ACTA MEDICA SALINIANA

CASE REPORT

MANAGEMENT OF CORONARY ARTERY PERFORATION IN ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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ABSTRACT

Background: Coronary Artery Perforation is a rare Percutaneous Coronary Intervention (PCI) complication that may lead to pericardial effusion frequently accompanied by tamponade. This complication occurs in 0.2 to 0.6% of all patients undergoing the procedures. The initial approach commonly comprises prolonged balloon inflation, placement of a covered stent graft (CSG), pericardiocentesis, or urgent coronary artery bypass graft (CABG) surgery.

The aim: To present a patient with a type III Ellis classification of coronary artery perforation, who had previously undergone primary PCI due to an acute extensive anterior STEMI, and discussed its management

Conclusion: Management of coronary artery perforation can be customized based on the categorization and hemodynamic condition of the hole. Our patient was effectively managed with extended balloon inflations and intra-coronary vitamin K administration without the need for surgical management intervention.

Keywords: Coronary Artery Perforation, Percutaneous Coronary Intervention, Prolonged Balloon Inflation, Stent

CASE ILLUSTRATION

A 49-year-old male was admitted to Emergency Department (ED) due to progressive onset of chest pain 2 hours before admission. Chest pain radiated to jaws and was followed by sweating and dyspnea. Previously, in 2014, he experienced the identical symptoms and was hospitalized due to Non-ST Segment Elevation Myocardial Infarction. Coronary angiography was performed at that time and showed severe LCx, Moderate LAD and Mild RCA disease. He then underwent elective PCI to distal LCx with Sirolimus 2.5 x 13 mm eluting stent implantation and Plain Old Balloon Angioplasty (POBA) to mid LAD and Diagonal Branch. A year later, Treadmill Test displayed positive ischemia response and another elective PCI was performed to distal LCx with Bare Metal Stent (BMS). In the next 4 years, he never did a routine control and poorly managed of medication.

Now, at the ED, physical examination revealed rhonchi in basal lungs. An electrocardiogram (ECG) was taken and showed ST elevation from V2-V4, I and aVL (figure 1). His chest X-Ray showed cardiomegaly with mild congestion at both lungs. Laboratory was markedly increased of Troponin T.

He underwent Primary PCI with initial assessment of Acute Extensive Anterior STEMI. The coronary angiography identified a huge thrombus burden at proximal LAD and mid LAD was the culprit lesion. There was a huge size difference between the proximal LAD and mid LAD segment. The operator decided to implant Sirolimus 2.75 x 32 mm eluting stent which was measured based on the proximal target (figure 2). Direct stenting was deployed to mid LAD and showed the presence of massive mid LAD perforation, graded as type III of Ellis classification (figure 3, Panel C). Therefore, the operator proceeded to use the same stent balloon to control of the perforated segment of the LAD.

