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Original Article

Isolated Native Pulmonary Valve Infective Endocarditis (PVIE) : Endocarditis of Native Pulmonary Valve

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Summary

Objective: The purpose of this article is to review the scarce data regarding the diagnosis and management of native pulmonary valve (PV) infective endocarditis (IE).

Material and Methods: Published literature on the clinical presentation, diagnosis and management of PVIE was identified from on-line sources. All the articles which provided adequate data and scientific information were reviewed by the senior author. The studies which met the primary objective were included.

Results : There are certain case series reported decades ago and a few recent ones. A lot of available data is in form of single case reports. The studies were tabulated to find out the underlying cardiac conditions, predisposing factors and causative organisms. The assembled data was systematically analyzed to obtain relevant diagnostic and management information.

Conclusions : Isolated native PVIE is rare in clinical practice. The etiology has changed over the years from congenital cardiac malformation to a variety of health care related procedures and medical co-morbidities. Modified Duke's criterions are difficult to apply and there is lack of evidence based guidelines for timing and indications for surgery. High index of clinical suspicion, echocardiography, blood cultures and newer imaging techniques can facilitate a rapid diagnosis. The strategy of conservative treatment or early surgery should be individualized. (Indian J Cardiol 2022;25 (3-4):26-35)

Key Words: Pulmonary valve, infective endocarditis, native pulmonary valve endocarditis

Introduction

Infective endocarditis (IE) is associated with high mortality and morbidity even when treated by appropriate antibiotics and surgical intervention. Endocarditis involving native pulmonary valve (PV) is rare as compared to other valves and accounted for only 1.5-2 % of all cases[1]. The incidence of PVIE was 1% in the International Collaboration on Endocarditis-Prospective Cohort Study (ICE-PCS)[2] and 2.4% in the EURO-ENDO registry of IE[3]. Both structurally normal and diseased valve can be affected. The infective process may be isolated to PV or concomitantly involve other valves[4-7].

Material and Methods

The English language literature was searched using PubMed, and Google Scholar for the key words and search terms :"pulmonary valve endocarditis", "pulmonic valve endocarditis", "native pulmonary/ pulmonic valve endocarditis", "isolated pulmonary/ pulmonic valve endocarditis", and" right sided endocarditis" . The relevant data pertaining to the

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primary objective was identified from earlier[4-7] and recent series[8-10] patient reports[11-37], autopsy data[38], blood investigations[39,40], echocardiography[41-44], newer imaging modalities[45-52], complications[53,54] and surgery[55-57]. During the recent years, incidence of prosthetic PV or conduit IE is increasing as is the prevalence of repaired congenital cardiac malformation(CCM)[58,59]. This review focuses exclusively on the diagnostic and management challenges of native PVIE.

Clinical Features

The recognition of PVIE is a challenge since there are marked differences in the clinical presentation as compared to left sided endocarditis. The diagnosis can be missed or delayed when endocarditis occurs in healthy individuals , without known risk factors and in absence of typical features of right sided IE[17, 32, 37]. In a large series, the average time of symptoms to diagnosis was over 3 months[9]. The clinical picture is characterized by fever, septic pulmonic emboli (SPE), pulmonary regurgitation (PR) with or without right sided congestive cardiac failure (CCF)[7]. Infective emboli can be a presenting manifestation and should alert the clinician[8,15,26,29]. SPE is defined as the embolization of septic thrombi from a primary infection site into the pulmonary circulation which is confirmed by computed tomography. Life threatening septicemia requiring an emergency valve replacement can be a rare presentation in a drug abuser[16]. The presentation can mimic infective pneumonia[8,37], common respiratory illness[12] or a viral fever during the flu epidemic[24]. Paradoxical embolization is an extremely rare manifestation. Signs of right sided CCF depend on the degree of PR which occurs as a sequel to valve destruction. The murmur is generally short and low pitched and there is often discordance between auscultatory and echocardiographic findings.

Predisposing Factors

Table 1 and 2 show the underlying predisposing factors.

