CASE REPORT

Bali Medical Journal (*Bali MedJ*) 2022, Volume 11, Number 3: 1811-1816
P-ISSN.2089-1180, E-ISSN: 2302-2914

Emergency thrombectomy in acute upper limb ischemia patient with diabetes mellitus

Yunita Arif[1*], Hermina Novida[2]

[1]Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga - Dr. Soetomo General Hospital, Surabaya, 60132, Indonesia; [2]Division of Endocrinology, Metabolic Diseases and Diabetes, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga - Dr. Soetomo General Hospital, Surabaya, 60132, Indonesia;

*Corresponding author:
Yunita Arif;
Metabolic Diseases and Diabetes, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga - Dr. Soetomo General Hospital, Surabaya, 60132, Indonesia;
yunita.arif@gmail.com

Received: 2022-10-07
Accepted: 2022-11-05
Published: 2022-11-30

ABSTRACT

Background: Acute limb ischemia is a sudden decrease in limb blood perfusion with an onset of less than 14 days, most commonly occurs in lower extremities. Diabetes mellitus, one of diseases underlying peripheral artery disease, complicates the pathophysiology of acute limb ischemia. In this case report we present a patient with type 2 diabetes mellitus and acute limb ischemia in the left upper extremity that had successful thrombectomy treatment.

Case presentation: A 58-year-old female patient came to the Emergency Care Unit of Dr. Soetomo General Hospital with the continuous pain in the five fingers to the palm of the left hand for the past two days. The numbness spread from the elbow to the left fingers, making them weak and difficult to grip. Those five fingers were pale and cold as well. The left hand had no pulsated radialis and ulnar artery, weak brachial pulse and undetected pulse oximetry result. The patient was diagnosed with type 2 diabetes mellitus since 2005 and has a controlled treatment. On the first day of hospitalization, the patient was scheduled for emergency thrombectomy at Dr. Soetomo General Hospital. Follow-up on the seventh-day post-surgery, the patient had no complaints of pain and pale in the left hand and the brachial, ulnar and radial arteries were palpable.

Conclusion: Acute limb ischemia is uncommon in the upper extremities but we found that it has an excellent prognosis with early revascularization, even in a patient with diabetes mellitus. The treatment must be in conjunction with aggressive diabetes mellitus treatment to avoid acute limb ischemia worseningt.

Keywords: acute limb ischemia, revascularization, diabetes mellitus, peripheral artery disease, Indonesia.
Cite This Article: Arif, Y., Novida, H. 2022. Emergency thrombectomy in acute upper limb ischemia patient with diabetes mellitus. *Bali Medical Journal* 11(3): 1811-1816. DOI: 10.15562/bmj.v11i3.3822

INTRODUCTION

Acute limb ischemia (ALI) is a condition where there is a sudden decrease in limb blood perfusion within 14 days. It is one of vascular emergency cases that causes a decrease in extremity viability and even dead limbs if left untreated immediately. Most ALI cases involve the lower extremities. The incidence of acute upper limb ischemia (AULI) is only about 10-15% of all ALI cases.[1] The anticoagulation with the adjusted dose based on the severity of ALI may help, but revascularization therapy remains the treatment of choice in patients with ALI.[2]

ALI is the part of peripheral artery disease (PAD) - a blood vessel disorder characterized by progressive narrowing and blockage of blood vessels mainly due to atherosclerosis, ranging from mild plaque formation to chronic vessel occlusion.[3-5] Risk factors for PAD include smoking, diabetes mellitus (DM), obesity, high blood pressure, high cholesterol, aging, genetics, heart disease and stroke.[3-5] DM is a major risk factor for PAD[6] and associated with other diseases.[7,8]

Epidemiological data show a strong relationship between PAD and DM with the prevalence of PAD in DM patients is 20%.[6] Every 1% increase in HbA1C level will increase the risk of PAD by 28%.[6] A common symptom of PAD is the presence of intermittent claudication, which is felt as pain or cramp during activity and improved by resting. However, in patients with DM, PAD symptoms may be asymptomatic due to peripheral neuropathy and cause a delay of therapy.[9] In this case report, we report a case of AULI with DM that was treated with thrombectomy and resulted an immediate improvement.

