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PENGANTAR EDITOR

Salam sehat,

Tiada ungkapan kata yang patut diucapkan selain puja dan puji syukur kehadirat Allah SWT karena berkat limpahan rahmat dan taufik-Nya maka jurnal Jambura *Medical&Health Science* dapat diterbitkan. Jurnal yang diterbitkan oleh Fakultas Kedokteran Universitas Negeri Gorontalo ini merupakan jurnal penelitian yang berkaitan dengan isu di bidang kedokteran dan ilmu kesehatan.

Dalam jurnal edisi perdana ini kami telah menerima 5 artikel yang terdiri atas 2 artikel penelitian, 2 laporan kasus dan 1 tinjauan pustaka. Semua artikel yang diterbitkan di jurnal ini telah diperiksa oleh tim reviewer jurnal. Kami sangat berharap kedepan akan semakin banyak akademisi dan peneliti di bidang kedokteran dan kesehatan yang mempercayakan tulisannya untuk diterbitkan di jurnal ini.

Kami mengucapkan terima kasih kepada dewan editor dan tim reviewer yang telah bersinergi dan berkolaborasi dalam penerbitan jurnal Volume 1 Nomor 1 Februari 2022 ini. Ucapan terima kasih dan penghargaan kami sampaikan kepada pimpinan Fakultas Kedokteran Universitas Negeri Gorontalo atas bimbingan, dorongan dan dukungan sehingga jurnal edisi perdana ini dapat diterbitkan pada momentum dies natalis ke-3 FK UNG.

Kami sangat mengharapkan saran dan masukan dari para pemangku kepentingan untuk perbaikan pelayanan dan penerbitan jurnal di waktu mendatang. Semoga kehadiran jurnal ini akan berkontribusi dalam pengembangan khazanah keilmuan di bidang kedokteran dan kesehatan serta dapat ikut meningkatkan kuantitas dan kualitas publikasi penelitian di Indonesia. Viva Medika.

Gorontalo, 1 Maret 2022

Dr.dr. Muhammad Isman Jusuf,Sp.N

Chief Editor

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TABLE OF CONTENT

Jambura Medical and Health Science Journal, Vol.1 No.1 (Februrary 2022)
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REVIEW ARTICLE **Open Access**

Anticipation and Management of Coronary Heart Disease for Hajj Pilgrims

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ABSTRACT

Background: The increasing number of Indonesian pilgrims yearly makes Indonesia the most significant country that sends pilgrims worldwide. But unfortunately, this is not accompanied by optimal health quality. One of the reasons is that most of these pilgrims are elderly and sometimes have suffered from degenerative diseases since they were in Indonesia.
Contents: Coronary heart disease is the leading cause of death in developed and developing countries. The pathophysiology of coronary heart disease is closely related to lifestyle and a combination of risk factors such as hypertension, dyslipidemia, smoking, diabetes mellitus, age and family history.
Conclusion: Early intervention on coronary heart disease's causes and precipitating factors can reduce morbidity and mortality, especially for pilgrims. For this reason, it is necessary to carry out good, and continuous collaboration between health workers and the pilgrims not only during the implementation of the pilgrimage in the holy land but has started when the pilgrims register as prospective pilgrims.

Keywords: Coronary heart disease, hajj management

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INTRODUCTION

The high interest of the Muslim community in Indonesia to perform the pilgrimage can be seen from the increasingly long queues of prospective pilgrims in almost every province. This phenomenon makes Indonesia the country with the most significant number of pilgrims globally. But unfortunately, this large number is not accompanied by the optimal health quality of the pilgrims themselves. One of the reasons is that most of these pilgrims are elderly and sometimes have been sick since they were in the country.

As we all understand, the pilgrimage requires optimal stamina and conditions; this is in line with the conditions for Hajj, namely istito'ah, which means physically capable.[1] This condition is necessary not only during the process of performing the pilgrimage itself but sometimes also conditions in other places. The field requires the congregation to walk for kilometres, for example, for those who get a hut far from the Grand Mosque, not to mention facing quite extreme weather conditions. This situation causes an increase in the physical and mental condition of the congregation, which indirectly increases the morbidity and mortality rates. Of course, there have been systematic and continuous efforts from the government, in this case, the ministry of health, to solve this problem, but on the other hand, regulations that tend to be less stringent from the government make the efforts made not optimal. One of the vital weaknesses of this regulation is that there is no age restriction and screening in the field tends to be inadequate and consistent.

Coronary Heart Disease (CHD) is the most common cardiovascular disease condition. Coronary heart disease is a general term used for all disorders involving obstruction of blood through the coronary arteries. This disease is also known as coronary artery disease (CAD). The process of atherosclerosis generally causes coronary heart disease. Where atherosclerosis is a process in which there is a thickening and hardening of a large or medium artery, such as the coronary, basilar, aortic, and iliac arteries, where lesions in these arteries result in impaired blood flow to primary tissues and organs such as the brain, heart and kidney which is manifested as coronary heart disease, stroke and peripheral artery disease.[2]

Ischemic heart disease (IHD) is an imbalance between oxygen supply and demand in the myocardium, which causes myocardial hypoxia and accumulation of metabolic waste caused by atherosclerotic disease in the myocardium. Coronary arteries. Angina pectoris is still the most common manifestation of ischemic heart disease, which means "a choking feeling in the chest". Although other conditions can cause similar discomfort, angina pectoris refers explicitly to an uncomfortable sensation in the chest and surrounding structures that

results from an imbalance between myocardial oxygen supply and demand..[2]

Stable ischemic heart disease (SIHD) is defined as angina symptoms that can be managed and do not progress progressively. In addition, there was also no history of previous infarction or procedural interventions such as stent placement (Percutaneous Coronary Intervention, PCI), coronary bypass surgery (Coronary Artery Bypass Graft, CABG) or significant signs of ongoing cardiac necrosis. SIHD is distinct from acute coronary syndrome (ACS), which includes unstable angina, ST-elevation myocardial infarction, and non-ST-elevation myocardial infarction. ACS is characterized by the presence of myocardial infarction, i.e. areas of myocardial necrosis caused by the prolonged cessation of blood supply; It most commonly results from an acute thrombus at the site of coronary atherosclerotic stenosis. This event may be the first clinical manifestation of ischemic heart disease, or there may be a history of angina pectoris before.[2]

Although ACS manifests the same general disease process as SIHD, risk stratification, diagnostic testing, and medical and procedural management usually differ significantly between the two conditions. In some circumstances, patients with low-risk unstable angina may be managed similarly to patients with SIHD. This article aims to summarize efforts to identify risk factors and manage all spectrums of coronary heart disease for pilgrims.

STABLE ISCHEMIC HEART DISEASE

Stable ischemic heart disease or chronic stable angina manifests as a predictable pattern of transient chest discomfort during activity or emotional stress. It is usually caused by an obstructive atheromatous plaque fixed in one or more coronary arteries. The pattern of symptoms is usually related to the degree of stenosis. Briefly, atherosclerotic stenosis narrows the luminal diameter of the coronary arteries by more than about 70%, followed by the reduction in flow capacity may be sufficient to meet the low oxygen demand of the heart at rest but not sufficient to compensate for the oxygen demands of strenuous activity. During physical activity, activation of the sympathetic nervous system results in increased heart rate, blood pressure, and contractility, increasing myocardial oxygen consumption. When oxygen demand exceeds available supply, myocardial ischemia develops, often accompanied by the chest discomfort of angina pectoris. Ischemia and symptoms persist until the increased need is addressed and oxygen balance is restored.[2]

Risk Factor Assessment

Hypertension and Obesity

Accurate blood pressure measurement requires that the patient rest for 5 minutes, and

when the blood pressure is measured, both feet are on the floor, legs not crossed, back supported, and arms kept at heart level. Bodyweight should be measured by calculating the body mass index (BMI). Obesity is most often determined by a BMI over 30 kg/m^2.[2]

Dyslipidemia

Several physical findings have been associated with dyslipidemia. Skin xanthomas (and xanthelasma of the eyelids) are localized collections of lipid deposits within the skin, often associated with an underlying lipid disorder. Corneal arcus is a deposition lipid-rich material deposition in the peripheral cornea, which can be directly visualized without a slit-lamp examination. [2]

Diabetes Mellitus

Acanthosis nigricans is a pigmented hyperkeratosis of the skin that usually occurs on the surface of the neck and flexors. Skin tags, also called acrochordons, are benign pedunculated growths common on the neck, armpits, and groin and are associated with diabetes and metabolic disorders..[2]

Tobacco Abuse and Chronic Obstructive Pulmonary Disease

Continued tobacco abuse increases the risk of recurrent cardiovascular events. The patients sometimes try to hide this use of tobacco cigarettes. Therefore, there are findings showing tobacco use can be used as a basis for providing education on the importance of quitting smoking to reduce the risk of cardiovascular events.[2]

Nicotine stains on teeth, fingers or nails, tobacco odour, or premature skin wrinkling may indicate previous tobacco abuse. In addition, chronic obstructive pulmonary disease (COPD) has been reported to be a risk factor for cardiovascular death, regardless of tobacco abuse. Examination findings of prolonged expiration, wheezing, and distant breath sounds may suggest COPD as a complication of smoking..[2]

Diagnostic Evaluation

Biochemistry Test

In patients with SIHD, metabolic abnormalities that are risk factors for CAD are frequently detected. These disorders include dyslipidemia and insulin resistance. In addition, chronic kidney disease is strongly associated with the risk of atherosclerotic vascular disease. All patients with a definite diagnosis or suspicion of CAD should have total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, serum creatinine checked (leading to estimated glomerular filtration rate [eGFR]), fasting blood glucose levels, and measurement of haemoglobin (Hb) A1c.[2,3,4]

Resting Electrocariography

Normal findings on a resting ECG are found in about half of patients with SIHD, and even patients with severe CAD may have normal ECG results at rest. The most common abnormality on the ECG in patients with SIHD is nonspecific ST-T wave changes with or without abnormal Q waves. [2,3,4]

Resting Echocardiography

Assessment of global LV function is one of the most valuable aspects of echocardiography. Identifying regional wall motion abnormalities may suggest CAD, whereas other findings such as valvular stenosis or pulmonary hypertension may point to an alternative diagnosis.. [2,3,4]

Chest Radiography

Chest X-rays are generally within normal limits in patients with SIHD, especially if they have normal findings on the resting ECG and do not have MI.. [2,3,4]

Stress testing

Non-invasive stress tests can often provide helpful information for establishing the diagnosis and predicting the prognosis in patients with suspected stable angina. [2,3,4]

Computerized Tomography

Cardiac CT has made substantial progress as a non-invasive approach to imaging atherosclerosis and its consequences. In addition to being a susceptible method for detecting coronary calcifications, cardiac CT scans also provide coronary artery angiography and quantification of ventricular function.. [2,3,4]

Coronary Magnetic Resonance Imaging (CMR)

CMR can visualize arteries in three dimensions and differentiate various tissues. CMR has attracted the interest of researchers as a method for assessing the character of arterial atheroma and assessing susceptibility to plaque rupture. [2,3,4]

Penilaian Invasif dengan Arterografi Koroner

Currently, coronary arteriography is still the appropriate standard for assessing coronary artery anatomy and the severity of CAD. [2,3,4]

Medical Management

Comprehensive management of SIHD has five aspects as indicated in Figure 1: (1) identification and treatment of associated diseases that can trigger or worsen angina and ischemia; (2) reduction of coronary risk factors; (3) application of pharmacological and non-pharmacological interventions for secondary prevention; (4) pharmacological management of angina; and (5) revascularization with PCI or CABG, if indicated. These five approaches

should be considered, often together, in each patient.[2]

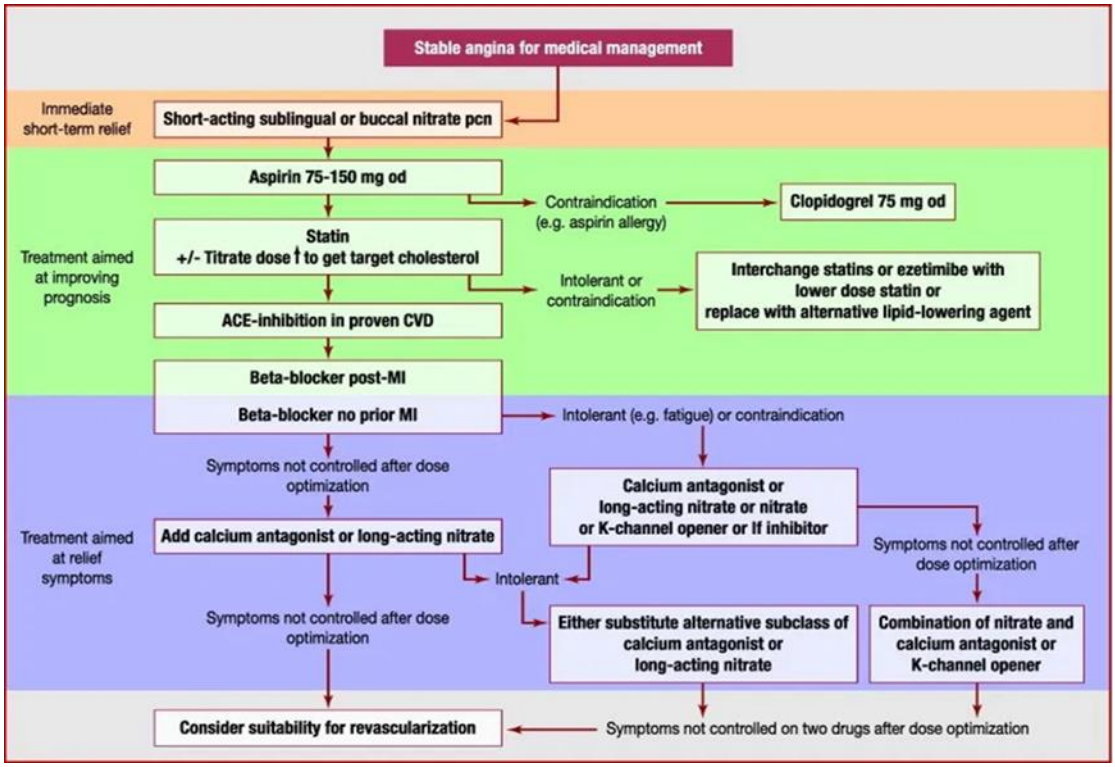

Figure 1. Medical Management of Stable Angina Pectoris.[2]

