# Pericardial calcification: Don't rush! The importance of invasive hemodynamic assessment

## *Calcificación pericárdica: ¡no se precipite! La importancia de la valoración hemodinámica invasiva*

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**To the Editor,**

Constrictive pericarditis (CP) is cause for diastolic heart failure due to the presence of a non-distensible pericardium. Diagnostic suspicion is key here because, even though it is a potentially curable condition, if untreated, its morbimortality rate is high.[1] Its differential diagnosis with restrictive cardiomyopathy can be a real challenge.[2] We present a case where the invasive hemodynamic assessment performed through cardiac catheterization showed its validity and diagnostic importance in the therapeutic decision-making process.

We present the case of a 59-year old hypertensive woman, former smoker and with a prior medical history of Hodgkin lymphoma at 18 years old —for which she was treated with tele-cobalt therapy and chemotherapy— and carcinoma located in her left breast that was mastectomized when she was 52 years of age. She was re-admitted twice in 2 months due to right heart failure with presence of the Kussmaul sign (video 1 of the supplementary data). Due to the poor acoustic window, the transthoracic echocardiography conducted only showed pericardial thickening and preserved biventricular systolic function without significant valvular heart disease. In the presence of severe pericardial calcification (figure 1) and due to the patient's clinical manifestations and prior medical history of chest radiation, cardiac catheterization was decided to confirm the suspicion of post-radiotherapy CP and indicate surgical pericardiectomy.

The hemodynamic assessment confirmed a reduced cardiac index (1.82 L/min/m$^2$) and a significant increase of pressure in the right cardiac cavities and other classical signs of constrictive pericarditis,[1-3] such as very deep x and y descent in the right atrium (figure 2A) and dip-plateau morphology in the right ventricular pressure curve (figure 2B). The simultaneous registry of pressures from both ventricles also showed the equalization of their end-diastolic pressures (figure 2C) and ruled out the expected increase of ventricular interdependence with the existence of parallel changes in both pressures with breathing movements (figure 2D). Also, the patient showed moderate postcapillary pulmonary hypertension (figure 2E,F). No significant coronary lesions were found, although the most distal branches of the right coronary artery were somehow fixed at the level of the inferior pericardial calcification

