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Salvation to Hearts in Distress under Shells of Stress: Subtotal Pericardiectomy in a Series of 9 Patients with Constrictive Pericarditis

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ABSTRACT

Objective: Chronic constrictive pericarditis (CCP) is a rare entity responsible of diastolic heart failure. The true prevalence is yet to be defined. The purpose of this study was to describe the clinical and para-clinical characteristics of patients with CCP, the therapeutic management, the outcomes and impacting factors.

Materials and Methods: We conducted a retrospective descriptive study from 2017 to 2020 including 9 patients hospitalized for CCP in our cardiovascular surgery department.

Results: The mean age was of 32.6 years. Majority were men (n=7). Dyspnea was the most common sign. Peripheral signs were dominated by signs of right heart failure. Cardiac ultrasonography showed pericardial thickening and calcifications with Doppler adiastolic signs in 90% of cases. Thoracic CT was performed in 7 patients, cardiac MRI in one patient, showing calcifications and measuring the pericardial thickening. Cardiac catheterization performed in 6 patients showed the aspect of Dip plateau. Tuberculosis etiology was retained in 55.6%; post-radiation origin in one patient and 33.4 % of cases were idiopathic. All of patients benefited from subtotal pericardiectomy with good results in the medium and long term. Two deaths occurred, the first patient died following multi-visceral failure, the second died 3 years later from neoplasia.

Conclusion: The CCP is a rare condition with poor prognosis. The diagnosis should be raised when there are signs of right heart failure associated with signs of hemodynamic adiastolia. The echocardiography, with computed tomography or cardiac MRI and especially cardiac catheterization confirm the diagnosis and also etiological orientation. Tuberculosis and idiopathic etiologies were the most common at our country. Medical treatment options are limited. Pericardiectomy remains the only radical treatment with good results in immediate, medium and long term.

KEYWORDS: Chronic Constrictive Pericarditis, Adiastolia – Dyspnea, Tuberculosis – Pericardiectomy, Survival.

INTRODUCTION

Chronic constrictive pericarditis (CCP) is a rare but serious disease with a dark prognosis in the absence of surgical treatment. It results from inflammation and fibrosis of the pericardium, ultimately leading to impairment of diastolic filling and right heart failure (1).

Generally, constrictive pericarditis is irreversible; the pericardium becomes a rigid inextensible shell, fibro-calcareous, hindering the diastolic expansion of the heart, defining adiastolia (2). This practically affects always both ventricles, increasing interventricular interdependence and resulting in equalization of the right and left filling pressures (3)

Currently, we recognize more of subacute forms with effusion due to earlier diagnosis. Tuberculosis remains a common cause of CCP in developing countries; Unlike the Western world where post-surgical, post-radiotherapic, neoplastic, viral and especially idiopathic become the main etiologies (4).

Although obtaining a history and physical examination are still the cornerstone of assessment, Imaging advances have made it easier to diagnosis, especially with the proper use of echocardiography doppler, high resolution tomography, magnetic resonance imaging (MRI), and specially the invasive hemodynamic measurements (5) which has been a very useful tool to confirm the diagnosis.

Medical treatment is only palliative; it helps to prepare patients for surgery which consists on a subtotal pericardectomy. The prognosis depends mainly on the precocity and success of it (6).

Through our study on CCP, based on 9 clinical files, we will describe the epidemiological, clinical, paraclinical, etiological characteristics, the course and treatment aspects of each medical file collected in cardiovascular surgery department at Mohamed V training military Hospital of Rabat then compared to data from the literature.

MATERIALS AND METHODS

Patients:

We performed a retrospective descriptive single-center study conducted in cardiovascular surgery department at Mohamed V training military hospital of Rabat, for a period of Three years from January 2017 to December 2020, involving nine cases of chronic constrictive pericarditis. Clinical characteristics, para-clinic data, indications, therapeutic means and methods, as well as results of treatment were collected and analyzed.

Inclusion criteria:

There were included in our study:

-All operated patients for chronic constrictive pericarditis confirmed by clinical, para-clinical and surgical data in that period.

Exclusion criteria:

The following were excluded from our study:

-Patients with pericardial effusion without constriction and active purulent pericarditis;

-Patients who have undergone a pericardectomy associated with a cure for another heart disease.

The statistical analyses:

Data analysis was performed using the SPSS 19.0 (Statistical software package of social science; SPSS 19.0, Chicago, Illinois, USA).

