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Toxoplasmosis and Heart Disease

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Abstract : This study is that of 12 patients with heart disease believed to be due to toxoplasmosis. A familial incidence of cardiomegaly, electrocardiographic and serological evidence was seen in several members of the families. Reliance has been placed on the serological findings. The usual picture was one of chronic heart disease. Some had only minimal symptoms, others were completely asymptomatic. Vascular manifestations without obvious involvement of the heart such as a sudden 'stroke' or arteritis were seen in some. It complicated other forms of heart disease. Toxoplasmosis as an aetiological factor should be looked for not only in patients with obscure heart disease of chronic nature but also in the acute forms with myocardial and or pericardial involvement. Toxoplasmosis might be commoner in Sri Lanka as an aetiological agent in obscure cardiac conditions than is realised.

1. Introduction

Toxoplasmosis is caused by a protozoon, *Toxoplasma gondii.* The world wide distribution and endemecity of toxoplasmosis was appreciated only after the introduction of the dye test by Sabin and Feldman in 1948. It is most common in warm and moist climates. Castellani[2] was probably the first to demonstrate the causative organism in man in Sri Lanka. Two forms of the disease have been suggested by Mohr:[13] (1) conatal and (2) postnatal. The conatal forms may manifest as malformations, neonatal deaths or may be latent. In the postnatal or acquired forms the protozoon may be disseminated throughout the body with clinical findings of multiple organ or system involvement. It may be localised from the very beginning and manifest itself as disease of a single organ ; or the disease may start as a generalised infection and then become localised.

Toxoplasma gondii can affect the myocardium as the primary target organ as the sole manifestation of the illness or as part of a generalised infection. Toxoplasmic myocarditis varies in its clinical manifestations and severity. It is potentially a curable illness and for this reason early recognition is of paramount importance.[2,6]

2. Patients and Methods

This study includes 12 patients with heart disease possibly due to toxoplasmosis and their family members; 4 of them were the subject of earlier reports.[14-16] All patients and their families underwent a complete diagnostic evaluation which included a history, physical examination and electrocardiographic, radiological and serological studies. They did not belong to any compact group such as farmers.

3—13786

The specific serological tests used in the diagnosis were the Indirect Haemogglutination test (IHA) and the Indirect Fluorescent Antibody test (IFA). The IHA was originally devised by Jacobs and Lunde.[8] The test is sensitive and specific and has been employed for diagnosis and sero-epidemiological studies in man and animals.

2.1. Case Reports

2.1.1. *Family A*

Case A1 : A 29-year old single male (D.A.) was admitted on 21.2.72 with a history of pain on both sides of the chest and neck of one day duration. The pain radiated down his left arm and was accompanied by sweating.

Examination revealed an average built, anxious individual. The blood pressure was 130/80 mm Hg, pulse 64/min and temperature 98.4° F. Examination of the chest revealed no abnormality in the heart or lungs. An electrocardiogram revealed the following changes : Sinus rhythm, rate 112/min, low voltage complexes, Q waves in L2, L3, AVF, V1 – V6 with ST elevation and inversion of T waves (Figure 1), the changes simulating an extensive postero-lateral myocardial infarction. SGOT 90 (23 – 110) units, serum cholesterol 168 mg/100 ml. IHA for toxoplasmosis 1 : 128 and 1 : 128 (2.3.72). He was treated with bed rest and sedation. He made an uneventful recovery. Teleradiogram revealed a slight enlargement of the cardiac shadow with hilar shadowing, and the electrocardiogram a slower heart rate and changes similar to the earlier record before discharge from hospital on 4.3.72.

Case A2 : A 32-year old sister of Case *A1* gave a history of repeated still-births and abortions (6 in all). She was in the 8th month of pregnancy at the time of examination. She had no complaints. Examination revealed no abnormality in her heart or lungs. IHA was 1 : 32 and electrocardiogram showed small Q waves in L1, AVL widened and slurred, QRS complexes with inverted T waves in L1, AVL, V4-V6 and QS waves in V2-V4 (Figure 1B). Pregnancy was terminated by Caesarean Section and a live, normal baby delivered. Histological examination of the placenta revealed no abnormality.

