



Mitral valve repair for functional regurgitation caused by Chagas' disease

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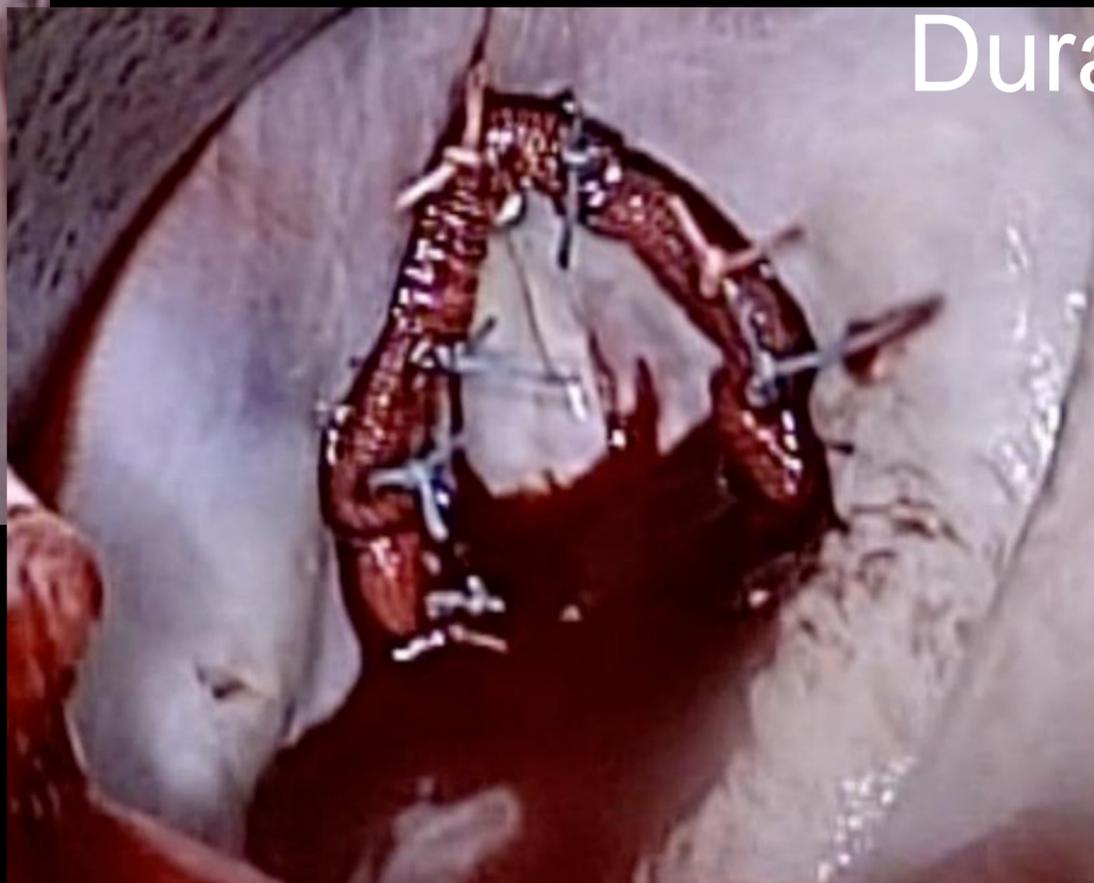
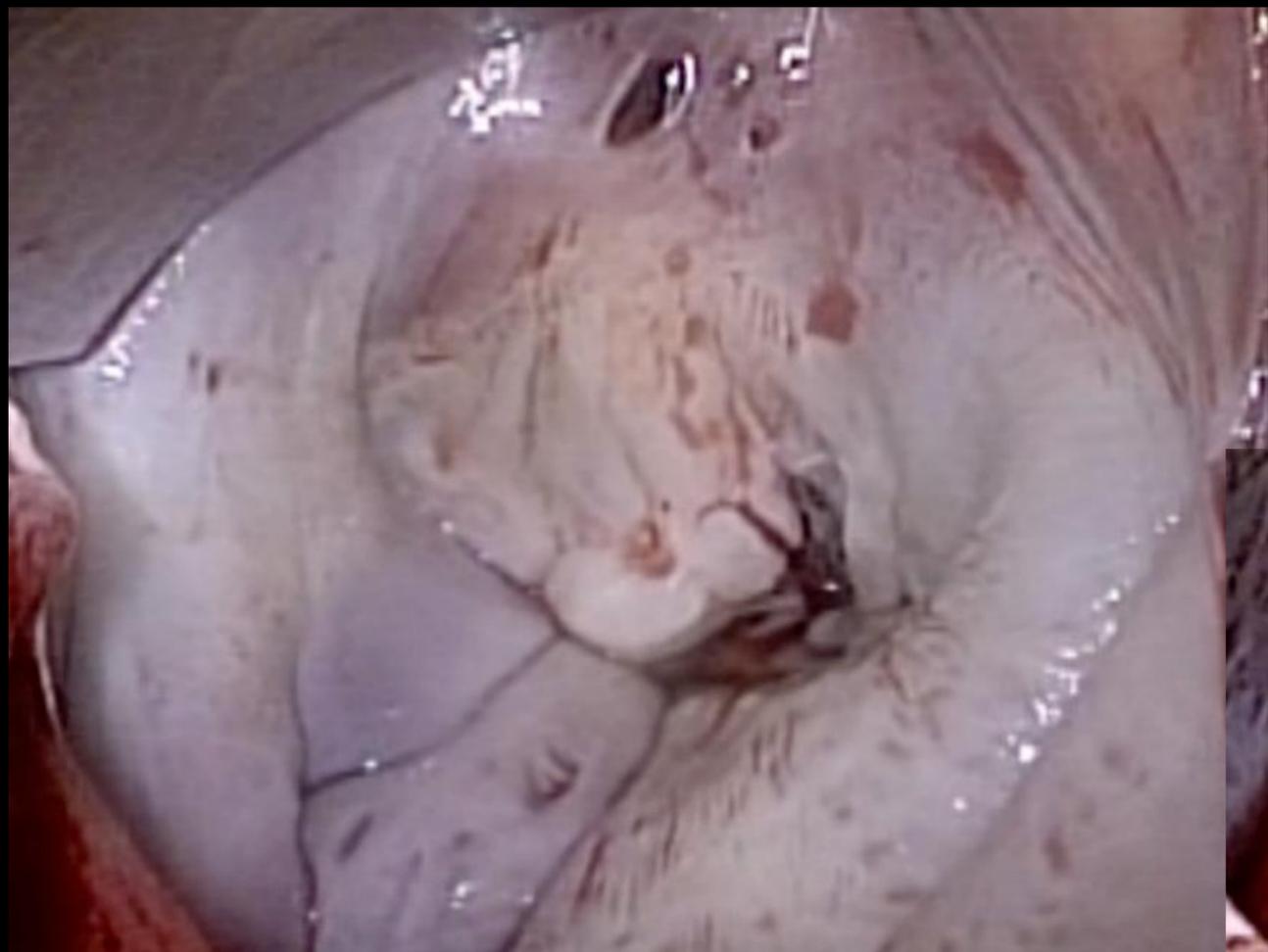
case summary

