Proceedings of the International Conference of Community Health and Medical Sciences, Vol. ,1 Issue. 1, 2023, pp. 115-123

ISSN 3021-677X online
DOI: https://doi.org/10.17501/3021677X.2023.1113

A 71-YEAR-OLD MAN WITH UNSTABLE ANGINA PECTORIS AND DM TYPE 2

Rahmadhanti S[1]*, Abduh M.S[2], and Tahitoe A.D[1]

[1]*Internal Medicine Department, Faculty of Medicine, University Muhammadiyah Semarang, Indonesia*

[2]*Cardiology Department, Rumah Sakit Roemani Muhammadiyah Semarang, Indonesia*

**Salsabilarahmadhanti.unimus@gmail.com*

Abstract: Angina pectoris is chest pain caused by insufficient blood and oxygen flow to the cor. Worsening chest pain that occurs at rest or with light activity without damage to the cor muscle is called unstable angina pectoris (UAP). According to the World Health Organization, in the United States every year there are one million patients hospitalized due to UAP where 6-8% of them have non-fatal infarction attacks or die within 1 year after the diagnosis is made. This case report describes a 71-year-old man who came to the emergency room with complaints of shortness of breath for the past 3 days and worsened when he was admitted to the hospital in the morning. Shortness of breath occurs when the patient is doing activities. Shortness of breath accompanied by pain like being crushed in the left chest and does not spread. Complaints accompanied by nausea and cold sweat. Physical examination found VAS 5 pain, percussion of the cor revealed cardiomegaly, auscultation of the chest revealed fine crackles at the bases of both lungs. Investigations revealed a random blood sugar test of 340 $mgdL^{-1}$, the EKG found T-wave inversion in leads V1-V5, while laboratory tests showed Troponin T of 0.21 $ngmL^{-1}$. Chest X-ray examination showed cardiomegaly and pulmonary edema. Echocardiographic examination revealed segmental hypokinetic impressions, left ventricular and right ventricular systolic function were good with an ejection fraction of 59.5%. Treatment of the patient in this case was given nasal cannula oxygen 3 liters per minute, Fondaparinux injection 2.5 mg once a day subcutaneously for five days, Furosemide injection 20 mg every 12 hours, Glimepiride 2 mg once a day, Metformin 500 mg every 12 hours, Ramipril 5 mg once a day, Bisoprolol 2.5 mg once a day, Clopidogrel 300 mg continued 75 mg once a day, Aspirin 160 mg continued 80mg once a day, Atorvastatin 20 mg once a day, and Nitrokaf retard 2.5 mg every 12 hours.

Keywords: unstable angina pectoris, DM type 2

Introduction

ACS (Acute Coronary Syndrome) is a collection of symptoms caused by MI (Myocardial Infarction) (Goyal Amandeep, 2022). The pathophysiology of ACS begins with the formation of atherosclerosis in the coronary arteries of the cor which provides food for cor cells. This event results in vasoconstriction and the formation of thrombus in the cor's blood vessels, thereby disrupting perfusion and ischemia and necrosis can occur (PERKI, 2015). Myocardial ischemia or necrosis that occurs causes manifestations such as chest pain, changes in myocardial electricity, and cardiac muscle

*Corresponding Author's Email: Salsabilarahmadhanti.unimus@gmail.com

secretion. There are 2 features of ACS namely Unstable Angina (UA) and Acute Myocardial Infraction (AMI) consisting of Non-ST-segment Elevation Myocardial Infarction (NSTEMI) and ST-Segment Elevation Myocardial Infarction (STEMI) (Departemen Kesehatan RI, 2020).

According to WHO, in the United States every year there are 1 million patients hospitalized due to UAP (Unstable Angina Pectoris) where 6-8% of them have infarction attacks that are not fatal or die within 1 year after the diagnosis is made. Meanwhile in Indonesia, the prevalence of cor disease in Indonesia is 1.5% with the highest prevalence in the province of North Kalimantan, which is 2.2% (Kemenkes, 2018).