Continuous variables were expressed as mean, as frequencies and percentage for categorical variables.

RESULTS

We exploited nine files of patients with CCP, we found results as follow:

Baseline Characteristics: (Table I)

The mean age of patients was 37,7±9,8 year-old. 78% were men with a sex ratio of 3.5. All of our patients were symptomatic. Dyspnea (66.67 %) was the most common sign, followed by the edema-ascitic syndrome 44.4 % (4 patients), chest pain 44.4% (4 patients), and hepatalgia at 44.4 % (4 patients). Four of our patients (45%) were on active smoking.

The screening for a personal medical and surgical history related to the occurrence of chronic constrictive pericarditis objectified the results showed in table 1 where tuberculosis was the common cause 55.56%; among patients with a history of tuberculosis, two patients were treated for pulmonary tuberculosis, two for recurrent tuberculosis pericardial tamponade (surgically drained) and one for tuberculosis lymph node.

67% of Patients had a preserved general condition, with an average body mass of 22.67±3.55kg/m²; perioral cyanosis was present in one patient. The peripheral signs were dominated by the turgescence of jugular veins (100%), hepatomegaly (89%) and peripheral edema at almost all patients. On cardiac examination, we found mostly tachycardia (55.5%), deafening of heart sounds (44.4%) and pericardial vibration (33.34%)

Characteristics	***Total (n=9)***
Mean Age (years)	37,7±9,8
Sex:	
Male	7 (78%)
Female	2 (22%)
Body mass index (kg/m²)	22.67±3.55
Antecedents:	
TBK	5 (55.56%)
Acute Viral pericarditis	1 (11.11%)
Tamponade	1 (11.11%)
Heart Failure	1 (11.11%)
Cardiac Traumatism / Thoracic surgery	0 / 0 1 (11.11%)

Thoracic irradiation	
Clinical features:	
NYHA II	4 (44.4%)
III	3 (33.3%)
IV	2 (22.2%)
Chest pain	4 (44.4%)
Edema-ascitic syndrome	4 (44.4%)
Hepatalgia	8 (88.89%)
Febricule	3 (33.3%)
Weight loss	3 (33.3%)
Examination Data:	
Tachycardia	5 (55.56%)
Gallop	4 (44.4%)
cyanosis	1 (11.1%)
Deafening of heart sounds	4 (44.4%)
Pericardial friction	2 (22.2%)
Pericardialvibrance	3 (33.3%)
Paradoxical pulse	0
Spontaneous turgor of the jugular veins	9 (100%) 9 (100%)
Edema of the lower limbs	1 (11.11%)
Collateral venous circulation	4 (44.4%)
Ascites	4 (44.4%)
Harzer sign	

Table I: Baseline characteristics of patients of the serie:
NYHA: New York Heart Association classification
TBK: tuberculosis

Paraclinic characteristics: (table II)

Pericardial thickening	4 (44.4%)
Pericardial Calcifications	5 (55.56%)
Pericardial Effusion	2 (22.2%)
Dilated right cavities	8 (88.89%)
Septal Bounce	7 (77.78%)
Respiratory variations in	
Mitral flow	9 (100%)
Tricuspide flow	9 (100%)
Dilation of the Hepatic veins and inferior vena cava	9 (100%) 4 (44.4%)
Minimal Mitral regurgitation	8 (88.89%)
Minimal Tricuspide regurgitation	8 (88.89%)
Dip plateau in PI flow	
Left Ventricle ejection fraction	1 (11.11%)
LVEF < 50%	8 (88.89%)
LVEF ≥ 50%	9 (100%)
LA dilation	9 (100%)
S/D < 1 (pulmonary venous flow)	
CT Scan	7 (77.78%)
Cardiac MRI	1 (11.11%)
Cardiac catheterization (*preoperative data):*	6 (66.67%) 18±4
*RA Pressure (mmHg)	25±10
*RV pressure (mmHg)	26±12
*PAP (mmHg)	22±6
*CP (mmHg)	

Table II: Paraclinical data of patients
*RA: right Atrium, LA: Left atrium, RV: Right ventricle, PAP: Pulmonary arterial pressure, CP: capillary pressure, PI flow: pulmonary insufficiency flow.

ECG showed 6 patients had sinus rhythm (66.67%) of which 4 had sinus Tachycardia, 5 had microvoltage (55.56%), 4 Hypertrophy of right ventricle (44.45%) and 3 patients were in atrial fibrillation (33.34%).
Chest x ray has objectified cardiomegaly in 55.56% of patients and pericardial calcifications (Figure 1) in 55.56% of cases.

