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Echoes of Deception: A Chronicle of Unusual Ventricular Pseudoaneurysm- A Case Series

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ABSTRACT

Background: Infective endocarditis (IE) is an infection involving the heart valves or the endocardial surface, typically resulting from bacteraemia. It can lead to serious complications, including perforation of the valve leaflets, annular abscesses, formation of fistulous tracts, and destruction of adjacent cardiac structures such as the subaortic region.[1,2]

Case Presentation: This case series illustrates an atypical and fatal complication of IE, which is left ventricular pseudoaneurysm (LVPA), an uncommon cardiac lesion that can be overlooked during life and often identified during postmortem examination due to its asymptomatic progression.[3,4] We present two cases of left ventricular pseudoaneurysms associated with fibrotic valvular vegetations and myocardial damage consistent with previously unrecognized infective endocarditis in apparently healthy males, revealed during the medicolegal autopsy.

Conclusion: These findings highlight the silent but deadly course of LVPA secondary to IE and emphasize the importance of vigilance in at-risk patients. Timely imaging and surgical management may prevent catastrophic rupture.[4,5] Autopsy remains vital in uncovering such silent, fatal complications.[6] We also suggest implications for public health and clinical awareness.

Keywords: Left ventricular pseudoaneurysm, infective endocarditis, aortic valve, sudden cardiac death, autopsy.

INTRODUCTION

Infective endocarditis (IE) is a condition caused by a bacterial infection affecting the heart valves or the endocardium.[7] It has an estimated incidence of 3–10 episodes per 100,000 person-years in the general population.[1] The disease occurs predominantly in older adults and individuals with underlying valvular or congenital heart disease, prosthetic heart valves, or indwelling cardiac devices.[1,2]

Complications of IE can be broadly categorized into cardiac and extracardiac manifestations. When the aortic valve is involved, it can lead to complications such as perforation of the valve, formation of a ring abscess, development of a fistula, or damage to the structures beneath the aortic valve in adjacent myocardial tissues.[2] One of the rare but life-threatening cardiac complications of infective endocarditis is the formation of a left ventricular pseudoaneurysm (LVPA).[8]

The rupture or perforation of the left ventricular free wall is a severe and fatal complication that usually develops within three to five days following an acute myocardial infarction.[4] A high risk of mortality is associated with this condition because of its high tendency to rupture into the pericardial space, leading to hemopericardium, cardiac tamponade and sudden death;[5,11] however, rare cases have documented long-term survival in patients with an unruptured left ventricular pseudoaneurysm.[3,12]

In rare cases, the formation of adhesions between the epicardium and pericardium, along with a parietal thrombus at the site of infarction, can result in a pseudoaneurysm (PSA), also called a false aneurysm.[4,13,14]

When the infection spreads beyond the valve structures into the surrounding myocardium, it can weaken the ventricular wall. Sometimes, the wall gives way, and the pericardium or fibrous adhesions temporarily contain the rupture. This leads to the formation of a pseudoaneurysm—an unstable sac that lacks the normal layered structure of the heart wall.[11,15,16] Staphylococcus aureus is the primary microorganism linked to this complication.[17] Other causative agents include nutritionally variant streptococci (NVS), such as *Abiotrophia* and *Granulicatella,* responsible for approximately 5–6% of streptococcal endocarditis cases.[8,18]

A true left ventricular aneurysm develops when the ventricular wall becomes thin and bulges outward while maintaining the integrity of all three layers of the heart, i.e. endocardium, myocardium, and epicardium. It typically forms after a myocardial infarction and communicates broadly with the left ventricle. The risk of rupture is low due to its fibrotic composition. On the other hand, a false or pseudoaneurysm arises when a rupture in the ventricular wall is sealed off by the pericardium or scar tissue, forming a cavity with a narrowed neck.[3,4] Early identification using imaging techniques such as echocardiography, CT scans, or cardiac MRI is essential, as pseudoaneurysms often require prompt surgical intervention to prevent life-threatening rupture.[5,11]

We present two cases of left ventricular pseudoaneurysm associated with infective endocarditis, diagnosed during medicolegal autopsy. Both patients were previously undiagnosed and presented with sudden cardiac death. These cases illustrate the importance of recognizing rare complications of IE and highlight the indispensable role of autopsy in revealing such fatal conditions. We also aim to create awareness of its subtle clinical symptoms, such as common constitutional symptoms of IE, which were missing in these cases, diagnostic modalities, infective cause, and autopsy findings.

