Complementary role of cardiac computed tomography angiography in the diagnosis of prosthetic aortic valve endocarditis and septic coronary embolism - a case report

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ABSTRACT

A 73-year old man presented with a posterolateral ST-elevated myocardial infarction 9 months after biological aortic valve replacement for aortic valve stenosis. Invasive coronary angiography showed a filling defect across the left main coronary artery bifurcation extending into the left anterior descending artery and the ramus circumflex. Transthoracic echocardiography revealed a thickened prosthesis leaflet with signs of slight stenosis. Cardiac computed tomography angiography showed a mass on the left coronary cusp of the valve prosthesis, suggestive for vegetation or thrombus. The scan also revealed central luminal filling defects, indicative for thrombus or septic emboli. Blood cultures proved positive for Propionibacterium acnes, therefore the patient was treated for prosthetic valve endocarditis. Computed tomography angiography offers high diagnostic accuracy for detecting infective endocarditis and renders complementary information about valvular anatomy, coronary artery disease and the extension of infections.

CASE REPORT

A 73-year old man presented with a posterolateral ST-elevated myocardial infarction 9 months after a biological aortic valve replacement (Sorin Crown, 27 mm) for aortic valve stenosis. He had radiating chest pain without other cardiac complaints or signs of fever.

Physical examination was unremarkable, his heart rate was 75 beats per minute, blood pressure was 135/80 mmHg and body temperature was 37.1 °C. Troponin T levels at admission were 0.145 µg/L (< 0.01 µg/L) and C reactive protein (CRP) was 14.3 mg/L (< 10 mg/L). Previous invasive coronary angiography (ICA) in the work-up for aortic valve replacement showed only slight wall irregularities in the left anterior descending artery (LAD). The patient was known with diabetes mellitus type 2 and experienced a stroke and a transient ischemic attack, 6 and 8 years earlier, respectively.

Transthoracic echocardiography (TTE) revealed a concentric hypertrophic left ventricle with good systolic function, a functioning aortic valve prosthesis with signs of slight stenosis (maximal pressure gradient 19 mmHg) and a thickened valve prosthesis leaflet. Following symptoms and
laboratory results, an emergency ICA was performed and showed a filling defect across the left main coronary artery bifurcation extending into the left anterior descending artery (LAD) and the ramus circumflex (LCx) (Figure 1). The thromboembolism was suspicious for valvular origin given the findings on echocardiography.

A retrospective gated cardiac computed tomography angiography (CCTA) (3rd generation dual-source CT scanner, Siemens Somatom Force, Forchheim, Germany) of the full cardiac cycle was performed to further evaluate valve function and to visualize the possible thickened valve leaflet and perivalvular abnormalities (abscesses). A non-mobile mass was detected at the left coronary cusp (7 x 9 x 12 mm) of the aortic valve prosthesis (Figure 2a) suggestive for vegetation or thrombus. Central luminal filling defects were visualized in the proximal LAD (Figure 2b), distal LAD, LCx and right coronary artery (RCA), indicative of thrombus or septic emboli. Distal to the large LAD filling defect a myocardial infarction in the LAD territory was confirmed by visualization of a low attenuation area in the left ventricular myocardium (Figure 3) and hypokinetic wall motion in the basal anterior left ventricular myocardium. Transesophageal echocardiography (TEE) confirmed the presence and location of the valvular mass (Figure 4).

Two days later the CRP rose to 117.9 mg/L and the body temperature of the patient fluctuated between 36.5°C and 38.7°C. Blood cultures proved positive for Propionibacterium acnes, therefore the patient was treated for prosthetic valve endocarditis (PVE) with heparin and penicillin for 6 weeks. The man improved clinically and was discharged from the hospital after 3 weeks of hospitalization.

**DISCUSSION**

Implanted prosthetic heart valves are susceptible to thrombus formation resulting in prosthetic valve dysfunction with an increased risk of thromboembolism [1]. In patients with prosthetic heart valves, coronary artery thromboembolism should be considered prosthesis-related until proven otherwise.