Case A3 : A 39-year old sister of Case *A1* had no symptoms. Her electrocardiogram was within normal limits. IHA was 1 : 1024.

2.1.2. *Family B*

Case B1 : A 29-year old man (AWAJ) was admitted on 24.4.70, with a history of severe retrosternal pain radiating down the left arm.

Examination revealed a young man of average build. The pulse was 92/min, blood pressure 170/80 mm Hg and temperature 98.4° F. The heart and lungs were normal clinically. Electrocardiogram showed Q waves in L3, AVF with ST elevation in L3, AVF. SGOT 180 (23 – 110) units, LDH 500 (100 – 400) units. He was treated as a case of acute myocardial infarction.

He was seen again in March 1971 with pain in chest. Electrocardiogram revealed a recent anterior myocardial infarction pattern. He made an uneventful recovery. IHA 1 : 512, IFA 1 : 256 (27.3.71) and IHA 1 : 1024, IFA 1 : 1024 (29.4.71).

He was re-admitted on 23.2.72, with a history of difficulty in breathing of 3 weeks duration. He had been treated by his doctor with Tetracycline and Hetrazan. Radiological examination on admission revealed no abnormality. He was breathless. His jugular pressure was not elevated, the blood pressure was 150/110 mm Hg and pulse 96/min. He was afebrile. The heart sounds were masked by the breath sounds. There were bilateral rhonchi with prolonged expiration in the lungs. The liver was not palpable. He was treated as a case of left ventricular failure with bed rest, diuretices, digitalis, aminophyllin and antibiotics. On the following day, there was no change in his condition with much cough and profuse expectoration. Electrocardiogram revealed sinus rhythm, rate 84/min, and changes consistent with old anterior and posterior infarction. The blood pressure was 130/80 mm Hg. As he showed no improvement Ampicillin and Cloxacillin were substituted for Tetracycline. An X'ray done on 1.3.72 showed no cardiac enlargement but a homogenous shadowing of both lung fields. White blood cell count was 9,600/mm,^{3} N 40% and E 9%. His condition remained the same and he continued to cough up whitish expectoration. On 4.3.72 he was given Prednisolone (30 mg daily) when he showed improvement with reduction in the cough, sputum and breathlessness. An X'ray done on 6.3.73 was normal. He was sent home on 7.3.72 and since then there has been no recurrence of symptoms. SGOT units, IHA 1 : 512.

Case B2 : A 24-year old wife of Case *B1* had no complaints. Electrocardiogram showed low voltage complexes, broad and slurred QRS, low and flattened T waves. IHA 1 : 256 and IFA 1 : 256.

Case B3 : A 1-year old male child of Case *B1* had a meningomyelocele at birth and was operated on. He had no other abnormality. No serological test was done.

2.1.3. *Family C*

Case C1 : A 26-year old male (SHPT-25669) was admitted with pain in chest and breathlessness. He was afebrile and no abnormality was detected. On the following day his pulse was 124/min, regular and blood pressure 130/80 mm Hg. There were no murmurs. That evening a pericardial rub was heard. He complained of pain in the back and had swelling over both buttocks. His temperature was 100° F and continued to remain so for the next 4 days. On the 10th day of his illness, a gallop rhythm was heard. Electrocardiogram showed sinus rhythm, QS waves in L1, AVL, V1-V4 with widened QRS complexes, ST elevation in L1, AVL, V1-V4. Serum cholesterol 284 mg%, ESR 8 mm, SGOT 240 (23-110) units. X'ray of chest revealed no abnormality. HAI for Arbo viruses was negative. IHA 1 : 64, IFA not done (12.9.72) ; IHA 1 : 64, IFA 1 : 256 (27.9.72) ; IHA 1 : 1024, IFA 1 : 512 (26.10.72). He made an uneventful recovery.

He was seen again on 20.2.73 when he had no symptoms. IHA 1 : 256 and IFA 1 : 256. The electrocardiogram showed right bundle branch block, QS waves in V2-V6, X'ray revealed no cardiac enlargement.

