Vol. 5 No. 2 (2026)

Pakistan Journal of Medical & Cardiological Review

https://pakjmcr.com/index.php/1/about

Online ISSN 3007-2387

Print ISSN 3007-2379

Shah et al., 2026

FREQUENCY OF PERICARDIAL EFFUSION AMONG MALE AND FEMALE IN TERTIARY CARE HOSPITAL

Jamal Shah
Department of Health and Biological Sciences Abasyn University Peshawar
Ayesha Naimat Ullah
Department of Health and Biological Sciences Abasyn University Peshawar
Zahid Ullah
Department of Health and Biological Sciences Abasyn University Peshawar
Wasif Khan
Department of Health and Biological Sciences Abasyn University Peshawar
Furqan Jehan
Department of Health and Biological Sciences Abasyn University Peshawar
Laiba Noor
Department of Health and Biological Sciences Abasyn University Peshawar
Shah Fahad Khalid
Department of Health and Biological Sciences Abasyn University Peshawar
Adnan Khadim*
Coordinator Cardiology Abasyn University Peshawar. Corresponding Author Email: adnan.khadim@abasyn.edu.pk

Author Details

Received on 22 May, 2026

Accepted on 26 June, 2026

Published on 28 June, 2026

Corresponding E-mails & Authors*:
Adnan Khadim
adnan.khadim@abasyn.edu.pk

Abstract

Background: Pericardial effusion involves the abnormal accumulation of fluid within the pericardial space, which can lead to significant hemodynamic impairment and progress to life-threatening cardiac tamponade if left unmanaged. **Objective:** To determine the frequency of pericardial effusion among male and female patients presenting to a tertiary care hospital and to analyze their clinical and demographic characteristics. **Methodology:** A descriptive cross-sectional study was conducted utilizing data from 196 patients at a tertiary care hospital. Demographic variables (age, gender) and clinical comorbidities (hypertension, diabetes mellitus, dyslipidemia, smoking status, and history of myocardial infarction) were documented. Echocardiographic evaluation was used to classify the

severity of pericardial effusion (mild, moderate, severe) and assess left ventricular (LV) function. **Results:** Out of 196 patients, 51.8% were female and 48.2% were male. The highest age frequency was observed in the 40–50 age group (40.8%). Comorbidities revealed a very high prevalence of hypertension (93.9%), while diabetes mellitus (6.6%) and dyslipidemia (7.7%) were lower. Smoking was reported by 20.4% of the cohort. Echocardiographic findings demonstrated that 99.5% of the suspected cohort had confirmed pericardial effusion. Severity distribution showed that mild pericardial effusion was highly prevalent at 87.2% (n=171), moderate effusion was present in 12.8% (n=25), and severe effusion was present in 10.7% (n=21). Cardiac tamponade was noted in 1.0% (n=2) of the cases. Left ventricular dysfunction was present in 42.3% of the patients, with 10.7% presenting with an ejection fraction below 35%. **Conclusion:** Pericardial effusion shows a slightly higher frequency among females than males in the tertiary care setting, with the middle-aged population (40–50 years) being most vulnerable. The vast majority of cases present as mild effusions, strongly associated with systemic hypertension and varying degrees of left ventricular dysfunction.

INTRODUCTION

1.1 Anatomy of the Heart

The heart of humans is a muscle, conically shaped, lying in the thoracic cavity, between the lungs, and inclined towards the left side. It rests in the mediastinum, protected by the pericardial sac. It acts as a pump and circulates the blood throughout the body through the circulatory system.

The heart consists of four chambers, namely two upper chambers named atria (right atrium and left atrium), and two lower chambers called ventricles (right ventricle and left ventricle). The right part of the heart processes the deoxygenated blood, whereas the left part processes the oxygenated blood. Deoxygenated blood enters the right atrium of the heart through superior and inferior venacav, and then it moves to the right ventricle through the tricuspid valve. Oxygenated blood enters the right ventricle, which pumps it into the lungs through the pulmonary artery to get oxygenated.

Oxygenated blood coming back from the lungs reaches the left atrium through the pulmonary veins, and it flows into the left ventricle through the mitral (bicuspid) valve.

Oxygenated blood coming from the lungs enters the left ventricle, having the thickest wall among all other chambers, and it gets pumped into the aorta, the major artery.

Three major components make up the heart wall, which include epicardium, myocardium, and endocardium. Myocardium is the thick layer and is accountable for the heart contractility. The strength and rhythmic activity of myocardium ensures the continuity of blood circulation.

Heart valves are present to enable the unidirectional flow of blood through the heart. Four valves, including tricuspid valve, pulmonary valve, mitral valve, and aortic valve, are present in heart, which help maintain blood flow direction and prevent any backward flow of blood.

Arteries play an important role in providing the heart with its own supply of blood. Coronary arteries arise from the root of aorta and provide oxygen-filled blood supply to the muscles of the heart. Occlusion in the coronary arteries could result in some serious problems, including myocardial infarction or heart attack. Intrinsic conducting system is responsible for coordinating all activities in the heart. Sinoatrial (SA) node present in the right atrium serves as the pacemaker, as it produces impulses responsible for initiating a cardiac cycle. These impulses pass through the AV node, bundle of His, and Purkinje fibers, leading to the contraction of heart muscles.

In conclusion, the heart is a very important organ, and its structure is highly sophisticated in order to maintain effective blood flow within the body. It is made up of several parts that ensure oxygen and nutrients are supplied to the body continuously and efficiently.(Betts et al., n.d.)

1.2 Physiology of the Heart:

Cardiac physiology is the study of the physiological processes involved in pumping blood in an effective manner by the heart. These processes include electrical activity, muscle contraction, and blood circulation.

1.2.1 Cardiac Cycle:

This refers to the processes occurring in the heart during one beat. There are two major processes in this phenomenon. These include diastole and systole. Diastole is a process whereby the muscle of the heart relaxes, allowing filling of blood in the heart. The atria receive blood, which flows to the ventricles through the open atrioventricular valves.

Systole is the process whereby the heart's ventricles contract to enable pumping of blood. The right ventricle pumps blood to the lungs via pulmonary arteries, whereas the left ventricle pumps oxygen-filled blood to the aorta for distribution to the rest of the body.

1.2.2 Heart Electrical Conduction System:

In the heart, the natural pacing system is responsible for controlling rhythm of the heart rate. Intrinsic electrical activity initiates from sinoatrial (SA) node found in the right atrium. This electrical activity spreads throughout the atria, resulting in their contraction. It passes to the atrioventricular (AV) node where there is some delay allowing filling of the ventricles. Afterwards, the electrical impulse spreads

1.3.3 Heart Rate and Its Regulation:

Normal heart rate for an adult is between 60 and 100 beats per minute. It is controlled by the autonomic nervous system. The sympathetic nervous system causes acceleration in heart rate and strength of heart muscle contractions whereas the parasympathetic nervous system controls its reduction when at rest. Some hormones affect heart function as well.

1.2.4 Stroke Volume and Cardiac Output

Stroke volume is defined as the quantity of blood pumped out of the ventricles during each heart beat. It measures about 70 mL. The cardiac output is the total volume of blood pumped out by the heart in a minute and equals:

Cardiac Output = Heart Rate × Stroke Volume

This provides for enough oxygen supply and nutrition to various parts of the body.

1.2.5 Blood Pressure and Flow:

Blood pressure refers to the force exerted by flowing blood against the walls of the vessels. The highest value of blood pressure is achieved during contraction of ventricles (systolic pressure), while the lowest one occurs during relaxation (diastolic pressure).

1.2.6. Coronary Circulation:

The myocardium also needs to receive constantly oxygen-supplied blood, which enters via the coronary arteries. The efficient coronary circulation is vital for proper functioning of the heart; otherwise, the lack of oxygen can result in such dangerous disorders as heart attack.

1.2.7. Electrocardiogram (ECG):

The electrical activity of the heart can be measured by using the electrocardiogram (ECG). Various kinds of waves may be detected, namely P-wave (depolarization of atria), QRS complex (depolarization of ventricles), and T-wave (repolarization of ventricles).(Betts et al., n.d.)

1.3. Anatomy of pericardium

The pericardium is a membranous sac formed by two layers; it encloses the heart along with its roots of the great vessels in the middle mediastinum lying between the two lungs. There are two parts of the pericardium including the fibrous pericardium as the outer part and the serous pericardium as the inner one. Fibrous pericardium is a dense connective tissue layer responsible for protecting the heart, preventing its excessive distension, and securing it in the thoracic cavity through attachment to the diaphragm from below, the sternum anteriorly, and great vessels like the aorta and pulmonary trunk from above. Within this sac, there is the serous pericardium, which is made up of two parts; parietal part covering the inner part of the fibrous pericardium, and epicardium or the visceral layer closely applied to the outer wall of the heart. The space between these two membranes contains about 10-50ml of serous fluid called pericardial cavity.

