Chapter

Chronic Constrictive Pericarditis (CCP) in Africa: Epidemiology, Etiology, Diagnosis, and Surgical Treatment

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"What has been accomplished does not die, but too often alas, the personality of those who have handed the torch from one generation to another soon fades into oblivion."

Harvey Cushing (as quoted in Harvey Cushing: A Biography by John F. Fulton, 1947)

Abstract

Chronic constrictive pericarditis (CCP) is not rare in Africa with tuberculosis as the most common etiology. A long history of visceral tuberculosis, typical symptoms of CCP because of late clinical presentation in most cases, make diagnosis almost easy to establish. Echocardiography and cardiac catheterization may be helpful as final investigation when differentiation between CCP, endomyocardial fibrosis, and restrictive ventricular heart disease is difficult to attest. Pericardiectomy remains the only efficient treatment to carry out. We review the African teams' surgical experiences on pericardiectomy and report their surgical results and those worldwide in literature overall. In African groups, early mortality varies from 0 to 22% versus 2.1 to 18.6% outside Africa associated with New York Heart Association Functional Class IV as the most common significant risk factor for early deaths in all series in the World, including Africa.

Keywords: chronic constrictive pericarditis, epidemiology, etiology, diagnosis, pericardiectomy

1. Definition

Constrictive pericarditis is a reduction or a quasi-absence of heart distension because of a chronic inflammatory, thickened, and compressive pericardium.

2. Historical remark

Pericardium and pericardial diseases, especially constrictive pericarditis, have always been a concern for physicians and surgeons in cardiovascular field. Some remarkable landmarks [1, 2] are as follows: the first anatomical description of the pericardium has been done by Hippocrates in 460 BC. Centuries after, surgical treatment started in the seventeenth century by Riolan, who performed

a pericardotomy for an effusion in 1649. Then, Vieussens, some years later in 1679 and 1715, observed some cases of pericardial adhesions; from that step, the concept of constrictive pericarditis was born. Afterward, in 1728, Lancisi confirmed the existence of constrictive pericarditis and its risks such as cardiac compression and death via necropsy studies. Those observations were also mentioned by Morgagni (1761), Senac (1749), Laennec (1819), and Cheevers (1842). Clinical patterns related to the right atrium and ventricle compression, mainly as the most important source in the compression process, has been emphasized by Wilkes (1870). Based on anatomical findings, clinical pictures were then clarified: Kussmaul (1873) pointed out the venous pressure elevation in case of constrictive pericarditis; Pick (1896) described a new entity named "Pericarditis pseudocirrhosis of the liver." Since then, Pick Syndrome was recognized as a component of constrictive pericarditis clinical presentation. From that, surgical treatment was considered as the most efficient therapy for releasing heart compression due to pericardium thickness and fibrosis. By then, Rhen and Sauerbruck (1913) in Germany, Hallopeau in France (1921), Schmieden and Fisher (1926) in Germany, Churchill (1929) and Beck (1931) in USA performed the first cases of pericardiectomy for constrictive pericarditis successfully.

3. Anatomy and function of the pericardium

The pericardium is a solid fibro-serous sac that maintains the heart in the anterior mediastinum in a closed thoracic cavity called the "pericardial cavity." The pericardium has two main components: (1) an external, fibrous pericardium ensuring the fixity of the pericardium and (2) an internal serous pericardium with two principal layers: the visceral and the parietal pericardium. In a normal heart, the parietal and visceral pericardium release the pericardial cavity filled with a fluid up to 50 ml allowing easy systolic and diastolic heart movements. Pericardium prevents the heart from inflammation, infection, damage, and excessive dilatation; it also ensures heart stabilization and anchoring [1–3].

4. Pathophysiology

In case of constrictive pericarditis, restricted diastolic heart distensibility does exist and may provoke a right and a left ventricular preload decrease with a reduction of stroke volume and cardiac output [2, 3]. In Africa, we use to face on late clinical presentation of patients with massive pericardial thickness and calcifications inducting myocardial atrophy, fibrosis, and severe systolic dysfunction that significantly affect the results after pericardiectomy.