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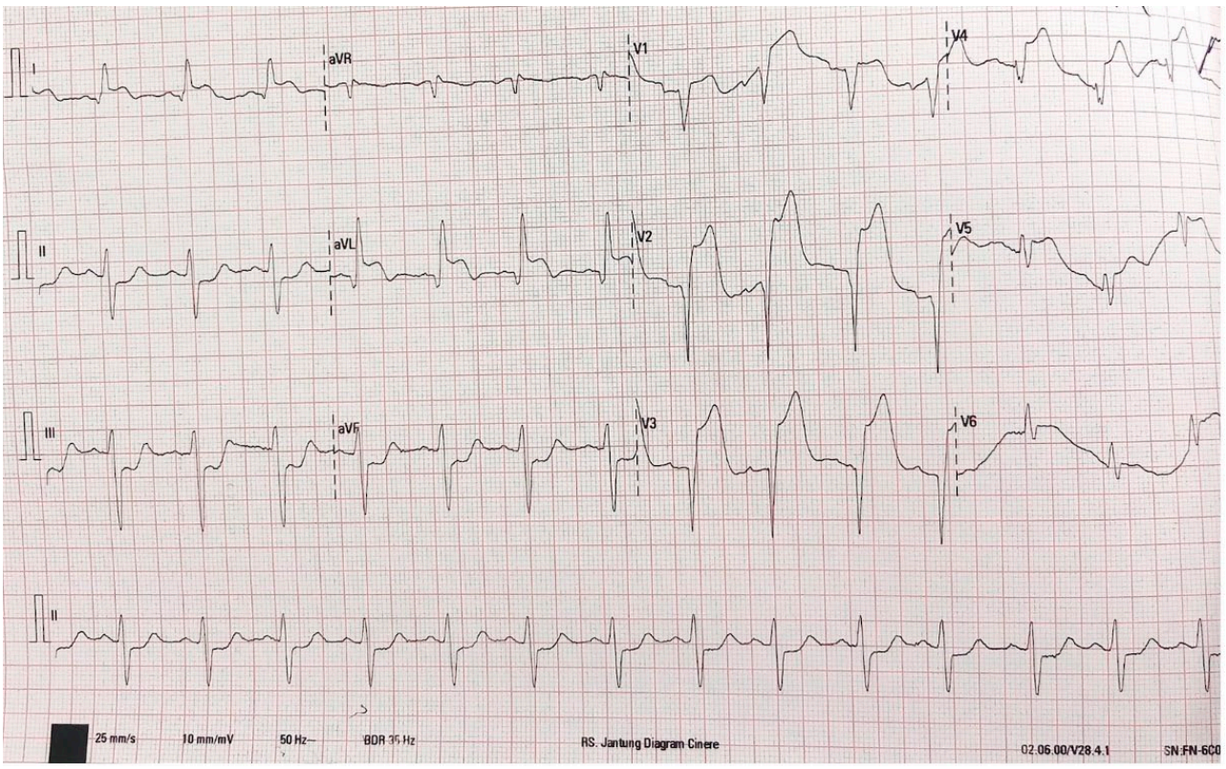

Figure 1. Electrocardiography (ECG) showed ST Elevation at the anterior and high lateral leads

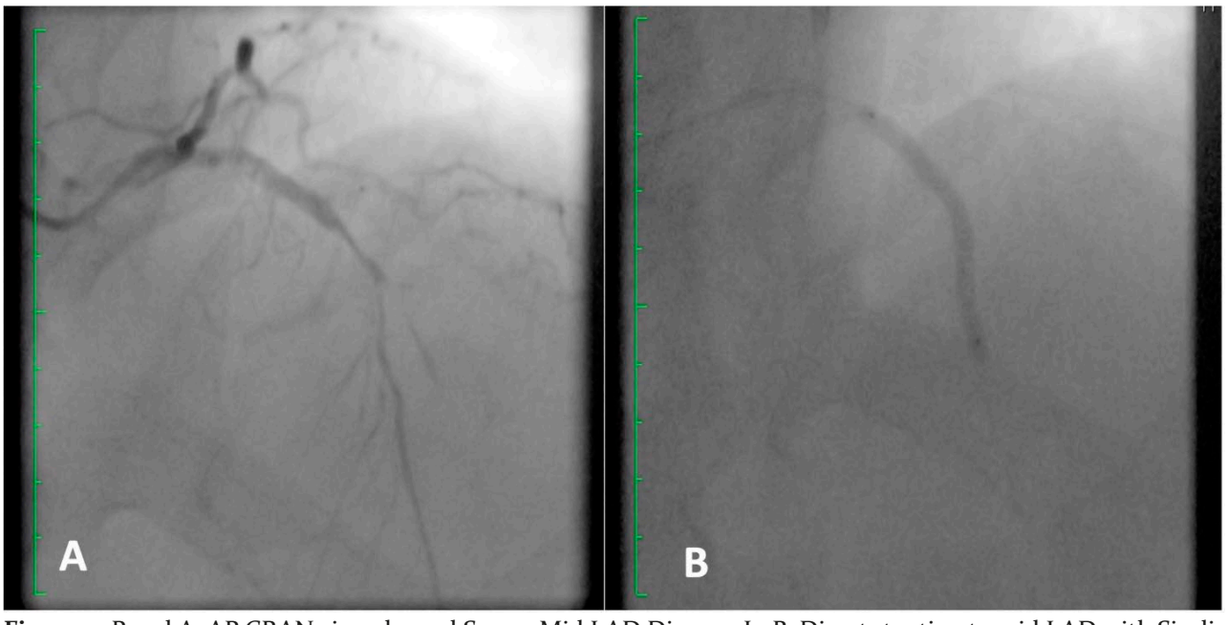

Figure 2. Panel A. AP CRAN view showed Severe Mid LAD Disease. In B. Direct stenting to mid LAD with Sirolimus 2.75 x 32 mm eluting stent.