The reflections of the serous pericardium at the great vessels constitute a set of recesses called transverse and oblique pericardial sinuses that play an important clinical role in cardiac surgeries. The pericardium derives its blood supply from the pericardiophrenic arteries arising from the internal thoracic arteries. Venous drainage takes place via similar vessels into the brachiocephalic veins. It is innervated by the phrenic nerves (C3-C5) providing sensory innervation for pain; referred pain from the pericardium may be felt in the shoulder region. Pain is mediated by vague and sympathetic nerves as well. Lymphatic drainage takes place into mediastinal and tracheobronchial lymph nodes. The pericardium is important for protection, heart positioning, and efficient functioning of the organ.

1.4. Pericardial Effusion:

Pericardial Effusion is an important clinical pathology involving the presence of an abnormal excess fluid in the pericardial space, which is defined as the potential space between the visceral and parietal layers of the pericardium. Pericardium itself is the fibro

serous double-layered sac surrounding the heart and its major arteries, which is of essential importance for the cardiac function and structural integrity. In normal physiological conditions, the pericardial cavity normally contains about 10 to 50 millilitres of serous fluid acting as a lubricant for the smooth heart movement during the cardiac cycle and preventing any contact between the pericardial layers, as well as facilitating the normal cardiac mechanics and protecting the heart from external mechanical injuries.(Ebrahimi et al., 2025a)

The equilibrium of the production and resorption of the pericardial fluid occurs under normal circumstances. The mesothelial cells that line the pericardium produce the fluid, whereas the lymphatics remove it. Any imbalance of this delicate process caused by pathologic conditions results in the buildup of excessive amounts of fluid, which constitutes pericardial effusion. The reasons causing the imbalance vary greatly and include inflammation, infections, tumor formation, metabolic disorders, and injuries. Pericarditis causes increased capillary permeability and thus an exudate forms; heart failure and hypoalbuminemia lead to transudates due to changed hydrostatic and oncotic pressures.(Yamani et al., 2022a)

The etiologies for pericardial effusion are wide-ranging and vary with geographical, demographic, and clinical parameters. Some of the most common causes of pericardial effusions include infectious diseases especially viral infections. Tuberculosis continues to be one of the most common causes of pericardial effusions in third-world nations, where cases are usually characterized by large volume pericardial effusions. In developed countries, malignancies are some of the most common causes of pericardial effusions followed by idiopathic causes. Malignant pericardial effusions are usually caused by tumors such as lung cancer, breast cancer, and lymphoma, indicating that the condition is at an advanced stage with a poor prognosis. Other autoimmune diseases that may contribute to pericardial effusions include systemic lupus erythematosus and rheumatoid arthritis.(Mori et al., 2024a)

The clinical manifestations of pericardial effusion can vary greatly based on the extent of the effusion and the rapidity of fluid build-up. Smaller effusions may be silent and detected by chance through diagnostic procedures conducted for other purposes. Symptoms associated with larger effusions or cardiac tamponade include chest pain,

dyspnea, fatigue, and cough. Other symptoms that may occur with large effusions or cardiac tamponade include hypotension, tachycardia, increased jugular vein distention, and distant heart sounds. Collectively, these symptoms represent Beck's triad, which is characteristic but not universal of cardiac tamponade. Other symptoms that may be seen with pericardial effusions include pulsus paradoxus, which is characterized by a marked reduction in systolic blood pressure during inspiration.(Revista De La Asociación & Americana, n.d.)

Diagnosis of pericardial effusion mainly depends on the imaging methods, and echocardiography is considered the most reliable test. The test is quite sensitive, detecting even a small amount of fluid in addition to the hemodynamic effects of the effusion. Chest X-rays can also be helpful in patients presenting with large effusions because they will demonstrate an increased cardiac silhouette. Electrocardiogram (ECG) findings may indicate low-voltage QRS complexes and electrical alternans. Computed tomography (CT) and magnetic resonance imaging (MRI) can give more detailed anatomical data and also help in finding the cause. Investigations that include inflammatory, renal, thyroid, and autoimmune studies are essential to determine the cause of the effusion.(Yamani et al., 2022b)

Pericardial effusion management is determined by the level of seriousness of the problem, symptoms presented, and the underlying cause of the condition. Mild cases of pericardial effusion can be managed without any invasive measures through periodic monitoring and management of the cause. Pericarditis can be treated with anti-inflammatory medication. Large pericardial effusions, on the other hand, will call for removal of fluid to prevent cardiac tamponade and death of the patient. It can be done with the help of pericardiocentesis, whereby an incision is made at the pericardium to drain the excess fluid from the sac. For recurrent or cancerous pericardial effusion, surgery like the creation of pericardial window or removal of the entire sac (pericardiectomy) may be needed.(Yamani et al., 2022b)

The pericardial effusion has far-reaching implications on the health of patients because of the many conditions it can cause. Its clinical significance, therefore, stems from the potential consequences that arise if proper measures are not taken early

enough. Understanding the pathogenesis, diagnosis, and treatment of the disease is crucial in dealing with it effectively.(Ebrahimi et al., 2025b)

1.4.1 Mechanisms Leading to Pericardial Effusion:

Development of pericardial effusion is caused by disruption in physiological regulation of formation and absorption of pericardial fluid. Normally, the pericardial cavity maintains a stable volume of serous fluid via balance between fluid filtration from capillaries and reabsorption by lymphatics. This balance is achieved due to hydrostatic and oncotic pressure, capillary permeability, and patency of lymphatics. In case of imbalance in this system, excess fluid starts accumulating within the pericardium, causing development of pericardial effusion. The causes of pericardial effusion are either related to excessive fluid production or decreased fluid absorption or their combination.

One of the most frequent pathophysiologic factors that cause pericardial effusion is high capillary permeability, often seen in conjunction with pericarditis, the inflammation of the pericardium. This inflammation can occur due to infections, including viral, bacterial, and tuberculous; autoimmune reactions; or after myocardial infarction. In cases of inflammation, different mediators, such as cytokines, histamine, and prostaglandins, are released, which leads to vasodilation and high capillary permeability within the pericardium. As a result, the substances present in the vessels penetrate into the pericardial space and cause an exudative pericardial effusion. Usually, these types of effusions contain large amounts of protein and cells, and sometimes also involve fibrin deposition, which further complicates the case through the creation of adhesions between the layers of the pericardium. The inflammatory mechanism plays an important role since it is not only related to fluid accumulation in the pericardium but also its structural changes.

Increased hydrostatic pressure in the pericardial capillary system is another key method that leads to accumulation of fluid by transudation. This is usually observed in clinical situations like congestive heart failure due to the high venous pressure leading to accumulation of fluid in the capillaries that will leak into the pericardial cavity. Clinical situations such as high systemic venous pressure, for example pulmonary hypertension and constrictive cardiac conditions, can also be involved. The fluid usually accumulated in this condition is devoid of protein and inflammatory cells making it different from

exudative fluid. Accumulation of this type of fluid usually takes time and allows stretching of the pericardium thus delaying clinical manifestations even with a considerable amount of fluid accumulated.

Low oncotic pressure is another cause of pericardial effusion. Oncotic pressure, which is mainly regulated by plasma proteins like albumin, ensures that fluid remains in the vascular system. The loss of oncotic pressure may occur in disorders like nephrotic syndrome, liver cirrhosis, or poor nutrition where there will be a significant reduction in plasma protein content leading to low oncotic pressure. This causes the movement of fluid out of the vascular system to neighboring tissues, including the pericardial cavity. Pericardial effusion due to low oncotic pressure is transudate in nature, but it is possible for there to be fluid retention in other cavities of the body as well.

Lymphatic obstruction or impairment is yet another important causative factor for pericardial effusion. Lymphatics serve an important function in eliminating any excess fluid from the pericardial cavity. However, when there is lymphatic obstruction or impairment, even the production of fluids at normal levels can lead to effusion. This process is generally seen in malignancies where the growth of tumors in lymphatic vessels causes obstruction or impairment of drainage. Malignant tumors like lung cancer, breast cancer, or lymphoma usually cause malignancy in pericardial effusion. Such pericardial effusion is characterized by the presence of malignant cells and is resistant to conventional treatments.

Another cause of fluid retention that arises from injury is direct injury to the pericardium. Injury can be from either blunt trauma or penetration trauma. This injury causes the person to bleed into the pericardium, forming a condition known as hemopericardium. Cardiac surgery, catheterization, and pacemaker insertion can also damage the pericardium and cause effusion. In this case, the formation of effusion is rapid, and there is a chance that it will progress to tamponade because of the quick build-up of pressure inside the pericardium. The effusion formed is usually hemorrhagic and requires immediate attention.

There are metabolic disorders that can cause pericardial effusion as well. Uremia is one of those conditions where pericarditis occurs and effusion follows soon after. It is caused by advanced kidney failure that causes the person to have metabolic toxins in

their body, which induces an inflammatory response that increases capillary permeability, causing the effusion. Hypothyroidism is yet another metabolic condition that can cause effusion. Effusion develops slowly in this case and is likely because the person loses capillary control and suffers from poor lymphatic drainage.

Infections are a significant etiologic factor, especially in developing countries, as tuberculosis is one of the major causes of pericardial effusion in these areas. Tuberculosis pericarditis causes chronic inflammation, granulomas, and an exudate that forms as a response to inflammation. Meanwhile, viral infections are more likely to occur in developed nations and cause acute pericarditis with moderate effusion. Purulent pericarditis, another form of infection-caused pericardial effusion, develops due to bacterial infections and is characterized by a purulent collection within the pericardial sac. The infection is usually severe and needs immediate medical intervention to avoid complications like sepsis or constriction pericarditis.