CASE PRESENTATION

A 58-year-old woman, came to Emergency Care Unit of Dr. Soetomo General Hospital with pain in the five fingers of the left hand. The pain was continuously felt from the five fingers to the palm for two days ago. The numbness from the elbow to the left fingers caused weak grip. The five fingers appeared pale in the last two days. When palpated, the five fingers were cold and blue with no swollen nor deformity (Figure 1). There was no stiffness nor trauma in the fingers. The patient also complained of nausea and vomiting 2–3 times a day during the same days span. There was no history of fever, cough and shortness of breath. The patient had lack of appetite due to nausea.

From the previous medical history, the patient has had type 2 DM (T2DM) since 2005, usually controlled at a health center

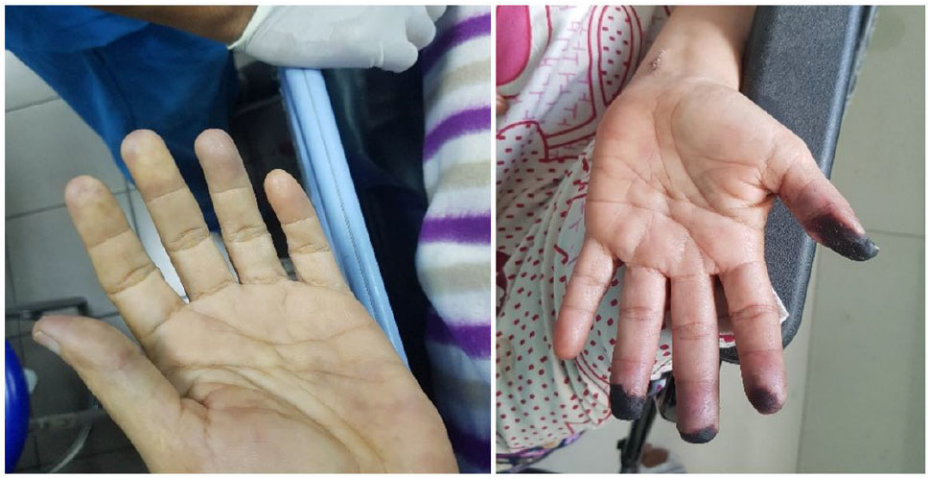

Figure 1. The pale palm and bluish decolorization of the fingertips in the left upper extremity.

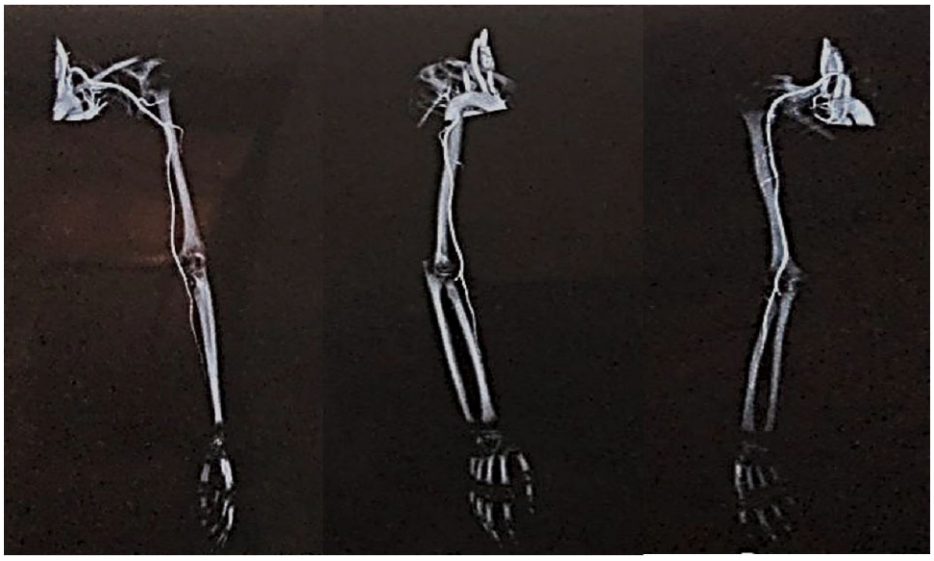

Figure 2. CT angiogram of the left upper extremity demonstrated a filling defect of ± 5 cm in the left radial artery at ± 15 cm from brachial artery. The left ulnar artery also had a filling defect of ± 0.9 cm at ± 0.9 cm from the brachial artery.

near the patient's house. The routine DM medications were glimepiride 2 mg once a day and metformin 500 mg 3 times a day. The patient also had hypertension but was not treated regularly, which was treated with amlodipine 10 mg daily. In 2018, the patient underwent surgery on her left toe due to a vascular blockage at Dr. Soetomo General Hospital. The patient's father also had DM.

