

RUPTURE OF A MYCOTIC ANEURYSM OF THE THORACIC AORTA

BY

S. N. JAVETT and E. KAHN

From the Transvaal Memorial Hospital for Children, Johannesburg

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Aortic aneurysms are rare in childhood. Calvin and Nichamin (1934), in a review of the literature up to 1934 were able to find 44 cases under the age of 18 years. They added two cases of their own. Since this time seven further cases have been reported bringing the total to 53 cases. The patient to be described was first seen 18 months before death and was suffering from streptococcal septicaemia. At that time a radiograph of the chest was normal. The fatal haemoptysis terminating the second admission to hospital occurred from an aortic aneurysm which presumably developed as a consequence of the initial illness.

Case Report

The patient, a girl aged 13 months, was admitted to hospital on October 12, 1948, with a history of feverishness and fretfulness for one month. The day before admission she suddenly became worse, was unable to sit up, manifested pain in the legs when the napkin was changed and ran a high temperature. On examination the child appeared ill. She was irritable, resented handling, and the neck was resistive to flexion. A spotty, purpuric rash was present all over the body, more marked on the buttocks. The temperature was 104° F., pulse rate 150. The heart sounds were closed, and râles were present at both bases. The right knee joint was red, swollen and tender. A presumptive diagnosis of septicaemia with complicating arthritis and meningitis was made.

Laboratory Studies. A blood count on October 12 gave the following results: Haemoglobin, 9.4%; colour index, 0.92; red cells, 3,290,000; white cells, 19,300 (neutrophils 77%, monocytes 2%, lymphocytes 19%, plasma cells 1%, metamyelocytes 1%). The red cells showed anisocytosis and polychromasia. Some polymorphs showed toxic granulation and there was a slight shift to the left.

The cerebrospinal fluid was examined on October 12, and showed 22 polymorphs, 5 lymphocytes and one disintegrated cell per c.mm. No bacteria were detected on direct or cultural examination. Total protein was 35 mg.%, chloride 715 mg.% and sugar 63 mg.%. Three days later the examination was repeated and 2 polymorphs and 2 lymphocytes per c.mm. were present. Occasional erythrocytes were seen. No bacteria were found on direct and cultural

examination. Total protein was 40 mg.%, chlorides 725 mg.% and sugar 60 mg.%.

Blood culture on October 12 recovered a non-haemolytic streptococcus.

Agglutination tests for typhoid and paratyphoid were negative on October 14.

A radiograph of the chest on October 13 (Fig. 1)

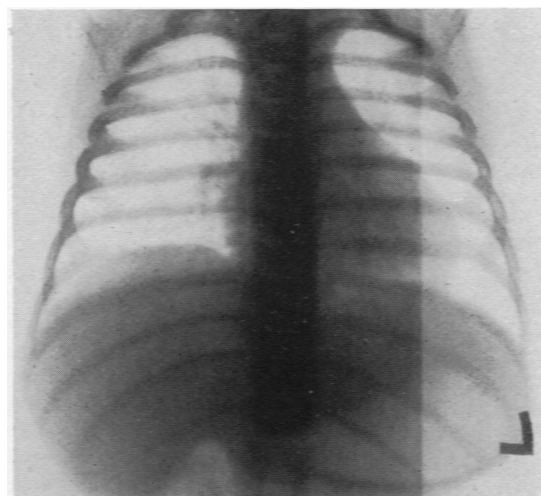


FIG. 1. Radiograph on the first admission showing a normal heart with no evidence of aneurysm.

showed that the lungs and heart were normal. The Mantoux test was negative.

The child was treated with penicillin, 50,000 units intramuscularly every three hours, with one sulphadiazine tablet, at the same time and half a tablet every four hours. Penicillin, 10,000 units, was given intrathecally on October 16.

The temperature subsided within three days and the child was discharged 10 days after admission. The final diagnosis was streptococcal septicaemia.

The child was re-admitted to hospital on April 24, 1950, at the age of 2 years and 7 months, with a history of having coughed up 2-3 oz. of bright red, frothy blood the previous day. She had been in good health since the previous admission except for frequent attacks of 'bronchitis'. The haemoptysis had occurred without warning and was followed a few hours later by a small haematemesis of presumably swallowed blood.