Treatment of the Related Disease

Several common medical conditions that increase myocardial O2 demand or reduce O2 supply can cause new angina pectoris or exacerbations of previously stable angina. These conditions include anaemia, latent thyrotoxicosis, fever, infection, and tachycardia. [2,3,4]

Controlling the Coronary Risk Factor

Several controlling strategy for the coronary risk factor, i.e. hypertension control, smoking cessation, management of dyslipidemia, regular exercise, and weight loss for obese patients. [2,3,4]

Pharmacotherapy for Secondary Prevention

Aspirin, low-dose anticoagulants, ACE inhibitors, beta-blockers, antioxidants and vitamins are beneficial for reducing the incidence of MACE in men and women with previous MI or stroke and post-CABG surgery. Counselling and Lifestyle Changes are also essential elements of the overall management of each patient. [2,3,4]

Pharmacology Treatment of Angina

Pharmacological prevention of angina symptoms during periods of activity classically involves using drugs that reduce myocardial oxygen demand or increase myocardial oxygen

supply in response to activity. Traditional anti-ischemic therapy includes three antianginal agents: nitrates, -blockers, and calcium channel blockers (CCBs). Currently, there are new antianginal drugs with entirely different mechanisms such as trimetazidine, ivabradine or partially such as nicorandil. [2,3,4]

Pendekatan Revaskularisasi pada Penyakit Jantung Iskemik Stabil

Each of the following considerations can be used to guide decisions regarding indications for revascularization: (1) onset of symptoms and severity of symptoms; (2) the physiologic significance of coronary lesions and other anatomic considerations; (3) the extent of myocardial ischemia and the presence of left ventricular dysfunction; and (4) other medical conditions that predispose to risk associated with percutaneous or surgical revascularization as well as conditions following revascularization. [2,3,4]

ACUTE CORONARY SYNDROME

Acute coronary syndrome (ACS) is a life-threatening condition that can occur at any time in patients with coronary artery disease. The syndrome forms a continuum from unstable angina pectoris to progress to a large acute myocardial infarction (MI), a condition of persistent necrosis of the heart muscle. A clinical diagnosis requires a condition suggestive of myocardial ischemia with some combination of myocardial necrosis detected by biochemical, electrocardiographic, or imaging modalities. A contemporary approach to treating patients with new-onset chest pain or worsening ischemic symptoms is to categorize them as an acute coronary syndrome (ACS), which includes a diagnosis of unstable angina (UAP), non-ST elevation MI (NSTEMI), and ST-segment elevation MI (STEMI). Despite advances in diagnosis and management, STEMI remains a significant public health problem in the industrialized world and is increasing in developing countries.[2,5]

Management

Successful management of ACS requires prompt initiation of therapy to limit myocardial damage and minimize complications as indicated in Figure 2. Therapy should be aimed at treating the intracoronary thrombus that triggers acute coronary syndrome and addressing the ischemic problem by administering anti-ischemic therapy to restore the balance between myocardial oxygen supply and demand. Although certain therapeutic aspects are common to all ACS, there are essential differences in patients with ST-segment elevation (STEMI) compared with patients without ST-segment elevation (UA and NSTEMI). Patients with STEMI usually have complete occlusion of the coronary arteries and therefore require rapid optimal reperfusion therapy (mechanical or pharmacological), whereas patients without ST elevation generally do not. [2,5]

Standard hospital procedures for any patient with ACS include close observation in the intensive care unit, where continuous ECG or arrhythmia monitoring is performed. The patient is initially treated in bed to minimize myocardial oxygen demand, and if hypoxaemia develops, additional oxygen may be provided (by mask or cannula) to increase oxygen supply. Analgesics, such as morphine, may be given to reduce chest pain and associated anxiety. [2,5]

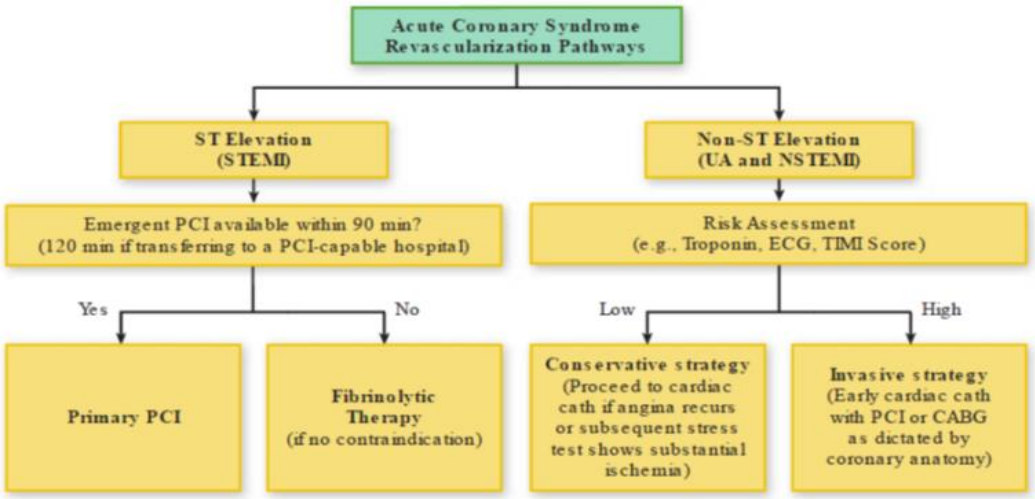

Figure 2. Flowchart for ACS Management.[2]

Acute Treatment of Unstable Angina and Non-ST-Elevation Myocardial Infarction

The main focus of treatment for UA and NSTEMI consists of an anti-ischemic treatment to restore the balance between myocardial oxygen supply and demand and antithrombotic therapy to prevent further thrombotic processes and facilitate resolution of the thrombus that nearly occludes the coronary artery. [2,5]

Anti-ischemic Treatment

The same pharmacological agents used to decrease myocardial oxygen demand in chronic stable angina are also appropriate for UA and NSTEMI but are often administered more aggressively. Beta-blockers decrease sympathetic impulses to the myocardium, thereby reducing oxygen demand and contributing to the heart's electrical stability. Nitrates help relieve angina through vasodilation, decreasing myocardial oxygen demand by reducing venous return to the heart (reducing preload and ventricular wall pressure). Nitrates can also increase coronary flow and prevent vasospasm through coronary vasodilation. In UA or NSTEMI, the sublingual route often gives nitroglycerin initially, followed by continuous intravenous infusion. [2,5]

Antithrombotic Therapy

The goal of antithrombotic therapy, including antiplatelet drugs and anticoagulants, is to prevent further development of partially occlusive intracoronary thrombi while at the same time dissolving them by endogenous mechanisms. Aspirin inhibits the synthesis of thromboxane A2, a potential mediator of platelet activation. It should be given promptly at initial presentation and continued indefinitely in patients without contraindications (e.g., allergy or underlying bleeding disorder). ADP is another necessary agonist which partially activates platelets by binding to the platelet P2Y12 receptor. This receptor's antagonist inhibiting platelet activation includes clopidogrel, prasugrel, and ticagrelor. There are also more vital antiplatelet agents used in ACS in some circumstances. Glycoprotein receptor (GP) IIb/IIIa antagonists (abciximab, eptifibatide and tirofiban) are potent antiplatelet agents that block the final pathway of platelet aggregation. [2,5]

Anticoagulant Drug

Intravenous unfractionated heparin (UFH) has long been the standard anticoagulant therapy in UA and NSTEMI. UFH also inhibits coagulation factor Xa, slows thrombin formation and further inhibits clot development. LMWH interacts with antithrombin but preferentially inhibits coagulation factor Xa. Fondaparinux is a subcutaneously administered agent that is a particular factor Xa inhibitor.[2,5] The resume of pharmacology agents in ACS is shown in Figure 3.

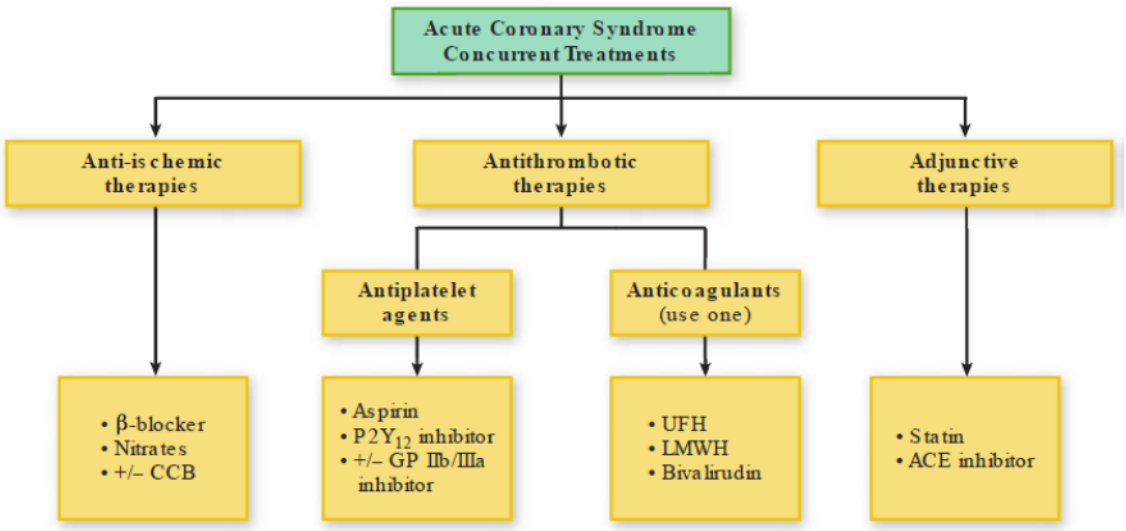

Figure 3. Pharmacology Agents in ACS.[2]

Acute Treatment of ST-Elevation Myocardial Infarction

In contrast to UA and NSTEMI, in STEMI, there is a total blockage of the coronary

arteries. Thus, to limit myocardial damage, the main focus of acute treatment is achieving rapid reperfusion of the threatened myocardium, using percutaneous coronary mechanical revascularization or fibrinolytic drugs. This approach reduces the extent of myocardial necrosis and dramatically improves survival. Effectively, this procedure should be done as quickly as possible; The sooner intervention is initiated, the greater the myocardium that can be saved. Decisions about therapy must be made within minutes of the patient's assessment, based on history and electrocardiographic findings, often before serum markers of necrosis will be elevated. In addition, as in the case of UA and NSTEMI, specific treatment should be initiated immediately to prevent further thrombosis and restore the balance between myocardial oxygen supply and demand. For example, antiplatelet therapy with aspirin can reduce mortality and re-infarction rates after STEMI. It should be given immediately at presentation (by chewing the tablet to facilitate absorption) and continued orally every day after that. Anticoagulants (e.g., intravenous UFH) are usually initiated to help maintain coronary vessel patency and are an essential adjunct to PCI and fibrinolytic regimens. Beta-blockers reduce myocardial oxygen demand and reduce the risk of recurrent ischemia, arrhythmias, and re-infarction. If there are no contraindications (e.g., asthma, hypotension, or significant bradycardia), oral beta-blockers should be given to achieve a heart rate of 50 to 60 beats/min. Intravenous beta-blocker therapy should be provided if the patient presents with elevated blood pressure, as this route of administration has been associated with an increased risk of cardiogenic shock in STEMI. Nitrate therapy, usually intravenous nitroglycerin, is used to help control ischemic pain and functions as a helpful vasodilator in patients with heart failure or severe hypertension. [2,5]

Primary Percutaneous Coronary Intervetion

The method of reperfusion therapy of choice for patients with acute STEMI is immediate cardiac catheterization and percutaneous coronary intervention in the causative lesion. This approach, called primary PCI, is a very effective method of restoring coronary perfusion. During the procedure, performed under fluoroscopy, a catheter is inserted into a peripheral artery and directed to the site of coronary occlusion. The balloon at the tip of the catheter is then inflated, compressing the thrombus and atherosclerotic plaque, and a stent is usually inserted, thereby restoring and maintaining coronary blood flow.[2,5]

Fibrinolytic Therapy

Primary PCI is the preferred reperfusion approach in acute STEMI, as it leads to more remarkable survival with lower rates of re-infarction and bleeding compared to fibrinolytic therapy. However, if PCI is unavailable or is likely to be delayed, fibrinolytic therapy is

alternative reperfusion. Fibrinolytic drugs accelerate the lysis of intracoronary occlusive thrombi in STEMI, restoring blood flow and limiting myocardial damage. Fibrinolytic agents currently in use include recombinant tissue-type plasminogen activator (alteplase, tPA), reteplase (rPA), and tenecteplase (TNK-tPA). Each drug functions by stimulating the natural fibrinolytic system, converting the inactive precursor plasminogen to the active protease plasmin, which lyses fibrin clots. Because the primary risk of thrombolysis is bleeding, contraindications to such therapy include conditions in which a necessary circulating fibrin clot may be compromised (e.g., patients with active peptic ulcer disease or an underlying bleeding disorder, patients who have recently had a stroke, or patients who are recovering). From recent surgery). [2,5]

