

Case report: isolated pulmonary valve endocarditis in a 39-year-old patient with intravenous drug abuse

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Background

Isolated pulmonary valve endocarditis is a very rare form of right-sided infective endocarditis. Due to the anatomy, in most cases, just the tricuspid valve is involved. Diagnosis can be challenging because of non-specific symptoms (fever, dyspnoea, haemoptysis, and pleuritic chest pain) and difficulty of detection by echocardiography. Risk factors include intravenous drug abuse, congenital heart disorders, alcohol abuse, male sex and central venous catheters, or pacemaker leads.

Case summary

A 39-year-old homeless male patient, who was a current intravenous drug user, presented with fever, dyspnoea, and haemoptysis. The chest X-ray showed bilateral infiltrates. Empiric antibiotic treatment was initiated. Blood cultures showed the presence of *Streptococcus dysgalactiae*. Atypical causes of pneumonia were excluded. Systemic embolism was suspected, and a computed tomography scan of brain, thorax, and abdomen was performed. Multiple septic embolic lesions were detected in both lungs. Echocardiography revealed an isolated pulmonary valve endocarditis. Penicillin G and gentamycin were administered intravenously for a duration of 6 and 2 weeks, respectively. The patient was discharged in stable condition but did not return for outpatient clinical appointments.

Discussion

To detect rare causes of right-sided infective endocarditis, repeated echocardiograms with special focus on the pulmonary valve may be required. Usually, antibiotic treatment alone leads to recovery. In special situations (heart failure, septic shock, or large vegetation size) surgery is required. Due to the high risk of postoperative complications, surgery in intravenous drug users should be avoided if possible.

Keywords

Right-sided infective endocarditis • Pulmonary valve endocarditis • Drug-abuse • *Streptococcus dysgalactiae* • Case report

Learning points

- An isolated pulmonary valve infective endocarditis is rare, but if clinical suspicion is high, transthoracic echocardiography/transoesophageal echocardiography should be repeated to rule out same.
- Antibiotic therapy is successful in controlling infection, though there are different approaches, general consensus is a 6-week course.
- Decision for surgery, especially in i.v. drug users, should be discussed in a heart team to avoid adverse outcomes.

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Introduction

Right-sided infective endocarditis occurs in 10% of all patients with endocarditis. In nearly all cases the tricuspid valve is involved. Isolated pulmonary valve endocarditis is a rare variant of the condition. One main risk factor is intravenous drug abuse.¹ We want to report a 39-year-old man with long history of intravenous drug abuse presenting with recurrent episodes of fever and respiratory symptoms, who was diagnosed with isolated infective endocarditis of the pulmonary valve.

Timeline

Date	Event
Day 0	Admission, patient presenting with general weakness, dyspnoea, intermittent episodes of fever, haemoptysis Chest X-Ray showing bilateral infiltrates, blood cultures were obtained, serum test showed elevated markers inflammation Diagnosis of community-acquired pneumonia i.v. antibiotics (Ampicilline und Clarithromycin) were started
Day 1	Blood cultures revealing <i>Streptococcus dysgalactiae</i>
Day 2	Search for alternative focus revealed spleen lesion, septic embolic process was considered, and normal transthoracic echocardiogram obtained
Day 4	Computed tomography showed septic embolism of the lungs as well as spleen infarction
Day 5	Final diagnosis isolated pulmonary valve endocarditis was made due to a second look transthoracic and transoesophageal echocardiogram
5 to 6 weeks after admission	Antibiotics were switched to penicillin G and gentamycin, fever disappeared, markers of inflammation decreased within 12 days, repetitive blood cultures, and echocardiograms, consolidation of the lesion
Day of discharge (after ca. 6 weeks)	Asymptomatic patient, multiple outpatient follow-up appointments were made
Follow-up	No follow-up available

Case presentation

A 39-year-old homeless male patient was admitted to our emergency department presenting with general weakness, dyspnoea, intermittent episodes of fever, episodes of coughing blood, and an unspecific feeling of pain. Clinical examination showed blood pressure of 105/55 mmHg, heart rate of 94 b.p.m., and a respiratory rate of 28 per