**Etiology & Demographics:**

Prosthetic valve endocarditis (PVE) is a form of infective endocarditis (IE) and is defined as an endovascular microbial infection occurring on parts of a valve prosthesis or on reconstructed native heart valves. PVE occurs in 1-6 percent of patients with highest prevalence in first post-surgical year [2]. Mechanical valves are more susceptible to thrombus formation than bio-prosthetic valves, which more closely resemble native valve tissue and function. The median age of patients with PVE ranges between 47 and 69 years old and the incidence is increased in persons of 65 years of age and older [2]. Male patients are more commonly affected by IE than females with a male to female ratio ranging between 3.2 and 9:1. Other risk factors include: intravenous drug use, poor dentition, structural, congenital or valvular heart disease, history of infective endocarditis, chronic hemodialysis or HIV infection.

PVE can arise early or late after surgery. This classification reflects the differentiation between infections directly related to the surgical procedure (early onset infection) and infections with a similar etiology to native heart valve endocarditis (NVE) (late onset infections). As the surgical material becomes endothelialized over the months following surgery, platelet-fibrin thrombi probably form on the prosthesis and serve as adherence sites for the organisms. In contrast to early onset PVE, the pathogenesis of late infections shows an increase in infections with community-acquired bacteria like Streptococcus spp and a decrease in hospital-acquired bacteria like methicillin-resistant staphylococcus and Gram-negative bacillus [3].

**Clinical & Imaging findings:**

A definitive diagnose of infective endocarditis is established by demonstrating microorganisms in vegetations by either culture or histology. Alternatively, a clinical diagnosis is established based on the Duke diagnostic criteria. These criteria are based on a combination of clinical, microbiologic, pathologic, and echocardiographic characteristics. The IE diagnosis according to the Dukes criteria requires either 2 major criteria, the combination of 1 major criterion and 3 minor criteria, or 5 minor criteria. Besides a positive blood culture, echocardiographic evidence of endocardial involvement is a major criterion for the presence of infective endocarditis. In native heart valves, TTE will be followed by TEE if positive and cardiac CT should be considered. In case of prosthetic heart valves, TEE should be acquired directly and cardiac CT or PET-CT or SPECT should be considered. Although current imaging guidelines advice TEE in suspected PVE, it has a lower diagnostic value in NVE. When negative, it does not rule out PVE and an alternative imaging modality (CTA or nuclear imaging) should be considered [2]. Recent evidence suggests that CT and TEE have similar diagnostic accuracy to detect infective endocarditis in patients with and without prosthetic valves [4]. CTA may render complementary information to TEE for assessment of valvular thrombus, vegetations, perivalvular abscesses and other complications of periannular extension of infections and is able to visualize coronary emboli, perfusion defects and heart (valve) function [5]. Imaging findings consist of a mass or thrombus on a (prosthetic) valve or supporting structure, a perivalvular/anular or myocardial abscess, indications of an intracardiac fistula, development of partial dehiscence of a prosthetic valve, decreased heart (valve) function or a pseudo aneurysm. In heart valve infective endocarditis CTA additionally renders quantitative information about the anatomy, heart valve calcifications, valve size, the aortic root and ascending aorta that is useful for treatment planning and potential surgical approach. In the presented case report, the non-mobile mass at the left coronary cusp of the aortic valve prosthesis (Figure 2a and Figure 4) was highly suggestive for PVE with vegetation formation, given that the blood cultures were positive for Propionibacterium acnes. The thrombus in the LAD and LCx were indicative for thrombus or septic emboli and most likely originated from the vegetation.

**Treatment & Prognosis:**

The treatment of PVE focusses on the identification of the causative organism and the selection of an effective
bactericidal antimicrobial regimen. Surgery may be required if antibiotic therapy fails and is particularly indicated in patients having signs or symptoms of congestive heart failure resulting from valve dehiscence or dysfunction. Other indications for surgery include paravalvular extension of the infection, intracardiac fistula, aortic abscesses or destructive penetrating lesions. Despite advances in knowledge, PVE remains a potential life-threatening disease with mortality rates ranging from 23% to 48% [6].

**Differential Diagnosis:**

**Hypo-attenuated leaflet thickening**

Hypo-attenuated leaflet thickening (HALT) is seen after surgical and transcatheter valve replacement (TAVR) and was diagnosed in 13-23% of patients in recent registries of TAVR patients with ~3 month post procedural CT scan. Most patients are asymptomatic and routine echocardiography may demonstrate an increased aortic valve gradient. The abnormality may resolve by anticoagulation therapy, that may be guided by CT in the follow up after TAVR [7]

**Thromboembolic disease**

Thromboembolic disease from an origin other than a (prosthetic) valve should also be considered (conduction disorders leading to arrhythmia, left atrial thrombus in atrial dilatation and/or atrial fibrillation, ventricular thrombus in dilatative cardiomyopathy or left ventricular aneurysm, intracardiac mass such as a myxoma, heart valve tumor (fibroelastoma).

