CONCLUSION

Complications are not uncommon during coronary angioplasty and stenting. Attention to basic steps of the procedure is mandatory. Calcified lesions should be adequately predilated before stenting. It is preferable to position the ends of the stent in healthy segment. If the stent landing zone is diseased, it is better to deploy at nominal pressures. If periprocedural dissec- tion occurs, the extent of dissection should be confirmed in atleast two perpendiculang angiographic views. Bladder retention leading to vasovagal response can be an unrecognised non-coronary cause of cardiac arrest during angioplasty.

### REFERENCES