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CHRONIC CONSTRICTIVE PERICARDITIS.

(A STUDY OF 51 CASES AT KENYATTA NATIONAL HOSPITAL(KNH)

BY

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Dr. Peter Nyagonchong'a Omboga. MBCh.B.

SUPERVISOR:

This dissertation has been submitted for examination with my approval as University Supervisor.



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S U M M A R Y

Chronic constrictive pericarditis is easily confused with other diseases, and the condition may be missed for periods of anything from many months to many years.

In our environment it is still a relatively common problem among cardiac lesions. In the years 1975 to 1980 a total of 2178 cases of cardiac diseases were seen at Kenyatta National Hospital. These included endocardial, myocardial and pericardial diseases. Out of these 135(6.10%) were of pericardial origin. In the same period 330 patients underwent cardiac surgery. Forty seven (14.24%), of these had chronic constrictive pericarditis for which they had pericardectomy done.

Like most cardiac lesions, the incidence of chronic constrictive pericarditis can be reduced appreciably by preventive measures. Once established however, the only treatment is surgical release of the constriction.

The study of chronic constrictive pericarditis in our country should encompass both epidemiological and pathological dimensions.

It is however possible to focus attention on modes and results of treatment and therefrom derive a very important insight into this disease whose early diagnosis and timely surgical treatment gives excellent results in most cases

To reappraise the state of the condition at Kenyatta National Hospital(KNH) 51 cases from the year 1973 - 1981, were studied, in terms of presenting features, investigations and treatment. Particular attention has been placed on features, which may assist in early recognition of the condition and the effects of late diagnosis and treatment vis-a-vis the eventual recovery and return to normal activity.

INTRODUCTION

Records on the structure and function of the pericardium go back to the time of Hippocrates who described it as "a smooth tunic which envelops the heart and contains a small amount of the fluid resembling urine". A good account of pericardial disease was made by Richard Lower (1631-1691).¹¹

A suggestion to perform pericardectomy for pericardial effusion by trephining the sternum was made by J. Riolan, but the technique was not adopted for the next two hundred years.¹¹

Several operations for constrictive pericarditis and pericardial effusion were performed by Romero (1819), Shuh (1840); and Karanaeff (1840); with variable success.¹¹

Towards the latter half of the last century advances were being made in interpreting the clinical picture of constrictive pericarditis in physiological terms, culminating in a critical exposition of the paradoxical pulse and the rise of venous pressure on inspiration by Kussmaul (1873).¹¹

Successful treatment of constrictive pericarditis by excision of the pericardium in the early part of this century was performed by Rehn and Sauerbruch in 1913 and Churchill in 1929.¹¹

The condition of chronic constrictive pericarditis has long been recognised in East Africa²⁹. Twenty years ago fifteen patients with this condition were treated by pericardectomy in Nairobi.

Structurally the pericardium consists of an outer sac, the fibrous pericardium, and an inner sac, the serous pericardium. The serous pericardium has two layers, the visceral layer which lines the heart and the parietal layer which lines the fibrous pericardium. The space between the two layers of the serous pericardium contains a thin film of fluid. Excess fluid can accumulate in this space, a state called pericardial effusion.

Functionally, the pericardium with its thin layer of fluid in the serous sac allows the heart a frictionless investment in which to function¹¹. The pericardium also prevents overdilatation of the heart and kinking of the major vessels. It may also prevent direct extension of infection from surrounding structures, to the heart. It is however noted that there is little disability after operative removal of the pericardium or in patients with congenital absence of the structure¹¹.

Absence of the pericardium may not produce disability in an individual but a diseased pericardium does. The pericardium may be involved in various disease states. One of these, is pericarditis. Pericarditis may present in an acute, a sub-acute or a chronic form. This study is concerned with the chronic constrictive type.

MATERIALS AND METHODS

An analysis of clinical records of 51 patients seen at Kenyatta National Hospital (KNH) between 1973 and 1981, and diagnosed as chronic constrictive pericarditis was made. A form into which to transfer the information from these records was made (see Appendix I).

The analysis included clinical presentation of these patients, in terms of age, sex, symptoms and signs. The aim here was to bring out the commonest and the most persistent presenting features in this condition. The history and the physical findings as recorded in the patients file were studied and the information transferred to the analysis form.

An analysis of the investigations was also made. This included data from the haemodynamic laboratory, radiological and histological investigations. Other investigations included haematological and liver function profiles.

There were three haemodynamic investigations done cardiac catheterisation electrocardiography (ECG) and echocardiography. All patients had ECG done. Catheterisation was done in 33 patients. Six patients had echocardiography done. These investigations were analysed in terms of reliability as confirmatory tests.

Radiological study was that of a plain chest x-ray. The aim here was two-fold to look for pericardial calcification and cardiothoracic ratio.

Bacteriological studies were carried out on sputum; pericardial, pleural and ascitic fluids. The studies included cultures and sensitivities for bacteria and acid fast bacilli.

Histological studies were carried out on the pericardium. Features of tuberculosis were looked for.

Liver function tests included estimates of bilirubin, plasma protein, and enzymes. This was to ascertain whether there is any significant liver function impairment in constrictive pericarditis.

