Management of complications of RV branch ellis type V artery perforation with handmade covered stent

Starry Homenta Rampengan*1, Jeffry1

INTRODUCTION
Coronary artery perforation is a rare and frightening complication of percutaneous coronary intervention. Several studies have reported variable number of incidence of coronary artery perforation from 0.29% to 3%. The risk will increase with the complexity of coronary lesions in patients, especially in patients with chronic total occlusion, type B2 lesions, type C lesions, lesions that are eccentric and more than 10 mm in length, small artery size, and also various other causes calcification of the coronary artery.1

At first the incidence of coronary artery perforation was mostly caused by the use of assistive devices such as rotablator and directional atherotomy, but over time the incidence of coronary perforation was mostly caused by inappropriate stent and balloon sizes, especially those that were too large, causing overstretching, as well as a hard and rigid guidewire commonly used in patients with chronic total occlusion or patients with severe coronary calcifications.

Based on the Ellis classification system, there are 5 types of coronary artery perforation, where the highest incidence is Ellis type II with a percentage of 37-61% of all cases and the rarest is Ellis type V, which is a perforation of the distal coronary artery generally caused by a rigid guide and hydrophilic. Coronary artery perforation itself can be detected as a hard and rigid guidewire commonly used in patients with chronic total occlusion or patients with severe coronary calcifications.

We reported a rare case of RV branch artery perforation complication, specifically Ellis type V perforation during percutaneous coronary intervention that was then treated with a handmade covered stent. This perforation occurred due to a total occlusion at the proximal RCA. An anomaly of the RV branch caused the guidewire to perforate the distal part of RV branch artery. As there was no improvement achieved with balloon inflation, a handmade covered stent was then made to close the RV branch, which was placed on the RCA covering the ostium of the RV branch.

Case Presentation: We presented a case of type V Ellis coronary artery perforation during percutaneous intervention. Patient came with persistent chest pain starting from the proximal radiating to her left side and also penetrated to her back. An echocardiography examination showed mild dilatation of the right atrium and ventricle, with the decrease of global left ventricular systolic function with an ejection fraction of 45%. There was also left ventricular diastolic dysfunction with impaired relaxation with increased LVEDP. A calcified aortic valve appeared with good function. A mild regurgitation of the mitral valve and mild-moderate tricuspid regurgitation was found with a low probability of pulmonary hypertension. Patient was diagnosed with temporary working diagnosis of Recent Inferoposterior STEMI RV TIMI 4/14 Killip I, AVB grade 1, PAC bigemini, controlled hypertension, and reactive leukocytosis. Patient underwent percutaneous coronary intervention in RCA on the 6th day of admission, in which an anomaly of the RV branch was found and perforation in the coronary artery occurred, thus a handmade cover stent was made to overcome the incidence. The procedure was finished with a total of 130 cc of contrast used and 100 cc of total bleeding. Patient was then admitted to the ICCU with stable vital signs and was evaluated for signs of tamponade using a Vscan every 3 hours. On the 8th day of hospital admission, patient had no complaints with stable vital signs. The final diagnosis at the time of outpatient was CAD 2VD post PTCA with 2 stents on RCA, RV Ellis type V branch perforation, STEMI Inferoposterior RV TIMI 4/14 Killip I, post AVB grade 1, post PAC bigemini, hypertension and reactive leukocytosis. Outpatient’s therapy included anticoagulants, statins, beta blockers, ACE-I, PPI, and sulfaftate.

Conclusion: We reported a rare case of RV branch artery perforation complication, specifically Ellis type V perforation during percutaneous coronary intervention that was then treated with a handmade covered stent. This perforation occurred due to a total occlusion at the proximal RCA. An anomaly of the RV branch caused the guidewire to perforate the distal part of RV branch artery. As there was no improvement achieved with balloon inflation, a handmade covered stent was then made to close the RV branch, which was placed on the RCA covering the ostium of the RV branch.

ABSTRACT

Introduction: Coronary artery perforation is a rare and frightening complication of percutaneous coronary intervention. One of the rarest is Ellis type V coronary perforation that involves a perforation of the distal coronary artery generally caused by a rigid guide and hydrophilic.

