Treating Acute Coronary Occlusion During PCI

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Acute occlusions are generally defined as sudden occlusions that occur either during or directly after percutaneous intervention (PCI) at the target lesion or at a site adjacent to the target lesion. Acute occlusions are a serious ischemic complication for PCI, and cause a large percentage of the deaths and myocardial infarctions that occur in PCI. The frequency of occurrence varies among reports, but can be said to be approximately 2.8%. At least half of those are said to occur in the catheter laboratory. Furthermore, of those occlusions that occur after the patient leaves the catheter laboratory, more than half occur within 6 hours of the PCI procedure.

Mechanism of Acute Occlusion

Our understanding of the following causes of acute occlusions has deepened due to observations from investigations of animal models, tissues examined at autopsy, and intravascular ultrasound and angioscopy.

a. Dissection of the intima or media, or formation of intramural hematoma, caused by balloon expansion.
b. Formation of thrombus due to platelet activation, caused by injury to the vessel wall.
c. Coronary artery spasm caused by the escape of vessel contractility agents and reduced NO production, due to endothelial injury.

The above three factors are thought to be the main causes of acute occlusion. Of these three, dissection of the coronary artery and hematoma cause the largest percentage of acute occlusions, followed by thrombus formation. Presently, thanks to the routine use of nitrites and calcium blockers during PCI, it can be suggested that occlusion due to spasm of the coronary artery are becoming the exception. There is also the possibility of acute occlusion occurring due to a spasm brought on by the first two factors. Furthermore, in the case of a coronary dissection or hematoma, thrombus may easily be caused by stagnation of blood flow and lead to coronary occlusion. When dealing with acute occlusion, it is important to be able to bring these three factors to mind.

Predictors of Acute Occlusion

Reported predictors of acute occlusion are listed in Table 1.

Table 1

Clinical predictors
Unstable angina
Diabetes mellitus
Angiographic predictors
Presence of coronary dissection
Intraluminal thrombus
Lesion length > 2 luminal diameters or > 10mm
Proximal tortuosity
Angle of bend > 45 degrees
Degenerated saphenous vein graft
Pre-angioplastic stenosis 90 – 99%
Post-procedural stenosis > 35%

Prevention of Acute Occlusion

1. Prevention by Drug Therapy

· Anti-platelet therapy
It is reported that administration of aspirin or ticlopidine during or directly after the procedure significantly lowers the incidence of acute occlusion.

· Heparin
Administering pre-procedural heparin to patients with unstable angina significantly inhibits the incidence of acute occlusion during PCI. If heparin is used during the operation to make the ACT (activated clotting time) 300 seconds or greater, the percentage of ischemic complications during and immediately after the operation is significantly reduced.
Opinions vary on whether continuing to administer heparin after PCI affects the incidence of acute occlusion.

· Thrombolytic agents
Administering a thrombolytic agent before PCI does not reduce acute occlusion. In fact, it has been reported that doing so may actually increase the risk of coronary dissections.

a. Anti-platelets, GP IIb/IIIa inhibitors
b. Nitrites, calcium blockers, and other vasodilators.

2. Prevention by PCI and Caution Points

It has been reported that acute occlusion can be prevented by low-pressure or gradual balloon inflation, or by using a long balloon for high-risk cases. It has also been reported that the risk of coronary dissection is significantly increased when the balloon:artery ratio is 1.3 to 1 or higher.

Treating Acute Occlusion

1. Stabilizing the hemodynamics

When acute occlusion occurs, assessing the condition accurately, without panicking, and determining the best treatment is essential. If the hemodynamics can be stabilized or maintained, more time is then available to assess the nature of the condition, which leads to the quickest or optimal treatment. There are several steps you can take to achieve stable hemodynamics, for example:

If bradycardia is evident:

- Inject intravenous atropine
- Insert a temporary pacemaker

If a drop in blood pressure is observed:

- Inject a intravenously vaso-pressor (noradrenaline, etc.)
- Try intra-aortic blood pumping (IABP)
- Heart massage, endotracheal intubation, etc.

These measures should be taken quickly in these cases.

2. Understanding the Causes of Acute Occlusion

As mentioned earlier, the causes of acute occlusion are 1. Coronary dissection or hematoma, 2. Thrombus, or 3. Coronary spasm. More than one of these factors are often observed when acute occlusion occurs, but in any case it is extremely important to understand the causes correctly and apply the proper treatment. While performing the steps under "Attempting to achieve stable hemodynamics," obtain the information needed to understand the causes as shown below.