Key observations are described:

1. Impairment of ventricular distensibility and filling coexisting with ventricular stroke volume reduction.

2. Abnormal rapid diastolic ventricular filling and abnormal venous pressure elevation followed by an early diastolic ventricular dip.

3. High diastolic plateau due to a small expansion of the ventricular volume at the late diastolic period.

All those consequences determine the so called "Dip-and-plateau waveform."

5. Epidemiology and etiology

Classically, constrictive pericarditis appears as a complication of acute or effusive pericarditis. From Yadav's study [4], approximately 9% of patients with acute pericarditis will contract a pericardial constriction. In Africa, the frequency of acute pericarditis varies widely with geographical location ranging from 2 to 11.3% among patients admitted in a hospital for cardiovascular diseases [5–7]; it affects mostly young male population with an average age between 26 and 42 years old and tuberculosis as the most frequent etiology from 33 to 69.5% in sub-Saharan Africa [5, 6, 8]. Over the past decades, the incidence of tuberculous pericarditis has risen up because of the HIV epidemic in sub-Saharan Africa [8, 9]. Noubiap et al. [10] have reported a comprehensive understanding of the epidemiology of pericardial diseases in Africa confirming clearly that tuberculosis remains as the leading cause of pericardial diseases in African Resource-Limited Settings with poor outcome marked by a mortality rate between 18 and 25% including a very high one of 40% within 6 months among patients with HIV/AIDS positive. However, pericardial tuberculosis frequency is variable according to authors such as Mayosi [11] and Thwaites [12] who found, respectively, pericardial tuberculosis in 69.5% of cases admitted for pericardiocentesis and in 10% of all hospitalized patients with heart failure. Moreover, the risk for developing constrictive is very high for tuberculosis or purulent pericarditis: 20–30% and in almost all the cases of tuberculous pericarditis as reported by Gupta [13]. In their prospective study on 500 consecutive cases, Imazzio et al. [14] have demonstrated that the evolution from non-constrictive pericarditis to constriction is different according to pericarditis etiology: the risk of constriction is greater for bacterial etiologies (tuberculosis or purulent pericarditis) than viral or idiopathic acute pericarditis; the incidence rate of constriction is, respectively, 31.65 cases per 1000 person-years for tuberculous pericarditis and 52.74 person-years for purulent pericarditis versus 0.76 person-years for viral or idiopathic pericarditis. The same observation has been described by Permanyer-Miralda et al. [15] in a prospective study of patients with occurrence of constriction in 56 and 35% of patients with tuberculous and purulent pericarditis, respectively, and in 17% of patients with neoplastic pericarditis after an acute pericarditis. In Africa, and in literature overall, the epidemiological pattern, incidence, and prevalence of CCP are not well elucidated. Nevertheless, it is known that tuberculosis is the most frequent etiology of constrictive pericarditis in Africa and emerging countries (40–90%) [16] versus other rare etiology in our practice such as constrictive pericarditis after surgery. In a recent study, Gaudino et al. [17] have concluded their study insisting on the fact that constrictive pericarditis after surgery has its own pathophysiological characteristics, but we still be ignorant on its real origin and pathogenesis. Therefore, we should be vigilant and keep in mind that any cardiac symptoms without explanation must be suspected and be treated surgically as soon as possible if there is any doubt of pericardial constriction.

6. Diagnosis

In African countries [18–26], the diagnosis of constrictive pericarditis is usually obvious for patients present late after the development of the constrictive process characterized mostly by advanced clinical manifestations of right-sided heart failure (50–100%), progressive New York Heart Association (NYHA) Functional Class III or IV (42–100%) associated with an evident antecedent pulmonary and extra-pulmonary tuberculosis such as tuberculous pericarditis (26–99%). Duration of illness prior to surgery may range from 1 month to 25 years with an average of 15 or 30 months found, respectively, by Ali et al. [26] and Yangni-Angate et al. [21].