Case Presentation: We presented a case of type V Ellis coronary artery perforation during percutaneous intervention. Patient came with persistent chest pain starting from the proximal radiating to her left side and also penetrated to her back. An echocardiography examination showed mild dilatation of the right atrium and ventricle, with the decrease of global left ventricular systolic function with an ejection fraction of 45%. There was also left ventricular diastolic dysfunction with impaired relaxation with increased LVEDP. A calcified aortic valve appeared with good function. A mild regurgitation of the mitral valve and mild-moderate tricuspid regurgitation was found with a low probability of pulmonary hypertension. Patient was diagnosed with temporary working diagnosis of Recent Inferoposterior STEMI RV TIMI 4/14 Killip I, AVB grade 1, PAC bigemini, controlled hypertension, and reactive leukocytosis. Patient underwent percutaneous coronary intervention in RCA on the 6th day of admission, in which an anomaly of the RV branch was found and perforation in the coronary artery occurred, thus a handmade cover stent was made to overcome the incidence. The procedure was finished with a total of 130 cc of contrast used and 100 cc of total bleeding. Patient was then admitted to the ICCU with stable vital signs and was evaluated for signs of tamponade using a Vscan every 3 hours. On the 8th day of hospital admission, patient had no complaints with stable vital signs. The final diagnosis at the time of outpatient was CAD 2VD post PTCA with 2 stents on RCA, RV Ellis type V branch perforation, STEMI Inferoposterior RV TIMI 4/14 Killip I, post AVB grade 1, post PAC bigemini, hypertension and reactive leukocytosis. Outpatient’s therapy included anticoagulants, statins, beta blockers, ACE-I, PPI, and sulfaftate.

Conclusion: We reported a rare case of RV branch artery perforation complication, specifically Ellis type V perforation during percutaneous coronary intervention that was then treated with a handmade covered stent. This perforation occurred due to a total occlusion at the proximal RCA. An anomaly of the RV branch caused the guidewire to perforate the distal part of RV branch artery. As there was no improvement achieved with balloon inflation, a handmade covered stent was then made to close the RV branch, which was placed on the RCA covering the ostium of the RV branch.

Keywords: Ellis type V perforation, coronary artery perforation, handmade cover stent

a fairly high mortality rate. Treatment of coronary perforation varies from using a covered stent, balloon inflation, thrombogenic coils, or by fat embolism.2

This study reports a patient with type V Ellis coronary artery perforation during percutaneous intervention. The patient presented with anomaly in the shape of the RV branch artery and was treated with a stent coated with Tegaderm, which acted as a covered stent.

**CASE REPORT**

A female patient with initial PCR, 69 years, with height of 156 cm, weight of 60 kg, normal nutritional status, address at Citra Raya Cikupa Tangerang, came to Prof. Kandou Hospital with complaint of chest pain since 3 days before admission. Chest pain was felt starting from the proximal radiating to her left side and also penetrated to her back. Patient could not precisely localize her pain. Patient described her pain as squeezing and felt uncomfortable; this occurred during rest time for more than 1 hour and finally dissipated itself. However, 4 hours afterwards, patient experienced another onset of chest pain, which was described as intense that it woke the patient from sleep and this time the pain did not go away. Patient also complained of shortness of breath during the attack, as well as cold sweats during the second attack of chest pain. Another complaint was nausea without vomiting also experienced during the attack. Fever, cough, runny nose, palpitations were denied by the patient. No complaints related to defecation and urination. This was her first experience of chest pain. There is no history of smoking and alcohol consumption, however patient has history of hypertension for the past 10 years and regularly consumes 5 mg of Amlodipine. Patient denies any history of type 2 diabetes mellitus, high cholesterol levels, kidney problems or cardiovascular treatment. The patient’s grand parents from her mother side had hypertension and died from a stroke, while the patient’s father also had a history of hypertension and died suspected of having a heart attack.

At first patient came to a private hospital in Tomohon, and was advised for immediate cardiac catheterization, but she refused and finally asked for outpatient treatment from the hospital. Patient continued to experience chest pain for 2 days afterwards and finally after 3 days from the first episode of chest pain, she went to Prof Kandou Hospital for treatment and agreed to cardiac catheterization.

On physical examination, general condition was found to be moderately ill, with awareness of compost mentis. On examination of vital signs, the patient’s blood pressure was 96/60 mmHg, pulse was 58 times/minute, respiratory rate was 20 times/minute, body temperature was 36.4°C and saturation was 98% without oxygen therapy. On head examination, there was no sign of anemia in conjunctiva and the sclera did not appear icteric. On neck examination, JVP 5+ cmH2O was found, and on palpation there was no palpable enlargement of the gland. On chest inspection, the shape was normal, there was symmetrical movement of the chest when breathing and iktus cordis was seen in the fifth intercostal space and anterior axillary line. There was no difference of tactile fremitus examination on the right and left lung fields, and on auscultation, vesicular breath sounds were heard from both lung fields, and no rhonchi or wheezing were heard from both lung fields. Heart sounds 1 and 2 were regular and no murmurs or gallops were heard. On the abdomen examination, the abdomen was flat, palpable soft, the liver was not palpable and no tenderness was felt on the abdominal area. On abdominal auscultation, bowel sounds were within normal limits. On extremities examination, the lower extremities were warm and there was no edema in both lower extremities.