Case C2 : Father of C1 aged 65 years with no symptoms. There was no history of chest pain. IHA 1 : 64, IFA 1 : 256 (16.10.72) and IHA-ve, IFA 1 : 64 (22.2.73). The electrocardiogram showed QS waves in L3, AVF, V1, V2, Q in V4, notched T waves in V1 and V2. T inversion in L1, AVL, V4-V6.

Case C3 : Mother aged 55 years with no symptoms. IGA 1 : 128, IFA 1 : 128 (16.10.72) and IHA 1 : 64, IFA 1 : 64 (20.2.73). Electrocardiogram was within normal limits.

Case C4 : Elder sister aged 35 years with no symptoms. IHA 1 : 64, IFA 1 : 64 (6.10.72).

Case C5 : Younger sister aged 23 years with no symptoms. IHA 1 : 64, IFA 1 : 64 (16.10.72) and IHA -ve, IFA 1 : 128 (20.2.73). ECG was within normal limits.

Cases C6 and C7 : Children of *C4* aged 5 and 4 years. Serology negative.

Case C8 : Servant aged 15 years with no symptoms. Serology negative.

The patients *D1*, *E1* and *F1* have been described in detail elswehere.[14,15] Only brief mention of the salient features will be made here.

2.1.4. *Family D*

Case D1 : A 29-year old male was seen on 5.7.71 in congestive heart failure. He had been in good health until 10 months earlier when he began to develop breathlessness on exertion followed by oedema of his lower limbs. Examination revealed a patient in congestive heart failure. Electrocardiogram showed sinus rhythm, right axis deviation, broad P waves, slurred QRS complexes, QS waves in L1, AVL and V4 with low T waves. X'ray showed enlargement of his cardiac shadow. IHA 1 : 512, IFA 1 : 32 (5.6.71) and IHA 1 : 1024, IFA 1 : 256 (28.7.71).

2.1.5. *Family E*

Case E1 : Female aged 32 years was seen in cardiac failure. She gave a history of breathlessness and swelling of legs over the last 3-4 years. Electrocardiogram showed complete left bundle branch block and left ventricular hypertrophy. X'ray showed cardiac enlargement CTR 12.5.22. IHA 1 : 512 (9.11.69) and 1 : 486 (22.12.69. She died a few months later following a cerebral embolus.

2.1.6. *Family F*

Case F1 : A 30-year old male (S) was admitted with a history of breathlessness of about 4 months duration. A diagnosis of old myocardial infarction and cardiomegaly had been made in another hospital. He was not in congestive heart failure. Electrocardiogram showed Q waves in L1, AVL and T inversion in L2, L3, AVF, V2 and V4. X'ray showed an enlarged heart shadow (CTR 15 : 22) and pulmonary congestion IHA 1 : 164 (9.11.69) and 1 : 162 (22.12.69).

The serological finding and electrocardiographic changes of the members of Families *D*, *E* and *F* are shown in Table 1.

TABLE 1. Serological and electrocardiographic findings in Families D,E,F.

Relationship	Serology IHA	Serology IFA	Electrocardiographic findings
Family D			
Patient *D1*	1 : 2048	1 : 256	RAD, broad P waves, slurred QRS complexes, QS waves in L1, AVL, V4 low T waves
Wife (24) *D2*	-ve	-ve	low flattened T waves.
Son (2½) *D3*	-ve	1:2	ND
Daughter (1½) *D4*	-ve	-ve	Q waves in L3, notched T waves V2 V4.
Family E			
Patient *E1*	1 : 512	ND	LBBB and LVH
Husband (40) *E2*	1 : 64	ND	normal limits
Son (14) *E3*	-ve	ND	prominent Q waves
Daughter (12) *E4*	-ve	ND	T inversion in L3, AVF, LVH
Son (9) E5	-ve	ND	prominent Q waves
Family F			
Patient *F1*	1 : 164	ND	Q waves and inverted T waves
Wife (25) *F2*	-ve	ND	not done

ND not done.