For this reason, prolonged stent inflation (20 minutes) was used to seal the perforation and bed-side echocardiography displayed minimal pericardial effusion without any sign of cardiac tamponade (Figure 3, Panel D). However, the perforation was not sealed completely (Figure 4, Panel E). Thus, another balloon inflation using Sapphire 3.0 x 15 mm was performed twice for 35 minutes and 5 minutes each and Vitamin K 20 mg intra-coronary was administrated between those two inflations (Figure 4, Panel F). The perforation was sealed with TIMI 3 flow achieved to distal LAD (Figure 4, Panel G).

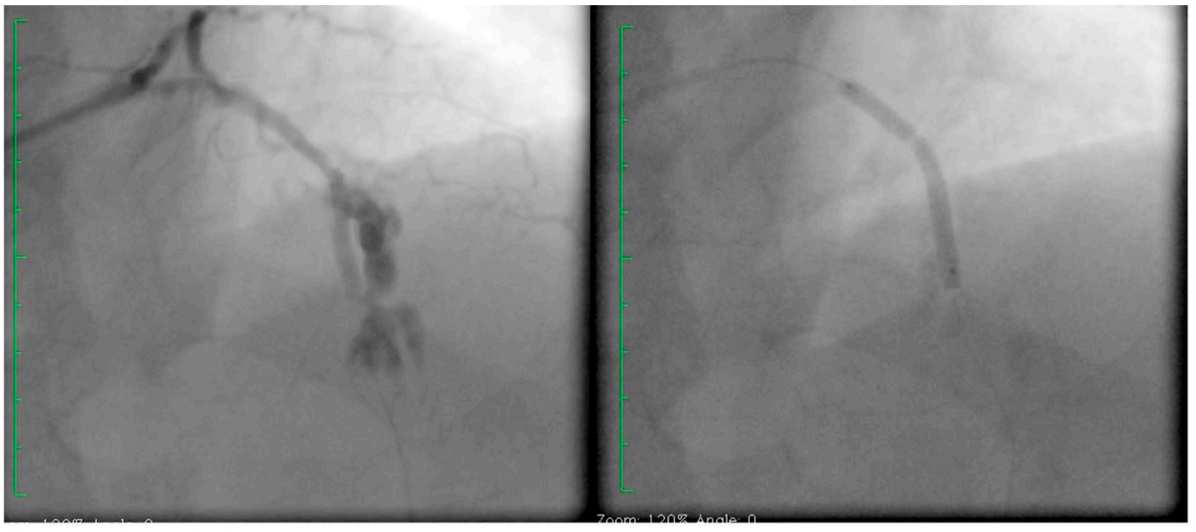

Figure 3. Panel C. Coronary artery perforation developed after stent dilatation up to 20 atm for 3 seconds. Based on Ellis classification, type III perforation was noted. Panel D. We sealed the perforation using the same stent balloon inflation up to 10 atm for 20 minutes. Immediate echocardiography was performed to identify pericardial effusion.

