Surgical resection of circumferential epicardial adipose tissue hypertrophy: Case report and systematic review of the literature

Hassiba Smail, MD,[a] Andrei Baciu, MD,[a] Jean-Nicolas Dacher, MD, PhD,[b] and Pierre-Yves Litzler, MD, PhD,[a] Rouen, France

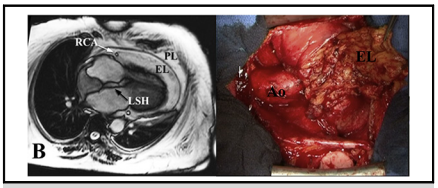
From the Departments of [a]Thoracic and Cardiovascular Surgery, and [b]Radiology, Rouen University Hospital Charles Nicolle, Rouen, France.

Disclosures: Authors have nothing to disclose with regard to commercial support.
Received for publication June 19, 2015; revisions received Aug 5, 2015; accepted for publication Aug 21, 2015; available ahead of print Sept 20, 2015.

Address for reprints: Hassiba Smail, MD, Department of Thoracic and Cardiovascular Surgery, Rouen University Hospital Charles Nicolle, 1, rue de Germont, 76000 Rouen, France (E-mail: Hassibasmail@yahoo.fr).

J Thorac Cardiovasc Surg 2016;151:e27-30
0022-5223/$36.00

http://dx.doi.org/10.1016/j.jtcvs.2015.08.083



Surgical resection of the epicardial lipomatous. *RCA*, Right coronary artery; *PL*, pericardial lipomatous; *EL*, epicardial lipomatous; *LSH*, lipomatous septal hypertrophy; *Ao*, aorta.

Central Message

The diagnosis of CLH can be misinterpreted. We performed a surgical resection with an excellent result.

See Editorial Commentary page e31.

JTCS VIDEO CLIP Video clip is available online.

CLINICAL SUMMARY

A 53-year-old woman with a medical history of metabolic syndrome and long-term nasal corticosteroid treatment was admitted for suspicion of acute coronary syndrome. The patient described recurrent chest pain and severe dyspnea. On examination, she presented a muffled heart sound with left pulmonary dullness and fever (38.5°C). Her body mass index was 31.6 kg/m^2 (weight: 75 kg, height: 154 cm) with a loss of 16 kg during the last 6 months. The electrocardiogram was normal, and chest x-ray revealed a left pleural effusion. Biological data showed an inflammatory profile and a normal troponin. Transthoracic echocardiography (TTE) showed images of moderate pericardial effusion (20 mm). Coronary angiography results were normal. Aside from a minimal elevation of the pulmonary artery pressure (systolic/diastolic mean) 36/25-31 mm Hg, right heart catheterization values were normal: mean pulmonary right atrial pressure was 9 mm Hg, right ventricular pressure (systolic/protodiastolic-telediastolic) was 39/4-12 mm Hg, pulmonary arteriolar vascular resistance was 1.8 Wood units, and systemic vascular resistance was 13.8 Wood units. On chest computed tomography (CT) scan, pericardial fat deposits and pericardial effusion of 2 cm were shown. Cardiovascular magnetic resonance (CMR) was performed, revealing a thick layer of circumferential epicardial fat pad measuring up to 30 mm and hypertrophy of the atrial septum (25 mm) with a typical dumbbell-shaped appearance (Figure 1, *A* and *B*). CMR showed severe muscle atrophy, but it did not confirm pericardial effusion. Functional data showed that epicardial adipose tissue (EAT) affected diastolic cardiac function. Thus, a diagnosis of EAT hypertrophy with concomitant lipomatous hypertrophy of the interatrial septum (LHIS) was made, both concurring with cardiac lipomatous hypertrophy (CLH).

Because of the occurrence of a tamponade leading to a severe dyspnea and recurrent chest pain, we decided to perform a decompressive pericardiectomy and a surgical excision of the excess epicardial fat.

After a median sternotomy, the pericardium was incised and the heart swelled out. There was a circumferential fatty tissue deposit around the heart that concealed the contraction. We created a cleavage plane between the epicardial fat and the myocardium by alternatively using a low level of electrocauterization and a soft swab stick to avoid any coronary lesions (Video 1). We resected the epicardial fat carefully on the right atrium, right ventricle, aorta, and pulmonary artery (Video 1). On the left ventricle, the resection was limited to the front side excluding the lateral and posterior wall. The whole procedure was performed without cardiopulmonary bypass considering the high risk of bleeding.

After surgery, the patient was extubated at 6 hours postoperatively, with an uneventful stay in the intensive care unit of 3 days. The symptoms quickly disappeared during the early postoperative period, and the patient was discharged at 7 days postoperatively.