CORONARY HEART DISEASE SCREENING IN PROSPECTIVE HAJJ CONTRIBUTORS

First Step of Health Assessment

The first stage of the health assessment is an essential examination of the hajj pilgrims, which can be carried out at the community health centre or clinic that has been designated as a health service facility for the hajj pilgrims. The first stage of health examination will produce results which will then be categorized according to the level of health risk, namely high health risk (risky) or low health risk (non-risky).[6]

At this stage, health workers at health centres and clinics can detect cardiovascular disorders, especially coronary heart disease in prospective pilgrims through history taking, physical examination and available supporting examinations, namely electrocardiography (ECG).[6]

Anamnesis

In this history, cardiovascular risk factors such as age, gender, past medical histories such as hypertension, diabetes, dyslipidemia and smoking can be identified. If the congregation is classified as an older adult and suffers from one of the diseases above, this prospective congregation should be suspected of suffering from coronary heart disease. This suspicion can increase if there are complaints related to cardiovascular, especially chest pain. Another complaint is shortness of breath during activity, long and frequent palpitations, and a history of previous fainting.[6]

Physical Examination

A physical examination can be done by checking vital signs first, especially blood pressure. If the blood pressure exceeds the standard limit, which is 140/90 mmHg, this prospective congregation should be suspected of suffering from hypertension. Next, the

height, weight and abdominal circumference can be measured. This examination can detect other risk factors such as obesity. A person is considered obese if the Body Mass Index (BMI) is> 25 kg/m2. Further physical examination was carried out as a general physical examination from head to toe. Sometimes, the physical examination may be regular in certain patients with ischemic heart disease.[6]

Additional Measurement

Suppose there are abnormalities on the ECG recording such as rhythm abnormalities such as atrial fibrillation, abnormalities in the ST segment and T waves that indicate an ischemic condition or infarction and the presence of ventricular hypertrophy. The existence of these findings will further strengthen the suspicion of cardiovascular disorders.[6]

Diagnosis and Health Risk Consideration

Suppose the history and physical and supporting examinations point to coronary heart disease. In that case, the prospective pilgrim can be diagnosed with coronary heart disease, in this case, ischemic heart disease and categorized as a high-risk pilgrim candidate.[6]

This high-risk category is under the Technical Instructions of the Minister of Health number 15 of 2016, namely: having health risk factors that can cause the inability to carry out the pillars and obligatory Hajj and threaten the safety of the pilgrims. Furthermore, the pilgrims are referred to a higher health centre such as a city or district hospital to undergo a second stage of health examination. [6]

Second Step of Health Assessment

The second stage of a health examination is an examination that must be followed by all prospective pilgrims at least 6 months before departure. Pilgrims who have attended the first stage of examination while waiting for the second stage of examination are required to undergo health coaching during the waiting period and maintain health conditions and receive integrated health guidance before the determination of istitoah status.[6]

The second examination stage is carried out by a team of specialist doctors at the city/district hospital who the health care team has appointed at the city/district level. At this stage, the criteria for istitaah will be determined for each prospective pilgrim. [6]

In particular, prospective pilgrims suspected of suffering from ischemic heart disease will be directed to the heart and blood vessel polyclinic to undergo a comprehensive examination by the relevant specialist. The examination stages are no different from the first stage examination, including anamnesis, physical examination and supporting examinations. At this stage, the specialist confirms the findings or previous data related to the cardiovascular condition of the prospective congregation.

Anamnesis

Evaluation of complaints, especially chest pain, needed to ischemic chest pain or not, with a more in-depth and directed history to rule out non-ischemic causes of chest pain. In addition, it was also investigated whether there were other complaints related to other cardiovascular diseases such as heart failure and arrhythmias. The history also explores cardiovascular risk factors that support ischemic heart disease and determines the supporting examinations that must be carried out.

Physical Examination

Physical examination at this stage is the same as in the first examination but is more focused on physical examination of the heart and peripheral blood vessels, such as xanthelasma of the eyelids associated with dyslipidemia; changes in arterial pulsation indicating circulatory abnormalities such as pulsus parvus et tardus, water hammer pulse etc.; increase in jugular venous pressure; abnormalities on cardiac examination: such as the presence of irregular or additional heart sounds and the presence of murmurs and abnormalities on pulmonary examination: such as additional sounds such as rhales or crepitations; abnormalities on abdominal examination such as hepatomegaly and ascites; oedema of the legs or feet.

Additional Measurement

At this stage, the supporting examinations that can be carried out are adjusted to the facilities available at the referral hospital. The standard is a complete laboratory examination to detect risk factors such as DM, dyslipidemia, and other chronic diseases. Another examination is a chest X-ray to detect abnormalities in the heart and lungs. The next check that can be done is a treadmill test. This examination is performed to confirm the suspicion of ischemic heart disease. Suppose from the above examination results there is a strong suspicion of ischemic heart disease. In that case, if there are more complete facilities, it can be continued with a coronary CT scan and coronary angiography.

Diagnosis and Recommendations

After it is proven that the prospective pilgrim suffers from ischemic heart disease based on the history, physical examination and support, the following action is to recommend pharmacological and non-pharmacological therapy. Giving this therapy is the treatment of comorbidities such as hypertension, diabetes, dyslipidemia with related drugs. Another therapy is drugs for secondary prevention of cardiovascular complications such as acute coronary syndromes and stroke, namely salicylic acid and statins. Therapy that is no less important is anti-ischemic drugs to reduce the symptoms of angina pectoris, such as nitrates,

beta-blockers and calcium channel blockers. Non-pharmacological therapy can be given in the form of recommendations for healthy lifestyles such as quitting smoking, avoiding food patterns that tend to be high in carbohydrates and fats, losing weight, and exercising regularly at least 3 times a week.[6]

Determination of the Health Istitaah of the Hajj Pilgrims

Based on the established diagnosis and taking into account the severity of the illness and the estimated prognosis at the time of the pilgrimage, the determination of istitaah can be enforced with the following criteria:

1. Meet the requirements of Hajj health istitaah
   Valid for prospective pilgrims who can be independent and suffer from mild ischemic heart disease and can be controlled by the therapy given
2. Meet the requirements of Hajj health istitaah with assistance
   Valid for prospective pilgrims who require minimal assistance from others and suffer from mild ischemic heart disease and can be controlled by the therapy given
3. Does not meet the requirements for temporary Hajj health
   Valid for prospective pilgrims who suffer from severe ischemic heart disease but can still perform revascularization measures such as coronary stent placement (Percutaneous Coronary Intervention, PCI or heart bypass surgery (Coroner artery Bypass Graft, CABG).
4. Does not meet the requirements for Hajj health
   Applies to prospective pilgrims who suffer from severe ischemic heart disease that does not improve with revascularization measures or are accompanied by other complications such as heart failure, kidney failure, stroke, peripheral arterial disease, so this pilgrimage is an absolute contraindication.

Third Step of Health Assessment

This stage of health assessment is carried out when prospective pilgrims are at embarkation. This assessment is carried out by an aviation health doctor appointed at each embarkation. The evaluation results are based on the current condition of the prospective pilgrims based on the recorded diagnoses. The results of the recommendations are in the form of airworthy or not airworthy. The prospective pilgrim is declared unfit to fly, especially for prospective pilgrims with ischemic heart disease who experience an acute attack episode such as acute coronary syndrome or stroke for less than 3 months. [6]

Evaluation of Pilgrims While in the Holy Land

Pilgrims who have been detected as suffering from coronary heart disease since embarkation has worn unique attributes such as bracelets which indicate that the congregation is included in the category of high risk (risti) pilgrims. In addition, it must be ensured to carry all medicines from the time of entry at embarkation, which these medicines must be sufficient for the duration of the pilgrimage from the time of departure until returning to the homeland. This policy is to prevent complications of coronary heart disease due to the depleted supply of medicines. These pilgrims must also be closely monitored by the crew of staff, especially health workers, while in the lodge and when carrying out the Umrah and Hajj processions. During the waiting period for the Hajj rituals, the congregation is trying to carry out routine worship rituals at the lodge. Unnecessary things, such as visits to historical places or markets, are strictly avoided. [6]

Other active crew members assist the group health workers in making regular visits to this risti congregation. If deemed necessary, can consult a specialist in the sector if chest pain symptoms do not decrease with rest or with the use of sub-lingual anti-ischemic drugs, or there are other complications such as infection etc. Health workers must also pay attention if pilgrims are not at high risk who complain of chest pain that leads to ischemic heart disease. This patient needs to take a history and physical examination and consult a cardiologist at the Indonesian Hajj Medicine Center (IHMC) for further examination. [6]

Suppose there is an emergency for the Risti pilgrims or other pilgrims, such as acute coronary syndrome ST-elevation myocardial infarction (ST-elevation Myocardial Infarction, STEMI) or requiring immediate revascularization or with impaired hemodynamics. In that case, referral to a Saudi Arabian hospital in coordination with specialist doctors is necessary—the heart of the BPHI and the Southeast Asian muasasah maktab officer. Before referral, initial therapy should be given, such as aspirin 2 tablets, clopidogrel 4 tablets and ISDN 5 mg sublingual. If you do not require immediate revascularization and are hemodynamically stable, you can be treated at the High Care Unit (HCU) at IHMC. The group device must be active to visit the Mecca IHMC or Saudi Arabian Hospital if congregants are cared for.[6]

At the hajj procession, all pilgrims need to be re-evaluated in collaboration with specialist doctors in the related sector and the Mecca IHMC to assess whether their health condition is appropriate to follow all hajj rituals with the group. Suppose the congregation's assessment is not feasible. In that case, the congregation can be referred to a Saudi Arabian hospital for further treatment or included in the wukuf safari team with the assistance of

health workers from the Mecca IHMC. If deemed worthy, the congregation can be included with the group with strict supervision from all group equipment, where the congregation is not allowed to do a lot of activities and specifically for rituals that require physical activities such as throwing the jumrah must be represented by other pilgrims.[6]

When performing the pilgrimage from Arafah to Mina, the congregation was tried to be in one tent with a group of equipment. If there is an emergency, the group's health officers must provide emergency assistance on the spot and contact the field Hajj officers in Arafah, Muzdalifah and Mina for referrals to emergency IHMC in Arafah and Mina or be referred directly to the nearest Saudi Arabian hospital in collaboration with Southeast Asian muasasah maktab officer. Furthermore, the sick congregation at the emergency BPHI will be referred to the Mecca IHMC.

The post-hajj ritual period is when there are many sudden deaths in boarding houses and places of worship, based on data from the Health Center of the Indonesian Ministry of Health. For this reason, the Risti congregation, especially those with ischemic heart disease, is sought to be sent home early or early tanazul with other groups without having to perform the sunnah arbain worship in Medina. While waiting for the return schedule of Risti's pilgrims, strict supervision is still being carried out, and efforts are made to rest more at the lodge. For pilgrims who are forced to be treated in a Saudi Arabian hospital when the group has to move to Medina or return to their homeland, the congregation will later be included in the list of late returning pilgrims or final tanazul. [6]

Conclusion

The series of pilgrimages from beginning to end requires optimal health stamina, so careful preparation is needed when in the homeland and the holy land. This preparation requires good cooperation between prospective pilgrims and health workers. Pilgrims with cardiovascular risk factors need a comprehensive evaluation to diagnose coronary heart disease. Congregants with non-istitaah evaluations are recommended to postpone the pilgrimage and those obliged to maintain their condition while in their homeland and while in the holy land.

The health workers of the group are obliged to carry out inherent supervision of these Risti pilgrims to prevent morbidity and mortality while in the lodge or during the Hajj procession. For this reason, it is necessary to carry out excellent and harmonious cooperation between fellow group officers health workers on duty in the sector and at IHMC and Southeast Asian muasasah maktab officers. To reduce the possibility of an emergency, the

Risti congregation can be included in programs held by IHMC, such as the Wukuf Safari and Tanazul Awal, so that it is hoped that all Risti pilgrims can maintain their health condition while in the holy land.

Conflict of Interest

Nothing to declare

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ORIGINAL ARTICLE **Open Access**

Glaucoma Prevalence in Diabetes Mellitus Patients at the Eye Policlinic, Prof. Dr. H. Aloei Saboe Gorontalo

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ABSTRACT

Introduction: Glaucoma is a disease of damage to the optic nerve that causes narrowing of the visual field and loss of visual function. One of the systemic conditions that can trigger glaucoma is diabetes mellitus. This study aims to determine the prevalence of glaucoma in diabetic patients at the Eye Polyclinic of RSUD Prof. Dr H. Aloei Saboe Gorontalo.
Methods: This research used a descriptive retrospective study design on medical records at Eye Polyclinic of RSUD Prof. Dr H. Aloei Saboe Gorontalo from January to December 2020.
Results: The results showed that from 12 cases of glaucoma in diabetes mellitus patients, there were more males, namely 8 patients (66.7%) and 4 patients (33.3%). Based on the age group, the most in the 40-60 year age group were 10 patients (84%). Based on the type of diabetes, all cases were found in type II diabetes, namely 12 patients (100%). Based on the length of time the patient had diabetes, it was found that 11 patients (92%) had diabetes >5 years.
Conclusion: In patients with diabetes mellitus, the prevalence of glaucoma was more in men who had diabetes for a long time.

