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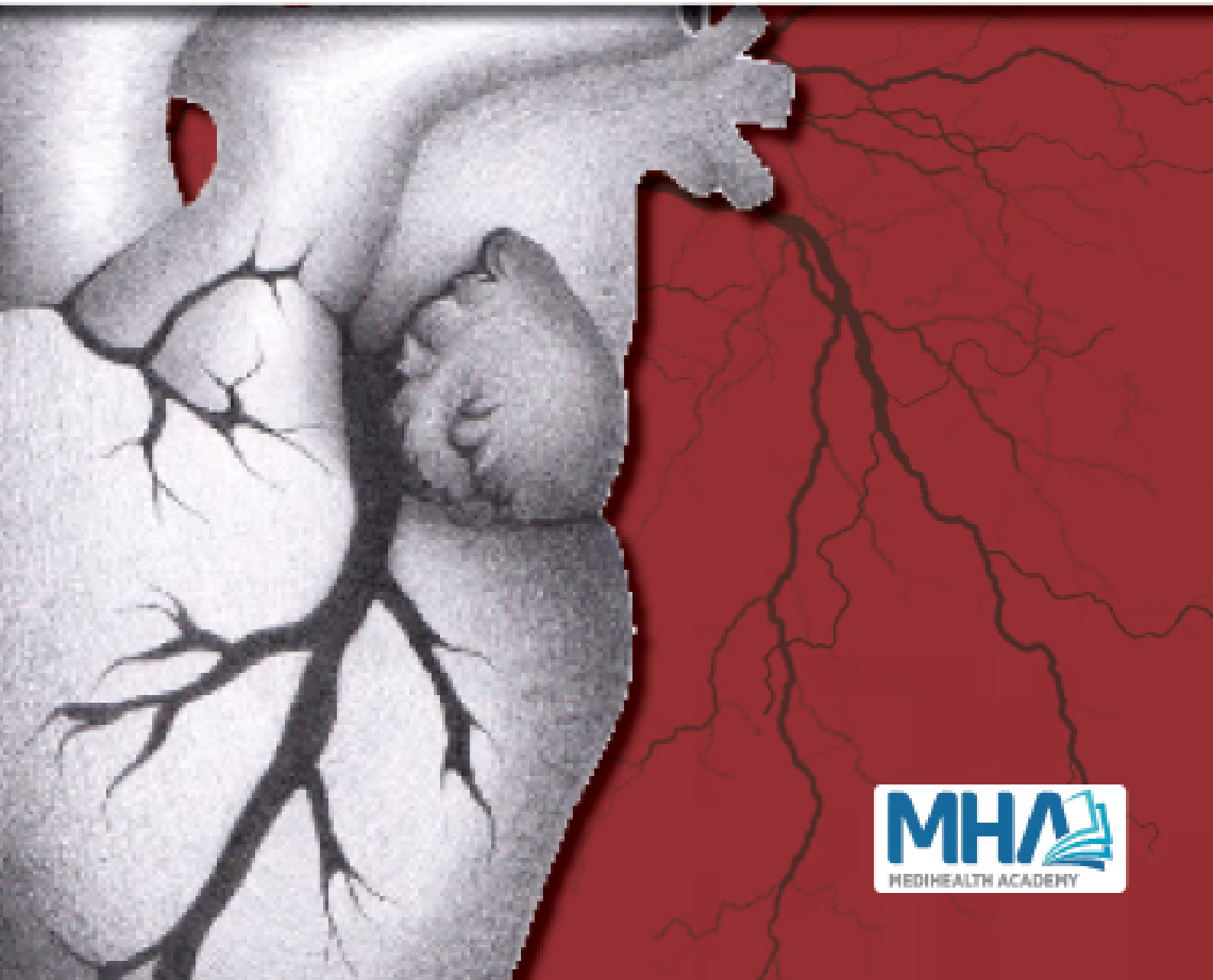

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ORIGINAL ARTICLE

CASE REPORTS

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Original Article

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Investigation of serum copeptin levels in patients with congestive heart failure

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ABSTRACT

Aims: Congestive heart failure (CHF) is characterized by symptoms and signs of volume overload and tissue perfusion insufficiency and accompanied by neurohormonal activation. Copeptin is a part of pre-pro-vasopressin and synthesized in equal molar amounts with vasopressin. We aimed to evaluate the sensitivity and specificity of copeptin in indicating the diagnostic value of CHF

Methods: This retrospective study included a total of 80 patients including 40 with heart failure and 40 healthy individuals. The groups were compared in terms of demographic features, laboratory findings including copeptin levels.

Results: There was no statistically significant difference between the groups in terms of gender ($p>0.05$). Age, hypertension, diabetes mellitus, smoking were statistically significantly higher in the heart failure group compared to the control group ($p<0.05$). In both groups, serum copeptin levels were higher in men than in women. Copeptin levels in the heart failure group were statistically significantly higher than in the control group ($p<0.05$). In the heart failure group, there was a negative correlation between serum copeptin levels and age, gender, hypertension, smoking, HDL cholesterol, hematocrit, creatinine levels.

Conclusion: Copeptin is a good indicator of the course of the disease in patients with heart failure.

Keywords: Congenital heart disease, risk factors, contemporary clinical practice

INTRODUCTION

Congestive heart failure (CHF) is a complex clinical syndrome that can be caused by any structural or functional disorder that impairs the ability of the ventricle to fill with and pump blood.[1] Heart failure is a physiopathologic condition defined as the inability of the heart to pump blood at a rate sufficient for the needs of metabolic tissues or to do so only with increased filling pressures. CHF, a complex syndrome characterized by symptoms and signs of volume overload and tissue perfusion insufficiency and accompanied by neurohormonal activation, is a biological disorder whose progression can be prevented.[1,2]

Copeptin is a part of pre-pro-vasopressin and synthesized in equal molar amounts with vasopressin. The advantage of copeptin is its long stability, rapid measurement from plasma and significance. It may remain stable for 14 days in plasma with EDTA and 7 days in plasma with heparin and citrate.[3] Copeptin is a 39 amino acid glycopeptide which is the C-terminal end of provasopressin.[4]

We aimed to evaluate the sensitivity and specificity of copeptin in indicating the diagnostic value of congestive heart failure (CHF) by comparing plasma copeptin levels in subjects with and without CHF (healthy volunteers), to demonstrate its diagnostic value at the bedside, and to define the relationship of copeptin levels with disease progression and recovery.

METHODS

This study was approved by Ethics Committee of Fırat University (Date:14/05/2010, Decision No: 23). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Between May 2010 and April 2011, 40 patients [mean age 56.37±8.71 years; 55% (n=22) male; 45% (n=18) female] who were hospitalized in the Cardiology Clinic of Fırat University Medical Faculty between May 2010 and April

2011 and who were diagnosed with heart failure based on anamnesis, physical examination, tele cardiography, electrocardiography, biochemical parameters and BNP levels and who had an ejection fraction (EF) of 40% or less measured echocardiographic ally by Simpson's method were included in the study; 55% (n=22) male; 45% (n=18) female] were included as heart failure group.