On physical examination, the general condition was weak, with Glasgow Coma Scale (GCS) 15, blood pressure 100/80 mmHg, heart rate 98x/min, respiratory rate 20x/min, body temperature 36.3°C, oxygen saturation 98%, visual analogue scale (VAS) score 6. Head and neck examination revealed no abnormalities, anemia, cyanosis, jaundice and dyspnea. The jugular venous pressure (JVP) was in normal range with no enlarged lymph nodes. Chest and abdominal examination showed no abnormalities. The examination of superior extremities showed that the left palm and fingers were pale, cold, no pulse on radialis and ulnar artery, weak brachial pulse and undetected pulse oximetry result (Figure 1). The patient was still able to move all five fingers. The ankle-brachial index (ABI) examination revealed 0.8 in the left side and normal in the right side.

The results of laboratory examination were: Hb 11.8 g/dL, leukocytes 13.320/µL, neutrophils 77%, platelets 275.000/µL, sodium 137 mmol/L, potassium 3.8 mmol/L, chloride 103 mmol/L, serum glutamic oxaloacetic transaminase (SGOT) 16 U/L, serum glutamic pyruvic transaminase (SGPT) 20 U/L, blood urea nitrogen (BUN) 20 mg/dL, serum creatinine 0.15 mg/dL, activated partial thromboplastin time (APTT) 25.4 sec, prothrombin time (PT) 9.8 sec and random blood glucose 300 mg/dL.

The chest radiography was normal. The electrocardiogram (ECG) showed sinus rhythm 92 x/min, inferior old myocardial infarct (OMI), anteroseptal ischemia with complete right bundle branch block (RBBB), right axis deviation (RAD) and counter-clockwise rotation (CCWR). The results of echocardiography showed the dimensions of all heart chambers were normal, systolic function of left ventricle (LV) was normal (ejection fraction; EF 68%), diastolic function of LV was abnormally relaxed, systolic function of right ventricle (RV) was normal, segmental analysis of LV was normokinetic, and left ventricle hypertrophy (LVH).

The CT-angiogram of the left upper extremity showed no contrast flow in the left radial artery at a distance of ±15 cm from brachial artery, ±5 cm long. The contrast flow was also not seen in the left ulnar artery at a distance ±0.9 cm from brachial artery, ±3 cm long (Figure 2). The results of left extremity Doppler ultrasound showed soft plaque >50% on the left radial artery at wrist and left ulnar artery at mid antebrachial level to the wrist. The peak systolic velocity (PSV) and pulsatility index (PI) of the arteries outside the lesion was within normal limits. The Doppler ultrasonography of all arteries in the left upper extremities had triphasic waveforms which was normal.

The patient was consulted to Cardiology Department and the patients was diagnosed with ALI in the left superior extremity, Rutherford IIB. The patient was treated with aspirin 100 mg once a day, cilostazol 50 mg twice a day, beraprost sodium 20 mcg 3 times a day and atorvastatin 40 mg once a day. The patient was also consulted to Thoracic and Cardiovascular Surgery Department under the same diagnosis and treated with

cito thrombectomy and heparinization 10 mg/kg/hour.

After all examinations and consultation, the patient was scheduled for the examination of fasting blood glucose (FBG), 2 hours post-prandial blood glucose (2hppBG), HbA1C, lipid profile, PT/APTT (every 6 h) and fibrinogen. The patient was then injected with actrapid for blood glucose regulation 4 units in 2 injections intravenously every 1 hour. After random blood glucose (RBG) was corrected to 187 mg/dL, the treatment was continued with insulin aspart (NovoRapid) injection 6 units sub cutaneous 3 times a day before meal, insulin detemir (Levemir) injection 10 units sub cutaneous once a day, diet for DM with cardiovascular diseases (diet-KV) 1900 kcal/day, tramadol injection 100 mg intravenously 3 times a day, lansoprazole injection 2 times a day intravenously, clopidogrel 75 mg orally once a day, cilostazol 100 mg orally twice a day, dorner 20 mcg 3 times a day, atorvastatin 40 mg per day once a day, unfractionated heparin (UFH) injection 2500 units followed by UFH 10 IU/kg/hour with the targeted PT/APTT was of 1.5-2x normal and blood glucose was 140–180 mg/dL. The patient was then hospitalized for the follow-up after surgery.