Blood laboratory examination was performed with the results of hemoglobin 11.6 g/dl, erythrocytes 4.11 106/µL, leukocytes 14.900/µL, platelets 299,000/µL, hematocrit 34.7%, basophils 1, eosinophils 0, rod neutrophils 8, segment neutrophils 57, lymphocytes 22 monocytes 12, SGOT 82 U/L, SGPT 62 U/L, urea 18 mg/dL, creatinine 0.8 mg/dL, blood sugar 117 mg/dL, sodium 136 mEq/L, potassium 3.74 mEq/L, chloride 99.7 mEq/L, Total CK 456 U/L, 1998 U/L CKMB, troponin T<47, quantitative non reactive anti HCV, quantitative non reactive HBsAg, non reactive anti HIV, PT 16.5, INR 1.24, and APPT 50.6. Anterior-posterior view of chest x-ray showed the bones were intact, the diaphragm was normal, there was no deviation from the trachea, the costophrenic sinus angles on both sides were sharp, no mass, the lung fields and aorta were normal. Soft tissue of the chest wall was normal, organs under the diaphragm were also normal and no enlargement of the heart, thus conclusion of the chest x-ray was without any significant abnormalities.

On electrocardiographic examination found sinus rhythm with premature atrial complex (PAC) bigemini, heart rate 78 beats per minute, axis deviation to the left, normal P waves, prolonged PR interval 0.24 seconds, Q and ST elevations in II, III, AVF, V3R, V4R, V7-V9, ST depression I and AVL with a QRS wavelength of 0.10 seconds. There was also an inversion of the T wave in leads III, AVF, V3R and V4R, no U wave was found with the overall impression of the ECG was STEMI Inferior Posterior RV accompanied by bigemini PAC. Patient also had an echocardiography examination from the previous hospital with the results of mild dilatation of the right atrium and ventricle, the left ventricle was not thickened, the global left ventricular systolic function decreased with an ejection fraction of 45%. Left ventricular segmental analysis showed severe inferior hypokinetik, inferoseptal-apicoseptal, basal inferolateral mild hypokinetik, with other segments normokinetik. There was left ventricular diastolic dysfunction with impaired relaxation with increased LVEDP. A calcified aortic valve appeared with good function. There was mild regurgitation of the mitral valve caused by ischemia, mild-moderate tricuspid regurgitation was found with a low probability of pulmonary hypertension. Decreased right ventricular contractility and diameter of the inferior vena cava 2.1 cm, collapsibility <50% with an estimated right atrial pressure of 15 mmHg.

Based on the history, physical examination and supporting examinations, patient was identified with temporary working diagnosis of Recent Inferoposterior STEMI RV TIMI 4/14
CASE REPORT

Killip I, AVB grade 1, PAC bigemini, controlled hypertension, and reactive leukocytosis. Patient was treated with Asplet 1x80mg orally, Clopidogrel 1x75mg orally, Enoxaparin 2x60mg subcutaneously, Pantoprazole 2x30mg intravenously, Sucralfate syrup 3x15cc, Atorvastatin 1x40mg orally, NaCl 0.9% 1500cc/24 hours intravenously, Dobutamine 5 mcg/kg/min intravenously and Ceftriaxone 1x2 intravenously.

Initially patient was planned to undertake further treatment in the ICU room but because the ICU was fully utilized, patient was treated in the intermediate room (IMED). On the 2nd day of treatment (October 24th, 2020) in the IMED room, the patient complained of intermittent chest pain that lasted less than 5 minutes and dissipated itself, without any shortness of breath. On physical examination found blood pressure 93/47 mmHg, pulse 82x/minute, respiration 20x/minute, and body temperature 36.4°C. For the ECG examination, the readings were still the same as the previous readings in the emergency room. Fasting blood laboratory was performed on patients with the results of leukocytes 15,300/µL, erythrocytes 3.72 106 L, hemoglobin 10.5 g/dL, hematocrit 31.4%, platelets 259,000, MCH 28.2 pg, MCHC 33.4 g/µL, eosinophils 0, basophils 0, rod neutrophils 6, segment neutrophils 57, lymphocytes 18, monocytes 19 and MCV 84.3, total cholesterol 123 mg/dL, HDL 35 mg/dL, LDL 66 mg/dL, triglycerides 110 mg/dL, HbA1c 6.2%, fasting blood sugar 92 mg/dL, uric acid 4.9 mg/dL, magnesium 2.1 mg/dL, albumin 3.08 g/dL, calcium 8.17 mg/dL, magnesium 2.1 mg/dL, phosphorus 2.6 mg/dL. The treatment for patient was still the same as given in the emergency room with the 3rd and 4th injections of Enoxaparin on the second day of treatment, and addition of Azithromycin 1x500mg orally. Based on the results of this investigation, patient was diagnosed with additional hypoalbuminemia and advised to increase protein consumption, especially egg whites or cork fish. Patient was scheduled for a cath conference on October 26th. Patient did not undertake re-echocardiography because there had been an echocardiography results from the previous hospital and only video was taken for the cath conference.