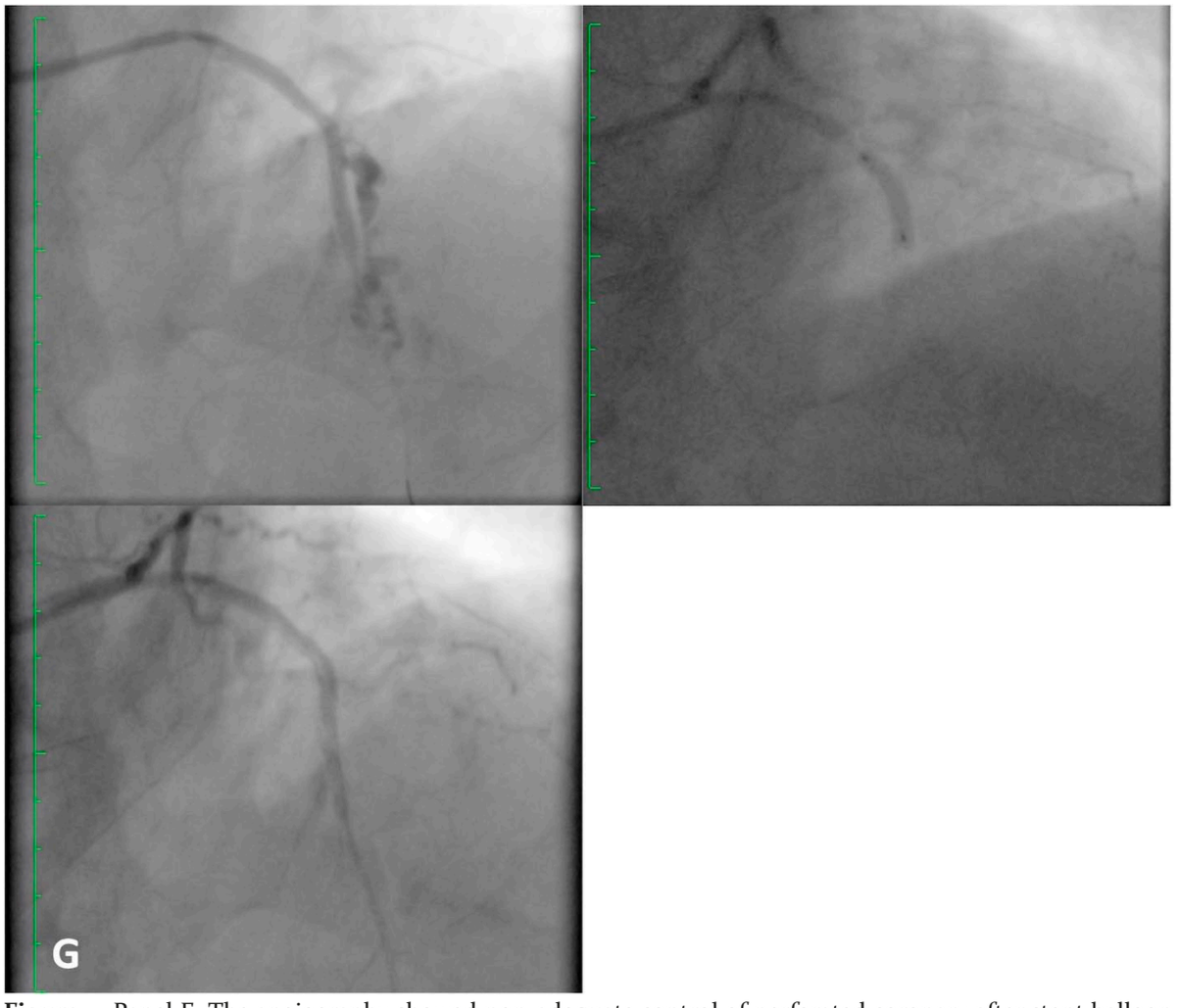

Figure 4. Panel E. The angiography showed non-adequate control of perforated coronary after stent balloon inflation. Panel F. Another prolonged balloon inflation to mid LAD using Sapphire 3.0 x 15mm (inflated up to 6 atm for 35 minutes and 5 minutes each). Panel G. The perforation was successfully sealed with TIMI 3 flow to distal LAD.

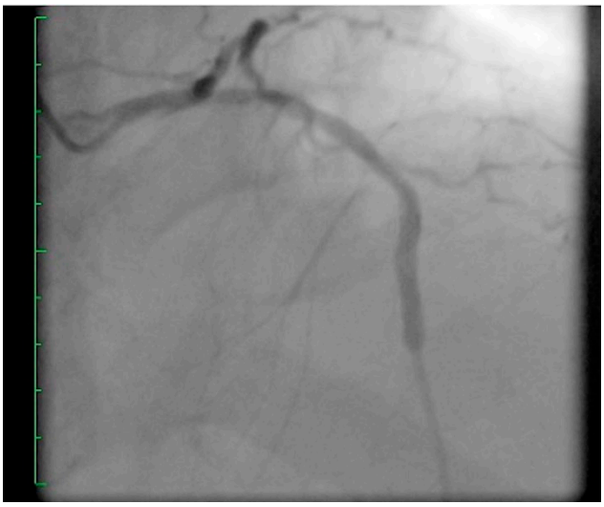

Figure 5. Staged Coronary Angiography acquisition performed after Recent Primary PCI (Taken 2 Days After PPCI), presented a big size gap at Mid LAD and Distal LAD Segment, but Coronary Flow Remains Excellent.

