

# The effect of transcatheter aortic valve implantation on pulmonary artery pressures in a patient suffering from chronic heart failure: a case report

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## Background

Pulmonary hypertension (PH) is most commonly caused by left-sided heart disease and may negatively affect the long-term prognosis and quality of life of patients with chronic heart failure (CHF). CardioMEMS (Micro-Electro-Mechanical-System) allows physicians to monitor pulmonary artery (PA) pressures remotely and optimize heart failure treatment based on haemodynamic parameters, which provides further insight into the effect of valvular interventions.

## Case summary

We present a case of a 61-year-old male patient with an ischaemic cardiomyopathy, poor LV function, moderate to severe mitral regurgitation, and severe aortic valve regurgitation in refractory heart failure. Right heart catheterization and CardioMEMS monitoring revealed severe pulmonary hypertension with mean PA pressures of 55 mmHg and a mean pulmonary capillary wedge pressure of 21 mmHg despite up titration of heart failure medication and sildenafil. Pulmonary and systemic causes of pulmonary hypertension were excluded. After heart team consensus, the patient underwent transcatheter aortic valve implantation (TAVI) which resulted in normalization of PA pressures and a significant improvement of functional performance.

## Discussion

To the best of our knowledge, this is the first case report describing the direct effects of TAVI on continuous PA pressures in a patient with poor LV function and severe aortic regurgitation. Elective TAVI appeared to be safe and very effective in reverting severe pulmonary hypertension. Most strikingly, drug interventions could not affect the elevated pulmonary pressures, but TAVI corrected the aortic valve insufficiency with normalization of left-sided pulmonary hypertension.

## Keywords

Case report • Heart failure • TAVI • CardioMEMS • Pulmonary hypertension • Remote monitoring

## Learning points

- CardioMEMS (Micro-Electro-Mechanical-System) pulmonary artery (PA) sensor is a reliable tool to monitor PA pressures even in patients with severe pulmonary hypertension.
- CardioMEMS PA sensor was able to monitor the impact of the actual valvular intervention on cardiac haemodynamics over time.
- Continuous PA monitoring might add important haemodynamic insights to the effect of valvular interventions and as such add a new dimension to current therapies.

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## Introduction

Pulmonary hypertension (PH) is often seen in patients with heart failure (HF), both in reduced and preserved left ventricular ejection fraction. Left heart disease is the most common cause of PH. Elevated pulmonary artery (PA) pressures may negatively affect the long-term prognosis and quality of life of patients with chronic heart failure (CHF). Chronic heart failure is often accompanied by valvular heart disease. Aortic regurgitation (AR) generates left ventricle (LV) volume and pressure overload, leading to eccentric hypertrophy, and progressive LV dysfunction.<sup>1-3</sup> Over the past decade, transcatheter aortic valve implantation (TAVI) has become a widely accepted method for the treatment of aortic valve stenosis and is now increasingly being used for the treatment of aortic regurgitation as well. In the field of HF, remote monitoring and telemonitoring have been introduced to relieve the large burden of CHF on health care systems. Among the most promising initiatives of remote monitoring is CardioMEMS (Micro-Electro-Mechanical-System) (Abbott Inc., Atlanta, GA, USA), a small implantable sensor capable of remotely measuring PA pressures on a daily basis. Remote monitoring by CardioMEMS was shown to be effective in the prevention of HF hospitalizations in the US CHAMPION Trial.<sup>4</sup> A trial to test the effectiveness of CardioMEMS for the Western European Setting is currently ongoing in the Netherlands.<sup>5</sup>

