

# Intramyocardial dissecting hematoma of the right ventricle secondary to acute myocardial infarction. Case report

*Hematoma intramiocárdico disecante del ventrículo derecho secundario a infarto agudo de miocardio. Reporte de caso*

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## ABSTRACT

**Background:** Intramyocardial dissecting haematoma is a rare form of cardiac rupture that can occur as a complication following acute myocardial infarction or during the remodelling process, with very scarce reports in medical literature. Usually caused by a haemorrhagic dissection among the spiral myocardial fibres. **Case report:** Here we report the case of a 60-year-old man with a previous infarction in the inferior wall extended to the right ventricle, in whom a dissecting intramyocardial haematoma in the free wall of the right ventricle was identified using transthoracic and contrast echocardiography, corroborated with cardiac magnetic resonance. **Results:** By this imaging modalities, it was possible to see the various acoustic densities of the progressive clotting of the hematoma, its extension through the haemorrhagic dissection, as well as its independency in relation to ventricular cavities and extracardiac space by confirming intact epicardial and endocardial layers. **Conclusion:** The patient presented a gradual improvement and hemodynamic stability, and basing on the existent medical literature, it was decided the medical treatment as the therapeutic option, maintaining asymptomatic after three months of follow-up.

## RESUMEN

**Antecedentes:** El hematoma intramiocárdico disecante es una forma rara de ruptura cardíaca que puede ocurrir como una complicación posterior al infarto agudo de miocardio o durante el proceso de remodelación del mismo, con informes muy escasos en la literatura médica. Generalmente causada por una disección hemorrágica entre las fibras espirales del miocardio. **Caso clínico:** Se presenta el caso de un hombre de 60 años con infarto previo en la pared inferior extendido al ventrículo derecho, en el que se identificó un hematoma intramiocárdico disecante en la pared libre del ventrículo derecho mediante ecocardiografía transtorácica y contrastada, corroborado con resonancia magnética cardíaca. **Resultados:** Por medio de estas modalidades de imagen se pudieron observar las distintas densidades acústicas de la coagulación progresiva del hematoma, su extensión a través de la disección hemorrágica, así como su independencia en relación con las cavidades ventriculares y el espacio extracardíaco, confirmando las capas epicárdicas y endocárdicas indemnes. **Conclusión:** El paciente presentó una mejoría gradual y estabilidad hemodinámica, y basándose en la literatura médica existente, se decidió el tratamiento médico como opción terapéutica, manteniéndose asintomático después de tres meses de seguimiento.

## INTRODUCTION

The left ventricular free wall rupture complicates less than 1% of cases of myocardial infarction, usually causing death;<sup>1</sup> in a minority of cases the overlying pericardium may contain the rupture leading to the formation of a pseudo-aneurysm. A less common complication of myocardial infarction is the subepicardial aneurysm (or pseudo-pseudo-aneurysm), a saccular left ventricle expansion communicating with the main cavity, with an outer border represented by a thin epicardial layer. Although the pathophysiology process should involve an initial tearing of the endocardium and subsequent hemorrhage into the myocardial layer, this sequence of events has never been documented. The intramyocardial dissecting hematoma (IDH) is an unusual form of cardiac rupture of the left ventricular wall. It is difficult to estimate the incidence of postinfarction IDH, since most reports are isolated cases and many may go unnoticed if the spontaneous evolution is favorable.<sup>1,2</sup> There are a few case reports of this complication involving the left ventricle, and fewer involving the right ventricle,<sup>3-5</sup> in our country there is no reported experience in this complication. We present a case of this complication during an acute infarct involving the right ventricle.

## CASE REPORT

Male, 60 years old, history of smoking, dyslipidemia and hypertension, both of long evolution.