A total of 80 subjects, including 40 healthy individuals [mean age 51, 22±8.70 years; 50% (n=20) males and 50% (n=20) females; mean age 51, 22±8.70 years; 50% (n=20) males and 50% (n=20) females) with preserved ventricular function and an echocardiographic ejection fraction (EF) of 60% or higher as measured by Simpson's method, without symptoms and laboratory findings of heart failure on anamnesis and physical examination, were included as the control group.

Exclusion criteria from the patient and control groups were those with malignancy, those with problems in hematologic parameters, those who did not accept the study and individuals under the age of 18 years. Copeptin levels were measured using an automated immunoassay.

Statistical Analysis

Statistical analysis was performed using SPSS 12.0 (Statistical Package for Social Sciences) program. Parametric data were expressed as mean ± standard deviation and nonparametric data as (%). Oneway Anova test was used to compare parametric data and normality was evaluated by Kormogorov-Smirnov test. Logarithmic transformations were applied to parameters that did not exhibit normal distribution characteristics before statistical analysis. Results were evaluated at 95% confidence interval and significance was evaluated at p<0.05 level.

RESULTS

There was no statistically significant difference between the groups in terms of gender (p>0.05). Age, hypertension, diabetes mellitus, smoking were statistically significantly higher in the heart failure group compared to the control group (p<0.05)(Table 1). In the heart failure group, 15 patients (37.5%) had a history of acute myocardial infarction.

Table 1. Demographic characteristics of the groups

	Control group (n=40)	HF* group (n=40)	p
Age (year)	51; 22±8.70	56.37±8.71	<0.5 (p=0.010)
Female n (%)	20 (50%)	18 (45%)	>0.05 (p=0.823)
Male n (%)	20 (50%)	22 (55%)	>0.05 (p=0.823)
Hypertension n (%)	-	18 (45%)	<0.05 (p=0.001)
Cigarette n (%)	21 (52.5%)	8 (20%)	<0.05 (p=0.005)
Diabetes mellitus	-	14 (35%)	<0.05 (p=0.001)

HF: Heart failure

When serum copeptin levels were analyzed between men and women in our study, serum copeptin levels were found to be 569±176.28 in men and 458.89±155.48 in women in the heart failure group and 394±56.13 in men and 394±56.13 in women in the control group. In both groups, serum copeptin levels were higher in men than in women. The difference between them was not statistically significant (p>0.05).

According to laboratory data, there was no statistically significant difference between the groups in hemoglobin, hematocrit and triglyceride levels (p>0.05). Total cholesterol, HDL cholesterol and LDL cholesterol levels were significantly lower in the heart failure group compared to the control group (p<0.05), and urea, creatinine and copeptin levels were significantly higher in the heart failure group compared to the control group (p<0.05) (Table 2).

Table 2. Characteristics of the groups from laboratory data

	Control group (n=40)	CHF group (n=40)	p
Hemoglobin (g/dl)	13.57±1.27	13.80±1.20	>0.05
Hematocrit (%)	40.29±4.29	41.55±4.38	>0.05
Total cholesterol (mg/dl)	210.88±58.68	179.15±40.36	<0.05
HDL cholesterol (mg/dl)	50.28±10.61	44.20±9.84	<0.05
LDL cholesterol (mg/dl)	130.32±24.74	117.23±31.02	<0.05
Triglyceride (mg/dl)	136.88±52.92	141.32±58.19	>0.05
Urea (mg/dl)	33.60±9.13	50.95±19.44	<0.05
Serum kreatinin (mg/dl)	0.97±0.16	1.120±0.249	<0.05
Serum copeptin (pg/mL)	387.50±65.8	519.50±174.22	<0.05

CHF: Congestive heart failure, HDL: High density lipoprotein, LDL: Low density lipoprotein

The difference in copeptin levels between the groups was statistically significant (p<0.05). Copeptin levels in the heart failure group were statistically significantly higher than in the control group. The copeptin level was 519.50 ± 174.22 in the heart failure group and 387.50 ± 65.8 in the control group. The difference was statistically significant (p<0.05, p=0.002) (Table 2, Figure).In the heart failure group (n=40), there was a positive correlation between serum copeptin levels and urea, smoking, total cholesterol, LDL cholesterol, triglyceride, hemoglobin and total cholesterol levels. However, in this group, serum copeptin levels were statistically significant only with smoking and gender (r: 0.416 and p= 0, 004 for smoking, r: -0, 319 and p= 0, 023 for gender) (Table 3).

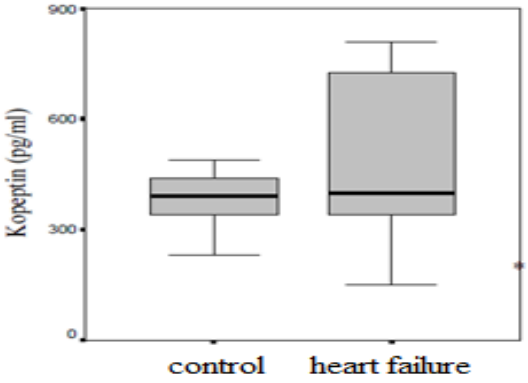

Figure. Serum copeptin levels

In the heart failure group, there was a negative correlation between serum copeptin levels and age, gender, hypertension, smoking, HDL cholesterol, hematocrit, creatinine levels.

In the control group (n=40), serum copeptin levels were negatively correlated with triglyceride, hematocrit, creatinine, urea, age, gender and smoking. In the control group (n=40), there was a positive correlation between serum copeptin levels and total cholesterol, HDL cholesterol, LDL cholesterol and hemoglobin (Table 4).

Table 3. Correlations of some parametric data with copeptin in the heart failure group

Heart failure group (n=40)	Copeptin (pg/ml)	
	r	p
Age (year)	-0.232	>0.05
Gender (male/female)	-0.319	<0.05
Cigarette	0.416	<0.05
Hypertension	-0.009	>0.05
Total cholesterol (mg/dl)	0.191	>0.05
HDL cholesterol (mg/dl)	-0.092	>0.05
LDL cholesterol (mg/dl)	0.011	>0.05
Triglyceride (mg/dl)	0.122	>0.05
Hemoglobin (g/dl)	0.024	>0.05
Hematocrit (%)	-0.043	>0.05
Urea (mg/dl)	0.036	>0.05
Serum kreatinin (mg/dl)	-0.254	>0.05

HDL: High density lipoprotein, LDL: Low density lipoprotein

Table 4. Correlations of some parametric data with copeptin in the control group

	Copeptin (pg/ml)	
	r	p
Age (year)	-0.138	>0.05
Gender (male/female)	-0.100	>0.05
Cigarette	-0.198	>0.05
Hypertension	-	-
Total cholesterol (mg/dl)	0.046	>0.05
HDL cholesterol (mg/dl)	0.160	>0.05
LDL cholesterol (mg/dl)	0.031	>0.05
Triglyceride (mg/dl)	-0.113	>0.05
Hemoglobin (g/dl)	0.124	>0.05
Hematocrit (%)	-0.071	>0.05
Urea (mg/dl)	-0.231	>0.05
Serum kreatinin (mg/dl)	-0.227	>0.05
Diabetes mellitus	-	-