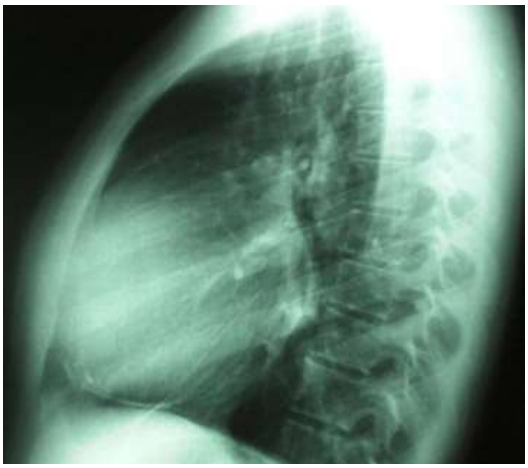

Figure 1: A profile Chest X ray showingpericardial calcifications

Pulsed and continuous Doppler echocardiography has the advantage of being non-invasive and provided hemodynamic data. The echographic signs findings are summarized in Table II.
All of our patients had pericardial anomaly represented by pericardial thickening (44.45%) and pericardial calcifications of 55.5% (figure 2).

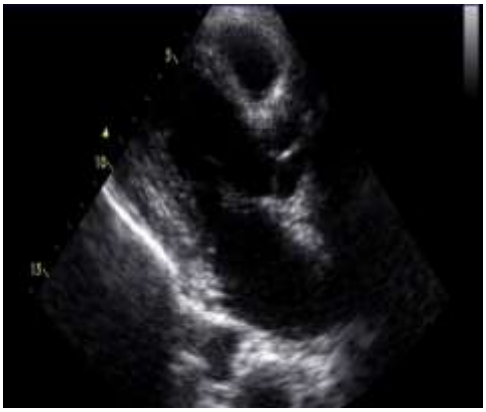

Figure 2 showing calcified pericardium
On a long-axis parasternal section

Almost all had dilation of their right cavities (88.87%) with septal bounce (77.7%) as shown in (figure 3).

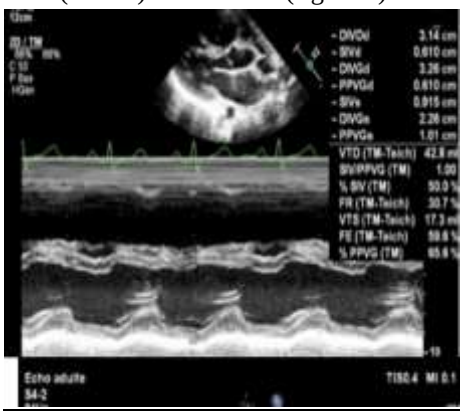

Figure 3: Showing the septal bounce in motion time mode

Aspect of dip plateau (figure 4) on the flow of pulmonary insufficiency was found in 89% of the cases; significant mitral and tricuspid respiratory variations (figure 5) a restrictive profile on tricuspid flow was found in all cases with a dilated inferior vena cava and dilated hepatic veins with little or no inspiratory compliance (figure 6). In all patients, the left atrium was dilated (average surface of 28.8 cm^2) with a wide and long A wave and reduced S wave on the pulmonary venous flow.

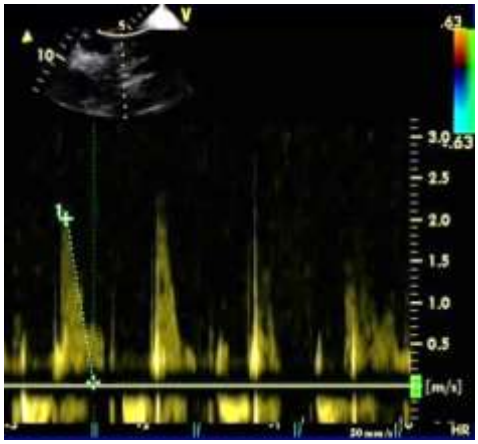

Figure 4. Showing aspect of Dip plateau on Pulmonary insufficiency flow

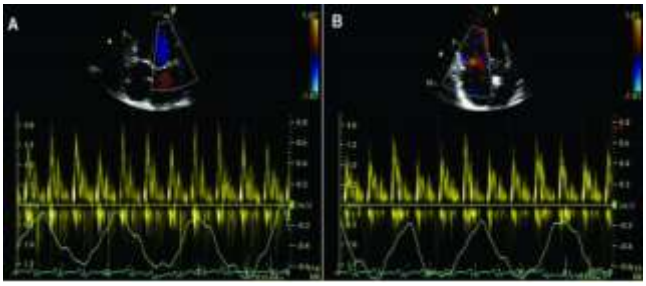

Figure 5: Respiratory variation of transmitral (A) and tricuspid (B) doppler flow