## Timeline

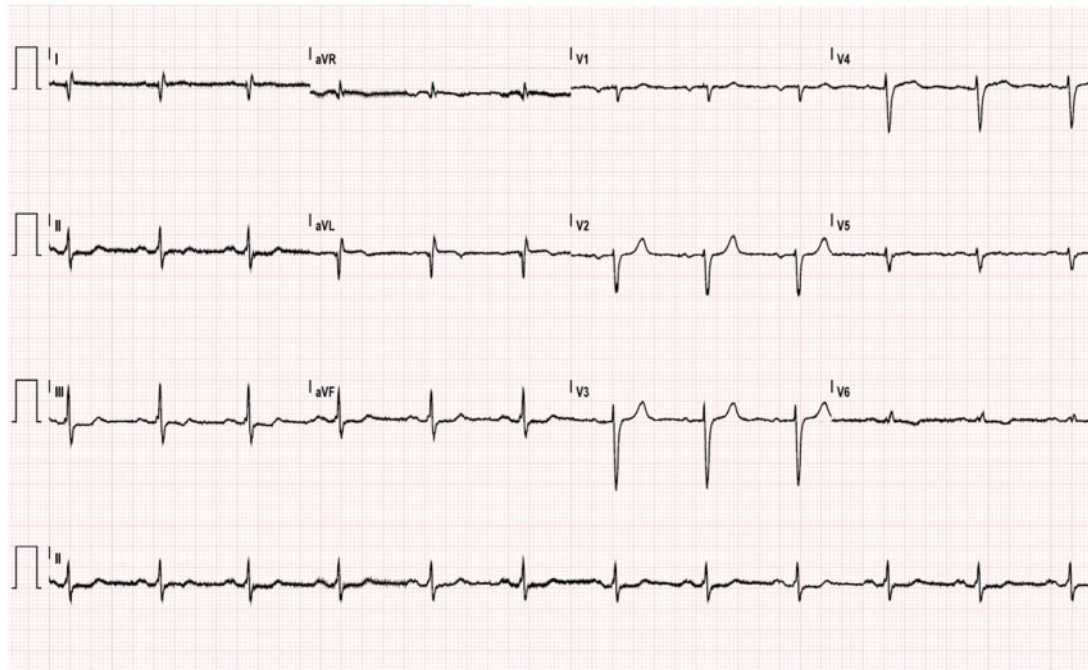
discharged after a long hospital admission for recurrent acute myocardial infarction and cardiogenic shock due to early in-stent thrombosis after elective PCI of the left anterior descending (LAD) artery. The patient was in New York Heart Association (NYHA) Class IIIb with the main complaints being fatigue and exertional dyspnoea despite optimal doses of HF medication according to the 2016 ESC HF Guidelines.<sup>6</sup> On physical examination, there were no signs of clinical congestion. The electrocardiogram showed a sinus rhythm of 68 beats per minute with a PR interval of 206 ms and moderate intraventricular conduction delay without any signs of a bundle branch block (Figure 1). Echocardiogram showed an impaired systolic left ventricular function with an ejection fraction of 28%, moderate aortic regurgitation, moderate mitral regurgitation, and an inferior vena cava of 15 mm with >50% collapse due to a high dose of diuretics in an euvoletic state. The laboratory results showed an estimated glomerular filtration rate of 38 mL/min, serum creatinine of 166 µmol/L, urea of 19.4 mmol/L, and N-terminal prohormone of brain natriuretic peptide (NT-pro-BNP) of 557 pmol/L. The patient first had an internal cardioverter-defibrillator implanted for primary prevention of sudden cardiac death and then had a CardioMEMS implanted to allow for remote haemodynamic monitoring and optimization of HF care. At right heart catheterization during CardioMEMS implant, the patient had severe pulmonary hypertension (PH) with a systolic, diastolic, and mean PA pressure of respectively 84, 38, and 54 mmHg, a mean pulmonary capillary wedge pressure of 21 mmHg, and right atrial pressure of 7 mmHg. In the months following CardioMEMS implantation, the mean pulmonary artery pressure (mean PAP)

Date	Events
1999	Inferior wall myocardial infarction
February 2017	Non-ST-segment elevation myocardial infarction, percutaneous coronary intervention (PCI) of circumflex artery
November 2018	Elective PCI of left anterior descending (LAD) and circumflex artery which was complicated by early LAD in-stent thrombosis and cardiogenic shock
March 2019	Ventricular inhibited internal cardioverter-defibrillator (VVI-ICD) implantation
May 2019	CardioMEMS (Micro-Electro-Mechanical-System) implantation after which severe pulmonary hypertension was noticed. Up titration of medication: <ul style="list-style-type: none"> <li>• High dose diuretics</li> <li>• Titrating higher dose angiotensin receptor-neprilysin inhibitor (ARNI)</li> <li>• Sildenafil</li> </ul>
July 2019	Analysis by pulmonologist for pulmonary/systemic causes of pulmonary hypertension which showed no clear non-cardiac causes
April 2020	Elective transcatheter aortic valve implantation for severe aortic valve regurgitation after which normalization of pulmonary artery pressures occurred