Table 1 : Underlying Congenital Cardiac Malformation

Type of CCM	Reference
• Tetralogy of Fallot	• (5-7, 22, 30)
• Ventricular septal defect	• (4-7, 29)
• Atrial septal defect	• (4, 6, 25, 36)
• Patent ductus arteriosus	• (4, 6, 24, 35)
• RVOT obstruction (valvar, infundibular, other)	• (4, 11, 25, 41)
• Transposition of great vessels	• (4)
• Unspecified CCM	• (8-10)

Table 2 : Health Care Related Infective Endocarditis

Predisposing Factor	Reference
• Intravenous drug abuse	• (8, 10, 16, 27)
• Insertion of PA diagnostic catheter	• (8)
• Cardiac implantable device therapy	• (9, 10)
• Immune-suppressive therapy	• (9, 10)
• Central venous catheter	• (8-10, 15, 18,21)
• Procedure related (Colonoscopy[12], Scrotal abscess[17], ERCP[20], Knee arthroplasty[22], Dental extraction[28], Sternal infection31	• (12,17,20,22,28,31)
• Others: Alcoholism, Bowel surgery, Liver or renal transplantation	• Quoted by (12)

Abbreviations: As in text

Congenital Cardiac Malformation (CCM)

Underlying CCM was the predominant predisposing factor in earlier series[4-7] and case reports in children and adults[24,25,29,30,35,36]. Almost all common variety of cardiac malformation have been associated with PV endocarditis (Table1) .Vegetations usually develop on the low pressure side of the defect with endocardial trauma, and downstream from the site of the lesion. In patients with patent ductus arteriosus (PDA),the vegetations first develop at the pulmonary end whereas in the ventricular septal defect (VSD) at the right ventricular margin of the defect. In sub-pulmonic VSD, the right coronary sinus bulges in to right ventricular outflow tract (RVOT) causing a pressure gradient .The infective process begins in the "jet lesion" and may extend from the deformed right coronary cusp to essentially normal PV[6].

The care of patients with CCM has improved with early surgical correction and in the recent series there are fewer patients with underlying congenital lesions who develop native PV endocarditis[9,10].

Drug Abuse and Health Care Associated Pvie (Table 2)

The etiology has shifted from CCM to drug addiction, devices, central lines to health care procedures. Pulmonary artery (PA)flotation catheter (22%) and intravenous (IV) drug abuse (41%)were major causative factors in a large series published in year 2001[8]. In narcotic addicts ,the PV involvement may be isolated or in association with tricuspid or other valves[6]. Life threatening septicemia and fungal endocarditis have been reported with drug abuse[16,27]. The crucial role of extra-cardiac co-morbidities have been highlighted in recent publications[9,10]. High prevalence of chronic immunosuppressive therapy, cardiac implantable electronic device (CIED) and central venous catheter (CVC)reflect the changing profile. Patients receiving immunosuppressive agents for rheumatoid arthritis ,myasthenia or post renal transplant care are susceptible[9]. Catheters used for dialysis, pharmacotherapy ,chemotherapy , nutritional supplementation and pacing have led to PVIE[15,18,21].

Health care related procedures like colonoscopy and polypectomy[12], scrotal infection[17], endoscopic retrograde cholangiopancreatography (ERCP)[20], knee arthroplasty[22], dental extraction[28] and sternal wire infection post coronary artery bypass graft surgery[31] have been complicated by PV endocarditis. Even, apparently healthy individuals with no identifiable risk factor can be affected[14, 19, 23, 26, 32, 37].

Pathological (Morphologic) Findings

Pathological findings obtained at surgery or autopsy data provide invaluable insights into the infective process and its sequel[4-7]. PV involvement was observed in 5.8% cases in an autopsy series[38]. The affected valve can be tricuspid, bicuspid or have