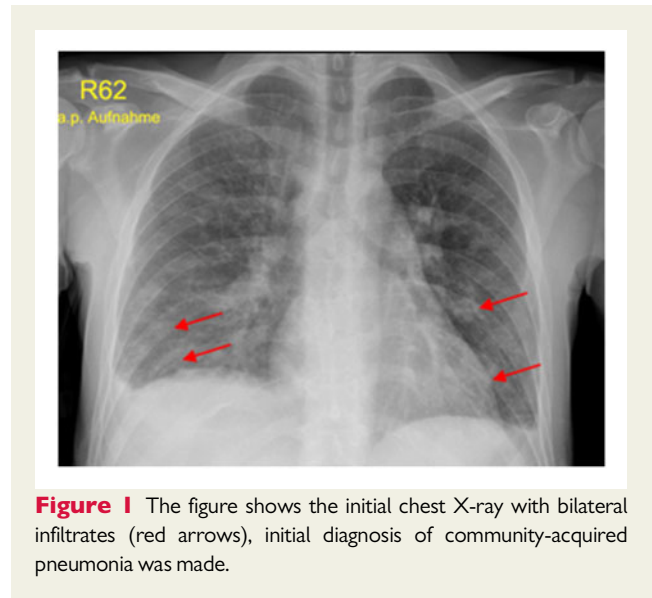


Figure 1 The figure shows the initial chest X-ray with bilateral infiltrates (red arrows), initial diagnosis of community-acquired pneumonia was made.

minute. Arterial oxygen saturation was 90%. Body temperature was 37.7°C. The dentition status was poor. However, there were no signs of an acute infection in that area. There were no peripheral stigmata of endocarditis present such as Janeway lesions or Osler's nodes. Further clinical examination was unremarkable.

The patient was a current user of intravenous heroin, amphetamine, and alcohol. He was not on any regular medication. There were no known allergies or other conditions present. There was no record of a prior episode of infective endocarditis.

Serum levels of C-reactive protein (286 mg/L; upper limit of normal <5 mg/L) and white blood cell count (16.5 cells/mm³; upper limit of normal <9.8 cells mm³) were elevated. Due to clinical presentation and elevated markers of inflammation, we performed a chest X-ray (Figure 1), which revealed bilateral diffuse pulmonary infiltrates. Community-acquired pneumonia was diagnosed, blood cultures were obtained and intravenous antibiotics (Ampicilline und Clarithromycin) were started. There was no evidence of tuberculosis in direct microscopy, culture or polymerase chain reaction (PCR). Other causes of atypical pneumonia were ruled out: *Aspergillus fumigatus*, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Pneumocystis jirovecii*. HIV was excluded based on PCR tests. Blood cultures showed the presence of *Streptococcus dysgalactiae*.

We performed abdominal ultrasound and transthoracic echocardiography (TTE) to evaluate alternative infective focus locations. The initial TTE showed no signs of endocarditis. Abdominal ultrasound showed two spleen lesions indicating spleen infarction. We assumed a septic embolic process and performed a computed tomography scan of the brain, thorax, and abdomen showing multiple lesions in both lungs classified as septic embolic lesions (Figure 2). Follow-up TTE as well as transoesophageal echocardiography (TOE) showed a round and mobile vegetation of 14 × 14mm in size, with the main part attached to the right leaflet of the pulmonary valve, as well as mid- to high-grade pulmonary valve regurgitation (PR) (Figure 3) without evidence of infection in the other valves (see [Supplementary material online](#)). There was no evidence of significant dysfunction of the right ventricle (RV) or pulmonary hypertension. Systolic pulmonary