Generally, male gender predominance is noted (60–80%), patients have an average age below 40 years; for instance in Morocco (32 years) [18], Ghana (33 years) [22], Senegal (23 years) [20], Gabon (36 years) [23], Cote d'Ivoire (28.8 years) [21], and Ethiopia (24.3 years) [26]. Main clinical findings often due to severe constriction include: hepatomegaly (74–100%), raised jugular venous pressure (76–100%), hepato-jugular reflux (67–100%), ascite (50–76%), peripheral edema (46–79%), complete "Pick Syndrome"(50–76%). Kussmaul sign is less detected (9.8%). In African setting, frequent radiographical findings at chest X-ray are as follows: enlarged cardiothoracic ratio or cardiomegaly (88–47%), calcifications (36–52.5%), and pleural effusion (44–63%); electrocardiography shows invariable modifications such as low QRS voltage (32.5–82.5%), atrial fibrillation (18–46%), and T wave abnormality up to 87.5%. Echocardiography is largely available and is useful for accurate assessment in revealing in most cases a thickened pericardium (56 and 100%), pericardial calcifications in 30.2%, 47.4% of cases according to authors and left ventricular septal motion abnormality (63.6%) with reduced ejection fraction below 0.60 in Rabat, Morocco [19]; cardiac catheterization performed only in a very few centers [21, 24, 25, 27] documents elevation and equalization of diastolic heart pressures with the typical dip-plateau waveform of constrictive pericarditis and evaluate the stroke volume, cardiac output, and myocardial systolic function. It still prevails to be the most final diagnostic assessment in sub-Saharan Africa. In his study, Yangni-Angate et al. [21] reported that cardiac catheterization confirmed a dip-and-plateau (square root sign), an equalization of end-diastolic pressures in right and/or left cardiac chambers ranged between 10 and 40 mmHg, a mean cardiac index (CI): 2.3 l/min/m^2 (extremes: 1.3–3.6). From this author, the constriction was limited to the right cardiac cavities called right constriction (n = 54, 45%) or to the right and left cardiac cavities called bilateral constriction (n = 66, 55%) and hemodynamic parameters and cineangiograms confirmed the diagnosis of pericardial constriction in all the patients. Omboga in Nairobi [25] showed the same observation with elevation of intracardial pressures in all cases, raised mean right atrium, end-diastolic right and left ventricular, and elevated mean pulmonary artery pressures at 18, 18, 20, and 27mmHg, respectively. Other imaging studies such as computed tomography and magnetic resonance imaging are rarely prescribed because they are inexistent usually. Those modern imaging techniques could be heavily useful in diagnosing constrictive pericarditis. When done, pericardial biopsy can be contributive for constrictive pericarditis etiology. Laboratory investigations regarding protein-losing enteropathy in patients with chronic constrictive pericarditis are not yet done for Africans. Differential diagnosis is always considered, and distinction from both constrictive pericarditis and restrictive cardiomyopathy due to endomyocardial fibrosis is really the usual situation to be clarified. Endomyocardial fibrosis (EMF) is a tropical heart disease with fibrous endocardial lesions lying in the right and/or the left ventricle. Endoventricular fibrosis is shown and confirmed in all cases by bi-dimensional echocardiography and angiocardiography [28].

7. Natural history

Constrictive pericarditis occurs mostly in our African context after a non-diagnosed, untreated tuberculous or pyogenic pericarditis or even as a sequel of a treated tuberculous pericarditis. Constrictive process starts with an acute pericarditis; then a subacute and chronic pericarditis marked in most of the cases by a fusion of the two layers of the pericardium and an occlusion of the pericardial cavity [3].

The delay from onset clinical symptoms to constriction is widely flexible from 1 month to 10 years even 25 years [2].

8. Surgical treatment

When diagnosis of constrictive pericarditis is confirmed and surgery is indicated, a pericardiectomy should not be delayed; surgery remains the only efficient and comprehensive treatment option. Pericardiectomy is frequently performed via a median sternotomy approach or a left anterolateral thoracotomy approach; it may be partial or complete. From the African teams' surgical experiences, cardiopulmonary bypass has not been used in all cases and excellent early surgical outcomes were reported.