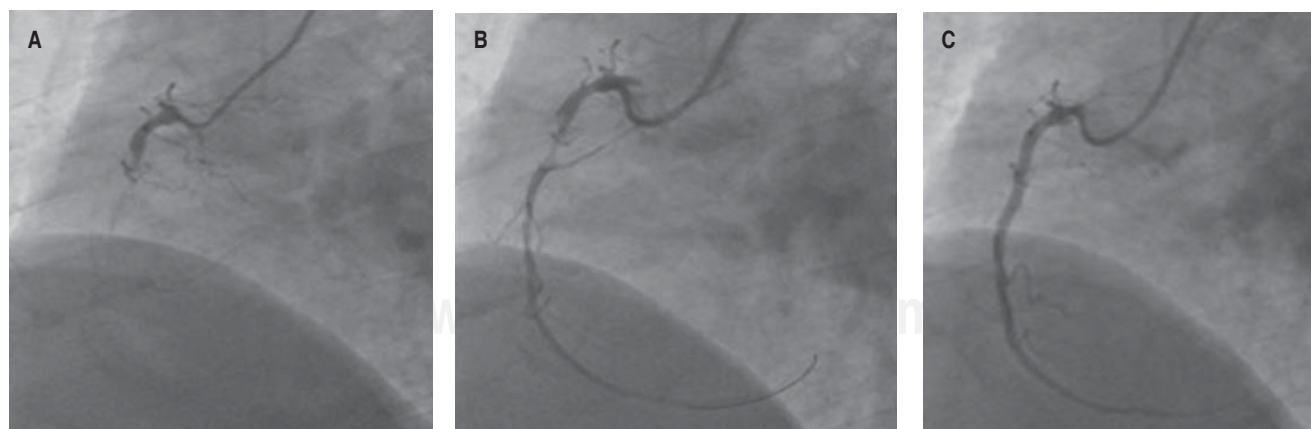
Suddenly began with typical chest pain with a minimal effort, dyspnea and diaphoresis lasting for two hours, as well as an episode of syncope. Required medical assistance and hospitalization in emergency room, diagnosing an ST-segment elevation myocardial infarction of the inferior wall complicated with right ventricle infarction, not reperfused. Was sent to our institution after 16 hours of ischemia, being initially diagnosed with cardiogenic shock (persistent systolic blood pressure < 90 mmHg for at least 30 minutes). The physical examination revealed a regular pulse rate of 60 beats/min and a blood pressure of 110/60 mmHg. Chest radiographs in the posteroanterior and lateral views

showed cardiomegaly with a cardiothoracic ratio of 0.6. His electrocardiogram (ECG) demonstrated signs of an inferior wall infarction with electrical extension to the right ventricle (significant ST-segment elevation in V3R and V4R derivations). Underwent continuous ECG and invasive hemodynamic monitoring, including the use of a pulmonary artery catheter (7.5-F Swan-Ganz thermodilution catheter, Edwards Lifesciences, Irvine, CA, USA) and an arterial line for mean arterial pressure (MAP). Registered a cardiac index (CI) of 2.1 L/min/m<sup>2</sup>, mean pulmonary artery pressure (MPAP) of 35 mmHg, pulmonary vascular resistance (PVR) of 285.3 dyne/seg/cm-5, systemic vascular resistance (SVR) of 1,313.5 dyne/seg/cm-5, right ventricular cardiac power index (rvCPI) of 0.11 W/m<sup>2</sup>, cardiac power index (CPI) of 0.28 W/m<sup>2</sup>, central venous pressure (CVP) of 20 mmHg, and pulmonary artery occlusion pressure (PAOP) of 29.6 mmHg. Due to the cardiogenic shock received norepinephrine 0.5 µg/kg/min and dobutamine 5 µg/kg/min, then underwent to coronary angiography via a transradial approach registering a multivessel coronary disease, the left coronary system with an angiographic stenosis of 85% in the middle segment of the anterior descending artery and normal distal flow, dominant right coronary artery with an acute total occlusion in the proximal segment requiring an elective percutaneous coronary intervention (PCI), advancing a universal guide wire through the total occlusion and predilatation with a 1.5 x 12 mm balloon with recovery of distal flow, finding an angiographic stenosis of 85% in the middle segment. A Resolute Onyx zotarolimus-eluting stent (ZES) (Medtronic, Inc., Santa Rosa, CA, USA) 2.5 x 26 mm was sailed in the middle segment and joined in the proximal segment with a Resolute Onyx ZES (Medtronic, Inc., Santa Rosa, CA, USA) 3.0 x 30 mm with a posterior review with Intravascular Ultrasound (IVUS), showing an adequate expansion of the stents without complications (*Figure 1*). The patient was transferred to a Coronary Intensive Care Unit after the procedure, showing a gradual improvement in their cardiovascular status, the norepinephrine was removed 12 hours later with an adequate pressure control, and the dobutamine was removed 24 hours later