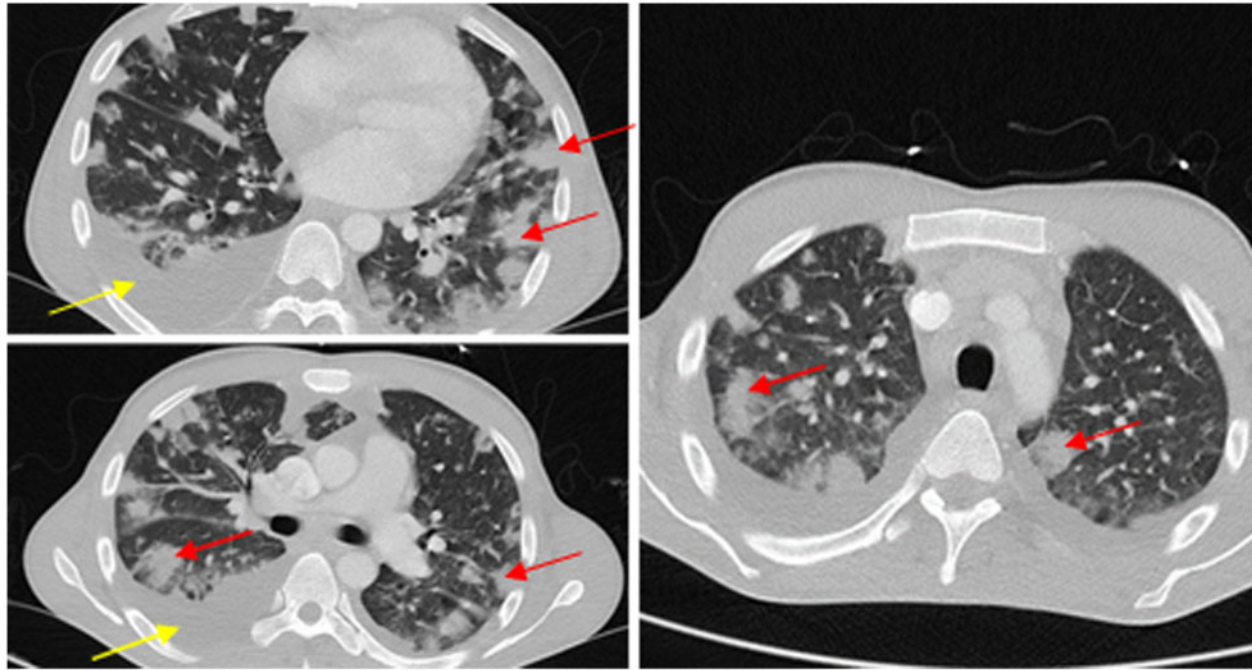


Figure 2 The figure shows a computed tomography of the thorax with different layers: the red arrows mark septic embolic infiltrates in both lungs and the yellow arrows mark coexistent pleura effusion dominant on the right side.

artery pressure was 32 mmHg (upper limit of normal <36 mmHg). The size of the right ventricle was almost within normal ranges: basal RV diameter (RVD1) 30 mm (normal range 25–41 mm), mid-cavitary RV diameter (RVD2) 37 mm (normal range 19–35 mm), and longitudinal RV diameter (RVD3) 82 mm (normal range 59–83 mm) (see [Supplementary material online](#)). Tricuspid annular plane systolic excursion indicated normal RV function as well (28 mm; lower limit of normal >17mm). The right ventricular outflow tract (RVOT) was slightly enlarged ([Figure 4](#)). We found no evidence for the presence of a patent foramen ovale (PFO) or other shunt.

We diagnosed isolated pulmonary valve endocarditis. Based on recent guidelines, we changed the antibiotic treatment to penicillin G and gentamycin according to available antibiogram². These were administered intravenously over a duration of 6 and 2 weeks, respectively. During that time, the initial symptoms of the patient disappeared. There was prolonged fever until 12 days of antibiotic treatment. Subsequently, serum markers of inflammation returned to normal (C-reactive protein 4.26 mg/L; white blood cell count 7.6 cells mm³) and surveillance blood cultures remained sterile. Follow-up TTE revealed consolidation of the vegetation as well as a reduction in size and remaining mid- to high-grade PR without clinical or echocardiographic signs of right ventricular heart failure ([Figure 4](#)).

An interdisciplinary heart team discussed the case and (with consensus of the patient) decided to continue medical treatment and against surgery. The patient's treatment was complicated by non-compliance. Although a multidisciplinary approach including social workers and psychiatrist was implemented, the patient was lost to follow-up.

Discussion

In right-sided infective endocarditis, the tricuspid valve is primarily affected,¹ involvement of the pulmonary valve is only observed in 1.0–1.5% of cases.³ Isolated infection of the pulmonary valve without infection of the other valves is even less common.⁴ Important risk factors for developing pulmonary valve endocarditis are intravenous drug abuse, congenital heart diseases, alcohol abuse, and male sex, as well as medical devices such as central venous catheters or pacemaker leads.⁵ The most common bacterial species found in isolated pulmonary valve endocarditis is *Staphylococcus aureus*.^{4,6} The spectrum also contains coagulase-negative staphylococci and Group B streptococci.^{4,7}

The diagnosis can be challenging due to relatively unspecific symptoms including fever and respiratory symptoms such as dyspnoea, haemoptysis, and pleuritic chest pain. Symptoms are often caused by septic pulmonary embolism. In our case, positive blood cultures and spleen lesions possibly due to septic embolism were major findings that led to diagnosis. The lesions were without contrast enhancement ruling out significant splenic abscess. Most common causes for a systemic embolism in right-side endocarditis are a PFO and physiologically pulmonary shunts.¹