Pericardial effusions can develop in patients with autoimmune disorders and connective tissue diseases, including systemic lupus erythematosus and rheumatoid arthritis. These diseases affect pericardial cells due to immune responses; thus, the body attacks the healthy tissue and increases vascular permeability, resulting in fluid accumulation. Exudates are usually associated with autoimmune diseases and tend to recur due to the chronic nature of the underlying condition.

In conclusion, the causes of pericardial effusion are many and varied and can be due to multiple factors. They include the increased permeability of blood vessels, raised pressure within blood vessels, decreased oncotic pressure, poor lymphatic drainage, tissue injury, metabolic disorders, infections, and autoimmune disorders among others. It is important to understand the various causes of pericardial effusion because they help determine the cause and therefore the best treatment method. The understanding of these causes will also enable clinicians to predict the progression and complications of the disease and intervene accordingly.

1.4.2 Clinical Significance and Hemodynamic Effects of Pericardial Effusion:

Clinical relevance of pericardial effusion arises from the wide spectrum of hemodynamic consequences associated with this condition, extending from incidental findings in completely asymptomatic patients to conditions with the potential for causing acute

circulatory collapse and death. The clinical relevance of pericardial effusion does not just involve the presence of an abnormality within the pericardial sac, but rather the physiological effects of this abnormality on cardiac filling, ejection volumes, and general circulation. Pericardium is a very low-pressure compartment, and even minute pressure alterations caused by pericardial fluid can have serious implications for cardiac function. Severity of symptoms is directly related to the rate of accumulation of fluid, the volume of the collected fluid, and the compliance of the pericardium itself.(Mori et al., 2024b)

One of the most significant clinical consequences of pericardial effusion is its impact on diastolic filling of the heart. The heart operates based on the principles of pressure pump mechanics, and sufficient filling of the heart cavities is necessary to maintain adequate stroke volumes and cardiac output. With pericardial effusion, as there is an increase in the volume of accumulated fluid in the pericardial sac, the intrapericardial pressure starts increasing. At first, it can accommodate small volumes without significant increases in pressure because of the limited elasticity of the tissue. Once it is exceeded, even minimal further increases in the volume cause rapid pressure growth. This pressure starts affecting the cavities of the heart by compressing them. Since the walls of the right atrium and ventricle are thin, they are especially vulnerable to this pressure growth and thus reduce venous return.

The decrease in stroke volume directly causes a reduction in cardiac output, which impacts the systemic circulation in many ways. The body tries to counteract the reduction by stimulating the sympathetic nervous system, causing tachycardia and vasoconstriction. These compensatory actions work well at first but gradually fail as the effusion worsens. Eventually, hypotension and shock symptoms occur due to inadequate compensatory efforts. This condition is a significant decline in hemodynamic status that can quickly lead to cardiovascular failure if not managed appropriately. (Ebrahimi et al., 2025b)

One of the essential hemodynamic features observed in pericardial effusion is the condition known as ventricular interdependence. As both ventricles are located within the single pericardial sac, any changes in the pressure level affecting one ventricle necessarily result in pressure changes for another ventricle. When the pericardium contains an abnormal amount of fluid, there is pressure on the right ventricle when this

chamber is relaxing. Thus, the septum separating these two chambers moves to the left chamber, reducing its filling, thus causing stroke volume and cardiac output to fall. This process becomes especially prominent when breathing is performed. Usually, inspiration results in filling up the right ventricle due to venous return increase. But in case of pericardial effusion, the rigidity of the pericardial sac does not allow filling and increases septal shift; thus, reducing cardiac output even more. This process results in pulsus paradoxus formation, i.e., the excessive drop of systolic pressure during inspiration.(Revista De La Asociación & Americana, n.d.)

Another important clinical consequence of pericardial effusion is its impact on venous return. With the increase in pericardial pressure, there will be a point where this pressure will start acting against the usual pressure gradient necessary for the entry of venous blood into the right atrium. The result is an increase in central venous pressure, which manifests as jugular venous distention. Increased venous pressure is also a cause of systemic congestion, leading to peripheral edema, hepatomegaly, and ascites when it gets very severe. All these changes are most pronounced in situations where pericardial effusion reaches the level of cardiac tamponade.

Respiratory variability in cardiac filling is another important hemodynamic change caused by pericardial effusion. Usually, during inspiration, negative intrathoracic pressure enhances venous return and right ventricular filling. But with pericardial effusion, the presence of non-compressible pericardium limits cardiac filling. Thus, there will be more exaggeration of respiratory variability of ventricular filling. The result is a reduction in stroke volume on inspiration, contributing to pulsus paradoxus.

In addition to cardiac output and venous return, pericardial effusion affects coronary perfusion. Coronary perfusion mainly takes place in diastole and adequate perfusion pressure is necessary for providing myocardium with oxygenated blood. Increased pericardial pressure can compress coronary arteries, specifically the arteries lying in subepicardial area, thus, decreasing coronary blood flow. It can contribute to myocardial ischemia in patients suffering from coronary artery disease. Decreased coronary perfusion results in decreased myocardial contractility, leading to another feedback mechanism worsening the situation and contributing to hemodynamic instability.(Ebrahimi et al., 2025b)

The clinical presentation of pericardial effusion varies from mild to severe manifestations. The small effusion may not be symptomatic and detected accidentally while performing echocardiography for another condition. The moderate effusion is presented with rather non-specific complaints, such as dyspnea, chest pain, fatigue, and coughing. These symptoms are not very noticeable and could be related to some cardiopulmonary disorders. The large effusion with rapid accumulation is presented with more severe symptoms that progress up to cardiac tamponade. The cardiac tamponade is marked by hemodynamic changes that are threatening for patient's life and include hypotension, tachycardia, increased jugular veins and muffled heart sounds, collectively called Beck's triad.

Another significant aspect in regard to pericardial effusion is its connection to certain underlying systemic diseases. In many instances, the presence of a pericardial effusion might be the key to diagnosis of such illnesses as malignancy, tuberculosis, autoimmune disorder, or renal failure. Thus, recognizing pericardial effusion is important not just for assessing cardiac problems but can give valuable information about other systemic diseases that the patient might suffer from. Consequently, it is necessary to conduct more research into the origins of the effusion as a clinical finding.(Ebrahimi et al., 2025a)

In summary, one can say that the hemodynamic consequences caused by the presence of a pericardial effusion can be attributed to intrapericardial pressure that inhibits the normal ventricular filling and decreases stroke volume, cardiac output, and ultimately affects the circulation system as a whole. Hemodynamic changes resulting from the presence of a pericardial effusion depend on how quickly and much the fluid accumulates and the stiffness of the pericardium itself. Pericardial effusions clinically vary from being asymptomatic up to causing heart tamponade.(Yamani et al., 2022a)

1.4.3 Diagnosis and Imaging of Pericardial Effusion

The diagnosis of pericardial effusion is a crucial point in the workup of the patient presenting with unexpected signs or symptoms concerning his cardiopulmonary system. Since the symptoms of pericardial effusion can vary widely, from totally asymptomatic cases to those with hemodynamic collapse caused by cardiac tamponade, a very high degree of suspicion is needed in diagnosing this disease. The diagnostic procedure

includes several stages such as clinical evaluation, testing, and investigation of the patient in order to diagnose the existence of fluid in the pericardium as well as the underlying cause of pericardial effusion. Imaging techniques have the key role in making this diagnosis.

Diagnosis starts with the clinical evaluation process. The patient's history might indicate that there are complaints like dyspnea, chest pain, tiredness, cough, or orthopnea. In a worse scenario, the symptoms might include hypotension, syncope, or even shock indicating possible cardiac tamponade. During physical assessment, there can be muffled or diminished heart sounds due to insulation from the pericardial effusion between the heart and the anterior chest wall. An increase in jugular venous pressure indicates venous obstruction whereas tachycardia is the compensatory mechanism for low cardiac output. At times, the pulsus paradoxus can be evident which is an increased fall in systolic blood pressure during inspiration which is very indicative of hemodynamic instability. However, despite all these findings, further tests must be conducted. The echocardiogram is the most crucial and commonly employed diagnostic test for pericardial effusion. This examination is non-invasive, easily accessible, and very effective in recognizing even minimal pericardial effusion. Transthoracic echocardiography is capable of visualizing the pericardial cavity directly and recognizing any pericardial effusion as an echo-free area between the visceral and parietal layers of the pericardium. The amount of effusion may be assessed according to the width of the echo-free space between the layers in the diastolic phase; therefore, effusions can be categorized as small, moderate, or large based on the measurement of the pericardial separation. Furthermore, this imaging test also offers essential clues regarding the hemodynamic effect of pericardial effusion. Another important finding on echocardiography in cases of cardiac tamponade is the right atrial collapse during systole and right ventricular collapse during diastole. This finding is secondary to an increased pressure in the pericardial sac that is more than the pressure inside the heart cavities, causing compression of the thin-walled structures of the right side of the heart. Other echocardiographic findings include dilatation of the inferior vena cava with low respiratory variation, which implies an increase in the central venous pressure. Abnormal respiratory variation in the mitral and tricuspid inflow velocities can also be observed

using Doppler echocardiography, indicating ventricular interdependence and abnormal filling.

