Aneurysm of the left ventricle: a report of two cases from Zambia

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Aneurysm of the heart is a rare condition and most cases have been diagnosed after death. Betsch (1945) found 1.5 cases per 1,000 in 7,200 post-mortem examinations. In this paper an indication is given of how an ante-mortem diagnosis may be arrived at.

The commonest cause of aneurysm of the heart in European races is myocardial infarction due to coronary occlusion: according to Sternberg (1914), this accounts for 85% of the cases. As for the incidence of aneurysm in myocardial infarction this varies from 3.5% to 22% in the various series published, as shown in the following table.

Authors	Year	Cases of infarction	Cases of aneurysm
Wartman & Hellerstein	1948	160	35 (22%)
Yater et al.	1948	114	4 (3.5%)
Betsch	1945	141	11 (8%)
Parkinson et al.	1938	Cases from observers	groups of (9%)

Aneurysms not due to myocardial infarction—15% of the total—may be due to a variety of causes: gumma, myocardial necrosis due to rheumatic fever, mycotic coronary arteritis, trauma, and congenital defects (Parkinson et al. 1938).

In African races, however, coronary atheroma is relatively rare, as is myocardial infarction arising from this disease. It is, therefore, not surprising that myocardial infarction is not the commonest cause of aneurysm of the heart.

CASE REPORTS

Patient 1

J.M., a 16-year-old male (village Nyandhana, chief Kanongesha, boma Mwinilunga) was admitted to the Ndola General Hospital on September 8th, 1965. The symptoms were shortness of breath on exertion and relieved by sitting up, cough, abdominal pain, loss of appetite and loss of weight. In the past he had experienced occasional pyrexial episodes but denied any other illness.

Physical examination. Heart rate was 102 per minute and the pulse volume was small and its rate regular. B.P. was 105/90. Jugular venous pressure was slightly raised. Sacral and pedal edema were absent. The outermost cardiac impulse was in the sixth interspace on the anterior axillary line. A pansystolic murmur was present at the mitral area. Crepitations were audible in the chest. The liver was 3 finger breadths enlarged with a positiv hepato-jugular reflux and the spleen was 2 finger breadths palpable. The clinical diagnosis was rheumatic mitral incompetence and congestive cardiac failure.

Investigations. Hb. 11.4G./100ml., WBC 4,000 (neutrophilis 95% lymphacytes 5%), ESR 15 mm., urine negative for protein, sugar and casts, stool negative for ova. The Mantoux test was strongly positive. Chest X-ray showed generalised heart enlargement, pulmonary venous congestion and possibly Kerley B. lines.

Progress. Response to digitalization and mersalyl was satisfactory and the patient was discharged on the ninth hospital day. He was readmitted 10 days after discharge in a moribund state and died soon after admission.

Post Mortem Examination. There was an excess of ascitic fluid and the liver showed a nutmeg appearance. The spleen was enlarged weighing 204 grammes. Both kidneys were small and rubbery and showed a "flea-bitten" appearance.

The heart was greatly enlarged weighing 403 grammes. Both ventricles contained ante-mortem thrombus. The left ventricle was both hypertrophied and dilated. The anterior wall of the left ventricle contained an aneurysmal dilatation commencing just below the commencement of the aorta. The aneurysmal sac was thin walled and was about 3 inches in length. Below the aneurysm was some endocardial thickening. Coronary arteries were normal.

Histologically the myocardium showed patchy fibrosis. The aneurysm contained adherent thrombus and there was no muscle in its wall.

Patient 2

First admission: R.C., an elderly man (village Kashita, chief Kazembe, boma Kawambwa) was admitted to the Ndola General Hospital in March, 1967. He stated that he had been ill since June, 1964 with heaviness in the chest and that recently his abdomen and his feet had started to swell.

Physical Examination: This revealed an elderly man with arcus senilis. B.P. was 140/80. The other physical signs were those of mitral incompetence and biventricular cardiac failure.