Keywords: Diabetes mellitus, glaucoma

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INTRODUCTION

Glaucoma is one of the leading causes of blindness globally and is the leading cause of permanent blindness. Glaucoma is a collection of symptoms with a characteristic optic neuropathy associated with visual field loss. According to WHO, 8% of all cases of blindness worldwide are caused by glaucoma.[1,2,3] Basic Health Research (Riskesdas) in 2007 reported that the prevalence of glaucoma in Indonesia was 0.46%, where 4 to 5 people out of 1000 population Indonesia has glaucoma. Based on online hospital application data (SIRS online), the number of visits to outpatient glaucoma patients at the hospital has increased. In 2017, there were 80,548 cases of glaucoma. Based on gender, more women than men.[4]

Glaucoma can be caused by systemic disease or local disease of the eye. One of the systemic disorders that can trigger glaucoma is diabetes mellitus (DM). In DM patients, there are increased tortuosity, focal dilatation and narrowing of blood vessels, the formation of microaneurysms and choriocapillaris blood vessel disorders in the form of decreased choroidal perfusion. It has reduced blood flow results in tissue ischemia and tissue death. Decreased choroidal perfusion can be found in normal pressure glaucoma associated with functional (field of view) and morphological (damage to the nerve fibre layer) disturbances.[1, 4-6]

Diabetes mellitus is a chronic disease in a metabolic disorder characterized by blood sugar levels that exceed normal limits. Diabetes mellitus was established when fasting blood levels more than 126 mg/dL, blood glucose 2 hours after loading more than 200 mg/dL, or blood glucose at 200 mg/dL accompanied by symptoms of frequent hunger, thirst and urination in large quantities and weight loss.

The results of Riskesdas 2018 revealed that the prevalence of DM in Indonesia based on a doctor's diagnosis at the age of 15 years is 2%. This data shows an increase compared to the prevalence of DM in the population 15 years at Riskesdas 2013 of 1.5%. However, according to the results of blood sugar examinations, the prevalence of DM increased from 6.9% in 2013 to 8.5% in 2018. This data suggests that only about 25% of DM sufferers know DM. Almost all provinces showed an increase in prevalence in 2013-2018, except for the province of East Nusa Tenggara. Gorontalo is included in the province category with the highest prevalence increase of 0.9%, along with Riau, Jakarta, Banten and West Papua.[4]

METHODS

This study used a retrospective descriptive method of patient medical record data at the Eye Polyclinic at Prof. Hospital. Dr H. Aloei Saboe Gorontalo from January to December 2020. The population of this study were patients with DM who had their eyes checked at the eye polyclinic at Prof. Hospital. Dr H. Aloei Saboe Gorontalo from January to December 2020. The research variables

were glaucoma sufferers, age, gender, and type of glaucoma.

RESULTS

From the data obtained from medical records at the Eye Polyclinic at RSUD Prof. Dr H. Aloei Saboe Gorontalo from January to December 2020, there were 12 cases of glaucoma. Based on Table 1, it was found that the distribution of glaucoma cases in patients with diabetes mellitus at the Eye Polyclinic at Prof. Hospital. Dr H. Aloei Saboe Gorontalo by gender, 8 male patients (66.7%) and 4 female patients (33.3%).

Table 1. Distribution of glaucoma cases by gender in patients with diabetes mellitus at the Eye Polyclinic at Prof. Hospital. Dr. H. Aloei Saboe Gorontalo

Gender	Total (n)	Proportion (%)
Male	8	66.7
Female	4	33.3
Total	12	100

Based on Table 2, it was found that the distribution of glaucoma cases in diabetes mellitus patients at the Eye Polyclinic at Prof. Hospital. Dr H. Aloei Saboe Gorontalo based on the most age group in the 40-60 year age group, as many as 10 patients (84%), followed by the age group <40 years with 1 patient (8%) and the age group over >60 with 1 patient (8%).

Table 2. Distribution of glaucoma cases in diabetes mellitus patients by age at the Eye Polyclinic at Prof. Hospital. Dr. H. Aloei Saboe Gorontalo

Age	Total (n)	Proportion (%)
<40 years	1	8
40-60 years	10	84
>60 years	1	8
Total	12	100

Based on table 3, it was found that the distribution of glaucoma cases all occurred in type II diabetes, namely 12 patients (100%) at the Eye Polyclinic at Prof. Hospital. Dr. H. Aloei Saboe Gorontalo. In addition, based on table 4, it was found that the distribution of glaucoma cases at the Eye Polyclinic at Prof. Hospital. Dr H. Aloei Saboe Gorontalo was aware that he had suffered from diabetes mellitus for > 5 years in 11 patients (92%) and suffered < 5 years in 1 patient (8%).

Table 3. Distribution of glaucoma cases by type of diabetes mellitus at the Eye Polyclinic at Prof. Hospital. Dr. H. Aloei Saboe Gorontalo.

Type of diabetes mellitus	Total (n)	Proportion (%)
Type I	0	0
Type II	12	100
Total	12	100

Table 4. Distribution of glaucoma cases in patients with diabetes mellitus based on length of suffering diabetes at the Eye Polyclinic at Prof. Hospital. Dr. H. Aloei Saboe Gorontalo.

Length of suffering DM	Total (n)	Proportion (%)
< 5 years	1	8
>5 years	11	92
Total	12	100

Discussion

Based on the recent results, there were 12 cases of glaucoma in patients with diabetes mellitus. Diabetes mellitus is a systemic disease that can be a risk factor for glaucoma. Patients with diabetes mellitus have three main complications, namely diabetic retinopathy, cataracts and glaucoma. Patients with diabetes mellitus are more at risk of developing glaucoma than individuals who do not have diabetes. Glaucoma is known as a cause of irreversible blindness if it is not treated promptly and appropriately; therefore, the detection of glaucoma cases is very important so that glaucoma sufferers are identified and treated before blindness occurs.

In this study, 12 cases of glaucoma were found in diabetic patients. The number of affected men was more than women. In a study conducted in RSUP Dr.M. Djamil Padang, it was found that more men suffer from glaucoma in diabetes because men tend to be less passive than women in the control and treatment of diabetes, thereby increasing the risk for glaucoma.[7]

In this study, based on the patient's age, it was found that the age group of 40-60 years had the most glaucoma in diabetes mellitus, namely 10 patients (84%) were 1 patient 8) was <40 years old and 1 patient (8%) was >60 years old. Risk factors for triggering and exacerbating glaucoma include age from 40 years, family history of glaucoma, myopia, hypermetropia, diabetes mellitus, hypertension, vasospasm, migraine, and corticosteroids central retinal vein obstruction, trauma, inflammation and eye surgery. The age group described that the flow of aqueous humour would be reduced by 30% during life and cause an increase in IOP. People aged >70 years are 3-8 times more

likely to develop primary open-angle glaucoma, while the age group of 55 to 65 is dominated by primary angle-closure glaucoma.[8]

Based on the type of diabetes mellitus in this study, all cases of glaucoma were found in type II diabetes. Type I and type II diabetes are at risk for diabetic retinopathy, cataracts, or glaucoma. Diabetes causes neurodegeneration where it will cause an increase in oxidative stress on eye cells which results in impaired vascularization and oxygen diffusion to eye cells. These things can cause damage to glial cells and endothelial cells of the eye resulting in hypoxia, resulting in changes in anatomy and nerve function in the eye. Type II diabetes causes an earlier depletion of the RNFL than individuals without diabetes.[9]

Based on the length of time patients had diabetes who had glaucoma, 11 (92%) had diabetes >5 years and 1 patient (8%) had diabetes <5 years. Zhao studied the duration of diabetes with the relationship of high risk of glaucoma. It was found that the longer the duration of a person affected by diabetes mellitus, the longer there will be damage to the eye's glial cells and neural function of the eye which will result in a higher risk of developing glaucoma.[10]

Conclusion

Based on the results of this study, it was concluded that there were more glaucoma sufferers in the male sex, where the most age group was 40-60 years, all glaucoma cases were found in patients with type II diabetes with the most duration of diabetes > 5 years.

Conflicts of Interest

Nothing to declare

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CASE REPORT **Open Access**

Trachea and Laryngeal Rupture Induced Pneumomediastinum in Psychiatric Patients : A Case Report

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ABSTRACT

Introduction: Laryngeal rupture is a potentially life-threatening aerodigestive injury, especially in cases with slow treatment, which can occur spontaneously, iatrogenic or as a result of trauma.
Case: We present a case of pneumomediastinum due to sharp trauma causing a laryngeal rupture in a patient with psychiatric disorders. Pneumomediastinum is one of the complications that occur.
Discussion: Trauma history and clinical complaints are very helpful in establishing the diagnosis, and imaging has an important role in describing pneumomediastinum and other air leak syndromes so that early treatment can be done immediately.
Conclusion: The approach to diagnosing pneumomediastinum with imaging in psychiatric patients has an important role because of the lack of information about complaints and mechanisms of pneumomediastinum.

Keywords: laryngeal rupture, pneumomediastinum, psychiatric disorder, sharp trauma.

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Introduction

Pneumomediastinum or mediastinal emphysema is defined as the presence of air in the mediastinal space that occurs spontaneously or secondary to thoracic and iatrogenic trauma resulting from surgical, diagnostic or therapeutic procedures.[1-3] Traumatic perforation of the upper aerodigestive tract can cause significant morbidity and mortality. Clinical symptoms that arise are varied and sometimes non-specific, so pneumomediastinum is often missed at the time of diagnosis. Complaints such as chest pain, shortness of breath, sore throat, dysphagia and fever are frequently reported in patients with pneumomediastinum and emphysema or cervical subcutaneous crepitus, or a combination of the two is considered diagnostic of aerodigestive injury.[1,4,5]

Imaging has a major role in establishing pneumomediastinums, such as chest X-rays, chest ultrasonography, and chest CT scans of pneumomediastinum.[2,5,7] Management of aerodigestive trauma in patients with psychiatric disorders is a challenge because of the lack of information about complaints and trauma mechanisms that can complicate the early diagnosis of pneumomediastinum.[6-8]

Case

A 32-year-old man was admitted to the Emergency Department through a referral from a regional hospital with complaints of stab wounds to the neck experienced since 8 hours earlier. According to the family, the patient suddenly stabbed a knife into his neck, and several stab wounds appeared on the front side of the neck. The patient had received treatment at the previous hospital in suturing on several wounds. There was a disturbance in the voice but no breathing problems, difficulty swallowing, a history of fainting, and blood coming out through the nose and mouth. The patient has a history of self-talk with a chaotic flow, is difficult to understand the meaning and often hears whispers from unknown sources. The patient also had a history of smoking and using drugs for a long time.

The patient appeared conscious with normal vital signs and good orientation to the surrounding environment on physical examination. From inspection, there were several wounds with stitches on the right neck with pain status 4-6/10 with continuous intensity, the voice sounded hoarse, the patient did not appear short of breath, the patient was quite calm, but often daydreamed, eye and verbal contact were present, blunted affect, talk constantly, the content of thought is quite relevant but sometimes blocking. On palpation, crepitus was found in the area around the stab wound and also tenderness.

On laboratory examination, it was found that the leukocyte value was increased (16.000/UL). The patient underwent a chest X-ray and CT scan of the neck without contrast. A chest x-ray examination was carried out in the AP position and obtained a picture of pneumomediastinum and emphysema subcutis (Figure 1).

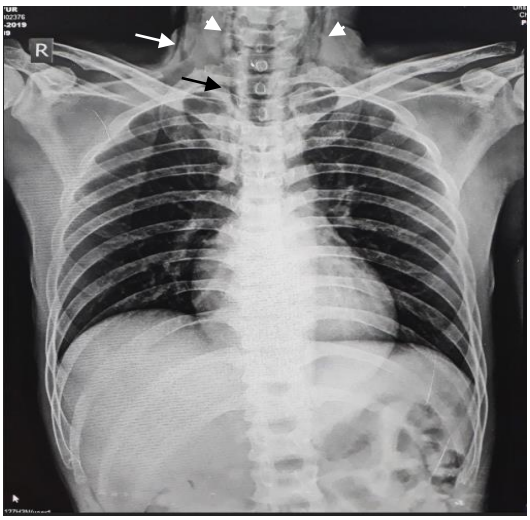


Figure 1. AP position chest radiograph showing linear lucency along the bilateral paratracheal planes (arrowheads). A lucent shadow surrounds the bilateral brachiocephalic trunk with a tubular artery sign (black arrow). And also found a lucent shadow in the Colli region impression on soft tissue (white arrow)

On CT scan of the neck without contrast, Pneumomediastinum was seen due to rupture of the trachea and larynx, skin defect of the anterior wall Colli at CV C5-C6 and right submandibular as high as CV C3 associated with lateral pharyngeal space, right submandibular fossa to retropharyngeal space, and emphysema subcutis neck region (Figure 2-4).

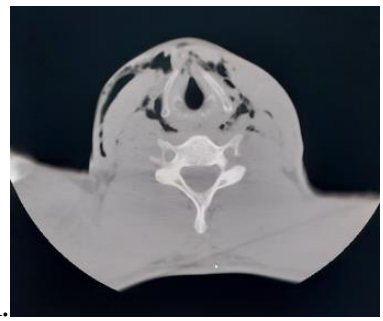
A.  B. 

Figure 2. A laryngeal discontinuity is seen in relation to the anterior mediastinal wall at the level of CV C7

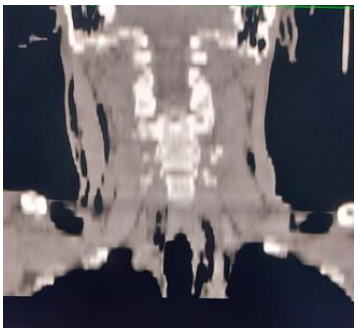
A.  B. 