2.1.7. *Family G*

Case G1 : A 40-year old single male was admitted on 3.3.72, with pain in chest from 8.2.72. The pain was inframammary with no relation to effort. There was no abnormality in the heart or lungs. Electrocardiogram showed Q waves in L3, AVF, low and flattened T waves in L2, L3, AVL and AVF. X'ray showed minimal cardiac enlargement with hilar congestion. WBC 6,400 mm^3. N 69%, L 28%, E 3%, ESR 15 mm, SGOT 60 units, IHA 1 : 128, IFA 1 : 128 (16.3.72) and IFA 1 : 64 (18.4.72).

2.1.8. *Family H*

*Case H*1 : A 30-year old single male (USA) was admitted with retrosternal pain of 4 days duration. No abnormality was detected in his heart or lungs. SGOT 90 units (23-110 units). ESR 3 mm, WBC 11,200 mm^3, N 50%, L 39%, E 11%. Electrocardiogram showed low voltage complexes, widened QRS complexes, low T waves in L1, L2, AVL with T inversion in AVF. IHA 1 : 128, IFA 1 : 32 (10.4.72) and IHA 1 : 64, IFA 1 : 64 (11.5.72).

2.1.9. *Family I*

Case I 1 : (*GS.*) This case has been described in detail elsewhere.[16] Male aged 22 years was admitted with breathlessness, fever and cough. On admission he was febrile with an extensive pericardial rub. A tentative diagnosis of tuberculous pericarditis was made. ESR 15 mm, WBC 5,400 mm^3, N 51%, L 46%, E 3%, Mantoux negative, ECG revealed auricular fibrillation with non-specific T wave changes. X'ray showed an enlarged heart shadow, the lung fields were clear. IHA 1 : 64, IFA 1 : 32 (23.5.71) and IHA 1 : 64, IFA 1 : 4096 (4.7.71). Five months later, a diagnosis of constrictive pericarditis was made and was referred to the Thoracic Surgeon and a pericardectomy was done. Histology of the pericardium revealed non-specific changes.

Case I 2 : Grandmother. IHA 1 : 128, IFA 1 : 32.

Case I 3 : Mother. IHA, IFA -ve.

Case I 4 : Brother. IHA 1 : 128, IFA 1 : 256.

2.1.10. *Family J*

*Case J*1 : This case has been described in another communication.[15] A 23-year old male was admitted on 9.9.69 with a hemiplegia of sudden onset. Clinically there was no abnormality in the heart. Electrocardiogram showed prominent Q waves in L2, L3, AVF with notched inversion of T waves in L2, L3, AVF, V2-V6. X'ray of chest and skull revealed no abnormality. IHA 1 : 512 (1.10.69) 1 : 4096 (9.11.69) 1 : 1456 (22.12.69) 1 : 128 (21.12.70) and -ve (25.5.71) and IFA 1 : 32.

The serological findings and electrocardiographic changes of some members in this family over a period of 21 months are shown in Table 2.

TABLE 2. Serological and electrocardiographic findings in Family J.

Relationship	Serology* IHA	IFA	ECG	Serology** IHA	IFA	ECG
Patient	1 : 4096	ND	Prominent Q waves, notched T waves	-ve	1 : 32	Prominent Q waves, T waves normal
Step-brother	1 : 4374	ND	same changes	1 : 64	1 : 256	same changes
Step-brother	-ve	ND	same changes	ND	ND	same changes
Father	1 : 162	ND	normal	1 : 32	1 : 4	normal
Step-brother	1 : 2	ND	Prominent Q waves, inverted T waves	-ve	1 : 64	Prominent Q waves, inverted T waves

* done at onset of illness (1.10.69)
* 21 months later (25.5.71).
ND not done.

2.1.11. *Family K*

Case K1 : A 48-year old male (SPRW) was admitted with hypertension. He complained of right-sided headache. Examination revealed a blood pressure of 190/95 mm Hg. There was no abnormality in the heart or lungs. The femoral arterial pulsations could not be felt on both sides. No bruit was heard over the abdomen or back. Ocular fundi : Grade 0-1 retinopathy. ESR 22 mm, Blood urea 40 mgm 100 ml, WBC 14,200 mm^3, N 76%, L 20%, E 4%, total proteins 6.7 gm 100 ml, Albumin 4.2 and globulin 2.5, Serum cholesterol 210 mg 100 ml, Latex flocculation test -ve ECG : low voltage complexes with widened and slurred QRS complexes in most leads. X'ray revealed no cardiac enlargement of the cardiac shadow with slight unfolding of the aorta. Arteriogram could not be done as he was sensitive to the dye. IHA 1 : 128, IFA 1 : 256.