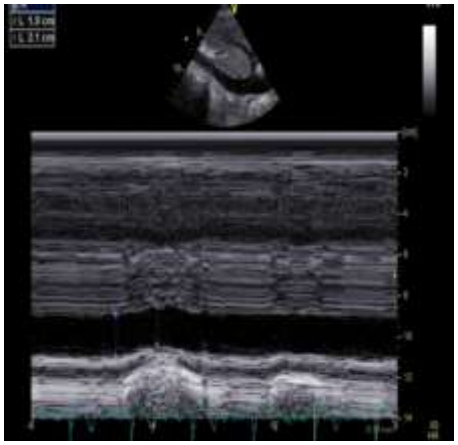

Figure 6: showing dilated inferior vena cava with a very poor compliance

Cardiac catheterization was performed in 6 of our patients, the pathognomonic signs of adiastolia and constriction were present; among which:

- Rise in the pressures of the right cavities,
- Equalization of end diastolic pressures of the cardiac chambers,
- Dip-plateau aspect of the pressure curve of the right ventricle,

The chest CT scan was performed in 7 patients and demonstrated pericardial calcifications with thickening (figure 7).

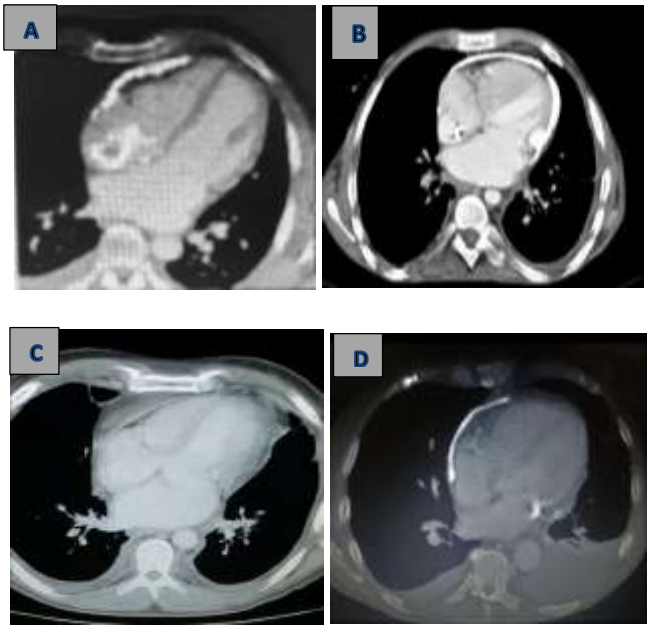

Figure 7: showing aspects in CT scan of a calcified (A+B) and Thickened (C) pericardium, also individualized in MRI (D) in patients of our series:

MRI was performed in one patient and showed pericardial thickening with dilated right cavities. (figure 7- image D)

All our patients had, in addition to the standard blood test (blood count, blood ionogram), and liver tests that showed a

spontaneously low Prothrombin level (52%) in half of our patients with moderate hepatic cytolysis; the mean blood creatinine was 10.8mg/l with normal potassium levels, the mean hemoglobin level was 12.9g / dL with a mean hematocrit of 39.9% and a mean C-reactive protein at 13.3mg/L. An Intradermal reaction (IDR) to tuberculin was performed with repeated Koch's Bacillus (BK) research in sputum as part of the etiological assessment. 45.5% had a positive reaction to IDR, with negative culture in the cytobacteriological examination of the sputum. An additive chemical and cytobacteriological study of ascites and pleural fluid was performed to guide the etiological diagnosis, showing in most cases a transudate.

The etiology (table III) that has been found in our series was tuberculosis at a rate of 55.6 %. One patient had a history of chest irradiation for breast neoplasia. The others three whose etiology was not determined were considered idiopathic 33.34%.

