CHRONIC CONSTRICTIVE PERICARDITIS

By

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Summary

11 patients of constrictive pericarditis with their clinical findings are described out of which 7 were subjected to Surgery. Tuberculosis appears to be the probably etiological factor in all the patients. None of cases showed pericardial Cacification as reported in Western Literature.

The condition results from dense fibrosis with thickening of peri cardium and obliteration of the pericardial space following acute sero fibrinous pericarditis. This condition which is relatively uncommon in Western Countries is frequently seen in Pakistan. In most of the series reported in Western Litrature the cause is obscure. In the series reported by Gimleete Histological evidence of Tuberculosis was present in 13 out of 41 specimens. The majority of the cases are believed to be the result of past tuberculosis infection which was not clinically recognised, more recently pericardial constriction is known to follow viral pericarditis, collagenopathies particularly rheumatoid Arthritis, amoebic pericarditis, Radiation, Pyogenic and Malignant infiltration.

Present Series

This consists of 11 patients. Out of which 7 were males and 4 females. The age distribution of these patients is as follows:—

Age	*Cases*
10—20	5
20—30	2
30—40	1
40—50	2
50—60	1

Table I Summarises the presenting symptom, physical signs past history, tuberculine tests Radiology and electrocardiogram. In the majority of patients Ascites was a symptom present in 10 out of 11 patients, Dyspnoea 11 out of 11 patients, Cough was present in 3 out of 11 patients and (dependent oedema 2 out of 11 patients. The conmonest physical findings was raised Jugular venous pressure and hepatomegaly. Pulses Paradoxus was felt only in 3 patients.

Abnormal and atypical widening of 2nd heart sound was heard in 5 patients and accentuated 3rd sound (&Pericardial knock) as reported by Beck was heard in 2 out of 11 patients. The duration of symptoms in this series reported is pretty long 4 months—2 years. 4 patients complained of a prolonged fever before the onset of symptoms. In the x-ray findings pericardial calcification which is reported in 50% cases in Western Literature was not seen in any single instance although it was carefully looked for in 3 projectings. Bilateral or Unilateral pleuraleffusion were seen in 4 out of 11 patients. Heart size was normal radiologically in 9 patients and slighly enlarged in two patients. The electrocardiographic findings were more or less uniform low voltage and generalized T-wave flattening or inversion except in one case where it was a normal voltage with T-wave inversion was seen.

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TABLE I

Serial No.	Presenting symptoms	Physical signes	Past history	Tuberculine Test	Radiology	E.C.G. Findings
1 S.M. 18 Male	Recurrent ascites Dyspnoea and cough—2 years	J.V.P. —10 cm Liver —2 fingers	Febrile illness before the onste of symptoms	Positive	Slight Cardiomegaly	Low voltage and generalize T wave inversion.
2 S.E. 14 Female	Recurrent Ascites Enlarged cervical glands—2 years	J.V.P. —15 cm Liver— fingers Splitting of 1st and 2nd heart sounds.	Nil	—— 20 mm wheel	Heart size normal Bilateral pleural effusion.	Low voltage generalize T wave in version.
3 R.B. 46 Female	Dyspnoea — 6months Ascites dependents — 4 months Oedema — 4 months	J.V.P. —12 cm Liver —2 fingers Pulsus pardoxus.	Febrile illness lasting 4 months—1 years.	—	Heart zize normal.	Low voltage.
4 N.A. 55 Males	Epigestric discomfort—4 months Dyspnoea—2 months Ascites Dependent Oedema	J.V.P. —15 cm Liver —3 fingers	Nil	—	Heart size normal.	Low voltage
5 B.K. 33 Males	Dyspnoea—6 months Palpitation—6 months Epigastric discomfort—6 months	J.V.P. —51 cm Assentuated 3rd Heart sound splitting 2nd heart sound pulsus paradoxus.	Nil	—	Heart size normal	Generalized T wave inversion.
6 F.M. M 50 Females	Ascites—1 year Cough—1 year	J.V.P. —13 cm Liver —3 fingers Splitting of 2nd heart sound loud 3rd heart sound. (Pericardial knock)	Febrile illness before the onset of symptom.	—	Heart size normal	Low voltage generalized T wave flattening.
7 S.M.B. 21 Females	Recurrent Asscites —1 year Abdominal pain —1 year Dyspnoea Ankle Oedema	J.V.P. —18 cm Liver — 4 fingers 1 year	Nil	—	Heart size normal	Low Voltage generalized T waves Flattening.
8 B.B. 30 Female	Recurrent Ascites —6 months Dyspnoea —6 months Fever —6 months	J.V.P. —15 cm M Liver — 5 fingers Split 2nd sounds	Daughter suffered from pleurisy	—	Heart size normal bilateral pleural effusion.	Low Voltage generalized T wave inversion.
9 M.K. 16	Dgspnoea 9 months Ascites —9 months Ankle Oedema —9 months	J.V.P. —15 cm Liver — 3 fingers Split 2nd sound	Nil	—	Heart size slightly enlarged.	Low voltage T wave inversion.
10 E.I.	Ascites —2 years Cough — years Dyspnoea —2 years	J.V.P. —15 cm Liver — 3 fingers Split 2nd sound	Febrile illness before the onset of symptom	—	Heart size normal Bilateral pleural effusion.	Low voltage T wave inversion.
11 Z.H. 19	Fever — 4months Chest pain —4 months Dyspnoea —2 weeks Ascites	J.V.P. —15 cm Liver — 3 fingers Pulsus Paradoxus	Nil	—	Heart size normal Right pleural effusion.	Normal voltage T wave inversion.