The risk factors for ACS are divided into 2, namely unmodified risk factors, which include: old age, family history, male gender. While the modified risk factors (modified risk factors), which include: smoking, DM (Diabetes Mellitus), dyslipidemia, obesity, and so on (Singh Anumeha, 2022). In recent years, many studies have linked hyperglycemia with mortality in ACS patients, including AMI patients. (Acute Myocardial Infarction). The mortality rate in AMI patients with DM accompanied by hyperglycemia reaches 30%, while ACS patients without hyperglycemia is 9%. Until now, hyperglycemia is still a topic that is often associated with ACS in the world, because high blood glucose levels in the cardiovascular system will reduce left ventricular function, which then stroke volume will decrease, recurrent mitral valve regurgitation, disturbances in diastolic filling time to increase the number risk for arrhythmias and thrombosis.

AMI is established if at least two of three criteria are met, any of the following, such as: specific chest pain (angina pectoris); altered electrocardiographic (ECG) patterns and increased cardiac biomarkers. Myocardial biomarker tests have an important role in diagnosing ACS, especially in patients with nonspecific symptoms and ECG. Troponin I is the most recommended biomarker because of its better sensitivity and specificity compared to other biomarkers (Chiha, 2016).

Case Report

Mr. D, 71 years old, came to the emergency room at Roemani Hospital with complaints of shortness of breath. The shortness of breath that has arisen since 3 days before entering the hospital and starting in the morning. Initially, complaints were felt when the patient was walking then felt shortness of breath. Shortness that is felt accompanied by pain like being stabbed on the left but does not spread to the shoulder or back. Shortness that is felt intermittently to disturbing activities. Complaints worsen when the patient performs activities. There are no mitigating factors for complaints. There are other complaints such as cold sweats, headaches, and nausea, which is recognized when complaints of shortness of breath recur.

The patient's past medical history, namely diabetes mellitus, was recognized as controlled by taking drugs in the form of metformin, glimepiride, pioglitazone since early 2023 and hypertension was recognized as controlled by consuming Ramipril since early 2023. Family disease history, such as cor disease, hypertension was recognized as belonging to the patient's biological mother, DM denied, and asthma denied.

Personal and socio-economic history, when he was a teenager the patient admitted that he liked to eat sweet and high-sugar foods. In addition, the patient also said that he rarely exercised. History of having pets, smoking, and contact with covid patients was denied.

Physical examination of the patient's general condition described the patient as having shortness of breath, compos mentis awareness with a GCS score of E4V5M6. Obtained blood pressure 116/76 mmHg, pulse 86x/minute, RR 26x/minute, temperature 36.7◦C, 98% oxygen saturation, and VAS pain scale (Visual Analague Score) 5/10, the impression is there is tachycardia. Examination of the generalized status of anemic conjunctival (-/-), icteric sclerae (-/-), pupil reflexes +/+ (3 mm/3 mm), symmetrical neck, enlarged lymph nodes (-/-), increased JVP (jugular Vein Pressure) (-). Examination of the thorax-lungs and cor on inspection found a normal chest shape (+), retraction (-), visible ictus cordis (+). Then on palpation we found normal crepitus (-/-), tactile fremitus (+/+), palpable ictus cordis on ICS V 1-2 cm medial to the left midclavicular line, lifting strength (+). Percussion examination found sonor throughout the lung fields, and on percussion of the cor found the lower right border at ICS V linea midclavicularis dextra, lower left border at ICS VI linea axillaris anterior left, for horizontal cor waist, upper limit at ICS II linea midclavicularis left. On auscultation examination, vascular lung base sounds were found in all lung fields, additional sounds in the form of fine wet rales (+) on ICS V-VI on the right and left pulmo. On examination of the abdomen, it was found that there were no injuries, masses, scar tissue. Then, on auscultation, there were peristaltic sounds (+) 10x/minute, on palpation of the mass (-), organ enlargement (-), tenderness (-), muscular defense (-). On examination of the tympanic percussion throughout the abdominal field (+), dullness (+) on examination of the liver, and there is no ascites. On examination of the superior and inferior extremities found warm acral (+), CRT <2 seconds, edema (-).