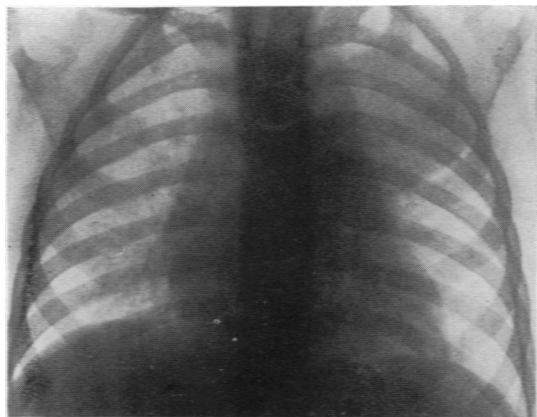


FIG. 2.—Aneurysm of arch of aorta.

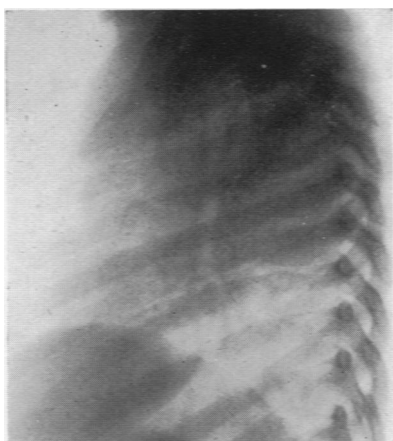


FIG. 3.—Lateral view of aortic aneurysm showing posterior location.

When first seen at the hospital, the child appeared comfortable and not seriously ill. There were loud râles and rhonchi in both sides of the chest. The heart seemed normal, apart from a tachycardia of 140 per minute. However, auscultation was rendered difficult by the loud râles and rhonchi in the chest. The child remained afebrile and asymptomatic. On the morning of April 27, three days after admission, the lungs were clear on auscultation, except for some coarse crepitations at the left apex. In addition, a fairly loud, blowing, systolic murmur was audible widely over the upper part of the chest on the left side, posteriorly as well as anteriorly. The heart sounds were closed at the apex. Radiographs (Figs. 2 and 3) taken on the previous day showed an opacity with a sharply defined lower edge at the apex of the left lung. This opacity had not been present when the child was radiographed in 1948 during her first stay in hospital. The diagnosis of an angiomatous tumour was mooted and fluoroscopic screening was to be carried out the following morning to define the limits of the mass in the left apex more clearly. However, on the same afternoon, again without any warning, the child coughed up 4.5 oz. of bright red blood.

After the haemoptysis the chest was again filled with

coarse crepitations and râles, and the systolic murmur could only be heard anteriorly over the left apex of the lung. On the following morning the child suddenly coughed up a large amount of blood, estimated at several pints, and died within a few minutes.

The following investigations had been carried out after admission: The Mantoux test was negative at 1:1,000. The prothrombin index was 97% of normal. A blood count gave haemoglobin 11.2%; colour index, 0.95; red cells, 3,820,000; total white cells, 19,600 per c.mm.: packed cell volume, 33.5%; sedimentation rate 30 mm. in one hour.

The stools contained cysts of *Giardia intestinalis*.

Necropsy. The upper lobe and the upper portion of the lower lobe of the left lung were bound to the posterior thoracic wall by dense, avascular adhesions. Similar adhesions had obliterated the fissure between the left upper and lower lobes. At the apex of the left upper lobe there were some bullous areas which were filled with blood. The rest of the heart and of the lungs was normal on inspection. In the substance of the apex of the left upper lobe, and extending a little into the apex of the lower lobe, a hard, round mass was palpable. This mass was completely surrounded by lung tissue and the dense adhesions just mentioned which glued together the two lobes of the lung. An incision through the apex into the mass (Fig. 4) showed an aneurysm, 1 in. in diameter, which was found to arise from the convexity of the aortic arch, just distal to the origin of the left subclavian artery, the communication being a clean, round hole 2 mm. in diameter. The interior of the aneurysm in the immediate vicinity of the opening into the aorta was covered by normal endothelium. The opposite wall consisted of concentric layers of avascular, organized clot. One portion of the upper wall, which was covered with fresh, ante-mortem clot, had given way and the aneurysm had ruptured into one of the bronchi of the left upper lobe.