When hemodynamics are stable, and there is time to spare:

a. First, inject nitrites to prevent coronary spasm.
b. Next, measure ACT.
c. Re-insert the wire and balloon to check whether the occlusion has re-opened.
d. Finally, use of IVUS is will give you even more information

When hemodynamics are unstable and there is no time to spare:

a. Re-inflate the balloon.
b. Next, try using a perfusion balloon.
c. If necessary, implant a stent.

3. Treatment after the Cause of the Acute Occlusion is Understood

a. Drug therapy
b. Treatment by re-peat PCI
c. Emergency bypass surgery

a. Drug therapy

The use of medicines for treatment in cases of coronary spasm or thrombus should be considered, but their effectiveness is limited.

- Nitrites

These are used to treat coronary spasm, but their effectiveness is extremely limited according to some reports. However, in cases where the main cause of the problem seems to be coronary spasm, injecting nitrites into the coronary artery should be tried, if the blood pressure allows.

- Thrombolytic agents

Consider using these medicines in cases where thrombus is thought to be present, buy, the recanalization rate is not high with these drugs, and in fact can lead to hemorrhagic complications, so great caution should be exercised when using these agents.

- Verapamil, Nicorandil, etc.

It has been reported that these are effective for arteriole embolism in cases where no reflow or other similar phenomena are observed.

b. Treatment by re-peat PCI

- Re-peat PCI with a balloon

  Press the dissection flap or thrombus to the vessel wall by using a balloon one size (0.5mm) larger and using a longer inflation time to preserve the lumen. Using an autoperfusion balloon allows for a longer inflation time, and a higher recanalization percentage can be obtained. In order to prevent thrombus

formation in the balloon lumen or inflation site at this time, it is necessary to use heparin to extend ACT sufficiently.

- Stenting
  Stent implantation gives the highest recanalization success rate. By implanting a stent, the dissection flap or thrombus is pushed against the vessel wall, and the effects of elastic recoil or spasm are reduced. There are cases of occlusion due to sub-acute stent thrombosis, but the frequency of this problem has been reduced by the availability and use of ticlopidine.
- Directional coronary atherectomy (DCA)
  When a large dissection flap is thought to be the cause of an acute occlusion, and if the location of the dissection flap can be seen with intravascular ultrasound, it may be possible to use DCA to excise the dissection flap.
- Intramural hematoma decompression using a guide wire
  If the acute occlusion is caused by an intramural hematoma, dilating the occluded site or implanting a stent may cause the intramural hematoma to advance distally. If there is a suitable side branch at the distal site, it may be possible to perform a decompression of the hematoma by inserting a stiff guide wire into the hematoma, directing it toward the side branch, and then re-entering the lumen.

c. Emergency Bypass Surgery

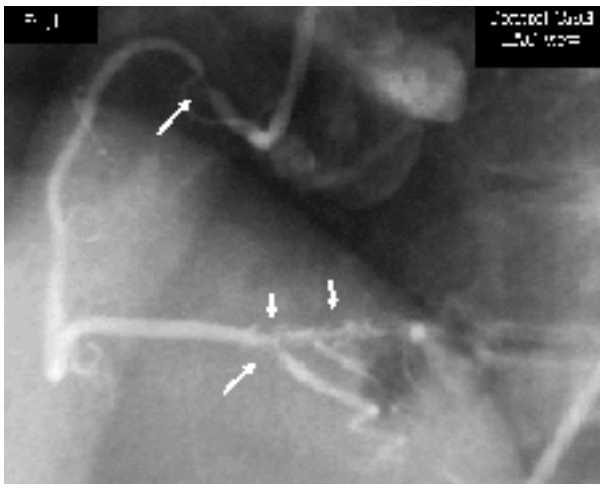
If the occlusion is resistant to both drug therapy and re-peat PCI, and/or a sudden change of in hemodynamics is suspected, emergency bypass should be considered. Of course, until bypass surgery standby is ready, every measure should be taken to try to maintain hemodynamics.