HDL: High density lipoprotein, LDL: Low density lipoprotein

DISCUSSION

Chronic CHF, which is a consequence of many cardiovascular diseases, is one of the leading causes of morbidity and mortality.[5,6] Despite the progressive decrease in the mortality of coronary artery disease and hypertensive cardiovascular diseases, the incidence and prevalence of HF increases proportionally with aging. The main reasons for the increase in the prevalence of HF are the increase in the elderly population and the prolongation of life span due to the development of diagnostic and therapeutic methods in cardiovascular diseases.[5,6]

Although advances have been made in the pathogenesis and treatment of heart failure, we still lack important insights into the underlying disorders, especially those at the cellular level. Despite the shortcomings, ongoing research at both basic and clinical levels will allow clinicians to better understand and treat this clinical syndrome.[7] Expectations are that biochemical markers such as BNP, copeptin and short echocardiograms will be used to screen for heart failure and thus prevent the onset of the syndrome by starting treatment at an earlier stage.[8]

Vasopressin is an antidiuretic and vasoconstrictor hormone.[9,10] and has effects on free water absorption, body fluid osmolality, blood volume and vascular tone. It is also thought to cause cell proliferation. All these effects are regulated through V2 (renal) and V1a (vascular) receptors. There are data showing that vasopressin is related with the severity of heart failure and the course of the disease.[11,12] Vasopressin is a hormone that is difficult to measure because it is mostly bound to platelets and cleared rapidly from the blood.[3] Copeptin is part of pre-pro-vasopressin and is synthesized in equal molar amounts to vasopressin. The advantage of copeptin is that it has a long stability, is rapidly measured in plasma and is significant. It can remain stable for 14 days in plasma with EDTA and 7 days in plasma with heparin and citrate.3 Copeptin is a 39 amino acid glycopeptide, which is the fragment at the C-terminal end of provasopressin.[4] The value of this marker as an indicator has been demonstrated in patients with critical illness,[4,13] coronary artery disease 14 and advanced heart failure.

Copeptin has been shown to be at least as valuable a marker as BNP in showing the course of the disease in patients with advanced heart failure.[8] Indeed, studies have shown that vasopressin is not only elevated in heart failure but also associated with the severity of the disease.[6,15] Many studies on copeptin have shown that it is a reliable marker in heart failure.

In our study, we found that serum copeptin levels were significantly increased in the CHF group compared to the control group in accordance with the literature.

In the 2007 University of Leicester study of copeptin after acute myocardial infarction (LAMP), copeptin was found to be a strong indicator of death and CHF in patients with acute myocardial infarction.[8] It has been observed that this marker provides additional contributions in determining the prognosis together with clinical findings in the classification of patients into low, intermediate and high risk groups.[8] Again in the SAVE study analysis, vasopressin was shown as an indicator of cardiac events that may develop after myocardial infarction.[16]

In the Optimaal study, BNP and copeptin were compared. As a result of the study, it was reported that copeptin was a strong and new marker for mortality and morbidity in patients who developed CHF after acute myocardial infarction.[17]

In our study, serum copeptin levels were significantly higher in patients who developed CHF after acute myocardial infarction.

In the Gruppo di Ricerca GISSI Heart Failure Trial (GISSI-Heart Failure), plasma concentrations of 4 components of the neurohormonal system were measured in patients with chronic and stable heart failure and their relationship with outcome was evaluated. These were atrial natriuretic peptide (MR-proANP), adrenomedullin (MR-proADM), C-terminal pro-endothelin-1 (CT-proET-1) and C-terminal pro-vasopressin (CT-proAVP or copeptin). At the end of the study, copeptin was evaluated as one of the best biological markers for prognostic information and risk stratification.18Nakamura et al.[11] showed that vasopressin

increased in patients with NYHA (New York Heart Association functional classification) functional capacity 3 and 4.

In our study, we found a clinical increase especially in patients with high functional capacity according to NYHA. Kelly et al.[19] found that C-terminal provasopressin (copeptin) was associated with left ventricular dysfunction, remodeling, death and heart failure after acute myocardial infarction. Neuhold et al.[20] compared copeptin and BNP levels in heart failure patients at the Austrian Medical University in 2008. As a result of this study, it was found that the predictive value of copeptin was superior to BNP in determining 24-month mortality and was the strongest single predictor of mortality in patients with NYHA functional class II. More recent data also showed that copeptid levels are important indicators of heart failure, its severity and prognosis.[21-23]

5. CONCLUSION

In our study, we found that serum copeptin levels were significantly increased in the CHF group compared to the control group in accordance with the literature. In conclusion, in our study, serum copeptin levels were significantly increased in CHF patients and in patients with CHF after acute myocardial infarction compared to the control group. This shows that copeptin is a good indicator of the course of the disease in patients with heart failure.

Measurement of serum copeptin levels may be a predictor of mortality and morbidity in heart failure patients; however, randomized, prospective long-term follow-up studies are clearly needed for this. Our study is one of the pioneering studies on this subject and we believe that it will shed light on further studies on this subject.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was initiated with the approval of the Fırat University Medical Faculty Clinical Researches Ethics Committee (Date: 15.05.2010, Decision No23).

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Case Report

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Primary left atrial rhabdomyosarcoma in a 42-year old patient: an unusual cardiac tumor presentation

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ABSTRACT

Left atrial rhabdomyosarcoma is an extremely rare and often overlooked form of primary cardiac malignancy that primarily affects the atria of the heart. The aggressive nature of atrial rhabdomyosarcoma necessitates early recognition, a comprehensive diagnostic workup, and prompt intervention to achieve the best possible outcomes. In this case report, we present the clinical details of a 42-year-old female diagnosed with left atrial rhabdomyosarcoma, who exhibited symptoms of recent pulmonary edema, weight loss, and alternating flushing. Diagnosis, surgical treatment, and the iatrogenic atrial septal defect complication due to interatrial septum invasion were discussed.

Keywords: Primary cardiac tumor, rhabdomyosarcoma, cardiooncology

INTRODUCTION

Atrial rhabdomyosarcoma is an extremely rare and often overlooked form of primary cardiac malignancy that primarily affects the atria of the heart. This neoplasm, characterized by its aggressive nature and predilection for young individuals, poses a distinctive challenge for both clinicians and researchers. Its scarcity and unique clinical presentation have contributed diagnostic challenges, and a dearth of well-documented cases in the medical literature.[1] The management strategy of such tumors is not well defined due to the insufficient data on primary malignant cardiac neoplasms.[1,2] In this case report, we shed light on the clinical, diagnostic, and therapeutic aspects of a case of atrial rhabdomyosarcoma presenting with symptoms of heart failure (HF) due to left atrial (LA) invasion, with the aim of enhancing our understanding of this rare cardiac tumor and its potential implications for patient care.

CASE

A 42-year-old female presented to the cardiology clinic with complaints of exertional dyspnea classified as New York Heart Association class 3, a history of recent pulmonary edema, weight loss and alternating flushing. The patient had no significant previous infections or operations. Her family history of cardiac disease was not significant. Her past medical history was unremarkable, and she did not take any medications, regularly consume alcohol, or use any illicit substances. Upon evaluation, the patient exhibited symptoms of dyspnea and orthopnea.