Figure 3. It is also seen that the tracheal discontinuity is as high as CV T3

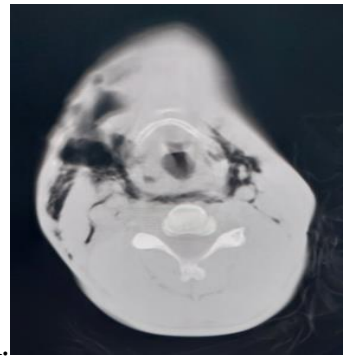
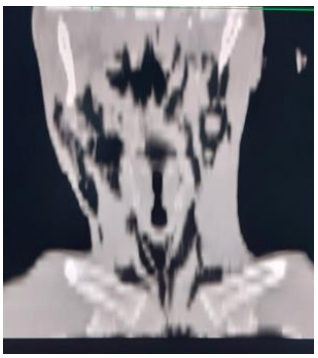
A.  B. 

Figure 4. Multiple soft tissue defects in the anterior neck and submandibular which indicate a subcutis emphysema

The patient was treated for three days by three different departments, namely the surgical department, the ENT department and the psychiatry department. The patient showed a stable general condition, and if there were a worsening, the surgical department would perform a mediastinectomy. The ENT section failed to install the NGT because the patient refused. The psychiatry department diagnosed the patient with schizophrenia with signs of depression and was treated to stabilize the patient further.

Discussion

Laryngeal rupture is a potentially life-threatening aerodigestive injury, especially in slow-treated cases that can occur spontaneously, iatrogenic or as a result of trauma. Traumatic injuries to the larynx and cervical trachea are rarely reported in all age groups because some victims die before being brought to medical services. Complications caused by aerodigestive injuries in the form of pneumomediastinum, pneumothorax, pneumopericardium, pneumoperitoneum, to subcutaneous emphysema are included in the air leak syndrome, which can be life-threatening due to severe airway obstruction. Early recognition of laryngeal damage is the key to the life-saving management of the patient.[1,6,8]

Pneumomediastinum or mediastinal emphysema is defined as the presence of air in the mediastinal space that can occur spontaneously or as a result of various processes that occur in the intrathoracic and extrathoracic. The causes of an iatrogenic pneumomediastinum include alveolar rupture, tracheobronchial branch laceration, bleb rupture, several conditions that cause increased intrapulmonary pressure, chronic lung diseases such as asthma, and violent coughing, and vomiting. At the same time, the causes of extrathoracic origin include facial trauma, laryngeal injury, tracheostomy action, from the retroperitoneum (e.g. from a diverticulum or duodenal ulcer), or the chest wall (subcutaneous emphysema around the tracheostomy drain). In our case, pneumomediastinum occurred due to sharp trauma to the larynx and trachea.[1,3,9]

Research conducted by Damore (1991) and Stack (1996) stated that pneumomediastinum is most often found in men than women due to a tendency to activities that increase the risk of barotrauma, such as diving or frequent work that holds your breath. Traumatic injuries to the cervical larynx and trachea are rare in all age groups. This condition can present with subtle symptoms and signs and may seem trivial but life-threatening. In diagnosing tracheal and laryngeal injuries, it is important to know the mechanism of injury, changes in voice, and the patient's respiratory status. And it becomes difficult to establish an early diagnosis in psychiatric patients who are less able to describe the mechanism of injury and complaints that describe the process and causes of pneumomediastinum. Other important symptoms and signs listed can be seen in Table 1.[2,9-11]

Table 1. Symptoms and signs of laryngeal or cervical tracheal injury

Pain over the anterior neck
Shortness of breath
Stridor
Inability to tolerate supine position
Inability to tolerate head extension
Hoarse, weak or absent voice
Haemoptysis
Bruising, lacerations or haematoma over the neck
Subcutaneous emphysema

Radiographic signs of pneumomediastinum depend on delineating the normal anatomic structures described by air as it leaves the mediastinum (Table 2).[2]

Table 2
Radiographic Signs of Pneumomediastinum

Subcutaneous emphysema
Thymic sail sign
Pneumoprecardium
Ring around the artery sign
Tubular artery sign
Double bronchial wall sign
Continuous diaphragm sign
Extrapleural sign
Air in the pulmonary ligament

Spinnaker sail sign

The spinnaker sail sign is a sign of the presence of air in the mediastinum seen on the chest X-ray of the neonate. This term refers to the visible appearance of the thymus being demarcated by air, the two lobes of the thymus being pushed laterally and seen as an elevated thymus resembling a "sail" image. Spinnaker sail sign (Fig. 5) is common with spontaneous anterior pneumomediastinum and usually resolves independently without specific treatment.[1,2]

a.

b.

Figure 5. a. CXR of the neonate shows Spinnaker's sail sign, the thymus is limited by air in the mediastinum, the thymus lobe is shifted laterally. Figure b. Normal neonatal thymus appearance.[1,2]

Pneumoprecardium

Pneumoprecardium is the presence of air in the anterior pericardium, namely between the sternum and the heart, which can be seen on the lateral radiograph. Spontaneous

pneumoprecardium is a rare case, especially in children. The most common cause was asthma (0.3%) and acute attack. Other causes are bronchiolitis caused by viruses, or respiratory tract irritation should also be considered.[1,2]

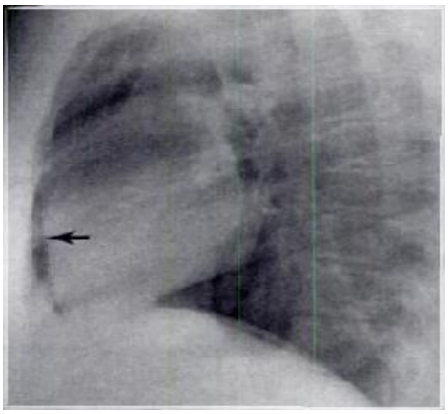


Figure 6. Pneumoprecardium in a post-tonsillectomy patient.[1,2]

Ring around the artery sign

The ring around the artery sign is an image of air surrounding the pulmonary artery or one of its main branches, which produces a ring around the pulmonary artery, especially when air surrounds the intramediastinal segment of the right pulmonary artery. Ring around the artery sign may be seen on lateral projection radiographs, seen as a lucency around or around the right pulmonary artery (Figure 7 and 8).[1,2]

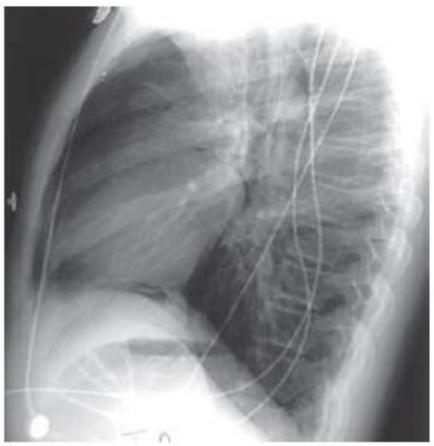


Figure 7. Lateral CXR, showing air around the right pulmonary artery. Free air is also seen in the anterior pericardium (pneumoprecardium).[1,2]

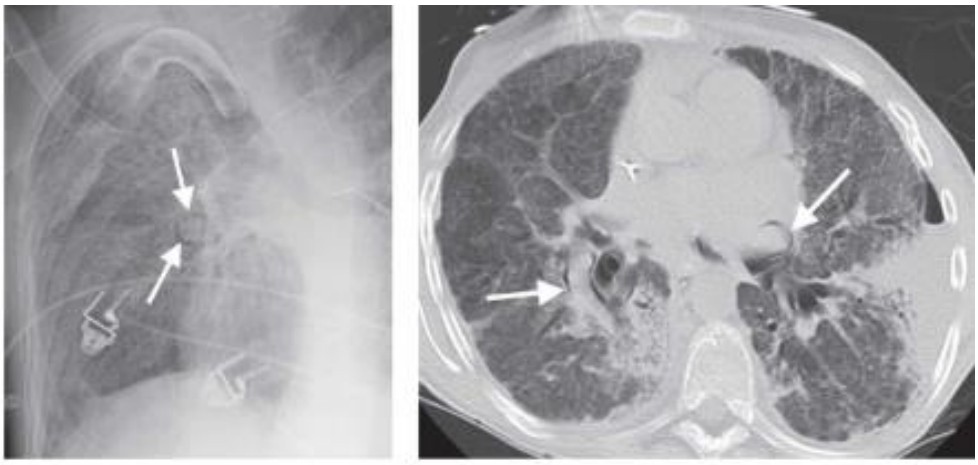


Figure 8. Ring around the artery sign. A: PA chest X-ray of a patient with acute respiratory distress syndrome showing a lucent ring around the right pulmonary artery (arrows) as a marker of pneumomediastinum. B: Thorax CT scan confirms air around both pulmonary arteries (arrows).[1,2]

Tubular artery sign

The tubular artery sign is the presence of air adjacent to the main branch of the aorta and depicts both sides of the blood vessel. Air in the mediastinum will outline the main lateral artery pulmonary and aortic arch, where this pleural line is formed from both the mediastinal parietal pleura and the visceral pleura (Figure 9-11).[1,2]

Figure 9. PA chest radiograph showing some of the typical signs of pneumomediastinum, namely air in the aortic branch called the tubular artery sign (white arrow), air at the border of the descending aorta (black arrow), air in the mediastinal pleura around the aortic arch (white arrow) and around the heart (black arrowhead).[1,2]

Figure 10. Lateral chest radiograph showing air surrounding the brachiocephalic vessels (black arrowhead). The lucent line is also seen in the prespinal soft tissue (white arrow). There is also a ring around the artery sign (white arrowhead).[1,2]

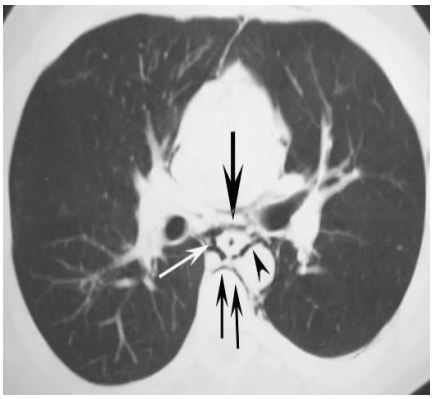


Figure 11. Axial CT scan image showing air surrounding the descending aorta (black arrowhead), azygos vein (white arrow), oesophagus (black arrow), and anterior portion of the spine (two black arrows).[1,2]

Double bronchial wall sign

The double bronchial wall sign is the presence of air in the mediastinum in the area around the bronchus, thereby clarifying and emphasizing both sides of the bronchus (Figure 12).[1,2]

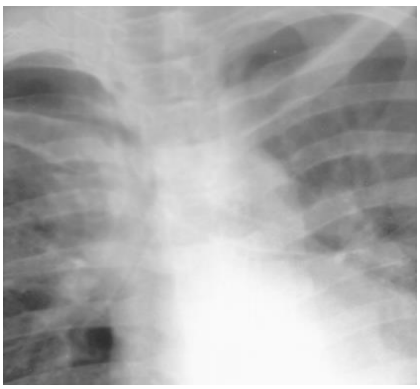


Figure 12. For a 35-year-old patient with status asthmaticus, the AP photo showed air in the mediastinum and left main bronchus, which was visualized by visible both sides of the bronchial wall.[1,2]

Continuous diaphragma sign

The continuous diaphragm sign is the presence of air in the mediastinum that forms a boundary on the superior surface of the diaphragm and appears to separate the diaphragm from the heart. This sign can be seen on the AP/PA radiograph when the air in the mediastinum separates the heart and the superior surface of the diaphragm, which can be seen in both the erect and supine positions. This sign resembles the pneumopericardium, but the pneumopericardium will show air that outlines the heart in a circle.[1,2]

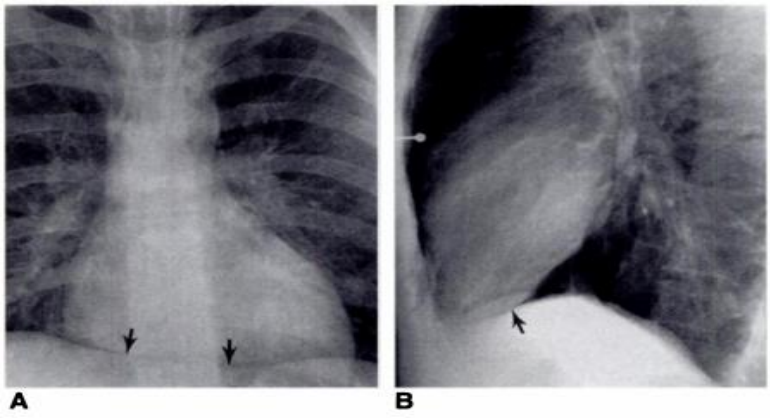


Figure 13. A. PA Thorax radiograph taken during expiration shows a thin band of air (arrow) between the heart and the diaphragm, making the diaphragm visible where normally obscured by the heart. This finding is known as the continuous diaphragm sign. B, Lateral chest radiograph, air outlines the superior surface of the left hemidiaphragm (arrow), which is usually obscured by the liver. This finding is the continuous diaphragm sign.[1,2]

Extrapleural sign

The extrapleural sign is the presence of air in the mediastinum, causing an area of lucency outside the pleura, usually at the lateral edge of the descending aorta (Fig. 13). The extrapleural sign can also form a radiolucent pocket, a sign of free air in the parietal pleura and diaphragm and posterior to the hemidiaphragm dome. In these circumstances, the air can disappear spontaneously within 10 days.[1,2]

