DISSECTING ANEURYSM WITH SURVIVAL FOR THREE MONTHS AFTER RUPTURE INTO THE PLEURA

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The following case of dissecting aneurysm is recorded because the patient survived for three months after a considerable amount of blood had ruptured into the pleural cavity.

It is hard to write of any aspect of dissecting aneurysm without making use of Shennan's (1934) monumental review of 300 cases. In recent dissecting aneurysms external rupture is much the most common cause of death. Where the intrapericardial aorta was involved (177 cases) rupture took place into the pericardium in 152, into the pleura in 6, and into the mediastinum in 5 cases. Where the aneurysm was completely extrapericardial (41 cases) rupture into the pleural cavity was relatively more common—into the left pleura 13 cases, into the mediastinum and pleura (generally left) 11, into the mediastinum 2, and into the lung 1 case. In the old "healed" dissecting aneurysms (79 cases) heart failure was the commonest cause of death (34 cases) but rupture into one of the cavities occurred 16 times and in some of these it was into the pleura.

The following cases are the only references I have found in Shennan's monograph to haemorrhage into the pleural cavities before the terminal episode, but his paper did not include a large number diagnosed during life. The case of Davy and Gates (1922) lived for 19 days and one of the reasons for the diagnosis was leakage of blood into the left pleura. The case of Barton (1930) lived for 17 days and fluid began to collect in the left pleura during the first day.

A woman was admitted to Guy's Hospital having collapsed after very severe pain in the chest and back with signs of fluid in the left chest. For ten years she had had occasional faints and for five years some pain on the left side of the chest, not clearly related to exercise. In September 1944 she had a right-sided hemiplegia from which she made a good recovery in a month; in December she had a similar attack, this time on the left side, and again recovered in the course of two months except for some slight residual signs.

On April 10, 1945, when 66, she was sitting on London Bridge Station waiting for a train when she was seized with agonizing pain in the centre of the chest and back. This was followed by breathlessness and collapse and she did not remember much about her admission to hospital. The blood pressure had fallen to 120/100 but soon rose to 220/130 and remained about this level after the first two days. The severest pain quickly subsided after morphia (1/4 grain and the same amount repeated) but she continued to have pain, especially in the back and needed a small dose of dilaudid (1/16 grain) most nights for some weeks. Her haemoglobin had fallen to 55 per cent and there was a leucocytosis of 15,000 with a normal differential count.

Cardiac infarction was suspected but the cardiogram only showed slight inversion of T I and there was no significant change in this a week later. She was found to have a left-sided pleural effusion (Fig. 1A) and a week later aspiration showed almost pure blood, not merely a blood-stained effusion.

At this stage I was asked to see her and on these findings (with a loss of 3 stone during the previous three years) thought she must have a growth of the lung, possibly with a secondary deposit causing cardiac infarction, although the past cerebral history favoured primary cardiovascular disease and without the pleural effusion would have been taken as supporting cardiac infarction in spite of the absence of cardiographic support.
Progress. She made slow but steady progress although there was no significant change in the physical signs in the left lung. Five radiograms of the chest showed no great difference. The "aortic" shadow was wide but this was accepted as part of her atherosclerosis. After her death re-examination showed that the width had increased slightly from 8·5 to 9·5 cm.

After two months in bed she was allowed up and after another month was looking forward to going home. On July 10 she went to the lavatory and while there called and said she felt faint: her pain was not very severe but she collapsed and was carried back to bed. She became unconscious within a few minutes and died in half an hour.

Post-mortem examination. This was carried out by Dr. Hopkins to whom I am indebted for the specimen illustrated but the weights and full details of the other viscera were lost.

The brain was not examined. The kidneys were rather small and granular. The heart showed moderate hypertrophy of the left ventricle. The aortic valves showed some atheroma and calcification. The ascending aorta and arch showed slight atheroma only.

In the arch of the aorta, about midway between the origin of the subclavian artery and the level of the pulmonary artery there was a recent tear of the aorta about 3·5 cm. long, with an escape of blood into the lung and pleural cavity. This recent tear and the large amount of recent clot made it difficult to be sure if there had been an original tear here with rupture through to the lung and pleural cavity and downward dissection, or if the original leakage into the lung at this point had come from the lower tear (see later) with retrograde dissection back to this level.

Below this tear for at least 15 cm. there was an extensive dissection with stratified layers of blood clot, partially organized and looking of some months duration (i.e. probably from the time of the collapse and hæmorrhax three months before). For 4 cm. below the tear (A to B, Fig. 2) there was a wider recent blood clot with a narrow tongue projecting a further 2 cm. (B to C), both on the inner side of the laminated clot, showing that the dissection had increased just before her death.

9 cm. below the fatal tear there was an old transverse tear 1·5 cm. long. The dissection extended below this and the stratified clot at this point looked at least as old as, and probably older than, anywhere else. This was certainly an old tear but it was not certain if it was the point of re-entry (though not the lowest point of the dissection) or, as seemed more likely, if it was the original tear causing the dissection that had spread upwards (as well as downwards) and had ruptured into the lungs and pleura near the point where the final tear had caused death. If this was so, it might explain why the second rupture into the pleural cavity had
caused death so quickly while the first had not. This uncertainty makes the case of little value except as an example of unusually long survival after a large hæmothorax had developed from a dissecting aneurysm.

**SUMMARY**

A case of dissecting aneurysm is described where the patient lived for three months with a large hæmothorax from the original tear and dissection of the aorta. She died suddenly from a second tear (also with some dissection) with rupture into the pleural cavity.

**REFERENCE**