Etiology of Pericardial Constriction:	***Total (n=9)***
Tuberculosis	55.56%
Post-Operative	0
Post-Radiation	11.1%
Idiopathic	33.34%

Table III: Etiologies of chronic constrictive pericarditis

Medical treatment:

Symptomatic medical treatment based on Diuretics and potassium has been prescribed preoperatively to all of our patients to relieve the functional symptomatology; anti-tuberculosis treatment was started preoperatively in five patients (55.5%) and received: Isoniazid, Rifampicin, Pyrazinamide, Ethambuthol, and sometimes streptomycin, which was continued postoperatively.

Surgical treatment:

All patients underwent a median sternotomy through which was performed a subtotal pericardectomy involving the entire anterior surface of the heart going from the phrenic nerve to the other, then the decortication is extended upwards towards the vessels of the base and downwards to the diaphragmatic surface and to the inferior vena cava as shown in figure 8 through different operative views (A, B, C and D) individualizing the thickened pericardium with or without calcifications and the ongoing procedure. Monitoring by Swan Ganz was carried out in 88% of patients. The use of cardiopulmonary bypass has never been necessary. This decortication allowed a good diastolic re-expansion of all cardiac chambers specially ventricles. There was an improvement in hemodynamics particularly the systolic blood pressure.

The average duration of the intervention was around 154 ±20 min and an average intensive care stay of 2.5 ± 1.4 days. The average duration of hospital stay was around 21 ± 4 days.

The fragments of removed pericardium were sent to anatomo-pathological analyze with screening for Koch Bacillus and culture on Lowenstein-Jensen media. In all cases, histological analysis revealed the presence of non-specified calcified fibrous pachypericarditis. Despite these results, antibacillary treatment was continued postoperatively in all of our patients with total duration of 09 to 12 months.

Post-operative follow-up:

The postoperative period was uneventful in 78 % of cases with improvement of dyspnea, as well as significant decrease in the pressure of the right atrium (from 18±4 mmhg preoperatively to 6.9±4.1mmhg postoperatively).The syndrome of low cardiac output occurred in 2 patients (22.2%) requiring pharmacological assistance, and tachyarrhythmia in 4 patients (44.45%).

Early and Overall Mortality:

Hospital mortality was of 22.2% (two patients). The first patient death occurred early at following multi-visceral failure with refractory cardiogenic shock and renal failure. The second died 3 years later from neoplasia.

The mean follow-up was of 48 months. Over this period, 06 patients are still asymptomatic and reported no recurrent symptoms, one patient was lost to follow-up and one patient as mentioned before, died 3 years later of a cancer.

DISCUSSION

Constrictive pericarditis results from inflammation and fibrosis of the pericardium, evolving into diastolic filling abnormalities and right heart failure [1]. Once the diagnosis is made, pericardiectomy is indicated in patients presented with symptoms of heart failure, especially those requiring increasing doses of diuretics [2].

CCP is a rare disease. Its frequency is about 0.5 to 2% of all heart diseases [6], and its incidence is about 2.4 cases per year [7]. In our series the incidence was of 2.9 cases per year. It is essentially a disease of the middle age, occurring in the end of the fifth or sixth decade [8], as shown in two of the largest studies on CCP with mean ages of 55 years [9] and 56 years (10). In our study, we find a younger age of patients with an average of 32.6 years, lower than those of these two studies, but similar to those found by NILGUN (32.2 years) [11] and TETTEY (33 years)) [12]. This difference in mean age could be explained by the predominance of tuberculous or idiopathic CCP in young subjects, unlike other etiologies found

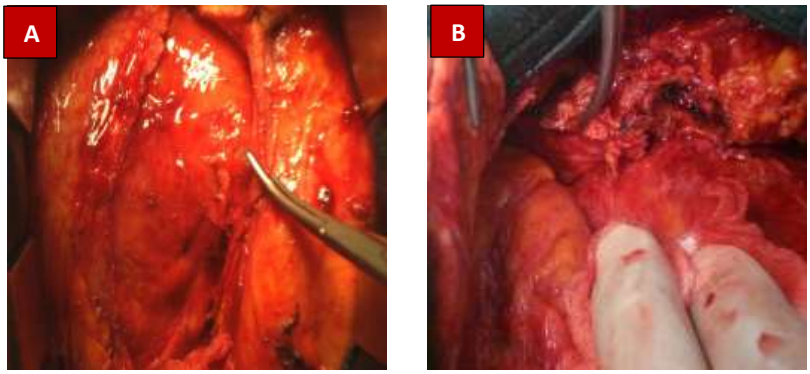

Figure 8: Operative view (A) showing Thickened pericardium and (B) showing localized thickened and calcified Pericardium in patients of our series.

