

Type: Case Study

Subject: Advanced Physiology and Pathophysiology

Subject area: Nursing

Education Level: Masters Program

Length: 7 pages

Referencing style: APA

Preferred English: AU English

Spacing Option: Double

Title: The question refers to the cases Acute Coronary Syndromes of Mr Hertz (Adult) and Mika (Paediatric).

Instructions: please kindly find the attached files of assignment details, mika (paediatric) case study, mr hertz case study and marking rubric guidelines

Important notes: references must not be less than 15 and should not be 5years older

UAC1617362223_Assignment 5 Rubric Marking Guidelines.pdf

Acute Coronary Syndromes

Name

Institutional Affiliation

Course

Instructor

Date

Acute Coronary Syndromes

Coronary cardiac disease (CHD) is a worldwide leading cause of death. More than 7,000,000 people die from CHD last year, representing 12.8% of all deaths. It is the leading cause of death before 75 in Europe, with approximately one in seven deaths in 2011, in the US, while in most developing countries, deaths from cardiovascular illness and CHD in men and women have fallen. In Britain, for example, between 1995 and 2010 there was a decline of more than half of CHD deaths per million men without diabetes. The Euro Heart Acute Myocardial Infarction Survey (AMI) reported that hospital-based mortality in 47 countries amounted to 6.2 per cent (Chang et al., 2018). Myocardial ischemia is typically the result of spontaneous atherosclerosis complications (plaque breakdown, erosion or dissection), which cause coronary thrombosis type 1 and 2 are by far the most common forms of AMI to such an extent that the prevalence of CHD in the general population can be estimated with proxy variables (Kim et al., 2020). We can now use the expression 'AMI,' which refers to AMI type 1 and AMI type 2. Highly sensitive biochemical markers like troponin (I or T) or creatine kinase MB (CK-MB) fractions can be detected by: electro-cardiographing shifts, or by techniques such as echocardiography, magnetic resonance imaging (MRI), or radionuclide imaging. Required diagnostic criteria for AMI include the modification (resurgence or fall) of cardiac biomarker values, along with at least one of these: ischaemic signs, usual electrocardiographic changes or structural or wall movement defects in the heart detected by imaging techniques. In addition, the understanding that acute coronary syndrome is not a uniform 'heart attack' type, but a continuum of pathophysiological processes, has led to the publication of separate guidelines for AMI with various therapeutic options presenting recurrent high-strength ST-segment presentations (STEMI) and non-STEMI (NSTEMI) (Libby et al., 2019).

According to a Euro Heart Survey, conducted by the European Society in Hospital, the mortality rate in unselected STEMI patient's ranges from 6% to 14%. Cardiovascular shock, heart failure, ventricular fibrillation and chronic ischemia are the most severe complications of AMI. Around 8% of AMI patients suffer from cardiovascular shock, but this remains true for 29% of the hospital admitted patients. According to the Global Acute Coronary Events Register (GRACE), cardiac insufficiency occurred in 15.6% of patients with STEMI and 15.7% of people with NSTEMI, but cardiac insufficiency occurred in just 13% of these people upon hospital admission (Chang et al., 2018). Ventricular fibrillation resulted in 1.9% of people with AMI and in 21% of people with chronic ischemia who suffered acute coronary syndrome and about half of them experienced this result in the first 24 hours. The GRACE has registered cardio insufficiency in 15.6% of those with STEMI and 15.7% of the NSTEMI sufferers; however, cardiac insufficiency occurred in only 13% of those with hospital admission. Ventricular fibrillation occurred in 1.9% of those with AMI, and 21% of those suffering from acute coronary syndrome with chronic ischemia, of which about half encountered this in the first 24 hours. Pericarditis, mitral failure, arrhythmias and conductive disorders are other potential complications of AMI (Libby et al., 2019).

A reperfusion treatment with either primary percutaneous coronary intervention (PCI), or thrombolytic treatment, has passed for less than 12 hours following symptoms starting at the height of contemporary administration of STEMI. While oxygen is biologically plausible, it can be harmful from the biological point of view (Australian Resuscitation Council, 2016). Potentially harmful methods include the paradoxical effect of oxygen on the reduction of blood flow in your heart and increase in intracoronary Doppler vascular resistance; decreased stroke volume and heart output; other adverse effects of hyper

oxidation, for example, increased vascular resistance (Schiavone et al., 2020). A systematic analysis of human research that included studies without randomization did not conclude that the use of oxygen reduces acute myocardial ischemia. In reality, there is evidence that oxygen can increase myocardial ischemia. There was also a cautionary note in another narrative study of oxygen therapy. Referencing a randomized controlled test (RCT) in 1976, the probability ratio of death (RR) for participants receiving oxygen to breathing air was 2, 89 (95% confidence interval (CI) 0, 81 to 10, 27). Despite this lack of solid proof of efficacy before our 2010 Cochrane evidentiary review was published, international guidelines strongly recommended administration of oxygen (Crea & Libby, 2017).