The heart was macroscopically normal. There was no evidence of endocarditis. Microscopic sections of the aorta in the region of the aneurysm showed replacement of muscle and elastic tissue by hyaline fibrous tissue (scar tissue). There were no signs of active inflammation or of rheumatic aortitis. The histological features of the sections were thought to be consistent with the diagnosis of old mycotic aneurysm.

Sections of the heart muscle showed no abnormalities.

Discussion

Our diagnosis of mycotic aneurysm was based on circumstantial evidence. The child suffered from an attack of septicaemia 18 months before death. A non-haemolytic streptococcus was recovered from the blood at that time. It is open to speculation whether this, or some other organism which responded to penicillin and/or sulphadiazine, was responsible for the septicaemia. A radiograph taken at the time did not show any signs of an aneurysm and it is presumed that this developed later. There were no signs of syphilis, either

macroscopically or microscopically, in the aorta and the heart. The aneurysm was not found in the situation of a ductus arteriosus. There were no manifestations of sepsis in the structures adjacent to the aorta, which might have led to its erosion. Finally, there was no sign of endocarditis.

Bronson and Sutherland (1918) quote the following aetiological factors in aneurysm formation in childhood: (1) atheromatous degeneration, (2) trauma, (3) erosion of the aorta from without, (4) congenital malformation, (5) acute infectious diseases. They consider the last group to be the most important one.

Calvin and Nichamin (1934) state that in their series of 44 cases there was 'an association with endocarditis in 10, and with rheumatic fever in 11 instances'. They continue:

'The predominance of rheumatic fever as an etiological factor in the production of aortic aneurysms in the young is thus apparent from a review of the literature, almost half of all the reported cases being in the rheumatic fever group.'

From a perusal of a number of case reports we have been left with considerable doubts whether some of the authors have not labelled their cases 'syphilitic' or 'rheumatic' occasionally with very little justification for such a diagnosis.

In three of the seven cases reported since 1943 the diagnosis seems to be well established. In Nicholson's (1940) case there was a calcified mycotic aneurysm associated with coarctation of the aorta and an arrested subacute bacterial endocarditis. Baer, Taussig and Oppenheimer (1943) demonstrated cystic spaces containing a coagulum in the media of the aorta in parts not affected by the aneurysm. The latter was due to a weakening of the aortic wall by these cystic spaces. The patient was also suffering from arachnodactyly and multiple congenital cardiac defects. Identical lesions in the media of the aorta have been reported in other cases of arachnodactyly. In McLaurin's (1945) case a dissecting aneurysm had originated in an atheromatous patch of the aorta. The kidneys showed evidence of subacute and chronic glomerulo-nephritis and the condition was probably secondary to hypertension.

Mycotic aneurysms 'metastatic' in nature, that is, neither caused by extension from suppurating neighbouring structures nor associated with bacterial



FIG. 4.—Necropsy specimen showing aneurysm 1 in. in diameter.

endocarditis, are very rare indeed. Revell (1945), when discussing this type of aneurysm occurring in any of the arteries and at all ages, was able to list only 23 reports in the medical literature. To these he added a case of his own. The youngest patient was 10½ years old. This type of aneurysm, rare though it is, seems to have a definite predilection for the thoracic aorta. In 11 of the cases the ascending aorta was the seat of the aneurysm. The arch was involved in five cases and the descending aorta in two cases. The abdominal aorta was affected five times and other arteries in the body seven times. In six cases two or more aneurysms were present in the same individual.

Summary

Fatal haemorrhage occurred in a child aged 2 years and 7 months from the rupture of an aneurysm of the aortic arch, probably mycotic in origin, into a bronchus of the left upper lobe of the lung. The aetiology of aortic aneurysm in childhood is discussed. The apparent predilection of mycotic aneurysm for the thoracic aorta is pointed out.

REFERENCES

- Baer, R. W., Taussig, H. B. and Oppenheimer, E. H. (1943). *Bull. Johns Hopk. Hosp.*, 72, 309.
 Bronson, E. and Sutherland, G. A. (1918). *Brit. J. Child. Dis.*, 15, 241. Cited by Calvin and Nichamin.
 Calvin, J. K. and Nichamin, S. J. (1934). *Amer. J. Dis. Child.*, 48, 780.
 McLaurin, J. W. (1945). *New Orleans med. surg. J.*, 97, 317.
 Nicholson, G. H. B. (1940). *Amer. Heart J.*, 20, 357.
 Revell, S. T. R. (1945). *Ann. intern. Med.*, 22, 431.