The patient's vitals showed a blood pressure of 126/87 mmHg, body temperature of 36.2°C, and a heart rate of 96 bpm. Physical exam was notable for inspiratory sounds on the left lower zone and a mild to moderate (3/6) holosystolic murmur at the apex. Additionally, her ECG revealed sinus tachycardia,

otherwise within normal ranges (QRS interval: 110 ms, QTc interval: 402 ms). The biochemical panel was within normal limits except high sensitive C reactive protein was elevated at 32 mg/L. Transthoracic echocardiography (TTE) demonstrated left ventricular ejection fraction to be 60%, moderate mitral regurgitation, and a suspicious mass in the LA. Subsequently, transesophageal echocardiography (TEE) was performed, and a 24x11 mm non-mobile mass was detected in the posterior wall of the left atrium which elongated through the interatrial septum (IAS) (Figure 1).

The mass was surgically removed by clean dissection along the atrial wall and IAS. Upon surgical inspection, the tumor encountered in the posterior atrial wall surface seemed to be roughened and elongated to the IAS. Tumor infiltration of the IAS was evident. A partial defect in IAS was left as a result of the extensive excision. Due to the high possibility of recurrence and the necessity of serial follow-up, IAS was not closed with a surgical patch. There was no evidence of pericardial or other adjacent structural spread of the mass.

Histological examination of the tumor revealed that the presence of spindle cells with eosinophilic cytoplasm and a fascicular pattern which stained positive for desmin and caldesmon, consistent with rhabdomyosarcoma (Figure 2).

In the postoperative period, a follow-up TTE and TEE did not detect any residual mass lesion in the left atrium (LA). However, a left-to-right shunt was observed from an approximately 10 mm sized defect in the posterior part of the interatrial septum (IAS) (Figure 3). This shunt was determined as restrictive, and close monitoring was recommended. With the oncology consultation, 8 cycles of adjuvant chemotherapy regimen involving vincristine, dactinomycin, cyclophosphamide (VAC) with serial cardiac function monitoring (TTE). Following treatment, the patient did not experience a reoccurrence of dyspnea or other symptoms at 9-month follow up and remains under medical surveillance. Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

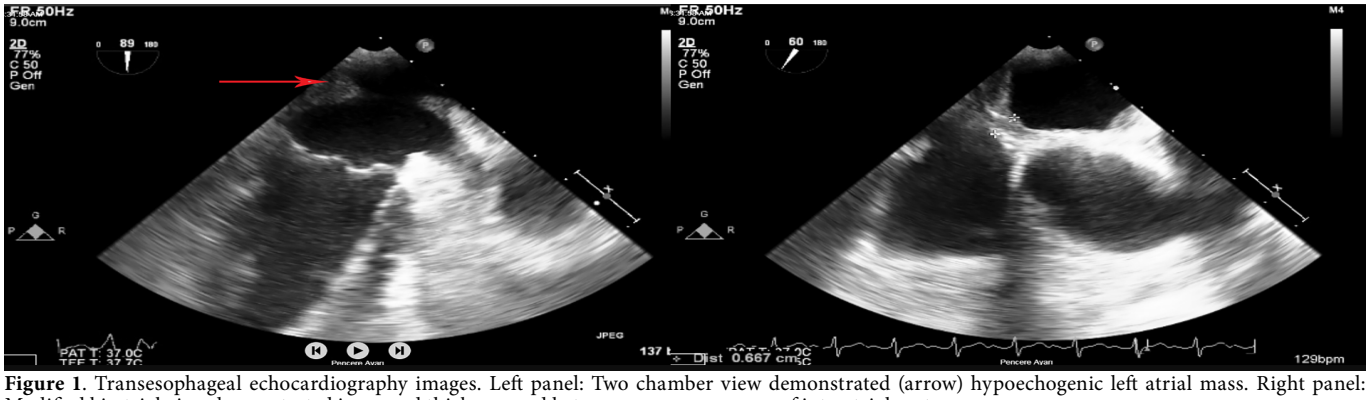

Figure 1. Transesophageal echocardiography images. Left panel: Two chamber view demonstrated (arrow) hypoechogenic left atrial mass. Right panel: Modified bi-atrial view demonstrated increased thickness and heterogenous appearance of interatrial septum.

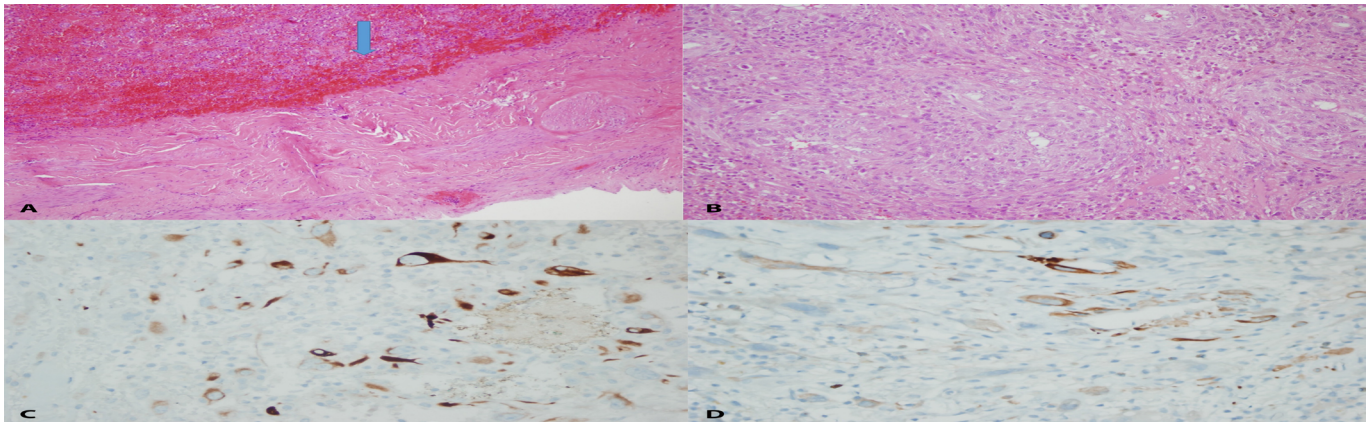

Figure 2. Histological examination of the tumor. A: Blue arrow demonstrated the infiltration of tumor in myocardial wall (H&E, X100 magnification). B: Tumoral cells forming fascicular pattern spindle cells with eosinophilic cytoplasm (H&E, X200 magnification). C: Intracytoplasmic reaction positive for Desmin (IP stain for Desmin, X400). D: Caldesmon positive tumor cells (X400 magnification). Abbreviations: H&E: Hematoxylin & Eosin stain, IP: immunoperoxidase stain.