On the 3rd day of treatment (October 25th, 2020), patient had no complaint of chest pain or shortness of breath. On examination of vital signs, blood pressure was found to be 95/60 mmHg with pulse 108x/minute, respiration 20x/minute, and body temperature 36.3°C. On physical examination there were no significant abnormalities and on the ECG examination, patient experienced a change from the previous ECG, which this day’s readings were sinus tachycardia of 108x/minute with axis deviation to the left and the patient’s PAC bigemini had disappeared, PR interval 0.22 seconds, ST elevation in the inferior segment and depression in I,AVL and V1-V3, T wave was not reversed. Treatment was still continued without any change in therapy. On the 4th day of treatment (October 26th, 2020), patient had no complaint of chest pain or shortness of breath. On physical examination, blood pressure was found to be 111/69 mmHg, pulse 92x/minute, breathing 18x/minute and temperature 36.2°C. On physical examination there were no significant abnormalities and on the ECG examination there were no significant changes, only the patient’s pulse rate had dropped to 92x/minute with the same ECG readings on the previous treatment. On this day the patient had a change in therapy where the administration of intravenous dobutamine was stopped and the administration of 0.9% NaCl was reduced by 500cc/24 hours and the total input and output were targeted to balance, patient was also given additional oral lactulose syrup 2x30cc. A cat conference was conducted with the results approved for percutaneous coronary intervention in this patient. Patient was also transferred for further treatment to the CVBC room on the 3rd floor.

On the 5th day of treatment (October 27th, 2020), patient felt chest pain again but with duration of <5 minutes for twice, but there were no complaints of shortness of breath. On physical examination, the pulse pressure was 104/62 mmHg, pulse 82x/minute, respiration 18x/minute and body temperature 36.5°C, no significant abnormalities were found on other physical examinations, and no changes were found in the ECG readings. Blood laboratory examination showed hemoglobin 9.7 g/dL, erythrocytes 3.41 106/µL, leukocytes 7.200/µL, platelets 261,000/µL, hematocrit 28.6%, basophils 0, eosinophils 0, neutrophils 0, neutrophils segment 56, lymphocytes 31 monocytes 13, SGOT 37 U/L, SGPT 54 U/L, urea 13 mg/dL, creatinine 0.6 mg/dL, sodium 138 mEq/L, potassium 3.54 mEq/L, chloride 103.3 mEq/L, calcium 7.83 mg/dL, magnesium 2.03 mg/dL, albumin 2.89 mg/dL, Ceftriaxone, enoxaparin, and azithromycin were discontinued, and at night for hydration, extra clopidogrel 300 mg and atorvastatin 80 mg orally were given as protocols for preparation of cardiac catheterization to be performed on the next day of treatment.

On the 6th day of treatment (October 28th, 2020), patient did not experience chest pain or shortness of breath. On physical examination, blood pressure was found to be 118/53 mmHg, pulse 92x/minute, breathing 18x/minute and temperature 36°C, on other examinations there were also no abnormalities. On this day, patient was planned for cardiac catheterization starting at 10 am. On coronary angiography, the left main artery (LM) was normal, the left anterior descending artery (LAD) showed 90-95% tubular stenosis in the middle artery, the left circumflex artery (LCx) was normal, and the right coronary artery (RCA) showed total occlusion in the proximal segment with grade V thrombus. Based on these results, it was continued with percutaneous coronary intervention in RCA. A JR 3.5/6F guiding catheter was inserted into the RCA, wiring was done with a hydrophilic guide wire to penetrate the lesion distally. A 2.0x20mm semi-compliant balloon was inserted and the lesion was evaluated, but still not visible. The balloon was replaced with a smaller semi-complain balloon measuring 1.5x15mm inflated many times from the distal to mid-artery to 10 atm, but the distal to the lesion was still not visible. After inflating the balloon several times there was a slight flow, and it turned out that the wire did not enter the distal RCA but into the distal branch of the RV. The patient had an anomaly of the RV branch, which appeared more angular forming the letter C and longer, thus resembled
the RCA. A perforation was seen distal to the RV branch artery classified as Ellis V. The hydrophilic guide wire was removed and could penetrate the lesion to the right distal RCA. The semi-compliant balloon was exchanged for a larger 2.0x20mm and inflated in the center region of the RCA before the estuary of the RV branch to 10 atm, and left for 20 minutes. After 20 minutes the balloon was deflated and on evaluation the perforation distal to the RV branch artery remained, and the 2.0x20mm semi-compliant balloon was re-inflated to 10 atm. A handmade cover stent was made using a 2.25x23mm Sirolimus drug eluting stent (DES) covered with Tegaderm. Then the balloon was deflated and the artificial stent cover was inserted into the middle of the RCA which when inflated would cover the mouth of the RV branch artery, then the stent was inflated to 14 atm. The Everolimus DES 2.5x28mm stent was placed in the proximal to mid-RCA overlapping segment with a handmade cover stent inflated to 14 atm and post-dilated with a balloon stent inflated to 16 atm. Coronary evaluation showed TIMI flow 3, thrombus (-), residual stenosis (-), no dissection and no flow to the RV branch arteries, thus the perforation was not visible again. The procedure was stopped with a total of 130 cc of contrast used and 100 cc of total bleeding. After the procedure, the patient’s blood pressure was 144/80 mmHg with a pulse of 90 beats/minute. Patient then admitted to have no complaints of chest pain or shortness of breath, but due to the distal perforation of the RV branch artery with Ellis type V classification, patient was planned to be observed in the ICCU first to monitor the risk of incidence of pericardial effusion and cardiac tamponade.