Lastly a review was made of the follow-up cases as seen in the clinic after surgery. This was to determine what percentage had resumed their normal duties, prior to onset of illness and what percentage still had disturbing symptoms and were on supportive medical treatment.

RESULTS

A) Sex (51 Cases)

There were 41 males (80.4%) and 10 females (19.6%)

B) Age (51 cases)

The age of the patients ranged from 2½ years to 74 years.

The peak incidence was between twenty and thirty years.

(see chart 1).

C) Duration of symptoms.

Most of the patients were diagnosed as having constriction within one year. The number diagnosed within this period was forty (78.4%). The rest of the patients (21.6%) were diagnosed between one year and four years. The earliest diagnosis was made in two months and the latest in three years and a half.

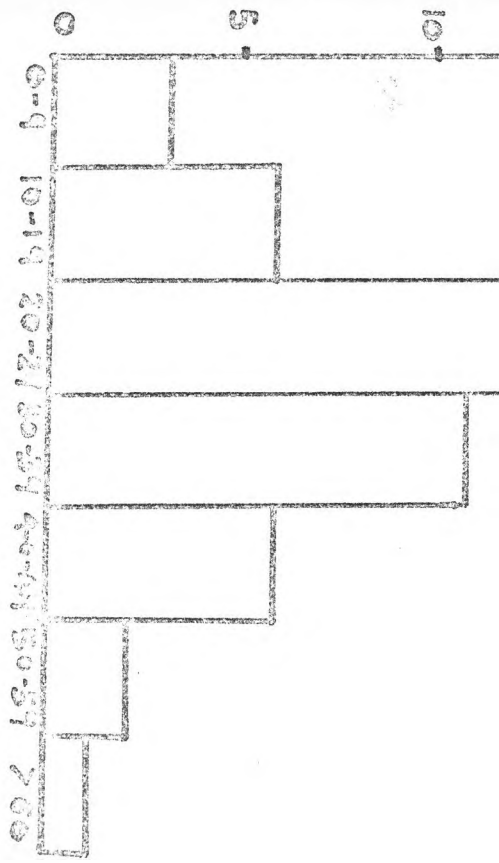
D) Symptoms when first seen (51 Cases)

Dyspnoea on exertion and abdominal swelling were by far the most common initial and disturbing symptoms. The commonest symptom was abdominal distension (76.4%) and the least common fever (11.8%). (See chart 2).

E) Physical Findings (51 Cases)

Hepatomegaly was the commonest physical finding (96.0%) followed by elevated jugular venous pressure (JVP) (90.1%). The least common physical finding was Kussmaul's sign, seen in 9.8% of the cases (see chart 3).