A comprehensive and emerging evidence base informs modern treatment of suspected and confirmed acute coronary syndrome (ACS). The main management elements of the enhanced clinical results for chest pain or ACS patients are addressed in the Australian clinical guidelines for the management of acute coronary syndrome 2016 on clinical practice. The guidelines are viewed as both evidence intensity and the probable absolute gain versus risk. The results are assessed (Chew et al., 2017). As practical points are often raised factors affecting the performance of particular therapeutics and management strategies. Including clinical evaluating and later functional and anatomical testing, these guidelines include recommendations on standardized assessment and treatment of suspected ACS patients. It also provides guidelines on: diagnosis and risk stratification for ACS; provision of acute and immediately post- fibrinolysis therapy for patients with ST segment myocardial infarction; risk stratification to inform routine and non-ST segment myocardial elevation ACS patient use and risk stratification to inform routine versus selective invasive treatment.

Some of the recommendations were more careful; for instance, the Australian Guideline did not prescribe regular application of oxygen to acute coronary syndrome (ACS),

whereas in hypoxemia it was only suggested by the Scottish Intercollegiate guide Network (SIGN) that the use of oxygen had not been clinically proven and there was no reference to animal models that showed reduced size (Kim et al., 2020). Since the 2010 Cochrane review, guidelines published have appeared to take a cautious approach that reflects the lack of evidence. For example, in 2010 the Guidelines for cardiopulmonary re-establishment and cardiovascular emergency treatment of the American Heart Association reported that "EMS providers use oxygen during initial tests of patients with suspected ACS. Since there is no collective assurance of oxygen use, several clinical studies have already been conducted or have been identified for reassessment (Chew et al., 2017). Practice should generally not be based on tradition, but on demonstrated advantage and protection. Since the 1976 study suggested a possible oxygen harm in suspected AMI, the evidence basis for existing and future guidelines on the role of oxygen therapy in heart attack patients should be systematically reviewed and updated, as appropriate, and further investigation undertaken if necessary to determine if this procedure does more harm than good (Libby et al., 2019).

Since there is no collective assurance of oxygen use, several clinical studies have already been conducted or have been identified for reassessment. Practice should generally not be based on tradition, but on demonstrated advantage and protection (Australian Resuscitation Council, 2016). Since the 1976 study suggested a possible oxygen harm in suspected AMI, the evidence basis for existing and future guidelines on the role of oxygen therapy in heart attack patients should be systematically reviewed and updated, as appropriate, and further investigation undertaken if necessary to determine if this procedure does more harm than good. If the only robust evidence indicates potentially serious harm even if the outcome is of no statistics, it confirms our view, while pathophysiological rationale is strong, that this intervention should not be used systematically (Notarangelo et al., 2017). Another problem that we have to address in evaluating the contemporary importance

and applicability of the earlier studies is that over the years the AMI concept has changed in order to represent a better understanding of the pathophysiological processes and advances in diagnostic methods like troponins with high sensitivity. In addition, it has been recognized that acute coronary syndromes reflect rather than a consistent form of "heart attack" a variety of pathophysiological processes. In particular, the different therapeutic choices now represent separate recommendations for STEMI and NSTEMI presentations (Crea & Libby, 2017).

Future research should take this range of ACS into consideration, as there may be a variable mortality rate (at least in hospital stays), but in a clinical sense a typical decision-making scenario for administration of oxygen is suspect of AMI, irrespective of final diagnosis following coronary angiography biomarker. Taking these factors into account, future oxygen studies in (suspected) AMIs should include pre-hospital participants. This presumably means that trials would recruit a large proportion of participants without an afterwards verified AMI diagnosis, given the difficulties posed by pre-hospital diagnosis. In order to minimize this issue, ongoing studies use wireless ECG transmission with cardiology analysis and additional patient discussion (Schiavone et al., 2020). In all cases, the study of true intention-to-treatment (ITT) can be done with regard to mortality and some other factors, although it cannot be feasible or suitable for all randomized patients to measure other clinical effects such as infarct size or complications. There is a special consideration to the optimum time to estimate the infarct size for clinical research using MRI (Notarangelo et al., 2017).

Oxygenation of tissue depends on the tissue being delivered optimally or properly. Growing inhaled oxygen concentrations are an efficient way to increase the blood oxygen's partial pressure and to correct hypoxemia. Simply put, oxygen therapy offers oxygen according to target saturation thresholds, in order to maintain normal or near normal levels of

oxygen saturation for acute and chronically diseased patients (in accordance with a health care professional or a hospital protocol (Notarangelo et al., 2017)).