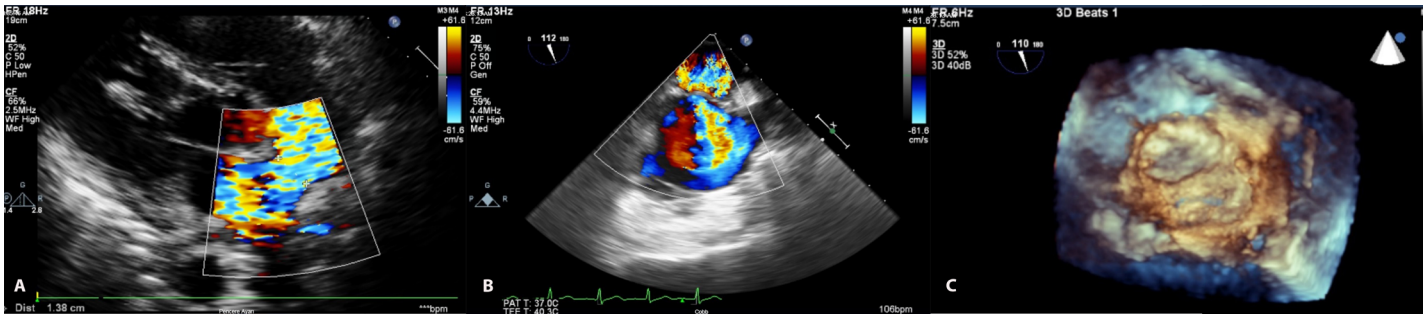

Figure 3. Postoperative TTE and TEE images. A: Modified subcostal plane imaging demonstrated large interatrial defect after surgery. B: Modified bi-caval view demonstrated left to right shunt after surgery. C: 3-Dimensional reconstruction demonstrated a slit-like defect in IAS. Abbreviations: TTE: Transthoracic echocardiography, TEE: Transesophageal echocardiography, IAS: interatrial septum.

DISCUSSION

Cardiac tumors, though relatively rare compared to tumors in other parts of the body, represent a diverse group of neoplasms that can affect various locations while mostly affecting cardiopulmonary functioning.[1,3] While a great portion of them present asymptomatically, we report a case of a patient who experienced HF symptoms as a complication of LA rhabdomyosarcoma with systemic symptoms.

Considering the fact that the heart is one of the rarest locations for tumorigenesis, primary cardiac tumors constitute a minority of all cardiac tumors which incidence was reported as 0.0017% to 0.28%.[1,2,4] Among these primary cardiac tumors, approximately 25% are malignant in nature, with rhabdomyosarcoma being one of the most prevalent malignant examples.[1,2] Malignant neoplasms encompass a range of tumors, including angiosarcomas, rhabdomyosarcomas, fibrosarcomas, and osteosarcomas.[5,6] The behavior of malignant neoplasms within the heart varies based on their specific subtype, histological characteristics, and invasiveness.[6] The determination of the subtype is best achieved through the application of immunohistochemical techniques.[6]

Clinical manifestations and symptoms associated with these cardiac tumors can differ based on their location within the heart, primarily found in the atria and their propensity for growth. They can be originated from various locations within the heart.[1] Although these neoplasms originate intramurally, they can present with complaints of dizziness and chest pain and can lead to arrhythmias and pericardial effusions, which can advance to tamponade, as a result of transmural invasion.[1] Alternatively, they can lead to symptoms similar to those in our patient, stemming from the obstruction of cardiac cavities, including the right and left atrium (RA/LA), inferior vena cava, and others in relation to the neighboring structure they are mainly affecting.[3,7,8] In cases where a tumor invades the right side of the heart, the presentation can often resemble that of tricuspid or pulmonary valve diseases, with signs of right heart failure.[1,7] Tumor metastases tend to be detected in various organs, including the lung, liver, thoracic lymph nodes, and pancreas.[2,3,8] Additionally, on rare occasions, cardiac rhabdomyosarcoma may give rise to cerebral embolisms.[2]

Atrial rhabdomyosarcoma may manifest with systemic symptoms such as weight loss, night sweats, and fever.[2,7,8] The presence of B symptoms and flushing can be the primary complaints of the patients. Recognizing these systemic symptoms is crucial for early detection and intervention, as they can provide valuable clues in the comprehensive assessment of patients with atrial rhabdomyosarcoma. Physicians should keep in mind about possible diagnosis of atrial rhabdomyosarcoma as a differential in patients presenting with similar complaints.

Various imaging studies, such as TTE, TEE, computed tomography, coronary angiography, and magnetic resonance imaging, play a pivotal role in both diagnosis and guiding surgical interventions.[7,8] Serial TTE and TEE should be considered throughout the presentation. However, definitive diagnosis relies heavily on tissue biopsy and subsequent histopathological analysis.

Classical treatment modalities for managing these cardiac tumors typically involve a combination of surgical resection, chemotherapy, and radiotherapy. In our patient's case, surgical resection, and chemotherapy of were employed, and a restrictive IAS defect was identified as a complication following tumor excision surgery. Through this case, we aimed to underline the importance of early recognition and multidisciplinary management in improving the prognosis of this intriguing and uncommon malignancy.

CONCLUSION

The aggressive nature of atrial rhabdomyosarcoma necessitates early recognition, a comprehensive diagnostic workup, and prompt intervention to achieve the best possible outcomes. In the absence of standardized treatment protocols for this rare malignancy, the establishment of an individualized, patient-specific therapeutic plan is paramount. This case report underscores the need to consider atrial rhabdomyosarcoma as part of the differential in cardiopulmonary disease, especially those in young, otherwise healthy individuals, as the limited data available hinders our understanding of this rare condition. Further investigations and clinical experiences are warranted to advance our knowledge and enhance the care provided to those affected by this exceptionally rare disease.

ETHICAL DECLARATIONS

Informed Consent

The patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Case Report

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Giant mobile thrombus in left atrium

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ABSTRACT

Giant mobile left atrial thrombus has a rare occurrence. Rheumatic mitral valve stenosis (RMVS), which is frequently seen in developing countries, atrial fibrillation, and left atrial dilatation contribute to faster and higher volume thrombus formation. A 46-year-old female suffering from dyspnea was admitted to the cardiology outpatient. Transthoracic and transesophageal echocardiography confirmed rheumatic mitral stenosis with 3.5x2.5 cm mobile circulating thrombus in left atrium. The patient underwent successful surgical thrombus removal, prostatic mitral valve replacement, and surgical ligation of the left appendage.

Keywords: Left atrial thrombus, rheumatic mitral valve stenosis, atrial fibrillation

INTRODUCTION

We often see left atrium thrombus formation in the left atrial appendage, and the most obvious etiology is atrial fibrillation.[1] Atrial fibrillation may be caused by valvular causes, especially mitral stenosis, or by non-valvular causes such as diabetes mellitus, heart failure, advanced age, alcohol and smoking.[2] Giant mobile left atrial thrombus has a rare occurrence. In addition to rheumatic mitral valve stenosis (RMVS), which is frequently seen in developing countries, the presence of atrial fibrillation also contributes to faster and higher volume thrombus formation.[3] Herein, we report a case with left atrial giant mobile thrombus due to RMVS.