with an acceptable cardiovascular tolerance. At 72 hours maintains hemodynamic stability and stratification is performed with perfusory scintigraphy (Thallium 201-chloride) reporting a nontransmural inferior and inferoseptal myocardial infarction of the left ventricle. Later underwent to a new coronary angiography via a transradial approach and elective PCI to the remainder stenosis. A Resolute Onyx ZES (Medtronic, Inc., Santa Rosa, CA, USA) 3 x 38 mm was sailed in the middle segment of the anterior descending artery and joined in the proximal segment with a Resolute Onyx ZES (Medtronic, Inc., Santa Rosa, CA, USA) 3.5 x 38 mm with a posterior review with IVUS, showing an adequate expansion of the stents without complications, considering the procedure as successful (*Figure 2*). Presenting a gradual improvement and hemodynamic stability. Discharged 24 hours after the procedure, it was performed a cardiac magnetic resonance for further evaluation of the IDH. Maintaining asymptomatic after three months of follow-up.

Echocardiographic follow-up: it was performed at 24, 48 and 96 hours after the symptoms onset. In the first echocardiogram it was reported a left ventricular end-diastolic volume of 96 milliliters, end-systolic volume of 38 milliliters, left ventricle ejection fraction of 60%, with dyskinesia in the inferior wall and hypokinesia in the posterior wall, without thrombi, tissue Doppler imaging revealed

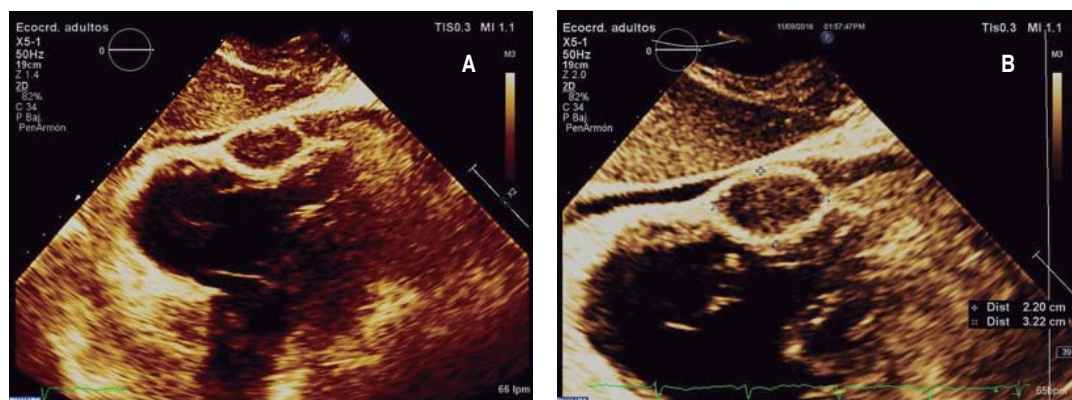
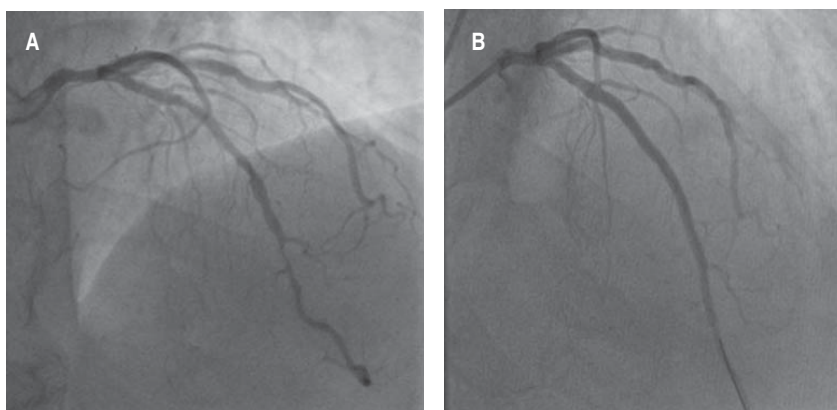
abnormalities of diastolic function ( $E < A$ ); dilated left atrium without thrombi and a left atrial volume index (LAVI) 49 mL/m<sup>2</sup>; mitral valve with mild regurgitation. Right ventricle: dilated, with hypokinesia in the lateral wall, mild depression of the systolic function, non-hypertrophic, tricuspid annular plane systolic excursion (TAPSE) 16 mm/m<sup>2</sup>, S wave of 6 cm/s, a circular echodense image delimited by the pericardial layer (right ventricular myocardium) is observed, the density is similar with the hematic content, locating most of its volume in the lateral and posterior wall, also presenting dehiscence towards the anterior and apical walls, with a total area of 6.5 cm<sup>2</sup> and diameters of 22 x 32 mm, corresponding with an intramyocardial dissecting hematoma of the right ventricle (*Figure 3*). Tricuspid valve structurally normal, with mild regurgitation and systolic pulmonary artery pressure (SPAP) of 25 mmHg. The second echocardiogram showed no progression and no increase in hematoma volume, without no depression of right and left ventricular systolic function and mild changes in the echodensity of the hematoma (*Figure 4*). The third study was performed with contrast echocardiography (CE) using an Epiq 7 device (Philips Medical Systems) and Bracco contrast agent, following intravenous infusion of SonoVue. This showed a thickened area of the lateral and posterior wall with an intramural cavity, non-communicated with the right