**Clinical perspectives**

The new European guidelines reinforce the role of CT in suspected infective endocarditis, but recommend ICA for the preoperative assessment of coronary artery disease (CAD) [9], hereby driving double diagnostics in patients presenting with infective endocarditis. Besides the similar diagnostic accuracy of CT and TEE for the detection of infective endocarditis [4], recent systematic reviews and meta-analyses showed high diagnostic performance of CTA for the exclusion of significant CAD in patients receiving heart valve surgery and TAVI [10, 11]. CTA could replace TEE to detect infective endocarditis in patients with and without prosthetic valves and serve as a gatekeeper to invasive coronary angiography for exclusion of significant CAD. It would serve as a less invasive alternative to TEE and reduce the number of invasive coronary angiography procedures performed. Hereby considering that invasive coronary angiography may be associated with the lodgement of aortic vegetations that may cause arterial embolisms. In addition to this risk, CCTA as first line diagnostics in patients suspected for infective endocarditis could result in a significant cost reduction [12] and could reduce physical and mental strain [13] by reducing the number of invasive tests performed. As a corollary, it could reduce the number of complications [14, 15] and reduce hospital admission duration, immobilization and absenteeism. For these reasons, CTA should be considered as the first line diagnostic approach to detect and evaluate infective endocarditis and to rule out significant coronary artery disease in haemodynamically stable patients.

**TEACHING POINT**

Prosthetic valve endocarditis (PVE) is a serious complication of heart valve implementation with mortality rates during admission of 20%. Concomitant coronary artery thromboembolism should be considered prosthesis-related until proven otherwise and CTA should be considered for the detection of PVE, given the similar diagnostic accuracy as TEE. It is less invasive and renders complementary information about the extension of the infections, valvular anatomy and CAD, that could be translated in enhanced diagnosis and improved treatment decisions (e.g. surgical planning).

**REFERENCES**


Cardiac Imaging: Complementary role of cardiac computed tomography angiography in the diagnosis of prosthetic aortic valve endocarditis and septic coronary embolism - a case report

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Figure 1: 73 year old male patient with suspected infective endocarditis.
FINDINGS: The black arrows indicate a filling defect across the left main coronary artery bifurcation, extending into the left anterior descending artery and the left circumflex artery.
TECHNIQUE: Invasive coronary angiography

Figure 2: 73 year old male patient with suspected infective endocarditis.
FINDINGS: A) Axial- and coronal oblique views of the aortic valve. A mass (7 x 9 mm x 12 mm) at the aortic valve leaflet is shown (Arrows), suggestive for vegetation or thrombus. B) Coronary artery curved multi-planar reformats. Central luminal filling defects (arrows) are shown in the right coronary artery, proximal and distal left anterior descending and proximal ramus circumflex, indicative of thrombus or septic emboli. These findings are in agreement with the results from invasive coronary angiography.
TECHNIQUE: Retrospective gated cardiac computed tomography angiography, 292 mAs, 120 kV, 0.6 mm slice thickness, 96 ml name contrast material (Iopromide 300 mg iodine/ml). The provided images were acquired at 70% of the R-R interval.
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Figure 3: 73 year old male patient with suspected infective endocarditis.
FINDINGS: Long-axis view (3 and 2 chamber) and basal short-axis view of the left ventricle showing an area of left ventricular myocardium with low attenuation (*), indicative for a low perfusion area at risk of myocardial ischemia and infarction and corresponding to the coronary territory with filling defects.
TECHNIQUE: Retrospective gated cardiac computed tomography angiography, 292 mAs, 120 kV, 0.6 mm slice thickness, 96 ml name contrast material (iopromide 300 mg iodine/ml). The provided images were acquired in 70% of the cardiac cycle.

Figure 4 (left): 73 year old male patient with suspected infective endocarditis.
FINDINGS: Short axis view of the aortic valve showing a non-movable valvular mass at left coronary cusp (*).
TECHNIQUE: Transesophageal echocardiography
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**Etiology**
Endovascular, microbial infection of the heart, occurring on valve prosthesis or reconstructed heart valves.