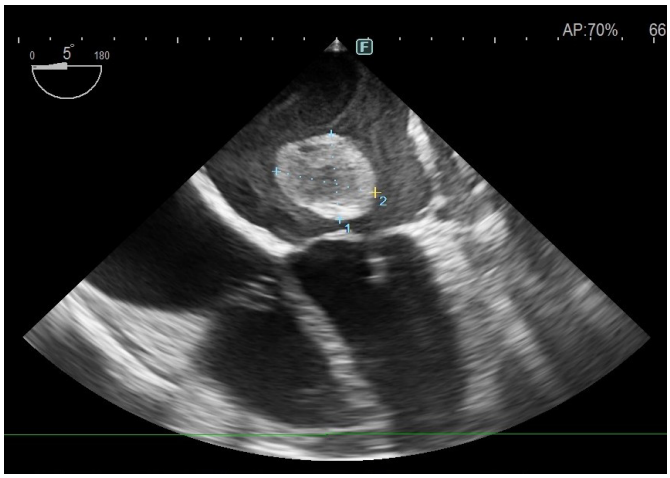
CASE

A 46-year-old female suffering from dyspnea was admitted to the cardiology outpatient clinic. She has no history of hypertension, diabetes mellitus, dyslipidemia or previous cardiac history. She had mild dyspnea for exercise for 3 months ,however dyspnea got worse in the last 3 days.

General examination was unremarkable. ECG showed atrial fibrillation without any ischemic ST/T wave changes. CK-MB, Troponin, and other hematological parameters were within normal limits.

2D Echocardiography showed: rheumatic mitral stenosis with maximum gradient of 19 mmHg and mean gradient of 9 mmHg and second degree aortic regurgitation. There was a mobile thrombus wandering in the left atrium (3.5× 2.5 cm). Third degree tricuspid regurgitation and minimal pericardial effusion were also observed.

The systolic pulmonary pressure was measured as 60 mmHg. Transesophageal echocardiography showed spontaneous echo contrast with 3.5x 2.5 cm mobile circulating thrombus in left atrium (Figure 1, Video 1). There was also extensive thrombus burden in left atrial appendage.



The patient was admitted to cardiology clinic and surgery was planned. The coronary angiography did not show any significant coronary artery disease. The patient underwent surgical thrombus removal, prostetic mitral valve replacement, and surgical ligation of the left appendage. Recovery was uneventful and she was discharged a week latter with medical therapy including warfarin.

DISCUSSION

Rheumatic heart disease, in which the mitral valve in mostly affected, remains the leading cause of cardiovascular mortality in children and young adults in developing countries, with an estimate of approximately 375,000 deaths per year in 1990 and 320,000 deaths in 2015.[4] With progressive dilatation of the left atrium, spontaneous echo contrast, and RMVS, the tendency to thrombus formation significantly increases. The risk of LA thrombus formation in moderate-severe RMVS patients is around 17%, and this rate doubles when AF is accompanied.[5,6] In our case, the absence of any known cardiac history, presence of LA dilatation, intense spontaneous echo contrast, accompanying AF, and the lack of anticoagulant use for AF created a highly prone situation for thrombus formation.

In the differential diagnosis of intracavity cardiac mass, thrombus, myxoma, lipoma and non-myxomatous neoplasm should always be kept in mind.[7] However, in our patient, we prioritized thrombus due to the predisposing factors we mentioned before and the imaging findings. Since intense thrombus was observed in the LAA, we hypothesized that the thrombus in the LAA grew considerably and then broke off, becoming free in the LA.

In patients with RMVS, the symptom status of the disease, atrial fibrillation, pulmonary artery pressure, and, rarely, the presence of free mobile thrombus with a high risk of systemic embolism, as in our patient, are the main factors that determine the type of treatment.[8] Mitral valve balloon valvuloplasty and surgery are the two main treatment options for RMVS, but in our patient, we preferred emergency surgery due to high burden of LAA thrombus and mobile large thrombus with high risk of embolism in the LA.

CONCLUSION

In countries where the prevalence of rheumatic valve disease is relatively high, large mobile LA thrombi may rarely be found on transthoracic echocardiography in RHD cases without any history. Since there is a significant risk of mortality and morbidity, diagnosis and treatment in a timely manner is very important.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Case Report

DOI: 10.51271/JCCVS-0033

Renal denervation for resistant hypertension in an elderly patient despite quintuple antihypertensive therapy: a case report

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ABSTRACT

Hypertension is the leading cause of cardiovascular morbidity and mortality. Renal nerves have critical roles in the regulation of blood pressure and fluid volume, and their dysfunction is closely associated with cardiovascular diseases. The denervation procedure is performed through a renal ablation catheter and has been shown in recent trials to provide impressive blood pressure reductions and a favorable safety profile in drug-resistant hypertension. We present a 78-year-old patient who underwent renal denervation due to unresponse to quintuple antihypertensive treatment.

Keywords: Hypertension, resistant, ablation, blood pressure

INTRODUCTION

Hypertension is the leading cause of cardiovascular morbidity and mortality. Renal nerves have critical roles in the regulation of blood pressure and fluid volume, and their dysfunction is closely associated with cardiovascular diseases.[1] Renal nerves consist of sympathetic efferent and sensory afferent nerves. Activation of efferent renal sympathetic nerves induces renin secretion, sodium absorption, and increased renal vascular resistance, resulting in increased blood pressure and fluid retention.[2] Afferent sympathetic stimuli release epinephrine/norepinephrine from the central nervous system. These stimuli increase renin/aldosterone release from the kidneys and adrenal glands, reduce glomerular filtration and renal blood flow, and increase salt/water retention. Drug-resistant hypertension is defined as BP > 140/90 mmHg despite the use of three or more antihypertensive drugs, one of which is a diuretic. It remains common despite the availability of several classes of effective antihypertensive agents. Sympathetic hyperactivity has long been known to be a major contributor to resistant hypertension, but surgical radical sympathectomy was abandoned several decades ago due to its significant side effects.[3] It has been shown that sympathetic activity increases by 45-60% in hypertension (HT), sympathetic activity decreases after renal denervation (RDN), and systolic/diastolic blood pressure decreases exponentially. The newly developed minimally invasive catheter-based method aims to partially block the renal sympathetic nerves. The denervation procedure is performed through a renal ablation catheter and has been shown in recent trials to provide impressive blood pressure reductions and a favorable safety profile in drug-resistant hypertension.[4] Although the long-term effectiveness and safety of renal denervation have not yet been determined, emerging data suggest that the benefits of renal denervation may extend beyond blood pressure control. Renal denervation studies continue to reveal a strong antihypertensive effect, especially in studies where drugs are discontinued subsequently.[5]

Herein, we present a 78-year-old patient who underwent renal denervation due to unresponse to quintuple antihypertensive treatment.

CASE

A 78-year-old female patient, who had been followed up for HT in the last 15 years, applied to our outpatient clinic because her blood pressure remained high in recent years despite a multi-drug treatment for HT. The patient presented with complaints of intermittent headache, shortness of breath, tinnitus, and fatigue. Her office BP measurement was 180/95 mmHg. The 24-hour ambulatory BP measurement was 168/105 mmHg during the day and 155/97 mmHg at night. Sinus rhythm and left ventricular hypertrophy findings were noted in the current ECG. An EF of 60%, hypertrophic interventricular septum, and diastolic dysfunction were detected in the ECHO. On physical examination, S1 and S2 heart beats were found to be displaced laterally, and +1 peritibial edema was observed. The drugs she took regularly were Ramipril 10 mg, Amlodipine 10 mg, Bisoprolol 10

mg, Furosemide 40 mg, and Doxazosin 8 mg. The reason spironolactone was not used in the patient's treatment is due to the patient's lack of response to previous diuretic treatments and the side effect profile(hyperkalemia). Additionally, secondary causes of hypertension were thoroughly evaluated, with particular consideration given to conditions like primary hyperaldosteronism, which are challenging to diagnose based on clinical findings.No renal artery stenosis was detected in the renal Doppler USG performed. There were no findings suggestive of secondary hypertension.