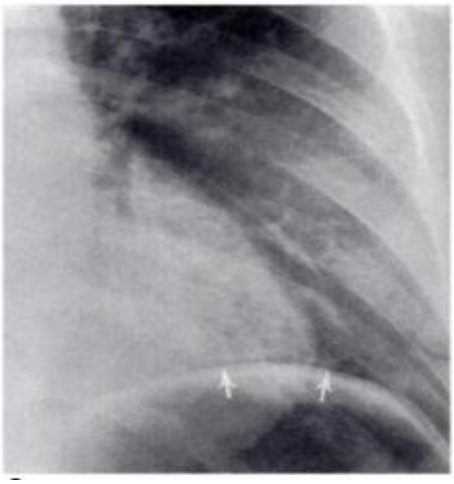


Figure 14. Pneumomediastinum from asthma. The radiograph shows gas (arrows) extending from the mediastinum and separating the pleura (parietal and visceral layers) from the diaphragm. This finding is known as the extrapleural sign.[1,2]

Naclerio's V sign

Naclerio's V sign can be seen on a frontal chest X-ray forming a 'V'-shaped air lucency in the lower-left area of the mediastinum. This sign is formed by air in the mediastinum, which gives the lower left lateral border of the mediastinum and is formed by air present in the parietal pleura and medial portion of the left hemidiaphragm. Usually, this sign is found in cases of oesophagal rupture, where air enters the mediastinum from the ruptured oesophagus. This sign can also be a complication of endoscopic examination, where it occurs in 1 in 1000 cases of patients with the endoscopic examination.[1,2]

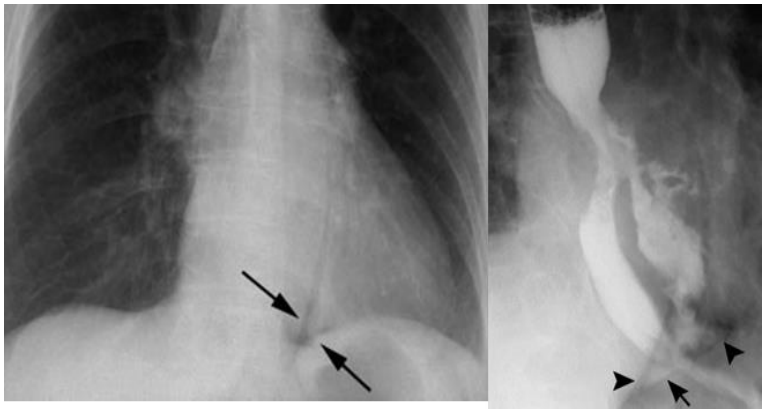


Figure 15. CXR image showing the pneumomediastinum forming the Naclerio's V sign (arrow). Using Iopamidol contrast material, it is clear that extra-passage contrast.[1,2]

Chest Ultrasonografi

The diagnosis of pneumomediastinum is usually based on a chest radiograph and physical examination. However, a chest radiograph may not be diagnostic, especially when there is a very little gas collection in the chest cavity. In some conditions, ultrasonography has a fairly good role in visualizing a pneumomediastinum, such as in infants who experience spontaneous pneumomediastinum that occurs after birth. The most common sonographic

finding is a thick echogenic line along the anterior border, lateral border and in the thymic parenchyma (Figure 16 and 17). [10,12]

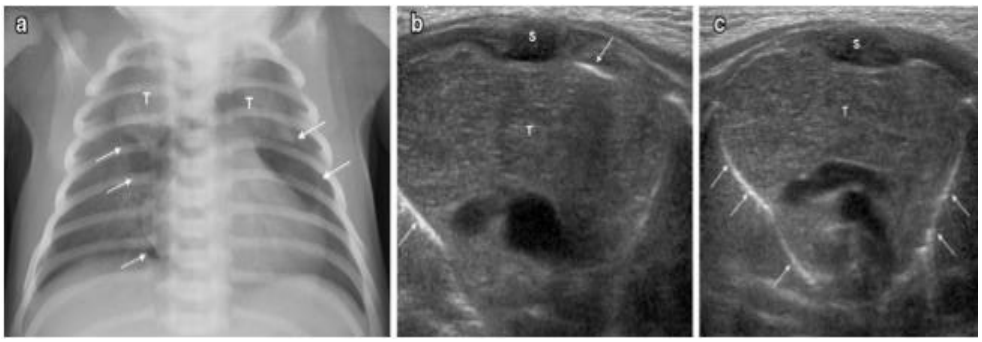


Figure 16. Pneumomediastinum in a 2-day-old boy with dyspnea. Chest radiograph shows abnormal radiolucency along both cardiac junctions (arrows) at thymus elevation. b, c Ultrasonographic scan of the transverse mediastinum showing thick echogenic lines (arrows) along the anterior and lateral borders of the thymus due to pneumomediastinum. Note that the sternum is cartilage, allowing the transmission of sound beams. T thymus, S sternum.[10,12]

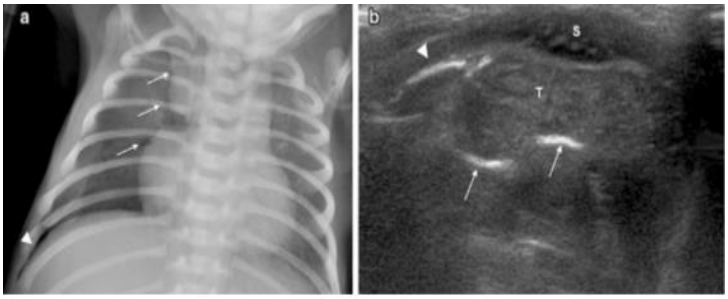


Figure 17. Pneumomediastinum in a 0-day-old boy with dyspnea. Chest radiograph shows an abnormal radiolucency (arrow) along the right border of the thymus and heart. The deep costophrenic sulcus (arrow) on the right indicates a pneumothorax. b Transverse sonogram reveals a thick curved echogenic line (arrow) between the posterior margin of the thymus and the great vessels due to the pneumomediastinum. An echogenic curvature (arrow) also outlines the right anterior lung, possibly due to a combined pneumothorax. T thymus, S sternum[10,12]

According to most authors, early surgical repair of healthy tissue remains the treatment of choice. Surgery is the preferred treatment for most patients with acute tracheal rupture. However, conservative management is an alternative for patients judged unsuitable for surgery and minor tears.[6,7,10]

Percutaneous drainage is a first-aid measure in the event of tension pneumomediastinum (a condition in which air is trapped in the mediastinum and cannot escape). This action is very important to overcome the decrease in venous return pressure.[7,9]

The prognosis of pneumomediastinum is known to be quite good. In stable patients, it is adequately treated with complete rest and analgesics. Morbidity or mortality of patients

with pneumomediastinum is usually caused not by the pneumomediastinum but by other causes that underlie the occurrence of pneumomediastinum.[6,7,10]

Complications of pneumomediastinum occur when the air in the mediastinal space cannot be completely absorbed, namely tension pneumomediastinum and mediastinitis. Tension pneumomediastinum causes compression of the large veins, resulting in decreased venous return and hypotension. Mediastinitis is an infection of the mediastinal space caused by massive and frequent vomiting, known as Boerhaave syndrome.[6,9]

The diagnosis of this patient was Pneumomediastinum rupture of the trachea et larynx due to a sharp trauma perforating wound, which was carried out by the patient himself with the discovery of several stab wounds that appeared on the neck, which resulted in tearing of the trachea and larynx. This tear causes the air that can spread to the surrounding area, such as the cuticle tissue, which causes subcutaneous emphysema and can even cause other complications such as pneumomediastinum and several other air leak syndromes. Patient complaints also support aerodigestive trauma such as hoarseness, painful swallowing and crepitus on examination of the cuticle tissue around the trauma site. Laboratory examination found an increase in leukocytes, which is a support for the occurrence of pneumomediastinum. On examination of the AP chest X-ray, a pneumomediastinum image was found with emphysema subcutis at neck regio, then the diagnosis on the AP chest X-ray was confirmed by the findings obtained on a non-contrast MSCT scan of the neck where pneumomediastinum was suspected to be from rupture of the trachea et larynx due to sharp trauma from a sharp stab wound. CT scan can also describe the expansion of air due to aerodigestive trauma in this patient. This result follows the earlier theory that CT scans can help confirm the diagnosis of a pneumomediastinum and explain the extent of air expansion in organs that are decomposed by air in cases of pneumomediastinum.

There is marked expansion of the air in this patient due to aerodigestive trauma. Several possible mechanisms may explain this phenomenon. First, the patient was a male patient with young adult age, whereas pneumomediastinum was most often reported in young patients and male gender. Second, the patient had a history of smoking and long-term drug use, both of which were also risk factors for pneumomediastinum.

Conclusion

We describe a case of pneumomediastinum resulting from laryngeal rupture due to sharp trauma in a psychiatric patient. This is a challenge in making a diagnosis because of the

lack of information about the etiology of trauma and complaints that can be fatal due to delay in therapy. Radiology is one of the best diagnostic supports in describing pneumomediastinum and other air leak syndromes either by chest X-ray, ultrasound or CT scan.

Conflict of Interest

Nothing to declare

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None

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CASE REPORT **Open Access**

Percutaneous Coronary Intervention in Single Coronary Ostium Presented with STEMI: A Case Report

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ABSTRACT

Introduction: Coronary artery anomalies presenting with ST-segment elevation myocardial infarction (STEMI) are uncommon and often are challenging to manage. We report a case of a successful percutaneous coronary intervention (PCI) of the right coronary artery (RCA) in a patient who presented with inferior STEMI and an anomaly of the left and right coronary arteries with a single coronary ostium in the right sinus of Valsalva.
Case: Male, 71 years old, was admitted to the emergency department with 8 hours onset of chest pain. The patient was diagnosed with STEMI Inferior 8 hours of onset Killip I TIMI score 5. The patient was given a dual loading antiplatelet, high-dose nitrate and statin, then we planned him for primary PCI. Primary PCI was performed on the right transradial through the single osteal coronary artery from the right sinus Valsalva. We got the TIMI flow 3 without other complications during the action.
Discussion: It is frequently difficult to identify the culprit lesion during primary PCI. The course of the anomalous vessel and the culprit lesion are clear often; the revascularization procedure hides several pitfalls.
Conclusion: Coronary artery anomalies presenting with STEMI are uncommon but often challenging. Percutaneous coronary intervention is considered the first choice in patients with coronary anomaly presented with STEMI.

Keywords: PCI, single coronary ostium, STEMI

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Introduction

Coronary artery anomalies presenting with ST-segment elevation myocardial infarction (STEMI) are uncommon and often are challenging to manage. The reported incidence of the right-sided origin of the left coronary artery (LCA) is only 0.02%-0.15%. An anomalous aortic origin of the left coronary artery (AAOLCA) coursing between the aorta and the pulmonary artery (PA) has a fatal risk of altered coronary flow due to the compression of the anomalous coronary artery between the great vessels during exercise.1 We report a case of a successful percutaneous coronary intervention (PCI) of the right coronary artery (RCA) in a patient who presented with inferior STEMI and an anomaly of the left and right coronary arteries with a single coronary ostium in the right sinus of Valsalva. The presentation of this rare coronary anomaly with its imaging features and possible management options were also discussed.

Case

Male, 71 years old, was admitted to the emergency department with 8 hours onset of chest pain. An active smoker and hypertension on treatment. His blood pressure 150/90, heart rate was 78 bpm, peripheral saturation was 99% in room air. The ECG on this patient showed ST elevation in the inferior lead with st depression in V1-V5, as shown in Figure 1. We diagnosed this patient as STEMI Inferior 8 hours of onset Killip I TIMI score 5. The patient has been given a dual loading antiplatelet, high-dose nitrate and statin, then we planned him for primary PCI.

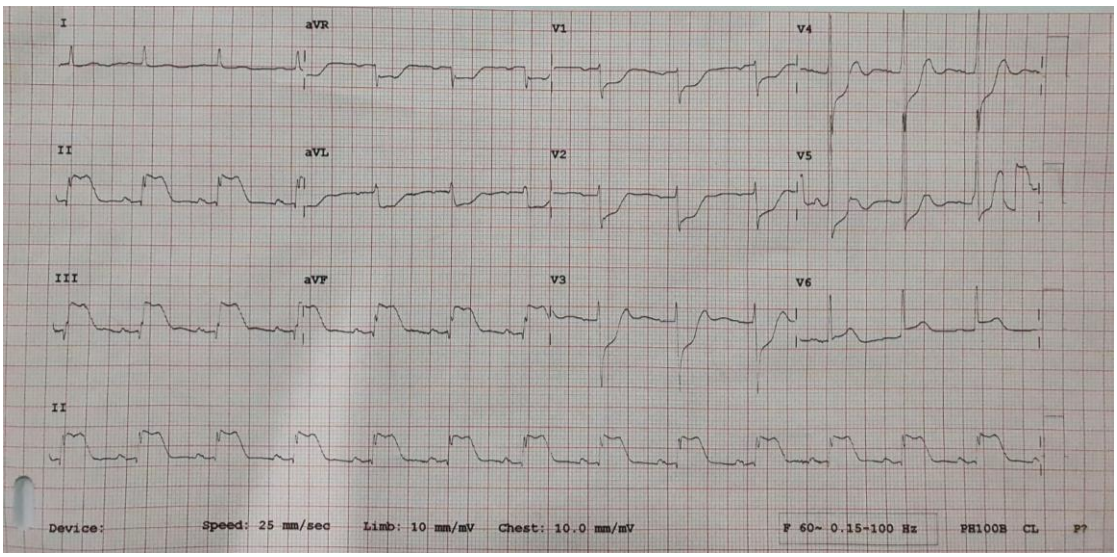

Figure 1. ECG findings on the patient

Primary PCI was performed on the right transradial. By the time we did the primary PCI, we had found it difficult to access the left main coronary artery. So we decided to access the right coronary artery. Surprisingly, there was an anomaly in the coronary arteries. We obtained a single osteal coronary artery arising from the right sinus Valsalva. On that osteal RCA, it branches off the left anterior descending artery. Total occlusion was found on distal RCA as presented in Figure 2A. Wiring is performed through the occlusion to distal RCA. Pre-dilation was performed with a 2.5 x 15 mm Saphine balloon in the mid to 16 atm. Then a 2.5 x 24 mm DES stent was implanted distally until the mid-PDA and expanded to 16 ATM. We got the TIMI flow 3 without other complications during the action, as shown in Figure 2B.