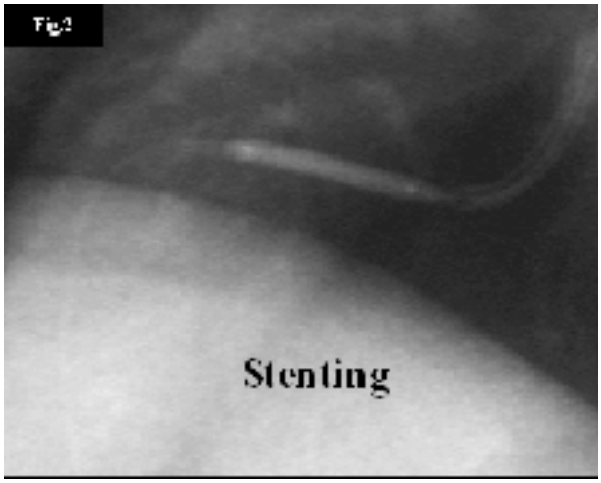
Follow-up Treatment After Recanalization of an Acute Occlusion

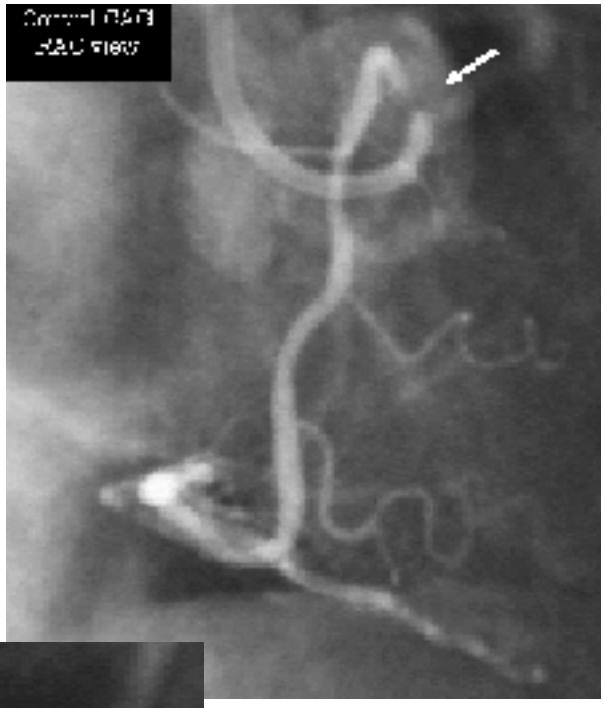
After successful recanalization of an acute occlusion, if the lesion site is unstable, the peripheral run-off is insufficient, etc., it is often helpful to insert an IABP. An IABP increases the coronary artery reperfusion pressure during inflation, and at the same time reduces stress on the heart.

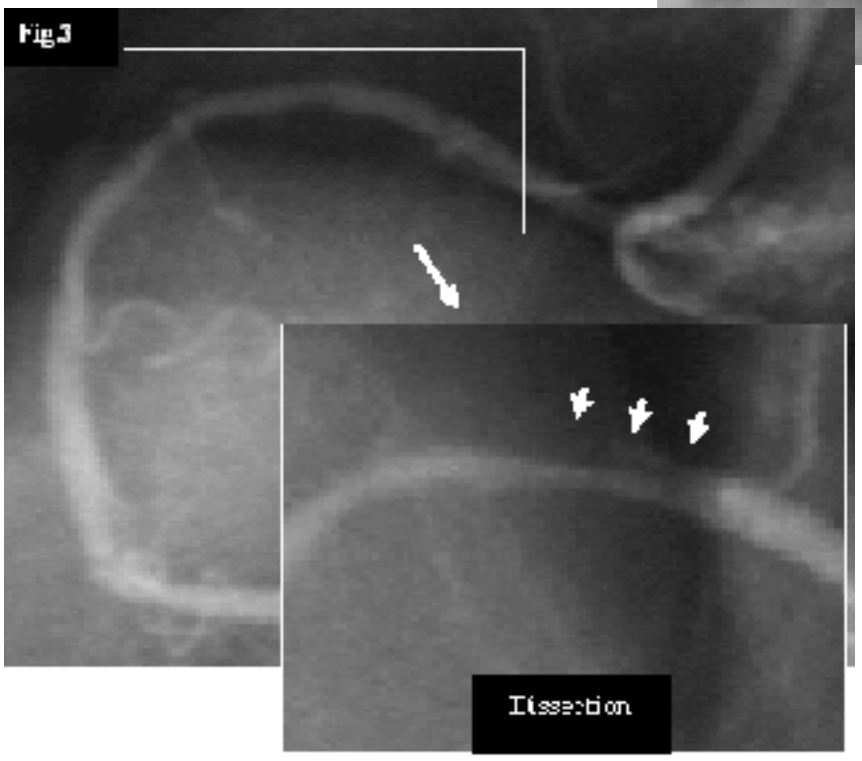
Case Presentation

This case involved an acute occlusion caused by a severe dissection during PCI. The patient was a 72 year-old woman, with a clinically-diagnosed old myocardial infarction. PCI was performed in the right coronary artery, to correct a *de novo* lesion in segment #1, and in-stent restenosis in #3. Image 1 shows the CAG prior to PCI. We performed direct stenting in #1, using a Multilink stent (3.0x 15mm) (Image 2). On the CAG taken after stent-implantation, a small dissection was seen at the distal edge of the stent (Image 3).
We then used a balloon to treat the in-stent restenosis