On the second day, the pain in the five fingers of her left hand had decreased, but still pale and cold. The general condition was weak, GCS 15, blood pressure 120/80 mmHg, heart rate 96 x/min, respiratory rate 18 x /min, body temperature 37.1°C and VAS score 3-4. Postoperative retrograde thrombectomy of the left ulnar artery showed a 0.5 cm of intraluminal thrombus at 13 cm of distal artery. Following the thrombectomy, the palpation was felt on the left axillary artery (+2), left brachial artery (+), left ulnar artery (+), but radial artery was difficult to evaluate. The laboratory results were FBG 243 mg/dL, 2hppBG 256 mg/dL, APTT 26.8 sec, PT 10 sec, potassium 3.4 mmol/L, sodium 144 mmol/L, chloride 102 mmol/L, albumin 3.7 g/dL, Hb 12.5 g/dL, leukocytes 21,318/µL, neutrophils 90%, platelets 291,000/µL and HbA1C 9.5. The patient was diagnosed with ALI of the left superior extremity post thrombectomy and T2DM. The patient was scheduled for lipid profile examination, hemostasis evaluation every 6 h with a targeted PT/APTT 2–3x normal, the patient was injected with NovoRapid 6 units sub cutaneous 3 times a day before meal, Levemir injection 10 units sub cutaneous once a day, diet-KV 1900 kcal/day, heparin 10 IU/kg/hour, lansoprazole injection twice a day intravenously, metoclopramide injection 3 times a day intravenously (if needed), clopidogrel 75 mg orally once a day, cilostazol 100 mg per 12 h orally, tramadol 100 mg in 100 cc NaCl 0.9% 3 times a day, ceftriaxone injection 1 gram intravenously twice a day.

The pain and pale were lessening and the patient was no longer weak on the third day. The general examination was GCS 15, blood pressure 120/80 mmHg, heart rate 96 x/min, respiratory rate 18 x/min, body temperature 37.1°C and VAS score 3. The left axillary, brachial and ulnar artery were all palpated (+2, +, +, respectively) and the radial artery was still difficult to evaluate. The PT/APTT were 9.6 and 23.6 sec, respectively at 12:00 am, 9 and 27 sec at 06:00 pm, 9.2 and 30 sec at 00:00 am, fasting blood glucose 188 mg/dL, 2hppBG 236 mg/dL, potassium 3.3 mmol/dL, sodium 135 mmol/dL, chloride 97 mmol/dL, RBG 196 mg/dL, Hb 12.7 g/dL, leukocytes 15900/µL, neutrophils 83%, platelets 340,000 /µL, SGOT 18 mg/dL, SGPT 20 mg/dL, BUN 14 mg/dL, serum creatinine 0.69 mg/dL, total cholesterol 236 mg/dL, low-density lipoprotein (LDL) 180 mg/dL, triglycerides (TG) 129 mg/dL, high-density lipoprotein (HDL) 39 mg/dL. Dyslipidemia was added to the patient's diagnosis. The patient was injected with Novorapid 10 units sub cutaneous 3 times a day before meal, Levemir injection increased to 12 units sub cutaneous once a day, diet-KV 1900 kcal/day, heparinization increased to 12 IU /kg/hour, CPG 1 x 75 mg orally, cilostazol 100 mg per 12 h orally, atorvastatin 40 mg orally once a day, ceftriaxone injection 1 gram intravenously twice a day, metamizole injection 1 ampoule intravenously 3 times a day, dorner 20 mg orally 3 times a day and pentoxifylline 400 mg orally 3 times a day.

On the sixth day of treatment, the patient had no pain in the left hand, the five fingers and the palm turned red. The physical examination revealed GCS 15, blood pressure 110/78 mmHg, heart rate 94 x/min, respiratory rate 18 x/min, body temperature 37.2°C and VAS score 0. The left axillary, brachial ulnar and radial artery was palpated (+, +2, +2 and +, respectively). The PT/APTT was 11.1/40.7 sec at 06:00 am, 11/49.2 sec at 12:00 am, 11.3/57 sec at 06.00 am, fibrinogen 110 mg/dL, FBG 110 mg/dL and 2hppBG 130 mg/dL. The patient was treated with NovoRapid injection 10 units sub cutaneous before meals 3 times a day, Levemir injection 12 units sub cutaneous once a day, diet-KV 1900 kcal/day, heparinization 15 IU/kg/hour, Lansoprazole intravenous injection 2 times a day, metoclopramide intravenous injection 3 times a day (if needed), clopidogrel 75 mg orally once a day, cilostazol 100 mg twice a day orally, ceftriaxone intravenous injection for 1 gram twice a day, dorner 20 mg orally 3 times a day, pentoxifylline 400 mg orally 3 times a day, atorvastatin 40 mg orally once a day.

