CASE REPORTS

THROMBOSIS OF THE INNOMINATE ARTERY

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Cases of thrombosis of the systemic arterial circulation have been reported from time to time. Various causes have been quoted and Allen *et al.* (1946) in their work on peripheral vascular diseases speak of inflammatory, degenerative, traumatic, and simple causes but there are also a number of cases of unknown ætiology. Aggeler *et al.* (1941) reported a case in which there was complete absence of pulsation in the carotid arteries and in the arteries of both upper extremities. There was no evidence of syphilis as had previously been reported with this rare syndrome. Apart from an increased platelet count, no abnormalities were demonstrated in the bleeding and clotting mechanism of this patient: it was concluded that thrombocytosis and autohæmagglutination may have been contributory factors. McCombs and McElroy (1937) reported a case of autohæmagglutination due to a cold agglutinin in a patient with peripheral vascular symptoms. Another interesting condition was reported by Nygaard and Brown (1937) as "essential thrombophilia," in which sudden widespread occlusion of major arteries and veins occurred with marked signs of circulatory insufficiency often resulting in gangrene. The histopathological changes in the involved vessels were insignificant. They also observed variations in the plasma coagulation time in some of their patients. Thrombosis seems to involve the arteries of the lower extremities more frequently than those of the upper part of the body and thrombus formation in the innominate artery seems to be especially rare. The following case with its remarkably long history may therefore be of some interest.

Case Report

A lorry driver, aged 46, was admitted to St. Andrew's Hospital on November 5, 1951, with very severe pain in his right arm which had started two days previously. On the morning of admission he had complained of dimness of vision and spots before his eyes. In the afternoon, after getting up, he fell forwards, was helped into a chair and appeared to be confused. His left arm, previously not affected, was now immobile and he was sweating profusely. His wife noticed a transient left facial weakness. The patient also complained of severe headache and loss of feeling and weakness in his left arm. He could neither walk nor stand up.

Previous history. There had been increasing pain in his right arm for about one year with weakness and limitation of movement due to the pain. He also noticed that his left arm went red in warm water but that his right arm remained white. A few months before admission his doctor found that there was weakness, coldness, and severe pain in his right arm as well as limitation of movement so that two weeks before admission he had to give up work. There was also a previous history of acute bronchitis, of a lump in his testicle two years before which disappeared on its own, and a shorter history of frequency of micturition and polyuria. The patient had been in the army and classified A1.

On examination. The patient was sweating and complaining of agonizing pain in his right arm. The heart sounds were normal and there were no signs of congestion. Apart from some scattered rhonchi, the lungs did not show any abnormalities. The tongue was normal and there were no abnormal signs in the abdomen.

In the nervous system the pupils were central, of equal size and shape, and extremely dilated. The right pupil reacted sluggishly to light and accommodation while the left pupil was immobile. The patient had some vision in his right eye; the left eye had light perception only and there appeared to be left hemianopia. Extra-ocular movements were normal and no papilloedema or retinal exudates or hæmorrhages were present. The other cranial nerves appeared normal. There was loss of power and decreased tonus in the left leg; the left plantar response was extensor but the other reflexes were normal. No sensory abnormality was found, and the right leg showed no neurological abnormality.

The signs in the arms could be contrasted thus:

Right arm

Some cyanosis. Cold to the touch. Marked loss of power. Very poor grip in hand. Normal pulsation of axillary artery. No brachial artery pulsation and no radial pulse felt. Agonizing pain. Blood pressure not recordable.

Left arm

Normal temperature and colour. Slight spasticity. Poor grip in hand. Normal pulsation of axillary, brachial, and radial arteries. B.P. 210/110.