9. Early outcomes and risk factors for early deaths

9.1 From cardiac centers in Africa

Yangni-Angate in Cote d'Ivoire [21], in his retrospective study related to 120 patients with CCP who underwent pericardiectomy through a median sternotomy approach (n = 117; 97.5%) found 15 early deaths (12.5%); the cause of hospital deaths was due to a low cardiac output (n = 12) and to a hepatic failure (n = 3). Class III or IV (NYHA) ($p = 0.01$), mitral regurgitation ($p < 0.05$), persistent a diastolic syndrome after surgery ($p < 0.05$) and low cardiac index ($p < 0.02$) were the important risk factors (**Table 1**). Age, size of cardiac X-ray silhouette, right and left ventricular diastolic pressures, ejection fraction, atrial fibrillation, and pericardial calcifications had no impact on early survival (**Table 2**).

Tettey in Ghana [22] reviewed the surgical management of constructive pericarditis and the post-operative challenges of 11 patients who had pericardiectomy via a median sternotomy in all patients with no early mortality and a significant improvement of functional capacity of all of the patients followed-up.

Mutyaba in South Africa [24] through a retrospective study of 121 patients who had undergone total (n = 105; 88.2%) or partial (n = 14; 11.8%) pericardiectomy for constrictive pericarditis at Groote Schuur Hospital, noted an early mortality of 14% (n = 14) mainly due to a low cardiac output syndrome. In this work, it has been statistically attested that serum sodium and pre-operative New York Heart Association Class IV versus combined Class I–III were independent predictors of early mortality. He also showed that early mortality after pericardiectomy was not influenced by HIV status and that of New York Heart Association Functional Class IV and hyponatremia were predictable factor for early mortality after pericardiectomy.

Ali in Ethiopia [26] has done a retrospective study at the Thoracic Surgical Unit, Tikur Anbessa Hospital, Department of Surgery, Medical Faculty, Addis Ababa University, Addis Ababa on 19 patients who underwent pericardiectomy for CCP by a median sternotomy approach (n = 15; 79%) often. One early post-operative mortality was registered. The author emphasized the benefit of pericardiectomy in terms of physical exercise improvement.

Ondo N'Dong in Gabon [23] has published his series on 18 patients with constrictive pericarditis treated surgically. All of them underwent a partial pericardiectomy via a left anterior thoracic incision in 17 patients and a median sternotomy incision in 1 patient. Four patients died in the early post-operative period due to low cardiac output; this study revealed pre-operative severe heart failure as a principal predictable risk factor for early death after pericardiectomy. This finding has been also noted In Rabat, Morocco by Nzondo [19]; in Fes, Morocco by Hind [18] and in Senegal by Ciss [20].

Nzondo [19] has retrospectively analyzed 11 patients who had undergone partial pericardiectomy via a median sternotomy approach for constrictive pericarditis. Early mortality was of 9.1% related to acute heart and multi-organs failure. Hind in