CASE: 1

A 26-year-old male was brought to a hospital with a history of sudden chest pain followed by loss of consciousness at home. He was brought dead to the emergency department. The corpse was shifted to the mortuary for a medicolegal autopsy examination. History from the family members revealed that he was a normal, healthy young individual with no known medical illness before the episode of chest pain.

An autopsy examination showed a well-built male with no congenital deformity seen externally. An infected open wound measuring 3cm x 3cm x tissue deep was seen on the shin of the left leg as shown in figure 1e. Postmortem changes such as lividity and rigor mortis were present. Cyanosis of the lips and nail beds of both hands was present. The oral cavity showed poor oral hygiene. Findings of the skull and the brain did not show any gross pathology. On the opening of the thoracic cavity, around 200 mL of straw-coloured fluid was found on both sides of the cavity, and both lungs were consolidated and adherent to the cavity at places with fibrous bands. The pericardium was intact with minimal serous effusion. The heart weighed 450 grams, and the anterior surface of the heart exhibited a noticeable bulge, with the upper anterior portion of the left ventricle below the atrioventricular junction, which was soft and slightly fluctuant upon pressure as shown in figure 1b. On sectioning, large irregular nodular vegetations were identified on the aortic valve's left and right coronary cusps as shown in figure 1a. The largest vegetation was seen, measuring 2 cm × 2 cm, with complete disruption of the left coronary cusp of the aortic valve as shown in figure 1c. The right coronary cusp displayed ulceration, thickening, and small nodular formations. Tiny fibrotic nodules on the non-coronary cusp were also noted, which were minimally affected as shown in figure 1c.

Further examination of the left coronary cusp vegetation revealed a mobile lesion with an underlying necrotic and hollow cavity concealed by the overlying vegetation. This cavity, located within the anterior wall of the left ventricular wall, measured 5 cm × 4 cm × 1 cm and contained blood clots. It was confined to the peri-annular region and myocardium without rupture to the epicardial surface as shown in figure 1c. These findings indicated a left ventricular pseudoaneurysm, a rare complication of infective endocarditis.

The coronary arteries remained patent, while the myocardium surrounding the lesion appeared soft and haemorrhagic, exhibiting dark mottling. The liver was enlarged and showed fatty changes. Spleen was enlarged and adhere to diaphragm with fibrotic band as shown in figure 1d. Examination of other internal organs, including the kidneys, and intestines, revealed congestion but no significant pathological abnormalities.

Rapid test antigen and polymerase chain reaction (RT-PCR) for COVID-19 were negative.

The cause of death was certified as cardiogenic shock as a result of aortic valve infective endocarditis complicated with peri-annular haemorrhage secondary to the left ventricular pseudoaneurysm.

Case 1:

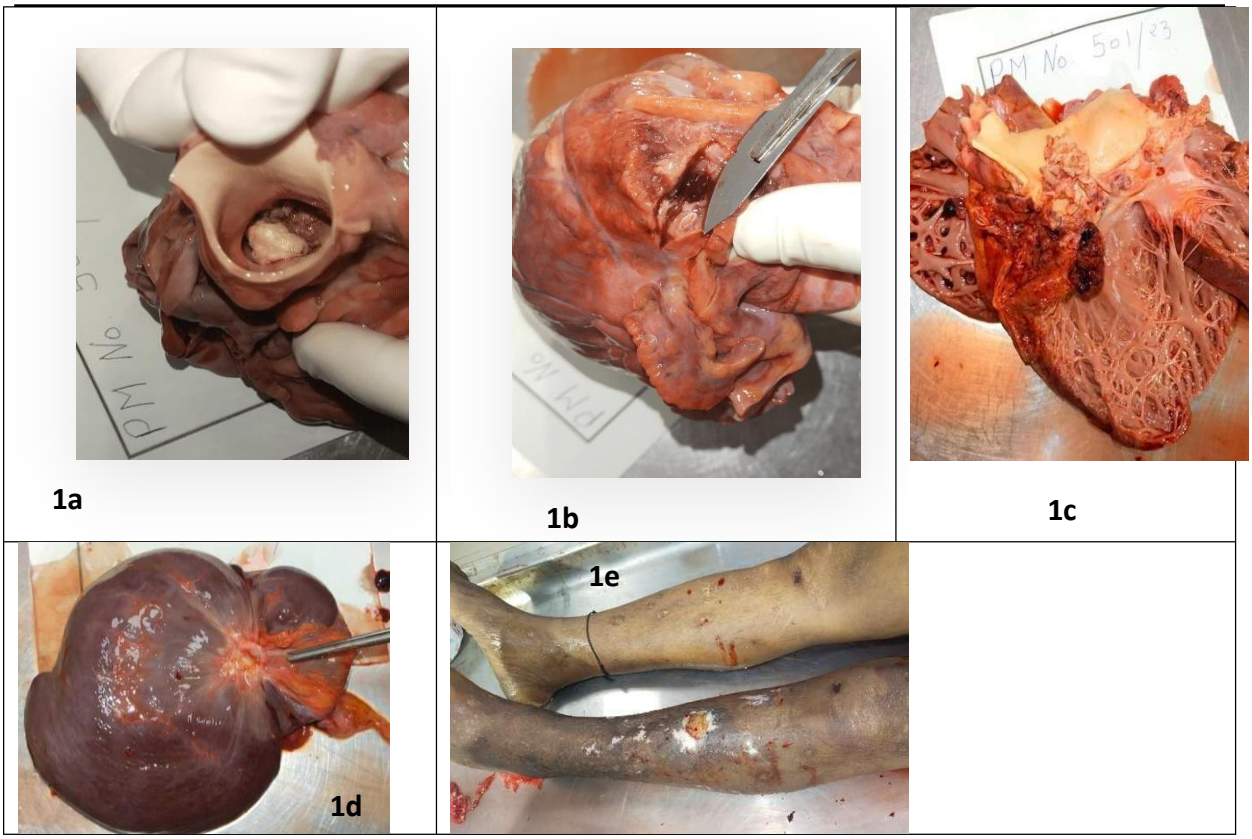

Figure 1:- Large vegetationof aortic valves (1a); Bulging sac-like pseudoaneurysm filled with infected blood clot on anterior wall of left ventricle (1b); infected lumen of pseudoaneurysm sac with infected valve, large vegetation and slit-like infected opening into the myocardium (1c); Septic feature on spleen (1d); Haemorrhagic and infected mesentery (1e).

CASE:2

A 55-year-old male was brought to a hospital with a history of sudden chest pain followed by loss of consciousness at home. He was declared dead on arrival. The corpse was shifted to the mortuary for a medicolegal autopsy examination. The corpse was later transferred to the mortuary for a medicolegal autopsy. According to family members, he had been a healthy individual with no known prior medical conditions before the onset of symptoms.

Autopsy findings revealed a well-built male with no visible injuries or congenital abnormalities. Cyanosis was noted on the lips and nail beds, suggesting oxygen deprivation. Oral cavity examination showed natural dentition with good oral hygiene. Internal examination of the skull and brain indicated an intact skull with no abnormalities in brain anatomy.

Both lungs were densely adherent to the cavity with fibrous bands and consolidated. Examination of the thoracic cavity revealed an intact pericardium filled with blood and clots (500 grams) as shown in figure 2a. The heart weighed 428 grams, and the posterior surface of the left ventricle appeared bulging, and a soft, slightly fluctuant with a 1 cm × 0.1 cm tear was identified as shown in figure 1b. Heart dissection showed irregular, nodular vegetations involving both left and right coronary cusps of the aortic valve, measuring 0.8 cm × 0.5 cm, had disrupted the non-coronary cusp as shown in figure 2d. The left coronary cusp exhibited ulceration, thickening, and small nodules, likely due to the spread of infection from an adjacent cusp. The non-coronary cusp was slightly involved, displaying a small fibrotic nodule.

Further inspection of the left coronary cusp revealed a mobile lesion with an underlying hollow, necrotic cavity concealed by vegetation. A cavity within the left ventricular wall, measuring 4.5 cm × 3 cm × 1 cm, was identified and contained blood clots as shown in figure 2d. This cavity was located within the myocardium. These findings were consistent with a left ventricular pseudoaneurysm, a rare complication of infective endocarditis.

Left anterior descending, left circumflex, and right coronary arteries showed atherosclerosis with luminal narrowing of about 50%, while the myocardium surrounding the lesion appeared soft and haemorrhagic. The anti-mesenteric surface of the intestine and mesentery were haemorrhagic and infected as shown in figure 2e.

The liver was enlarged and showed fatty changes. Examination of the spleen, and kidneys showed congestion but no significant pathological abnormalities.
Rapid test antigen and polymerase chain reaction (RT-PCR) for COVID-19 were negative.
The cause of death was certified as cardiogenic shock as a result of aortic valve infective endocarditis complicated with peri-annular haemorrhage, secondary to the pseudoaneurysm.