## Case presentation

A 61-year-old male with a history of inferior wall myocardial infarction and non-ST-segment elevation myocardial infarction (NSTEMI) treated by percutaneous coronary intervention (PCI) of the circumflex artery was seen in the outpatient clinic. The patient was recently

remained elevated between 50 and 55 mmHg. Up titration of loop diuretics (bumetanide maximum tolerated daily dose of 7.5 mg), mineralocorticoid receptor antagonists (eplerenone daily dose of 50 mg), sacubitril/valsartan (maximum tolerated daily dose of 96/104 mg), and sildenafil to a maximum tolerated daily dose of 60 mg did not improve PA pressures nor symptoms. The patient was



**Figure 1** The electrocardiogram on presentation in the outpatient clinic.

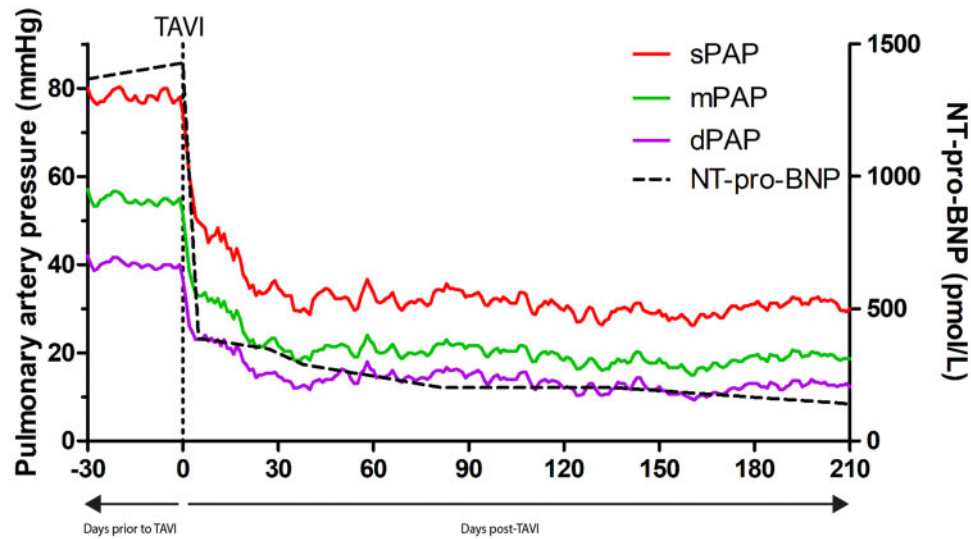
referred to the pulmonologist for analysis of non-cardiac causes of PH. Interstitial lung disease, chronic thrombo-embolic pulmonary hypertension, and sleep apnoea were excluded and the PH was classified as WHO Type 2 after multidisciplinary consultation between the cardiologists and pulmonologists. In compassionate use we tried high dose sildenafil to lower right-sided pressures, unfortunately without effect. A new transthoracic echocardiogram showed a tricuspid aortic valve with severe regurgitation and a pressure half-time (PHT) of 310 ms, a left ventricular ejection fraction of 31%, moderate to severe mitral regurgitation, and an estimated systolic right ventricular pressure of 60 mmHg on echo. At this stage, it was unclear to what extent the aortic valve regurgitation or the mitral regurgitation contributed to the PH and symptoms and to which valve the intervention should be targeted. The Heart Team accepted the patient for TAVI to first reduce LV volume overload. MitraClip was not selected due to a potential clinical course towards LV assist device (LVAD) therapy. Surgical aortic valve replacement would have been a high-risk procedure because of severely impaired LV function. Furthermore, this could mean re-operation for the potential course towards LVAD implantation in the future. Besides, it was not fully clear to what extent AR contributed to symptoms and pulmonary hypertension because of concomitant severely impaired LV function. Surgery was thus deemed undesirable and TAVI was preferred. EuroSCORE II and Society of Thoracic Surgeons Score were 4.85% and 1.35%, respectively. On CT-scan, the annulus area was 496 mm<sup>2</sup>, annulus diameter was 25.1 mm and the Agatston score for the aortic valve was 326 indicating little calcification. Based on these findings, a 27 mm LOTUS Edge valve was implanted through the transfemoral way without any complications. Mean PAP (mPAP) was 56 mmHg on