Further progress and treatment. The excruciating pain in the patient's right arm was much improved by administration of morphia, at first given intravenously and then subcutaneously, and the pain had much diminished on the day after admission. He was put on priscol 25 mg. thrice daily, and noticed soon after "pins and needles" in the fingers of his right hand. There was no change in any of the physical signs. Two days after admission, he complained of headache and was very drowsy and stuporous. There was no aphasia. His right arm seemed to be warmer than before. He was sweating and the temperature rose to 99° F. on two occasions. There was restriction of medial movements of the left eye and a left hemiparesis. The pupils were large and equal and reacted to light. There was left hemianopia and left facial weakness. Co-operation was rather poor and the patient was now hiccupping. No neck stiffness could be detected. Later in the day he became more comatose but he still responded to painful stimuli. His pulse rate was 100 and his temperature 100° F. The pupils were equal and the right reacted sluggishly to light. Both eyes deviated now to the right side, the right eye more than the left. The blood pressure in the left arm had fallen to 140/70. Administration of penicillin was started.

Investigations. E.S.R., 11 mm. in 1 hr.; Blood urea, 47 mg. per 100 ml. W.B.C., 20,000 (Polym., 83%; Lymphocytes, 12%; Monocytes, 5%); Urine contained no albumin or sugar. X-ray of chest; no evidence of aneurysm or of calcification of the aorta. Heart not enlarged.

Spinal fluid (7/11/51). Pressure 80 mm.; no block; clear colourless fluid. W.B.C., 1; sugar 80 mg. per 100 ml.; protein, 65 mg. per 100 ml.; chlorides, 770 mg. per 100 ml.; globulin, faint excess; culture sterile., W.R., Lange and Kahn, negative.

X-Ray of shoulder showed no evidence of calcification of arterial trunk, but arthritic changes in right acromio-clavicular articulation.

The condition of the patient deteriorated and he died three days after admission.

Post-mortem examination (Dr. Donald Teare). Well nourished adult man. Skeletal system normal.

Circulatory system. The right side of the heart was very widely dilated. The left ventricle was normal in size. The myocardium was quite healthy. There was no valvular disease. The foramen ovale was closed. Atheromatous changes were present in the coronary arteries and the aorta, but not very marked. Thrombosis had started in the innominate artery with a large thrombus attached to it at one point. The tail of the thrombus which was free, lay in the right carotid artery and the right internal carotid artery was also completely blocked. The entire right cerebral hemisphere showed very intensive softening.

The other systems showed no significant changes.

Microscopical examination (Dr. Levene). Microscopically the section showed a large thrombus within the atheromatous artery. The thrombus was undergoing organization as was shown by the outgrowth from the artery of fibroblasts and capillaries. At the base of the thrombus was gross fatty change, which was also present in a marked degree in the intima of the underlying artery. One of the notable features was the large number of polymorphs in the intima of the artery and the base of the thrombus (Fig. 1).

Discussion

On admission a tentative diagnosis of a dissecting aneurysm of the thoracic aorta had been made. Syphilitic aortitis was very unlikely as the W.R., Kahn and Lange tests were negative. There was also no history of chest pain, dyspnoea, or hypertension as described by Aggeler *et al.* (1941). Periarteritis nodosa, thromboangiitis obliterans, and giant cell arteritis were unlikely and

FIG. 1.—Large organizing thrombus developing in an atheromatous innominate artery. Gross fatty change at the base of the thrombus is also present in the intima of the underlying artery. Large numbers of polymorphs are present in the intima of the vessel and at the base of the thrombus.

none of these conditions were found after death. The absence of gangrene in the right arm in spite of the long history suggested that there was at no time complete occlusion of the axillary artery in which pulsation could be readily felt. No evidence of trauma was given in the history. Whether thrombocytosis was present cannot now be determined as no platelet counts were done at the time. The slight rise of the sedimentation rate, and blood urea, and the polymorphonuclear leucocytosis as well as the small rise in the protein and globulin of the C.S.F. could be attributed to the terminal illness. The neurological signs were due to the complete occlusion of the internal carotid artery with associated intensive softening of the entire right cerebral hemisphere. It seems likely that this was a case of thrombus formation in an atheromatous vessel due to infection although no cause of such infection could be found in the clinical history or in any of the laboratory investigations.

Summary

A case of thrombosis of the innominate artery with extension of the thrombus into the right common and internal carotid arteries together with intensive softening of the right cerebral hemisphere has been described.

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