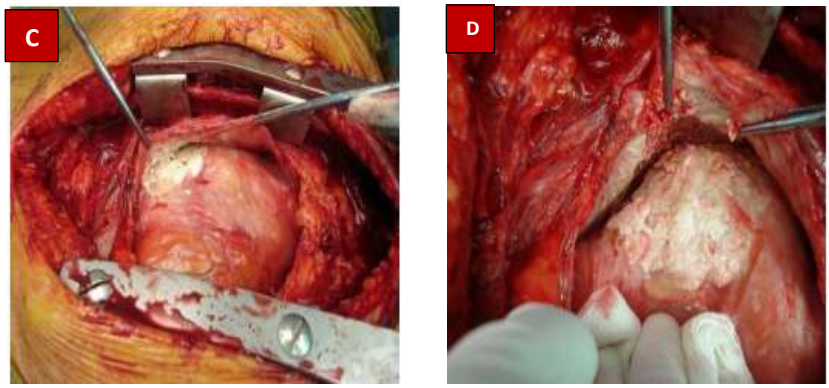

Figure 8: Operative views (C+D) showing the ongoing Partial pericardial resection by median sternotomy in one patient. Note the presence of pericardial calcifications.

In the literature, particularly the post-surgical and post-radiation, which are more common in the elderly.

CCP is a classic etiology of right heart failure. The fibrotic and calcified pericardium is thickened and isolates the heart chambers within the thorax, modifying their response to respiratory variations. The intra-pericardial heart volume remains fixed. There is also a ventricular filling disorder which is rapidly limited, the ventricular diastolic pressure, suddenly increasing, is classically equal to one third of the value of the systolic pressure. It is the phenomenon of adiastolia that is responsible for the dip-plateau on catheterization curves. It has two consequences: firstly, during inspiration, while the left ventricular pressure remains constant, the pressure in the pulmonary veins decreases, resulting in a decrease in left ventricular filling; on the other hand, the right venous return increases and so does the filling of the right ventricle. Since the cardiac volume is constant, it results in a ventricular interdependence which results in a septal dyskinesia, the increase of the filling of the right ventricle at the expense of the left ventricle, being thus dependent on the respiratory cycle. Finally, it is during diastole, when the ventricular volumes are at their maximum, that venous return is made difficult and the pressure in the different cavities is equalized [6, 16]

The functional symptoms is dominated by an exertional dyspnea [5] it was found in 44% of patients in HALEY [13]. In our series, 66.67% of patients were in NYHA class II and III. The other functional signs found in our series are dominated by weight loss, chest pain and signs of tuberculous impregnation: asthenia, night sweats and fever. These same accompanying signs are found in the literature [12, 13].

On clinical examination, the classic PICK syndrome is rarely complete [6, 14]. The most commonly described peripheral physical signs in literature, at frequencies of 80 to 98% [17], are those found in our study: hepatomegaly, spontaneous turgor of the jugular veins and hepato-jugular reflux. Ascites, edema of the lower limbs and pleural effusions are also found, at lower frequencies.

Tachycardia (55.5%), deafening of heart sounds (44.45%), pericardial friction and pericardial "vibration" (S3) was found in 33.34% of our patients which reflects the diastolic plateau that appears earlier if the constriction is severe [1]; S3 was observed in 47% of Mayo Clinic patients, while 16% had pericardial friction [9]. In the Stamford series [18], only 5% of patients had pericardial vibration and 4% had friction.

The sign of KUSSMAUL [19] as well as the paradoxical pulse were not found in our patients, unlike LING and Al [9] who detected the presence of this sign in 23% and paradoxical pulse in 19% of their patients who underwent pericardectomy for CCP. Cases of pericardial constrictions without paradoxical pulse have been explained by the fact that the

stiffed pericardium isolates the heart from the effects of respiration [20, 21].

ECG may show sinus tachycardia or nonspecific ST-segment and T-wave changes. A minority of patients with constriction have been reported to have low voltage or atrial fibrillation. [6, 16].

The chest X-ray objectifies the presence of eggshell calcifications, better visualized in profile incidence. It was found in 77.8% of our patients. Same results was reported by others authors [10, 22, 33]. Cardiomegaly, pulmonary vascular congestion and pleural effusion are nonspecific (6, 15, 24).