On the seventh day of treatment, the patient had no complaints of pain in the left hand, the five fingers and the palms were red. The general examination showed GCS 15, blood pressure 120/78 mmHg, heart rate 94 x/min, respiratory rate 18 x/min, body temperature 37.2 °C, and VAS score 0. The left axillary, brachial, ulnar and radial artery were palpated (+, +2, +, and +, respectively). The PT/APTT was 11.1/32.7 sec when checked at 06:00 am, FBG 105 mg/dL and 2hppBG 123 mg/dL. The patient was discharged with the treatments were NovoRapid injection 10 units sub cutaneous 3 times a day before meal, Levemir injection 12 units sub cutaneous once a day, clopidogrel 75 mg orally once a day, cilostazol 100 mg orally twice a day, dorner 20 mg and atorvastatin 40 mg orally once a day.

DISCUSSION

The mechanism of PAD is progressive narrowing and blockage of blood vessels mainly due to atherosclerosis, ranging from mild plaque formation to chronic occlusion of blood vessels. PAD symptoms' severity depends on the blockage's severity and the presence or absence of collateral vessels.[10] ALI is a form of PAD that requires immediate treatment. The clinical features of ALI can occur within minutes, hours, or days and vary from intermittent episodes

of claudication to pain at rest, or sudden pain in the affected extremity, numbness of the extremity, absence of pulsations of the distal part to the occlusion, cold skin, pale and may be accompanied by motor impairment. These signs and symptoms are commonly abbreviated as 6Ps: paresthesia, pain, pale, pulselessness, poikilothermic and paralysis.[1,2,11] The most common cause of AULI is embolic process (90%) originating from the heart, trauma or embolism from aorta and its branches. AULI most often occurs in the brachial, radial and ulnar arteries.[12]

In this case, the patient complained of pain in the five fingers to the palm accompanied by numbness to the left elbow, the left hand felt weak so it was difficult to grip. This complaint appeared for 2 days before she was admitted to the hospital. There was no history of such complaints. On physical examination, the left palm looked pale, cold, and there was no left ulnar and radial artery pulsation, and weak brachial artery pulsation.

ABI is a first-line examination in patients with suspected ALI accompanied by PAD symptoms. This test measures the systolic blood pressure of the arm (brachial artery) and ankle (dorsalis pedis and posterior tibial artery) in the supine position using a sphygmomanometer or Doppler ultrasonography. This test is non-invasive and easy to perform. Interpretation of ABI score was > 1.3 was non-compressible, 1-1.3 was normal, 0.9-1 was borderline, 0.71-0.9 was mild, 0.41-0.7 was moderate and 0.0- 0.4 was severe PAD.[11,13] If the result of ABI is positive for PAD (0.9 or lower), it is necessary to carry out further supporting examinations related to ALI. The patient in this study had ABI 0.8 for the left extremities, which means mild PAD, and 1 for another side.

The common scoring system used to determine the clinical state of PAD is Fontaine and Rutherford classification.[14,15] This classification may help determining the right therapy for PAD patients. The Rutherford classification is most often used to determine the management of ALI. This classification may determine the prognosis of ALI, sensory function, motor function, and arterial and venous blood flow of the extremity.[1,2] In this study, the patient had numb fingers of the left hand to the elbow. The motor function of the left hand decreased, the five fingers were difficult to move due to continuous pain and there was no tissue damage in the extremities. The Doppler ultrasonography showed soft plaque > 50% on the left radial artery at wrist level and left ulnar artery at mid antebrachial to wrist level. The PSV and VI in Doppler ultrasound were normal in all arteries and the triphasic waveform was detected. Based on this classification, the diagnosis of the patient became ALI Rutherford IIB of the left superior extremities.

Among Vascular Doppler, Doppler ultrasound (DUS), Computed tomography (CT) angiography, magnetic resonance angiography (MRA), digital subtraction angiography (DSA), CT angiography is the gold standard for diagnosing ALI.[10] Digital subtraction angiography (DSA) is the most accurate test to determine the anatomy and vascular abnormalities, but the examination is invasive so the risk of bleeding, infection, and rupture is high. Doppler ultrasound (DUS) is non-invasive, non-radiative, inexpensive and requires a relatively short time to examine. It also provides important information about hemodynamics (proximal and distal to obstruction) and patency of vascular flow. The limitation of DUS is its dependence on the operator and inability to provide a clear picture of the arteries. A more precise imaging technology is therefore required.[2] The imaging modality of choice in ALI patient is CT angiography, with a 98% specificity and 96% sensitivity. It clearly shows calcifications, stents, location, level of occlusion and patency of blood vessels. Computed tomography angiography has a high enough risk in patients with renal impairment (glomerular filtration rate of less than 60 mL/min). Magnetic resonance angiography can still be used in patients with renal impairment with a filtration rate of less than 30 mL/min, but MRA cannot detect arterial calcification, thus providing limited information for anastomotic site selection and cannot be performed in patients with pacemakers and metal implants.[1,10]