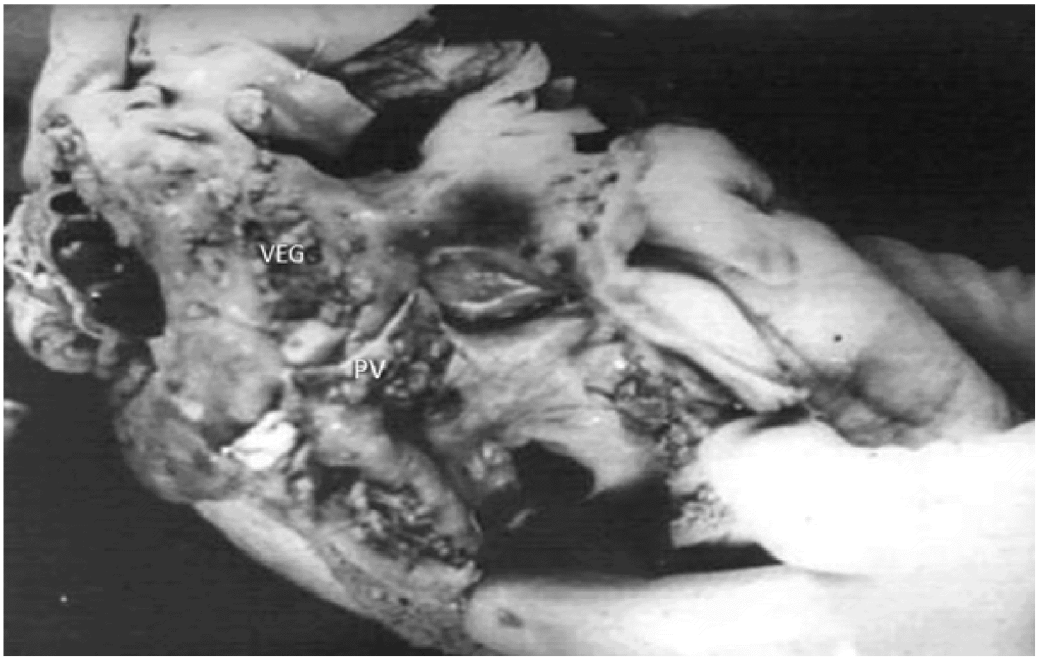


Fig. 1 : (a) Heart at autopsy reveals extensive vegetations (Veg) of the PV extending into PA.

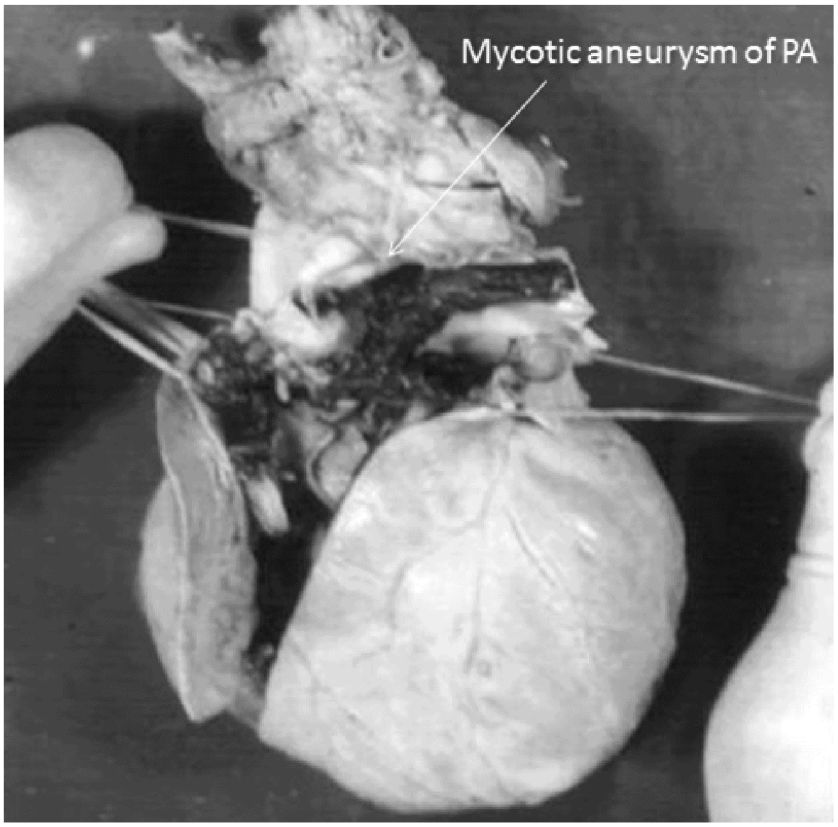


Fig. 1 : (b) Mycotic aneurysm of PA and vegetations in a different patient at post-mortem.