The results of the investigation carried out at the emergency room on June 14 2023 obtained a random blood sugar test of 340 mg/dL, then the EKG found sinus rhythm results, regular HR 79x/minute, RAD, normal transition zone, T inverted at V2-V5, so that the conclusion is ischemic anterior septal myocardium. On complete blood laboratory examination, Hb 13.1 g/dL L, Leukocytes 8200/mm3, Platelets 323000/mm3, Urea 16 mg/dL, Creatinine 0.87 mg/dL. Then from the results of the clinical chemistry examination, the results showed Total Cholesterol 211 mg/dL H, Triglycerides 194 mg/dL H, HDL-Cholesterol 35 mg/dL, LDL-Cholesterol 97 mg/dL, Troponin T 0.21 ng/mL. Chest X-ray

shows the results: in Cor the apex of the cor is shifted to the laterocaudal; flattened waist of the cor, elevation of the left main bronchus; in pulmonary, the bronchovascular pattern appears to be increased with vascular blurring; and consolidation appears in both lung fields; right hemidiaphragm at the level of the 10th posterior rib; sharp left right costophrenic sinus. Impression: Cardiomegaly (LV, LA) and stage 3 pulmonary edema. Meanwhile, echocardiography showed segmental hypokinetic impression, good systolic LV + RV FS, diastolic LV dysfunction, LA dilatation, and concentric LVH, good EF : 59.5%.

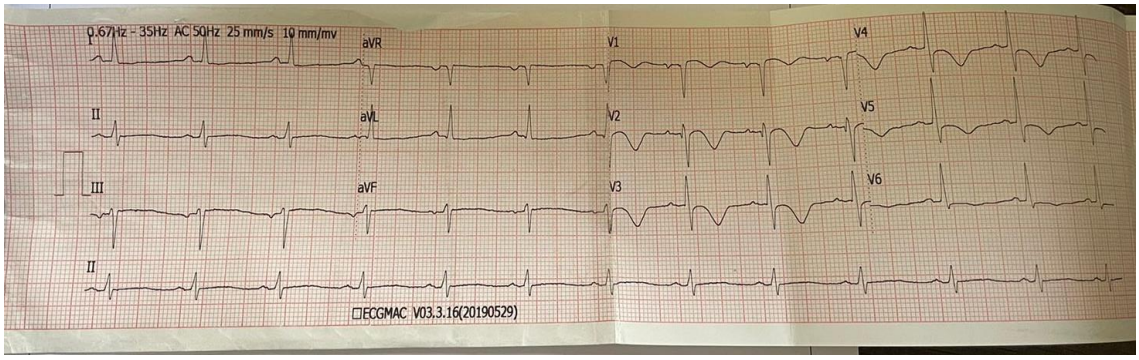

Figure 1. ECG Results

The therapy given to the patient is nasal cannula 3 liters per minute, injection NaCl 10 tpm, injection Furosemide 20mg ever 12 hours, sliding scale/ 6 hours which was then replaced with Glimepiride 2 mg once a day and Metformin 500 mg every 12 hours, Ramipril 5 mg once a day, Bisoprolol 2.5 mg once day, Clopidogrel 300 mg once a day (loading dose) followed by 75 mg once a day (maintanance), Miniaspi 160 mg (loading dose) followed by 80 mg once a day (maintanance), Atorvastatin 20 mg once a day, ISDN sublingual if needed, injection Fondaparinux 2.5 mg subcutaneous once a day, Nitrocaf retard 2.5 mg every 12 hours.

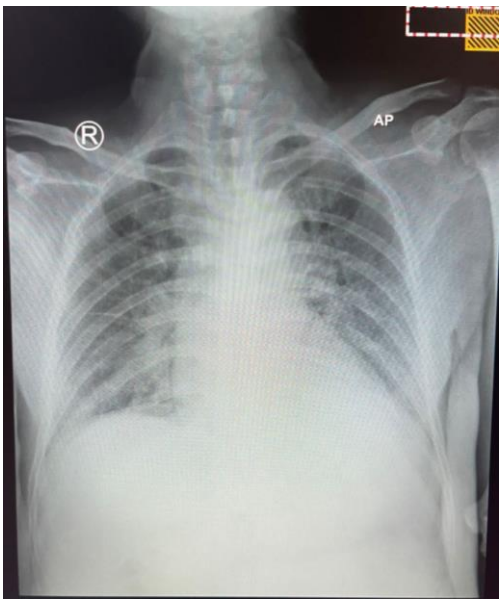

Figure 2. X-Photo Thorax

Discussion

Patients who have a suspicion of ACS should be evaluated sooner. The diagnosis of Unstable Angina Pectoris in this patient was made through anamnesis, physical examination, and supporting examinations.