We planned a renal denervation procedure for our patient. After light sedation, we placed a 6F sheath into the femoral artery under local anesthesia. We cannulated the right renal artery with the JR4 guiding catheter and obtained images of the renal artery and its branches. We reached the distal part of the renal artery and its branches with a soft-tipped 0.014 mm wire. With the renal denervation catheter, the distal branches of the renal artery with a diameter greater than 3 mm, the bifurcation point, the junction of the renal artery, and the renal artery in the abdominal aorta were ablated one by one. (Figure) Then, left renal artery angiography was performed. With the renal denervation catheter, the bifurcation points of the renal artery branches with a distal diameter greater than 3 mm and the abdominal aorta/renal artery ostium were ablated. Then the renal denervation catheter was removed, both arteries were checked with angiography, no complications such as dissection, rupture, embolization, etc. were observed, and the procedure was completed successfully.

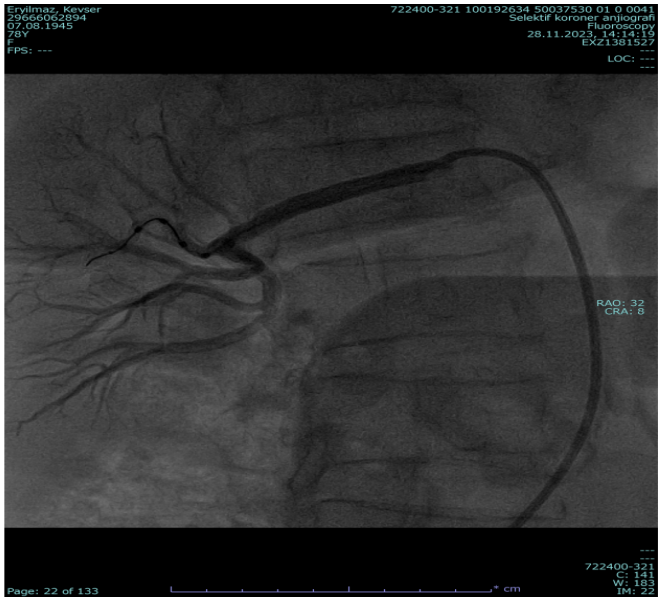

Figure. Image during the renal denervation procedure

DISCUSSION

Approximately 12% of patients with essential hypertension are considered refractory to conventional therapy, whose blood pressure remains persistently elevated despite the use of three or more pharmacological agents. Catheter-based RDN, a modern incarnation of a historically effective therapy, has recently emerged as a new treatment strategy. Subsequently, randomized data of BP reduction collected while using the Symplicity® catheter (Medtronic Inc. Minneapolis, MN, USA) in a group of patients taking an average of five antihypertensive medications at months 1, 3, 6, and 12, respectively, showed office BP decreases of 20/10, 24/11, and 25/11 mmHg. Early blood pressure decreases after RDN have been reported in some cases. However, in other cases, the response does not always appear immediately or may take several months to appear. The role of RDN in the treatment of resistant hypertension remains unclear, but the sustained and dramatic BP reductions observed in these and other cases should stimulate further research to improve understanding of the mechanisms mediated by hypertension and to identify patients likely to achieve the most dramatic responses with RDN.[3] In our case, we performed renal denervation for essential hypertension resistant to regular and quintuple antihypertensive treatment and gradually discontinued the antihypertensive drugs afterwards. At the 1st month follow-up, the ambulatory systolic blood pressure value decreased by an average of 25 mmHg.

Recent studies have discussed the long-term efficacy and safety of RDN. However, there is a need for more extensive studies involving larger patient groups and long-term follow-ups. This case study aims to contribute to filling this gap in the literature.[6,7,8]

CONCLUSION

Renal denervation can be an effective option for patients who have resistant hypertension.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Journal of
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Case Report

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A large pericardial cyst causing persistent chest tightness and dyspnea: a case report

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ABSTRACT

Pericardial cysts are a rare cause of mediastinal masses with an incidence of about 1 in 100,000 patients. Most of the cases are secondary to congenital incomplete fusion of the pericardial sac. More than two-thirds of pericardial cysts are located in the right cardiophrenic angle. Over 50% of pericardial cysts are asymptomatic and discovered incidentally during thoracic imaging such as chest X-ray, computed tomography (CT) scans and transthoracic echocardiograms. Symptomatic cases commonly present with non-specific symptoms such as dyspnea, chest pain, and persistent cough. A 48-year-old female was referred for assessment of shortness of breath and atypical chest tightness for several years. The echocardiography revealed a large cystic-appearing mass presenting with slight compression of the right-atrium. A cardiac CT scan and magnetic resonance imaging (MRI) scan were performed, confirming the presence of a large pericardial cyst with no signs of complications like tamponade or pericarditis. As the patient had symptoms and cyst had compressive effects, surgical resection was done.

Keywords: symptomatic pericardial cyst, incidental pericardial lesion, pericardial cyst management

INTRODUCTION

Pericardial cysts (PCs) are a rare benign mass of the mediastinum with an incidence of about 1 in every 100,000 persons.[1] Most cases are congenital but can also be acquired after cardiothoracic surgery, inflammatory conditions or following trauma.[2] Pericardial cysts are most commonly found in the right cardiophrenic angle (70% of cases) followed by the left cardiophrenic angle (22%) and other parts of the mediastinum (8%).[3] Typically found in the third or fourth decade of life, PCs have no gender predilection in their prevalence.[4]

More than 50% of pericardial cysts are asymptomatic and often diagnosed incidentally during chest X-rays, CT scans, or echocardiography.[2,3] In symptomatic cases, the most commonly reported symptoms are dyspnea, chest pain, and persistent cough.[2,3] Symptoms mostly arise usually due to compression of adjacent structures like the lungs or right atrium.

In our study, we illustrated an incidental finding of the pericardial cyst in a patient who presented with shortness of breath and atypical chest tightness for several years.

CASE

A 48-year-old female presented to the gynecology department for diagnostic laparoscopy due to chronic pelvic pain and frequent vaginal infections. An abdominal ultrasound detected bilateral ovarian cysts and endometriosis, prompting a cardiac evaluation prior to laparoscopy. The patient was referred for assessment of shortness of breath and atypical chest pain persisting for several years. Physical examination was unremarkable, and an ECG showed normal sinus rhythm. A chest X-ray revealed a well-defined, oval mass in the right cardiophrenic angle. Transthoracic echocardiography (TTE) showed preserved systolic function without regional wall motion abnormalities or significant valve disease but revealed a cystic-appearing mass causing slight compression of the right atrium.