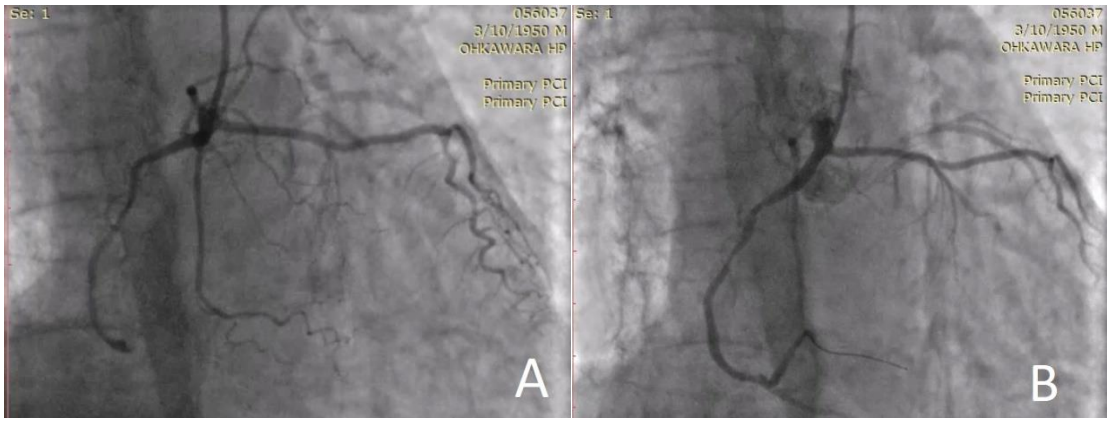

Figure 2. Coronary angiography of the patient. A. Initially, before the PCI procedure, shown a total occlusion on distal RCA. B. After PCI, the contrast appeared, filling the lower RCA blood vessels.

Discussion

The incidence of coronary anatomy anomalies is approximately 0,6% - 1,3%. There is various type of coronary artery anomaly, and the rarest condition is the anomalous aortic origin of the coronary artery from the opposite coronary sinus. The variant, in this case, a single ostium in the right sinus Valsalva is exceedingly rare, and only a few cases have been described in the literature.[2,3]

In nearly 15% of patients with SCA, myocardial ischemia can develop in the absence of atherosclerosis. During exercise, a mismatch between myocardial demand (which is increased during exertion) and myocardial oxygen delivery, dependent upon the single coronary blood flow, may fail to increase demand or may even decrease during exertion. This limited or diminished coronary blood flow is probably the result of one or many high risk anatomic and physiologic factors: a). flap closure of the slit-like deformation of the coronary

ostium; b). the acute (non-orthogonal) angle of take-off and kinking of the coronary artery as it exits from the aorta; c.) hypoplasia and stenosis of the intramural segment, particularly at the level of the valvular commissure.[4,5]

Currently, the ideal imaging tool for diagnosing and delineating coronary artery anomalies is angiography supported by CT angiography. The culprit lesions in an inter-arterial course may pose a great challenge in stented cases due to the compression of the vessel during strenuous exercise. It is important to know the course of the arteries before any intervention in PCI of anomalous coronary arteries. CTA is a useful diagnostic tool to delineate a probably malign variation to be stented or the anatomical relationship of the anomalous coronary trunk.[6]

During primary PCI, it is frequently difficult to identify the culprit lesion. The course of the anomalous vessel and the culprit lesion are clear often; the revascularization procedure hides several pitfalls. It is still a challenge for an interventional cardiologist in the settings of myocardial infarction in a patient with coronary anomaly where complete anatomy is unknown.

Conclusion

Coronary artery anomalies presenting with STEMI are uncommon but often are a challenge. Percutaneous coronary intervention is considered the first choice in patients with coronary anomaly presented with STEMI. The interventional cardiologist needs to know the full range of aberrant coronary anatomy, particularly in emergency cases where rapid identification of the culprit's vessel is crucial.

Conflict of Interest

We do not have any potential conflict of interest

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REVIEW ARTICLE **Open Access**

Diagnostic Evaluation in Primary Intraocular Lymphoma : A Review

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ABSTRACT

Background: Although very rare, there has been an increasing incidence of primary intraocular lymphoma (PIOL) in recent years. In addition, PIOL is often missed in diagnosis and is often treated without a standard, resulting in frequent recurrence and involvement of the central nervous system. Investigations have an important role in establishing the diagnosis of this case.
Contents: The latest diagnostic supporting tests applied to PIOL are optical coherence tomography, fundus fluorescence angiography, indocyanine green angiography, fundal autofluorescence, ocular ultrasonography, vitrectomy sampling, and head MRI. Histopathological examination with Giemsa stain or hematoxylin-eosin is the gold standard in this case. Molecular studies and genetics have also recently played an important role in supporting PIOL diagnosis.
Conclusion: PIOL has similarities with other vitreoretinal and uveal tract diseases, so it is not easy to diagnose. High clinical suspicion needs to be followed up with the latest investigations to establish PIOL diagnosis.

Keywords: Diagnostic approach, intraocular lymphoma, primary

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Introduction

Primary Intraocular Lymphoma (PIOL) or often called primary vitreoretinal lymphoma (PVRL), is a neoplasm, most commonly B-cell, and rarely T-cell, that originates from or initially arises in the subretinal pigment epithelium (RPE), retina, and vitreous.[1-4] Definitions PIOL is a subset of PCNSL and a heterogeneous group of malignant lymphocytic neoplasms affecting the retina with or without vitreous or optic nerve involvement and evidence of brain or cerebrospinal fluid involvement. Hodgkin's lymphoma is from a large diffuse network of B-cell histologic types, and the difference is that PIOL is a subtype of primary central nervous system lymphoma (PCNSL).[6] Once there is a manifestation of central nervous system involvement, the disease entity changes to PCNSL.[7]

Although primary orbital tumours are very rare compared to other tumours, this subtype of PIOL is the most common primary ocular lymphoma malignancy.[8] In the United States, the incidence of PCNSL was 0.46 per 100,000 population in the period 2004-2007. The incidence was higher in males (0.54) than females (0.39), with a male: female ratio of 1.38. Although there are contradictory reports that intraocular lymphoma is more common in women.[9] PCNSL with ocular involvement and PIOL is estimated to account for 1% of non-Hodgkin's lymphomas, 1% of intracranial tumours, and ocular involvement and PIOL are estimated to account for 1% of non-Hodgkin's lymphoma, 1% of intracranial tumours, and <1% of intraocular tumours.10 There is a trend of increasing incidence of PIOL in recent years which may be due to the increase in cases of immunodeficiency, immunosuppression, and the development of diagnostic tools.[7]

PIOL commonly presents in older patients as non-specific, chronic and relapsing uveitis known as Masquerade's syndrome that does not respond to steroid therapy, making the diagnosis sometimes difficult.[11] Eye disorders are usually bilateral (64-83% of cases).[12] Although Unilateral involvement is present in 30% of cases but may be bilateral in about 85% of patients. Approximately 25% of patients with PCNSL will have a PIOL, and at least 60% of patients presenting with a PIOL will have central nervous system disease. The patient's most frequent symptoms are decreased visual acuity and floaters, and infrequent complaints include redness of the eye, photophobia, and ocular pain.[13]

The gold standard in establishing PIOL diagnosis is the finding of lymphoma cells in the intraocular compartment by histopathological examination.[7] However, starting the diagnostic procedure must be based on a high clinical suspicion. Some clinical indications that increase the suspicion of PIOL according to Chan et al. (2011)[10] are older adults with

non-infectious uveitis who do not respond to inflammatory therapy, visual acuity that is not correlated and is better than the severity of the vitreous found on fundoscopic examination, and there are a characteristic yellow-white subretinal infiltrate lesion on fundoscopic examination. This article will then review further investigations to establish the diagnosis of PIOL.

Optical Coherence Tomography (OCT)

Direct infiltration of the retina by lymphoma cells with focal proliferation creates a semi-opaque appearance that appears homogeneous on optical coherence tomography (OCT). Infiltrating lymphomatoma can be seen as a hyperreflective signal in the form of dots, bands and nodules at or above the RPE with the spectral-domain OCT2 (Figure 1). This hyperreflective signal must be distinguished from age-related macular degeneration or diabetic macular oedema.[14,15] Granular subretinal lesions (between Bruch's membrane and the retinal pigment epithelium) may be seen when a subretinal lesion is present. OCT can be used to monitor lymphoma progression or regression.[16]

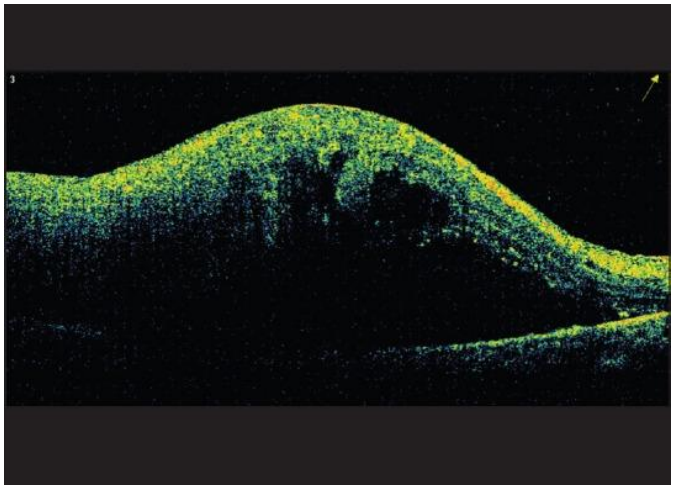

Figure 1. Overview of Subretinal Lymphoma Cell Infiltration on OCT imaging.[7]

Fundus Flourescence Angiography (FFA) and Indocyanine Green Angiography (ICGA)

FFA can be used to demonstrate hypofluorescence in the early to late stages of PIOL involving the outermost retinal layer (Fig. 2). Other signs that can be seen on this examination are punctuated hyperfluorescent window defects, round hyperfluorescent lesions, vasculitis, fluorescence leakage along the retinal veins, and periarteriolar staining.[17,18] In the early stages of PIOL, hypofluorescent lesions will appear on ICGA examination but become less visible in the late stages of PIOL.[19] The FFA and ICGA examinations simultaneously had sensitivity and specificity values of 89% and 85%, respectively.

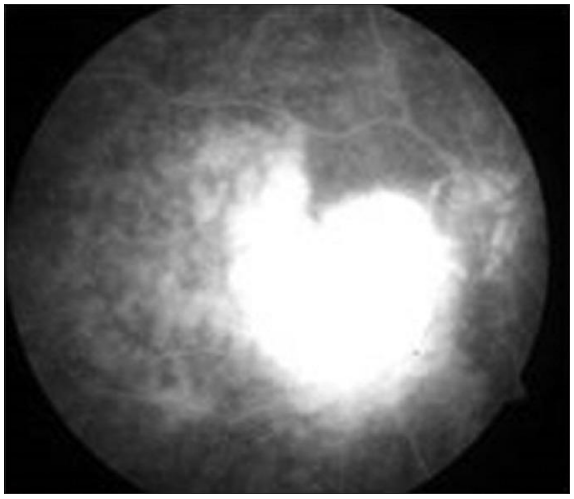

Figure 2 Small hypofluorescent lesion in the lesion area with fluorescent leak seen in late stage PIOL on FFA examination.[21]

Fundal Autoflorescence

This examination is to determine where the lymphoma infiltrating the RPE is located. On fundal autofluorescence (FAF) examination, the typical finding in PIOL cases is a granular pattern consisting of hyperautofluorescent patches surrounded by a hypoautofluorescent ring as shown in Figure 3. In the lymphomatous infiltration in the sub-RPE space, a hyperautofluorescent spot is seen surrounded by a hypoautofluorescent ring. On the other hand, if the lymphomatous infiltrate is above the RPE, it will produce a granular pattern of hypoautofluorescent dots surrounded by a hyperautofluorescent ring. The hypofluorescent spots (leopard spots) seen in FFA are identical to the hyperautofluorescent spots in FAF.[19,22]

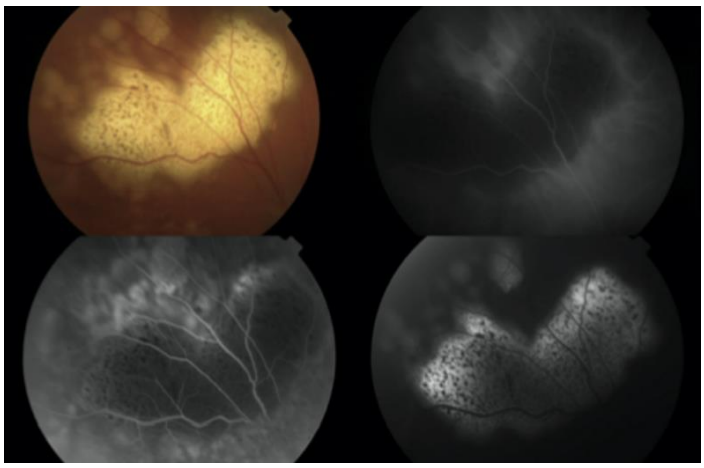

Figure 3. PIOL lesion on fundus autofluorescence.[22]