During monitoring in ICU, he complained of recurrent chest pain and the ECG showed ST elevation in lead I and aVL (Figure 6). Reinfarction was noted and decided to perform coronary angiography once more. A subsequent angiogram has revealed severe proximal LAD with mild residual thrombus burden, patent LAD and LCx stents and an adequate control of LAD perforation. In view of TIMI 3 flow and thrombus to proximal LAD, it was decided to defer the procedure and staged PCI to LM-LAD after 6 months. He was discharged after 7 days with markedly decreased of Left Ventricular Ejection Fraction but remained hemodynamically stable.

The patient returned 30 days after the PCI procedure for outpatient consultation. He remained asymptomatic and showed no adverse cardiac events at the six-month follow-up.

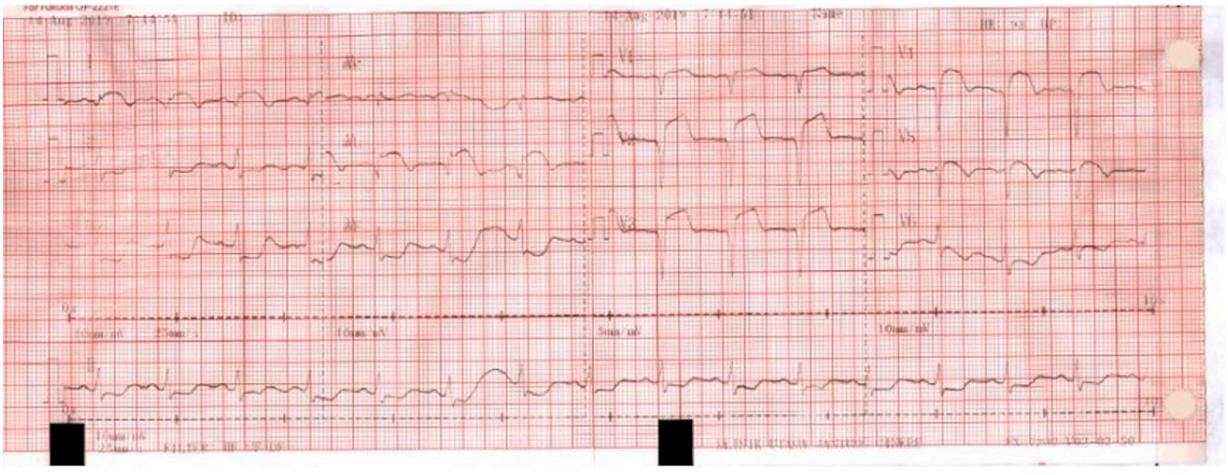

Figure 6. ECG at The Onset of Acute Reinfarction After PCI

DISCUSSION

During PCI, coronary perforation, which mainly happened in a large vessel, may occur as a consequence of guidewire advancement, balloon/stent advancement, balloon/stent inflation, over-sizing, or ruptured balloon/stent. It is reported that perforation after stenting is mainly caused by excessive over-dilatation or oversized stent implantation.[1]·[2] The incidence of coronary perforation, whilst low, its rate may be higher when using atherectomy devices or in complex lesion procedures.[3]·[4] Once the Coronary Artery Perforation (CAP) develops and the blood fills the pericardial space, it may lead to cardiac tamponade, resulting in cardiogenic shock and a high mortality rate. [4]

The severity of coronary perforation is arranged using the Ellis classification.[5] This wide scheme is based on the angiographic appearance of the CAP: type I is defined by extraluminal crater without extravasation, type II is defined by pericardial or myocardial blush without contrast jet extravasation, and type III is defined by contrast jet extravasation through frank (> 1 mm) perforation and type III cavity spilling (CS) indicates contrast jet extravasation into an anatomic cavity (e.g. cardiac chamber or coronary sinus).[3]·[5] Lemmert et al reported the cases based on the Ellis type I in 2.9%, Ellis type II in 40.4%, Ellis type III in 54.8%, and Ellis type III cavity spilling in 1.9%.[6] This case appears to be a case of type III with cavity spilling.