Infarction of a myocardial (MI) in children is very uncommon. They may have various etiologies. The etiologies commonly identified include Kawasaki disease, myocarditis, hypercholesterolemia of the family, idiopathy and cardiomyopathic, collagen vascular coronary disease, substance abuse (cocaine, sniffing of cardiovascular glues), trauma, complications of congenital cardiovascular operations, genetic disorder such as progeria, pseudo-xanthoma-elastic, mucopolysaccharidosis, etc. The case of Mika indicates a potential cause of coronary- and cerebral artery thrombotic occlusion, caused MI and stroke in this child. Ischemic cardiovascular disease is a rare psychiatric disorders complication. There are few confirmed cases in the world. The 11-year old girl with a diagnosis of nephrotic steroid-reaction syndrome due to mycosis and anteroseptal MI. However, there was no proof of atherosclerotic coronary artery disease in cardiac catheter insertion. The cause of the case, similar to our patient, was the probable thrombotic occlusion of the left anterior decreasing artery. MI could occur due to hypercoagulable states - nephrotic and anti-phospholipid disease syndrome (Hughes' syndrome), coronary artery spasm and coronary embolization and myocardial bridging in children with an angiographical 'usual' coronary artery. Infants are often rarely affected by accelerating atherosclerosis, sudden dissections and aneurysms, ectasia and anomalous origins of the coronary artery. No controlled tests are available in this age group to direct early MI care. In conclusion, MI can result in various causes in children needing different approaches to management. A conventional therapy for acute myocardial infarction (AMI) patients has become morphine, oxygen, nitrates, antiplatelets (MONA). Oxygen is a drug that saves lives. Oxygen seems to have become a clinician's knee-jerk reflex response for patients with anticipated clinical emergencies. The myocardial infusion has been compromised by AMI and the myocardium

hypoxia event arises. The oxygen supplied by ischemic myocardial tissue improves the oxygenation and reduces ischemic pain in such situations seems quite logical and biologically feasible. This implies that in Milka's case, oxygen should be supplied up to the required saturation level to prevent further complications.

In acute myocardial infarction, adults with a diagnosed acute coronary syndrome should be evaluated based on the general proposition of myocardial infarction criteria. In Herz's case, cardiac care providers should maintain the universal concept of myocardial infarction standards would be used to determine the presence of an acute myocardial infarction for adults with a suspected acute coronary syndrome. Health practitioners should be aware of the overall concept of myocardial infarction and may determine the presence of acute myocardial infarction on the basis of the Universal Definition criteria, in adults with a suspected acute coronary syndrome (Australian Resuscitation Council, 2016). In order to diagnose the acute Myocardial Infarction of adults suspected of acute coronary syndrome, health practitioners should provide service with personnel with experience to use the criteria under the universal concept of myocardial Infarction. In Herz's case saturation should be removed and the patient should continue being supplied with oxygen. Although there are potential physiological adversities, almost 90 percent of presumed ACS patients continue receiving supplementary oxygen. This has been founded on the belief that supplemental oxygen could increase oxygen delivery to myocardial ischemic and therefore decrease myocardial injury and is endorsed in laboratory experiments, older clinical studies, evident benefit of hyperbaric oxygen, and intracoronary aqueous oxygen clinical testing.

In healthy patients with satisfactory levels of oxygen saturation, oxygen should be decreased or discontinued. The medical emergency is hypoxemia or hypoxia and should be treated immediately. If oxygen treatment is not initiated, the patient can be badly damaged.

Oxygen therapy is essential to ensure that the saturation rate is monitored and oxygen supplied in line with target saturating rates (Crea & Libby, 2017). While all medicines administered in the hospital need a prescription, oxygen therapy may be initiated in emergencies without a doctor's order. Most hospitals will have a procedure in place to allow providers of health care to use oxygen in emergencies. The oxygen administration provider monitors the patient's answer and maintains the levels of oxygen saturation within the specified limit. Acute hypoxemia associated with pneumonia, shock, asthma, cardiac disorder, lung embolus, myocardial infarction that causes hypoxemia, postoperative conditions, pneumothorax, and haemoglobin defects include the most common reasons for initiating oxygen treatment. The most common reasons are hypothesis. Oxygen treatment has no potential side effects if there are therapeutic signs (Schiavone et al., 2020).