In the anamnesis the patient complained of shortness of breath since 3 days of SMRS. Shortness that the patient complained of initially when the patient was walking then shortness of breath. Shortness that is felt accompanied by pain like being stabbed in the left chest but does not spread. Shortness that is felt intermittent and heavy when doing activities. In addition to tightness and chest pain, patients also complain of cold sweats, headaches, nausea when complaints of tightness recur. The patient has a history of DM and controlled high blood pressure since the beginning of 2023. There is a family history of high blood pressure, namely in the patient's biological mother. During adolescence, the patient likes to eat sweet foods or drinks.

On physical examination, blood pressure was 116/76 mmHg, HR 86x/minute, RR 26x/minute, temperature 36.7◦C, SpO2 98% with nasal cannula 3 lpm, then for VAS pain scale 5/10. On the thorax-cor percussion the lower right border is at ICS V linea midclavicularis dextra, the lower left is at ICS VI linea axillaris anterior left, for the waist the cor is flat, the upper limit is at ICS II linea midclavicularis left. On auscultation of the lungs there are coarse wet crackles on the right and left ICS V-VI.

On supporting examination, the patient's random blood sugar test reached 340 mg/dL, then the EKG found anteroseptal myocardial ischemia, while laboratory tests showed a decrease in Hb levels of 13.1 g/dL, leukocytes 8200/mm3, platelets 323000/ mm3, Urea 16 mg/dL, Creatinine 0.87 mg/dL. Then from the results of the clinical chemistry examination, the results showed Total Cholesterol 211 mg/dL H, Triglycerides 194 mg/dL H, HDL-Cholesterol 35 mg/dL, LDL-Cholesterol 97 mg/dL, Troponin T 0.21 ng/mL. On a chest X-ray, the results show: on Cor the apex of the cor is shifted to the laterocaudal; flattened waist of the cor, elevation of the left main bronchus; in pulmonary, the bronchovascular pattern appears to be increased with vascular blurring; and consolidation appears in both lung fields; right hemidiaphragm at the level of the 10th posterior rib; sharp left right costophrenic sinus. Impression: Cardiomegaly (LV, LA) and stage 3 pulmonary edema. Meanwhile, echocardiography showed segmental hypokinetic impression, good systolic LV + RV FS, diastolic LV dysfunction, LA dilatation, and concentric LVH, good EF : 59.5%.

Patients suspected of having ACS in a stable condition should be included in the indications for hospitalization and bed rest with the aim of monitoring continuous ECG rhythms and will be observed further to monitor the possibility of recurrent ischemia. High-risk patients, including those with a history of DM, will experience continuous chest discomfort, especially on the left side and/or hemodynamic instability, should be treated in the coronary care unit for further observation for 24-48 hours.

Risk factors that influence the occurrence of UAP in patients are a history of DM accompanied by hyperglycemia in the patient's blood glucose and dyslipidemia. A history of Type 2 DM in ACS patients confirms the existence of a related theory that Type 2 DM is a risk factor for ACS. Diabetes Mellitus is associated with an increased rate of cardiovascular system disease including coronary artery disease. The Framingham study explains that the incidence of cardiovascular disease in men with DM increases twofold with DM (Chiha, 2016).

In patients with diabetes mellitus, it is characterized by vascular endothelial dysfunction and accelerated development of atherosclerosis. DM impairs every homeostatic mechanism that the endothelium uses to prevent atherosclerosis. The endothelium is the synthetic site for bioactive substances including NO (Nitrit Oxide), a potent vasodilator which also inhibits the proliferation of smooth muscle, blood vessels, inhibits leukocyte adhesion and platelet adhesion and aggregation. Hyperglycemia can inhibit eNOS (endothelial Nitric Oxide Synthase) activity to reduce NO production. Endothelial dysfunction is associated with insulin resistance, hyperglycemia, and excess release of free fatty acids. These disturbances increase NO swelling by increasing PKN (Protein Kinase C) activation, increasing ROS (Reactive Oxygen Species) production and releasing endothelial NO synthase (Babes, 2022).