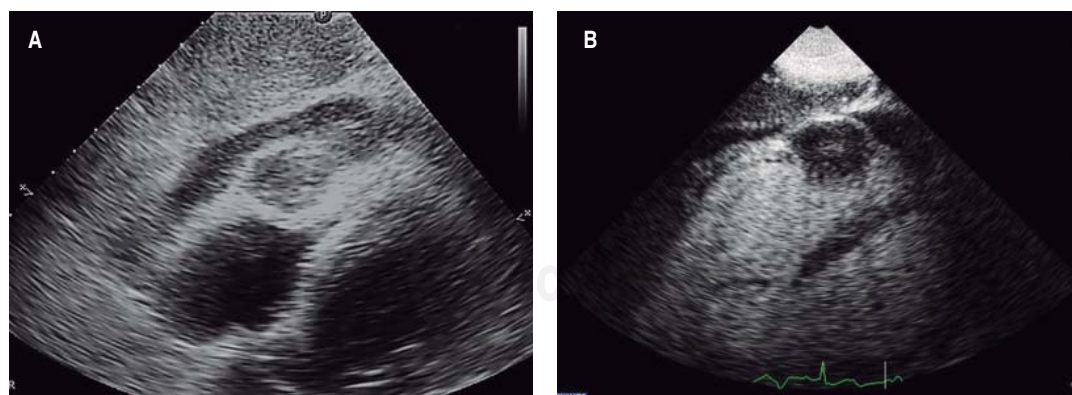
**Figure 1.** Coronary angiography of the right coronary system. **A)** LAO 40° RCA angiogram showing segment 1 acute total occlusion. **B)** After advancing a universal guide wire through the total occlusion, predilatation with a 1.5 x 12 mm balloon was made with recovery of distal flow. We found an angiographic stenosis of 85% in segment 2. **C)** LAO 40° RCA post PCI final angiogram (see text for further details).

**Figure 2.**

Coronary angiography of the left coronary system with an angiographic stenosis of 85% in the middle segment of the anterior descending artery (A). Successful elective PCI. Two Resolute Onyx ZES (Medtronic, Inc., Santa Rosa, CA, USA) were delivered in segment 6 and 7, showing an adequate expansion of the stents without complications (B). See text for further details.



**Figure 3.** Transthoracic 2D-echocardiographic views of intramyocardial dissecting hematoma. A) After 24 hours of symptoms onset, subcostal view showing the myocardial dissection and pericardial effusion. B) Measures: total area of 6.5 cm<sup>2</sup> and diameters of 22 x 32 mm.



**Figure 4.** Transthoracic 2D-echocardiographic views of intramyocardial dissecting hematoma. A) After 48 hours of symptoms onset, subcostal view showing mild changes in the echodensity of the hematoma. B) After contrast injection, of the endocardial border is clearly shown.