Blood tests that were examined including complete blood count, hemostatic function, LDH, D-Dimer, BUN, creatinine, lactic acid, potassium, blood gas analysis, blood glucose. However, laboratory examination does not really contribute in establishing the diagnosis of ALI. Laboratory examinations are only to assess general health status and assess comorbidities.[2,10]

Risk factors for PAD include smoking, hypertension, dyslipidemia, age, gender, and diabetes mellitus.[6,16] DM is a very important risk factor for PAD due to abnormal conditions in DM such as hyperglycemia, insulin resistance and dyslipidemia, which may trigger the changes in the function and structure of blood vessels. Mechanisms of changes that occur such as increased oxidative stress, impaired intracellular signal transduction by protein kinase C (PKC) and receptor for advanced glycation end-products (RAGE) activation.[17] As a result, there will be a decrease in nitric oxide (NO) and an increase in endothelin which are strong vasoconstrictors, activation of proinflammatory transcription factors such as nuclear factor kappa B, AP-1 and an increase in the prothrombotic factor plasminogen activator inhibitor.[10,17,18] The risk factors in this patient were a history of T2DM since 2005, dyslipidemia and a postoperative history due to PAD on the left toe in 2018.

In all ALI cases, initial anticoagulant therapy such as unfractionated heparin (UFH) should be given immediately before and after the procedure to reduce thrombus propagation. Revascularization is the main therapy for ALI patients with the technique is based on the type of occlusion (thrombus or embolism), location, duration of ischemia, comorbidities and Rutherford classification.[2]

There are some endovascular treatment techniques such as catheter-directed thrombolysis (CDT), percutaneous thromboaspiration (PAT) with or without thrombolytic therapy and percutaneous mechanical thrombectomy (PMT). Catheter directed thrombolysis is not recommended in ALI class IIb and III, because it takes a long time to reperfusion. In addition, ALI lasting longer than 14 days makes it challenging to insert the catheter through the thrombus, and this treatment is not advised in individuals who have thrombolysis contraindications. Approximately 25% of patients with CDT

therapy require additional open surgical revascularization, despite the fact that this method is extremely effective. Although PAT, which has a 30% effectiveness rate, is extremely effective, some studies claim that using thrombolytic drugs in conjunction with this treatment may improve outcomes. Percutaneous mechanical thrombectomy is the first choice of therapy in ALI class IIb, because the time to reperfusion is shorter when compared to CDT. Patients with contraindications to thrombolysis and who are at high risk for surgery can undergo PMT. In patients with a high risk of bleeding, PMT can be used to remove thrombus masses before local thrombolysis and distal embolic complications that occur after CDT.[1,2]

Open surgical revascularization is usually performed in severe ALI cases where the affected extremity is not viable, in ALI with onset of more than 14 days, or in which thrombolysis is contraindicated. This open surgical revascularization consists of: thrombectomy with a Fogarty balloon catheter, bypass surgery, and adjuncts such as endarterectomy, intra-operative thrombolysis and patch angioplasty. Patients with suspected embolism and no ipsilateral pulse of an ischemic extremity are best treated with Fogarty catheter balloon thrombectomy. This bypass surgery is usually performed in patients with ALI caused by thrombosis with underlying PAD. These patients are not suitable for balloon catheter thrombectomy, because thrombus may reoccur. Intraoperative thrombolysis, this procedure is rarely used and is only performed in special cases such as secondary ALI due to thrombus from popliteal artery aneurysm with tibial occlusion.[1,2]

There are some treatments for ALI based on its classification. ALI class I, normal motor function without sensory loss and with intact capillary refill, usually only requires systemic anticoagulant therapy and stabilization of the patient in the early management phase. ALI class IIa, slow capillary refill with limited sensory loss and movement, must be revascularized. The treatment of choice for ALI class IIb, sensory loss spreading more than toe and rest pain, is also revascularization. Amputation is the primary option in ALI class III due to its permanent tissue damage or gangrene.[2] A cytologic thrombectomy was performed with a Fogarty balloon catheter.