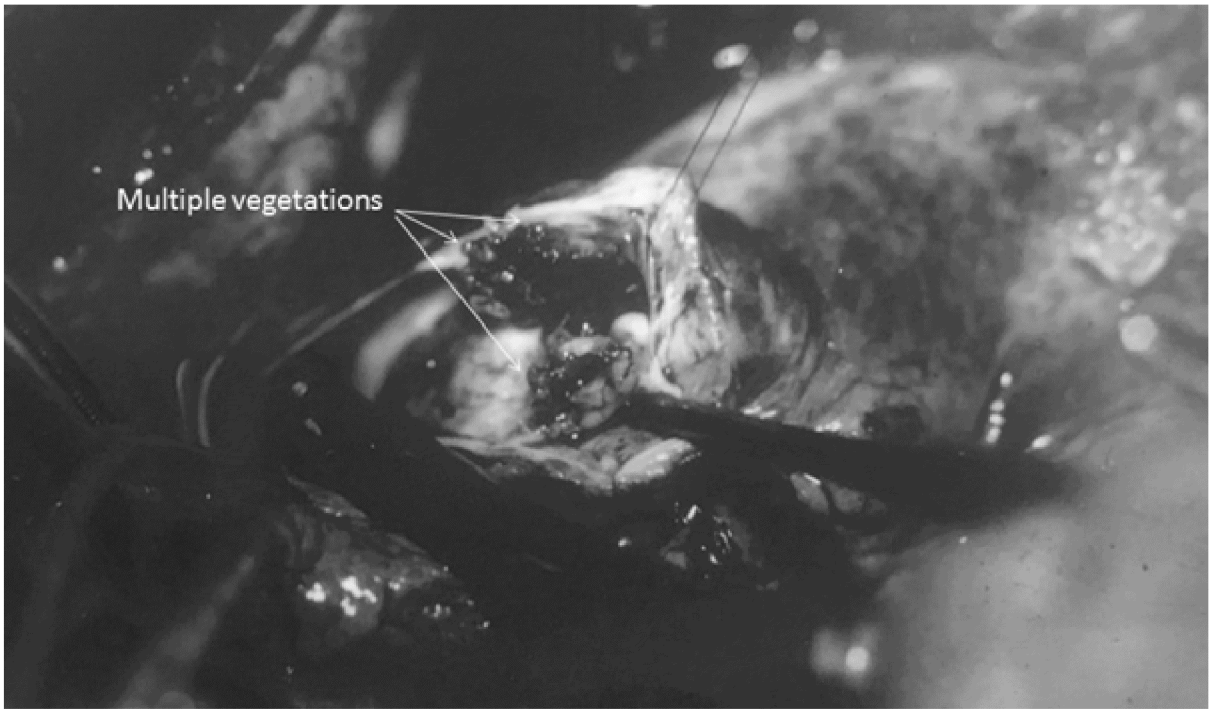


Fig. 1 : (c) Multiple small vegetations (arrow) are visualized at surgery in yet another patient

one cusp. Vegetations can be sessile, polypoid, vary from 2-3 mm to few centi-meters and be active, healing or healed (Figure 1 A and B). They can be limited to PV or extend to RV, pulmonary arteries and tricuspid valve. Perforation of valve, prolapse or complete destruction of cusps lead to PR. Mycotic aneurysm of PA is an extremely rare complication of PVIE (Figure 1C). Lungs show changes secondary to infective emboli.

Diagnosis and Investigations

Modified Duke's criteria based on clinical, echocardiographic and blood culture findings are utilized for the diagnosis of IE. Accordingly, the diagnosis can be definite, possible or rejected[39]. There is no large study validating the utility of Duke's criteria in PVIE. In a febrile patient, diagnostic suspicion may be strengthened by non-specific laboratory signs such as elevated erythrocyte sedimentation rate (ESR), leukocytosis and anemia .C-reactive protein(CRP) and pro-calcitonin have not been particularly useful[40]. The chest roentgenogram can show infiltrates, consolidation, pleural effusion, nodular densities or infarction. It also provides useful information about underlying CCM, device or central lines. Right atrial (RA), RV enlargement or RBBB (right bundle branch block) on electrocardiogram can be useful clues to SPE or PR.

Blood Culture

Blood culture is a key investigation and a positive result provides the diagnosis, identifies bacteria and allows susceptibility testing.The microbiological spectrum of the disease keeps changing. A number of gram positive and gram negative organisms have been identified according to the underlying risk factor (Table 3). Isolated fungal endocarditis of PV is exceptional with scattered reports in those on chronic dialysis[21] or drug abuse[27]. Candida Albicans and Glabrata carry high recurrence and poor prognosis[21,27]. Blood culture negative infective endocarditis (BCNIE) poses considerable diagnostic and therapeutic dilemma[22, 26, 29, 30, 36]. Prior antibiotic therapy or lack of refined culture techniques is usually responsible.

