Restrictive Cardiomyopathy due to Myocardial Cysticercosis

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There is no description of cysticercosis affecting heart function. In the present report, the authors describe the case of a 46-year-old woman with cardiac cysticercosis and heart failure, presenting with echocardiographic findings suggestive of restrictive cardiomyopathy and myocardial microcalcifications suggestive of cardiac infiltration by the disease.

Cysticercosis is a disease caused by an infestation of the larval form of *Taenia solium*. In human cysticercosis, the person is an accidental intermediate host. The infection is caused by the ingestion of eggs containing viable oncospheres that invade the intestine, enter the vasculatory system and lodge in tissues. The global prevalence of cysticercosis caused by *cisticercus cellulosae* has been estimated at three hundred thousand people. Although any organ or tissue can house the cysts, the most commonly affected areas are the brain, skeletal musculature and subcutaneous tissue. While the topic of neurocysticercosis has been documented in numerous reports, cardiac cysticercosis and particularly myocardial cysticercosis are rare and consequently few studies have been conducted.

**CASE REPORT**

The patient is a 46 year old female born in the city of Jacobina – BA, with a history of seizures since 1970, believed to be caused from epilepsy, using phenobarbital on a regular basis. In 1996, a simple x-ray of soft tissues revealed microcalcifications [Fig. 1](#). Examination of the cerebrospinal fluid revealed a positive hemagglutination reaction of 1:4 for cysticercosis and a positive ELISA reaction for the detection of the antibody *Cysticercus cellulosae*. A CT scan of the brain revealed cerebral and right cerebellar calcifications which are also compatible with neurocysticercosis [Fig. 2](#). As such, the patient was diagnosed with neurocysticercosis.

In 1998 she began to present dyspnea while performing out-of-the-ordinary exertions and palpitations. An electrocardiogram revealed a sinus rhythm, strain on the left ventricle (LV), secondary alterations of repolarization and supraventricular ectopic activity.

In 2001, the dyspnea progressed to ordinary exertions, fatigue, edema in the lower limbs, ascites and hepatomegaly. The Doppler echocardiogram revealed normal LV systolic function and restrictive diastolic impairment. The Doppler did not reveal any mitral valve respiratory flow variations and there was no paradoxal movement of the interventricular septum. The unusual...
aspect of the diffusion of myocardial microcalcifications in both ventricles called attention, in view of the aspect of other sites attacked by the disease (fig. 3). Additionally, a concentric increase in the LV wall thickness was observed and slight dilation of the left atrium.

A hemodynamic study and angiocardiography showed normal coronaries. The diagnosis was therefore confirmed as diastolic heart failure as a result of restrictive cardiomyopathy and due to the pattern of calcification is probably secondary to myocardial cysticercosis.

**DISCUSSION**

The clinical picture presented by the patient starting in 2001 indicated a syndromic diagnosis of heart failure. The Doppler echocardiogram revealed alterations compatible with a restrictive cardiomyopathy and presence of microcalcifications suggestive of cysticercosis. In this case a myocardial biopsy was not performed to confirm the diagnosis, however, the lesions found on the echocardiogram are very suggestive of cysticercosis since they correspond to the calcification pattern in other areas.

There are other possible causes of the diastolic impairment that could be suggested, such as: hypertensive heart disease, constrictive pericarditis, endomyocardial fibrosis and Loeffler’s idiopathic hypereosinophilic syndrome.

Hypertensive heart disease is unlikely since the patient did not present a prior history of systemic hypertension. In reference to constrictive pericarditis, the Doppler echocardiogram did not reveal any indicative signs of this disease, such as mitral valve respiratory flow variations, paradoxal movement of the interventricular septum or morphological alterations of the pericardium.

The increased thickness of the LV walls and the absence of signs of endocardial fibrosis on the echocardiogram, such as abnormal echoes in the left ventricle apical region, inflow pathway or apex of the right ventricle did not substantiate a diagnosis of endomyocardial fibrosis or Loeffler’s idiopathic hypereosinophilic syndrome.

The distribution of topographic locations of cysticercosis varies among different papers on the subject. Nevertheless, there is a relative consensus that the encephalic and muscular skeletal forms have a greater prevalence.

Reports in medical literature are controversial regarding the prevalence of cardiac cysticercosis. Gobbi et al. and Lino et al. discovered cysticerci in the hearts of 26% and 22% of cysticercosis autopsies, respectively, while Vianna et al. discovered them in only 8%.

The cardiac variety can cause functional diastolic alteration which is a minor symptom in the majority of cases.

Most cases related in medical literature of myocardial cysticercosis consist of anatomical pathological studies of autopsy series and there are few data about the functional importance of these alterations and clinical evolution of these patients’ condition.
The patient’s condition progressed with significant clinical improvement after specific treatment for restrictive cardiomyopathy and was referred to a cardiologist for outpatient treatment.

The relevance of this clinical case is the significant clinical manifestation of cardiac disease in the case of myocardial cysticercosis; there are no documented cases in medical literature of restrictive cardiomyopathy associated with this disease. Additionally, medical literature lacks data about echocardiograms for myocardial cysticercosis and this case illustrates these alterations.

**Conclusion**

Cardiac cysticercosis presents an unusual echocardiograph aspect and can provoke a significant manifestation of diastolic heart failure.

**References**