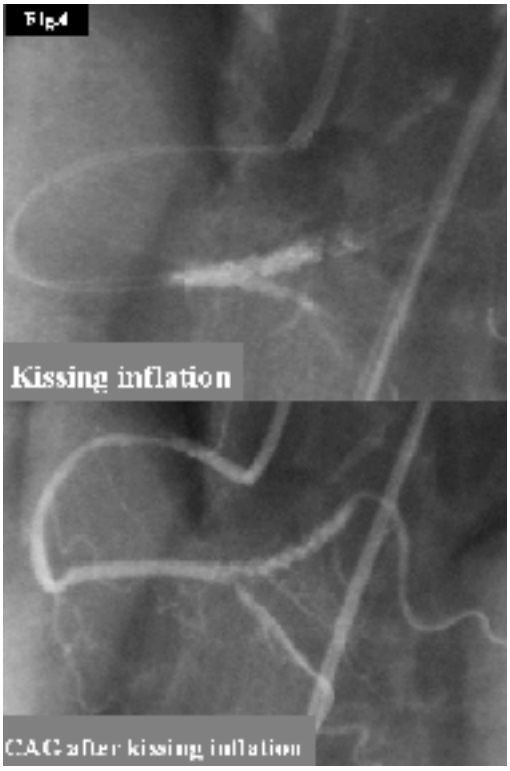


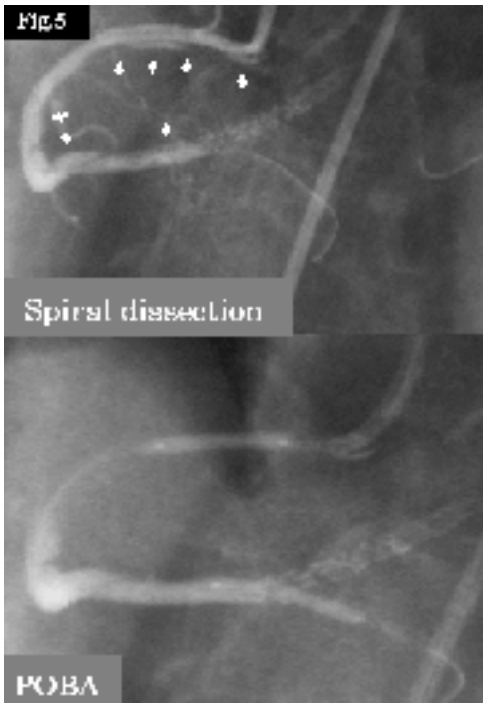


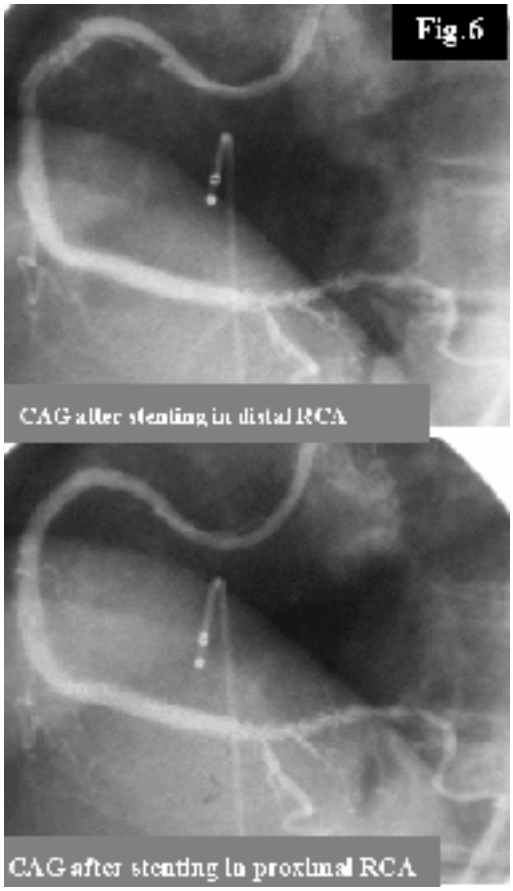




in #3. On the CAG taken directly after balloon inflation, no worsening of the dissection was detected. However, on the next CAG, an arterial dissection from # 1 to #3 was observed. Blood flow was interrupted immediately in front of the stent in # 3, due to a hematoma caused by the arterial dissection (Image 4). ST segment elevation of the inferior wall was seen on the ECG, but there was no evidence of hypotension or arrhythmia, such as bradycardia, atrioventricluar block, etc. We inflated a balloon at both the proximal and distal areas of the dissected lumen, but no improvement was seen (Image 5).