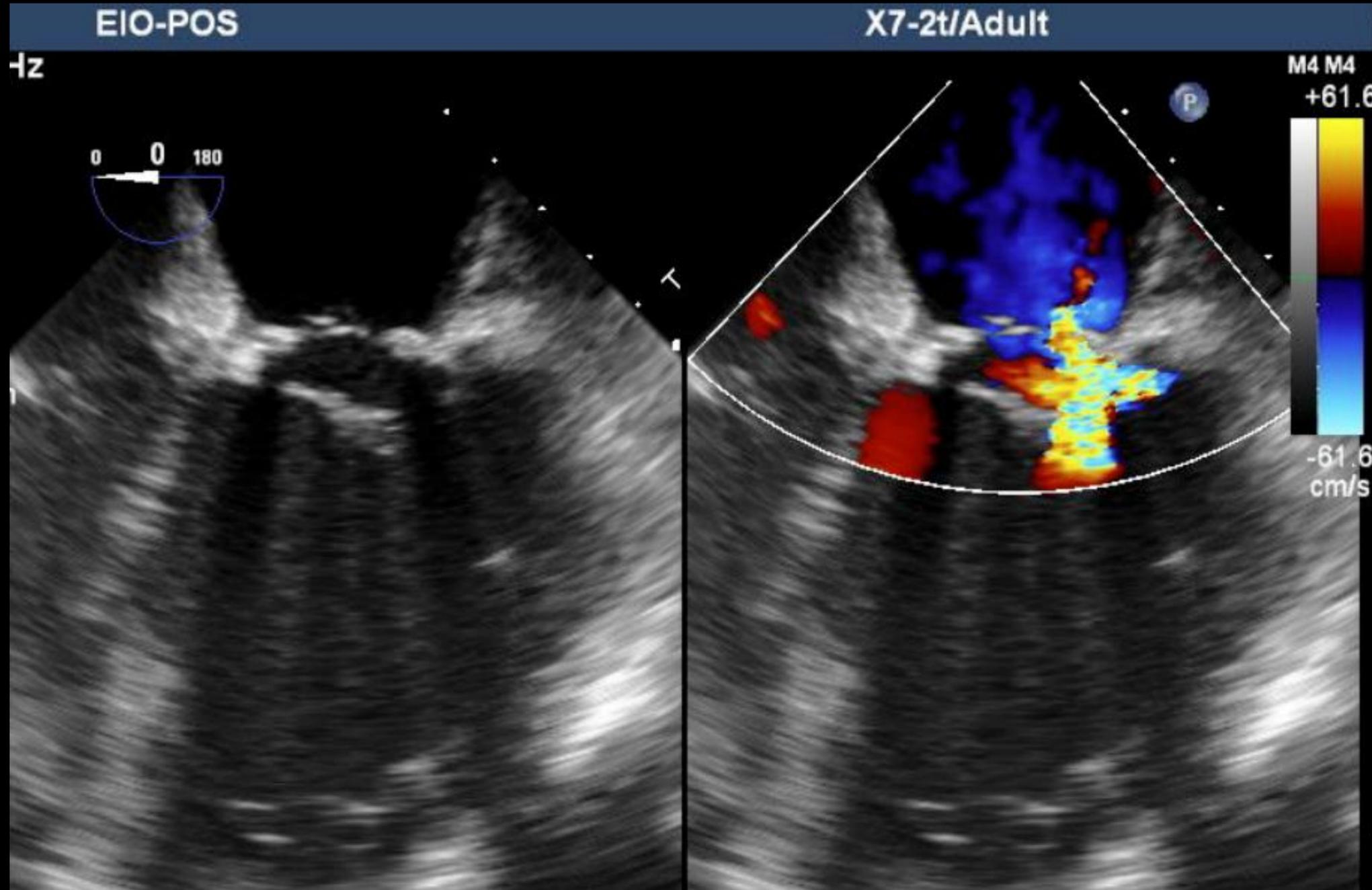
- Female, 60 yo
- Diagnosed with Chagas' disease 10 years
- Progressive symptoms of heart failure, presented in NYHA III
- Severe mitral regurgitation (4 +), LVEF 30 %