2.1.12. *Family L*

Case L1 : A 45-year old male (NGF) was admitted with a history of palpitations of sudden onset. He had had similar epsiodes 2 years earlier. Examination revealed that he had auricular fibrillation and a mid-diastolic murmur, ECG showed auricular fibrillation and ischaemic T wave changes. X'ray showed enlargement of the heart shadow with 'mitralisation'. Screening revealed an enlarged left auricle, right ventricle and a quiet heart. IHA 1 : 256.

Case L2 : Wife aged 35 years had no complaints. Electrocardiogram showed rSR complexes in L3, AVF with fiat T waves. IHA 1 : 64.

3. Results

The study revealed a spectrum of clinical manifestations of varying severity. The following classification of the disease spectrum to include the various clinical forms encountered has been adopted (Table 3).

TABLE 3. Classification.

Acute myocardial involvement
Chronic myocardial involvement
Pericardial involvement, acute, chronic, constrictive
Asymptomatic
i. abnormal ECG, + ve serology
ii. abnormal ECG, — ve serology
iii. normal ECG, + ve serology
Extracardiac-vascular manifestation— 'stroke', arteritis
Familial heart disease
Summation with other heart disease

3.1. Clinical features

3.1.1. *Acute:* There were 3 patients in this group (*A*1, *B*1 and *C* 1). They were all relatively young and were admitted with severe pain in the chest simulating an acute myocardial infarction. Their electrocardiograms showed sinus tachycardia, low electrical complexes, Q waves, ST-T wave changes and bundle branch block. In 2 patients the transaminase levels were elevated. Their cholesterol levels were normal. The significance of their serological titres will be discussed.

3.1.2. *Chronic:* There were 5 patients in this group (*D*1, *E*1, *F*1, *G*1 and *H*1). They were all below 40 years. Two had pain in chest, 4 were in cardiac failure when first seen. Laboratory investigations revealed normal erythrocyte sedimentation rates, normal white cell counts and transaminases. Electrocardiograms revealed low voltage complexes conduction defects, Q waves, ST-T wave changes. The cardiac shadow was enlarged in 4 on X'ray with pulmonary congestion in 3. Their IHA titres ranged from 1 : 64 to 1 : 1024 and IFA 1 : 32 to 1 : 256.

3.1.3. *Pericardial involvement:* There were 2 patients (*C*1 and *I*1) who had pericarditis. Case *I*1 subsequently went on to constrictive pericarditis requiring surgery after an illness lasting 8 months. His ESR was normal repeatedly, Mantoux negative and normal white cell counts. There was a rise in the IFA titre from 1 : 32 to 1 : 4069.

3.1.4. *Asymptomatic:* A study of the family members of patients with myocardial toxoplasmosis revealed that the majority had no clinical symptoms though they showed abnormal electrocardiograms with positive serology, abnormal electrocardiograms with negative serology and normal electrocardiograms with positive serology.

3.1.5 *Extra-cardiac manifestations:* *J1* in this study was a male aged 23 years who was admitted with a stroke. Electrocardiograms showed prominent Q waves in L2, L3, AVF with notched inversion of T. waves in the anterior chest leads. X'ray of the chest showed no abnormality. IHA 1 : 512 and rose to 1 : 4096. In another (*K1*) the femoval arteries were occluded.

3.1.6. *Summation with other heart disease:* Patient (*L1*) who had evidence of chronic rheumatic heart disease on screening had a quiet heart. His wife (*L2*) had a serological titre with minute changes in her electrocardiogram.

3.1.7. *Familial heart disease:* As mentioned earlier, several members had either positive serology with or without electrocardiographic changes. One (*A2*) had repeated still-births and the 7th pregnancy ended in a Caesarian Section. The baby was normal at birth and the placenta revealed no abnormality. Bistologically *A2* had electrocardiographic changes while her sister had a serological titre of 1 : 1024. but a normal electrocardiogram. In Family *B*, the father and mother had a positive titre and electrocardiographic changes and the child born to them had a meningo-myelocele. A study of other patients in this series show that several members had evidence of infection (past or present) together with electrocardiographic abnormalities.