Case 2:

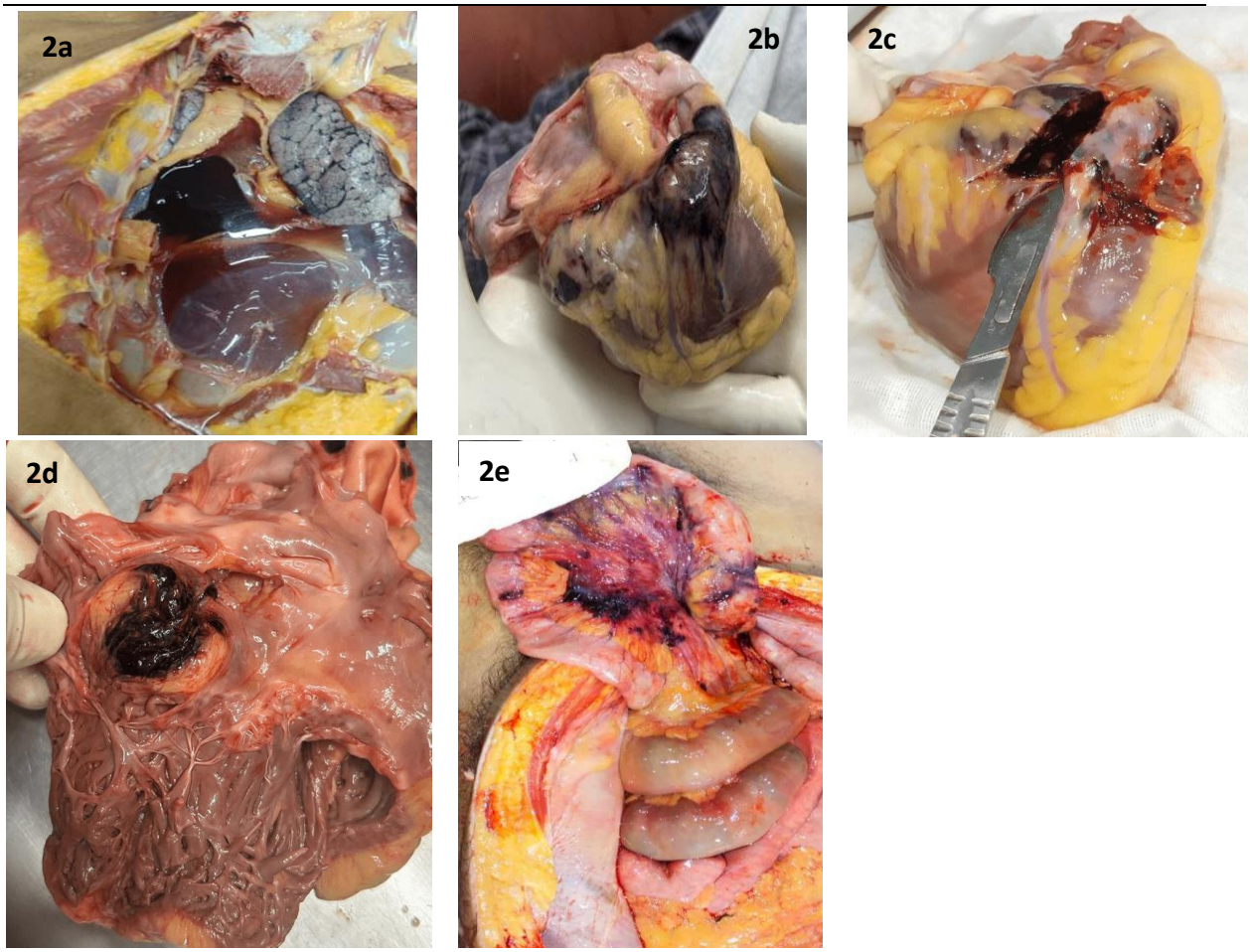

Figure 2:- Gross haemorrhage in pericardial cavity (2a); Bulging sac like pseudoaneurysm filled with blood clot and ruptured site on posterior wall of left ventricle (2b and 2c); infected slit-like opening with infected valve (2d); Haemorrhagic and infected mesentery (2e).