Investigations. Haemoglobin 12.2G./100ml., WBC 3,800 (neutrophilis 38%, lymphocytes 58%, monocytes 2%, cosinophils 2%). Stool and urine were normal. Blood urea 12mg.%, electrolytes normal, total serum

proteins 7g% (albumin 3). The chest X-ray showed gross generalised cardiac enlargement, unfolding of the aorta and marked pulmonary congestive changes. The ECG

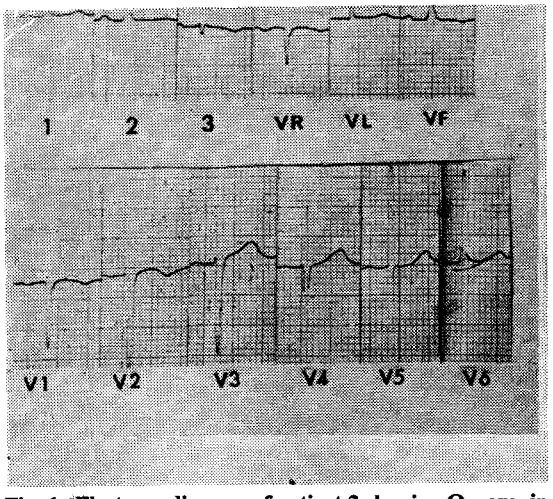

Fig. 1. Electrocardiogram of patient 2 showing Q wave in V4, raised ST in V2 and V3 and T wave inversion in V2. photograph retouched for greater charity.

(Fig. 1) showed sinus, rhythm, a normal axis, a deep narrow Q wave in V4, raised ST segments in V2 and V3, and well marked T wave inversion in V2.

Progress. Response to the standard regime for congestive heart failure was satisfactory and the patient was discharged on the 13th day after admission. The diagnosis was mitral incompetence due either to rheumatic heart disease or cardiomyopathy, probably the former in view of the rapid response to therapy.

A month after discharge the patient was seen again at follow up; there were no signs of congestive failure.

In mid-May 1967 there was a brief admission to the Kitwe Central Hospital because of cough and swelling of abdomen and of feet.

Second Admission. This took place on May 25th, 1967. The signs again were those of mitral incompetence and biventricular failure.

Investigations. Hb. 10.8G/100ml. blood urea 40mg.% electrolytes normal, serum protein 7.3g.%, albumin

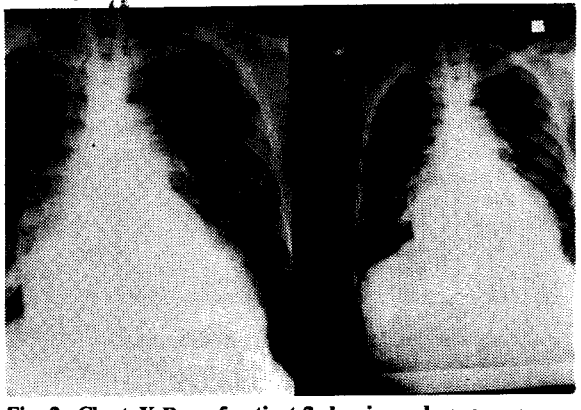

Fig. 2. Chest X-Ray of petient 2 showing pulmonary congestion, very large cardiac shadow and bulge on left border with greater radiolucency (delimited by arrows).

3.2, globulin 4.1, serumbilirubin 2.8mg%, alkaline phosphotase 8.8 King Armstrong units. Chest X-ray (Fig. 2) again showed marked pulmonary congestion and an even larger heart shadow than previously. The left cardiac border showed a large bulge that was more radiolucen than the rest of the cardiac shadow. These signs were, however, only observed on careful perusal after the patient's death.

Progress. Response to therapy was not as rapid as on the first admission and the patient was discharged after a hospital stay lasting two months.

Third Admission. This took place after one month's discharge from the second admission. The patient had lost much weight as a result of avoiding food due to abdominal pain on eating. B.P. was 100/65 and the signs were as previously described with a heaving left ventricular impulse. There was initially a good response to therapy, but death took place suddenly on the 12th day.

Post-mortem Examination. Liver showed signs of chronic venous congestion. The kidneys were normal in size, the capsules stripped easily to reveal a granular surface with some small shallow scars. The pericardium was somewhat thickened with the parietal layer adherent to the heart but without constriction. The heart was enlarged, especially the left ventricle which was dilated without a remarkable degree of hypertrophy. Arising from the left ventricle a little to the left of the interventricular septum and on the left border of the heart was a thin walled sac 4 inches in diameter with its opening half an inch wide: the orifice lay behind the chardae tendineae of the inferior papillary muscle, and the sac contained small adherent clots. Microscopically the sac wall consisted of acellular fibrous tissue. The heart valves were normal, as were the atria. There was a marked degree of aortic atheroma but very little ulceration. The coronary arteries were normal.