No. of



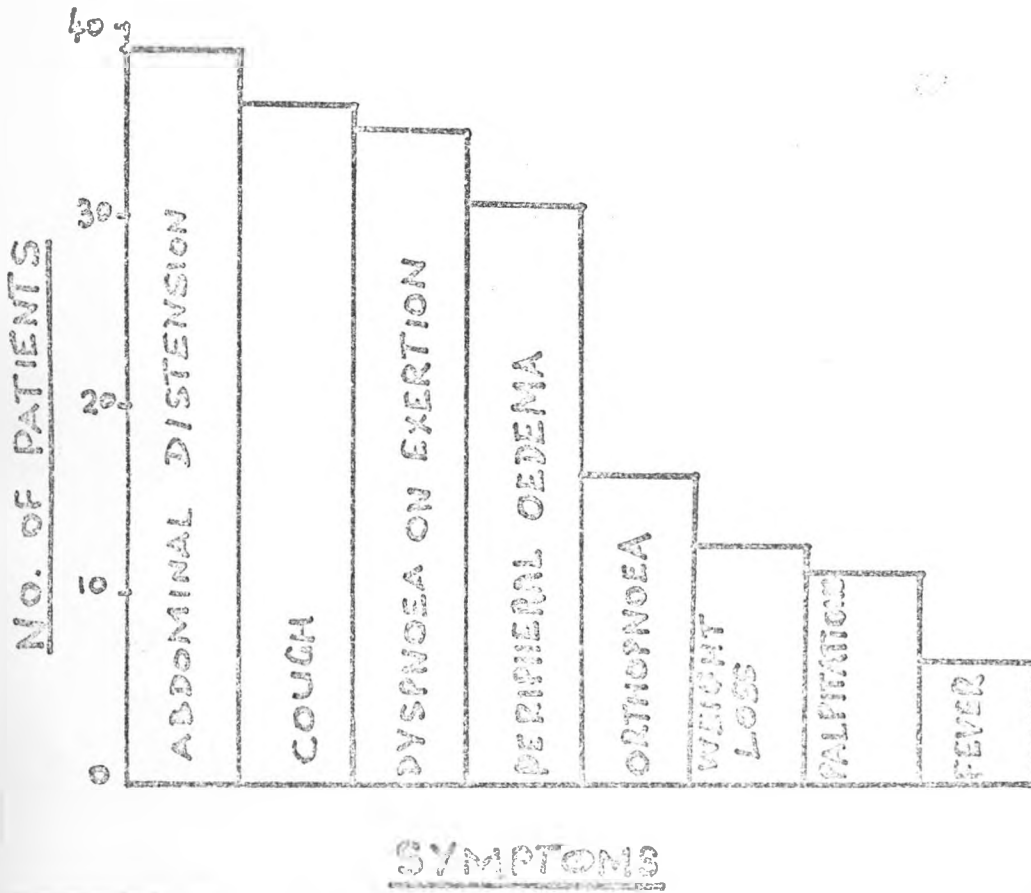
AGE IN YEARS

PATIENTS

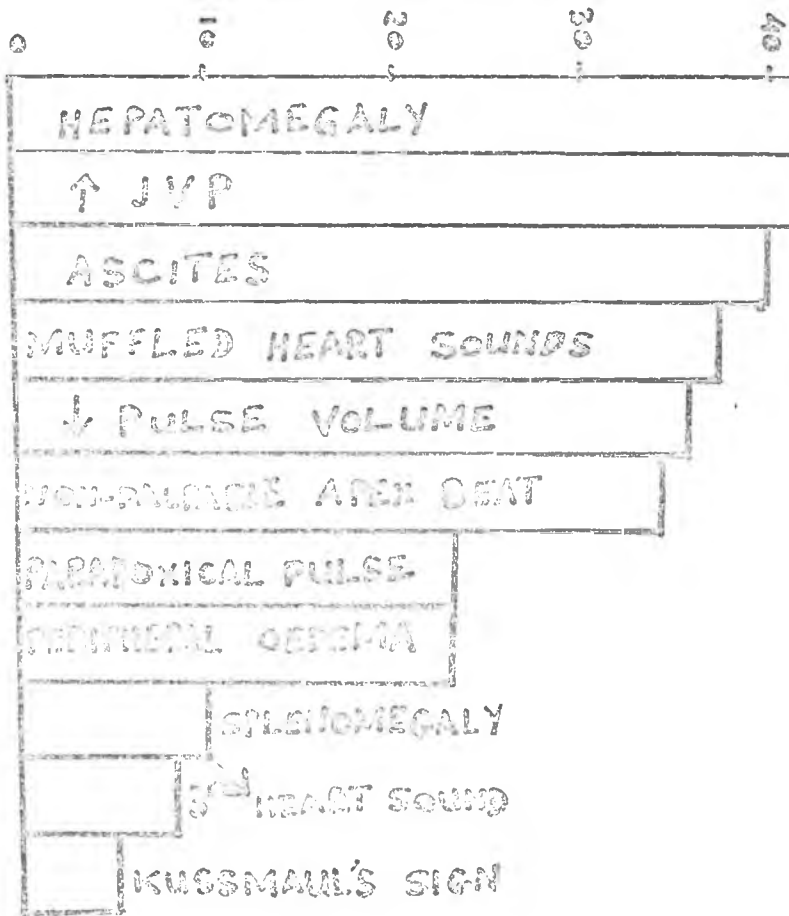


CHART 1- AGE DISTRIBUTION

CHART 2 - PRESENTING SYMPTOMS.



No. of PATIENTS



PHYSICAL FINDINGS

CHAPT 3- PHYSICAL FINDINGS

50 "



F) Laboratory Findings

Various investigations were done from the more basic ones to more sophisticated ones.

Ten patients (19.6%) were found to have a haemoglobin of less than 10g%. This has been attributed to anaemia of chronic illness.

Twelve patients (23.5%) were found to have an elevated bilirubin-of more than 1mg%.

Five patients (7.8%) were recorded as having had a positive Mantoux test.

All patients had bacterial work-up on their sputum, and where available ascitic, pleural and pericardial fluids. Only one patient had tubercle bacilli isolated in his sputum. In three patients culture for tubercle bacilli was positive. In one patient culture of pericardial fluid grew staphylococcus aureus. There were no parasites isolated in this series.

Radiological Findings

All patients had chest x-rays done. In eight patients (15.6%) the chest roentgenograph showed pleural effusion. In five (9.8%) there was pericardial calcification, (see plate 1a and 1b). Twenty-two patients, (43.1%), were found to have a reduced cardio-thoracic ratio.

Electrocardiographic (ECG) Findings

This investigation was carried out on all patients.

The most common features were generalized low voltage, and T-wave changes, (see Figure 1).

Thirty-eight patients, (74.5%), had generalised low voltage, more marked on peripheral leads. There was T-wave inversion in thirty-one patients, (60.7%).

Eleven patients, (21.5%), were found to have T-wave flattening. Atrial fibrillation was found in eight patients, (15.6%), and S-T segment changes in five.

Cardiac Catheterisation

In all, thirty three patients had cardiac catheterisation done.

Intracardiac pressures were found to be elevated in all cases, approaching the same level in all chambers (see Table 1).

Characteristically there was a rise in the right atrial mean pressure with rapid x and y descents. There was also a rise in right ventricular end diastolic pressure, mean pulmonary artery pressure and left ventricular end diastolic pressure.

Echocardiographic Findings

Six patients in this series had echocardiography done.

In two patients pericardial effusion and pericardial thickening were apparent, (see plate 2).

