Left ventricular aneurysm of unusual aetiology: report of two cases

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Two young patients aged 14 and 25 with left ventricular aneurysms are described. In both, a clinical picture initiated by attacks of supraventricular tachycardia led to the discovery of the condition.

In the first patient the clinical picture clearly suggested a traumatic aetiology. In the second, the angiographic and histological findings strongly favoured a congenital origin. Both patients underwent successful aneurysmectomy. The literature on the incidence and aetiology of traumatic or congenital ventricular aneurysms is discussed.

Most left ventricular aneurysms (LVA) are due to coronary occlusion (Sternberg, 1914). Other aetiologies that have been described include trauma (French, 1912), rheumatic carditis (Parkinson, Bedford, and Thomson, 1938), syphilitic myocarditis (Sohval, 1935; Aronstein and Neuman, 1941), tuberculosis (Jones and Tilden, 1942), interstitial myocarditis (Beaudet and Boermans, 1947), and mycotic infections (Eichhorn and Garvin, 1943). Burn, Hollander, and Crawford (1943), Clearkin and Bunjé (1955), Björk (1965), and Maloy et al. (1971) described congenital LVA, and Abrahams et al. (1962) reported an unusual type of annular subvalvular LVA in Nigerians which they considered to be of congenital origin. Chesler et al. (1965) described the same condition in the South African Bantu. However, whatever the aetiologies, LVA in young persons is rare. The following is a report of two additional examples of LVA in patients aged 14 and 25, one traumatic and the other most probably of congenital origin.

CASE REPORTS

PATIENT 1 M.S., a 14-year-old boy, was born in Morocco and his family history was non-contributory. Three months before his first admission to hospital on 24 August 1963, he was hit heavily on the left chest with a ball during a football game. He lost consciousness for 15 minutes, recovered spontaneously, and complained only of weakness for the following two days. One month after this episode he became aware of retrosternal pains on effort with radiation to the left shoulder, palpitations, and shortness of breath. Palpitations recurred a number of times, and he was readmitted to hospital. The electrocardiogram (ECG) showed a supraventricular tachycardia which disappeared after treatment with digitalis. One month later he was readmitted to hospital with the same clinical picture but at this time the supraventricular tachycardia responded only to intravenous procaine amide. The plain chest radiograph showed a localized bulge in the left ventricular border (Fig. 1a) which had not been seen on chest films taken before the chest injury. The patient was transferred to our institute for further evaluation.

On physical examination there were no signs of heart failure; examination and palpation of the anterior chest wall were normal, and blood pressure was 120/80 mmHg. The heart sounds were normal and a grade I/VI systolic murmur was heard at the apex. The ECG showed signs of inferolateral myocardial infarction with persistent ST elevation in leads II, III and V5 to V8. The serum aspartate aminotransferase (SGOT) and lactate dehydrogenase (LDH) were within normal

¹Quoted by Clearkin and Bunje (1955)
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FIG. 1. (a) Plain chest radiograph shows a prominent localized bulge of the left heart border. (b) Selective right heart angiocardiology, frontal view, demonstrates a large apical aneurysm.

limits. Right heart catheterization showed normal pressures and oxygenation. Angiocardiography revealed a large apical aneurysm of the left ventricle with paradoxical pulsations (Fig. 1b). The remainder of the ventricular wall showed good contractions. Surgical intervention was advised in order to check the attacks of tachycardia and to prevent further evolution of the lesion.

At open-heart surgery an apical aneurysm, 5–6 cm in size, with fibrotic but very thin walls was found. The aneurysm was resected almost completely except for a small rim which was
consolidated with a Teflon patch. The left anterior
descending coronary artery and the circumflex
artery appeared normal on inspection and on
palpation. The postoperative course was un-
eventful. Histological examination of the wall of
the aneurysm showed fibrous tissue only.

On follow-up examination four months later
the patient's only limitation was with effort;
normal daily activities presented no problems. On
auscultation both a third and a fourth heart
sound were audible with a grade I/VI midsystolic
murmur audible at the apex. The ECG at that
time revealed disappearance of the ST elevation,
the Q waves remaining in leads II, III, and V5
to V8. The chest radiograph again showed a
localized bulge of the left-heart border, however
it was less prominent than before. A repeat
angiocardiogram showed slight dyskinesia of the
papillary wall of the left ventricle.