DISCUSSION

Corvisart reported the earliest case of ventricular pseudoaneurysm in 1797.[4] Left ventricular pseudoaneurysm (LVPA) is a rare but serious complication of infective endocarditis. Infective endocarditis poses a serious health risk with high morbidity and mortality rates in individuals of all age groups with congenital and acquired risk factors. Intravenous drug users, patients undergoing haemodialysis, and those with immunosuppression are also at increased risk.[1,2] In 16% of cases, intravenous drug use is a significant contributing factor in North America. Microorganisms can be injected directly into the bloodstream from the skin and soft tissue, which increases the risk of infective endocarditis in approximately 5% to 20% of cases.[19,20] Additional risk factors include prosthetic heart valves, dental infections, and certain medical procedures.[7] In our study, one case of LVPA was recognized in a young male.

Infective endocarditis can lead to complications, which include structural damage to the heart. In some instances, the infection spreads deep into the myocardium, which can result in localized necrosis and weakening of the ventricular wall, leading to rupture contained by the pericardium, forming a pseudoaneurysm.[2,4] Since it is consistently found to communicate with high-pressure chambers like the left ventricle or aorta, it is believed to originate from a preexisting weakened abscess that has undergone necrosis.[17] The elevated pressure inside the left ventricle is thought to force its way through the abscess, extending into the underlying structure.

Most commonly, LVPAs are complications of myocardial infarction or cardiac surgery; however, they may also result from chest trauma, infective endocarditis, or inflammation.[4,5]

The time frame between the onset of infective endocarditis (IE) and the development of a left ventricular pseudoaneurysm (LVPA) varies among patients. According to available literature, LVPA can be formed one month after surgery for active endocarditis,[15] two months later in mitral valve replacement for infective endocarditis[16] and twelve years after aortic valve replacement surgeries for infective endocarditis in adults.[21] In some patients, the diagnosis of pseudoaneurysm is made many years after myocardial infarction.[13] There are occasional reports of prolonged survival of a patient with an unruptured left ventricular pseudoaneurysm.[12]In 1963, Hurst et al. documented a case of a patient who lived for six years with a pseudoaneurysm following an acute posterolateral myocardial infarction.[22]

Available literature also suggests that members of the nutritionally variant streptococci (NVS) within the viridans group are one of the infective organisms. It is typically part of the normal human oral cavity and intestinal tract flora.[8,18,23] While infections caused by nutritionally variant streptococci are relatively rare, this microorganism has been associated with infections in the central nervous system, ocular and respiratory tracts.[18] Infective endocarditis caused by this microorganism typically presents in a subacute or chronic form. Although uncommon, it is usually linked to severe disease.[18,23]

Adam et al. documented a case series of four instances of infective endocarditis caused by this microorganism, with peri-valvular abscess complications occurring in 11% of the cases. All of these patients had identifiable risk factors, including poor oral hygiene, recent dental extraction, and the presence of a prosthetic heart valve.[23] In our study, the risk factors of IE leading to PSA remained unidentified, leaving an uncertain route through which the microorganism entered the bloodstream. However, it was suspected to have originated from the oral cavity or from skin infections as shown in figure 1e. It was also hypothesized that both deceased individuals likely experienced mild and nonspecific symptoms before the final episode, which may not have raised sufficient concern for their relatives to seek timely medical attention.

Left ventricular PSA is usually associated with clinical symptoms, though 10% of patients may not exhibit any.[11,14] When a pseudoaneurysm follows a myocardial infarction, patients can experience recurrent anginal chest pain and congestive heart failure, while arrhythmia, syncope, and systemic embolism are less commonly observed.[4,5,24] Its diagnosis is further complicated by the overlap of primary symptoms, including heart failure, dyspnoea, and chest pain, with those of coronary artery disease. In some cases, patients report vague symptoms like dizziness, cough, or altered mental status, which do not immediately suggest the presence of a pseudoaneurysm.[11] This can also associated with arrhythmias, thromboembolism and heart failure.[11]

Grossly, LVPA appears as a fibrous sac-like cavity under pericardial tissue without myocardial tissue,[11] small, narrow-necked, which communicates with the left ventricle,[26] and the cavity of PSA may contain mural thrombi, which can be partially organised and typically found in the posterior, lateral, or inferior wall.[11,15,24]

The left ventricle pseudoaneurysm of the upper anterior wall is a rare location for PSA but can occur in infective endocarditis where aortic valves are involved.[15,16,21] In one of our cases (case 1), the left ventricle PSA, formed in the anterior wall, often originates from abscesses in the mitral-aortic intervalvular fibrosa (MAIVF). The MAIVF is a relatively avascular structure prone to infection and injury, resulting in pseudoaneurysm formation.[9,10] The pseudoaneurysm relieves pressure by creating a fistulous connection with the left atrium or by rupturing through the epicardium, resulting in bleeding into the pericardial sac, which can lead to cardiac tamponade and immediate death, as seen in our case 2. Additionally, the size, location, and severity of the structural abnormality may exert pressure on the coronary artery, potentially triggering an acute myocardial infarction.[6,17] Similar case was also reported by Rahimi et al.[8]