Sometimes, transthoracic echocardiography may not offer enough information regarding the presence and extent of the pericardial effusion, especially in obese patients, those who require mechanical ventilation, and critically ill patients. In such situations, transesophageal echocardiography (TEE) may be performed to have more detailed images of the pericardium. TEE has greater sensitivity to detect small and loculated effusions and is indicated for critical care patients. Another imaging method that is widely employed in the assessment of pericardial effusion is chest radiography, but this technique is less accurate when compared to echocardiography. For patients who have medium and large-sized effusions, chest radiography will help identify an enlarged, globular appearance of the heart, which is referred to as a "water bottle heart." This occurs as a result of the accumulation of fluid around the heart, which makes it assume an abnormal shape. Chest radiography is unable to identify the presence of small-sized effusions, meaning that a normal chest radiograph cannot rule out the existence of fluid in the pericardium. Another diagnostic test that helps determine pericardial effusion is electrocardiography (ECG). Although the results of an ECG do not specify the diagnosis, they may give additional clues about its existence. For example, one of the most characteristic signs of significant pericardial effusion is the presence of low voltage QRS complex, which develops because of the insulating effect of the fluid around the heart. In some cases, electrical alternans may be detected, with alternating amplitude of the QRS complex from beat to beat, caused by swinging movements of the heart inside a sac filled with fluid. Furthermore, sinus tachycardia, which is a nonspecific symptom, is often observed. If there is accompanying pericarditis, diffuse ST segment elevation and PR segment depression are also characteristic features. Newer modalities such as CT and MRI give an accurate anatomical picture and are useful when there is diagnostic uncertainty or complexity. CT scan is very effective in quantifying the amount of fluid in the pericardium and in detecting related changes such as thickened pericardium, calcified or mass lesions. It is especially important in diagnosing malignant effusions and examining the mediastinum. MRI gives excellent contrast resolution and is

ideal for studying pericardial inflammation, fibrosis, and hemodynamics. It is very useful in distinguishing constrictive pericarditis from restrictive cardiomyopathy.

In addition, laboratory investigations have an essential role to play in the diagnostic assessment of pericardial effusion in relation to its etiology. Blood tests may involve complete blood count, inflammation markers, including ESR and CRP, renal and hepatic function tests, thyroid function tests, and autoimmune tests. High levels of inflammation markers may imply that the etiology involves either infection or autoimmune factors, whereas renal dysfunction may imply that the pericardial effusion is uremic. In cases where infection and cancer are involved, pericardial fluid analysis by pericardiocentesis will be necessary.(Yamani et al., 2022a)

1.4.4 Management Overview of Pericardial Effusion

Management of Pericardial Effusion is a sequential procedure which involves many considerations such as the volume of the effusion, the velocity at which fluid is accumulating, the status of hemodynamic stability, and etiology. Major objectives in managing this condition include stabilizing the patient if an acute condition exists, releasing the cardiac pressure if necessary, treating the underlying disease, and preventing future occurrences. Since this condition occurs in a continuum where one end represents mild and asymptomatic fluid retention, and the other end represents fatal cardiac tamponade, it calls for personalized management of this condition. For patients with pericardial effusions that are relatively small and asymptomatic, as well as hemodynamically stable, conservative treatment is generally considered adequate. Conservative treatment involves close surveillance and follow-up without any invasive procedures. Serial echocardiograms may be conducted to determine whether the effusion is increasing or improving over time. In these situations, it is necessary to consider only the causes of the problem and not the accumulation of fluid in itself. For instance, when pericardial effusion is a result of a virus infection, it can be managed by giving support and anti-inflammatory drugs. If the effusion is as a result of renal failure, improved dialysis will cause the effusion to improve.(Yamani et al., 2022c)

When there is evidence of inflammation along with pericardial effusion, anti-inflammatory treatment becomes the cornerstone of medical management. Nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used as primary drugs

because of their efficacy in reducing inflammation and helping relieve chest pain. Colchicine may be used alongside NSAIDs as secondary medication when treating recurring or idiopathic pericarditis-related pericardial effusion, as it is proven to reduce recurrences and improve outcomes. Corticosteroids may also be used if patients present with severe or refractory conditions and pericardial effusions linked with underlying autoimmune diseases like systemic lupus erythematosus, or if patients are unresponsive to NSAIDs and colchicine. However, corticosteroids are usually recommended for specific indications owing to the risks of recurrence and other side effects that accompany these medications. However, if there happens to be moderate to significant amounts of pericardial effusion, as well as clinical symptoms associated with it, then it becomes important to adopt an active approach towards dealing with this problem. Whether an active approach needs to be used or not will depend on the patient's clinical presentations, as well as hemodynamic instability that can be assessed using an echocardiogram. Patients that present with signs of pericardial constriction, like low blood pressure, increased heart rate, jugular venous distention, as well as pericardial chamber collapse detected from the echocardiogram, will require urgent attention for their pericardial fluid removal. Percutaneous drainage using pericardiocentesis becomes important in such cases. It entails putting in a needle into the pericardium and aspirating fluid.

Pericardiocentesis is a diagnostic and therapeutic procedure. Apart from alleviating the compression effect on the heart, the aspirate fluid can be subjected to various laboratory tests such as cytology, biochemistry, microbiology, and immunology. Laboratory tests will aid in establishing the causative factor responsible for the effusion. For instance, the identification of cancer cells in the pericardial fluid is an indication of metastasis, whereas bacteria in the fluid suggest purulent pericarditis. However, pericardiocentesis poses potential complications such as myocardial perforation, arrhythmia, and coronary artery damage. Therefore, the procedure should be conducted by trained medical professionals using proper imaging techniques. The management of pericardial effusion will also be done through managing the underlying disease that caused the excess fluid in the body. For instance, if the condition is due to an infection like tuberculosis, then the patient must undergo the appropriate antimicrobial therapy

for several months. If it is a result of bacterial infection, the use of broad-spectrum antibiotics will be initiated first before tailoring it according to culture findings. Chemotherapy, radiation, or even palliative management may also be used to manage malignancy-related pericardial effusion depending on the stage of disease. Meanwhile, in patients who have renal failure, optimizing their dialysis is very important since uremic toxins play a big role in causing inflammation of the pericardium and fluid accumulation. For those with hypothyroidism, administration of thyroid hormones leads to the gradual resolution of the problem.

In addition to medical management, supportive care is also important, especially for patients who are admitted in hospitals. It involves monitoring the vital signs, administering supplemental oxygen if needed, and monitoring the fluid status. Monitoring the cardiac function continuously should also be done for patients with unstable hemodynamics. Some patients may need to be managed in the intensive care unit (ICU).

Long-term management should include prophylaxis and monitoring for potential complications. Good prognosis can be expected in patients suffering from idiopathic or viral pericardial effusions as well as appropriate treatment; however, in the case of malignant or tuberculous pericardial effusions, the prognosis remains more problematic. Repeated clinical and imaging examination are necessary to ensure complete resolution of the effusion and early detection of its possible recurrence. Education of the patient is also important, especially in regard to symptoms which need prompt medical care due to the development of the disease or its recurrence.(Ebrahimi et al., 2025b)

MATERIAL AND METHODOLOGY

This descriptive cross-sectional study was conducted at Lady Reading Hospital over a period of 6–8 months. The sample size was calculated using OpenEpi Version 3 with a population size of 1,000,000, an assumed prevalence of 20%, a 5% margin of error, a 95% confidence level, and a design effect of 1, yielding the required sample size.

The study included all adult patients (⩾18 years) presenting to or admitted to the hospital during the study period with echocardiography-confirmed pericardial effusion of any severity (mild, moderate, or severe). Only patients with complete baseline demographic and echocardiographic data were included. Patients younger than

18 years and those with poor-quality echocardiographic images that did not allow accurate assessment of pericardial effusion were excluded.

Data were entered and analyzed using SPSS (Statistical Package for the Social Sciences). Continuous variables were summarized as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The results were presented using appropriate tables and graphical displays, with a p-value of <0.05 considered statistically significant.

RESULTS & DATA ANALYSIS

The statistical analysis of 196 patients evaluated for pericardial effusion at a tertiary care hospital provides a comprehensive demographic and clinical profile. The detailed distributions are categorized below:

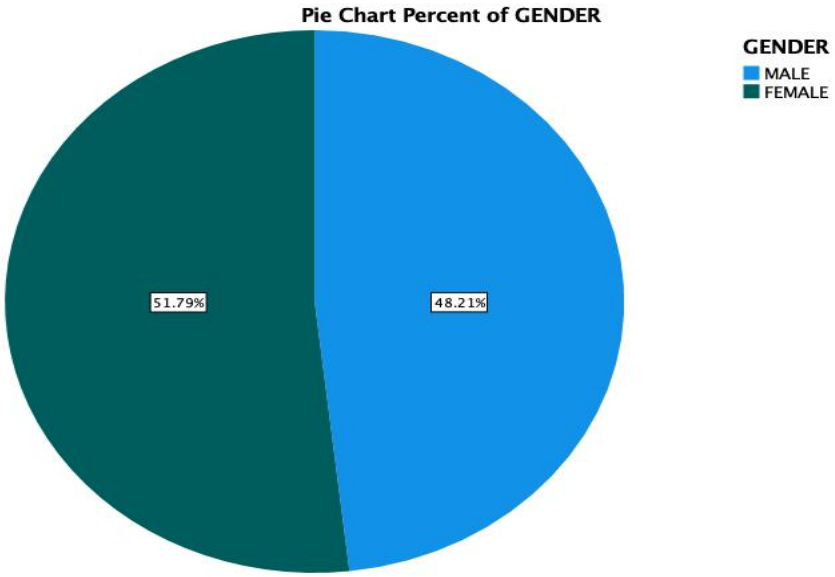
GENDER WISE DISTRIBUTION:

The data indicates a slightly higher frequency of females presenting with or evaluated for the condition compared to males.