The transthoracic echocardioghraphy (TTE) has acquired real expertise in this area. It can reproduce the hemodynamic data of cardiac catheterization by visualizing a dip-plateau on the pulmonary insufficiency flow; also it shows non-invasively the respiratory variations to which the ventricular filling flows are subjected. It was performed in all of our patients. In fact, the finding of pericardial thickening, effusion, calcifications, dilation of right cavities, the inferior vena cava and supra hepatic veins and septal bounce have been very evocative of CCP for us. These same echocardiographic results are found in patients with CCP in several studies [16, 25]. In a study at the Mayo Clinic, increased pericardial thickness was observed in 37% of patients, abnormal septal movement in 43%, and atrial enlargement in 61% [9]. Cardiac Doppler suggests the diagnosis of CCP by showing the hemodynamic picture of adiastolia, with a decrease in proto-diastolic mitral flow (E wave) on inspiration while at the tricuspid level, the reverse phenomenon occurs [26]. This sign is important in cases where the decrease in the height of the mitral E wave is at least of 25%. It is highly specific of constriction of the heart [2]. HANCOCK [27] describes a sensitivity and specificity between 85 and 90%.

Chest computed tomography (CT) scan was performed in 77.8% of our patients; it revealed pericardial thickening and calcifications. One patient underwent cardiac MRI. In the series by MAHDHAOUI and colleagues [14], thoracic CT scan was performed in 50% of patients; it revealed thickening and pericardial calcifications, dilation of the inferior vena cava; MRI was done in 10% of their patients and confirmed the findings of the CT scan. However, CT scan and MRI do not allow a dynamic study like echocardiography; but still confirm the pachy-pericarditis and provide in particular a precise measurement of pericardial thickness and appreciate the degree of severity of the constriction.

In addition, cardiac MRI allows visualizing a very localized thickness of the pericardium (> 4 mm), immobile regardless the time of the cardiac cycle, associated to a dilation of the vena cava, tubular aspect of the right ventricle, paradoxical septum and dilation of the atria [3]. Certain dynamic sequences, in particular those using the "tagging" technique, visualize precisely the contraction of the myocardium; in case of CCP, the epicardium is not very mobile, adhering to the pericardium, indicating the diagnosis of epicarditis [6, 28]. This fibrosis extended to the myocardium is confirmed by the persistence of a late enhancement localized to the epicardium ten minutes after the injection of contrast product. This situation explain postoperative myocardial failure after decortication.

Cardiac catheterization was performed in our series for diagnostic confirmation, considered necessary for patients in whom diagnosis could not be made based on clinical presentation and typical characteristics of non-invasive examinations. NISHIMURA [26] reported that the greatest interest of catheterization in the diagnosis of pericardial constriction was the dynamic respiratory variations between the right and left ventricular pressure curves. TALREJA and al reported a sensitivity of 90% and a predictive value of CCP of 100% [29]. Out of 143 patients of the Mayo Clinic [30] who were operated on for CCP, 78 patients (54.5%) had undergone cardiac catheterization. In our series, 6 patients (66.67%) had undergone it. The hemodynamic changes found in our patients are as those obtained in the series by MAC CAUGHAN and Al [31] and KIRKLIN and Al [32] which were the elevation of the tele-diastolic pressures of the right cavities, the equalization of the right and left pressures and the characteristic and constant dip-plateau aspect.

However, the other causes of end-diastolic pressure equalization should always be eliminated before the diagnosis of pericardial constriction is retained such as: tamponade, restrictive cardiomyopathy, a dilated cardiomyopathy seen at late stage, defect of interatrial septum, chronic restrictive pulmonary disease, pneumothorax, a right atrial myxoma, tricuspid valve dysfunction should also be ruled out [25]. Nephropathies and nephrotic syndrome, superior vena cava obstruction, cirrhosis and liver disease, as well as abdominal tumors should be considered in differential diagnoses [4].

The etiology of CCP has changed considerably over the past decades in the Western world [33]. In developing countries tuberculosis remains the main cause of constrictive pericarditis [34, 35, 36]. This is shown by the results of the different series carried out in India [37], Gabon [38], and Ghana [12] with rates respectively at 40%, 39%, 63%. Tuberculous pericarditis would affect 1 to 2% of patients with tuberculosis, by direct extension of the mediastinal lymph nodes and, occasionally by hematogenous or contiguous dissemination from the myocardium [35]. In our country, tuberculosis was responsible for 55.6% of CCP in our series. The same results was already obtained in a previous study carried out at the Military Hospital of Rabat over a period of fifteen years from July 1994 to November 2009 that found tuberculous etiology in 43% of cases [39].