3.2 Electrocardiographic abnormalities

Table 4 shows the frequency of the electrocardigraphic findings. All 12 patients had ECG abnormalities. Of the 27 family members on whom ECG had been done, 11 had normal records. Little more than half had abnormal electrocardiograms with Q waves and T wave changes being most frequently encountered.

TABLE 4. Frequency of electrocardiographic findings.

Findings	Patients	Family members	Total	
	12	26	38	
Normal	0	11	11	
Atrial fibrillation	2	0	2	
Leftbundle branch block	1	0	1	
Intraventricular conduction defect	3	3	6	
Low voltage complexes	3	3	6	
Q waves	7	11	18	
P wave abnormalities	2	0	2	
L.V.H.	1	1	2	
T wave abnormalities				
Notched	1	7	8	22
Biphasic, inverted flat or low	8	6	14	

4. Discussion

In this study much reliance has been placed on the serological findings in the diagnosis. Testing for toxoplasmosis frequently yields positive results. Only a small number of the patients infected manifest the disease. A purely serological diagnosis can only be made in a current infection when 2 samples show a 4-fold rise or fall in titre. When a single specimen shows a titre above that found in the 'normal' person, a presumptive diagnosis may be made. Though only a rise in antibody titre is proof of active infection, it is unusual for this to be seen in toxoplasmosis because by the time a test is done the patient has often developed a steady high antibody titre. Reliance cannot be placed entirely on serological tests alone for a percentage of adult population shows titres depending on past exposure to toxoplasmic infection. However, very high titres have more significance than low ones.

In Sri Lanka, Kulasiri *et al.*[10] in a study of 1069 sera from predominantly male blood donors aged between 17 to 48 years found a titre of the highest frequency of distribution for IHA to be 1 : 128 and for IFA 1 : 64. According to them, if the titres in both tests were below the respective titres of the highest frequency of distribution the infection could be considered to be at its early stages or it had spent itself. If the values were at or above the respective titre of the highest frequency, a further determination a few weeks later is indicated. An increase of at least a 4-fold dilution would indicate a recent infection. Values of both IFA and IHA over 1 : 4096 would indicate acute or convalescent phase.

Though the IHA appears to be deficient in detecting antibodies in the acute stage of the infection, a good qualitative correlation of this test with the dye test has been reported.[20] However, in evaluating the reliability of the dye test in the laboratory diagnosis of toxoplasmosis, De Saram, *et al.*[4] compared two sets of sera and performed the dye test, complement fixation test, haemagglutination test and indirect fluorescent antibody test in all of them. In the 1st set of 100 sera where the illness was established, aetiology other than toxoplasmosis, 21 sera from 15 patients gave a positive dye test ranging from 1 : 16 to 1 : 512. All 100 sera, however, were negative by the other 3 tests. The 2nd set of sera was from 110 patients unrelated to toxoplasmosis but with dye test ranging from 1 : 16 to 1 : 1024. Only 15 of the sera gave a positive reaction with the other 3 tests (CFT 1:8-1:32, IHA 1:8-1:128, IFA 1:16-1:64). Since there was no clinical evidence of toxoplasmosis in them they were assumed to be the result of past infections. The titres they report are very much lower than those found in this study.

According to Ludlam and Beattie[11] high toxoplasmic antibody level can be due to an anamnestic increase brought about by another infection and vice versa. In a case described by Sabin[22] with pneumonitis due to histoplasmosis the toxoplasma dye test rose from 1:16 - 1:1024.

The antibody titres tend to wane with time and be no higher than in the 'normal' population and according to Beattie[1] normal titres do not exclude the diagnosis of toxoplasmosis as is seen in Family *J* where the antibody titres had fallen to normal after 21 months.

Besides the serological titres there was clinical, electrocardiographic and radiological evidence and this together with the results of the family studies are in support of the diagnosis.

