Case Reports

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Multiple Emboli from Left Ventricular Myxoma

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We recently encountered a patient who, over a 2-year period, suffered repeated, mainly cerebral, embolism. At necropsy a left ventricular myxoma was discovered. The rarity of this condition prompted the following case report.

Case Report

A 25-year-old man was admitted on June 17, 1967, with a right hemiplegia of sudden onset. Three episodes of pain, swelling, and red blotchy discoloration of both feet and legs, each lasting 2 to 4 weeks, had occurred over the preceding year. During this period, he noticed transient burning and tingling in his fingers.

He was apyrexial and normotensive; pulse rate 90/min.; atrial fibrillation was evident. Patchy reddish-blue discoloration of both feet and toes was noted, and the right fifth toe was cyanotic. The right foot was colder than the left, with diminished pulsation of the right dorsalis pedis artery. Apart from irregularity, auscultation of the heart was normal.

The patient was drowsy and restless, and moderate motor dysphasia was present. There was a right homonymous hemianopia with impairment of conjugate gaze to the right, a marked right lower facial weakness, a right spastic hemiplegia with right-sided hyperreflexia, right extensor plantar response, absence of the abdominal and cremasteric reflexes on that side, and a right hemihypalgesia.

Relevant investigations were as follows: Hb 15.7 g./100 ml., haematocrit 48 per cent, white cell count 12,300, 92 per cent polymorphs, 8 per cent lymphocytes, platelet count 209,000/cu.mm. The erythrocyte sedimentation rate was repeatedly normal except on one occasion (38 mm./hr.). Serum lactic dehydrogenase 670 U (normal less than 500 U). Four blood cultures were sterile. X-rays of the chest and skull were normal. The cerebrospinal fluid was normal in pressure and content.

The electrocardiogram on admission confirmed atrial fibrillation. The following day it showed sinus rhythm with first degree heart block (P-R interval 0.22 sec.). Occasional dropped beats were noted, due at times to sino-atrial, at other times to atrioventricular block. All subsequent electrocardiograms were normal.

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On the third hospital day, a brain-stem episode occurred, evidenced by the sudden onset of bilateral ptosis, limitation of upward gaze, an almost complete right facial palsy, and deviation of the tongue to the right. These signs resolved in three days. Anticoagulation was started.

On the eighth day, further evidence of peripheral embolism appeared. The right foot and the index and ring fingers of the left hand became cyanotic, and several subungual splinter haemorrhages were observed. On several occasions since admission, a temperature up to 37.4°C. was recorded. Despite negative blood cultures and absence of cardiac murmurs, the patient was started on intravenous penicillin 20 mega-units/day and intramuscular streptomycin 1 g./day. The former was continued for 6, the latter for 4 weeks. Thereafter his temperature stayed normal.

Over the next week, recurrent cyanotic discoloration of fingers and toes occurred. This usually appeared suddenly, often involving only a longitudinal half of a digit at a time, and resolved in several days.

On the fifteenth day, a sudden attack of generalized cyanosis and transient apnoea supervened, and was followed by dyspnoea lasting several hours. There was now deterioration of his conscious state, coarse nystagmus to the left, and a left Horner's syndrome. Intravenous heparin was substituted for phenindione, and the fresh neurological signs gradually regressed over two days.

After this stormy course, no further embolic episodes occurred, and anticoagulation was maintained by oral phenindione. Two months after admission right cardiac catheterization was performed. The following pressures were obtained: right atrium 0 (mean), right ventricle 14/0 mm. Hg, pulmonary artery 10/2 mm. Hg, pulmonary wedge 0 (mean). The cardiac output was estimated at 4.2 l./min. Right pulmonary cine-angiography showed no abnormality in the pulmonary vessels or left atrium. The patient was discharged on August 22, 1967, with residual right hemiparesis. He was able to walk with the aid of a tripod and still had a slight motor dysphasia. Anticoagulants were discontinued.

Over the following 8 months, he made some functional recovery and was able to walk unaided. Speech had virtually returned to normal. In October and December 1967, and April 1968, major non-focal fits

occurred, and after the second he was started on diphenylhydantoin 100 mg. three times a day. He was readmitted on May 25, 1968, in status epilepticus, which responded satisfactorily to intravenous diazepam. On the day of admission, frequent isolated right-sided fits were witnessed but subsequently these were completely controlled by intramuscular diphenylhydantoin.

He was unconscious and Cheyne-Stokes breathing was evident. A soft mid-systolic murmur was heard over the apex and along the left sternal edge. The right fundus was normal; the left showed a pale disc and white intraluminal material segmented along the course of the retinal arterioles. The right pupil measured 3 mm. in diameter and reacted readily to light. The left was dilated and unreactive. Both eyes were turned to the right, but deviation to the other side was complete on passive head turning. Upward eye movement was obtained bilaterally on corneal stimulation. Spasticity and hyperreflexion involved all limbs, being more conspicuous on the right side. The plantar responses were bilaterally extensor. Painful stimuli produced a decerebrate posture on the right, but flexion of the limbs on the left.