TABLE 1: *GENDER WISE DISTRIBUTION:*

Gender	Frequency	Percent (%)	Valid (%)	Percent	Cumulative (%)	Percent
Male	94	48.0	48.2		48.2	
Female	101	51.5	51.8		100.0	
Missing System	1	0.5	Missing		-	
Total	196	100.0	100.0			

FIGURE 1: GENDER WISE DISTRIBUTION



AGE WISE DISTRIBUTION:

The age distribution shows that the condition is most prevalent in middle-aged individuals, particularly between 40 and 50 years of age.

TABLE 2: *AGE WISE DISTRIBUTION*

Age Group	Frequency	Percent (%)	Valid (%)	Percent	Cumulative (%)	Percent
30–40	31	15.8	15.8		15.8	
40–50	80	40.8	40.8		56.6	
50–60	56	28.6	28.6		85.2	
60–70	28	14.3	14.3		99.5	
56 (unclassified)	1	0.5	0.5		100.0	
Total	196	100.0	100.0			

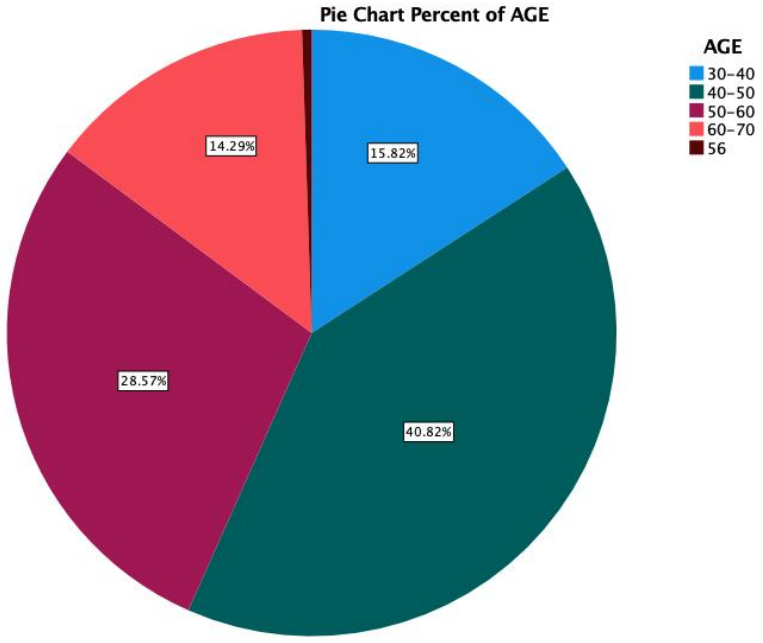


FIGURE: 2 AGE WISE DISTRIBUTION

DISTRIBUTION OF COMORBIDITIES & RISK FACTORS

Analysis of comorbidities revealed that hypertension is almost universally present in the study population, whereas clinical rates of diabetes and dyslipidemia were lower.

TABLE 3: *DISTRIBUTION OF COMORBIDITIES & RISK FACTORS*

Comorbidity / Risk Factor	Category	Frequency	Percentage (%)
Hypertension	YES	184	93.9
	NO	12	6.1
Diabetes Mellitus	YES	13	6.6
	NO	183	93.4
Dyslipidemia	YES	15	7.7
	NO	181	92.3
Smoking Status	NO	154	78.6

	YES	40	20.4
	EX-SMOKER	2	1.0

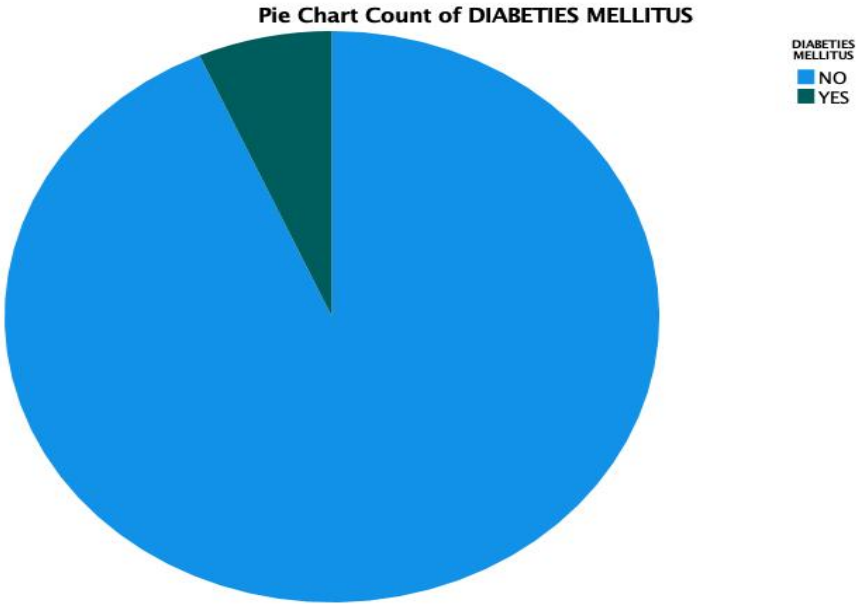


FIGURE 3: DIABETIES MELLITUS

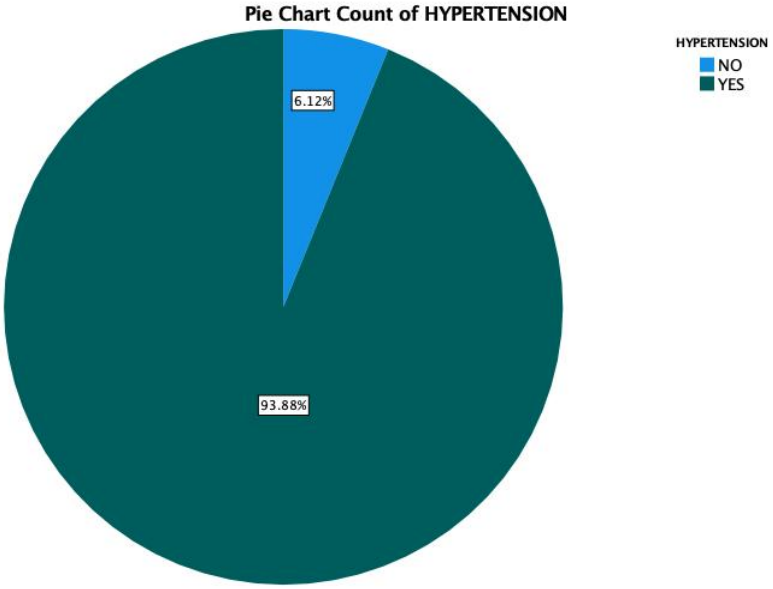


FIGURE 4.4 : HYPERTENSION

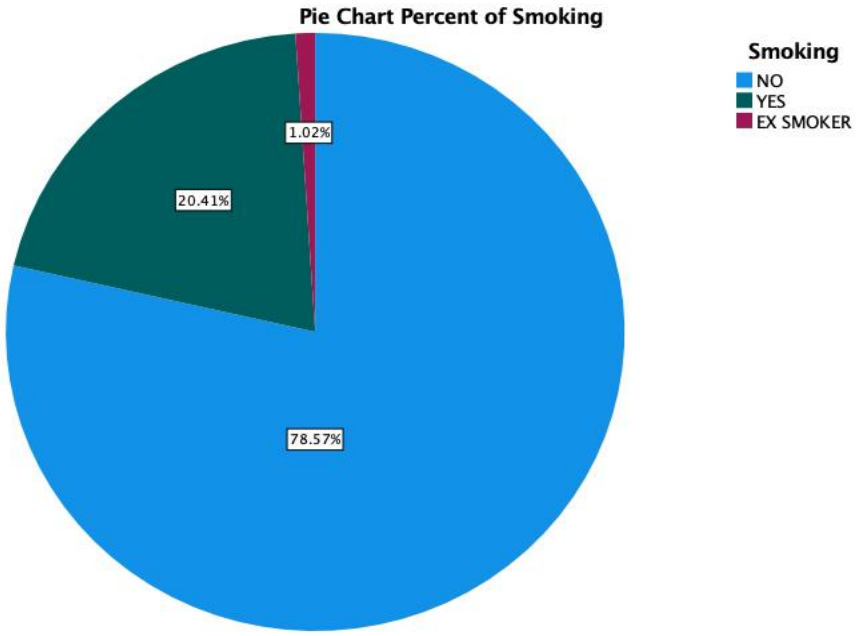


FIGURE 5: SMOKING

CARDIAC HISTORY & PROCEDURES:

A significant proportion of patients had a history of Old Myocardial Infarction, with a subset having undergone Percutaneous Coronary Intervention (PCI).