Author	Country	Study period	Cases (n)	Aetiology	Early deaths (%)	Causes of deaths	Early risk factors
Omboga et al. [25]	KENYA	1973–1981	51	TB (30%) Idiopathic (70%)	8.5	• Pulmonary embolism (n=3) • Massive haemorrhage (n=1)	–
Yangni–Angate et al. [21]	Cote d'Ivoire	1977–2012	120	TB (99%)	12.5	• Low cardiac output (n=12) • Hepatic failure (n=3)	• Class III or IV (NYHA) • Low cardiac index • Mitral regurgitation • Persistent diastolic post-operative syndrome
Ondo N'Dong et al. [23]	Gabon	1986–1999	18	TB (50%) Idiopathic (33%) Infection (17%)	22.2	Low cardiac output (n=4)	Class IV (NYHA)
Mutyaba et al. [24]	South Africa	1990–2012	121	TB (91%) Idiopathic (5%) Miscellaneous (4%)	14	Low cardiac output (n=11) Acute kidney injury (n=6)	Class IV (NYHA)
Zamani et al. [27]	Morocco	1994–2009	23	TB (43%) Idiopathic (57%)	17	Low cardiac output (n=2) Severe dysarythmia (n=1) Haemorrhage (n=1)	Class IV (NYHA)
Ali et al. [26]	Ethiopia	1996–2005	19	TB (31.6%) Infection (10.5%) Miscellaneous (67.9%)	5.3	–	–
Ciss et al. [20]	Senegal	1996–2008	32	TB (63.6%)	6.25	Low cardiac output (n=1)	Class IV (NYHA)
Nzondo et al. [19]	Morocco	1996–2010	11	TB (45.5%) Idiopathic (54.5%)	9.1	Low cardiac output (n=1)	Class IV (NYHA)
Tettey et al. [22]	Ghana	2000–2004	11	TB (63.6%)	0		
Hind et al. [18]	Morocco	2003–2013	43	TB (58%) Idiopathic (48%)	4.6	Low cardiac output (n=2)	Class IV (NYHA)

TB: Tuberculosis.

Table 1.
Surgical early results after pericardiectomy for chronic constrictive pericarditis.

Risk factors for immediate deaths	Alive (n = 105)		Deceased (n = 15)		P
	Average	Extremes	Average	Extremes	
Age (years)	30.4 ± 16.6	10–51	28.4 ± 10.1	8–46	0.09
CTR	0.55 ± 0.05	0.45–0.70	0.53 ± 0.3	0.50–0.59	0.34
RVEDP	20.6 ± 7.8	7–40	16.2 ± 10.3	15–40	0.12
LVEDP	20.1 ± 6.1	10–30	24.6 ± 7.7	16–35	0.07
EF	50.4 ± 16	31–67	54.3 ± 5	49–59	0.24
CI	2.42 ± 0.7	1.3–3.6	1.63 ± 0.2	1.4–2	0.02
WPAP	20.6 ± 9.9	10–40	25 ± 10.4	18–37	0.36
SPAP	27.3 ± 11.1	21–66	38.2 ± 17.9	21–66	0.08
Functional class NYHA III–IV	42/105		15/15		0.01
Atrial fibrillation	18/105		3/15		0.10
Calcifications	54/105		6/15		0.07
Mitral insufficiency	6/105		9/15		0.00
Persistence of post-operative constriction	9/105		5/15		0.00
Bilateral constriction	61/105		15/15		0.04

CTR, cardiothoracic ratio; RVEDP, right ventricle end-diastolic pressure; SPAP, systolic pulmonary arterial pressure; WPAP, wedged pulmonary artery pressure; LVEPD, left ventricle end-diastolic pressure; CI, cardiac index; EF, ejection fraction.

Table 2.
Risk Factors for immediate deaths after pericardiectomy for constrictive chronic pericarditis—from Yangni-Angate et al. study [21].

Fes, Morocco [18] on a study of 43 patients with constrictive pericarditis focused on 41 who had a partial pericardiectomy through a median sternotomy approach; hospital mortality was 4.6% (n = 2).

Finally, Ciss et al. [20] in their study on 32 patients with constrictive pericarditis undergoing partial pericardiectomy via a median sternotomy approach reported an early mortality of 6.25% in 2 patients out of 32 caused by hemodynamic and severe instability added to a poor NYHA functional class pre-operatively.

Omboga in Nairobi [25] in his study of 47 patients out of 51 who underwent surgery through median sternotomy (82.9%) and left lateral thoracic (17.1%) approaches has mentioned 8.5% of hospital mortality not due to acute failure as mentioned in previous studies in Africa but attributable to massive hemorrhage in the operative table (n = 1) and pulmonary embolism (n = 3) secondary to deep venous thrombosis.

None of African teams mentioned above used robotic approach; this minimally invasive modern approach seems to be less painful, more adequate for a complete release of the pericardial constriction [29].

