

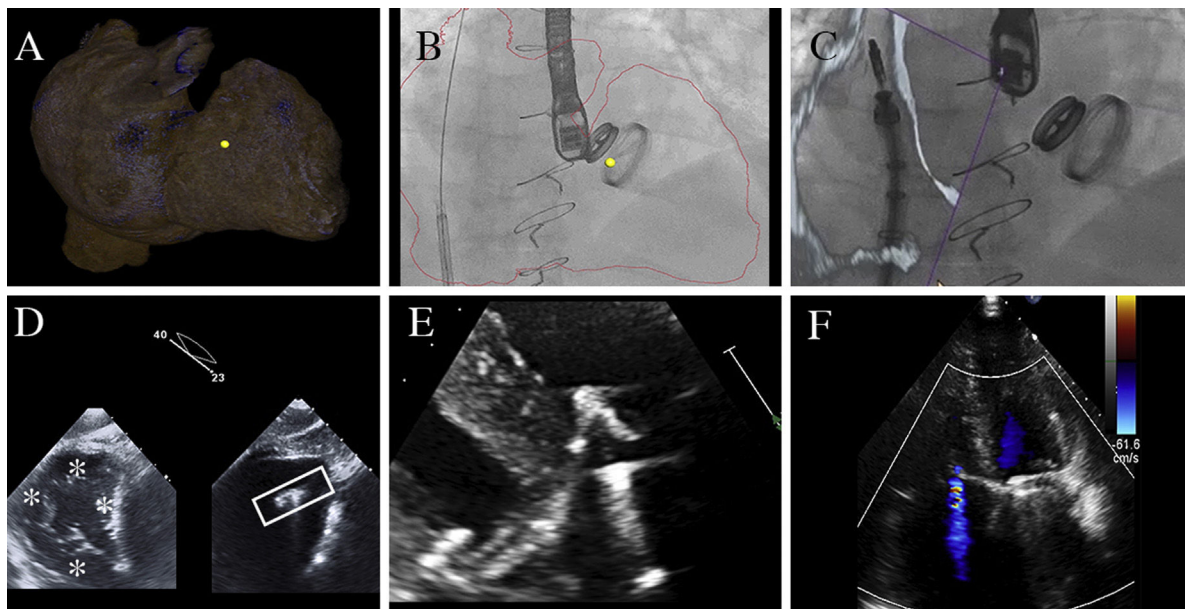


Figure 2. Multimodal imaging during implantation. Fusion of images with computed tomography (A-B); the marker that identified the desired clip position can be seen between the septal and anterior leaflets (yellow dot). C: fusion of images with echocardiography. D: echocardiography to assess coaxiality. E: grasping. F: final result.

vertical inclination, rather than the usual horizontal inclination that shows 2 orthogonal planes, from a transgastric position (Figure 2D). The 40° plane showed the valve scallops (Figure 2D left, asterisks) and the simultaneous plane was parallel, with 23° vertical inclination. This second plane showed the orientation of the clip (Figure 2D right, box). Good coaxiality of the device to the septal-anterior scallops was identified (Figure 2D, orientation of the box), albeit in a posterior position, which was corrected.

Leaflet grasping could not be seen adequately on the transesophageal window. Therefore, we opted to change to transthoracic echocardiography, which revealed that the septal leaflet had not been grasped and there was tricuspid regurgitation between the clip and the leaflet. Grasping was therefore done with transthoracic echocardiographic guidance, and was successful on the third attempt, with good visualization of the grasp (Figure 2E). Next we checked the indirect signs of adequate leaflet grasping, as in a mitral clip procedure, which were favorable. The regurgitation was reduced from severe to mild (Figure 2F) and the patient progressed well. One month later, magnetic resonance imaging showed reduction of right ventricular volume to 77 mL/m², reduction of the regurgitant fraction to 19% and a preserved ejection fraction of 60%. Follow-up at 4 months showed significant clinical improvement with reduction of diuretic use.

Severe tricuspid regurgitation is a condition that is often related to surgically-corrected left-sided valve disease, and has a poor long-term prognosis.¹ Transcatheter treatment can nowadays be studied with a multitude of devices that act at different levels.² Multimodal imaging plays an important role in the continuity of the process. It is key to understanding the functional anatomy of the lesion and personalize the repair, which means optimal patient selection. The vast experience with MitraClip has made it the first tested device, independently of the regurgitation mechanism, with a 13% residual severe regurgitation rate.³ The Spanish experience so far is based on 2 cases⁴; here we present the first to show the usefulness of using the maximum commercially-available technology for patient selection, planning, and follow-up.

In conclusion, multimodal imaging provides precision and safety in the process of patient selection, implant guidance, and subsequent follow-up.

CONFLICTS OF INTEREST

C.-H. Li and D. Arzamendi have received honoraria from Abbott.

SUPPLEMENTARY MATERIAL



Supplementary material associated with this article can be found in the online version available at <https://doi.org/10.1016/j.rec.2018.04.027>.