TABLE 4: *CARDIAC HISTORY & PROCEDURES*

Clinical Variable	Category	Frequency	Percentage (%)
Old Myocardial Infarction	YES	68	34.7
	NO	128	65.3
PCI History	YES	30	15.3
	NO	166	84.7
CABG History	YES	1	0.5
	NO	195	99.5

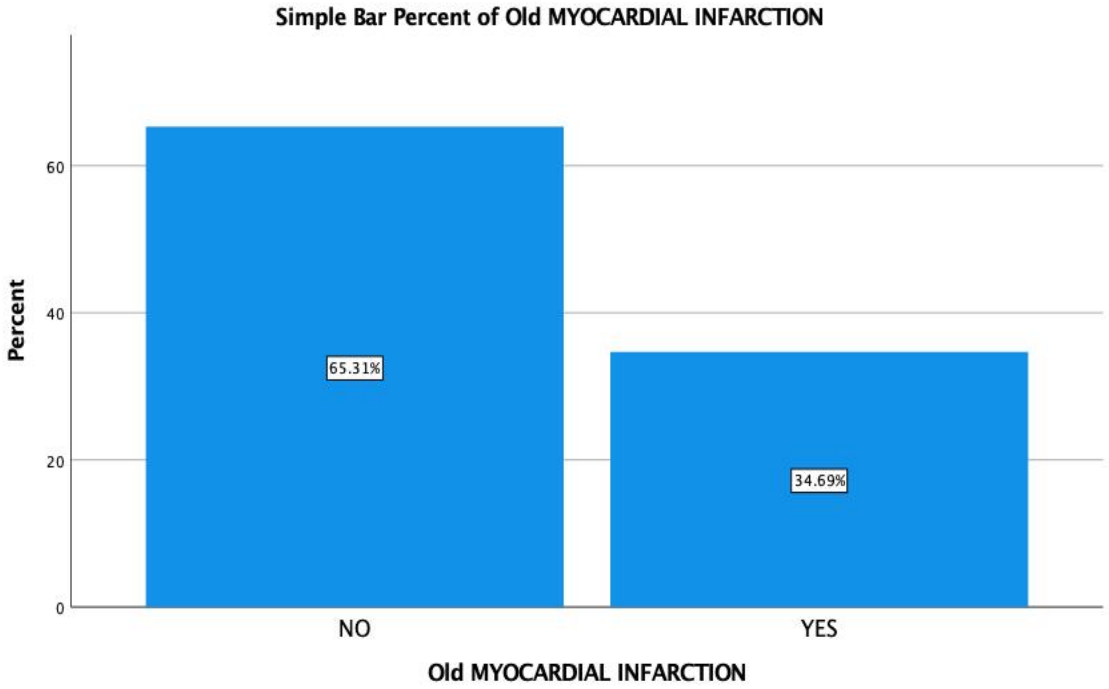


FIGURE 6 OLD MYOCARDIAL INFARCTION

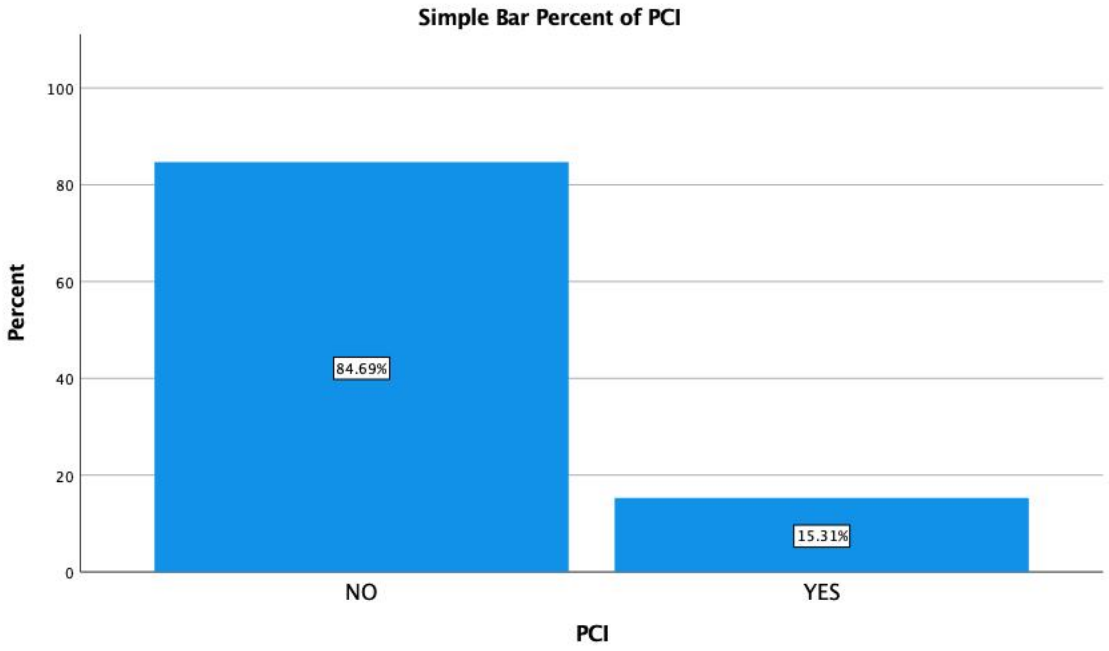


FIGURE 7 PCI

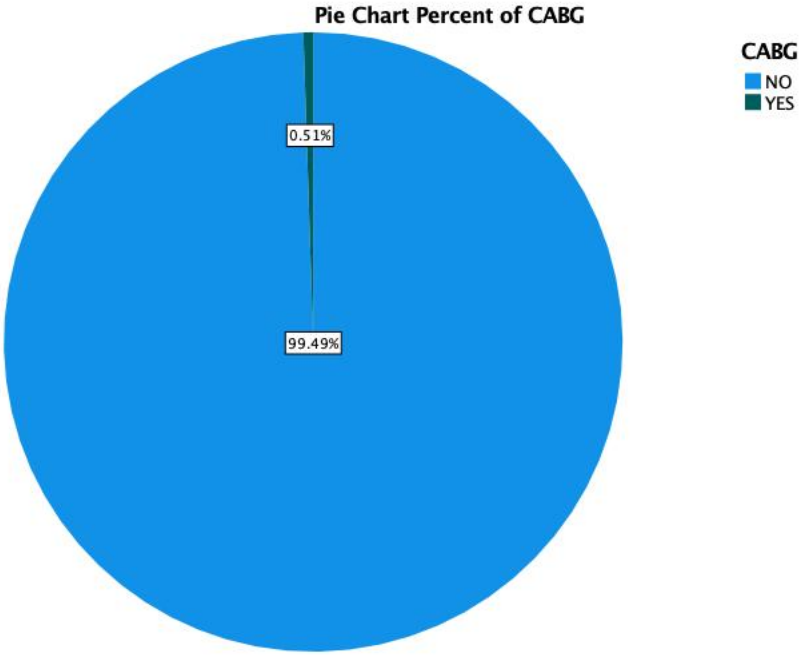


FIGURE 8 CABG

ECHOCARDIOGRAPHIC SEVERITY & HEMODYNAMICS:

Echocardiographic classification highlights that while nearly the entire analyzed cohort had fluid presence, it was predominantly mild in severity. Severe cases or progression to tamponade were rare.

TABLE 5: *ECHOCARDIOGRAPHIC SEVERITY & HEMODYNAMICS:*

Echocardiographic Variable	Category	Frequency	Percentage (%)
Pericardial Effusion Presence	YES	195	99.5
	NO	1	0.5
Mild Pericardial Effusion	YES	171	87.2
	NO	25	12.8
Moderate Pericardial Effusion	YES	25	12.8
	NO	171	87.2

Severe Pericardial Effusion	YES	21	10.7
	NO	175	89.3
Cardiac Tamponade	YES	2	1.0
	NO	194	99.0