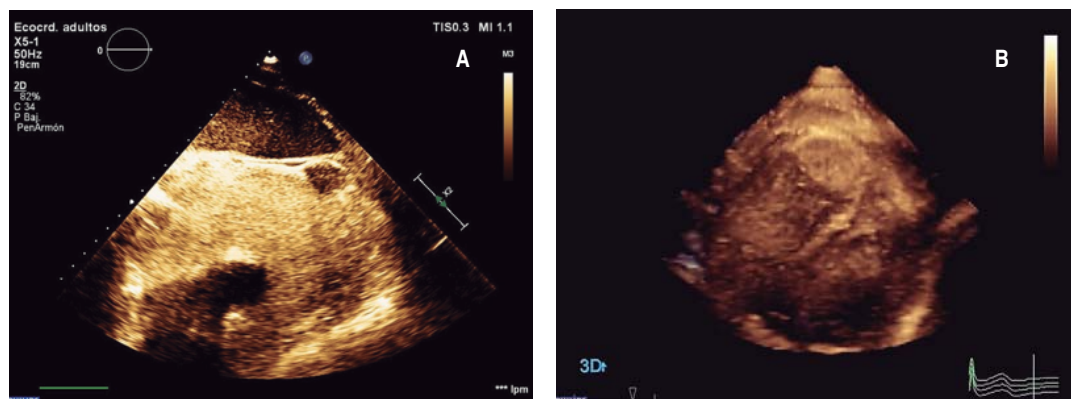
ventricle cavity. Echo-Doppler and CE found no communication with the left ventricle (Figure 5).

After 15 days of the symptoms onset it was performed a cardiac magnetic resonance on Siemens 3T device. HASTE, SSFP or cine sequences are used for the evaluation of right ventricular function, STIR sequence in edema research, double IR with and with fat sat for anatomical evaluation and fat infiltration, as well as T1 GRE post-administration gadolinium to assess myocardial viability. Finding a right ventricle with basal inferior hypokinesia, rest of the walls with conserved contractility, right ventricular

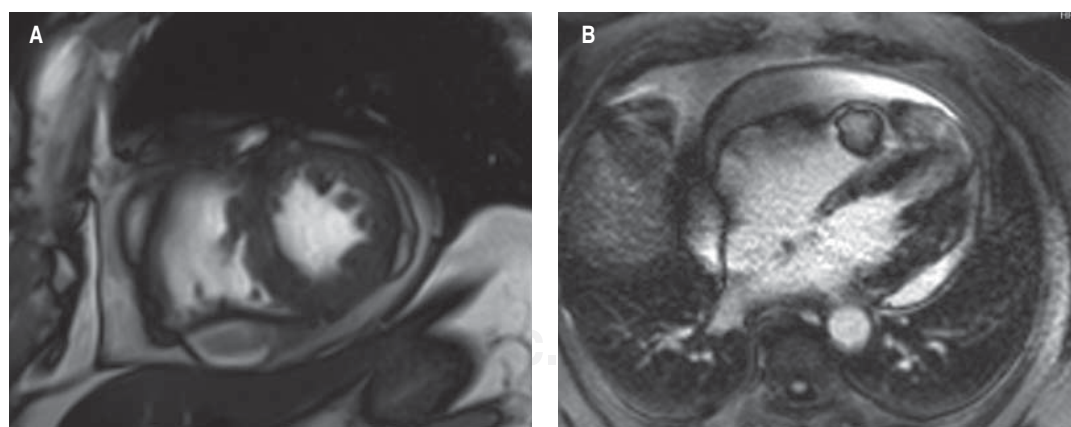
ejection fraction of 34%. It is identified in the inferior wall a mural hematoma wich measures 22 x 23 mm, inferior transmural late reinforcement without viable tissue, as well as transmural late reinforcement of the inferior wall of the right ventricle (Figure 6). Left ventricular systolic function preserved with ejection fraction of 64%.

## DISCUSSION

The IDH is an unusual form of cardiac rupture of the left ventricular wall, most reports are isolated case studies and many may go unnoticed if the



**Figure 5. A)** Transthoracic 2D-echocardiographic views of intramyocardial dissecting hematoma after 96 hours of symptoms onset, after contrast injection showing no chamber communication. **B)** 3D echocardiography showing the total volume and integrity of the endocardial and epicardial layers.



**Figure 6. Magnetic resonance imaging rest perfusion study at midventricular level showed a large hyperintense expansive lesion in the right ventricular inferior wall (A) and a severe subendocardial perfusion defect in the midventricular inferior wall. (B) ECG gated short-T1 inversion-recovery (STIR) four-chamber magnetic resonance imaging showing the integrity of the epicardial and endocardial layers.**

spontaneous evolution is favorable. IDH usually occurs few days after acute infarction, within 7-30 days; however, we cannot be certain about the exact time of the event.