TTE at discharge showed an ejection fraction of 62%; histology confirmed benign EAT, and pleural fluid was nonspecific. One month after surgery, CMR showed a significant decrease of the epicardial fat volume (15 mm) and correct diastolic function. The patient stopped the

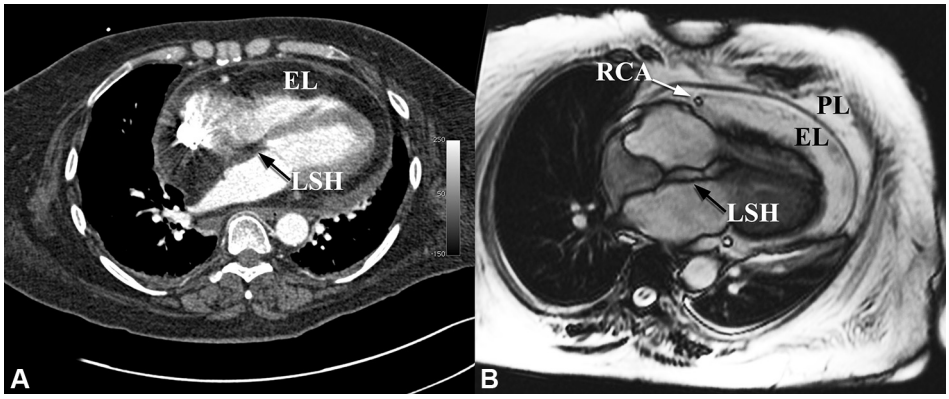


FIGURE 1. Preoperative chest CT scan (A) and CMR images (B). Images showing extensive epicardial lipomatous hypertrophy and LHIS. *EL*, Epicardial lipomatous; *LSH*, lipomatous septal hypertrophy; *RCA*, right coronary artery; *PL*, pericardial lipomatous.

corticosteroid therapy, and better control of the metabolic syndrome was initiated.

Three years after surgery, the patient remains asymptomatic from chest pain and dyspnea. CMR showed stable epicardial fat volume (15 mm) and stable morphology of the LHIS (Figure 2).

SYSTEMATIC REVIEW METHOD

Search Strategy

We searched 3 databases (PubMed, Medline, and Embase) in an attempt to locate all existing English-language case reports on the surgical management of epicardial fat hypertrophy with acute presentation of diastolic dysfunction. Search terms were ''epicardial lipomatous hypertrophy'' or ''epicardial adipose tissue hypertrophy'' or ''cardiac lipomatous hypertrophy.'' These terms were combined with ''diastolic dysfunction,'' ''tamponade,'' and ''surgical resection.''

Inclusion and Exclusion Criteria

Only original case reports of surgical management of EAT hypertrophy with acute presentation of diastolic dysfunction or tamponade were included in this review. We excluded all literature reviews, clinical trial articles, and all analyses of LHIS. Two authors independently screened the titles and abstracts of all articles found in the initial search.

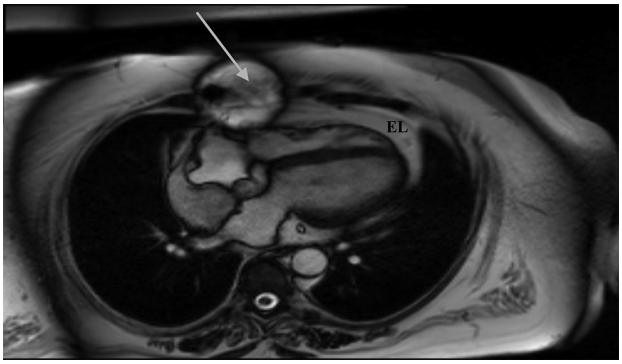


FIGURE 2. CMR imaging at 3-year follow-up showing stability of epicardial lipomatous hypertrophy (15 mm) compared with immediate postoperative CMR imaging (*arrow*: metallic artefact from a sternal wire). *EL*, Epicardial lipomatous.

Data Extraction

We extracted the author, year of publication, age, clinical presentation, imaging diagnosis, and treatment, when provided.

RESULTS

By using the predefined strategy, as indicated by the Preferred Reporting Items for Systematic review and meta-Analysis protocol,[1] 236 references were retrieved for initial screening, of which a total of 233 were excluded (Figure 3). Of the remaining 3 case reports, only 2 met our inclusion criteria. The patient and surgical characteristics from these 2 case reports[2,3] are summarized in Table 1. Our systematic review did not identify any published description of surgical resection of epicardial fat.

DISCUSSION

CLH is not a well-known cardiac pathology, and its diagnosis remains difficult; nevertheless, the anatomic and radiologic descriptions in our case confirmed a diagnosis of EAT hypertrophy with concomitant LHIS. CLH is defined as an infiltration of fat within the interatrial septum and an increase of the EAT thickness.[4]

Our systematic review identified that the most frequently published anatomic expression of CLH is LHIS (137 articles). It is characterized by excessive fat deposition in the interatrial septum and a thickness of more than 20 mm. The diagnosis of LHIS is most often made incidentally or during arrhythmia. Imaging diagnosis consists of the typical dumbbell-shaped appearance of the interatrial septum, and LHIS is associated with hypertrophy of EAT in 75% of cases.[5,6] In our case, the