However, even if right-sided infective endocarditis is in the main focus of diagnostic efforts, it can still be difficult to detect: a prior case series evaluating the diagnostic value of TTE and TOE found that in patients with prosthetic pulmonary valve and pulmonary conduit endocarditis, TTE had a sensitivity of 62% and TOE of 53%.⁸ The combination of the two methods led to a sensitivity of 88%.⁸ In our

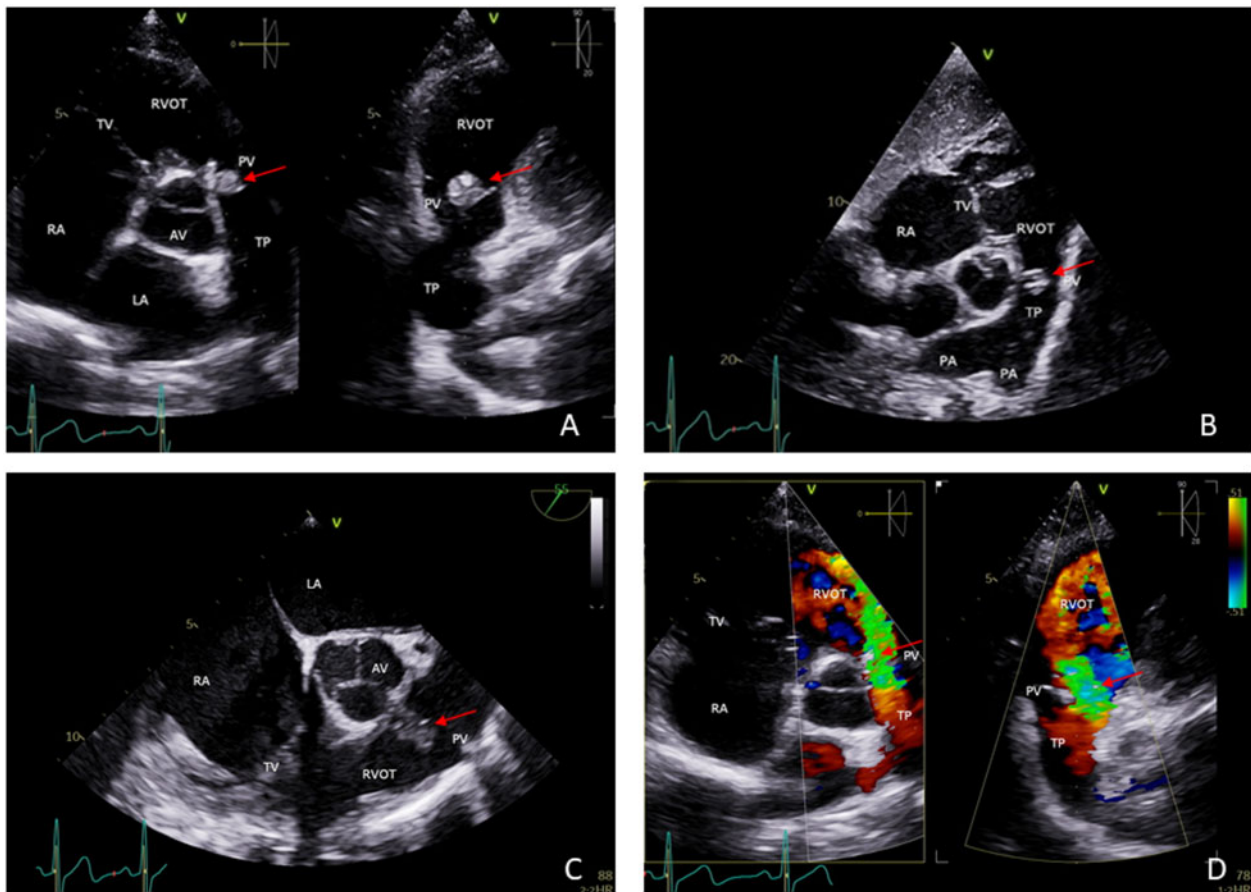


Figure 3 (A) Transthoracic echocardiography parasternal short-axis view (biplane): AV, aortic valve; LA, left atrium; PV, pulmonary valve; RA, right atrium; RVOT, right ventricular outflow tract; TP, truncus pulmonalis; TV, tricuspid valve. The red arrow marks the endocarditis lesion attached to the pulmonary valve. (B) Transthoracic echocardiography subcostal short-axis view: PA, pulmonary artery. The red arrow points at the endocarditis lesion. (C) Transoesophageal echocardiography short-axis view: the red arrow marks the lesion attached to pulmonary valve. (D) Transthoracic echocardiography biplanar parasternal short-axis view: the arrow marks the remaining mid- to high-grade regurgitation jet after therapy.