Surgery

Minimally
invasive mitral
valve repair

- Ring annuloplasty - Duran Ancore®





post op echo
minimal mitral
regurgitation

Follow up

- Multiple episodes of ventricular tachycardia (common in Chagas' patients)
- Received Implantable Cardioverter-defibrillator and discharged from hospital 10th day post-op

1 month



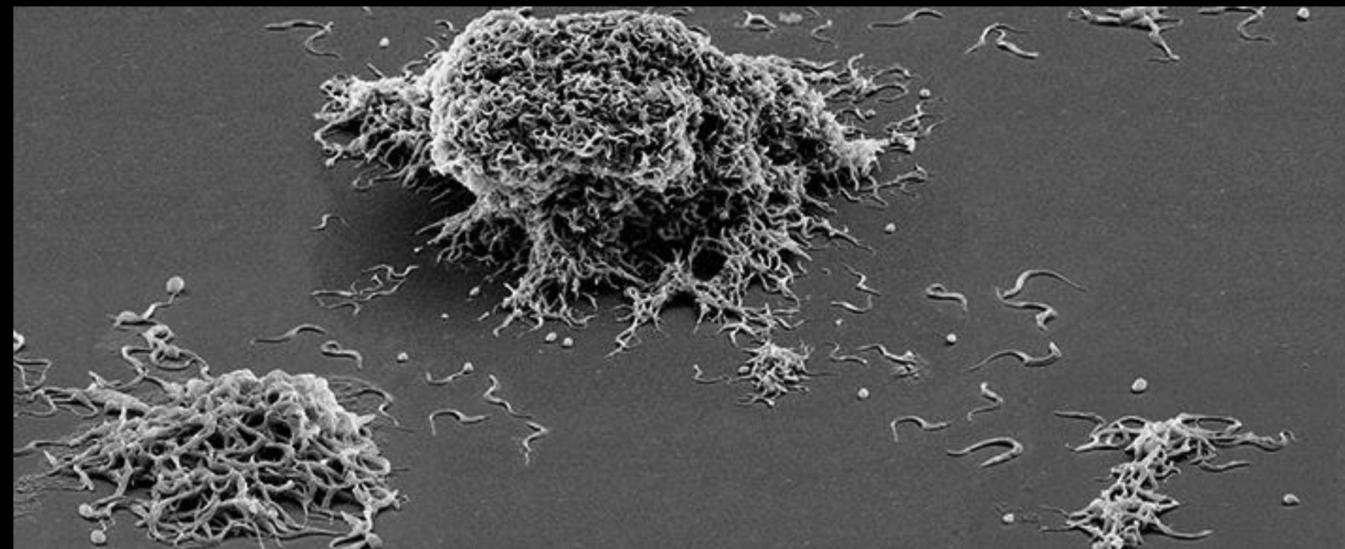
6 months

- NYHA I
- LVEF 35 %
- minimal mitral regurgitation
- no VT episodes
- refuses new TEE