TABLE II

Serial No.	*Operative Finding*	*Histology*	*Treatment*	*Results*
1. S.M. 18	Pericardium thick, Pockets of pus and cheesy material.	Not available	Standard Anti-tubercular Treatment.	Cured
2. S.B. 14 Females	Thick Cartilargious pericardium layers adherent.	No specific lesion recognized.	———do———	Died 10 hours after operation.
3. R.B. 46 Females	Thick pericardium layers adherent.	Diffuse infiltration with mono nuclear cells, area of necrosis Dg. chronic pericarditis.	———do———	Cured.
7. S.M.B. 21 Female	Pericardium thick with adherent layers.	Granulomatous lesion composed of epithelial cells and gaint cells. (Tubercular Pericarditis)	———do———	Completely relieved of symptoms.
8. B.B. 30 Female	Thick hard pericardium with adherent layers.	Not available.	———do———	Completely relieved of symptoms.
9. N.K. 16 M	Pericardium dense and thick layers adherent.	Thick fibrotic pericardium with new formed blood vessels nodular round cells infiltration diagnosis non specific pericarditis.	———do———	Improved post-operative.
10. E.I.	———do———	———do———	———do———	Completly relieved of symptoms.

7 out of these patients were subjected to surgery. 1 patients died 10 days post operatively and six were cured. The results are summarized in table III.

All patients had standard anti-Tuberculosis treatment at least one month before operation followed by continouation of anti-tubercular therapy post operatively. Thus in the histology findings in one case, a clear out diagnosis of tubercular pericarditis was given, in 3 cases round cell infiltration was reported. All the 6 patients who survived operation was relieved of symptoms and gained weight on Anti-tubercular regime, those who refused operation were treated symptomatically with diuretics and anti-tubercular drugs, also improved.

Discussion

Although convincing evidence of tubercular etiology was present in only 1 patient. Probably the majority of the cases are due to tubercular etiology. This is suggested by the past history of febrile illness in 4 patients positive tuberculine reaction in all the patients and in the operative findings of pus and cheesy material in in case No. 1 in which the histology was not available. The presence of round cells infilteration in 3 histology reports is also suggestive of tubercular etiology. The response to anti-tubercular drugs, has been uniformly satisfactory which also supports tubercular etiology. Two more patients of proved tuberculous pericardial effusion not included in this series developed constriction within 6 months of diagnosis inspite of Anti-tubercular therapy.

References:

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