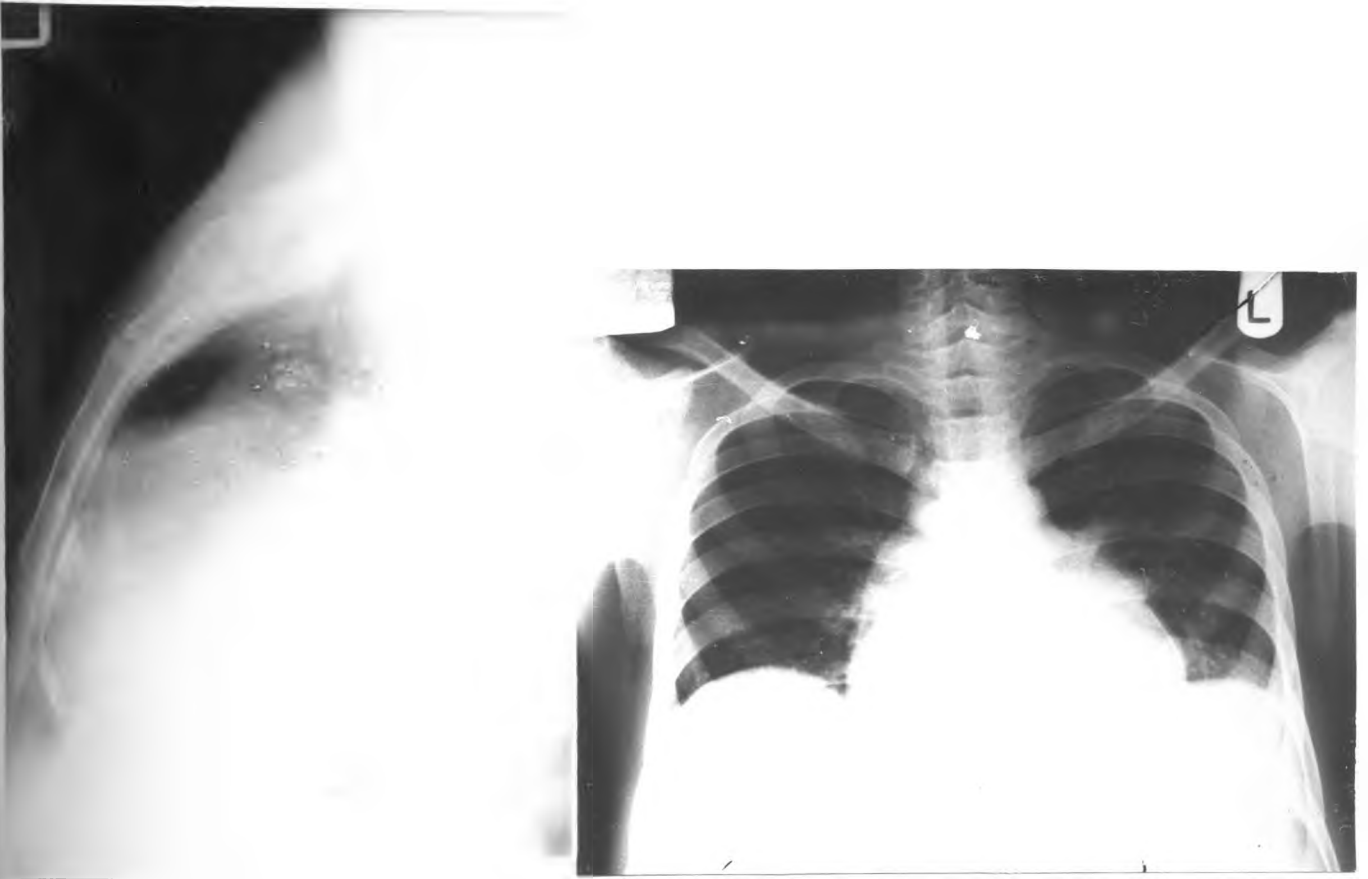


PLATE 1 Chest x-ray of patient with constrictive pericarditis.

The lateral view shows pericardial calcification, which is not very well shown in the antero-posterior view. Pericardial calcification if present is best demonstrated by lateral views of the chest.