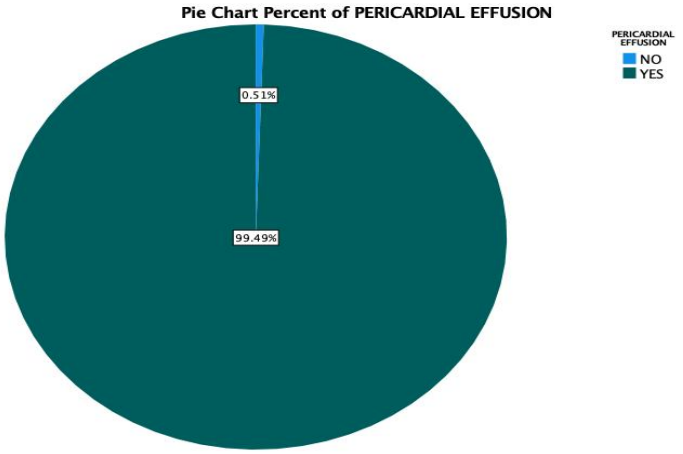


FIGURE 4.9 PERICARDIAL EFFUSION

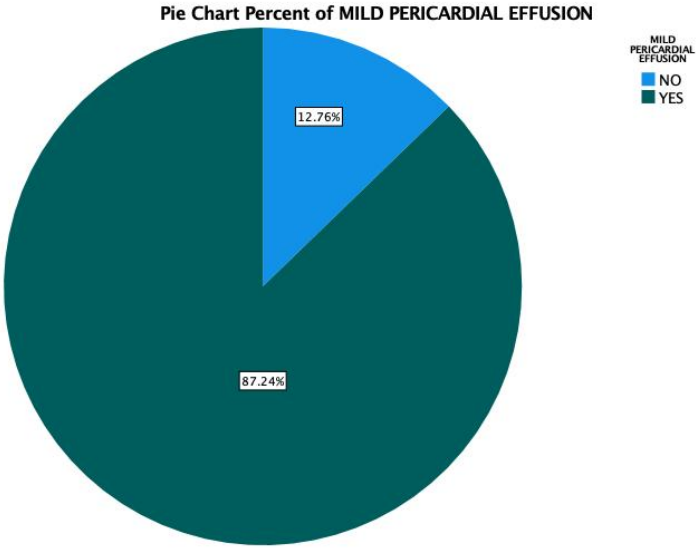


FIGURE 9 MILD PERICARDIAL EFFUSION

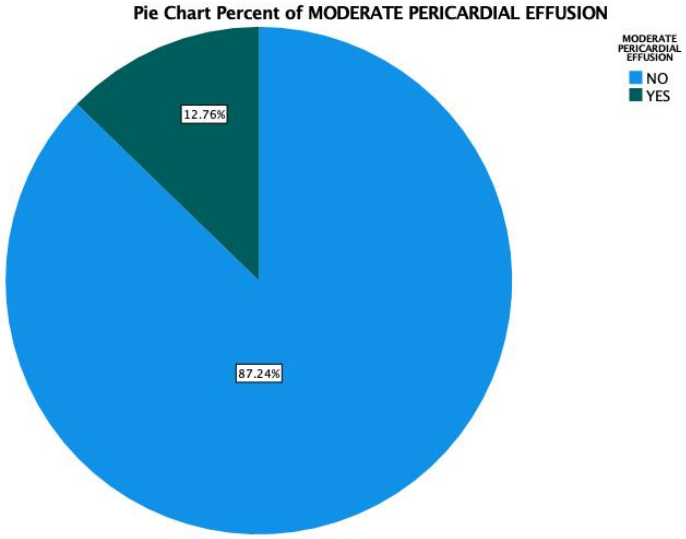


FIGURE 10 MODERATE PERICARDIAL EFFUSION

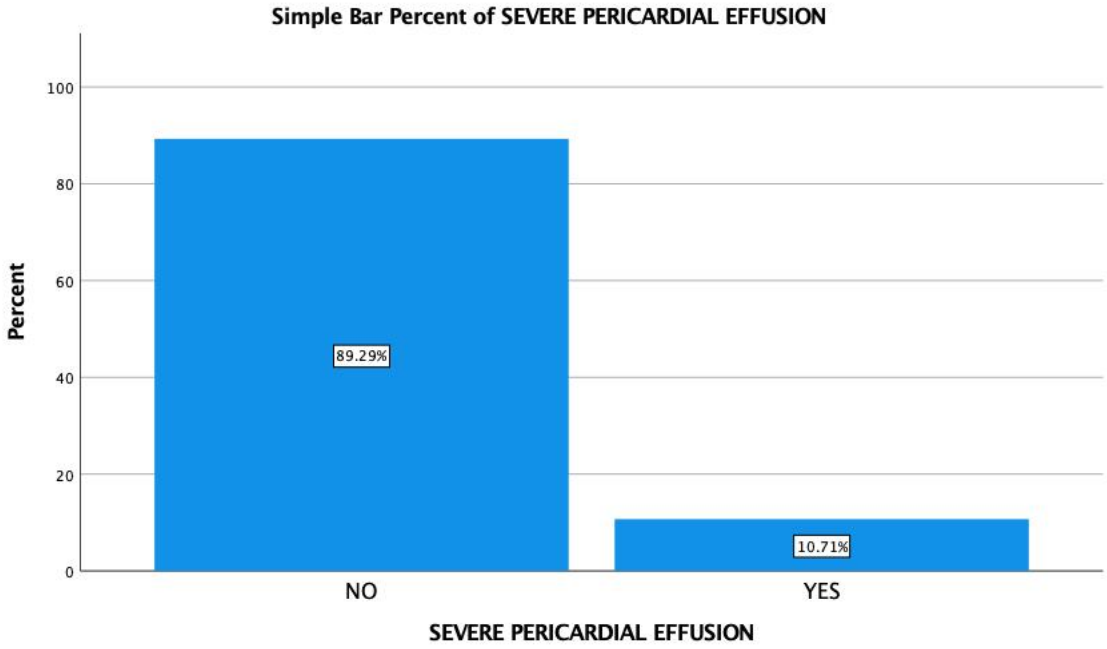


FIGURE 11: MILD PERICARDIAL EFFUSION

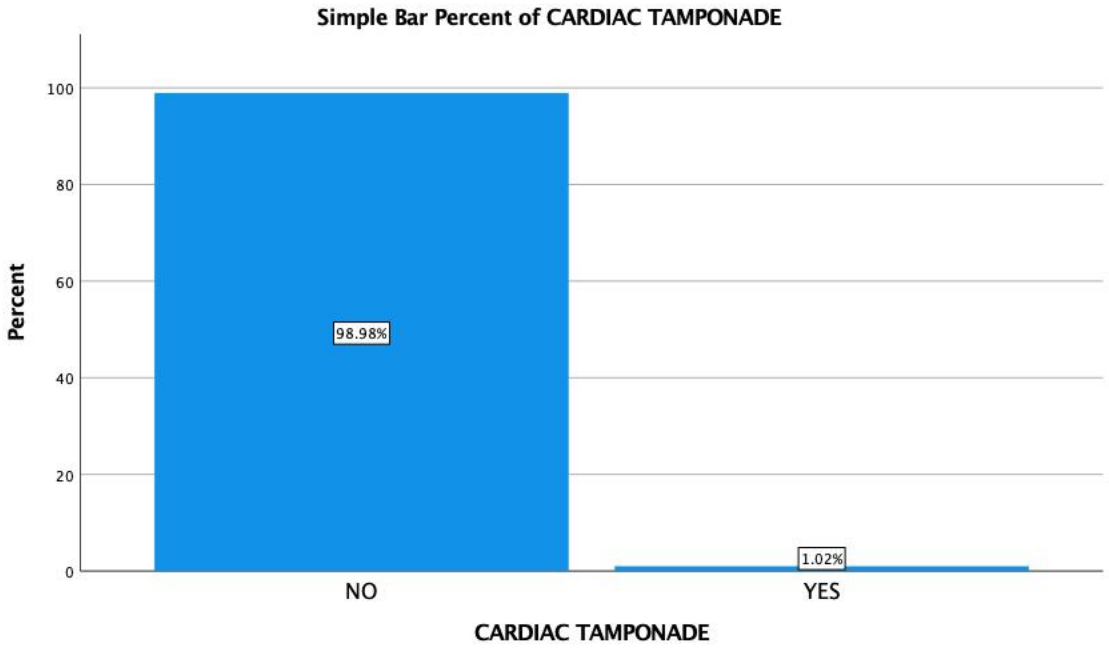


FIGURE 12 CARDIAC TEMPONADE

Left Ventricular (LV) Function & Ejection Fraction

Left ventricular impairment was present in over 40% of the sample, which may contribute to the altered hydrostatic pressures leading to effusion.