PATIENT 2 H.D., a 25-year-old man, was born
in Tunisia. At the age of 15 years he had fallen
from his bicycle and sustained a neck wound
which was sutured. Apart from that he was in
good health.

In November 1973 (10 years later) he was ad-
mitted to hospital because of palpitations and
chest pains associated with nausea, vomiting, and
shortness of breath. These symptoms had ap-
peared suddenly while he was playing football.
The blood pressure was 90/70 mmHg. The ECG
showed supraventricular tachycardia with aberrant
ventricular conduction. This reverted to sinus
rhythm during an attempt to introduce an oeso-
ophageal electrode. The plain chest radiograph
showed normal pulmonary markings; the heart
shadow was not enlarged but a curvilinear calcification
was observed at the apex (Fig. 2a). He
was transferred to our institute one month
later for further evaluation. On admission physical
examination was negative. The heart rate was
78/min and regular; blood pressure was
120/70 mmHg. No signs of heart failure were
present. The ECG was normal. The laboratory
data, including enzymes (SGOT, creatine
phosphokinase, LDH) and Casoni and Paul-Bunnel tests,
were normal. Right and left heart catheterization
showed normal pressures and oxygenation. Left
ventriculography showed a 3 × 3 cm aneurysm in
the apical region, connected to the ventricle by an
elongated neck (Fig. 2b). The aneurysmal sac
showed diminished contraction but its neck dis-
played forceful and simultaneous contractions
with the ventricular wall. Selective coronary
arteriography showed normal coronary arteries.
Surgery two months later confirmed the presence
of an aneurysm (3 × 3 cm) connected to the apex
by a muscular neck. The anterior wall of the re-
sected aneurysm was fibrotic, and the posterior
wall was calcified and very stiff with a smooth
inner surface and normally shining endocardium
and trabeculae. The mitral valve and the chordae
tendineae appeared normal. Pathological examina-
tion of the resected specimen showed fibrous tissue
intertwined with muscle fibres and normal
coronary arteries. The postoperative course was
uneventful and the patient was discharged after
10 days in very good condition.

DISCUSSION

Both these patients were young, with a past
history of non-penetrating injury and with
rhythm disturbances in the form of attacks of
supraventricular tachycardia. The aetiology of the
aneurysms was probably traumatic in the first
case and congenital in the second.

In the first patient, the temporal relationship
between the appearance of the aneurysm and
the traumatic episode is convincing. The patient
complained of repeated attacks of palpitation fol-
lowing the injury. Investigation of these led to
the discovery of the aneurysm. The history of this
patient resembles that presented by Cuendet et al.
in 1966 and by Ecoffier et al. in 1969.

In the majority of cases, cardiac damage from
non-penetrating chest injury is neither recognized
nor diagnosed immediately, but is recognized
later by its complications—rhythm disturbances,
tamponade, cardiac failure, or embolic pheno-
mena (Arenberg, 1943; Liedtke and DeMuth,
1973). The frequency of arrhythmias after cardiac
 trauma attests to the fact that the injured heart
is electrically unstable; ventricular fibrillation
may occur at that time even though no gross or micro-
scopic cardiac lesion can be demonstrated
(DeMuth and Zinsser, 1965).

The mechanism of the aneurysm formation
after contusive heart injuries has been elucidated
by the clinical and experimental work of Bright
et al. (quoted by Pupello et al., 1970). After injury
there is a softening and fibrotic replacement of the
contused myocardium. Thinning of this area may
then lead to dilatation and paradoxical systolic
pulsations of a localized area of the left ventricle—
this constitutes a true aneurysm.

The coronary arteries are relatively resistant to
injury by blunt chest trauma. Laceration of these
to vessels is uncommon and thrombosis rare. Of


Left ventricular aneurysm of unusual aetiology: report of two cases

FIG. 2. (a) Plain chest radiograph. A curvilinear calcified rim is seen at the apex (arrows). (b) Selective left ventriculography, right oblique view, shows an apical aneurysm (A) with a narrow neck (between arrows): LV = left ventricle.
546 patients with fatal non-penetrating cardiac injury reported by Parmley, Manion, and Mattingly (1958), only 10 had lacerations of the coronary artery with no findings of thrombosis within a coronary artery despite the frequency of myocardial contusion adjacent to extramural coronary vessels. Very often patients suffering from blunt chest trauma have other injuries which may cause hypovolaemia and shock. It is possible that these factors play a more decisive role in producing the occasionally found coronary thrombosis than direct injury to the coronary vessels (Glancy, Yarnell, and Roberts, 1967).