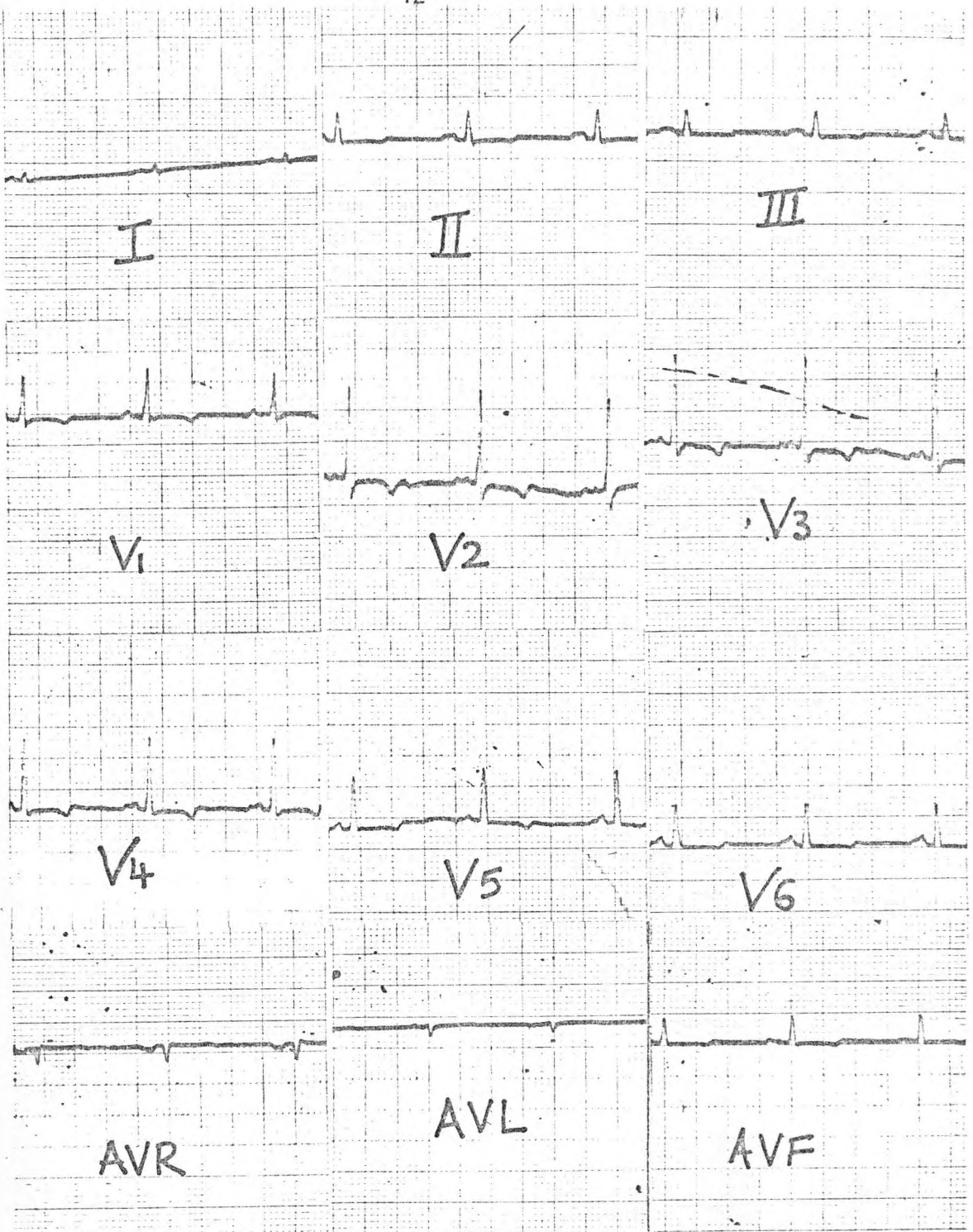


FIGURE 1 Electrocardiogram of patient with constrictive pericarditis.

Note - peripheral low voltage, depressed S-T segment ($V_2 - V_6$), and flattened T-wave (I and a VF).

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SITE	PRESSURE (mmHg)		
	NORMAL	PHASIC	MEAN
S.V.C.	5		
I.V.C.	5		
RIGHT ATRIUM	5	a=26 v=25	18
RIGHT VENTRICLE	25/5	44/18	
PULMONARY ARTERY	25/12	44/23	27
PULMONARY CAPILLARY	12		
LEFT ATRIUM	12		
LEFT VENTRICLE	140/12	110/20	
AORTA	140/90	110/76	
BRACHIAL ARTERY	140/90		

TABLE 1 - Catheterisation data of a patient with constrictive pericarditis. Note the high mean right atrial pressure and elevated right ventricular and left ventricular end diastolic pressures.

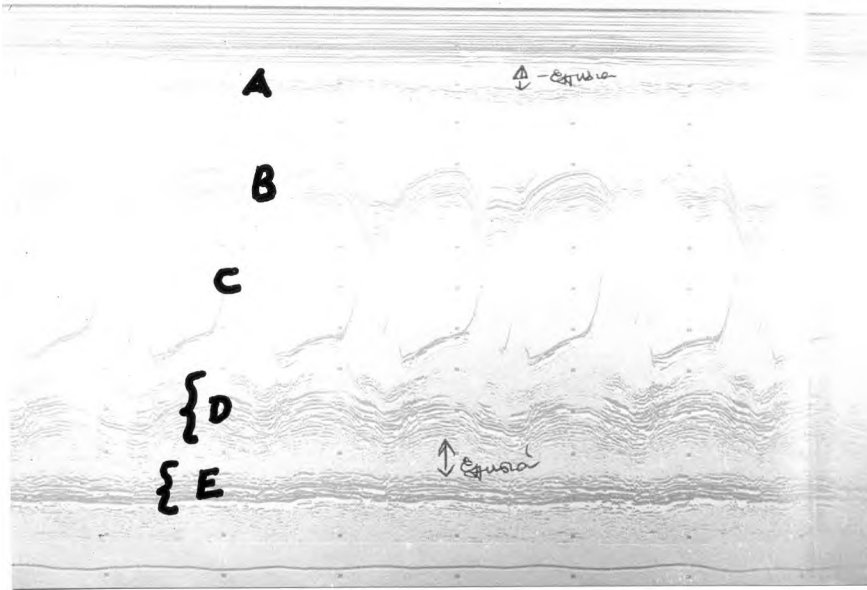


PLATE 2 Echocardiogram. In this plate there is pericardial effusion shown both anteriorly and posteriorly. The pericardium is markedly thickened posteriorly-E and there is reduced movement of the posterior wall of the left ventricle shown here as a flattened wave-D. A-Anterior Right Ventricular wall, B-Interventricular septum. C-Mitral valve, D-Posterior left ventricular wall, E-Pericardium.