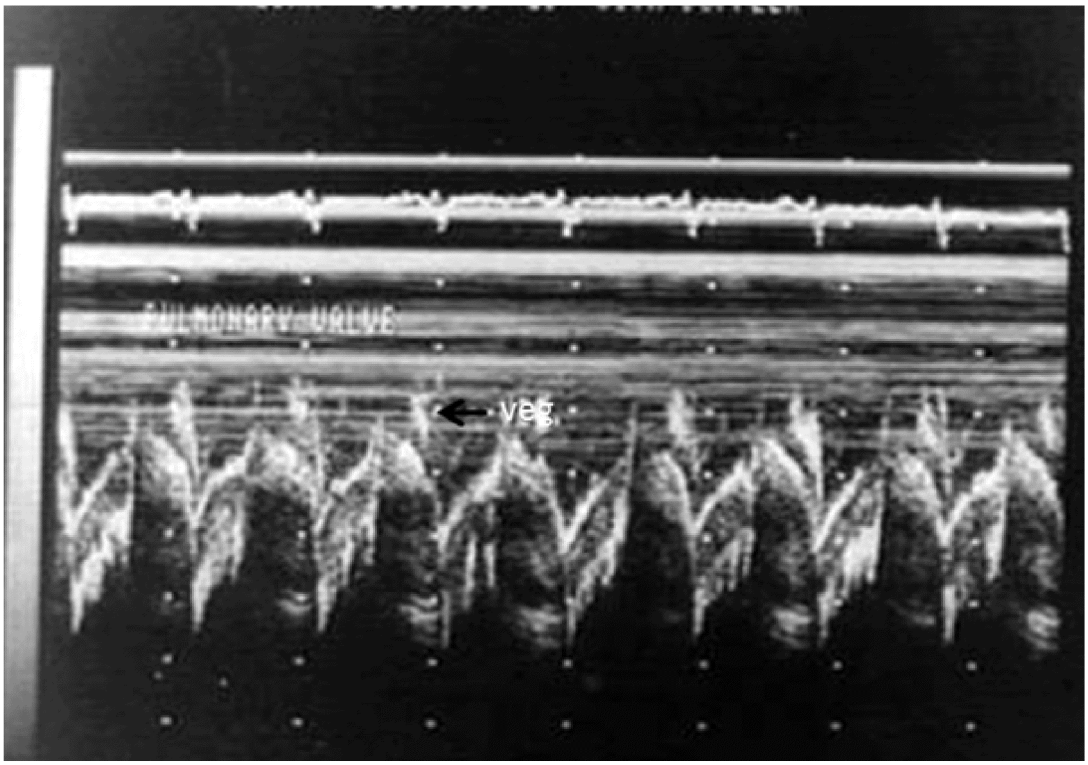
Table 3 : Microorganisms in PVIE

Organism	Risk Factor	Reference
• Viridans Streptococci (Mitis, Sanguius, Pneumoniae, Viridans)	• CCM, Dental extraction, normal individual	• (14,17,24,28,35)
• Staphylococci (Aureus, Hominis, Coagulase negative, Hemolyticus, Epidermidis, MRSA)	• CVC, IV drug use, Dialysis, CCM, Sternal infection, no risk factor	• (7,13,16,18,19,25,31)
• Enterococcal Fecalis	• Colonoscopy and polypectomy, No obvious cause	• (12,23)
• Gram Negative (Klebsiella pneumoniae, Citrobacter koser)	• ERCP	• (20)
• Fungus (Candida Albicans, Glabrata)	• IV Drug abuse, CVC	• (21,27)