In the ICCU, the patient’s vital signs were stable and an evaluation for signs of tamponade was performed using a Vscan every 3 hours. On V-scan examination, pericardial effusion was found 0.3 cm in the posterior and apex, and 0.4 cm in the anterior. The Vscan was continued every 3 hours and after 3 repetitions with an interval of 3 hours the pericardial effusion did not increase and there were no signs of tamponade. At 22.00, patient was then transferred back to the intermediate room.

On the 7th day of treatment (October 29th, 2020), patient had no complaint of chest pain or shortness of breath. On physical examination and ECG examination, no significant changes or abnormalities were found from the previous day’s treatment. The patient’s diagnosis progressed to 2VD CHD post PTCA with RV branch perforation, and minimal pericardial effusion without signs of tamponade. Patient was re-examined with Vscan and no increase in pericardial effusion was found. Some additional therapy was given to the patient, which were ramipril 1x2.5 mg and KSR 3x600mg. Patient was then planned to move to the regular room.

On the 8th day of treatment (October 30th, 2020), patient had no complaints, with blood pressure 103/57 mmHg, pulse 90x/minute, breathing 18x/minute and temperature 36.1°C. On physical examination, no significant abnormalities were found. Patient had laboratory examinations to evaluate percutaneous coronary intervention with the results of hemoglobin 10.3 g/dL, erythrocytes 3.6 106/µL, leukocytes 10,500/µL, platelets 329,000/µL, hematocrit 30.9%, basophils 1, eosinophils 0, rod neutrophils 0, neutrophil segment 69, lymphocytes 19 monocytes 11, urea 10 mg/dL, creatinine 0.7 mg/dL, sodium 139 mEq/L, potassium 3.72 mEq/L, chloride 99.8 mEq/L. Patient was re-evaluated for pericardial effusion and there was no change in the results compared to the previous examination, thus patient was advised for outpatient treatment. The final diagnosis from the patient at the time of outpatient was CAD 2VD post PTCA with 2 stents on RCA, RV Ellis type V branch perforation, STEMI Inferoposterior RV TIMI 4/14 Killip 1, post AVB grade 1, post PAC bigemini, hypertension and reactive leukocytosis. Outpatient therapy was as follows: aspirin 1x80mg, clopidogrel 1x75mg, atorvastatin 1x40mg, ramipril 1x2.5mg, bisoprolol 1x1.25mg, lansoprazole 2x30mg and sucralfate 3x15cc.

**DISCUSSION**

Since the introduction of percutaneous coronary intervention in 1977 together with newer adjunctive tools and therapies, the use of percutaneous coronary intervention has continued to increase in the management of both simple and complex coronary lesions. Along with the increasingly complex lesion of the existing coronary, it is followed by the increasing incidence of complications, which are perforation of the coronary arteries. These complications are rare but often fatal. This coronary artery perforation can cause pericardial effusion with or without cardiac tamponade, which if not diagnosed and treated properly, can potentially lead to death.1

Coronary artery perforation occurs when a dissection or tear of the intima layer of the coronary artery completely penetrates the entire layer of the artery wall, this can be a small or large tear visible on angiography with the help of contrast extravasation. This extravasated blood will cause pericardial effusion, which can result in cardiac tamponade if left untreated. The incidence of coronary artery perforation varies depending on the medical center at which study was conducted, from the lowest of 0.1% to 3%, where the highest percentage occurs in patients with chronic total occlusions (CTO) and followed by a fairly high 30-day mortality rate that can reach 10.7% overall.2

Coronary artery perforation is reported to be associated with complexity of the existing coronary lesions, and the following are some risk factors for coronary artery perforation. Basically, these risk factors are divided into three, namely modifiable risk factors, non-modifiable risk factors and risk factors related to coronary anatomy and catheterization. The first non-modifiable risk factor is age, where the older the patient, the higher the incidence of coronary artery perforation. From a study conducted by Hendry et al., it showed that patients of the age over 65 had the higher risk of coronary artery perforation. The second risk factor is gender, where the percentage of coronary artery perforation is higher in female. The next risk factor is history of coronary artery bypass graft (CABG) where the incidence of perforation is also greater in this patient. Another risk factor is previous use of clopidogrel, where Doll et al found that patients with prior clopidogrel use had higher incidence of coronary artery perforation.