A chest CT scan showed a cystic formation in the inferior anterior mediastinum measuring 62mm x 40mm with a density of 6 HU.(Figure 1)

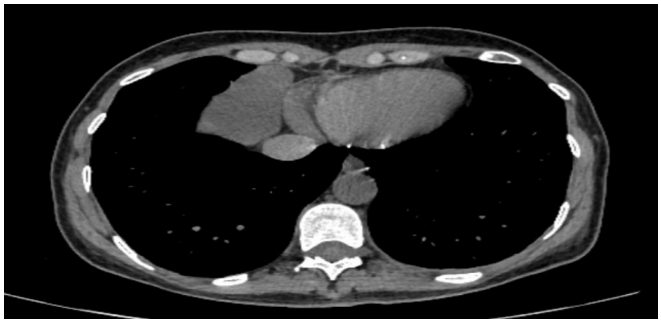

Figure 1. CT chest showing pericardial cyst

Further evaluation with cardiac magnetic resonance (CMR) revealed an ovoid pericardiac structure in the inferior anterior mediastinum at the right cardio phrenic angle, with dimensions of 82 mm x 34 mm x 33 mm (APxLLxSI).

In cine-MR images (Figure 2a) the cyst is presented with clear contours, and a generally thin capsule attached to the corresponding pericardium above the right atrium, with homogeneous isointense/slightly hyperintense internal structure in relation to the blood. Near the lateral face inside the cystic formation, the presence of a slightly thickened longitudinal hypointense structure (about 22 mm) can be outlined, with the pattern of a septum, which encloses a small peripheral space in the shape of a microcyst. T1w image shows hypointense and T2w image homogeneous hyperintense signal (Figure 2b and 2c); the presence of late enhancement (LGE) at the level of the surrounding capsule and lateral paracapsular (inside the structure) (Figure. 2d)

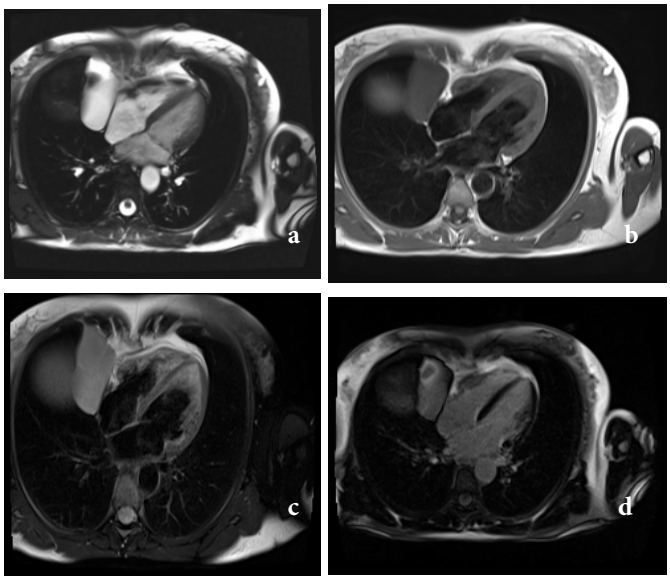

Figure 2. CMR showing pericardial cyst measuring 8.2 x 3.4 x 3.3 cm (a. cine-MR image, b. T1w image, c. T2w image, d. LGE)

Considering the elevated risk of cyst rupture the patient was referred to a cardiac surgeon. After lateral thoracotomy, the pericardium was opened. No fluid or pathologic finding was seen in the pericardium. On the right side and outside the pericardium, a pericardial cyst was totally excised. The pathologic report confirmed the diagnosis of pericardial cyst. (Figure 3 and 4).

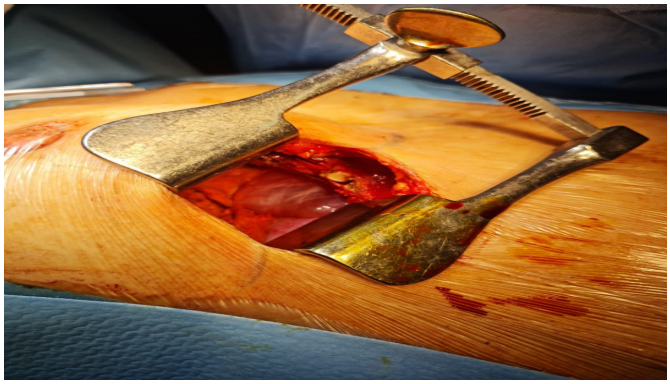

Figure 3. The surgical removal of pericardial cyst

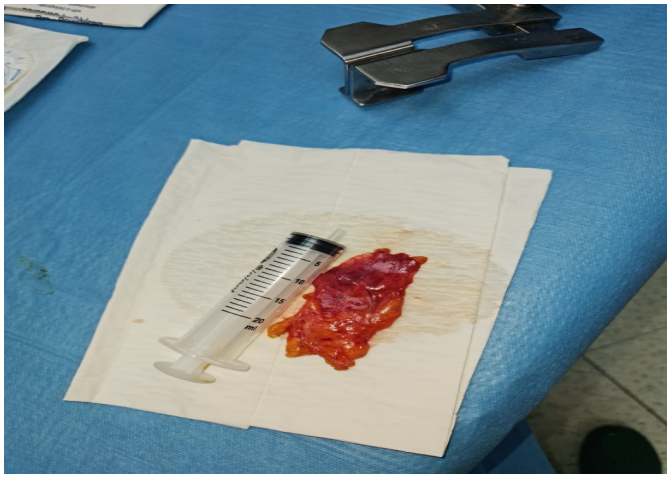

Figure 4. The surgical removal of pericardial cyst

DISCUSSION

Pericardial cysts are infrequent benign causes of mediastinal masses commonly asymptomatic.[5] Symptomatic cases typically arise from complications such as compression, inflammation, hemorrhage, and rupture.[6] A patient may experience dyspnea, chest pain, or palpitations in the event the cyst causes cardiac compression or irritation of the nearby structures. Our patient had persistent chest tightness and dyspnea for several years. However, the presence of a pericardial cyst in a typical location or, less frequently, in an unusual location, still poses a diagnostic challenge in distinguishing it from other intracardiac or mediastinal benign or malignant lesions. Further imaging studies are necessary for accurate diagnosis.

CT scans and especially cardiac magnetic resonance with its unique capabilities of tissue characterization are excellent for differentiating pericardial cysts from other mediastinal masses.[7]

Management decisions should be based on the presence or absence of symptoms, cyst size, hemodynamic state, and compression of important structures. Asymptomatic patients generally undergo conservative treatment depending on the location and size of cyst. If the patient is asymptomatic and the cyst is small, serial transthoracic echocardiograms are sufficient. If the patient is symptomatic and/or the cyst is large, resection is considered.

CONCLUSION

Even though PCs are a rare occurrence, physicians should not neglect to include them in their differential diagnosis for patients presenting with chest tightness, shortness of breath, or other nonspecific symptoms. Further investigations are crucial for differentiating simple pericardial cysts from other pericardial lesions. Based on the symptoms, size, and compression effect of the cyst, management may vary from serial echocardiogram to aspiration or surgical resection. Conservative management with regular echocardiographic follow-up is recommended in asymptomatic patients. However, surgical excision is considered a choice in symptomatic cases.

ETHICAL DECLARATIONS

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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