Discussion. The probable cause of the aneurysm in the two cases is a congenital diverticulum of the left ventricle. Myocardial infarction can be excluded since the coronary arteries were healthy, and although ECG changes were present in the second case there was no pathological evidence of myocardial disease. There was likewise no evidence of gumma, rheumatic fever, mycotic coronary arteritis, or trauma.

A number of authors have reported African cases of left ventricular aneurysm not due to myocardial ischaemia (Macfie & Ingram, 1920, from Ghana; Robertson & Jackson, 1960, from Nigeria; Abrahams et al., 1962, from Nigeria; and from South Africa, Lurie 1960, and Chester et al. 1965).

Abrahams et al. (1962) described 12 cases and Chester et al. (1965) 6 cases. The former regarded their cases as congenital in origin and the latter were also inclined to this view. Lurie (1960) described 3 of his 4 cases as "idiopathic", and since this term implies the absence of a known disease process it is hard to see how these cases differ from congenital ones.

The anatomical features of the aneurysms in our cases were similar to those found by Agrhams et al.

Continued on Page 163

Aneurysm of the left ventricle

Continued from Page 145

CLINICAL PRESENTATION OF LEFT VENTRICULAR ANEURYSM

and Chester et al., and also to some of Lurie's cases. The aneurysms reported by these authors originated from the left ventricle and arose either below the aortic valve (as in our case 1) or below the mitral valve (as in our case 2).

Both our patients presented with the signs of congestive cardiac failure and mitral incompetence. This combination also appeared in 7 of the cases of Abrahams et al., and in 3 of the cases of Chester et al. The signs of mitral incompetence alone were present in 10 of the cases of Abrahams et al. and in 5 of those of Chester et al. Anginal pain was a prominent symptom in 3 of Chester's cases.

Chester considered that a submitral aneurysm should be suspected in an African subject with mitral incompetence, a localised bulge on the left cardiac border radiologically, and cardiographic evidence of ischaemia. Similarly a sub-aortic aneurysm should be suspected when signs of aortic incompetence and ischaemia occur in the absence of evidence of syphilis and of bacterial endocarditis.

The electro-cardiogram of the second patient of this report is consistent with antero-septal ischaemia.

We have often found difficulty in determining the underlying cause in cases presenting with mitral incompetence. Such difficulty is common where idiopathic cardiomyopathy, endo-myocardial fibrosis and rheumatic heart disease are common W.H.O. Chronicle, 1967). Realisation that left ventricular aneurysms often present with mitral incompetence should be of assistance in this connetion.

SUMMARY

Two cases of aneurysm of the left ventricle occurring in Zambians are described: the probable congenital nature of such aneurysms usually found in the African races is contrasted with the origin from myocardial ischaemia generally found in the European races.

The condition often presents with mitral incompetence and congestive cardiac failure, as it did in the two cases described.

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Notes and News

Election to Fellowship

The entire medical profession in Zambia will join in congratulating Dr. J. C. Davidson, founder and editor of this journal, on his recent election to Fellwoship of the Royal College of Physicians of Edinburgh.

This honour, the highest that the Royal College can bestow, is just recognition for Dr. Davidson's clinical and scientific work in Malawi and Zambia.

M.N.L.

Medical Research Committee

The National Council for Scientific Research has set up various specialist committees one of which is the Medical Research Committee.

Its chairman is Dr. J. C. Davidson (a member of the council) and its members are, Dr. J. Collins, Professor V. Elizarov, Dr. G.H. Fletcher,Dr. C. J. Goosen, Dr. J. H. Kasonde, Professor M. King Dr.C. MacDougall, Mr. E. C. Thomson, and Dr. H. L. Wolfe.

The Committee has already met and is anxious to encourage medical research in Zambia.

A circular has already been sent to all doctors in Zambia asking for information on the following points:

(a) Are you or have you been engaged in research work in Zambia or Central Africa?

(b) If so, what is or was the nature of your research work?

(c) Have you published any papers on this work and, if so where were they published?

(d) In your present or future research work are there any ways in which you think that the National Council for Scientific Research might be of assistance?

Any replies to this circular should be sent to Dr. Gamal Gad, Secretary General of the N.C.S.R.

Special number of the Journal

A special issue of the *Medical Journal of Zambia* will be available in December with the papers presented at the Medical Congress of the Zambia Medical Association held in Kitwe in May this year.