9.2 From worldwide outside Africa

Compared to African series, where the most common etiology of CCP is TB, in developed world CCP etiology profile is different with idiopathic as most frequent. However, there is no significant difference among either surgical operative approach and hospital mortality or risk factors for early deaths. This fact is on the same line with McCaughan's consideration cited by Kirklin and Barratt-Boyes [2] who demonstrated in 1985 that most of early deaths are due to acute heart failure

and that of pre-operative NYHA class III and IV are significant risk factor for early death after pericardiectomy as noted in African surgical experiences. Chowdhury et al. [30] in a study including 338 patients (85.6%) who underwent total pericardiectomy (group I), and 57 patients (14.4%) undergoing partial pericardiectomy (group II), has demonstrated better perioperative and late mortality rates after total pericardiectomy; in addition, duration of symptoms, advanced functional class, partial pericardiectomy, pre-operative high right atrial pressure, hyperbilirubinemia, renal dysfunction, atrial fibrillation, pericardial calcification, thoracotomy approach, were significant risk factors for death.

In a recent study from Porta-Sanchez in Spain [31], 140 consecutive patients who underwent pericardiectomy for constrictive pericarditis over a 34-year period in a single center were analyzed. Most frequent etiology found was idiopathic in 76 patients (54%). Median sternotomy was done in all patients. Perioperative mortality was 11%. There was no difference in mortality between etiologies.

Mayo Clinic Experience with pericardiectomy for constrictive pericarditis over eight decades [32] related to 1071 pericardiectomies in 1066 individual patients. Patients were divided into two intervals: an historical (pre-1990) group (n = 259) and a contemporary (1990–2013) group (n = 807). This study showed a significant change in constrictive pericarditis etiology with a similar overall survival over decades and a significant decrease of 30-day mortality from 13.5% in the historical era to 5.2% in the contemporary era ($p < 0.001$). Another article from North America with no significant disparity is the Montreal Heart Institute Experience over a 20-year period [33]; it involved 99 consecutive patients with constrictive pericarditis; idiopathic was the most frequent etiology (61%) and hospital mortality 7.9% after isolated pericardiectomy.

Busha et al. [34] revealed an higher mortality death of 18.6% after pericardiectomy in 97 consecutive patients with constrictive pericarditis and no different risk factors for early death such as reduced left ventricular ejection fraction (LVEF) (35% < LVEF <55%) and right ventricular dilatation on multivariable analysis. He also found no difference in early mortality between patients with isolated pericardiectomy and those with concomitant surgery ($p = 0.62$).

10. Late outcomes

In African context, lack of substantial late outcomes after pericardiectomy is observed due to a significant number of patients lost to follow-up in general; however, in his series from Cote d'ivoire, Yangni-Angate [21], after an average follow-up of 4 years (extremes: 1–10 years), no late death was observed. A functional class I or II (NYHA) was mentioned in all survivors. Among them, those who underwent cardiac catheterization late post-operatively, a significant reduction even a normalization of the right and/or the left ventricular end-diastolic pressures, of the pulmonary capillary wedge pressure ($p < 0.05$) and of the right atrial pressure ($p < 0.05$) and absence of the dip-and-plateau after pericardiectomy were certified (**Table 3**).

Significant reduction of right atrial mean pressure from 17 ± 6 mmHg pre-operatively to 7.1 ± 4.2 mmHg after pericardiectomy for CCP has been also shown by an African team work published by Zamani in Rabat, Morrocco [27].