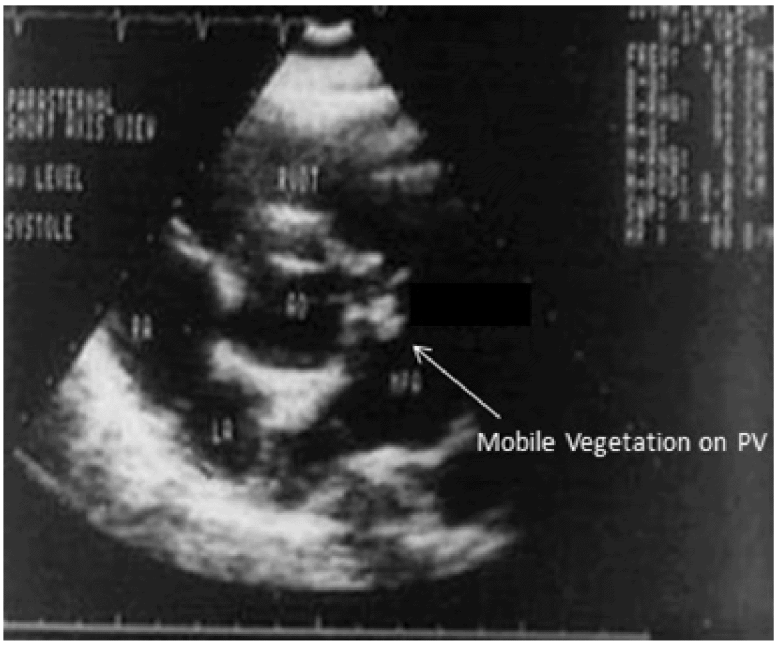
Abbreviations: As in text

Echocardiographic Diagnosis

Echocardiography remains the principal diagnostic imaging technique. Trans-thoracic echocardiography (TTE) is usually the initial test, but a trans-esophageal imaging is recommended when the initial TTE is negative in the setting of high clinical suspicion. PV is the most difficult valve to be visualized and has three cusps: the anterior, left posterior and right posterior. It is extremely difficult to differentiate between the two posterior cusps due to lack of any anatomical landmark. M-mode echocardiographic (MME) detection of PV vegetations was described by Kramer[39] to be followed by increasing recognition[41-44]. MME is able to visualize only the posterior PV cusp due to limited access and detect vegetations only in 29% of cases[18]. The echocardiographic description of vegetations on PV is similar to other valves. Vegetations produce thickening of valve along with echo dense mass or globular densities described as "soft", "shaggy," or "fluffy" shadows .Two- dimensional TTE provides better morphological information and diagnosis in



(A)



(B)

Fig. 2 : (A) M- mode echocardiography shows thickened , shaggy echoes and 2DE in parasternal short axis view reveals a mobile mass (B) suggestive of PV vegetation. Abbreviations : RVO = right ventricular outflow, AO = aorta , LA = left atrium, RA = Right atrium

91% cases[9]. Parasternal short and long axis, RV inflow short axis and subcostal short axis views are recommended to assess the PV. Parasternal short axis view is most useful and provided diagnosis in 87%[18]. Figure 2A and 2 B show utility of MME and two dimensional TTE in detection of pulmonary valve vegetations.

Trans-esophageal echocardiography (TEE) of the anteriorly located PV can be challenging for most echo-cardiographers and has been used as an initial modality for diagnosis in 22% .The mid esophageal RVOT, upper esophageal aortic and trans -gastric short axis (30-60 degree) views provide excellent assessment of PV. An experienced operator can provide superior images and better Doppler assessment for PR on TTE than TEE. TEE provides better assessment of other valves and was superior in evaluating CIED lead involvement[9].

Adjunctive Imaging Modalities

It is desirable to have an adjunct imaging modality that assesses infection in extra cardiac regions (primary culprit/and or embolic source of infection) and diagnoses complications. Multi-slice computed tomography (MSCT), ECG gated cardiac CT angiography (CTA), nuclear imaging and magnetic resonance imaging (MRI) play an important role.

MSCT and CTA:

MSCT can demonstrate cardiac and non-cardiac manifestations and complement TTE and TEE[45, 46]. Pulmonary complications (SPE, abscess, infarction) seen in 50% of cases are best detected by this modality. The technique can diagnose large (>10mm, mobile) vegetations which usually cause embolization. The high resolution images provided by CTA are valuable in diagnosis of pseudo-aneurysm, fistulas, valve perforation, abscess and valvular dehiscence. Its best utility is in evaluation of RVOT or pulmonary arteries which are difficult to be assessed by echocardiography[46].

Nuclear imaging

18F- flurodeoxyglucose (FDG) positron emission tomography/computed tomography (PET/CT) provides functional molecular imaging of the whole body[47-49]. FDG targets the inflammatory cells and accumulates with high concentration at the site of infection and has a potential role in the diagnosis of endocarditis affecting CIED.

Ventilation /Perfusion (V/Q) lung scan is useful for diagnosis of PE. A normal ventilation scan in conjunction with impaired perfusion signifies a high specificity for SPE in setting of IE[50]. Figure 3 shows segmental perfusion defects in right lung secondary to pulmonary infarction due to SPE.

