Surgical Treatment of Constrictive Pericarditis

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Summary

A review of 20 cases of Chronic Constrictive Pericarditis is presented in this paper. They were operated upon in Cardiothoraeic Surgical Unit, PGMI, Lady Reading Hospital, Peshawar. Age ranged from 14 years to 65 years, with mean age of 36.8 years. 14 were males and 6 females. Perioperative mortality was 10%. Most of them were due to in NYHA - class III & IV. All of them were due to tuberculosis. Post-operatively all of them were in NYHA- Class II or I.

Introduction

Chronic constrictive pericarditis (C.C.P.) is a chronic inflammatory process that involves both layers of the pericardium leading to pericardial thickening and thus causing constriction of the ventricles. There is thus reduced diastolic filling and reduced cardiac output. In our experience it was almost always due to tuberculosis though there are other causes as well. All the patients had severe symptoms and signs including fatique, dysponea on exertion and even at rest. They had raised JVP, bilateral pleural effusions,

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hepatomegaly, ascites and sometimes edema feet. Most of these patients were in NYHA class - III & IV.

Patients and Methods

A total of 20 patients of constrictive pericarditis have been operated from December 1989 to September 1991. All of them had pericardiectomy. Age ranged from 14 years to 65 years with an average age of 36.8% years. Sex ratio was 14 males and 6 females. All these were severely symptomatic. Some of them could not get up from the bed (NYHA Class-IV). These patients had massive asciites and bilateral pleural effusions. Those patients who were able to walk, could not perform their duties.

Investigations

Apart from complete blood, urea, electrolytes, blood grouping and cross-matching, Chest X-ray was most useful. It showed bilateral pleural effusion and sometimes calcified pericardium. Echocardiography was performed in all these cases and most of the times it would give diagnosis. In doubtful cases cardiac catheterization was done.

There were some changes in the ECG: ST segment depression and low voltage QRS complexes.

None of the cases had CT scan done.

Surgical Approach

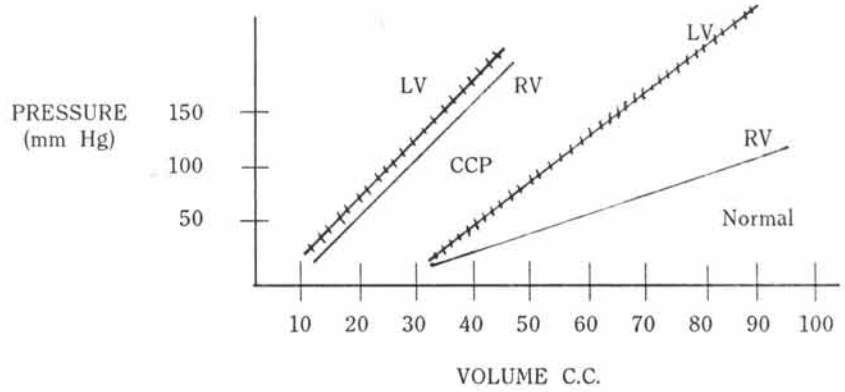
A mid-line sternotomy was performed in all the cases for C.C.P. 3-4 units of blood was arranged. Pericardium was found to be extremely thick. Both visceral and parietal pericardial layers were thick and would restrict the heart. Both pleurae and peritoneum were opened to suck out effusions and ascites: this enabled the anaesthetist to do better ventilation. First the pericardium from the left ventricular side was released. If it is done vice versa, then the lungs get flooded and there is danger of pulmonary odema. Both the phrenic nerves were preserved. Then the pericardium from pulmonary

artery, inferior vena cava and superior vena cava was excised until heart was found to be beating nicely. Sometimes there is danger of the tear specially (R) atrial appendage which is sutured with prolene. The diaphragmatic surface of the heart was also freed. The patients were not given too much fluids.

Two chest tubes were used. Sternum was closed with stainless steel wires and all of them spent 2-3 days in I.C.U.

Discussion

(a) *Pathophysiology*: The pathophysiology of C.C.P. has been debated over half a century. Burwell and colleagues[1] suggested that it is the constriction of both left and right ventricles which leads to C.C.P. Isaac-et-all[2] demonstrated in dogs that a change in the volume elasticity curve of the two ventricles resulted from experimentally - produced C.C.P.



In C.C.P. there is reduction of ventricular diastolic distensibility leading to reduced ventricular filling thus causing reduced stroke volume and reduced compliance.

Morphology

Pericardium has two layers, parietal and visceral. Both layers are involved in the process. When the layers are free there is usually some fluid between them. In advanced pathology both layers fuse together leading to constriction. The heart is thus encased in thick fibrous sheat which is solid and calcified. More importantly, it leads to myocardial atrophy. Thus very sick patients would still be in failure even after successful pericardiectomy but their symptoms are much better controlled medically after pericardiectomy.

History

Gallen described C.C.P. in 160 AD in animals. Lower[3] described it in human beings in 1669. Delorme in 1889 and Weil in 1895 suggested pericardiectomy for C.C.P.[4].

The causes are:

1. Tuberculosis
2. Idiopathic
3. Post-traumatic
4. Post-cardiac surgery
5. Post-radiation
6. Rhematoid disease and sarcoidosis

Results

Two patients died within 30 days of operation. The mortality was thus 10%. One of the two died on 3rd post-op. day due to severe C.C.P. The other died in strange circumstances. He was depressed post-op. He disappeared from the ward on the 10th night post-op. and was found dead in the water tank of the Hospital.

A retospective study from Mayo clinic of 231 patients from 1836 to 1983 shows 32 deaths (14%). The mortality depends on the state of NYHA class:

NYHA I or II	0.5%
NYHA III	10%
NYHA IV	46%

Conclusion

1. All patients with C.C.P. need pericardiectomy

2. The mortality increases with higher NYHA class.

3. It is not only the constriction but myocardial atrophy as well which leads to sick patients.

References

1. Burwell CS Constrictive Pericarditis. Circulation. 1957, 15, 161.

2. Issacs, J.P. The Pathologic Physiology of Constrictive Pericarditis. Bull Jobus Hopkanin Hosp. 1952, 90, 259.

3. Lower, 1969, 104-107.

4. Delorme, E. 1889, 24, 918.