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Available online 7 July 2018

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<https://doi.org/10.1016/j.rec.2018.04.027>
1885-5857/

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Myocarditis as a Form of Presentation of an Inflammatory Autoimmune Myopathy Associated With Anti-signal Recognition Particle Antibodies



Miocarditis como forma de presentación de una miopatía inflamatoria autoinmunitaria asociada con anticuerpos antipartícula de reconocimiento de señal

To the Editor,

Necrotizing autoimmune myopathy (NAM) is a rare but serious condition that can feature cardiac involvement.¹ We report an unusual and difficult-to-diagnose case of NAM-induced myocarditis that highlights the usefulness of autoimmune studies in patients with idiopathic myocarditis. The disease and the associated cardiac involvement are reviewed.

An 82-year-old woman presented to the emergency department with a 4-week history of dyspnea on moderate exertion, with no symptoms or signs of heart failure. The patient had a history of untreated hypercholesterolemia, with no previous history of diabetes mellitus, hypertension, or heart disease.

Examination on admission revealed elevation of troponin T and creatine kinase (Figure 1 A) and complete left bundle branch block, which had been present on previous electrocardiograms. The blood count, coagulation test, basic biochemistry, and chest X-ray were all normal. The combination of dyspnea with indicators of myocardial injury suggested cardiac involvement, and the patient was kept in hospital for further evaluation.

Transthoracic echocardiography showed normal overall and segmental systolic functions, with no sign of valve disease. A coronary angiogram detected no lesions.

The persistent elevation of myocardial injury markers in the absence of apparent ischemia suggested myocarditis. Despite the persistently elevated muscle enzymes, tests eliminated a possible

infectious or toxic origin. A specific antibody profile showed a positive result for anti-signal recognition particle (anti-SRP) autoantibodies.

The patient's condition thus appeared to be a case of NAM associated with anti-SRP autoantibodies. Thoracic, abdominal, and pelvic computed tomography and tumor marker analysis eliminated cancer as the cause of the autoimmune profile. Neurophysiological analysis and a muscle biopsy were compatible with NAM (Figure 1B and C).

To resolve the co-occurrence of elevated myocardial injury markers with the inconclusive transthoracic echocardiogram, we requested a cardiac magnetic resonance imaging (MRI) scan. The scan showed a nondilated left ventricle with normal systolic function, and late gadolinium enhancement revealed intramyocardial areas in the basal inferolateral segments that were consistent with fibrosis (Figure 1D).

The patient was treated with aspirin, beta-blockers, and corticosteroids (3 bolus doses of 250 mg 6-methylprednisolone followed by prednisone at 0.4 mg/kg/d). Given the seriousness of the patient's condition, the decision was taken to administer 1 g of intravenous rituximab, with a repeat dose after 2 weeks. The patient was maintained on decreasing doses of prednisone, and after 3 months she was free of dyspnea and her myocardial enzymes had normalized.

Autoimmune myopathies (AIM) are a heterogeneous group of muscular disorders characterized by muscle weakness, elevated creatine kinase, and electromyographic alterations; in addition, AIM patient muscle biopsies typically show evidence of inflammatory infiltration, necrosis, or anomalous expression of major histocompatibility complex 1 (MHC1).¹

Inflammatory myopathies have historically been classified according to anatomical and pathological criteria into the following categories: polymyositis, dermatomyositis, inclusion