**Table 1** Echocardiographic parameters pre-transcatheter aortic valve implantation and post-transcatheter aortic valve implantation

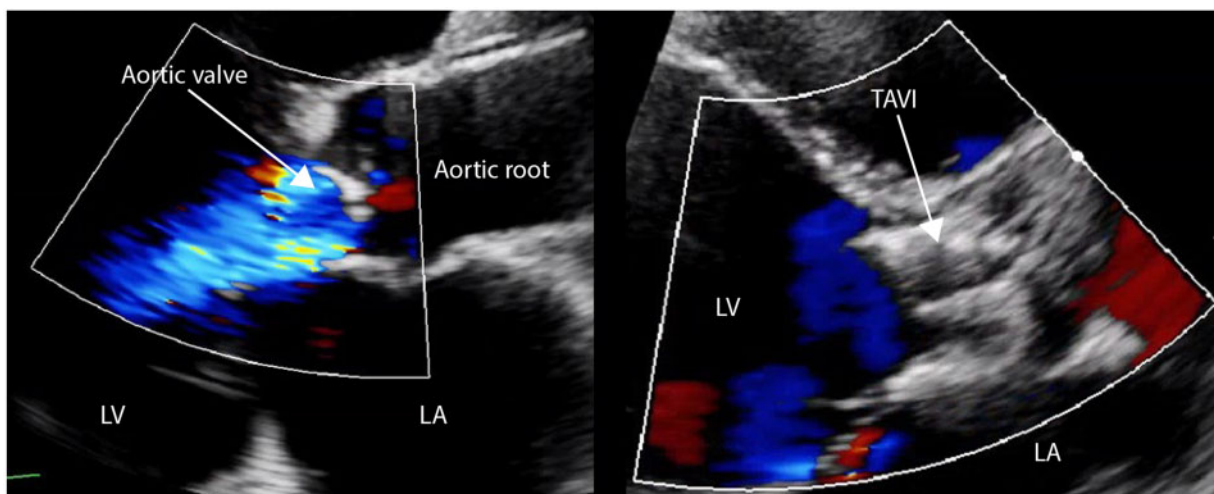
	Pre-TAVI	Post-TAVI
<b>Aortic valve</b>		
PHT	310 ms	
V <sub>max</sub>	402 cm/s	194 cm/s
Visual assessment	Severe regurgitation	Trace regurgitation
<b>Mitral valve</b>		
PISA	6 mm	
Visual assessment	Severe regurgitation	Trace regurgitation
Tricuspid valve gradient	55 mmHg	29 mmHg
Right ventricular systolic pressure	60 mmHg	29 mmHg
LVEF	31%	30%
LVEDD	67 mm	66 mm
LA diameter	47 mm	42 mm
Ascending aortic diameter	33 mm	30 mm

LA, left atrium; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; PHT, pressure half-time; PISA, proximal isovelocity surface area; TAVI, transcatheter aortic valve implantation.

the day of TAVI. After successful implantation, both the aortic valve insufficiency and mitral regurgitation were reduced to a trace and the mPAP decreased to 42 mmHg 1 day post-procedural. Several



**Figure 2** Pulmonary artery pressures pre- and post-transcatheter aortic valve implantation.



**Figure 3** Left: Mid-diastolic severe aortic regurgitation prior to transcatheter aortic valve implantation. Right: Mid-diastolic trace aortic regurgitation post-transcatheter aortic valve implantation.

echocardiographic parameters pre-TAVI and post-TAVI have been summarized in *Table 1*. In the following weeks, mPAP further decreased to 20 mmHg without any additional pharmacological interventions (*Figures 2 and 3*). After the mPAP had been stable around 20 mmHg for several weeks, sildenafil was completely stopped and the diuretics were decreased to less than 50% of the original daily dose. PA pressures remained stable after lowering the diuretics and the patient improved from NYHA Class IIIb to II. During the last contact in the outpatient clinic 7 months post-TAVI, the mean PA pressure had remained stable in the normal range around 20 mmHg. Furthermore, the heart failure-related complaints had decreased significantly and the patient improved further to NYHA Class I.