A similar experience as African teams series has been published by Bicer et al. [35] with a predominance of tuberculous constrictive pericarditis (48.8%); other etiologies were: idiopathic (31.9%), malignancy (6.4%), prior cardiac surgery (4.3%), non-tuberculosis bacterial infections (4.3%), radiotherapy (2.1%), uremia (2.1%), and post-traumatic (2.1%). They had performed pericardiectomy in all patients via a sternotomy approach with a very low early mortality of 2.1%, while

Hemodynamic measurements	Average		Extremes		P
	Preop.	Postop.	Preop.	Postop.	
RAP	16	7.4	10–36	5–10	0.04
RVEDP	21	10	7–40	5–15	0.02
SPAP	29	23	8–66	17–30	0.09
WPAP	21	14	10–40	9–19	0.00
LVEDP	21	13	10–35	4.5–20	0.02
CI	2.3	2.7	1.2–36	1.92–3.5	0.15

Significant ($P < 0.05$); Non-significant ($P \geq 0.05$); Preop., Pre-operative; Postop., Post-operative; RAP, right atrial pressure; RVEDP, right ventricle end-diastolic pressure; SPAP, systolic pulmonary arterial pressure; WPAP, wedged pulmonary artery pressure; LVEPD, left ventricle end-diastolic pressure; CI, cardiac index.

Table 3.
Comparison of hemodynamic measurements: pre-operative versus post-operative in patients who underwent pericardiectomy for chronic constrictive pericarditis—from Yangni-Angate et al. study [21].

late mortality was 23.4%, and actuarial survival rates at 1, 5, 10 years were 91, 85, and 81%, respectively. Those rates are closed to the Montreal Heart Institute experience [33] characterized by tolerable long-term clinical outcome: 79% of patients were in NYHA class I or II post-operatively with an overall survival rate of 87% at 5 years and 78% at 10 years. At 10 years survival, Bicer et al. [35] estimated rate was worse (64%) because of poor prognosis of pericardiectomy after constrictive pericarditis post-mediastinal irradiation.

In a 24-year experience on pericardiectomy in patients with constrictive pericarditis, based on Kaplan-Meier survival curves demonstration, Szaboa et al. [36] have confirmed poor prognosis of post-irradiation constrictive pericardiectomy and shown better and comparable long-term survival after in other etiologies as idiopathic tuberculosis myocardial infarction, and uremia; the author noted no survival after 5 years with post-radiation constrictive pericarditis; it is widely accepted radiation etiology is a negative factor affecting long-term survival results as well indicated by Nishimura in Japanese population [37] and Avgerinos et al. [38]. On his study related to 46 patients with a mean age of 59 years and various classic etiologies, Nishumuna et al. [37] have described the very high severity of radiation etiology with death within only 1 year after pericardiectomy confirming overall literature results.

Avgerinos et al. [38] reported his 36 patients study who underwent pericardiectomy for constrictive pericarditis over 15 years; he has no hospital mortality and 1-, 5-, 10-, and 15-year survival rates were 97.2, 94.6, 86.5, and 78.3%, respectively; he fund risk factors for increased long-term mortality such as: pre-operative heart failure, elevated pre-operative total bilirubin (>2.7 mg/dl, hazard ratio 6.8, $p = 0.02$), and elevated creatinine (>1.4 mg/dl, hazard ratio 3.1, $p = 0.05$). Subsequently, he demonstrated from Kaplan-Meier survival analysis a significant decrease in survival of all the patients with post-radiation etiology associated ($p = 0.05$) or with impaired cardiac, hepatic or renal dysfunction.

According to Porta-Sanchez et al. [31], from a Cox-regression analysis in his study, age at surgery, advanced New York Heart Association Functional Class (III–IV) and previous acute idiopathic pericarditis were associated with increased mortality during follow-up ranging from 0.1 to 33.0 years with a mean follow-up of 12 years.

Predictor factors of prognosis and mortality after pericardiectomy have been largely documented by many studies; one of them from Kang [39] has even stipulated that an echocardiographical measurement of higher early diastolic mitral

inflow velocity in predicting mortality after pericardiectomy was 71 cm/s (sensitivity of 84.6% and specificity of 52.2%); that value may also be useful in predicting late survival ($p = 0.029$).

Because of satisfactory long-term results, pericardiectomy can be considered as a safe and efficient technical procedure [40]; it can be achieved in most cases with minimal hospital mortality, post-operative functional class significant improvement and substantial reduction of heart diastolic pressures and absence of any recurrence if completely performed.

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