The underlying mechanism is a hemorrhage dissecting among the spiral myocardial fibers, creating a neocavitation limited by the myocardium, echodense structures can be found in the interior and may be related to thrombi in different stages. Because of the potential risk of fatal myocardial rupture, surgical correction was the preferred method of management for the initial cases in literature.<sup>6,7</sup> However, high operative mortality and good clinical course of conservatively treated patients changed the approach nowadays with spontaneous resolution in a very short period of time or spontaneous resolution in several months.<sup>8</sup>

A revision about the evolution of postinfarction dissecting hemorrhage into intramural hematoma suggests a mechanism of a «pin-hole» dissecting hemorrhage which allows the initial communication between the left ventricle cavity and pericardium leading to cardiac tamponade. Subsequently, the small tear favored by the absence of antiplatelet agents, sealed, causes the expansion of blood into the necrotic infarcted myocardium which leads to intramural hematoma formation. Intramycardial dissecting hematoma tends to be more frequent in patients with acute ST segment elevation myocardial infarction, and has been associated with reperfusion injury following infarction, mainly after thrombolysis or percutaneous revascularization,<sup>6</sup> as seen in our patient.

About the electrocardiographic findings, a persistent ST segment elevation is a distinctive feature of this condition, particularly more than 72 hours in all patients,<sup>1-6</sup> a characteristic found in this case.

In terms of diagnosis the half of the cases are diagnosed upon postmortem autopsy. Is usually confirmed by echocardiography and other imaging techniques such as transesophageal echocardiography, cardiac tomography and magnetic resonance.<sup>9</sup> The echocardiographic diagnosis is based on the establishment of a neocavitation entirely contained in the myocardium, and the visualization of a low-speed with contrast injection.<sup>10</sup> A careful examination of the endocardial and epicardial

layers is extremely important, and contrast material is useful to delimit the endocardial borders and distinguish prominent ventricular trabeculations, thrombi, or pseudoaneurysms, whereas color Doppler ultrasound is able to detect the presence of a communication with the endocardial or pericardial cavities.<sup>2</sup> Hematoma consists in a cystic-like, echolucent cavity, variable in size, adjacent to severely hypokinetic or dyskinetic infarct-related segments. Hematoma acoustic characteristics depended on time of evolution. Serial echocardiography is useful in determining its evolving nature, and may guide the outcomes and need of surgical treatment.<sup>7</sup> Nowadays, it can all be better assessed by 3D echocardiography techniques which, as demonstrated in our patient, allow us to visualize the isolation of the myocardium and the confirmation of the IDH.<sup>11</sup>

This case suggests the importance of prolonged and intensive imaging monitoring. The differential diagnosis of IDH includes prominent ventricular trabeculations, intracavitary thrombi, spontaneous hematoma and pseudoaneurysms.<sup>12-14</sup> The importance of cardiac magnetic resonance lies on its ability to identify the severity and hemodynamic consequences following a right ventricular infarction, as well as its ability to differentiate between other associated pathologies, as seen in our case, the diagnostic accuracy can be improved associating more than one image study.

In this case we decided to maintain a conservative treatment, with a strict blood pressure control and standard postinfarction medical treatment, previously there was no evidence about the better treatment to this patients. In 2016 a review by Ying Zhao, et al; showed the total mortality rate was not different between surgical group and medical treatment group (33.3% vs. 54.3%,  $p = 0.08$ ). The mortality was not different compared surgical and medical treatment in the left ventricle free wall and septal groups (20.0% vs. 40.9%,  $p = 0.25$  and 46.2% vs. 60.0%,  $p = 0.60$ ), but the surgery improves survival significantly in right ventricle free wall group (as our patient, 30.0% vs. 87.5%,  $p = 0.015$ ).<sup>15</sup> The principal limitation of this review was the total number of patients in the right ventricle free wall group ( $N = 18$ ) which is still limited to provide a strong

conclusion for the best treatment option that this subgroup should undertake.

As previously commented, the experience in our country, as in our center, in the treatment this complication is non-existent. Being this the first clinical case described in our literature, and the first patient in ambulatory follow-up with medical treatment as the preferred treatment option for a further future analysis.

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### Conflict of interest statement

None declared.

### Ethical approval

No ethical approval is required. All identifiable patient information has been removed from this manuscript.

### Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor of this journal.

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