The next risk factors are modifiable. One example of modifiable risk factors is
the presence of comorbid hypertension, peripheral arterial disease, and a history of previous heart failure. These three are associated with a higher percentage of coronary artery perforation. The next two risk factors are low BMI and patients with low creatinine clearance, both of which are also associated with a higher incidence of coronary artery perforation.\(^3\)

The last group of risk factors is those related to the anatomy of coronary and also complexity of the catheterization procedure. A complex coronary lesion such as those of type B2, and C according to the division of the ACC/AHA. Patients with CTO lesions, severe calcification, high angle, tortoise and small coronary size also have an increased incidence of coronary artery perforation. And the last is the use of atheroablative devices during percutaneous coronary intervention and also the use of hydrophilic guide wire, all of which will increase the risk of coronary artery perforation. During percutaneous coronary intervention, coronary perforation usually occurs due to movement of the guide wire, balloon/stent, oversizing balloon/stent and when the balloon/stent is inflated. According to Witzke et al., initially coronary perforation was more often caused by debulking techniques than non-debulking and 50% of coronary perforations were associated with guide wire.\(^4,5\)

Coronary perforation often occurs during movement of the distal guide wire from the coronary, especially when the guide wire is trying to pass through the lesion. Hydrophilic guide wire has a higher risk of causing coronary perforation because of its low friction coefficient and easy distal displacement, this type of guide wire is indeed an option, especially in patients with severe stenotic lesions. Analysis by Kiernan et al showed that manipulation of the wire accounted for 66% (89% using hydrophilic guide wire), 16% due to stenting, angioplasty alone 8% and 11% due to rotational atherotomy. From another study said that the perforations caused by guidewires were generally small, thus the leakage of blood into the pericardial cavity occurred slowly, causing asymptomatic at first, but this was dangerous because often it became symptomatic when tamponade eventually occurred. Table 1 is the clinical picture in patients with perforation of the coronary arteries from the study conducted by Miguel et al.\(^6\)

Ellis et al based on their research conducted in 1994 classified coronary perforation into several types. The first is Ellis type I characterized by the presence of an extraluminal dome but without extravasation of contrast, the next is Ellis type II characterized by the presence of myocardial or pericardial blushing without the presence of jets of contrast extravasation, and Ellis type III characterized by the presence of contrast extravasation and a clear perforation measuring more than 1 mm, which is also accompanied by contrast streaming spilling. The last one is Ellis type IV or often also called as Tioe III CS, which is similar to Ellis type III but accompanied by cavity spilling. In 2008 Muller et al proposed adding another type, namely Ellis type V, where this type of perforation is characterized by perforation of the distal coronary due to the use of rigid or hydrophilic guide wires. Based on a study by Miguel et al., the most common occurrence of Ellis type III was with an incidence rate of 54.8%, followed by Ellis type II at 40.4%, where the other 3 types of Ellis had a total incidence rate of just under 5%.\(^7\)

Cardiac tamponade is a risk that can occur in patients with coronary perforation, and the incidence was mostly due to Ellis type III coronary perforation. In patients with cardiac perforation, nearly 40% of patients develop pericardial effusion and about 10-20% develop cardiac tamponade. Often the picture of tamponade is not immediately visible even the symptoms only appear 1 week after percutaneous coronary intervention. The incidence of coronary perforation will also significantly increase the risk of death in patients after percutaneous coronary intervention.