Trana et al reported from several studies from 1990 – to 2011 that the incidence of type III CAP ranges from 0.06 to 0.49%.[7] Reports showed a five-fold rise in in-hospital mortality as a result of cardiac tamponade and hemodynamic compromise. The 30-day mortality after perforations varied from 6.6% to 15.5% with a significant upward trend and 17.8% in 1 year. [1]·[6]·[8]·[9]

The development of CAP has several predictors. Patients with CAP were more often women and older, with a greater burden of comorbidity including hypertension, hypercholesterolemia, and previous myocardial infarction (MI) respectively, and underwent more complex PCI procedures. [9] Although, another recent study showed that cases of CAP highly occurred in men and most patients had presented with stable angina.

Treated lesion type was B2/C in 94.6%, and 31.3% were chronic total occlusions. [6] Likewise, Al Ramee et al added the bulk of perforations were placed in LAD (44.6%) with the mid part was likely the lesion location (46.4%).[10] The clinical predictors for CAP which present in this case are male, history of non-ST elevation MI, with hypertension as comorbid, and underwent Primary PCI of mid LAD.

The risk of CAP begins once the guidewire is inserted and remains throughout the PCI procedure. Meticulous attention to guidewire position, appropriate sizing of the balloon or stent prior to inflation, and avoiding overdilation or high-pressure inflation exceeding the balloon's burst pressure. Extra care must be taken in high-risk lesion types, e.g. calcified, angulated, bifurcation lesions, or chronic total occlusion, as well as during the usage of the debulking device. [3]·[11]

The management of CAP depends on its severity (e.g. size of the perforation, the extended contrast medium extravasation) and the hemodynamic status of the patient. The approaches are ranging from watchful waiting to prompt balloon inflation until the vessel is closed properly and hemodynamic stability is achieved.[12]·[13] Consequently, the management of CAP remains challenging, involving continuous assessment of the hemodynamic status while carrying out an attempt to seal the perforation (Figure 7).

Once coronary perforation is confirmed, the same balloon responsible for the perforation should immediately be positioned at the perforation site to achieve hemostasis in a short time. The balloon should be carried out at the lowest pressure to assist hemostasis as verified by contrast injection at regular intervals: usually, inflations at 2 to 4 atm for approximately 5 to 10 min are sufficient. In a view of incomplete sealing, the balloon should be placed in the correct position and inflated at higher pressure. If the perforation involves the left main artery, a perfusion balloon or a covered stent should be considered the most effective technique to seal the perforation while maintaining vessel patency.[12]·[13]

Once the vessel is occluded by the balloon, the patient's hemodynamics may normalize; however, it is recommended to use intravenous fluids, vasopressors, and periodically mechanical circulatory support may be required once the hemodynamic compromise occurs. The presence of coronary perforation should also encourage immediate echocardiography, and when a large pericardial effusion is associated with tamponade, emergent pericardiocentesis and surgery are indicated. In addition, the use of protamine as a reversal agent of heparin is suggested.[13]

Our case report appears to be the case of type III coronary artery perforation. We dropped a surgical option