Today, the most common causes of the disease in developed countries are cardiac surgery, particularly coronary bypass surgery, idiopathic pericarditis, and mediastinal radiotherapy

[36]. LING and Al showed that open heart surgery and chest irradiation appeared to be the most common causes of heart constriction in wealthy countries [9]. In our study, no patient had a history of heart surgery, one patient had a history of chest radiotherapy; The idiopathic etiology, frequent in both West and developing countries, would correspond for some authors to old viral pericarditis for which infectious proof has not been made [6].

While the episodes of acute decompensation require symptomatic treatment based on the use of digitalo-diuretics and punctures to evacuate any effusions, in PCC the effective treatment is surgical requires pericardectomy. Anti-tuberculosis therapy is started for a period of six to nine months when this etiology is confirmed or suspected in the presence of an evocative context [6, 40]. It takes effect two weeks to one month before the intervention [41]. With a non-negligible intraoperative mortality rate at around 6%, surgery nevertheless results in a favorable outcome in the majority of cases, central venous pressure returning to normal within one month [28].

However, in very advanced situations, there may be an extension to myocardium leading to myocardial fibrosis, modifying its properties and explaining poorer results characterized by low cardiac output [6, 42].

The extent of pericardial decortication remains debated [31, 43, 44]. All of our patients had a subtotal pericardectomy through a median sternotomy without cardiopulmonary bypass, resecting the entire pericardium on both sides of both phrenic nerves; with excellent results in improving the functional status of our patients and reducing perioperative mortality. This was the same observation in the series of TETTEY [12] who, like our team, chose subtotal pericardectomy by median sternotomy. Although CULLIFORT and al [45] advocated that delayed clinical improvement and the persistence of constriction symptoms are most often the results of incomplete decortications. He associated radical pericardectomy with good postoperative results [45] Same as CHOWDHURY and Al in their study between 1985 and 2005 [46] who demonstrated the advantage of total resection over partial, and concluded that it was associated with lower perioperative mortality, reduction in postoperative low-cardiac output syndrome, shorter hospital stay and better long-term survival.

The outcome was favorable; more than 80% of patients presented a marked improvement in their symptoms [6]. The short, medium and long term functional results are satisfactory [47] except in case of incomplete intervention with recurrence of the constriction [6, 8] and in patients operated on at a late stage of the disease [51], the associated myocardial involvement remains a prognostic element since it is often irreversible [31].

Perioperative mortality was of 11.1% in our study (one patient). These results are close to those of many recent series which report a rate between 5 and 9.1% [42, 43, 46, 48]. The causes of hospital mortality are due in 75% of cases to acute heart failure [9, 31]; then hemorrhage and respiratory failure. In our patient, it was due to multi-visceral failure with refractory cardiogenic shock.

Long-term survival appears to be impacted by the etiology of CCP. In fact, the survival of patients with post-surgery CCP is better than that of post-chest radiotherapy, and lower than that of idiopathic CCP. In addition to etiology, advanced age, severe systolic left ventricular dysfunction, pulmonary arterial hypertension, preoperative NYHA stage III and IV, hepatic damage, renal failure and hyponatremia are considered independent predictors of overall survival [9, 10, 22].

LIMITATIONS OF THE STUDY

There are limits to our study. First, there could be a biased selection of patients due to the retrospective nature of the study, given the small number of our patients and their mode of recruitment. In addition, the distribution of etiologies is specific to our institution. Thus, it would be difficult to extrapolate our results to those of other teams. In fact, our study could be of a use to pave the way for other work with a more large number of patients, on national level and even in Africa.

CONCLUSION

In an appealing context such a right heart failure, the diagnosis of CCP must be quickly evoked. The presence of calcifications in chest X-ray and TTE especially, often makes it easy to confirm the diagnosis by underlining the hemodynamic abnormalities. Computed tomography or cardiac MRI and Cardiac catheterization are elements of interest in front of a less caricatural clinical presentation, particularly in the absence of calcifications or in a patient with lower echogenicity.

Tuberculosis is still an important etiology evolving into pericardial constriction in our country and in developing countries, unlike the Western world where post cardiac surgery constriction occupies the first place.

Medical treatment is purely symptomatic, most often associated with antibacillary treatment for confirmed or suspected tuberculosis etiologies. Subtotal pericardectomy through median sternotomy has been our treatment strategy, with good results in the short, medium and long term.

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