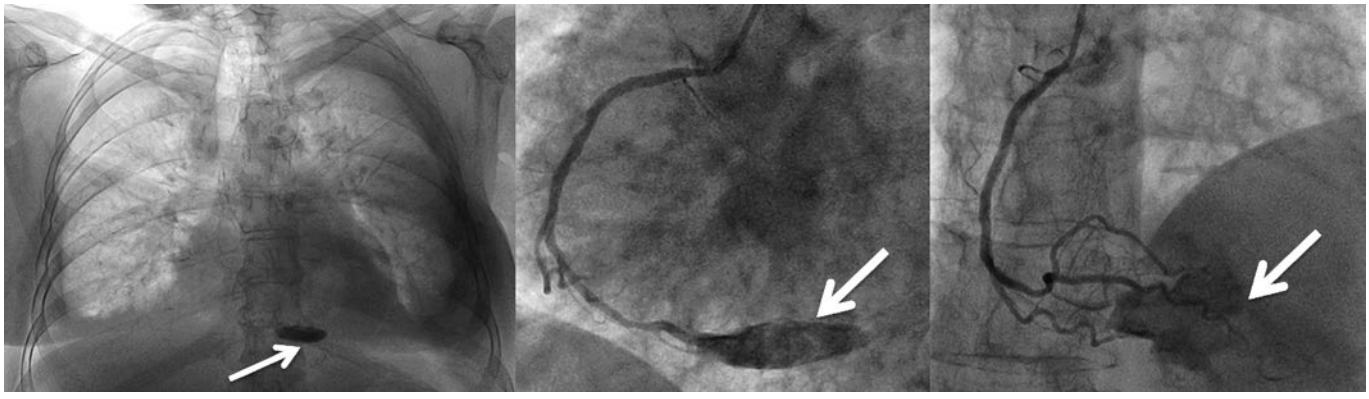

**Figure 1.** Inferior pericardial calcification (arrows).

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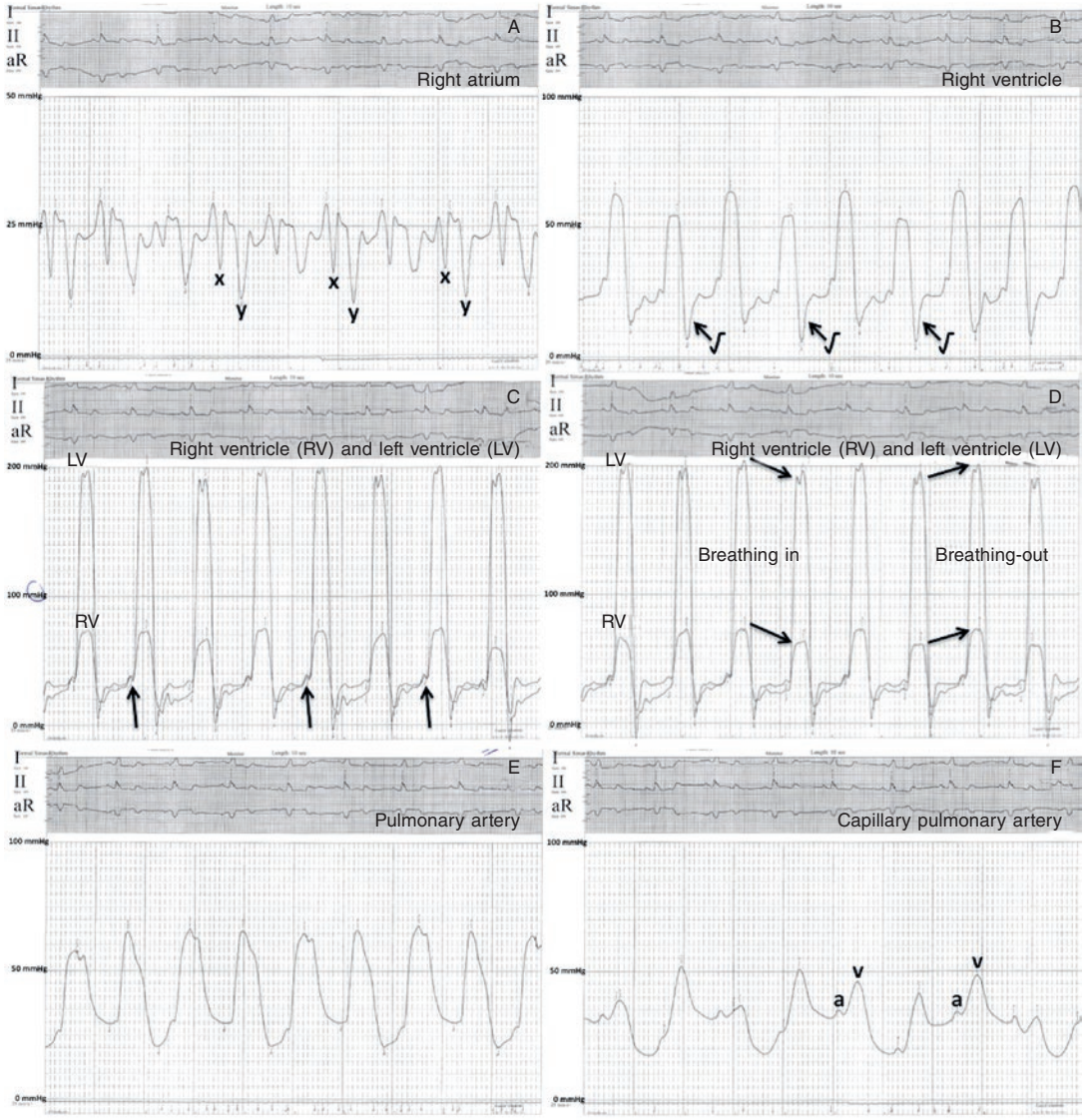


**Figure 2.** Registry of pressures. A: right atrium (average 22 mmHg) with deep sinuses x and y; B: right ventricle (61/10-27 mmHg) with square root symbol (arrow); C and D: right and left ventricles with equalization of both end-diastolic pressures (arrows), parallel decrease (ventricular match) of pressure curves while breathing in parallel increase while breathing-out; E: pulmonary artery (65/25(42) mmHg); and F: capillary pulmonary artery (31 mmHg) with a prominent v wave.