Ultrasound B-Scan

Ocular ultrasonography is not specific for PIOL but can be used in cases where the posterior segment is difficult to visualize. Ultrasound findings suggesting a PIOL include vitreous debris, retinal detachment, enhancement of the chorioretinal lesion, and optic nerve widening, as shown in Figure 4.[19] B-scan ultrasound allows objective measurements with good repeatability, descriptive and easy follow-up. Therefore, ultrasonographic B scan of the eye should provide the basis for clinical diagnosis and follow-up of PCNSL with intraocular involvement as a cost-effective method with high accuracy and efficiency for diseases with high and low malignancy rates.[23]

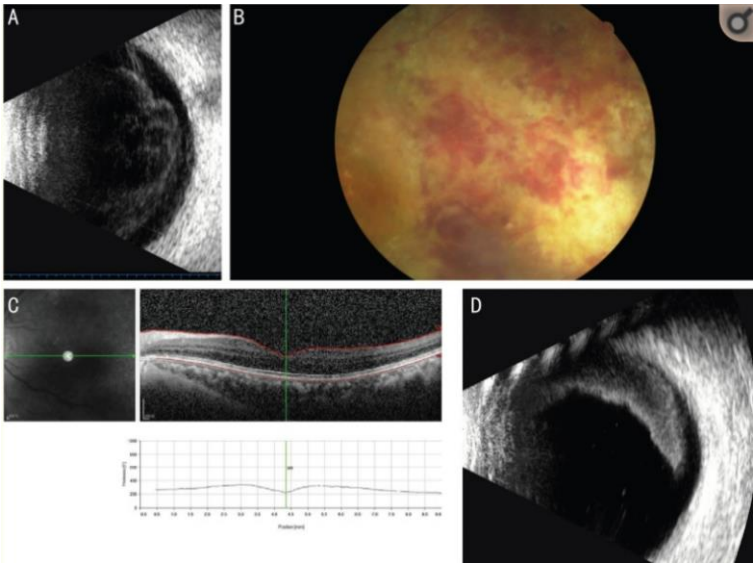

Figure 4. PIOL lesions on Ultrasound B scan and OCT. A: The punctate echo group with moderate and high peaks was observed on ultrasound B-scan of the eye; B: Yellow deposits on the retina observed on fundoscopy; C: Normal central foveal thickness detected by OCT scan; D: Suspected eccentric mass observed on ultrasound but not on slit-lamp examination.[23]

Sampling Vitrectomy

Two methods can be used to perform vitrectomy sampling: vitreous biopsy and pars plana vitrectomy (Pars Plana Vitrectomy, PPV). The vitreous biopsy takes a sample from the central vitreous of 1-2 ml of the undiluted specimen while in PPV samples from the central vitreous and cortical 50-100 ml of diluted vitrectomy cassette fluid.[24] To ensure adequate and maximum cellular viability, samples should be taken to the laboratory without delay within 30 minutes of sampling. Suppose the sample cannot be brought to the laboratory. In that case, fixatives such as ethanol, methanol, propranolol solutions (8:1:1 ratio), Shandon cytofix or HEPES-glutamic acid-mediated organic-solvent protection effect (HOPE) buffer can be used.

PPV examination has advantages over vitreous biopsy because it can maximize sample volume, improve patient vision by cleaning vitreous debris, and PPV's greater cellularity than vitreous biopsy, allowing additional tests such as immunohistochemistry, DNA extraction for monoclonal studies and polymerase chain reaction (PCR) to be performed.[24]

Head Magnetic Resonance Imaging (MRI)

Since PIOL is part of PCNSL, it is necessary to perform a systemic neurologic examination to exclude the possibility of central nervous system involvement. This aim can be done by magnetic resonance imaging (MRI) of the brain, examining the cerebrospinal fluid, and a brain biopsy.[7] CNS lesions that may be seen on brain MRI are uni- or multi-focal hypodense on T1-weighted and hyperdense on TRI-weighted MRI as shown in Figure 5.[19,25] Cerebrospinal fluid examination for the detection of lymphoma is important to avoid invasive diagnostic procedures such as vitrectomy. 25% of patients with CNS lesions have positive cerebrospinal fluid cytology.[26] Imaging-guided brain biopsy should be performed in patients with suspicious MRI lesions but negative CSF cytology.[7]

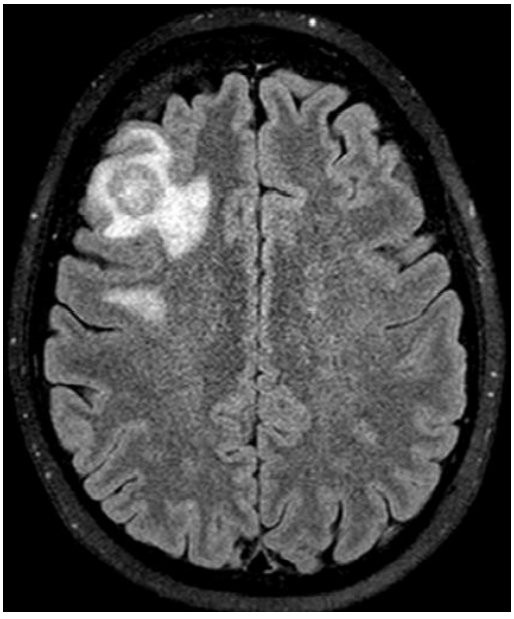

Figure 5. MRI image of PIOL with CNS involvement, showing a mass in the right medial frontal gyrus accompanied by vasogenic oedema.[27]

Standard Histopathologic Examination

Standard histopathological examination of PIOL using a light microscope. The most

recommended tissue stains are Giemsa or Diff-Quick because they can give a better picture of the characteristics of lymphoma cells. However, other stains such as Papanicolou or Hematoxylin-eosin can also be used. Under light microscopy, PIOL or PVRL cells are seen clustered in the sub-RPE space and appear as large lymphoid cells with little basophilic cytoplasm and a large visible nucleus that may be round, oval, or granular or hyper-segmented with dense prominent nucleoli and mitoses (Figure 6). In addition, a background image of inflammatory cells and necrotizing lymphoma cells can also be seen.[7]

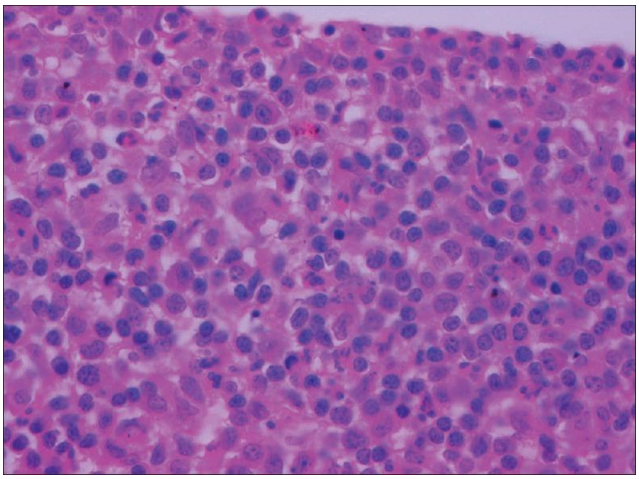

Figure 6. Vitreous biopsy examination in PIOL cases. Large cells have scanty or moderate cytoplasm and prominent nucleoli (HE staining 400x magnification).[7]

Immunohistochemistry and Flow Cytometry

The immunohistochemical examination determines whether lymphoma originates from B cells or T cells. In addition, this examination aims to determine the ability of lymphoma cell proliferation, which shows the degree of aggressiveness. Most cases originate from B cells where the markers CD20, CD79α are positive, and CD3 are negative. Rarer cases where lymphoma originates from T cells where the markers are CD3 positive and CD20 negative. These two subtypes generally show high expression of the Ki-67 marker so that they have extensive proliferative abilities (Fig. 7-9).[11]

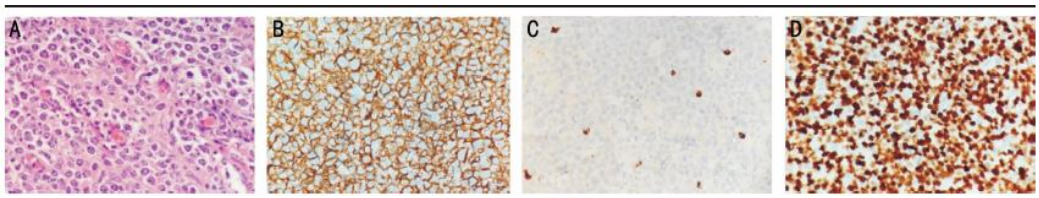

Figure 7. Histopathological and immunohistochemical examination of PIOL derived from B cells. (A) Atypical cells with pleomorphic nuclei with prominent nuclei and very little cytoplasm (HE staining 200x magnification); (B) Immunohistochemical examination which

showed positive CD20 (200x magnification); (C) Immunohistochemical examination which showed negative CD3; (D) Immunohistochemical examination which showed high expression of the proliferative marker Ki-67 (>80%) indicated its extensive proliferative nature.[11]

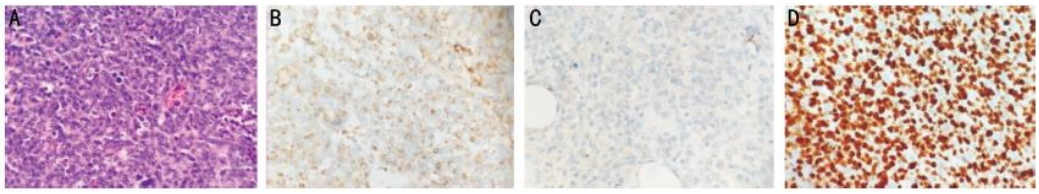

Figure 8. Histopathological and immunohistochemical examination of T cell-derived PIOL. (A) Moderate to large lymphoid cells with atypical nuclei (HE staining 200x magnification); (B) Immunohistochemical examination which showed positive CD3 (200x magnification); (C) Immunohistochemical examination, which showed negative CD20; (D) Immunohistochemical examination which showed high expression of the proliferative marker Ki-67 (>80%) indicated its extensive proliferative nature.[11]

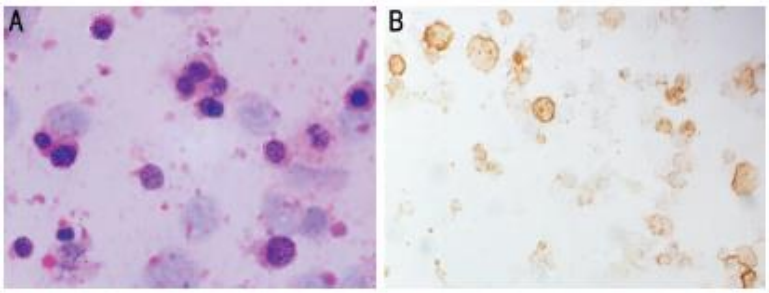

Figure 9. Cytological examination of the vitreous specimen of a PIOL patient. (A) large, atypical and highly stained lymphoid cells with irregular nuclei and coarse chromatin (400x magnification); (B) After an immunocytochemical examination, a positive CD20 expression was found, indicating B cells' origin.[11]

The flow cytometry examination can examine the cell surface marking and show a monoclonal B cell population. IOLs usually consist of monoclonal B cells with restricted chains. The ratio 2 : 3 or 0.6 is a very sensitive marker for lymphoma.[28]

Other Laboratory Examinations and Genetic Test

The molecular examination detects immunoglobulin genes involved in lymphoma cells and the cytokines expressed. On vitreous fluid analysis will show an increase in interleukin (IL-10) with a ratio of IL-10: IL-6> 1.0 very helpful for diagnosis. PCR was used to examine the amplification of the immunoglobulin heavy chain DNA. In B-cell lymphoma, molecular analysis can detect rearrangements of the IgH gene, whereas, in T-cell lymphoma, rearrangements of genes making up the T-cell receptor can be detected. PCR analysis of the EB virus in aqueous humour may be useful to support the diagnosis of intraocular NK cell lymphoma.[11]

PIOL detection can also be done through chromosomal analysis. The presence of a BCL-2 translocation (14;18) is suggestive of an IOL diagnosis. Microdissection with a minimum of 15 atypical lymphoid cells has been shown to have a diagnostic efficiency of 99.5% using PCR.[29,30] In addition, HIV tests, complete blood counts, and specific tests can also be performed to determine the cause of uveitis.[7]

Conclusion

PIOL is a rare clinical entity that is often misdiagnosed as an infectious or inflammatory condition. There is a high index of clinical suspicion, especially when there is a suboptimal response to standard steroid treatment or recurrence in suspected vitreous/uveitis. This high clinical suspicion needs to be continued with the latest investigations to establish PIOL diagnosis. Initial investigations that can be performed are optical coherence tomography, fundus fluorescence angiography, indocyanine green angiography, fundal autofluorescence, ocular ultrasonography, vitrectomy sampling, and head MRI. Molecular and genetic testing can also support the diagnosis of PIOL. In addition, histopathological examination with Giemsa stain or hematoxylin-eosin is the gold standard in this case.

Conflict of Interest

Nothing to declare

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