Management of coronary artery perforation is highly dependent on the severity of the perforation based on the Ellis classification system and the vital signs of the patient. The main goal of treatment for coronary artery perforation is to close the perforation as quickly as possible so that the pericardial effusion does not develop into a very dangerous cardiac tamponade. Definite treatment pathways for coronary artery perforation do not yet exist because studies and guidelines are minimal in this regard. Therefore, there are various approaches in the management of coronary perforation. Management strategies vary widely from observation to immediate corrective surgery, this is influenced by many factors

<table>
<thead>
<tr>
<th>Clinical events during hospital admission</th>
<th>Results %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tamponade treated with pericardiocentesis</td>
<td>48.0</td>
</tr>
<tr>
<td>Tamponade treated with surgery</td>
<td>2.7</td>
</tr>
<tr>
<td>Pericardiocentesis after undiagnosed coronary perforation</td>
<td>75.7</td>
</tr>
<tr>
<td>Pericardiocentesis after insertion of covered stent</td>
<td>52.8</td>
</tr>
<tr>
<td>Pericardiocentesis after insertion of coil</td>
<td>100</td>
</tr>
<tr>
<td>Pericardiocentesis after conservative procedure or balloon inflation</td>
<td>32.9</td>
</tr>
<tr>
<td>Cardia surgery</td>
<td>12.7</td>
</tr>
<tr>
<td>Periprocedural myocardial infarction</td>
<td>34.0</td>
</tr>
<tr>
<td>Bail out CABG</td>
<td>5.3</td>
</tr>
<tr>
<td>Death</td>
<td>8.0</td>
</tr>
<tr>
<td>Post-hospital mortality</td>
<td></td>
</tr>
<tr>
<td>Death in 30 days</td>
<td>10.7</td>
</tr>
<tr>
<td>Death in 1 year</td>
<td>17.8</td>
</tr>
</tbody>
</table>
but the important factors that play a role are the type of perforation based on Ellis’ classification, the hemodynamic status of the patient, the availability of equipment at the hospital where we work, and the ability of the operator.\(^1\),\(^3\)

The initial action that can be done with almost all types of Ellis classification is inflation of low pressure balloons ranging from 2-6 atm with a minimum duration of 10 minutes to close the perforation area. With low-pressure balloon inflation, it is hoped that hemostasis will occur so that the perforation can close spontaneously, especially in type I and II. If after inflation extravasation still exists, the balloon is re-expanded while planning for the next steps to handle this perforation. Protamine sulfate to convert the effects of heparin, this in itself is still contradictory because several studies reported that administration of protamine sulfate would cause aggregation of platelets and could cause thrombosis of existing stents. It is important to note that patients who have previously used NPH insulin will experience adverse events from the use of protamine sulfate. The use of newer antithrombotics such as GpIIb/IIIa inhibitors and bivalirudin should generally does not require special treatment, because it will often close spontaneously. However, several reports have described cases with high risk of tamponade. The operator should be prepared for pericardiocentesis if the patient has signs of tamponade. Often this becomes difficult because pericardiocentesis is performed in patients who tend to be restive and there is no much fluid in the pericardial cavity. If the tamponade has been handled and the patient tends to be stable, then the covered stent can be placed to close the perforated area on the coronary, if the covered stent is unable to close the perforation then the last resort will be corrective surgery.

Type IV Ellis artery perforation generally does not require special treatment, because it will often close spontaneously. This type also very rarely causes cardiac tamponade, but more often ischemia or myocardial infarction due to coronary extravasation into the cardiac chambers. If this incidence occurs then a covered stent may be considered. The last type of perforation is Ellis type V which is different from other types because perforation occurs at the end of the coronary artery. This can occur as a result of unintentional movement of the guide wire or if there is a sharper angle of the RV branch that is initially suspected as an RCA in a patient with total occlusion proximal to the RCA. The following is a specific algorithm for the treatment of Ellis type V.\(^8\),\(^9\)

Similar to type I-IV Ellis coronary perforation, the initial procedure for type V Ellis coronary perforation is balloon inflation proximal to the coronary artery perforation. If the patient is hemodynamically stable, the perforation can be checked again after 5-10 minutes of balloon inflation, but if the patient is unstable during balloon inflation, a call for help from the other teams of anaesthetists, surgeons, and echocardiographers is needed. In hemodynamically unstable patients, cardiac tamponade generally has occurred; therefore in unstable patients the catheterization team must be ready for immediate pericardiocentesis.

If balloon inflation fails to close there are three things that can be done. Anticoagulant reversal as in other types can be considered but at the time of reversal performed the balloon and guide wire must be removed first. Furthermore, injection of thrombin can directly stimulate fibrin formation. This procedure requires proper administration of thrombin-containing fluid at the perforated area. This injection process can use the smallest available balloon lumen. Next fat embolization can also be utilized. The utilized fat can come from subcutaneous tissue in the abdominal or femoral area, this fat tissue can be a physical barrier from blood leakage, also this can add up to fat tissue that will stimulate the coagulation pathway thus closing the coronary artery perforation. This procedure requires a small adipose tissue so it can pass through the thrombo-aspiration catheter depending on the size, if using a 6F it would be size <1mm, 7F with size <1.2mm. The next choice of procedure is autologous blood clots,
CASE REPORT

blood clots can be obtained easily and will be biocompatible to all patients and do not add to the cost of the procedure. Blood clots combined with contrast and saline have been reported by Tanak et al to close the perforation and will not undergo lysis 6 months later even with antplatelet therapy given to the patient.