One patient showed pericardial thickening without any effusion.

There was pericardial effusion in two patients with reduced movement of the left posterior ventricular wall, with no obvious pericardial thickening. In the last patient the echocardiogram showed flattening of the left ventricular posterior wall in diastole.

Histological Findings.

Of the forty-seven operated cases histology reports of the pericardium were available in thirty-eight of them.

Seven patients (14.9%) showed features of tuberculosis.

Four patients (8.5%) had calcium deposits on the pericardium.

The rest, twenty-seven (57.1%) showed features of non-specific chronic inflammation.

G) Results of Treatment

Of the fifty-one patients diagnosed as chronic constrictive pericarditis, forty-seven underwent surgery, and it was possible to perform radical pericardectomy in forty-two of them.

Four out of the original fifty-one did not have surgery. One was a case of terminal hepatoma. One declined surgery and there is no reason available why the other two did not have surgery.

The surgical approach in thirty-nine, (82.9%), of the patients was by a midline incision through the sternum. In the rest eight, the approach was through a left lateral thoracic incision.

Nine patients (19.1%), out of those who underwent surgery died. One died of massive haemorrhage on the operating table. Three died of pulmonary embolism as a result of deep vein thrombosis within two weeks post-operatively. One died of atrial fibrillation ten months after surgery. The only patient who underwent pericardectomy on the open heart machine died three months later while still in intensive care, of respiratory failure. The causes of death in the rest three patients are not available.

H) Post-Operative follow-up at Kenyatta National Hospital.

Out of the forty-seven patients who had pericardectomy, thirty-four were followed up at the cardiothoracic clinic at Kenyatta National Hospital.

After one year of follow-up twenty one patients (61.7%) resumed normal activity. Normal activity is taken to mean that the patient is symptom-free and *and has resumed normal duties.* is not on any supportive medical treatment. If on the other hand the patient still has all or some of the previous disturbing symptoms and is on supportive medical treatment, then he is deemed not to have resumed normal activity. Patients in this category are on treatment for congestive heart-failure.

In this series there were thirteen patients (38.3%) who still had symptoms and were on medical treatment one year after surgery.

DISCUSSION

Pathophysiology

In chronic constrictive pericarditis the process of healing of the pericardium has produced fibrosis and contraction. This results in the encasing of the heart interfering with diastolic filling. As a result of this inadequate filling of the ventricles during diastole there is a reduction in stroke volume. There is also an elevation of the end-diastolic pressures in both ventricles, as well as the mean pressures in the atria, pulmonary and systemic veins to about the same level.

It is also observed that the Central Venous and atrial pressures display an M-shaped wave with steep rises of 'a' and 'v' waves and steep 'x' and 'y' descents, the 'y' being more marked. This is in contrast to cardiac tamponade where the most prominent deflexion is that of the x wave. In constriction also, the ventricular pulses exhibit the square root phenomenon.

The decreased stroke volume and therefore reduced cardiac out-put causes exertional dyspnoea²⁹. Venous congestion is initially caused by impaired ventricular filling due to the restrictive action of the inelastic pericardium. It is, however, possible that the fibrotic process may extend to the myocardium and venous congestion may then be attributed to both myocardial and pericardial involvement. This may also explain why some patients do not do well even after surgical release of the myocardium by pericardectomy.

In a normal cardiovascular system there is an inspirational fall and an expirational rise in blood pressure which is normally no more than 6mmHg. This is called the paradox. An accentuation of this phenomenon of more than 10mmHg difference results in a paradoxical pulse where the pulse may completely disappear

on inspiration. This may be found in constrictive pericarditis. It is however more common in cardiac tamponade.

An inspirational rise in central venous pressure, called Kussmaul's sign is also observed in both cardiac tamponade and chronic constriction.

The effects of constriction are initially inconspicuous. In the sub-acute form of this condition the heart is constricted by fibroelastic bands which constrict the heart in both diastole and systole¹⁴. The heart then, is still connected to the thorax hydrodynamically, so that the intrapleural pressures are still transmitted to the heart in the normal way. Paradoxical pulse, seen in 47.0% of the cases in this series, depends on the normal augmentation of venous return and is therefore prominent in this form of constriction.

In chronic constriction, the rigid shell constricts the heart in diastole only, when the heart needs to expand to the maximum volume. Transmission of interpleural pressures to the heart is lost, so that it is hydrodynamically isolated from the thoracic cavity. This leads to loss of inspirational augmentation of cardiac filling and therefore to inspirational rise of venous pressure - Kussmaul Sign. ^(seen in 9.8%) This sign is therefore seen more in the chronic form of constriction.