TABLE 5: *LEFT VENTRICULAR (LV) FUNCTION & EJECTION FRACTION*

LV Function Variable	Category	Frequency	Percentage (%)
LV Dysfunction	YES	83	42.3
	NO	113	57.7
Ejection Fraction (EF)	ABOVE 55	100	51.0
	45–55	40	20.4
	35–45	35	17.9
	LESS THAN 35	21	10.7

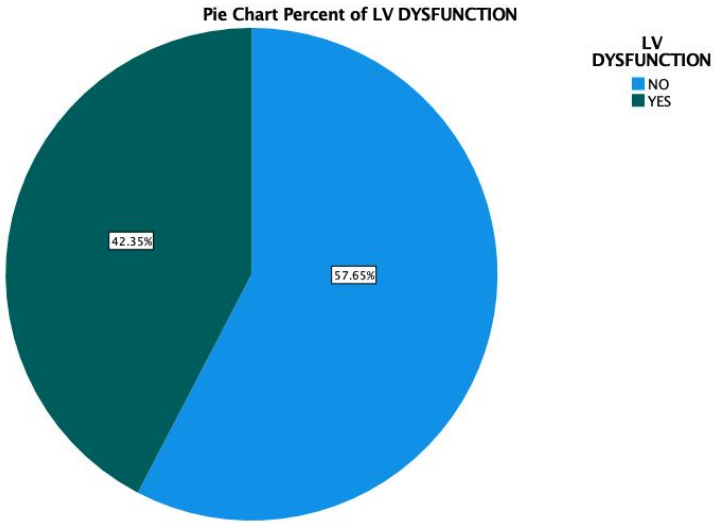


FIGURE 13 LV DYSFUNCTION

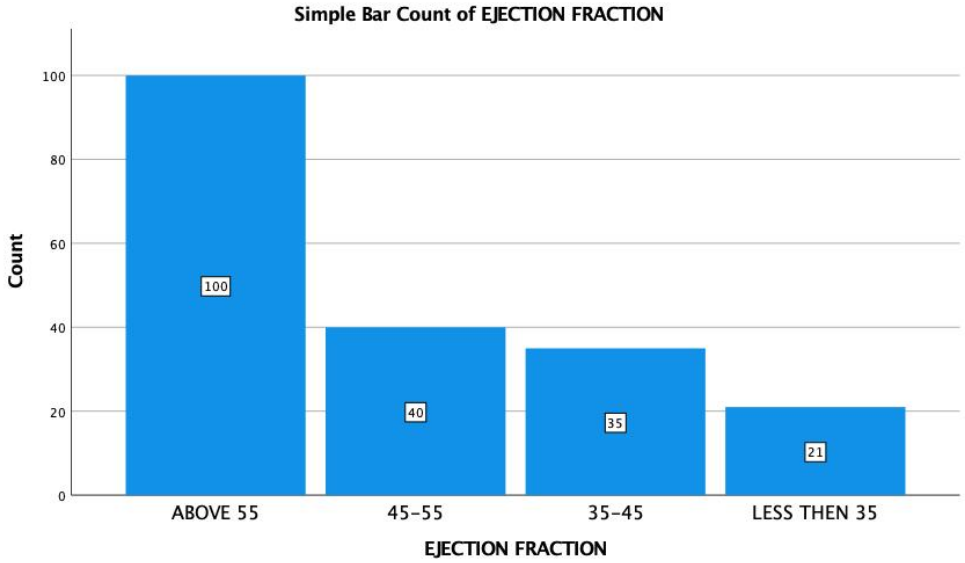


FIGURE 14 EJECTION FRACTION

DISCUSSION

The findings of this study demonstrate that pericardial effusion is a highly relevant clinical condition in tertiary care settings, showing a slight female preponderance (51.8% vs. 48.2%). This aligns with various literature suggesting that autoimmune and specific inflammatory pathways prevalent in females might increase susceptibility to fluid accumulation. The age peak between 40 and 50 years underscores the necessity of

targeted screening in the middle-aged population before advanced cardiovascular deterioration occurs.

Crucially, the statistical overlap between severe systemic hypertension (93.9%) and the high frequency of mild pericardial effusion (87.2%) indicates that elevated systemic and venous pressures heavily drive transudative fluid leakage into the pericardial space. Furthermore, the presence of left ventricular dysfunction in 42.3% of the patients supports the pathophysiological mechanism where altered hydrostatic pressures due to heart failure promote effusion development. Although a major proportion of cases remain mild and hemodynamically stable, the presence of 10.7% severe effusions and 1.0% cardiac tamponade cases highlight the critical window for rapid diagnostic imaging and therapeutic intervention (such as pericardiocentesis) to prevent cardiovascular collapse.

CONCLUSION

This study concludes that pericardial effusion in tertiary care hospital exhibits a higher frequency among females and individuals aged 40–50 years. The clinical presentation is predominantly mild fluid accumulation, strongly associated with chronic systemic hypertension and left ventricular impairment. Echocardiography remains the definitive, indispensable gold-standard tool for diagnosing, grading, and tracking the hemodynamic impacts of these effusions.

RECOMMENDATIONS

1. Mandatory Echocardiographic Screening: Implement routine echocardiographic evaluations for all patients presenting with chronic uncontrolled hypertension or significant left ventricular dysfunction to catch fluid accumulation at an early, manageable stage.
2. Targeted Monitoring for Vulnerable Demographics: Clinicians should maintain a higher index of suspicion and initiate closer follow-up for female patients and individuals in the 40–50 age range presenting with cardiopulmonary symptoms.
3. Establish Standardized Management Pathways: Develop clear hospital guidelines distinguishing conservative management for mild/asymptomatic effusions (e.g., anti-inflammatory optimization) from immediate invasive procedures (pericardiocentesis) for severe or tamponade cases.

4. Multi-Center Prospective Studies: Conduct broader, long-term multi-center trials incorporating fluid analysis (exudate vs. transudate classification) to definitively map the underlying etiologies (tuberculous, malignant, vs. idiopathic) unique to the local region.

LIMITATIONS OF THE STUDY

1. **Single Centre Study**

This study was conducted at only one tertiary care hospital, which may limit the generalizability of the findings to other hospitals or regions. Patient characteristics, disease patterns, and healthcare practices may differ across different healthcare settings.

2. **Limited Sample Size**

The study included 196 patients, which provides valuable information but may not fully represent the broader population of patients with pericardial effusion. A larger sample size would improve the statistical power and reliability of the findings.

3. **Cross-Sectional Study Design**

As this was a cross-sectional study, data were collected at a single point in time. Therefore, it was not possible to establish causal relationships or assess the long-term progression, treatment outcomes, or recurrence of pericardial effusion among the study participants.

REFERENCES

1. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases. *Eur Heart J.* 2015;36(42):2921–2964. doi:10.1093/eurheartj/ehv318.
2. Imazio M, Gaita F. Diagnosis and treatment of pericardial effusion. *Heart.* 2015;101(14):1159–1168. doi:10.1136/heartjnl-2014-306362.
3. Little WC, Freeman GL. Pericardial disease. *Circulation.* 2006;113(12):1622–1632. doi:10.1161/CIRCULATIONAHA.105.561514.
4. Sagristà-Sauleda J, Mercé J, Soler-Soler J. Diagnosis and management of pericardial effusion. *World J Cardiol.* 2011;3(5):135–143. doi:10.4330/wjc.v3.i5.135.
5. Imazio M, Adler Y. Management of pericardial effusion. *Eur Heart J.* 2013;34(16):1186–1197. doi:10.1093/eurheartj/ehs372.

6. Otto CM, Bonow RO. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine.* 12th ed. Philadelphia: Elsevier; 2022.
7. Kumar V, Abbas AK, Aster JC. *Robbins and Cotran Pathologic Basis of Disease.* 11th ed. Philadelphia: Elsevier; 2021.
8. Hall JE. *Guyton and Hall Textbook of Medical Physiology.* 15th ed. Philadelphia: Elsevier; 2024.
9. Betts JG, Young KA, Wise JA, Johnson E, Poe B, Kruse D, et al. *Anatomy and Physiology.* Houston: OpenStax; 2023.
10. Zipes DP, Libby P, Bonow RO, Mann DL, Tomaselli GF. *Braunwald's Heart Disease.* 12th ed. Philadelphia: Elsevier; 2022.
11. Yamani N, Patel N, Singh A, et al. Pericardial Effusion. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2024.
12. Mori M, Matsuura Y, Takagi H, et al. Malignant pericardial effusion: Current diagnosis and management. *J Cardiol.* 2024;83(2):105–114.
13. Qasba PK, Ahmad M, Khan A, et al. Contemporary management of pericardial effusion: A systematic review. *Cureus.* 2025;17(1):e00000.
14. Ebrahimi R, Shafiee A, Hosseini M, et al. Recent advances in diagnosis and treatment of pericardial effusion. *J Clin Med.* 2025;14(3):765.
15. Spodick DH. Acute cardiac tamponade. *N Engl J Med.* 2003;349(7):684–690.
16. Maisch B, Seferović PM, Ristić AD, et al. Guidelines on the diagnosis and management of pericardial diseases. *Eur Heart J.* 2004;25(7):587–610.
17. Ristić AD, Imazio M, Adler Y, et al. Triage strategy for pericardial effusion. *Heart.* 2014;100(24):1968–1974.
18. Hoit BD. Pericardial effusion and cardiac tamponade in the new millennium. *Curr Cardiol Rep.* 2017;19(7):57.
19. Hancock EW. Subacute effusive-constrictive pericarditis. *Circulation.* 1971;43(2):183–192.
20. Levy PY, Corey R, Berger P, Habib G, Bonnet JL, Levy S, et al. Etiologic diagnosis of pericardial effusion in adults. *Arch Intern Med.* 2003;163(5):567–573.
21. (Ezquerro et al., 2024)Ezquerro L, Coimbra R, […] Moreno-Azanza MGeoscience Frontiers (2024) 15(5)

22. (Qasba et al., 2025)Qasba R, Cangut B, [...] Hameed IJournal of Clinical Medicine
23. (Betts et al., n.d.)Betts J, College T, [...] Young K
24. (Ebrahimi et al., 2025a)Ebrahimi P, Taheri H, [...] Shahid FJournal of Cardiothoracic Surgery
25. (Yamani et al., 2022a)Yamani N, Abbasi A, [...] Unzek SAnnals of Medicine and Surgery
26. (Mori et al., 2024a)Mori S, Bertamino M, [...] Ameri PCardio-Oncology