True aneurysm of the left ventricle consists of remnants of the myocardial wall or its fibrous tissue replacement. Pseudoaneurysm refers to a false myocardial sac, the walls of which are formed by either the pericardium, other adjacent extracardiac fibrotic tissue (Bjornsson, 1964), or both, and which communicates with the ventricular lumen through the site of previous laceration, rupture, or cardiotomy incision (O'Reilly, Kazenelson, and Spellberg, 1970). Without pre-existing fibrous thickening and adhesions of the pericardium sufficient to localize and retain the escaping blood within a restricted area, cardiac tamponade and rapid death would result (Hurst, Fine, and Keyes, 1963). When the escaping blood is encapsulated in a localized area a picture of pseudoaneurysm is produced. In the light of these facts, our first patient can be considered to have had a true aneurysm.

The clinical distinction between true and false aneurysm is difficult (Hurst et al., 1963). Left ventriculography may aid in the differential diagnosis (a narrow neck and the absence of contractions of the aneurysmal bulge would support the presence of a false aneurysm (Killen et al., 1969)). However, the distinction is not of major importance for the surgical approach since it is similar in both instances.

Killen et al. (1969), in a review of the literature, reported only 19 examples of true or false post-traumatic LVA. Until recently no further reports had been published as far as we have been able to determine. Most of the patients reported underwent surgery.

In our second patient there does not seem to be any correlation between the development of symptoms and trauma, and therefore the lesion is likely to be congenital; the neck wound is probably unrelated to the cardiac findings. Furthermore, the histological and radiological findings point to a congenital aetiology; the histological findings showed that the aneurysmal wall consisted of fibrous tissue intertwined with muscle fibres but with the inner wall of the aneurysm layered by a smooth endocardium; normal trabeculae were present. Furthermore, neither vasculitis of any kind nor arteriosclerotic changes were seen in the small coronary vessels. In addition, the left ventriculography findings suggested a congenital aneurysm, since they showed an aneurysmal sac connected to the left ventricle by a narrow neck, both the neck and the aneurysm displaying strong contractions synchronous with the left ventricular contractions. As far as the presence of calcification in the aneurysmal wall is concerned, this is not a specific finding for any of the various aetiologies and is rather a function of time, indicating a long-standing lesion.

The patient resembles the 12-year-old boy reported by Shabetai and Spitz (1966) in whom a calcified left ventricular aneurysm was found. The radiographs showed cardiac enlargement and a U-shaped calcification of the cardiac apex. Angiocardiography revealed a left ventricular aneurysm connected to the apex by a narrow neck.

Only a few examples of congenital ventricular aneurysms have been described in the literature, and even in these there is some doubt as to the correct classification. For example, in some, the presence of Aschoff bodies (Burn et al., 1943) or luetic lesions (Clearkin and Bunjé (1955) makes probable an aetiology other than congenital.

Vivas-Salas (1948) classified congenital aneurysms into three groups:

Group 1 includes cardiac diverticula. A cardiac diverticulum is considered to be a distinct entity and consists of an elongated muscular sac usually connected to the left ventricular apex and commonly associated with omphalocele, diaphragmatic hernia, and cardiac or extracardiac malformations (Ormond, Joffe, and Chesler, 1973; Galioto et al., 1974).

In group 2 the congenital aneurysm is due to a developmental anomaly of the muscle bundles. Burn and associates (1943) described an aneurysm arising from the fibrous band of the mitral ring in a 13-year-old girl. Clearkin and Bunjé (1955), Bertrand and Cooley (1955), and Björk (1965) described similar patients in whom the aneurysms arose from a primary defect in the development of the muscle bundles of the left ventricle. Our second patient can be included in this classification.

The third group is where there is an anomalous origin of the left coronary artery from the pulmonary artery. In this circumstance, inadequate oxygenation of the ventricular wall results in a
ventricular aneurysm with or without papillary muscle dysfunction.

Complications of cardiac aneurysms include any one or more of the following: rhythm disturbances, systemic emboli, congestive heart failure, rupture or cardiac tamponade. In our patients rhythm disturbances dominated the clinical picture and constituted the main indication for surgery.

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