Laboratory and other findings.

Routine laboratory data have not been found helpful¹⁶.

The haematocrit was found decreased in 19.6% of the cases.

An elevation of bilirubin of more than 1mg% was observed in only 23.5%.

Chest roentgenographs, done in all patients showed pericardial calcification

in only 9.8%. It seems that too much emphasis has been placed on radiographic demonstration of pericardial calcification²⁹. It is however an important finding when present. Pericardial calcification is best demonstrated by lateral views of the chest, (see plate 1a and 1b).

A reduced cardiothoracic ratio is not always seen in constriction. In some cases there is, in fact, an increase in the size of the cardiac shadow. In this series 43.1% of the patients had a small heart.

Electrocardiographic abnormalities are always seen in chronic constriction. Atrial fibrillation considered the most common arrhythmia in constrictive pericarditis, was observed in eight patients (15.6%). The presence of atrial fibrillation with pronounced right axis deviation is considered as a sign of more severe disease.

A more common observation is a generalised low voltage of the QRS complex seen in thirty eight patients (74.5%). The low voltage is more pronounced in peripheral leads (see plate 2). It is a reliable diagnostic feature when present. Its absence does not exclude the diagnosis of constrictive pericarditis.

In thirty-one patients (60.7%), there was T-wave inversion, while in eleven (21.5%) there was T-wave flattening. T-wave changes are attributed to atrophy and myocardial fibrosis in constriction.

S-T segment changes were only seen in five patients.

Echocardiographic studies were carried out in six patients. Evidence of pericardial thickening was observed in three of them using this technique.

Echocardiography is a non-invasive technique which utilises the quality of different types of tissues to transmit different amounts of sound waves. The waves not transmitted are reflected back as echoes, and utilising these echoes depths and mobility of tissues can be determined. The sound waves are those beyond the hearing range of the human ear. In constrictive pericarditis various observations can be made. Pericardial thickening can be discerned more clearly when there is pericardial effusion (see plate 3).

The motion of the left ventricular posterior wall is impaired, producing a flat profile on the echocardiogram instead of the wave like motion. This is because in diastole there is an abrupt transition of rapid ventricular filling to diastasis in those with constrictive pericarditis³¹.

The most definitive diagnostic procedure is cardiac catheterisation. Intracardiac pressures are elevated in all chambers as a result of diastolic restriction by the rigid pericardium. All thirty-three patients who had this investigations done showed abnormal atrial pulse waves and elevated intracardiac pressures. Elevated intracardiac pressures are also seen in restrictive cardiomyopathy, and this may present a problem in the differential diagnosis, vide infra.

In some cases diagnosis may not still be reached, even after all these investigations. Diagnostic thoracotomy would then be justified.

Aetiology

In most cases of chronic constrictive pericarditis no underlying aetiology is

evident clinically or histologically^{16,22}. Most cases of this condition are attributed to infection by tubercle bacilli, though this bacillus is very seldom isolated⁷.

In this series seven patients (18.4%) were proven by histological examination of the pericardium to have had tuberculosis, as the cause of pericardial constriction. Tubercle bacilli were demonstrated by staining and culture in only four patients. Nine patients had a previous history of tuberculosis for which they were treated, though in these patients there was no evidence of tuberculosis on pericardial histology and culture.

Other aetiologic factors have been mentioned. In this series three patients were shown to have pyogenic pericarditis. They were all below ten years old. In two of them it followed acute osteomyelitis and in the other an episode of pneumonia. All three developed constriction within ten weeks. It seems then that, in children chances of developing pyogenic pericarditis are higher than in adults, and that constriction develops much faster^{5,10,26}. It is therefore justified to perform pericardectomy in pyogenic pericarditis even if there is no evidence of constriction.

Viruses, fungi and parasites are other aetiologic agents in constrictive pericarditis²¹. Others include connective tissue disorders, trauma, neoplasia and radiation^{7,16,29}.

In thirty-four patients (72.3%) in this study no underlying aetiology could be established. However all patients, except those with pyogenic pericarditis

were on treatment against tuberculosis. Considering the relatively high incidence of tuberculosis in this region this action is justified.

Differential Diagnosis

Chronic constrictive pericarditis commonly presents problems in diagnosis. It may present as a variety of other conditions including liver cirrhosis, congestive heart failure, nephrotic syndrome, intra abdominal malignancies and cardiomyo-
7, 16, 29
pathies .

In this series twenty patients (39.2%) were initially diagnosed as cirrhosis of the liver, 31.3% as cases of congestive heart failure, 9.8% as nephrotic syndrome and 19.6% as constrictive pericarditis.

In the full blown case the most disturbing and earliest symptoms are abdominal distension, cough and dyspnoea on exertion. Orthopnoea, a prominent feature in congestive heart failure was only seen in 33.3% of patients in this study. Marked peripheral oedema, cardiac murmurs and cardiomegaly are features more common in congestive heart failure than in constriction.