Other supportive therapies for PAD patients are cilostazol, pentoxifylline, lipid-lowering drugs and antiplatelet drugs. Cilostazol is a phosphodiesterase inhibitor that inhibits platelet aggregation and exhibits a vasodilating effect, administration of cilostazol 200 mg/day for 12 to 24 weeks increases the maximum walking distance without claudication.[14] Pentoxifylline can reduce blood viscosity and increase erythrocyte flexibility. Antiplatelet effectively reduces the incidence of PAD.[14,19] This patient was also given oral therapy with cilostazol 50 mg twice a day, pentoxifylline 400 mg once a day, clopidogrel 75 mg and atorvastatin 40 mg once a day. Management of PAD in addition so interventional and pharmacological therapy also includes controlling risk factors, for instance, blood glucose, blood pressure, cholesterol, smoking habits, and weight loss. The targets for blood glucose is HbA1C < 7, triglycerides < 150, LDL < 100, HDL > 35, low homocysteine, and blood pressure < 140/90.[20,21]

The insulin is given in 0.5 – 1 unit/kgBW/day, with the targeted HbA1C < 7. In critical DM patients, the targeted blood sugar is 140 – 180 mg/dL.[22] The patient in this study was given a diet-KV of 1900 kcal/day, injection of subcutaneous Levemir 10 units at night and subcutaneous NovoRapid 6 units every 8 h before meals. The insulin dose was titrated based on fasting and postprandial blood glucose levels.

Following the surgery, observations were conducted based on patient complaints, arterial pulsation, complete blood count, coagulation function, motor and sensory function, and CT angiography to determine the progression of ALI. In certain cases, reperfusion after revascularization was inadequate, necessitating amputation. The condition of the post-thrombectomy in this study showed that the pain was decreased, palpable radial and ulnar artery pulses, no numbness, warm with the last PT/APTT was 11.1 and 32.7, respectively.

CONCLUSION

Although ALI is uncommon in the upper extremities, we discovered that it has an excellent prognosis with early revascularization, even in diabetic individuals. The medication must be carried out in conjunction with intensive DM treatment to prevent disease development.

PATIENT CONSENT

The patient provided informed consent to include the case as case report.

ACKNOWLEDGMENTS

The authors would like to thank to the patient and Dr. Soetomo Hospital.

DISCLOSURE OF CONFLICTS OF INTEREST

We do not have no conflict of interest

FUNDING

This study received no external funding.

AUTHOR CONTRIBUTION

Both authors contributed significantly to the case-report.

REFERENCES

1. Gunawansa N. Atraumatic Acute Limb Ischemia: Clinical Presentation, Classification, Assessment and Management-A Review. Int J Vasc Surg Med [Internet]. 2017;3(3):46–52. Available from: http://dx.doi.org/10.17352/2455-5452.000029
2. Olinic DM, Stanek A, Tătaru DA, Homorodean C, Olinic M. Acute Limb Ischemia: An Update on Diagnosis and Management. J Clin Med [Internet]. 2019 Aug 14;8(8):1215. Available from: https://pubmed.ncbi.nlm.nih.gov/31416204
3. Mulia E, Dewi I, Julario R, Budiarto M. Percutaneous Aspiration Thrombectomy in Acute Upper Limb Ischemia: A Case Report. Iranian Heart Journal. 2021;22(1):117-24.
4. Givani CL, Novida H. Critical Limb Ischemia In a Diabetes Mellitus Patient with Atrial Fibrilation. Curr Intern Med Res Pract Surabaya J [Internet]. 2020;1(2):25. Available from: http://dx.doi.org/10.20473/cimrj.v1i2.21472
5. Budiarto R. Muhammad MD, Hasan MRD. Aorto Femoral Bypass Graft in Chronic Limb Ischemia Patient. Cardiovasc Cardiometabolic J [Internet]. 2021;2(1):41. Available from: http://dx.doi.org/10.20473/ccj.v2i1.2021.41-49
6. Yang S ling, Zhu L yun, Han R, Sun L lei, Li J xia, Dou J tao. Pathophysiology of peripheral