These cases are incidentally detected on echocardiography or other imaging studies, although pseudoaneurysms are usually associated with a significant risk of rupture, necessitating surgical repair.[10] They can also contribute to heart failure due to their inability to contract appropriately or cause embolic events stemming from thrombus formation within the stagnant blood flow. Congestive heart failure is the most common presentation, followed by angina, ventricular arrhythmias, and embolization.[13] In our study, PSA was identified only through a medicolegal autopsy.

Over many years, despite advancing diagnostic methods and advanced treatment modalities, the mortality rate for IE remains relatively high.[19] A retrospective study by Fernandez Guerrero et al. reinforced the importance of autopsy in evaluating the quality of care and providing data for the management of IE.[6]

In this case series, both deceased succumbed to death after having sudden chest pain. Both were healthy with no known risk factors. Kashyap et al. reported a similar case of a young, previously healthy man with no known risk factors who was diagnosed with severe infective endocarditis. He presented with left-sided abdominal pain, fever, and vomiting. A prompt and accurate diagnosis enabled effective treatment with antibiotics and aortic valve replacement surgery, resulting in a complete recovery.[7,8]

A histopathologic examination was performed in our case, and the PSA diagnosis was confirmed.

Thrombus can also be found in the left ventricular PSA.[25] Embolization of thrombotic material, induced by stagnant blood flow patterns, has also been reported with large pseudoaneurysms (>3 cm in diameter).[11,25] We also found a thrombus in the cavity of left ventricular PSA in our case 2.

As observed in our case series of undiagnosed infective endocarditis, we propose that damage caused by the infectious process weakens the myocardium, facilitating dissection. The presence of fibrotic vegetations and chronic inflammatory changes suggested a subacute to chronic disease course. The slit-like opening of the pseudoaneurysm, resembling myocardial dissection, further supports the theory of extravalvular abscess remodelling. Consequently, the pseudoaneurysm primarily contains a thrombus with minimal infectious material. In the absence of medical intervention, the pseudoaneurysms likely ruptured or caused fatal haemodynamic compromise, as we have observed in case 2.

LIMITATIONS

This case series is limited by including only two confirmed cases of left ventricular pseudoaneurysm detected at autopsy, making it difficult to draw general conclusions. The study's retrospective design also means only limited clinical information was available, and no ante-mortem diagnosis had been made in either case. Left ventricular pseudoaneurysm can be difficult to detect early, as it often lacks specific clinical signs. These cases highlight the critical role of autopsy in identifying fatal complications that might be missed during standard clinical assessments. A larger case series and stronger integration of clinical and pathological findings are necessary to improve understanding. Greater clinical awareness and improved diagnostic tools are essential for the timely recognition and management of this potentially fatal condition.

CONCLUSION AND RECOMMENDATIONS

This case report highlights a rare occurrence of a left ventricular pseudoaneurysm. Although PSA is a complication that can cause sudden death due to rupture and heart failure, it has been thought that the life duration of a patient may be extended by preventing any sudden death resulting from myocardial rupture at an early stage with a kind of repair mechanism by PSA. If the diagnosis is made and the treatment is given after the development of PSA, the life duration of a patient will be longer. Nevertheless, as in our case, if the diagnosis is not done and if any treatment is not given, it may become a fatal complication.

We emphasise public awareness regarding subtle clinical symptoms of IE, especially in individuals with skin injuries or poor dental hygiene. Preventive measures, including routine dental care and timely treatment of skin infections, are essential to reduce the risk of bacteraemia and IE. Continue follow-up is also recommended in diagnosed cases of IE till their death. Suspected IE cases found at autopsy should incorporate microbiological cultures and histopathology to aid in identifying causative organisms and contributing to epidemiological insights.

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List Of Abbreviations

IE= Infective Endocarditis
LVPA=left ventricular pseudoaneurysm
PSA= pseudoaneurysm
NVS=Nutritionally variant streptococci
RT-PCR= Rapid test antigen and polymerase chain reaction
MAIVF= Mitral-aortic intervalvular fibrosa

Consent: Informed written consent was taken from the Next-of-kin.

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