case, we failed to detect the lesion in the first TTE. The combination of TTE and TOE, as well as repetition of the procedure was required to find the correct diagnosis. Common views to evaluate the pulmonary valve in TTE are parasternal long-axis view (angulate the transducer in dorsal direction) and short-axis view (at the level of the aortic valve), subcostal short-axis view (at the level of the aortic valve) as well as a 'dextrocardiographic' view accessible via apical two-chamber view and in TOE short-axis view at 40–50 degree (at the level of the aortic valve).

Most patients with infective pulmonary valve endocarditis can be treated using medical therapy only.² The prognosis is much more benign compared to left-sided infective endocarditis, even in higher grade valve regurgitation.⁹ The most widely used empiric therapy is anti-staphylococcal β -lactam antibiotics combined with aminoglycosides.¹⁰ When the pathogen is unknown, therapy strategy should consider *S. aureus* being the most common cause. However, in our case the organism was known at the time of diagnosis and an antibiogram was also available. According to present ESC guidelines, we administered Penicillin G and Gentamycin over period of 6 weeks.² In

uncomplicated cases, a short duration of intravenous treatment over 2 weeks is possible.¹¹ Alternatively, in intravenous drug users, oral antibiotic regimes have also been successfully trialed.¹²

Surgical treatment is not often required in isolated pulmonary valve or right-sided endocarditis, because the conditions respond well to medical treatment.⁹ Indication for surgical treatment is persistent bacteraemia especially when the infection is caused by a fungi, *S. aureus* or *Pseudomonas aeruginosa*.² In case of persistent valve vegetations >20 mm and signs of right ventricular heart failure due to repetitive pulmonary embolism or secondary due to severe valvular regurgitation surgical treatment should also be considered.²

The main principles of surgery are radical debridement of vegetations and infected tissue as well reduction of regurgitation size. There are 'non-prosthetic' and prosthetic surgical techniques. Common 'non-prosthetic' procedures are vegetectomy and valvectomy. The most common prosthetic procedure is Carpentier's remodelling annuloplasty.¹³ However, in patients with intravenous drug abuse, prosthetic techniques should be avoided because there is a high risk of prosthetic-associated complications such as prosthetic