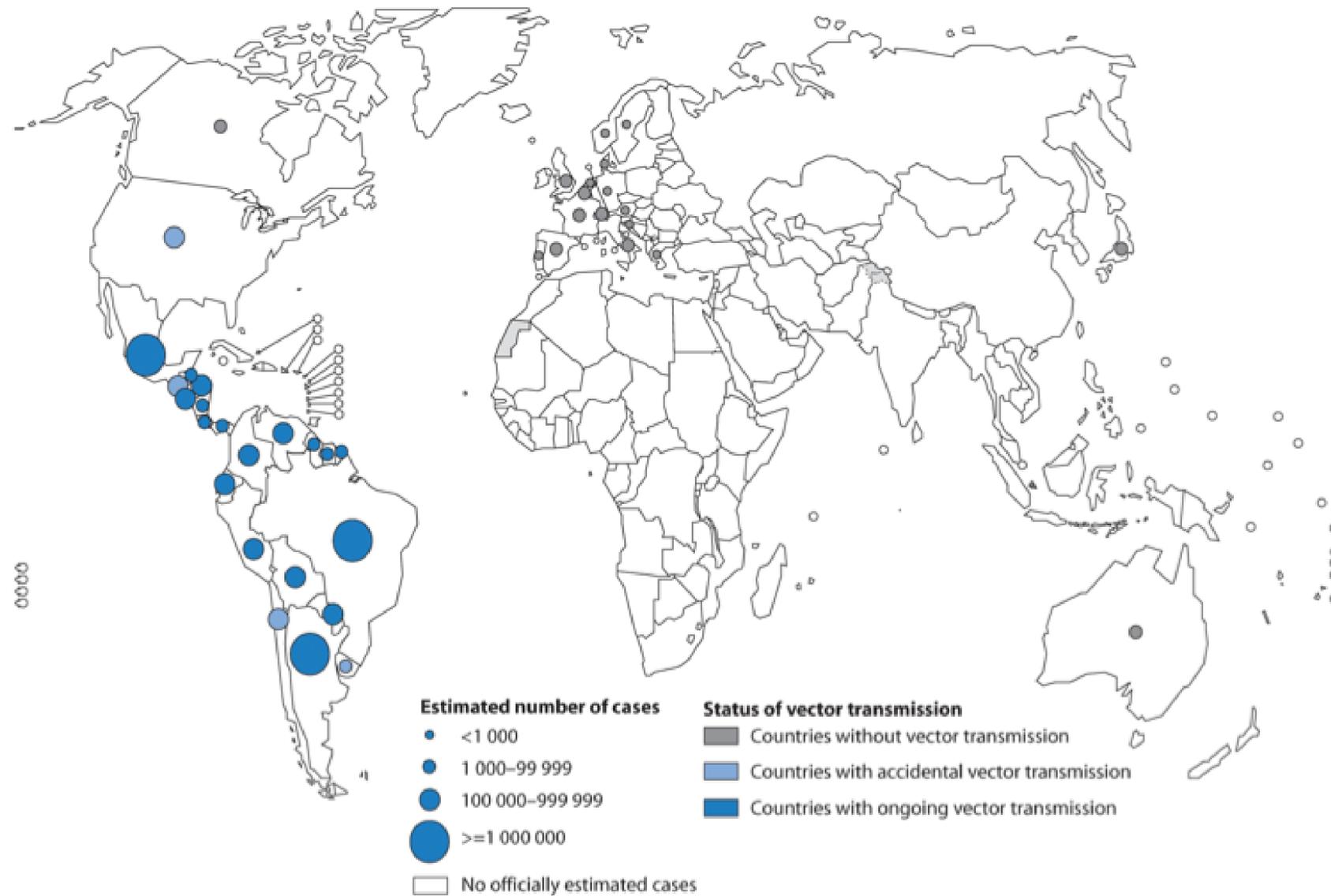


Foto: Arquivo/Fiocruz - FIOCRUZ MULTIMAGENS

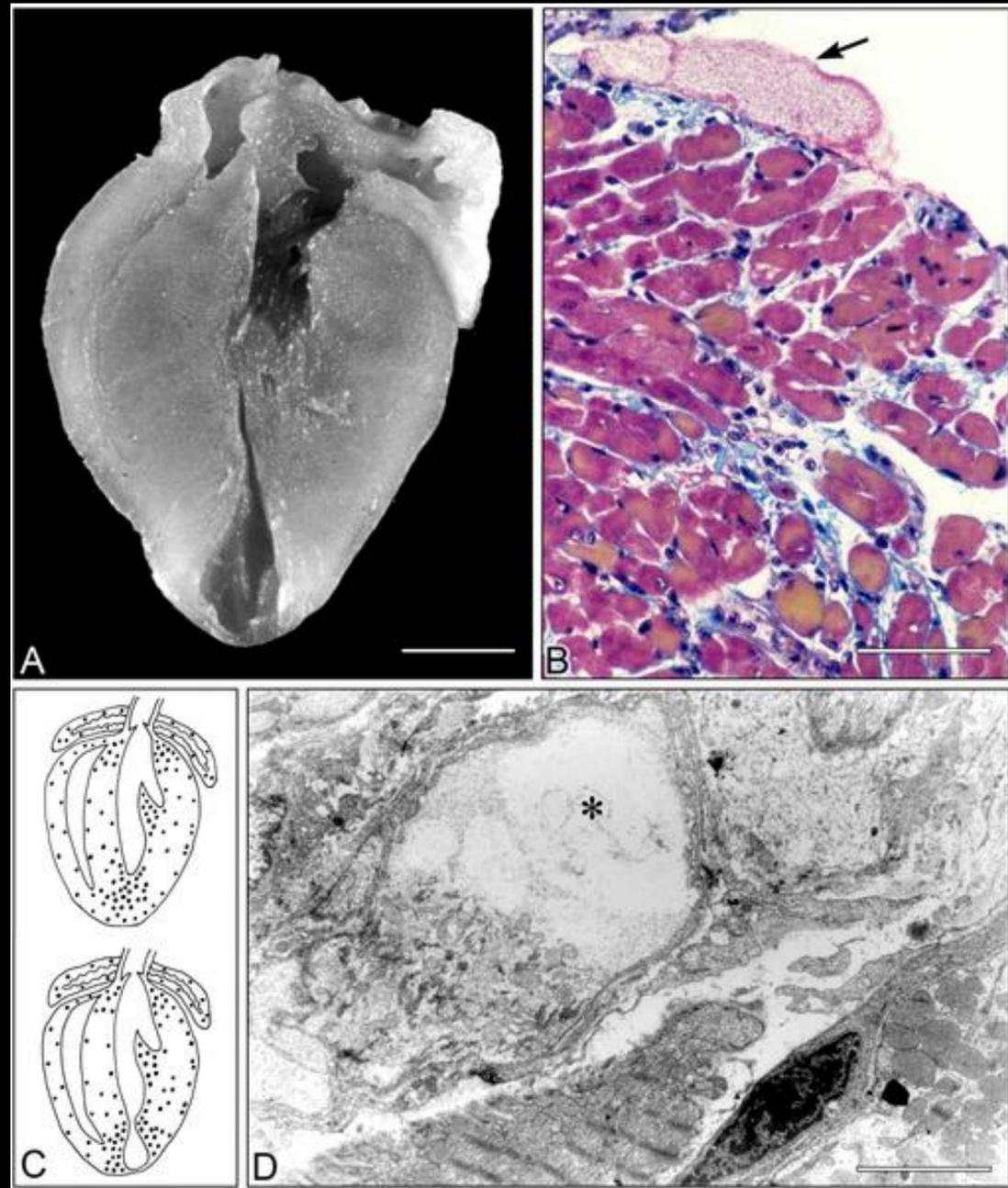
- Chagas' disease, or American trypanosomiasis, is a potentially life-threatening illness caused by the protozoan parasite, *Trypanosoma cruzi* (*T. cruzi*).
- discovered by Carlos Chagas in 1909, it is transmitted through infected triatomine insects - they bite exposed skin area of sleeping people and defecate around the lesion
- other forms of transmission are blood transfusion, organ donation or during childbirth