**Incidence/ prevalence**
1-6% of patients, with highest prevalence in first post-surgical year.

**Gender ratio**
The male: female ratio ranges from 3:2 to 9:1.

**Age predilection**
Older age is associated with increased risk.

**Risk factors for IE**
Age >60 years, male sex, injection drug use, poor dentition, structural, congenital or valvular heart disease, prosthetic heart valve or other intravascular device, history of infective endocarditis, chronic hemodialysis or HIV infection.

**Microbiology of PVE**
*(<2 months, 2 to 12 months, >12 months)*
- Streptococci (2%, 13%, 30%), Pneumococci (0%, 0%, 1%), Enterococci (8%, 11%, 11%), Staphylococcus aureus (30%, 13%, 22%), Coagulase-negative staphylococci (28%, 36%, 12%), HACEK group (0%, 0%, 4%), Gram-negative bacilli (10%, 4%, 5%), Fungi (9%, 8%, 1%), Polymicrobial/miscellaneous (3%, 8%, 4%), Polymicrobial/miscellaneous (4%, 0%, 2%)

**Treatment**
Antibiotic treatment. Surgical replacement of the prostheses if antibiotic treatment fails.

**Prognosis**
Mortality rates ranging from 23% to 48%.

**Findings on imaging**
Valvular treatment. Surgical replacement of the prostheses if antibiotic treatment fails.

**Table 1: Summary table of infective endocarditis.**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>TEE</th>
<th>CTCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoattenuated leaflet thickening</td>
<td>• Thickened valve leaflet visible as a radiolucent mass</td>
<td>• Thickened valve leaflet, visible as a hypodense mass (contrast filling defect)</td>
</tr>
<tr>
<td>Haemolysis</td>
<td>• Turbulent flow through prosthetic valve or between the sewing ring and native valve, visible with colour Doppler</td>
<td>• Gap between annulus prosthetic valve, visible due to filling with contrast material</td>
</tr>
<tr>
<td>Thromboembolic disease</td>
<td>• Right ventricle dilatation and/or dysfunction</td>
<td>If sufficient contrast in the pulmonary veins:</td>
</tr>
<tr>
<td></td>
<td>• Inferior vena cava dilatation with lack of collapse during inspiration</td>
<td>• Central low attenuation filling defect</td>
</tr>
<tr>
<td></td>
<td>• Interventricular septum flattening</td>
<td>• Local vessel enlargement due to impaction of thrombus by pulsatile flow</td>
</tr>
<tr>
<td>Primary cardiac neoplasms</td>
<td>• Cardiac mass adjacent to the valve leaflet or other endocardial structure</td>
<td>• Hypodense mass (contrast filling defect) adjacent to the valve leaflet or other endocardial structure</td>
</tr>
<tr>
<td>Atrial myxoma</td>
<td>• Atrial mass</td>
<td>• Hypodense atrial mass</td>
</tr>
<tr>
<td>Cardiac conduction disturbances</td>
<td>• Cardiac asynchrony</td>
<td>• None</td>
</tr>
<tr>
<td></td>
<td>• Reduced ejection fraction</td>
<td></td>
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<tr>
<td></td>
<td>• Decreased contractility</td>
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**Table 2: Differential diagnoses table for infective endocarditis.**

<table>
<thead>
<tr>
<th>ABBREVIATIONS</th>
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<tbody>
<tr>
<td>CAD = coronary artery disease</td>
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<tr>
<td>CCTA = Cardiac computed tomography angiography</td>
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<tr>
<td>CRP = C reactive protein</td>
</tr>
<tr>
<td>CT = Computed tomography</td>
</tr>
<tr>
<td>ICA = invasive coronary angiography</td>
</tr>
<tr>
<td>LAD = left anterior descending artery</td>
</tr>
<tr>
<td>PVE = prosthetic valve endocarditis</td>
</tr>
<tr>
<td>LCx = Left circumflex artery</td>
</tr>
<tr>
<td>RCA = Right coronary artery</td>
</tr>
<tr>
<td>TEE = Transoesophageal echocardiography</td>
</tr>
<tr>
<td>TTE = Transthoracic echocardiography</td>
</tr>
</tbody>
</table>

**KEYWORDS**
Computed tomography; prosthetic heart valve endocarditis; computed tomography coronary angiography; invasive coronary angiography; prosthetic heart valve thrombosis; coronary embolization; transesophageal echocardiography

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