The next option is to use microspheres/microparticles. Microparticles are spherical, hydrophilic, non-absorbable particles with sizes varying from 1-1500 micrometers that can be placed into the perforation area using a micro catheter. Because of the wide variety of sizes, embolization can be easily performed on any size of blood vessel. In general, the use of these microparticles is an option in more peripheral blood vessels and their use in addition to coronary artery perforation can also be used to close the arterial supply from tumors and arteriovenous malformations.

Coil is a permanent metallic agent usually made of stainless steel or platinum accompanied by synthetic wire wool or dacron fiber and a thrombogenic agent. The coil can be placed at the site of the perforation with a normal guide catheter, which is commonly used in percutaneous coronary intervention. The choice of coil size must be slightly larger than the size of the blood vessel in order for complete embolization to occur, if the size is too large then the coil will be pushed proximally, if it is too small then the coil will be pushed to the distal area of the perforation. This coil is the choice for distal perforations such as the Ellis type V and also small segment caliber.

If the distal coronary perforation occurs in a branch of a major vessel then a covered stent may also be used as it was in this patient. Initially covered stent was designed to treat the incidence of in-stent restenosis but over time covered stent has an important role as an important action in the treatment of coronary perforation, especially in the proximal segment measuring more than 2.75 mm. In general, covered stent is the main choice in the treatment of Ellis type III but can also be used for type V Ellis perforation of the main vessel branch, as in this case there was perforation of the RV branch artery and a handmade covered stent was placed on the RCA. Covered stent itself consists of several types with 2 main groups forming the graft, namely polytetrafluoroethylene (Grandmaster, Begraft) and polyurethanes (PK Papyrus). Types with graft-forming polyurethanes are a newer generation compared to polytetrafluoroethylene. In this newer generation stent placement time is shorter, but there is no significant difference in the incidence of stenosis, MACE, or mortality. In this case report, because at the time of coronary perforation a covered stent was not available, a DES coated with Tegaderm was used.

Tegaderm itself is generally used for wound management where its use aims to provide a barrier between the skin and wound, absorb exudate and reduce pain at the wound site. It can be used in wounds with as little as 1 mm depth, as it is adherent and remains intact even if the wound is mobile or subject to friction. Tegaderm bandages can be removed and re-applied as often as necessary, which allows for the continuous care of the wound and prevents wound infection. Tegaderm is known for its easy application and removal, which is important in cases of wound care.

Figure 2. Treatment algorithm of Ellis type V coronary perforation.
to keep the wound sterile and moist thus faster healing process can take place and formation of scar tissue will reduce. Tegaderm consists of a polyurethane membrane coated by an adhesive, and is impermeable to liquids. Due to the limited equipment available and the urgency of the situation, the operator decided to use a handmade covered stent where DES was coated with Tegaderm where basically this Tegaderm has a basic composition that is more or less the same as the latest generation covered stent.\textsuperscript{11}

In general, the incidence of coronary perforation can be treated with percutaneous intervention alone, but if percutaneous intervention also cannot resolve the perforation, the last resort is surgical correction. This surgery can repair the perforation or ligate the perforated coronary and followed by bypass grafting process if there is a stenosis that has not been treated at the time of percutaneous coronary intervention. Therefore, the treatment of coronary perforation requires a skilled and capable team, as well as adequate tools, but in medical centers with limitations in Indonesia, options such as formation of a handmade covered stent can be used as a solution.\textsuperscript{12}

CONCLUSION

We reported a case report of a Recent STEMI Inferoposterior+RV patient with RV branch artery perforation complication at percutaneous coronary intervention that was treated with a handmade covered stent. Coronary perforation occurred in this patient was Ellis type V. This occurred due to total occlusion at the proximal RCA and the patient had anomaly of RV branch shape that resembles the RCA, so that the guidewire perforated distal of the RV branch artery. After trying to treat it with balloon inflation, the perforation was not resolved, then it was planned to close the RV branch using handmade covered stent that was placed on the RCA covering the ostium of the RV branch.

ETHICAL STATEMENT

The research has received ethical approval based on the ethics approval certificate by the Health Research Ethics Commission of Universitas Sam Ratulangi, Manado, North Sulawesi.

AUTHOR CONTRIBUTION

All authors have the same contribution in writing this article.

FUNDING

This research did not receive any funding from public, commercial or non-profit sector.

CONFLICT OF INTEREST

The authors declare there is no conflict interest in the making of this manuscript.

REFERENCES

APPENDIX

ECG from the Previous Referral Private Hospital

ECG 23/10/2020
CASE REPORT

ECG 24/10/2020

Echo Readings from the Referral Hospital

ECG 25/10/2020
Catheterization images