These factors are also responsible for the diffuse nature of atheroma and impaired cardiac function. Diffuse coronary disease can limit collateralization which can lead to an increase in infarct size. Increased production of ROS, especially superoxide anion, reduces the bioavailability of NO from the endothelium and oxidative stress inhibits the production of a compensatory vasodilator (protacyclin) (Babes, 2022).

Dyslipidemia is one of the factors that causes acute coronary syndrome because people pay less attention to eating a balanced diet and prefer to eat junk food. The majority of people who only live at home do not do physical activity so that the unhealthy food consumed does not burn properly which causes buildup in the artery walls and atherosclerosis. Atherosclerosis is a cause of acute coronary syndrome (Muhibbah, 2019).

Dyslipidemia causes damage to the vascular endothelium. If endothelial death occurs as a result of oxidation that causes an inflammatory response. Where the angiotensin II response causes impaired vasodilatation and triggers a prothrombotic effect by involving platelets and coagulation factors. This results in a protective response in which fibrofatty lesions and fibrous, atherosclerotic plaques are triggered by inflammation. Plaque that occurs can become unstable and rupture resulting in ACS (Faridah, 2016).

EKG examination is very meaningful in establishing the diagnosis of angina pectoris, especially UAP. ECG examination can also play a role in the risk stratification of UAP patients. Abnormalities on ECG examination are found in almost 50% of angina pectoris patients. The following are typical EKG abnormalities found in angina pectoris:

Stable angina pectoris: ST-T segment abnormalities consistent with mycardial ischemia

UAP or NSTEMI: recent ST segment depression suggests acute ischemia, whereas a negative T wave is indicative of ischemia or NSTEMI

Other atypical ECG features such as arrhythmias, bundle branch block, bifascicular or trifascicular block, if there are accompanying cardiovascular abnormalities (Adi, 2014).

Echocardiography examination is very meaningful in determining the diagnosis of angina pectoris because it can determine the extent of ischemia, especially when examining pain. In addition, echocardiography can also detect left ventricular function, mitral insufficiency, and regional wall motion abnormalities of the cor (Adi, 2014).

ACS therapy is divided into 3 phases, namely the acute phase, the intensive care phase (2 x 24 hours), and the usual care phase. In the acute phase, the initial therapy that must be carried out in ACS patients without ST-segment elevation is total bed rest, oxygen 2-4 L/minute, IVFD installation, drugs such as Aspilet 160 mg chewable, Clopidogrel (for ages < 75 years and not taking Clopidogrel) 300 mg or Ticagrelor 180 mg, Nitrates sublingual 5 mg, can be repeated 3 times if there are still complaints, followed by iv nitrates if complaints are persistent, cardiac monitoring, risk stratification to determine invasive strategies (TIMI Stratification Score). On the intensive care basis (2 x 24 hours) the recommended drugs for ACS therapy are Simvastatin 20-40 mg or Atorvastatin 20-40 mg or Rosuvastatin 20 mg once a day if the LDL level is above the target; Aspilet 1 x 80-160 mg; Clopidogrel 75 mg once a day or Ticagrelor 90 mg every 12 hours; Bisoprolol 5-10 mg once a day if kidney function is good, or 12.5 every 12 hours mg if kidney function decreases, the dose can be uptitrated (given if there are no contraindications); Ramipril 10 mg or Lisinorpil 10 mg once a day, Captopril 25 mg every 8 hours or if LV function decreases EF < 50% and given if there are no

contraindications; if intolerant to the ACE-I group, ARB class drugs can be given in the form of Candesartan 16 mg once a day or Valsartam 80 mg once a day; Diazepam 5 mg every 12 hours, heparin, cardiac monitoring, fasting 6 hours, cardiac diet 25-35 kcal/kgBB/24 hours, total fluid 25-35 cc/kgBB/24 hours, lipid profile examination (total cholesterol, HDL, LDL, triglycerides), and uric acid. Furthermore, in the usual care phase, drugs in the intensive care phase are continued without using heparin, risk stratification for prognostics, rehabilitation and secondary prevention (Adi, 2014).