(figure 1 and video 2 of the supplementary data), a finding considered specific of CP.[1]

We should consider the diagnosis of CP in the presence of mainly right heart failure-like symptoms. Clinical findings and non-invasive diagnostic modalities allow to diagnose it in 70% of all cases.[1] However, the remaining patients may need cardiac catheterization for a correct differentiation between CP and restrictive cardiomyopathy, especially those with a prior medical history of radiotherapy or heart surgery in whom pericardial and myocardial damages are usually coexistent.[2,3]

The classical criteria of CP (eg, early fast ventricular filling or end-diastolic pressure equalization of the 4 cardiac chambers, lack of pulmonary hypertension, etc.) have low sensitivity and specificity for differential diagnosis purposes when it comes to restrictive cardiomyopathy.[1-3] To this day, it is widely accepted that the dynamic breathing variations of intracardiac pressures are very accurate[1,2] and have a 97% sensitivity rate and a 100% positive predictive value for the identification of patients with CP.[3] In CP there is a dissociation between intrathoracic and intracardiac pressures with reduced intrathoracic pressure not transferred to the cardiac cavities during breathing, which leads to reduced left ventricular filling. In the presence of a non-distensible pericardium with a relatively fixed intrapericardial volume, this reduced left ventricular filling simultaneously increases to the right ventricular filling. The opposite happens when breathing out. This increased ventricular interdependence automatically translates into impaired ventricular systolic pressures. In other clinical scenarios there is an increase and decrease of both pressures consistent with the breathing cycle (ventricular match), but in the case of CP, these variations simply do not match,[1,2] which is a highly sensitive and specific marker[3] not found in our patient.

Even though the Doppler echocardiogram can assess these changes during the respiration phase using the analysis of transmitral

flow pattern and septal movements,[1] patients who have undergone heart surgery or received radiotherapy in the past usually show acoustic windows that complicate this assessment. As it has already been confirmed, the detection of thickening or pericardial calcification in the different imaging modalities available today does not necessarily reflect a purely constrictive pathophysiology.[3]

Our hemodynamic assessment shows a probable mixed condition of myocardial constriction and restriction due to chest radiation sustained by the patient in her youth. With the data presented here, we believe that the case reinforces the need to perform very precise hemodynamical assessments of these patients before indicating any surgical interventions[2] especially when the perioperative mortality rate of pericardiectomy of these patients can exceed 20%.[1] That is why we initially took a conservative approach and waited on the patient's clinical progression after a slow and adequate optimization of the medical therapy.

## SUPPLEMENTARY DATA

Supplementary data associated with this article can be found in the online version available at https://doi.org/10.24875/RECICE.M19000027.

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# Pulmonary artery pseudoaneurysm as a Swan-Ganz catheter complication

## *Seudoaneurisma de arteria pulmonar como complicación de catéter de Swan-Ganz*

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**To the Editor,**

We present the case of a 65-year-old woman without any known drug allergies and with a previous cardiac history of atrial fibrillation treated with warfarin and rheumatic poly-valve disease with double mitral valve lesion (severe mitral stenosis and moderate-to-severe mitral regurgitation) and severe tricuspid regurgitation with indirect data of pulmonary hypertension admitted due to progressive worsening of her usual dyspnea until becoming dyspnea of minimal exertion and with important limitations in activities of daily living.

She was examined at the cardiovascular surgery unit and on April 17, 2018 she was intervened to replace her mitral valve for a mechanical prosthesis and tricuspid annuloplasty back on April 17, 2018 without intraoperative incidents or complications in the immediate postoperative period neither at the intensive care unit nor at the hospital ward from where she was discharged in due course.

Back in May 31, 2018, the patient was admitted to the emergency room due to exacerbated dyspnea that became dyspnea on moderate exertion accompanied by an increased abdominal perimeter and swelling in both her lower limbs. The blood test confirmed the presence of elevated levels of natriuretic propeptides. The chest x-ray conducted showed congestive signs and the image of a right lung base pulmonary nodule suggestive of phantom tumor not detected in previous x-ray studies. After depletion therapy another follow-up x-ray was performed showing a round well-established image. A chest CT scan was requested that confirmed the presence of a partially thrombosed pseudoaneurysm of the pulmonary artery.

The case was discussed with the cath lab and the embolization of the pseudoaneurysm was decided in June 20, 2018. Using the right femoral access, a 5-Fr JR 4 diagnostic catheter was advanced towards the right pulmonary artery. Then, the catheter was changed over a 0035″ guidewire with a MP 4-Fr catheter to perform

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