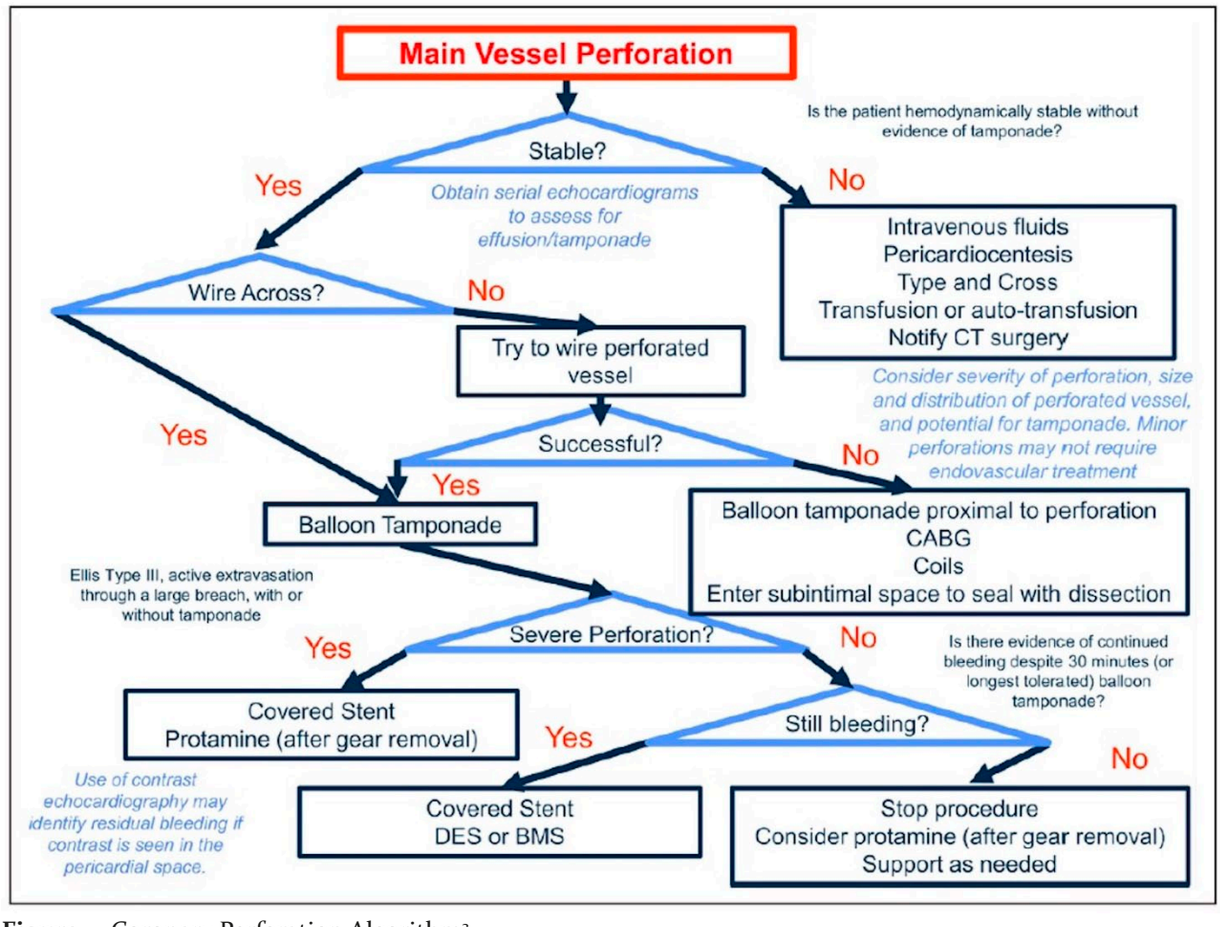

Figure 7. Coronary Perforation Algorithm[3]

because of the limitation for performing CABG in our institution and in view of immediate echocardiography which revealed minimal pericardial effusion (4-6 mm diameter) without any sign of cardiac tamponade. On the other hand, the use of covered stents represented an alternative and more prompt treatment but was not available at that time. The operator then tried to proceed with prolonged balloon inflations for 60 minutes of total duration.

In agreement with the previous report, Meguro et al stated the use of prolonged balloon inflation (average of 48 minutes) was necessary for the management of CAP. [4] We showed much longer prolonged balloon inflations were effective and indispensable to achieving the hemostasis. In addition to administering Vitamin K intra-coronary within the inflation, the management of coronary perforation brought a good result.

CONCLUSION

Coronary artery perforation related to PCI was an uncommon complication but a potentially fatal outcome. Management of coronary artery perforation can be tailored according to the classification of the coronary artery perforation and the hemodynamic status. Most patients with coronary artery perforation can be treated successfully with non-surgical management. Although, surgical intervention should be provided quickly if coronary artery perforation does not respond to the non-surgical therapies. Our case was successfully treated with the use of prolonged balloon inflations and administered vitamin K intra-coronary without having to undergo surgical repair. Furthermore, patients should be monitored carefully as a late cardiac tamponade and post-procedural infarction might occur even in less advanced types of coronary artery perforation.

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