References

- Australian Resuscitation Council. (2016, January). *ANZCOR Guideline 14.2 – Acute Coronary Syndromes: Initial Medical Therapy*. New Zealand Resuscitation Council. <https://www.nzrc.org.nz/assets/Guidelines/ACS/ANZCOR-Guideline-14-2-Jan16.pdf>
- Chew, D. P., Scott, I. A., Cullen, L., French, J. K., Briffa, T. G., Tideman, P. A., ... & Aylward, P. E. (2017). Corrigendum to 'National Heart Foundation of Australia & Cardiac Society of Australia and New Zealand: Australian Clinical Guidelines for the Management of Acute Coronary Syndromes 2016' *Heart Lung and Circulation* volume 25,(2016) 898-952. *Heart, Lung and Circulation*, 26(10), 1117.
- Boles, U., Rakhit, R., Shiu, M. F., Patel, K., & Henein, M. (2013). Coronary artery ectasia as a culprit for acute myocardial infarction: review of pathophysiology and management. *Anadolu Kardiyol Derg*, 13(7), 695-701.
- Budzianowski, J., Pieszko, K., Burchardt, P., Rzeźniczak, J., & Hiczekiewicz, J. (2017). The role of hematological indices in patients with acute coronary syndrome. *Disease Markers*, 2017.
- Chang, H. J., Lin, F. Y., Lee, S. E., Andreini, D., Bax, J., Cademartiri, F., ... & Min, J. K. (2018). Coronary atherosclerotic precursors of acute coronary syndromes. *Journal of the American College of Cardiology*, 71(22), 2511-2522.
- Crea, F., & Libby, P. (2017). Acute coronary syndromes: the way forward from mechanisms to precision treatment. *Circulation*, 136(12), 1155-1166.

Di Fusco, S. A., Rossini, R., Flori, M., Pollarolo, L., Ingianni, N., D'Aquino, M. M. C., ... & Colivicchi, F. (2021). Pathophysiology and management of recreational drug-related acute coronary syndrome: ANMCO position statement. *Journal of Cardiovascular Medicine*, 22(2), 79-89.

Helwani, M. A., Amin, A., Lavigne, P., Rao, S., Oesterreich, S., Samaha, E., ... & Nagele, P. (2018). Etiology of acute coronary syndrome after noncardiac surgery. *Anesthesiology*, 128(6), 1084-1091.

Kaski, J. C. (2004). Pathophysiology and management of patients with chest pain and normal coronary arteriograms (cardiac syndrome X). *Circulation*, 109(5), 568-572.

Kim, J. M., Stewart, R., Kang, H. J., Kim, S. Y., Kim, J. W., Lee, H. J., ... & Yoon, J. S. (2020). Long-term cardiac outcomes of depression screening, diagnosis and treatment in patients with acute coronary syndrome: the DEPACS study. *Psychological medicine*, 1-11.

Kim, J. M., Stewart, R., Lee, Y. S., Lee, H. J., Kim, M. C., Kim, J. W., ... & Yoon, J. S. (2018). Effect of escitalopram vs placebo treatment for depression on long-term cardiac outcomes in patients with acute coronary syndrome: a randomized clinical trial. *Jama*, 320(4), 350-357.

Libby, P., Pasterkamp, G., Crea, F., & Jang, I. K. (2019). Reassessing the mechanisms of acute coronary syndromes: the “vulnerable plaque” and superficial erosion. *Circulation research*, 124(1), 150-160.

Nieuwsma, J. A., Williams Jr, J. W., Namdari, N., Washam, J. B., Raitz, G., Blumenthal, J. A., ... & Sanders, G. D. (2017). Diagnostic accuracy of screening tests and treatment

for post-acute coronary syndrome depression: a systematic review. *Annals of internal medicine*, 167(10), 725-735.

Notarangelo, F. M., Maglietta, G., Bevilacqua, P., Cereda, M., Merlini, P. A., Villani, G. Q., ... & Ardissino, D. (2018). Pharmacogenomic approach to selecting antiplatelet therapy in patients with acute coronary syndromes: the PHARMCLO trial. *Journal of the American College of Cardiology*, 71(17), 1869-1877.

Palur Ramakrishnan, A. V. K., Varghese, T. P., Vanapalli, S., Nair, N. K., & Mingate, M. D. (2017). Platelet activating factor: A potential biomarker in acute coronary syndrome?. *Cardiovascular therapeutics*, 35(1), 64-70.

Schiavone, M., Gobbi, C., Biondi-Zoccai, G., D'ascenzo, F., Palazzuoli, A., Gasperetti, A., ... & Forleo, G. B. (2020). Acute coronary syndromes and Covid-19: exploring the uncertainties. *Journal of clinical medicine*, 9(6), 1683.

Wadei, H. M., Mai, M. L., Ahsan, N., & Gonwa, T. A. (2006). Hepatorenal syndrome: pathophysiology and management. *Clinical Journal of the American Society of Nephrology*, 1(5), 1066-1079.

