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Constrictive Pericarditis: A Case Study

Series Editor: A. Maziar Zafari, MD, PhD, FACC

*Assistant Professor of Medicine, Division of Cardiology, Department of Medicine, Emory University School of Medicine, Atlanta, GA;
Director, CCU, Atlanta Veterans Affairs Medical Center, Decatur, GA*

Contributors: William J. Nicholson, MD

*Cardiology Fellow, Division of Cardiology, Department of Medicine, Emory University School of Medicine, Atlanta, GA;
Cardiology Fellow, Atlanta Veterans Affairs Medical Center, Decatur, GA*

Andro G. Kacharava, MD, PhD

*Assistant Professor of Medicine, Division of Cardiology,
Emory University School of Medicine, Atlanta, GA;
Staff Cardiologist, Atlanta Veterans Affairs Medical Center, Decatur, GA*

Table of Contents

Introduction	2
Clinical and Physical Findings	3
Physiology and Pathology	5
Diagnostic Imaging Modalities	5
Hemodynamics	7
Treatment	7
Summary Points	10
References	11

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Constrictive Pericarditis: A Case Study

William J. Nicholson, MD, and Andro G. Kacharava, MD, PhD

I. INTRODUCTION

Constrictive pericarditis (CP) is a relatively rare postinflammatory disorder with different causes. The diagnosis of CP and its distinction from restrictive cardiomyopathy (RCM) remain notoriously difficult. Patients with both conditions present with increased left-sided and right-sided filling pressures, and their symptoms may resemble congestive heart failure (CHF). It is paramount to diagnose and distinguish these 2 entities because surgical pericardiectomy is potentially curative in CP.

Constrictive pericarditis is a condition in which the pericardial sac undergoes a process of progressive thickening and fibrosis; the natural elasticity of the pericardial sac is lost, subsequently placing the heart in a nonpliable and rigid encasement, impairing diastolic filling of the ventricles. *Restrictive cardiomyopathy* refers to idiopathic or systemic disorders involving the myocardium characterized by restrictive diastolic filling of the ventricles. Many clinical and hemodynamic findings are common to both CP and RCM, making the 2 disorders difficult to distinguish. The purpose of this review is to discuss the clinical presentation of CP and to focus on the hemodynamic, echocardiographic, and radiologic findings differentiating it from RCM. Although the diagnosis can usually be achieved via these noninvasive and invasive studies, surgical exploration or biopsy occasionally is needed to make definitive diagnosis. A case patient is presented to illustrate the management of CP.

ANATOMICAL CONSIDERATIONS

The pericardium is an elastic, closed fibroserous sac surrounding the heart. It consists of 2 layers of tissue: an external (fibrous) and an internal (serous) layer. The internal or serous layer is made up of both visceral and parietal pericardium. The visceral layer is a thin monolayer adherent to the heart's epicardium. The parietal pericardium is adjacent to the visceral pericardium on one side and becomes tightly opposed to the external fibrous layer on the other side. The space created between the visceral and parietal layers of the pericardium contains approximately 30 to 50 mL of serous fluid, which acts as a lubricant to minimize fric-

tion between the 2 layers of pericardium during the heart's movement throughout the cardiac cycle.¹ This fluid-filled space defines the pericardial cavity. The fibrous pericardium envelops the entire heart and extends over approximately 3 cm of the great vessels where it then attaches. Therefore, much of the ascending aorta, the main pulmonary artery, all 4 pulmonary veins, and portions of the inferior and superior venae cavae are contained within the pericardium.²

Fibrosis and scarring can affect each of the layers of the pericardium either separately or simultaneously. Development of adhesions and calcifications between the layers can lead to obliteration of the pericardial cavity, creating a rigid inelastic "shell" around the heart with resultant pathophysiologic consequences (ie, CP). As the pericardial cavity is obliterated in patients with CP, even the physiologic amount of pericardial fluid may disappear; however, excessive amounts of effusive fluid may be present in some patients. As this fluid is subjected to the constricting effects of the scarred pericardium, increased pressure can result in cardiac compression or tamponade with resultant hemodynamic deterioration; this entity is known as *effusive CP*.^{3,4} In such cases, the constrictive hemodynamics are masked by tamponade and may only become obvious after the pericardial fluid is drained by pericardiocentesis.

ETIOLOGIES

Many different conditions can produce acute pericarditis, and nearly all are capable of inducing constriction, albeit some more frequently than others (Table 1).⁵ The notable exception is acute rheumatic fever, which can produce extremely dense pericardial adhesions that rarely lead to constriction.⁶ As with many other diseases, the dominant causes of CP have changed over the years. Tuberculosis was historically the leading cause of CP and remains a dominant cause in developing countries. Neoplasm, collagen vascular disease, infectious etiologies, radiation therapy, and previous cardiac surgery are some of the more common causes of constriction in modern developed countries.⁷ However, in numerous cases (even after microscopic and culture examination of pericardial scar tissue), an inciting etiology is not found. In fact, idiopathic or presumed viral etiologies are now the leading