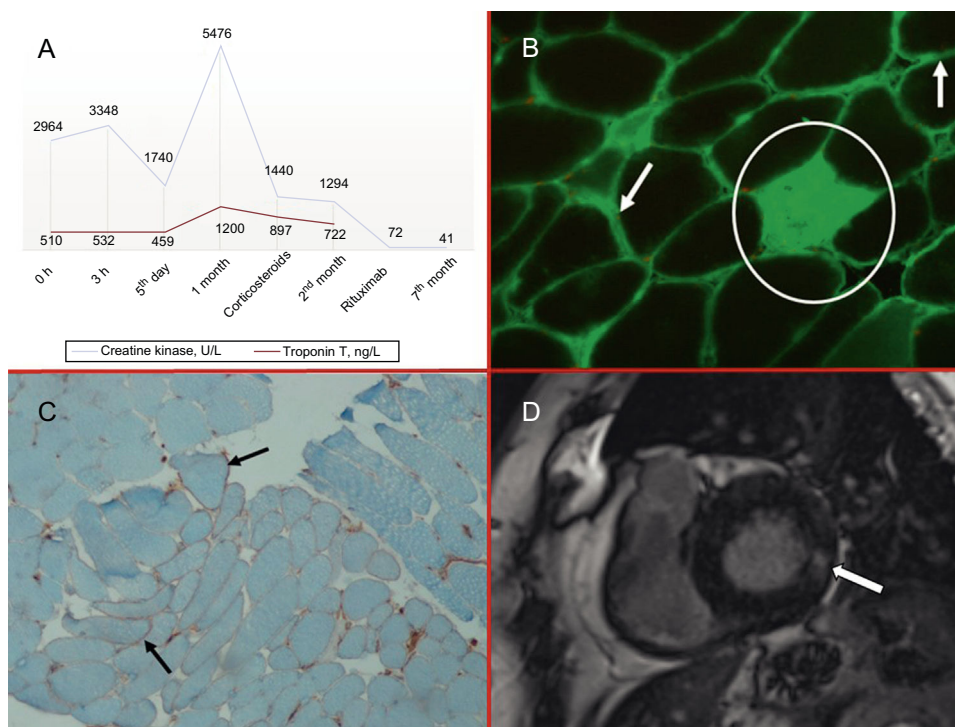


Figure. A, Myocardial injury markers; both enzymes were elevated, especially creatine kinase. B, Muscle biopsy: IgG immunofluorescence ($\times 10$). The arrows indicate the accumulation of muscle fibers in the plasma membrane, and the circle marks a necrotic endomysial fiber. C, Muscle biopsy: MHC1 immunohistochemical staining ($\times 10$). MHC1 overexpression in muscle fiber plasma membranes (arrows). D, Cardiac magnetic resonance imaging: late gadolinium enhancement sequence. The arrow indicates intramyocardial basal inferolateral capture. MHC1, class 1 major histocompatibility antigen.

body myositis, necrotizing myositis, and nonspecific myositis. The recent description of new antibodies and the availability of techniques for their detection have prompted calls for a reclassification under the term AIM, given that almost all forms of the disease feature autoimmune antibodies.²

The case of NAM reported here differs from other reported instances of AIM in showing minimal or absent inflammation in the muscle biopsy, despite the presence of areas of marked necrosis and regeneration.

NAM can occur alone or in conjunction with viral infection, cancer, scleroderma, or statin therapy. The condition is associated with 2 types of antibody: anti-SRP antibodies and antibodies to 3-hydroxy-3-methylglutaryl coenzyme-A reductase²; however, up to a third of patients are seronegative.

Anti-SRP antibodies are highly specific and are associated with more acute forms of disease, higher creatine kinase concentrations, and more pronounced damage to the respiratory and esophageal muscles. Cardiac injury is less frequent.^{1–3}

The first description of cardiac involvement in AIM was provided by Oppenheim in 1899. The prevalence of cardiac injury in AIM patients remains uncertain, ranging from 6% to 75% depending on patient selection and the method used. However, cardiac injury is considered a major clinical manifestation of AIM and one of the principle causes of death.^{3,4}

Clinical expression of cardiac involvement is relatively infrequent (3%–6%), with the most frequent cardiac manifestation being myocarditis (40%). Recurrent myocarditis is believed to cause fibrosis of the cardiac conduction tissue, the vasculature, and the myocardium; the final outcome is heart failure, which is the most frequent cardiovascular cause of death (20%).^{5,6} Nevertheless, the rate of subclinical involvement ranges from 13% to 72% and shows a wide variety of manifestations, including alterations to the electrocardiogram (arrhythmias and conduction and repolarization alterations), the echocardiogram (diastolic dysfunction and takotsubo pattern), and MRI. Cardiac MRI stands out as the best method for detecting the initial myocardial inflammation and myocardial fibrosis in the chronic phase.³

The case reported here demonstrates the advisability of achieving an etiological diagnosis of nonischemic myocardial injury and shows that cardiac involvement can be the only manifestation of AIM. This patient's case also emphasizes the importance of cardiac MRI, autoimmunity studies, and above all

multidisciplinary collaboration for an appropriate clinical approach to these disorders.

CONFLICTS OF INTEREST

C. Morís de la Tassa is a proctor for Medtronic and sits on its advisory board.

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Available online 30 June 2018

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<https://doi.org/10.1016/j.rec.2018.04.024>

1885–5857/

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A 3D Printed Patient-specific Simulator for Percutaneous Coronary Intervention



Simulación impresa en 3D de intervención coronaria percutánea específica para un paciente

To the Editor,

Advances in 3-dimensional (3D) printing over the past 10 years have converted this technology from a specialized industrial process into a widely used tool with applications in many areas, including cardiology.^{1,2}

The 3D printing methodology that has expanded most is fused deposition modeling, and commercially accessible printers today provide 20 μm resolution with practically no geometric limitation.

Previous reports have shown the usefulness of 3D printing for pretreatment trials,³ training,⁴ physiological simulations,⁵ and other applications. To our knowledge, the approach described here is the first specifically designed for training in percutaneous coronary intervention that reproduces structures while preserving

the spatial distribution found in the patient and maintaining high-precision anatomic detail.

The model was built from synchronized cardiac computed tomography images. The ascending aorta and coronary arteries were segmented using the HOROS open-code software program (The Horos Project), resulting in a mesh of the intravascular volume of these structures. Subsequent processing in the Rhinoceros 3D modeling program produced vessels with a defined wall thickness while maintaining the intravascular lumen dimensions. A bespoke stand was then designed to hold the model in the correct anatomical orientation. The model was prepared for printing in the CURA program (Ultimaker inc).

The resulting model precisely replicated the spatial orientation of the intravascular lumens in the aorta and coronary tree. Blood flow was simulated by continuous pumping of saline solution through the model in an open hydraulic circuit.

A 7-Fr, 3.75 extra-backup curve guide catheter (Medtronic) was introduced into the left coronary artery through a 7-Fr introducer