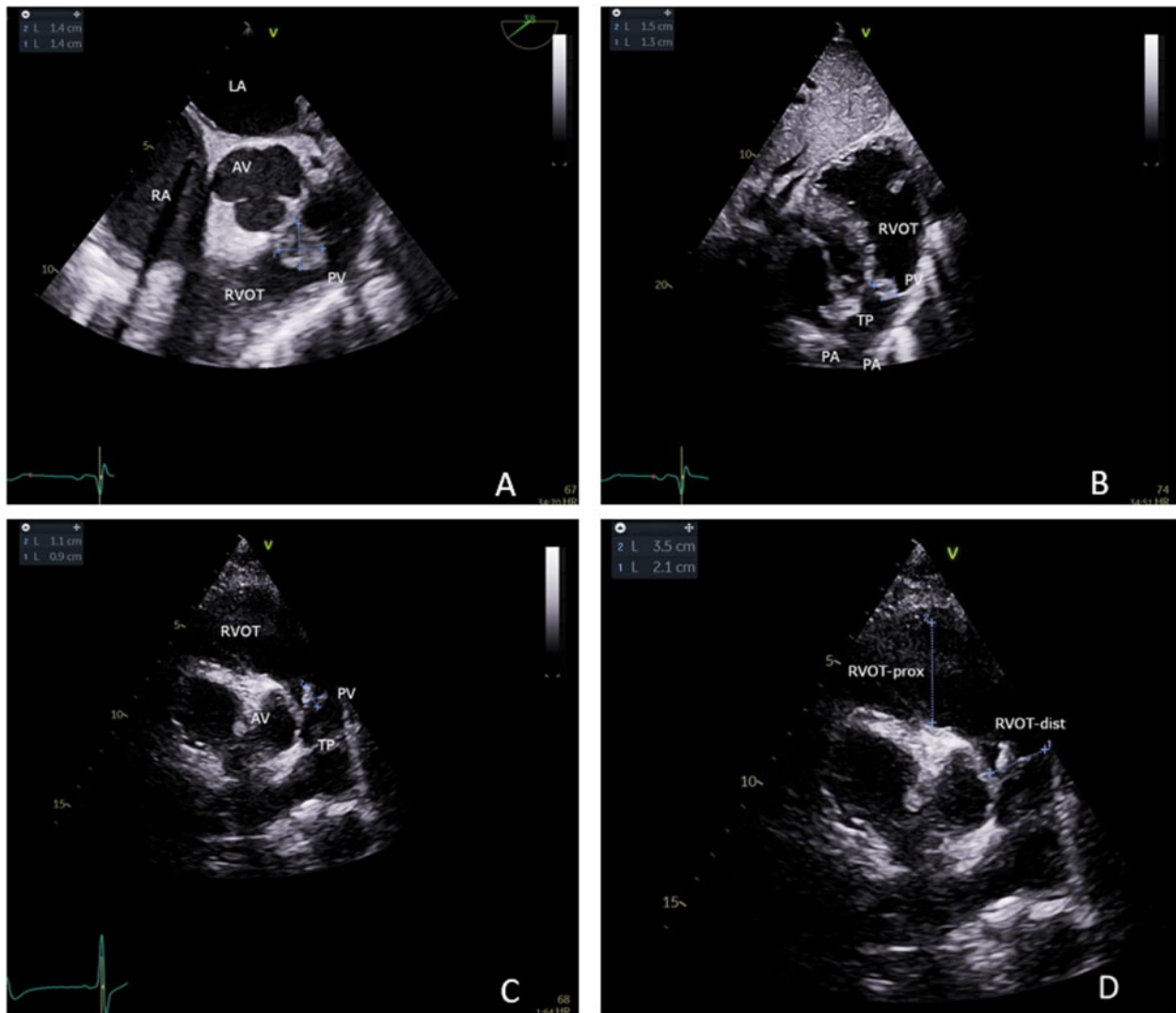


Figure 4 (A) Transoesophageal echocardiography short-axis view: endocarditis lesions at the time of diagnosis. The lesion was 14 mm × 14 mm in size. The blue lines show the diameters which were measured. The vegetation was located at the aortic near commissure between left and right leaflet. (B) Transthoracic echocardiography subcostal short-axis view shows the endocarditis lesion before treatment. In this view, we measured the lesion 15 mm × 13 mm. (C) Transthoracic echocardiography parasternal short-axis view: the consolidated lesion after treatment which was reduced in size and measured 9 mm × 10 mm. (D) Transthoracic echocardiography parasternal short-axis view shows diameter of right ventricular outflow tract (distal and proximal) according to current guidelines, right ventricular outflow tract distal 21 mm (normal within <27mm), right ventricular outflow tract proximal 35–37 mm (normal within <35 mm), right ventricular outflow tract proximal long axis was 35 mm (not shown, normal within <30 mm), right ventricular outflow tract was enlarged due to volume overload because of remaining pulmonary valve regurgitation, tricuspid annular plane systolic excursion 28mm, no significant tricuspid valve regurgitation.

infection.^{13,14} Witten *et al.*¹⁵ showed that reoperations were significantly more often observed in injection drug users.

In our case, the patient responded well to antibiotic treatment. After therapy, there was a consolidated lesion on the pulmonary valve with moderate-to-severe PR. However, right ventricular function and systolic pulmonary artery pressure were in normal ranges. Taking the intravenous drug abuse and significant social and personal problems into consideration, we decided against surgical treatment which corresponded with the wish of the patient.

Conclusions

In summary, the diagnosis of isolated pulmonary valve endocarditis might be challenging because symptoms are often not specific. When only the pulmonary valve is affected, repeated TTE or/and TOE might be necessary since echocardiographic imaging of the pulmonary valve remains challenging. In most cases, medical therapy led to successful treatment. However, social problems and low therapy adherence among patients with intravenous drug abuse can lead to decreased

mid- and long-term results. Therefore, we recommend a multidisciplinary approach including social workers to secure the initial achievements of the main therapy.

Lead author biography



Martin Richard Platz graduated from medical school of Leipzig University in 2016. He works as a resident at the Department of Cardiology at the University Hospital of Leipzig, Germany. His field of interest includes general cardiology and internal medicine as well as echocardiography, anticoagulation therapy, and emergency medicine. In 2021, he will finish his special training in internal medicine.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance. The consent has been documented in the medical record. A digital copy is available.

Conflict of interest: none declared.

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