## Discussion

Remote monitoring by the CardioMEMS PA sensor has been shown to be effective in lowering the risk of HF hospitalizations in NYHA Class III HF patients. Currently, studies are being conducted to assess its efficacy in the Western European setting and a broader patient population.<sup>5,7</sup>

Valvular interventions, especially the less invasive percutaneous methods such as TAVI and MitraClip, are increasingly applied in chronic HF.<sup>8</sup> The efficacy of these interventions is mainly evaluated by (subjective) anamnestic and physical signs and echocardiography. Unfortunately, these methods do not allow for an objective and continuous assessment of the actual haemodynamic effects of valvular

intervention. Usually, what happens between valvular intervention and the moment of the first contact at the outpatient clinic several weeks to months later is a complete black box.

To the best of our knowledge, this is the first case report describing the direct effects of TAVI on continuous pulmonary artery pressures. We have shown that CardioMEMS is very well suited for continuous monitoring of PA pressures, even in severe PH, and that it provides valuable insights in understanding the haemodynamics and success of valvular intervention.

CardioMEMS monitoring showed severe pulmonary hypertension despite maximum tolerated doses of heart failure medication including angiotensin receptor-neprilysin inhibitor (ARNI) and sildenafil. After pulmonary disease was excluded, we started thinking about the role of AR earlier and decided within the Heart Team that the AR probably did contribute to pulmonary hypertension and therefore the patient was accepted for TAVI. Without CardioMEMS, decisions would have been made based on clinical findings, the patient would likely have remained in the same impaired condition and the severity of AR would probably not have become apparent until acute worsening heart failure would have occurred. CardioMEMS post-TAVI made it possible to accurately assess the haemodynamic effects of valvular intervention daily, which would not have been possible with traditional serial echocardiography. Furthermore, daily evaluation of pulmonary artery pressures enabled us to guide medical therapy with down-titration of HF medication and improved functional performance as a result.

CardioMEMS can help assess the need for valvular intervention based on haemodynamic parameters, especially when it is unclear to what extent the valvular heart disease contributes to the severity of heart failure and symptoms. The presented case is a perfect example of this hypothesis.

The additive value of continuous PA monitoring by CardioMEMS on top of the standard evaluation of valvular interventions seems clinically plausible and might become clearer in the future when CardioMEMS will be applied more routinely as a standard of care in patients with CHF. Future reports are needed to further elucidate the potential of this hybrid construction of CardioMEMS and valvular intervention. In this case of severe AR, TAVI appeared to be safe and effective in lowering PA pressures and optimizing the heart failure treatment for this patient.

## Conclusions

CardioMEMS may help identify factors other than congestion, such as valvular heart disease, that contribute to pulmonary hypertension despite maximum tolerated doses of heart failure medication. In this case, severe aortic valve regurgitation contributed significantly to the severity of pulmonary hypertension and mitral regurgitation. Most strikingly, although drug interventions could not intervene in the severely elevated pulmonary pressures, elective TAVI appeared to be safe, led to a significant decrease in volume backflow of the aortic valve regurgitation and consequently had major impact on the mechanism and severity of left-sided pulmonary hypertension. CardioMEMS allows physicians to optimize heart failure therapy and is well suited for evaluation of both pharmacological and non-pharmacological interventions, such as monitoring the success of valvular interventions like TAVI in this illustrative example.

## Lead author biography



Sumant Radhoe graduated in Medicine and Clinical Research from the Erasmus University Rotterdam in 2018. He is currently pursuing his PhD degree at the department of Cardiology of the Erasmus University Medical Center in Rotterdam. His main academic interests include heart failure, remote monitoring of heart failure patients and advanced heart failure therapy.

## Supplementary material

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

**Slide sets:** A fully edited slide set detailing these cases 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 has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** J.B. has received independent research grants and speaker fees from Abbott. N.V.M. has received research grant support and advisory fees from Abbott, Boston Scientific, Medtronic, Daiichi Sankyo and PulseCath B.V. S.R. and J.V. have no conflict of interest to declare.

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