In cirrhosis of the liver a raised JVP is lacking, seen in a striking 90.1% in constriction. Liver function tests are more likely to be abnormal in cirrhosis than in constriction. The diagnosis of nephrotic syndrome is given away by massive proteinuria, and the marked constitutional symptoms give a clue to malignancy.

The most difficult to distinguish from pericardial constriction are the cardiomyopathies, especially restrictive cardiomyopathy. Clinical features may be similar in these two conditions and cardiac catheterisation in restrictive cardiomyopathy may

show similar features with pericardial constriction.¹⁶

Findings in favour of cardiomyopathy include episodes of pulmonary oedema, very rarely seen in constriction; a prominent displaced apical impulse evidence of left ventricular hypertrophy or bundle branch block on the E.C.G. Haemodynamically the intracardiac pressures between the two ventricles do not approach the same level in cardiomyopathy and the difference between them is usually greater than 6mmHg. This value is always less in constriction.

With these features in mind diagnosis of pericardial constriction should not be very difficult.

Treatment

Forty-seven out of fifty one patients diagnosed as having pericardial constriction underwent surgery.

The aim is surgical treatment is radical removal of both parietal and visceral pericardium.

The most common approach used in this series is through a median sternotomy.

The left ventricle is freed first to avoid pulmonary congestion when release of the right ventricle increases pulmonary flow and volume¹⁶.

In most patients haemodynamic parameters return to normal over the next one year¹⁶. However in some patients this does not occur. This has been attributed to myocardial fibrosis and atrophy which occurs in some patients with this condition.

CONCLUSION

In conclusion, misdiagnosis of chronic constrictive pericarditis occurs often. If only to free a patient from suffering and disability, attempts should be made to reach early diagnosis and to follow it with early surgical treatment.

There is however a more important reason for early recognition of this condition. In this study those who were diagnosed and treated early showed better prognosis. Of the twenty-one patients who resumed normal activity fifteen were those who were diagnosed within six months of the symptoms. Of the thirteen who still had symptoms, ten were diagnosed six months after onset of symptoms. It seems then that early diagnosis and treatment gives better prognosis.

Finally, prevention is possible, taking into consideration the aetiological factors. The emphasis on early diagnosis and treatment should automatically be followed by a fall in incidence as preventive measures also stem off new cases.

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APPENDIX I (i)

SURGICAL PERICARITIS

<u>Name</u>	<u>Age</u>	<u>Sex</u>
<u>Tribe</u>	<u>Residence</u>	<u>Occupation</u>

Medical History

1. Date of onset of illness
2. History of PTB
3. Length of Rx
3. Other illness
 - (a) Furulent - Acute PyogenicSource of entry

4. SYMPTOMS

- (a) Cough
- (b) Weight loss
- (c) Fever
- (d) Shortness of breath
 - on exertion
 - Orthopnoea
- (e) Swelling of Abdomen
 - legs
- (f) Palpitations.

5. PHYSICAL EXAMINATION

- (a) Nutritional Status
- (b) Glands
- (c) Jaundice
- (d) Pulse
 - Rate
 - Volume
 - Paradoxical
- (e) JVP
- (f) Heart Sounds
 - Loud
 - Muffled
 - Murmurs
 - 3rd Sound
- (g) Apex Beat
 - Palpable
 - Not Palpable

APPENDIX I (ii)

(h) Abdomen

- Ascites
 - Mild
 - Mod
 - Gross
- Hepatomegally
- Splenomegally

(i) Ankle Oedema.

6. INVESTIGATIONS

- (a) Haemogram - Hb
- WBC
- ESR

(b) LFTs

<u>Alk Phosphatase</u>	<u>Total Fr</u>	<u>Bilirubin</u>
SGOT	Alb	Total
SGPT	Globulin	Direct Indirect

(c) CARDIAC ENZYMES

(d) Mantoux

(e) Urinalysis

(f) CXR

- Caediothoracis Ratio
- Pleural Effusion
- Lung Fields
- Pericardial Calcification.

(g) Histology

(a) Pericardium

- Features of Tb
- Calcification
- Other

(b) Liver

- Cardiac Arrhosis.

(h) ECG

Features of constriction

(i) ECHO

Features of Constriction

(j) CATHETERISATION

Features of constriction

(k) BACTERIOLOGY

- Gram Stain
- AFB

APPENDIX I (iii)

7. SURGERY

- (a) Date (b) Time (c) Duration
- (d) Incision
- (e) Pericardium
- Percital
 - Visceral
- (f) Fleura
- Opened
 - Not Opened
- (g) (R) Atravin
- Pentorated
- (h) Caral Onifices
- (i) Blood Loss

8. POST OF

- (a) Blood Loss - Day 1 2 3
- (b) Urine - cut put chart
- (c) Weight Loss
- (d) Electrolytes
- (e) Mobilisatibn
- 1st Out of Bed
- (f) Ascites
- Recession
- (g) Hepatonegdy
- Recession
- (h) Stitches
- Removal
- (i) Discharge

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9. COMPLICATIONS

- (a)
- (b)
- (c)
- (d)

10. FOLLOW UP

- (a) Resumption of Normal Activity
- (b) Antifailure Therapy