Potts and Wilkinson[19] reported an acute fatal case of myocardial toxoplasmosis in a 30-year old male. He had a febrile illness with bronchitis and was seen in acute pulmonary oedema. X'ray revealed moderate cardiac enlargement and electrocardiogram showed left bundle branch block. Patients (*A*1, *B*1and *C*1) in this study simulated myocardial infarction. Such changes in electrocardiogram, however, are known to occur in conditions other than ischaemia. Gau *et al.*[6] in a study showed that Q wave pattern on the electrocardiogram provided insufficient evidence of ischaemic heart disease. In 6 of their 10 patients with congestive cardiomyopathy and 3 of 8 with hypertrophic obstructive cardiomyopathy the electrocardiograms were misleading. Two had elevated transaminase levels. Coltman[3] has drawn attention to the place of SGOT in the diagnosis of myocarditis. The question is whether serological titres imply that their illness was due to toxoplasmic infection or whether they in fact have acute myocardial infarction due to coronary atherosclerosis or both. It could also be surmised that an infection could precipitate an attack of acute myocardial infarction in a person at risk ; for instance it could alter the fibrinolytic activity of the blood thereby causing clotting. There has been a rise in the titre in patients *B*1 and *C*1 and several members of their families had serological titres with electrocardiographic abnormalities.

The more usual picture is that of chronic heart disease.[15] Pericarditis has been observed as part of a generalised infection[9] or as an isolated infection.[7] That toxoplasmosis can lead to constriction is not yet certain[24] but Jones *et al.*[9] reported a case where at pericardiotomy for cardiac tamponade there was pericardial thickening.

Paulley *et al.*[17,18] described 5 cases. One of them was a young man of 24 whose mode of presentation was that of patient *J*1 in this study. His sister too died of an embolus. Toxoplasmosis may be one cause of a 'stroke' in a young adult (Cases *E*1 and *J*1) and of sudden death. Intracardiac thrombi and embolisation are not uncommon in cardiomyopathies. Ready *et al.*[21] suggested that toxoplasma infection may be an aetiological factor in nonspecific aortitis when the aorta and branches may be involved.

Very little is known about the effect of various infections in a patient with pre-existing heart disease. Toxoplasma may be contributing to the 'myocardial factor' associated with other forms of heart disease.

A familial incidence with cardiomegaly, electrocardiographic changes and serological titres in several members is seen at times.[15,17,18] In one of the families studied by Paulley *et al.*[17,18] the mother of the patient and a brother had died at 39 and 16 years respectively and at autopsy the hearts were enlarged. One other sister who had a right bundle branch block on the electrocardiogram died suddenly with embolism. Two other sisters had abnormal electrocardiograms, one with positive, the other negative serology. Similar findings were seen in the families studied by others. Paulley *et al.* [17,18] have stressed that toxoplasmosis should be excluded in all forms of obscure cardiomyopathy especially familial cardiomegaly.

The electrocardiogram shows disturbances of rates, rhythm, conduction defects and abnormalities such as ventricular hypertrophy, Q waves and low electrical complexes. Notched T waves and Q waves were encountered in several patients and members of their families.

Clinical and pathological studies over several years indicate that coronary artery disease in general and myocardial infarction in particular are relatively uncommon in Jamaica. Population studies, however, have shown that the findings of 'ischaemic' ECG changes are common. It is also known that electrocardiographic changes characteristic of infarction can occur in Jamaican cardiomyopathy. Further population studies have revealed that Q waves show an invariably high proportion in which muscle damage is in the anteroseptal region possibly suggesting focal ischaemic necrosis associated with disorder of the small coronary arteries. It was considered possible that some other myocardial disease simulating ischaemia contributed to these epidemiological findings.[25]

More recently, in a reappraisal of cardiovascular surveys in Jamaica by Miller and Ashcroft[12] it was found that many who were thought to have idiopathic cardiomegaly or an obscure form of ischaemic heart disease based on unexplained electrocardiographic abnormalities were subsequently found to have no clinical abnormalities. They were of the opinion that in their study there was no evidence as to whether the electrocardiographic changes were related to ethnic or to environmental factors.

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