In this case the patient was given nasal cannula therapy 3 lpm, Ramipril 5 mg once a day, Bisoprolol 2.5 mg once a day, ISDN if necessary, Atorvastatin 20 mg once a day, Miniaspi 160 mg once a day, Clopidogrel 300 mg once a day, administration of a low cardiac salt diet DM, and there is additional therapy in the form of injection of Omeprazole 40 mg every 12 hours and injection of Furosemide 20 mg every 12 hours during the acute phase. During the intensive care phase, the patient was still treated with the same drugs and replaced the novorapid sliding scale with Glimepirid 1 mg once a day and Metformin 500 mg every 12 hours, the maintenance dose was changed to Miniaspi 80 mg once a day and Clopidogrel 75 mg once a day, and there is additional therapy in the form of injection Fondaparinux 2.5 mg once a day, and Nitrocaf retard 2.5 mg every 12 hours.

Conclusion

ACS (Acute Coronary Syndrome) is a collection of symptoms caused by MI (myocardial infarction). Unstable angina (UA) is part of the ACS besides Acute Myocardial Infraction (AMI). Prompt evaluation and appropriate management of patients with suspected ACS is required. Prevention of this disease can also be done by avoiding or controlling the risk factors that influence the occurrence of UAP in patients, one of which is diabetes mellitus and dyslipidemia.

The prognosis of ACS cannot be described explicitly because there are many factors that affect the prognosis of the disease. Based on the TIMI risk score, it can be used to predict the probability of mortality in unstable angina pectoris. In this case, the patient has a mortality risk of 13%.

References

Adi RP. Pencegahan dan Penatalaksanaan Aterosklerosis. In: Alwi I, et all. (2014) Buku Ajar Ilmu Penyakit Dalam Jilid II (6th ed). Jakarta : Interna Publishing

Babes Elena E, Bustea C, Behl Tapan, Abdel-Daim M. M, et all. (2022) Acute Coronary Syndromes in Diabetic Patients, Outcome, Revascularization, and Antithrombotic Therapy [Internet]. Elsevier. Available from : https://doi.org/10.1016/j.biopha.2022.112772 (cited : April 2022)

Chiha M, Njeim M, Chedrawy EG. (2016) Diabetes and Coronary Cor Disease : a Risk Factor for The Global Epidemic. International Journal of Hypertension. Available from: http//wwwhindawi.com/journals/ijhy/2012/69/7240/ [cited Jan 10 2016]

Departemen Kesehatan RI. (2020) Pharmaceutical Care Untuk Pasien Penyakit Jantung Koroner : Fokus Syndrom Koroner Akut. Jakarta : Direktorat Jenderal Bina Pelayanan Medik

Faridah, E.N., Pangamenan, J.A. & Rampengan, S.H.. (2016) Gambaran Profil Lipid pada Penderita Sindrom Koroner Akut di RSU Prof. DR. R. D. Kandou Periode Januari - September 2015. Manado : Universitas Sam Ratulangi Manado

Goyal Amandeep, Zeltser Roman. (2022) Unstable Angina Pectoris [Internet]. Available from : https://www.ncbi.nlm.nih.gov/books/NBK442000/ [cited September 18 2022]

Kementerian Kesehatan RI. (2018) Hasil Utama Riset Kesehatan Dasar (Riskesdas) 2018. Jakarta : Kementerian Kesehatan Badan Penelitian dan Pengembangan Kesehatan

Muhibbah, etc. (2019) Karakteristik Pasien Sindrom Koroner Akut Pada Pasien Rawat Inap Ruang Tulip di RSUD Ulin Banjarmasin. Indonesian Journal for Health Sciences, 3 (1), 6-12

Perhimpunan Dokter Spesialis Kardiovaskular Indonesia (PERKI). (2015) Pedoman Tatalaksana Sindrom Koroner Akut. Jakarta: PERKI

Singh Anumeha, Museedi S. Abdulrahman, Grossman A. Shamai. (2022) Acute Coronary Syndrome [Internet]. Available from : https://www.ncbi.nlm.nih.gov/books/NBK459157/ [cited July 11 2022]