Distribution of cases of *Trypanosoma cruzi* infection, based on official estimates and status of vector transmission, worldwide, 2006–2009

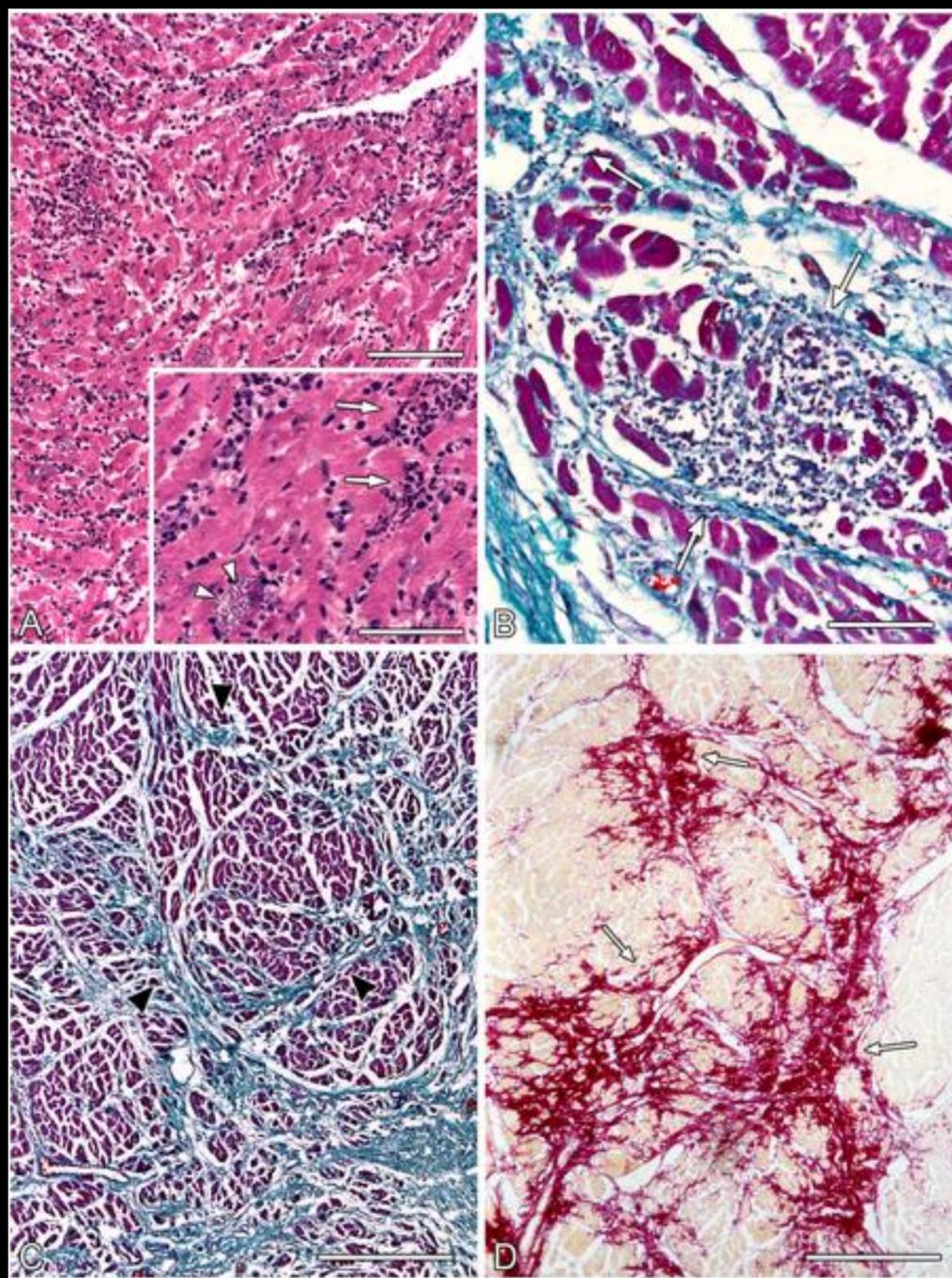


once restricted to endemic areas (blue circles), it has spread due to population migration (grey dots)



Benznidazole or nifurtimox are effective drugs for using during the acute phase. Once the infection has become chronic, they are less effective and congestive heart failure develops in 30 % of those patients.

Study in mice chronically infected with *T. cruzi* demonstrating involvement of microcirculation. (A) Enlarged heart of a mouse infected with *T. cruzi* 100 days post-infection showing marked thinning of the apex of the left ventricle (apical aneurism). Bar = 2 mm. (B) Myocardium of an infected mouse stained by the Carstairs method for demonstration of platelets. An occlusive platelet thrombus is seen in a small epicardial vessel (arrowhead). Bar = 50 mm. Mononuclear cell infiltration, interstitial edema and fibrosis, and foci of myocytolytic necrosis. (C) Schematic representation of coronal sections through mice hearts infected with *T. cruzi* 100 days post-infection without (upper panel) and with (lower panel) apical aneurism, showing the extent of foci of myocytolytic necrosis. These areas are scattered throughout the ventricular and atrial myocardium, but are more numerous in the subendocardial and subepicardial regions in the apex, papillary muscles, and base of the ventricles. (D) Electron micrograph showing complete dissolution of myofibrils within a myofiber (*) of an infected mouse with characteristic myocytolysis or myocytolytic necrosis.



possible pathological mechanisms are

- direct tissue destruction
- autoimmune mechanisms
- autonomic abnormalities
- microvascular heart lesion

heart failure is cause of death in 58% of patients

arrhythmias/sudden death in 36,5%

Micropathology of Chagas heart disease. (A) Acute myocarditis with foci of myocytolytic necrosis and degeneration are seen with an intense inflammatory infiltrate around ruptured pseudocysts of parasite (arrows, in the inset). Intact intramyocyte parasite nest without inflammatory response (arrow heads, in the inset). Hematoxylin and eosin staining. Bar = 100 mm; inset bar = 50 mm. (B) Chronic fibrosing myocarditis. Foci of myocytolytic necrosis associated with mononuclear inflammatory infiltrate and incipient interstitial fibrosis appearing in light blue (arrows). Gomori trichrome staining. Bar = 100 mm. (C) Chronic fibrosing myocarditis. Predominantly perimysial interstitial fibrosis extending to the endomysium (arrow heads) appearing in light blue associated with mononuclear inflammatory infiltrate. Gomori trichrome staining. Bar = 500 mm. (D) Chronic fibrosing myocarditis. Interstitial and diffuse fibrosis manifested by increased amount of thick collagen fibers surrounding muscle fiber bundles (perimysial matrix) and around intramural coronary vessels, combined with a less pronounced increase in the endomysial matrix. Picosirius red staining.