arterial disease in diabetes mellitus. J Diabetes [Internet]. 2016;9(2):133–40. Available from: http://dx.doi.org/10.1111/1753-0407.12474
7. Zahra Z, Ramadhani CT, Mamfaluti T, Pamungkas SR, Firdausa S. Association between depression and HbA1c levels in the elderly population with type 2 diabetes mellitus during COVID-19 pandemic. Narra J [Internet]. 2022;2(1). Available from: http://dx.doi.org/10.52225/narra.v2i1.51
8. Mahmudiono T, Setyaningtyas SW, Rachmah Q, Nindya TS, Megatsari H, Indriani D, et al. Self-efficacy in physical activity and glycemic control among older adults with diabetes in Jagir Subdistrict, Surabaya, Indonesia. Heliyon [Internet]. 2021 Jul 15;7(7):e07578–e07578. Available from: https://pubmed.ncbi.nlm.nih.gov/34355086
9. Thiruvoipati T, Kielhorn CE, Armstrong EJ. Peripheral artery disease in patients with diabetes: Epidemiology, mechanisms, and outcomes. World J Diabetes [Internet]. 2015 Jul 10;6(7):961–9. Available from: https://pubmed.ncbi.nlm.nih.gov/26185603
10. Obara H, Matsubara K, Kitagawa Y. Acute Limb Ischemia. Ann Vasc Dis [Internet]. 2018 Dec 25;11(4):443–8. Available from: https://pubmed.ncbi.nlm.nih.gov/30636997
11. Baker S, Diercks D. Acute Limb Arterial Ischemia. Emerg Med [Internet]. 2018;50(3):65–71. Available from: http://dx.doi.org/10.12788/emed.2018.0083
12. Bae M, Chung SW, Lee CW, Choi J, Song S, Kim SP. Upper Limb Ischemia: Clinical Experiences of Acute and Chronic Upper Limb Ischemia in a Single Center. Korean J Thorac Cardiovasc Surg [Internet]. 2015/08/05. 2015 Aug;48(4):246–51. Available from: https://pubmed.ncbi.nlm.nih.gov/26290835
13. Fowkes FGR, Aboyans V, Fowkes FJI, McDermott MM, Sampson UKA, Criqui MH. Peripheral artery disease: epidemiology and global perspectives. Nat Rev Cardiol [Internet]. 2016;14(3):156–70. Available from: http://dx.doi.org/10.1038/nrcardio.2016.179
14. Rhee SY, Kim YS. Peripheral Arterial Disease in Patients with Type 2 Diabetes Mellitus. Diabetes Metab J [Internet]. 2015 Aug;39(4):283–90. Available from: https://pubmed.ncbi.nlm.nih.gov/26301189
15. Hardman RL, Jazaeri O, Yi J, Smith M, Gupta R. Overview of classification systems in peripheral artery disease. Semin Intervent Radiol [Internet]. 2014 Dec;31(4):378–88. Available from: https://pubmed.ncbi.nlm.nih.gov/25435665
16. Gulati A, Garcia L, Acharji S. Epidemiology of Chronic Critical Limb Ischemia [Internet]. Critical Limb Ischemia. Springer International Publishing; 2016. p. 9–14. Available from: http://dx.doi.org/10.1007/978-3-319-31991-9_2
17. Nativel M, Potier L, Alexandre L, Baillet-Blanco L, Ducasse E, Velho G, et al. Lower extremity arterial disease in patients with diabetes: a contemporary narrative review. Cardiovasc Diabetol [Internet]. 2018 Oct 23;17(1):138. Available from: https://pubmed.ncbi.nlm.nih.gov/30352589
18. Marso SP, Hiatt WR. Peripheral Arterial Disease in Patients With Diabetes. J Am Coll Cardiol [Internet]. 2006;47(5):921–9. Available from: http://dx.doi.org/10.1016/j.jacc.2005.09.065
19. Jackson MR, Clagett GP. Antithrombotic Therapy in Peripheral Arterial Occlusive Disease. Chest [Internet]. 2001;119(1):283S-299S. Available from: http://dx.doi.org/10.1378/chest.119.1_suppl.283s
20. Shrikhande GV, McKinsey JF. Diabetes and Peripheral Vascular Disease: Diagnosis and Management: Springer Science & Business Media; 2012.
21. Gerhard-Herman MD, Gornik HL, Barrett C, Barshes NR, Corriere MA, Drachman DE, et al. 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease. J Am Coll Cardiol [Internet]. 2017;69(11):e71–126. Available from: http://dx.doi.org/10.1016/j.jacc.2016.11.007
22. American Diabetes Association Professional Practice Committee. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2022. Diabetes Care. 2022;45(Suppl 1):S17-S38. doi: 10.2337/dc22-S002.