Intravenous heparin was started and continued throughout. No improvement occurred in the neurological state, and the clinical course was complicated by bronchopneumonia which was treated with ampicillin and chloramphenicol. The patient died in shock on June 7, 1968, 13 days after admission.

Laboratory investigations this time revealed no additional abnormalities. The erythrocyte sedimentation, serum proteins, and electrophoresis remained normal.

Pathological Findings (Dr. C. R. Knappett). The necropsy was performed four hours after death. The skull and meninges were normal. The brain weighed 1230 g. Several large old depressed infarcts were present in the left middle cerebral artery territory. In the right hemisphere, a more recent pigmented infarct was seen on the inferior surface of the temporal lobe, and a recent haemorrhagic infarct on the upper surface of the parietal lobe. The cerebral arteries, and the great vessels in the neck, appeared normal.

The heart weighed 380 g. The left ventricle showed slight mural thickening. Both atria, their appendages, and heart valves were normal. From the posterior wall of the left ventricle, a red and yellow, smooth, multilobulate tumour, 3.5 cm. in diameter, was present, arising from the surface by a short pedicle 3 mm. in diameter (Fig.). Several discrete fibrous scars were present in the wall of the left ventricle, measuring up to 1 cm. in diameter, none being situated in the immediate vicinity of the tumour site. The coronary arteries and their ostia were normal. Several pale shrunken cortical infarcts were present in both kidneys. No infarcts were seen in the spleen. Recent haemorrhage had occurred into the substance of both suprarenal glands and the surrounding connective tissue. Changes associated with a terminal bronchopneumonia were present in both lungs.

Histologically, the tumour showed the typical appearances of an intracardiac myxoma. Sections of the myocardial scars showed localized areas of replacement fibrosis. No myxomatous emboli were observed. The small arterioles were normal in appearance. Section of

FIG.—Interior of the left ventricle showing the myxoma *in situ* below the aortic ring.

myocardium below the origin of the tumour did not show fibrosis. It was considered that the most probable explanation of the myocardial scarring was coronary embolism occurring secondarily to the ventricular myxoma. The heart muscle showed no other abnormality.

Discussion

The salient features of six previously reported cases of left ventricular myxoma are summarized in the Table.

Normal catheter and cine-angiographic findings excluded a left atrial myxoma. The possibility of a ventricular myxoma was not appreciated at the time. A review of the cine-angiogram showed only a small portion of the left ventricular outflow tract and no tumour was seen. It seems, therefore, that when a myxoma is suspected, the left ventricle should receive as close a scrutiny as the left atrium, and retrograde left ventriculography is essential before a myxoma can be excluded with certainty.

During the two symptomatic years embolization was intermittent, suggesting periodic release of fragments of myxoma. The case reported by Kay *et al.* (1959) also showed intermittency of embolization over 17 years. Björk and Björk (1965) observed that after embolization, symptoms of left ventricular outflow tract obstruction diminished, suggesting decrease in the size of the myxoma due to tumour detachment.

Obstructive symptoms which occurred in our patient on the fifteenth hospital day of his first admission were associated with embolization, but

thereafter no further emboli occurred for 9 months.

The small number of reported cases of left ventricular myxoma limits useful comparison with left atrial myxoma.

In both, female sex predominance is evident. Whereas left atrial myxoma usually presents between 30 and 60, left ventricular tumours tend to present earlier. Embolization and obstructive symptoms are common to myxomas at both sites, but constitutional symptoms appear to be less common in left ventricular myxoma: a raised erythrocyte sedimentation rate was recorded in only 1 of the 6 reported cases; in our patient with one exception the sedimentation rate continually fell within the normal range. The validity of these observations will need to be confirmed in the light of further reports of this uncommon condition.

Summary

The case history of a 25-year-old man presenting with systemic embolization intermittently over a period of 2 years is described. A left ventricular myxoma was found at necropsy. During life, the possibility of a left atrial myxoma was considered and excluded. The importance of extending the studies to include the left ventricle is stressed.

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TABLE
REPORTED CASES OF LEFT VENTRICULAR MYXOMA

Reference	Age at onset of symptoms (yr.)	Sex	Duration of symptoms	Site of emboli	Obstructive symptoms	Diagnosis established	Outcome
Young and Hunter (1947)	10	F	1 wk.	Aorta; renal; ?cerebral	Absent	Necropsy	Undiagnosed at death
Fayen and Baglio (1959)	45 at death	F	None	None	Absent	Necropsy	Incidental necropsy finding
Kay <i>et al.</i> (1959)	15	F	17 yr.	Aorta (5); cerebral; multiple peripheral	Absent	Thoracotomy	Successful removal
Thomas <i>et al.</i> (1963)	14	F	6 mth.	None	Present	Retrograde left ventriculography	Post-operative death
Björk and Björk (1965)	47	F	5 yr.	Aorta	Present	Microscopy of embolus; pulmonary artery angiography; retrograde left ventriculography	Successful removal
de Paiva <i>et al.</i> (1967)	14	M	2 yr.	Aorta; femoral; renal; cerebral	Present	Retrograde left ventriculography	Post-operative death
Present case	24	M	2 yr.	Cerebral; renal; multiple peripheral ? cardiac	Present	Necropsy	Undiagnosed at death

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