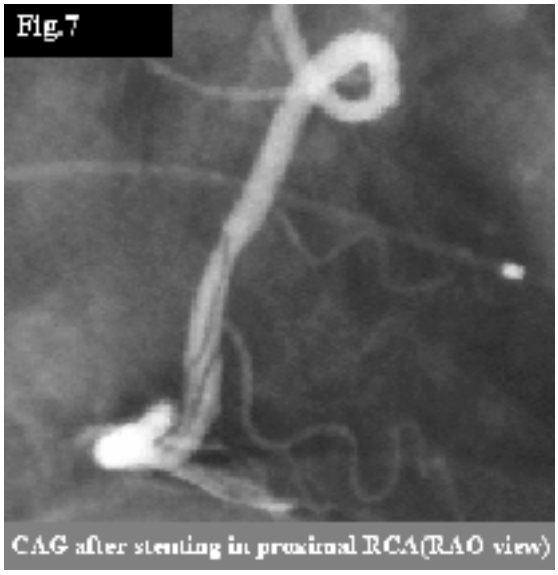


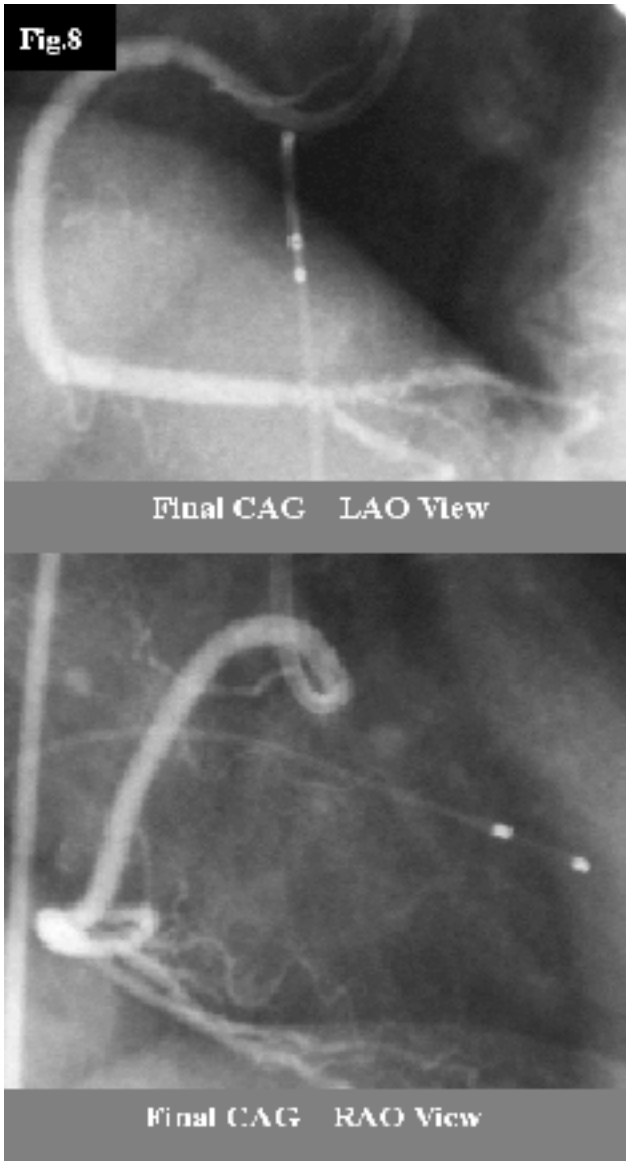




To prepare for arrhythmia during re-opening of the occlusion, we inserted a temporary pacemaker, after which we implanted a stent (S-670, 4.0 x 30mm) at the distal site of the dissection to compress the hematoma. We were able to re-open the occlusion. We also implanted a stent at the proximal site (S-670, 4.0 x 30mm) (Image 6).

The post-stenting CAG and intravascular echocardiogram are shown in Image 7.





From this view, we decided that there was residual hematoma in the unstented segment #2-3 that was causing a slight stenosis, so we implanted an additional stent (S-670, 4.0 x 30mm). The final angiogram is shown in Image 8.