Surgical treatment of hydatid cyst infiltrating into myocardium and causing mitral valve regurgitation

Miyokarda infiltre olan ve mitral kapak yetmezliğine neden olan kist hidatığın cerrahi tedavisi

ABSTRACT

Hydatid cyst is a parasitic disease caused by Echinococcus granulosus. It is endemic in Asia, Africa, South America, and the Mediterranean region. Cardiac involvement is rare. Surgery is the definitive treatment of cardiac hydatid cysts; however, to avoid recurrence after surgery, medical therapy should be also continued. The left ventricular free wall is the most common location, followed by the right ventricle and interventricular septum. Involvement of mitral valve is extremely rare. Herein, we present a case of hydatid cyst with myocardial involvement leading to severe mitral valve regurgitation which was successfully treated with surgery.

Keywords: Heart, hydatid cyst, mitral valve, myocardium.

Herein, we present a case of HC with myocardial involvement leading to severe mitral regurgitation (MR) which was successfully treated with surgery.

CASE REPORT

A 63-year-old female who suffered from dyspnea and palpitation was admitted to our hospital with exacerbated symptoms. She underwent femoral artery embolectomy one year ago. Transthoracic echocardiography (TTE) showed severe MR (regurgitation volume [RV]: 62 mL, regurgitation fraction [RF]: 50%, regurgitation area: 13.2 cm²) and moderate tricuspid regurgitation (RV: 43 mL, RF: 38%, tricuspid annulus: 41 mm, tricuspid annular...
plane systolic excursion/TAPSE: 1.9 cm). Calcific degeneration was present on the leaflets and posterior leaflet’s movement was restricted by a calcified mass of 2×2 cm size. Transesophageal echocardiography revealed two masses (1.7×1.4 cm on the posterior, 2×1.9 cm on the anterior leaflets) and patent foramen ovale (PFO). Hyperintense mass (20×17 mm) at the intersection of the LV and mitral annulus adjacent to the posterior mitral leaflet (PML), hyperintense mass (19×14 mm) at the level of anterior mitral leaflet (AML), and another T₁A isointense, T₂A hypointense intramyocardial mass at the junction of the leaflets were detected in cardiac magnetic resonance imaging (MRI) (Figure 1). There was no infiltration into the tricuspid valve. Masses demonstrated peripheral opacification after contrast injection and were interpreted as HC.

Although indirect hemagglutination test was positive, other blood tests including serology and complete blood count were normal. Abdominal ultrasonography revealed no other organ system involvement. A written informed consent was obtained from the patient. Preoperative coronary angiography showed significant stenosis on the left anterior descending artery (LAD). The patient was operated under cardiopulmonary bypass (CPB). Three yellowish intramyocardial cysts adjacent to the PML were dissected, caseification necrosis was drained and flushed with saline. Calcified germinative membranes were removed. Specimens were collected for pathological and microbiological examination. Layers of the mitral valve were abutted, the remaining cyst walls were occluded with pledgeted sutures (Figure 2), and calcified mitral valve was replaced with a 27-mm Sorin Pericarbon bioprosthesis (Sorin Group, Saluggia, Italy). The PFO was closed and tricuspid annuloplasty was performed with a 33-mm Medtronic Duran AnCore ring (Medtronic, Minneapolis, Minnesota, USA). Left internal thoracic artery-LAD bypass was performed. Pathological examination showed hemosiderin containing macrophages, destructed erythrocytes, and *Echinococcus granulosus* scolex and hooklet fragments. Postoperative first month TTE revealed trace paravalvular MR (E/A:1.3/1.1, max/mean gradient: 7/3 mmHg) and trace central tricuspid regurgitation (max/mean gradient: 5/2 mmHg). Albendazol therapy at a dose of 400 mg q.i.d. was continued for six months.

**DISCUSSION**

The prevalence of cardiac involvement in all HC patients is 0.5 to 2% and, unlike our case, it is usually secondary involvement.[2,3] Cyst embryos which enter into the intestine are transported to the liver and, then, to the right atrium by venous circulation.[1] In our case, PFO was probably responsible for the left heart cysts. After the larvae reach the myocardium through coronary arteries, cysts are formed in about one to five years and usually become symptomatic after adulthood.[4]

Electrocardiography and chest X-ray are not usually diagnostic.[4] Casoni’s intradermal test, Weinberg
reaction, and eosinophilia in the peripheral blood smear have a high false negative rate with a low diagnostic value. Enzyme-linked immunosorbent assay (ELISA) and indirect hemagglutination tests are highly sensitive and specific for HC. Transthoracic echocardiography is an effective method for screening and diagnosis of HC and it also helps to localize the cyst. Computed tomography and MRI are essential for definitive diagnosis and planning for surgery.

The majority of the patients are asymptomatic. Symptomatic patients as our case, suffer from dyspnea, palpitations, and angina pectoris. Pulmonary or systemic embolism, anaphylactic shock and sudden cardiac death are less common complications of cardiac HC. In our case, medical history revealed femoral embolectomy, possibly caused by HC. Cysts may also expand toward the conduction system and provoke heart blocks or arrhythmias. Septal cysts may cause LV or RV outflow tract obstruction. Stenosis or regurgitation of the valves may occur, as in our case.

Surgery is the optimal treatment for recovery and to avoid postoperative complications even in asymptomatic patients. We performed surgery on CPB, as the cyst was invading the myocardium and mitral annulus. Placing an additional filter to the venous side of the circuit is advised to block the passage of hydatid cyst fragments to the pump. Manipulation of the heart must be avoided before cross-clamping. Pulmonary artery can be clamped to elude embolism during surgeries for right heart cysts. To reduce the contamination risk, puncturing the cyst to aspirate the cyst content and irrigating the fibrous space with hypertonic solution are recommended as in our case. There are, however, some concerns about how to manage the potential space left after the excision of cyst membrane. In our case, we stabilized the annulus with pledgeted sutures that we used for valve implantation to close the space after drainage. Leaflet tissue adjacent to the cysts was widely calcified and deformed and, therefore, we preferred replacing the valve. The space left was not large and we did not need to reconstruct the annulus with a synthetic or pericardial patch. In cases with adequate leaflet tissue, repair or reconstruction would be a better option. Although recurrence after surgery is rare, the patients should be followed with echocardiography and serological markers for few years after surgery. A six-month albendazole therapy is recommended to avoid recurrence.

In conclusion, cardiac hydatid cyst is a rare disease and extension to the mitral valve is even more infrequent. In patients with a history or suspicion of hydatid cysts, cardiac examination should also be performed, particularly in patients with symptoms relating to cardiac involvement. Echocardiography and magnetic resonance imaging are effective and reliable imaging methods in this group of patients both for the diagnosis and surgical decision. A meticulous surgical intervention is essential in these patients to avoid contamination and possible myocardial damage during cyst excision.

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**REFERENCES**