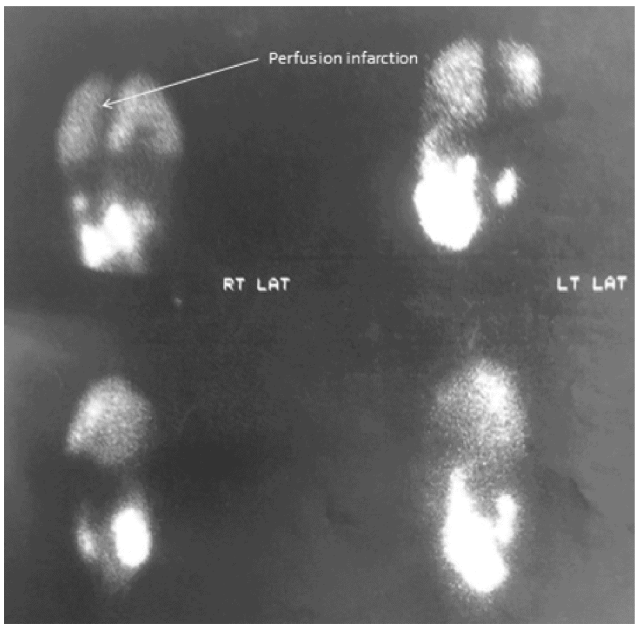


Fig. 3 : Pulmonary ventilation - perfusion scintigraphy reveals perfusion defects in right upper lobe apical segment and posterolateral aspect of lower lobe due to pulmonary infarction resulting from dislodged vegetations .

Cardiac Magnetic Resonance Imaging (CMRI)

CMRI provides both anatomical and hemodynamic information .It can quantify PR, define RV volumes and function and visualize para-valvular area, PA wall, lateral wall of RV and diagnose abscess, fistula or mycotic aneurysm in these locations[19, 22, 23, 51, 52]. Complications: The complications are related to septicemia, SPE and structural deterioration of PV. Septicemia can result in multi-organ dysfunction, acute kidney injury, shock and death. Pulmonary complications include abscess, infarction and pleural effusion. The valve damage leads to varying degree of PR and hemodynamic instability. Para-valvular abscess, mycotic aneurysm and intra-cardiac fistula result from para-valvular extension and endothelial erosion. Mycotic aneurysm of PA is an extremely rare complication and has been reported in association with right heart endocarditis and PDA.[53,54]. The aneurysm is caused either by contagious spread from an adjacent area with infection or by SPE. These aneurysms are inherently unstable, can rupture with catastrophic consequences.

Management

It is sometimes argued that right sided IE is better tolerated and more likely to respond to medical treatment than infection of the left sided valves. Conservative management is the initial strategy and surgery is considered in specific situations. The main objective is to eradicate infection by intensive antibiotics/ antifungal treatment guided by culture sensitivity. Sick patients require diuretics, oxygen, noninvasive or invasive ventilation, inotropic agents, dialysis and supportive care. The data from case reports suggest that approximately 50% had conservative treatment, 10% were too sick or refused operation and the remaining underwent surgery[11-36]. The surgery involves correction of underlying congenital lesion and infected valve. High mortality ranging from 20 to 66% has been observed[4-5,7,10]. Whereas for left sided endocarditis there are evidence based guidelines ,the indications for surgery and its timing in PVIE are not clear[60]. Highly mobile vegetations, repetitive PE, and failure of intensive medical treatment are well recognized indications for early surgery. In a large series, 56% of patients needed surgical intervention[9]. There is little evidence regarding the optimal surgical strategy and the options include debridement of the infected area, vegetation excision with valve preservation, repair or replacement. Removal of vegetations by preserving the valve is an option in the early stage of the disease. Valvectomy (resection of valve) achieved eradication of infection with no recurrences and well tolerated PR at 5-10 years follow up[56]. Den and colleagues reported rapid post operative recovery and sustained short term benefits following pulmonary valve repair using autologous pericardial patch[57]. Preservation of the native valve is recommended whenever feasible, a homograft [16,18,30,31,34]. or stent-less bio-prosthesis[17,21] is preferred when replacement is required.

The recognition of native PVIE remains challenging due to it being very rare and change in the underlying etiology. A high clinical suspicion in patients with CCM, bacteremia, central venous lines, devices or extra-cardiac co-morbidities may enable rapid diagnosis of this entity.

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